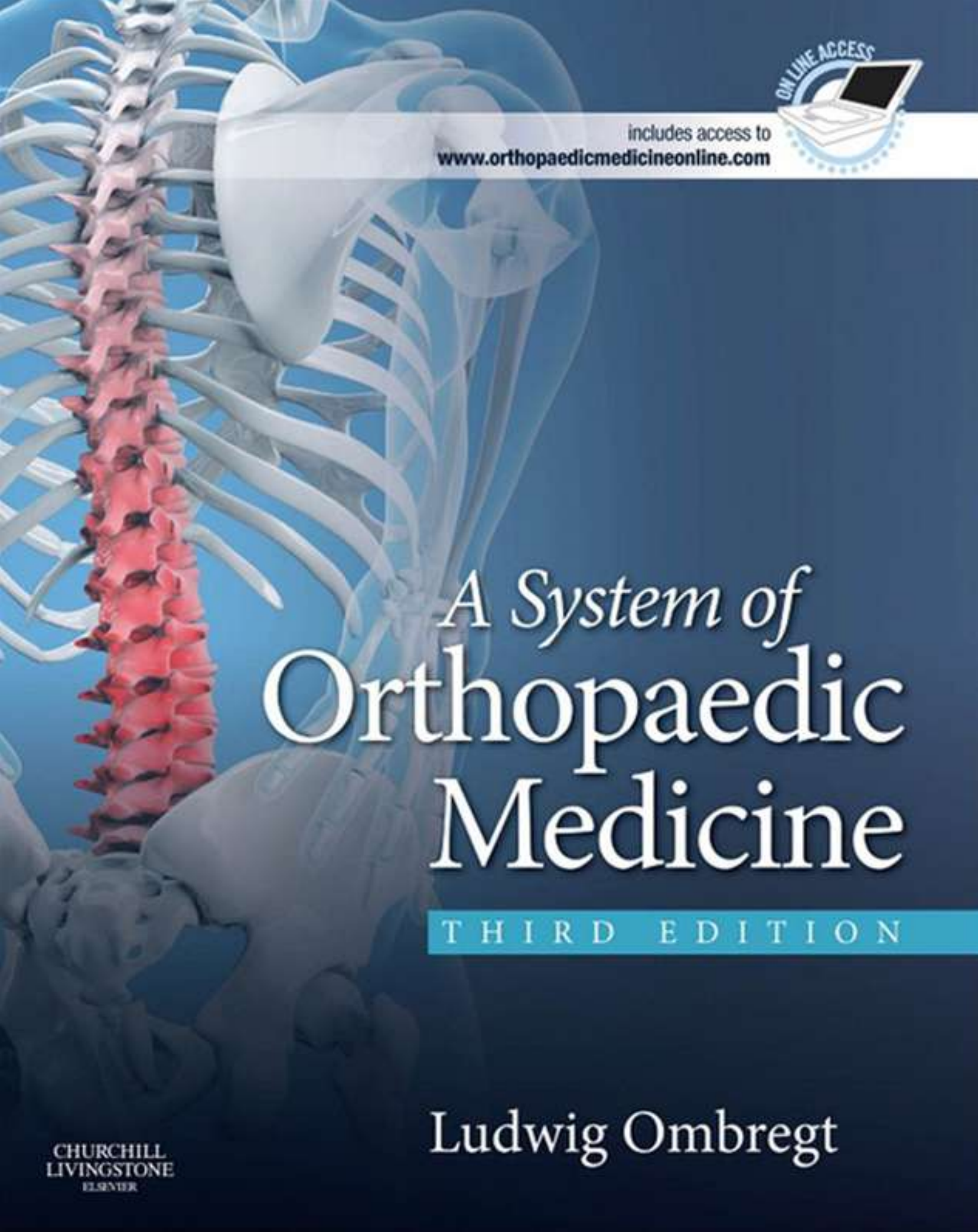

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A System of Orthopaedic Medicine

THIRD EDITION

Ludwig Ombregt

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A System of Orthopaedic Medicine

For Elsevier

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THIRD EDITION

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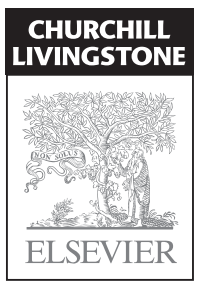

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Preface to the third edition

Since its first publication, almost two decades ago, *A System of Orthopaedic Medicine* has proven to be a reliable guide and a dependable working tool for many physicians and therapists working in the field of orthopaedic medicine. During these two decades we have witnessed a quite astonishing advance in technology that has made it possible to see even the slightest anatomical changes. Computed tomography, magnetic resonance imaging, sonography and arthroscopy have changed the practice of medicine to an unbelievable degree. So have new surgical techniques that address the restoration of anatomical defects. Despite the great progress that these procedures represent, they are not an unmixed blessing. There is an almost irresistible tendency to overuse them. This not only plays an important part in escalating the cost of medical practice, but may also result in an unnecessary hazard to the health of the patient. Another danger of the booming technology is that physicians who rely too much on easy-to-order tests are in danger of losing their clinical ability to diagnose and think. In the end, looking substitutes for thinking, which is not without danger if one considers the huge number of asymptomatic anatomical changes that exist in a normal population.

The outline of this third edition remains unchanged. The book still focuses mainly on clinical reasoning and diagnosis to counterbalance the massive present-day emphasis that is given to technical investigation. The 'System' relies first of all on a systematic clinical assessment of the joints involved. Interpretation of the results obtained then groups the disorders and conditions into clinical syndromes. Finally, the natural history and the conservative treatment of each condition are discussed. For this third edition, each chapter has been revised completely and the references fully adapted. This new edition also contains a huge online resource that consists of (a) Over 100 video clips of examination and treatment techniques, (b) all the references with access to the abstracts on Medline, (c) all the applied anatomy chapters, illustrated with artwork from Elsevier anatomy books, and (d) less important chapters taken out from the book.

Ludwig Ombregt
2013

Preface to the first edition

This book is based entirely on the Cyriax approach to orthopaedic medicine. James Cyriax (1904–1985), the 'father' of orthopaedic medicine, devoted his whole life to developing a logical system of clinical examination designed to elicit accurate diagnoses of locomotor disorders. His system is founded on a few elementary theories: *referred pain* and *selective tension* – an injured structure hurts when tension is put on its fibres. A detailed clinical examination using these simple principles outlines clinical patterns that can be interpreted on the basis of the known facts of applied anatomy and physiology. Logical conclusions can then be drawn regarding diagnosis, treatment and spontaneous recovery.

The authors of this book were all trained by Dr Cyriax and subsequently taught with him until his death in 1985. Ever since, they have continued his work, and as far as possible improved it. The fact that they are all practitioners, using the system in their daily clinical work, has helped to add considerable complementary clinical experience.

Orthopaedic medicine has traditionally been a grossly neglected area of medical education, despite the large number of patients suffering from non-surgical orthopaedic conditions. Any family practitioner will confirm that between a fifth and a quarter of his/her work consists of patients complaining of musculoskeletal pain. This combination of inadequate education and a high incidence of disorders leads to a large number of patients for whom there is no clear diagnosis nor proper treatment. Chronicity, neurosis and compensation claims are, then, in many cases, inevitable consequences.

In recent years another trend has had a major impact on orthopaedic medicine. Outstanding advances such as computed axial tomography (CAT), magnetic resonance imaging (MRI), arthroscopy and echography have undoubtedly enhanced the clinician's ability to see detailed anatomical images and structures. However, these procedures have brought mixed blessings. Too often they are employed unquestioningly before a good clinical diagnosis has been established and may contribute needlessly to the escalating costs of health care. More importantly, the 'lesions' these investigations reveal are often not the real cause of the problem and may seriously mislead the examiner, resulting in either inappropriate or potentially harmful treatment for the patient. Technology has become, in too many cases, a substitute for clinical skills, and the ability to diagnose and think remains poor.

An accurate clinical diagnosis is the first and most important obligation for every orthopaedic doctor and therapist. Since these skills are not formally taught, the purpose of this book is to provide a systematic approach to clinical examination which can lead to accurate diagnosis. The method described is logical and consists of assessing in turn the function of each moving tissue, the positive and negative responses forming a pattern. These patterns are then translated into clinical syndromes. The system relies entirely on basic clinical skills and requires none of the diagnostic apparatus found only in hospitals. For this reason, it will be of particular value to family doctors, physiotherapists, rheumatologists, orthopaedic surgeons and particularly those working in sports medicine. Especial emphasis is given to differential diagnosis. Warning signs are highlighted to alert both examiner and therapist to possible pitfalls and therapeutic dangers.

Most of the treatments described were devised by Cyriax. They are: injections, infiltrations and deep transverse friction for muscular, tendinous and ligamentous lesions; manipulation for some ligamentous disorders and for reducing displaced fragments of cartilage in the different joints; traction for some types of lumbar disorders and capsular stretching for some capsular disorders. Although there may be many alternative equally effective treatments practised by others, the authors have confined themselves to those with which they have considerable personal experience. The techniques described here are simple and have, through time, confirmed their effectiveness. For any treatment to be successful, accurate diagnosis must be followed by carefully focused therapy to the affected area. For this reason, considerable detail is given on the correct performance of each therapeutic technique, supported by numerous clear, explanatory photographs and diagrams.

Finally we wish to stress the importance of close cooperation between physician and (physio)therapist. The system of orthopaedic medicine promoted in this book requires teamwork. Both doctor and therapist examine the patient in the same way, speak the same language and share their assessments and diagnoses. As to treatment, some disorders will respond better to physical treatment and others will require a medical approach, but both professions need to be fully aware of the possibilities, modalities and potential results.

Ludwig Ombregt
Pierre Bisschop
Herman J. ter Veer
Tony Van de Velde
1995

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1

Pain

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Definition of pain

Pain is the presenting symptom in almost every orthopaedic patient. A complaint of pain is always indicative of some variety or degree of dysfunction[1] and results from a combination of physical and psychological causes, although sometimes one or the other predominates. All pain must be regarded as real. Pain entirely devoid of somatic cause is labelled 'psychogenic pain': although no peripheral tissue damage exists, the pain is just as distressing as somatic pain[2] (see Section 16).

The taxonomy committee of the International Association for the Study of Pain defined pain as: 'an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage'.[3] Pain is thus not a 'primary sensation' in the sense that smell, taste, touch, vision and hearing are, but is an 'emotional state', like sorrow, love or hate. The consequence is that it is extremely difficult to explain one's pain to another person. This is reflected in the numerous words that patients use to describe intensity and quality of pain: twinge, ache, distress, discomfort, soreness, cramp, suffering, misery, agony, torment, anguish.[4] The fact that pain is always a subjective experience provides the first difficulty in its use in diagnosis. The language used is not always easy to understand, and the examiner usually needs a high level of competence and understanding to translate patients' subjective descriptions into more objective and useful statements.

However, unlike the other affective states, pain is always felt in some particular *part* of the body. Having said this, the localization of the pain very often lacks precision, and it is often experienced at some distance from its source – 'referred pain'. This constitutes the second problem in using the symptom of pain as a diagnostic aid.

Perception and modulation of pain

The intensity of pain does not depend only on the intensity of irritation of the peripheral nociceptive system (receptors and their afferents). Centripetal transmission of peripheral nociceptive stimulation is subject to varying degrees of facilitatory and inhibitory modulation at different synapses during its course to the cerebral cortex. An important modulation site, of major concern to the orthopaedic physician, is the gateway synapse in the basal spinal nucleus, but there are also

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modulation systems in the spinal grey matter, in the thalamus and in the cerebral cortex itself.[5]

Peripheral nociceptive system

Nociceptive receptors are defined as nerve endings that are sensitive to noxious or potentially noxious (mechanical and chemical) stimuli. The perceptual aspect of the nociceptive system consists of unmyelinated free nerve endings, distributed three dimensionally throughout skin, subcutaneous and adipose tissue, fasciae, aponeuroses, ligaments, tendons, muscles, periosteum and bone.[6,7] Clinically, three distinct areas of pain perception may be considered: the skin (superficial somatic pain); the locomotor system (deep somatic pain); and the viscera (visceral pain). Of these, only the skin is adapted to localize pain exactly in the region of injury. Deep somatic and visceral pain are often felt in unusual locations (see Referred pain, p. 6).

In normal circumstances, this nociceptive receptor system remains largely inactive. The unmyelinated free nerve endings are depolarized only by the application of mechanical forces sufficient to deform or damage the tissue that contains them or after exposure to sufficient concentrations of irritating chemical substances (lactic acid, serotonin, prostaglandins and histamine), released from local inflammatory cells and from the peripheral terminals of the primary afferent fibres themselves.[8–10]

Another important influence on nociceptor sensitivity is the pH of the tissue. High local concentrations of protons are known to occur in inflammation and the consequent reduction in pH contributes to the sensitization of nociceptors.[11,12]

Afferent nociceptive system

Nerve impulses generated at the nociceptive receptor system are delivered into the spinal cord by small myelinated and unmyelinated nerve fibres (5 μm or less in diameter), that mainly belong to the Ad and C groups of afferent nerve fibres (Fig. 1.1). Their cell bodies are located in the dorsal root ganglia of the spinal nerves. The very small diameter of the C nerves explains their slow conduction velocity (1 m/s), and their extreme sensitivity to blockade by local anaesthetic drugs. The myelinated Ad fibres are slightly larger and have a faster conduction velocity (10 m/s).[13]

The nociceptive afferents enter the spinal cord, where they divide into short ascending and descending branches, before they terminate at synapses on various groups of relay neurones in the dorsal horn of the spinal grey matter. Most of the connections are to the neurones in the basal spinal nucleus (at the base of the dorsal horn).[14,15]

The efferents of these cross the cord obliquely to turn upwards on the contralateral side and form the anterolateral spinal tract, which connects the basal spinal nucleus with the thalamic nuclei and has therefore traditionally been called the 'spinothalamic tract'. Most of the fibres in this tract, however, do not directly ascend to the thalamus without interruption, but instead synapse with neurones in the brainstem reticular system, while others re-enter the spinal grey matter to synapse

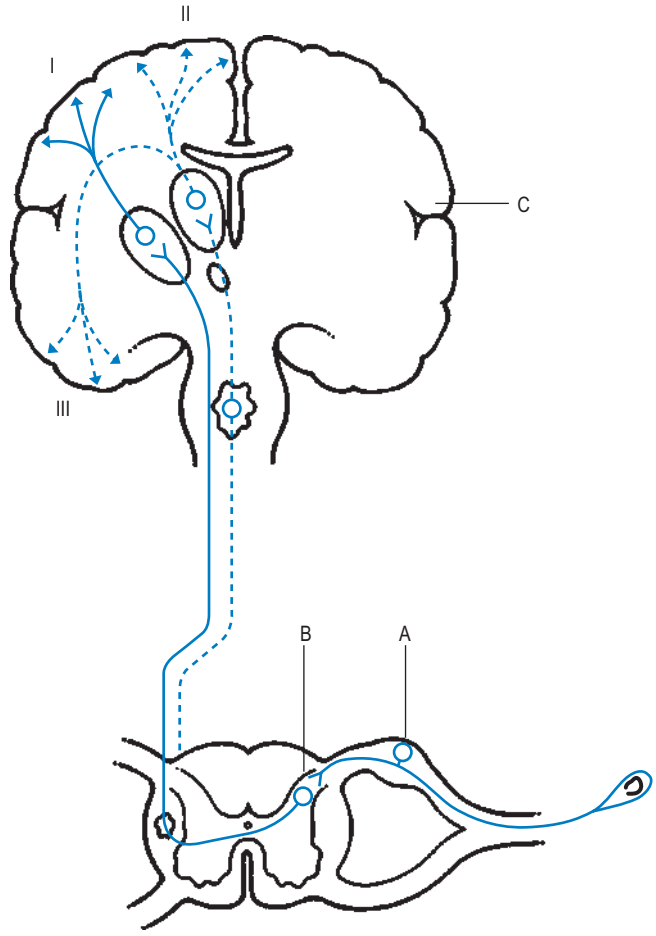

Fig 1.1 • The afferent nociceptive systems. Projection areas: I, perceptual area; II, emotional area; III, memory storage. Three levels of sensory neurone: A, primary sensory neurone; B, dorsal horn cell (gateway synapse); C, thalamic relay.

with internuncial neurones.[16] However, the majority of the ascending nociceptive inputs terminate (sometimes after crossing several synapses) in the thalamic nuclear relay sites.[17] It should be emphasized that not only do the neurones in the thalamic centres respond to peripheral noxious stimulation but they can also be activated by mechanoreceptor peripheral stimulation (see Pain modulation systems below).

The axons of the thalamic nuclei then ascend to the neurones of the cerebral cortex. Three thalamocortical projections can be defined: those responsible for perception; those related to the emotional experience; and those responsible for memory.[18]

The first project to the superior paracentral region of the cerebral cortex and seem to contribute to the so-called '*perceptual component*' of pain – the patient's ability to perceive whereabouts (in which segment of his body) the pain is localized.[19]

The activation of the second thalamocortical projection system, projections that pass from the medial and anterior thalamic nuclei to the frontal lobes, evokes the *emotional disturbances* related with pain.[20] Thus, a stimulus 'hurts' only when the nociceptive afferent projections arrive at the frontal cortex.

A third thalamocortical projection system links some of the medial thalamic nuclei to the cortex of the ipsilateral temporal lobe. Here the recent and *long-term memory storage systems* of the brain are located.[21,22]

A fourth projection system exists which relates some thalamic nuclei to subjacent hypothalamic nuclei in the ventral diencephalon. It is very probable that this thalamohypothalamic system provides the means whereby nociceptive afferent activity entering the brain evokes the complex of *visceral reflex* (cardiovascular and gastrointestinal) *effects* and hormonal changes that are so often associated with the experience of pain.[23]

In conclusion, activity of nociceptive receptors distributes pain into four different projecting systems in the brain, each contributing to a specific component of the global experience 'pain'. However, the projection of pain from a peripheral receptor to the brain is not via a straight-line system. The intensity of pain is not only determined by the intensity of peripheral stimulation but also depends largely on peripheral and central modulation systems at the various synaptic stages in the course of the afferent pathway within the central nervous system. These modulation systems account for the large variation in the intensity of pain experienced. Patients with apparently comparable pathological lesions undergo widely different degrees of suffering; even in an individual patient, the intensity of experience of pain varies widely with the prevailing emotional mood, with concentration on the problem or with suggestions from others.

Pain modulation systems

There are both peripheral and central pain modulation systems.

Peripheral modulation of pain

One of the most important sites at which a synaptic modulation operates from both the peripheral and central sources is at the synapses in the basal spinal nucleus. In 1965, Melzack and Wall,[24] basing their theory mainly on the work of Noordenbos,[25] published an article entitled: 'Pain mechanism: a new theory'. They called their concept of peripheral pain modulation the *gate control theory* (Fig. 1.2), which is based on three premises:

- Afferent nerves contain two types of fibres: small fibres (P), as described above, and large-diameter afferents (M), which are derived from the various mechanoreceptors in the articular capsule, ligaments and muscle spindles. These fibres produce information about static joint position, pressure changes in the joints, joint movement and stresses that develop in the joint at the extremes of movement. The fibres of mechanoreceptor transmission have a lower stimulation threshold and a faster conduction velocity than the smaller and mostly unmyelinated fibres of the nociceptive system.
- In the substantia gelatinosa (SG) of the dorsal horn, both afferent systems converge and interrelate, with the overall effect that the large-diameter afferents have an inhibitory effect on the relay neurones located in the basal spinal nucleus. This inhibition is presynaptic and is reduced only when there is a massive input from the small nociceptive afferent fibres. The latter thus facilitates central transmission of pain. The interaction between both systems is gate control: impulses travelling along the larger fibres close the gate, and those in the small fibres open the gate so that impulses to thalamus and cortex can pass through.
- The activity of the gate is not only modulated by impulses from nociceptive and mechanoreceptor systems but also receives a descending and regulating feedback from the reticular system, the thalamus and the cerebral cortex.

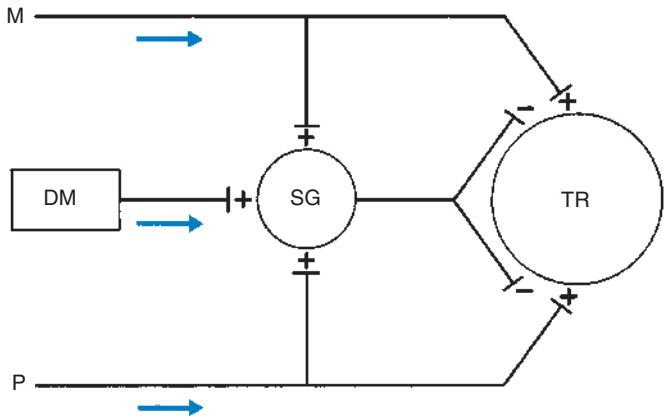

Fig 1.2 • Gate control theory (after Melzack and Wall[24]): P, small nociceptive fibres (pain); M, large mechanoreceptive fibres; TR, transmission cells (relay neurones in the basal spinal nucleus); DM, descending modulation; SG, substantia gelatinosa; +, excitatory effect; –, inhibitory effect.

This peripheral modulation of pain has considerable clinical importance. It indicates that centripetal projection into the central nervous system of afferent activity from the nociceptive receptor systems is not passed straight to any 'pain centre' in the brain but receives constant modulation at its synaptic portal of entry into the neural axis at the level of the basal spinal nucleus. The modulation stems from the concurrent activity of the mechanoreceptors located in the same tissues, and from feedback through projection systems descending from the brainstem and cerebral cortex. This effect is one of the reasons why movement and selective stimulation of mechanoreceptors can cause inhibition of pain.

Central modulation of pain

Awareness of pain is also modulated at the central projection systems.

At the reticular formation

A modulation system at the reticular formation in the brainstem exerts a continuous inhibitory effect on the projection neurones in the spinal nucleus ganglion via the reticulospinal tract, which is discharging continuously at varying frequencies throughout life.[26] The inhibitory effect on nociceptive afferent transmission is augmented when the attention of the patient is distracted from the site of pain. This is what occurs when

another painful site elsewhere in the body is stimulated (counter-irritation), when the patient concentrates on work or other activities or when hypnosis is induced.[27] The inhibitory effect of this reticular system also increases when the blood concentration of catecholamines is very high, as can be the case in states of great emotional tension.[28] Also some drugs (chlorpromazine, diazepam and morphine) may selectively increase the activity of the reticular neurones that operate this inhibitory system.[29]

Inhibitory reticular activity is depressed and pain is enhanced when attention is concentrated on the painful site, or following the administration of barbiturates, caffeine or theophylline.[30]

The cerebral cortex

The cerebral cortex, especially the sectors located in the frontal and paracentral regions, in turn regulates the activity of the reticular formation. Reticular activity is increased, and perception of pain thus inhibited during rest and sleep and after the ingestion of alcohol. Conversely, depression of reticular activity is seen during increasing cortical activity, for example with anxiety, uncertainty and fear.

Referred pain

Introduction

When the skin is pricked with a pin, the patient can exactly pinpoint the injury. This ability to localize the pain is limited to skin and does not apply when the source of the pain is in deep tissue. Deep somatic pain and visceral pain are often felt far from their point of source. In consequence, the examiner needs to know the patterns of pain reference so as not to be misled about where to search for the seat of the trouble. Diagnosis of orthopaedic lesions often rests entirely on history and clinical examination and is therefore almost impossible if the rules and conditions relevant to referred pain are not clearly understood.

Those who originally studied pain reference soon noted that although it appeared erroneous and anarchic, some rules of presentation did exist. For instance, pain from specific structures is always referred to the same parts of the body: colic from a ureteral stone to groin and testicle, diaphragmatic disorders typically to the shoulder, angina pectoris to one or both arms, and the pain caused by arthritis of a hip very often to the ipsilateral knee. Also pain is, in the main, referred distally and its localization depends in a certain way on the severity of the lesion.

In 1905, Sir Henry Head described referred pain in the abdominal wall caused by a visceral disease.[31] Using the dermatological appearances in herpes zoster, he constructed schemes of segmental innervation of the skin.[32] He also described dermatomic zones that became painful in the event of provocation of a related visceral structure. His theory of pain reference was built on the concept of the segmental organization of the human body and its nociceptive system. Further experiments in this sphere were conducted by Sir Thomas Lewis in 1936.[33] In 1938 and 1939, Kellgren published the results of a systematic examination of the phenomena of referred pain, demonstrating segmental radiation and failure to cross the midline.[34,35]

His experiments were confirmed by others.[36–38] Later, the concept of segmental reference of pain was refined[39,40] and exact borders of the different dermatomes mapped out.[41–44]

Possible mechanisms

The fact that referred pain is an error in perception was first pointed out by John Hunter in 1835 (cited by Cyriax).[45] It was obvious that if pain is felt elsewhere than at its true site, the nociceptive mechanism is reacting inappropriately. However, since there seems to be logical consistency in the way the errors are made (pain from specific lesions is always referred to the same areas), there must also be a logical explanation for 'failures'. (If a machine always makes the same mistake, a structural or functional disorder must exist.) The basis for the inadequacy must therefore be sought in a miscalculation in the pain mechanism. Theoretically, the defect can lie anywhere along the afferent pathway, from the peripheral receptors to the synapses in the spinal cord and the reticular area and projection zones in the sensory cortex.

During the last century, numerous investigators have studied referred pain. Two main hypotheses have been put forward:

- *Error at the level of the spinal cord*. Most authors have opted for this hypothesis. Mackenzie described an 'irritable focus' in the grey matter of the spinal cord as being responsible for the phenomenon.[46] Also Livingston[47] placed the basis of the error at synapses in the dorsal horns. Wedell *et al*[48,49] and Pomeranz[50] accepted a double origin for the sensitive neurone – afferent fibres of a somatic structure, and those coming from a related visceral structure synapse with the same spinal ganglion. Taylor *et al*[51] and Wells *et al*[52] also made a plea for the spinal explanation of referred pain. Their view is that separate peripheral sensory nerves (deep somatic, skin and visceral) converge on to the same cell in the dorsal horn of the spinal cord (Fig. 1.3).
- *Failure at the sensory cortex*. A number of authors have proposed that the misinterpretation is at the projection area of the sensory cortex rather than at the spinal level.[33,53,54] The concept was clinically elaborated by Cyriax who based his theory on a number of premises:
  - Referred pain is a pain experience felt elsewhere than at its true site of origin.
  - Skin is an organ adapted to localize the pain accurately.
  - Pain is experienced in the sensory cortex, which is organized dermatome by dermatome.
  - The skin is represented accurately in the sensory cortex.
  - A memory storage system is located in the sensory cortex. This is fed by constant input from the skin. Input from deeper somatic structures is very rare in a normal and healthy individual.

As pointed out previously, pain is experienced at three different locations in the cerebral cortex. Perception – of the site of

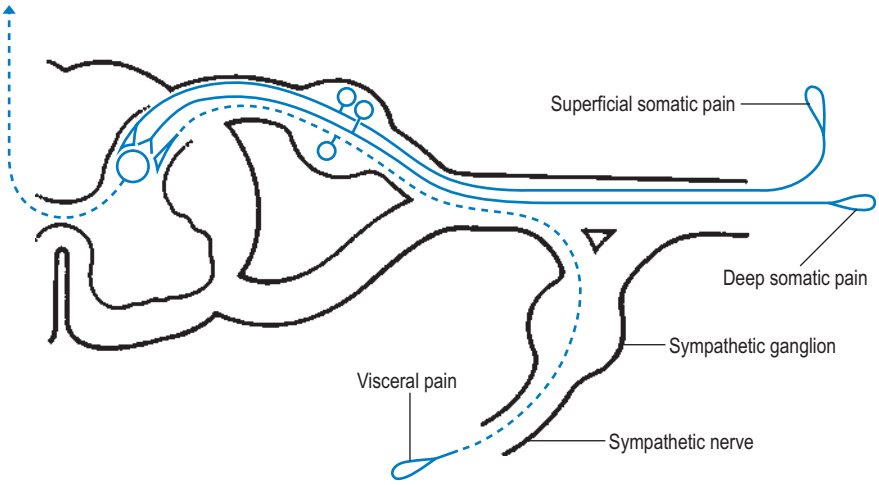

Fig 1.3 • Separate peripheral sensory nerves converge onto the same cell in the dorsal horn of the spinal cord.

pain – is located in the superior paracentral cortex. The frontal lobes evoke the emotional disturbances related to pain, and the memory store is in the temporal lobes.

The ability to localize pain in the region of injury is limited to skin and does not apply when the source of the pain is in deep tissue. In due course, a certain pain memory is built up in the temporal lobes, and achieves a high degree of anatomical precision. The efficacy of the long-term memory storage system is not simply a function of the intensity of the painful experience but also relates to the length of time a painful experience lasts or to the frequency with which it is repeated.[55,56] Since the frequency of painful stimuli coming from the skin is much higher than the frequency of stimuli coming from deeper structures, it will be obvious that pain memory will centre around painful experiences from the skin. When the same cortical cells receive a painful message arriving from a deep-seated structure, the memory will interpret it on the basis of past experience; in that the sensory cortex is arranged segmentally, the pain will be ascribed to the correct segment but the system will fail to localize it accurately at the site of the lesion. The brain therefore 'places' it in the tissue it has a reference for – the skin. Pain is thus felt under the surface area connected with the particular cells that belong to the same segment as the tissue from which the nociceptive afferents originate. The pain is felt deep to the skin of the relevant dermatome and not accurately in the skin.

Clinical consequences

The concept of referred pain is extremely important to the orthopaedic physician, who has to deal daily with the problem. If the principles of erroneous localization by the cortex are clearly understood, the examiner can turn a misleading phenomenon to diagnostic advantage. In the Cyriax concept, referred pain obeys certain rules. The inadequacy in the sensory cortex is structural and therefore can easily be accommodated. To a certain degree, referred pain can be compared with the refraction of light when it falls on a water surface. The observer does not see objects under the water surface at their exact localization. However, since the error of perception is structural and obeys particular physical rules and laws, it is easy to correct what is seen (provided the observer knows the correction formula) and so locate the object accurately. The same applies to referred pain. The examiner must constantly ask if the localization of the pain is also the exact localization of the disorder and, if the answer is negative, what corrections must be made to arrive at the exact localization.

Before this discussion of referred pain is continued, it should be stressed that root pain reference does not necessarily mean that a nerve is involved. The false idea that wide radiation of pain is evidence of involvement of nerves is still strongly held by some and is usually the most important obstacle to a logical understanding of referred pain. To approach the problem of referred pain with an open mind, the reader must constantly remember that referred pain is an *error of perception*. Although the nerve supply to these peripheral structures is distributed on a segmental basis, it does not indicate that referred pain 'runs down' a somatic nerve. For instance, pain at the anterior aspect of the leg does not necessarily mean that a nerve structure (L3, femoral nerve or peripheral branches of the femoral nerve) is involved. Although inflammation of the dural sleeve of the L3 nerve root does of course lead to pain extending in the L3 dermatome, the same pain can be provoked by a lesion in any other tissue belonging to the L3 segment (e.g. hip joint or psoas bursa). There will not be any difference in the nature and extent of the pain. The only distinction between pain as the result of a compressed and inflamed nerve root and pain originating from trauma to other structures is the appearance of paraesthesia (see Pressure on nerves, Ch. 2).

Rules of referred pain (Box 1.1)

The first rule – reference of pain within the borders of the skin area that belongs to the same segment as the tissue lesion that causes pain – follows directly from the premise that the nociceptive mechanism is organized on a segmental basis. Nociceptors, afferent fibres and the sensory cortex are all arranged segmentally. Afferents from skin, deep somatic structures and visceral organs from the same segment relay on the same dorsal neurones and project to the same area in the sensory cortex. Pain reference is therefore confined to, and remains within, the borders of the cutaneous area (or dermatome) that belongs

embryologically to the same segment as the tissue from which the pain is arising.

To understand the segmental organization of the nociceptive mechanism, it is necessary to reconsider embryogenesis (Fig. 1.4).

Embryogenesis

Primitive body

When a fetus is between 4 and 6 weeks old, 42 pairs of somites develop: four occipital, eight cervical, 12 thoracic, four to six lumbar, five sacral and eight to 10 coccygeal.[57] The first two and the last seven or eight pairs disappear early in development. The ventral aspect of each somite differentiates into the *sclerotome* which, together with the chorda around the neural tube, forms the origin of the axial skeleton. The other part of the somite becomes the *myotome*, covered by the *dermatome*. Each pair of somites develops its own segmental innervation which later leads to the development of the spinal ganglion and spinal nerve.

In due course, the dermatome differentiates into skin and subcutaneous tissue, the myotome into muscles, tendons, ligaments, capsules and bursae, and the sclerotome into bone and fibrous septa. Although the original form of most segments is modified as the limbs are formed, their segmental innervation remains constant throughout life. The projection area in the cerebral cortex also remains segmentally organized.

Limb formation

After the first month of intra-uterine life, two pairs of buds originate at the lateral sides of the fetus. The proximal papules appear first at the base of the neck, followed by the formation of two distal buds in the caudal area. These extensions gradually project from the cylindrical fetal segments from which they originate. As the limbs grow further and further laterally, some dermatomes become totally disconnected from the trunk (Fig. 1.5).

For the upper limb, the dermatomes C5, C6, C7, C8 and T1 leave the trunk completely to form the covering of the arm. T2, although also present at the inner aspect of the upper arm, connects with the trunk again, and borders with C4.

In the lower limb, parts of L2, L3 and the whole dermatomes L4–L5 and S1 withdraw from the trunk to form the lower limb. S2 is present partly in the limb and partly in the buttock, where it borders with L3.

During limb formation, some muscles undergo centripetal migration and others centrifugal. As a rule, however, dermatomes distally project further than myotomes, and sometimes a muscle becomes completely dissociated from the covering dermatome. An example of dermatome migration is

Box 1.1

Rules of referred pain

- The pain radiates segmentally and does not cross the midline
- The pain is usually felt deeply
- The pain is referred distally within the dermatome
- The pain does not necessarily cover the area of the causative lesion
- The pain is felt anywhere in the dermatome but not necessarily in the whole dermatome

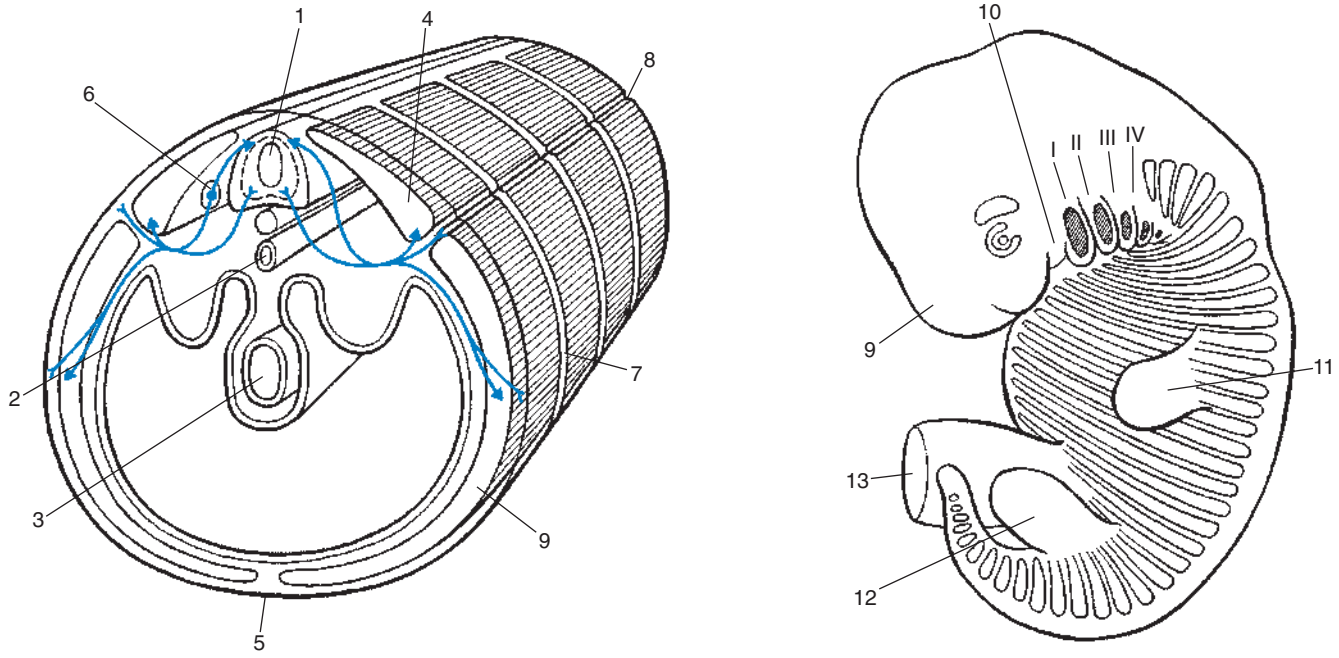

Fig 1.4 • Embryogenesis: 1, neural tube; 2, aorta; 3, intestine; 4, myotome; 5, dermatome; 6, primitive spinal ganglion and spinal nerves; 7, myoseptum between the segments; 8, horizontal septum; 9, frontal knob; 10, maxillar knob; 11, cranial limb bud; 12, caudal limb bud; 13, umbilical cord; I–IV, gill arches.

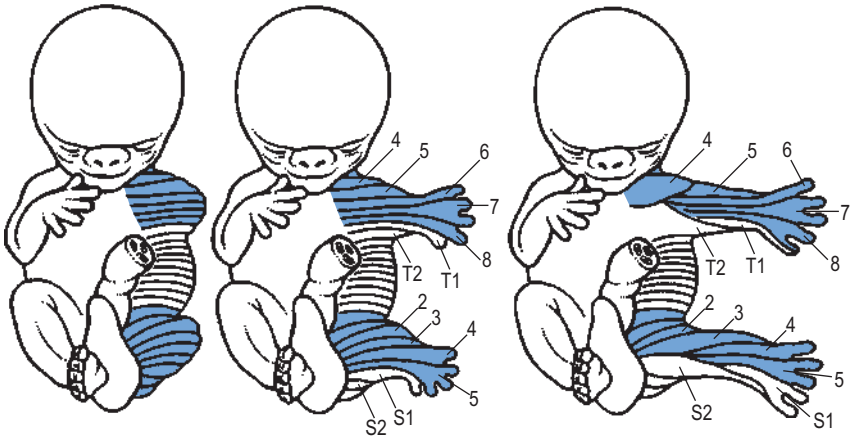

Fig 1.5 • Limb formation. As the limbs grow further and further laterally, some dermatomes become anatomically disconnected from the trunk.

the C5 segment in the upper limb: the myotome does not extend beyond the elbow, but the fifth cervical dermatome descends to the radial styloid. An example of centrifugal dissociation between a muscle and its relevant dermatome is the diaphragm, which has a C4 origin: the C4 dermatome ends at the scapular spine and under the clavicle, and is therefore completely separated from the thoracic localization of the muscle. Another instance of muscle migration is the latissimus dorsi muscle (C7–C8) which shifts its origin to the iliac crest. The dermatomes of the seventh and eighth cervical segment, however, do not occupy the trunk, so dissociating the muscle almost completely from its corresponding dermatomes.

Because of these muscle migrations, with overlaps and discontinuous areas, it is very difficult to draw accurate maps of the myotomes. However, in the appropriate chapters of this book, we will indicate to which segment each of the structures under discussion belongs.

Dermatomes

The cutaneous area supplied by one spinal nerve is a dermatome. The first clinicians to draw dermatomic maps were Head and Campbell.[58] Their diagrams are the basis for the classical drawings in standard neurological textbooks. However, they did not take into consideration the significant degree of variability and overlap in dermatomic borders.[57,58] Later investigators such as Keegan and Garrett, and Fukui *et al*[59,60] have demonstrated that dermatomes of adjacent spinal nerves overlap markedly.

An example of this is pain at the anterior aspect of the thigh, which can be of second or third lumbar origin. The second lumbar dermatome spreads from the groin, down the front of the thigh, to the patella. Pain of third lumbar origin again spreads along the anterior aspect of the thigh and the patella but it can continue down the anterior aspect of the leg, to just above the ankle. Pain at the anterior aspect of the thigh therefore can be of second or third lumbar origin but if it spreads further down below the patella then its origin is third lumbar.

Another example of overlap between dermatomes is referred pain in the hand and fingers: C6 pain refers to the dorsal aspect of the hand, the thumb and the index finger, whereas C7 refers also to the dorsum of the hand and the index finger, as well as to the long and ring fingers. In a patient complaining of pain at the back of the forearm or at the dorsum of the hand and the index finger, it is difficult to decide whether the pain is of sixth or seventh cervical origin.

We use the charts drawn by Foerster[41] in 1933 and corrected by Cyriax in 1982,[45] in order to give the broadest possible information about the area to which pain from a particular segment may refer. The extreme importance of accurate localization of pain in orthopaedic medicine requires the physician to use a map of dermatomes that is as close as possible to clinical reality. The figures showing the dermatomes are based on Foerster,[41] Cole *et al*,[61] Cyriax,[45] Conesa,[62] Wakasugi,[63] and Mitta.[64]

Cervical and thoracic dermatomes

C1 to C4 occupy the scalp (C1), the back of the neck and the temporal area, the upper half of the ear and the upper half of the face (C2), the neck, lower mandibular area and chin (C3), and the lower half of the neck, shoulder area, front of the upper chest and the area above the spina scapulae (C4) (Fig. 1.6).

C5 to T2 are projected from the trunk to form the covering of the upper limb (Figs 1.7 and 1.8).

C5 covers the deltoid area and the outer aspect of the arm up to the base of the thumb.

C6 is the anterolateral aspect of the arm, the thenar eminence, thumb, dorsum of hand and index finger.

C7 comprises the back of the arm and hand, together with index, long and ring fingers.

C8 is the inner aspect of the forearms, the hypothenar area and palm, together with the three ulnar fingers.

T1 includes the inner aspect of the forearm as far as the hypothenar eminence.

T2 is Y-shaped and overlies the inner aspect of the upper arm and the axilla, where it divides into an anterior and a posterior component. This latter part of the dermatome borders with the inferior aspect of the C4 dermatome.

If the upper limb is held outstretched horizontally with the thumb pointing upwards, the original position of the embryological bud is recreated, and one can reconstruct the way the dermatomes project from the trunk. This is a good way to

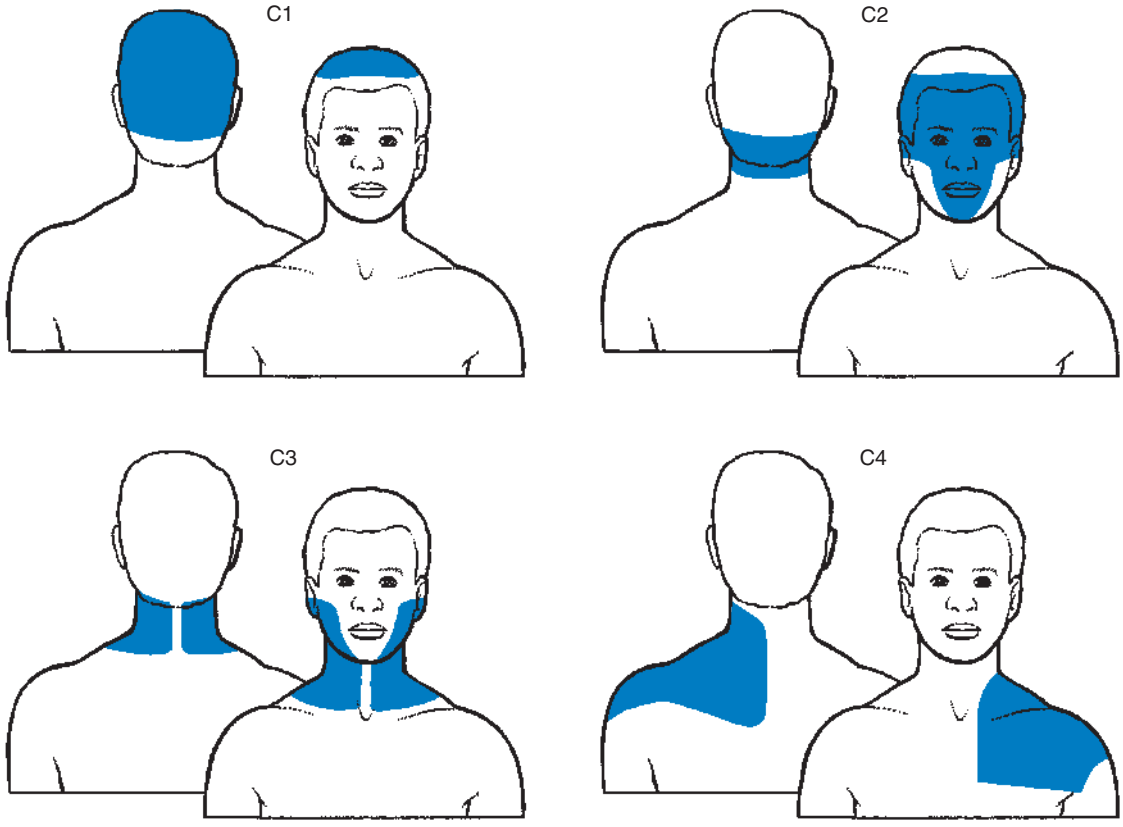

Fig 1.6 • C1 to C4 dermatomes.

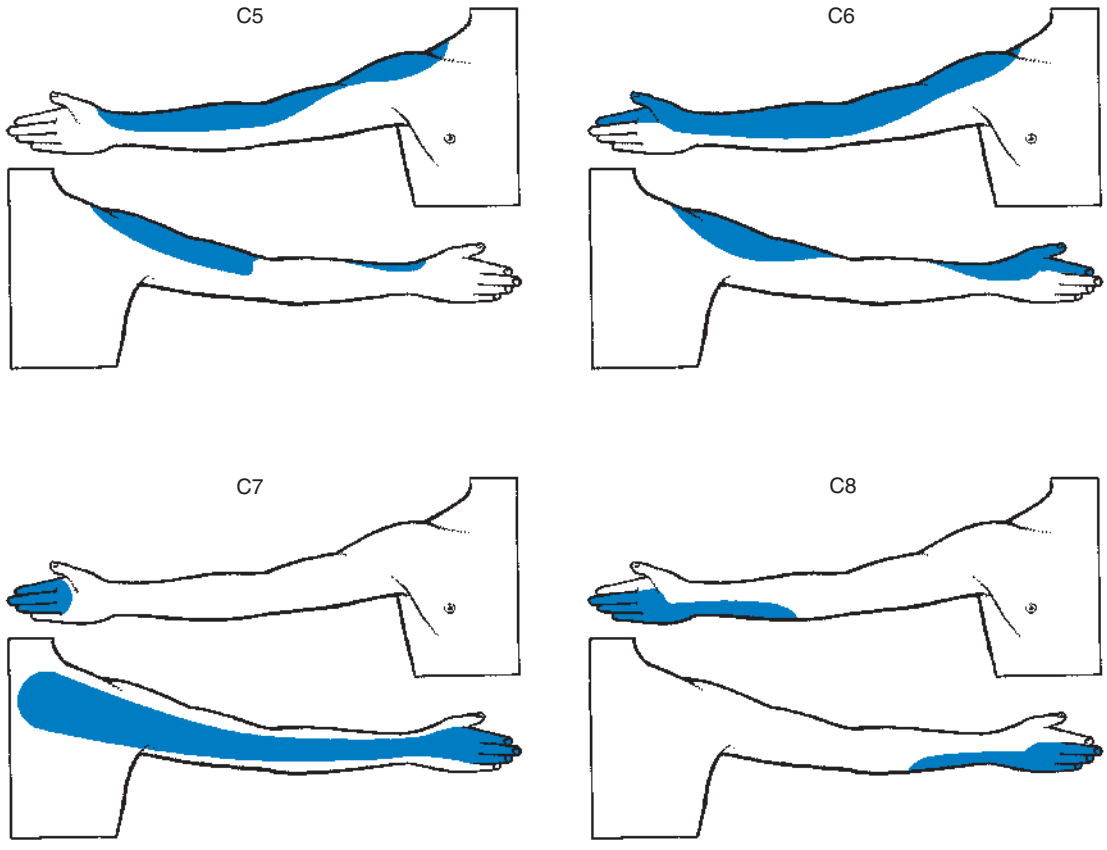

Fig 1.7 • C5 to C8 dermatomes.

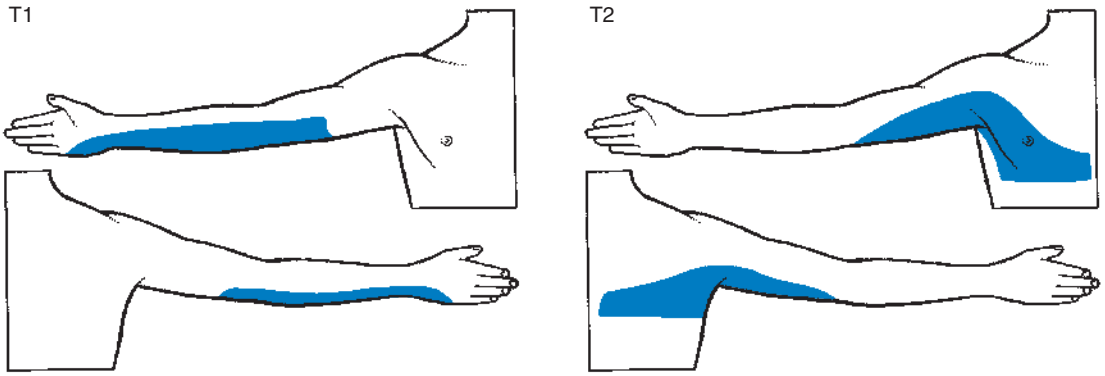

Fig 1.8 • T1 and T2 dermatomes.

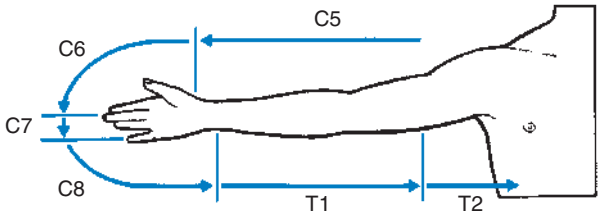

Fig 1.9 • Outstretched arm with arrows showing the dermatomes.

memorize the position of the separate dermatomes in the upper limb (see Fig. 1.9).

From T4 to T12, the dermatomes encircle the trunk, more or less following the original segmental construction of the embryo (Fig. 1.10).

T4 constitutes the axilla and a patch on the front of the chest.

T4 encircles the trunk at the level of the nipple.

T7 reaches the lower costal margin and covers the xiphoid process.

T10 is level with the umbilicus, and T12 reaches to the groin, and probably also the area between the femoral trochanter and iliac crest.

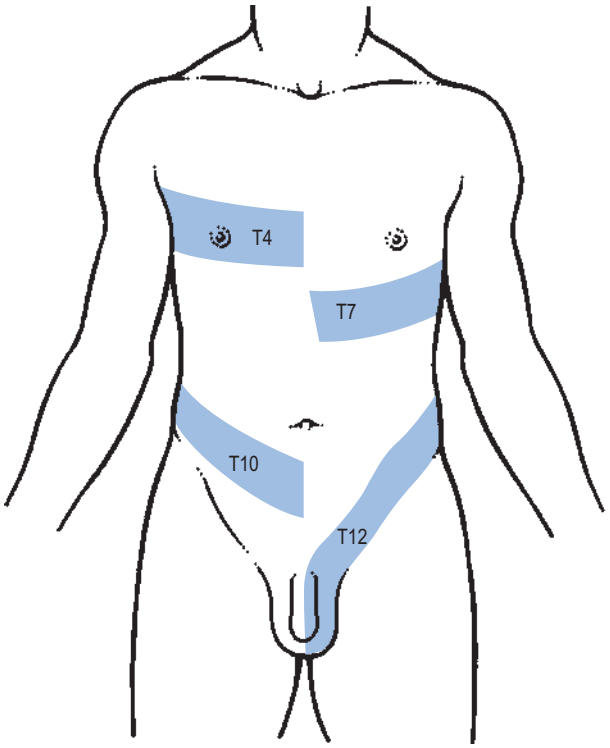

Fig 1.10 • T4, T7, T10 and T12 dermatomes.

Lumbar and sacral dermatomes

L1 is also more or less circular (Fig. 1.11). It comprises the lumbar region from the second to the fourth lumbar vertebrae, and runs along the upper aspect of the buttock and the iliac crest to the lower abdomen and the groin.

L2 and L3 are two discontinuous areas, one in the lower lumbar region and upper buttock, and one in the leg (Fig. 1.12). The areas in the buttock largely overlap. Also, in the leg, there is considerable overlap between L2 and L3: L2 involves the whole front of the thigh, from the groin to the patella. L3 also takes in the anterior aspect of the thigh, but spreads further down, as far as the anterior and medial aspects of the ankle.

L4, L5 and S1 are completely disconnected from the trunk, overlying the surface of the leg and foot (Figs 1.13 and 1.14).[64] In consequence, the third lumbar dermatome lies adjacent to the upper border of the second sacral dermatome at the lower buttock.

L4 occupies the lateral aspect of the thigh, crosses the leg above the ankle, and ends at the medial malleolus, the inner border of the foot and the big toe.

L5 consists of the outer aspect of the leg and crosses the ankle above the lateral malleolus, to end on the dorsum of the foot. L5 also comprises the big, second and third toes, and the inner half of the sole.

S1 includes the calf, the heel, the lateral malleolus and foot, the two outer toes and the whole sole of the foot (Fig. 1.15). Sicard and Leca[65] demonstrated that the fifth lumbar and first sacral dermatomes also comprise a small vertical band at the

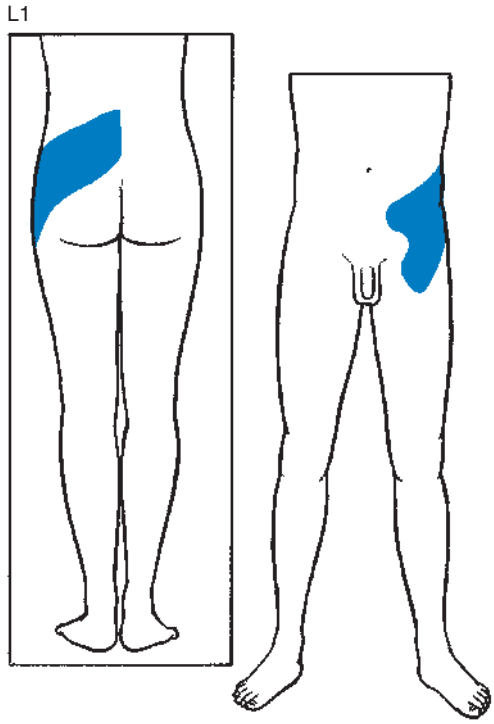

Fig 1.11 • L1 dermatome.

posterior aspect of the thigh, which could account for the thigh pain commonly described by patients suffering from L5 or S1 sciatica.

S2 is large and comprises the plantar aspect of the heel, the calf, the back of the whole thigh and the lower buttock. In the buttock, it borders with the lumbar part of L3 (Fig. 1.15).

S3 is a narrow zone at the inner side of the thigh, where it borders with L2 anteriorly and S2 posteriorly (Fig. 1.16). The tip ends just proximal to the knee. The upper extent reaches the inguinal ligament where it adjoins the 12th thoracic and first and second lumbar dermatomes. It follows that the groin is a confluence of dermatomes, and pain, apart from that of local origin, may be referred from a 12th thoracic, a first or second lumbar, or a third sacral origin. The groin is also a common site for extrasegmental dural pain reference.

S4 comprises the saddle area, anus, perineum, and scrotum and penis or labia and vagina.

S5 is the coccyx.

As in the upper limb, the original position of the distal embryological bud can be reconstructed by abducting the thigh to 90°, and by lateral rotation until the big toe points upwards. This position demonstrates the way the dermatomes were projected from the trunk and also constitutes a good way of memorizing the position of the various dermatomes in the lower limb (Fig. 1.17). Proximally to distally and then turning proximally again, the following are encountered: L2 at the

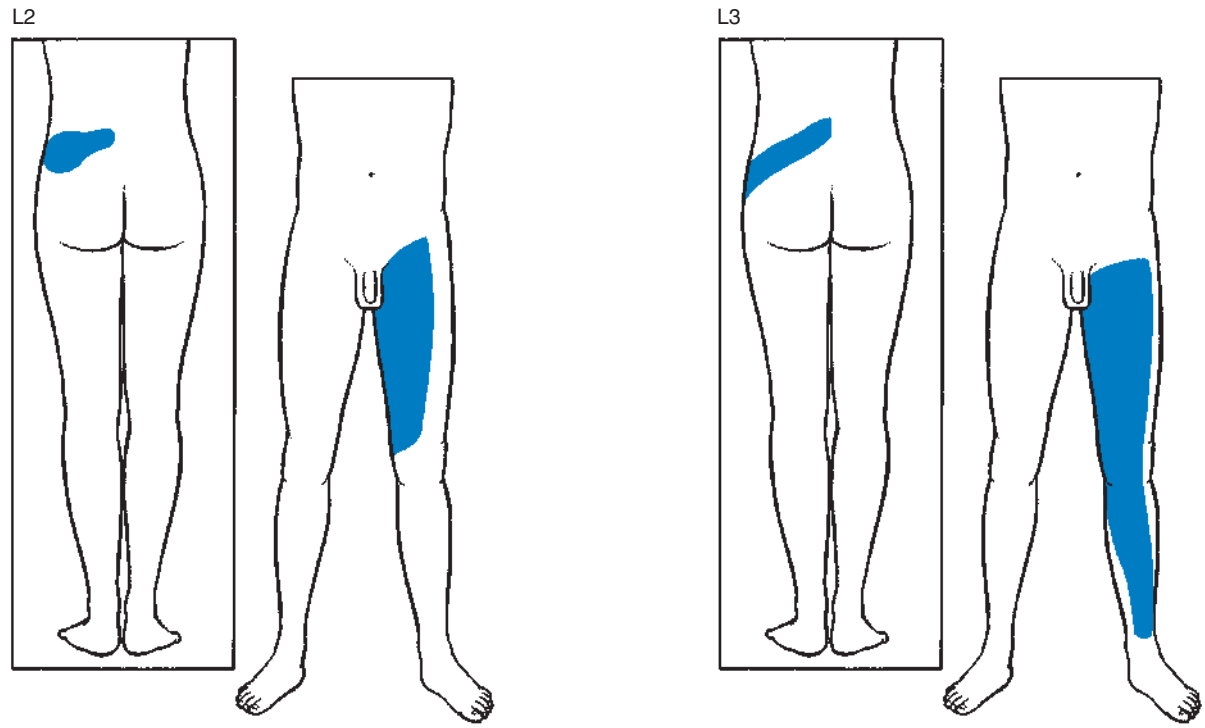

Fig 1.12 • L2 and L3 dermatomes.

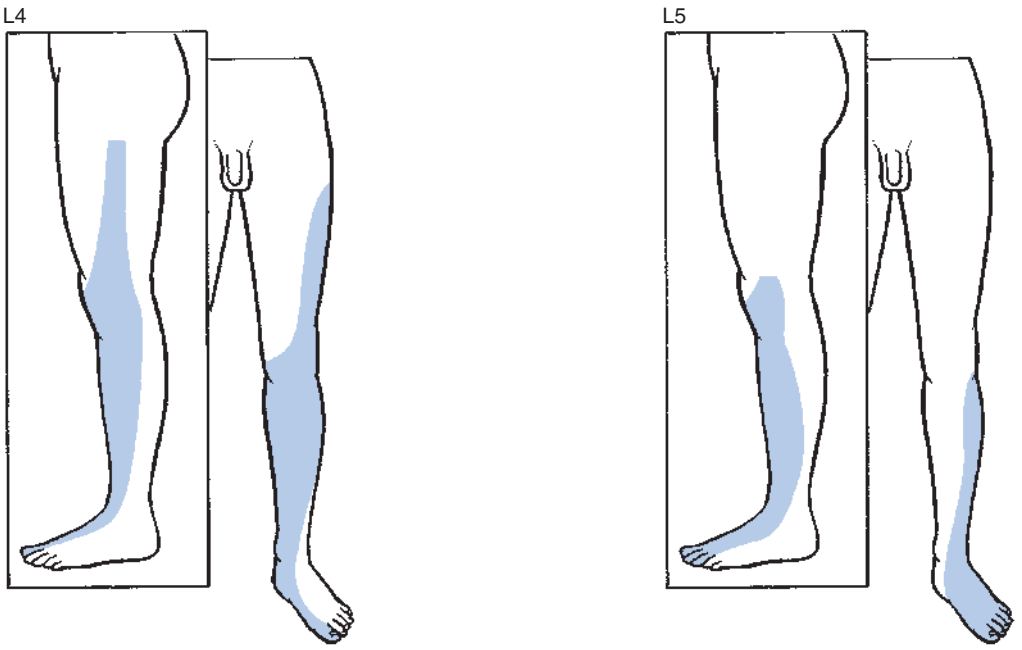

Fig 1.13 • L4 and L5 dermatomes.

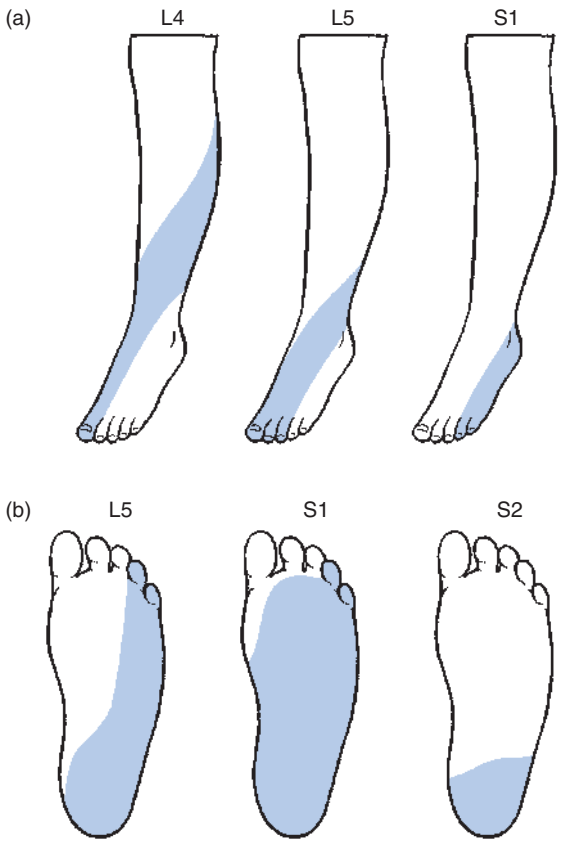

Fig 1.14 • Dermatomes of the foot, (a) dorsum (b) sole.

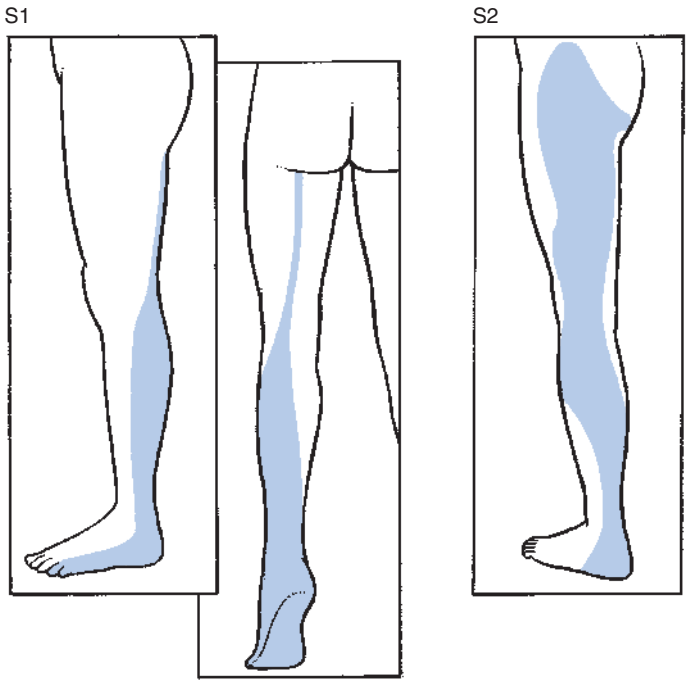

Fig 1.15 • S1 and S2 dermatomes.

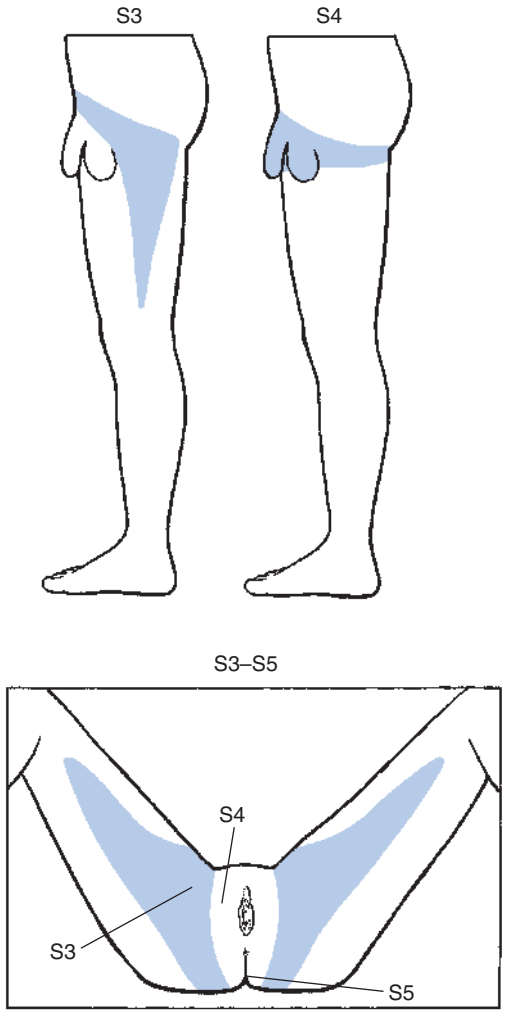

Fig 1.16 • S3 to S5 dermatomes.

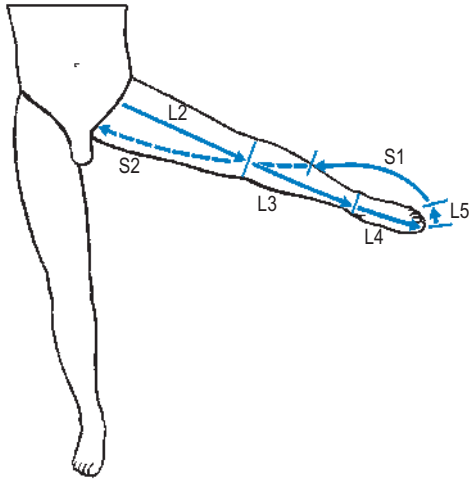

Fig 1.17 • Outstretched and rotated leg, with arrows showing the dermatomes.

anterior thigh, L3 at thigh and leg, L4 at the lateral aspect of the leg, anterior aspect of ankle and inner border of foot up to the big toe, L5 at the dorsum of the foot and the three inner toes, S1 at the lateral aspect of the foot, outer malleolus and calf, S2 at the posterior aspect of the leg; turning back to the trunk in the gluteal area, the boundary zone in the perineum, between leg and trunk is comprised of S3 and S4.

Discrepancies between dermatomes and myotomes

We have mentioned already that, as the outcome of embryological development, dermatomes do not always precisely cover the underlying myotomes. Cyriax described eight areas in the human body where the skin and the structure it covers have completely different embryological derivations (Cyriax[45]). These are the head, scapular and pectoral region, hand, intrathoracic and intra-abdominal region, buttock and scrotum.

Head

The skull, head and face are derived from the two remaining occipital somites, originally situated at the back of the neck. During development, a pair of frontal knobs and two mandibular arches form and fold forwards to create the skeleton and soft tissues around the buccal cavity. The skin of the head and face, however, are formed from the upper two cervical segments.

Scapular region

The growth of the protuberances that are to become the upper limbs draws some segments out from the cylindrical cervical and thoracic structures. At the same time, the scapula and its muscles, together with the latissimus dorsi (C5–C7) move centripetally between the skin of the thorax (circularly arranged thoracic dermatomes) and the underlying ribs and intercostal muscles (circularly arranged thoracic sclerotomes and myotomes). Therefore, pain in the scapular area can have both scapular (cervical) and thoracic origins.

Pectoral region

During the growth of the upper limb bud, the same phenomenon as occurs in the scapular region takes place at the pectoral region. The pectoral muscles, derived from cervical segments (C6–C7) move centripetally between the thoracic dermatomes and their myotomes.

The hand

The thenar muscles form part of the eight cervical and first thoracic myotomes, but the skin is formed from the fifth and sixth cervical dermatomes. The interosseus muscles are C8 and T1, but the skin of the dorsum of the hand, except at the ulnar border which is also C8, is derived from the C6 and C7 segments.

Intrathoracic region

It is obvious that there are numerous discrepancies in origin between the thoracic cage and its content. The diaphragm, for instance, is derived from the third and fourth cervical segments

and thereafter descends. Hence a lesion of the diaphragm may cause pain felt in the neck and at the upper scapular and pectoral region, even though it lies at the lower thoracic level. The heart is derived from C8 to T4. Therefore myocardial pain may radiate to the chest, the shoulder and the inner aspect of the arm, as far as the ulnar border of the hand. It is presumed that a small part of the myocardium, probably the auricles, has a third cervical origin, which could explain the well-known clinical fact that the pain of angina often radiates to the neck anteriorly. The oesophagus is T4–T6 and the lungs have a T2–T5 origin.

Intra-abdominal region

The abdominal wall has a more or less circular construction, from T7 at the xiphoid process, over T10 at the umbilicus, to L1 at the iliac crest, inguinal ligament and groin. In the abdominal wall, the dermatomes exactly overlie the myotomes.

Most of the intra-abdominal content also has a mid- and lower thoracic origin. The embryological derivation of the stomach and duodenum (T6–T10), liver (T7–T9 right), gall bladder (T6–T10 right),[66] pancreas (T7–T8) and small intestine (T9–T10), fit very well with their actual localization in the abdominal cavity, and therefore pain derived from these organs approximates with their surface representation. Structures of lower thoracic, lumbar or even sacral origin, however, show a more complicated pattern of referred pain. The kidney and ureter, for instance, have a T11–L1 derivation and, although they are localized high up in the abdomen, referred pain can reach the inguinal fossa and the groin (T12–L1). The colonic flexure is from L2 to L3, which allows the pain not only to radiate to the lower back but also to the front of the thigh. The sigmoid colon and rectum have S3–S5 origin. Hence, in diseases of the sigmoid and the rectum, pain can be felt in the iliac fossa, perineum, penis, vulva and inner aspect of the thigh.

The buttock

The skin of the lower lumbar area and the outer buttock is derived from L1. At the upper buttock, there is considerable overlap with the lumbar patches of the L2 and L3 segments (Fig. 1.18). The skin of the lower buttock is derived from S2.

The gluteal muscles forming the buttocks are derived from the fourth lumbar to the first sacral segments. Consequently, the dermatomes descend further distally than the myotomes they cover.

Scrotum

The testicles are derived from T11–T12 and L1. The epididymis has a T10 origin. The scrotum, however, belongs to the S4 dermatome. A trauma to the testicle therefore may cause not only local pain but also pain spreading along the iliac crest posteriorly and up to the lower thoracic region. Testicular disease frequently produces pain in one or the other iliac fossa.

Referred pain in visceral diseases

It is important to emphasize that referred pain is not a phenomenon of orthopaedic medicine only. As described earlier, many visceral diseases also cause referred pain. For the convenience of practitioners often faced with thoracic or abdominal pain, a list of the segmental derivations of the viscera, based on the work of Cyriax[45] (see his p. 30), William and Warwick,[67] Lindsay *et al*[68] and Guyton (cited by Van Cranenburgh[69] is given in Box 1.2).

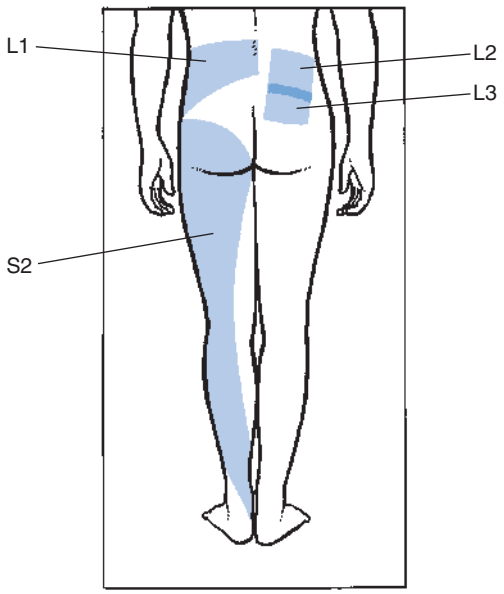

Fig 1.18 • Overlap of the dermatomes at the back and the buttock.

Box 1.2

Referred pain in visceral diseases

Heart (auricles?)	C8–T4 (C3?)
Lungs	T2–T5
Oesophagus	T4–T6
Diaphragm	C3–C4
Stomach and duodenum	T6–T10
Liver and gall bladder	T7–T9 right
Spleen	T7–T10 left
Pancreas	T8
Small intestine	T9–T10
Appendix	T10–L1
Kidney	T10–T12 (L1)
Ureter	T11–T12
Suprarenal glands	T11–L1
Ovary and testis	T11–T12 (L1)
Epididymis	T10
Colon: ascending	T10–L1
flexure	L2–L3
sigmoid	S3–S5
Rectum	S3–S5

Referred pain is felt deeply and distally in the dermatome

An important difference between local pain and referred pain is that in the latter the pain is felt deeply and vaguely. The

patient thus does not point to a localized and precise area, but outlines an approximate one and tends to describe it as 'deep'.

That pain – with some exceptions – is always referred distally, remains a purely empirical clinical observation and has hitherto not been explained on neurophysiological grounds. The fact that pain that arises from the proximal part of a segment can be felt distally in the dermatome but a distal lesion is not referred proximally within this same segment, remains an inconsistency that is hard to rationalize. However, this clinical observation is very important to the clinician confronted with referred pain, for the lesion must never be sought in a structure that is localized distally of the painful area. Pain at the distal aspect of the L3 dermatome (knee and lower limb) can have a proximal origin (spine, hip), but pain only in the hip or the groin, cannot be caused by a lesion at the knee or the thigh.

Segmentally referred pain does not cross the midline

The segments in the body are arranged in pairs, each of which has its own segmental innervation and its own projection area in the cerebrum. Hence it is obvious that the cerebral cortex will easily differentiate between a left-side or a right-side pain, and no one will question that a C5 pain on the left side has a left-side origin and vice versa. The fact that pain stemming from a unilateral structure does not cross the midline, becomes important in the interpretation of more or less centrally localized pain, for instance in aches in the neck or back. It is evident that a lesion of a unilateral facet joint will not cause pain radiating all over the lower back. Only centrally localized structures (vertebral body, longitudinal ligaments, intra- and supraspinal ligaments and dura mater) can theoretically be responsible for a bilaterally radiating pain at both sides of the midline. A pain felt centrally or bilaterally must originate from a central structure, or from two bilateral structures (two facet joints or two sacroiliac joints) but it can never be the result of a lesion in a unilateral structure.

Dura mater an 'exception' to segmental reference

Pain originating from the dura mater has a rather peculiar behaviour. First, in that the dura is a midline structure, it is innervated from both sides, so that pain refers bilaterally. Second, pain stemming from the dura has a very broad reference and seems to cover several consecutive dermatomes. For instance, pressure of a lumbar disc on the dura at the L5 level can cause pain in the back which radiates to the abdomen and groins, down to the anterior and posterior aspect of both thighs and legs, and upwards to the back of the lower chest. This type of pain reference is inexplicable in terms of segments. We therefore call it '*extrasegmental reference*' of pain. Because in orthopaedic medicine, the dura is the exclusive source of this type of pain radiation, it is also called *dural reference*. Pain of this nature can be very difficult to interpret if it is felt in a part or parts of the possible reference area. As in purely segmental reference, dural reference can be to only a part of the respective dermatomes. Thus, instead of the broad radiation to the whole back, both glutei and both legs, dural pain sometimes only affects a small part, for instance one groin, or one buttock or the whole posterior aspect of the thigh. The differential diagnosis from a more segmental pain, for instance as the result of a nerve root compression, is then difficult.

A common clinical finding in a cervical disc protrusion that impacts on the dura is unilateral interscapular pain or pain in the trapezius or pectoral area. In the latter instance, suspicion of angina pectoris may then easily arise. Also the removal of an appendix for dural pain of lumbar origin referred extrasegmentally into the iliac fossa and groin is not at all exceptional.

A possible explanation for the misleading pain reference of the dura may lie in its multisegmental origin, which is reflected in the great overlap between the fibres of the consecutive sinuvertebral nerves innervating its anterior aspect.[70–73] More recent researchers describe division of the nerves into ascending and descending branches which ramify variously, to give off longitudinally and transversely orientated branches.[74,75] In a recent study that used the very sensitive acetylcholinesterase method, more ramifications between the nerve branches were demonstrated (Fig. 1.19).[76] Ascending branches up to four segments cranial to the level of entry into the dural nerve plexus and also descending branches extending up to four segments caudally were observed. In addition, many vertical and horizontal interconnections between the various ascending and descending branches were seen. The conclusion is that dural nerves may spread over *eight segments* and that a great overlap exists between adjacent and contralateral dural nerves.

These findings may form an anatomical explanation for the clinical observations of Cyriax on the limits of extrasegmental reference of dural pain.[77] Upwards, pain originating in the lower cervical part of the dura may spread to the occiput, skull and forehead (Fig. 1.20). Downwards it can descend to T7 which corresponds with the lower angles of the scapulae. Anteriorly the pain can occupy the whole pectoral area. Extrasegmental pain does not extend beyond the upper half of the arms.

A middle thoracic disc lesion can cause dural extrasegmental pain that may radiate to the base of the neck, and downwards to the whole abdomen and to the upper lumbar region (Fig. 1.21).

Dural extrasegmental reference from a low lumbar level may reach the lower thorax posteriorly, the lower abdomen deep to the umbilicus, the groins, the buttocks, and the sacrum and coccyx (Fig. 1.22). Unlike the cervical dura – which does not radiate far into the arms – extrasegmental reference from a lumbar origin may also involve the legs, both the anterior and the posterior aspects, and descend to the ankles.

Referred tenderness

There seems to be more to referred pain than just mislocation by the patient. Within or near the area of referred pain, it is often possible to find small *trigger points* which are exquisitely

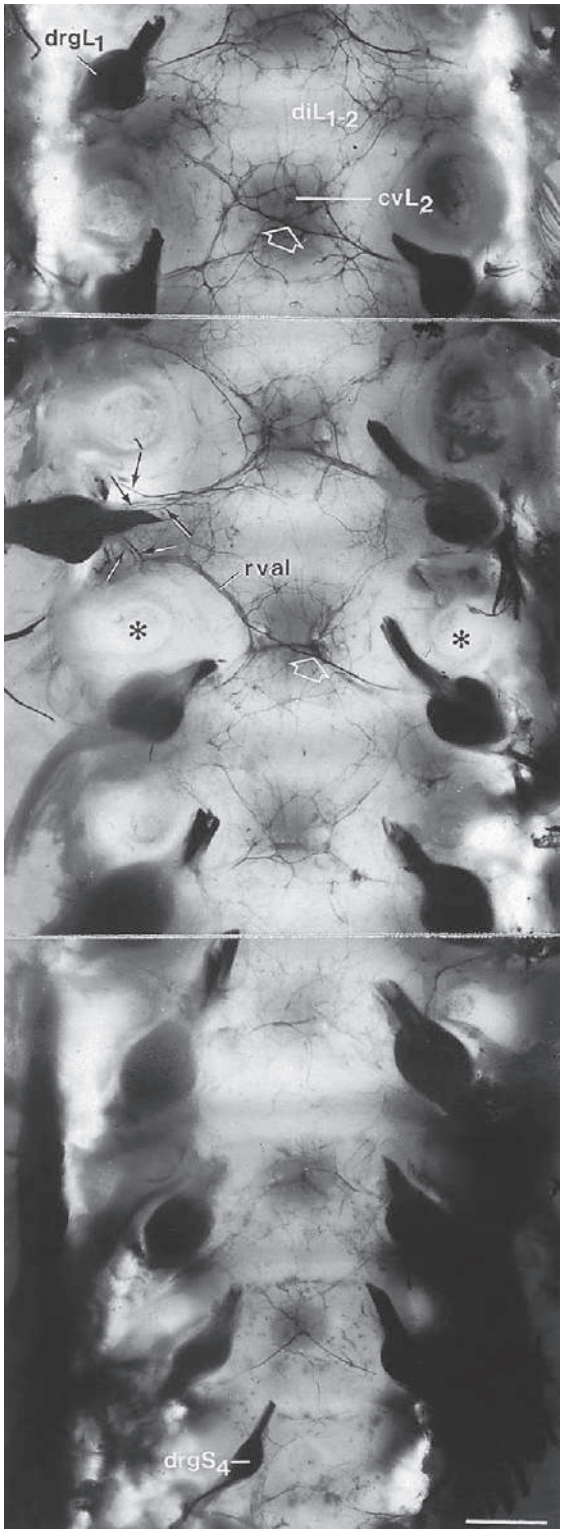

Fig 1.19 • The nerve plexus of the lumbosacral posterior longitudinal ligament (L1 to S4). Dorsal view after complete laminectomy and after removal of the spinal cord and the ventral dura. *, cut pedicle of a ventral arch; cv, vertebral body; di, intervertebral disc; drg, spinal ganglion; rval, ventral ramus of the spinal branch of the lumbar artery; small arrows, sinuvertebral nerves entering the vertebral canal; open arrows, crossing sinuvertebral nerves. Reproduced with permission from Groen GJ. Nerves and nerve plexuses of the human vertebral column. *Am J Anat* 1990;**188**:282–96.

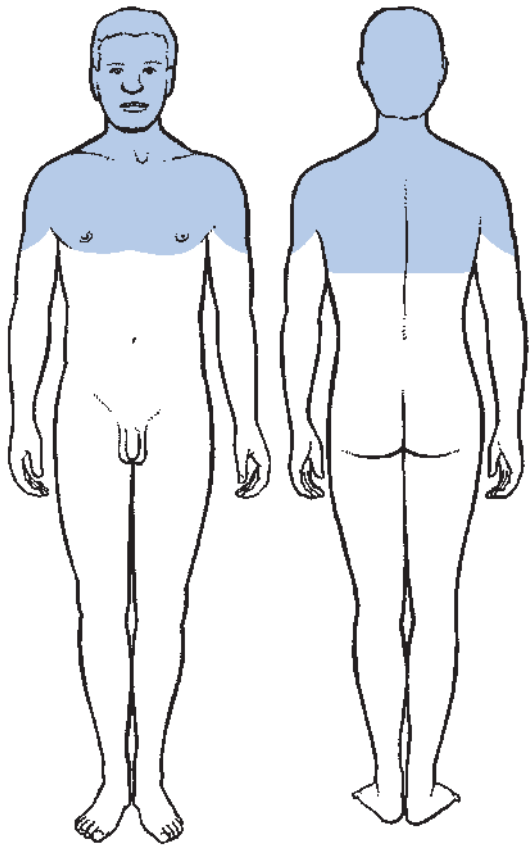

Fig 1.20 • Limits of dural multisegmental pain of cervical origin.

sensitive. Pressure on one of these immediately produces a deep and radiating tenderness which is identified by most patients as the source of their symptoms. Classic localizations of these tender spots are:

- In cervical dural compression, the upper border of the trapezius, the scapular muscles or the base of the neck.
- In lower lumbar dural involvement, the sacroiliac region and the upper part of the buttocks.

If the tender area is palpated without the prior conduct of a proper functional examination of the neck or lumbar spine, the tenderness is regarded as the primary lesion, the more so because the patient insists that it is the apparent origin of the pain. It is no wonder then that 'fibrositis', 'myofibrositis' or 'myofascial pain' syndromes have long been regarded as primary lesions. The 'fibrositis' concept has been used to explain the cause of lumbago.[78] Lewis[33] was the first to recognize that trigger points and myalgic spots were not primary lesions and that the painful area, although tender to the touch, did not contain a focus.

Cyriax considered that the localized tender area is a secondary effect of pressure on the dura mater.[79,80] He derived his theory from the simple clinical observation that the tender spot shifted from place to place over a few seconds after a successful manipulation and that, when a full and painless range of motion had been restored, the tenderness disappeared.

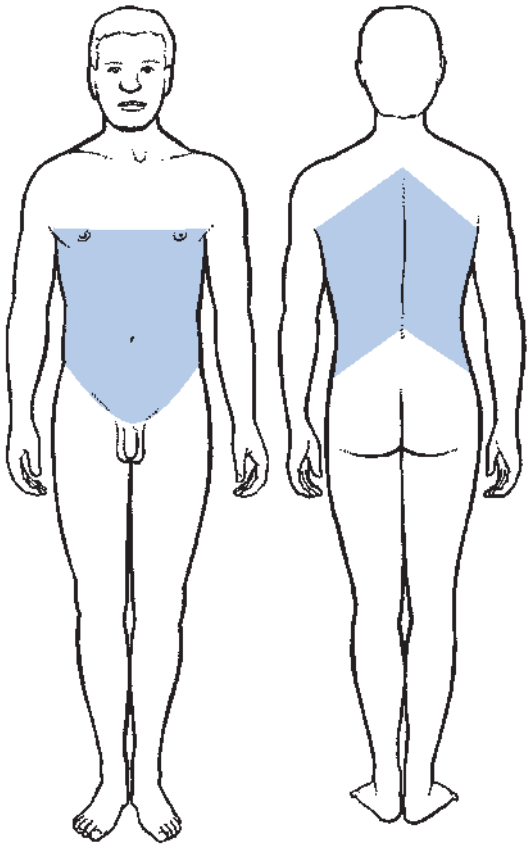

Fig 1.21 • Limits of dural multisegmental pain of thoracic origin.

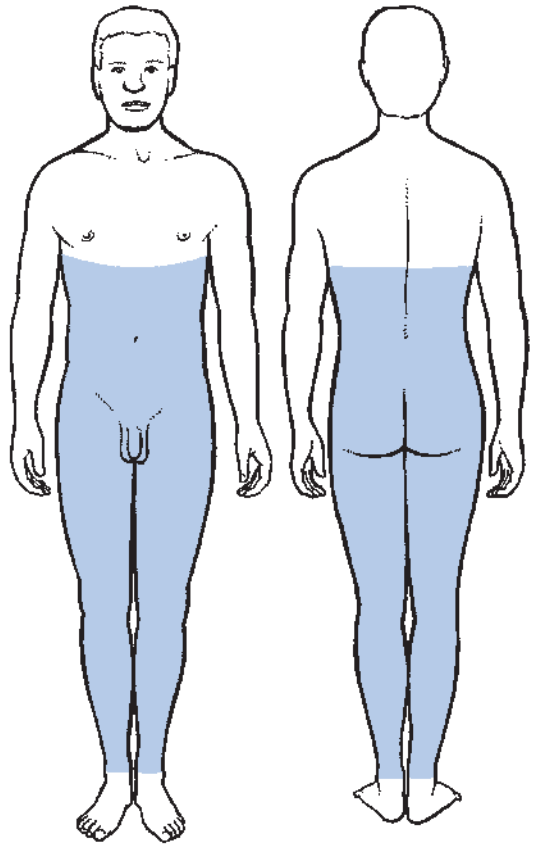

Fig 1.22 • Limits of multisegmental pain of lumbar origin.

However, referred tenderness has also been demonstrated in muscular and fibrous tissue lesions,[81] in visceral lesions such as ischaemic heart attacks[82] and in pathological viscera. The phenomena are not completely understood as yet but it is reasonable to assume that trigger points are caused by summation mechanisms which can be understood in terms of gate-control mechanisms.[79]

Summation – the excitatory effect of converging inputs – is an important pain mechanism. Pain may be triggered by two sources of nerve impulses: a major one from the lesion; and a minor one from normal skin which add together. If one source is removed, it becomes more difficult for the other to trigger the feeling of pain.[83]

Factors determining reference of pain

Referred pain is a faulty perception of the origin of a pain. In orthopaedic medicine it is common to see marked differences in the extent of reference between segments, between distinct affected tissues and between different degrees of the same condition.

The degree of reference – that is the distance between the localization of the perception and the site of the lesion – depends on four different factors (Box 1.3). Some can be explained on the basis of the known pathways, others remain unexplained and result purely from clinical observations.

Box 1.3

Factors favouring reference of pain

Strength of the stimulus:

- The stronger the stimulus, the more reference of pain

Position of the affected tissue:

- The more central the lesion, the more reference of pain
- The more distal the lesion, the less reference of pain

Depth of the affected structure:

- More reference from deep-seated structures
- Less reference from superficial structures

Nature of the affected tissue:

- Little reference: bone and periosteum
- More reference: muscle
- Much reference: capsule, ligament, bursa, tendon, dura, dural sleeve and perineurium

The strength of the stimulus

The greater the stimulus, the more extensive the reference of pain. In other words, intense stimulation radiates the pain widely and slight irritation localizes the pain closer to its origin.

This phenomenon is used in orthopaedic medicine to estimate the degree of irritation or to evaluate treatment. A

classic example is the C5 pain that results from shoulder arthritis. In this disorder there is a typical development of pain which gradually increases. At first, pain is felt only in the deltoid area, close to the shoulder. During the following months it gradually spreads to the arm, first above the elbow but later also to the forearm. At its worst, the pain can even be felt at the distal end of the radius and the base of the thumb. If treatment is successful or the condition regresses, the pain gradually leaves the forearm and moves upwards until it is only at the shoulder area. That the area of reference of pain becomes smaller indicates that the strength of the stimulus is decreasing and inflammation improves. Serious sacroiliac arthritis can provoke pain in the whole of the S1 and S2 dermatomes, with pain at the back of the thigh and the leg, down to the heel and the sole. If the condition improves, pain first leaves the heel and the calf. Further regression of the arthritis will probably result in localized pain in only the neighbourhood of the sacroiliac joint, with some reference to the buttock.

The mechanism of this phenomenon is probably based on the fact that the more the peripheral sensory nerve fibres are stimulated, the more there is cerebral cortical activity.[84]

The position of the affected structure

Pain seems to refer distally only; this being so, the closer to the midline the affected structure lies, the greater the possibility of extensive reference of pain. The further the lesion lies from the midline, the more accurate will be the localization of the lesion by the pain it provokes. As a rule, a lesion in the wrist or in the ankle does not give rise to diffuse pain and the patient usually knows quite well where the source is. Lesions in the hand or foot can therefore be precisely indicated. Also lesions in the elbow and knee cause quite well-defined pain that does not radiate enough to confuse either the patient or the examiner. Lesions that involve the shoulder, the hip, the sacroiliac joints and the spine usually provoke extensive pain reference. The segments to which the shoulder, hip and sacroiliac joints belong (C5, L3 and S1–S2) have the longest dermatomes of the body; as a consequence, pain can be referred a long way distally – calf pain in sacroiliac arthritis or knee pain in arthritis of the hip.

The depth of the affected structure

As early as 1939, Kellgren[35], and Lewis and Kellgren[85] stated that the localizing ability of a structure depended largely on its depth from the surface. This was confirmed later by other studies.[38,44]

Pain originating from a superficial lesion is usually pinpointed correctly by the patient but deep lesions can cause wide reference. This follows immediately from the way referred pain originates. We have seen that referred pain is an error of perception and that pain memory is based upon the experience gathered by recurrent stimuli through the skin, which is adapted correctly to localize pain. It follows that the deeper structures lie from the skin, the less the chance that they will be stimulated by external factors. When internal (pathological) factors activate the nociceptors in these deeply situated structures, the memory mechanism is inadequate and places the pain within the affected segment.

The nature of the structure

There are many discrepancies in our knowledge of pain referral and further research is required to clarify why some structures give more pain reference than others. For example, pain originating in bone or periosteum, although usually located deeply, hardly radiates at all. This does not mean that bone pain cannot be very severe, but it seldom gives extensive reference. This phenomenon is of great value in clinical diagnosis. Serious but localized pain always points to the possibility of a lesion of the bone. For example, an intense but localized pain in the lumbar spine is typical of a bony lesion, such as fracture, infection or new growth at a vertebra. Also intense, deep, but very localized pain in a limb draws attention to bone disease. In addition, severe but localized pain at the shoulder, precisely felt at the true site, is always an indication of a bony lesion.

Pain stemming from a lesion in a joint capsule, bursa, ligament or tendon is referred in an uncharacteristic way – the degree of pain reference is not determined by the type of tissue involved. Pain originating in a muscle seems to cause less reference than pain stemming from the tendon or the tenoperiosteal insertion.

Intense pain reference can also result from pressure on the different parts of the peripheral nervous system. Depending on the localization of the compression, the reference will be segmental or extrasegmental (see earlier).

Access the complete reference list online at **www.orthopaedicmedicineonline.com**

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Pressure on nerves

2

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Anatomy

Peripheral nerves contain both neural and supportive elements. A large multifascicular nerve is composed of a number of different bundles of nerve fibres or fasciculi (Fig. 2.1). These are bound together by the epineurium, a condensation of areolar connective tissue derived from the mesoderm. In humans, the epineurium normally constitutes 30–50% of the total cross-sectional area of the nerve bundle: it contains fibroblasts; collagen (types I and III); variable amounts of fat (possibly to cushion the nerve fibres it surrounds); lymphatic; blood vessels (vasa vasorum); and free nerve endings. In a monofascicular nerve, the epineurium only surrounds the fasciculus and is fused with the perineurium.

The perineurium surrounds and protects one fascicle. It has two different layers; an outer collagen-rich connective one and an inner epithelial layer of contiguous cells. The perineurium has an important role in maintaining the osmotic milieu and fluid pressure within the endoneurium and also acts as a barrier against chemical and bacterial invasion.[1]

The connective tissue of peri- and epineurium possesses blood and lymph vessels – the so-called vasa vasorum.[2,3] Also free nociceptive nerve endings which come from the related multifascicular nerve trunks are embedded in the perineurium and epineurium.[4,5]

Enclosed in the perineurium is the fasciculus – a bundle of nerve fibres bound together and protected by the endoneurium. The latter consists of long collagen fibres running with the nerve fibres. The fibrous and cellular components of the endoneurium are bathed in endoneural fluid.[6]

The nerve fibres are axons – the distal offshoots of nerve cells (Fig. 2.2). Most axons are surrounded by a myelin sheath formed from the compressed and concentric Schwann cell membranes. Axons range in diameter from 0.2 μm (small non-myelinated nociceptive axons) to 20 μm (large and myelinated efferent motor axons) and in length from 1 to 100 cm. They contain most of the cell volume.

From central to peripheral, the nervous system can be clinically divided into four zones (Fig. 2.3):

- The spinal cord
- The spinal nerve, which contains fibres belonging to one segment
- In the brachial and sacral area, and distal from the spinal ganglion, the different spinal nerves form a nerve plexus, from which originate the large multifascicular nerve trunks
- Further distally the trunks split into peripheral nerves, with motor, sensory or combined function.

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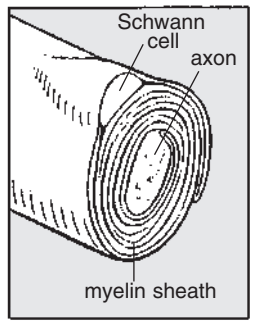

Fig 2.1 • A peripheral nerve is composed of a number of different fasciculi.

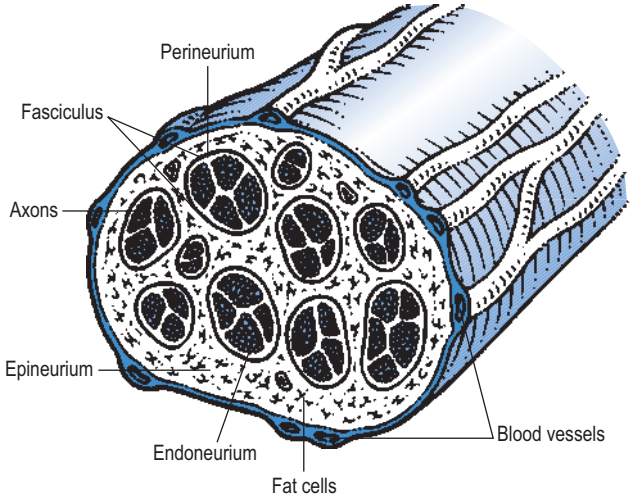

Fig 2.2 • Axon, surrounded by myelin sheath.

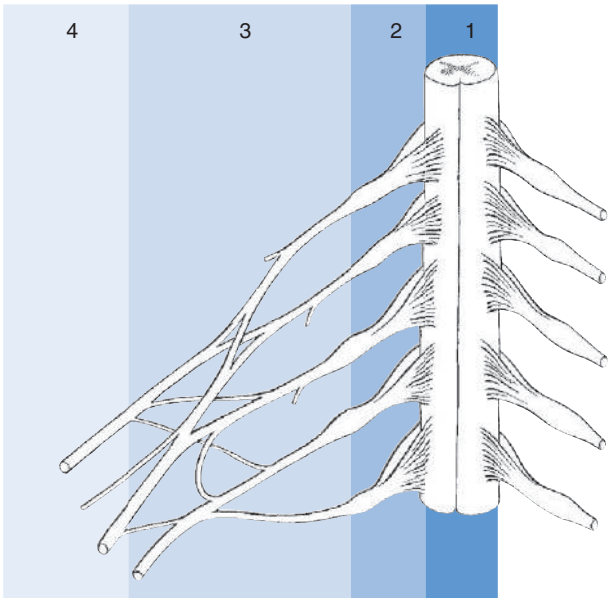

Fig 2.3 • Anatomy of the peripheral nervous system: 1, spinal cord; 2, nerve root; 3, nerve plexus and nerve trunk; 4, peripheral nerve branches.

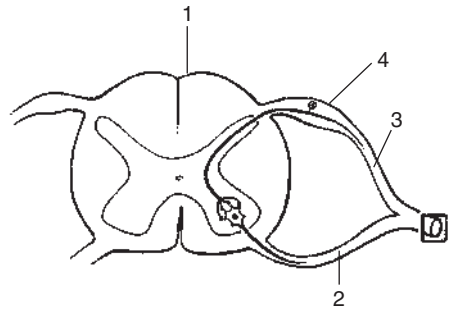

Fig 2.4 • Structure of an intraspinal root: 1, spinal cord; 2, ventral root; 3, dorsal root; 4, dorsal ganglion.

Although the fasciculi, from their exit from the central nervous system to the distal extremity of the nerve, exhibit the general morphology summarized above, the structure and behaviour of the connective supportive elements differ considerably. This may explain the different clinical behaviour of a compressed cord, a compressed nerve root (within or without its dural sleeve), a compressed nerve trunk and a compressed small peripheral nerve.

Roots

The course of the spinal nerve within the spinal canal, from the emergence of the rootlets at the anterior and posterior aspect of the spinal cord to the outer border of the foramen, is called the intraspinal root (Fig. 2.4).[7] Being inside the meningeal membranes of the spinal cord, the posterior and anterior roots are devoid of the elaborate epineural and perineural membranes that are characteristic of peripheral nerves.

Proximally, the rootlets float freely within the cerebrospinal fluid which is the main source of their metabolic needs. In this intrathecal part of the intraspinal root, the rootlets are held together by the endoneurium which is more loosely arranged than is typically seen in peripheral nerves.[8] Further distally, the nerve root becomes enclosed in the dural sheath – a tubular prolongation of the dura. In this dural investment the nerves do not lie freely but are bound by the arachnoid membrane (Fig. 2.5).[9,10] This area is known as the extrathecal part of the intraspinal nerve root – that length of the root *and* the dural sleeve between the main dural sac and the exit from the foramen. The extrathecal portion is short in the cervical region but becomes longer with the increasing obliquity of the intraspinal roots in the thoracolumbar and lumbar regions.

Distal to the posterior root ganglion, at the level of the foramen, the posterior and anterior roots are fused into one single bundle. Here the nerve root becomes extraspinal. The tissue of the epidural pouch becomes more condensed and blends with the epineurium of the extraspinal nerve root.

The segment of the spinal root which is liable to compression, whether by a disc protrusion, an osteophytic outgrowth or a narrow lateral recess, is the extrathecal part of the intraspinal root. To understand the symptom of a compression or inflammation of this part of the peripheral nerve system, it is necessary to recognize the importance of the dural investment

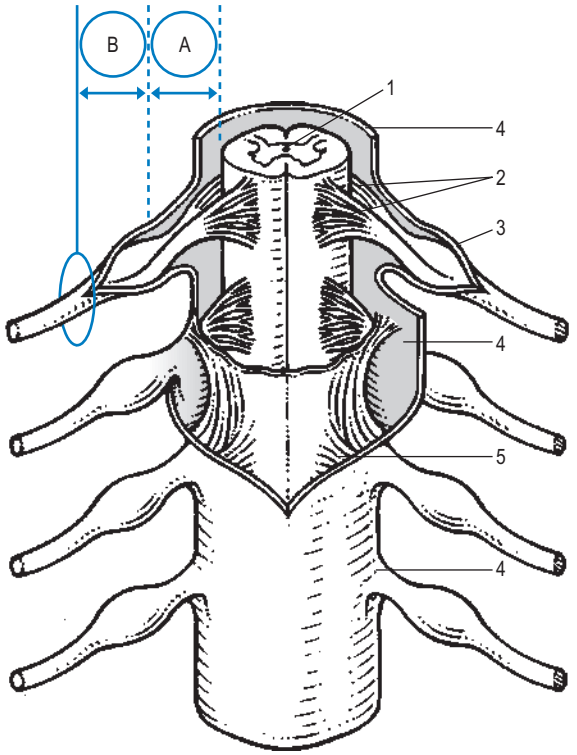

Fig 2.5 • Course of spinal nerve: A, intrathecal part of the nerve root; B, extrathecal part of the nerve root; 1, spinal cord; 2, ventral and dorsal rootlets; 3, spinal ganglion; 4, dura mater; 5, arachnoidea.

– the nerve root sheath. The dural sheath has considerable sensitivity:[11] it has many nociceptive nerve endings, especially at the anterior aspect where it receives its innervation from the sinuvertebral nerve belonging to the same segment.[12–14] Pain arising from the dural sheath is segmental and obeys the rules of segmental reference of pain (see Ch. 1). There is no evidence that root pain arises from involvement of the axons. For example, pressure at the extraspinal nerve root, which misses the nerve root sleeve, as happens in some types of spondylolytic compression, causes not pain but only paraesthesia and neurological deficit.

From a clinical point of view, it is important to consider this structural duality: the outer investment is responsible for segmental pain, whereas the parenchymatous content is responsible for the paraesthesia and the conduction deficits.

Nerve plexus and nerve trunk

Immediately distal to the foramen, the single fasciculus of the extraspinal root is enclosed in a thin, but strong, perineurial sheath, external to which is the epineurial areolar connective tissue. Within a few millimetres of its formation, the single fasciculus of the spinal nerve divides into several bundles which form the plexuses. Motor and sensory fibres of one nerve root mix, and more distally there is a redistribution of the fasciculi of various consecutive nerve roots.[15] The brachial plexus is thus formed of the anterior rami of roots C5–T2, and the sacral plexus of the roots L2–S5. Distally, the fasciculi continue in the large nerve trunks of the limbs.

The fasciculi of plexus and trunks do not differ significantly from those of the roots or the peripheral nerves. The connective support tissue, however, has some anatomical particularities. Because the monofascicular spinal nerve changes into a multifascicular structure, there is an increased amount of epineurial tissue, forming a protective packing for the nerve tissue. The perineurium is also reinforced by elastin fibres. The fasciculi have an undulating course, whereas the collagen fibres run more longitudinally. This structure ensures that the nerve fibres are protected from mechanical deformation (compression and elongation) during normal movements of the limbs.[16] Although the epi- and perineurium contain nociceptive nerve endings, these seem to be relatively insensitive.[17]

Small peripheral nerve

A small peripheral nerve is the distal termination of a branching nerve trunk. The nerve ending is often monofascicular. The epineurium is then fused with the perineurium. Peripheral nerves can either have only a motor or a sensory function, or both can be combined. They all have nociceptive nerve endings in their supporting connective tissue.

Terminology

Lesions of the peripheral nervous system are characterized by a pathognomonic sensation: paraesthesia ('pins and needles'). Although all tissues in the human body which contain nociceptive structures can be a source of pain, pins and needles will only arise when some part of the peripheral nervous system is at fault. Hence, the medical world tends to use the term 'neuritis' when pain is accompanied by pins and needles. Strictly, however, the suffix '-itis' implies inflammation. Therefore the word neuritis should only be used when the peripheral nerve is affected by infectious or toxic irritation – i.e. there is an intrinsic disorder of the nervous parenchyma. Classically, these lesions are classified into mono- and polyneuritis. They are not discussed in this book, except in the shoulder region, where the clinical appearance of three mononeurites and neuralgic amyotrophy of the shoulder girdle is reviewed (see online chapter *Nerve lesions and entrapment neuropathies of the upper limb*).

When external pressure is applied to a normal nerve, pins and needles arise, although the nerve tissue is initially not inflamed. If the compression is not severe, the nerve continues to conduct normally. This situation of extrinsic pressure on a normal nerve is not a 'neuritis' and requires a totally different therapeutic approach. In this situation, the terms 'pressure on nerve' or 'entrapment neuropathy' are preferred.

Pain originating from the peripheral nerve system

Nociceptive pain

Peripheral nociceptors in the connective tissue of the peripheral nerves are stimulated, and via Ad and C fibres of the nervi

nervorum conducted to the spinal cord and thence to the pain projection areas in the cortex.[18] There are indications that most of the pain that stems from direct irritation of the peripheral nervous system is of nociceptive origin.[19,20] 'Nerve pain' thus behaves identically to other peripheral pain,[21] obeys exactly the rules of referred pain and is not to be distinguished from pain of ligamentous, tendinous or arthrogenic origin (see Ch. 1).

Neuropathic pain

This type of pain, also called 'de-afferentation or neuralgic pain' is less common than nociceptive pain and results from prolonged damage to peripheral nerve tissue, such as avulsion, dissection or amputation.[22] The pain is felt in the anaesthetic area, is continuous and burning and is independent of posture or movement, although local pressure can increase the pain considerably.[23]

Chronic damage or formation of scar tissue seems to provoke pain mechanisms without involvement of peripheral nociception. Also, the formation of a neuroma leads to increased sensitivity and spontaneous pain.[24] Research on experimental neuromata has shown that regenerating axons have a spontaneous excitability and an increased sensitivity to mechanical stimuli. An action potential in one axon probably leads to an impulse in a nearby axon. This mechanism of 'cross-talking fibres' accounts for the repetitive train of action potentials in a bundle of regenerating axons.[25] A small stimulus thus leads to a self-perpetuating series of action potentials, and excessive and long-standing pain.[26]

Another mechanism that may account for neuropathic pain is the loss of inhibitory effects of the large diameter mechanoreceptor afferents in a traumatized nerve. This leads to a relative increase of the activity from the small nociceptive afferents, and thus an opening of the gate at the dorsal horn.[27] (see Ch. 1).

Superficial dysaesthetic pain

This type of pain is also rare, and is typical of diffuse polyneuritis, for example in diabetes,[28] vitamin B_1 deficiency or chemical irritation. Damage to small C fibres leads to sprouting of small offshoots in the regenerating axons. This leads to increased excitability, which results in unpleasant painful sensations during normal stroking of the skin (allodynia).[29] The patient also complains of a burning feeling and 'electrical sensations' when the skin is gently touched (dysaesthesia), and there is also some analgesia (see Box 2.1 for an overview of neurogenic pain).

Box 2.1

Neurogenic pain

Nociceptive pain

- Common
- Irritation of nociceptive structures in the connective support tissues of the nerve
- 'Normal' pain sensation, obeys the rules of segmental reference

Neuropathic pain

- Rare
- Results from damage to the nerve tissue
- Intense pain, sharp, burning

Superficial dysaesthetic pain

- Rare
- Polyneuritis: irritation of C fibres
- Dysaesthesia, allodynia and anaesthesia in the innervation area of the peripheral nerve

Behaviour of nervous tissue during pressure

Entrapment of peripheral nerve tissue is defined as mechanical compression of the nerve, which includes the reduction of radial dimensions in the neural cells, the neural support elements or any combination of these. Depending on the degree and the duration of compression, the effects can be subtle or can lead to displacement, deformity and morphological changes in the compressed tissue (neural tissue or neural support tissue).[30]

The clinical effects of nerve compression are pain, paraesthesia and loss of function (see Box 2.2 for an overview of pressure on nerves).

Pain

The pain mechanism in entrapment phenomena is usually nociceptive: free nerve endings in the connective tissue of the nerve or in the dural investment of the nerve root are depolarized by application of mechanical forces or after exposure to irritating chemical substances, released from inflamed tissues.[31] The pain stems from irritation of the support tissue enclosing the nerve fibres and only exceptionally does it result from pathological processes in the nerve tissue itself (neuropathic and dysaesthetic pain). This has the following clinical consequences.

The pain will depend largely on the density of the nociceptive receptors in the support elements. It follows that the intensity of the pain depends not only on the intensity of compression but also on the localization along the course of the peripheral nerve. Pressure on a nerve root, for instance, will be more painful than an equal degree of force applied on a nerve plexus.

Because an external force acts first on the outer supporting structures of the nerve, pain will usually be the first symptom and it sometimes appears before involvement of the parenchyma is present. A chronic but moderate pressure that is insufficient to impair conduction solely influences the outer structures and results in pain only. It is thus possible to have a completely normal examination of the peripheral nervous system, even though the patient does have nerve compression.[32]

Paraesthesia

Pins and needles are pathognomonic of involvement of the peripheral nervous system in that the sensation cannot be produced in any way other than compression or inflammation of nerve tissue.[33] Paraesthesiae are always felt in the cutaneous area supplied by the nerve tissue involved and distal to the site of the lesion. It is therefore extremely important to ascertain the precise site of the symptom, in that this helps to determine the site of compression.

Provocation of pins and needles by movements (distant movements or local pressure) or by stroking over the affected skin demonstrates an external origin for the symptoms. In primary afflictions of the peripheral nerve (neuritis), the pins and needles come and go spontaneously and movements do not influence them.

Loss of function

The epineurium and perineurium initially buffer the fasciculi from constrictive effects, but with a greater amount of compression, structural changes of the elements within the endoneurium follow.[34] Recent research has demonstrated that the intraradicular oedema caused by alteration of the blood–nerve barrier is the most important factor in the nerve root dysfunction of chronic compression.[35,36]

Sometimes only the Schwann cells are affected, without damage to the axons. Destruction of the myelin sheath then results in loss of conduction. This type of lesion (lesion of Schwann cells without lesion of the axon fibre) is termed 'neuropraxis'. If the compression has been only temporary, recovery of the Schwann cells will not take more than 2 weeks. This is the type of lesion responsible for the 'Saturday night palsy' seen after prolonged pressure on the radial nerve, or the 'gardener's palsy' seen after prolonged traction on the peroneal nerve.

If considerable compression is maintained for a longer period, atrophy of the nerve tissue occurs and is followed by Wallerian degeneration of the distal part of the axon. Oedema, cellular proliferation and ingrowth of connective tissue also follows.[37] If the compression is maintained for long periods, fibrotic degeneration appears at the site of the lesion, which makes recovery most unlikely.[38]

Clinical syndromes

Cyriax[39] (see his pp. 37–39) distinguished four different syndromes in entrapment phenomena, corresponding to the site of compression along the peripheral nerve: at the small peripheral sensory nerve, at the nerve trunk/plexus, at the nerve root and at the spinal cord (see Fig. 2.3).

Depending on the localization of compression, the peripheral nervous system behaves differently. These differences are a major help to the clinician trying to ascertain the precise site of the lesion.

Small peripheral nerves

Pressure on a small peripheral sensory nerve results in pain, paraesthesia and numbness. Pain is usually moderate and the main symptom is numbness. Together with some paraesthesia, these symptoms occupy the appropriate area of supply, which is usually well defined with clear-cut borders. The patient can tell precisely where the cutaneous analgesia is felt and where sensation is still normal. The centre of the region is often completely anaesthetic. A typical example is 'meralgia paraesthetica', which results from compression of the lateral cutaneous femoral nerve of the thigh.

Nerve trunk/plexus

Minor and intermittent pressure on a nerve trunk or a plexus causes paraesthesia and numbness. Sudden and serious tissue damage may provoke neuropathic pain. Constant pressure on a nerve trunk leading to parenchymatous damage does not usually provoke pain nor paraesthesia but only loss of motor and sensory function.

If the pressure is intermittent, a neurological deficit does not appear, even after many years. Paraesthesiae do not appear during the time of compression but only when the pressure on the nerve trunk has been released. It is common knowledge that pressure on the sciatic nerve while sitting causes only vague analgesia in the affected area or no symptoms at all. The shower of pins and needles then only appears when the subject relieves the pressure by standing up. The interval between the cessation of the pressure and the onset of the tingling depends on the duration of the compression: the longer the pressure is applied, the longer is the interval between the relief of pressure and onset of symptoms.

There is also a relation between the duration of compression and the duration of paraesthesia. Thus, after 15 minutes of pressure, the pins and needles appear 20–60 seconds after the release and last only 1 or 2 minutes. After release from 15 hours' compression, paraesthesiae will probably appear only after an interval of some hours, then persist for 1 to 2 hours before recovering spontaneously. Cyriax[39] calls this strange and hitherto unexplained phenomenon the 'release phenomenon' (see his p. 37). Lundburg and Rydevik have demonstrated that fluctuations in membrane permeability of the structures within the endoneurium are more noticeable when compression on the nerve trunk is released and oedema appears, than during the compression of the nerve and its supplying blood vessels.[40] This might explain the release phenomenon.

Another characteristic of paraesthesia induced by compression at the level of the plexus is that active movements of the limb or the digits, or stroking over the analgesic area of skin usually brings on or increases the pins and needles. It is a common experience that the paraesthesia in the feet that comes on after relieving pressure on the sciatic nerve increases when the subject walks around or stamps the feet on the ground.

Paraesthesia and numbness are usually felt in the distal part of the cutaneous area supplied by the compressed plexus or nerve, no matter at what point in its course the compression

occurs. The lesion lies proximal to the upper edge of the paraesthetic area but not always close to it. Therefore it must not be thought that, if movement of the foot provokes the pins and needles, the lesion lies in the ankle or foot. Clinical examination must include the entire length of the nerve involved.

The release phenomenon is typical of compression of the sacral plexus and the nerve trunks of the lower limb. The phenomenon is also provoked by compression at the brachial plexus (thoracic outlet syndrome) but ceases to operate when a nerve trunk distally to the thoracic outlet is compressed. It is a curious clinical fact that pressure on a nerve in the upper limb only provokes paraesthesia during the moment of compression. Pins and needles in the two ulnar fingers, caused by compression of the ulnar nerve at the elbow, immediately stop when the compression is released. Similarly, in carpal tunnel syndrome, the paraesthesiae are felt during the moment of compression.

Compression of the nerve root

Pressure on the extrathecal intraspinal nerve root results in a typical set of symptoms (pain and paraesthesia) and signs (motor and sensory deficit) strictly related to the segment involved. Contrary to the pins and needles brought on by the release of pressure on a nerve trunk, the paraesthesiae only appear during the period of compression, and cease immediately thereafter.

The sequence of appearance of pain, paraesthesia and deficit and their mutual interrelation have important clinical significance and are often of considerable help in the diagnosis of nerve root compression.

Pain

The nerve root has a dural sheath, which is innervated by the sinuvertebral nerve.[12] The latter is derived from the corresponding nerve root. Therefore pain originating from the dural sheath is strictly segmental and follows the rules of segmental reference of pain. Compression applied to the dural sleeve of the nerve root thus results in pain occupying all or any part of the dermatome. Pain felt in a particular dermatome in combination with other symptoms of nerve compression, immediately draws attention to an impingement on the nerve root.

Paraesthesia

Pins and needles stem from pressure on the parenchyma itself. In root pressure, they accompany the segmental pain, or appear at a later date. Pins and needles indicate that the parenchyma itself is irritated, whereas radicular pain is a symptom of compression of the dural sheath.

In nerve root compressions, paraesthesiae are felt in the distal extremities of the dermatomes, which are areas often not supplied by a particular nerve trunk or nerve. As in compression of a nerve trunk, stroking the skin may provoke or increase the pins and needles, but moving the digits does not influence them.

Deficit

The absence of the protective packing by epineurial tissue renders the nerve roots more susceptible to direct compression than nerve trunks. Compression disturbs nerve conduction by interfering with the blood supply of the nerve fibres.[41] Loss of function of the nerve fibres results in sensory and motor deficit. Paraesthesiae usually disappear with the onset of cutaneous analgesia.

Progressive compression of a nerve root within its dural sleeve causes a typical sequence of symptoms: pain, paraesthesia and numbness will follow each other, rather than coincide. This is typically the case in a progressively increasing pressure exerted by an evolving disc lesion: slight compression on the epidural sheath of the nerve root causes pain only (Fig. 2.6a). As the pressure increases, paraesthesia and muscle fasciculations – symptoms of parenchymatous hyperexcitability – appear[42] (Fig. 2.6b). In the final stage, pressure has induced such ischaemic damage to the nerve root that function is completely lost, including the conduction of pain (Fig. 2.6c). The patient then complains of weakness and numbness, but pain and paraesthesia have disappeared.

Spinal cord

Pressure on the anterior aspect of the spinal cord results, *inter alia*, in bilateral paraesthesia. Pain is absent if the compression is slowly progressive, but dural pain may accompany the paraesthesia if the compression is sudden. The main cause

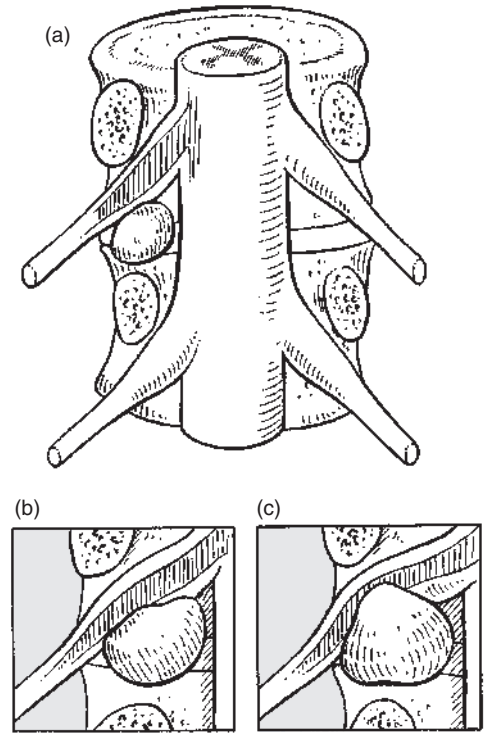

Fig 2.6 • Progressive compression of a nerve root.

Box 2.2

Pressure on nerves: Summary

Peripheral nerve	Numbness > paraesthesia > pain
	Clearly delineated area
Nerve trunk	Paraesthesia > numbness > pain
	Vaguely delineated area
	Release phenomenon:
	—The pins and needles appear after the compression has ceased
	—There is a direct relation between the interval of onset and duration of compression
Nerve root	Pain > paraesthesia > numbness
	Segmental distribution of symptoms
	Chronological sequence of symptoms
	Compression phenomenon:
	—The pins and needles appear during the compression
Spinal cord	Completely painless (sometimes dural pain)
	Extrasegmentally and bilaterally distributed paraesthesia
	Positive neck flexion

of spinal cord compression is spinal stenosis at the cervical or thoracic level. When the cord is compressed over the thoracic region, the paraesthesiae are felt only in the limbs. In compression at the cervical level, pins and needles will be present in all limbs or in the lower limbs only. The paraesthesiae are usually bilateral and extend beyond the borders of the areas of the cutaneous innervation of any spinal nerve, nerve trunk or peripheral nerve. For instance, the patient may complain of pins and needles in both hands and forearms at both aspects or in both legs from the knees to all the toes.

The symptoms are provoked neither by movements of the limbs nor by stroking the skin. Neck flexion is the only way to bring on the pins and needles (L'Hermitte's sign). Together with the extrasegmental and bilateral distribution of the painless paraesthesiae, positive neck flexion provides the clue to the diagnosis of incipient spinal cord compression.

Access the complete reference list online at
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Connective tissue

3

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Structural composition

Together with muscle, nerve and epithelium, connective tissue is one of the basic components in the human body. It binds structures together, helps in mechanical and chemical protection and also plays a principal role in reparative processes.

Connective tissues are defined as those composed predominantly of the extracellular matrix and connective tissue cells. The matrix is made up of fibrous proteins and a relatively amorphous ground substance. Many of the special properties of connective tissues are determined by the composition of the matrix, and their classification is also largely based on its characteristics.

Connective tissue cells

Cells of general connective tissues can be separated into the resident cell population (mainly fibroblasts) and a population of migrant cells with various defensive functions (macrophages, lymphocytes, mast cells, neutrophils and eosinophils), which may change in number and moderate their activities according to demand.

Fibroblasts, the majority of cells in ordinary connective tissue, arise from the relevant undifferentiated mesenchymal stem cells[1] and are involved in the production of fibrous elements and non-fibrous ground substance (Fig. 3.1). During wound repair they are particularly active and migrate along strands of fibrin by amoeboid movements to distribute themselves through the healing area to start repair. Fibroblast activity is influenced by various factors such as the partial pressure of oxygen, levels of steroid hormones, nutrition and the mechanical stress present in the tissue.[2]

The other cell types are migrant cells and only occasionally present, such as: *macrophages*, *lymphocytes*, *mast cells*, and *granulocutes* (Table 3.1).[3,4]

Extracellular matrix (ECM)

The extracellular matrix is composed of insoluble protein fibres, the fibrillar matrix and a mixture of macromolecules, the interfibrillar matrix. The latter consists of adhesive glycoproteins and soluble complexes composed of carbohydrate polymers linked to protein molecules (proteoglycans and glycosaminoglycans), which bind water. The extracellular matrix distributes the mechanical stresses on tissues and also provides the structural environment of the cells embedded

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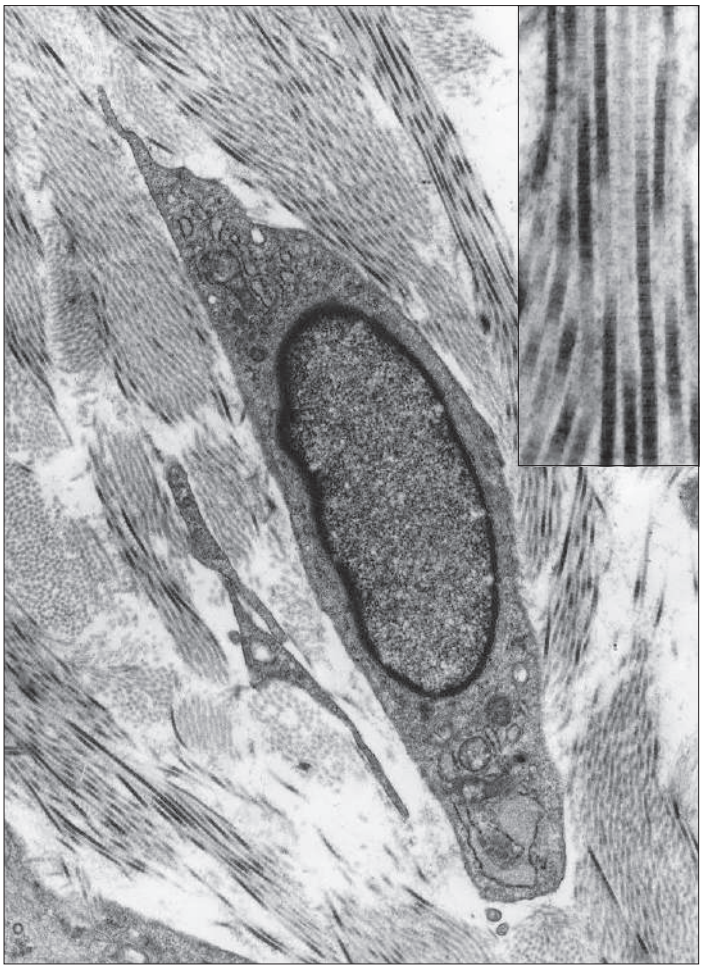


Fig 3.1 • Electron micrograph of a fibroblast in human connective tissue, surrounded by bundles of finely banded collagen fibrils (shown at high magnification in the insert) which they secrete. From Standring, Gray's Anatomy, 40th edn. Churchill Livingstone, Edinburgh, 2008 with permission.

Table 3.1 Connective tissue cell types

Connective tissue cells	Resident cells	Fibroblasts (adipocytes) (mesenchymal stem cells)
	Migrant cells	Lymphocytes Mast cells Granulocytes Macrophages
Extracellular matrix	Fibrillar matrix	Collagen Elastin
	Interfibrillar matrix	Proteoglycans Glucoproteins Water

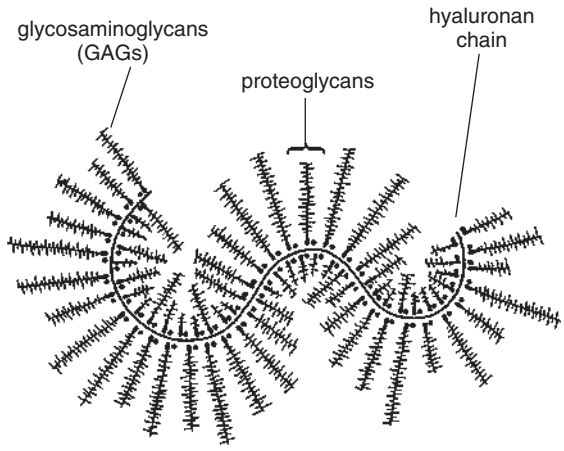


Fig 3.2 • The complex shape of proteoglycans. Polysaccharide molecules (GAGs) bound to a central protein core: proteoglycans are in turn bound to a long central chain of hyaluronan to form a proteoglycan aggregate. This arrangement is typical of cartilage. In other types of connective tissue hyaluronan chains may be absent. Redrawn from Walker PS[6] with permission.

in it, forming a framework to which they adhere and on which they can move.[5]

Non-fibrous ground substance

The interfibrillar ground substance is composed of proteoglycans (a family of macromolecules) which bind a high proportion of water (60–70%) and glycoproteins. The latter have a complex shape and are soluble polysaccharide molecules (glycosaminoglycans) bound to a central protein core. In cartilage, the proteoglycans are in turn bound to hyaluronan (a long chain of non-sulphated disaccharides) to form a proteoglycan aggregate – a bottlebrush three-dimensional structure (Fig. 3.2).[6] Glycoprotein secures the link between proteoglycan and hyaluronan and also binds the components of ground substance and cells.

The three-dimensional structure of the proteoglycan aggregates and the amount of water bound gives ground substance its high viscosity. A semi-fluid viscous gel is formed within which fibres and fibroblasts are embedded, so facilitating normal sliding movements between connective tissue fibres. In structures subject to high compression forces (e.g. articular cartilage), there is a large amount of proteoglycans but the content is relatively small in tissues such as tendons and ligaments exposed to tension forces.

Fibrous elements

The fibrous elements are collagen and elastin – both insoluble macromolecular proteins. Collagen is the main structural protein of the body with an organization and type that varies from tissue to tissue. Collagen fibres are commonest in ordinary connective tissue such as fascia, ligament and tendon. The fibrillar forms have great tensile strength but are relatively inelastic and inextensible. By contrast elastin can be extended to 150% of its original length before it ruptures. Elastin fibres return a tissue to its relaxed state after stretch or other considerable deformation. They lose elasticity with age when they

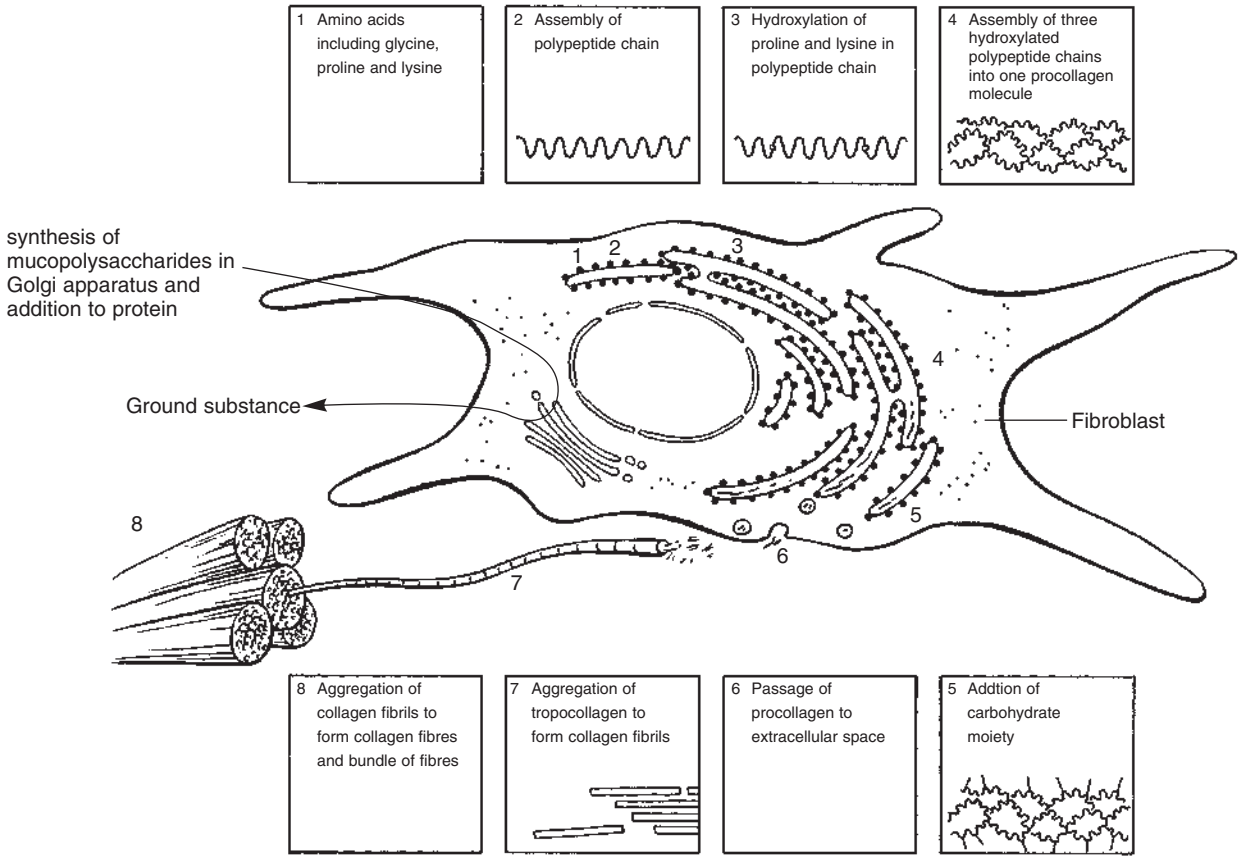


Fig 3.3 • The successive steps in collagen synthesis by fibroblasts.

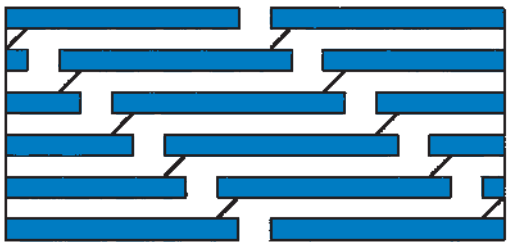


Fig 3.4 • Crosslinking and interspaces between head and tail of neighbouring tropocollagen molecules. They overlap each other by a quarter of their length. Molecules in the same parallel row are separated from each other by small interspaces.

tend to calcify. Box 3.1 outlines the components of connective tissue.

The basic molecule of collagen is *procollagen*, synthesized in the fibroblast, illustrated in Figure 3.3, steps 1–4. It is formed of three polypeptide chains (α-chains). Each chain is characterized by repeating sequences of three amino acids – glycine, proline and lysine joined together in a triple helix. The helical molecules are secreted into the extracellular space where they slowly polymerize and crosslink (Fig. 3.4). They overlap each other by a quarter of their length, lie parallel in rows and are collected into large insoluble fibrils. The fibrils unite to form fibres, finally making up a bundle. An aggregate of bundles makes up a whole structure such as a ligament or a tendon. The individual bundles are in coils, which increases their structural stability and resilience, and permits a small physiological deformation before placing the tissue under stress, and in consequence permits a more supple transfer of tractive power in the structure itself and at points of insertion (Fig. 3.5). The process of collagen synthesis is stimulated by some hormones (thyroxine, growth hormone and testosterone), although corticosteroids reduce activity.

Box 3.1

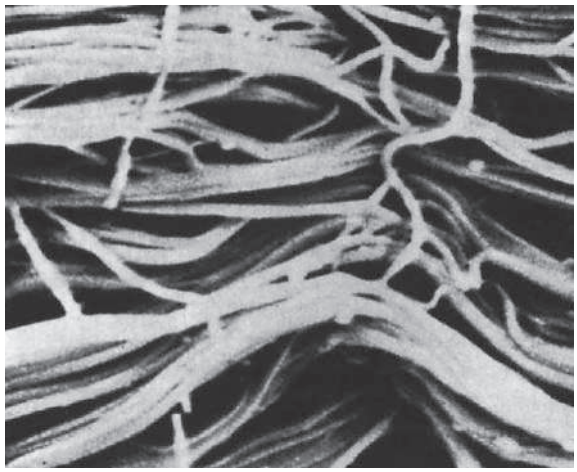
Components of connective tissue

Cells

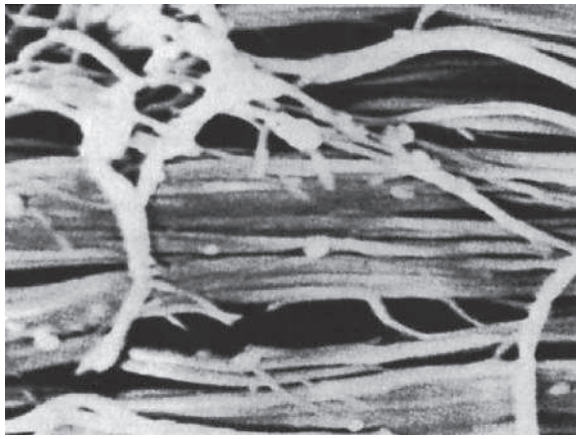
- fibroblasts → fibrous connective tissue
- chondrocytes → cartilage
- osteoblasts and osteocytes → bone

Extracellular matrix (ECM)

- fibres: collagen → framework of the ECM
- elastin → extensible element of ECM
- proteoglycans: hydrators, stabilizers and space fillers of ECM
- glycoproteins: stabilizers and linkers of ECM
- fluid



(a)



(b)

Fig 3.5 • (a) Unloaded collagen fibres in a human knee ligament. (b) Physiological deformation after stress. From Kennedy *et al*[7] with permission (http://jbjs.org/).

Connective tissue collagen can be classified into different types of which at least 14 are now genetically characterized and the others are being investigated. In the context of this book the most important are:

- *Type I*: the most abundant of all collagen. Strong thick fibres packed together in high density. It predominates in bone, tendon, ligament, joint capsule and the annulus fibrosus of the intervertebral disc.
- *Type II*: thin fibres found in articular cartilage and the nucleus pulposus of the intervertebral disc. They particularly function in association with a high level of hyaluronan and sulphated proteoglycans to provide a hydrated and pressure-resistant core.
- *Type III*: essentially present in the initial stages of wound healing and scar tissue formation. It secures early mechanical strength of the newly synthesized matrix. These relatively thin, weak fibres are replaced by the strong type I fibres as healing proceeds.

In relation to the degree of orientation of fibrous tissue elements, ordinary connective tissues can also be classified into *regular* and *irregular* types.

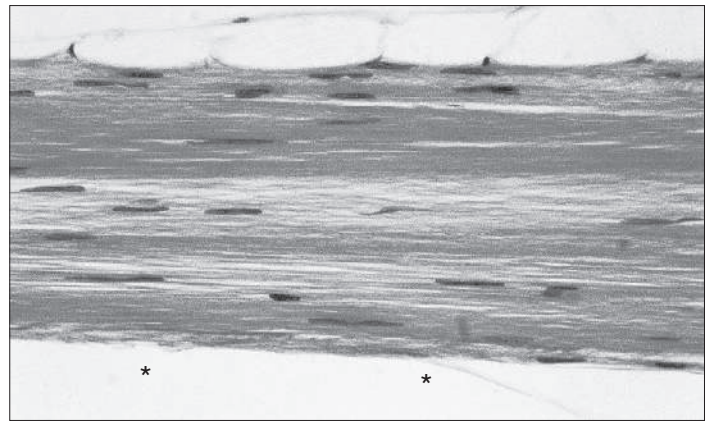


Fig 3.6 • Dense regular connective tissue in a tendon. Thick parallel bundles of type 1 collagen (asterisks) give tendon its white colour in life. The elongated nuclei of inactive fibroblasts (tendon cells) are visible between collagen bundles. From Standring, Gray's Anatomy, 40th edn. Churchill Livingstone, Edinburgh, 2008 with permission.

Regular types

Highly fibrous tissues such as ligaments, tendons, fascia and aponeuroses are predominantly collagenous and show a dense and regular orientation of the fibres with respect to each other. The direction of the fibres is related to the stress they experience. Collagen bundles in ligaments and tendons are very strong and rupture usually takes place at the bony attachments rather than by tearing within their substance (Fig. 3.6).

Irregular types

The irregular types consist of collagen and elastin interlacing in all directions. It is loose, extensible and elastic and found between muscles, blood vessels and nerves. It binds partly together, although allowing a considerable amount of movement to take place. In the sheaths of muscles and nerves and the adventitia of large blood vessels, the tissue is more dense with a high proportion of collagen fibres to protect these structures against considerable mechanical stress. The dura mater is also an example of an irregular connective tissue sleeve.

Vascularization

Connective tissue is poorly supplied with blood vessels. In dense fibrous tissues these usually run parallel to and between the longitudinal bundles with communicating branches across these.

Lymphatic vessels are more numerous, especially in loose connective tissues such as the dermis. They are also abundant in tendons and tendon sheaths.

Innervation

Dense connective tissues, for example, ligaments, tendons and fascia, have a rich supply of afferent nerve endings. The various sensory receptors transmit information to the central nervous system about changes in length and tension which allows constant monitoring of the position and movement of a joint as well as of injurious conditions that threaten these structures.

Structural and physiological studies[8,9] have shown the presence of at least four types of receptor. Three of these have encapsulated endings but the fourth consists of free unencapsulated endings:

- *Type 1* (Ruffini endings) are present in the superficial layers of a fibrous joint capsule. They respond to stretch and pressure within the capsule and are slow adapting with a low threshold. They signal joint position and movement.
- *Type 2* are particularly located in the deep layers of the fibrous capsule. They respond to rapid movement, pressure change and vibration but adapt quickly. They have a low threshold and are inactive when the joint is at rest.
- *Type 3* are found in ligaments. They transmit information on ligamentous tension so as to prevent excessive stress. Their threshold is relatively high and they adapt slowly. They are not active at rest.
- *Type 4* are free unencapsulated nociceptor terminals which ramify within the fibrous capsule, around adjacent fat pads and blood vessels. They are thought to sense excessive joint movements and also to signal pain. They have a high threshold and are slow adapting. Synovial membrane is relatively insensitive to pain because of the absence of these nerve endings.

All these receptors influence muscle tone via spinal reflex arcs which are formed by the same nerves that supply the muscles which act on the joint. Parts of the joint capsule supplied by a given nerve correspond with the antagonist muscles. Tension on this part of the capsule produces reflex contraction of these muscles to prevent further overstretching of the capsule. In consequence all receptors have an important function in stabilization and protection of the joint. After rupture of a capsule, ligament perception is considerably disturbed because of the disruption of the transmission of afferent information. For example in a sprained ankle there is loss of control of locomotion. Even months after repair of ligamentous and capsular tissues has taken place, perception may still be distorted.

Structures containing connective tissue

Synovial joints (Fig. 3.7)

In synovial articulations, the bones involved are linked by a fibrous capsule, usually containing intrinsic ligamentous thickenings, and often also internal or external accessory ligaments. The articulating bony surfaces are generally not in direct continuity but are covered by hyaline articular cartilage of varying thickness and precise topology. Smooth movement of the opposing articular surfaces is aided by a viscous synovial fluid, which acts as a lubricant, and whose production requires the presence of a synovial membrane which is one of the defining characteristics of the joint type.

Fibrous capsule and ligaments

In synovial articulations the bones are linked by a fibrous capsule of parallel and interlacing connective tissue fibres – a cuff that encloses the joint cavity. With some exceptions, each end is attached in a continuous line around the articular ends of the bones concerned. Within this the capsule is lined by synovial membrane. A fibrous capsule usually exhibits local thickenings of parallel bundles of collagen fibres, called capsular (intrinsic) ligaments, that are named by their attachments. Some capsules are reinforced by tendons of nearby muscles or expansions from them. Accessory ligaments are separate from capsules and may be extracapsular or intracapsular in position.

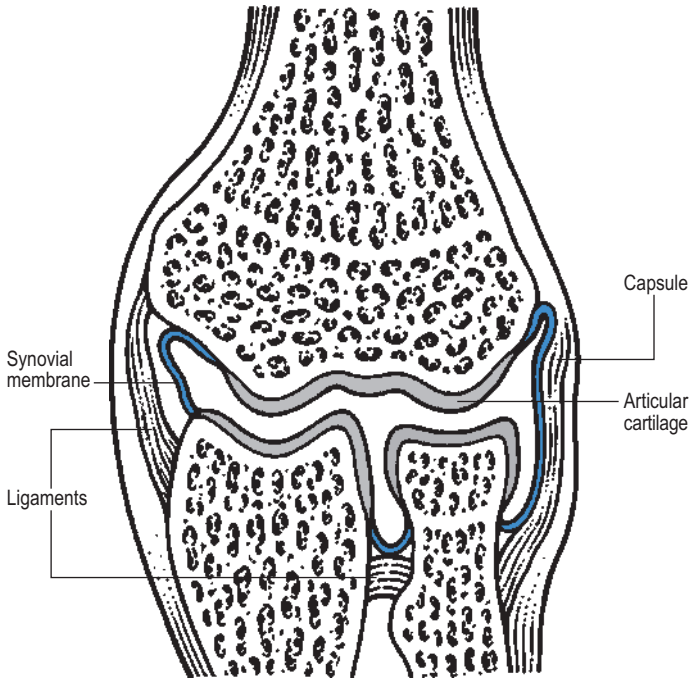


Fig 3.7 • Example of a synovial joint.

All ligaments are slightly elastic: collagen comprises about 70–80% of the dry weight, elastin 3–5%. They are taut at the normal limit of a particular movement but do not resist normal actions, since they are designed to check excessive or abnormal movements. Further they are also protected from excessive tension by reflex contraction of appropriate muscles.

The mechanical response of a ligament to a load can be represented on a load–deformation curve (Fig. 3.8). In the first part of such a curve (its foot) the ground substance is almost completely responsible for absorbing the stress and displaces the fibres in the direction of the stress. When the load is increased, ligamentous tissue responds slowly and maximum resistance to distraction is only possible if there is enough time for realignment of the collagen bundle. The linear part of the curve shows the slow elastic stretching of the collagen. During this stage, recovery of the original shape of the tissue occurs when the deforming load is removed. This slow rate of deformation is known as 'creep deformation'. Even in this linear part of the curve, breaking of intermolecular crosslinks begins. For this reason it is assumed that, in physiological circumstances, the load on ligaments is kept within that shown in the foot of the curve, where collagen is not yet under undue strain and the role of ground substance is maximal.[10] The composition and the amount of gel ground substance are therefore

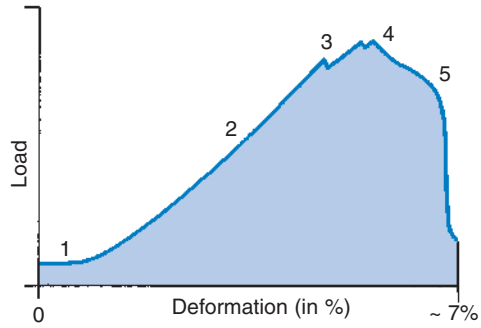


Fig 3.8 • Mechanical response of the anterior cruciate ligament of the knee to a load. 1. Foot of the curve, the ground substance alone almost completely absorbs the stress. 2. Linear part of the curve, slow elastic stretching of the collagen which is known as 'creep deformation'. 3. Yield point, a non-elastic or plastic deformation occurs. 4 and 5, the ligament progressively ruptures. Redrawn from Frankel[33] with permission.

important in load bearing. On reaching the yield point, a non-elastic or plastic deformation occurs and the ligament progressively ruptures. Some investigators have found that in bone–ligament–bone preparations, separation occurs at the point of insertion.[11]

In normal circumstances, mechanical stress induces early firing of mechanoreceptors in capsuloligamentous tissues. This causes a well-balanced reflex action of all musculotendinous units acting across the joint to avoid inert tissue becoming overloaded and damaged. If this muscular defence fails, strain falls on the ligament which is unable to stabilize the joint and so ruptures.

Synovial membrane and fluid

The synovial membrane lines the non-articular parts of synovial joints such as the fibrous capsule and the intra-articular ligaments and tendons within the margins of articular cartilage. The internal surface of the membrane has a few small synovial villi which increase in size and number with age. It also has flexible folds, fringes and fat pads. These accommodate to movement so as to occupy potential spaces and may promote the distribution of synovial fluid over the joint surfaces (Fig. 3.9).

Structurally the membrane consists of a cellular intima which is one to four cells deep that rests upon a loose connective tissue subintima and contains the vascular and lymphatic network which has an important function in the supply and removal of fluid. On ultrastructural examination, two cell types (A and B) are apparent. These are closely involved not only with the production of synovial fluid[12] but also in the absorption and removal of debris from the joint cavity. The A cells especially have marked phagocytic potential.[13] Some synovial cells can also stimulate the immune response by presenting antigens to lymphocytes if foreign material threatens the joint cavity.[14]

Synovial fluid is a clear, viscid (glairy) substance formed as a dialysate containing some protein. It occurs not only in synovial joints but also in bursae and tendon sheaths. Secretion and

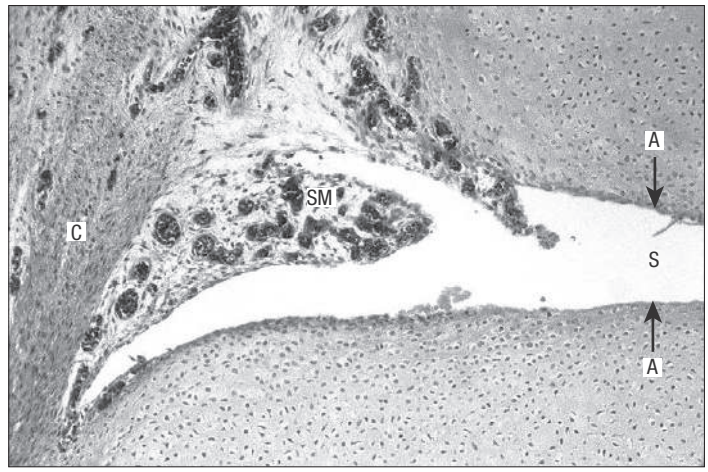


Fig 3.9 • A section of a synovial joint and its associated highly vascular synovial membrane in a human fetal hand. The two articular cartilage surfaces (A, arrowed) are separated on the right by a layer of synovial fluid (S) secreted by the synovial membrane (SM) which extends a short distance into the joint space from the capsule (C). From Standring, Gray's Anatomy, 40th edn. Churchill Livingstone, Edinburgh, 2008 with permission.

absorption are functions of the cells of the intima and of the vascular and lymphatic plexus in the subintima. The synovial initima cells also secrete hyaluronan molecules into the fluid and much evidence has accumulated to show that the viscoelastic and plastic properties of the fluid are largely determined by its hyaluronan content. Chains of hyaluronan bind proteins; these complexes are negatively charged and in turn bind water. The biophysical process is similar to that of the proteoglycans in the matrix of connective tissue and a thick viscous liquid which resembles egg white is formed. Its viscosity varies widely according to circumstances. With a low rate of shear, water is driven out of the hyaluronan–protein complexes and the fluid becomes highly viscous; increase in shear lowers viscosity and the fluid tends to behave more like water. In contrast to viscosity, elasticity increases with higher rates of shear. Both viscosity and elasticity decrease with increasing pH and temperature.[15]

Cartilage

Articular cartilage is essentially a specialized type of connective tissue.

Composition

Although the same three tissue elements – cells, ground substance and fibres – are present, their properties differ from ordinary connective tissue and determine its biochemical and biomechanical behaviour.

The composition of proteoglycans in the ground substance changes with increasing depth

In the superficial layer, chrondroitin sulphate is a prominent constituent in the GAGs but the deeper layers contain more and more keratan sulphate. A high concentration of chrondroitin sulphate stimulates the condensation of thin collagen fibres to form a dense network at the surface but keratan sulphate

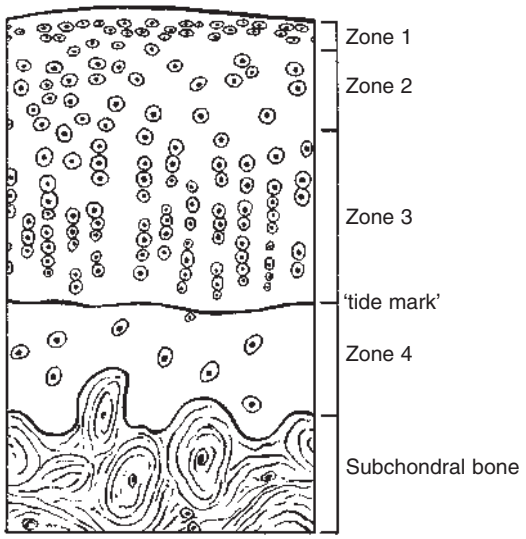


Fig 3.10 • Zones in articular cartilage. Zone 1, cells, small and flattened, disposed parallel to the surface; zone 2, cells become larger and more rounded; zone 3, cells are largest and arranged in columns, perpendicular to the surface; zone 4, mineralized cartilage. The border between mineralized and non-mineralized cartilage is called the 'tide mark'.

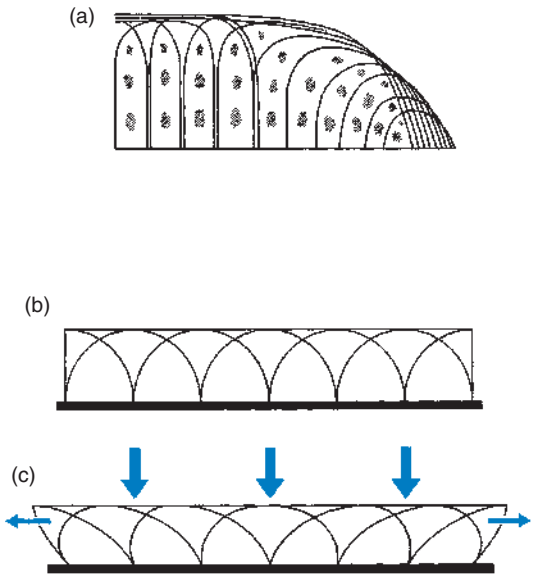


Fig 3.11 • (a) Arrangement of collagen fibres in articular cartilage. (b) and (c) Functioning of collagen fibres in articular cartilage. (b) non-load condition, (c) during load they are stretched in a direction perpendicular to the direction of force.

enhances synthesis of thick, easily movable fibres in the deeper layers. This adapted synthesis influences the local architecture and strength and resistance to compressing and shearing forces.

Cartilage cells or chondrocytes occupy small spaces in the matrix

They are involved in the production and turnover both of type II collagen and ground substance, processes stimulated by variation in load.

Chondrocytes change with increasing depth from the surface[1,16] (Fig. 3.10). In the superficial stratum (zone 1), cells are small, flattened and disposed parallel to the surface. They are surrounded by fine tangentially arranged collagen fibres. A thin superficial layer of this zone has been shown to be cell-free. Cell metabolism in this part is low, which is consistent with the absence of wear and tear in normal healthy tissue. The cells of the intermediate stratum (zone 2) are larger and more rounded and those in the radiate stratum (zone 3) are large, rounded and arranged in columns perpendicular to the surface. In these deeper zones, cells are screened from the coarse fibres by a coat of pericellular matrix bordered by a network of fine collagen fibres. In this way, cells are protected against the stresses generated by load conditions.

The collagen fibres vary in structure and position with increasing depth from the surface (Fig. 3.11a)

In the superficial or tangential stratum, a dense network of fine fibrils is arranged tangential to the articular surface to resist tensile forces that result from compression on certain points of the articulating surface during normal activities. Analysis has also shown the existence of certain 'tension trajectories' in accordance with the more or less fixed patterns of tensile forces that take place during movements. These preferential directions have been elaborated during growth as a result of forces acting on the joint. Near the border of the joint, the fibrils blend with the periosteum and joint capsule.

In the intermediate stratum, collagen fibres are coarser and more spread out to pursue an oblique course that forms a three-dimensional network. In non-load conditions the fibres are orientated at random but when load is applied they are immediately stretched in a direction perpendicular to that of the applied force (Fig. 3.11c). When the load is removed the fibres return to their original oblique position. This behaviour partly explains the resilience and elasticity of cartilagenous tissue.

In the radiate stratum, collagen fibres are arranged radially and correspond with the fibrous architecture of the subchondral and osseous lamina. The result is a series of arcades which extend from the deepest zone towards the surface.

Characteristics of cartilage

These include low metabolic and turnover rates, rigidity, high tensile strength, and resistance to compressing and shearing forces while some resilience and elasticity is retained. The proportion of collagen in matrix increases with age.

On the basis of variations in the matrix and the number of fibres present, cartilage in the locomotor system is divided into two types: hyaline and fibroelastic.

Most cartilage is hyaline: exceptions are the surfaces of the sternoclavicular and acromioclavicular joints and of the temporomandibular joints, all of which are of dense fibrous tissue. Although the light microscope appearance of hyaline cartilage is translucent, electron microscopy shows a system of fine fibrils and fibres. The water content is up to 80%. Strength, resistance and elasticity are the results of the proteoglycans in the ground substance together with the specific properties of the collagen fibres. Negatively charged proteoglycans bind a

large number of water molecules and causes the cartilage to swell. Swelling is limited, however, by the increasing tension of the collagen fibre networks in the superficial and deep layers which are closely interconnected. The result is an elastic buffer that, together with the elasticity of the periarticular structures, dissipates the effect of acute compressive forces. It also provides the articular mechanism with some degree of flexibility, particularly at the extremes of range. If the load is applied over a very short time, cartilage deforms in an 'elastic' way almost without disturbance of its water content. However, if compression is maintained for hours, water is displaced to surrounding regions that are under less or no compression and the compressed cartilage undergoes 'plastic' deformation. In engineering terms, this slow predictable rate of deformation is known as 'creep' and is greatest within the first hour of compression. When the deforming load is removed, recovery of the original shape of the tissue occurs at a rate that is specific for each form of cartilage.

Water transport during dynamic load conditions probably also has significance in the transport of nutrients and metabolites to and from the chondrocytes.

Articular cartilage lacks a nerve supply and is also completely avascular. Nutrition is derived from three sources: synovial fluid, vessels of the synovial membrane and vessels in the underlying marrow cavity which penetrate the deepest part of the cartilage over a short distance. This last source is available only during growth because, after growth is completed, the matrix at the deepest part of the cartilage becomes impregnated with hydroxyapatite crystals which form a zone of calcified cartilage impenetrable by blood or lymph vessels (Fig. 3.10, zone 4).

Articular discs and menisci consist of fibroelastic cartilage and are predominantly fibrous. They separate certain articular surfaces that have a low degree of congruity (e.g. the knee and the radiocarpal joint). Their functional roles are to improve the fit between joint surfaces, distribute weight over a larger surface, absorb impacts and spread lubricant.

With age, articular cartilage becomes firmer but also thinner and more brittle. The number of cells decreases. In normal healthy joints these changes are extremely slow. Erosion particularly occurs when joints become dehydrated or when synovial fluid viscosity permanently alters. Replacement of an eroded surface by proliferation of deeper layers has not been demonstrated. Deposits of calcification and surface ruptures are signs of degeneration. Except in young children, regeneration cannot be expected. However, there is evidence that a defect can be filled with newly synthesized collagen.

Synovial bursae

In situations where skin, tendons, muscles or ligaments move in relation to other structures under conditions that involve fluctuating pressure, synovial bursae are formed to reduce friction. They can be compared with flattened sacs of synovial membrane which create discontinuity between tissues and provide complete freedom of movement over a short distance. A capillary film of synovial fluid on their internal surfaces acts as a lubricant. Depending on their position they are classified as subcutaneous, subtendinous, submuscular or subfascial bursae. Sometimes they communicate with the joint cavity with which their synovial membranes are continuous.

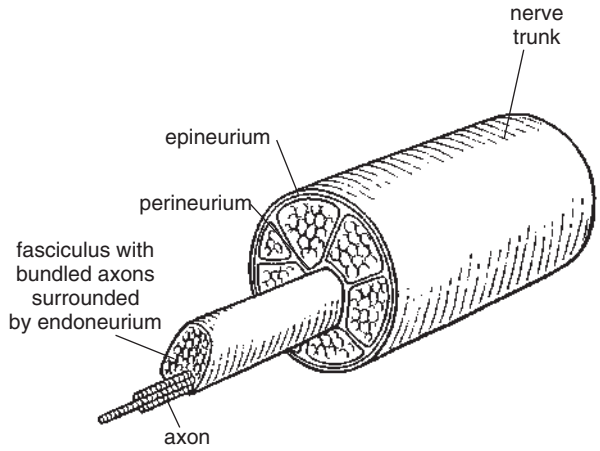


Fig 3.12 • Connective tissue in peripheral nerves: epineurium, a collagen coat that encases the nerve trunk; perineurium, fine collagen and laminae of fibroblasts that surround each fasciculus; endoneurium, loose delicate collagen that surround axons in the fasciculi.

Nerves

Peripheral nerves also possess supporting connective tissue. Within the nerve trunk the efferent and afferent axons are grouped together in a number of fasciculi (Fig. 3.12). The bundled axons in the fasciculi lie roughly parallel, surrounded by loose delicate collagen fibres running longitudinally along them. Both structures show a wavy appearance which disappears when gentle traction is applied.

Each fasciculus is surrounded by a fibrous *perineurium*, a regular structure of flattened laminae of fibroblasts alternating with fine collagen, running in various directions. These fibroblasts are connected together and form a diffusion barrier against noxious chemical products, bacteria and viruses. In this way, the enclosed axons are to some extent isolated from the external environment. Inside this perineural tube a protein-poor liquid flows centrifugally. This axoplasma is cerebrospinal fluid, which is re-assimilated into the blood circulation at the end of the peripheral nerve. In this respect, the spinal canal and the endoneural spaces are continuous (Fig. 3.13).

The *epineurium* encases the nerve trunk as a collagen coat with little regular organization. Connective tissue surrounding nerves serves as an important mechanical protection to maintain the conductile properties of the nerve.[17] During movement, nerves are potentially exposed to tensile forces that can be avoided by mobility in relation to surrounding structures. Here, the wavy form of both axons and surrounding collagen fibres is an important consideration: this 'waviness' of the axons is paramount, allowing them to remain relaxed even when the collagen fibres are stretched. Thus, within the normal range of movement, the axons will be protected by the tensile force of the collagen component. When there is a severe sprain or fracture perhaps with dislocation, the range of plastic

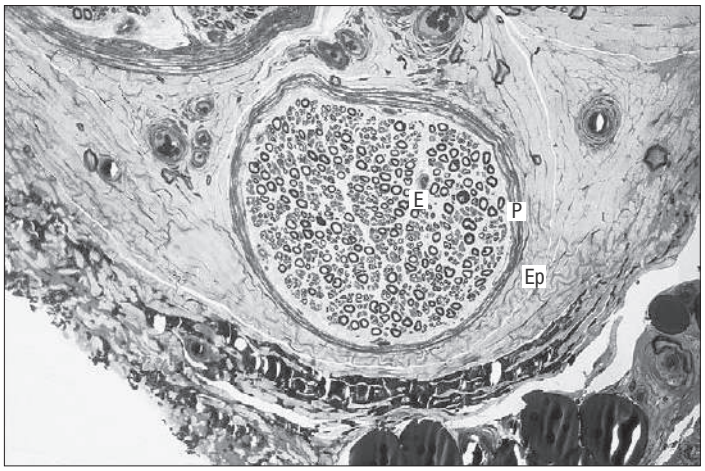


Fig 3.13 • Transverse section through a human peripheral nerve, showing the arrangement of its connective tissue sheaths. Individual axons, myelinated and unmyelinated, are arranged in a small fascicle bounded by a perineurium. P, perineurium; Ep, epineurium; E, endoneurium. Courtesy of Professor Susan Standring, GKT School of Medicine, London, in Standring, Gray's Anatomy, 40th edn. Churchill Livingstone, Edinburgh, 2008 with permission.

deformation of collagen can be exceeded and ultimately rupture of collagen fibres and neurotmesis results.

The tolerance of nerves to tension is much greater than it is to compression. However, the mobility of nerves allows them to move laterally, so avoiding a compressive force. When space is inadequate for such movement or the nerve is firmly anchored – which is the case in cervical nerve roots – the epineurium may absorb a certain amount of pressure but sooner or later the blood supply within the nerve is affected by increasing compression. Further compression may result in interference with the conductile properties of the nerve. In such circumstances, the Schwann cells and subsequently the myelinated sheath are damaged. Although the axons remain intact, action potentials become blocked, leading to loss of sensory and motor function (see Ch. 2).

Muscles

Muscular tissue consists of specialized cells or myofibrils embedded in a network of fine connective tissue that transmits the pull of the muscle cells during contraction to the adjacent parts of the skeleton. For this purpose, connections exist between the muscle cells and the finest collagen fibres of the connective tissue network.

The muscle cell or myofibril consists of sarcomeres or myofilaments – the basic contractile units of a muscle – arranged in parallel (Fig. 3.14). In each sarcomere two types of filament are distinguishable, chemically characterized as actin and myosin. The actin filaments are each attached at one end to the inner side of the cell membrane forming the so-called Z-line. At the other end they are free and interdigitate with the central myosin filaments. During muscle contraction, the actin filaments slide in relation to the myosin towards the centre of the sarcomere which brings the attachments at

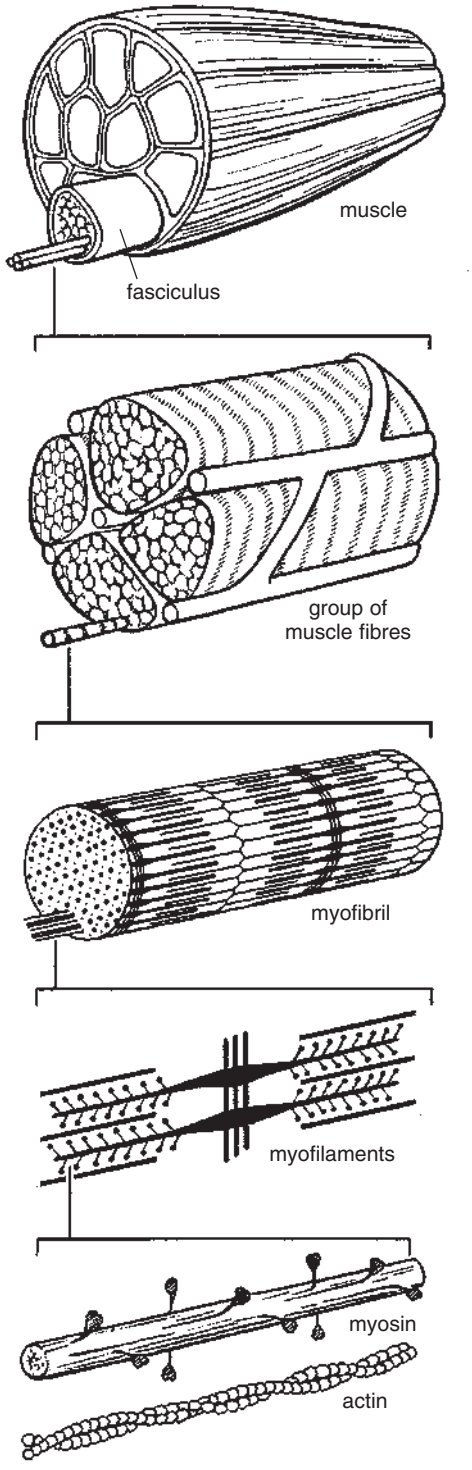


Fig 3.14 • The various levels of organization within a skeletal muscle, from whole muscle through fasciculi, fibres, myofibrils, myofilaments, down to molecular dimensions. The myofilaments are the basic contractile units of a muscle. Groups of myofilaments are arranged in parallel to form muscle fibres. These, in turn, are arranged in bundles or fasciculi. A network of connective tissue transmits the pull of the muscle cells during contraction via a tendon to the adjacent parts of the skeleton.

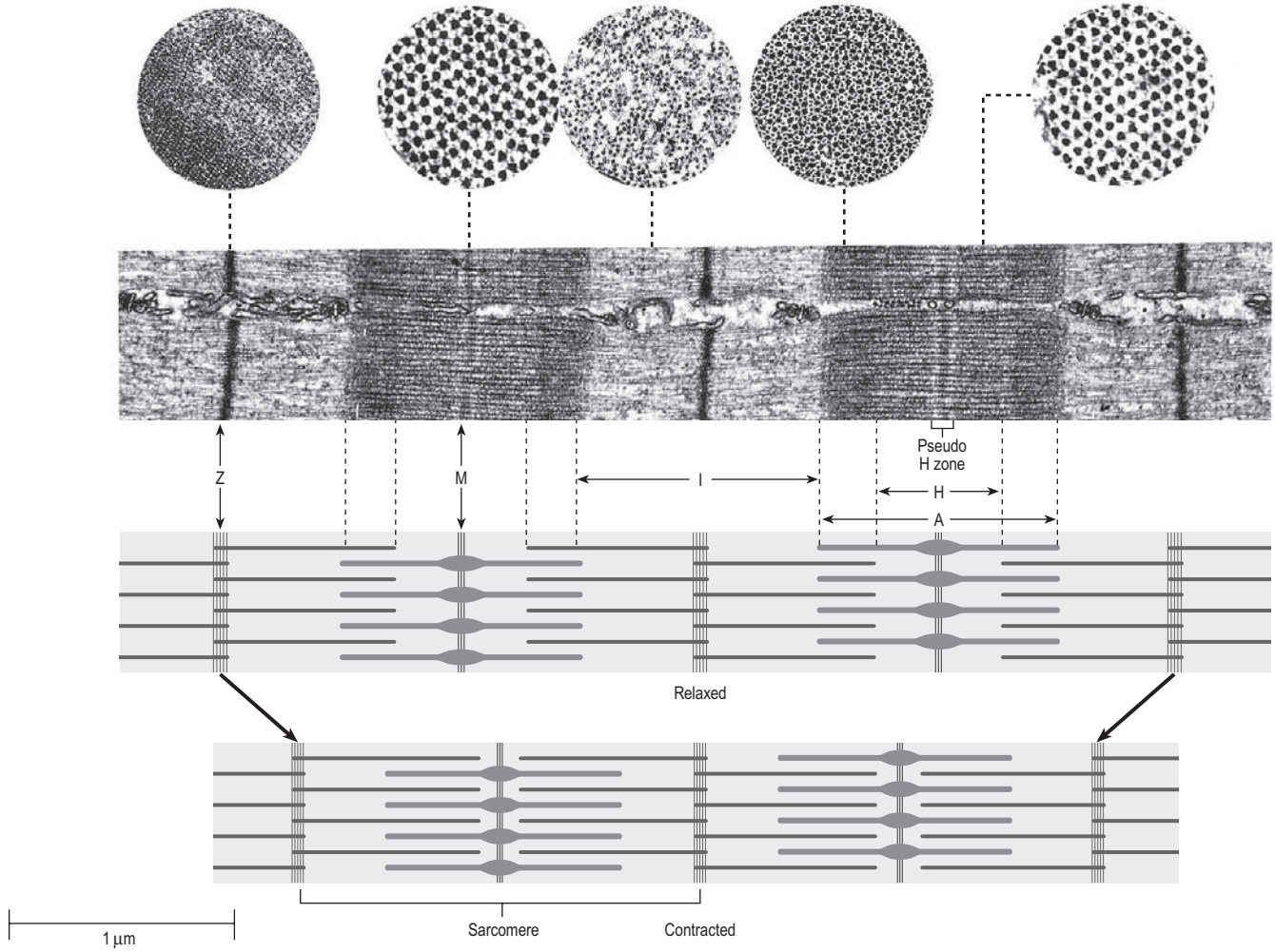


Fig 3.15 • Sarcomeric structures. The drawings below the electron micrograph (of two myofibrils sectioned longitudinally and with their long axes, orientated transversely) indicate the corresponding arrangements of thick and thin filaments. Relaxed and contracted states are shown to illustrate the changes which occur during shortening. Insets at the top show the electron micrographic appearance of transverse sections through the myofibril at the levels shown. Note that the packing geometry of the thin filaments changes from a square array at the Z-disc to a hexagonal array where they interdigitate with thick filaments in the A-band. Photographs courtesy of Professor Brenda Russell, Department of Physiology and Biophysics, University of Illinois at Chicago, in Standring, Gray's Anatomy, 40th edn. Churchill Livingstone, Edinburgh, 2008 with permission.

the Z-lines closer together with shortening of the whole contractile unit (Fig. 3.15).

Exercises increase the number of myofibrils (hypertrophy). During periods of immobilization cell volume decreases (atrophy).

Groups of sarcomeres are arranged in parallel to form muscle fibres. These in turn are arranged in bundles or fasciculi of various sizes within the muscle.

The network of the fine collagen fibrils within a fasciculus is known as the *endomysium* and fills the spaces between muscle fibres. In this way, each muscle fibre is surrounded by a thin sheet of connective tissue that provides the pathway for the capillaries, which lie mainly parallel with the muscle fibres and facilitate the exchange of metabolites between muscle fibres and the capillary bed.

The *perimysium* is the stronger connective tissue that surrounds each fasciculus. It consists of parallel bundles of collagen that are partly arranged in a circular manner around the muscle fibres as well. These bundles are in close connection with the collagen of the endomysium.

Finally, the whole muscle is surrounded by the stout *epimysium*, which is continuous with the septa of the outer perimysium and blends with the connective tissue that forms the tendon, fascia or aponeurosis (Fig. 3.16).

At the myotendinal junctions the connective tissue of the endo-, peri- and epimysium becomes very fibrous and thickens, whereas the muscle fibres taper or flatten and show terminal expansions. The connection is so strong that rupture seldom occurs at the myotendinal junction.

Nerve supply to muscles

Nerves supplying muscle are frequently referred to as 'motor nerves', but they contain both motor and sensory components. The motor fibres comprise α-efferents, which

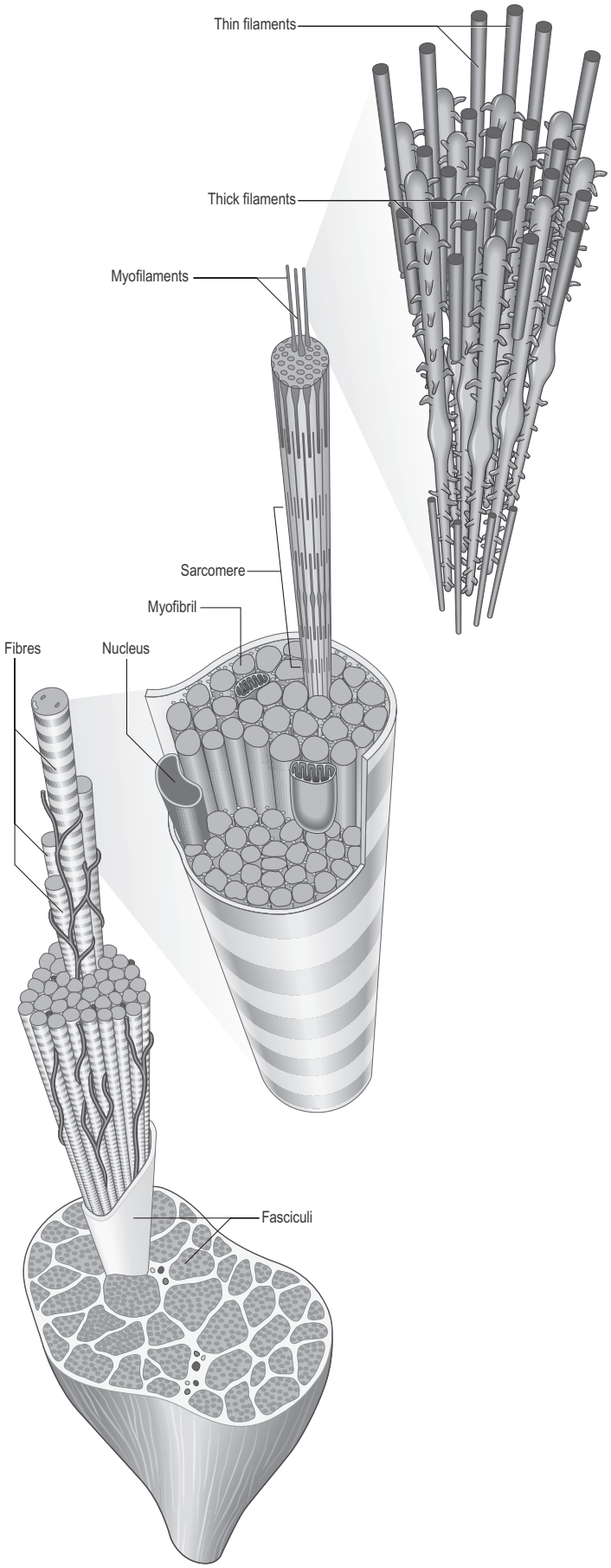


Fig 3.16 • Levels of organization within a skeletal muscle, from whole muscle to fasciculi, single fibres, myofibrils and myofilaments. From Standring, Gray's Anatomy, 40th edn. Churchill Livingstone, Edinburgh, 2008 with permission.

supply extrafusal muscle fibres, γ-efferents, which run to the muscle spindles, and autonomic efferents, which supply the smooth muscles of the vascular wall. The sensory component consists of large, myelinated IA and smaller group II afferents from the neuromuscular spindles and fine myelinated and non-myelinated axons which convey from free terminals in the connective tissue sheaths of the muscle.

Tendons

These structures are largely composed of collagen fibres with a low amount of proteoglycans. On a dry weight basis, collagen represents 60–80% of the total weight of tendon.[18]

Tendons, which consist of fascicles of collagen fibres running parallel and partly interweaving, are highly resistant to extension. Although elastin is absent and their collagen is difficult to stretch, tendons are nevertheless slightly stretchable. The wavy form of the fibres, together with the interweaving pattern of the fascicles, results in a slight elongation at the moment of muscle contraction which damps any abrupt pull on the insertion.

At the surface, the epitendineum or tendon sheath consists of irregularly arranged condensed collagen as well as elastin fibres. It is continuous with the loosely arranged connective tissue that permeates the tendon between its fascicles and provides a route of ingress and egress for vessels and nerves. At the insertion, the collagen bundles of the tendon permeate into bone. It has been shown[19,20,21] that the insertion of the connective tissue of ligaments into bone involves a transition from non-mineralized through mineralized fibrocartilage to bone.

In young growing tendons, fibril diameter and tensile strength can be increased by exercise. In adults, however, the effect is minimal although regularly applied tension is necessary to maintain structural integrity. Immobilization has demonstrated loss of tensile strength (see p. 46).

The nerve supply to tendons appears to be entirely afferent. Vascularization of tendons is low – the reason they appear white. Small arterioles ramify in the interfascicular intervals and are accompanied by veins and lymphatic vessels. Passage of vessels through the teno-osseous junction seems not to occur.

Where tendons pass under ligaments or through osteofibrous tunnels, synovial sheaths are formed which separate the tendon completely from its surroundings. These synovial sheaths have two concentric layers, separated by a thin film of synovial fluid and form a closed double-walled cylinder (Fig. 3.17). The fluid acts as a lubricant and ensures easy mobility of the tendon. The internal (visceral) layer is attached to the tendon and the external (parietal) layer to neighbouring structures such as periosteum and retinaculum.

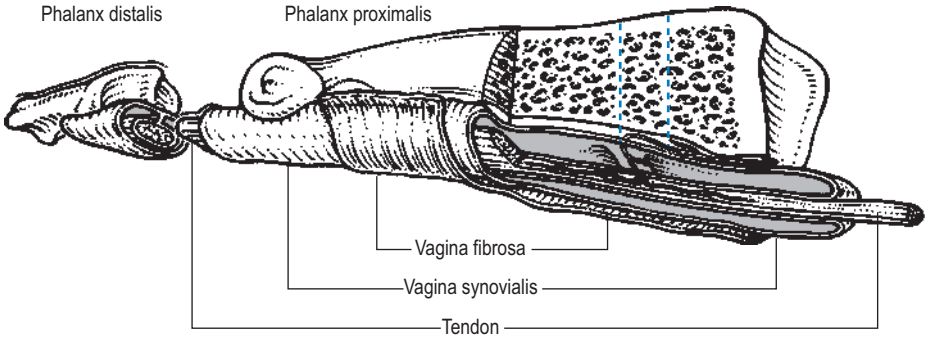


Fig 3.17 • Synovial sheath of the deep flexor tendon of a finger.

Table 3.2 Stages of repair after mechanical damage to soft connective tissue

Stage	Reaction
I	Inflammation: clearance of debris and preparation for repair Vasoconstriction (5–10 min) followed by vasodilatation and increased capillary permeability leading to: Exudation Liquid component Fibrinogen Cellular component
II	Granulation: formation of scar tissue (48 h to 6 weeks) Vascular infiltration Fibroblast proliferation
III	Remodelling (starts at the third week and may continue for 1–3 years): Devascularization Maturation Remodelling

Trauma to soft connective tissue

Introduction

Soft tissue injury involves damage to the structural elements of connective tissue with rupture of arterioles and venules. A general inflammatory reaction follows (Table 3.2), one role of which is defensive in that it prompts the subject to restrict activities while recovery takes place.

Regardless of the site of injury and the degree of damage, healing comprises three main phases: inflammation, proliferation (granulation) and remodelling. These events do not occur separately but form a continuum of cell, matrix and vascular changes that begin with the release of inflammatory mediators and end with the remodelling of the repaired tissue. Connective tissue regenerates largely as a consequence of the action of inflammatory cells, vascular and lymphatic endothelial cells and of fibroblasts.[22,23]

Inflammation

The first reaction is vasoconstriction of small local arterioles that lasts about 5–10 minutes and is followed by active vasodilatation and increased blood flow for 1–3 days. In major injuries with damage to blood vessels, blood escapes to form a haematoma that temporarily fills the injured site. Within the haematoma, fibrin accumulates and platelets bind to collagen fibrils to form a clot that provides the framework for invasion of vascular cells and fibroblasts.[24] The vascular changes and further inflammatory reactions are initiated by chemical mediators released from destroyed tissue cells.[25,26] Mast cells release heparin (anticoagulant) and histamine (vascular dilator). Plasma cells produce bradykinins and substance P (pain and vasodilatation). Platelets produce serotonin, prostaglandins and growth factors that stimulate migration, proliferation and differentiation of cells.[27,28] In addition, mediators cause migration of leukocytes into the injured area and swelling of the endothelial cells that line vascular channels. The endothelial cells pull away from their attachment to each other to leave sizeable gaps between cells that increase the permeability of vessels and so allow plasma, cells and proteins to escape. As a result, the presence of these proteins enhances the flow by osmosis of more plasma into the injured extracellular space. The whole process is the *exudative phase*. The liquid part of the plasma exudate dilutes potentially noxious substances and products of cell destruction and helps in their elimination by the supply of globulins and enzymes.

Another important substance is fibrinogen which forms an extensive network of fibrin into which fibroblasts can migrate along with other reparative cells.

The cellular parts of the exudate are:

- neutrophil granulocytes responsible for phagocytosis and proteolysis of the products of cell destruction
- lymphocytes which increase permeability and help to activate the phagocytosis of damaged cells
- macrophages whose role is probably to engulf and digest protein and to supply amino acids to the fibroblast; macrophages remain present throughout the entire inflammatory phase to assist in the phagocytosis of tissue debris and are also key cells in repair.

The well-known clinical signs of inflammation are: *swelling*, *warmth*, *pain*, *tenderness* and *functional loss* – a defensive

reaction of the body that prompts the subject to restrict activities while recovery takes place.

Repair

It is worthwhile to mention that only the synovial capsules of the joints, skeletal muscle and bone are, to some degree, capable of *regeneration*. All other connective tissues heal by *repair* with the formation of collagen and thus scar tissue.

Once the exudative phase has cleared debris by dilution and phagocytosis, fibroblasts and capillaries migrate along the fibrin network. The process of vascular infiltration, fibroblast proliferation and the deposition of collagen usually begins within 48 hours of injury overlapping with the end of the exudative phase and the later remodelling phase. Repair is begun and directed by the release from macrophages of chemotactic agents which attract fibroblasts and endothelial cells, secrete growth factors which stimulate these cells to proliferate and produce lactic acid which enhances the synthesis of collagen by fibroblasts. High levels of corticosteroids prevent the migration of macrophages.

During their proliferation, fibroblasts develop into cells termed myofibroblasts that generate a traction-like activity on the matrix required for the reduction of any gap in the healing area.[29]

Capillaries, at the edge of the injured area, send forward buds which then turn and meet each other to form new capillary loops capable of maintaining a circulation that ensures oxygen and nutritional supply in the relatively hypoxic region where the healing tissues meet and, at the same time, enables the removal of metabolic waste products. These new capillaries are fragile and stay within the support of newly synthesized collagen which has already been deposited ahead of the formation of the capillary loops. The highly vascular mass produced gives the surface of the tissue its granular appearance and hence its name – *granulation tissue*.

By the fourth or fifth day after injury the amount of collagen is significant and there is a progressive but gradually slower increase up to 6 weeks after injury. Corticosteroids decrease the number of fibroblasts and result in a diminished formation of collagen fibres and possibly a weaker fibrous scar. Normally the initial arrangement of collagen fibres is at random but after 6 weeks, tensile strength continues to increase because of orientation of fibres along the lines of stress in the injured tissue (remodelling).

Remodelling

Around the end of the third week maturation begins – the process of reshaping and strengthening the scar tissue by removing, reorganizing and replacing cells and matrix. A better structural orientation and increase in tensile strength result.[30] The remodelling phase can be divided into a consolidation and maturation stage:[31]

- First, vascularization decreases and many of the new vessels atrophy and disappear as the blood supply becomes appropriately adjusted to the needs of the scar tissue.
- Second, the amount, form and strength of scar collagen changes: the immature and weak tissue with a random orientation of fibres in three planes is remodelled into linearly arranged bundles of connective tissue. The process is the result of a number of factors, including turnover of collagen, fibre linkage and increased intermolecular bonding.

It is now generally recognized that internal and external mechanical stress applied to the repair tissue is the main stimulus for remodelling. Tension by gentle movements in functional directions reorientates the collagen and breaks any weak or unnecessary crosslinks that may have formed. Mechanical stress thus has its greatest influence on remodelling at this time. Non-functional collagen is cleared away by phagocytosis.[32–36]

Remodelling may continue for years although more slowly as time passes. The tensile strength of replaced or repaired collagen in ligaments reaches 50% of normal by 6–25 months after injury and 100% only after 1–3 years.[37] The strength of a scar formed in an injured muscle increases faster because of its superior vascular supply.[25]

Self-perpetuating inflammation

The linear sequence of events described above – an inflammatory reaction followed by repair and remodelling – is typical for acute wounds, either accidental or surgical. Orthopaedic medicine, however, also deals with chronic repetitive strains and tissue disruptions, overuse phenomena and excessive tension on devitalized tissues. Here the reaction of the tissues involved is often not linear; the inflammation may be prolonged and the formation of scar tissue excessive and inappropriate.

Rest usually initiates adhesion formation in and around the healing breach. Oedema raises tissue tension and causes pain, so impeding functional movements that are extremely important in the early stage of regeneration. Without proper movement there is no balance between formation and lysis of the regenerating elements of the involved tissue. Proper alignment of collagen does not result and the final form of the scar tissue tends to remain ill-organized. Any small stress applied to an inappropriate tissue is sufficient to disrupt newly formed fibres in the healing breach. This in turn starts another inflammatory response and a vicious circle of chronic repetitive disruptions of inferior quality connective tissue will result. If such a state of chronic inflammation is maintained, the function of the affected area continues to deteriorate and leads to further tissue damage.

Cyriax drew attention to such chronic types of inflammation of soft tissues that began as a result of trauma but continued long after the cause had ceased to operate – *self-perpetuating inflammation* (see Fig. 3.18) – particularly prone to happen after a minor injury to a ligament. Occasionally it also occurs as an overuse phenomenon in a tendon. With knowledge of the inflammatory reaction in traumatized soft tissues, it is clear that lack of movement during the period of repair and remodelling which leads to adhesive scar tissue formation can be responsible for some chronic lesions.

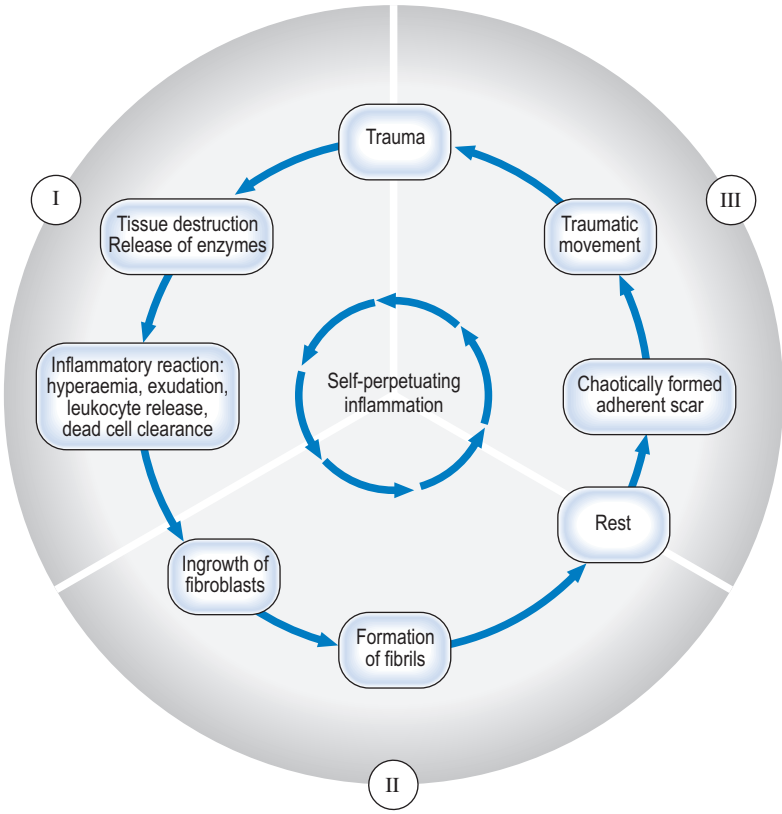


Fig 3.18 • Self-perpetuating inflammation: rest initiates adhesion formation; stress applied at the inappropriate time disrupts the newly formed fibres, which starts another inflammatory response. I, inflammation; II, repair; III, remodelling.

The decision whether a lesion requires rest or movement cannot be taken by the patient, who feels pain and loss of function and interprets these symptoms as a potential threat that can be reduced by immobilization. The main goal in the treatment of musculoskeletal lesions is therefore to guide the healing soft tissues through the stages of inflammation and repair by the provision of sufficient and appropriate motion that can restore painless function. If a chronic self-perpetuating inflammation has become established, a local infiltration of corticosteroid may interrupt the process. The scar becomes painless and the tissue, no longer deprived of its functional motion and appropriate stress, starts to remodel. Another approach which helps to reduce the amount of disorganized scar tissue is to perform deep transverse friction followed by manipulation (see p. 54).

Effects of immobilization on healing

Joint capsule and ligaments

Disturbance of the blood and lymph stream in the synovial membrane influences the supply of nutrients and the scavenging of metabolic products and destroyed cells. Joint immobilization reduces synovial fluid hyaluronan concentration and is accompanied by changes in the synovial intimal cell populations.[38]

In a study on the effects of immobilization of knee joints of dogs, deposition of excessive connective tissue was noted.[39] In the course of time, mature scar and intra-articular adhesions were found which restricted joint motion. Within the matrix a 4.4% loss of extracellular water and a significant reduction in GAG content (30–40%) was established. Ingrowth of new capillaries at the edge of injured tissue was diminished. Other workers studied the effects of immobilization on the knee joint of the rabbit.[40,41] They confirmed the findings in the dog but also postulated that loss of water and GAG content would decrease the space between collagen fibres and thus restrict normal interfibre movement. Random orientation of newly generated fibrils and the formation of crosslinks between newly regenerated fibrils and pre-existing collagen fibres were other findings responsible for decreased collagen mobility and restricted movements. These matrix changes are relatively uniform in ligament, capsule, tendon or fascia. Some specific studies on collateral and cruciate ligaments have demonstrated laxity, destruction of ligament insertion site and failure at a lower load after immobilization for 3 months.[42–44]

Cartilage

Several authors have also demonstrated the deleterious effects of immobilization on cartilage:[44–51]

- Shortening and thickening of fibrous articular capsule gives rise to a three-fold increase of the compression of articular cartilage, which may eventually initiate degenerative changes in the joint.
- Loss of water content and GAGs in cartilage decreases its elastic properties.
- Decreased capsular blood supply leads to a deposit of some end-products of metabolism at the joint surface.

Table 3.3 Effects of immobilization and mobilization on soft tissue injuries

Tissue	Immobilization	Mobilization
Joint capsule	1. Distribution of blood and lymph flow 2. Intense synovitis 3. Loss of extracellular water and GAG content 4. Deposition of excessive connective tissue 5. Decreased collagen mobility 6. Intra-articular adhesions 7. Laxity and destruction of ligament insertion site	1. Increased circulation 2. Prevention of abnormal adhesions 3. Beneficial influence on the remodelling process 4. Increase of strength of connective tissue in ligaments
Synovial fluid	1. Alteration of viscoelastic properties	
Cartilage	1. Increase of compression 2. Deposit of end-products of metabolism 3. Decrease of elastic properties 4. Autolysis of cartilage	1. Beneficial effect on assimilation of nutrients
Muscles	1. Atrophy 2. Decrease of strength 3. Increase of amount of connective tissue 4. Disturbance of neuromuscular coordination of muscle groups	1. Increased circulation 2. Increase of muscle strength and endurance 3. Maintenance of proprioceptive reflexes which ensure active joint stability

- Lysosomal enzymes released from dead chondrocytes lead to an autolysis of cartilage which is proportional to the time of immobilization.

Muscle

Muscular reactions to immobilization have also been investigated.[52,53] There is:

- decreased capillary density and muscle atrophy
- decrease in muscle strength most dramatically during the first week of immobilization. After 2 weeks in a plaster cast, there is 20% loss of maximum strength. Slow muscle fibres, with predominantly oxidative metabolism, are more susceptible to immobilization atrophy than are fast fibres
- an increased amount of connective tissue. Proliferation first takes place in the perimysial spaces, followed sometimes by the endomysial spaces. It is suggested[53] that this may impair the vascular supply of muscle fibres and could facilitate degeneration and also could make regeneration more difficult. Although muscle structure, metabolism and function are severely impaired after immobilization, almost complete recovery is possible provided that the training programme starts with very moderate exercises and avoids maximum voluntary efforts of regenerating muscle fibres
- disturbance of neuromuscular coordination of muscle groups.

The same studies drew attention to the reactions of organ systems, such as the cardiovascular, respiratory, locomotor and autonomic, which may also become disturbed.

Effects of mobilization on healing

The benefit of early mobilization in most soft tissue lesions was advocated by Hippocrates more than 2400 years ago. Capsular circulation is increased (Table 3.3) which aids the supply of nutrients and elimination of cartilagenous debris. Physical joint movements have a beneficial effect on the assimilation of nutrients by the cartilage.[54]

Experimental findings[11,55,56] on the influence of physical activity on ligaments and tendons support the view that the strength of connective tissue is increased with exercise training and decreased with immobilization, provided that the exercise programme is of an endurance nature. Trained animals have significantly heavier ligaments, stronger ligament–bone junctions and junction strength to body weight ratios. Similar effects pertain in repaired ligaments,[42,57] which show significantly higher strength values after repair is complete if they have not been immobilized. Early mobilization also considerably influences the remodelling process and prevents formation of abnormal adhesions that may restrict joint movements.[58]

Another advantage of early mobilization is the positive effect on skeletal muscles,[57,59,60] with increased circulation, muscle strength and endurance and maintenance of proprioceptive reflexes, which ensure the active stability of the joint.

Treatment of traumatic soft connective tissue lesions

The overall aim of treatment in orthopaedic medicine is to restore painless function of the connective tissue. During the last decades it has become clear that application of functional movement to healing connective tissue is extremely important and should be the first and principal objective for the therapist. Of course, the selection of the techniques will depend on several factors such as the stage of the lesion, the tissue involved, the severity of the lesion, the irritability of the tissue and the pain perception of the patient.

Muscular lesions

Delayed muscle soreness, contusion, (minor) rupture, myosynovitis and myositis ossificans are different types of lesion which can occur in skeletal muscle.

Delayed muscle soreness

A delayed, specific soreness sometimes appearing 12–24 hours after intense exercise is well known in athletics and may be caused by the disturbance of metabolism, with a high concentration of lactic acid and the resulting inflammatory reactions: vasodilation, increased capillary permeability and intercellular oedema. Swelling and oxygen deficiency may irritate free nerve endings and lead to muscle spasm.[61] Another, more recent, theory is that there is injury to sarcomeres and intramuscular collagen fibres and, in consequence, an inflammatory reaction.[62]

Passive stretching and active contraction cause discomfort. Pain lasts 3–4 days, gradually diminishing during the subsequent days. Improper warm-up, unusual exertion, early season training and running before muscles are properly conditioned are some conditions that are thought to bring on the pain.

The best way to avoid this condition is to stay 'in shape' between seasons. Other precautions to be advised are:

- warm up before beginning an exercise programme
- include stretching exercises in the warm-up
- gradually increase the load and duration of the exercises
- avoid excessive tension on muscles
- allow the muscles to dissipate waste products (warm-down).

Muscular contusion

This results from a direct blow on the muscle belly. There is a variable degree of severity, characterized by pain and intra- or intermuscular bleeding with extensive swelling. Intramuscular bleeding is more serious and lasts longer because of difficulty in dispersing the haematoma. Such a blow does not cause much discomfort or pain while the athlete is warm and actively taking part in sport; it is some hours afterwards that stiffness and disability set in. Active contractions against resistance cause discomfort and passive stretching of the muscle is painful and limited.

Treatment involves aspiration of the haematoma (within 3 days), after which compression is applied immediately, using an elastic wrap. A short period of rest may be prescribed but rest should never be total and prolonged.

After a few days, treatment with gentle deep transverse frictions and active contractions with the muscle in a fully shortened position can be started (see Minor muscular tears).

Minor muscular tears (or 'muscle strain injuries')

Acute lesions

These are the common 'muscle pull' or 'strain' injuries that result from sudden and over-violent effort or movement in the muscle and usually cause immediate disability. Tears occur most often during unusual contractions,[63] which produce significantly higher muscle force than when the muscle is held at the same length or is allowed to shorten. It has also been shown that muscles crossing two joints, such as hamstrings and gastrocnemii, are particularly at risk. The vulnerable site appears to be near the muscle–tendon junction. The immediate response to the trauma is inflammation, associated oedema and localized haemorrhage. Excessive oedema and haemorrhage should be reduced as much as possible. Blood collections are not confined to the muscle proper but escape through the perimysium and fascia into the subcutaneous space. As a rule, the degree of pain is consistent with the extent of the rupture.

Treatment

The immediate induction of *local anaesthesia* at the site of the lesion effectively blocks the nociceptive impulses which are responsible for muscle spasm at the site of damage. Cold therapy as an alternative has been criticized.[64] Although it has a positive effect on pain threshold,[65,66] physiological effects and procedures of application are still chiefly based on empirical and clinical findings. Van Wingerden suggests that cold therapy, especially when applied in the acute phase of injury, could lead to increased oedema, inhibition of the healing process and even to increased inflammatory reactions.[64]

From the next day on, *active or electrical contractions* follow, with the muscle in a fully shortened position to maintain mobility by broadening the muscle belly. Meshwork of regenerating fibrils in the healing breach may hinder the broadening capacity of the muscle fibres during contraction. In the later stages of healing (granulation and remodelling), intramuscular formation of abnormal crosslinks takes place and the inappropriate scar tissue will form a mechanical barrier to broadening during contraction, a chief muscle function (Fig. 3.19).

Deep transverse friction also imitates broadening and prevents newly formed bonds from matting muscle fibres together. It should be started the day after injury, as it can be expected that repair has begun by this time. A gentle type of massage is performed daily for a short period of time (see Ch. 5). At this stage, it should not be done to an extent that interferes with the capillaries and fibrils consolidating themselves in the healing breach. Both intensive passive stretching and resisted movements may cause damage at the site of injury and must be avoided until recovery is well established. It has been demonstrated[67,68] that contractile ability recovers rapidly.

Once the patient is free from pain and has a full range of mobility, repetitive stretching exercises seem to make an important contribution to the future prevention of these injuries.[69–72] Return to sport can be allowed when the strength of the injured limb has been restored to within 10% of that of the unaffected limb (usually after 3–6 weeks).[73]

Chronic lesions

Scarring takes place in chronic lesions, matting fibres together transversely. The range of broadening is impaired and contractions against resistance are painful. A painful area can be palpated, although it may be difficult when the lesion is deeply situated, for example, the belly of biceps brachii or the extensor carpi radialis brevis. Pain probably results from overstretching at the junction between normal and scar tissue, from local variations in tension. This explanation seems logical because

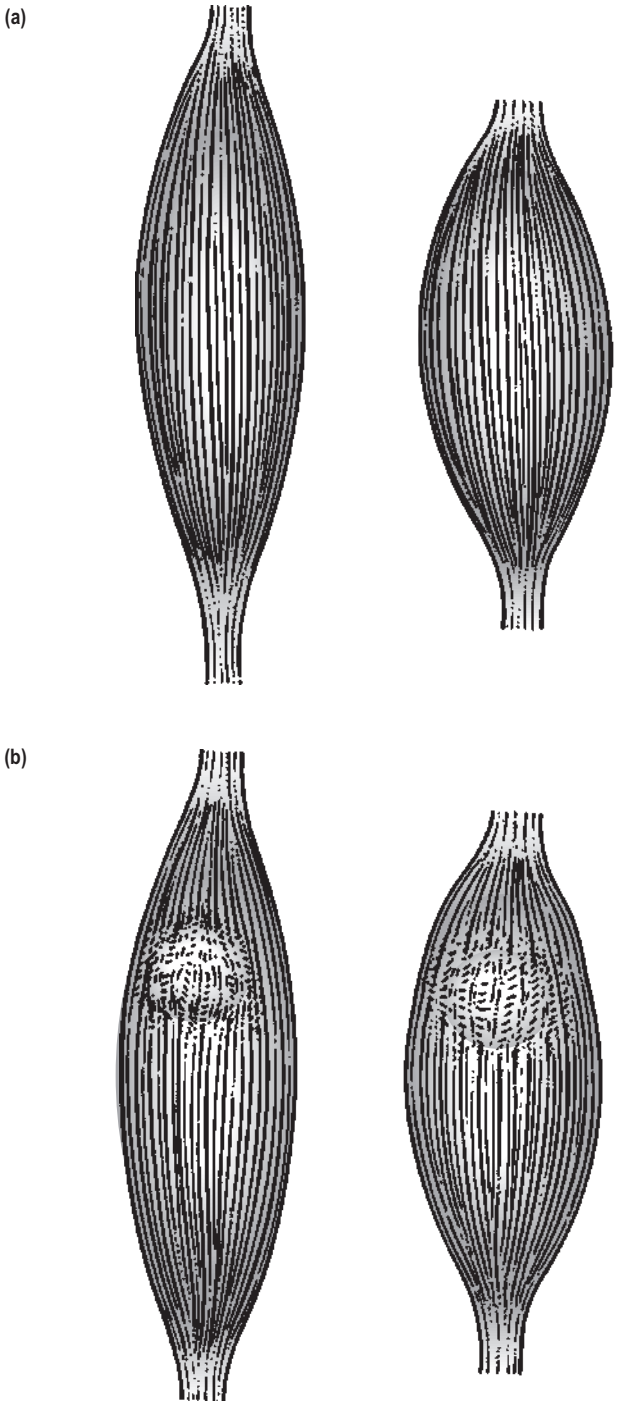


Fig 3.19 • (a) The muscle belly broadens during contraction; (b) scar tissue and intramuscular formation of connective bridges form a mechanical barrier to broadening during contraction.

pain is absent in diffuse fibrosis after sclerosing injections and in ischaemic contracture.

Treatment

Deep transverse friction for about 20 minutes twice a week to the site of the established scar is indicated. The muscle fibres are teased apart, abnormal crosslinks ruptured and mobility restored. To maintain the effect of the friction, *active* and *electrical contractions* with the muscle belly in a fully relaxed position should follow. The muscle contracts to its fullest extent and this is repeated for 5 minutes at regular times. When the lesion is at the musculotendinous junction, active or electrical contractions are ineffective and treatment is by deep friction only.

Steroids do not have a place in the treatment of muscular lesions. Recent research in an animal model indicates that corticosteroids may be beneficial in the short term but they cause irreversible damage to healing muscle in the long term, including disordered fibre structure and a marked diminution in force-generating capacity.[74]

Myosynovitis

Myosynovitis is a painful condition described by Cyriax,[75] arising from a muscle as the result of overuse. In severe cases it is accompanied by crepitus on movement. This uncommon condition seems to occur only in the bellies of the long abductor and the extensor muscles of the thumb and in the musculotendinous junction of the tibialis anterior. The last disorder is a well-known complaint in new military recruits who march unaccustomed distances.

Myositis ossificans

This condition is characterized by progressive benign heterotopic bone formation, which may occur after severe contusion to muscle fibres, connective tissue, blood vessels and underlying periosteum. The pathogenesis is poorly understood. It is seen most often in males, aged between 15 and 30 years. Highly common sites are the brachialis and quadriceps muscles. The condition is sometimes found in the hip adductors and pectoralis major and the bony deposit is often connected to the underlying bone.

There is the following triad of symptoms:

- Increasing pain
- Palpable and increasing firm mass in the affected muscle
- Gradual decrease in the range of movement of the neighbouring joint(s).[76,77]

The history of severe contusion to the affected muscle is helpful in the early evaluation of these patients because radiographic changes become evident only 2–4 weeks following the trauma. The condition can mimic benign or malignant bone tumour and osteomyelitis.[78,79]

Specific treatment is not recommended. Bone formation may resorb with time but recovery may take from 1 to 2 years. Early surgery is to be avoided because it may exacerbate the bone formation. Removal can be considered if symptoms persist, but only after the heterotopic bone is mature and no further radiological changes occur.[80,81]

Tendinous lesions

Tendons transmit power from the muscle belly to bone. In that the tendon is always less in diameter than the muscle, the load transmitted to the tendon will be much greater per unit diameter than in the muscle belly. Dysfunction may result from changes either within the tendon or in the surrounding tissues of the tendon (paratenon).

Terminology

Within the literature, there is much confusion about the terminology and over the last decades, numerous terms have been used to describe the pathology of tendons. The most common term is 'tendinitis' which focuses on clinical inflammatory signs. Puddu et al.[82] proposed the term tendinosis as a histological description of a degenerative pathology with a lack of inflammatory change. As these terms are often used interchangeably and without precision[83] it may be more appropriate to refer to a symptomatic primary tendon disorder as a tendinopathy as this makes no assumption as to the underlying pathological process.[84]

'Tendinitis'

When strain on a tendon tears some fibres, it always seems to occur in those parts of the tendon where vascularization is relatively poor. The insertion into bone and sometimes a specific part of a tendon are such 'critical' vascularized zones. Good examples are a zone in the Achilles tendon about 2.5 cm above its insertion into the calcaneus[85,86] and the supraspinatus tendon close to its insertion on the major tubercle.[87]

Above the age of 25, vascularization of tendinous tissues also decreases (ultimately by about 30%) which enhances their vulnerability.

Lesions result principally from overuse. However, the mechanical response of a tendon to a load not only depends on the amount of the externally applied force but also is closely bound up with the state of the tendon involved. Overuse may disturb the microcirculation which, especially in zones of hypovascularity, will negatively influence metabolic processes. If this continues, a process of degeneration starts, called 'tendinosis'. In the beginning of the process this is a degenerative condition without accompanying inflammatory reactions and therefore clinical signs and symptoms may be totally absent.

From the moment that a normal load strains the tendon and tears some fibres, an inflammatory reaction begins. Long-standing treatment with corticosteroids or periods of immobilization will further negatively influence the condition of collagen and lead to further deterioration and decrease in the number of fibrocytes and fibres. In sports, a cold climate, bad equipment and wrong training procedures (e.g. lack of warm-up including absence of stretching, too progressive an increase of load and duration of the exercise programme, bouncing exercises and jerky muscle contractions) may all negatively influence the condition.

The inflammatory reactions in tendinitis are located not only at but also around the ruptured fibres, in the areolar tissue between the fasciculi. Here oedema, growth of capillaries, migration of leukocytes and invasion with mesenchymal cells take place, which induces new generations of fibroblasts and leads to a large proliferation of connective tissue around the site of the lesion.

Acute traumatic lesions

In acute traumatic lesions 48 hours after the injury, newly generated collagen fibrils start to close the tissue defect by the formation of scar tissue. Scars may remain lastingly painful and often have an inferior tensile strength, especially if the initial inflammatory reaction was excessive and the granulation and remodelling phases inadequate.

Treatment

To prevent excessive inflammation and to stimulate better remodelling, gentle mobilization and transverse friction are used to orient the randomly distributed collagen in a functional direction. This also prevents the formation of abnormal adhesions. Treatment starts the day after the injury and massage should be superficial and light in order not to rupture newly generated fibrils. Treatment along these lines is continued daily for the first week and on alternate days for the second and third weeks. After 3 weeks, active unloaded contractions can be added. A tape or bandage to support the structure and prevent undue movement is advisable. Careful evaluation is necessary, gauging the degree of activity against the degree of local reaction.

The patient should be very cautious about returning to full activity until the lesion is completely pain free.

Chronic (overuse) lesions

Treatment

In chronic tendinous lesions, transverse friction massage applied precisely to the exact area may be an extremely useful technique and can often bring lasting relief.

Modalities and effects of this treatment form are discussed in the online chapter *Applied anatomy of the cervical spine* but transverse friction aims to achieve transverse movement of the collagen structure of the connective tissue. In this way adhesion formation is prevented and existing adhesions are mobilized. The cyclic loading and motion of the healing connective tissues may also stimulate formation and remodelling of the collagen.

Another approach is to infiltrate locally a low dose of corticosteroid. This will quickly reduce the chronic inflammatory reaction but it also affects the proliferative and even the remodelling phase – the biosynthesis of collagen is altered by corticosteroids. For this reason, local steroid infiltrations should only be at the tenoperiosteal insertion and never in relation to the body of a tendon for fear of possible tendon rupture. A further disadvantage of steroid infiltration is the higher recurrence rate (about 25%) after initial therapeutic success.

Persistent pain is thought to be caused by irreversible degenerative changes within the tendon. Removal by surgical treatment may then be indicated. Again treatment may include sessions of transverse friction applied in a progressive way until the patient is free of symptoms. Thereafter, a programme of rehabilitation is undertaken.

Tenosynovitis

Tenosynovitis is a well-known lesion at the ankle or the wrist where tendons possess a sheath. Roughening of the gliding surfaces of a tendon and the internal or visceral layer of the sheath results from an inflammatory reaction; commonly occupational overuse or strain. During movement, pain is evoked as the roughened surfaces move against each other. In severe cases fine crepitus is palpable and swelling obvious. Coarse crepitus is a warning sign that points to rheumatoid disease or tuberculosis.

Treatment

This consists of deep transverse friction whether the condition is recent or chronic but it is indicated only in posttraumatic lesions. Friction restores the smooth and painless movement of the tendon in relation to the internal layer of the sheath.

An injection of a small amount of steroid suspension between tendon and sheath is an alternative. Some lesions respond better to the massage and for others injection is the treatment of choice (see Ch. 5).

All activities that cause pain should be avoided until symptoms have ceased.

Tenovaginitis

Tenovaginitis is primarily a lesion of the tendon sheath itself and is often associated with considerable swelling and tenderness but there is never crepitus. It may have a detectable cause in overuse but also occurs spontaneously. Non-specific types of tenovaginitis should be differentiated from those with a specific cause, such as bacterial infection, rheumatoid arthritis, gout and gonorrhoea.

Treatment

For the non-specific types of tenovaginitis, treatment is the introduction of a small amount of steroid between tendon and sheath, which brings quick relief. In persistent cases, deep transverse friction can be tried but incision of the tendon sheath is often necessary.

In specific tenovaginitis, treatment is that of the underlying disease.

Tendinosis

This is a degenerative condition of the tendon without accompanying inflammatory reactions and therefore often without clinical symptoms. The lesion is characterized by visible discolouring of the tissue and loss of the mirror-like gloss of the tendon surface and is typically described as 'mucoid degeneration'.[88]

Microscopically, tendinosis is characterized by a marked loss of collagen. The fibres show an irregular entangled course and an irregular waving pattern. Between the fibres, cavities filled with fluid loosen the tissue. The number of fibroblasts is decreased and their nuclei transformed.

Besides this local destruction, there are also signs of regeneration as shown by slight fibroblast proliferation, formation of capillary sprouts and new mesenchymal cells, which synthesize young collagen fibrils to fill up the tissue defect. If this process of granulation is insufficient, necrosis and calcification result.

Tendinosis and mucoid degeneration often lead to spontaneous ruptures (long head of the biceps, Achilles tendon). It is suggested that the 'typical' histopathological changes characterized by tendon degeneration may not necessarily be directly linked to increased nociception giving the patients warning signals; while in painful 'tendinitis' the mechanically weaker tendons may be protected from ruptures due to decreased impact levels since painful activities will be avoided.[89]

Complete rupture

Complete rupture of a tendon usually results from indirect trauma. It always seems to occur at the midsubstance of the tendon. Acute tears are those occurring suddenly, usually as a result of a single injury. Chronic tears are those that are insidious in onset and result from repetitive loading of a degenerated and weakened tendon (see above). Tendinous ruptures occur predominantly at the shoulder, wrist and heel. At the shoulder, the incidence of full thickness rotator cuff tears in cadaveric populations ranges from 30%[90] to 60%.[91]

Ruptures of Achilles and tibialis posterior tendons are also quite common. The special anatomical situations of the flexor and extensor tendons of thumb and fingers subject them to high loads which may alter their histological structure and finally rupture them.

Healing of a ruptured tendon depends on the capabilities of both the tendon itself as well as on reactions from the surrounding tissues. Some tendons such as the Achilles tendon have a remarkable ability to heal after rupture, whereas others do not. Rotator cuff tears for instance usually do not heal and, if they do, a permanent weakening results. Healing of a ruptured Achilles tendon does not differ from the general tissue reactions after mechanical damage: the initial stage of exudation is followed by vascular granulation and fibroblast proliferation. Collagen synthesis begins within the first week and reaches its maximum after about 4 weeks. It then continues for about 3 months. Maturation and remodelling begin at the end of the third week and continue for up to 1 year after the injury.

Mechanical strength of a healing tendon is related to the histological process of repair. During the second phase, strength increases but is still insufficient to prevent further stretching of the healing wound. For this reason, the region always has to be immobilized until the process of maturation has begun (about 3 weeks after injury). At that time the arrangement of collagen fibrils is not organized and remodelling depends entirely upon the presence of repetitive tensile forces applied to the scar tissue. Several studies support the concept that controlled cyclic passive loading of the healing tendon, performed after the initial healing phase (3 weeks) is effective in decreasing the formation of abnormal adhesions and increasing the tensile strength of the healing tissue.[92–94]

Local swelling of a tendon

This is not infrequently seen in the digital flexor tendons in the palm of the hand. When these swellings are localized at the level of the heads of the metacarpals, they engage and may become fixed in the distal part of the tendon sheath at the moment of flexion. Extension is often only possible with help. This symptom is known as 'trigger' finger or thumb.

Treatment

Infiltration of steroid, to influence the inflammatory component of the swelling, is sometimes effective. If this fails, surgical removal of the swollen tissue (scar and/or calcium deposits) or slitting of the narrowed part of the sheath is the alternative.

An overview of the localization and treatment of musculotendinous lesions is given in Figure 3.20.

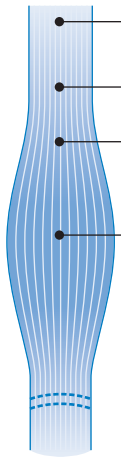
Musculotendinous lesions	Localization	Treatment
	1 Tenoperiosteal	Infiltration with triamcinolone or deep friction
	2 Tendinous	Deep friction
	3 Musculotendinous	Deep friction
	4 Muscular	Infiltration with local anaesthetic Deep friction Active and electrical contraction
	5 Tenosynovitis	Infiltration with triamcinolone or deep friction
	6 Tenovaginitis	Infiltration (or deep friction)

Fig 3.20 • Localization and treatment of musculotendinous lesions.

Ligamentous lesions

Rationale for treatment of acute and chronic lesions

Treatment regimes remain the subject of controversy and range from a policy of no treatment through early mobilization and strapping to plaster immobilization. However, experimental studies of the past decades confirm the existing clinical feeling that sprained ligaments heal better and stronger under functional loading than they do during rest. The effects of loading on healing ligaments have been studied extensively and the available evidence indicates that the remodelling of the repair tissues responds extremely well to cyclic loading and motion.[95] Long-term studies in ankle sprains have shown better results when early mobilization is used.[96,97] Other prospective and randomized studies also show the best results with early functional treatment (see Cyriax[98] p. 8, Larsen[99] and Freeman[100]). Because late reconstruction of ruptured ligaments at the ankle still gives very good results,[101–103] there is no need for early surgical treatment and conservative treatment with early mobilization must always be tried first. At the knee joint, several studies have also demonstrated that the non-operative management of an isolated medial collateral ligament rupture gives results equally as good as surgical repair but with significantly quicker rehabilitation.[104–109]

In spite of these findings, most physicians and surgeons dealing with the management of ligamentous ruptures reason 'anatomically': if a rupture is suspected or proven radiologically, the medical approach must be to repair the defect as soon as possible. This is achieved by partial or total immobilization or by early suture – the same approach as is taken in fractures, where the separated pieces of bone are fixed in apposition. This anatomical way of thinking does not correspond to the functional reality of connective soft tissue lesions – the function of a ligament is in no way comparable with the function of a bone. Where bone must be strong and solid, a ligament must allow and control movements within certain limits. To serve that purpose, ligamentous tissue must be mobile enough to change its position continuously. The same properties apply to scar tissue, which must not only be strong to prevent excessive movement but must also be mobile enough to allow sufficient movement. If this principle is neglected and a scar becomes unduly adherent (e.g. to bone), continuous functional problems will result. Early mobilization prevents such adhesions within or around the healing structure. Tension during the state of collagen deposition aligns newly generated collagen fibrils in the direction of stress and also prevents the formation of crosslinks in a random pattern. Consequently, the scar is strong in the direction along which force is applied. Tension also prevents scar tissue becoming adherent to bone. Movement stimulates proteoglycan synthesis important in lubrication of connective tissue and maintaining the critical distance between pre-existing fibres.

It is most effective to start mobilization from the onset, before the newly generated fibrils develop crosslinks in an abnormal and irregular pattern. This may be effectively achieved by deep transverse friction and passive movements. However, the serious traumatic inflammation and the intense pain during the slightest movement are very strong impediments to early mobilization of connective tissues. In these circumstances, Cyriax advocated the infiltration of localized lesions with small amounts of triamcinolone as soon as the patient is seen. This shortens the acute phase of the inflammatory process and therefore encourages the patient to move the injured part at an early stage with all the associated beneficial effects. In diffuse lesions this approach is impractical, and deep transverse massage and passive movements are substituted, although exercise and movement have to be modified until pain has abated.

'Sprain' of a ligament is the result of excessive joint movement with lack of muscular control. The transitional zone between mineralized fibrocartilage and bone is the site of most separations between ligaments and bones.[11] However, sprain may also occur in the substance of a ligament. A good example of this is the medial collateral ligament at the knee, where tears are often situated in the midportion or just below the joint line adjacent to the tibia. Experiments with strength and failure characteristics of rat medial collateral ligaments[110] have shown that this especially results from a large load that is rapidly applied. The failure point is reached before significant elongation can take place. The same load applied more slowly results in failure at the transitional zone between mineralized fibrocartilage and bone, where the connective structure is weakest.

In accordance with the degree of the injury, ligamentous lesions can be divided into three grades:

- *Grade 1*: a slight overstretching with some micro-tears within the ligamentous structure
- *Grade 2*: a more severe sprain with partial rupture of the ligament
- *Grade 3*: the ligament is completely torn across or is avulsed from the bony attachment.

This classification is rather arbitrary and, although it might be possible to distinguish a small lesion from a total separation, the difference between grades 1 and 2 is always subjective.

Ligamentous lesions can also be classified according to the time that has elapsed since the causative accident:

- *Acute*: within 48 hours
- *Subacute*: 48 hours to 6 weeks
- *Chronic*: more than 6 weeks.

This classification is of importance in relation to treatment.

Sprains with incomplete ligament rupture are often quite painful and accompanied by muscle spasm and pseudolocking. This makes clinical examination difficult, and diazepam or general anaesthesia may be needed to complete a thorough examination.

In complete tears there is rarely pain of a significant degree and in knee and ankle sprain the patient can often walk without aid. It is a fear of 'giving way' that characterizes the lesion and prevents the patient from doing more strenuous activities such as going up or down stairs, jumping or doing full squats.

Clinical evaluation of an acutely injured joint should be carried out as soon as possible – within a few hours of the accident – otherwise pain, swelling and muscle spasm will often make it impossible to perform proper ligamentous tests. This is particularly necessary in first and second degree lesions, in which these symptoms are so evident. History and knowledge of the mechanism of the injury are important aids in diagnosis and point to the injured ligament(s). Tenderness and localized oedema indicate the anatomical site of the tear in most instances.

Treatment

Minor ligament sprain should be treated conservatively. Ligaments will heal but it is essential not to strain them again during the first part of the granulation stage. For example, after damage to the medial collateral ligament of the knee joint, full extension should be prevented during the first 10 days.

In the acute stage, traumatic reactions such as pain and swelling should be kept to a minimum. Therefore, early application of compression and an elevated position of the extremity are most important.

In a sprained ligament of the knee or ankle, crutches should be used if movement is necessary. The next day, physiotherapy is started. Effleurage diminishes swelling and pain, which are impediments to movement. Thereafter transverse friction is performed to move the damaged tissue to and fro over the subjacent bone in imitation of its normal behaviour. This prevents the random orientation of newly generated fibrils and formation of abnormal crosslinks between newly regenerated fibrils and pre-existing collagen fibres. In this acute stage, really deep transverse friction will take no more than 1 minute since there is no question of breaking down strong scars. However, it should still be as gentle as is compatible with securing adequate movement of the damaged tissue. Then the joint is moved passively through its greatest possible range without causing pain. The same movement(s) is repeated actively. There should be no attempt to increase this range in the acute or subacute stage. In a sprained joint of the lower limb, instruction on gait follows. Strapping the joint to protect it from unwanted movements is a useful additional measure, especially if the patient seems anxious. Patients treated along these lines recover most rapidly.

An infiltration with a small amount of steroid at the site of the lesion is an alternative. The injection should be given during the first 48 hours – the initial and exudation stage. It reduces traumatic inflammation and prevents most structural and reflex changes. Pain also disappears, which enables the patient to move the joint in a normal way. Deep transverse friction then loses its efficacy.

Steroid injections during granulation and repair may lead to fewer fibroblasts, a diminished collagen fibre formation and a weaker scar.[111] However, these effects are not seen after a single injection during the acute, inflammation stage.[112]

Overstretching a ligament often leads to permanent laxity with consequent instability of the joint. Cyriax emphasized the propensity of ligaments not controlled by muscles to develop such permanent laxity and cited the sternoclavicular, acromioclavicular, sacroiliac and sacrococcygeal ligaments, the symphysis pubis, the cruciate ligaments at the knee and the inferior tibiofibular ligaments as examples.[75] After trauma, reflex muscle spasm is not capable of stabilizing these joints. In intracapsular ligaments (e.g. the cruciate ligaments), unsuccessful repair may also result from the synovial environment, limited fibroblast migration and reduced vascular ingrowth.

The subsequent traumatic inflammation can be reduced by the means already described. To prevent laxity, it is a principle of treatment to completely avoid movements which can be achieved by immobilization of the joint or by surgical repair. Such measures are best executed within 7 days.

After recovery, minor laxity is compatible with excellent function, whereas painful chronic laxity becomes painless in due course or can be converted into painless laxity by use of a local steroid infiltration.

In joints controlled by muscles, permanent laxity is much less likely to occur. Reflex muscle spasm efficiently stabilizes the joint. Grade I and II lesions treated on the lines set out will heal adequately. The results of conservative treatment in grade III lesions are also successful in almost all cases but it is essential that the lesion is isolated.[106,113] It is then advisable to immobilize the joint partially so as to prevent any unwanted movement taking place during recovery. Lasting instability in these joints can be more or less compensated for by tautness of the muscles and tendons passing over the joint. Strength-building exercises are of great importance and must be on an exact and planned basis. If necessary, strapping or braces may provide added support.

In surgical repair, dense fibrous tissue is used to reconstruct the ligament – the fascia lata, part of the patellar tendon on other tendons. Following transplantation a graft heals to the recipient tissues but never approaches the strength it had before transplantation. Nevertheless grafts can significantly improve the stability of joints.

Capsular lesions

Traumatic arthritis, capsulitis or synovitis all have an identical meaning: inflammation of the entire capsule as a result of a more or less recent trauma. This is invisible on X-ray and therefore this investigation cannot exclude the diagnosis. If trauma has damaged the lymph and vascular network in the

subintima, the integrity of the synovial membrane and therefore its cellular function will be severely disturbed. Increased permeability of small venules allows plasma to leak through and effusion results. Intra-articular swelling increases tension within the capsule and further irritates various sensory receptors. Pain and reflex inhibition of muscles result which can further affect the joint.

If the patient states that the joint suddenly became very painful and swollen over a few minutes, haemarthrosis is suspected. The speed of appearance of the effusion and the severe pain by far exceed that caused by clear fluid. Palpation of the joint also shows it to be hot and more tense than if the effusion is clear. Blood in the joint is a strong irritant and has an erosive action on the cartilage. It must be aspirated at once, after which the remaining blood-tinged synovial effusion is removed a few days later.

After injury, joints supported by muscles soon develop limitation of movement with a capsular pattern (see Ch. 4). In recent arthritis such limitation results from defensive muscle action which is reflexive and controls movements that could further irritate the capsule. Muscles spring into action, which can be felt (*hard end-feel*) and seen on gently forcing a particular movement.

In the chronic stage, inflammation is maintained by the effects of cartilagenous debris, blood elements and enzymes from destroyed cells which activate cells of the synovial membrane to produce excessive fluid of inferior quality containing hyaluronic acid molecules that are decreased in size and concentration. Friction resistance between joint surfaces is consequently increased and inhibition of excessive collagen proliferation reduced. Contraction of the capsule occurs and restricts movements, in the capsular pattern. In the absence of muscle spasm, end-feel then becomes rather hard, resembling leather being stretched.

In joints without muscles surrounding them to control movements, no limitation of movement can result, however severe the arthritis. Muscle spasm cannot occur and there is pain at the extremes of range only.

Table 3.4 Symptoms, signs and indications for therapy in traumatic capsulitis

Inflammation stage	Symptoms and signs	Therapy
Active	Constant pain Wide reference of pain Inability to bear weight on the joint Local warmth Spastic end-feel	Intra-articular steroid suspension Alternative technique: Slight distraction techniques
Chronic	No pain except on forced movement Little reference of pain Ability to bear weight on the joint Elastic capsular end-feel Resistance to passive movements is perceived before appreciable pain is evoked	Capsular stretching

Box 3.2

Contraindications of forced movements in traumatic arthritis of peripheral joints

- Elbow joint
- Hip joint
- Interphalangeal and metacarpophalangeal joints
- Lower radioulnar joint
- Acromioclavicular joint
- Sternoclavicular joint
- Sacroiliac joint

Treatment (Table 3.4)

Movements retain mobility at a joint. They have the effect of keeping a joint structure normal.[98]

In recent trauma, pain limits active movement and it is essential, particularly in middle-aged and elderly people, in whom posttraumatic adhesions are apt to form, to restore a full range of movement as soon as possible. Gentle assisted active and passive movement increases vascular supply, removes cartilagenous debris, resorbs oedema or joint effusion more quickly, aids in the transport of nutrients and metabolites to and from cartilage cells, and prevents the formation of adhesions and capsular contraction. Movements should be performed to the point of discomfort but not of pain. All possible directions of movement should be attempted, one by one, and a small but definite increase in range of movement should be achieved each day. If this fails, intra-articular corticosteroid injections are indicated.

In the chronic stage, stretching out the capsule requires many repetitions of a long steady push, maintained as long as the patient can bear it – e.g. 1 minute. Dense capsular adhesions finally yield but no increase in range of movement can be expected after the first few sessions. During the period when progress is slow, therapist and patient must show forbearance and persistence, respectively. Short-wave diathermy given for 15 minutes before mobilization diminishes the pain of capsular stretching.

Symptoms and signs of activity may contraindicate forced movements. Spontaneous pain, especially at night, wide reference of pain and inability to bear weight upon the affected joint all indicate that the lesion is in an active stage and that forced movements will increase the problem. Local warmth, effusion and muscle spasm are signs accompanying such an event and are indications for an injection of a steroid suspension. If the patient refuses, slight distraction techniques in the neutral position of the joint are an alternative. The therapist coaxes painless distraction of minimal amplitude, intermittently and with slight irregularity in depth and timing. Performed daily for the first week and on alternate days for the next week(s), pain abates and range increases slowly, although not in all cases of traumatic inflammation.

Forced movements should not be done in traumatic arthritis of the following peripheral joints (see also Box 3.2):

Table 3.5 Treatment of ligamentous lesions

Phase	Treatment (1)	Treatment (2)
Acute phase		
Joints controlled by muscles		
First day	Compression Elevation	Alternative (within 48 hours) Steroid infiltration
Following days	Effleurage + Deep transverse massage Controlled movements (active and passive) Gait instruction	Controlled movements (active and passive) Gait instruction
Joints not controlled by muscles	Deep transverse massage Immobilization	Infiltration (steroid or sclerosant) Immobilization
Chronic phase		
Adhesive scar formation	Deep transverse friction + Manipulation	(Steroid infiltration)
Lasting instability	Strength-building exercises Proprioceptive training	Surgical reconstruction (Infiltration with sclerosant)

- *Elbow*: passive mobilization for recent posttraumatic stiffness is apt to diminish rather than increase the range of movement; moreover, it is contraindicated because of the ever-present danger of setting up myositis ossificans. An intra-articular injection with corticosteroid suspension is indicated and will give quick recovery
- *Hip*: traumatic arthritis is best treated by rest in bed
- *Interphalangeal and metacarpophalangeal joints of the hand and foot*: these joints respond badly to forced movements. Joints of the toes can be injected with corticosteroid suspension
- *Lower radioulnar joint*: aggravation of symptoms can be expected even with active exercises.
- *Joints not under voluntary control*: after an injury the formation of adhesions is not to be expected. Forced movements are useless and harmful. Rest, support and corticosteroid injections are then good alternatives.

Treatment of acute and chronic ligamentous lesions is summarized in Table 3.5.

Access the complete reference list online at **www.orthopaedicmedicineonline.com**

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Clinical diagnosis of soft tissue lesions

4

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Introduction

The major part of this book is about making a clinical diagnosis: a system of clinical reasoning leading to a proper diagnosis. The final stage of the diagnostic procedure is the precise anatomical description of the lesion, for example: supraspinatus tendinitis at the superficial aspect of the tenoperiosteal junction, chronic subdeltoid bursitis, lesion at the origin of the extensor carpi radialis brevis muscle, periostitis at the anteroinferior surface of the fibula, annular disc protrusion at the L4–L5 level irritating the fifth lumbar nerve root.

During the last decades, new technology has revolutionized diagnosis and decision making in orthopaedic medicine. Previously, soft tissue lesions were characterised by a lack of objective findings. This has changed dramatically since the arrival of sonography, computed tomography and magnetic resonance imaging (MRI). These new techniques can demonstrate anatomical changes in soft tissues and therefore contribute significantly to the understanding of non-osseous orthopaedic lesions. However, they do not make clinical assessment redundant. Contrary to popular belief, diagnosis is not made by only looking at the result of a technical investigation. In the best case, an anatomical picture may be the ultimate confirmation of the clinical diagnosis. If imaging is undertaken too early during the diagnostic process it will create more problems and questions than it resolves, and often puts the examiner on the wrong track and leads to wrong therapeutic decisions.

First of all, not every detected anatomical lesion causes pain or dysfunction. Asymptomatic lesions do exist and are present in numbers that are much larger than previously assumed: asymptomatic herniations in cervical, thoracic and lumbar spine are present in up to 50% of the population.[1,2,3] Also the high prevalence of rotator cuff tears in elderly asymptomatic individuals is very well known.[4,5] It is estimated that in the general population, approximately two-thirds of all rotator cuff tears are asymptomatic.[6,7] Large numbers of asymptomatic lesions have also been demonstrated in the knee. A recent MRI study on asymptomatic soccer players demonstrated one or more MRI abnormalities in no less than 64%. Another study with MRI scans performed on the knees of asymptomatic male professional basketball players demonstrated an overall prevalence of articular cartilage lesions of 47.5%[8] and meniscal lesions of 20%.[9]

Another shortcoming of technical investigations is that they only detect an anatomical lesion (defect, swelling or other structural changes) and not the functional deficiency (weakness, limitation, laxity). In other words, the behaviour of the tissue during activity is not assessed.

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For all these reasons, there is no place for high-tech visualization techniques in the beginning of the diagnostic process and they should never be used as screening tests. A biased examiner who is looking for a particular lesion will often find that lesion, whether it is responsible for the complaints or not. Too many and too early technical investigations substantially increase the cost of medical care but do not give a better outcome. On the contrary, in the hands of an unprofessional doctor, high-tech investigations are potentially dangerous as they may lead to major, unwanted and unnecessary surgery.

Principles of diagnostic procedure in orthopaedic medicine

Clinical examination is all about behaviour of the tissues involved. The examiner must have a very good knowledge of the behaviour of the lesions that he is dealing with and of the behaviour of the normal tissue. Tissue behaviour is described by the patient during the inquiry and checked by the examiner during the functional examination. Looking for tissue behaviour, the following general principles are important.

1. Look for 'inherent likelihoods'

Some things are likely to happen

Soft tissue lesions behave in a very typical way and the examiner will therefore regularly be faced with the same history and the same response to functional testing. The symptoms and signs are closely related to the lesion present. The examiner should therefore try to recognize 'inherent likelihoods', a term defined as *the sequence of symptoms and/or signs that belong to the clinical picture of a certain pathological disorder and that are likely to be found, more or less in a sequence which is typical for that disorder*.

For example: in the history, a patient with lumbar pain may mention that on some days the pain spreads down the lower limb; tennis elbow is characterized by sudden twinges when objects are picked up; and in lumbar disc lesions pain may shift from one side to the other.

Functional examination can also show some inherent likelihoods. When resisted extension of the wrist hurts at the elbow, a tennis elbow is suspect and can be confirmed by positive responses to resisted extension of the wrist with the fingers held actively flexed and to resisted radial deviation of the wrist. In tendinitis at the radial insertion of the brachial biceps, apart from pain on resisted flexion and supination of the elbow, full passive pronation is also painful. In L5 sciatica, pins and needles in the medial three toes may be accompanied by numbness in the same area and by weakness of the extensor hallucis longus and peroneal muscles.

The examiner who has a knowledge of what is likely to happen should recognize this and compare the pattern to the 'unlikelihoods' presented by some patients, which indicate either a non-organic lesion, a somatic but non-orthopaedic problem or an unusual lesion. These inherent probabilities can of course only be recognized if the clinical examination is performed thoroughly.

2. Look for objective physical signs

Examination of the moving parts is an exercise in applied anatomy

The examination should include questioning (history taking) and testing which provokes or elicits symptoms and/or signs that can be assessed, judged and interpreted as objectively as possible.

The soft tissues of the locomotor system have the advantage that their functional anatomy is well known. It is clear how joints behave, how capsules and ligaments guide and limit movements, how muscles function and what movements they provoke.

Therefore, examination of the moving parts is an exercise in applied anatomy. Each tissue or group of tissues in turn must be tested and the answer interpreted in the light of the anatomical possibilities.

3. Avoid palpation as much as possible

The function of the different tissues is known

Although *palpation* is very often used as a diagnostic procedure, it is unreliable for several reasons:

- Some regions in the body are always tender to touch (e.g. lesser tuberosity at the shoulder, lateral epicondyle at the elbow, border of the trapezius muscle).
- Some structures lie too deeply and cannot be reached by the palpating finger (e.g. capsule of the hip joint, cruciate ligaments at the knee).
- The painful area does not always correspond to the site of the lesion (referred pain) and referred dural tenderness is sometimes present.
- Some patients with altered perception or desire to deceive the examiner may produce misleading responses.

It is easy to understand that, in these circumstances, palpation offers no help at all or, even worse, may misdirect the examiner.

Diagnosis, therefore, rests largely on the correlation of a series of semi-subjective data, obtained from a proper *functional examination* – an indirect approach. By assessing the function of each tissue in turn and interpreting the signs in the light of the anatomical knowledge, the examiner should be able to come to a correct description of the lesion.

The patient is asked to answer some very precise questions. A patient with an organic lesion exactly describes what is felt and gives the examiner a fairly precise clinical picture. The neurotic or malingering patient will feel the need to embellish so as to give a colourful description of suffering rather than of the symptoms.

4. Functional testing: the principle of 'selective tension'

The soft tissues can be put under tension

The different tissues of the moving parts can be subjected to strain which may increase the pain and tests are used to elicit or influence the patient's symptoms.

The possibility of making a diagnosis by selective tension depends largely on the characteristics of each tissue and on its capacity either to contract or to become stretched.

Muscles and tendons may be stressed by isometric contraction of the muscle or by passive stretching in the opposite direction. By contrast, ligaments and joint capsules can be put under tension by passive stretch.

If a certain test is positive, in that it provokes the symptom for which the patient consults, it establishes the relationship between the structure that becomes stretched, squeezed or contracted and the lesion.

It is important to try to use movements that put tension on one structure only, so that interpretation is as simple as possible. If a movement tests more than one tissue, accessory tests or palpation may be required to obtain further information that can differentiate between potential causes. For example, when testing the lateral ligaments at the ankle, a combined movement of passive plantar flexion and inversion is performed. If this is positive and, later in the examination, passive internal rotation at the mid-tarsal joints is negative, involvement of the calcaneocuboid ligament is excluded. In examination of the shoulder, painful resisted flexion of the elbow incriminates either the biceps or brachialis muscle; if resisted supination of the elbow is also positive, the lesion lies in the biceps muscle.

5. Use physiological movements as much as possible

Normal movements may become disturbed

This approach has some advantages:

- The structures that participate in the movement are well known (applied anatomy).
- The movements are easily controllable and reproducible. Pain may be provoked, but also limitation can be seen and weakness is not difficult to detect. The inter- and intra-tester reliability is quite high.[10–15]
- Patterns can be found: pain patterns, patterns of limitation and patterns of weakness. The recognition of a known pattern confirms the symptoms and signs presented.[16]

6. Distinguish between inert and contractile tissues

Soft tissues are either inert or contractile

This distinction is one of the pillars on which the whole system of orthopaedic medicine rests. The soft tissues of the locomotor system can be divided on the one hand into tissues that can contract (the contractile structures) and on the other hand, tissues that do not possess this capacity (the non-contractile or inert structures) (Box 4.1).

Contractile structure

The complex of muscle origin, muscle belly, musculotendinous junction, body of tendon, tenoperiosteal junction and also the bone adjacent to the attachment of the tendon are considered clinically as contractile (Fig. 4.1).

Box 4.1

Inert and contractile tissues

Inert tissues	Contractile tissues
• Joint capsules • Ligaments • Bursae • Aponeuroses • Dura mater • Dural sleeve of the nerve root • Peripheral nerves	The complex of: • Muscle–bone attachment (origin) • Muscle belly • Musculotendinous junction • Body of tendon • Tenoperiosteal junction (insertion) • The bone adjacent to the attachment of the tendon

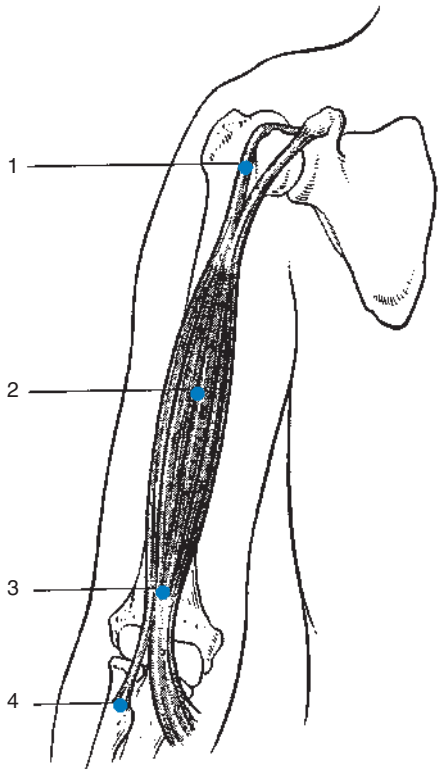


Fig 4.1 • Contractile structures – the musculotendinous unit: 1, tendon; 2, muscle belly; 3, musculotendinous junction; 4, insertion.

The only worthwhile method of testing these structures is by maximal contraction against resistance. The movement should be performed isometrically so that the applied tension, which causes pain, falls only on the muscle and the structures attached to it.

Passive movement in the opposite direction, which stretches the contractile tissue, can also elicit the pain but cannot be used as a specific test for it because non-contractile tissues are also stretched. For example, a lesion in the subscapularis tendon at the shoulder gives rise to pain on resisted internal rotation. Full passive external rotation may also hurt. This sign fits the clinical picture of subscapularis tendinitis but has neither diagnostic nor localizing value because the passive

movement also stretches the anterior joint capsule and the pectoralis major muscle and tendon.

However, a pain elicited by resisted movement does not invariably mean that a contractile tissue is at fault. If the bone close to the tendinous insertion is affected (fracture or other bony disorder), pain is evoked by the pull of the muscle. A contraction may also squeeze an underlying structure such as a lymphatic gland or bursa. When such tissues are inflamed, squeezing may evoke pain. The same applies when there is a disorder adjacent to muscles, for example an abscess. This explains why, for example, contraction of the sternocleidomastoid muscle may be painful in glandular fever and why contraction of the gluteal muscles can hurt in a trochanteric bursitis.

Inert structure

An inert structure does not possess an inherent capacity to contract and relax and can thus be tested only by passive stretching or squeezing. The inert tissues are shown in Box 4.1.

Active movements may also stretch or squeeze an inert structure but, because they also activate the contractile tissues, interpretation is subject to ambiguity and they cannot be used to test inert structures. For example, during active elevation of the arm, many muscles are in action (deltoid, supraspinatus, serratus anterior, trapezius). At the same time, certain parts of the joint capsule and some ligaments are stretched (acromioclavicular, sternoclavicular, conoid and trapezoid ligaments) and other structures are compressed (subacromial bursa, inferior acromioclavicular ligament, tendinous insertions of supraspinatus, infraspinatus, subscapularis and biceps).

7. Concentrate on 'the' pain

'The' pain is that pain for which the patient consults

When tests evoke pain, the examiner must make sure that this is the pain that is the patient's complaint. It is possible that some movements elicit pain in a certain area and that other tests provoke another pain in another region: one of these will be recognized by the patient as the presenting symptom. The examiner should then concentrate on this pain alone.

The situation often occurs because combined lesions are quite common. A patient may come to see the doctor with pain down the arm. If, after the history has been taken, it is not clear whether the pain originates either from the cervical spine or shoulder girdle or from the shoulder itself, the preliminary examination aims to clarify the situation. It may show some discomfort at the base of the neck when cervical movements are tested (especially in middle-aged and elderly people) but if only shoulder movements elicit the pain complained of, then this pain ('the' pain) is the primary problem; the other pain ('a' pain) is secondary. The arm pain will, of course, be dealt with first and only when this problem is solved is the other problem (if still present) approached.

Difficulties may arise in hypersensitive patients who report every tension they experience and for which they use different words: 'it hurts, it aches, it pulls, it stretches, …'.

8. The patient's cooperation is vital

The patient knows the symptoms

The patient's cooperation is essential, and it is vital that the questions put are understood. Details are sought on what activities have an influence on the symptoms and how symptoms behave over time. Except in psychologically disturbed patients, the more precise the questioning, the easier it is to obtain accurate answers. The patient must realize that, during functional examination, the examiner is looking for tests or movements that *elicit* symptoms. Most difficulties arise with those who are in constant pain, in that they tend to answer every question positively. It is the task of the examiner to explain carefully that movements that *alter* the pain are being sought. Not only tests that make the pain worse (a frequent occurrence) but also those that decrease the pain are considered important.

9. Take into account the patient's personality

The patient is a person

The history in particular will give an idea about the patient's personality. The reaction to pain can be assessed and a picture built up of the extent of disability. The findings can then be related to what is actually found when the examination takes place.

The view obtained from the history and physical examination may have therapeutic significance: for example, most patients can cope with active treatment such as manipulation or deep transverse massage but the clinician may obtain a 'perception' for those who cannot.

10. Keep the balance between credulity and excessive scepticism

Objectivity is a fair attitude

Orthopaedic medical disorders produce symptoms and signs that may be difficult to analyse objectively. Patients who have a reason to assume disorders for some type of personal gain, therefore, commonly use clinical features in the locomotor system to try to establish their credibility (see online chapter *Psychogenic pain*).

Although the examiner must be on guard against feigned illness, great care must also be taken to maintain a dispassionate attitude during the clinical encounter. The diagnosis of 'psychogenic pain' must not be made too quickly. Only when many inherent unlikelihoods are encountered during the history and functional examination should the examiner be suspicious about the veracity of the patient's story. Also, the discovery of a series of lesions is self-contradictory, because the development of several problems at the same time is most unlikely.

11. Request technical investigations only when necessary

Looking is not a substitute for thinking

Clinical testing is the first approach in orthopaedic medicine. Technical investigations, although sometimes very valuable, are only asked for in some situations:

- To exclude major lesions for which the functional testing has not been sufficient.
- To exclude contra-indications for some therapeutical actions (manipulations or infiltrations).
- To confirm the tentative diagnosis made after the clinical examination.

Warning

Incorporated into this system of clinical evaluation are *warning signs*: certain symptoms or combination of symptoms and signs indicate that something unprecedented is taking place and so alert the examiner to the possible presence of a potentially serious condition. Possible warning signs are, for example, pain in the upper lumbar area, deficit of more than one nerve root in the cervical spine, or a capsular pattern of the hip in children.

The presence of such warning signs will put the examiner on guard and indicate accessory clinical tests, further technical investigations or reference to a neurologist, an internist, a cardiologist or an oncologist.

These warning signs will be discussed further in later chapters.

Clinical evaluation

History

History is of prime importance in reaching a diagnosis. It is so well known as a method of determining symptoms that most examiners fail to realize how much information can be gained from it.

Patients are the best source of information in that they are suffering from the lesion and can best report precisely what is felt. It is then the examiner's task to translate the subjective *symptoms* into anatomical and functional conclusions.

Cyriax said: 'Every patient contains a truth. He will proffer the data on which diagnosis rests. The doctor must adopt a conscious humility, not towards the patient, but towards the truth concealed within the patient, if his interpretations are regularly to prove correct'.[17]

History taking is a slow business that requires time, patience and concentration; the examiner must do everything possible to gain the maximal detailed information. Vague, general description of the complaints should not be accepted but precise and detailed answers sought.

Most patients, and certainly when they are frank, are able to provide precise answers to the examiner's questions or can spontaneously give a well-structured, detailed and chronological account. They try to be as helpful as possible and are visibly pleased to talk to an interested physician. However, taking the history becomes more difficult in those who cannot express themselves or give a disjointed story. It is the examiner's task then to make sure that the right questions are asked in order to get useful answers. The same applies to talkative patients who try to be too helpful by adding all manner of irrelevant details. In these circumstances the examiner should concentrate on the important items only and bring the patient back to the point whenever there is a digression.

Patients with a clinical presentation that may rest in non-organic causes try to escape from precise questioning. They offer a garbled story full of internal contradictions.

Remarks

Questions should be asked in such a way that the account of the symptoms is given in *chronological order* which enables the examiner to get an idea of the duration and behaviour of the condition present. Knowledge of different dermatomes and of the possible likelihoods will help in interpretation of the evolution of the patient's symptoms.

Leading questions should be avoided, because they suggest to the patient what answer is expected. *The questions should be neutral*, so that the patient has to think about what is felt. An honest patient will have no problems in giving exact answers; one who dissembles has the opportunity to make mistakes and display inconsistencies.

Examples of questions that should be recast are as follows:

- Not: does the pain spread down your leg?
  - But: does the pain spread at all?
    If so, where to?
- Not: is it painful to cough?
  - But: what happens when you cough?
- Not: is it painful to bend forwards?
  - But: does anything bring the pain on?
- Not: do you feel pins and needles in your hand?
  - But: have you got any pins and needles?
    And if so, where?

When there is a *relationship between the patient's symptoms and rest, exertion, certain activities or certain postures*, then it is probable that the patient suffers from a lesion of the locomotor system. The main exceptions are angina and intermittent claudication. Questions should therefore be asked about the movements and positions that evoke, increase or influence symptoms, for example:

- What brings the symptoms on?
- What makes the symptoms disappear?

Some information can be obtained only from the history, and not from any other diagnostic procedure. For example, to ascertain the stage of shoulder arthritis, to find out whether a displaced fragment of cartilage is stable or unstable, to determine whether sciatica is caused by a primary or secondary posterolateral disc protrusion, depends on the answers to some very specific questions. These are not only diagnostically important but also have a prognostic value and can determine correct treatment.

For lesions of the knee or spine, the history is of extreme importance; the examiner must go into great detail and if this is done the diagnosis becomes apparent. For example, a patient may mention that pain started in the centre of the back, soon spreading unilaterally towards the buttock, and later radiating down the lower limb into the lateral border of the foot and the two little toes while at the same time pain in the back and buttock disappeared. After a while pins and needles began to

occur in the same toes and additionally they would go numb. The patient has revealed everything: the normal evolution of a protruded fragment of disc at the L5–S1 level, compressing the first sacral nerve root, is immediately apparent.

In some other joints, such as the shoulder, the history matters less but examination will disclose the lesion.

Taking the history

Age, sex, profession, hobbies and sports

Some disorders are confined to certain age groups so that the age of the patient may indicate diagnostic possibilities. For example, a patient of 14 who mentions internal derangement at the knee probably suffers from osteochondritis dissecans. The same story in a patient of 20 suggests a meniscal problem and at 60 years points to a loose body in an arthrotic joint. The same applies to the hip: trouble at the age of 5 is probably due to Perthes' disease; at 15 it could be the result of a slipped epiphysis; at 30 ankylosing spondylitis is a possibility; and at 50 arthrosis is more likely. A similar age distinction applies in root pain of cervical origin: under the age of 35 it is extremely rare that this is caused by a disc protrusion.

Certain disorders are more typical for men (e.g. primary sciatica and ankylosing spondylitis) and others occur more often in women (e.g. de Quervain's disease and the first rib, thoracic outlet syndrome).

The profession of the patient may sometimes give an idea about the causative strains that have acted on the affected joint. Also it may – in conjunction with hobbies or sports – have an influence on the decisions to be taken on treatment. Treatment for acute lumbago will be different in an employee who sits most of the day than in a docker who has to do heavy work; a patient with regular attacks of sudden backache will be advised against tennis, a sport full of quick movements.

Initial symptoms

The examiner should get an accurate picture of *the moment the symptoms first appeared*. The patient should be encouraged to recall that period and questions should proceed from that first instant.

The *onset* of the symptoms has to be clear. If they came on after an *injury*, a very detailed description of the accident should be elicited. The events immediately following the accident must be ascertained, in that compensation may be claimed because of inadequate or inappropriate management. The subsequent condition of the joint may not allow complete examination and therefore an idea of the direction of the forces acting on that joint and the position in which the joint was held at the moment of the accident are essential and will give a notion of the possible structures affected. The knee is an important example. In a 'sprained' knee the inflammatory reaction that follows the accident is so spectacular (swelling, limitation of movement) that proper functional testing becomes difficult, which means that in the acute phase the examiner has to rely mainly on the history to get an idea of what has happened. If the pain is felt at the medial aspect of the joint and the patient mentions a valgus injury, the medial collateral ligament or the medial meniscus are most likely to have been damaged.

Swelling of a joint after an injury may have come on immediately, in which case blood is the cause, or after a few hours, which is typically the result of a reactive effusion.

When the patient mentions a *spontaneous onset*, this may be either sudden or gradual. Apart from diagnosis this distinction may have therapeutic consequences. Backache, as the result of disc protrusion, that comes on suddenly is annular and requires manipulation, whereas a gradual onset suggests nuclear displacement, which is treated with traction.

The patient must exactly define the *first localization* of the symptoms. The area where the pain was first felt very often lies quite close to the site of lesion, referred pain usually coming on later. This does not apply to 'pins and needles'. They are mostly felt distally in the limb, from wherever along its length the nerve is affected.

Questions are also asked about *what influenced* the symptoms. The examiner looks for a relationship between activities, movements or posture and the symptoms.

Progression/evolution

The symptoms may be present without interruption from their onset. However, it is also possible that the patient describes a recurrence (see Box 4.2).

The progression of symptoms since their first onset is ascertained. The condition may have continued *uninterrupted*, in which case details are asked about the development of the severity of the symptoms and of the localization of pain. If the latter has remained *unchanged* from the beginning, this indicates that the lesion is quite stable and not evolving. When pain has *diminished* it usually indicates an improvement, although there are conditions (e.g. nerve root atrophy and certain cases of mononeuritis), in which the pain disappears long before the condition has resolved. Pain becomes *worse* as the condition progresses: in such circumstances it is important to know the length of time for which it has been present. This has diagnostic significance: it is clear that conditions such as metastases have quite a short time course. In contrast, slowly worsening pain is characteristic of some other conditions such as a neurofibroma. When the patient describes *intermittent* pain, details are sought about the occasions on which pain is

Box 4.2

Evolution of symptoms

- Uninterrupted
  - Unchanged
  - Diminished
  - Worsening
  - Intermittent
- Recurrent

felt. Nocturnal pain, for example, suggests an inflammatory condition.

A very important distinction should be made between the following definitions.

Reference of pain

Referred pain is a very typical feature in non-osseus lesions of the locomotor system. It is mostly segmental and thus experienced in a single dermatome, which indicates the segment in which the lesion should be sought. Reference of pain is influenced by the severity of the lesion: the more severe it becomes, so giving rise to a stronger stimulus, the further distally does the pain (usually) spread. The reverse also holds: reduction in the distal distribution is synonymous with improvement.[18,19]

It is therefore always important not to forget the question: 'Where was the pain originally and where has it spread since?'

Shifting pain

Pain coming on in one place as it leaves another indicates a *shifting lesion*.

This extremely significant phenomenon is well known in internal medicine: for example, when a renal calculus moves from the kidney down the ureter to the bladder and urethra, the pain experienced will follow the displacement. Pain is felt in the loin first, then in the iliac fossa, later in the groin and finally in the genitals. When the pain leaves one point, it is felt in another *instead*.

The same happens in soft tissue lesions. A good example is central backache, which becomes unilateral, then later on shifts to the buttock and finally to the lower limb – *the backache has become sciatica*. This shift can only be explained as follows: a structure lying in the midline and originally compressing the dura mater (backache) has shifted to one side and now compresses the dural sleeve of the nerve root (root pain). To be able to shift, that structure has to lie in a cavity and, because the pain was originally central, this has to be a central cavity. The only structure lying in a central cavity and able to change its position is the intervertebral disc: there is no other possibility (Fig. 4.2).

The same situation is encountered when a loose fragment of cartilage moves within a peripheral joint as, for example, often happens in the knee. Dependent on the position of the loose body in the joint space, the pain can be felt at the inner aspect, anteriorly or posteriorly and on other occasions even at the lateral side. Such moving pain indicates a moving lesion.

Expanding pain

This is synonymous with an *expanding lesion* – one that grows, for example a tumour. When it appears in another region the pain does not leave the area where it originated. It spreads, even beyond the boundaries of dermatomes. A patient may describe a pain that begins in the centre of the back and then becomes bilateral. It spreads to one buttock, and later to both, also increasing in the back. Later it spreads to one leg and even subsequently to both, while still becoming worse in the back as well as in the lower limbs. Such a course is one of expanding pain, as the lesion becomes more extensive.

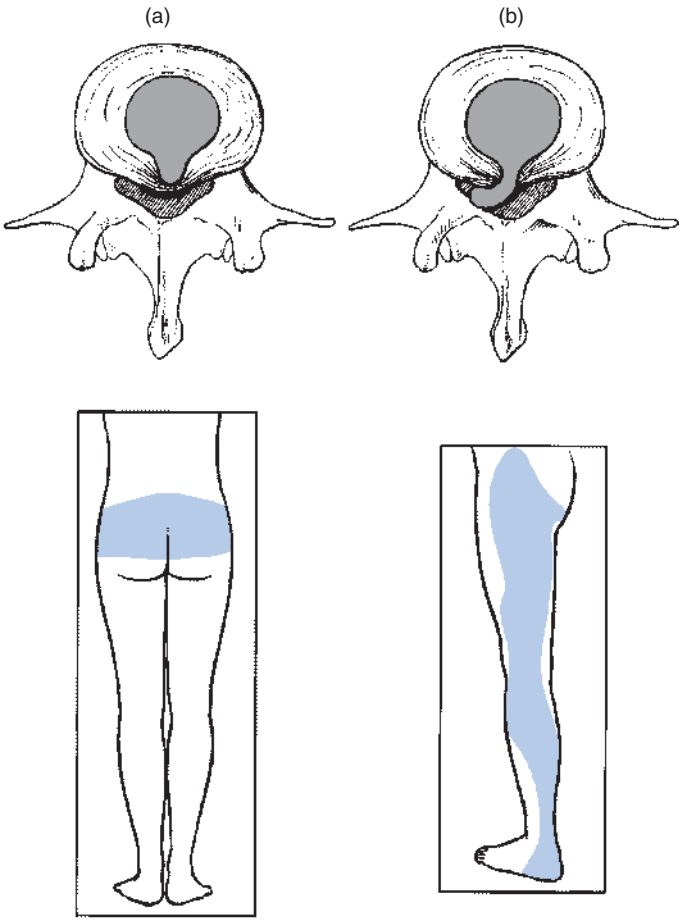


Fig 4.2 • Shifting pain: posterocentral disc protrusion causing central backache (a); shifts to posterolateral position causing unilateral sciatica (b).

Warning

An expanding pain, in which pain spreads but does *not* regress at the original site (in the way that a shifting pain does), is indicative of an expanding lesion and is usually the manifestation of a serious condition.

Another course is *recurrence*. Certain disorders, such as those causing internal derangement or of rheumatoid type, have a recurrent character. Some occur suddenly, others more gradually. If the symptoms occur intermittently, it is important to know whether the patient is or is not free of pain between attacks, because this has both prognostic and therapeutic consequences. Freedom from symptoms for a certain period of time suggests that the same may happen again. In internal derangement, regular recurrence implies that the loose fragment of cartilage or bone is unstable, in which case the maintenance of reduction will be the main concern of the therapist. A patient who is doing heavy work and who gets lumbago every 2 years must be regarded as having a stable lumbar disc which is completely different from a man with a light job who gets

Box 4.3

Pain

- Description
- Character: mechanical
  inflammatory
- Severity: therapeutic consequences
- Localization: diagnostic significance

Box 4.4

Characteristics of mechanical and inflammatory pain

Mechanical pain	Inflammatory pain
• Starting pain/stiffness	• Nocturnal
• During loading	• Morning stiffness
• Worse at evening	
• Moving in bed	

lumbago three times a year. In the first case reduction suffices, whereas the second will need other prophylactic measures to maintain the disc in place.

The onset of pain may vary from one attack to another. Backache that starts suddenly on some occasions, but gradually on others, very strongly suggests discal trouble. The localization of the pain may also change from one attack to another: it may be felt on one side of the body or of a joint and on the next occasion on the other side. This shifting pain is very typical of internal derangement, although there are some other conditions that may present the same picture (e.g. alternating buttock pain in sacroiliac arthritis caused by ankylosing spondylitis and alternating headache in migraine).

Actual symptoms

After having built up a complete picture of the patient's symptoms, information is sought about what is experienced at the time of interview.

Most patients consult the doctor because they have pain but other symptoms may also be described: pins and needles, numbness, limitation of movement, twinges, weakness and vertigo. These are sometimes forgotten by the patient and therefore the examiner must inquire about them. Every symptom must be given due weight and examined in detail.

Pain (Box 4.3)

There are many different ways of describing pain: it is amazing how much variation patients can achieve in their vocabulary and how many different descriptive terms can be used for the different sensations perceived. The reason lies in the fact that pain is mainly an unpleasant emotional state that is aroused by unusual patterns of activity in specific nociceptive afferent systems. The evocation of this emotional disturbance is contingent upon projection to the frontal cortex.[20,21] The nature of the pain may have some diagnostic value: everybody knows the throbbing pain of migraine, the stabbing pain of lumbago or the burning sensation of neuralgic conditions. Although the way the patient describes the pain may sometimes point to a certain disorder, it can also indicate the emotional involvement of the patient with the lesion.

Pain may have either a mechanical or an inflammatory character (Box 4.4). Mechanical pain (e.g. in arthrosis) is characterized by pain and stiffness at the beginning of a movement; augmentation when load is put on the joint; pain at the end of the day and absence of pain at rest, although moving in bed may also be uncomfortable. Inflammatory pain (e.g. rheumatoid arthritis, gout or infectious arthritis) wakes the patient at night and gives rise to frank stiffness early in the day.[22]

The severity of pain may be a determinant of the type of treatment that is chosen. For example, although sciatica without neurological deficit is not immediately an indication for surgery, discectomy may become the treatment of choice when the pain has become unbearable.

Finally, localization has some diagnostic significance. Pain may be felt centrally (on the midline), bilaterally or unilaterally. Central and bilateral pain usually point towards a lesion lying in the midline. A bilateral lesion is another possibility, but this is much less frequent. It should be realized, however, that central symptoms do not arise from a unilateral structure. And, although some structures lie very close to the midline (facet joints, costovertebral joints, erector spinae muscles), they are still unilateral and can only give rise to symptoms felt unilaterally. Unilateral pain originates in a unilateral structure or, when dealing with the spine, in a central structure that moved to one side and compresses nerve tissue unilaterally (e.g. a disc).

When the lesion is in the locomotor system, there should still be a relationship between symptoms and rest, exertion, activities, movements or posture. When coughing, sneezing or breathing hurts in an area other than in the chest, the dura mater could very well be responsible. Dural pain can be felt in the trunk far beyond the relevant dermatome.

Of special interest to the examiner are 'twinges': sudden short bouts of pain, which last only one second and are often associated with momentary functional incapacity. The occurrence of painful twinges may be the result of one of the following:

- Internal derangement
- Tendinous
- Neurological.

A momentary subluxation of a loose fragment of cartilage in a joint

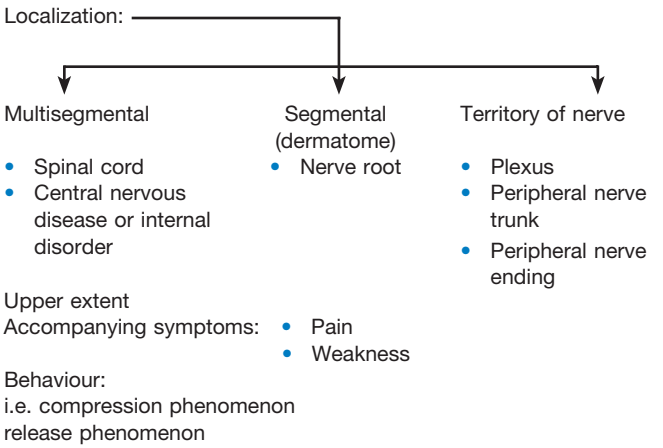
This happens quite often in the lumbar spine, the knee and the hip and less frequently in the elbow, ankle and subtalar joints. If there are any signs found during clinical examination, they will be articular – a non-capsular pattern (see p. 74). The combination of twinges and articular signs is pathognomonic of the existence of internal derangement.

A tendinous lesion

The patient recounts that, especially when the tendon is involved in movement, there are bouts of painful momentary

Box 4.5

Paraesthesia



weakness which arrest movement. This is common in tennis elbow, where the lesion lies at the origin of the extensor carpi radialis brevis muscle from the lateral epicondyle. It also occurs, although less frequently, in tendinitis at the shoulder, especially of the supraspinatus.

A neurological condition

These include tabes, post-herpetic or trigeminal neuralgia and Morton's metatarsalgia.

Paraesthesia

Non-painful sensory disturbances, paraesthesia, are strongly indicative of a condition that originates in a nerve (Box 4.5). They may result from an intrinsic lesion (primary neuritis or secondary polyneuropathy) or from an extrinsic cause (compression). They may also vary in quality and in intensity. In orthopaedic medicine the variation lies between numbness and real pins and needles. It is very often described as 'tingling'.

The moment the patient mentions the presence of pins and needles, the examiner should go into detail and ask the following questions:

- What brings on the pins and needles?
- What makes them disappear?
- How far proximally do they extend?
- Where exactly are they felt?

In entrapment neuropathies, knowledge of what brings the pins and needles on will show whether a compression phenomenon or the release phenomenon is acting (see pp. 26–27). For example, pressure on a small distal nerve gives rise to paraesthesia and analgesia in the cutaneous area of that nerve during the time of compression (e.g. meralgia paraesthetica). However, when a nerve trunk or nerve plexus becomes compressed, the paraesthesia are felt in a larger area, corresponding with the territory of that nerve and occur only after the compression has ceased (e.g. thoracic outlet syndrome). Nerve root compression results in segmental pain and paraesthesia felt within the corresponding dermatome (e.g. sciatica). Multisegmental bilateral paraesthesia indicates a lesion in the spinal cord.

It must be remembered that the site of compression always lies proximal to the proximal extent of the paraesthesia. They are usually felt in the distal part of the extremities. The more accurately the patient describes the area, the more distal the compression lies.

A paraesthesia-like feeling, especially vague tingling, may be experienced in some circulatory conditions, such as Raynaud's syndrome, but this is usually accompanied by changing of the colour of the skin in the distal part of the limb.

Functional disability

Often, functional disability is complained of. It comprises limitation of movement, internal derangement, weakness and incoordination and instability.

Limitation of movement

When limitation of movement is mentioned, its nature will have to be determined during the functional examination: limitation of active movements only, or limitation of both active and passive movements, and in this case whether it is of the capsular or of the non-capsular type. End-feel at the end of the passive movements and the relationship between pain and end-feel must also be ascertained (see pp. 73–74).

Internal derangement

Symptoms caused by internal derangement are irregular in nature. There are moments when the joint feels normal and that the patient is able to do everything, and other occasions when the joint does not work well. Typical symptoms are sudden twinges, shifting pain, giving way of the limb, locking of the joint and a feeling of insecurity.

If the answer to the question 'Does the joint lock?' is positive, further inquiry should make clear in what position locking occurs and how it is released. For example, a knee that locks in extension and unlocks spontaneously suggests a loose body, and one that locks in flexion and has to be unlocked manipulatively is a meniscal problem.

Weakness

This symptom should lead the examiner to concentrate during the examination on both active and resisted movements to see if the weakness is physically apparent or not. If weakness is confirmed, the distinction has to be made between a lesion in the muscle itself (e.g. atrophy or rupture) or in the nervous system, which is more often the case.

Incoordination and instability

These complaints are indications that something is amiss with either deep (proprioceptive) sensibility, the vestibular system or the cerebellum.

Further questions

Other questions, if appropriate, are asked about similar symptoms, past or present, in other parts of the body, especially other joints (see Box 4.6). If the answer is positive, conditions such as rheumatoid arthritis, spondylitic arthritis, Reiter's disease and gout should be suspected and further examination is required.

Box 4.6

Inflammatory disorders

Rheumatoid pattern	Reactive pattern
• Several joints	• Few joints
• Small joints	• Large joints
• Symmetrical	• Asymmetrical

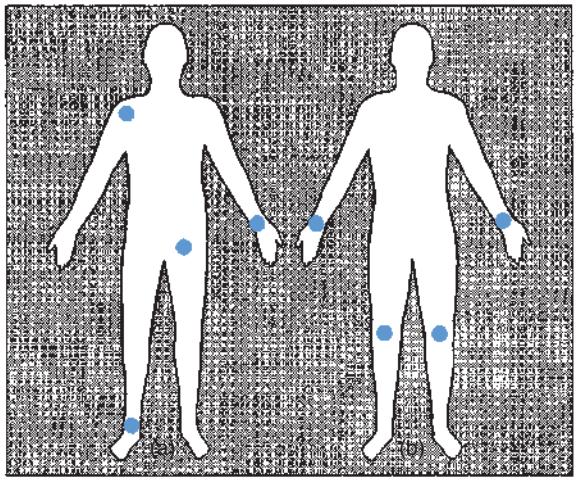


Fig 4.3 • Patterns of: (a) reactive arthritis; (b) rheumatoid arthritis.

Disorders of rheumatoid type (rheumatoid arthritis, lupus erythematosus, systemic sclerosis, dermatomyositis) are characterized by the symmetrical joint involvement, usually of the small joints (e.g. metacarpophalangeal joints). Arthritis of reactive type (e.g. peripheral joint involvement in ankylosing spondylitis, ulcerative colitis, Reiter's disease, sarcoidosis or psoriatic arthritis) affects a few large joints (e.g. shoulder, hip or knee) asymmetrically (Fig. 4.3).

Questions about the general state of health are asked to find out whether there is the possibility of a serious disorder (e.g. cancer).

The patient should also reveal present medication, and a doctor or therapist who considers manipulation should make sure that the patient is not taking anticoagulants: these are a contraindication because of the danger of haemorrhage.

Inquiries should also be made about previous treatments, which may give some idea of the chance of success of the proposed therapy. Previous surgery, its timing and indication are noted – it is not impossible that the present condition is the outcome of previous intervention (Box 4.7).

Box 4.7

Summary of history

1. Age, sex, profession, hobbies, sports
2. Initial symptoms:
   - Onset: • traumatic
     • spontaneous: sudden/gradual
   - Localization
   - Influence
3. Progression/evolution:
   - Uninterrupted
   - Changing: • reference of pain
     • shifting pain
     • expanding pain
   - Recurrences
4. Actual symptoms:
   - Pain
   - Paraesthesia
   - Functional disability: • limitation of movement
     • internal derangement
     • weakness
     • incoordination/instability
5. Further questions

Inspection

Inspection begins the moment the patient enters the room. If gait is disturbed, the way of walking may be diagnostic. An experienced examiner usually has the analytical ability to recognize, for example, the following typical patterns: a subluxated meniscus at the knee; a 'tennis leg'; the elderly patient with arthrosis of the hip; acute lumbago; a spastic gait; the patient with parkinsonism; a Trendelenburg gait; a drop foot; a ruptured Achilles tendon; and finally the patient with a non-physical condition.

Further features may be noted while the history is taken. A normally seated patient should have at least 90° flexion at hip and knee which is later confirmed on functional examination. The face may reveal the extent of pain or may disclose parkinsonism. The attitude in which a limb is held during the inquiry may be informative. Finally, the way the patient takes off clothes and shoes can provide further information on disability.

Deformities or deviations are easily seen: for example, in acute torticollis or in lumbago, the patient stands with the head or the lumbar spine held in deviation, usually towards one side. Real deformities can be the result of one or more fractures. Pathological fractures of a vertebral body lead to angular kyphosis or, if they occur at several levels, to a 'shortened' patient. Every examiner knows the typical kyphosis of adolescent osteochondrosis. Other examples are genu valgum and varum, which may be physiological up to a certain age but are pathological in adults. The presence of a deformity is not always relevant. It is quite possible that it has nothing to do with the presenting condition. A short leg or a long-standing scoliosis are very often seen but occur in asymptomatic as well as in symptomatic patients.

Soft tissues, such as skin and muscles, may show abnormalities. The colour of the skin may be different from that of other parts of the body: red in inflammation, blue when a haematoma is present or when a venous disorder has developed, white in arterial problems. Visible muscle wasting may be obvious. If swelling is present, the examiner should ascertain whether it is general and diffuse or localized.

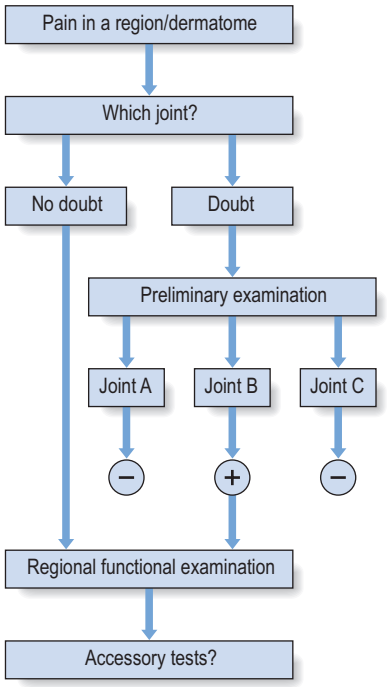


Fig 4.4 • Role of the preliminary examination.

An inflammatory condition may show quite spectacular signs such as redness of the skin, swelling and warmth.

Preliminary examination

After having taken the history and amassed evidence from inspection, the examiner should have an idea of which part of the body the symptoms originate from. Regional examination follows. When the history alone is not diagnostic, so that doubt over the origin or localization exists, a quick survey of the tissues forming the relevant segment may be necessary.

This 'preliminary' examination (Fig. 4.4) includes tests for the different joints. For example, the preliminary examination for pain down the arm includes tests for the cervical spine, shoulder girdle, shoulder, elbow, wrist and hand (Table 4.1).

When some tests are positive and indicate that the lesion lies about a certain joint, this joint will then be tested more rigorously. When no abnormalities are found, it is assumed that the lesion lies outside the moving parts. If all movements hurt, or when the patient presents a contradictory pattern, the question of a psychogenic disorder arises.

Functional examination

In lesions lying within the locomotor system, pain is brought on mainly by tension. Therefore, during examination the different tissues are selectively put under tension and the results noted.

As the function of the different tissues is well known, functional testing is really an exercise in applied anatomy. This indirect approach to diagnosis is purely mechanical.

Table 4.1 Movements tested in the preliminary examination of the upper limb

Root	Region	Type
C1	Neck movements	Active Resisted
C2–C4	Shoulder movements	Active Resisted
C5	Arm movements Shoulder	 Active Resisted
C6–C7	Elbow Wrist	Passive Resisted Resisted
C8	Thumb	Resisted
T1	Fingers	Resisted

The examination used is standardized, which permits a systematic search for *signs* in such a way that, with a minimum of tests, maximum information is obtained. Too many tests in the standard examination can confuse the examiner and make interpretation very difficult.

This does not imply that the diagnosis will always be reached after the standard functional examination. It may sometimes be necessary to add accessory tests (see pp. 66–67).

Procedure

The purpose of functional examination is to reach a decision on *which structure* is affected. However, even that may be insufficient. In a patient with a shoulder problem it is not enough to say that there is a lesion in the brachial biceps muscle: is it in the long head or the short head? In a lesion of the long head, is it localized in the bicipital groove or in its intra-articular course or at its insertion at the glenoid? Therefore, once the structure at fault is known, the next step must be to determine exactly *where, within that structure*, the lesion lies.

Examination is usually performed on both sides. Certainly, if the range of a passive movement is assessed, it has to be compared with the contralateral normal joint. When muscle strength is evaluated, comparison with the non-affected side is again necessary.

The patient as well as the examiner should adopt a good position from which to start. When passive movements are performed, the examiner must make sure that they can be done until the end of range (if present) is reached: movement should not be limited by the examiner's body, or by the couch or even by clothing. For resisted movements, which are done isometrically, the examiner's position must be such that the examiner can exert more power than the patient can. It may be of interest to put the patient in front of a mirror so that reactions can be observed.

The movements that are used to test the different structures should be 'pure' ones, i.e. testing only one structure or

Box 4.8

Aims of testing active movements

- To ascertain patient's willingness and ability
- To determine range
- To assess muscle power

one group of structures at a time. This facilitates subsequent interpretation.

Questions asked during examination should be as neutral as possible. The examiner must not impose on the patient an expected answer. Questions such as 'Does this movement hurt?' or 'Is this painful?' should be avoided. It is better to say 'How is that?' and ask the patient to report anything experienced. It must also be understood that the examiner is looking for tests that elicit the symptoms or, if they are constant, those that influence them.

Movements

The functional examination contains active movements, which are not always necessary, passive tests and movements against resistance. Positive tests are always repeated on the opposite side in order to compare the normal with the abnormal.

Active movements

Active movements are mainly used to determine quickly the area from which symptoms originate and what structures to test in detail with passive and/or resisted movements (Box 4.8).

Examination by active movements goes against the principle of trying to test one structure at a time. When a limb is actively moved, muscles, tendons, ligaments, bursae and capsule are all put under stress. In consequence such tests are non-specific and therefore not always necessary. However, they can be of interest because they suggest what the patient is able and willing to do. In most cases, they also give an idea of the range of movement possible (normal, limited or excessive) and of muscle power. They are always executed first to provide a criterion with which to compare subsequent passive and resisted movements.

When a normal range is later found on passive testing and normal muscle strength is apparent when tested against resistance, there is no reason for any abnormality or restriction of active movements. Thus if, in these circumstances, it is claimed that the active movement cannot be done, there must be refusal, either involuntary (psychogenic) or voluntary (intention to deceive). There are, of course, organic lesions where a discrepancy exists between active and passive or resisted movement but only within certain limits. For example, a mononeuritis of the long thoracic nerve, leading to weakness of the serratus anterior muscle, can make full active elevation of the arm impossible even though the passive range and muscle power of the shoulder are normal. A further example is total rupture of the Achilles tendon, in which resisted plantiflexion is weak and active movement impossible despite the range of passive movement at the ankle remaining unaltered.

Active movements are usually required when the spine (cervical, thoracic and lumbar), the shoulder girdle and the shoulder are examined but they may also be tested at any other joints if they can supply additional information.

Passive movements (Fig. 4.5)

These tests are meant to examine the inert tissues. The information they give is precise, provided they are performed correctly. The patient should completely relax the muscles and let the examiner do the movement. It is important to realize that non-inert tissues may also be stretched by the manœuvre: if this gives rise to pain, it must be determined whether there is a 'contractile tissue pattern', in which case active contraction in the opposite direction will also be painful (see pp. 77).

The criteria for passive testing are pain, range and end-feel (Box 4.9).

Pain

The patient must understand that the examiner is looking for 'the' pain – the reason for presentation – and that each time this pain is evoked or influenced a response is required. When straight-leg raising is performed in a patient with low back pain radiating down the posterior aspect of the thigh, the examiner must make sure that the sensation felt during the test is the exact pain complained of and not just a tight feeling in shortened hamstring muscles.

Pain evoked by a certain movement requires the further information of exactly *where* it is and *at what stage of the movement*. Pain may come on at a certain moment but the movement is not necessarily limited. For example, straight-leg raising may become painful at 60° but continue to full range without increased discomfort. Excessive gentleness may fail to elicit information. Passive movements must be performed as comfortably as possible for the patient but, if a proper answer can only be obtained by pushing a bit harder and thus hurting the patient, this should be done. It would be a diagnostic error to interpret a perfectly full movement as being limited by a few degrees, if the movement was stopped because of pain. The same applies to a painful arc. This may be so uncomfortable that the patient cannot get beyond it by active movement. If an arc is suspected, a harder push during passive movement may succeed and show its presence. Interpretation would be totally different: on the one hand, limitation of movement and, on the other hand, full range with a painful arc.

The presence of joint signs in root lesions is a misleading phenomenon. When a cervical or lumbar nerve root becomes compressed as the result of a disc protrusion, passive movement in a neighbouring joint may occasionally affect the root pain. Thus passive movements for the shoulder joint may hurt at the end of range so focusing attention on the shoulder. As pain of cervical origin is also felt down the arm the picture becomes very confused. The same occurs in lumbar root pain; passive hip movements are not limited but they may influence the pain when they are brought to full range – they are

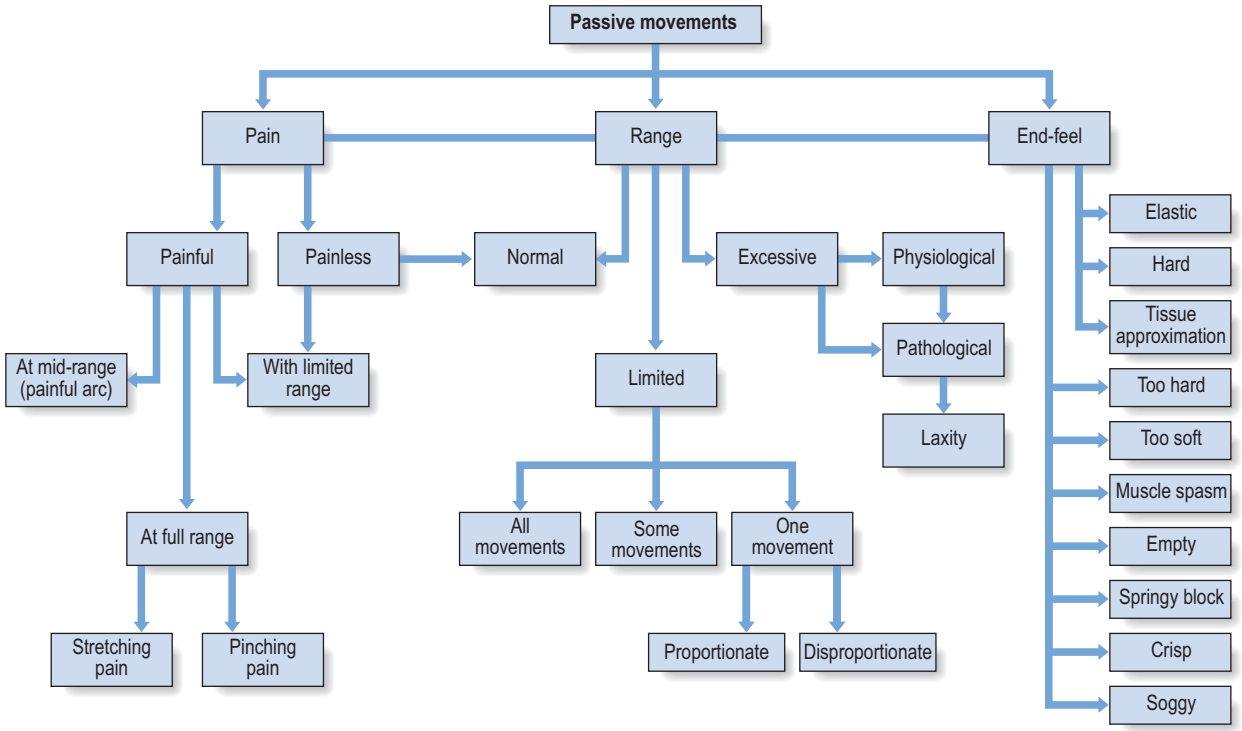


Fig 4.5 • Passive movements.

Box 4.9

Aims of testing passive movements

- To assess pain
- To determine range
- To characterize end-feel

probably capable at their extreme of altering the tension on the nerve roots in a minor way, analogous to straight-leg raising.

Amplitude

The range of movement is noted and is always compared to the unaffected side. The response should be interpreted in the light of the patient's age and general condition.

Limited movement requires the examiner to ascertain whether the limitation is in all directions or only in some directions or in one direction only. If only one movement is reduced, a 'proportionate' or a 'disproportionate' limitation may be present (see p. 76).

Pain at full range suggests that the pain is provoked by stretching or pinching of the affected structure. The localization of the pain will very often be indicative.

Excessive range may be pathological but laxity is sometimes purely physiological: for example, most women can hyperextend the elbow. When a joint moves further than is normally accepted, great care must be taken that the symptoms are not too readily ascribed to that phenomenon. Hypermobility with a normal end-feel is usually not of significance.

End-feel

This is a term typical of Cyriax. It describes the sensation that the examiner experiences at the end of the passive movement. The hand that performs the passive movement is not only motor but also sensory. When the movement comes to the end the examiner should assess the sensation. When no limitation is present the end-feel is at the end of the normal range. When limitation exists, the end-feel is judged at the end of the possible range.

End-feel is diagnostically important as it gives an idea of the structure or condition that stops the movement.[23] In addition it has therapeutic consequences. Especially during attempted spinal manipulations, the sensation imparted to the operator's hand will indicate whether or not to proceed. Likewise, in the shoulder or hip, the decision whether to undertake capsular stretching depends on end-feel.

For accurate judgement of the nature of end-feel, experience is required. Passive movement should be executed with extreme gentleness, especially during the final degrees of range. The moment resistance is first noted, the rate of movement is reduced so that the feeling can be assessed before movement comes to a complete stop. In normal circumstances each joint movement has a characteristic end-feel, which can be considered as 'physiological'. In pathological conditions, end-feel may change. The examiner should know what the normal feeling of each (passive) movement of a joint is like in order to be able to judge every change in end-feel.

Physiological end-feel

This can be hard, elastic or related to tissue approximation:

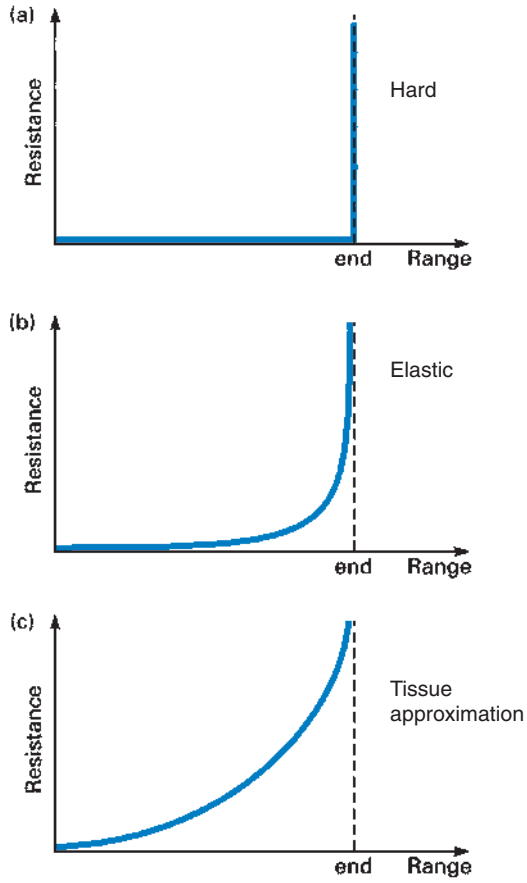


Fig 4.6 • Hard (a), elastic (b) and soft tissue approximation; (c) end-feels.

- *Hard*: the movement comes to an abrupt stop, either because two bony surfaces meet (bone-to-bone end-feel) or because a ligamentous structure prevents further movement (Fig. 4.6a). Extension of the elbow has a bone-to-bone end-feel, because the main factor that stops the movement is the engagement of the olecranon process in the olecranon fossa. Extension of the knee also has a hard stop, though less clearly direct than the elbow; here, the main structure that stops movement is the posterior cruciate ligament, which does not stretch.
- *Elastic*: at the end of range the examiner feels resistance but further stretching over a few degrees is possible (Fig. 4.6b). The feeling is one of two pieces of tough rubber being squeezed together or of a piece of thick leather being stretched. This end-feel is typical of a normal joint capsule and is felt during most rotation movements in shoulder, elbow, hip, knee, cervical and thoracic spine. The tern 'elastic' has the same meaning as 'capsular'.
- *Tissue approximation*: this end-feel is normal in those movements where extra-articular soft tissues engage against each other (Fig. 4.6c). Flexion at elbow and knee give this 'soft', 'muscular' end-feel. At the elbow the muscles of the forearm come in contact with the biceps and at the knee the calf muscles engage against the hamstrings. This 'extra-articular' end-feel does not occur in patients with poorly developed muscles: they present an 'articular' end-feel, either hard or elastic.

Pathological end-feel

A number of types of end-feel are pathological.

- *Too hard*: the normal elastic, or tissue approximation, end-feel may become harder when the quality of the tissue that stops the movement alters under the influence of pathological conditions. This change very often accompanies limitation of movement because of capsular contracture, osteophytic outcrops of bone, myositis ossificans or a malunited fracture close to the joint. Dependent on the condition, the end-feel may vary from a slight increase in hardness (early arthrosis) to a bony block (ankylosing spondylitis). The latter forms a contraindication to further stretching (i.e. during manipulation).
- *Too soft*: in those instances where a hard end-feel is expected, the movement may come to a soft stop. This is especially the case in a loose body blocking a movement as may happen at the elbow: extension is limited by a few degrees with a soft end-feel.
- *Muscle spasm*: this involuntary muscular contraction usually indicates severe disorder. The muscles contract with a sudden, vibrant 'twang' even when the movement is performed very gently. This is reflex protective spasm and may happen in acute conditions, such as arthritis in the acute stage. It may also be a sign of more serious lesions such as recent fracture (e.g. scaphoid fracture at the wrist) or secondary deposits (e.g. in a cervical vertebra). Such an end-feel is always an absolute contraindication to manipulation.
- *Empty*: movement may be so painful that the patient begs the examiner to desist. The latter feels that further movement would be possible because there is no organic resistance but stops the movement because of the perceived pain or out of sympathy for the patient. Further forcing would result in active voluntary muscular contraction. This situation always indicates a serious problem. Acute bursitis, extra-articular abscess or neoplasm is a possibility, but there is nothing wrong with the joint itself. This end-feel may also indicate laxity in a joint or a non-organic lesion. In the latter case there is initial strong resistance, which yields to sustained pressure, disclosing a full range of motion.
- *Springy block*: this finding always indicates internal derangement: when part of an intra-articular cartilaginous tissue displaces, it may prevent a full range of movement which leads to a rebound at the extreme of the possible range. The commonest examples are a displaced meniscal fragment in the knee and a buckled end-plate in the lumbar spine.
- *Crisp*: this is typical for a disc protrusion and is best felt in the cervical spine. The feeling is the result of small involuntary muscular contraction at the end of the possible range, known as 'muscle guarding'. The condition is not acute, as a result of which there is no real muscle spasm.

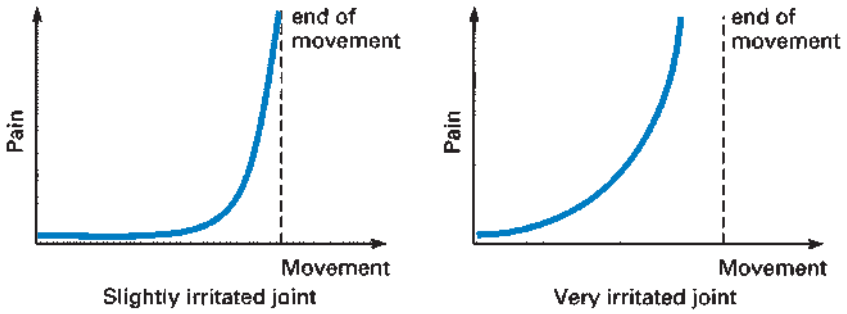


Fig 4.7 • Relationship between pain and end-feel.

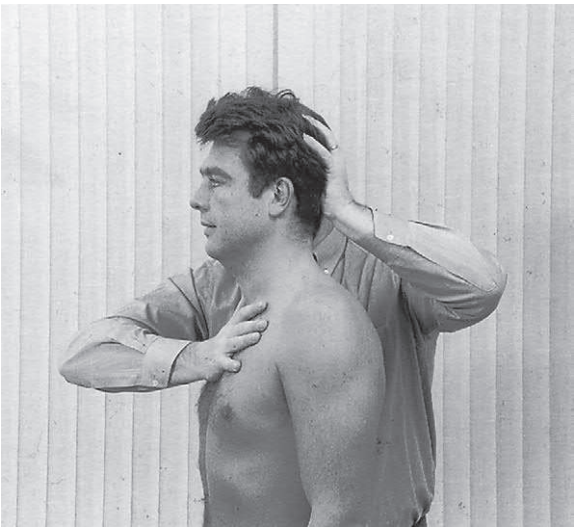


Fig 4.8 • Resisted extension of the neck.

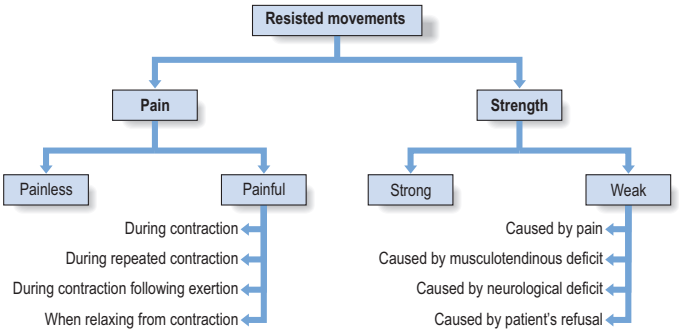


Fig 4.9 • Resisted movements.

- *Soggy*: this end-feel is seldom encountered and can be situated somewhere between a soft and an empty feeling. It is typical of rheumatoid arthritis in the upper cervical joints and is a strong contra-indication to manipulation.

Relationship between pain and end-feel

It is also important to look for the relationship between the moment of appearance of pain and that of the end-feel. Usually the pain comes on at the same time as the end of range is sensed (Fig. 4.7). Earlier pain implies that the joint is in a very irritated state which has therapeutic implications.

Resisted movements

Movements against resistance are performed isometrically. The joint is put in a neutral position and should not move when resistance is applied. This method ensures that stress on inert tissues is minimal or absent.

The position of the examiner must be such that the force exerted by the patient can be resisted. A maximal contraction is asked for and held for a few seconds. Counterpressure is therefore taken by the other hand at the opposite side. For example, to test resisted extension of the neck, one hand is put on the occiput and another hand on the sternum (Fig. 4.8).

Box 4.10

Aims of testing resisted movements

- To assess pain
- To determine muscle strength

Similarly, to test resisted flexion, one hand is placed on the forehead and the other between the scapulae.

The criteria when testing against resistance are pain and strength (Box 4.10).

Pain

In a lesion that lies in the contractile tissue, the pain should be elicited during the contraction. In mild disorders the test may initially be negative but repetition of the movement may eventually provoke symptoms. Similarly, slight muscular or tendinous problems may give pain only during exertion and resisted movements may remain painless. If pain cannot be elicited by repetitive contractions, the patient should be seen and tested in the circumstances when pain is experienced. The resisted movements may then be found positive.

There are cases of tendinitis in which the pain is only felt when the patient relaxes after muscle contraction. This is not uncommon and can also be interpreted as a positive resisted movement.

A positive answer on resisted movement is usually only one – but the most important – part of a 'contractile tissue pattern'

in which the passive movement that stretches or pinches the affected part is also painful.

The examiner must be aware of inert tissue lesions that can become painfully squeezed or moved by muscular contraction (e.g. gluteal bursitis).

Strength

The movement should be strong, but weakness may occur and an experienced examiner will immediately have an idea of what is the cause. It may be the result of pain; in that instance, the examiner feels a sudden cessation of power – the patient stops the contraction when the pain is felt. This often happens in partial muscular or tendinous ruptures when sufficient fibres are torn to diminish strength.

In total contractile tissue ruptures, there will not be pain but complete absence of muscle power. This pattern is completely different from a neurological weakness. Here there still is a force that has to be overcome: the examiner is stronger than the patient and so can push the patient's limb away while still feeling continuing resistance. The latter can vary from an almost normal sensation – slight paresis – to very little (scarcely detectable) force – complete paralysis. This may be the result of a lesion that has completely stopped motor innervation or it may be a consequence of the patient's refusal to undergo the manœuvre.

Accessory tests

After a well-balanced basic functional examination, interpretation of the pattern that emerges should, in most cases, make it possible to single out the tissue at fault. Difficulties are, of course, encountered and further accessory testing is then required to reach a precise diagnosis, to positively confirm an existing but tentative diagnosis or to disclose the precise point affected within the structure.

An important feature is that a distinction should be made between standard functional examination and accessory tests. The basic examination is always done in its entirety, whereas the accessory tests are applied selectively (Box 4.11).

It is wrong to do accessory tests without a prior idea of the nature of the problem, and in the hope that more tests would automatically provide more complete information. On the contrary, the more tests the more confusing the picture may become and in the end the 'wood cannot be seen for the trees'. Accessory tests therefore should be goal-oriented. They are performed in the following circumstances.

Box 4.11

Aims of accessory tests

- To differentiate within a group of structures
- To confirm a tentative diagnosis
- To unravel a difficult pattern
- To extend a negative examination
- To make a differential diagnosis
- To understand unusual signs

After testing a group of structures

When a resisted movement has tested a group of structures, further differentiation is sometimes necessary to find in exactly which structure the lesion lies.

In 'tennis elbow', resisted extension of the wrist is painful. To find out whether the lesion lies in the extensors of either the wrist or of the fingers the test is repeated with the fingers held actively flexed, so inhibiting the finger extensors. A negative answer implicates the wrist extensors, which can then be differentiated by executing resisted radial and resisted ulnar deviation: when resisted ulnar deviation hurts, the extensor carpi ulnaris is at fault; whereas if resisted radial deviation is painful, the lesion clearly lies in one of the radial extensors of the wrist – true tennis elbow. Whether the tendinitis is situated at the extensor carpi radialis longus or brevis cannot be found by further testing – palpation is called for.

The same applies to the hamstring muscles. A muscular or tendinous lesion about the knee will give rise to pain on resisted flexion. Differentiation between either (a) the femoral biceps or (b) the semitendinosus and semimembranosus is achieved by testing resisted external and internal rotation. Positive lateral rotation incriminates the biceps and medial rotation draws attention to the other two muscles. Palpation identifies exactly where the lesion lies.

To confirm a tentative diagnosis

An accessory test may help, especially in uncommon conditions, to confirm a tentative diagnosis. For example, when the diagnosis of mononeuritis of the spinal accessory nerve has been suspected, weakness on resisted scapular approximation will show the diagnosis to be correct.

In difficult cases

A clinical picture can sometimes be difficult to interpret and accessory tests may then resolve doubt. When pain is thought to be due to squeezing of a structure, diagnostic traction may help to confirm this. For example, in difficult cases of internal derangement of the cervical spine, pain and/or paraesthesia can be temporarily abolished by applying manual or mechanical traction. Traction on the arm can diminish pain in subacromial bursitis.

When the functional examination is negative

A negative examination does not necessarily mean that a lesion is absent. There may be a problem in a neighbouring tissue which was not systematically tested in the standard examination. A negative shoulder examination, for example, invites the examiner to test the coracobrachialis muscle. In anterior knee pain a negative examination is followed by tests of the patello-femoral joint.

In differential diagnosis

Different conditions may give rise to similar positive answers. An accessory test can then help to elucidate which structure is at fault. For example, limitation of external rotation at the shoulder is the primary sign of anterior capsular contraction as well as of a subcoracoid bursitis. Passive external rotation with the arm held in abduction will be positive in a capsular lesion and negative when a bursitis is present.

A test for distant pain can be performed when a lesion lies in a long bone. When a fracture is suspected on the dorsal aspect of a rib, pressure on the anterior aspect or on the sternum may provoke the pain. This technique can also be used to differentiate a rib problem, a lesion in an intercostal muscle or at a costovertebral joint.

When unusual signs are found

Discrepancies between individual findings on functional examination may suggest an unusual and possibly serious lesion, a psychogenic problem or an attempt to deceive. In the former, the patient is sent for further specialized examination. When neurosis or deception is suspected, the patient is subjected to a number of accessory tests: additional movements that cannot possibly influence the alleged symptoms or repetition of tests in other positions. The opportunity for the patient to make mistakes is thus presented, which in a positive way can confirm the examiner's suspicion (see online chapter *Psychogenic pain*).

Palpation

Palpation can be a very useful aid to diagnosis when performed at the right moment. However, it is frequently done too early in the examination, with the risk of an incorrect diagnosis. When, after functional testing, the examiner has interpreted the different movements and the pattern that emerged, and still thinks that more information could be forthcoming about the lesion, palpation is undertaken. Abnormal sensations that are imparted to the hand in a stationary as well as in a moving joint are sought.

Palpation of the stationary joint

During palpation of a motionless joint (Fig. 4.10a) the examiner may assess temperature (either warmth or cold), swelling, synovial thickening or structural changes. Palpation may also elicit tenderness.

Warmth

Warmth is best palpated with the dorsum of the hand and may be diffuse or localized. Spurious warmth can be the result of a rubefacient ointment or of a bandage that has been removed just before examination. Otherwise, warmth always indicates activity. The lesion may be osseous (e.g. fracture or metastases) or articular (e.g. arthritis, haemarthrosis, loose body, or a disorder of a meniscus or ligament) or tendinous (e.g. rheumatoid-type tendinitis). A purely mechanical tendinitis will not cause any warmth. When the warmth occurs suddenly and twinges are also present, this indicates persistent subluxation of a fragment of cartilage. The heat may or may not diminish when the joint is kept still. Sometimes (e.g. in loose body) the warmth may be elicited merely by the examination.

Cold

When the extremity feels cold an arterial problem is suspected. Palpation will then follow for arterial pulsations. Cold may also occur during an attack of sciatica, especially that which causes muscular weakness. When the foot becomes cold only after exertion, the probable cause is an iliac thrombosis. The cause may also be neurological.

Swelling

Swelling is the result of an articular reaction to a lesion and may be localized or generalized. Swelling that came on after injury may be the outcome of haemorrhage or effusion. If it is caused by bleeding, the joint fills up within a few minutes; effusion develops over a few hours. Blood also fluctuates: the swelling moves *en bloc*. The presence of pus is exceptional and indicates an infection with microorganisms. Periarthritic oedema may pit. In a localized swelling, its consistency should be ascertained: a soft swelling indicates subcutaneous clear fluid or a thickened bursa, a fluctuating mass may result from a haematoma or from a mucocele and a hard but still fluctuant swelling is typical of a cyst or a ganglion. When the consistency is bony the cause is usually a callus, a bony subluxation (e.g. capitate bone at the wrist or cuboid bone in the midfoot), an osteophytic outcrop or any other bony deformity which results from a destructive process (e.g. osteitis deformans, neoplasm).

Thickening of the synovial membrane

This is found in rheumatoid, bacterial or inflammatory arthritis (e.g. gout, tuberculosis, gonorrhoea, Reiter's disease, ulcerative colitis, spondylitic arthritis or psoriatic arthritis). It is absent in mechanical conditions, such as traumatic arthritis, post-immobilizational arthritis and arthrosis. To palpate capsular thickening, the examiner has to seek the reflexion of the membrane where it overlies a bony prominence.

Gaps

A gap may be palpated at the site of the rupture of a muscle or tendon.

Tenderness

Palpation for tenderness is sometimes performed to determine the exact localization of a lesion. For this purpose, it is done only in a structure that has already been found, by clinical examination, to be affected and only when it is within reach of the finger. Eliciting tenderness is only necessary when, after clinical examination, the diagnosis still lacks precision. When a localizing sign has been found, palpation is of course

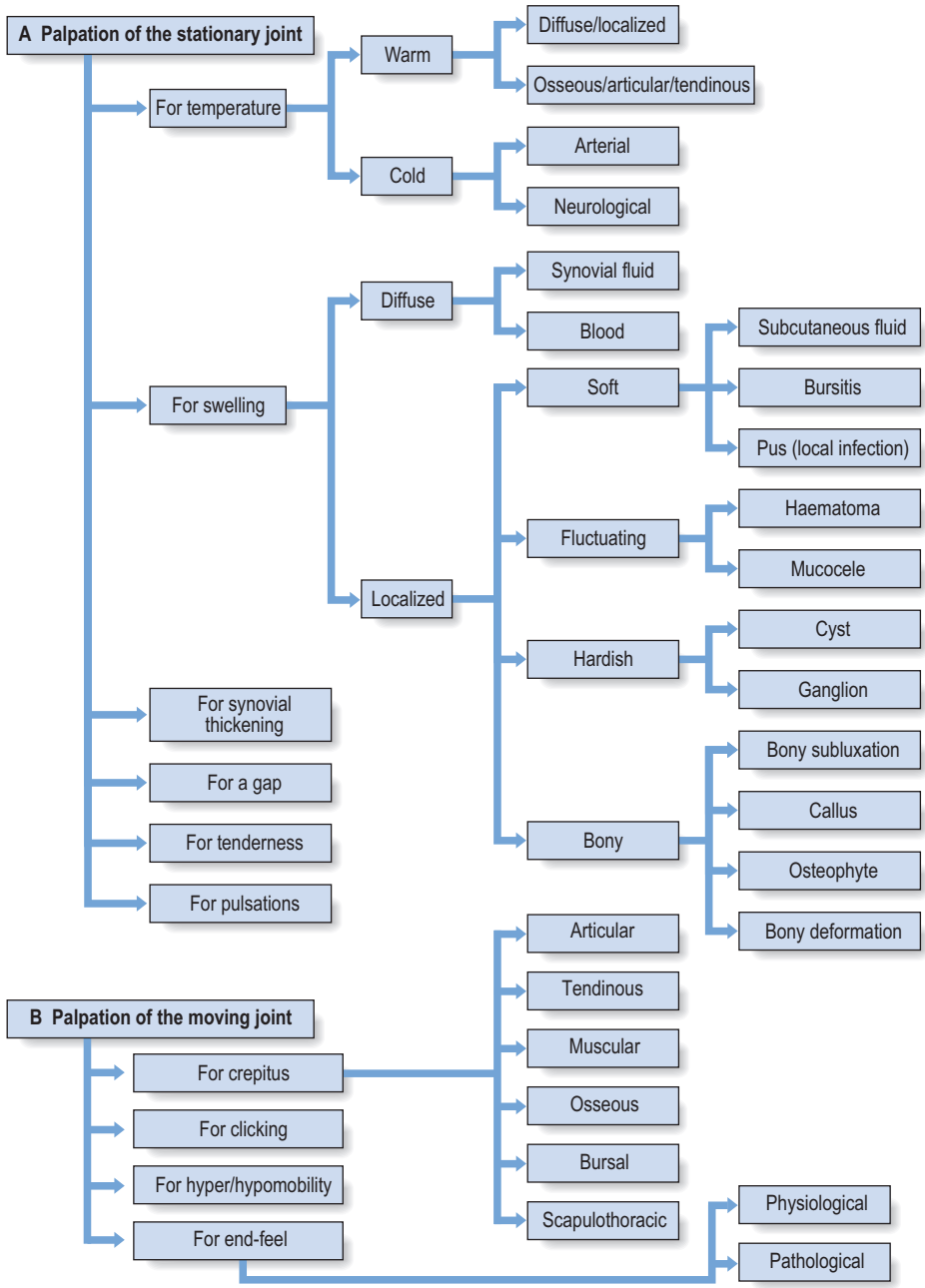


Fig 4.10 • Palpation of (A) the stationary and (B) the moving joint.

superfluous. To search for tenderness is of much more value in a distal joint (wrist, hand, ankle, foot) than in a proximal joint (trunk, shoulder, hip), because the localization of the pain is much more accurate.

Difficulties may arise when two tissues overlap and when no certainty exists whether the lesion lies in the superficial or a deep structure. This situation arises most in the chest and abdomen. To ascertain whether the tenderness lies in a superficial or a deep muscle or in another structure (e.g. a viscus), palpation is performed during contraction and relaxation of the superficial muscle. Greater pain when the superficial muscle is tense suggests that the muscle is at fault, the converse that deeper structures are affected.

Other misleading phenomena are the presence of either a 'referred' tenderness or an 'associated' tenderness.

Localized, deep referred tenderness may occur within a painful region, for example, as the result of compression of the dura mater usually by a small displaced fragment of the intervertebral disc. This phenomenon is described in Chapter 1 and can be very deceptive unless the examiner is familiar with the rules of pain reference.

Associated tenderness is a very well-localized tender area very close to the site of the lesion. There is a relation between the two because both pain and tenderness disappear when the patient is cured. Associated tenderness is most common at the wrist and the elbow. In de Quervain's disease – tenovaginitis

of the abductor longus and extensor brevis muscles at the wrist – the styloid process of the radius is extremely tender, as is the posterior aspect of the lateral epicondyle in tennis elbow, especially when the lesion lies at the tenoperiosteal origin of the extensor carpi radialis brevis.

Negative palpation does not necessarily mean that the diagnosis is wrong, except perhaps in very superficial lesions, because there are tissues that are not explicitly tender when affected (e.g. the supraspinatus tendon).

Pulsation

Palpation for pulsation in the arteries may have an important diagnostic value and is indicated when symptoms are mentioned or signs are found that point to a vascular condition (e.g. claudication, a cold limb, or blue or white skin).

Palpation of the moving joint

Next, the moving joint is palpated. The examiner may either experience crepitus or clicks, or can assess end-feel or mobility (see Fig. 4.10b).

Crepitus

Crepitus always indicates a pathological situation. It can often be heard and can also be felt by the palpating hand placed on the moving part during active or passive movement. There are circumstances in which the crepitus is only felt during a movement against resistance. Crepitus may be articular, tendinous, muscular, osseous or bursal.

Articular crepitus

Crepitus which originates from the joint surfaces gives an idea of their ability to glide over one another. Fine crepitus indicates slight roughening and occurs in mild arthrosis or in long-standing rheumatoid arthritis. The latter gives rise to the characteristic 'silken' crepitus. Coarse grating is felt in advanced arthrosis and results from considerable fragmentation of the cartilaginous surface. In severe arthrosis the cartilage has wholly worn through which leads to intermittent creaking of bone against bone ('cogwheel' phenomenon).

Tendinous crepitus

Crepitus in relation to a tendon establishes tenosynovitis. Fine silky crepitus occurs in acute mechanical cases as the result of a traumatic roughening of the tendon surface and of the inner aspect of the tendon sheath. Coarse crepitus is felt in chronic rheumatoid or tuberculous tenosynovitis.

Muscular crepitus

This is observed in two situations only:

- Tenosynovitis of the two extensors and of the long abductor of the thumb in the distal part of the forearm is usually accompanied by crepitus that can be felt locally. The crepitus is, however, sometimes felt throughout the muscle bellies, possibly as far up as the elbow (see p. 524).
- Localized crepitus is usually felt in a lesion at the musculotendinous junction of the tibialis anterior muscle.

Osseous crepitus

A fracture may crepitate when the limb is moved and the two ends of the fractured bone move against each other but, except in pathological fractures, an attempt to elicit crepitus usually causes so much pain that it should not be done.

Bursal crepitus

Subdeltoid bursitis is the characteristic situation. Some time after a bursitis with effusion has subsided, creaking on moving the arm can be felt (see Shoulder, p. 333).

Scapulothoracic crepitus

This is unique. The posterior thoracic wall can become roughened at an area just beyond the lateral edge of the iliocostal muscle, with localized crepitus felt on shoulder movement (see the online chapter *The shoulder girdle: Disorders of the inert structures*.

Clicks

Clicks can be produced in several ways. Certain tests on clinical examination or certain manœuvres during a manipulative session may provoke movement of an intra-articular fragment of cartilage. This often happens when a loose body is present in a joint (e.g. knee or elbow) or when a meniscus subluxates (e.g. knee or jaw) or a fragment of disc moves (e.g. spinal joints). Clicking during a manipulative manœuvre is of interest in that it is often associated with an improvement of the physical signs, clearly indicating that a block to movement has been removed. In ligamentous laxity (as can be seen in subluxation of the clavicular joint, in capsular overstretching of the shoulder, or in rupture of the medial collateral ligament at the knee), a click is produced when one bone moves in relation to its fellow. Irrelevant clicks also appear, for example painless clicking of a costal cartilage or a patellar click during active extension of the knee.

End-feel

End-feel can be assessed when a passive movement comes to a stop. It is either physiological or pathological and has important diagnostic and therapeutic consequences (see pp. 64–65).

Hypermobility and hypomobility

These are terms that are currently used by osteopaths and manual therapists.[24–25] During their so-called 'segmental examination' they claim to be able to feel the difference in mobility between different, mostly spinal, joints.[26] To establish hyper- or hypomobility would afford an excellent guide to the level of the lesion *if* it were always at the joint in which the fault lay, which is not in fact the case. Moreover, these findings are so subjective that they are not verifiable or reproducible. It has been shown on different occasions that even experienced osteopaths or therapists cannot agree about the findings.[27–31] The main mistake, however, consists in attributing diagnostic significance to such differences in mobility. When movement

in a hypomobile or hypermobile joint strongly evokes the patient's symptoms, this has to be considered as the best criterion and clearly shows where the lesion must lie. However, when the abnormal mobility does not elicit the known symptoms, the problem clearly has nothing to do with the hypermobile joint.[32–34] Furthermore, there are no universal standards to determine abnormal mobility. What is normal for one person can be abnormal for another. Hypermobility can be physiological up to a certain level, especially in women, and hypomobility can also be considered as normal, particularly in the elderly. When hypermobility gives rise to symptoms, then and then only can it be considered as pathological. The terms 'laxity' and 'instability' are then used. The end-feel will add greatly to this conclusion.

Diagnostic infiltration or aspiration

Diagnostic local anaesthesia

When faced with lesions of the locomotor system, a diagnostic infiltration with a weak local anaesthetic is extremely valuable and most effective in confirming the diagnosis. There is no other comparably effective test. By using infiltration in every suitable case, the physician makes the patient the judge of the correctness of the diagnosis.

Between 2 and 10 ml of a weak local anaesthetic (e.g. procaine 0.5%) is infiltrated at the assumed site of the lesion. Five minutes later the movements that were previously positive are retested. This allows the patient to determine whether the infiltration has altered the pain. When the tests have become completely or partly negative, the exact site of the lesion has clearly been reached, confirming the diagnosis. When the tests are still as painful or as limited as before, the wrong place was infiltrated, i.e. the diagnosis is wrong.

This approach is useful in the following circumstances:

- When the examiner is not certain about a diagnosis or when the examiner wants positive confirmation of a tentative diagnosis, the induction of a local anaesthetic may help.
- For lesions in the extremities, infiltration is particularly useful and gives reliable answers. However, it is not useful on the posterior aspect of the trunk: paraspinal infiltrations may give temporary relief but have no diagnostic value.
- In difficult cases this approach may be the final 'court of appeal'.

Aspiration

From the diagnostic point of view it is often important to aspirate a swollen joint or a diffuse extra-articular swelling in order to ascertain what sort of fluid is present.

Technical investigations

It should be stressed again that there still remains a huge discrepancy between discovered anatomical changes and existing pain and disability. During the last decades the medical profession has focused on, and been fascinated by, techniques of imaging that can clearly show existing anatomical aberrations. However, very often, these anatomical changes are not the source of the pain because they appear to exist also in large groups of symptomless individuals. For example, the prevalence of herniated lumbar discs in a normal asymptomatic population ranges from 35%–50%.[35–41] Evidence of cervical discopathy is also found in asymptomatic subjects[42–44] and there is not always good concordance between the different imaging techniques.[45] In the shoulder, echography can discover partial thickness and full thickness ruptures of the rotator cuff even in large asymptomatic groups.[46–49] The finding of asymptomatic anatomical changes in knee joints during arthroscopy is not uncommon.[50,51]

In orthopaedic medicine, technical examinations are not routinely performed but are instead used only when necessary to refine the clinical diagnosis, to exclude certain lesions or to clarify differential diagnosis. They are always undertaken when the history and/or functional examination have revealed warning signs (Box 4.12).

The clinician should not ascribe too much diagnostic importance to technical investigations – a positive answer does not always identify with certainty the condition present. The reverse also holds: a negative picture does not always mean that there is nothing wrong. Knowing when to ignore a positively or negatively misleading picture comes only from proper clinical examination (see Box 4.13). The latter always remains the most important of the tools available and it is far more dangerous to omit a proper clinical examination and rely on technical investigations than to do the reverse.

Later chapters outline when and when not to fall back on technical investigations and complement the clinical findings.

Interpretation

Interpretation is only possible if the clinical examination has been performed correctly: a detailed history, followed by inspection and a careful functional examination. Every part of the examination may be important, and consequently a complete examination should be carried out on each occasion. Cyriax stated 'Omission of part of this examination, because the diagnosis seems obvious or to save time, is the common source of error'.[52] Examining patients may become a routine and repetitive, with the same features being frequently encountered so that it is tempting to omit that part of the examination

Box 4.12

Technical investigations

Only when necessary:

- To refine the clinical diagnosis
- To exclude certain lesions
- To clarify differential diagnosis
- When warning signs are present

Box 4.13

Summary of functional examination

1. Inspection
2. Preliminary examination
3. Functional examination
   a. Active movements
   b. Passive movements:
      Pain: • where • when
      Amplitude: • limited • full • excessive
      End-feel: • physiological • pathological
   c. Resisted movements
      Pain
      Strength: • normal • weak
4. Accessory tests
5. Palpation:
   a. Motionless joint
   b. Moving joint
6. Diagnostic infiltration or aspiration:
   a. Diagnostic local anaesthesia
   b. Aspiration
7. Technical investigations

Box 4.14

Patterns found in clinical examination for suspected soft tissue lesions

Active movements are positive

- In combination with passive movements
- In combination with resisted movements
- In isolation:
  Limitation of active movements only
  Presence of a painful arc
  A 'snap' occurs
  A 'crack' is heard

Passive movements are positive

- Limitation of range:
  The capsular pattern
  Non-capsular patterns
- Full range
- Excessive range
- No movement possible

Resisted movements are positive

- In combination with passive movements
- In isolation
- In combination with other resisted movements

No movements hurt

which is expected to be negative. By doing this, the presence of a double lesion can easily be overlooked or, even worse, the signs of an uncommon (possibly serious?) disorder (one of the classic 'warning signs') may be missed.

The functional examination gives positive answers balanced by negative ones, the latter corroborating the former. A pattern is found, which may be capsular, non-capsular, muscular or partly or fully articular, and this has to be interpreted so that logical conclusions of incontestable validity are drawn. Interpretation requires experience because examination is subjective and such features as weakness, end-feel and resistance can only be interpreted by comparison, which needs time and patience.

In practice one of a number of patterns can emerge (see Box 4.14).

Impairment of active movements

In combination with impairment of passive movements

The response to active movements (Fig. 4.11) may correspond with that found on passive testing. When an active movement is found to be *painful*, passive movement in the same direction will usually be more painful. In this case, the passive movement must be considered as the important one and the response to it is used for interpretation (see pp. 73–77). When the active movement is *limited*, passive movement is usually also restricted, although it should be possible to go a little further in the limited direction.

In combination with impairment of resisted movements

When an active movement *hurts* and the same movement performed against resistance is also painful, the latter has to be considered as being most important (see pp. 77–78). *Limitation* of an active movement and not of the corresponding passive one indicates gross weakness. The same movement performed against resistance usually also proves to be weak. Sometimes, however, other (accessory) tests against resistance must be done to uncover the lesion.

In isolation

One or more active movements are limited but are full range on passive movement

This occurs when one or more muscles are out of action. The resisted movements aimed to test the same muscles will also be weak. Weakness is quite often encountered in orthopaedic medical disorders. It may be of organic type or functional type (Fig. 4.12).

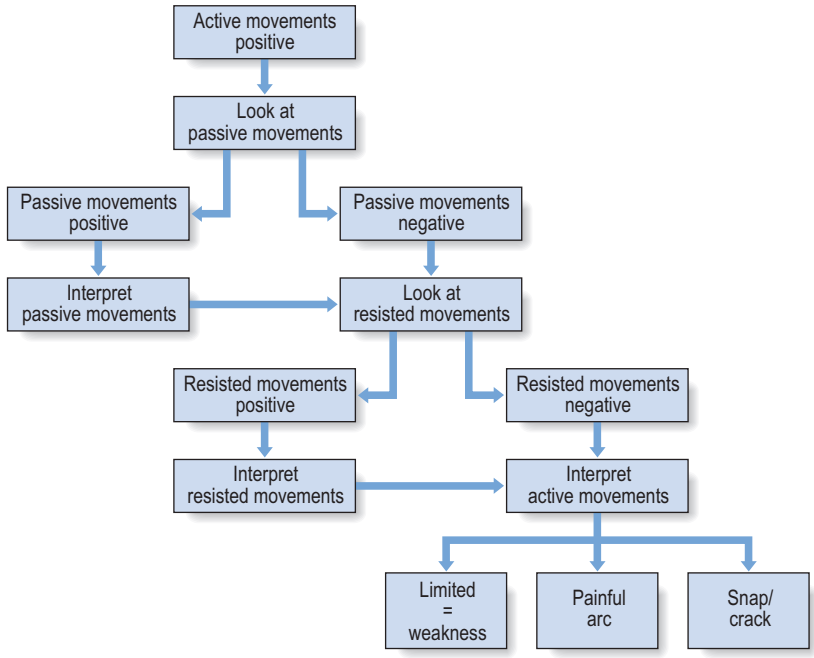


Fig 4.11 • Response to active movements.

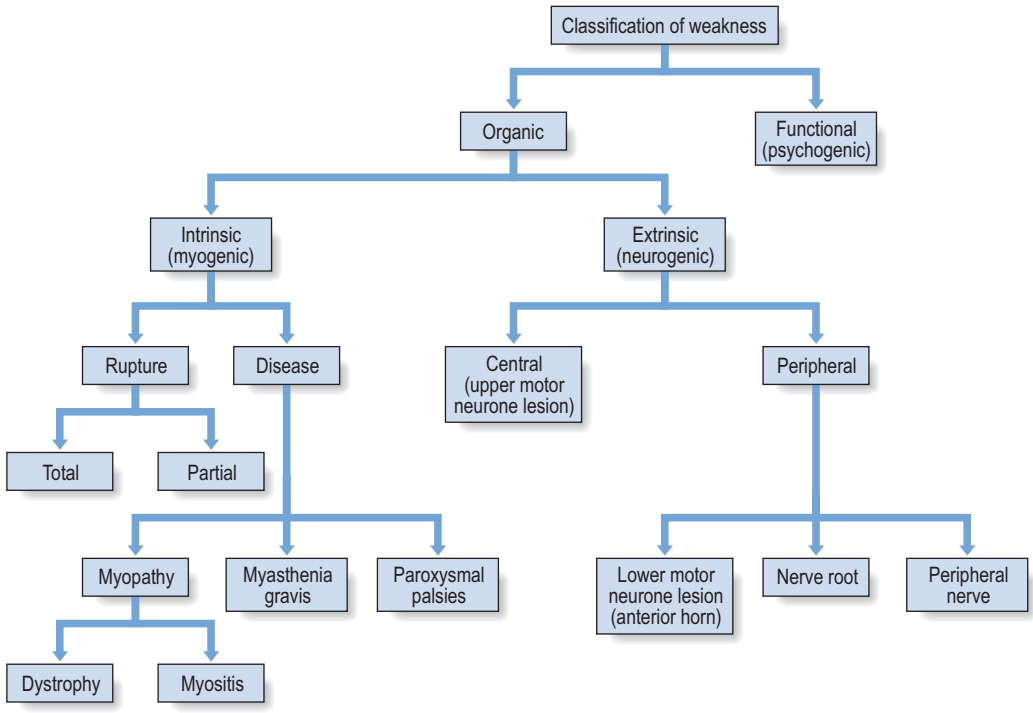


Fig 4.12 • Classification of weakness.

Functional palsies are mostly psychogenic. Organic palsies are the result of either an anatomical pathological condition or of a physiopathological (usually biochemical) lesion. The cause may be intrinsic or extrinsic.

Intrinsic (myogenic) lesions are the result of either a structural disorder, such as a severe partial or a total rupture of muscle or tendon, myopathy (e.g. muscular dystrophy or myositis) or a biochemical disorder (e.g. myasthenia gravis or paroxysmal palsies). Extrinsic (neurogenic) lesions may be either central or peripheral (Table 4.2).

Central palsy

A central palsy is the result of a lesion of the upper motor neurone (i.e. the corticospinal and/or corticonuclear tracts), caused by a vascular condition, a tumour, trauma or disease (e.g. encephalitis or multiple sclerosis) in the region between

Table 4.2 Differences between central and peripheral palsies

Central	Peripheral
Muscular hypertonus	Muscular hypotonus
Hyperreflexia	Hyporeflexia
No muscular atrophy	Muscular atrophy
Upper skin reflexes disappear	
Lower skin reflexes appear	

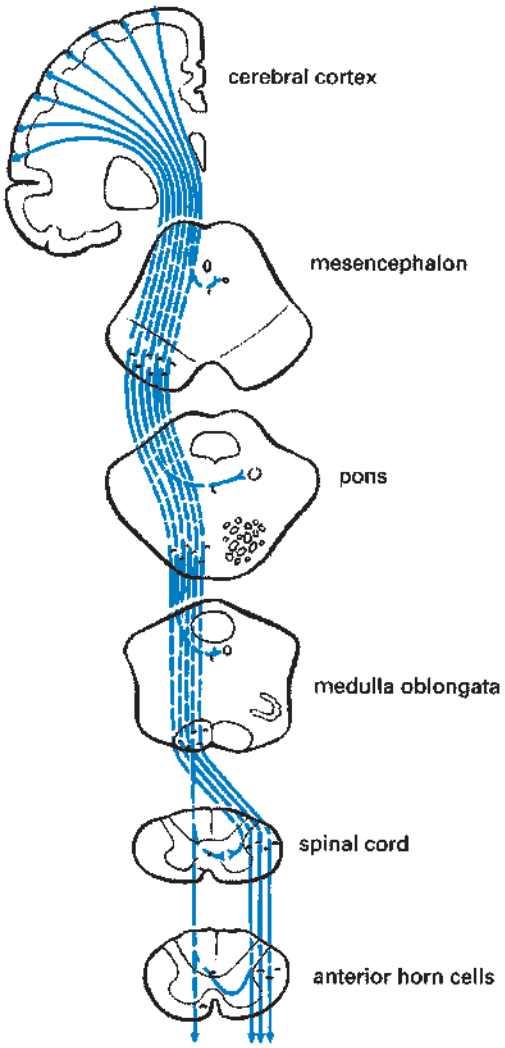


Fig 4.13 • Corticospinal tract (pyramidal tract).

the cerebral cortex and the spinal cord (Fig. 4.13). Hemiplegia, monoplegia, paraplegia or quadriplegia follows, characterized by muscular hypertonus, hyperreflexia, disappearance of the upper skin reflexes and appearance of abnormal skin reflexes (e.g. the Babinski reflex). Because the cells in the anterior horn of the spinal cord are not affected, there is no muscular atrophy apart from some slight wasting because of the patient's inactivity.

Peripheral palsy

A peripheral palsy may have its origin in the cells of the anterior horn of the spinal cord (nuclear palsy or lower motor neurone lesion). Typical anterior horn disorders are acute anterior poliomyelitis, giving rise to asymmetrical paralysis of the proximal muscle groups, and degenerative conditions such as chronic anterior poliomyelitis and amyotrophic lateral sclerosis (ALS), which cause symmetrical bilateral paralysis starting in the distal muscle groups.

Anterior nerve root lesions (radicular palsy) give rise to segmental paralysis or paresis. Nerve root palsy is usually preceded by severe segmental pain.

When a peripheral nerve or nerve trunk is affected, only the muscles innervated by that nerve are weak. When a plexus is affected, the weakness is more extensive. The clinical features are muscular hypotonus, hyporeflexia and atrophy.

A painful arc is present

The moment the patient shows a painful arc the examiner knows that the lesion lies in a position that is subject to compression. Interpretation is on anatomical grounds: the examiner knows what structures can and cannot become momentarily compressed (see later chapters on specific joints).

A snap occurs

When this is present, the patient usually mentions it as a previous occurrence. It may be detected at examination, especially on active movements.

A snap may be felt when a tendon catches against a bony prominence and then slips over it. Examples are the shoulder ('snapping shoulder'), if the long head of biceps slips in and out of the upper end of the bicipital groove as the result of rupture of the transverse humeral ligament; the ankle ('snapping ankle'), when the peroneal tendons are loose in their groove on the posterior surface of the fibula and slip forwards over the malleolus; and the hip, when the greater trochanter catches against the edge of the gluteus maximus muscle. Snapping is not necessarily painful. Therefore, when a patient complains about a joint that also snaps on certain movements, the cause of the pain and the snapping are not always related.

A crack is heard

Joints may crack when they are brought to the end of range, either actively or passively. The cause is the momentary formation of an intra-articular air bubble from the synovial fluid as a result of the partial vacuum created by traction. This often happens in the finger joints and is also very common in the spinal facet joints during manipulation.

Impairment of passive movements

Positive passive movements in combination with negative resisted movements draw attention to the inert structures, although they do not unconditionally exclude a lesion of contractile tissue (see Fig. 4.14).

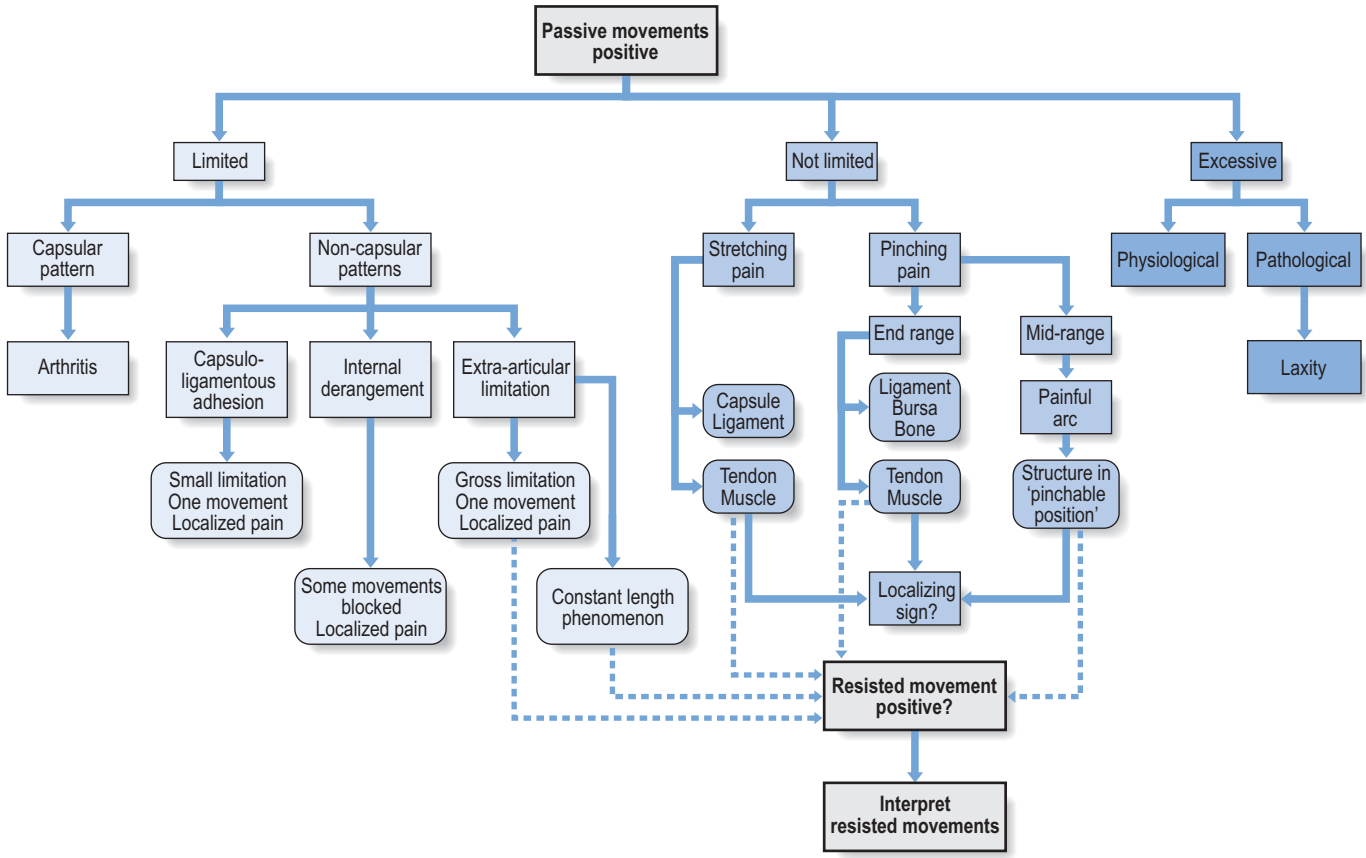


Fig 4.14 • Positive passive/negative resisted movements.

Limited range

Limitation in the literal sense of the word can only be detected on passive testing. Although a patient may present with limitation on active movement, this does not necessarily mean that the joint cannot move to its full range. The examiner should meticulously check whether a movement really is limited. Even the slightest decrease in range has significance. When one or more passive movements are limited the major question arises: 'Is this limitation in the capsular proportions or not?' Or, in other words, 'Is there a capsular pattern present or is the pattern non-capsular?'

The capsular pattern

> The capsular pattern = Arthritis

In a peripheral joint, a lesion of either the fibrous capsule or of the synovial membrane with which it is lined gives rise to limitation of movement, to a characteristic proportion. It does not matter whether the irritation is synovial only (as in recent sprain or haemarthrosis), capsular only (as in arthrosis) or both (as in rheumatoid arthritis) – the same pattern results. This limitation of movement to a proportion typical for the joint capsule is initially caused by a protective mechanism (muscle spasm) and later by fibrous contracture. This pattern of limitation always indicates that a lesion of the whole joint is present. The condition is called capsulitis, synovitis or arthritis, the latter being the best name as it clearly indicates involvement of the entire joint.

This phenomenon is easy to check in clinical daily practice but is difficult to explain. In acute cases the muscles clearly contract in a way that anticipates the situation that might be created when later the capsule 'contracts'. So there must be an in-built mechanism in the brain which acts in imitation or in correspondence with the normal evolution of the movement.

The capsular pattern varies from joint to joint. It is not clear why some joints limit in certain directions and other joints in other directions. In arthritis of the shoulder it consists of a certain degree of limitation of scapulohumeral abduction, more limitation of external rotation and less limitation of internal rotation. A capsular lesion in the hip by contrast manifests itself with most restriction of internal rotation and less limitation of flexion, abduction and extension, external rotation remaining full range. There are joints in which the capsular pattern includes complete absence of certain movements: the joint is fixed in a certain position. Examples are the subtalar joint, which fixes in full valgus, and midtarsal arthritis in which the joints are fixed in full abduction and external rotation.

The full articular pattern

At the spinal joints, a similar situation is found. When the whole intervertebral joint complex is affected a full articular lesion exists which gives rise to a pattern comparable to the capsular pattern in peripheral joints. However, the lesion is not always capsular and therefore is better described as a 'full articular' pattern, which covers all possible conditions.

Joints not spanned by muscles

Where muscles are not present to cause any limitation of movement, joints are supported by ligaments only. Restriction of movement caused by irritation of the capsule (synovitis) is not possible. There is pain only at the extremes. This phenomenon is found at the acromioclavicular, sternoclavicular, sacroiliac, sacrococcygeal and lower tibiofibular joints, and also at the symphysis pubis.

Limitation with a bony block end-feel

Slight painless limitation of movement with a bony block end-feel occurs in neuropathic arthropathy. The movement causes an ache in gross osteophytosis, myositis ossificans or a malunited fracture.

Non-capsular patterns or partial articular limitation

When the limitation of movements in a certain joint does not correspond to the capsular pattern of that joint, the lesion is clearly not capsular, which means that it does not affect the entire joint. So arthritis is not present.

In contrast to the capsular pattern a non-capsular pattern is less well defined, but for each joint different patterns may occur, all of which do differ from the capsular pattern (see Fig. 4.15).

Partial articular pattern

In the spine the term *partial articular pattern* is more appropriate, indicating that only part of the intervertebral joint complex is affected.

Non-capsular conditions fall into three main categories.

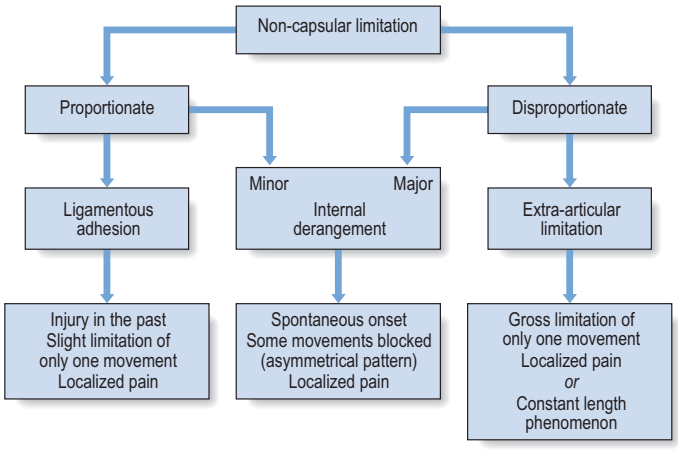


Fig 4.15 • Classification of non-capsular limitation.

Ligamentous adhesions

When a post-traumatic ligamentous lesion heals in the absence of movement macro-adhesions may form (ligament to capsule or underlying bone), which impair function.

One movement is usually limited by localized pain but others may be painful at their extremes. The limitation is *proportionate*: slight restriction in one direction – the one that stretches the affected part of the ligament – and full range in other directions.

Typical examples are the medial collateral ligament of the knee and the lateral ligaments of the ankle. At the knee, flexion is usually 10–15° limited, with pain very localized over the course of the ligament. At the ankle, the combined movement of plantiflexion and inversion is slightly restricted and the tender spot lies in the affected ligament (anterior talofibular, calcaneofibular or calcaneocuboid).

Internal derangement

When fragments of cartilage or bone become loose in a joint, the clinical picture is of internal derangement. The knee is the most common site followed by the spinal joints, the wrist and the jaw and less so the hip, the elbow or the tarsal joints.

Displacement comes on quite suddenly and blocks part of joint movement with localized pain on one aspect and limitation of some movements but not others.

Two different types of limitation occur:

Proportionate derangement. When the displaced fragment is small and lies in such a position that it does not greatly hinder the function of the joint, restriction of movement is also small by comparison with normal movement. The picture may very well mimic the proportionate limitation in ligamentous adhesions but the absence of an injury and the short history exclude this possibility. A typical example is the loose body in an arthrotic knee: flexion is 15–30° limited and the pain is felt at the inner side.

Disproportionate derangement. A large displacement causes gross limitation of movement in some directions and full or almost full range in other directions – an asymmetrical pattern. This is a well-known clinical picture in acute lumbago. Two or three trunk movements are totally blocked and one movement, usually one lateral flexion, is scarcely limited.

Extra-articular limitation

A tissue outside the joint may be damaged and prevent movement in one direction only. A *disproportionate* limitation is then seen: one movement is grossly limited and all other movements are of full range. Such disproportion is only possible when the tissue that causes the limitation lies extra-articularly. In some cases it does not allow stretching, in others it is resistant to compression. Rupture of some muscular fibres (in the quadriceps or in the calf) is followed by localized muscle spasm which prevents stretching. In acute subdeltoid bursitis, gross disproportion is also found between the very limited scapulohumeral abduction and the slight limitation of both rotations.

The 'constant-length' phenomenon. In this phenomenon the degree of limitation of a movement in one joint depends on the position in which the adjacent joint is held. This is only

possible when the structure that prevents stretching spans the two joints. Straight-leg raising is a typical example, with the hamstring muscles spanning the hip and knee joints. The amount of flexion at the hip depends on the position of the knee: when done with the knee extended the movement stops when the hamstring muscles become tight, but flexion of the knee allows full flexion at the hip. Another example is Volkmann's contracture: the fingers cannot be extended without flexing the wrist.

Full range

When no limitation of movement exists on passive testing, the picture may be difficult to interpret. The site of pain usually helps to determine whether it is caused by stretching or by pinching of the affected tissue (Fig. 4.16).

Pain is elicited on stretching

Pain at the extremes of several movements may be found in those joints that are not spanned by muscles. As the capsule of these joints does not have muscular protection the passive tests stretch the ligaments. When, for example, the acromioclavicular joint is affected, the extremes of passive movements at the shoulder are painful.

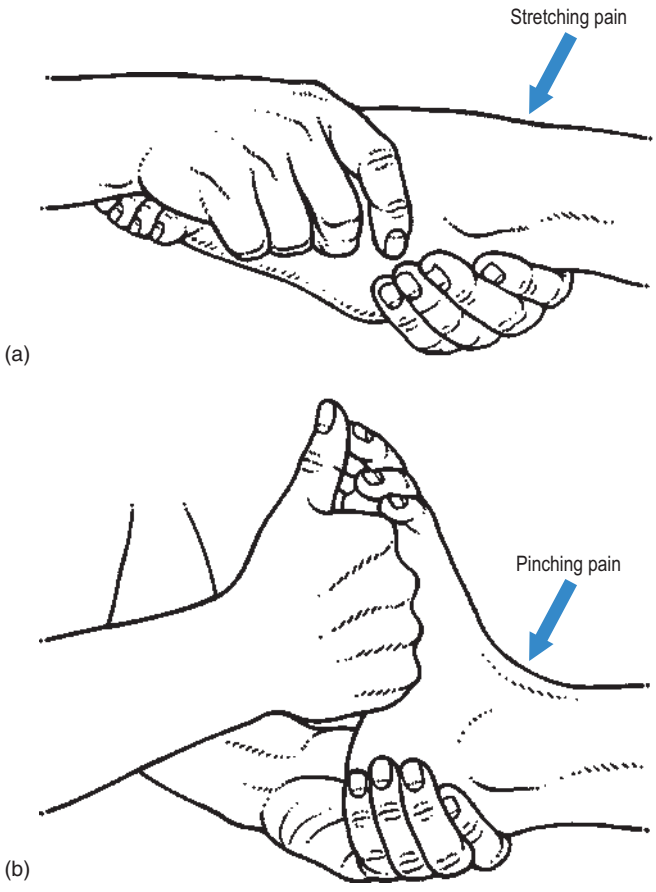


Fig 4.16 • (a) Pain on stretching, (b) pain on pinching.

When only one passive movement causes pain as a result of stretching, the examiner should think in terms of a ligamentous, tendinous or muscular tissue being stretched. In the last of these, resisted movement in the opposite direction is also painful – the 'contractile tissue pattern' (see below). However, an early arthritis in shoulder and hip may, for a short period, show pain at the end of one movement only.

Pain is elicited on pinching

Pinching of a tender tissue may happen at the end of range or at mid-range. In both cases, the pain is the result of the same mechanism. When a passive movement elicits pain at *full range* but the main test result is a resisted movement, this sign sometimes has localizing value. This is seen for example at the shoulder in supraspinatus and subscapularis tendinitis, at the elbow in biceps tendinitis, at the hip in rectus femoris tendinitis and at the heel in Achilles tendinitis. For more details, see the appropriate chapters.

In other cases, logical thinking based on anatomical grounds is required to find out what structure has been painfully pinched. It may be an inert structure (bone, capsule, ligament) or a contractile structure (muscle, tendon). The latter will, of course, show with pain on resisted movements as well. Palpation may then often help to determine the exact localization and local anaesthesia can be used to confirm this.

Pain felt somewhere near *mid-range* in a movement, either passive or active, is called a 'painful arc'. The pain is absent when the movement starts, appears at a certain moment during the movement and disappears again before the end of range is reached. To be called 'painful arc' the pain should disappear at either side of the arc. An arc indicates that the affected structure becomes momentarily compressed between two bony surfaces. Only some structures in the body lie in a position that they can cause a painful arc. It is quite common at the shoulder and in the lumbar spine.

Sometimes the painful arc can be considered a *localizing sign*: its presence shows exactly where the lesion lies. In supraspinatus tendinitis the presence of a painful arc indicates that the lesion lies at the superficial aspect of the tenoperiosteal insertion of the tendon into the greater tuberosity of the humerus, and a painful arc accompanying the signs of a subscapularis tendinitis shows the lesion to lie at the upper part of the insertion at the lesser tuberosity.

Excessive range

Hypermobility is seen in capsuloligamentous laxity and is then a pathological finding. However in some circumstances excessive range can be purely physiological (e.g. passive elbow extension in most women). The matter is considered earlier in this section under *palpation*.

No movement is possible

This is not often encountered but it may result either from a severe muscle spasm, protecting a very acute or irritated lesion or from fibrous or bony ankylosis.

Impairment of resisted movements

In combination with impairment of the passive movements

If a movement against resistance is painful, passive movement in the opposite direction is usually also painful. This is the 'contractile tissue pattern'. When the lesion is tendinous there is a full range of movement. In muscular rupture, the movement that stretches the muscle may be not only painful but also limited because of a localized protective muscular spasm of the fibres around the breach. This phenomenon helps to differentiate between a tendinous and a muscular lesion.

In isolation

One resisted movement is positive

This indicates one muscle or one muscle group is involved. In the latter case, one or more accessory tests are necessary to find the muscle or tendon at fault. For example, resisted extension at the wrist tests the extensors of fingers and wrist; repetition of the test with the fingers held actively flexed distinguishes between finger extensors and wrist extensors. Radial and ulnar deviation against resistance differentiates between the radial extensors (extensor carpi radialis longus and brevis) and the extensor carpi ulnaris.

In combination with impairment of other resisted movements

Two resisted movements are positive

This can occur when two contractile structures are affected or, more probably, when one muscle is affected which combines the two functions. The brachial biceps muscle produces both flexion of the elbow and supination of the forearm. A lesion in this muscle leads to both movements being positive. At the shoulder, the combination of positive resisted external rotation and resisted adduction of the shoulder draws attention to the teres minor, which combines these functions.

Three or more resisted movements are positive

When this occurs, the chance that there is a lesion in a contractile tissue is much less. It is more logical to think of either transmitted stress to a very painful space-occupying lesion, such as a tumour, metastases, abscess or very irritated bursa, or a psychogenic problem.

All resisted movements are positive

This excludes a musculotendinous lesion and has the same interpretive significance as multiple resistance. The examiner should also make sure that the patient does not misinterpret the questions: a hypersensitive patient may equate effort with pain and say that every movement against resistance is painful. Careful comparison of both sides is helpful in avoiding misinterpretation.

Table 4.3 Relationship of pain and strength in resisted movement

Pain	Strength	Disorder
–	Normal	–
+	Normal	Contractile tissue
–	Weak	Nervous lesion Complete rupture
+	Weak	Serious lesion Partial rupture

Pain develops on repetition

Pain developing on repetition of a movement may be indicative of an arterial problem leading to intermittent claudication.

Relationship of pain and strength

Resisted movements test for strength and pain. The patterns that emerge are usually a combination of strength or weakness and the presence or absence of pain. In practice, four combinations may occur (Table 4.3).

Painless and strong

Negative resisted movements – no pain and normal strength – exclude a lesion of a contractile tissue. This axiom is often neglected. For example, patients with pain and local tenderness in the trapezius area are often diagnosed as having a 'trapezius syndrome'. The clinical examination, however, shows the trapezius muscle to function normally. In this case there is no reason to incriminate the muscle. Pain and tenderness are clearly referred, usually from the cervical spine. Another example is when a patient has pain over the long head of the biceps but the tests for that muscle are completely negative, then it is apparent that the muscle is normal and the pain is referred.

Painful and strong

When the resisted movement causes pain but the patient is capable of exerting normal strength, the condition must be a minor lesion in a contractile structure. The possibilities are a muscular strain, a lesion at the musculotendinous junction, a tendinous problem (tendinitis, tenovaginitis or tenosynovitis) or a lesion at the tenoperiosteal junction. Typical examples are supraspinatus tendinitis at the shoulder, lesion of the extensor carpi radialis brevis at the elbow (tennis elbow), tenovaginitis of the extensor pollicis brevis and abductor pollicis longus at the thumb (de Quervain's disease), infrapatellar tendinitis at the knee or peroneal tenosynovitis at the ankle.

Painless (or constant pain) and weak

The patient is either pain-free or in constant pain. The test against resistance, although weak, does not elicit or alter the pain. This is usually the result of a nervous condition, either extrinsic or intrinsic. Dependent on the severity of the lesion the weakness detected may vary from very moderate to complete paralysis.

Complete rupture of a muscle or a tendon also leads to a painless total loss of strength (see earlier).

Painful and weak

This combination suggests a major lesion. The movement is painful and its strength is reduced, either because the muscle is functioning poorly or because the attempt to use it increases the pain sufficiently to inhibit full contraction. This pattern occurs in serious disorders, such as fracture or metastases. Usually there are also articular signs. When after an 'ankle sprain' resisted eversion proves painful and weak, a fracture of the tuberosity of the fifth metatarsal bone is suggested. Metastases in the upper femur or in the cervical spine give rise to weakness in resisted movements of the hip or the neck.

If only one movement is painful and weak and the joint moves normally, a partial rupture of a muscle or tendon is probably present, although a severe tendinitis may cause an identical picture.

Positive resisted movements in lesions of non-contractile structures

There are three situations in which muscular contraction causes pain in the absence of any musculotendinous lesion.

The first is a bony lesion (e.g. fracture or metastases) close to the insertion of a muscle or tendon: when the muscle is tested against resistance, the contraction causes pull at the site and the test is painful and possibly associated with weakness. Stress fracture of the pubic bone causes pain felt on resisted adduction of the hip and in an anterior upper rib fracture contraction of the pectoralis major muscle hurts.

The second is compression of a tender structure by a contracted muscle belly which is often encountered in the buttock where an irritated gluteal or trochanteric bursa can be compressed by contraction of the gluteus medius muscle.

The third is in patients with psychological problems, when resisted movements are quite often more painful than passive or active ones (see online chapter *Psychogenic pain*).

Absence of pain on functional testing

For a given region a negative functional examination, including accessory functional tests, implies that the pain is referred. Other parts of the locomotor system from which the pain might originate are then examined. When nothing is found, the lesion is clearly non-orthopaedic, most probably nervous but possibly visceral. Also small bony lesions (e.g. osteoid osteoma) along the shaft of a long bone may cause localized pain without influencing the function of an adjacent joint.

Warning

Every patient with a clear pattern of symptoms in the absence of positive signs on clinical examination should be referred for more specialized investigations.

Summary

The interpretation of resisted passive movements is summarized in Figure 4.17.

Diagnostic difficulties

Very slight pain

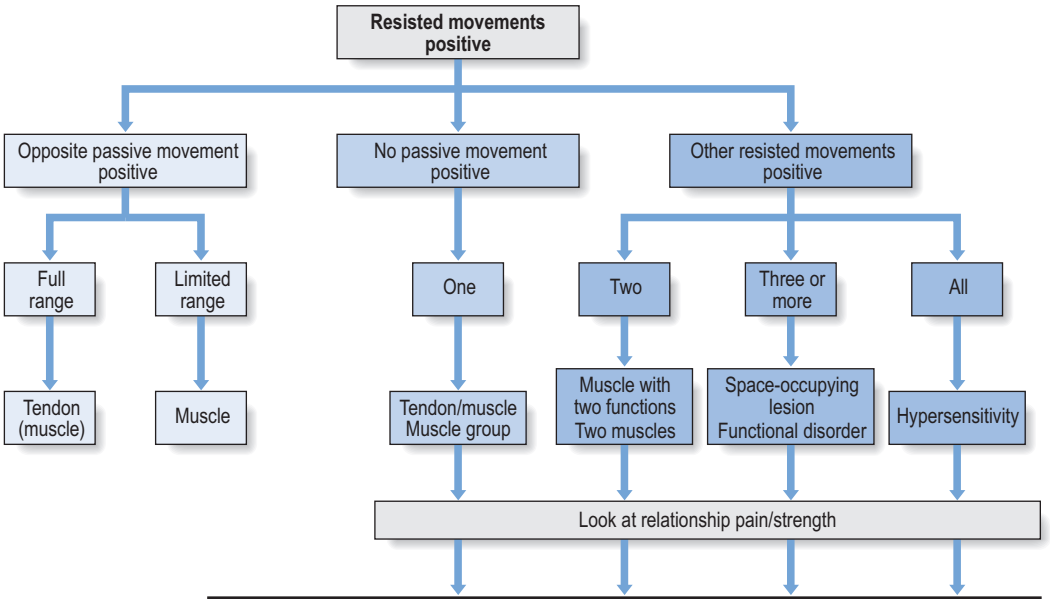
When the patient has very little pain, examination can be totally negative. Palpation is very dangerous in these cases for the reasons mentioned earlier. It is better to re-examine 1–2 weeks later. If spontaneous recovery has not taken place, it is likely that some signs will have developed. Another way to proceed is to seek to provoke signs by having the patient exercise sufficiently to produce symptoms. Clearly a diagnosis is impossible as long as signs are absent.

Very severe pain

Certain conditions are so painful that it is hard for the patient to state accurately where and when pain is felt. Functional testing and its interpretation are also difficult, either because excessive physical signs are presented or because the patient cannot move and is opposed to carrying out potentially diagnostic manœuvres. Severe pain may interfere with the ability to cooperate so that, for example, a resisted movement cannot be done properly. After an injury, swelling and/or haematoma may also render palpation impossible: for example, in the first days after an ankle sprain the condition may be so acute that no clear pattern emerges on examination; lumbago may be so painful that any movement becomes impossible. History is then of great importance, particularly the presence or absence of warning signs. The number of conditions that can provoke such severe pain is limited and the examiner must try to interpret the symptoms in the light of the realistic possibilities.

Non-orthopaedic disorders

Some neurological, vascular or visceral disorders may cause diffuse pain which resembles the symptoms of a lesion of the locomotor system. A patient who complains of vague pains, especially in the trunk, but has negative findings on locomotor and soft tissue examination should be referred for further neurological, vascular or internal examination.



Pain	One contractile tissue	Double lesion Space-occupying lesion	Space-occupying lesion	Space-occupying lesion
Pain and weakness	Partial rupture Fracture Space-occupying lesion			Fracture Serious disorder Space-occupying lesion Functional disorder
Weakness	Complete rupture		Neurological lesion: peripheral nerve or nerve root Gross neurological lesion Functional disorder	

Fig 4.17 • Interpretation of resisted and passive movements.

Difficult clinical pictures

Conditions affecting the spinal column may give rise to vague, diffuse pain felt somewhere in the trunk. If the anterior longitudinal ligament becomes affected in ankylosing spondylitis, pain may be referred to the sternum or to the epigastrium. For those who are not familiar with the phenomenon of dural pain, the extensive and vague radiation of multisegmental pain can be very confusing.

A patient may present the symptoms and signs of a genuine, although minor, lesion which is overshadowed by psychogenic features. The picture is correct in quality, but grossly excessive in quantity. The examiner recognizes the truth within the patient's story but finds that it is interwoven with unlikelihoods and improbable signs are mixed up with the genuine findings (see online chapter *Psychogenic pain*).

The occurrence of a double lesion is not so uncommon and it quite often does not cause a problem, especially when the two conditions can be clearly distinguished because they cause pain in different places or show different sets of signs. Interpretation is more difficult when they lie close together or when the symptoms and signs of one clearly dominates. The examiner can make a diagnosis based on those symptoms and signs that are understood and wait until one problem is solved before attempting to interpret the remaining features. It is indeed a sound principle never to treat two conditions at the same time. The choice of which to treat first can be based on several criteria: pain, frequency, importance and the 'approachability' of the lesion. It is logical to try to improve the more painful lesion first. If doubt exists as to which structure is at fault, the condition that is statistically more common is treated. A problem in a joint – usually arthritis – is dealt with first. Finally, when it is known that one condition can be more quickly treated than another, this condition is treated first.

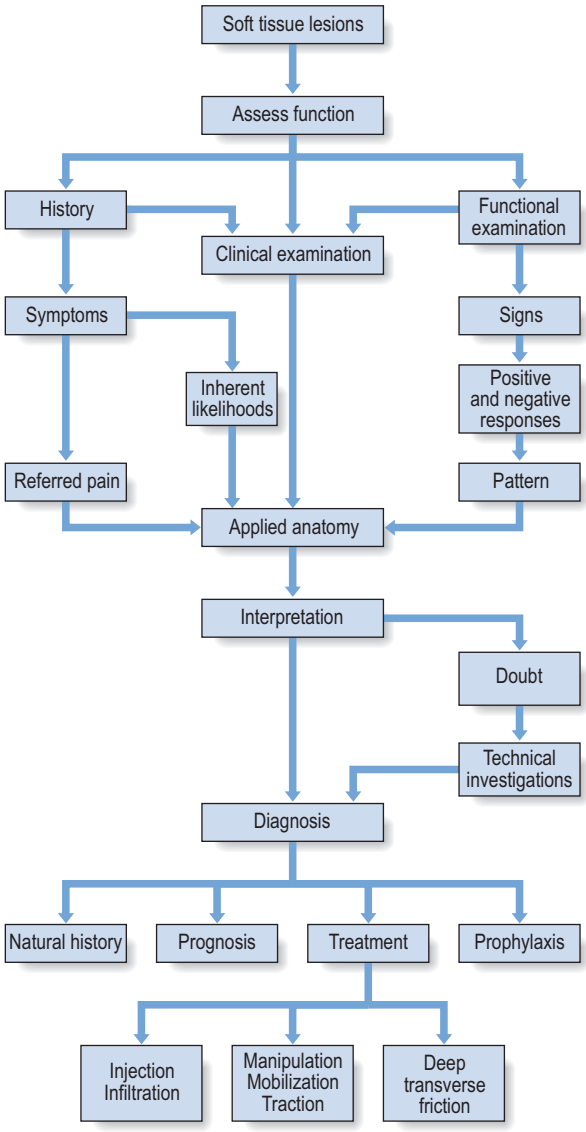


Fig 4.18 • Clinical approach in orthopaedic medicine.

The clinical approach in orthopaedic medicine is summarized in Fig. 4.18.

Psychogenic problems

The reader is referred to the online chapter *Psychogenic pain*.

Access the complete reference list online at **www.orthopaedicmedicineonline.com**

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5

Principles of treatment

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Introduction

Cyriax had a straightforward opinion about treating orthopaedic problems:

- All pain arises from a source
- All treatment must reach the source
- All treatment must exert a beneficial effect on it.

It is obvious that the method of treatment will depend largely on the existing type of disorder.

In orthopaedic medicine, disorders may be grossly categorized as follows:

- *Traumatic* – an injury resulting either from one single trauma or from multiple small traumas, the so-called overuse injuries
- *Inflammatory* – rheumatoid: poly- or monoarticular, infectious, traumatic
- *Degenerative*
- *Internal derangement* – loose bodies and displaced menisci in peripheral joints and intervertebral disc displacements in the spine
- *Functional disorders* – instability, weakness, proprioceptive disturbances
- *Psychogenic pain* – there is no existing functional or anatomical explanation for the pain.

However, most 'disorders' have a combined aetiology: traumatic inflammation or repetitive internal derangement may lead to functional instability or to weakening of the proprioceptive reflexes; long-standing functional disorders may lead to psychogenic decompensation.

Before any form of treatment is undertaken, precise diagnosis is mandatory; it is the type, extent and position of the disorder present which determines treatment. Therefore training in orthopaedic medicine must put great emphasis on how to reach a proper diagnosis. It is more difficult and requires considerable delicacy of approach to teach and learn how to diagnose and so to propose therapy chosen on logical grounds, than it is to instruct and learn treatment techniques. Other aspects must also be taken into account: how much pain can the patient bear? To what extent does the lesion interfere with normal activities? How eager is the patient to receive a quick

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cure? And what is the patient's attitude towards certain therapeutic methods such as corticosteroids and manipulation?

Orthopaedic medicine based on a detailed functional examination requires more knowledge, skill, time and effort from the physician than just to order technical investigations, but leads to greater professional interest, more appropriate diagnosis and a higher degree of patient satisfaction. Clear diagnosis and consequent selection of treatment on logical grounds also leads to better understanding between doctors and therapists. Because the two groups work with the same types of patient, they must share a common approach. Therapists should no longer be regarded purely as technicians who listen to the physician and carry out orders. On the contrary, they should be aware that they have diagnostic and therapeutic responsibilities. Their opinion must be taken seriously and is important to avoid unnecessary delay in achieving a satisfactory outcome.

Techniques

The treatment techniques used in orthopaedic medicine thus depend entirely on the type of disorder. The different types of treatment we describe are:

- *Manipulation techniques* (rapid, small-amplitude, thrusting passive movement – also called 'grade C mobilization') are used to reduce small cartilaginous displaced fragments both in the spine and in peripheral joints (loose bodies). Manipulation is also called for to restore normal mobility in a joint restricted by ligamentous adhesion and in subluxation of bones.
- Gentle *passive mobilizations* (grade A and B mobilizations) are used to stretch capsular adhesions and to improve the function of ligaments and tendons. In the treatment of traumatic injuries they are often used in combination with deep transverse massage.
- *Active movements* and proprioceptive training are needed in the treatment of functional disorders and instability. In the treatment of minor muscular tears they are very useful in avoiding the formation of abnormal intralesional adhesion formation.
- *Injection and infiltration techniques* are used to reduce traumatic or rheumatoid inflammation. They are most valuable in arthritis, bursitis, ligamentous and tendinous lesions and in neurocompression syndromes.
- *Deep friction* is a very useful technique in treating traumatic and overuse soft tissue lesions. The rationale for using deep friction (which is in fact a form of soft tissue mobilization) is supported by experimental studies of the past several decades that confirm and explain the beneficial effects of activity on the healing musculoskeletal tissues (see Connective tissue).

Repair and remodelling of healing tissues respond to cyclic loading and motion.[1] Early motion and loading of injured tissues is not without risk, however, and excessive loading can inhibit or stop healing. Deep transverse friction imposes cyclic loading without bringing too much tension on the healing longitudinal structures of tendon or ligament and can therefore be considered as beneficial.

Deep transverse friction

Deep transverse friction (although the word friction is technically incorrect and would be better replaced by 'massage') is a specific type of connective tissue massage[2] developed in an empirical way by Cyriax.[3]

Transverse massage is applied by the finger(s) directly to the lesion and transverse to the direction of the fibres. It can be used after an injury and for mechanical overuse in muscular, tendinous and ligamentous structures.[4–6] In many instances the friction massage is an alternative to infiltrations with steroids. Friction is usually slower in effect than injections but leads to a physically more fundamental resolution, resulting in more permanent cure and less recurrence. Whereas steroid injection is usually successful in 1–2 weeks, deep friction may require up to 6 weeks to have its full effect.

The technique is often used before and in conjunction with mobilization techniques. In minor muscular tears, friction is usually followed by active movement, in ligamentous tears by passive movement and in tendinous lesions by active unloaded movements until full resolution has been achieved.

It is vital that transverse massage is performed *only at the site of the lesion*. The effect is so local that, unless the finger is applied to the exact site and friction given in the right direction, relief cannot be expected.

Over the years, and unfortunately enough, the technique has developed a reputation for being very painful for the patient. However, pain during friction massage is usually the result of a wrong indication, a wrong technique or an unaccustomed amount of pressure. Friction massage applied correctly will quickly result in an analgesic effect over the treated area and is seldom a painful experience for the patient.

Mode of action

Transverse massage should be used empirically for what it is and what it achieves; there is no scientific proof for any postulates about the underlying mechanism of action.

Only a few studies exist,[7,8,9] and more research is urgently needed. However, experienced therapists know in what kind of soft tissues they can expect good results with transverse massage and where the technique does not work. Transverse massage either is effective quickly (after 6–10 sessions) or not at all. Advice on indications, contraindications and modalities of the technique that are given in this book rely solely on the experiences of the authors and not on scientific research.

However, although the exact mode of action is not known, some theoretical explanations have been put forward. It has been hypothesized that friction has a local pain-diminishing effect and results in better alignment of connective tissue fibrils.

Relief of pain

It is a common clinical observation that application of local transverse friction leads to immediate pain relief – the patient

experiences a numbing effect during the friction and reassessment immediately after the session shows reduction in pain and increase in strength and mobility. The time to produce analgesia during the application of transverse friction is a few minutes and the post-massage analgesic effect may last more than 24 hours.[10] The temporary relief at the end of a session may prepare the patient for treatment with mobilization not otherwise possible, such as selective rupture of unwanted adhesions.

A number of hypotheses to explain the pain-relieving effect of transverse massage have been put forward:

- Pain relief during and after friction massage may be the result of modulation of the nociceptive impulses at spinal cord level: the *gate control theory* (see Ch. 1). The centripetal projection into the dorsal horn of the spinal cord from the nociceptive receptor system is inhibited by the concurrent activity of the mechanoreceptors located in the same tissues. Selective stimulation of the mechanoreceptors by rhythmical movements over the affected area thus 'closes the gate for pain afference'.
- According to Cyriax, friction also leads to increased destruction of pain-provoking metabolites, such as Lewis's substances. This metabolite, if present in too high a concentration, provokes ischaemia and pain.[3]
- It has also been suggested that prolonged deep friction of a localized area may give rise to a lasting peripheral disturbance of nerve tissue, with local anaesthetic effect.
- Another mechanism through which reduction in pain may be achieved is through diffuse noxious inhibitory controls, a pain-suppression mechanism that releases endogenous opiates. The latter are inhibitory neurotransmitters that diminish the intensity of the pain transmitted to higher centres.[11–13]

Effect on connective tissue repair

Connective tissue regenerates largely as a consequence of the action of inflammatory cells, vascular and lymphatic endothelial cells and fibroblasts. Regeneration comprises three main phases: inflammation, proliferation (granulation) and remodelling. These events do not occur separately but form a continuous sequence of changes (cell, matrix and vascular changes) that begins with the release of inflammatory mediators and ends with the remodelling of the repaired tissue (see Ch. 3). Friction massage may have a beneficial effect on all three phases of repair.

Friction stimulates phagocytosis

It has been suggested that gentle transverse friction, applied in the early inflammatory phase enhances the mobilization of tissue fluid and therefore increases the rate of phagocytosis.[14]

Friction stimulates fibre orientation in regenerating connective tissue

During maturation, the scar tissue is reshaped and strengthened by removing, reorganizing and replacing cells and matrix.[15]

It is now generally recognized that internal and external mechanical stress applied to the repair tissue is the main stimulus for remodelling immature and weak scar tissue – with fibres that are oriented in all directions and through several planes – into linearly rearranged bundles of connective tissues.[16] Therefore, during the healing period, the affected structures should be kept mobile by normal use. However, because of pain, the tissues cannot be moved to their full extent. This problem can be solved by friction. Transverse friction massage imposes rhythmical stress transversely to the remodelling collagenous structures of the connective tissue and thus reorients the collagen in a longitudinal fashion. Friction is thus a useful treatment to apply early in the repair cycle (granulation and beginning of remodelling stage): the cyclic loading on and motion of the healing connective tissues stimulates formation and remodelling of the collagen.[17]

Friction prevents adhesion formation and ruptures unwanted adhesions (Fig. 5.1)

In that transverse friction aims to achieve transverse movement of the collagen structure of the connective tissue, crosslinks and adhesion formation are prevented. In the early stages of proliferation when crosslinks are absent or still weak, friction must be very light so as to cause only minimal discomfort. Therefore, in the first day or two following an injury, friction is given with slight pressure only and over a short duration, e.g. 1 minute.

At a later stage when strong crosslinks or adhesions have formed, more intense friction is needed to break these

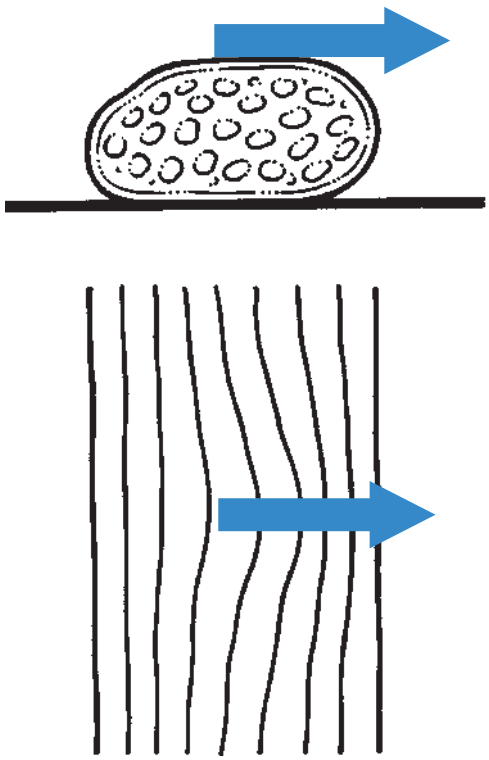


Fig 5.1 • Friction achieves a transverse movement between longitudinally arranged collagenous fibres.

down.[18–21] The technique is then used to soften the scar tissue and to mobilize the crosslinks between the collagen fibres and the adhesions between healing connective tissue and surrounding tissues. This, together with the local anaesthesia produced, prepares the structures for mobilizations that apply longitudinal stress to the structures and rupture the larger adhesions.

Friction induces traumatic hyperaemia

Forceful deep friction produces vasodilatation and increased blood flow to the area. It may be hypothesized that this facilitates the removal of chemical irritants and increases the transportation of endogenous opiates, so causing a decrease in pain. Such a forceful friction, resulting in hyperaemia is only desirable in chronic, self-perpetuating lesions.

Indications

Diagnosis

The reduction in pain achieved after a few minutes of localized transverse friction may be very helpful to define the exact location of the lesion. In muscular, tendinous or ligamentous lesions, a few minutes of massage on the suspected area results in diminished pain on testing immediately thereafter, so confirming the diagnosis as accurately as an infiltration with local anaesthesia.

Preparation for mobilizations and manipulation

Transverse massage is often applied before and in conjunction with other mobilizing techniques. In muscular lesions, friction is given before active or electrical contractions on an unloaded muscle. The purpose is to allow broadening of the muscle and so the prevention of adhesion formation between adjacent muscle fibres and/or bundles.

For reasons of pain relief, transverse massage is usually required before manipulative breakage of ligamentous adhesions is performed. This may be indicated in chronic ligamentous lesions at knee and ankle.

Deep and thorough friction also precedes manipulation of the elbow in type II tennis elbow. The technique is used for its desensitizing and softening effect which makes the manipulation more tolerable.

Therapy

Muscle bellies

Friction is given to a healing muscle belly after contusion, in minor muscular tears and in so-called 'myosynovitis'. In minor muscular tears the friction is often part of combined treatment because it is usually applied after an infiltration with local anaesthesia and is followed by active contractions.

The aim of treatment in muscular tears is to allow the torn fibres to heal in such a way that normal increase in breadth on contraction remains possible, a characteristic that can be disturbed by abnormal adhesion formation. Transverse friction aims to achieve a transverse sweeping movement over the longitudinal muscular fibres without pulling on the tear, so to prevent (in the early stage) or to break down (in the chronic stage) adhesion formation between the individual fibres and between individual fibres and the surrounding connective tissue. It is obvious that to break down crosslinks in a chronic stage, the friction can be given forcefully and for a duration of 15–20 minutes, whereas in more recent lesions the technique must be applied more gently and for a shorter duration. Friction to a muscle belly is always given with the muscle well relaxed.

In recent tears, especially in the large muscles of the lower limb, friction is followed by active or electrical contractions with the muscle in a position of maximal relaxation and without weight bearing, so that tension does not fall on the healing breach.

To avoid early recurrence, friction is given for 1 week after all clinical tests have become negative. During the period of treatment, all movements or activities that bring on pain should be avoided by the patient.

Theoretically, friction can be used for all muscle belly lesions. However, some lesions respond so well to local anaesthetic infiltration that friction is not used. This is the case in type IV tennis elbow (lesion at the muscle belly of the extensor carpi radialis). On the other hand, sometimes no alternatives exist to treatment with deep transverse friction (Box 5.1). A lesion of the subclavius or intercostal muscles for instance can be treated only by deep transverse friction.

Musculotendinous junctions

It is a common clinical experience that all musculotendinous junctions (containing both muscular and tendinous fibres) throughout the whole body can be treated only by deep transverse friction. It would seem that no alternatives exist: local anaesthetics, so curative for some lesions of muscle bellies, and steroids, so effective for tenoperiosteal lesions, have not the slightest effect on musculotendinous lesions, whereas deep transverse friction usually has.

Tendons

All overuse tendinitis can be treated by deep massage except for the tenoperiosteal origin of the extensor carpi radialis brevis (type II tennis elbow), which is best treated by an infiltration with corticosteroid or, in refractory circumstances, sometimes by manipulations.

Box 5.1

Muscle belly disorders that can be treated only by deep transverse friction

Subclavius
Brachialis
Supinator
Adductors of the thumb
Interosseus muscles of the hand
Intercostal muscles
Oblique muscles of the abdomen
Interosseus muscles of the foot

Tenosynovitis also usually responds well to deep transverse massage. In this condition, occurring in long tendons with a sheath, inflammation and roughening of the gliding surfaces of both tendon and sheath give rise to pain and sometimes to crepitus. Friction rolls the sheath around the stretched tendon, so facilitating functional movement between the tendon and its sheath. The technique is useful in both acute and chronic lesions.

Lesions at the tenoperiosteal insertion can be treated either with corticosteroid infiltrations or with deep transverse massage. Corticosteroid suspension quickly converts an inflamed and painful scar into one free of inflammation. However, the recurrence rate is rather high, between 20% and 25%.[3] The aim of the massage is to get rid of the self-perpetuating inflammation by breaking up the disorderly scar tissue and adhesion formation by converting it into properly arranged longitudinal connective fibres. This takes longer but once cure is achieved there will be less of a tendency towards recurrence. It may therefore be a policy to start treatment with infiltrations but if the trouble recurs after a few months to substitute with massage.

As a rule, however, friction is always selected as the treatment of choice in athletes or when the tendon is weakened (partial rupture). It cannot be denied that repeated use of corticosteroids, even in small doses and correctly applied, temporarily weakens a tendinous structure. Steroids also take away inflammation and pain, so giving the patient the false impression of being cured. The combination of a weakened tendon and abolition of pain can be disastrous – rupture may ensue.

There exist also a few conditions that seem to respond only to deep transverse friction. Steroid infiltrations are useless here. This is so in tendinous lesions of the interosseus in the hand and at the quadriceps expansion at the patella.

Lesions in the tendinous body, either traumatic or resulting from overuse, are contraindications for infiltration with corticosteroids. Ruptures have been reported after intralesional steroid infiltrations of long tendons and therefore deep frictions are the treatment of choice here.[22,23]

It is obvious that during the whole period of treatment of tendinitis, tenosynovitis or tenovaginitis, the patient must avoid all activities that provoke the pain, especially the loading of the affected contractile tissue.

Ligaments

Transverse massage is an excellent treatment in acutely sprained ligaments, especially in ligaments of the knee and ankle. The background, mode of action and technique differ considerably and depend on the stage of the lesion.

It has been explained (see Ch. 3) that early mobilization is extremely important for swift and full recovery of ligamentous sprains. However, in advocating this, one main difficulty is encountered: the intensity of the initial inflammatory reaction. The slightest movement causes pain which forces the patient to immobilize the joint and the ligaments. However, during immobilization, regenerating fibrils quickly start to form randomly organized scar tissue, leading to crosslinks and adhesion formation. This problem can be solved by gentle transverse frictions. Rhythmic movement across the inflamed ligament eases the pain and the tissue can be moved to and fro in an imitation of its normal behaviour.

In recent cases the friction need not last long nor be very vigorous – 1 or 2 minutes of daily gentle transverse sweeping movement over the regenerating fibrils is enough. As pain diminishes over subsequent days friction is progressively increased to about 4–5 minutes for 2 or 3 days and finally to a full duration of 15–20 minutes. From the third day, friction is followed by passive and active movements within the limits of pain to maintain normal gliding of the ligament over adjacent bones. When the lower limb is involved, the patient should be instructed to walk as normally as possible but without provoking too much pain.

In chronic ligamentous lesions, frictions are also used but in a totally different way. Here adherent scar tissue has formed abnormal attachments as the result of healing during a period of insufficient movement. As a result of the reduced mobility of the ligament, vigorous use of the joint re-sprains the ligament and in due course leads to recurrent sprains.

Treatment will consist of rupturing the adhesions by manipulation, for which vigorous deep friction to the site of the adhesions prepares the ligaments. The massage weakens and desensitizes the structure, making the forced movement practicable and painless.

Experience has shown that particular ligamentous lesions can be treated only by friction. This is the case for the posterior carpal ligaments at the wrist and the tibiotalar ligaments.

Joint capsules

Deep transverse friction can be applied to the capsules of the trapezium–first metacarpal joint, the temporomandibular joint and the cervical facet joints. The indication is traumatic arthritis or osteoarthrosis. Results are fair, provided the arthrosis is not too advanced. Indications and contraindications to friction are outlined in Table 5.1.

Contraindications

Ossification and calcification of soft tissues

Extensive ossification in muscles, tendons, ligaments or capsules is a bar to all active treatment. However the minor calcifications that may occur after a sprain can be managed by

Table 5.1 Indications and contraindications to friction

Indications	Contraindications
Diagnostic difficulties	Ossification and calcification of soft tissues
Preparative massage	Bacterial and rheumatoid-type tendinitis, tenosynovitis and tenovaginitis
Therapeutic massage:	Skin problems such as ulcers, psoriasis or blisters
• To muscle bellies	Neighbouring bacterial infection
• To musculotendinous junctions	Bursitis and disorders of nerve structures
• To tendons	Haematoma, if large
• To ligaments	
• To joint capsules	

friction. In supraspinatus tendinitis, calcification is regarded as responsible for complaints when the insertion is very tender to touch and a radiograph shows calcification. These findings are a contraindication to friction. In contrast, when calcification is present in the absence of severe tenderness, transverse massage can be given.

Bacterial and rheumatoid-type tendinitis, tenosynovitis, tenovaginitis

All types of bacterial and rheumatoid disorders, no matter at what stage of inflammation, are absolute contraindications to friction.

Skin problems such as ulcers, psoriasis and blisters

When normal skin has been abraided – sometimes by friction – massage should not be given. In skin disorders, it must be abandoned when stable skin–finger contact becomes impossible and friction aggravates the skin problem.

Neighbouring bacterial infection

Because these may be reactivated by or may extend if friction is used, it must be postponed until the infection has resolved.

Bursitis and disorders of nerve structures

When bursitis is mistaken for a tendinous or ligamentous disorder and friction is given, the problem will either increase or, at best, the pain will remain unchanged – it certainly will not improve. Friction to a nerve is also harmful.

Haematoma

A haematoma in a muscle belly or after an ankle sprain is not a contraindication to friction. Even if the haematoma is the result of deep friction, treatment may be continued unless the effusion is large.

Technique

Introduction

Transverse massage is not an easy technique. In order to produce results, three conditions must be satisfied.

First, the therapeutic movement should be applied to the exact site of the lesion which may occupy only a very small volume of tissue. In other words, an identification of the site to within 1 cm must be achieved which relies entirely on clinical diagnosis and palpation of the lesion, based in turn on anatomical knowledge. In some instances it will be necessary to palpate carefully the entire structure at fault so as to find the point that reproduces the patient's pain.

Secondly, friction should be applied transversely across the longitudinally orientated fibres, with sufficient sweep to reach all the affected tissue and firmly enough to produce movement between the individual connective tissue fibres of the affected structure.

Third, the movement can only reach deeply seated structures if the deep friction technique of Cyriax is used; that implies attention must be paid to different elements such as the position of the patient and of the therapist's hand, which fingers are used, the amount of pressure, the duration and frequency of the sessions. The patient's skin and the therapist's finger must move as one, so that the deep layers of the skin move over the affected fibres. Therefore all cream, ointments, powder or any other procedure, such as previous heat, that makes the skin sweat, must be avoided. Six to 12 treatments are normally necessary. Except in acute ligamentous disorders they are not given more often than every other day because otherwise the site of the lesion may still be too tender from the previous treatment to permit adequate massage.

Position of the patient

The patient's position must be *comfortable* because it must be maintained for up to 15–20 minutes. Sitting or lying is preferable.

The lesion must be brought *within finger's reach*. In some structures this can be easily attained but others such as the supraspinatus insertion and the anterior aspect of Achilles tendon, require more specific positioning of the patient.

In addition, positioning must place the affected structure under the *required amount of tension*. Full relaxation is necessary for a muscle belly in order not only to treat its surface but also to access a deeply seated lesion. Tendons with a sheath must be kept taut otherwise friction will be ineffective between tendon and sheath. The same applies in ligamentous lesions, which are also placed in tension but within the limits of pain.

Position of the therapist and the hands

The bodily position of the patient should be the most comfortable and least tiring for the therapist. Working height is of chief importance, so an adjustable high–low couch is ideal. To have some economy of effort the therapist should adopt a position that utilizes body weight to a maximum. Usually this is standing and with the patient on a slightly lower plane. The therapist should avoid flexed positions. The shoulder should also not be in abduction because this quickly leads to pain and cramp in the neck and shoulder girdle.

Massage is performed by the whole arm and is not just an activity of hand and digits. Movement is generated in the shoulder and conducted via elbow and forearm to the digits. One set of muscles is used to apply force and another to provide movement, for example pressure with the fingers, movement with the arms. Digits, hand and forearm should generally form a straight line and are kept parallel to the direction of movement.

The majority of friction techniques are performed in two phases: *an active movement*, usually as a result of flexor muscular activity and a *passive movement*, when the arm and hand are returned to the starting position. At the end of the passive phase there should also be a moment of rest during which the therapist fully relaxes the muscles.

The hands can be used in a variety of ways depending on the tissues to be treated and the surface worked on. The wrist and metacarpophalangeal joints should be kept in an almost neutral position. The interphalangeal joints are slightly flexed to avoid traumatic arthritis.

Three main techniques can be distinguished.

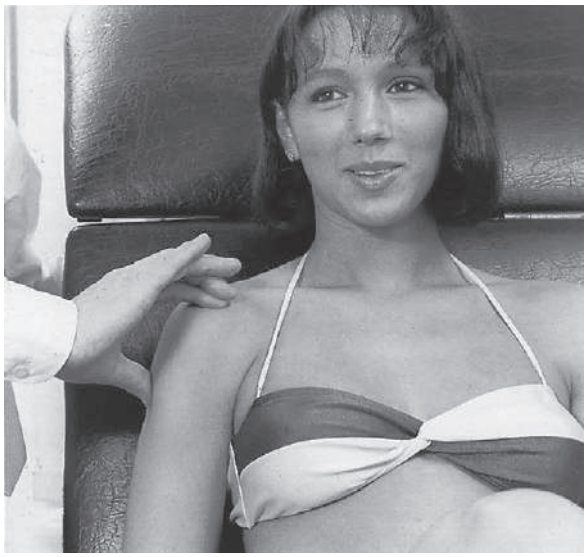
To-and-fro movements

These are used in the treatment of dense, round or flat collagenous bundles (tendons or ligaments) and in the treatment of tenosynovitis. The active phase is a sweep with the tip(s) of one or two digits across the tendinous structure. During the passive relaxation phase the finger is returned to the starting position, without losing contact between finger and skin. Movement is with the arm; friction is given by use of the pulpy part of the finger (Fig. 5.2). In large lesions, as in peroneal tendinitis, two or three adjacent fingers are used together. In deep-seated lesions as in tendinitis of the long head of biceps in the bicipital groove or at its insertion on the radius or in infraspinatus tendinitis, the thumb performs friction.

Counterpressure is usually provided to enable a good sweep. The finger(s) applying counterpressure and stabilization are most important in bringing those applying friction into the right position and also determining the direction of the friction. The thumb is used (to give counterpressure) when the sweep is performed by a movement of the index reinforced by the middle finger or the middle finger aided by the index finger. When the thumb does the massage, counterpressure is from the fingers (Fig. 5.3). The most common way of applying friction around a round edge on a flat surface is to use the index reinforced by the middle finger. Sometimes the opposite is done: the middle finger is reinforced by the index. Sometimes counterpressure is not given, for example in friction to the quadriceps expansion or intercostal muscles.

Pronation–supination

This technique is often used where the lesion is difficult to reach: the anterior aspect of the Achilles tendon, popliteus tendon and the dorsal interossei of the metacarpals. Massage



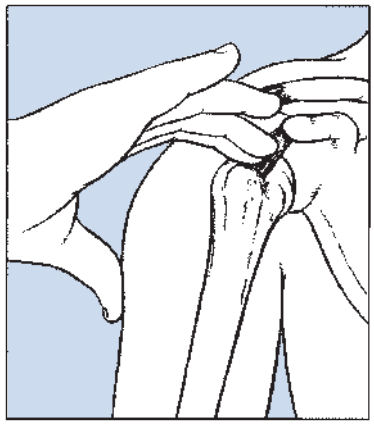


Fig 5.2 • Friction to the supraspinatus tendon: counterpressure is by the thumb.

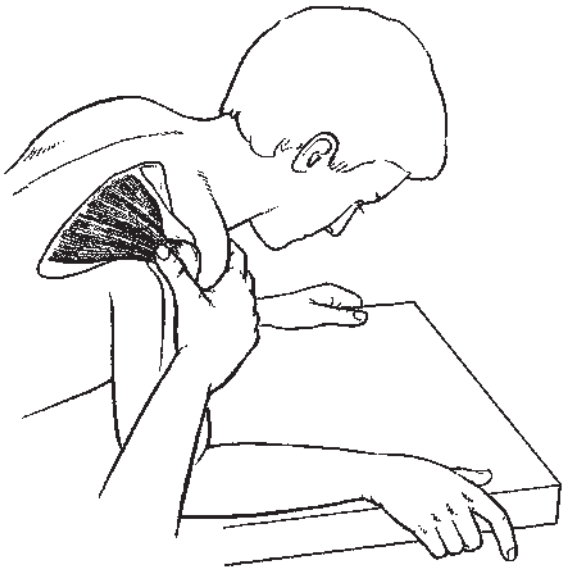


Fig 5.3 • Friction to the infraspinatus tendon: counterpressure is by the fingers.

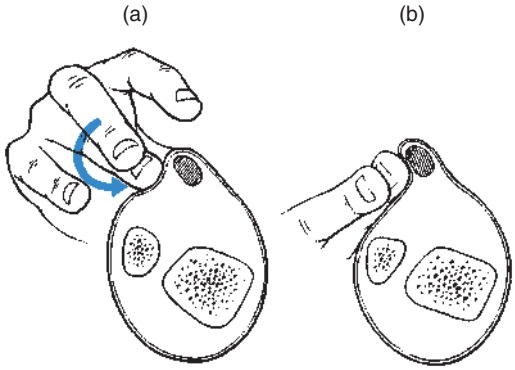


Fig 5.4 • Active phase of pronation–supination friction technique to the anterior aspect of the Achilles tendon. a, starting position; b, end of supination (active) movement.

is performed with the pulpy part of the third finger (long finger), reinforced by the index finger. The long finger is used because its long axis is the prolongation of the axis of pronation–supination rotation of the forearm (Fig. 5.4).

The active phase is usually on supination. No counterpressure is given. Caution is taken not to move the finger on the skin but rather to move the skin and the fingertip as a whole. The passive phase is the pronation movement that brings the frictioning finger back to the starting position without losing contact with the skin.

Pinch grip

This is the normal technique for a muscle belly. The pinch is between the thumb and the other fingers. The muscle is fully relaxed. The fingers are placed at one side of the affected area and the thumb at the opposite side (Fig. 5.5). By drawing the fingers upwards over the affected area, the therapist feels the muscle fibres escape from the grip until only skin and subcutaneous tissue remain.

During the passive phase the fingers are slightly relaxed and moved downwards into the previous deep position where the same movement starts again.

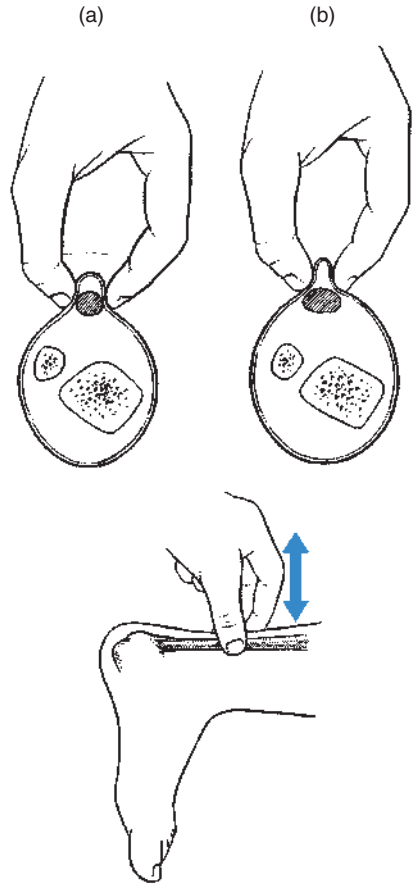


Fig 5.5 • Pinch grip friction to the Achilles tendon. a, starting position; b, end of active phase.

Sometimes the same technique is used in tendinous lesions, for example, at the sides of the Achilles tendon.

No movement between finger and skin is allowed

Deep friction can only be effective when skin and subcutaneous fascia are moved over tendon ligaments or muscles. No movement is allowed between the therapist's finger and the

patient's skin. If movement occurs between finger and skin, blistering soon takes place and usually indicates faulty technique. Sometimes it can be avoided by keeping the skin dry by the use of 95% alcohol in water and/or by placing a piece of cotton in between the finger and the skin.

In the obese, subcutaneous soreness and/or ecchymosis may occasionally occur and sometimes a nodule may form. For this reason the finger should not be in continuous contact with the same area but should displace the skin slightly to one or other side, before pressure is applied.

Direction of friction must be transverse to the tissue fibres

Longitudinal massage improves the circulation of blood and lymph but has no effect on musculoskeletal lesions. On the contrary, because lesions of tendons, muscles and ligaments are normally caused by a longitudinal force, longitudinal massage can possibly be harmful in that it may separate the ruptured ends further. To restore and/or maintain full mobility of a lesion, massage must be given across the fibres, so moving all fibres in relation to each other. To achieve this, the therapist must have a good anatomical knowledge of the direction of the fibres.

Sweep

The main goal of friction is to move fibres in relation to each other and adjacent structures. Enough sweep must be given to the friction for this purpose, so the frictioning finger starts at the far side of the lesion, glides over it and ends at the near edge. Pressure alone, however hard and painful it may be, is totally ineffective. Adequate sweep is sometimes limited by the amount and elasticity of the overlying skin. Initial displacement of skin over the lesion from the near to the far side may help increase sweep and reduce the risk of blistering.

Amount of pressure

Over recent decades, friction has been held in some disrepute in that it was perceived by some as synonymous with very painful treatment. Though it cannot be claimed as wholly pain-free, the pain should not be unbearable. When excessive pain is provoked, this is usually the result of a failure to understand the meaning of the term 'deep', which means 'as deep as needed to reach the lesion'. Many therapists misinterpret this in such a way that they feel that they always have to work hard physically, which obviously leads to pain and may do more harm than good.

The amount of pressure applied depends on three elements:

- *The depth of the lesion*: that friction must always reach sufficient depth to move the affected fibres in relation to their neighbours and sometimes the underlying bone or capsule, increased pressure must be applied to deeper structures.
- *The 'age' of the lesion*: recent sprains and injuries require only preventive friction because crosslinks or adhesions have not had time to form. In long-standing cases more pressure is needed to get rid of these. However, pressure should always be associated with movement and should not replace it because pressure alone is both painful and ineffective.
- *The tenderness of the lesion*: in severely inflamed lesions that are very tender to touch, friction with the usual amount of force may be very painful. Pain can be avoided by starting with a minimal amount of pressure – just enough to reach the lesion – and progressively increasing the force as treatment proceeds.

In order to avoid painful sessions of deep transverse friction it is good practice to grade its application. Begin with a sweep that is gentle and continue this for a few minutes; some numbness of the treated area follows which allows slight intensification of the amount of pressure, which in turn leads to more numbness. Finally, it will be possible to give effective massage that is practically painless to the patient.

Duration and frequency

Friction is usually given for about 10–20 minutes and, because of tenderness, on every second day. The ideal timing of the next treatment is when local tenderness caused by the previous session has resolved. If tenderness persists after 2 days, the pressure used during friction should not be diminished but the interval between sessions must be increased.

Massage immediately after a ligamentous sprain or a minor muscular rupture may be applied daily for the first week but should be of very low intensity and short duration.

Treatment is stopped once the patient is pain-free during daily activities and functional tests are totally negative. Local tenderness may persist longer but disappears spontaneously because it is the outcome of repetitive hard pressure. However, in a minor lesion of a muscle belly, massage is continued for 1 week after full clinical recovery to prevent recurrence (see Table 5.2; see also Box 5.2).

Passive movements

Treatment by passive movement is otherwise known as *mobilization*. It cannot be performed by the patient and requires the intervention of a therapist. Depending on its velocity and the range of movement that is aimed for, it can be graded as A, B and C *mobilization*:

- Grade A mobilization is a passive movement performed within the pain-free range.
- Grade B mobilizations are passive movements performed to the end of the possible range. The latter is indicated by an end-feel. All stretching and traction techniques are grade B mobilizations.
- Grade C mobilization is a minimal thrust with a high velocity and over a small amplitude. It is performed at the end of the possible range, i.e. the moment the therapist has reached the end-feel. Another word for grade C mobilization is *manipulation*.

Table 5.2 Transverse massages/modalities

Indication	Duration (min)	Pressure	Frequency	Combined treatment
Diagnostic	15–20	High	Once	
Acute ligamentous	30 sec	Very low	Daily	Effleurage before and active movement after the treatment
Subacute ligamentous	3–10	Low	Daily–3 times/week	Passive grade B movements after
Chronic ligamentous	15–20	High	3 times/week	Passive grade B movements after
Ligamentous adhesions	15–20	High	Once	Before manipulation (grade C mobilization)
Tendinitis – tenoperiosteal	15–20	Grading	3 times/week	Relative rest – unloaded active movement
Tenosynovitis	15–20	Grading	3 times/week	Relative rest – unloaded active movement
Musculotendinous	15–20	Grading	3 times/week	Relative rest – unloaded active movement
Myosynovitis	15–20	Grading	3 times/week	Relative rest – unloaded active movement
Muscular tear – acute	5–10	Low	Daily	Procaine infiltration before and active unloaded contractions after treatment
Muscular tear – chronic	10–15	High	3 times/week	Active unloaded contractions after treatment
Capsular lesions	15–20	Grading	3 times/week	

Box 5.2

Summary of deep friction technique

1 Position of the patient must:
be comfortable
bring lesion within finger's reach
be appropriate for the type of structure at fault:
- tendon/ligament under tension
- muscle belly: relaxed

2 Position of the therapist must:
be comfortable
facilitate economy of effort:
- alternating active and passive phases
- using large muscles

3 Use of the hand:
To-and-fro movement
Pronation/supination
Pinch grip

4 Use of the fingers:
Counterpressure
Friction using the fingers

5 Other points:
Fingers and skin move as one unit
Direction of friction must be transverse
Sufficient sweep must be used
The pressure must be appropriate
Duration and frequency must be appropriate

Indications

Grade A mobilizations

To promote healing of injured connective tissue

Passive movements within the pain-free range are usually called for in the treatment of injured connective tissue. A comprehensive literature evaluation and meta-analysis of experimental studies of the past several decades have demonstrated that regeneration of injured connective tissue is significantly better with the application of continuous passive motion. If the healing tissues are not loaded, regeneration results in unstructured scar tissue. Under functional load, the collagen fibres are oriented in a longitudinal direction and the mechanical properties are optimized.[24]

Grade A mobilizations are therefore applied early in the treatment of sprained ligaments to promote orientation of the regenerating fibres. They are given in conjunction with gentle transverse massage and within the pain-free range. Care should be taken not to bring the fibres under longitudinal stress in order not to disrupt the healing breach. The movements are of short duration but repeated often.

Distractions at the shoulder

Grade A mobilizations are also used on the capsule of the shoulder in stage III arthritis when stretching and intra-articular steroids are contraindicated (see Ch. 14). In this condition, long-standing stimulation of the nociceptors has increased neuro-sympathetic activity, giving rise to vasoconstriction, muscle spasm and pain.

Gentle and rhythmical grade A movements are performed in such a way that the fibres are stretched longitudinally, stimulating the mechanosensor mechanisms in the joint and so

inhibiting somatosympathetic reflexes that are co-responsible for the increased inflammation of the joint.

Deformity correction

Some cases of lumbago show persistent spinal deviation even after the pain has ceased. A quick thrust manipulation so effective in relief of pain is not effective in correcting the remaining deformity, but sustained translatory gliding in the opposite direction is most helpful. The movement is performed slowly and care is taken to keep the gliding within the pain-free range (see Ch. 40).

Reduction of an intra-articular displacement in a peripheral joint

When a meniscus or some other piece of intra-articular cartilage (with or without an osseous nucleus) becomes displaced and locks a joint, the logical treatment is either to remove it or manœuvre it into such a position that the joint can again move freely over a normal range. The technique needed for the latter is usually a series of manipulative movements which normally contain elements of traction combined with movements of rotation and flexion or extension. In general, these are first performed in the less painful direction of movement and repeated several times with progressively increasing force.

Unlike manipulations in the spine, the manœuvre to reduce an intra-articular loose body is not a grade C mobilization because the movement is not performed at the end of range nor does it contain a 'thrust' element. The flexion–extension movement is over a wide range and stops before the end-feel is reached. The rotation movements are performed to the end of range where end-feel is sensed by the therapist. The 'manipulation of an intra-articular displacement in a peripheral joint' is therefore a combination of grade A and grade B mobilizations.

Grade B mobilizations

To maintain a normal range at the joint

Paralysed muscles may lead to a loss of normal range of motion of the corresponding joint. This can be avoided by gently stretching the capsule, starting as soon as possible after the onset of paralysis. The approach should also be considered for joints that have been injured or subjected to surgery. In such circumstances, there may be the paradox that immobilization is needed for a fracture to heal but that movement is required to prevent loss of capsular elasticity. Often the problem can be solved by adapting the technique of capsular stretching so that it does not influence the site of the fracture.

To stretch the capsule of a joint

Grade B mobilizations may be required to stretch the joint capsule in non-acute arthritis and in early osteoarthrosis. The technique will be further referred to as *capsular stretching*. Capsular stretching is particularly useful in shoulder and hip joints but is applicable in all 'non-irritable' capsulitis. The condition is characterized by:

- a limitation in the capsular pattern (see Ch. 4)
- demonstration of a hard-elastic end-feel to restricted movements (see Ch. 4).

In the very beginning of arthritis, muscle spasm forces the joint to be held in a position of ease, so restricting movement in some directions more than in others (see Capsular pattern, Ch. 4). Immobilization and inflammation cause disordered deposition of collagen fibres in the joint capsule and lead to the formation of capsular adhesions, which in turn are responsible for more restriction of movement and pain. *Stretching* aims at restoring mobility and function by breaking micro-adhesions and producing elongation of the shortened capsule. To be applicable, however, the ligamentous end-feel must be reached before the protective muscle spasm begins. To be successful, the therapist should therefore be able to differentiate between an elastic and a spastic end-feel.

The technique is a slow and steady pressure, performed at the end of range over about 30 seconds to 1 minute with as much force as is reasonable for the patient to bear. Tension is slightly diminished for a few seconds, so affording the patient some respite, and then again increased. From time to time the procedure is completely interrupted. If tension is released too quickly, some pain may be felt and it is therefore wise to bring the limb back into neutral position under traction. The technique is not painless. The stretching causes some micro-ruptures, which result in an inflammatory response and after-pain that lasts for a few hours.

Normally, capsular stretching is given for 15–20 minutes, three times a week. The therapeutic effect is slow.

Capsular stretching can be preceded by application of heat, either through short-wave diathermy or ultrasound. This can relieve some pain and seems to lower the viscosity of the collagenous tissue, allowing more movement for less force. *In vivo* studies on the effects of heat on ligament extensibility have shown that sustained force applied after elevating tissue temperature produced significantly greater residual elongation.[25,26]

Manipulation of a joint capsule under anaesthesia is a grade C mobilization and is only considered for postoperative intra-articular adhesions. A joint that has been manipulated under anaesthesia requires daily intensive mobilization immediately afterwards in order to prevent the formation of new intra-articular adhesions.

To stretch a muscle

Children with short calf muscles can be helped by sustained stretching. The procedure consists of a series of alternating passive stretchings and active contractions. Stretching is maintained for about 8–10 seconds and is followed by full relaxation and active contraction of the muscle. These alternating movements are performed six to eight times per session, preferably daily but at a minimum of three times a week. The earlier the stretching is started, the better the result. Above the age of 15 not much improvement can be expected.

Traction

Traction is used to separate articular surfaces from each other and can be employed in two ways: as an accessory to manipulation or as the sole treatment. Reducing a displaced fragment is obviously easier when the bone ends between which it lies are pulled apart. If the fragment projects beyond the articular edge, tautening of the ligaments and capsule also provides a

centripetal force. In that traction diminishes the pressure on the fragment, pain decreases, which allows the patient to relax the muscles more.[18] In the cervical and thoracic spines, traction is a built-in safety measure for protecting the spinal cord during manipulation (see below) although the use of traction for this purpose and at these sites does not imply that manipulation can be performed on a basis of 'try and see what happens' without a proper diagnosis.[27]

In the spine, traction is used as the sole treatment only in nuclear disc protrusions, which are rare at the cervical and thoracic levels but are more common in the lumbar area. Spinal traction is always mechanical and is performed with the help of a harness (lumbar or low thoracic) or a sling (cervical or upper thoracic). Spinal traction distracts the intervertebral disc spaces. It also pulls the apophysial joints apart and slightly widens the intervertebral foramina.[27–31] At the same time, negative intradiscal pressure is produced with centripetal 'suction' on any protrusion. The posterior longitudinal ligament is tightened, which may help reduce a displaced fragment. All these elements are helpful in the progressive reduction of a nuclear disc protrusion. Reduction of herniated bulges has been demonstrated on epidurography[31–33] and on CT scan[34] during and after traction. The effect of traction depends on the amount of force applied, the length of time per session, the interval between each session and the total number of sessions.[35]

Grade C mobilizations

Grade C mobilizations or manipulations are forceful passive movements, performed at the end of range. Spinal manipulations are mainly to interrupt discodural or discoradicular contact. At the peripheral joints the purpose of a manipulation is to rupture unwanted adhesions between bone and ligament or bone and tendon or to reduce small bony subluxations in the wrist or foot.

Rupture of ligamentous adhesions

Small ligamentous adhesions sometimes develop between a healing ligament and bone. They usually result from a sprained ligament that has been immobilized during the healing process. The usual presentation is at the lateral ligaments of the ankle and at the medial collateral ligament of the knee. The clinical features are local pain during exertion and a small limitation of movement in one direction only. The adhesions can be ruptured by a high-velocity, small-amplitude thrust manipulation, after preparation of the affected ligament with intensive deep transverse friction.

The joint is stretched as far as possible in the limited direction and manipulated with a single firm thrust, during which a typical 'snap' is often heard. Harm is not caused to the ligament nor to the other parts of the joint because the adhesions bear the brunt of the force. The manipulation is almost painless and after-pain is not to be expected. A successful manipulation should achieve an immediate result. Active movements during the following days to maintain function should be highly encouraged.

Rupture of tenoperiosteal adhesions

In type II tennis elbow (tendinitis of the attachment of the extensor carpi radialis brevis), adherent and disorganized scar tissue causes a self-perpetuating inflammation. The manipulation aims to rupture the adhesions and produce a permanent elongation of the tendon. The high-velocity manœuvre is preceded by thorough deep transverse friction in order to numb and to weaken the spot. The manipulation is performed only once per session; 10–15 sessions may be required to achieve a result.

To reduce a bony subluxation

A subluxation of one of the carpal bones or of the cuboid bone can easily be reduced by digital pressure combined with translatory movement during traction.

Manipulation of the spine

Spinal manipulative therapy is a major part of treatment techniques in orthopaedic medicine and is discussed thoroughly below.

Contraindications to forced movements

Contraindications to spinal manipulations are discussed later in this chapter.

Capsular inflammation

Forced movements should not be performed when signs and symptoms of capsular inflammatory activity are present. These are spontaneous pain, pain especially at night, wide reference of pain, inability to lie on the affected side at night or to bear weight on the affected side. Local warmth and effusion are other pointers of a highly inflamed joint. However, if these symptoms and signs are present but the rest of the clinical examination demonstrates internal derangement (e.g. knee, hip, ankle), manipulation is indicated and can safely be performed.

Muscle spasm

Grade C mobilizations should never be applied to a joint that is protected by a muscle spasm. Grade B mobilizations may be used unless the end-feel of the movement that is intended to be forced through is also spastic.

Severe osteoporosis

Grade B mobilizations, for instance stretching of the shoulder or hip joint in elderly people, should always be carried out with caution for fear of fracturing the humerus or the neck of the femur.

Joints and ligaments not under voluntary tension control

Mobilization is also contraindicated for those joints and ligaments on which the tension is not under voluntary control. This is the case for the acromioclavicular, the sternoclavicular and the sacroiliac joints and the sacrococcygeal ligament.

Manipulation of the spine

Introduction

Spinal manipulative therapy includes all procedures of mobilizing or adjusting the spine by means of the hands. As in the peripheral joints, grade A and B mobilizations are movements of low velocity with varying amplitude but remaining within physiological limits and within the patient's tolerance and control.

A manipulation or grade C mobilization usually implies a single thrust of high velocity performed at the end of a passive movement after the 'slack' has been taken up, and over a small amplitude. It goes beyond the physiological limit but remains within the anatomical range. Precision of the movement and control of the applied force are required.[36] Spinal manipulative therapy is a valuable method in the treatment of mechanical spinal disorders. Although it has not been scientifically validated, some studies have shown beneficial effect.[37–40] However, its potential benefit should not be overestimated and the indications must be well defined and based on a sound clinical diagnosis. It must never be done as a test to see if it is effective. Therefore it should not be used on all those with back and neck pain although it may well cure a proportion who actually require it. To use McKenzie's words:

> *Even if you have a hammer in your hand not everything you see is a nail. Therefore indiscriminate use of spinal manipulative therapy must not be made if the criticisms that have been justifiably levelled at chiropractice and osteopathy are to be avoided. The development of postgraduate courses in manipulation is welcome, although some have overvalued the benefits of manipulative therapy. All who undertake manipulation have experienced the feeling of pride and joy in producing cure. It is the duty of those who have more experience of the benefits and limitations of manipulative therapy to moderate the understandable enthusiasm of those entering the field – a few successes may quickly lead to the temptation to manipulate every patient for any disorder.*[41]

Manipulation either helps quickly or not at all. Therefore if improvement does not occur after one or two sessions, manipulation is not likely to be successful and it is pointless to continue with it.

Historical note

Manipulation is as old as medicine and embraces both medicine and mankind in general. In recent times, the medical aspect has become structured and different methods have been developed which are subject to controversy and competition.

Osteopathy

The concept of osteopathy was introduced by A. T. Still (1828–1917) and developed out of frustration with traditional medicine. His ideas were based on two principles: (1) the body has within itself the processes to combat all disease, and (2) the cause of all disease is dislocated bones, abnormal ligaments or contracted muscles with consequent mechanical pressure on blood vessels and nerves.

Diagnosis is mainly based on palpation for restricted spinal mobility and treatment consists of a manipulative system in which joints are forced by a distant leverage. Cure is sought for all kinds of visceral and musculoskeletal disorders.[27]

Chiropraxy

This method was started in 1885 by D. Palmer. It is based on a revision of techniques that originated with Hippocrates and is also influenced by osteopathy. Chiropraxy was long regarded as maintaining osteopathic dogma in its most primitive form and having a strong commercial character.

Chiropractors also claim to cure visceral diseases via the musculoskeletal system. Diagnosis is made on palpation for vertebral displacement and manipulative pressure is applied directly to the bone.

Orthopaedic medicine

This term describes the system of diagnosis and treatment of musculoskeletal lesions introduced by J.H. Cyriax. It is the system on which this book is based. Diagnosis rests on careful history and functional examination. Treatment depends mainly on the type of lesion, and manipulation is applied only when indicated. In spinal manipulation, Cyriax proposed a fixed set of high-velocity, small-amplitude thrusts performed at a certain distance from the lesion and, characteristically for this method, usually under strong traction. The objective of Cyriax's spinal manipulative techniques is to alter the discodural or discoradicular interaction by moving a displaced cartilaginous fragment away from the sensitive dura mater and dural nerve sleeve. Spinal rotation manipulations apply a torsion stress throughout a whole part of the spine, not at just one level. With an intact posterior longitudinal ligament and annulus fibrosus, some of this torsion force exerts a centripetal force by suction on the protruding disc material.[42] This effect is not confined to one level and full reduction is not absolutely necessary for pain relief, in that when contact between dura and disc has ceased the problem is frequently solved.

Manual therapy

Treatment is characterized by rhythmic repeated movements within the physiological range. Oscillatory techniques had already been used by E. Cyriax (father of J.H. Cyriax) but were more widely employed by Maitland and later slightly changed by the different schools of manual therapy (Cyriax:[3] p. 40). Pressure is applied to what is believed to be the appropriate level.

Orthopaedic medicine technique

Before any manipulation is done an exact diagnosis must be made. The decision to manipulate is followed by choice of the correct manœuvre. The patient is put in a comfortable position and the manipulator adopts a stable stance. The floor and shoes should not be slippery, so that there is no risk of inappropriate movement.

Attention must be given to the following general matters, which are important for all manipulations.

Traction during manipulation

Most types of spinal manipulation in orthopaedic medicine are performed under traction. For the cervical and thoracic spine, traction is applied by the manipulator with the help of a fixing belt or by one or two assistants. At the lumbar level, traction is usually already built into the manœuvre. Traction facilitates the reduction of a displaced fragment and provides an important safety element against the possibility of a protrusion contacting the spinal cord during manipulation.

End-feel on taking up the 'slack'

All spinal manipulations are performed over only a small amplitude. Therefore all 'slack' must be taken up by moving the vertebral joints passively to the end of the normal passive range of movement. At this stage it is absolutely necessary to have a clear idea of the end-feel, which is nominally elastic for the entire spine. An end-feel that does not correspond with this – muscle spasm, or hard or empty end-feel – is an absolute contraindication to any manipulation and the manœuvre is not continued.

Final thrust

Immediately after the slack has been taken up in the surrounding tissues, a minimal amplitude, high-velocity thrust is given to affect the target tissue. The velocity is of great importance because tissues loaded quickly are stiffer so that the manœuvre will affect only the displaced fragment of disc and will not damage the surrounding structures.[43]

The amount of force used for the final thrust depends mainly on the patient and manipulator in that a tall manipulator will have to use less force in a small patient and vice versa.[44] The length of the lever (see later) is also important. The force should always be kept reasonable and may be progressively increased, according to the immediate result.

The manipulation thrust is often accompanied by an audible 'pop'.[29,45] Although it is a common belief that pops or clicks are provoked by the formation of a temporary vacuum, as occurs in small peripheral joints put under traction,[46–48] this is not definitely established for the spine. An alternative and more likely explanation is movement of cartilaginous fragments, as may be heard during manipulation for a loose body in the knee or hip. If the clicks were simply the result of the collapse of a vacuum they should also be – but are not – heard during mechanical traction, in which the traction force is much higher.

Leverage

The amount of force used depends on the length of the lever. If for example a rotation of the lumbar spine is forced via the shoulder and pelvis, the lever offered by the shoulder is the same length as that offered by the pelvis, so an equal amount of force must be used by both hands. But if the femur is used instead of the pelvis, the length of the pelvic lever doubles. The hand on the shoulder must apply double the amount of force that is used on the knee. The longer the lever, the less force is needed.

Reassessment

After each manœuvre the patient is assessed, the criteria of success being the absence of symptoms and the restoration of pain-free movement. The patient and not the manipulator is the arbiter.[49] Depending on the immediate outcome, the therapist decides whether to repeat the same manipulation, probably with increased strength, to try another manœuvre or to refrain from further manipulation.

It should be appreciated that after successful manipulation the anatomical lesion is still present: a piece of cartilage, although put back in place or into a neutral position, persists and may redisplace. For this reason, those who undertake manipulation should note the results obtained and what manipulations were used, in case of recurrence.

Figure 5.6 outlines the assessment of spinal lesions and their manipulation.

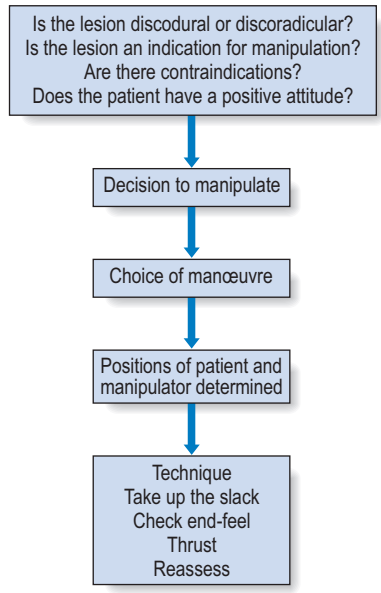


Fig 5.6 • Spinal manipulation.

Selectivity of a manipulative treatment

Selectivity must be considered both in diagnosis and therapy.

Selectivity of diagnosis

Osteopaths and manual therapists claim to have developed the clinical skills to localize by palpation the exact site of fixation, and are therefore able to perform the manipulation at the required level. Diagnosis is mainly based on segmental mobility tests: joint play, springing test or tests of passive physiological movements. Movement can be tested by exerting local pressure at one side of a vertebra while counterpressure is applied to the contralateral side of the vertebra above or below. For the lumbar spine, it can be done with the patient on the side with both hips flexed to 90°. Small movements of the thighs cause the lumbar spine to flex or extend which can be detected by palpation of the spinous processes.

Other practitioners look mainly for palpable soft tissue changes, such as local subcutaneous thickening or exquisite tender spots (trigger points) in muscles, ligaments (iliolumbar,

sacroiliac) and over bony prominences. All these are considered to be important diagnostic and therapeutic factors.

The great variability in the extent of spinal stiffness between subjects, or at different levels within the one subject, makes the determination of areas of abnormally increased stiffness difficult. Increased stiffness may in fact be a normal variant and bear no relationship to the patient's presenting symptoms. Few of those advocating segmental mobility tests have seriously examined the value of their tests. They have generally presumed that the tests were useful because their patients got better.[50] However, several studies have failed to demonstrate the reliability of these tests.[51–62] Therefore it must be made clear that judgement of small changes in the range of movement of a segment, in the absence of full restriction of movement, remains a very subjective finding, which depends mainly on the personal conviction of the examiner rather than on objective measurements. Moreover, in soundly based tests, findings must be reproducible and must show correspondence when performed by other investigators. In the establishment of 'joint play' the inter-observer discrepancy is too large to be acceptable.[63] In 1973, Cyriax attended a demonstration in which five therapists, all of whom specialized in mobility testing, examined over a period of a few minutes a patient with a neck problem. There was no agreement between these specialists about the level of the lesion (C2, C3, C4, C5, C6 or T2), or about the direction of restriction (see Cyriax:[64] p. 108). Similarly, a patient who had congenital fusion of the sacroiliac joints was examined by 10 manipulators. Each had his own diagnosis, such as left anterior sacrum, right anterior sacrum and bilateral posterior sacrum, although 'the tests were very positive' for all of them (see Maigne[65] and Cyriax:[64] p. 363).

Even if it were possible to identify with certainty localization of the hypomobile segment, the question remains as to whether this is also the site of the lesion. Studies have shown that frequently the lesion does not lie at the joint where motion is restricted but at one which appears to be normal.[55] Moreover, other disorders such as osteophytosis, congenital fusion and ankylosing spondylitis all give rise to restricted movement which is usually painless.

Selectivity of manipulation

Manipulation is often accompanied by immediate relief of symptoms and signs which, since success has been obtained, is logically taken as absolute confirmation of the precision of diagnosis and treatment. Such a deduction may be – and often is – totally wrong. The only thing proved is that the manipulation was efficacious. The erroneous reasoning that successful manipulation necessarily confirms the diagnosis has been and is still today an important argument for the false belief of some schools that manipulation can cure all kinds of disorders even including visceral diseases. A typical example is pectoral pain, resulting from a thoracic discodural interaction which is misdiagnosed as angina. The patient goes to an osteopath who manipulates the thoracic spine and the pectoral pain ceases immediately. Both patient and manipulator, misled by the wrong diagnosis, will believe that the manipulation has altered autonomic tone and cured the angina, whereas what it actually did was interrupt the discodural interaction.

In orthopaedic medicine most manœuvres used are non-specific long-lever manipulations. These include all procedures in which a force is exerted on a part of the body some distance away from the area where it is expected to have its beneficial effect. Levers may include the shoulder, transverse processes and parts of the skull, pelvis or thigh (Frymoyer *et al*:[45] p. 1594). Although some criticize the crudity of long-lever high-velocity manipulation it should be realized that it is not elegance, impressiveness, specificity or technical difficulty which count but effectiveness and safety. Furthermore, the use of a lever enables the manipulator to reach the lesion more effectively. During the preparative phase – on taking up the slack – all the normal joints are brought to their anatomical end position except for the joint that is blocked. When the additional thrust is given, the final extra pressure falls inevitably first and to the greatest degree on the deranged joint. The manœuvre thus becomes specific even though in general the techniques are regarded as non-specific.

Long-lever manipulations are in full contrast to what are called 'specific' short-lever high-velocity manipulations. Here the goal is to act specifically at what is believed to be the level of the lesion. The spinal segment and the facet joints adjacent to the lesion are locked by moving the spine to the physiological limit of passive movement and a high-velocity small-amplitude thrust is given to the short vertebral lever (transverse process or spinous process) in the specific direction that will liberate the restricted movement. However, it is technically not possible to lock all other joints and then to manipulate at just one level (Cyriax:[64] p. 108). It was even demonstrated that by mobilizing the sacroiliac joints after locking of the lumbar spine, the largest movement took place between L4 and L5.[66] Furthermore, if diagnosis fails to be absolutely right, how can there be certainty as to the exact level of the lesion to work on? Fortunately for those who employ 'specific' short-lever manipulations, these are much less specific than they think, because the manipulations actually cover a much larger part of the spine and so unintentionally also include the lesion.

Specificity is a false attribute. The methods that claim to lead to specific localization in both diagnosis and treatment are scientifically unacceptable. Claims of specificity are made in order to give prestige to manipulators who claim to feel something that cannot be felt. Manual therapists, chiropractors and osteopaths over-complicate their teaching and often create excessive patient dependency, instead of providing the patient with independence. Indeed, patients are encouraged to return at regular intervals for pointless prophylactic adjustment.[41] We support R. McKenzie's conclusion that demystification of spinal manipulative therapy is an urgent priority.[41] Chiropraxy, manual therapy and osteopathy, however, thrive by creating the impression that there is something complex and exclusive about the practice of passive end-range motion that only experts in these practices can understand or have the skills to feel. The belief is strong that expertise in the understanding and delivery of spinal manipulative therapy requires 3 or 4 years' training. The main advantages of the methods discussed in this book are that the manipulations are much simpler and at least as effective as those advocated by chiropractors, osteopaths and manual therapists. Non-specific long-lever manipulations are quickly effective, do not take long to perform and are

simple to learn. Moreover, they can take only about 180 hours of tuition, provided that the student has already gained qualifications in medicine or physiotherapy.

Mode of action of spinal manipulation

To date, the mode of action of manipulation has not been totally clarified, although many different models have been put forward. All pose unsolved questions, lack objective confirmation and are subject to dispute. Different attitudes towards spinal disorders determine theories and explanations.[67]

Those who believe in 'posterior facet joint' syndrome as a frequent source of back pain, believe that manipulation corrects a posterior joint dysfunction, in which either a minor subluxation of a facet together with an entrapment of the synovial fold, or of part of a small intra-articular meniscus, is held responsible, both of which may give rise to a blocked vertebral joint (Kirkaldy-Willis:[29] p. 296; Mathews and Yates[68]).

Others suggest that manipulation ruptures periarticular connective tissue adhesions or that it abolishes a muscle spasm.[69] In our opinion, a manipulation does not directly affect muscle spasm; instead, the latter disappears secondarily when the underlying problem is solved.

It has also been suggested that manipulations influence the mechanism of cutaneous pain tolerance as the result of release of endorphins, or that stimulation of mechanoreceptors of the facet joint capsules, ligaments and annulus fibrosus influences the neurophysiological gate that controls the perception of pain.[70] This could conceivably abolish pain immediately after manipulation but does not explain longer term improvement.

Some believe that manipulation may move an inflamed nerve away from the herniation[71] or that relief is achieved via a return to normal of neurogenic reflex activity.[42,72]

We strongly believe that spinal pain is the result of disc protrusion that gives rise to a conflict between the posterocentral or posterolateral rim of the disc and the pain-sensitive dura mater or dural nerve sleeve, and that a displaced fragment of an intervertebral disc can be moved by manipulation.[42,73–76]

This was the hypothesis of Cyriax[3] (see his pp. 38–50) and Maigne[77] and has been supported by the observations of Mathews and Yates,[68] who have shown by epidurography that in acute lumbago small lumbar disc protrusions diminished in size after manipulation. Manipulative interruption of contact, moving the displaced cartilaginous rim away from sensitive structures, is the objective for relief of pain and is best obtained by a non-specific long-lever, high-velocity manipulation.

Indications for spinal manipulation

Spinal manipulation is useful for all annular disc protrusions in the absence of any contraindications or of any signs or symptoms that indicate that manipulative reduction would not succeed. All these factors may vary for the cervical, thoracic and lumbar spine, and the indications are discussed in detail in later chapters.

Contraindications to spinal manipulation

All effective treatments are potentially dangerous and therefore possess contraindications. Although manipulative techniques are not hard to learn, years of experience are needed to learn when to manipulate, when not, and what sort of manœuvres to use.

Contraindications to manipulation are bleeding disorders, softening of bone, rheumatoid conditions, neurological deficit and danger to the spinal cord.

Box 5.3

Anticoagulants contraindicate spinal manipulation

Coumarin derivatives:
- Heparin
- Warfarin
- Phenprocoumon
- Acenocoumarol

Indandione derivatives:
- Diphenadione
- Phenindione

Bleeding disorders and anticoagulant use

When normal clotting of blood is not guaranteed, as in congenital or acquired (liver disease) bleeding disorders or because of the administration of anticoagulants (Box 5.3), spinal manipulations are potentially dangerous. Disastrous results can follow, such as intraspinal haemorrhage with the formation of a haematoma that may lead to sensory and motor deficit, to paraplegia, quadriplegia or death.[78] For this reason, a coagulopathy is an absolute contraindication to spinal manipulations. Manipulation can be safely performed only after blood clotting tests have returned to normal.

Spinal tumours, unstable fractures, vertebral infections and severe osteoporosis

(see Grieve:[27] p. 829)

These all result in weakening of bone with risk of further damage by manipulation. Long-lever manipulation is not safe in severe osteoporosis.

Rheumatoid arthritis, psoriatic arthritis, Reiter's syndrome and ankylosing spondylitis

The first three of these may be associated with ligamentous laxity and gross destruction of the joint with subsequent instability. Manipulation must not be undertaken. The same applies for the inflammatory stage of ankylosing spondylitis. In the unlikely event of a patient with this disorder developing a disc lesion, manipulation is not at all safe, especially in the cervical spine, where luxations, fractures and cord compression have been described.[79]

Neurological deficit and spinal cord compression

Segmental neurological deficit is characterized by disturbance of either motor function, with impaired reflexes and/or muscular weakness, or sensory function, with diminished sensibility. The two may be combined. Segmentally referred pins and needles due to compression of the sensory fibres of a nerve are not regarded as neurological deficit and are therefore not a contraindication. In neurological deficit, manipulation is usually

without value because deficit suggests a large protrusion that cannot be reduced. This opinion is not universally supported,[80,81] and some still manipulate when minor neurological signs are present. However, in all instances where progressive neurological deficit is present, manipulation must not be done.

A similar rule applies in cord compression or cauda equina syndrome when a very large posterocentral protrusion threatens the spinal cord or the cauda equina. This is true not only for obvious signs but also in the presence of even the slightest symptoms, such as extrasegmental pins and needles or pain in the S4 dermatome.

Hypermobility

In several schools of thought on manipulation, too much emphasis is placed on hypermobility. Although it is true that special attention must be given to hypermobility in the cervical spine, it is not important elsewhere. A hypermobile joint may move further than an ordinary joint, but once extreme range is reached it stops in exactly the same way as does an ordinary joint. More often the disc fragment itself is hypermobile, which may lead to frequent recurrences. This circumstance does not contraindicate manipulation but calls for extra measures to achieve stabilization, for example by infiltration with ligament sclerosant (see Cyriax:[3] p. 38).

For the cervical spine a history of frequent disc problems, alternating from one side to another, may be suggestive of hypermobility. When doubt exists the following tests may be useful: spinal flexion with putting the palms flat on the floor with knees straight, passive apposition of the thumb to the flexion aspect of the forearm, passive hyperextension of the fingers parallel to the external surface of the forearm and hyperextension of the elbow and/or of the knee of 10° or more. If one or several of these are positive hypermobility is likely.[82] Also, when the end-feel on passive rotation, extension or on preparation for a straight-pull manipulation is too elastic, further manipulation must cease.

Spinal manipulation under anaesthesia

Spinal manipulation should never be done in an unconscious patient. First, anaesthesia is unnecessary. Second, the final warning of potential danger offered by increased pain or abnormal end-feel on taking up the slack is completely lost, as is clinical assessment after each manœuvre. Consequently, complications are more frequent after manipulation under anaesthesia.[83]

Patients who have difficulties in relaxing during manipulation may benefit from being given diazepam before a session.

Evaluation of the effectiveness of spinal manipulation

Although several studies, mainly done on the lumbar spine, support the view that many patients benefit from spinal manipulation,[39–41,84–87] agreement on the effectiveness and the role of manipulation in spinal disorders is still lacking. Reports from clinical trials are not always clear and the effects of manipulation are often difficult to interpret[88] because of a small number of patients and their selection by symptoms rather than diagnosis. To make trials scientifically valuable a precise definition of manipulation is necessary – a record of which types of technique have been used and for what type of disorder and its duration. It should also be said that a double-blind randomized trial on spinal manipulation is not possible because of the absence of a placebo and the obvious knowledge by the patient and operator of what is being done.

Sims-Williams published a clinical trial on 94 patients with non-specific lumbar pain, who took part in a trial in which placebo physiotherapy was compared to Maitland mobilization and manipulation. Studies mainly showed that manipulation hastened improvement chiefly in those patients whose severity and duration of symptoms did not require specialist referral.[80,89]

Bergquist-Ullman and Larsson,[90] Coxhead *et al*[81] and Farrell and Twoney[39] found a significantly shorter duration of complaints in patients receiving manipulation.

Chrisman *et al*[91] stated that half of those suffering from sciatica showed clinical improvement after manipulation, but the improvement could not be objectively confirmed by myelography.

Hadler *et al*[86] compared the results of spinal mobilization to high-velocity thrust manipulation in patients with acute, uncomplicated low back pain and tried to exclude all elements of chronicity or compensation insurance. Patients treated by manipulation improved to a significantly greater degree and more rapidly than those treated by mobilization.

In one study, in which rotational manipulation was compared with simulated short-wave diathermy, those who received the former fared better immediately after the session.[92] However, when pain relief was compared 7 days after treatment there was no significant difference between the two groups.

In another study, 24 patients with complaints of less than 3 weeks' duration were followed. It showed that 92% of those treated by rotational manipulation were cured in less than 2 weeks.[93] Of those who received diathermy, only 25% got better in the same period of time.

A further trial in acute disc herniation compared conventional physiotherapy with manipulation and showed that the manipulated group scored significantly better.[94] All manipulated patients were able to return to work, whereas only 26% of the physiotherapy group could do so.

In a randomized clinical trial Koes *et al* compared the effectiveness of manipulative therapy, physiotherapy, treatment by the general practitioner and placebo therapy in 256 patients with non-specific back and neck complaints of at least 6 weeks' duration. They concluded that manipulative therapy showed a faster and greater improvement in physical functioning compared to the other three therapies.[95,96]

Controlled studies performed by Mathews *et al* clearly demonstrated that manipulation treatment for low back pain scored significantly higher in the subgroup with limited straight leg raising.[97,98]

Generally it can he concluded that manipulation and mobilization hasten both pain relief and the resolution of objective signs such as limitation of straight leg raising and of articular movement. Manipulation with high-velocity thrust seems to work better and quicker than more gentle mobilization techniques. The results are also better in recent-onset cases than

in those of longer duration. Manipulation helps either quickly or not at all; which means that manipulative therapy continued over a long period of time is not appropriate.

Complications of spinal manipulation

General considerations

Manipulation is, as with anything in medicine, not without danger and it must be admitted that even in experienced hands accidents may happen. Therefore manipulative treatment must never be undertaken recklessly, or on a 'hit or miss' basis.[3] Although it is probable that more accidents have occurred than have been reported, the risks must not be overstressed.[40,99] In 30 years of clinical practice Kirkaldy-Willis[29] (his p. 293) did not have one patient who was made worse by a manipulation. Dvorak and Orelli estimated that in 1 out of 400 000 manipulations severe neurological damage results, and in 1 out of 40 000 slight neurological signs appear.[100] It should not be forgotten that traditional treatment with non-steroidal anti-inflammatory drugs is also not without complications, in that they may give rise to severe gastrointestinal problems (mucosal bleeding, or reactivation of previous ulcers or initiation of new ones), haemolytic anaemia, leukopenia, thrombocytopenia and even fatal agranulocytosis, salt and water retention, albuminuria, nephritis, acute renal insufficiency, or to allergic reactions in all degrees of severity. No matter what treatment is given there will always be an inherent risk, but this should be reduced to a minimum by taking all necessary precautions.

Complications of manipulation can range from a temporary slight increase in pain to severe neurological deficit and death.

Postmanipulation pain

Immediate postmanipulation pain – which is usually not severe and is present in the whole area – can be relieved by using special techniques at the end of a manipulation session such as a lateral glide at the cervical spine or the rhythmic extension technique in the lumbar spine.

A degree of pain for a short duration – due to stretching of the muscles and ligaments – is normal in the elderly. It disappears spontaneously within 1 or 2 days. To avoid undue anxiety, patients should be warned of this. For the same reason, in older patients some manipulation-free days must be planned between sessions.

Increased pain

Worsening of the condition immediately after a manipulative manœuvre manifests by either an increase in original pain or by its movement more laterally. The implication is that the protrusion has been further displaced. This may occasionally occur in a manipulation which was perfectly performed and does not necessarily mean a poor technique. Further worsening can usually be avoided by performing a different manœuvre, very often by changing the direction of rotation. The same course is adopted when, after manipulation, pain moves to the other side – an indication that overcorrection has occurred.

Fracture

Exceptionally, manipulation may be complicated by a rib fracture, sternal fracture or fracture of a transverse process. These usually occur in the elderly and can be avoided by not using certain techniques in patients above 60 years of age.

Onset of neurological deficit

Sometimes uncomplicated root pain becomes complicated by neurological deficit, and it may occur after a manipulation that was correctly performed, but which has resulted in the protrusion being moved further laterally or the compression being increased. This event must be considered when root pain does not improve. Neurological reassessment must be done and, as a consequence, further manipulations will cease and, if necessary, epidural injections or sinuvertebral nerve blocks may be given. It is very seldom that the problem is so severe that neurosurgical intervention is needed.

Vascular interference and cord compression

Most severe problems arise from compression of blood vessels resulting in temporarily or permanent ischaemia of the cerebrum, brainstem or spinal cord, or as a result of direct compression of the spinal cord by a disc fragment or as a sequel to vertebral fracture or luxation. Para- or tetraplegia may follow, sometimes leading to death. These complications are usually encountered in the cervical spine and are extremely rare at the thoracic level. When they occur at the lumbar spine, they may give rise to a cauda equina syndrome, often followed by definite neurological features.

Risks in relation to level

The risks involved in manipulation differ at the cervical, thoracic and lumbar levels.

Cervical level

Serious neurological complications have been reported after manipulation of the cervical spine. The techniques that are blamed to cause most of the injuries are hyperextension manœuvres accompanied by excessive rotation.[45,102,103]

During a recent inquiry over a period of 2 years among 468 Californian neurologists, 55 strokes following osteopathic cervical manipulation were surveyed. Most of the patients continued to have persistent neurological deficits 3 months after the onset and about one-half were marked or severe. Nearly all of the strokes involved the posterior circulation and almost one-half were angiographically proven.[109] A Danish survey reported the incidence of cerebrovascular incidents (CVI) after chiropractic manipulation to be about 1 for every 120 000 cervical treatment sessions, manipulation to the upper neck being about four times more commonly associated with CVI than treatment of the lower neck.[110]

Neurological complications are mainly the result of a dissection of the vertebral artery[104–108]. Vertebral artery dissection (VAD) is an uncommon vascular wall condition that typically involves a tear at some point in the artery's lining and the formation of an intimal flap (see Chapter 9). The latter triggers off an arterial narrowing or even a complete obstruction of the lumen.[111,101] Although the pathophysiology of a dissecting vertebral artery is well understood, the underlying cause of intimal tears remains uncertain. Most experts link VAD to traumas of varying degrees of severity and maintain that because tearing occurs, previous trauma was necessarily involved. An article reviewing 606 cases of VAD reported that 371 (61%) were spontaneous. The remaining 39% were associated with trivial or other trauma, which included manipulation in 9% of the total cases.[112] However, VADs more commonly occur after

very minor trauma, and even everyday activities such as reversing a vehicle, coughing, vomiting, unusual sleeping positions or having one's hair washed at a beauty salon[113]. For that reason, evidence is mounting that the association between spinal manipulation and stroke is coincidental rather than causal and reflects the natural history of the disorder[114].

Thoracic level

Complications at this level are usually due to vertebral metastases or to severe osteoporosis resulting in fractures of vertebrae, ribs or sternum.

Lumbar level

The main complication is compression of the cauda equina because of a massive posterior sequestration of a lumbar disc.[115] Compression of the cauda equina complicating lumbar manipulations without anaesthesia is extremely rare, when it is considered that in the US in 1975 about 124 million office visits to chiropractors took place. Between 1911 and 1992 only 13 cases were recorded.[116] This potential complication should never be taken as contraindication to manipulation. However, anyone who manipulates must be able to recognize the syndrome immediately, so that no time is lost before surgical decompression is performed.

Measures to prevent complications

Although complications may occur and cannot be totally excluded by any means, each manipulator should try to reduce the eventuality to the lowest possible degree. It would be wrong to condemn the manipulator simply because a mishap or a complication occurred. If this was done it would mean that, instead of getting on with his work, the practitioner would forever be 'looking over his shoulder'. It is not possible to arm oneself against unpredictable events. Even if they do occur occasionally, this argument should not be used to condemn manipulation.[116]

Obviously all measures must be taken to decrease the risks as far as possible. In orthopaedic medicine the necessary steps are taken during the whole procedure, starting with the history, through clinical examination, technical investigations and ending with the manipulation itself. Safety measures and acknowledged warning signs are incorporated in each stage. These are discussed in detail in the relevant chapters and are only briefly outlined here.

History taking

Before any manipulation, history taking must be sufficiently thorough to elicit information on drug intake (anticoagulants) and the existence of other (visceral–rheumatoid) diseases. Severe pain in the absence of movement or pain worse at night may indicate that the lesion – whatever it may be – is in a highly inflamed state and unsuitable for manipulation. Expanding pain, excessive weight loss and recent operations may indicate metastases and must always be excluded. All these are severe warning signs.

Cervical root pain of more than 2 months' duration or root pain at lumbar level for over 6 months (unless there is still scapular or lumbar pain influenced by cervical or lumbar movements) does not respond to manipulative reduction. Hence no attempt to manipulate should be made.

Clinical examination

A complete clinical examination must *always* follow. In particular, in relation to the cervical spine, full attention must be paid to the end-feel on passive rotations and on extension. Even if the clinical examination suggests an ordinary disc lesion, if the end-feel shows muscle spasm is empty or if it comes to a bony hard stop, manipulation should not be done before serious disorders have been excluded. Muscle or empty end-feel may point to a disorder such as metastases, fracture or infection. A bony block end-feel implies that it is impossible to increase the range beyond this point, and therefore the lesion is unsuitable for manipulation.

A patient suffering from acute lumbago or sciatica usually cannot put weight on the joint, may suffer from constant pain day and night and on clinical examination may be found to have deviation and muscle spasm. When more severe disorders such as metastases, rheumatoid arthritis and ankylosing spondylitis have been excluded, a disc lesion is most likely and manipulation can be safely performed.

A protrusion of disc substance larger than the aperture from which it emerged – usually characterized by a neurological deficit – means that reduction is impossible. The same accounts for nuclear protrusions, which require traction.

Manipulation should never be used simply because 'no contra-indication has been discovered, no one can think what else to try, and a friend was recently relieved by this means' (Cyriax:[117] p. 31). If the diagnosis is unclear or if too many inconsistencies are found, manipulation should be avoided. Once again it should be stressed that manipulation follows only if the diagnosis of a discodural or discoradicular interaction has been established. For safety, the following must be added: 'If the diagnosis is established but it is uncertain if manipulation will work, then try. If the diagnosis is uncertain and it is unclear whether or not manipulation is safe, do not try.' (Cyriax[3]).

Radiographic appearances

Radiological changes are signs of structural alterations that usually cannot be treated, though it is of most importance to find out if they are relevant to the symptoms present. Osteoarthrosis of the spine cannot be cured by manipulation, but a manipulation can easily reduce a displaced disc fragment even in an osteoarthrotic spine.

Although the diagnosis of tumours of the lumbar spine is largely dependent on radiography examinations, it must be remembered that 30% of the osseous mass of a bone must be destroyed before this type of lesion becomes radiologically evident.[118] Therefore radiographs do not reveal early disease, and too much reliance on negative radiographic appearances can give rise to a false feeling of security. Radiographs that show no evidence of bone disease should not be taken to indicate that manipulation is safe (Cyriax:[64] p. 292). A decision to manipulate because the radiograph is normal carries a high risk of worsening the patient's condition and may even lead to disaster. The diagnosis of lesions in bone in the early stage is mainly dependent on history and clinical examination. Special attention must be paid to 'warning signs'. In several disorders, such as early invasion of bone by a secondary neoplasm, the clinical signs may precede the radiographic findings. In

doubtful cases, when routine radiography fails to support the clinical impression, a radioisotope scan must be obtained.

Appropriate technique

Manipulative techniques contain important safeguards against possible accidents. This is chiefly so for the cervical spine, in which dangerous techniques such as extension in combination with rotation should never be used.[36,119,120] Those with special interest in musculoskeletal disorders should not only read textbooks but must be prepared to take courses in order to learn the necessary theoretical and practical skills before undertaking manipulation.

Once a manipulation has started, the operator must always concentrate on the type of *tissue resistance* (end-feel) *while taking up the slack* just before the final thrust is given. If the end-feel is abnormal, he must stop immediately and must not manipulate. To push through muscle spasm protecting a joint should never be attempted.[117]

To prevent compression of the spinal cord all manipulations must he performed under *traction*.

The major aim of manipulation should always be to gain maximal benefit with the use of minimal force. Therefore it is good sense to start gently and progressively to increase the force if needed.

Each manipulation must always be followed by re-assessment. If the patient is worse, the same manœuvre should not be repeated but another manœuvre can be tried. If the latter also increases signs or symptoms, further manipulations should be abandoned.

Avoidance of overtreatment

Once symptoms and signs have cleared, treatment must be stopped. There is no sense in asking the patient to come back at regular intervals for 'prophylactic' manipulations.

Box 5.4 summarizes measures to prevent complications.

Box 5.4

Measures to prevent complications

History:
- Exclude anticoagulants
- Check for warning signs
- Ascertain duration of root pain

Clinical examination:
- Check for warning signs
- Assess end-feel
- Exclude neurological deficit

Radiographic appearances:
- Exclude structural alterations
- Do not rely on radiography alone; negative radiography does not guarantee absence of disease

When manipulating:
- Avoid dangerous techniques
- Check end-feel
- Use traction
- Start gently
- Reassess after each manipulation
- Do not overtreat

Active movements

The effects of immobilization on skeletal muscle, tendon, ligament, joint capsule and articular cartilage are dramatic and have been described thoroughly in Chapter 3. Physical activity is also the primary stimulus for the repair of musculoskeletal tissues. Therefore, most therapeutic modalities that are advocated in this book are in conjunction with movement. Active movements are defined as those undertaken by the patient via muscular contraction. In orthopaedic medicine they are not so often used as a sole therapy; their main role is in combination with other therapeutic techniques. However, there are a few situations (both therapeutic and prophylactic) where active movements play a particular role.

Simple active movements to gain or preserve normal range in a joint

A few examples make the above clear. Immobilization of the shoulder leads to the development of an arthritis with limitation of all movements in a capsular way if the shoulder is not brought daily through a full range of movement. Total rupture of the infraspinatus tendon leads to limitation of external rotation of the shoulder if the patient is not performing daily lateral rotations.

Treatment of sprains is essentially treatment with movement (see p. 48). Although the first measure is friction, passive and active movements within the limits of pain to maintain normal gliding of the ligament over adjacent bones should be encouraged. When the lower limb is involved, the patient should be instructed to walk as normally as possible but without provoking pain.

Ligamentous adhesions that have developed as the consequence of a chronic sprain at ankle or knee need to be ruptured manually: the joint is forced through its normal range of movement by a high-velocity, short-amplitude thrust manipulation (see p. 12). After the manipulation and in order to retain the mobility achieved, the patient should repeat the same movement actively and on a daily basis. After rupturing of adhesions under anaesthesia, active movements in addition to passive ones are also necessary to maintain the range that has been regained. They should be performed several times a day.

Isometric contractions

Isometric contraction is the development of tension within a muscle without significant change in its fibre length. Joint motion or work is not achieved.

In orthopaedic medicine isometric contractions are mainly performed to strengthen stabilizing muscle groups. The main example is in treatment of shoulder instability: in order to provide a firm foundation for the scapula, the muscles of the shoulder girdle (trapezius, serratus anterior, rhomboids and pectoralis minor) are strengthened by isometric training.

Isotonic contractions

Isotonic exercise is classically defined as the movement of a load at constant resistance through an arc of motion.

In orthopaedic medicine, isotonic contractions are performed in the following situations:

- *In minor muscular tears* after the lesion has been prepared by gentle transverse friction (see p. 4). The contractions are carried out with the muscle in a position of maximal relaxation and with minimal resistance so that no tension falls on the healing breach. They begin as soon as possible after the injury and are preceded by an infiltration with local anaesthesia and by transverse friction. The idea is to promote movement between adjacent muscular fibres to prevent abnormal formation of adhesions because these can disturb the normal increase in breadth on contraction.
- *To strengthen weakened muscles* as in arthritis or after local or generalized immobilization. If passive movements are limited, say by the arthritis, the isotonic exercises should be performed within the painless range to avoid increase of synovial inflammation.
- *To strengthen muscles* so they can protect joints or inert structures from being painfully overstretched. One example is the strengthening exercises of the short plantiflexor muscles and the lumbricals of the foot in the treatment of splay foot and chronic metatarsalgia. Strong muscles that contract properly at each step will take most of the body weight and relieve the metatarsal heads.

Eccentric contractions

Eccentric (lengthening) actions are characterized by elongation of the muscle during active contraction.[122] Over the last fifteen years, eccentric exercises have been promoted as treatment strategies for tendinopathies and muscle strains, especially for the lower limb (quadriceps and Achilles tendinopathy). Greater forces can be produced during eccentric contraction than during concentric (shortening) actions,[123] which leads to positive changes in tissue structure and mechanical properties. One study found that Type I collagen synthesis increased after eccentric training in a group of twelve soccer players with unilateral Achilles tendinosis, offering a possible explanation for the mechanism of tendon healing.[124] Another study described a decrease in tendon thickness and normalized tendon structure measured by ultrasound in a group of subjects with chronic Achilles tendinosis who were trained using an eccentric training protocol.[125] Systematic review of the literature revealed that eccentric exercise may reduce pain and improve strength in subjects with lower extremity tendinosis,[126,127] but whether it is better than other forms of rehabilitation has yet to be determined.

Electrical contractions

In some circumstances a strong voluntary muscle contraction is not possible. Muscles can be (temporarily) paralysed or they are affected by severe injury. In such a situation, daily application of electrical stimulation (ES) may retard the loss of muscle strength or even improve it in already weakened musculature. The gains are not long-lasting, however, and electrical stimulations should only be used temporarily while awaiting neurological recovery. ES can then be replaced by a good exercise programme.

Coordination exercises

During the last decades it has become clear that rehabilitation should not be restricted solely to procedures that improve mobility, strength and endurance but also that functional exercises allowing a better coordination of particular muscle groups should be included in the rehabilitation programme. This kind of training is particularly important in the treatment of problems of instability (e.g. in shoulder, knee and ankle).

The ability to control the position of a joint during active motions (proprioception) and to produce a voluntary muscular contraction to stabilize the joint and/or to alter the joint position so as to prevent excessive joint displacements is referred to as *reactive neuromuscular control*.

Proprioception is a specialized sensory modality that gives information about extremity position and direction of movement: stretch-sensitive mechanoreceptors within skin, capsular ligaments and tendons (see Ch. 3) are activated by tension, thus producing a muscular contraction to protect these structures at the extreme of motion.[128] This type of afferent sensory feedback is extremely important in mediating muscular control over joints and thus preventing them from overstretch and (sub)luxation.[129]

Numerous authors have recommended the training of proprioceptive skills and proper muscular coordination to re-establish the reactive neuromuscular control abilities in the joints at risk and to create a functional joint stability.[130–134] Several techniques exist, from the see-saw block or tilting board in the treatment of functional instability of the ankle to the use of dual channel electromyography biofeedback systems in the treatment of patellofemoral dysfunction[135] and glenohumeral instability.[136] However, most functional training techniques used to re-establish the proprioceptive skills make use of *eccentric training*.[137] Eccentric activation refers to the situation in which the muscle–tendon unit is lengthened while active. Small weights and multiple repetitions of the movements are used.

Proprioceptive neuromuscular facilitation (PNF) techniques can also be used to gain or improve neuromuscular stability at a joint. PNF techniques may be of value in the prophylaxis of recurrent periarticular shoulder lesions. PNF may be defined as a method *promoting or hastening the neuromuscular mechanism through stimulation of the proprioceptors*. Hence, PNF refers to the improvement of flexibility through stimulation of the nerves and muscles internally. The technique involves the use of the principles of reciprocal innervation and the stretch reflex: as a muscle is passively or actively stretched, it is brought to a point of limitation before pain develops. This is the point at which the proprioceptive organs send a message to the central nervous system to terminate the movement before further elongation occurs. At this point the muscles being stretched (antagonist) are contracted for a few seconds

at an intensity of 50% of maximum. This contraction allows for the inhibition of the muscle spindles and Golgi tendon organs and a subsequent initiation of the stretch reflex of the stretch receptors. After releasing the contraction, the muscle is stretched to a new point of limitation and held for another few seconds. The interested reader is referred to Alter's *Science of Stretching*,[138] that offers over eight PNF techniques and exercises.

Injection and infiltration

Musculoskeletal disorders such as tendinitis, minor muscular ruptures, ligamentous sprains and arthritis can usually be treated by infiltration. For an optimal effect the product administered must he put directly into the lesion and not in its surroundings. The descriptive terms 'injection' and 'infiltration' are used; each has a well-defined meaning and expresses a different way by which the product is administered. In *injection* the tip of the needle is placed in exactly the right place and all the product is deposited at one single push, as is done in an ordinary intramuscular injection. This technique is mainly used for intra-articular and caudal epidural injections. Once the tip of the needle has been brought within the joint or the epidural space, the full amount of product is put in. Local administration of a drug into a structure, as in bursitis, tendinitis, tenosynovitis, tenovaginitis, lesions of a muscle belly and also in ligamentous problems, is usually performed by *infiltration*. In this, maximal beneficial effect is obtained when all the different areas within the lesion receive some of the product. This can only be achieved if the tip of the needle is displaced several times while injecting a small amount of the product at each point. An infiltration is therefore a series of injections, given at slightly different places, within the lesion. Although the aim of all infiltrations is the same, the specific technique may vary depending on the type of lesion and its location.

In orthopaedic medicine three types of product are used: local anaesthetics, corticosteroids and sclerosant solution. Each of these has its own specific indications, contraindications and side effects. Although, in each group, several agents are available, it is better to use only one as this leads to optimal therapy.

General principles

To obtain maximal benefit with minimal side effects the following general rules must be observed.

Exact diagnosis

Any treatment must reach the lesion. Infiltrations, like deep friction, need a diagnosis accurate within a millimetre or two and must be directed to the precise site of the lesion. The steroids used in orthopaedic medicine are all suspensions of insoluble particles, so their action is mainly confined to the place where they are administered. An exact preliminary diagnosis must be made, together with proper localization of the lesion.[139]

Choice of product

Many different products are used in musculoskeletal disorders. Local anaesthetics and corticosteroids are most frequently used but occasionally a sclerosant solution containing phenol, glycerol and dextrose is needed. The type of the product, its concentration and volume depend on the structure affected, the nature of the lesion, the degree of inflammation and other additional elements such as age, activities and the general condition of the patient. Of main importance is increased liability to – or the presence of – general or local infections and allergy to the products.

A lesion of the muscle belly can be treated by infiltration of a local anaesthetic, usually procaine, whereas steroid is never used. Musculotendinous insertions do not respond to any product and therefore are never therapeutically infiltrated but are best treated by deep transverse friction. Procaine is of no curative value in tenoperiosteal insertions or periosteal attachments of ligaments; for these, steroid is used or friction is given. In lesions of the body of a tendon, steroid is never infiltrated into the tendon itself, but is put at the surface along the tendon.

For optimal results the appropriate agent at the indicated dose and concentration must be used. For example, 20 mg of triamcinolone at a concentration of 10 mg/ml means that a total volume of 2 ml is administered. This is not the same as 0.5 ml of a 40 mg/ml suspension, as the steroid administered in this way will be concentrated over a much smaller area and may not be sufficient in volume to reach the whole lesion. Moreover, a strong concentration of steroid spread over too small an area may cause an increased risk of tendinous or ligamentous rupture.

Equipment

If a certain amount of product is indicated, it should always be put into a syringe of the corresponding volume. If this is not done, infiltration may fail because of counterpressure offered by the tissue. Care should be taken to fit the needle firmly to the syringe because an infiltration into a ligamentous or tenoperiosteal insertion may demand considerable pressure on the plunger, which could lead to detachment of the syringe.

The needle to be used must be of appropriate length and be as thin as possible. These characteristics are always indicated throughout the text in subsequent chapters. An appropriate needle enables the therapist to recognize the type of tissue by the resistance it offers as the needle penetrates the structure: for example, ligaments and tenoperiosteal junctions have a totally different resistance from that of a muscle belly. Moreover, the thinner the needle the easier it is to recognize differences in resistance on the plunger during infiltration. Both types of resistance are important in giving a good indication of the localization of the tip of the needle.

The main characteristics of the needle types referred to in this book are given in Table 5.3.

Position of the patient

Before any attempt is made to infiltrate, the patient is positioned in the way that renders the lesion most accessible. For

Table 5.3 Main characteristics of needles used

Needle type*	Gauge		Length	
	G	mm	inches	mm
Preparation	19	1.1	$1\frac{1}{2}$	40
2.5 cm	25	0.5	1	25
3 cm	22	0.7	$1\frac{1}{4}$	30
4 cm	21	0.8	$1\frac{1}{2}$	40
5 cm	22	0.7	2	50
7 cm	20	0.9	$2\frac{3}{4}$	70
Spinal	22	0.7	$3\frac{1}{2}$	90

*Terminology used in this book.

each injection, the ideal positions from which to start are presented in later chapters; positions are usually the same as for deep friction. For intra-articular injections, different positions have been described. We present those which are the most simple to perform and which offer the least risk in relation to other neighbouring structures. The landmarks required and sometimes also the affected structure should be palpated and, if necessary, should be marked on the patient's skin.

Aseptic care

Careful aseptic precautions are always necessary in order to avoid, for example, a suppurative arthritis. Aseptic techniques include:

- Hand antisepsis
- Skin antisepsis at the site of insertion
- Disinfecting the rubber septum with alcohol prior to piercing it
- Using a new sterile syringe and sterile needle to draw up the fluid while preventing contact between the needle and the hands
- Removing the needle and using a fresh one for the injection
- Using a no-touch-technique: neither the tip of the needle, nor the skin at the site of insertion should be touched after skin antisepsis
- Using gloves? Gloves can only be used if an assistant handles the non-sterile packages and vials and prepares the syringes. In our opinion, the use of gloves does not add to aseptic safety, if the rules mentioned above are strictly followed.

Technique of infiltration

Two major techniques are considered here: dynamic infiltration, in which the product is injected during movement of the needle, and a static infiltration, in which the product is administered with the needle at rest.

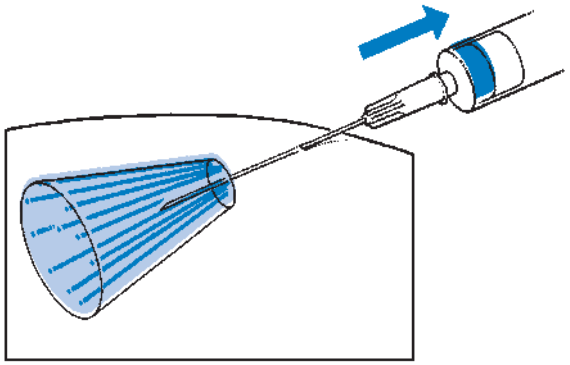


Fig 5.7 • Conewise infiltration.

Dynamic infiltration

This is used mainly when large amounts of product have to be administered in extensive lesions. Three different techniques are used, mainly conewise, fanwise and cylindrical infiltration. A further technique is used for static infiltration.

Conewise infiltration

This is the usual technique for a lesion of a muscle belly. The limb is brought into such a position that the muscle is well relaxed. The tender part is pinched between the thumb and fingers and the needle is inserted obliquely in between them until its tip passes beyond the farthest edge of the lesion. Some of the product is now administered while the needle is withdrawn until the tip is at a point just beyond the nearer border of the lesion. It is then reinserted at a slightly different angle and more product is infiltrated while the tip is again withdrawn (Fig. 5.7). This is repeated several times until the entire lesion has received some of the product. During the whole procedure, the fingers that hold the lesion verify and control the infiltration.

Cone infiltration is also used in bursitis lying within the reach of the fingers. After the lesion has been carefully defined, the needle is directed to the centre of the tender area and towards the underlying bone. The site of tenderness usually cannot be kept between the fingers. The product is infiltrated by a series of partial withdrawals and reinsertions at a slightly different angle.

Fanwise infiltration

This technique is similar to conewise infiltration but is two-dimensional. It is used in subacromial bursitis. The needle is directed towards the centre of the deep part of the subdeltoid bursa and to its full length. By a series of partial withdrawals and reinsertions in a horizontal plane, to the left and to the right from the centre (Fig. 5.8), the full amount of product is applied during withdrawal.

Cylindrical infiltration

A cylindrical infiltration is used to place the agent along the surface of a tendinous body. The tendon is first stretched to provide a stiff flat surface. The needle is tangentially inserted along the tendon between the tendon and its sheath, until the tip reaches the far edge of the lesion. During partial withdrawal as the needle traverses the surface of the tendon, constant pressure on the plunger maintains flow of the suspension. The

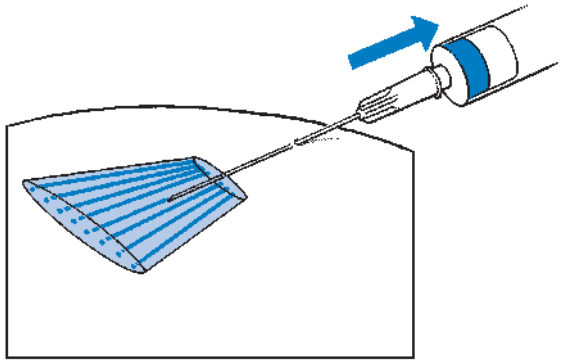


Fig 5.8 • Fanwise infiltration.

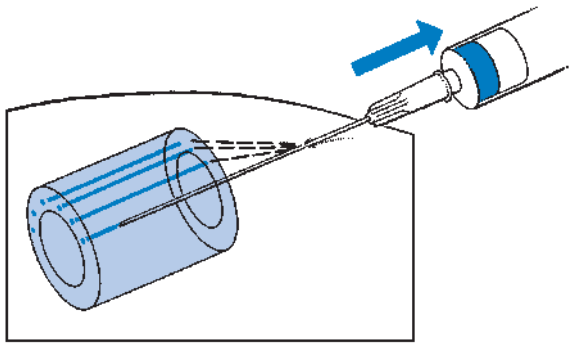


Fig 5.9 • Cylindrical infiltration.

needle is then reinserted parallel to its first position but 2 mm to one or the other side and the manœuvre repeated (Fig. 5.9). This is repeated four or five times until the full amount has been given.

Static infiltration

This technique is mainly used to put a small amount of fluid into a well-delineated area. It is indicated for tendinous insertions and ligamentous attachments to bone. These offer considerable resistance to depression of the plunger. Hence the smallest syringe must be used to provide enough purchase to force the product into the structure. When the tip of the needle lies at the exact site, a typical counterpressure is felt during insertion, just before its point hits the bone. Now a few droplets are injected with the tip of the needle in bony contact. The needle is then five to ten times partly withdrawn and inserted at slightly different angles and depths (Fig. 5.10) until the full amount has been administered and the whole lesion has been treated. The technique is sometimes referred to as *peppering*. During the entire infiltration a typical resistance is felt, much more marked than during infiltration of a muscle belly or a bursa, where resistance is minimal.

Follow-up and after-care

For maximal benefit, all infiltrations in any part of the contractile structures are followed by relative rest for 1–2 weeks. If full activity is continued without this rest, relapse is likely. When tendinous structures are infiltrated by steroid there is a second reason for rest, in that the agent may temporarily weaken the tendon.

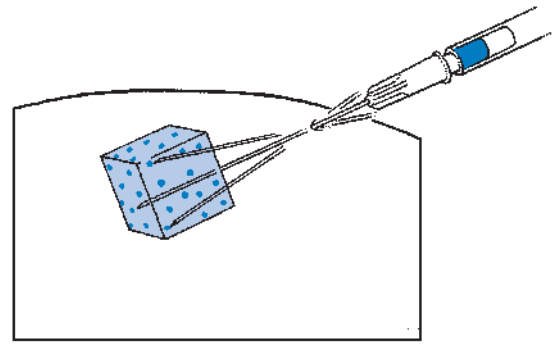


Fig 5.10 • Static infiltration.

The patient should be reassessed after 14 days and, if pain still remains, a second infiltration is given. A partial rupture of a muscle belly offers an exception to this in that it is infiltrated only once, followed next day by deep transverse friction, active exercises and electrical stimulation in the position of maximal relaxation of the muscle. Stress at the site of healing is avoided for about 3 weeks.

After infiltration into a sprained ligament, the joint is immediately used as fully as possible, allowing movements within the limits set by pain but avoiding stretching of the damaged structure. This leads to good functional results in a short period of time.

The advantage of this type of treatment is its rapid effect but on some occasions it may lead to recurrence in tendinous lesions. Recurrences are best treated by deep transverse friction.

Local anaesthetics

Cyriax first started using procaine purely for diagnosis. If the suspected lesion is infiltrated by local anaesthetic, the pain on functional testing should disappear almost immediately, so confirming the diagnosis. He was most surprised when some of those initial patients reported persistent improvement some time later. He continued to use procaine because he felt that the therapeutic results were better than with the alternative local anaesthetics. Nowadays local anaesthetics are still used for both diagnostic and therapeutic purposes.

Because prolonged action of the anaesthetic is never needed in orthopaedic medicine, adrenaline (epinephrine) is not added. In addition, vasoconstriction restricts the spread of the agent so diminishing any beneficial effect.

Types of local anaesthetic

Two major types of local anaesthetic are available: procaine and those belonging to the amide group (e.g. bupivacaine, lidocaine (lignocaine)) (Table 5.4).

Table 5.4 Local anaesthetics and their effects[141]

Name	Strength	Toxicity	Maximum dose (mg)	Latency (min)	Duration (min)
Procaine	1	1	500	5–10	45
Lidocaine (lignocaine)	4	2	200	2	60
Prilocaine	4	1, 5/4	400	2	60
Mepivacaine	4	2	350	1	100
Bupivacaine	16	8	150	2–5	360

Procaine

Procaine is an ester of para-aminobenzoic acid and was first synthesized in 1904 by Einhorn. Until 1943 it was the only anaesthetic available.[140] It is quickly and locally metabolized by an esterase, giving rise to the acid itself, which may act as an allergen. Its effect begins after 5–10 minutes and lasts approximately 45 minutes. The maximum dose used is 250 mg, which corresponds to 50 ml of a 0.5% concentration.

Although classical texts emphasize the hazard of allergic reactions, we have used procaine several times daily for over 20 years without any problems. Cyriax estimated the risk as 1 per 500 000 administrations.

The usual concentrations employed are 0.5 and 2%. Indications for 0.5% procaine are lesions of muscle bellies, most cases of chronic bursitis and caudal epidural injection. Procaine 2% is used to dissolve calcifications in tendons and bursae, and in sinuvertebral nerve blocks.

Amides

Newer local anaesthetics are the amides. They are metabolized in the liver and seldom give rise to allergic reactions. We use lidocaine (lignocaine) and prilocaine to anaesthetize only certain structures so allowing other interventions. For this reason, they are used to anaesthetize the skin and the intercornual ligament before inserting an epidural needle, and for skin and tenoperiosteal anaesthesia before a tenotomy in type II tennis elbow. Because their local anaesthetic effect is stronger and faster acting than that of procaine, they are also used for diagnostic infiltrations around nerves and in tendons, ligaments and bursae.

In countries where procaine is not available, or there is patient allergy, bupivacaine 0.125% is an alternative. The maximal dose used is 60 mg, which corresponds to about 50 ml of a concentration of 0.125%. The immediate effect starts after 2–5 minutes and continues over 3–6 hours.

Side effects

Side effects of local anaesthetics are traditionally divided into three groups:

- Psychogenic reactions
- Toxic side effects
- Allergy.

Psychogenic side effects

These minor side effects are mainly caused by somatosympathetic reactions on pain and fear and are not related to the drug as such. Nevertheless the symptoms may be quite similar to those of real toxic reactions: pallor, cold sweating, dizziness, nausea, yawnings, palpitations and vasovagal collapse with syncope all may be present.[142] They should be taken seriously because if the patient is not placed supine quickly, cerebral hypoxia may follow, with unconsciousness, tremor and convulsions.

To avoid these effects, all patients who require an infiltration should at least sit or lie down and be given a full explanation of what is to happen. If symptoms do occur, the patient is immediately placed in Trendelenburg's position (head and thorax low, legs up) and oxygen given. The patient usually improves promptly after 1–2 minutes.

Toxic reactions

These are dose-dependent side effects related to the amount of product that reaches the blood circulation.[143] They may be the result of unintentional intravascular injection, absolute overdose, swift absorption or delayed elimination of the drug.[144]

If local anaesthetics are used at the indicated dose, which is usually less than half of the maximum dose allowed, and in a proper way – taking care during the administration that it is not directly intravascular – toxic reactions should never occur. However, if they do occur during an injection, administration must stop at once.

Types of reaction

Toxic side effects can be divided into two groups. They may be related to the central nervous system or to the cardiovascular system or to both.

Effects on the central nervous system

Although local anaesthetics may provoke stimulation or depression of both cortex and medulla, stimulation is the more frequent but is less severe than depression. Normally the depression period is preceded by a stimulation stage, but it may come on at once without a prior stage of excitement.[145]

Some *stimulation* of the cerebral cortex and the upper centres may sometimes occur even with a low dose. It is

characterized by anxiety, excitement, logorrhoea, hypertension, headache, dizziness, tinnitus, diminished hearing, disturbed vision, metallic taste, muscular fasciculations around the mouth and tremor. None of these symptoms is severe as such, but they are all signs of the presence of a toxic reaction that could lead to cardiovascular collapse. Further stimulation may be followed by convulsions, which indicate that severe cortical stimulation is present and may progress to postconvulsive depression.[145] Stimulation of the emetic centre causes nausea and vomiting. Stimulation of the cardiovascular centre in the medulla may cause tachycardia and hypertension; respiratory stimulation is characterized by an increased depth and frequency of breathing.

The first feature that draws attention to *depression* of the cortex is sleepiness, together with dysarthria and a feeling of coldness. If injection is not stopped at once and the necessary measures instituted, the outcome may be coma and death. Depression of the medulla may suppress vasomotor control resulting in pallor and hypotension, later followed by syncope and cardiac arrest. Respiratory depression is first characterized by irregular breathing with periods of apnoea and dyspnoea and often cyanosis. Finally, total respiratory arrest may result. A depressive effect is far more dangerous than is stimulation.

Effects on the cardiovascular system

A direct peripheral effect on the blood vessels causes vasodilatation, leading to hypotension. Local anaesthetics have a depressive effect on the myocardium, which can lead to bradycardia, arrhythmia and cardiac arrest. In patients with already weakened myocardium, heart failure may result.

Local anaesthetics can alter the blood pressure in different ways. Stimulation of the CNS leads to hypertension, depression to hypotension. Hypotension may be intensified via a direct vasodilatory effect on the blood vessels and by direct myocardial depression. Furthermore, in lumbar epidural injection, hypotension may be due to blockade of the sympathetic nerves.[146]

Clinical appearance

Toxic side effects may clinically appear in two ways:[147]

- *A delayed reaction* may occur within 5–30 minutes after the infiltration. This is the most frequent reaction and is due to slow absorption until a toxic level is reached. It usually first gives rise to stimulation of the cortex before respiratory symptoms and cardiovascular collapse occur. For this reason the patient should always remain under supervision during the first half hour following an injection.
- *An immediate reaction* may occur within seconds to minutes of the administration, with all toxic reactions coming on at once. It is usually the result of intravascular injection or of swift absorption. Collapse may occur very quickly and death may follow rapidly if resuscitation is not begun at once.

Treatment

The most important measures, even if only minor signs of CNS or cardiovascular involvement are present, are to stop further infiltration at once and to administer oxygen. These may be sufficient to stop further development towards more severe conditions. The rest of the treatment should be in relation to the degree of toxic reaction:

- *Fear or anxiety attacks* are best treated by intravenous diazepam (5–10 mg).
- *Cerebral signs* – tremor and convulsions – require immediate administration of 100% oxygen via a mask. Small intravenous doses (5–10 mg) of diazepam may be given. Barbiturates and short-acting muscular relaxants should only be used by the experienced and should always be preceded by the administration of oxygen.[144]
- *Hypotension:* the patient is put into the Trendelenburg position. If this is not sufficient, administration of vasoactive drugs such as dopamine or noradrenaline (norepinephrine) may be required.[143] In circulatory depression, intravenous fluid must also be given. Bradycardia can be treated by atropine.
- *Cardiac arrest:* oxygen and external cardiac massage are given.
- *Respiratory depression* is treated by oxygen and artificial respiration (intubation) (see Box 5.5).

Allergic (anaphylactic) reactions

Side effects not related to dose can occur even after the administration of small amounts of the agent. Previous exposure and sensitization to the active product or preservatives (methylparaben) or any other accessory material present is an essential precondition.

Two types of allergic reaction may occur: an anaphylactic reaction, which may be life threatening, and a local reaction

Box 5.5

Summary of treatment of side effects of local anaesthetics

Always:
- Stop further infiltration
- Give oxygen at once

Tremors and convulsions:
- 100% oxygen via mask
- Diazepam i.v. 5–10 mg

Vascular collapse:

Hypotension:
- Trendelenburg position
- Vasoactive drugs i.v. (dopamine, isuprenaline hydrochloride, adrenaline (epinephrine))
- Fluid i.v.

Bradycardia:
- Atropine

Cardiac arrest:
- External cardiac massage

Respiratory depression:
- Give oxygen
- Artificial respiration (intubation)

presenting as dermatitis from local contact with the skin. The latter is usually encountered only in professionals who use the product, rather than in patients.

Although allergic reactions are frequently mentioned in relation to local anaesthetics they are in fact rare.[147] According to Cyriax, anaphylactic reactions occur in 1/50 000 procaine infiltrations, though they are more frequently encountered with procaine than with the amides.[144,146,148] Since an anaphylactic reaction may be very dramatic, with a mortality rate of about 3.4%, one must always be aware that it is possible.[149] Before local anaesthetics are administered the patient must always be asked for evidence of an allergic constitution. If a patient claims to be allergic to local anaesthetics, a careful and detailed history must be taken because 99 out of 100 reactions are due to toxic or psychogenic side effects and not to an allergy as such.[147] An intradermal test with a small amount of agent can be useful but is not 100% reliable.[147]

An anaphylactic reaction may come on immediately after the injection or some time later, up to about 30 minutes. It is the immediate type which is often dramatic. The initial feature of an anaphylactic reaction is often *flushing* occurring within 20 minutes after administration. This may be quickly followed by dyspnoea due to *bronchiolar constriction* and localized *oedema of the larynx and glottis*. It may end in *respiratory obstruction* which is the main cause of death.

The respiratory symptoms are accompanied by vasodilatation which leads to *hypotension* and *shock*. Occasionally other anaphylactic reactions such as *urticaria* and *angioneurotic oedema* may also present immediately, but are far less dramatic.

Treatment

Treatment is different for immediate and delayed allergic reactions. The treatment is outlined in Tables 5.5 and 5.6.

Immediate reaction

Even when only minor signs of anaphylactic reaction are present 0.3–0.5 ml of *adrenaline (epinephrine)* 1/1000 (0.3–0.5 mg) is immediately administered by the subcutaneous route to provoke vasoconstriction, bronchiolar dilatation and resorption of oedema. Since adrenaline (epinephrine) is broken down quickly, it should be repeated every 20 minutes. Subcutaneous administration of adrenaline can only be done if the general circulation is intact. When blood pressure is too low it must be carefully administered intravenously. In this event 3–5 ml of adrenaline (epinephrine) 1/10 000 must be administered in repeated shots of 1 ml (0.1 mg) until effect has been attained.[150] Small repeated shots are necessary to avoid ventricular fibrillation. Some also advise inhalation of a 1/10 000 adrenaline (epinephrine) solution via aerosol in case of laryngeal oedema. *Steroids* inhibit allergic reactions but act too

Table 5.5 Classification of anaphylactic reactions of increasing severity and their treatment

Severity	Symptoms	Treatment
I	Urticaria Red conjunctivae Fever	Antihistamine
II	Hypotension Dyspnoea Tachycardia Nausea Diarrhoea	Trendelenburg position 100% oxygen Adrenaline (epinephrine) Antihistamine Corticosteroid Aminophylline
III	Shock: circulatory collapse and angioneurotic oedema Life-threatening spasms of the bronchi	Trendelenburg position 100% oxygen Intubation Infuse: Adrenaline (epinephrine) Antihistamine Corticosteroid Aminophylline
IV	Cardiac arrest Respiratory arrest	Heart massage Artificial breathing

Table 5.6 Medication used in anaphylactic reactions

Drug	Dose	Notes
Adrenaline (epinephrine)		
Adults		
Normal blood pressure	1/1000, 0.3–0.5 ml s.c.	To be repeated every 20 min
Severe hypotension	1/10 000, 3–5 ml i.v.	1 ml per shot
Children	1/1000, 0.01–0.03 ml/kg s.c.	
Antihistamine (clemastine)	2 mg i.m./i.v.	Every 6 h for up to 24 h
Corticosteroids (dexamethasone)	4–8 mg i.v.	Every 6 h for up to 24 h
Aminophylline		
Adults, initial dose	240 mg i.v.	
Children		
Initial dose	5 mg/kg i.v./i.m.	
Follow-up dose	0.4 mg/kg/h i.v.	

slowly in this situation. Oxygen should also be given but may be of little value if the airway is severely obstructed. In the latter case, *aminophylline* should also be injected.

The patient should remain under supervision during the first few hours after the anaphylactic reaction.

Delayed reaction

If the allergic reaction comes on only after 30 minutes, administration of an antihistamine drug is sufficient.

Corticosteroids

The adrenal corticosteroids are classified into two groups: mineralocorticoids such as aldosterone, with sodium-retaining activity, and glucocorticoids, which influence the intermediary metabolism (nitrogen catabolism, increased glucogenesis) and have a strong anti-inflammatory and anti-allergic effect. The ability to suppress inflammation has made the glucocorticoids very useful but also potentially harmful. Administered in high doses, they may cause Cushing's syndrome.[151]

All corticosteroids used in orthopaedic medicine are glucocorticoids. The first was hydrocortisone, extracted from the adrenal cortex by Kendall in 1936, and first intra-articularly injected by Thorn in 1950.[152] Since then many other steroids have been synthesized. Efforts to increase the anti-inflammatory effect and to diminish the influence on metabolism have remained largely unsuccessful so far. Thus, unnecessary use should be avoided.

The ideal steroid should meet the following criteria: little discomfort to the patient during and after injection, a low level of absorption into the systemic circulation (if absorption does occur, it should be slow), together with a prolonged local effect without general and local side effects.[153] Some preparations are formulated specifically for local use and are available as a crystalline suspension. The less water soluble they are, the less they are absorbed into the general circulation. As a consequence they have a more prolonged local effect and fewer general side effects. Intrasynovial administration has the advantage of a maximal local benefit with minimum systemic side effect.[154]

The agent we use most often is triamcinolone acetonide, in a concentration of 10 mg/ml. Throughout this book, this is the product and concentration intended, unless indicated otherwise in the text. It has a mean duration of activity of about 14 days, whereas triamcinolone hexacetonide has more prolonged activity.[155] Normal doses used are 5 mg for small joints, 20 mg for medium-sized joints and 50 mg for the hip and knee.

Therapists who prefer another type of corticosteroid should always administer the dose equivalent to the one indicated in the text (see Table 5.7).

Table 5.7 Corticosteroids

Product	Anti-inflammatory effect
Hydrocortisone	1
Cortisone	0.8
Prednisone	2.5
Prednisolone	4
Methylprednisolone	4
Triamcinolone	5
Betamethasone	28
Dexamethasone	28
Beclometasone	40

Effects of local corticosteroids

Corticosteroid injected locally has a local anti-inflammatory effect due to stabilization of the lysosomal membrane with decreased liberation of cytotoxic enzymes. Steroids also impair the proliferation of fibroblasts and decrease the rate of production of mature collagen. They decrease plasma fibrinogen as well and have an increased fibrinolytic activity.[156] Furthermore, corticosteroids reduce oedema formation and the escape of plasma protein across the capillary membrane, and diminish the number of leukocytes in exudate at an inflammatory site. All these effects lead to reduction of pain and fibrosis.[157–161]

When injected into a joint, steroid is partly broken down by enzymes from the synovial membrane and is partly resorbed into cells of the synovial fluid and cells of the synovium. A small amount enters the general circulation.

Indications for local corticosteroids

Joints

Beneficial effects occur in traumatic arthritis, monoarticular steroid-sensitive arthritis, rheumatoid arthritis, crystal-induced arthritis (gout and pseudogout), ankylosing spondylitis, lupus erythematosus and psoriasis.[153] Steroids are ineffective in Reiter's disease (Cyriax:[3] p. 52).

Monoarticular steroid-sensitive arthritis is of spontaneous onset without any other signs of rheumatological disorders and resolves equally spontaneously over months or years. The joints affected are the glenohumeral, the elbow, the knee, the hip, the ankle and the temporomandibular. The treatment of choice is intra-articular steroids. Usually, the joint must be kept under continuous anti-inflammatory influence for some time. Hence a specific sequence of injections must be observed.

Improvement is subjectively felt by the patient in that pain and stiffness diminishes, and objectively shown by a decrease of local heat and effusion together with improved function. Patients should be warned against overactivity at weight-bearing joints for the first days after injection to avoid further destruction of cartilage.[162] Moreover, the efficacy of an injection is greater with relative rest.

In arthrosis, lasting improvement is not to be expected.[163] Some authors even suggest, although this has never been scientifically proven, that it may accelerate the degenerative process.[162] If synovitis complicates arthrosis, intra-articular injection may well be beneficial.

Tendons

Steroids may be indicated in tendinitis, tenosynovitis and tenovaginitis. It is often used in tendinitis of the supra- and infraspinatus tendons and the subscapularis tendon at the shoulder.

The same applies for type II tennis elbow and for golfer's elbow at the tenoperiosteal insertion. In the lower limb, steroid injection is mainly used in supra- and infrapatellar tendinitis and for tendinitis of the peroneal tendon.

To avoid rupture, infiltration should be at the tenoperiosteal insertion or between tendon and tendon sheath, never into the body of the tendon.[162,164–167] Ruptures have been reported mainly after intratendinous infiltration with agents of high anti-inflammatory effect or at too high a concentration or when too frequently administered. When the rules are respected as set out in this chapter, there should be no danger of rupture.

Ligaments

In the acute stage – within 24 hours of the injury – of a ligamentous sprain at the ankle or tarsus or of the medial and lateral collateral ligaments at the knee, a steroid infiltration quickly helps but should only be done at the ligamentous insertions. Because the inflammatory reaction ceases shortly after administration, the ligamentous lesion can heal in the presence of movement. This leads to better functional results and to absence of adhesions, which are later often the reason for chronic pain.

In sprain of the cruciate ligaments of the knee, steroid infiltration is the only possible treatment, no matter what the stage of inflammation. Steroid infiltration is also an important part of the treatment in sprains of the radial and ulnar collateral ligaments of the wrist and in plantar fasciitis.[168]

Bursae

Depending on the type and location of bursitis, steroid is used at once or after an infiltration with procaine has been found to be unsuccessful. Pain, local tenderness and functional impairment are all reduced.[169]

Nerves

Compression of the median nerve in the carpal tunnel of the ulnar nerve at the ulnar sulcus or of any nerve root by an unreducible disc protrusion all usually benefit from an infiltration with triamcinolone around the nerve.[170]

Local side effects

Unwanted side effects of local corticosteroids are minimal compared with those of even low doses of oral steroids.[153] They are traditionally divided into local and general side effects.

Musculoskeletal side effects

There are a number of musculoskeletal side effects of local corticosteroid injection.

Iatrogenic infectious arthritis

This complication is the most feared although it is seldom encountered. The incidence varies from author to author: it follows between 1/1000 and 1/40 000 injections.[153,171] The usual organism is *Staphylococcus aureus*; less often, Gram-negative organisms are involved.[172] The condition should not be regarded as a real side effect caused by the steroid but is due rather to inadequacy of aseptic care or contamination, chiefly occurring during preparation of the syringe. Patients who suffer from diabetes, or who are receiving oral steroids or are immunosuppressed (leukaemia, AIDS, drug abuse) are more vulnerable.[173] Suppurative arthritis may often lead to therapeutic difficulties, and death may follow.[174]

Obviously this complication should be avoided by good antiseptic care, especially when the injection is made into a high-risk patient and when infiltrating in the neighbourhood of a joint or in the joint itself. Although skin lesions in psoriasis are highly colonized by bacteria, it has not been documented whether intra-articular injections given through such plaques increase the likelihood of an infectious arthritis.

Destruction of joint cartilage and evolution of steroid arthropathy

It has been suggested that intra-articular steroids may hasten the process of arthrosis by a deleterious effect on cartilage, leading to changes that closely resemble a Charcot-like neuroarthropathy. Steroid depresses the synthesis of collagen and proteoglycans, which may result in a loss of stiffness of the cartilage. In non-weight-bearing joints this has little or no effect. In weight-bearing joints it may result in fissure formation at the surface of the cartilage and cystic degeneration in the middle zone.[175–178] However, rapid progression to degeneration has not been proved with certainty.[171,179] Cases have been reported of multiple intra-articular injections in some joints not leading to any abnormalities on later radiography.[180,181] Often effects of local steroid cannot be differentiated from those caused by general administration of the product,[182] prompting the question whether it is a local or general effect. Because it is difficult to differentiate the possible destructive effects of the steroids from the natural progression of the osteoarthrosis, even though the risk may appear small, it is a good habit to avoid frequent injections. Moreover, injection into cartilage should never be done. When too much counter-pressure on the plunger is present, the tip of the needle must be replaced. It is also wise to rest weight-bearing joints for 24–48 hours after an injection.[139]

Flare up of crystal-induced arthritis

During the first 48 hours after an intra-articular injection of a crystalline suspension, a synovitis may flare up as response to the crystals. The mechanism is the same as in gouty arthritis. An equally painful inflammatory reaction is occasionally encountered after a local tenoperiosteal infiltration. Normally it disappears within 12–48 hours. If the reaction is prolonged, iatrogenic infection must be excluded.

Rupture of ligament or tendon

Tendon ruptures have been described after one or multiple infiltrations.[166,183,184] Infiltration of steroids into acutely injured ligaments in the rat significantly impaired the healing process relative to a non-injected ligament at 10 days and at 3 weeks after injury.[185] However, after 6 weeks the tensile strength (the ultimate stress) of the ligaments that had been injected with the steroids returned to a value that was equal to that of the controls that had not received an injection.[186] Although other studies have not confirmed these findings,[187,188,189] infiltration into the tendon body should never be done. In tenosynovitis, steroid can safely be infiltrated between tendon and tendon sheath and at the tenoperiosteal insertion, although multiple repetitive infiltrations must be avoided.

Calcification

Punctate calcifications of joint capsules and pericapsular calcifications are common after intra-articular and periarticular injections. Steroid paste can be found on the surface of infiltrated tendons. These observations are not clinically important.

Neurovascular complications

Direct intrafascicular injection of steroid in peripheral nerves may provoke permanent damage, whereas extrafascicular injection does not appear to be harmful.[190] The mechanism of injury is multifactorial and can be related to direct trauma with the needle, to ischaemia and to a neurotoxic effect of the steroid or of the buffer agents and additives such as polyethylene glycol and benzyl alcohol. The first features are severe radiating pain and numbness or paraesthesia in the sensory territory of the nerve, together with motor deficit. The pain usually responds poorly to narcotics and may persist for many years.[191] As to the steroid itself, triamcinolone hexacetonide and hydrocortisone are most hazardous and dexamethasone causes minimal damage, whereas triamcinolone acetonide is intermediate.[190,192]

Damage to the nerve should be treated conservatively for about 12 weeks. If the neurological deficit does not improve after this time, neurological consultation is necessary.

Dermatological side effects

Fat necrosis, atrophy of skin and subcutaneous tissue and depigmentation of the skin may be encountered. These are due to a faulty injection technique or to leakage of the product after an intra-articular injection. The latter occurs often in smaller joints if the volume injected is excessive. Consequently, if resistance during the injection increases markedly, the injection should be stopped.

General side effects

Although injected intra-articularly or into the soft tissues, corticosteroids do enter the circulation. The causes are leakage and absorption. Therefore general side effects depend on the administered dose, the frequency of injections, the number of joints injected and the aqueous solubility of the agent. The greater the aqueous solubility, the higher the absorption rate. Less soluble microcrystalline suspensions remain within the joint for longer. Administration of a given dose equally divided between two joints produces more general side effects than if the same amount was injected into a single joint – the result of a larger absorptive surface.[172]

The majority of the more severe systemic effects can be avoided if no more than two joints are treated at the same time, using a maximum of 40 mg triamcinolone and allowing an interval of 1 month between two injections.[193] If treatments are performed as recommended in this book general side effects are extremely rare.

Endocrinological side effects

There are five categories of potential endocrinological side effects.

Disturbance of hormonal equilibrium

This can result in either hyperglycaemia or suppression of the adrenal cortex via depression of plasma cortisol levels:

- *Hyperglycaemia*: glucocorticoids have an important influence on the intermediary metabolism. They increase circulatory glucose via stimulation of gluconeogenesis and decrease the intracellular use of glucose. Therefore it is wise to advise diabetic patients to check their blood sugar more closely for the first few days after the use of steroid.
- *Suppression of the adrenal cortex via depression of plasma cortisol levels*: small doses of steroid may provoke some suppression of the adrenal cortex.[194] This seems to occur not only when steroids are administered orally but also after intra-articular injections.[195] Prolonged administration renders the adrenals atrophic and provokes suprarenal insufficiency with symptoms such as hypotension, anorexia, fever and generalized joint or muscular pain.

Iatrogenic Cushing's syndrome

The full syndrome with weight gain, depression, insomnia, amenorrhoea, diminished libido, thinned skin, muscular weakness, polyuria and polydypsia is seldom encountered after local use of steroids.[180] Occasionally facial hirsutism and acne may be seen.

Flushing

In the first few days after an intra-articular injection, some patients suffer from erythema and warmth in face, neck and chest. This is a totally benign but rather frequent sensation which is more common after the use of triamcinolone.

Shaking and chills

Very rarely patients suffer from chills and shaking after the use of steroid, a reaction that normally abates within 24–48 hours.[154]

Interference with the menstrual cycle

In female patients repeated injections with steroid may lead to dysfunctional uterine bleeding. Steroids may also interfere with hormonal contraceptives. There is no evidence for a teratogenic effect.[196]

Musculoskeletal side effects

In addition to the local musculoskeletal side effects already mentioned, there can be systemic musculoskeletal effects.

Osteoporosis and increased risk of fractures

It is difficult to estimate the exact incidence of osteoporosis provoked by corticosteroids. Nevertheless, special care should be taken when administering them to post-menopausal females because of the increased likelihood of these side effects.[193,197]

Steroid myopathy, spontaneous tendon ruptures and aseptic necrosis

Any of these may occur, although they are all very rare.[198]

Immunological side effects

Suppression of the inflammatory mechanism sometimes leads to a disappearance of symptoms in non-injected joints. Chronic use of steroids may increase the liability to infections.

An allergic reaction to steroids is not expected because they all have immunosuppressive and anti-inflammatory activity.

However, a number of well-documented allergic anaphylactic (type I) reactions to corticosteroid medications have been reported.[199] Recent reports have warned of potential (though rare) anaphylactic reactions after intra-articular or intralesional injection of triamcinolone acetonide.[200–202]

One recent study indicates that the triamcinolone acetonide component responsible for the patient's reaction is the suspending agent carboxymethylcellulose.[203] It is therefore suggested that care be taken and component testing eventually considered when patients experience allergic-type reactions to drugs.

Miscellaneous side effects

Central nervous system, gastrointestinal and ophthalmological side effects have been reported:

- Prolonged use of steroids may provoke changes in personality and mood.
- In patients who have previously suffered from peptic ulcer and pancreatitis, steroids should he cautiously used.
- After long-term use of steroids some patients may complain of worsening of vision. This may be due to glaucoma or cataracts.[204]

Indications for and contraindications to local corticosteroids

The indications for corticosteroid injection are given in Box 5.6. Contraindications may be divided into absolute and relative (Box 5.7).

Viscosupplementation

Over the past 2 decades, the use of intra-articular viscosupplementation in the non-operative management of patients with osteoarthritis has become quite popular. Viscosupplementation refers to the concept of synovial fluid replacement with intra-articular injections of hyaluronic acid (HA) for the relief of pain associated with osteoarthritis.[205] HA is a high molecular weight polysaccharide and is an important component of synovial fluid and extracellular matrix of articular cartilage. It contributes to the elasticity and viscosity of synovial fluid. HA acts as a fluid shock absorber and it helps to maintain the structural and functional characteristics of the cartilage matrix.[206,207] It also inhibits the formation and release of prostaglandins, induces proteoglycan aggregation and synthesis, and modulates the inflammatory response.[208] Any degradation of HA is therefore associated with increased vulnerability to articular cartilage damage. Treatment with HA is indicated for patients who are functionally limited due to osteoarthritic pain and who have failed to respond adequately to standard treatment options and wish to postpone or avoid surgery. Viscosupplementation is currently accepted as a useful therapeutic modality in treating patients with osteoarthritis of the knee or hip,[209,210] with beneficial effects on pain, function and patient global assessment.[211] Recently, promising results for viscosupplementation in grade I and II ankle osteoarthritis have also been published.[212,213]

Box 5.6

Indications for corticosteroid injection

Tendons

- Tendinitis at a tenoperiosteal junction
- Tenosynovitis/tenovaginitis: injection between tendon and tendon sheath, never into tendon

Joints

- All non-supportive types of synovitis
- *Exception*: Reiter's disease

Bursae

- Acute subdeltoid bursitis
- Retrocalcaneal bursitis
- Ischial bursitis
- Bursitis intractable to local anaesthetics

Ligaments

- Acute stage of a sprained ligament
- Plantar fasciitis
- Sprained cruciate ligaments at the knee
- Sprain of radial and ulnar collateral ligament at the wrist

Spinal disc lesions

- Epidural injection: in patients intractable to local anaesthesia
- Sinuvertebral block

Box 5.7

Contraindications to corticosteroid injection

Absolute contraindications

- Infection of a joint
- Severe infection of neighbouring skin
- Osteomyelitis in neighbouring bones
- Bacteraemia
- Bacterial endocarditis
- Active tuberculosis and herpes corneae
- Immune deficiency (leukaemia, AIDS)
- Allergy to steroid or its vehicle
- Osteochondral fracture
- Joint prosthesis
- Uncontrolled clotting disorder

Relative contraindications

- Anticoagulant treatment: the risk of bleeding from the needle puncture is small, but clotting should not be excessively delayed[171]
- Haemarthrosis
- Reiter's disease
- Markedly unstable joint
- Poorly controlled diabetes
- Adjacent abraded skin: chance of infection is real

Table 5.8 Phenol preparation for injection (P2G)

Component	% by weight
Phenol	2
Anhydrous dextrose	25
Glycerol	30
Water	43

Sclerosing agents

Introduction

Chemical agents such as phenol and dextrose are infiltrated into weakened ligaments and tendons in order to create the formation of strong, thickened fibrous tissue. Because of its proliferative effect on connective tissue, the technique is called *prolotherapy* or *sclerotherapy*.

Product

The original solution, used by Hackett and consisting of zinc sulfate and carbolic acid, provoked painful reactions and was not totally risk free.[214] The mixture used nowadays is the one chosen by Ongley which has a safe reputation as it was regularly used to sclerose varicose veins. It is formulated as described in Table 5.8 and is mentioned under the name P2G.[215]

Mode of action

The mechanism of action for prolotherapy has not been clearly established. The main hypothesis is that the infiltration produces a local inflammatory reaction which is followed by an increased proliferation of fibroblasts and production of new collagen fibres. The final outcome is tightening, reinforcement and loss of normal elasticity of the ligaments.

Liu *et al*[216] and Maynard *et al*[217] studied the histologic effect of injections with a sclerosant. Biopsies of infiltrated medial collateral ligaments of the knee of the rabbit showed not only increase in ligamentous mass and strength but also a normal alignment of the fibres. In other words, the newly produced connective tissue did not have the chaotic appearance of scar but appeared much the same as normal tissues, except that they were thicker, stronger, and contained fibres of varying thickness.

Klein *et al*[218] performed biopsies of posterior sacroiliac ligaments in three patients with chronic low back pain both before and after prolotherapy injections. After six injections at intervals of a week they found an increase in the average ligament diameter measured by electron microscopy from 0.055 micrometres to 0.087 micrometres. Light microscopy showed an increase in collagen-producing fibroblasts. The ligament orientation was organized and linear, as in normal ligaments.

In addition to the effects on fibrous tissue, phenol also has a neurolytic effect. When injected in or around the medial branches of the posterior ramus, it provokes a chemical denervation. This may explain the quick relief (sometimes from the day after the injection) obtained by a number of low back pain patients, treated with sclerosant injection.[219]

Another possible mode of action is through the sclerosing of pathologic neovessels that are frequently associated with painful tendinopathy.[220,221] Finally, the potential of prolotherapy to stimulate release of growth factors favouring soft tissue healing has also been suggested as a possible mechanism.[222]

Table 5.9 Prolotherapy: Indications

Ligaments	Lumbar spine	Lumbar instability
		Chronic postural backache
		Intractable backache
	Sacroiliac joints	Sacroiliac dysfunction (ligamentous pain)
	Peripheral joints	Carpal instability
		Tibiofibular ligaments
		Acromioclavicular ligaments
Tendons		Achilles tendinitis (midportion)
		Infrapatellar tendinitis
		Tennis elbow type II
		Recurrent supra- and infraspinatus tendinitis

Indications (Table 5.9)

Ligaments

1. The main indication for sclerosant therapy is at the lumbar spine. In recurrent disc protrusions or in chronic backache from postural ligamentous pain (see p. 459), a series of infiltrations is made in all the dorsal ligaments of L4–L5–S1 motion segments.

   Prolotherapy is also used for pain relief in 'intractable backache' of the lumbar spine (see p. 582). The positive effect after the injections is thought to be caused by the neurolytic effect of the phenol. The agent is infiltrated around the lateral or medial branch of the posterior ramus and may cause pain relief of months' or years' duration.
2. Sclerosing injections are also used in treating sacroiliac strains. Infiltration is always at the ligamentoperiosteal junction (see p. 608).
3. Recurring carpal subluxations or in the inferior tibiofibular ligament. Infiltration is into the remains of the ligament at the ligamentoperiosteal attachment. The injections can also be used in the treatment of recurrent subluxations and strain of the *acromioclavicular joint*.

Tendons

During the last decade some reports of good results with prolotherapy in the treatment of painful chronic mid-portion Achilles tendinosis were published.[223,224] It is suggested that neovessels play a key part in causing chronic tendon pain, hence the curative effect of their sclerosis.[225,226] Prolotherapy has also been used in the treatment of chronic infrapatellar tendinitis,[220] hip adductor tendinopathy[227] and chronic plantar fasciitis refractory to conservative care[228]. Also type II tennis elbow (tendinopathy of the origin of the extensor carpi radialis

brevis) in which previous infiltrations with steroid have given poor or only temporary relief is a good indication for sclerosing injections.[229,230]

Finally, proliferant infiltrations may also be required in recurrent supra- or infraspinatus tendinitis. Again the injections should be given tenoperiosteally.

Side effects and complications

Infiltration of a sclerosant is quite painful. Therefore 25% of a strong local anaesthetic should always be added to the sclerosant solution. After an hour and up to 2 days thereafter there is considerable after-pain, sometimes to such an extent that the patient needs strong analgesics. Apart from the after-pain there are very few reported side effects.

In 1993 Dorman published a survey of prolotherapy injections performed on a total of 494 845 patients.[231] Of these, 343 897 were treated for low back pain. Only 66 minor complications were reported. These included 24 reports of allergic reactions and 29 instances of pneumothorax. All of these resolved without serious problems. There were also 14 reports of major complications, defined as the patient needing hospitalization or having transient or permanent nerve damage.

Results

A double-blind controlled study of prolotherapy was published in *The Lancet* in 1987.[215] The investigators used a strict set of criteria (such as no litigation, long-standing pain, no severe medical illnesses and a diagnosis of ligamentous back pain). One half of a group of 81 patients received prolotherapy injections with a solution of dextrose, phenol, lidocaine (lignocaine) and glycerin, while the others were injected with saline. The average length of time of symptoms was 8.98 years in the treatment group and 10.72 years in the placebo group. The results showed a statistically significant difference in the two groups, with the prolotherapy group showing a marked decrease in subjective pain as compared to the saline groups ($p < 0.00l$ at 6 months). A later double-blind study in 1993 showed similar results.[232]

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Clinical examination of the cervical spine

6

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History

History taking in patients with problems in the neck, trapezius or shoulder region should be as detailed as possible and great care should be taken to define every symptom precisely. Especially when a controversial treatment, such as manipulation, is to be considered, it is vital that nothing has been forgotten that could constitute a contraindication.

Age may be important, because some disorders do not occur before or do typically occur at a certain period of life. For instance, torticollis in a baby is probably congenital. The same clinical picture in a 5-year-old child is more likely to be caused by contracture of the sternocleidomastoid muscle after, for example, glandular swelling or abscess formation. During and after adolescence it is more probably the result of a disc protrusion.

The same argument applies to root pain. Under the age of 35 it is scarcely ever caused by a disc except after trauma, in which it may occur from 30 years upward. Radicular pain in a young person is usually the result of a neurofibroma, while in the elderly it is commonly the result of compression by an osteophyte or invasion from secondary deposits in the spine, rather than a disc protrusion.

Early morning headache is typical in the elderly and is often the result of contracture in upper cervical ligaments. Headache from temporal arteritis also occurs late in life.

Whenever symptoms appear in a patient of the 'wrong' age group, further investigation should be requested in order to exclude more serious disorders.

The patient's *work*, *hobbies* or preferred *sport* may give an idea of postures, movements or strains that may be causative or provocative.

Cervical spine lesions may lead to the following symptoms: pain, paraesthesia, vertigo or symptoms related to the vertebral artery, and incoordination and spasticity.

Pain

Pain is the most common symptom. Its localization may give an idea of the position of the lesion. Details about its evolution and behaviour help to determine the nature of the disorder.

If this is the patient's first presentation with a problem in the cervical spine, questions are asked about the current complaints (see below). In a recurrence, a detailed and chronological reconstruction of the past history should be made.

The patient may present with acute, subacute, chronic or recurrent pain. Acute and subacute episodes are characterized by difficulty in moving the head without exacerbation of the pain. Except in young people with torticollis or after injury, acute lesions are not as common as more chronic complaints. Chronic lesions come and go for months or years without any tendency towards spontaneous recovery. Most problems in the cervical spine, however, are recurrent, which implies that the course is characterized by definite attacks of short duration. The examiner should then find out whether the patient

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is totally free from pain in between the attacks, for how long the symptoms last and whether the pain is always felt on the same side.

Onset

Next, the following questions are asked about the onset of the pain.

- Where did it start?
  - Pain of cervical origin very often starts at the cervical spine but frequently spreads or shifts to another region quite quickly, so that the cervical source may pass unnoticed.
  - Interscapular onset of pain is typical of a lower cervical disc lesion that compresses the dura. In contrast, it is very unusual for pain to begin in the arm. Should this occur, the possibility of a neurofibroma, compressing a nerve root, has to be considered in young people. In the elderly, an osteophyte or even a malignant process is more probable.
- When did it start?
  - Pain of cervical origin may occur in discrete attacks, especially when a disc lesion is responsible. It is important to encourage the patient to recall the first episode and to ask for a chronological account. In discal root pain, a normal period of spontaneous relief should be recognizable.
- How did it start?
  - The origin may be spontaneous, either acute or chronic, but may also be the consequence of injury. In the latter case, more details should be sought about the type of injury (e.g. a fall or whiplash). It is then necessary to seek further investigations to exclude fractures or luxations.

Evolution

More detailed information is then obtained about the development of the complaints in relation to localization, duration and intensity.

The *localization* may change, either because the pain shifts to another place or because it spreads. Pain that spreads and gradually expands over a larger area is typical of an expanding lesion and should always arouse suspicion. On the other hand, pain that shifts from the scapular area to the upper limb is highly indicative of a shifting lesion (or disc lesion). The fragment of disc substance first displaces posterocentrally and compresses the dura mater, which results in central, bilateral or unilateral scapular pain; it then moves laterally and impinges on the dural investment of a nerve root. The scapular pain disappears and is replaced by a radicular pain down the upper limb. In order to interpret the distribution and evolution of the pain correctly, the mechanism of dural pain should be understood. Because the anterior aspect of the dura mater is innervated by a dense network of branches of sinuvertebral nerves originating at several levels, extrinsic compression and subsequent irritation of the dura may give rise to pain felt in several dermatomes. This phenomenon is called 'multisegmental pain' and is described in Chapter 1. Because the dural investment of the nerve root is only innervated from its own recurrent nerve, irritation here results in pain strictly felt in the corresponding dermatome, thus strict segmental pain.

Also *the duration of the pain* is informative. Most benign cervical disorders are intermittent. If pain progressively worsens, then the presence of an irreversible lesion such as metastases must be borne in mind, particularly in the elderly. Root pain as the result of a disc protrusion lasts for a variable but limited period and then ceases as spontaneous remission takes place (see Ch. 8). Hence, root pain that lasts longer than 6 months should arouse suspicion of another, possibly progressive cause.

As cervical disorders are frequently episodic, the patient should be asked to describe *previous episodes* in as detailed a way as possible. Often a recurrence can be ascribed to instability of the affected structure. Once an intervertebral disc fragment has displaced, it may do so again, because the cartilage remains unhealed. Further displacement may be in a different direction and it is thus possible for the pain to be variable and not on the same side. Therefore, pain that changes sides from one attack to another very strongly suggests the presence of a disc lesion. Also the duration of the previous bouts may have some prognostic value, as it can give an idea of how long the current symptoms may be expected to last. The patient should be questioned about previous successful treatment because, if the present episode is a recurrence, it is very likely that it will respond to the same treatment. Has the patient been totally free of pain between attacks? Freedom from symptoms indicates that the patient went into complete remission and this result could be obtained again. Failure of complete remission requires a search for the reason: possibly previous treatments have not been completed. Alternatively, age may be a factor in that some conditions have a tendency to become more persistent with advancing years.

Current pain

After the patient has given a thorough description of the onset and evolution, the moment has come to ask for details about the pain experienced at present: its localization, the influence of posture and movements, and how it is affected by cough.

Localization

The localization may vary: headache, pain in the face, neck, scapular area, pectoral area or down the upper limb are all possible.

Headache

If headache is referred from the cervical spine, the patient will usually mention an association between the symptoms and certain postures and/or movements. The pain may be bilateral or unilateral, and can be either segmental or multisegmental. Segmental pain originates from the upper cervical segments (C1 and C2). Disorders at the joints between occiput and atlas, or between atlas and axis, may give rise to pain felt at the centre of the upper neck and spreading to the occiput, the vertex (C1) and/or the temples and forehead. As the pain is

not always felt in the neck, localization to the head only can divert attention from the cervical spine. Multisegmental headache results from irritation of the dura at any cervical level. The pain often radiates from the mid-neck up to the temple, the forehead and behind one or both eyes, but rarely to the bridge of the nose. If, apart from this distribution, there is also downward reference of pain to the scapular area, the dural origin is clear.

Some types of headache can be recognized by paying attention to the history.

Early morning headache in elderly patients is a typical example. The patient wakes every morning with headache and/or occipital pain. After some hours the symptoms ease and have completely disappeared by midday. Symptoms do not recur until the next morning. The sequence is repeated daily without fail and, as the years go by, pain tends to last longer into the day. This type of headache responds spectacularly to manipulative treatment (see p. 201).

Migraine is another typical history. Symptoms usually start during adolescence and may persist for many years. The vascular origin of this disorder has been widely accepted and the following features are well known: an 'aura' which frequently includes visual hallucinations, photophobia, nausea, vomiting and other bizarre experiences, often precedes an attack; pain is severe, unilateral and well localized, though may change sides for each attack and is usually described as throbbing or bursting.

C*luster headache* is very severe, is more common in men, and occurs on a regular basis. The pain is always felt on the same side, mainly above the eye, and may be accompanied by a partial Horner's syndrome.

Pain in the face

This may either have a local origin or be referred. Local causes include sinusitis, dental problems, temporomandibular lesions, lesions of the facial bones, neuralgia and arteritis. Referred pain may be a segmental C2 pain or a multisegmental dural pain. The latter should always be borne in mind because, if a local cause is not found, treatment to the cervical spine may be curative.

Pain in the neck

Local pain in the upper neck is usually the outcome of a local lesion: one of the ligaments of the upper cervical segments or one of the upper facet joints. Rarely, the muscular insertions on the occiput are responsible. However, upper cervical pain may also have a multisegmental dural origin. Mid- or lower cervical pain is most often caused by a mid- or lower cervical discodural conflict, especially if the pain is felt centrally or bilaterally. Unilateral and lower cervical pain that is well localized often originates from a facet joint.

Pain in the trapezioscapular area

This is the most common pain reference for cervical lesions. The majority of pain in the trapezius or scapular area has a cervical origin, and must usually be considered as the multisegmental reference of a discodural conflict (Fig. 6.1). The pain may be unilateral, bilateral or interscapular. Depending on the patient's age, it may be intermittent or constant; the older the patient, the more likely the pain will last over longer periods. Upper scapular pain or pain in the trapezius area may also have a C4 segmental origin. Other sources of trapezioscapular pain are a thoracic lesion, a local scapular lesion or a shoulder girdle problem.

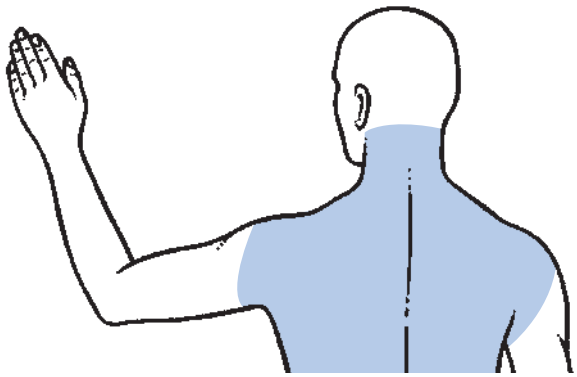


Fig 6.1 • Multisegmental scapular pain.

Pain in the pectoral area

Another rare manifestation of multisegmental dural reference is pain in the pectoral area. Because the pain is usually felt deeply and there is a strong popular association between pectoral (retrosternal) pain and cardiac disease, the pectoral discomfort can be initially misjudged as angina.

Pain down the upper limb

Dural pain never presents down the arms. Therefore, upper limb pain is always segmental in origin and is referred within its dermatomal borders. If the lesion is discoradicular, the normal time sequence of the symptoms must be appreciated: neck pain first, followed by unilateral scapular pain and then finally segmental pain. It is therefore important to ask whether or not the arm pain has been preceded by neck and/or scapular pain. If not, a non-discogenic origin of the pain is to be considered.

Exacerbation of pain by cough

This is uncommon in cervical lesions but may occur in a disc prolapse, when the pain is usually felt in the scapular region. Pain in the arm on coughing is one of the symptoms that draws attention to a neuroma (see Ch. 9).

Paraesthesia

Paraesthesia is a very common symptom which may originate from any nerve fibre in the cervicoscapular area or in the arm (Table 6.1). Paraesthesia is often experienced as a 'pins and needles' sensation. In other instances, the patient may describe the feeling as 'numbness'. The moment the patient mentions the presence of such symptoms, the examiner should carefully determine how proximal they are because, as has been explained in Chapter 2, the point of compression always lies proximal to that of the paraesthesia. The lesion may lie at any one of a number of different levels but the vaguer the distribution of the pins and needles, the more proximally the lesion needs to be sought.

Table 6.1 Paraesthesia

Level	Cause/site of cause	Symptoms
Cervical	Myelopathy:	No pain
	Intrinsic	Multisegmental paraesthesia on neck flexion
	Extrinsic	Lhermitte's sign
	Nerve root	Pain Segmental paraesthesia Compression phenomenon
Shoulder girdle	Brachial plexus	Vague paraesthesia Release phenomenon
Arm	Nerve trunk	Defined area of paraesthesia Specific tests
	Nerve ending	Cutaneous analgesia (Paraesthesia)

At the cervical spine

Spinal cord

External pressure on the spinal cord is characterized by painless paraesthesia in the upper and/or lower limbs felt distally and in a multisegmental distribution. The paraesthesia comes and goes in a wholly irregular way, most marked by day. Neck flexion usually increases the symptoms, or Lhermitte's sign may be present: an electric shock sensation in the trunk and/or upper limbs following forceful passive flexion of the cervical spine. Differential diagnosis has to be made from pernicious anaemia, diabetes and peripheral neuritis.

Nerve root

When a nerve root is affected, paraesthesia appears distally in the corresponding dermatome. The pins and needles come and go in an erratic fashion and do not last for more than an hour at a time. Pain resulting from external pressure on the dural sleeve may precede or accompany the paraesthesia.

At the shoulder girdle

Lesions of the brachial plexus at the thoracic outlet give rise to paraesthesia in one or both hands and affect all digits. When there is external pressure and because of the release phenomenon, pins and needles are only felt after the compression has ceased (see Ch. 2). They are often nocturnal, waking the patient after some hours' sleep.

In the arm

When one of the peripheral nerves in the upper limb is involved, paraesthesia (and sometimes pain) is felt in the territory of that nerve (radial, median or ulnar). The well-known distribution of paraesthesia (three and a half radial, or one and a half ulnar fingers) often gives a clue. Differentiation from radicular paraesthesia is generally not difficult. In case of doubt, specific tests to elicit the pins and needles can be performed (e.g. Tinel's test). The pins and needles are often accompanied by cutaneous analgesia, which may be marked, especially in distal lesions.

Vertigo or symptoms related to the vertebral artery

It is well known that vertigo may be the consequence of vertebrobasilar vascular insufficiency. However, cervical disorders without any impairment of the vertebral artery can also give rise to vertigo. The explanation is that the cervical spine, together with the eyes and the labyrinths of the ear, is an important source of proprioceptive information that influences the sense of balance. When vertigo is a dominant symptom, the examiner must carefully look for its origin, especially if a manipulative treatment is considered (see Ch. 11).

Incoordination and spasticity

These complaints are indications that something is amiss with either deep (proprioceptive) sensibility, or the vestibular system or the cerebellum.

Medication

Current medication may have a bearing on both diagnosis and management, and should be carefully ascertained, especially because anticoagulants are contraindications to manipulative therapy (see Ch. 5).

A summary of history taking is given in Box 6.1.

Inspection

Inspection and examination are performed on the standing patient. The examiner stands behind the patient and looks at the neck and scapular region. First the posture of neck and head is observed. The head should be in the midline, with the chin above the manubrium. A normal neck shows a slight lordotic curve. Loss of the lordotic curve, with the head fixed in flexion, suggests a block at the back of an intervertebral joint and is most common after whiplash injuries. Next, neck length is ascertained. A short neck is present in Klippel–Feil syndrome (due to fusion of several vertebrae)[1] and a webbed neck in a girl is typical of Turner's syndrome.[2] Then the examiner notices if the head is tilted or rotated in one direction, which indicates torticollis. A careful analysis of the torticollis is then made. The neck may be held in lateral deviation or in a combined position of lateral deviation and rotation. The lateral tilt as well as the rotation can be towards or away from the painful side. In acute discodural torticollis, the head is usually deviated in lateral flexion away from the painful side. There is usually no fixation in rotation. Shortening of the sternocleidomastoid muscle (congenital or acquired) leads to a deviation of the head in lateral flexion towards the pain combined with rotation to the other side (see Ch. 8).

Box 6.1

Summary of history

Age

Work, hobbies, sport

Pain

Onset

1 Where?
2 When?
3 How?

Evolution

1 Localization
2 Duration

Current pain

1 Localization
   a Headache
      i Segmental/multisegmental
      ii Typical histories
         ◦ early morning headache
         ◦ migraine
         ◦ cluster headache
   b Pain in the face
      i Local lesions
      ii Referred pain
         ◦ segmental
         ◦ multisegmental
   c Pain in the neck
      i Upper neck
         ◦ local lesion
         ◦ multisegmental
      ii Mid- or lower neck
   d Scapular pain
      i Segmental
      ii Multisegmental
      iii Thoracic lesion
      iv Scapular lesion
   e Pectoral pain: multisegmental
   f Arm pain: segmental
      i Spinal origin
      ii Non-spinal origin
2 Influence of coughing

Paraesthesia

Lesion at the cervical spine

1 Spinal cord
2 Nerve root

Lesion at the shoulder girdle (brachial plexus)

Lesion in the arm (peripheral nerve)

Vertigo or symptoms related to vertebral artery

Incoordination and spasticity

Medication

The scapular and deltoid areas are surveyed. Deviations are noted in the position of the scapulae and in the muscular contours of neck, trapezius and shoulder muscles.

Functional examination

A complete examination of the cervical spine is comprised not only of neck movements but also of shoulder and upper limb tests. Many of the symptoms that occur in the upper limb originate from the neck. Hence an upper limb scanning examination must always be performed to distinguish them from local lesions. The examination includes articular tests, root tests, cord tests and tests for the peripheral joints. Before the examination starts, the examiner asks the patient if anything can be felt at that moment. If the answer is affirmative, the next questions will be: what is felt, and where? During the subsequent test session the examiner will find out if the movements have any influence on these symptoms or evoke other symptoms.

Neck movements

The movements are first performed actively and then repeated passively; after this, resisted movements are tested.

Active movements

The patient is asked to flex and extend the head and to rotate and tilt it in both directions (Fig. 6.2). The amplitude of each movement is noted and the patient indicates whether it is painful and, if so, where. Neck flexion must be considered not only as an articular test for the cervical spine but also as a dural test for the thoracic spine in that it stretches both the cervical and the thoracic dura. Hence, if pain is elicited in the upper thoracic region, it may be the consequence of a dural impingement at either a cervical or a thoracic level. The relation of the movements shows either a symmetrical (full articular) pattern (Fig. 6.3) or an asymmetrical (partial articular) pattern (Fig. 6.4). The full articular pattern of the cervical spine is: some or great limitation of extension, equal degree of limitation of both rotations and both lateral flexions and no limitation of flexion. All other combinations of pain (and limitation) are classified as partial articular patterns.

Passive movements

Next, the same movements are performed passively. However, it is not always necessary to carry these out. Flexion and both lateral flexions are done in doubtful cases only, but passive extension and both passive rotations are always executed with great care. Pain, limitation and the end-feel are assessed. The normal end-feel is capsular. Abnormal end-feels are muscle spasm, bone-to-bone, crisp, empty, soggy and elastic rebound. They point towards particular pathological entities (see Ch. 4).

Passive extension

The examiner asks the patient to extend the neck. Both forearms are then placed against the patient's scapulae with the fingers on the patient's forehead. Simultaneous radial deviation of both hands tests the end-feel in extension. By executing the test very gently, hard axial pressure is avoided.

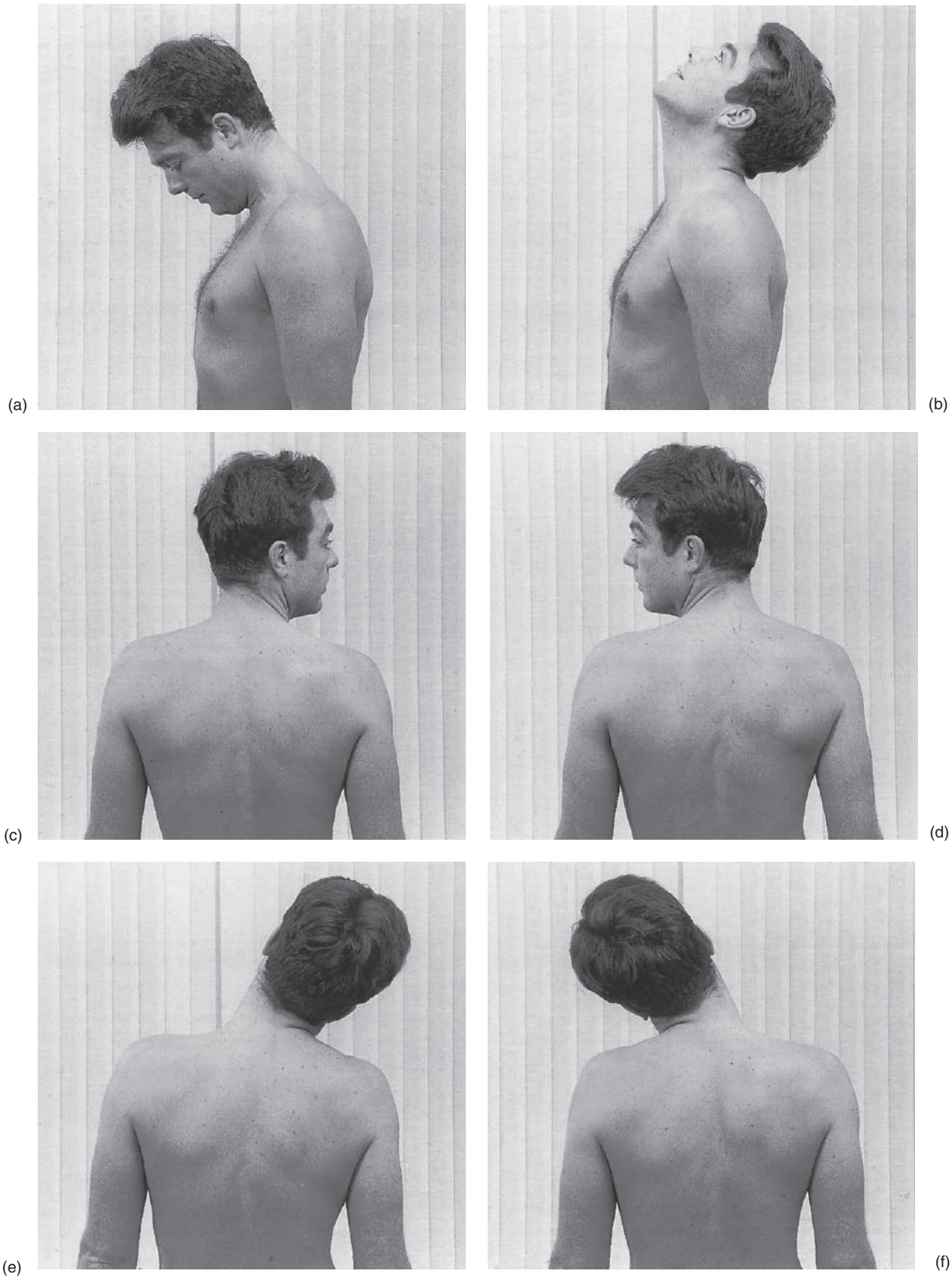


Fig 6.2 • Active movements: flexion (a), extension (b), rotation (c, d) and lateral flexion (e, f) of the head.

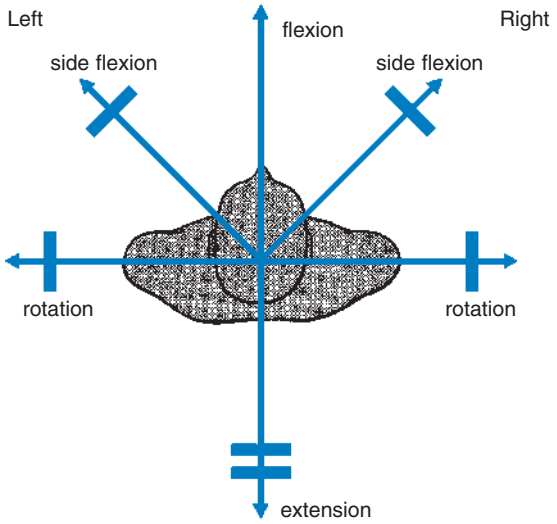


Fig 6.3 • The full articular pattern.

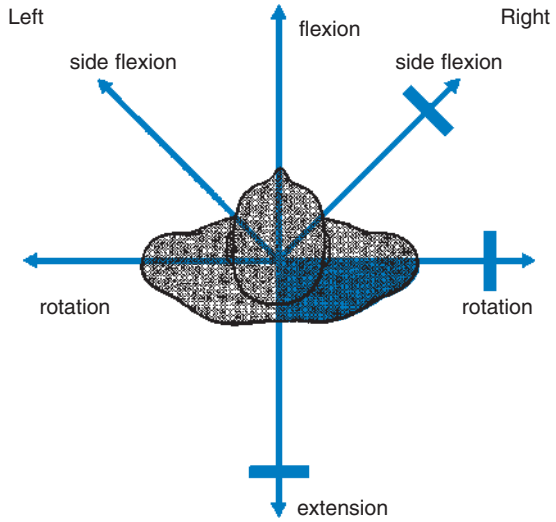


Fig 6.4 • Example of a partial articular pattern (colour indicates pain).

Rotation

The patient turns the head in rotation, first to one side, then the other. To avoid trunk movements, the examiner stabilizes the patient's body using the forearms. One is placed against the patient's scapula at the side towards which the patient turns the head and the other at the anterior aspect of the other shoulder. The hand of the arm that overlies the scapula moves into ulnar deviation and grasps the patient's forehead on the contralateral side. The other hand moves into radial deviation and grasps the patient's occiput on the side facing the examiner. Movement is performed by a simultaneous action of both hands. As the head turns, end-feel is again noted.

Lateral flexion

The examiner places the ipsilateral hand laterally against the patient's shoulder. With the other hand placed above the ear at the opposite side, gentle side flexion is undertaken.

Flexion

The patient bends the head and the examiner gently assists it into greater flexion. Excessive pressure should be avoided.

Resisted movements

After active and passive tests, resisted movements are performed. Conduction of both rotations suffices. The other movements are only carried out in case of doubt and for the purpose of differential diagnosis. Pain and/or weakness are ascertained. Interpretation of positive resisted movements must be done in the light of the overall clinical picture. The response can be obscured in acute lesions, where the slightest effort augments the pain as the result of transmitted stress. Muscular and tendinous lesions are extremely uncommon at the cervical spine. The movement also gives information on motor conduction of the first cervical nerve root.

Primary test: rotation

The patient holds the head in the neutral position. The examiner places the forearms against both scapulae with the fingers just above the patient's ears and the fingertips pointing forwards so that they lie on the temples (Fig. 6.5). The patient is asked to rotate the head and the movement is resisted with the fingertips.

Secondary tests: extension, flexion and lateral flexion

These are illustrated in Figure 6.6.

Flexion

The examiner stands beside the patient and places one hand on the upper interscapular area and the other hand on the forehead. The patient is asked to push the head forwards and resistance is provided.

Extension

The examiner stands beside the patient. One hand is placed laterally on the sternum and the other on the occiput. The patient is asked to pull the head backwards and resistance is provided.

Lateral flexion

The examiner stands behind the patient. One hand is placed laterally on the opposite shoulder and the other above the ear. The patient is asked to push the head against the hand. Resistance is exerted.

Scapular movements

Scapular movements are performed to establish the mobility of the scapula in relation to the thorax. They also test the shoulder girdle and the thoracic part of the dura mater.

Active movement

The patient is asked to elevate the shoulders (Fig. 6.7). Pain and/or limitation are checked.

The mobility of the scapula on the thoracic cage, together with the integrity of the acromioclavicular joints, the sternoclavicular joints and the joints between the first ribs and the vertebral column, is assessed. Any pain and/or limitation

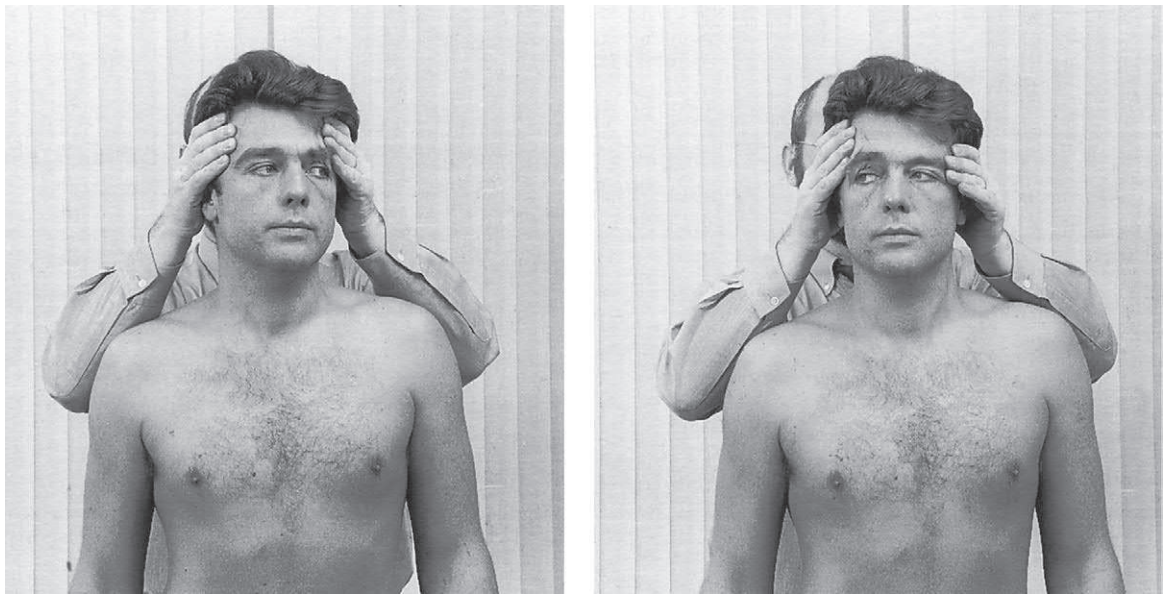


Fig 6.5 • Primary resisted movement: rotation.

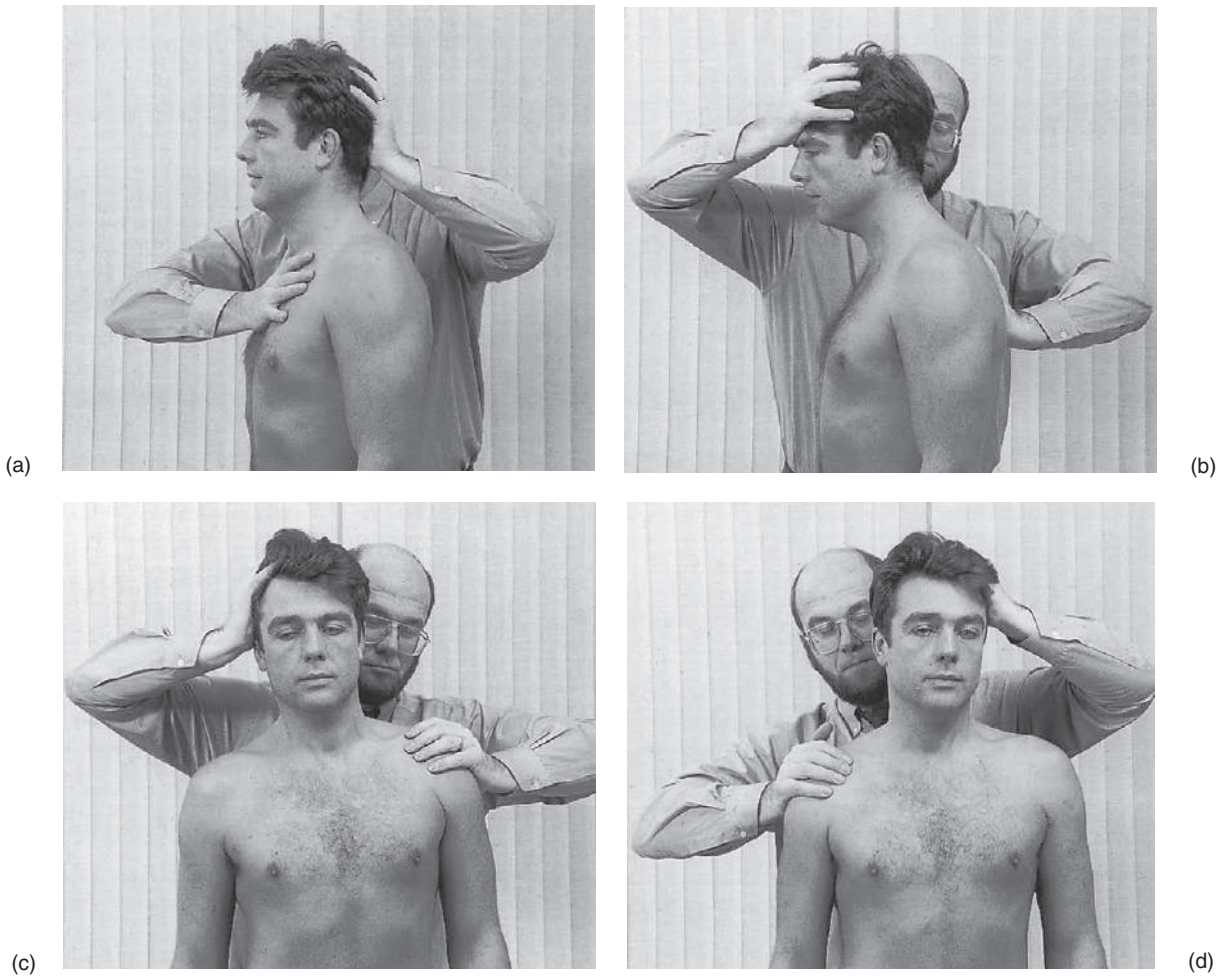


Fig 6.6 • Secondary resisted movements: extension (a), flexion (b) and lateral flexion (c, d).

Fig 6.7 • Active elevation of the shoulders.

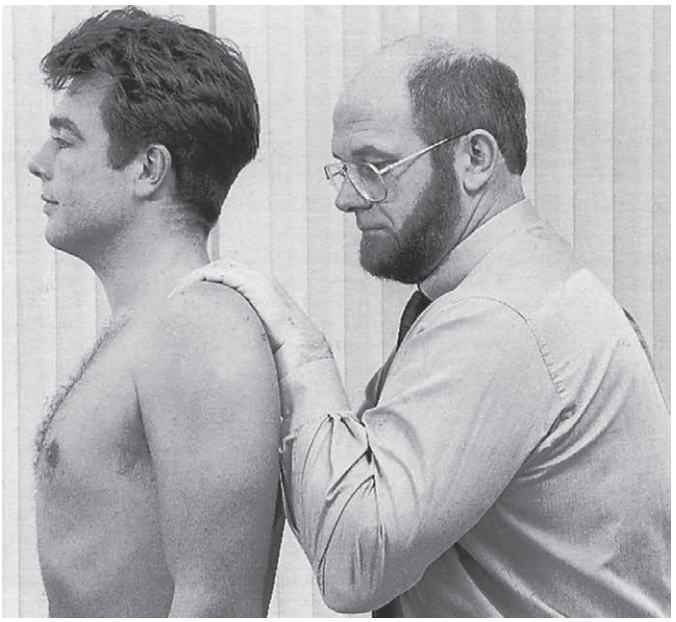


Fig 6.8 • Resisted elevation of the shoulders.

suggest the need for a full shoulder girdle examination (see p. 210).

Resisted movement

The patient is asked to keep the shoulders elevated while the examiner applies a downward force on them (Fig. 6.8). This tests the trapezii muscles and the motor conduction in C2–C4. Normally the trapezii are stronger than the downward pressure.

Arm tests

Arm movements test the integrity of the muscular system.

If pain is elicited, alternative causes of pain down the upper limb should be sought. When one or more movements are weak a neurological problem is responsible. The pattern presented will indicate the possible level.

Active elevation

The patient is asked to elevate both arms sideways, as high as possible (Fig. 6.9). This is a swift scan for shoulder and shoulder girdle problems. If there is pain and/or limitation, a complete shoulder examination should follow (see Ch. 12).

Resisted movements

Isometric resisted tests are done bilaterally, so that strength may be compared. These are mainly tests for motor conduction and so strength is noted, but, if the test elicits pain, the possibility of a local lesion should be considered.

The tests are illustrated in Figures 6.10–6.13. Detailed execution of the different tests is discussed in the relevant chapters on the shoulders, elbow and wrist.

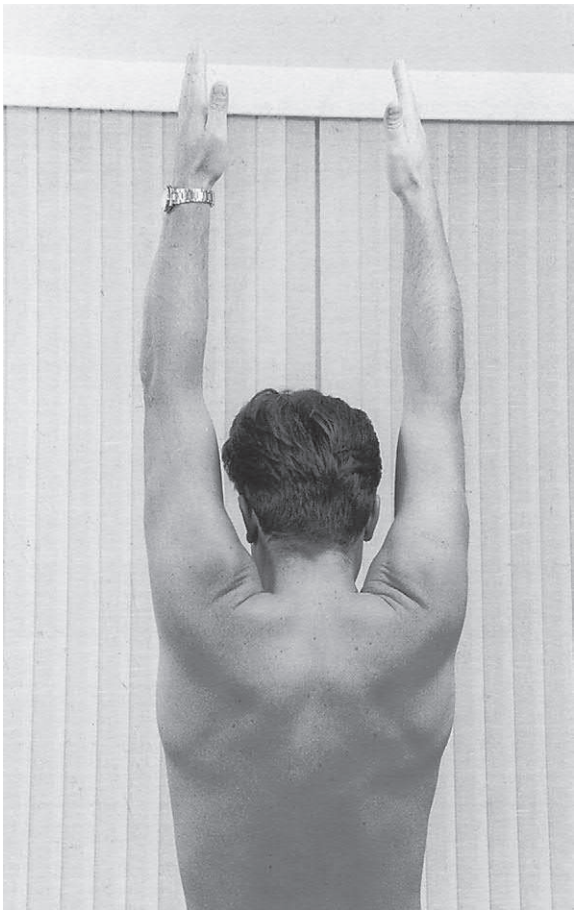
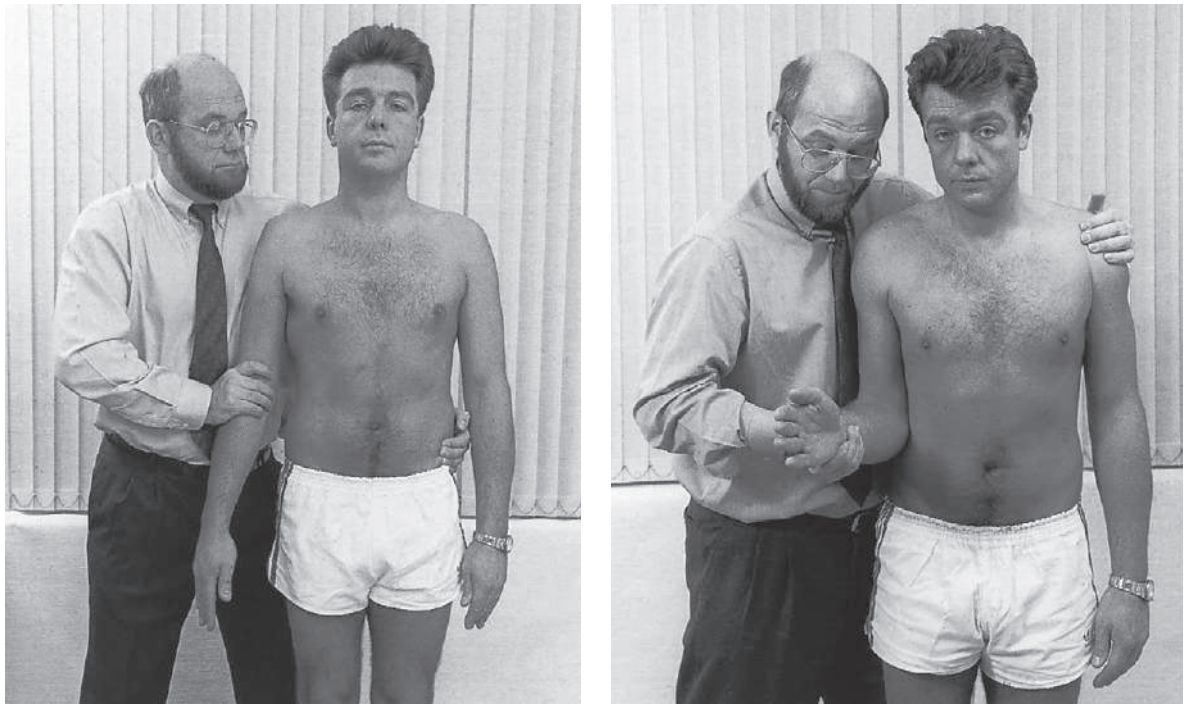
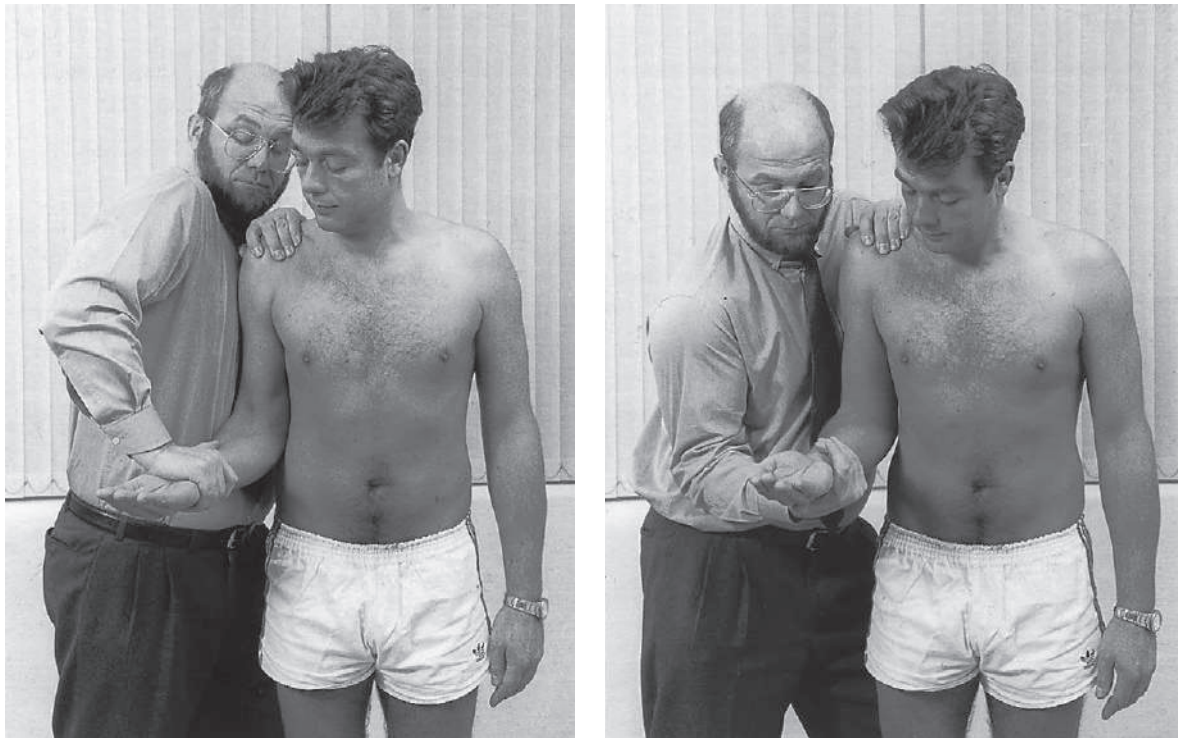


Fig 6.9 • Active elevation of the arms.



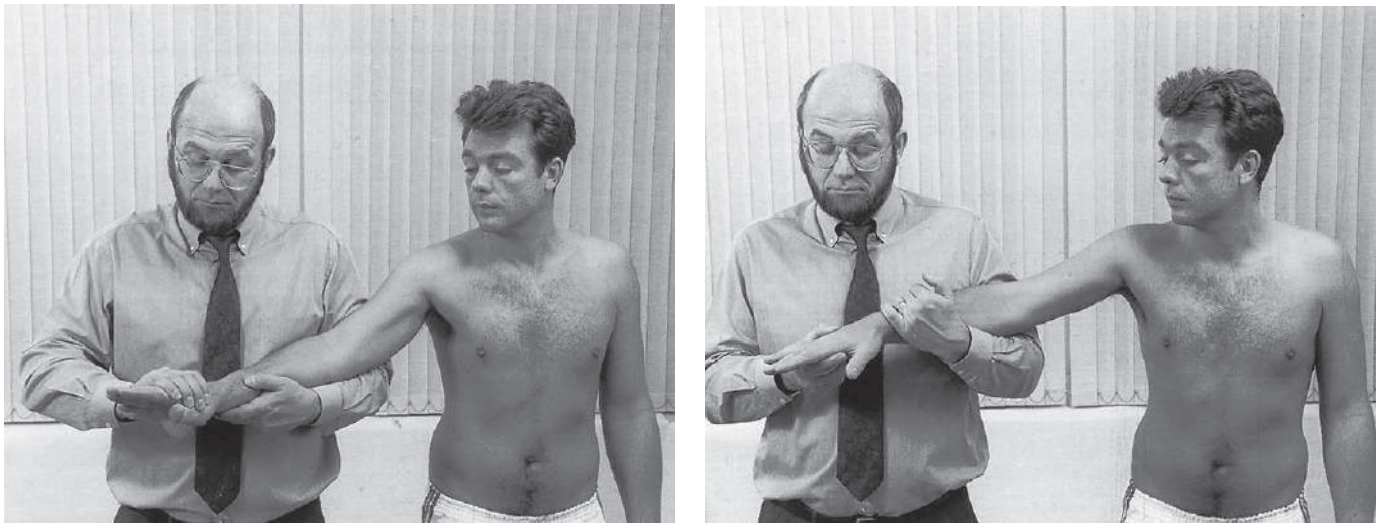
(a) (b)

Fig 6.10 • (a) Resisted abduction of the shoulder tests the C5 nerve root and the abductor muscles. (b) Resisted external (or lateral) rotation of the shoulder tests the C5 nerve root and the external (or lateral) rotators.



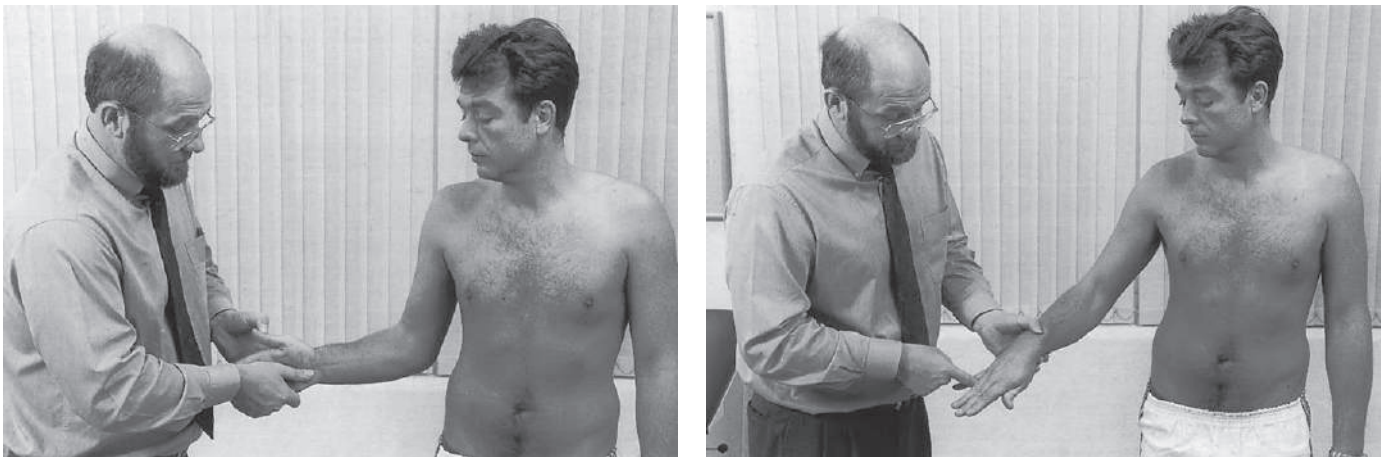
(a) (b)

Fig 6.11 • (a) Resisted flexion of the elbow tests the C5 and C6 nerve roots and the elbow flexors. (b) Resisted extension of the elbow tests the C7 nerve root and the elbow extensors.



(a) (b)

Fig 6.12 • (a) Resisted extension of the wrist tests the C6 nerve root and the wrist and finger extensors. (b) Resisted flexion of the wrist tests the C7 nerve root and the wrist and finger flexors.



(a) (b)

Fig 6.13 • (a) Resisted extension of the thumb tests the C8 nerve root and the thumb extensors. (b) Resisted adduction of the fifth finger tests the T1 nerve root and the intrinsic muscles of the hand.

Resisted abduction of the shoulder

This tests the C5 nerve root and the abductor muscles of the shoulder (deltoid and supraspinatus). The test is performed with the arm hanging down, a few degrees of abduction being permitted. The patient is asked to resist the examiner's attempt to push the arm inwards.

Resisted external rotation of the shoulder

The C5 nerve root is tested as well as the external rotators of the shoulder (infraspinatus and teres minor). The patient is asked to bend the elbow to a right angle and to hold it firmly into the side. The examiner's attempt to push the forearm medially should be resisted.

Resisted flexion of the elbow

This test examines nerve roots C5 and C6 and, at the same time, the flexors of the elbow (biceps, brachialis and brachioradialis). The patient holds the elbow in 90° flexion and supination, and resists the examiner's attempt to extend the elbow.

Resisted extension of the elbow

This tests nerve root C7 and the extensor of the elbow (triceps). The elbow is again held at a right angle and the examiner's attempt to bend the patient's elbow is resisted.

Resisted extension of the wrist

The C6 nerve root is tested, as well as the extensors of the wrist (extensores carpi) and fingers (extensor digitorum

communis). The elbow is held in extension. The patient resists the examiner's attempt to flex the wrist.

Resisted flexion of the wrist

This test examines nerve root C7 and the flexors of the wrist (flexores carpi) and fingers (flexores digitorum). The elbow is held in extension. The examiner's attempt to extend the wrist is resisted.

Resisted extension of the thumb

The C8 nerve root is tested, as well as the extensors of the thumb (extensores pollicis). The examiner's attempt to flex the thumb is resisted.

Resisted adduction of the fifth finger

This tests the T1 nerve root and the intrinsic muscles of the hand, i.e. the adductors of the little finger. The patient squeezes the examiner's finger between the fourth and fifth fingers. The examiner assesses the strength.

Sensory conduction

The examiner passes his fingers over the patient's skin in the different dermatomes (Fig. 6.14). The patient is asked if the sensation is the same over all areas.

One arm is compared with the other, and each dermatome is compared with the others in the same limb.

Testing reflexes

The main reflexes are tested and note is taken of whether they are normal, diminished, absent or inverted. Each side is always compared with the other.

Biceps reflex

The patient's elbow is held at a right angle and is well relaxed. The tendon is stretched by the pressure of the examiner's thumb on which the hammer is tapped. The C5 and C6 nerve roots are tested. The reaction is elbow flexion (Fig. 6.15).

Fig 6.14 • Testing sensory conduction.

Brachioradialis reflex

The patient's elbow is held at a right angle and is well relaxed. The hammer taps on the distal end of the radius. This tests the C5 nerve root. The reaction is elbow flexion (Fig. 6.16).

Triceps reflex

The patient's elbow is 90° flexed and well relaxed. The hammer strikes the triceps tendon, just proximal to the olecranon. The C7 nerve root is tested. The reaction is elbow extension (Fig. 6.17).

Plantar reflex

To test the plantar reflex, the examiner uses the sharp end of the reflex hammer to stroke the sole of the patient's foot, starting at the lateral aspect of the heel and moving along the

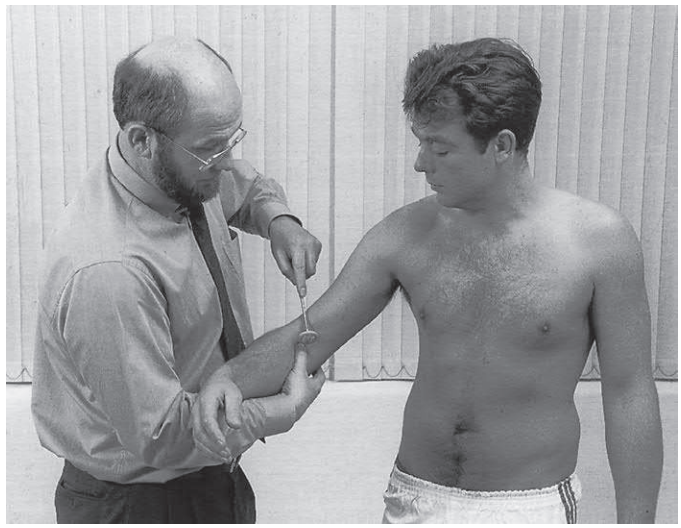


Fig 6.15 • Testing the biceps reflex: C5 and C6 nerve roots. The reaction is elbow flexion.

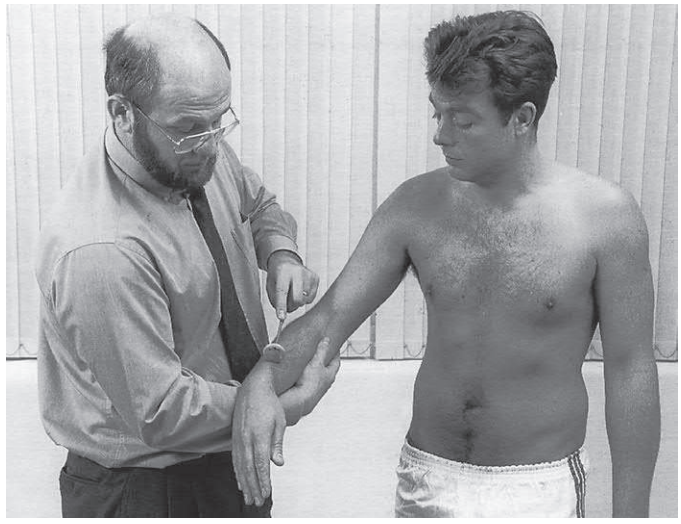


Fig 6.16 • Testing the brachioradialis reflex: C5 nerve root. The reaction is elbow flexion.

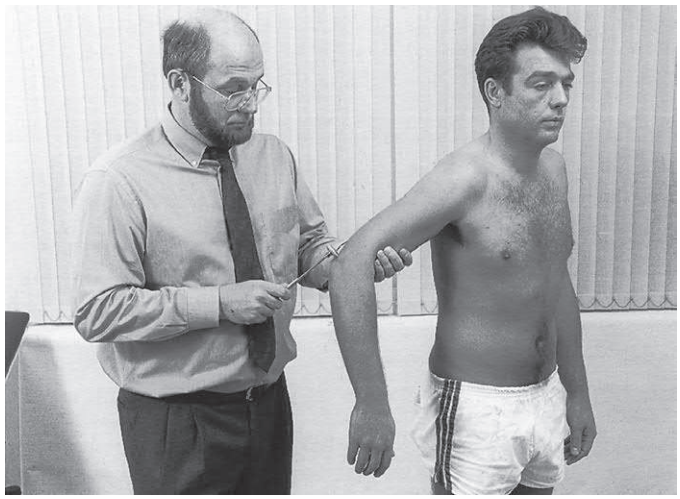


Fig 6.17 • Testing the triceps reflex: C7 nerve root. The reaction is elbow extension.

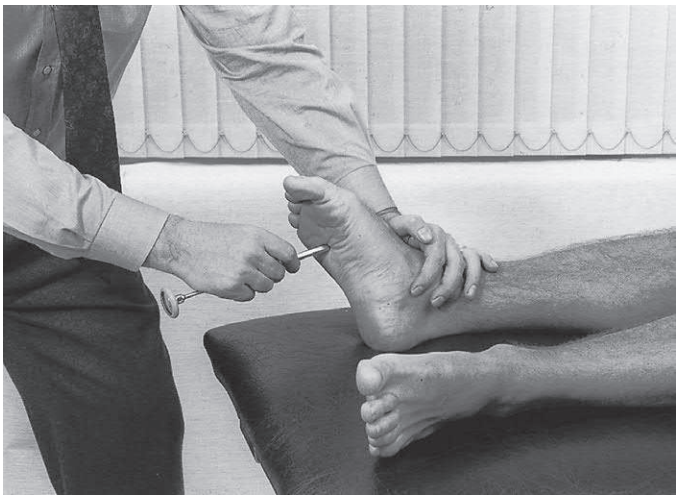


Fig 6.18 • Testing the plantar reflex: central nervous system. The reaction is flexion of the toes.

lateral border of the foot to the base of the fifth metatarsal bone and then onwards to the base of the big toe at the medial aspect of the foot. The normal reaction, as described by Strümpell, is flexion of the toes and withdrawal of the foot (Fig. 6.18). The pathological reflex – Babinski's sign – is a slow extension of the big toe, combined with spreading of the other toes and flexion of knee and hip. The presence of Babinski's sign indicates a (severe) central disorder.

Hoffmann's sign

The hand is supported and pronated so that wrist and fingers fall into slight flexion. The middle finger is firmly grasped and partially extended. The nail is then flicked by the examiner's thumbnail. This flicking should be done with considerable force. The sign is considered to be positive when quick flexion of both the thumb and the index finger results.[3] A positive sign is indicative of possible pyramidal tract pathology (Fig. 6.19).[4]

A summary of the neurological deficit at each level is given in Table 6.2.

A summary of the functional examination is given in Box 6.2.

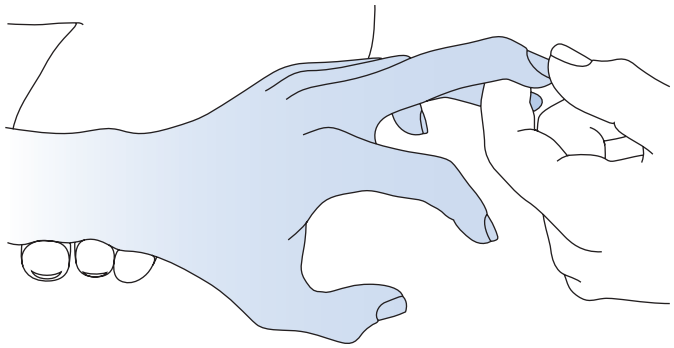


Fig 6.19 • Hoffmann's sign is elicited by flicking the distal phalanx of the long finger. Flexion of the thumb at the interphalangeal joint is a positive response.

Technical investigations

Technical investigations have become a routine measure in the evaluation of patients with neck pain for several reasons: to assist diagnosis, to complement the clinical diagnosis, to meet the patient's request for 'radiography', or for medicolegal reasons.

During recent decades there has been a tendency to reduce the time spent on history taking and clinical examination and to proceed immediately with technical investigations in order to detect the anatomical changes that are held to be responsible for a patient's condition. This trend not only has serious financial consequences but also leads to diagnostic errors. It is important to realize that imaging, whether it is X-ray, computed tomography (CT) or magnetic resonance imaging (MRI), does not reveal the source of the pain but only shows anatomical changes that may or may not be consistent with the patient's description of the pain. Most of these anatomical alterations reflect painless degenerative changes that are normal at certain ages and which may also be present in asymptomatic individuals. The examiner should constantly keep in mind the fact that the presence of anatomical and morphological changes does not automatically imply causality. For example: CT and MRI are widely used to demonstrate the existence of discal disorders, which also exist in a large number of asymptomatic people. Boden[5] cites a figure between 14 and 28% and Teresi[6] a figure of 23%. According to Matsumoto *et al*, degeneration is present in 86% of the discs of asymptomatic individuals of over 60 years of age. Posterior disc protrusion is observed in 7.6% of asymptomatic subjects.[7]

However, technical procedures may be of great help in complementing the clinical findings, i.e. to confirm a tentative diagnosis or to exclude serious disorders. The examiner should always remember, however, that a clinical diagnosis is a basic requirement and that imaging techniques should never be used

Table 6.2 Neurological deficit at each level

Level	Deficit
C1–C2	Tingling in the occipitoparietal region. Muscular weakness is rare
C3	Paraesthesia or numbness at the lower pinna, the posterior part of the cheek, the temporal area and the lateral aspect of the neck. Weakness is clinically not detectable. Cutaneous analgesia is uncommon (the lateral aspect of the neck)
C4	A horizontal band of cutaneous analgesia along the spine of the scapula, the mid-deltoid area and the clavicle. Slight weakness of the trapezii. Paraesthesia does not occur
C5	Weakness of the supraspinatus, infraspinatus, deltoid and brachial biceps muscles. The biceps and brachioradialis jerks are sluggish or absent. Paraesthesia and sensory deficit do not occur
C6	Paraesthesia in thumb and index fingers. Analgesia at the tips of thumb and index fingers. Weakness of the biceps, brachialis, supinator brevis and the extensores carpi radiales muscles. The biceps jerk is sluggish or absent
C7	Paraesthesia in index, middle and ring fingers. Cutaneous analgesia at the dorsal aspect of the index and middle fingers. The weak muscles are the triceps and the flexor carpi radialis. The triceps jerk may be affected
C8	Paraesthesia in the middle, ring and little fingers. Cutaneous analgesia at the little finger. The weak muscles are both extensors of the thumb, the extensor and flexor carpi ulnaris, the adductor pollicis, the common extensor of the fingers and the abductor indicis
T1	Paraesthesia and cutaneous analgesia at the ulnar aspect of the hand, and weakness of the intrinsic muscles of the hand
T2	Paraesthesia and motor and sensory deficit are very uncommon

Box 6.2

Summary of the functional examination

Neck movements

6 active movements:
pain/limitation

3 (or 6) passive movements:
pain/limitation/end-feel

2 (or 6) resisted movements:
pain/weakness – C1

Scapular movements

Active elevation
pain/limitation → Shoulder girdle examination?

Resisted elevation
C2–C3–C4

Arm tests

Tests for neurogenic integrity and alternative causes of arm pain

Active elevation:
pain/limitation → Shoulder examination?

Resisted movements?
(tests for motor conduction):

shoulder:
- abduction – C5
- external rotation – C5

elbow:
- flexion – C5/C6
- extension – C7

wrist:
- flexion – C7
- extension – C6

thumb extension – C8
little finger adduction – T1

Sensory conduction

Reflexes:
Biceps – C5/C6
Brachioradialis – C5
Triceps – C7
Plantar – CNS
Hoffmann's – CNS

as screening tests. Also, decisions about treatment procedures should be made on clinical grounds but may be influenced by specific findings on imaging.

Radiography may show congenital anomalies but quite often these are not clinically significant. Another reason for obtaining plain films is to exclude fractures and luxations.[8] Plain radiography has a specificity of 100% in low-risk patients.[9,10] However, in high-risk patients, helical CT scan seems to be the preferred initial screening test for detection of cervical spine fractures.[11,12] The most important consideration with cervical X-rays is perhaps the risk of missing tumours and infections. It is important to remember, however, that bony disorders are not always visible on plain X-rays in their initial stages, and a negative examination may give a false feeling of security.

Computed tomography is a good diagnostic tool for bony disorders, such as fractures and luxations, where it is often more effective than plain radiography and MRI.[13] It is useful to demonstrate osseous neural foraminal stenosis, bone destruction or bone proliferation and ossification of the posterior longitudinal ligament. It is also effective for detecting cranial migration of the odontoid process in rheumatoid arthritis patients.[14]

Magnetic resonance imaging – because of its superior depiction of soft tissue anatomy – is a preferred tool for the diagnosis of soft tissue disorders such as pre- or paravertebral haemorrhage or oedema, ligamentous lesions and disc herniations.[15] It is also excellent for demonstrating cord compression and nerve root injuries.[16] It appears to be the most reliable imaging study for diagnosing spinal infections[17] and tumours.[18] Dynamic functional MRI may provide additional information in patients with rheumatoid arthritis.[19]

Scintigraphy is most useful for the detection of quantitative changes in the skeleton in bone metastases and inflammatory and metabolic bone diseases.

Electromyography has a low sensitivity and specificity, and is therefore only secondary in the diagnosis of cervical nerve

root palsies, which can easily be detected by clinical testing. It can have a certain importance in cases with medicolegal implications.

Conclusions

- Technical investigation should not replace clinical examination.
- It may be used to clarify the clinical picture or to exclude a serious disorder.
- The results must be evaluated with great care and only in the light of the clinical picture.
- In doubtful cases, clinical evaluation will be more important than technical investigations.
- Treatment decisions should never be taken on the outcome of imaging studies alone.

Access the complete reference list online at **www.orthopaedicmedicineonline.com**

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7

Interpretation of the clinical examination of the cervical spine

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A standardized clinical examination enables the examiner to recognize clinical patterns. He/she will easily distinguish common patterns from uncommon ones and recognize so-called 'warning signs' (see Ch. 9). Neck, shoulder girdle and shoulder problems are differentiated and a distinction is made between 'mechanical' conditions, such as disc lesions or capsuloligamentous lesions, and 'non-mechanical' conditions, such as rheumatological, neurological or infectious disorders.

Interpretation of the history

Most symptoms (elements taken from the history) are purely subjective. They are: pain, paraesthesia, numbness and dizziness. Others, such as diminished mobility and weakness, are more objective and can be checked during the functional examination.

Pain

Pain is usually the leading symptom. Pain behaviour is extremely important and is defined by the following elements: localization, onset, evolution, influencing factors, duration and accompanying symptoms.

Localization

The actual site of the pain is a first rough pointer. Pain may be localized or vague, and is felt either in the neighbourhood of the lesion or at a distance (see Referred pain, Ch. 1).

Very localized pain, accurately indicated by the patient, is often a ligamentous or facet joint problem. Bony lesions also give rise to localized pain. Pain that is vaguely defined and spreads over a larger area is usually referred. It is then distal to the lesion (see Rules of referred pain, Ch. 1). Referred pain that is felt in a particular dermatome (segmentally referred) is often radicular in origin but may also result from any soft tissue lesion in the region of the neck. The source is usually an inflammation and/or compression of a mid- or lower cervical nerve root, giving rise to pain in the shoulder area (C4) or in the upper limb (C5–T2). It often indicates a discoradicular interaction, but other causes of root pain should also be considered, such as degenerative conditions or a space-occupying lesion in the radicular canal.

Pain that is felt in several adjacent dermatomes at the same time is quite common and indicates a multisegmental type of reference (see Fig. 7.1). Multisegmental pain reference may be either the result of multiradicular involvement – which is extremely uncommon in the cervical spine and should immediately arouse suspicion (see Warning signs, Ch. 9) – or the result of a discodural interaction. In the latter case other dural symptoms may also be found and the functional examination shows a clinical picture of internal derangement (see Ch. 8). It should be stressed that dural symptoms are mostly discogenic in origin but may also occur in any space-occupying lesion in the spinal canal interfering with the sensitivity or the mobility of the dura mater. Dural pain is usually felt at the lower neck, in the trapezius area and the upper scapular and interscapular region, either centrally or unilaterally. The pain may spread further, upwards to the head, face and upper neck or the mid-scapular, pectoral and axillary regions.

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Onset (Fig. 7.2)

Pain may come on suddenly, gradually or as the result of an injury. Pain that starts suddenly is activity-related. It is a manifestation of sudden internal derangement of an intervertebral joint, mostly the result of the displacement of a discal fragment. It is then usually accompanied by sudden twinges when moving. It comes and goes in an irregular way and tends to recur. Functional examination shows the articular involvement. Pain that comes on gradually is not very informative because many different conditions begin in that way. If the pain is related to specific activities, a mechanical condition (see Ch. 8) is probable; if such a relation between symptoms and movements or postures is not found, a non-mechanical condition should be considered (see Ch. 9). If an injury is responsible for the development of the patient's symptoms, further technical investigation will be necessary to exclude serious disorders such as fractures and luxations.

Evolution (Fig. 7.3)

Pain may shift from one place to another, mostly from the centre of the neck to one side or from the cervicotrapezioscapular area to the upper limb. The meaning of pain that moves is important: the pain shifts because the lesion shifts. There are not that many lesions that may change their position; a loose fragment of disc is one of the few possibilities. Since discodural or discoradicular interactions occur in episodes, pain may switch sides from one attack to another. Pain may also expand, which means that it increases in extent and in intensity. Expanding pain is always a serious warning sign in that it indicates an expanding lesion: for example, a tumour. In most cases of discoradicular interactions, pain has started proximally in the neck, trapezius and/or scapular area before it shifts to the upper limb. However, in younger patients, discoradicular pain may be felt in the arm from the very beginning. The lesion is then called a primary posterolateral disc protrusion. Other instances that start with pain in the arm without previous neck or cervical pain are neurofibromas and root compressions by osteophytic outcrops or metastases.

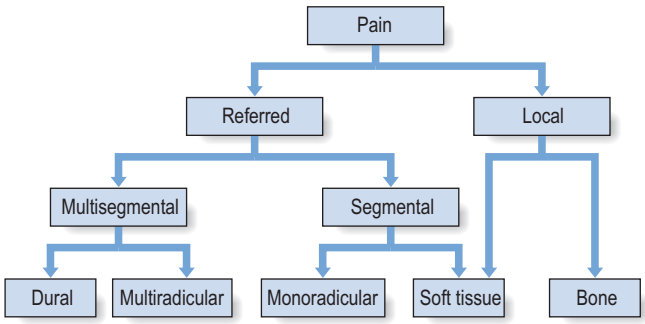

Fig 7.1 • Reference of pain.

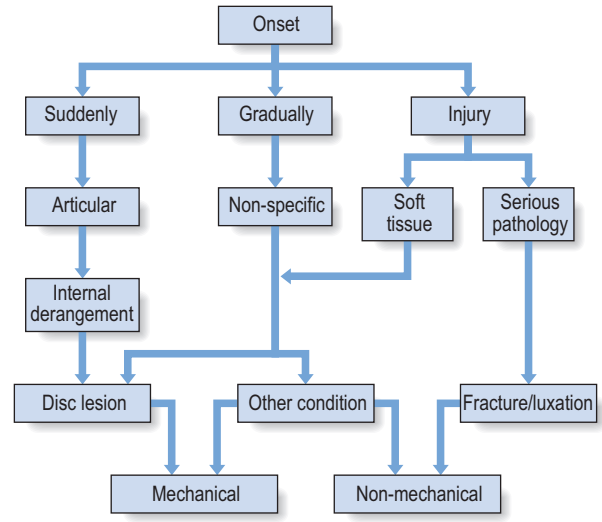

Fig 7.2 • Onset of pain.

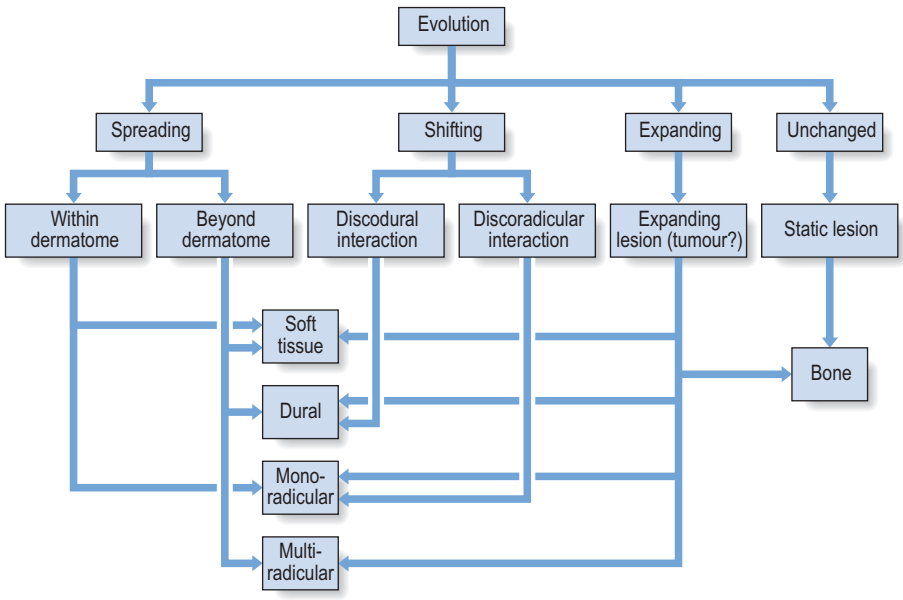

Fig 7.3 • Evolution of pain.

Factors influencing pain

The questions: What brings the pain on? and What makes the pain disappear? are necessary to find out whether or not the condition is related to activity or posture. Most disorders are activity-related: discodural or discoradicular interactions, degenerative conditions, muscular and capsuloligamentous lesions. The type of activity that has an influence on symptoms may help in determining the possible type of lesion. Disc lesions are affected by certain movements, especially towards kyphosis, as well as by certain postures, whereas ligamentous conditions are mostly purely postural. In the latter case, maintenance of positions will aggravate pain and altering the position relieves the pain. When the condition is non-activity-related it is of the non-mechanical type and suggests bony lesions, rheumatoid-type conditions, infections, intraspinal or neurological disorders, or visceral pathologies.

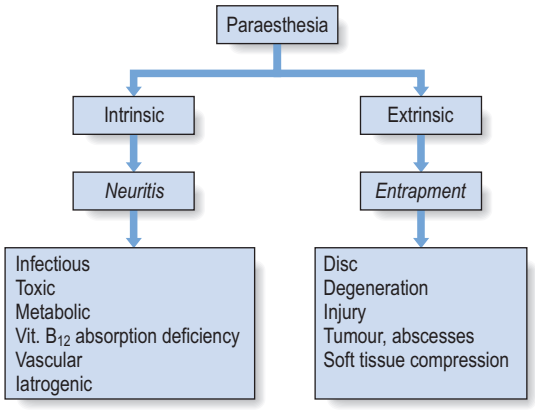

Fig 7.4 • Paraesthesia.

Accompanying symptoms

Coughing or sneezing that causes pain in the trapezius or scapular region is a common symptom in discodural or discoradicular interactions, although any space-occupying lesion in the spinal canal may elicit such symptoms. Pain in the arm on coughing is considered unusual and mandates a closer look: the patient could be suffering from a neurofibroma. Twinges – sudden bouts of pain resulting from movement of the head – are a typical articular symptom. They very much suggest internal derangement in the intervertebral joint – a disc protrusion. Morning pain is typical of arthritic and rheumatoid-type conditions such as ankylosing spondylitis. It also occurs in capsuloligamentous contracture following degeneration ('the elderly man's morning headache' – see Ch. 8). Nocturnal pain is generally accepted as being of the inflammatory type, although mechanical pain may occur when the patient has a poor sleeping posture in which the head is put into a painful position.

Duration of pain

Except in acute torticollis, which recovers spontaneously within a maximum of 10 days, neck pain has no tendency to spontaneous recovery. It may improve after some time but it may also persist. If the cause is internal derangement it usually does improve but tends to recur, certainly in young people. In the elderly the tendency to spontaneous remission is inclined to diminish. Neck pain that not only continues but also increases in intensity is very suggestive of malignancy. Root pain that is the result of a discoradicular interaction has a spontaneous remission over a period of 3–4 months reckoned from the moment of the 'shift': the pain appears in the arm and the scapular pain disappears. Root pain that has not diminished after several weeks is probably the result of another condition, either degenerative or malignant.

Paraesthesia

Paraesthesia is the major differential diagnostic feature distinguishing lesions in nervous tissue and other structures. This may vary from real 'pins and needles' to 'numbness' and may evolve towards a sensory deficit. If paraesthesia is mentioned and is clearly related to the lesion, a nervous disorder or a lesion affecting a nervous structure is present. The problem may be intrinsic ('neuritis') or extrinsic ('entrapment') (see Fig. 7.4).

Intrinsic neuritis may result from infectious (mononucleosis), toxic (ethyl alcohol or lead poisoning), metabolic (diabetes mellitus) or vascular conditions (arteriosclerosis), may have an iatrogenic cause (injection) or may follow vitamin B_{12} absorption deficiency.

When the lesion is the result of *external pressure on the nerve*, the cause must be sought in the intervertebral disc (discoradicular interaction), degenerative conditions (lateral recess stenosis), injury (fractures or luxations), space-occupying disorders (tumour, abscess, haematoma, aneurysm) or other soft tissue compression (fibrous bands, scars).

In nerve tissue compression three major features have to be interpreted: proximal extent, localization and behaviour. The patient has to understand clearly the difference between pain and paraesthesia because both behave differently. The lesion responsible for the development of pins and needles always lies proximal to their *proximal extent*; in other words, the area of paraesthesia is always felt distally to the site of compression. The *localization* of the paraesthesia is defined to a multisegmental area (spinal cord), a segmental area (nerve root) – dermatome – or the territory of a peripheral plexus or nerve. The *behaviour* of the symptoms depends on which part of the nervous system is involved and subsequently on which mechanism is active: spinal cord, nerve root, nerve trunk or nerve ending (see Complete information on pressure on nerves in Ch. 2).

Interpretation of the functional examination

The functional examination of the cervical spine does not cause technical problems. Active, passive and resisted movements

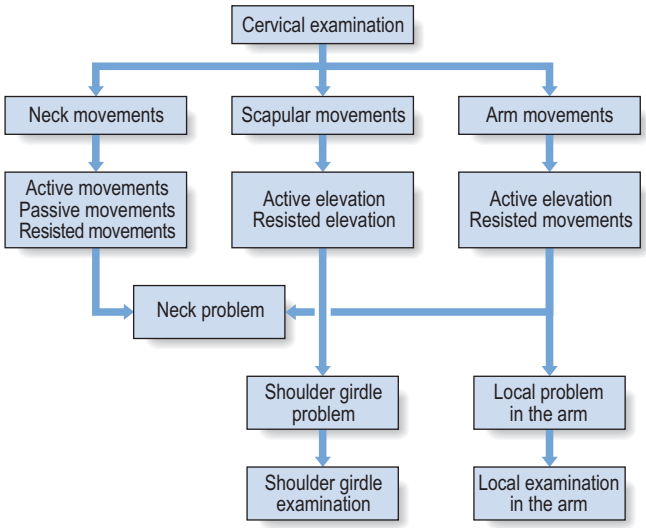

Fig 7.5 • Cervical examination.

are easy to perform and easy to assess; the patient reports pain and the examiner detects limitation, changes in the end-feel and weakness. Interpretation rests on these physical findings and clinical patterns will emerge. Because the examination comprises tests at three different levels – neck, shoulder girdle and arm – it is possible to establish the level at which the lesion should be sought (Fig. 7.5). If signs are found at all three levels, it is logical to look for the lesion 'half-way', i.e. in the shoulder girdle.

If *neck movements* – active, passive and/or resisted – are clearly disturbed, the problem must lie in the neck. When, on the contrary, they are totally negative, a lesion in the neck is very unlikely. A neck that moves perfectly well in all directions, actively as well as passively, with a normal end-feel, cannot have a serious articular problem. Passive movements that cover the full range and are painless indicate that the inert structures are neither painfully stretched nor compressed. Negative isometric tests show that the contractile tissues are also functioning well and that there is nothing wrong with the bone to which they are attached. Pain in the absence of any positive test must therefore be referred or at least of the non-mechanical type: for example, inflammatory.

Positive scapular movements usually point towards a lesion in the shoulder girdle, although it is possible to find slight positive signs in a disorder of the cervical spine; elevation and/or approximation of the scapulae also act as a dural sign. If a shoulder girdle problem is suspected, a complete shoulder girdle examination should follow (see online chapter *Clinical examination of the shoulder girdle*).

Positive arm tests may occur in cervical disorders such as spinal cord or nerve root palsy, but if they provoke pain felt in the arm, they usually indicate a local lesion in the upper limb – shoulder arthritis, subdeltoid bursitis or tendinitis. Elevation of the arm is thus also used to detect alternative causes of pain down the upper limb. Peripheral nervous lesions will also be detected during routine arm tests. In all these instances, an appropriate and full examination of shoulder, elbow or wrist must follow.

Interpretation of neck movements

(Fig. 7.6)

Neck movements are usually disturbed in two ways: either both active and passive movements, or active and resisted movements are positive. If all three sets of tests are positive, the examiner should be careful: a serious condition could be present.

Active and passive movements are positive

Painless active limitation is not usually a reason for consultation unless it interferes with certain activities: for example, reversing a car. Painful limitation will disclose a certain pattern, which can be confirmed by passive testing; the same limitation will be found but the end-feel will add some information about the structure that causes the restriction. Two main 'patterns of limitation' may be recognized: the full articular pattern or any other pattern that deviates from the full pattern.

Limited movement: the full articular pattern

The full articular pattern at the cervical spine is no limitation of flexion, equal degree of limitation of side flexion and rotation, and some or serious limitation of extension (see Fig. 7.7).

Painless limitation with a hard end-feel in an elderly person indicates that the patient has arthrosis of the cervical spine. Apart from some stiffness and a vague ache, this condition does not really cause serious problems.

Painful limitation in a full articular pattern, however, points towards a more serious condition, such as ankylosing spondylitis, rheumatoid arthritis, recent fracture or luxation, postconcussional syndrome, bone disease or a lesion of the upper cervical complex. The history and the end-feel can add more information about the aetiology, but usually further paraclinical investigations are required.

Limited movement: partial articular patterns

A partial articular pattern is a clear asymmetrical presentation of pain and/or limitation. This common finding usually indicates internal derangement (displacement of a disc fragment with discodural or discoradicular interaction). The complete clinical picture must, of course, be compatible with history and end-feel – see Chapter 8.

Specific types of partial articular pattern – the convergent type and the divergent type – may indicate a lesion of a facet joint (see Ch. 7), although it is not impossible for the same pattern to arise in an ordinary disc lesion too.

Full range: partial articular patterns

The same asymmetric pain patterns may occur in the absence of any clinically detectable limitation. This usually has the same significance, although the examiner has to be even more careful because a non-activity-related condition could easily be responsible.

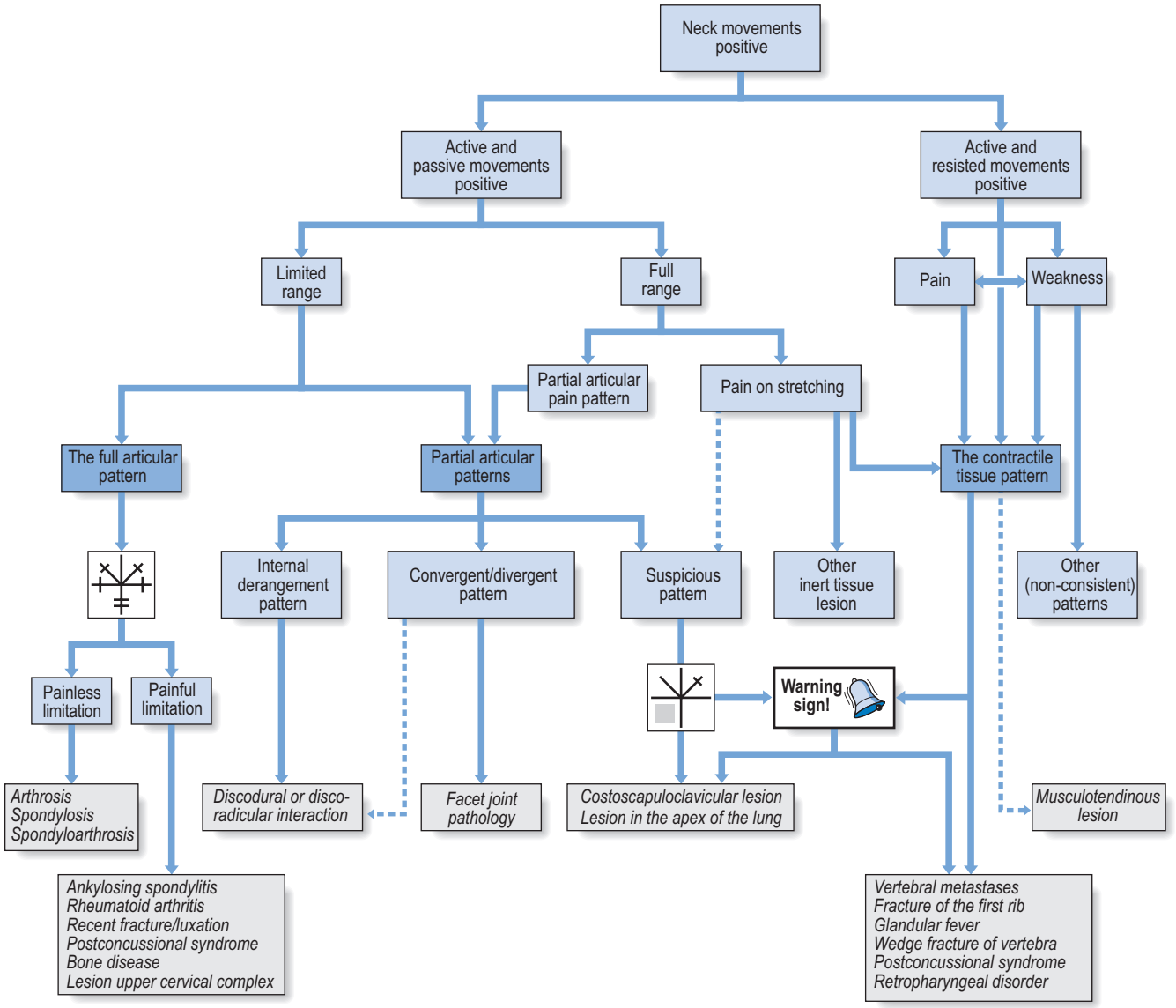

Fig 7.6 • Interpretation of neck movements.

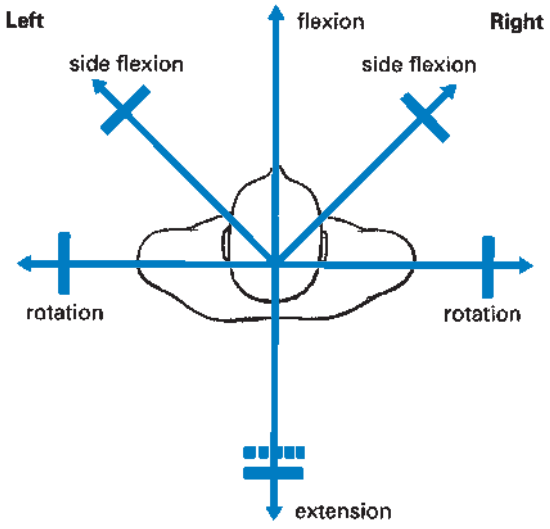

Fig 7.7 • The full articular pattern.

Limited movement: a suspicious partial articular pattern

Warning

The partial articular pattern in which side flexion away from the painful side is the only painful movement is an important warning sign (Fig. 7.8).

A displaced fragment of disc is very unlikely to disturb side flexion while leaving all other movements free. Often, the reason for this particular limitation of movement lies outside the cervical column (neck, mediastinum or apex of the lung).

Active and resisted movements are positive

Isometric testing – movements performed against maximal resistance – may cause pain, weakness or a combination of both. The muscular contraction augments the pressure in the intervertebral joints. It is therefore not totally impossible to find positive resisted movements in an articular lesion, especially when the condition is in an acute or subacute stage – for example, acute torticollis – or when there is a high degree of inflammation. The passive tests, however, should still dominate.

When a clear *contractile tissue pattern* is found – pain on isometric testing in one direction, but also on active and passive stretching in the opposite direction – the possibility of a lesion of a musculotendinous structure, although not so common, should be considered. When resisted movements are clearly more positive than active and passive ones, or when the pattern includes weak movement(s), this has to be considered as a warning sign. Vertebral metastases, fracture of the first rib, glandular fever, wedge fracture of a vertebral body, postconcussional syndrome and retropharyngeal tendinitis or abscess should be excluded.

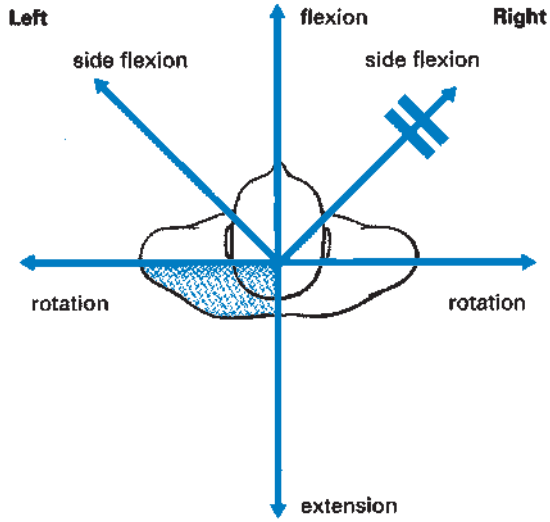

Fig 7.8 • A suspicious partial articular pattern (colour indicates pain).

Warning

- Isometric contraction that is more painful than active or passive movements warrants a closer look.
- Weakness of one or more neck movements indicates a serious disorder. Further investigations are required.

When no clear pattern of pain and/or weakness is detected or when the pattern is inconsistent, diagnosis on clinical grounds is extremely difficult, and the patient should be sent for further investigations.

Interpretation of shoulder girdle movements (Fig. 7.9)

Shrugging the shoulders involves quite a number of different structures: acromioclavicular joint, sternoclavicular joint, upper rib joints, costo- and coracoclavicular ligaments, costocoracoid fascia, upper lung, brachial plexus and muscular structures – elevators are at work, while depressors become stretched.

Pain when the shoulders are shrugged may occur in a lesion of the shoulder girdle that becomes painfully stretched: for example, sternoclavicular arthritis, healed apical tuberculosis, and lesions of the first or second thoracic nerve root, subclavius, costocoracoid fascia or first costotransverse joint. It is important to remember that this test also stretches the thoracic dura mater and thus provokes pain in a thoracic discodural interaction.

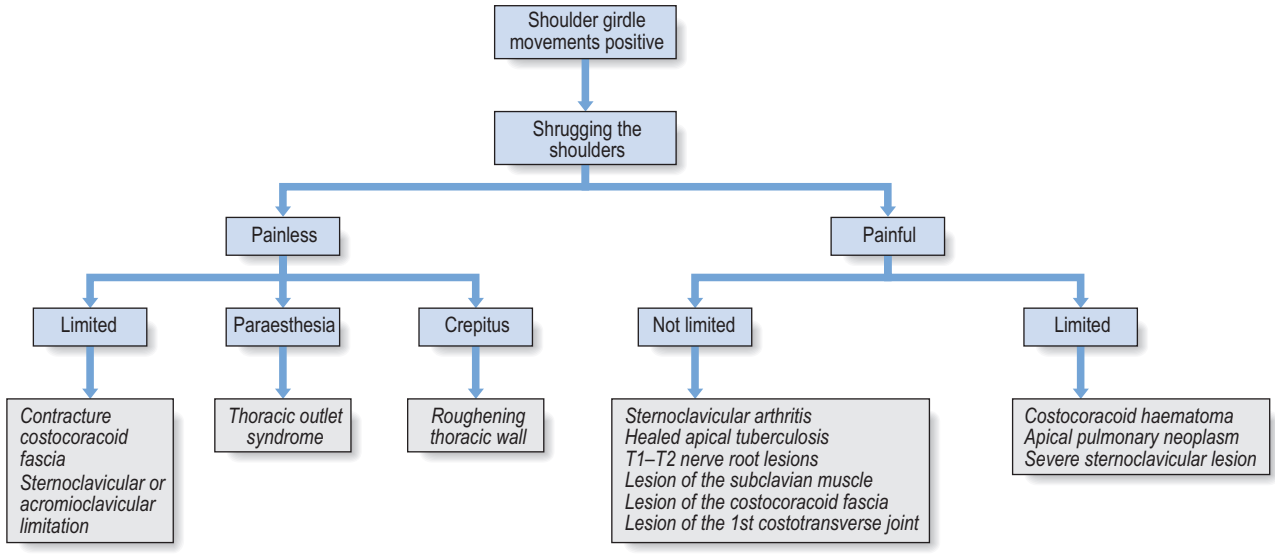

Fig 7.9 • Shoulder girdle movements.

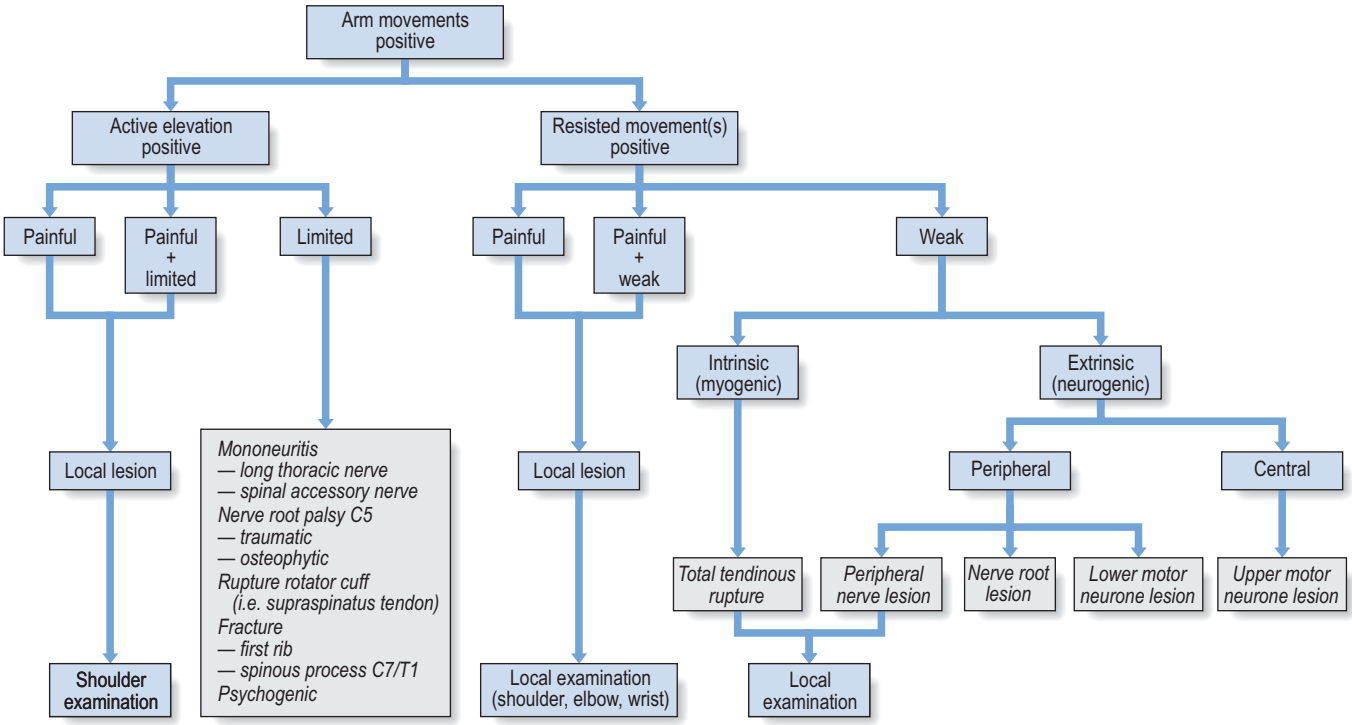

Fig 7.10 • Arm movements.

Painless limitation shows that the mobility of the scapula in relation to the thorax is impaired, either by contracture of soft tissue at the shoulder girdle or by gross lesions at the sternoclavicular or acromioclavicular joint.

Painful limitation is rare and is indicative of a lesion, such as a haematoma in contact with the costocoracoid fascia, an apical pulmonary neoplasm or a severe sternoclavicular lesion.

Pins and needles in one or both hands on sustained movement suggests the thoracic outlet syndrome. Crepitus indicates roughening of the posterior thoracic wall (see online chapter *Disorders of the inert structures*).

Interpretation of arm movements (Fig. 7.10)

Active elevation of the arm is positive

When active elevation is *painful* but not limited, the examiner should ascertain where the pain is felt. If positive neck signs have been found and the shoulder movement hurts in the neck region, this is probably the result of 'transmitted stress' and thus irrelevant. If the movement hurts in the shoulder and/or down the arm, the problem is local and full examination of shoulder, elbow or wrist should follow.

Active elevation that is *painful and limited* is probably the result of a local condition in the shoulder girdle or in the shoulder. A proper examination of shoulder girdle and arm should follow and disclose the lesion.

A number of conditions *limit* active but not passive elevation of the arm. Apart from more generalized disorders, the following neurological lesions can be responsible: mononeuritis of the long thoracic or spinal accessory nerves, osteophytic or traumatic root palsy of C5. Tendinous ruptures, especially of the supraspinatus tendon, may also limit arm elevation, as does fracture of the first rib or of the spinous process of C7 or Tl. Psychogenic pain is another possibility.

Resisted arm movements are positive

When one or more isometric tests of the arm are painful and/or weak, the lesion must be sought in shoulder, elbow or wrist. The appropriate local examination then leads to a diagnosis.

Painless weakness is sometimes difficult to detect. The patient is asked to perform a maximal contraction and the examiner assesses the strength. Weakness may vary from a subtle paresis, typical of nerve root compression by a fragment of disc, to complete paralysis, as found in more serious neurological disorders or in complete tendinous rupture.

The examiner should be able to establish whether the condition is myogenic or neurogenic. Neurological weakness either has a central cause – upper motor neurone lesion – or is the result of a peripheral nerve lesion – lower motor neurone, nerve root, plexus or local peripheral nerve. When the weakness is segmentally distributed, the problem must lie in the cervical spine. In more generalized neurological conditions (e.g. diseases of the central nervous system and cerebrovascular accidents), the weakness is more extensive. Painless myogenic weakness is probably the result of a complete rupture of a tendon.

The most common condition causing weakness down the arm is nerve root compression – usually C7 – by a displaced fragment of disc. In orthopaedic medicine the most important non-mechanical causes of weakness down the arm are mononeuritis of the suprascapular nerve, vertebral metastases, Tl nerve root lesion, neuralgic amyotrophy, traction palsy of C5 and neurofibroma.

8

Mechanical disorders

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Mechanical disorders of the cervical spine affect the structures of the locomotor system. They are often related to the consequences of the ageing spine. Degenerative changes at the level of the intervertebral discs may lead to disc displacements and also to anatomical and biomechanical changes in ligaments, capsules, nervous tissue and vascular structures. In addition to this degeneration being responsible for the development of certain disorders, injuries and overuse may also lead to soft tissue lesions in the region of the neck (Box 8.1).

Mechanical disorders are activity-related, which means that their symptoms are influenced by movement and/or posture. Questions in the history taking, such as 'What brings your symptoms on?' or 'What makes your symptoms disappear?' elicit answers that may suggest the type of condition from which the patient suffers.

Degeneration and anatomical changes

Degenerative changes in the cervical spine are part of the normal ageing process and are almost ubiquitous in older people.[1,2] Very often they remain asymptomatic.[3,4] Therefore diagnoses such as 'degeneration', 'arthrosis', 'osteophytosis', 'spondylosis' or 'spondylarthrosis' based on incidental radiological findings are still too easily made. Throughout the literature, a discrepancy between the discovery of structural changes and the presence of symptoms is a consistent finding.[5]

Ageing of the cervical spine

As a result of weight bearing in bipedal humans, the axial spine is submitted to increased forces acting on the different structures. The disc, being entirely avascular, suffers most from this situation; degeneration starts quite early in life and progresses with age. The degenerative process in the intervertebral disc is followed by other structures of the spine, such as bone, ligaments, capsules and zygapophyseal joints.

Ageing of the cervical disc

In healthy individuals the cervical intervertebral disc has a structure which is very similar to that of the lumbar disc: an annulus fibrosus which contains a nucleus pulposus. As long as the disc is not submitted to excessive asymmetrical forces, the hydraulic mechanism within the disc functions perfectly. It becomes deficient as the result of excessive forces occurring during everyday activities, or following absence of movement. The annular fibres become strained and sprained, and the nucleus starts to dry out. This is a characteristic sign of disc degeneration.

It has been shown that, in the first two decades of life, lateral tears occur in the annulus fibrosus. They tend to develop joint-like structures – the uncovertebral joints – that then begin to undergo transformation.[6] In the second and third decades, the lateral tears enlarge towards the medial part of the disc, often ending in a complete transverse splitting of the disc into equal halves. These anatomical changes cause instability of the

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Box 8.1

Mechanical disorders of the cervical spine

Disc disorders
- Disco-dural interactions
- Disco-radicular interactions

Degenerative disorders
- With localized pain
  - Arthrosis at the upper two cervical joints, leading to ligamentous contracture: morning headache in the elderly
  - Subacute arthritis of the atlantoaxial joint
  - Arthrosis at a facet joint
- With radicular pain: osteophytic root compression
- Cervical spondylotic myelopathy

Capsuloligamentous disorders

Musculotendinous lesions
- Lesions of the semispinalis or splenius capitis muscle
- Lesions of the longus colli muscle (retropharyngeal tendinitis)

disc and favour the possibility of cartilaginous displacements, either of the annulus fibrosus or of the nucleus pulposus (see below: disc displacements).

In the fourth and fifth decades, the nucleus pulposus dries out further because of the loss of proteoglycan matrix with, as a result, diminution of the height of the intervertebral space and flattening of the lordotic curve. The uncovertebral joints start to bear weight and tend to flatten. Degenerative changes at this level threaten the spinal nerve and the vertebral artery.

From the fifth and sixth decades on, the disc tends to collapse, further diminishing its height. The borders of the vertebral bodies come into apposition, with the formation of hyperostosis and osteophytes as a reactive result. The spine stiffens and the articular segments stabilize again.

See Chapters 31 and 32 for an extensive explanation of the function and behaviour of the lumbar disc, which is highly comparable to the situation in the cervical spine.

Consequences of disc ageing for surrounding structures

Effects on the ligaments

Instability of the intervertebral joints results in instability of the entire segment. The excessive and irregular movements of the related vertebrae lead to traction at the site of osseous attachments of the annulus fibrosus to the vertebral body. Lifting of the anterior and posterior ligaments results in spur formation at the anterior and posterior borders of the vertebral body, and subsequently to the formation of osteophytes. In addition, the ligamentum flavum is submitted to traction forces. It becomes deformed and enlarged, and will buckle into the spinal canal during extension.

Effects on the facet joints

The prevalence of cervical facet joint degeneration is very high in individuals aged 50 years and more, with a tendency to increase in severity with age. Diminution of the height of the disc and the subsequent reversal from lordosis to kyphosis result in a greater transmission of shearing forces to the facet joints. They start to bear weight and this furthers the degenerative process. This is characterized by fibrillation and erosion of the articular cartilage, partial or complete denudation of the cartilage, and new bone formation.[7] Later in life, subperiosteal osteophytes with periarticular fibrosis develop, resulting in a gross decrease in mobility.[8]

All levels of the middle and lower cervical spine are affected to almost the same degree, whereas in the lumbar spine there is an increase in degeneration towards the lower levels.[9]

Effects on the uncovertebral joints

With progressive diminution of disc height, the bony protuberances about the uncovertebral articulations approach each other and are pressed together. The intervening (pseudo-) articulations degenerate, leading to osteophytes that enlarge the joint surfaces and project from the posterior edge of the vertebral body into the disc space and the intervertebral foramina. Compromise of adjacent neurovascular structures (nerve root and vertebral artery) is a potential complication.

Effects on the spinal canal

Circular disc displacement, as is seen when the disc has degenerated entirely, may further the development of osteophytic outcrops on all sides, and thus also into the spinal canal, threatening the spinal cord.[10] Once the bony parts formed by the uncovertebral joints touch each other, further loss of height can only happen anteriorly. Because of the decrease in height in the anterior column of the cervical spine, cervical lordosis may diminish and even disappear, resulting in the typical position of the degenerated neck – the chin projected forwards. Retrolisthesis in the mid-cervical area (C3–C6) is a common finding in spondylarthrosis. Also, buckling of an enlarged ligamentum flavum narrows the spinal canal, especially during extension of the neck.[11,12]

Effects on the intervertebral foramen

The association of diminished intervertebral space, osteophyte formation at both the uncovertebral and zygapophyseal joints, and hypertrophy of the facet joint capsules causes diminution of the intervertebral foramen and eventual neural foraminal encroachment.[13] Further diminution of the foraminal space is caused by the backwards slip of the upper vertebra during extension.[14]

Radiological changes

Degenerative changes of the cervical spine are common in both symptomatic and asymptomatic adults. At the age of 50, spondylosis is visible on plain X-rays in more than 50% of asymptomatic people.[15] A magnetic resonance imaging (MRI) study on 500 asymptomatic subjects could detect disc

degeneration in 17% of discs in men and in 12% of those of women in their twenties, and 86% and 89% of discs in both men and women over 60 years of age. Posterior disc protrusion with demonstrable compression of the spinal cord was observed in 7.6% of subjects over 50 years of age.[16,1] The clinical question is whether the patient's symptoms are caused by the changes that are seen on imaging. Unfortunately, even the most sophisticated imaging studies do not show pain, but only structural abnormalities that may or may not be the cause of the patient's symptoms.[4,17]

Pathology

Disorders of the disc: disc displacements

Symptomatic disc displacements in the cervical spine are more common than is generally believed. They can occur at any age from childhood onwards but present with different clinical syndromes, depending on the age group.[18]

Cartilaginous displacements are most frequent at the C6–C7 and C5–C6 joints. They are occasionally found at the C4–C5 or C7–T1 joints, and rarely at the C2–C3 or C3–C4 joints.[19] Nuclear prolapses are very uncommon, except in young people. It was to Cyriax's credit that he recognized the clinical pictures associated with disc displacements and made an inventory of symptoms and signs. A distinction can be made between posterocentral and posterolateral displacements resulting respectively in discodural and discoradicular interactions. It is important to remember that diagnoses are made on clinical grounds, following the patient's history and the functional examination. Technical investigations that rely only on anatomical findings lead very easily to misinterpretation.[17] They can sometimes be used to confirm a diagnosis, rather than to make one. Discs may displace in two ways: either they move posteriorly to compress dura and/or cord, or they move posterolaterally to compress the nerve root.

Posterocentral displacement

Posterior migration of disc tissue beyond the posterior limits of the intervertebral joint space causes pressure on the posterior longitudinal ligament and, via the ligament, on the anterior part of the dura mater. This produces multisegmental pain with dural reference, mostly in the trapezius and scapular area but possibly towards the head and/or pectoral area (see Ch. 7). The pain is the result of a discodural interaction: irritation of the anterior structures in the spinal canal is responsible for the dural symptoms. Pure posterocentral compression gives rise to pain felt centrally and/or bilaterally. Compression just laterally from the midline will result in pain that is felt unilaterally but is still multisegmental. The internal derangement in the intervertebral joint provokes articular signs demonstrable on functional examination.

Posterolateral displacement

If the extruded disc material runs into the nerve root canal, discoradicular interaction occurs. This posterolateral migration of disc tissue causes pressure on the nerve root, which results in a series of symptoms: first segmental pain following compression of the dural sheath (root pain), then segmental paraesthesia and/or neurological deficit as the result of the parenchyma becoming pinched. The disc displacement may be primary or secondary.

Secondary posterolateral displacement

This is the most common situation. The patient has suffered from a few posterocentral displacements with neck, trapezius and/or scapular pain in the past. During the most recent attack, as the disc fragment moves from the centre to one side, the pain shifts from the neck to the upper limb. Segmental root pain builds up while the multisegmental neck pain disappears.

Primary posterolateral displacement

From the outset the pain is felt in the forearm. There has been no previous neck pain because originally there was no compression of the dura mater but rather of the nerve root directly. The segmental pain can be accompanied by paraesthesia and/or neurological deficit. Because this condition is rather uncommon, differential diagnosis should be made with non-discal nerve root compression or with intrinsic conditions of the nervous system.

Stages of disc displacement

Disc displacements also behave according to their actual status of degeneration. Cyriax described seven stages of disc lesions, each one typical of a certain age group and characterized by a typical history (Fig. 8.1).[20]

Stage 1

Acute torticollis in adolescents or young adults: acute neck pain, with total immobilization and gross deviation. This is

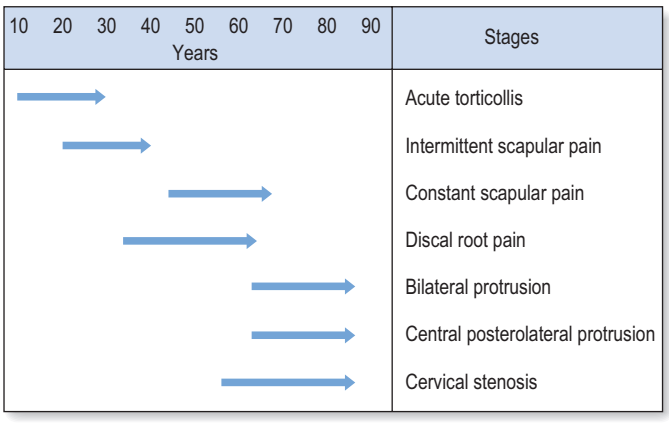


Fig 8.1 • Stages of disc displacement.

the result of a large discodural interaction, usually of the nuclear type. The lesion undergoes spontaneous cure within 10 days.

Stage 2

Intermittent scapular pain, mostly unilateral but not always on the same side. The patient is in his/her late 20s or older. The symptoms may last several weeks.

Stage 3

Constant scapular pain in a patient over 50. Due to developed spondylosis and stiffness of the neck, there is no spontaneous reduction of disc material.

Stage 4

Pain in the upper limb after initial scapular pain. The patient is between 35 and 65. The pain typically increases during the first 2 weeks then remains unchanged over the next 4–8 weeks, recovering spontaneously after 8–12 weeks. There may be paraesthesia and neurological deficit.

Stage 5

In elderly patients, a bilateral protrusion may cause aching in both upper limbs and paraesthesia in all the fingers.

Stage 6

Constant bilateral aching in head, neck and scapulae may be experienced in elderly patients as the result of a fixed posterocentral protrusion that leads to constant discodural interaction.

Stage 7

The disc compresses the spinal cord. This results in paraesthesia in the hands and/or feet.

Osteophytes may obliterate the anterior spinal artery, leading to progressive paraplegia.

Findings on examination

It is believed that disc degeneration and disc displacements are of themselves painless events. It is only when pain-sensitive structures in the neighbourhood pick up on the disc abnormality and translate mechanical pressure and inflammatory changes into pain that signs and symptoms originate. The reader is referred to Chapter 33 – the 'dural concept' – for a thorough description of this dural hypothesis.

Disc displacements in the cervical spine will give rise to the same set of signs and symptoms as in the lumbar spine. However, in contrast to the lumbar spine, disc lesions at the cervical spine may threaten the spinal cord. Therefore, not only the usual dural, articular and root signs and symptoms, but also cord signs and symptoms may be present here and must be sought.

Dural symptoms

When a fragment of disc (at any level) protrudes posterocentrally, it may compress the dura mater either in the midline, which results in central or bilateral pain, or slightly to one side, leading to pain felt unilaterally. The dura mater, being a multisegmentally innervated tissue, translates this compression into multisegmental pain (see Ch. 1). Pain reference from the cervical dural mater may thus spread up to the head and down to the mid-thoracic region, and may be felt anywhere in this area (Fig. 8.2). However, the pain is most commonly experienced in the region of neck, trapezius and scapula (Fig. 8.3a). Occasionally it is felt in the clavipectoral area or in the axilla (Fig. 8.3b).

Patients often mention a tender spot, usually somewhere at the superior border of the trapezius, which they identify as the source of their pain. On palpation the examiner indeed finds localized tenderness. However, during the examination, movements designed to test the trapezius muscle are found to be negative. Furthermore, the tender spot may shift during neck manipulation and disappears when a full and painless range is

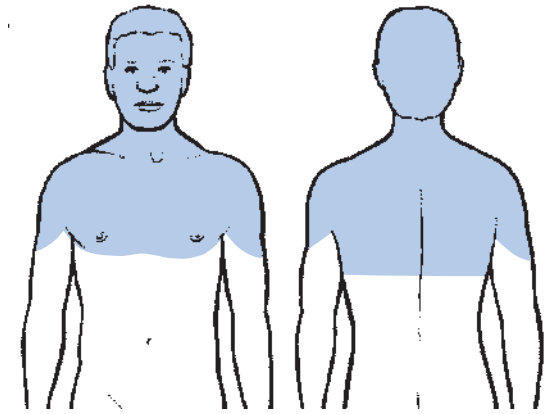


Fig 8.2 • The boundaries of multisegmental reference of pain of cervical origin.

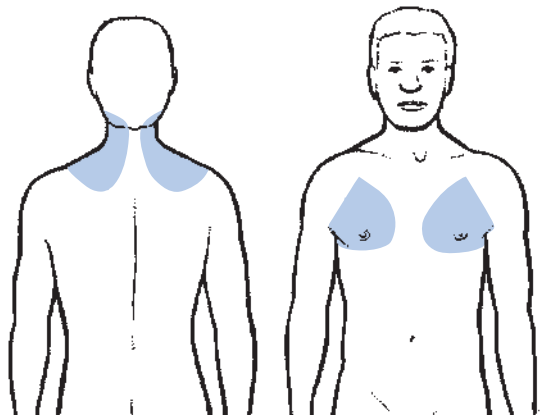


Fig 8.3 • Multisegmental pain: (a) the most common distribution involves trapezius and interscapular area; (b) less commonly, pain is felt in the pectoral and axillary areas.

obtained. This confirms that the tenderness is clearly a referred phenomenon (see Ch. 1) without localizing or diagnostic value. This multisegmental tenderness is one of the dural symptoms and results from dural compression at any level.

In contrast to a lumbar discodural problem, a cough seldom hurts. Very exceptionally, swallowing may be uncomfortable, although the mechanism causing this symptom is unclear.

Dural signs

Dural signs are not found on examination. Although neck flexion stretches the dura mater, the pain very often experienced during this movement must be regarded as an articular sign rather than a dural one. Neck flexion can only be regarded as being a dural test if it stretches the dura from a distance, which is not the case here. There is also no equivalent to the straight-leg raising test in the lumbar examination where a simple movement (hip flexion with an extended knee) allows differentiation between dural pain, root pain and muscular tightness. Some nerve roots (C4–C6) are tethered to the gutter of their respective transverse process,[21] but apparently not to the foramen.[22] Some authors have developed and discussed 'upper limb tension tests' (ULTT) as a valuable examination procedure for patients with neck problems with or without radiation down the upper limb.[23,24] The aim would be to create stress, transmitted to the structures in the spinal canal via the peripheral nerves (median–radial–ulnar). The peripheral nerves in the upper limb however, have, unlike those in the lower limb, a more complex course. It is therefore more difficult to carry out 'pure' movements and, although these tests stress the neurological tissues, they also stress some contractile and inert tissues in neck, shoulder girdle and arm. Because of their low specificity, [25–27] they are not integrated into the standard functional examination of the cervical spine.

Articular symptoms and signs

The symptoms are activity-related: pain is elicited or influenced by movements, activity and posture. Sometimes there are twinges during movement of the neck.

On examination, a partial articular pattern of internal derangement is found: some active movements are painful and limited or cause pain at the end of range, while others are negative or clearly less positive. The asymmetrical pattern that occurs may vary from very subtle, with hardly any limitation, to very pronounced with some movements completely blocked, forcing the head in a deviated position. A minimal partial articular pattern (Fig. 8.4a) emerges when the internal derangement is minor and the discodural interaction slight. It is hypothesized that pronounced signs result from a severe discodural interaction (Fig. 8.4b). Passive movements usually hurt more than active ones, though cases may be encountered in which passive movements ease the pain. Resisted movements are negative, except perhaps resisted flexion. The latter may slightly increase the pain in more acute cases, presumably as a result of the consequent compression strain on the affected joint.

When neck flexion elicits pain in the upper thoracic area, further examination of neck, shoulder girdle and thorax must

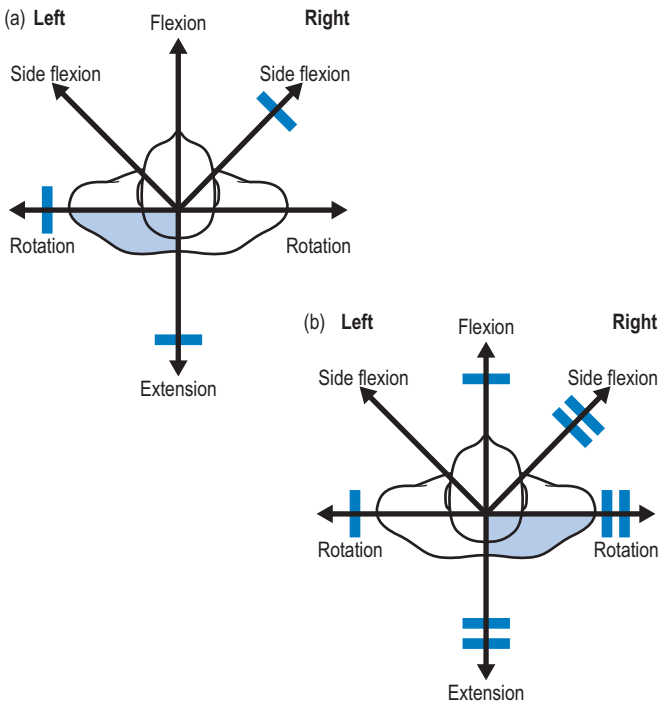


Fig 8.4 • Examples of partial articular patterns: (a) minimal; (b) severe (colour indicates pain).

differentiate between a cervical lesion with pain reference to the upper thorax and a lesion in the shoulder girdle or upper thoracic spine. The movement is an articular sign for the cervical spine but is also regarded as a dural sign for the thoracic spine. During neck flexion the dura mater is stretched and drawn forward against an eventual upper thoracic protrusion (see Ch. 25).

A painful arc may be found on active or passive testing. This sign sometimes occurs during rotation movement in patients with unilateral symptoms and during extension in patients with central or bilateral pain, and is pathognomonic of a disc lesion.

If only one movement proves positive, it is usually rotation towards the painful side. Caution must be taken when the only painful movement seems to be side flexion away from the painful side. This sign may occur in any costoscapuloclavicular lesion, but may also suggest a lesion at the apex of the lung. Further investigations are necessary.

In discodural problems either the normal leathery end-feel is expected or, in more severe cases, the more typical end-feel of muscle guarding ('crisp'). This is clearly unlike the hard end-feel of arthrosis or the muscle spasm end-feel of more serious disorders.

Root symptoms and signs

By analogy with the lumbar spine, where backache may evolve into sciatica, cervicoscapular pain may be replaced by brachial pain. The original multisegmental cervicoscapular pain disappears and severe and strictly segmental pain down the arm supervenes. The examiner must be aware that patients sometimes omit to mention the original pain in the trapezius or scapular area but concentrate only on the severe symptoms at

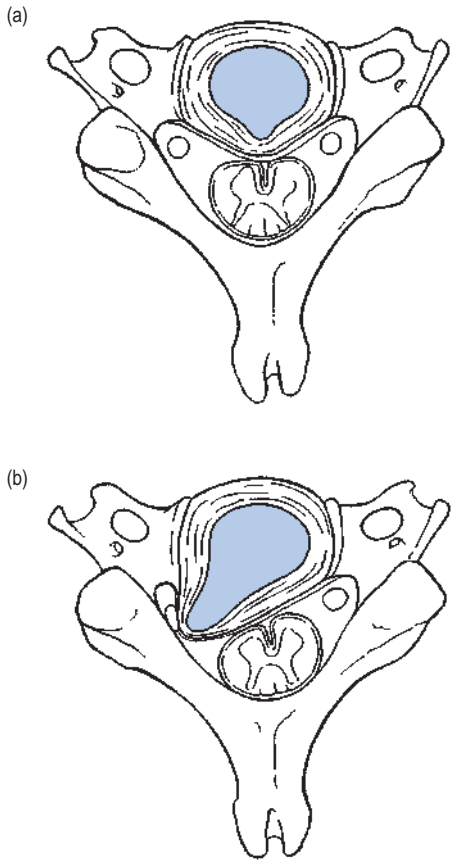


Fig 8.5 • Pressure on the dura mater (a) ceases when the disc moves laterally and compresses the nerve root (b).

presentation. This change in pain localization is the result of the disc moving laterally (Fig. 8.5). Pressure on the dura mater ceases when the dural sleeve of the nerve root becomes compressed and radicular pain appears. Increasing compression of the nerve root is translated into increasing symptoms and signs. Paraesthesia and/or numbness point towards involvement of the nerve root fibres and are usually felt in the distal part of the corresponding dermatome.

Conduction may become impaired, leading to neurological deficit, which manifests as a minor motor palsy (paresis), a sensory palsy (cutaneous analgesia) or both. Reflexes may become sluggish or absent. Because the cervical nerve roots emerge horizontally, the symptoms and signs are strictly monoradicular, in contrast to the situation in the lumbar spine where the nerve roots have an oblique course and one disc displacement may compress several nerve roots. However, the C6 and C7 nerves are sometimes slightly more oblique and may become compressed by two discs.

Additional tests to provoke radicular pain can be added. During the Spurling test, the patient side-flexes the head to the affected side first, followed by the unaffected side. Meanwhile the examiner presses straight down on the head.[28] A test result is classified as positive if pain radiates into the arm towards which the head is side-flexed during compression. Alternatively the head is rotated and side-flexed to the same side during compression.[29] This is called the 'maximal cervical compression test'. Great care is needed while performing this test as it also compresses the vertebral artery.

Table 8.1 Findings on examination

	Symptoms	Signs
Dural	Multisegmental pain Multisegmental tenderness (pain on coughing)	
Articular	Pain on movement/posture Twinges	Deviation Partial articular pattern Painful arc Crisp end-feel
Root	Segmental pain Segmental paraesthesia Segmental numbness	Motor deficit Sensory deficit Sluggish reflex
Cord	Paraesthesia in all limbs Lhermitte's sign Sensory disturbance in feet	Gait disturbance Spastic palsy lower limbs Hyperreflexia – clonus Positive Babinski–Hoffmann

Cord symptoms and signs

In posterocentral disc protrusion, the spinal cord may also become compressed. Pressure on the cord itself is not painful. It leads to multisegmental paraesthesia, usually felt in both hands and/or feet and very often provoked or influenced by active or passive neck flexion. Sensory disturbance soon supervenes. In more severe cases, the gait will become disturbed, the plantar reflex may become positive (Babinski's sign) and the patient may develop spasticity, incoordination and extensive weakness in the lower extremities.

Findings on examination are summarized in Table 8.1.

Common syndromes

The following clinical pictures are all caused by cervical disc protrusions. Symptoms and signs are determined by size, localization and degree of degeneration of the displaced discal fragment and the nature of the involved structures.

Acute torticollis: unilateral pain with asymmetrical picture

Unilateral acute torticollis is caused by a discodural interaction that is quite common between the ages of 15 and 30. An acute torticollis under the age of 12 is rarely caused by a disc (see Differential diagnosis).

History

Most patients wake in the morning with a stiff neck. Pain and stiffness increase as soon as they adopt an upright position. Any attempt to move the neck results in unilateral pain, radiating to trapezius and/or upper scapular area. Occasionally the symptoms come on suddenly as the result of a certain

movement (e.g. bending over a washbasin), trivial trauma (e.g. a blow to the head[30]) or coughing.

Neck movements make matters worse, and even arm movements may cause considerable pain. The pain diminishes when lying down with the neck supported but it is very difficult to change position and the patient has to hold the head firmly between the hands to do so.

Previous attacks, not necessarily with symptoms on the same side, may be mentioned. Many patients also feel giddy during an attack of acute torticollis.

Inspection

The patient holds the head in an asymmetrical position. In acute torticollis that results from a disc lesion, the deviation is usually purely lateral, in most cases away from the painful side and without any rotatory component. Slight deviation in flexion occasionally occurs.

Examination

On examination, gross and obvious articular signs are found: a striking and extremely painful limitation of one rotation and one lateral flexion towards the same side, usually the painful side. Often no movement is possible in either of these directions (Fig. 8.6). The other movements are much less painful and are scarcely limited, except possibly extension. The end-feel is empty or one of severe muscle guarding.

Resisted movements may be painful and even weak. The patient stops the contraction because muscular action causes a momentary change of pressure in the intervertebral disc and intensification of pain.

Arm movements may also be impaired. On active elevation of the ipsilateral arm, a false painful arc may be present and in very acute cases the patient may even be unable to bring the arm up voluntarily above the horizontal. These secondary signs are clearly the result of transmitted stress: when the arm reaches the horizontal, the muscular effort is at its greatest and secondary tautening of the cervical muscles takes place, leading to such pain that the arm falters and a false shoulder sign appears. There are no root or cord signs.

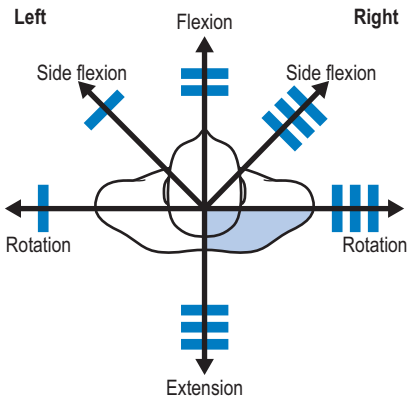


Fig 8.6 • The partial articular pattern in acute torticollis (colour indicates pain).

Diagnosis

The pattern of limitation is diagnostic. Acute torticollis is the most striking example of a partial articular pattern, which indicates that part of the joint is blocked by an intra-articular displacement. In most instances the displacement is nuclear: the nucleus has oozed out slowly during the night, while the patient has being lying for some hours with the neck in side flexion or rotation. The dura mater becomes compressed and irritated, which causes the cervicoscapular pain. A sudden onset suggests an annular displacement. It usually occurs in patients over 30. This mechanism was hypothesized by Cyriax more than 50 years ago and was recently proven by two MRI studies. Maigne *et al* performed MRI in a 15-year-old male adolescent, a few hours after the onset of torticollis. Increased signal intensity compatible with a fluid collection was seen in the right uncovertebral region at C2–C3, causing excessive lateral pressure and pushing C2 outwards. A new MRI taken 3 weeks after resolution of symptoms was unremarkable.[31] Another MRI study performed by Gubin *et al* in ten children and adolescents with acute torticollis showed similar findings: high-intensity zones were seen near uncovertebral zones C2–C3 or C3–C4. The zones were always on the side where the patients felt pain and disappeared spontaneously after a few days.[32]

The characteristics of acute unilateral torticollis are listed in Box 8.2.

Box 8.2

Summary of unilateral acute torticollis

Definition

- Acute torticollis is a sudden attack of severe cervicoscapular pain, owing to a large posterior shift of disc material, compressing the dura mater and resulting in a discodural interaction

Onset

- Sudden: annular (age >30)
- Slow: nuclear (age <30)

Symptoms

- Articular
  - (Twinges)
  - Pain on active movements
- Dural
  - Dural pain: large reference

Signs

- Deviation in side flexion (or flexion)
- Gross partial articular pattern

Treatment

- >30 years: manipulation under traction
- <30 years: progressive sustained movements

Spontaneous recovery

- Takes 7–10 days

Differential diagnosis

Discogenic torticollis is the most common form of twisted neck or 'wry neck' and should be differentiated from many other types of torticollis.

Torticollis in infancy and childhood

This can be congenital or acquired. Congenital types include those resulting from osseous, muscular or neurogenic factors.

Osseous lesions are mostly anomalies of the atlas,[33] such as hemiatlas, partial atlantic aplasia[34] or partial atlanto-occipital fusion,[35] and Klippel–Feil syndrome. Klippel–Feil syndrome is congenital fusion of two or more cervical vertebrae. The classic clinical triad is: lower neckline, short neck, and restriction of head and neck movements.[36] In patients with moderate involvement, this classic triad may not be seen and sometimes there is only some scoliosis deformity or moderate torticollis.[37]

Congenital muscular torticollis (CMT) is caused by shortening of the sternocleidomastoid muscle. This leads the head to bend toward the affected side and the chin to turn to the opposite side, the so-called cock robin position of the neck. CMT is the third most common congenital musculoskeletal anomaly after dislocation of the hip and clubfoot,[38] with a reported incidence of 0.3–1.9%.[39] Various theories have been proposed but the true aetiology of torticollis remains uncertain.[40] As a result of fibrotic changes of the sternocleidomastoid muscle, the unilateral contracture may subsequently result in plagiocephaly, skull and facial asymmetry.[41] When diagnosed early, CMT can be managed conservatively and seldom requires surgery.[42] In fact, a simple stretching programme leads to spontaneous resolution in most patients.[43] In children older than 1 year, corrective surgery has both cosmetic and functional benefits, the best outcomes being obtained between the ages of 1 and 4. After the age of 5, the form and efficacy of treatment are controversial.[44]

Acquired torticollis in children should always be taken seriously, as it may result from a number of underlying causes, some of which are severe and life-threatening. Spontaneous subluxation of the atlantoaxial joint following peripharyngeal inflammation is known as *Grisel's syndrome*. Children between 5 and 12 years are most frequently affected. However, the condition has been reported in patients ranging from infancy to the seventh decade of life.[45] Torticollis may occur shortly after the onset of the underlying infection or it may follow mild trauma to the neck. The syndrome has been reported in association with rhinopharyngitis,[46] cervical osteomyelitis, rheumatic conditions, and following surgical procedures such as tonsillectomy or adenoidectomy,[47] choanal atresia repair and mastoidectomy. It is hypothesized that distension and abnormal laxity of the ligaments surrounding the atlantoaxial articulation result from direct spread of inflammation from the pharynx and nasopharynx.[48] This leads to C1–C2 instability with subluxation and (sometimes) devastating neurological sequelae (between 10 and 15% of patients have neurological signs or symptoms, with serious consequences including quadriplegia and sudden death[49]). The condition begins with a spasm of the irritated sternocleidomastoid muscle. This contraction leads to a typical position of the head, which is easily differentiated from the position in acute discogenic torticollis. In a disc lesion, the deviation is purely in lateral flexion or flexion or a combination of both. When a spasm of the sternocleidomastoid muscle is responsible, the neck is held in flexion, side flexion towards and rotation away from the painful side (signe du merle or 'blackbird's sign') (Fig. 8.7).[50]

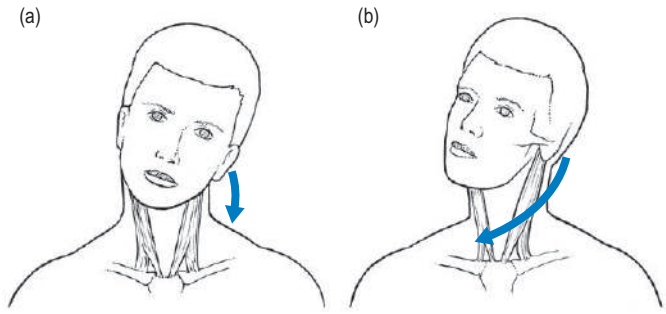


Fig 8.7 • (a) Discogenic torticollis – the deviation is pure in lateral flexion. (b) Grisel's syndrome: spasm of the sternocleidomastoid muscle forces the head in side flexion towards and rotation away from the painful side.

The diagnosis of atlantoaxial subluxation requires radiological investigation. Plain radiographs of the cervical spine may show asymmetry between the facet joints on the anteroposterior projection and an increased atlanto–dens interval on the lateral projections.

Initial treatment consists of collar immobilization and broad-spectrum antibiotic therapy. In the case of persistent torticollis with no improvement following 1 week of conservative treatment, the patient should be referred for neurosurgical consultation.

Benign paroxysmal torticollis is a relatively uncommon, self-limiting condition occurring during infancy. It resolves by the age of 2–3 years. Periodic episodes of torticollis may randomly alternate from side to side and be associated with vegetative symptoms. The aetiology is unknown and no treatment is effective.[51]

Spasmodic torticollis

Spasmodic torticollis or cervical dystonia is the most frequently occurring focal dystonia, with an incidence of 8.9 per 100 000 people.[52] Dystonia is defined as sustained, involuntary muscle contractions, which often cause twisting or repetitive movements or abnormal postures. The origin of the symptoms is still unclear. The condition often results in cervical pain and disability as well as impairments affecting postural control.[53]

The inspection is diagnostic. The patient is seen to rotate the head suddenly, always in the same direction, by an apparently irresistible active movement. This involuntary movement can be prevented by manual pressure, which can also be used to overcome the muscles and rotate the head back into the neutral position. The muscles give way in a manner that suggests neurological hypertonus.

Therapy of spasmodic torticollis includes behaviour modification, such as training the patient to readjust the position of the head, and biofeedback, hypnosis and anticholinergic drugs.[54] When these procedures are not successful, local application of botulinum toxin A offers a highly effective treatment

technique. Botulinum toxin A is injected into the overactive, dystonic cervical musculature to provide a graded, reversible denervation of the neuromuscular junction by preventing the release of acetylcholine from the presynaptic axon of the motor end plate. Botulinum treatment showed success rates ranging from approximately 60 to 90%.[55,56] Surgical intervention is used when a patient has not responded to other interventions. Although a variety of surgical techniques have been studied, the most commonly used approach is selective peripheral denervation.[57]

Psychogenic torticollis

The patient keeps the head and scapula fixed in an impossible position, which immediately demonstrates its non-organic nature: the head is held in side flexion towards the alleged painful side and the scapula at the same side is kept hunched. No organic lesion exists which leads to fixation of the head in side flexion and whereby the patient is obliged to keep the scapula elevated.

On examination, all active and passive movements are very painful and limited. Resisted movements are painful and weak. This again is very suspect. Finally, the end-feel discloses active muscular contraction but sustained passive pressure during persuasion soon leads to a full range of movement and a normal end-feel, which confirms the diagnosis.

Parkinsonism

The neck may gradually become stiff and painful because of muscle rigidity that is the result of increased extrapyramidal tone. The passive range being much larger than the active one affords a clue, and inspection of the face is confirmatory, as are other typical findings (e.g. tremor, gait).

Cervical scoliosis

The presence of a compensatory thoracic curve following cervical scoliosis indicates the possibility of adolescent scoliosis, unilateral cervical rib, Klippel–Feil deformity[58] or previous thoracoplasty.

Meningitis

The differential diagnosis is not very difficult. In meningitis, the patient develops acute pain in the neck with headache. There is nausea and sometimes vomiting and fever. The neck is fixed in extension. On examination very pronounced dural symptoms and signs are found: even straight-leg raising is positive (Kernig's sign and Brudzinski's sign).

Natural history

Untreated, the pain is constant and severe for 2 or 3 days; the condition recovers spontaneously over the course of the following week.

Treatment

Treatment is by manipulation. The technique is adapted according to the clinical picture. Acute torticollis in patients under 30 often behaves in a nuclear manner; these patients should be treated with 'nuclear' techniques – progressive sustained rotation and/or lateral flexion, whereby an attempt is made to push the displaced nuclear material slowly back. The less common annular cases should be treated with the usual 'annular' manipulation techniques (see Ch. 11).

Unilateral cervicoscapular pain

This is the most common neck complaint. Symptoms may start from the late 20s on and can occur at all ages. The pain has a recurrent character with varying localization and eventually becomes more or less chronic.

History

The patient describes periods of unilateral cervicoscapular pain, which may come on spontaneously or after a minor trauma. The pain may be localized, usually at the mid- or lower neck, or refers to the trapezius area, scapular area, shoulder region or head. The multisegmental character of the reference shows the pain to be of dural origin. The pain is aggravated by certain movements – a common complaint is difficulty in twisting the head to one side so as to reverse a car. Certain postures, especially flexion maintained for a certain time (e.g. when reading), may increase the pain. There is usually no pain at night, except on turning in bed. The painful episode lasts a few days to several weeks and disappears spontaneously. There is no pain between the bouts. A new attack is not always felt on the same side. Over the age of 50, pain may not disappear completely between two episodes of aggravation.

Examination

A mild partial articular pattern is found (Fig. 8.8). As a rule, two, three or four movements out of the six are painful, and four, three or two prove painless. Passive movements are usually more painful than active ones. There may be minor limitation, or a painful arc is found. The end-feel is rather crisp, indicating the muscle guarding typical of a disc lesion, but it is not uncommon to find a normal, capsular end-feel. Root and cord signs are absent. It is not easy to determine the level at which the

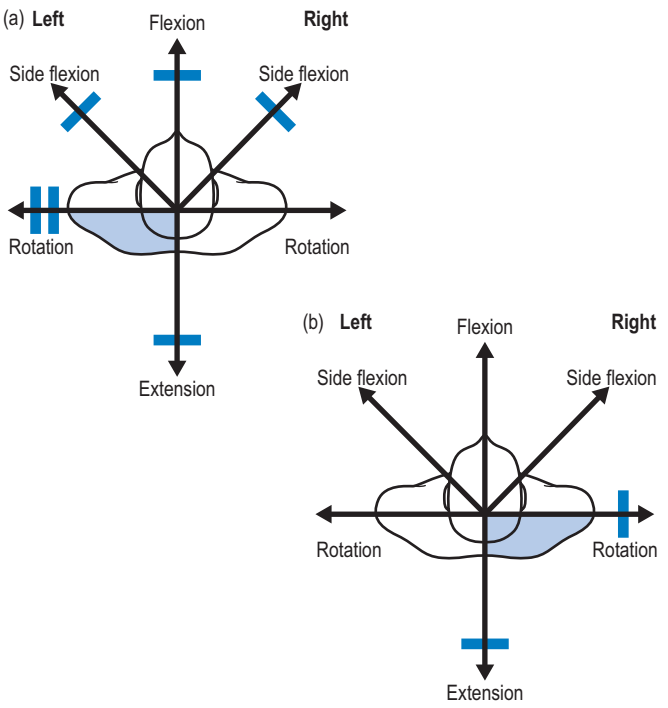


Fig 8.8 • Examples of partial articular patterns in unilateral cervicoscapular pain (colour indicates pain).

protrusion lies, because any disc at any level gives rise to the same symptoms (multisegmental pain) and signs (partial articular pattern). Oscillatory techniques on a sitting or prone lying patient or anteroposterior gliding movements with the patient lying supine may sometimes provoke the pain and give an idea as to the level but this cannot always be relied upon.

Diagnosis

The diagnosis is based on the typical history, together with a partial pattern on examination. The history is that of pain, coming and going in episodes and influenced by particular movements and postures. The pain is of the multisegmental type and spreads over several dermatomes. The asymmetrical pattern of painful movements shows that part of the intervertebral joint is blocked by a displaced fragment of disc, usually annular.

Differential diagnosis

The differential diagnosis includes lesions in the shoulder girdle (e.g. posterior sternoclavicular syndrome, see online chapter *Interpretation of the clinical examination of the shoulder girdle*), in the upper thoracic area (see Ch. 26), internal disorders (e.g. in the heart, lungs or diaphragm, see Ch. 28) and neurological conditions (e.g. long thoracic or spinal accessory neuritis, see online chapter *Nerve lesions and entrapment neuropathies of the upper limb*).

Natural history

In patients under 50, there is a tendency to spontaneous cure in a few weeks. Nevertheless a number of patients are not completely cured and are left with a minor ache between the periods of aggravation.

Treatment

Unilateral pain in the cervicoscapular area is highly suitable for manipulation. Usually a few sessions suffice to restore a full and painless range of movement. The patient must be informed that the disc may move again, however, and that recurrences are not uncommon. Each new attack should be treated in a similar way. However, when there is a tendency to recurrence, prophylactic measures should be taken (i.e. position at work, sleeping position, collar, exercises – see Ch. 13).

See Box 8.3 for a summary of unilateral pain in the neck, trapezius or scapular area.

Unilateral root pain

Root pain as the result of a disc protrusion pressing on the dural investment of a cervical nerve root is quite common. It has an incidence rate of 1/1000 per year with a peak age between 45 and 54. The lesion very seldom occurs in adolescence. In younger patients suffering from cervical root pain, other disorders must be excluded before a disc lesion is considered. Over the age of 60, root pain caused by a discoradicular interaction is also rare, whereas an osteophytic or metastatic compression becomes more likely (Box 8.4).[59]

History

As a rule, the patient has had a history of recurrent bouts of unilateral cervicoscapular pain over the past years. The present symptoms start with the usual scapular pain, but this time they do not resolve spontaneously. On the contrary, one day the scapular pain may have become much worse and strictly unilateral, subsequently shifting to shoulder and arm (Box 8.5). The scapular pain then disappears or diminishes to a degree that it is no longer worthy of mention, whereas pain in the arm increases and remains severe for 1–2 months. The ache is sharp and permanent, and prevents the patient from sleeping. Sometimes the patient can only achieve rest by bringing the arm above the head – a position that relieves tension on the roots but also widens the intervertebral foramen and diminishes the pressure.[60,61] This sign is highly suggestive of a discoradicular conflict, but is unlikely to be present in radiculopathy caused by spondylosis.[62] The arm pain may be accompanied by pins and needles in some fingers, later followed by numbness. In order to make the differential diagnosis with other lesions that provoke paraesthesia, it is vital to collect more information

Box 8.3

Summary of unilateral pain in the neck, trapezius or scapular area

Definition

- An attack of moderate pain in the cervical, trapezial or scapular area as the result of a posterior shift of disc material intermittently compressing the dura mater and resulting in a discodural interaction
- Pain is usually unilateral but may change sides

Onset

- Sudden: annular
- Slow: nuclear

Symptoms

- Articular
  - Pain on movements
- Dural
  - Multisegmental pain reference possible

Signs

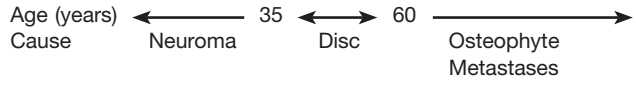
- Moderate or slight partial articular pattern
- Crisp end-feel

Treatment

- Manipulation
- (Traction)
- Prevention

Box 8.4

Cause of root pain according to age



Box 8.5

Discoradicular symptoms and signs

Symptoms	Signs

• Moving pain: scapular pain is replaced by root pain
• Sequence of radicular symptoms:
  • Segmental pain, followed by
  • Localized paraesthesia and
  • Distal numbness
	• Minimal articular sign
	• Neck movements provoke radicular pain/paraesthesia
	• Segmental motor deficit
	• Segmental sensory deficit

about its behaviour: how far proximally does the paraesthesia spread, which fingers and what parts of them are involved, and when precisely are the pins and needles felt? A typical feature is that the paraesthesia has neither edge nor aspect within the fingers. Stroking the skin may provoke or increase pins and needles but moving the digits has no influence. Paraesthesia comes and goes, day or night, in an erratic fashion, appears without any definite reason and does not last for more than an hour at a time. In difficult cases, diagnostic traction may help: if the paraesthesia disappears during manual traction at the head and comes back when traction is released, the cause is clearly cervical.[63] In most cases pain and paraesthesia disappear spontaneously, 6–10 weeks after the onset of the brachial pain. It may take months for the numbness to disappear completely.

Inspection

In very acute cases a pain-avoiding posture may be present: the head is held in side flexion, mostly away from the painful side. Severe root pain may force the patient to keep the arm at the side, well supported by the other hand.

Examination

The patient is asked about the precise location of the pain, which will enable the examiner to determine the dermatome and affected root. However, it is important to mention that the localization of the arm pain is not always a reliable localizing sign. Due to the high incidence of intradural connections between the dorsal rootlets of C5, C6 and C7 segments, there can be overlap between dermatomes, with one dermatome encompassing one or two adjacent segments.[64,65] It may therefore be possible for an individual to have a dermatomal distribution that fails to match the classic pattern precisely. A better pointer to the level of compression is paraesthesia, and therefore, if the patient mentions pins and needles, more details should be sought about their localization.

Articular signs may be present but are not paramount. Usually only a slight partial articular pattern of internal derangement is seen; as the protrusion lies more outside the joint and the articular movements no longer interfere with it, articular signs are less important than root signs. Some active and passive movements may provoke or increase pain in the scapular area. The end-feel is crisp. It is possible, although not at all common, for neck movements to influence the pain and/or the pins and needles down the arm. The Spurling manœuvre (a downwards pressure on an extended and rotated neck) may also elicit radicular pain and paraesthesia.[28] Resisted movements of the head are negative.

At the end of the normal course of the disorder, neck movements become completely painless but the arm still hurts considerably. At this point the best diagnostic clue (apart from the history) is the characteristic pattern of root palsy on examination of the upper limb.

Weakness indicates which nerve root has become compressed. The degree of motor deficit can be very subtle. The examiner should therefore compare both sides carefully.[65] Gross weakness (3+ or 4) is a warning sign, indicating that the lesion is very probably non-discogenic in nature. Cutaneous analgesia is sought at the fingers and the reflexes are tested. An uncomplicated discoradicular interaction does not cause cord signs.

Diagnosis

The history shows the typical evolution of a posterolateral disc displacement: initial attacks of multisegmental cervicoscapular pain, shifting to segmental pain, down the upper limb. As the pain increases, the accompanying symptoms follow a strict chronology:

pain → paraesthesia → numbness → weakness

The examination confirms the history: the combination of a partial articular pattern and positive root signs.

Different root syndromes and their differential diagnosis

The nerve roots most frequently compressed by a disc are, in order of frequency, C7 (56–70%), C6 (19–25%), C5 (2–14%) and C8 (4–10%).[66] Each nerve root lesion, whatever its cause may be, gives rise to pain felt in the skin area corresponding with the segment to which the nerve root belongs. The pain is the result of involvement of the dural sheath surrounding the nerve fibres. When the nerve parenchyma becomes affected too, paraesthesia will be felt down the nerve distribution in the arm. However, variations in the nerve distribution may occur,[67–69] and pain and paraesthesia may be felt in areas which do not totally correspond to one specific dermatome. More involvement of the nerve fibres causes weakness of the muscles belonging to the same segment and cutaneous analgesia in some (mostly the distal) part of the dermatome. One should bear in mind that other (non-neural) soft tissue lesions may lead to exactly the same segmental pain.[70] Therefore resisted tests, undertaken to examine nerve root conduction, also serve to test for alternative causes of pain down the limb.

C1 and C2 nerve roots

The absence of discs at the first two cervical levels implies that nerve root compression by discs cannot occur. Reference of pain to the C1 or C2 dermatomes (headache; Fig. 8.9) does not always mean that C1 or C2 nerve roots are involved; the pain may be multisegmental and the result of compression of the lower cervical dura by a posterocentral disc protrusion.[71,72]

True segmental C1–C2 pain may be found in elderly people and is usually bilateral. It is hypothesized to be the result of arthrosis at the upper cervical joints involving the capsular and ligamentous structures.

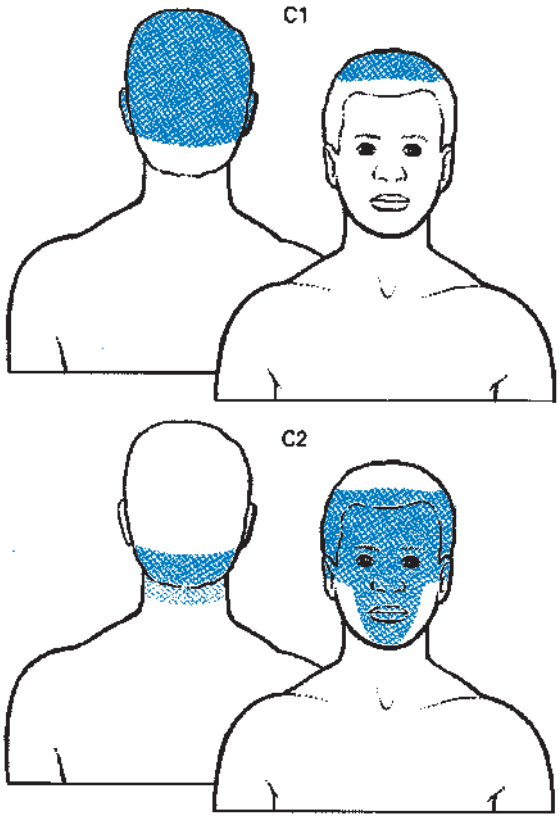


Fig 8.9 • The C1 and C2 dermatomes.

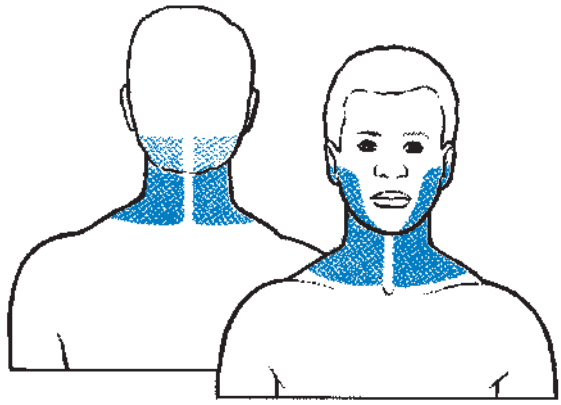


Fig 8.10 • The C3 dermatome.

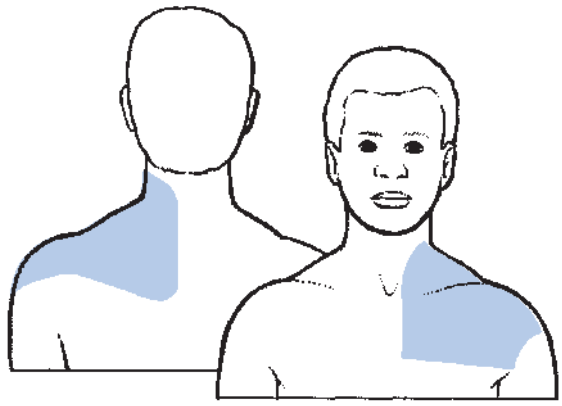


Fig 8.11 • The C4 dermatome.

The C2 spinal nerve has a close proximity to the lateral capsule of the atlantoaxial (C1–2) zygapophyseal joint and innervates the atlantoaxial and C2–3 zygapophyseal joints; therefore, trauma to or pathologic changes around these joints can be a source of referred headache. This occurs especially in elderly patients with gross unilateral arthrosis of the atlantoaxial joint. It causes unilateral deep or dull pain that usually radiates from the occipital to parietal, temporal, frontal and periorbital regions. Ipsilateral eye lacrimation and conjunctival injection are common associated signs.[73] Clinical examination shows a marked full articular pattern in which rotation is especially limited.

C3 nerve root

Compression of the third cervical nerve root by a posterolateral disc displacement is very rare.

Pain is felt at the lateral aspect of the neck and is referred to the occipital and temporal regions and the posterior part of the cheek. Pins and needles or numbness may be present (Fig. 8.10). Weakness is clinically not detectable. Cutaneous analgesia is uncommon, but if it occurs, it occupies any part of the lateral aspect of the neck.

Differential diagnosis. Most C3 pain stems from the C2–3 zygapophyseal joint. This joint and the third occipital nerve appear most vulnerable to trauma from acceleration–deceleration ('whiplash') injuries of the neck.[74] Pain in this region may also result from pressure on the dura mater at any level and is then multisegmental. Heart disease may also give rise to reference of pain in the C3 dermatome. Another possibility is a trigeminal neuritis, which is characterized by paroxysms of severe, lancinating, electric bouts of pain restricted to the distribution of the trigeminal nerve.[75]

C4 nerve root

Discogenic lesions of the fourth cervical nerve root are rare. Pain spreads from the lower half of the neck towards the point of the shoulder (Fig. 8.11). Paraesthesia seems not to occur at this level. In theory, weakness of the trapezius muscle should be found but in practice muscular weakness is not detectable. A horizontal band of cutaneous analgesia may be found along the spine of the scapula, the mid-deltoid area and the clavicle.

Differential diagnosis. It is important to remember that the upper scapular area is very often the site of multisegmental dural pain, resulting from a discodural interaction at any level. Upper scapular pain also occurs in: (a) disorders within the shoulder girdle, e.g. posterior sternoclavicular syndrome or a lesion of the first costotransverse joint; (b) some neurological conditions, e.g. mononeuritis of the long thoracic, spinal accessory or suprascapular nerves; and (c) some pulmonary or visceral disorders, e.g. inflammation of the gallbladder. Pain felt at the shoulder on deep breathing is typical in diaphragmatic pleurisy.

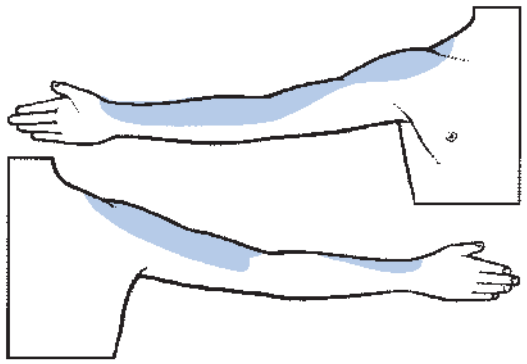


Fig 8.12 • The C5 dermatome.

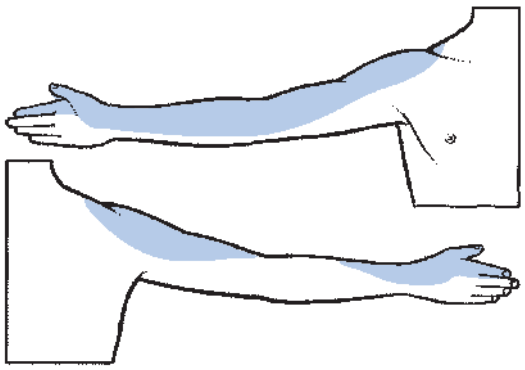


Fig 8.13 • The C6 dermatome.

C5 nerve root

A C5 nerve root involvement is usually not the result of a disc lesion but is often the outcome of either a traction injury or compression by an osteophyte. Traction palsy of the C5 nerve root occurs after an injury that depresses the shoulder girdle and osteophytic palsy caused by encroachment into the fourth intervertebral foramen. Both result in gross weakness of the C5 muscles without pain.

Pain is felt along the lateral aspect of the arm and forearm as far as the base of the thumb (Fig. 8.12). Pins and needles do not seem to occur in C5 nerve root compression.[76] Weakness may be found in supraspinatus, infraspinatus, deltoid and brachial biceps. A C5 sensory palsy from a disc is rare. The biceps jerk may be sluggish or absent; the brachioradialis jerk is sluggish, absent or inverted.

Differential diagnosis. The differential diagnosis (see online chapter *Nerve lesions and entrapment neuropathies of the upper limb*) includes some peripheral nerve lesions and all pathology of the shoulder (see Chapters 14 and 15):

- *Axillary nerve palsy after dislocation of the shoulder*: this results in painless weakness and atrophy of the deltoid muscle and consequent weakness of abduction of the shoulder. Rotation movements remain strong.
- *Mononeuritis of the spinal accessory nerve*: there is painless weakness of the trapezius muscle which results in slight limitation of active elevation of the arm.
- *Mononeuritis of the long thoracic nerve*: this presents with painless weakness of the serratus anterior muscle. Active elevation of the arm is grossly limited.
- *Mononeuritis or traumatic palsy of the suprascapular nerve*: there is painless weakness of the infraspinatus (and supraspinatus) muscles.
- *Neuralgic amyotrophy*: this leads to gross weakness in several muscles.
- *Herpes zoster*: there is pain and a typical skin eruption in the C5 dermatome.
- *Shoulder arthritis, supraspinatus tendinitis or infraspinatus tendinitis*: pain down the C5 dermatome can be referred from the joint capsule or from a rotator cuff tendon.
- *Rupture of the supraspinatus muscle*: partial rupture leads to painful weakness and complete rupture to painless weakness of abduction at the shoulder.
- *Rupture of the infraspinatus muscle*: lateral rotation is painful and weak in partial rupture, and weak only in complete rupture.
- *Metastases or fracture in the scapula*: passive mobility of the scapula is limited and the muscles attached to it are weak.
- *Myopathy*: bilateral wasting of the supraspinatus and infraspinatus muscles is suggestive of myopathy. Arthritis from immobilization of the shoulder is then a painful complication.

C6 nerve root

The sixth cervical nerve root can become compressed by the C5 disc or by osteophytic outcrops originating from the uncovertebral joint or from the facet joint.

Pain runs along the anterior aspect of the arm, the volar aspect of the forearm and the radial side of the hand as far as the thumb and index finger (Fig. 8.13). Pins and needles are felt in the thumb and index finger.[77] There is weakness of the biceps, brachialis, supinator brevis and the extensors of the wrist. Sometimes the subscapularis muscle is affected too. Cutaneous analgesia is sometimes detectable at the tips of thumb and index finger. The biceps jerk is sluggish or absent.

Differential diagnosis. The differential diagnosis is from the following disorders:

- *Thoracic outlet syndrome*: nocturnal pins and needles in one or both hands are often a conspicuous feature. Searching for the release phenomenon by scapular testing provides the clue (see online chapter *Nerve lesions and entrapment neuropathies of the upper limb*).
- *Compression of the median nerve in the carpal tunnel*: there are pins and needles in the three and a half radial fingers. The history is indicative (see online chapter *Nerve lesions and entrapment neuropathies of the upper limb*).
- *Palsy of the radial nerve from compression at mid-humerus*: there is paralysis of the extensors of wrist and fingers, together with weakness of supination of the elbow and of extension of the thumb (see online chapter *Nerve lesions and entrapment neuropathies of the upper limb*).
- *Tendinitis or partial rupture of the biceps muscle*: there is pain or painful weakness on resisted flexion of the elbow (see Ch. 15).

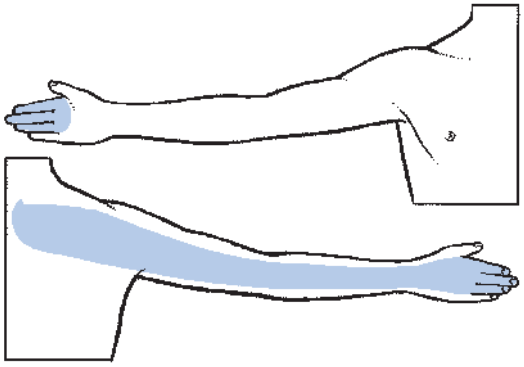


Fig 8.14 • The C7 dermatome.

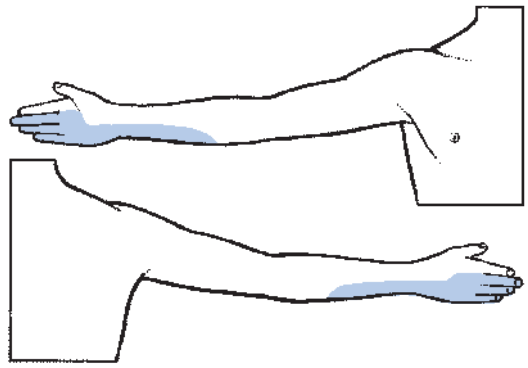


Fig 8.15 • The C8 dermatome.

- *Tennis elbow*: tendinitis at the origin of the extensor carpi radialis brevis may be so acute that the patient often winces and lets the hand go when asked to extend it against resistance (see Ch. 19).

C7 nerve root

Compression of the seventh cervical nerve root by the C6 disc is far more common than compression at any other level.[66]

Pain is felt at the posterior aspect of the arm and the dorsal aspect of the forearm as far as the second, third and fourth fingers (Fig. 8.14). Rarely, pain is experienced at the anterior and upper aspect of the chest and not in the arm. Pins and needles are mostly felt in the index, middle and ring fingers. The most conspicuous feature is weakness of extension of the elbow (triceps muscle). Flexion of the wrist may also be weak (flexor carpi radialis). In severe cases, resisted adduction of the arm is slightly weak and there may even be some visible wasting of the mid-portion of the pectoralis major. The examiner also notices that voluntary elevation of the arm is lacking over the final 5°. In the unusual circumstance of a larger part than usual of the serratus anterior muscle being derived from the C7 segment, winging of the scapula may be seen.[78] Cutaneous analgesia is sought at the dorsal aspect of the index and middle fingers. The triceps jerk is seldom affected, even in quite severe cases.

Differential diagnosis. Differential diagnosis is from the following disorders:

- *Lead poisoning*: one of the first signs is often bilateral weakness of extension of the wrist (see online chapter *Nerve lesions and entrapment neuropathies of the upper limb*).
- *Carcinoma of the bronchus*: this may also lead to bilateral weakness of extension of the wrist.
- *Tennis elbow*: a painful twinge with weakness of extension of the wrist may be found in acute cases (see C6 root).
- *Golfer's elbow*: pain on resisted flexion of the wrist is the primary sign (see Ch. 19).
- *Tendinitis of the triceps muscle*: there is pain on resisted extension of the elbow (see Ch. 19).
- *Fracture of the olecranon*: resisted extension of the elbow is painful and weak but this picture is complicated by articular signs at the elbow joint (see Ch. 18).

C8 nerve root

The eighth cervical nerve root emerges between the seventh cervical and first thoracic vertebrae and can be compressed by the C7 disc.

Pain is felt at the inferior part of the scapular region and the ulnar aspect of the hand, over the middle, ring and little fingers and the ulnar and distal aspect of the forearm (Fig. 8.15). Pins and needles are experienced in the middle, ring and little fingers). The condition is easily confused with ulnar nerve problems ('pseudo ulnar palsy').[79]

Both extensors of the thumb are weak. Other muscles affected are the ulnar deviators of the wrist, the adductor pollicis, the common extensor of the fingers and the abductor indicis. Occasionally the triceps muscle is also slightly weak. Cutaneous analgesia is found at the little finger.

Differential diagnosis. The differential diagnosis includes:

- *Thoracic outlet syndrome*: this may result in pins and needles in the ulnar fingers and sometimes in weakness of the intrinsic muscles of the hand too (see online chapter *Nerve lesions and entrapment neuropathies of the upper limb*).
- *Metastases in the C1 or T1* vertebra: involvement of the C7 and C8 nerve roots at the same time is very suggestive of malignant disease.
- *Angina pectoris*: heart disease should always be considered when pain radiates to the left arm, especially to the ulnar aspect of the hand and forearm.
- *Traction palsy of the lower trunk of the brachial plexus*: this also affects the fibres derived from the C8 nerve root (see online chapter *Nerve lesions and entrapment neuropathies of the upper limb*).
- *Frictional neuritis* of the ulnar nerve at the elbow or pressure on the ulnar nerve at the wrist (see online chapter *Nerve lesions and entrapment neuropathies of the upper limb*).
- *Thrombosis of the subclavian artery*: there is pain in the upper limb and an absent radial pulse.

T1 nerve root

This segment includes the upper limb and is therefore examined with the neck. Compression of the first thoracic nerve root by a T1 disc protrusion is very uncommon and never leads to a palsy.

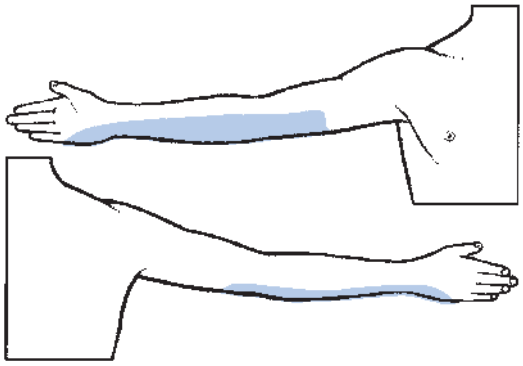


Fig 8.16 • The T1 dermatome.

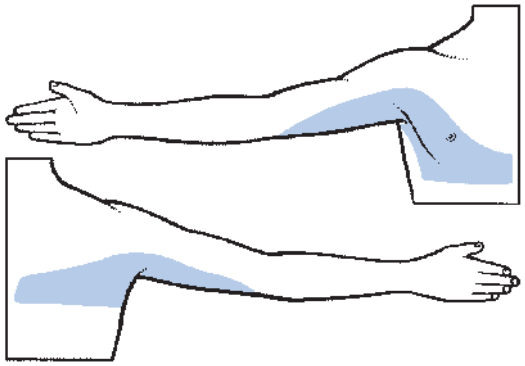


Fig 8.17 • The T2 dermatome.

T1 pain is felt in two distinct sites: the pectoroscapular area and the ulnar aspect of the forearm (Fig. 8.16). The thoracic pain may be influenced by coughing and neck flexion. The T1 root is stretched by a forward movement of the scapulae, and by abducting the arm and flexing the elbow so that the hand comes to lie behind the neck.[80] Pins and needles are felt at the ulnar side of the hand. A T1 palsy is characterized by weakness of the intrinsic hand muscles, but is never caused by a disc lesion. Also, numbness in the T1 area is always the result of a non-discal condition. A first thoracic palsy, together with Horner's syndrome, is one of the main symptoms in Pancoast's tumour[81] and also vertebral metastases may involve the T1 nerve root.

Differential diagnosis. Differential diagnosis of a T1 lesion should be made from the following conditions (see online chapter *Nerve lesions and entrapment neuropathies of the upper limb*):

- *Cervical rib*: this may compress the lower trunk of the brachial plexus, which is partly derived from the T1 nerve root.
- *Compression of the median nerve*: in carpal tunnel syndrome, pain may sometimes spread to the forearm. Most of the muscles in the hand that are supplied by the median nerve are also partly derived from the T1 nerve root.
- *Compression of the ulnar nerve*: this may cause pins and needles in the ulnar border of the hand. All the small muscles of the hand that are innervated by the ulnar nerve are also partly derived from the T1 nerve root.
- *Amyotrophic lateral sclerosis*: weakness and atrophy of the small intrinsic muscles of the hand is often one of the first signs of amyotrophic lateral sclerosis.[82]

T2 nerve root

The second thoracic nerve root is examined with the neck because it gives rise to symptoms felt in the upper limb.

A disc lesion at this level is extremely rare. If it does occur, it gives rise to pectoroscapular pain radiating down the inner aspect of the arm as far as the elbow (Fig. 8.17). The pain is influenced by neck flexion, approximation of the scapulae and sometimes stretching of T1.

Natural history

In discoradicular interactions, spontaneous cure is the rule for both pain and neurological deficit.[83] Patients with neurological deficit usually recover in about 3 months, and those without in 4 months, reckoned from the onset of the pain down the arm. The more marked the palsy, the more quickly the pain abates. Muscle strength will almost certainly have returned to normal 3–6 months after the pain ceased. The only exception is the C8 root, which may take 6 months or more to recover. The sensory disturbance recovers very gradually over a year. Mochida *et al* performed follow-up studies, both clinical and with MRI, on 38 patients with lateral cervical disc herniations. All of them were treated with conservative therapy. The authors' conclusion was: 'migrating, lateral-type herniations regress so frequently that conservative treatment should be chosen not only for patients with radicular pain, but also for those with upper limb amyotrophy.'[84]

Treatment

There are a number of approaches to treatment of nerve root problems.

Prophylaxis

Root pain can be avoided when the original cervicoscapular pain is recognized as the early stage of a disc protrusion and treated with manipulative reduction.

Manipulation

This can be tried in patients without neurological deficit and with favourable articular signs (neck movements hurt in the scapular area). Reduction in a few sessions is possible in the first 2 months after the onset of the brachial pain. After this period, the results are poor. Manipulation is almost impossible in patients with unfavourable signs (neck movements hurt down the arm or provoke pins and needles in the hand).

When the root pain has been present for some time or neurological deficit has set in, it is better to explain the mechanism of spontaneous cure to the patient and to wait for it. In the meantime, nerve root blocks are given to control the pain. Unilateral scapular pain and root pain of more than 6 months' standing can often be helped with two or three manipulation sessions. The scapular pain ceases and the mechanism of spontaneous cures is restarted.

Infiltration

In those patients in whom manipulation fails or is no longer indicated, 1–6 infiltrations with 2 mL of a steroid solution at the nerve root may afford considerable relief. Pain and

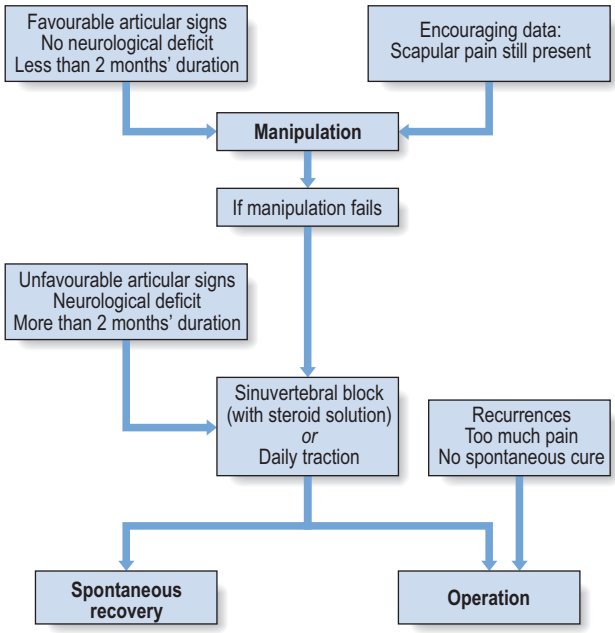


Fig 8.18 • Treatment of cervical nerve root involvement.

Box 8.6

Cervical disc syndromes

Common

- Discodural interactions
  - Acute torticollis – equivalent of acute lumbago
  - Cervicoscapular pain – equivalent of discodural backache
- Discoradicular interaction
  - Cervical root pain – equivalent to sciatica

Less common

- Acute torticollis with central pain and symmetrical picture
- Chronic central cervicoscapular pain

inflammation are diminished during the period of spontaneous healing.[85]

Daily mechanical traction and sometimes wearing a collar for 2–3 weeks may also help to make the pain tolerable.

Surgery

Neck surgery for discoradicular interactions is performed far too often. The condition has such a favourable spontaneous cure rate that surgical intervention should only be considered in exceptional situations: when the condition recurs, when pain is intolerable or when spontaneous cure fails to occur. Furthermore, the long-term results of surgery are no better than when spontaneous cure has been awaited.[86]

Recurrences

These are rare after spontaneous recovery from root pain with neurological deficit but are otherwise not infrequent.

Treatment of cervical root nerve involvement is shown in Figure 8.18. The characteristics of root syndromes in the neck and upper limb are summarized in Figure 8.19.

Summary

These three clinical syndromes are most common (Box 8.6) and may be compared to the three pictures found at the lumbar spine (see chapter 33).

Less common syndromes

The syndromes described in this section are also based on disc lesions that, in contrast to the lumbar spine, may imperil the spinal cord. Cyriax believed that posterocentral disc protrusions may form the first step in the evolution towards spinal stenosis. An unreduced central disc displacement exerts continuous pressure on the posterior longitudinal ligament. The latter draws the periosteum away from the vertebral bone. Bone grows towards the periosteum and osteophytes form. These diminish the anteroposterior diameter of the spinal canal and spinal stenosis syndrome develops (see p. 164).

The clinical pictures that appear during the first stage of this development are still reversible by simple manual techniques. Once bony narrowing of the spinal canal has started, however, reduction is impossible.

Acute torticollis with central pain and a symmetrical picture

History

A young or middle-aged person develops severe central neck pain, either spontaneously or as the result of trauma, usually a whiplash injury. The pain fixes the head in a flexed position. Shortly afterwards, both upper limbs may also become painful with a feeling of weakness. Paraesthesia appears distally in both arms or in all four limbs.

Examination

In acute cases, the neck is usually fixed in flexion, which may be so extreme that the chin touches the sternum, extension being totally impossible. The movement pattern is symmetrical, with both rotations and both lateral flexions equally limited. Active elevation of both arms is painful and sometimes limited. There are no root signs. Cord signs are usually absent, except in extreme cases where pyramidal tract signs may be found.

Diagnosis

Diagnosis rests on the history (sudden onset) and on the clinical examination, which shows an extreme limitation of extension.

Differential diagnosis

Differential diagnosis includes conditions that present with a full articular pattern: for example, fracture of a vertebral body and other bony disorders. A similar pattern with gross limitation of extension is also seen in severe ankylosing spondylitis.

Natural history

This type of torticollis tends to recover spontaneously, although very slowly.

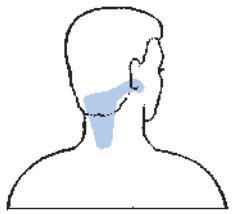
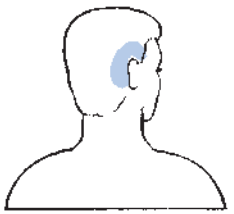
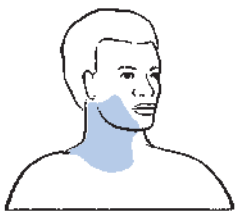
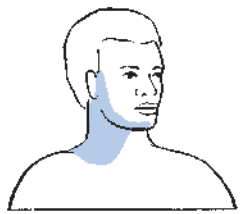
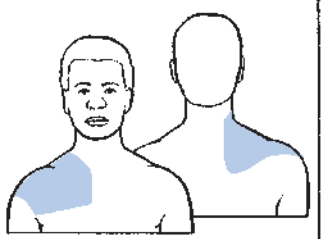
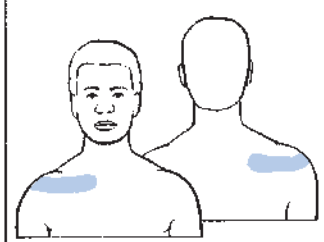
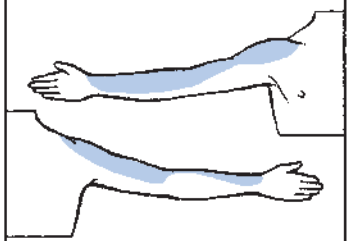
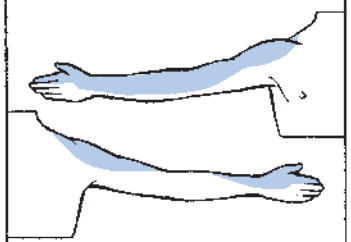
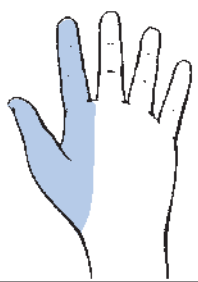
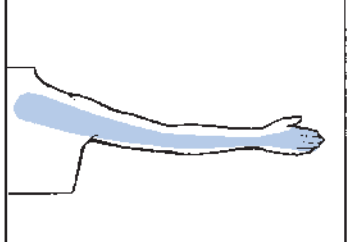
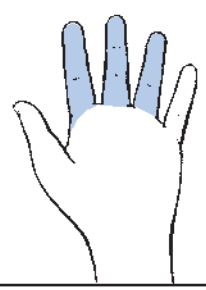
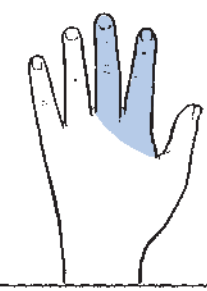
ROOT	SHEATH	NERVE FIBRES			DIFFERENTIAL DIAGNOSIS
	Pain	Paraesthesia	Motor deficit	Sensory deficit	
C2 (not a disc)			None	None	Dural reference Osteophyte at the atlanto-axial joint
C3			None		Dural reference
C4		None	None (trapezius muscle?)		Dural reference

Fig 8.19 • Root syndromes in neck and upper limb.

Continued

ROOT	SHEATH	NERVE FIBRES			DIFFERENTIAL DIAGNOSIS
	Pain	Paraesthesia	Motor deficit	Sensory deficit	
C5		None	Supraspinatus Infraspinatus Deltoid Biceps Jerks: biceps, brachioradialis	None	Combination of a cervical disc lesion + shoulder lesion C5 root traction palsy Axillary nerve palsy after dislocation Mononeuritis spinal accessory, long thoracic, suprascapular nerve Traumatic suprascapular nerve palsy Herpes zoster Myopathy (deltoid, supra-, infraspinatus muscles) + pain from the capsule of the shoulder joint Rupture of the supra- or infraspinatus tendon Metastases in the scapula Diaphragmatic pleurisy
C6			Biceps Brachialis Supinator Extensor carpi radialis (Subscapularis) Wasting: brachioradialis muscle Jerks: biceps		Pressure on the median nerve in the carpal tunnel Pressure on the brachial plexus by first rib or a cervical rib Tendinitis or partial rupture of the biceps muscle Pressure on the radial nerve at mid-humerus Rheumatoid perineuritis (complication of a monoarticular rheumatoid arthritis of the shoulder) Tennis elbow
C7			Triceps Flexor carpi radialis (Extensores carpi radiales) (Latissimus dorsi) (Serratus anterior) Wasting: pectoralis major		Lead poisoning (bilateral) Carcinoma of the bronchus Tennis elbow, golfer's elbow Tendinitis of the triceps muscle Fracture of the olecranon

Continued

Fig 8.19 • Root syndromes in neck and upper limb (cont'd).

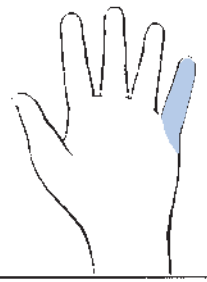
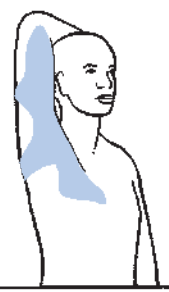
ROOT	SHEATH	NERVE FIBRES			DIFFERENTIAL DIAGNOSIS
	Pain	Paraesthesia	Motor deficit	Sensory deficit	
C8			Extensor and flexor carpi ulnares Extensors and adductores pollicis Extensor digitorum communis (Triceps)		Pressure on the brachial plexus by a cervical rib Pressure on the brachial plexus by the first rib C7 or T1 neoplasm Pancoast's tumour (apex of the lung) Angina Traction on the inferior trunks of the brachial plexus with neurological deficit Frictional neuritis of the ulnar nerve at the elbow Pressure on the ulnar nerve at the wrist Thrombosis of the subclavian artery
T1			Palsy = not a disc	Palsy = not a disc	Cervical rib Pulmonary sulcus tumour Vertebral metastases Pressure on the median nerve
T2		None	None	None	None

Fig 8.19 • Root syndromes in neck and upper limb (cont'd).

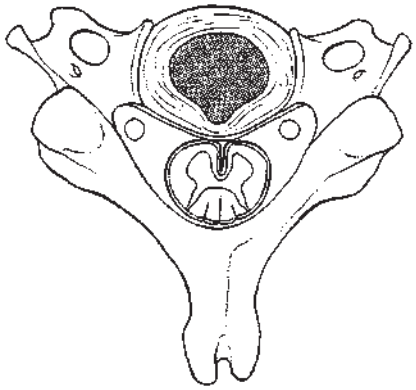


Fig 8.20 • Posterocentral disc displacement.

Treatment

Manipulative reduction is the treatment of choice but rotation manœuvres are avoided. The therapist should choose the techniques with great care and perform them very cautiously to avoid further displacement towards the spinal cord or stretching of the posterior longitudinal ligament.

Central or bilateral pain in the neck, trapezius or scapular area

The condition causes central neck pain, with or without bilateral radiation to occiput, trapezii and scapulae, although slow painless development also occurs. Pain is the result of the pressure exerted on the dura mater (Fig. 8.20). Cord symptoms indicate involvement of the spinal cord. Cord signs point to irreversible impairment of conduction. The evolution from pain, through cord symptoms to irreversible cord signs may take several years to develop.

History

Patients are usually middle-aged. They start complaining of central neck ache, which later spreads bilaterally from the occiput to both trapezii. Gradually the pain expands, radiating down to the interscapular area and both scapulae. There may come a time when both upper limbs start to ache. Pain in the arms may alternate: if it is more severe on one side, it is correspondingly less so on the other. By the time the pain has become constant, pins and needles may start, first in both hands, some time later in both feet too. Neck flexion may bring on or increase the pins and needles.

In the middle-aged or elderly patient, the whole process may be entirely painless. It starts with pins and needles in all four limbs: first in both hands, later radiating into the lower limbs, from the front of both knees to the feet. Gradually the gait may become disturbed and weakness appears in both hands.

Examination

Articular signs may be quite inconspicuous. Pain is constant but is not very much influenced by articular movements. If the patient is elderly, a full articular pattern of limitation may be found with a hard, arthrotic end-feel. If any movements are painful, these are usually flexion and extension. As long as the pressure on the spinal cord is not considerable, symptoms alone dominate the presentation and there are no root or cord signs.

Increased compression leads to the manifestation of the clinical syndrome of cervical myelopathy (see p. 165): a positive plantar reflex, incoordination and spasticity of the lower limbs and weakness of the upper limbs.[87]

Diagnosis

The history is very important, especially at the beginning of the process, before cord signs and symptoms have supervened. The patient will probably have had attacks of torticollis or of unilateral cervicoscapular pain in the past. Centralization of the pain as age advances, together with a painless full articular pattern on examination, suggests this type of disc lesion. When involvement of the spinal cord is suggested (multisegmental paraesthesia, gait disturbance), examination becomes decisive.

Differential diagnosis

When central or bilateral pain is present, great care should be taken to make a differential diagnosis from other lesions that may cause similar symptoms: arthrosis, ankylosing spondylitis, rheumatoid arthritis, recent fracture, postconcussional syndrome, and bone disease. In the presence of bilateral paraesthesia, differential diagnosis must be made from sensory stroke and disseminated sclerosis.

Natural history

There is a tendency towards spontaneous cure but regression of the disc protrusion is slow.[88]

Treatment

Posterocentral disc protrusions not compressing the spinal cord can be reduced by manipulation, provided rotatory manipulations are avoided. The manipulation must be performed under sufficient traction and by an experienced therapist. When symptoms and/or signs of spinal cord compression are present, manipulation is strongly contraindicated and surgery is called for.

Degenerative disorders

Arthrosis of the cervical spine is usually referred to as cervical spondylosis or spondylarthrosis. It starts very early in life with disintegration of the disc. The ageing of the cervical disc and the consequences for the surrounding structures have been discussed earlier in this chapter. Degenerative changes in vertebrae and discoligamentous structures are found in 75–90% of people aged over 50.[15,16,89] From middle age on, degeneration is evident on plain X-rays and on MRI in more than 50% of asymptomatic people.[90,91] Therefore, most authors emphasize that there is no consistent correlation between abnormal radiographic appearances and symptoms.[92,93] The conclusion that pain originates from degeneration of the cervical spine is all too often made by looking at imaging alone. The importance of arthrotic changes is overestimated because, in most instances,

the degenerative condition is not painful in itself and leads only to some stiffness. It is the clinician's task to determine the possible relationship between the degenerative changes and the patient's symptom.[94]

Non-symptomatic arthrosis at the cervical spine shows painless limitation of movement in the capsular proportions whereby the movements are merely uncomfortable at the end of range. The end-feel is hard.

Symptomatic cervical spondylosis should be diagnosed purely on clinical grounds, through history and clinical examination. When cervical movements influence the patient's symptoms, the problem clearly lies within the spine but this does not necessarily imply that arthrosis is responsible. Although degeneration is not painful of itself, it can lead to symptomatic conditions such as: ligamentous contracture, overstretching of a facet joint, radicular compression by osteophytes or cervical spinal stenosis. The diagnosis of these lesions should be made on clinical grounds and not on imaging.[95]

Local pain

Pain in the neck, either unilateral or bilateral, and with or without radiation to the head, occurs in degenerative conditions affecting the upper cervical ligamentous complex or the facet joints. This takes place in arthrosis of the upper two cervical joints, in subacute arthritis of the atlantoaxial joint, in facet joint arthrosis or in traumatic osteoarthrosis.

Morning headache in the elderly

'Morning headache' in the elderly is believed to stem from ligamentous contracture at the atlanto-occipital and atlantoaxial complexes. The hypothesis relies on the typical distribution of pain in the C1–C2 dermatomes,[96,97] the leathery end-feel on clinical examination and the uniformly good results with mobilization.

As age advances, increasing pain develops in the C1–C2 dermatomes: the middle of the upper neck and the occiput, the temples and the forehead (C2). Local pain may be totally absent, so that the patient complains of occipitofrontal headache only.[98] The headache is only on waking and happens without fail every morning. After some hours the pain begins to diminish and by midday all symptoms have ceased, only to return the next morning. Later in the course, the pain may last longer into the day.

On examination a full articular pattern is found. The movements are not very painful but merely stiff and uncomfortable. The end-feel is hard but not bone-to-bone; rather it is leathery, showing capsular contracture. Radiography demonstrates a certain degree of arthrosis, normal for the patient's age.

The differential diagnosis is between traumatic arthrosis and postconcussional headache. In both, the history is distinctive (see pp. 164 and 168).

The condition is very easily cured by manipulation: 1–4 sessions of slow capsular stretching make all symptoms disappear. Age is not a contraindication to manipulation but the older the patient, the more gradually the therapist must work: fewer manœuvres are carried out in one session and the interval between the sessions is longer (see Ch. 11 and summary in Box 8.7).

Box 8.7

Morning headache in the elderly

- Headache on waking
- Full articular pattern
- Good response to slow stretching manipulation

Subacute arthritis of the atlantoaxial joint

Subacute arthritis of the atlantoaxial joint is rare. The patient, aged between 25 and 40, develops stiffness and discomfort in the middle of the upper neck, and over the following weeks these symptoms gradually increase. On clinical examination, extension, both side flexions and flexion movements are full and painless. Both rotations are equally painful and very limited – only 10–20% is possible. Even in a supine position, the range remains unchanged. The end-feel is soft. This combination of symptoms and signs is clearly a warning sign but further investigations, such as blood tests and imaging, remain negative. The aetiology remains unclear but the disorder recovers in a couple of months with anti-inflammatory therapy.[99]

Arthrosis of a facet joint

The prevalence of cervical facet joint degeneration is very high in individuals aged 50 years and more, with a tendency to increase in severity with age. Contrary to the lumbar spine, where there is an increase in degeneration towards the lower segments, in the cervical spine all levels of the middle and lower cervical spine are affected to almost the same degree.[100] However, the relation between the high prevalence of radiographic zygapophyseal arthrosis and neck pain is not clear.[101] It is not well understood why arthrosis at a facet joint becomes symptomatic in one person and not in another. Cyriax hypothesized that pain results from the formation of adhesions and a 'self-perpetuating inflammation', which follows overstretching of the arthrotic joint capsule.[20]

Diagnosis of a facet joint lesion is not easy and is always tentative. Some elements from the history and clinical examination may help in differentiating a facet joint lesion from a discodural conflict. In doubtful cases, local infiltration with lidocaine may prove diagnostic.[102]

The pain has a spontaneous onset, is unilateral and does not spread. When the facets are bilaterally affected, the pain is bilateral, sparing the centre of the neck. Dural symptoms are absent. Pain is felt when certain positions have to be maintained for a period of time.

On examination, a partial articular pattern is found: some movements, especially passive ones, hurt. The end-feel is rather hard. The fact that the pain is quite localized and does not radiate indicates the possibility of a facet joint lesion. Troisier[82] distinguishes a 'convergent' and a 'divergent' pattern of painful movements (Figs 8.21 and 8.22). In the first, there is pain on extension and on lateral flexion and rotation towards

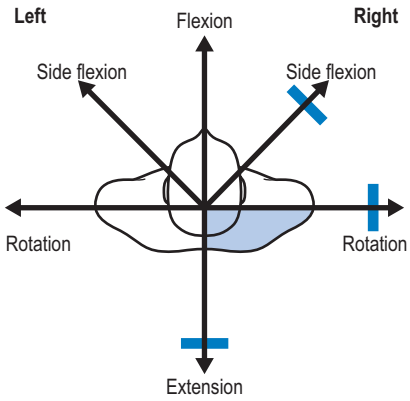


Fig 8.21 • The convergent partial articular pattern in arthrosis at a facet joint (colour indicates pain).

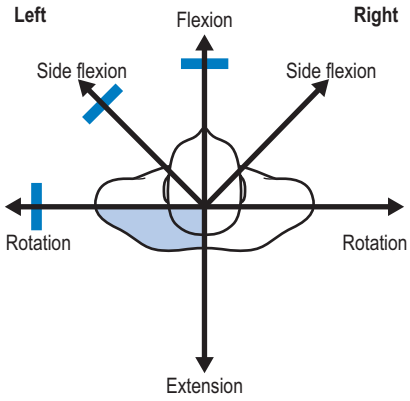


Fig 8.22 • The divergent partial articular pattern in arthrosis at a facet joint (colour indicates pain).

Box 8.8

Symptomatic facet arthrosis

- Sprain of an arthrotic joint capsule?
- Unilateral, localized pain
- No dural symptoms
- Partial articular pattern (divergent or convergent pattern)
- Diagnostic infiltration?
- Local steroids or friction/mobilization

the painful side. In the second, the opposite is found: flexion, lateral flexion and rotation away from the painful side hurt. Such a pattern is compatible with facet joint involvement but does not exclude a discodural interaction. Differentiation between these lesions is always difficult.

This condition responds to deep transverse massage at the joint capsule and slow stretching manipulation. Infiltration with a steroid suspension is an alternative (Box 8.8).

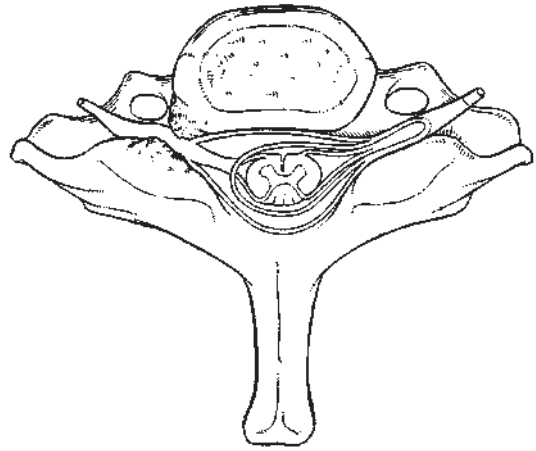


Fig 8.23 • Osteophytic nerve root compression.

Radicular complaints: osteophytic root compression

A nerve root can become compressed by osteophytes and bony spurs in the neural foramen. The root normally occupies about one-third of the space in the foramen and is accompanied by radicular arteries and veins. The spinal nerve root is vulnerable to compression, anteriorly by an osteophyte derived from the uncovertebral joint and posteriorly by one originating from the facet joints, or from both (Fig. 8.23). These phenomena are secondary to degeneration of the intervertebral disc, with subsequent dehydration and collapse. This disintegration causes increased mechanical stress at the uncovertebral joints and the facet joints, leading to the formation of subperiosteal bone and osteophytic outgrowth, and finally resulting in narrowing of the intervertebral foramen and encroachment of the nerve.[103,104]

Symptoms of osteophytic radiculopathy usually develop insidiously, but are sometimes triggered by trauma. The patient is over 50 and complains of paraesthesia and weakness in the arm, sometimes pain. Contrary to a disc that causes radicular pain, in osteophytic root compression there is or was not much pain in the neck or scapular area, merely some stiffness. Also the root pain is very moderate, though it may be present for several months. The main complaints are paraesthesia in the arm or fingers and a gradually increasing weakness of the arm.

On examination, a full but painless articular pattern with a hard end-feel is found, indicating arthrosis. If any movement does hurt, it is usually side flexion towards the affected side, and a twinge may travel down the arm. Alternatively, waves of paraesthesia may be felt in the distal part of the relevant dermatome and are clearly the result of the osteophyte being pushed against the nerve root. The Spurling manœuvre (a downward pressure on an extended and rotated neck) may also elicit radicular pain and paraesthesia.[28] Isometric testing of the upper limb shows segmental weakness. The most frequent localization is C4–C5. A combination of higher uncinate process, smaller anteroposterior diameter of the intervertebral foramen, and a longer course of the nerve root in close proximity to the uncovertebral joint may explain the predilection of

Table 8.2 Differential diagnosis between discal and osteophytic root compression

	Disc	Osteophyte
History	>35 years of age	Elderly: >50 years of age
	Shifting pain (scapula > arm)	Arm pain
	Typical evolution	No evolution
	Intense pain	Slight pain
	Pain > weakness	Weakness > pain
Examination	Partial articular pattern	Full articular pattern
		Compression pain
	Subtle weakness	Serious weakness
	C7 root	C5 root

nerve root compression at this level.[105,106] The C5 nerve root thus becomes compressed, identified by the difficulty or sometimes even inability to bring the arm up. In severe cases, the deltoid may become wasted.[107]

The differential diagnosis is from root compression by a disc protrusion or by a neuroma or metastases, and can usually be made on a clinical basis (Table 8.2). The osteophyte grows slowly and lies quite far laterally so that it does not exert pressure on the dural investment of the nerve root. Therefore intense pain, as would occur in a discoradicular interaction, is seldom present. The patient complains mainly of paraesthesia and of a progressive weakness of the upper limb developing over the course of a few months. Nerve root compression by a disc protrusion is characterized by a definite chronology: a period of bouts of multisegmental pain, referred to the scapular area, is followed by the occurrence of segmental pain, followed by segmental paraesthesia and often moderate neurological deficit of the muscles belonging to the same segment. The condition undergoes spontaneous cure in 2–3 months. A neuroma usually starts in the opposite way: distal paraesthesia first, followed by pain that starts distally and spreads in a proximal direction. Root pain due to a neuroma typically occurs in younger people, while root pain from a disc lesion occurs more frequently in the middle-aged. A tumour or metastasis leading to root compression usually has a quicker progression than an osteophyte. Further differential diagnosis includes all the upper limb entrapment syndromes and neuralgic amyotrophy (see online chapter *Nerve lesions and entrapment neuropathies of the upper limb*).

The diagnosis can be confirmed by radiography. Anteroposterior and oblique views show the encroachment. The uncovertebral osteophytes are best seen on an anterior view and the posterior osteophytes on an oblique one. Computed tomography (CT) is often used to complement radiography because it provides superior imaging of bone and better defines the anatomy and size of the neural foramen.[108] When the clinical diagnosis of an osteophytic root palsy has been made and the X-ray confirms its presence, the diagnosis is clear. Nevertheless, it should be borne in mind that the prevalence of asymptomatic osteophytes is very high. Therefore, the clinical examination is decisive.

Manipulation is, of course, contraindicated. Two infiltrations of triamcinolone suspension around the nerve root – nerve root block – at an interval of 2 weeks may be tried. It alleviates the inflammation at the level of the compressed nerve root, so that the pressure diminishes.[109,110] The discomfort disappears but the anatomical situation has not changed. When there is gross muscular weakness or weakness has a tendency to increase, surgical treatment is indicated. During the last few decades, a minimally invasive posterior cervical foraminotomy technique has been developed[111] that is highly effective and results in long-lasting pain relief.[112]

Box 8.9

The mushroom phenomenon

- Elderly patient
- Increasing pain in any position, except lying
- Diminution of symptoms under traction
- Full articular pattern
- Radiographic evidence

Compression phenomenon: the mushroom phenomenon

This phenomenon is extensively described in later chapters on the thoracic and lumbar spine (see Chs 27 and 35) and is summarized in Box 8.9. A cervical mushroom phenomenon is very rare. It occurs in advanced degeneration of the disc, which displaces mainly in the anterior and anterolateral directions. The intervertebral space becomes so narrow that the vertebral bodies lie in apposition. This phenomenon, together with folding of the posterior longitudinal ligament and enlargement of the arthrotic facet joints, can cause considerable narrowing of the spinal canal and the lateral recess, which may result in compression of dura or nerve root during axial loading.

An elderly patient will state that there are no symptoms as long as recumbency is maintained: in that position the head can be moved easily in every direction without any problems. However, on standing or sitting for a certain amount of time, vague, central and bilateral pain in the neck develops and spreads to both arms and hands. All symptoms disappear when traction is applied or the head lifted upwards with the hands. On examination, a full articular pattern is found: painless limitation of movement with a hard end-feel. There are no radicular signs and no cord signs; consequently the diagnosis rests mainly on the history. A radiograph shows severe arthrosis with gross narrowing of the intervertebral space.

The patient can be helped either by a weight-relieving collar or by surgery (arthrodesis).

Cervical spondylotic myelopathy

Cervical spinal myelopathy (CSM) is defined as spinal cord dysfunction, secondary to intrinsic compression from degenerative disease of the cervical spine. It is the most common cause of spinal cord dysfunction in patients who are aged over 55.[113] The presence of a congenitally narrow spinal canal is a critical predisposing factor in patients with spondylotic

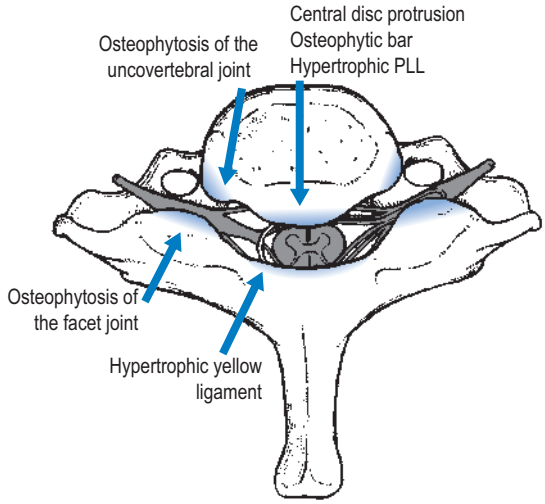


Fig 8.24 • Mechanical factors involved in cervical spondylotic myelopathy (coronal section). PLL, posterior longitudinal ligament.

myelopathy.[114] The anteroposterior diameter of the cervical spinal cord is 10 mm on average and normally the sagittal diameter of the spinal canal averages ± 18 mm. It is therefore generally accepted that a spinal canal with a diameter between 10 and 13 mm is at risk and can only sustain a narrowing of 2–4 mm before developing myelopathy.[115,116]

Pathogenesis

Symptoms of myelopathy occur in a wide variety of combinations. In its most severe form the clinical presentation is a spastic gait. There may be atrophy, sensory disturbance and spasticity in the hands. Sometimes impairment of sphincter function also occurs.[117] The variable clinical picture reflects the many complex factors that may affect the spinal cord. The primary pathophysiologic abnormality is a reduced sagittal diameter of the spinal canal. In that narrowed canal, two different mechanisms – a mechanical and a vascular one – can cause pathological changes in the spinal cord. White and Panjabi[118] divide the mechanical factors involved in the pathogenesis of CSM into two groups: static and dynamic. Static factors include the well-known arthrotic changes that narrow the canal: a spondylotic bar anteriorly, degenerative osteophytosis of the uncovertebral joints and facet joints laterally, and hypertrophic ligamenta flava posteriorly (Fig. 8.24).[119,120] Dynamic factors are abnormal forces placed on the spinal column and spinal cord during flexion and extension. Diminution of the diameter of the spinal canal occurs during extension (because of folding of the posterior longitudinal ligament and of the yellow ligament[121]) and during flexion (the spinal cord is pulled against the osteophytes).[122] Also degenerative cervical spondylolisthesis may cause excessive movement.[123,124] During extension, backward gliding of the vertebrae, in combination with folding of the posterior ligaments, may result in cervical cord encroachment (the pincer effect) (see Fig. 8.24).[125]

Another mechanical theory is that CSM is caused by tensile stresses transmitted to the spinal cord from the dura via the dentate ligaments.[126] A spondylotic bar pushes the spinal cord posteriorly, but this displacement is resisted by the dentate ligaments that are anchored by the dural root sleeves in the lateral foramen.[127] This theory could explain the neuropathological findings in CSM where damage to the spinal cord is most severe at the lateral columns, and in them the involved areas are often wedge-shaped, with the apex medial and the base lateral.[128]

Other authors consider spinal cord ischaemia to be a major cause of CSM, either through compression of the anterior spinal artery and its branches in the spinal cord, or through the radicular arteries in the intervertebral foramina. This vascular theory is not widely accepted and most believe that a combination of both mechanical and vascular causes has to be considered (Box 8.10).[129,130]

Box 8.10

Factors involved in cervical spondylotic myelopathy

- Developmentally narrow canal (<13 mm)
- Mechanical factors
  - Static
    - Central disc protrusion
    - Osteophytic bar
    - Osteophytosis of the uncovertebral and facet joints
    - Hypertrophy of the yellow and posterior longitudinal ligaments
  - Dynamic
    - Spondylolisthesis
    - Compression during flexion and extension
    - Traction via the dentate ligaments
- Vascular factors
  - Ischaemia of the anterior spinal artery
  - Ischaemia of the radicular arteries

Diagnosis

CSM usually develops insidiously, although episodes of abrupt deterioration can occur.

Symptoms

A subtle gait disturbance is usually the first and most common presentation.[131] Although CSM can cause a variety of signs and symptoms, spastic gait occurs first, followed by upper extremity numbness and clumsiness. There is loss of fine motor control of the hands and difficulty in writing. Surprisingly, neck pain is not as common a symptom as one might expect, maybe because some pain and stiffness is accepted as normal for the person's age.

After some years, when the situation gets worse, patients develop a typical 'spastic gait' that is broad-based, hesitant and jerky. This gait is one of the main characteristics of cervical myelopathy. Nurick has classified CSM largely on the basis of gait abnormality – typically the most common clinical concern of the patient (Table 8.3).[132]

There are also sensory changes in the lower extremities, which the patient typically describes as 'walking on cotton wool'. Bowel and bladder dysfunction may supervene; according to Epstein *et al*, 20% of patients with CMS over the age of 65 have bladder dysfunction, mostly associated with urinary retention.[133]

Table 8.3 Nurick's classification of disability in spondylotic myelopathy

Grade	Symptoms and signs
0	Root symptoms and signs No evidence of cord involvement
I	Signs of cord involvement Normal gait
II	Mild gait impairment Able to be employed
III	Gait abnormality prevents employment
IV	Able to ambulate only with assistance
V	Chair-bound or bedridden

Signs

Examination of the neck is usually not very painful but reveals a gross full articular pattern of limitation with a hard end-feel, suggesting severe arthrosis. Flexion and extension may elicit electric shock sensations in the extremities (Lhermitte's sign[134]), which is present in about one-quarter of myelopathic patients.[135,136]

Neurological examination

Compression of the spinal cord typically leads to 'upper motor neurone' findings distal to the level of compression (i.e. spasticity, hyperreflexia, clonus, positive Babinski and Hoffmann signs). These often occur in combination with 'lower motor neurone' findings at the level of compression (e.g. weakness, hyporeflexia and atrophy). For example, in a lesion at C4 level, with compression of both C5 nerve roots and of the spinal cord at that level, everything above C5 is normal. C5 shows weakness of abduction and external rotation of the shoulder and some weakness of flexion of the elbow. There is no spasticity and the biceps jerk is sluggish or absent. Below C5 there is spasticity, with clonus and hyperreflexia: an exaggerated triceps jerk and clonus at the patella and the ankle.

Other functional tests can be performed and, when positive, are very suggestive of CSM, though none of these tests in itself has pathognomonic value. Ono *et al*, for instance, observed a characteristic abnormality in the hands of patients with CSM. There was inability to extend the ulnar two or three fingers, despite the relatively well-preserved function of the wrist, thumb and index finger. Rapid extension of the fingers was impossible, even in patients with less advanced disease.[137] Also Hoffman's sign is very often positive. The sign is described as follows.[138] The pronated hand is supported so that it is completely relaxed and the fingers partially flexed. The middle finger is firmly grasped and partially extended, and the nail is flicked by the examiner's thumbnail. The flicking should be done with considerable force. The sign is present if quick flexion of both the thumb and the index finger results. A positive sign is highly indicative of possible pyramidal tract pathology.[139] Shimizu *et al* described the scapulohumeral reflex, which is positive in more than 95% of patients with high cervical cord compression.[140] The scapulohumeral reflex is elicited by tapping the tip of the spine of the scapula and acromion in a caudal direction and is positive when elevation of the scapula or abduction of the humerus is seen. The reflex centre of the scapulohumeral reflex is clinically presumed to be located between the posterior arch of C1 and the caudal edge of the body of C3. A hyperactive scapulohumeral reflex therefore provides useful information about dysfunction of the upper motor neurones cranial to the level of the C3 vertebral body. As the disease develops, Babinski's sign and its variants, Chaddock (stimulation below the external malleolus causes extension of the great toe) and Oppenheim (stroking down the medial side of the tibia causes extension of the great toe), become positive.[141]

In addition, sensory disturbances are very often present. There may be loss of contralateral pain and temperature sensation because of compression of the spinothalamic tract. The neurological level is several segments below the area of compression. Ipsilateral proprioception and vibration sense may be disturbed as a result of a lesion of the posterior columns. Dermatomal sensation (appreciation of light touch as well as tactile discrimination in pinprick tests) can be affected by a dorsal nerve root compression.

Differential diagnosis

The differential diagnosis of CSM is not easy. It is important to exclude both multiple sclerosis and upper motor neurone disease (amyotrophic lateral sclerosis), as their presentations are very similar to that of CSM.[142] A thorough neurological examination is therefore necessary. Most importantly, CSM does not affect the cranial nerves or the normal jaw reflex, whereas the other two disorders may. Other disorders in the differential diagnosis include spinal tumours, cerebrovascular disorders that cause spastic hemiplegia and peripheral neuropathy.

Amyotrophic lateral sclerosis

Amyotrophic lateral sclerosis (ALS) is a disorder of the anterior horn cells and of the pyramidal tract (first motor neurone).[143] The disease starts between the ages of 40 and 70 in one (commonly upper) limb and advances progressively to the contralateral upper and later the lower limbs. Depending on the localization in the upper or lower motor neurone, spastic or weak paralysis will predominate. The classic triad is atrophic weakness of the hand and forearm muscles, minor spasticity of the legs and generalized hyperreflexia, but without sensory changes; as a pure motor neurone disease ALS does not affect sensation. A diagnosis of ALS is further favoured if there are muscular fasciculations in the face, tongue or lower extremities. The diagnosis is supported by typical findings on electromyography (EMG).[144]

Multiple sclerosis

Multiple sclerosis (MS) is a chronic inflammatory disease of the central nervous system with the morphological hallmarks of inflammation, demyelination, axonal loss and gliosis. The spinal form of MS in particular may mimic the clinical course of CSM: there is a generalized spastic paralysis and clonus, and the patient also experiences paraesthesia. However, the patient is younger – the average age of onset is 30 years. Diagnosis relies on EMG findings, typical appearances on MRI[145] and cerebrospinal fluid analysis.

Radiographic studies

In the past, the radiological diagnosis of CSM was dependent on radiography, myelography, CT and CT myelography. More recently, MRI has proven to be the most valuable tool, not only to depict anatomically how the spinal cord is compressed, but also to demonstrate the pathologic changes in the spinal cord.[146] Takahashi *et al* described the MRI findings of intramedullary high signal intensity (SI) on T2-weighted MR images in CSM. They include oedema, ischaemia, demyelination, gliosis and cavities.[147]

Natural history

The natural history seems to be one of static neurological deficit or episodic progression. The course is quite variable. Often, symptoms are mild and do not progress. Such cases are best treated conservatively with a cervical collar and physical therapy. However, once clinical CSM is evident, progression may occur despite the best of treatments. Elderly patients often experience faster progression of symptoms and more serious neurological impairment.[148] It was found that those who deteriorated were more likely to be female and to have significantly more cervical mobility when compared to those patients whose disability remained static. It is proposed that measurement of cervical mobility may help to select patients who are more likely to deteriorate and thus more likely to benefit from surgical intervention.[149]

Treatment

Minimal symptoms without hard evidence of gait disturbance or pathologic reflexes warrant non-operative treatment, but surgery should be recommended to arrest progression of myelopathic symptoms in patients whose general condition is satisfactory.[150] Both anterior and posterior approaches have been utilized for surgical treatment of cervical myelopathy. Anterior decompression frequently requires corpectomy at one or more levels and strut grafting with bone from the ilium or fibula. Multilevel laminectomies were initially used for posterior decompression but now are either combined with fusion or replaced by laminoplasty.[151]

Prognosis is good when the diagnosis is made early in the course but poor in late diagnosis, when myelopathy has become more severe. It is therefore important to operate on patients with CSM as early as possible before neurological deficits are too pronounced.

Patients who are treated by surgery seem to have fair outcomes. The progression of myelopathy is arrested in about 95% and the improvement rate of the gait is estimated to be around 51–85%.[152,153]

Some recommend preventive decompression surgery in individuals who have spondylotic encroachment on the cervical spinal cord without clinical signs and symptoms of myelopathy. The reasoning is that these patients are at increased risk of spinal cord injury if they experience minor trauma. A recent meta-analysis of the literature, however, concluded that there is insufficient evidence for an increased risk and that recommendations for preventive decompression surgery cannot be made.[154]

The characteristics of CSM are shown in Box 8.11.

Box 8.11

Summary of cervical spondylotic myelopathy

Definition

- Spinal cord dysfunction, secondary to intrinsic compression from degeneration of the cervical spine

Onset

- >55 years

Pathogenesis

- Narrowed canal: developmental
- Static factors
- Dynamic factors

Symptoms

- Spastic gait
- Clumsy hands
- Sensation of walking on cotton wool
- Sphincter dysfunction

Signs

- Upper limbs: lower motor neurone signs
- Lower limbs: upper motor neurone signs

Differential diagnosis

- Amyotrophic lateral sclerosis
- Multiple sclerosis
- Spinal tumours
- Cerebrovascular disorders

MRI

Treatment

- Surgery

Capsuloligamentous disorders

Capsuloligamentous disorders of the cervical spine are sometimes described under the heading 'postconcussional syndrome'. This is a term used to describe the occipital headache and/or upper cervical pain that remain after a concussion which may have sprained the upper cervical ligaments and, less often, the muscles. The pain is believed to be caused by ligamentous adhesion formations.

However, the diagnosis is often tentative and, as the patient is very often claiming compensation, the examiner has to take into account the possibility of neurosis or aggravation. Careful history taking and clinical examination should enable the examiner to come to a precise diagnosis. When the patient presents a history with inconsistencies or unlikely combinations of findings and this is followed by an examination during which the signs do not correspond, the patient is probably confabulating. However, when adhesions in the occipito-atlantoaxial ligaments are responsible, the picture is one of pain at the extremes of extension, both rotations and both lateral flexions.

The condition can easily be treated by mobilization. One to three sessions of quick stretch manipulations break the adhesions, after which all symptoms should disappear.

Disorders causing pain on resisted movements of the neck

Musculotendinous lesions

The presence of a clear contractile tissue pattern is suggestive of a musculotendinous lesion but this is not common in the region of the neck. Only two conditions are rarely encountered: a lesion of the semispinalis or splenius capitis muscle and a lesion of the longus colli muscle, so-called retropharyngeal tendinitis.

Lesions of the semispinalis or splenius capitis muscle

It is understandable that a patient who has had an accident severe enough to cause concussion may also suffer from musculotendinous lesions in the suboccipital muscles. This is infrequent but after recovery from concussion the patient may be left with unilateral or bilateral occipital pain, sometimes radiating to the head. In most instances, a lesion of the occipital muscles does not occur in isolation but in combination with a lesion of the occipito-atlantoaxial ligaments.

On examination, resisted movements are positive. In unilateral pain, resisted extension and resisted side flexion towards the painful side are both painful. In bilateral cases, resisted extension and resisted flexion to both sides may be positive. Palpation shows the lesion to lie at the insertion of the semispinalis capitis muscle, rarely at that of the splenius capitis.

Treatment consists of two or three sessions of deep transverse massage. In more chronic cases, up to 6 weeks' treatment may be required.

Lesion of the longus colli muscle (retropharyngeal tendinitis)

Retropharyngeal calcific tendonitis, also known as acute calcific prevertebral tendinitis, is a clinical syndrome that was described originally by Hartley in 1964,[155] and was later demonstrated to be secondary to calcium hydroxyapatite deposition in the longus colli muscle.[156] This muscle is a paired neck flexor that comprises the prevertebral space (Fig. 8.25). Classically, calcification affects the superior oblique portion of the longus colli muscle at the C1–C2 level (Fig. 8.26). The condition affects adults within a reported age range of 21–81 years, with its greatest distribution between 30 and 60 years. The incidence of this condition is rare, although its true incidence is probably higher than previously thought.[157]

The history and clinical examination are very characteristic. The patient quite suddenly develops a severe, bilateral pain occupying the whole head and neck area and can hardly move the head. Swallowing is very painful and hurts so much that the head is held with both hands. The pain is felt not in the throat, but in the neck. There is no fever. After a few days the pain diminishes, and disappears completely after a few weeks.

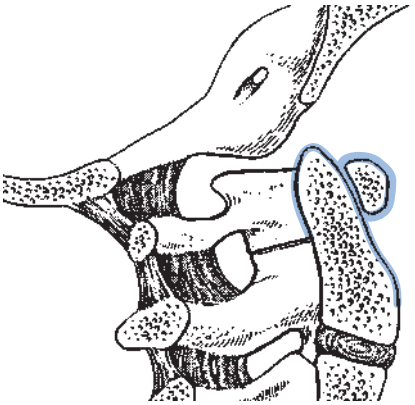


Fig 8.25 • The superior oblique fibres of the longus colli muscles are involved in acute calcific prevertebral tendinitis.

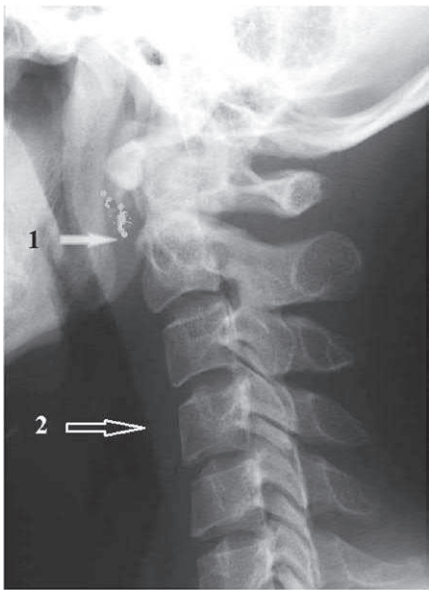


Fig 8.26 • Retropharyngeal tendinitis: localization of the calcium deposits. Arrow 1 indicates amorphous calcification anterior to C2. Arrow 2 indicates swelling of the retropharyngeal space.

On examination a full articular pattern of limitation of movement is found on active testing. Passive movements show a different pattern: when they are done gently, flexion and both side flexions can be performed to full range; extension and both rotations remain very limited and the end-feel is spastic. Resisted rotations and resisted flexion are painful.

The lateral radiograph shows swelling of the retropharyngeal space and amorphous calcification anterior to C1–C2. The thickening of the shadow thrown by the longus colli muscles is such that it increases from the usual 3 mm to 10 or 15 mm.

CT shows the pathognomonic tendinous calcifications within the longus colli and can also demonstrate sterile fluid smoothly expanding the retropharyngeal space.[158] In the presence of a fluid collection, the possibility of infection must be considered, specifically abscess from lymphadenitis.[159]

The differential diagnosis includes traumatic fracture/dislocation of the vertebra, retropharyngeal abscess, meningitis and infectious spondylodiscitis.

Treatment consists of rest and administration of oral non-steroidal anti-inflammatory drugs for 1–2 weeks. The condition undergoes spontaneous cure in 2–3 weeks; pain eases, motion returns, and swelling and calcification disappear almost simultaneously.

Serious disorders

Resisted movements of the neck may be painful and/or weak in more serious conditions, because the muscular contraction pulls at an affected structure (i.e. bone) or squeezes a tender structure (i.e. inflamed lymph glands or abscess). This combination of signs has to be considered a warning sign. The possible conditions are:

- Vertebral metastases
- Fracture of the first rib
- Fracture of the spinous process of C7 or T1
- Wedge fracture of a vertebral body
- Glandular fever
- Retropharyngeal abscess
- Postconcussional syndrome
- Lesion of the sternoclavicular joint (see the online section *The shoulder girdle*).

Disorders causing symptoms on active and/or resisted shrugging of the shoulders

Examination of the shoulder girdle is part of the scan examination of the cervical spine. Some conditions in the scapular area and shoulder girdle may provoke positive signs during this examination. These lesions are extensively discussed in the online section *The shoulder girdle*. They are:

- Lesion of the costocoracoid fascia
- Lesion of the sternoclavicular joint
- Lesion of the first costotransverse joint
- Lesion of the conoid/trapezoid ligament
- Lesion of the subclavian muscle
- Lesion of the levator scapulae muscle
- Thoracic outlet syndrome
- Upper lung disorder (warning sign).

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Non-mechanical disorders: warning signs

9

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Pathology: see the online content

Most cervical spine syndromes are activity-related or mechanical. They result from disc lesions and/or degeneration of the spine. Syndromes that are not activity-related are called non-mechanical disorders. They stem from inflammatory diseases, tumours and metabolic disorders. Though they can influence mobility, they are intrinsic diseases of the cervical structures and do not have a mechanical origin.

Warning signs

Mechanical lesions of the cervical spine usually demonstrate very typical behaviour. They present with so-called 'inherent likelihoods' – the sequence of symptoms and signs that make up the clinical picture of a certain pathological disorder and that are likely to be found. Hence, when symptoms and signs come forward during the clinical assessment which show rather unlikely behaviour, examiners should be on their guard. Such symptoms and signs are called 'warning' signs. The examiner should consider them as the hallmark of a (serious) non-mechanical disorder until there is proof to the contrary. An unusual disorder is immediately suspected and further complementary investigations (blood tests, radiography, CT scan, bone scan, MRI) should be requested.

Warning signs disclosed by the history

Gradually increasing pain

Mechanical lesions tend to present with acute pain, as episodic pain that comes and goes in a rather irregular way, or as a constant, unchanged pain over a considerable length of time. Sometimes with a disc lesion, pain gradually increases over a short period of time, after which it remains unaltered for a certain period and then diminishes again. If, in contrast, the intensity of the pain increases progressively over a couple of weeks, then a serious lesion is likely.

Expanding pain

Pain typically changes location with a disc lesion. In a discodural conflict the pain moves within the zone of multisegmental reference, and when the conflict becomes discoradicular it shifts from the neck or scapular area to the arm. In other words, it shifts from one area to another. In the case of expanding pain, however, the evolution is different: for example, the pain starts in the centre of the neck, then becomes bilateral and spreads to the scapular area, and may finally radiate down one upper limb or both limbs. Another possibility is pain that develops in one dermatome and gradually spreads beyond its borders into other dermatomes. Increasing scapular pain together with an increase of brachial pain is also suspect. Pain that expands very often indicates a lesion that expands (a tumour or metastasis).

First-time neck pain in an elderly person

A middle-aged or elderly patient who, for the first time in life, complains of neck pain or who describes rapidly increasing pain and neck stiffness, coming on in the course of 1 or 2 months, should be suspected of having malignancy in the cervical spine.

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Bilateral arm pain

A discal lesion seldom causes pain in both arms. Either the disorder is discodural, causing bilateral cervicoscapular pain, or there is a discoradicular conflict, in which case pain will be felt in one arm only. A disc displacement that is large enough to cause pain in both arms would certainly also threaten the spinal cord. Other possibilities are: large osteophytes at one particular level and other bony lesions of the cervical spine. Bilateral arm pain is therefore a warning sign for a serious disorder.

Arm pain in a person younger than 30

Root pain as the result of pressure by an ordinary cervical disc is very rare in people under the age of 30. Therefore, if there is evidence of radicular pain and the patient is younger than 30, another cause has to be sought.

Arm pain lasting longer than 6 months

Most lateral disc protrusions tend to resolve spontaneously. For lumbar disc lesions this may take up to 12 months (see Ch. 33). For cervical disc protrusions causing discoradicular pain, the process is faster and most protrusions will have disappeared after 2–4 months, depending on the size of the protrusion (the larger the protrusion, the faster it fades).[1] Therefore, if the pain lasts longer than 4 months, it is unlikely that an ordinary discoradicular lesion is responsible and another cause should be sought.

Wrong time sequence

The symptoms that result from discoradicular root pain follow a typical timeline. Initially there is cervicoscapular pain that later moves distally to become strictly segmental pain. Other symptoms follow: first pins and needles and later numbness. When the reverse is true – root pain starting distally and later moving proximally, or distal paraesthesia occurring before the root pain starts – examiners should again be on their guard because this time sequence makes a discal problem very unlikely.

Warning signs disclosed by the functional examination

Full articular pattern

A full but painless articular pattern with a hard end-feel is normal in elderly patients, and results from the normal degeneration of the cervical spine. However, a full articular pattern that is painful and has a more spastic end-feel points to a serious disorder and warrants further investigation.

Muscle spasm on passive movements

Muscle spasm is a defence mechanism. It is an involuntary muscular contraction that stops the passive movement, thereby protecting the cervical spine against further painful and possibly harmful movement. The muscles contract with a sudden vibrant twang, even when the movement is performed gently. This phenomenon is typical of very acute conditions, like acute arthritis or fractures, and never occurs in ordinary disc lesions; therefore it is always suspicious.

Resisted movements of the neck that are painful and weak

Resisted movements may be painful in acute discal conditions but are never weak. If an attempt to contract the muscles against the examiner's resistance is immediately stopped because it is too painful, full strength is not perceived and the movement is thus interpreted as being weak. This is always a sign of a serious disorder. Painful weakness is mainly caused by a pull on bony lesions (tumours and metastases).

Side flexion away from the painful side as the only painful movement

As mentioned earlier, this partial articular pattern is always suspicious and suggests a costoscapuloclavicular lesion (see online section *The shoulder girdle*) or a visceral lesion in the upper part of the thorax: the apex of the lung or mediastinum.

Limited scapular elevation

Limitation of active elevation alone is extremely rare. In theory, it could result from a C4 palsy or from a spinal accessory palsy, but in practice this is not detected. Limitation of both active and passive elevation of the scapula points to a lesion of the shoulder girdle and not to a cervical lesion. When this sign is found, the examiner should perform a more thorough examination of the shoulder girdle and upper thorax.

Horner's syndrome

This syndrome is caused by an interruption of the sympathetic nerve supply to the eye and is characterized by the classic triad of miosis (constricted pupil), partial ptosis, and loss of hemifacial sweating (anhidrosis). Horner's syndrome may result from several nervous conditions: a lesion of the primary neurone, a brainstem stroke or trauma to the brachial plexus. When the syndrome is accompanied by cervicoscapular pain, the cause is involvement of the cervical sympathetic ganglia at the base of the neck that results from a tumour of the lung apex (e.g. Pancoast)[2] or a dissecting carotid aneurysm.[3]

Hoarse voice

Paralysis of the vocal cords gives rise to typical hoarseness (when a lot of air is released). The cause may be local lesions or involvement of the recurrent laryngeal nerve by an invasive lesion in the neck or upper thorax (bronchial and oesophageal carcinoma, malignancy of the mediastinal lymph nodes and aortic aneurysm).

T1 palsy

Palsy of the first thoracic nerve root leads to atrophy and weakness of the intrinsic hand muscles. The palsy is never caused by a disc lesion. Therefore, the detection of weakness in the

adduction movement of the little finger is always a sign that must be taken seriously. It is one of the first signs of amyotrophic lateral sclerosis, but is also often present in the initial stages of cervical spondylotic myelopathy and in brachial plexus lesions.

Excessive loss of power

Root compression as the result of a protruded disc results in a slight muscular paresis (stage 1 or 2). When more extensive paralysis is found, the condition is, by definition, serious.

Involvement of two or three nerve roots

Unlike in the lumbar spine, root syndromes resulting from cervical discoradicular lesions are always monoradicular. It may be, however, that a radicular weakness does not precisely match the classic pattern. This is somewhat exceptional and is explained by intradural connections between the ventral rootlets of C5, C6 and C7 segments. So it is possible for an individual to have some overlap between myotomes, with one myotome encompassing one or two adjacent segments.[4] It may also be possible to have two disc lesions at two (consecutive) levels at the same time. These situations are rare, however, and therefore, when there is involvement of two or more nerve roots, a more serious lesion should be assumed until there is proof to the contrary.

Muscular weakness in the absence of root pain

In a discoradicular interaction, segmental pain is one of the most striking features and results from a compressed and inflamed dural nerve root sleeve. It is only when the compression continues and increases that paraesthesia and neurological deficit will also follow. For a parenchymatous lesion to develop in the nerve root that is large enough to cause neurological damage, there must have been considerable pressure and pain. Silent radicular weakness therefore means that the cause of the damage is not acute external pressure from a protruded disc.

Warning signs and symptoms are summarized in Box 9.1.

Box 9.1

Warning signs and symptoms

Warning symptoms	Warning signs
• Gradually increasing pain • Expanding pain • First time neck pain in an elderly person • Bilateral arm pain • Arm pain in a person younger than 30 • Arm pain lasting longer than 6 months • Wrong time sequence	• Full articular pattern • Muscle spasm on passive movements • Resisted movements of the neck that are painful and weak • Side flexion away from the painful side as the only painful movement • Limited scapular elevation • Horner's syndrome • Hoarse voice • T1 palsy • Excessive loss of power • Involvement of two or three nerve roots • Muscular weakness in the absence of root pain

Warning signs and imaging techniques

It is the clinical approach that suggests serious disorders. If history and/or clinical examination show one or more warning signs, the examiner should consider the disorder as a serious, non-mechanical disorder until there is proof to the contrary. The presence of a normal X-ray or computed tomographic scan is not always a guarantee of the absence of a serious lesion; nor is the presence of degeneration, bulging discs or herniated discs proof of a symptomatic mechanical lesion.

Access the pathology of non-mechanical disorders of the cervical spine and the complete reference list online at **www.orthopaedicmedicineonline.com**

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10 Whiplash-associated disorders

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Previous accounts used different terms – whiplash injury, hyperextension injury, acceleration injury, soft tissue neck injury, cervical strain, cervical sprain – to describe the lesion that may occur after and the consequences of a typical car accident: a rear-end motor vehicle collision.

When a vehicle is struck from the rear, the occupants rarely have any warning and do not brace the muscles to prevent head movement. As a result, the body is propelled forwards and the neck hyperextends backwards well beyond the normal range of allowable movement (Fig. 10.1). This violent motion is followed by a less rapid forwards recoil into flexion that can often result in a head injury if the head impinges on the windscreen.

Although rear-end impacts are most common and result in hyperextension–hyperflexion injuries, other types of car accident may also cause whiplash-type injuries and, in any trauma, complex head and neck movements may occur, leading to different lesions that resemble whiplash.

Definition

Medical literature, in an attempt to find a proper definition, has so far described 'whiplash injury' in terms of the mechanism of the accident, the type of lesion that is caused or the clinical appearance after the injury. In 1995[1] the Quebec Task Force (QTF) proposed the following definition:

> Whiplash is an acceleration–deceleration mechanism of energy transfer to the neck. It may result from rear-end or side-impact motor vehicle collisions but can also occur during diving or other mishaps. The impact may result in bony or soft tissue injuries (whiplash injury), which in turn may lead to a variety of clinical manifestations (whiplash-associated disorders).

The term 'whiplash-associated disorders' (WAD) indicates the clinical features that result from an accident in which two elements – acceleration, followed by deceleration – are responsible for the traumatic forces that act on the cervical spine and related structures.

Incidence

As the result of increases in availability and use of cars worldwide, motor vehicle accidents have become very frequent with, as a result, an enormous increase in whiplash-type trauma. It is one of the most common mechanisms of injury to the cervical spine.

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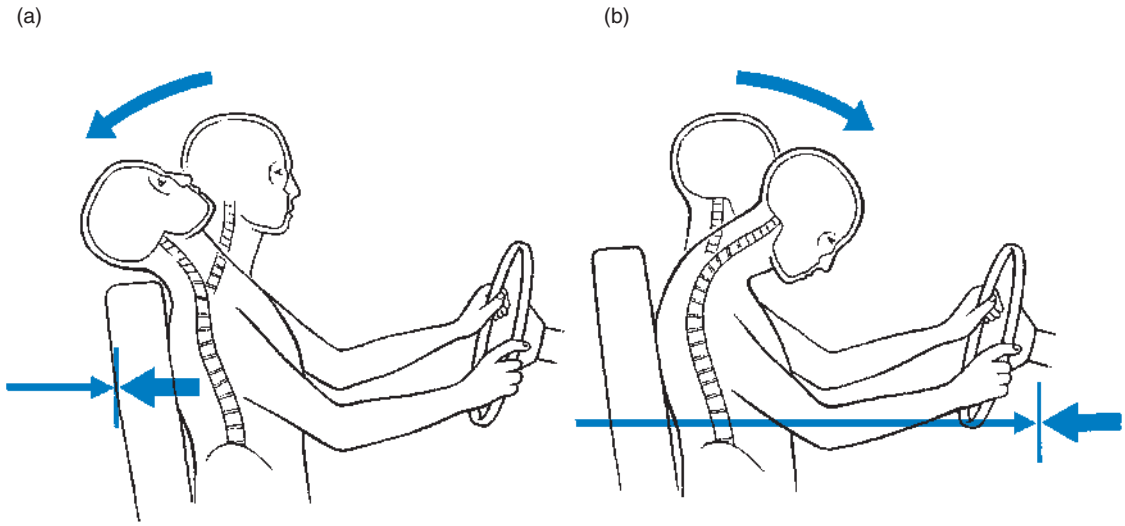


Fig 10.1 • Whiplash injury: (a) hyperextension on impact, followed by (b) recoil into flexion.

The incidence is not precisely known. A figure of 1 per 1000 people per year has been suggested.[2]

The QTF mentions figures on whiplash injuries in Canada. In 1987 in the province of Quebec there were approximately 131 whiplash injuries per 100 000 vehicles per year – 70 injuries per 100 000 inhabitants. This cost the Canadian government CAN $19 000 000, of which 70% was income compensation. The female : male ratio is about 1.5 : 1 and the main age group 20–24 years.

Other studies in Canada mention 5000 whiplash cases a year in the province of Quebec, accounting for 20% of all insurance claims after motor vehicle accidents.[3,4]

In the United States 11 300 000 car accidents were reported for the year 1991, of which 2 690 000 were rear-end collisions and caused 85% of all whiplash injuries.[5]

Classification

The QTF, persuaded that proper diagnosis is difficult to achieve, has proposed two classifications: one according to the severity of the symptoms and signs (grades) (Box 10.1) and one according to the time elapsed since the accident (stages) (Box 10.2).

Neither classification suggests what lesion is present nor stipulates the type of tissue damage. They reflect only the clinical appearances that occur after acceleration–deceleration injury.

Pathology

Depending on the movement of the head during the accident, several lesions may occur, ranking from severe to moderate to slight. Hyperextension is the most common mechanism, followed by hyperflexion and lateral flexion.[6]

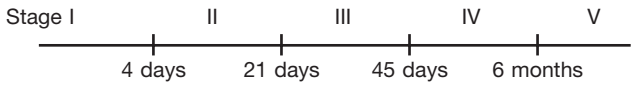
Box 10.1

Clinical (Quebec) classification of whiplash-associated disorders

Grade	Clinical presentation
0	No neck complaint No physical sign(s)
I	Neck complaint involving pain, stiffness or tenderness only No physical sign(s)
II	Neck complaint AND Musculoskeletal sign(s)
III	Neck complaint AND Neurological sign(s)
IV	Neck complaint AND Fracture or dislocation

Box 10.2

Classification according to the time elapsed since the accident



Severe lesions

Hyperextension and distraction of the neck may rupture the anterior longitudinal ligament as well as some discs. A ruptured disc can lead to backward displacement of the vertebra lying

above it – the upper facets then slide downwards on the lower – with damage to the spinal cord as a result.[7] Spinal cord injuries after motor vehicle accidents occur most often in young car users in the 15–24 year age group.[8,9]

Pure hyperextension may also cause compression of the spinal cord in those cases in which retrolisthesis or spinal stenosis already existed. In other instances, compression fractures of the posterior elements may occur.

Hyperflexion injury may lead to fractures of the vertebral body – most fractures of the atlas[10] and of the axis[11] are the result of motor vehicle accidents – and/or to disruption of posterior ligaments and occasionally facet joint luxation.

Less frequently, lesions of arteries, veins, neural structures, oesophagus and retropharyngeal tissues may occur.

Other lesions

Less severe lesions are much more frequent and may involve the intervertebral discs, the zygapophyseal joints, and the cervical ligaments and muscles. These lesions may occur in isolation but are more often combined and therefore are sometimes difficult to recognize. The common complaint is neck pain.

Discodural and discoradicular interactions

Recent retrospective studies have shown that the occurrence of disc lesions after whiplash injury is quite high[12,13] and one prospective study indicates the value of clinical diagnosis.[14] Most disc lesions are endplate avulsions and ruptures of the anterior annulus fibrosus.

As the result of the hyperextension element during the trauma the disc may have fissured. The subsequent flexion or hyperflexion element causes displacement of disc material in a posterior direction. Davis *et al* describe a number of posterolateral disc lesions with radicular symptoms as the result of a hyperextension whiplash trauma.[12] These herniations seemed to develop only after the acute phase and it took a few weeks for the radicular symptoms to appear. In postmortem studies Taylor *et al* describe the intervertebral disc as the most frequently damaged structure.[15–17] Jónsson *et al*[18] Also confirmed the large number of disc lesions after whiplash, and during surgery were able to confirm the findings from magnetic resonance imaging (MRI).

Posterocentral protrusions lead to central, bilateral or unilateral pain in a multisegmental distribution: pain in the neck, trapezius and upper scapular area. On examination a symmetrical (mimicking a full articular pattern) or asymmetrical pattern of limitation is found. In acute cases the picture may be torticollis-like. For a detailed description of disc pathology, see page 145.

Facet joint problems

Whiplash may also lead to problems at the level of the zygapophyseal joint capsules.[19] Lord *et al* undertook a placebo-controlled prevalence study after whiplash and found that chronic cervical facet joint pain was common.[20]

Pain is felt unilaterally and is usually quite localized. A convergent or divergent motion pattern may occur, although any asymmetrical pattern is compatible. (Facet joint pathology is discussed on p. 163).

Ligamentous lesions

Ligaments can become overstretched, leading to minor lesions,[12] or may become adherent as the result of post-traumatic immobilization. They present with vague stretching pain felt at the end of range of those movements that stretch the ligament (see p. 168).

Muscular lesions

Muscular lesions, mostly anteriorly, are described in clinical studies,[21,22] on echography,[23] in experiments in animals[24,25] and in postmortem studies.[26] Muscles, particularly their occipital insertions, can be strained during injury. The subsequent pain will be quite localized and can be elicited during either contraction or stretching – the contractile tissue pattern (see p. 169).

Medicolegal consequences

As WAD automatically involve compensation claims, they have considerable consequences. If a significant number of patients remain with some disability – and this number still seems to be increasing – the costs of diagnosis, treatment and indemnity become progressively higher.

The different parties involved in the approach to WAD are:

- The patient who is seeking help and a refund of money. For most patients the two elements do not influence each other but for a number of people the compensation claim is essential. This may result in absence from work, illness behaviour, social disability, malingering and fraud.
- Doctors who are looking for a diagnosis. Physicians treating the patient wish to reach a diagnosis and to confirm it with technical investigations. They also – because of the often-complex nature of the syndrome – use an extensive pattern of treatment techniques. Doctors who work for the insurance company are sometimes biased and tend to over-diagnose the condition as 'psychogenic' or 'simulation'.
- Insurance companies who are trying to minimize their payments. As the result of the lack of consensus about diagnosis and treatment of WAD, insurance companies see their compensation payments and indemnities rise considerably. They exert pressure on governments in order to keep these expenses under control.
- Lawyers who are protecting either the insurance company or the patient. Discussions about confirmation of the lesions and the consequences for patients' professional activities lead to an increase in litigation.

It is clear that eligibility for compensation for pain and suffering furthers the perseverance of symptoms and a tendency to

chronicity. Where this compensation can be eliminated, a decrease in incidence and an improved prognosis are seen.[27]

Psychological problems

Most WAD start as an ordinary trauma followed by purely physical disturbance. When subsequent treatment is unable to rehabilitate the patient after a short period of time, secondary emotional and psychological changes may supervene. They raise the patient's awareness of neck pain and subsequently aggravate and perpetuate the pain, or even turn a simple neckache into chronic pain and disability.[28]

Diagnosis

Clinical picture

Making a diagnosis in patients who have undergone a whiplash-type injury is no different from other patient groups. It requires proper history taking, inspection and careful functional examination, including a neurological evaluation.

Severe lesions should be recognized and the patient immediately treated as necessary. Most of these conditions are classified as grade IV and fall outside the scope of this book. They will probably be recognized clinically when warning signs are present but certainly be detected by performing the necessary technical investigations: radiography, computed tomography (CT), scintigraphy and/or MRI.

Technical investigations in post-traumatic neck patients are mandatory but the decision as to which imaging technique to use should be based on clinical grounds. Radiographs in patients with soft tissue injuries are often negative: no fractures or luxations are found. The finding most commonly obtained is loss of the normal cervical curvature on a lateral view.[29] CT and MRI are not very helpful in recent cases but may become important when the condition persists, although their use is still controversial. Scintigraphy may be useful to screen for occult fractures.[30]

The reader is referred to the section on non-discogenic disorders (see Ch. 9) for further information about the diagnosis of non-mechanical conditions.

In most cases the condition is *not severe* and clinical examination initially suggests some of the above-mentioned conditions, although the picture may sometimes be vague and difficult to interpret.

Diagnostic difficulties

Most patients present a genuine clinical pattern. The symptoms and signs are clear and not too difficult to interpret. Some patients give a more diffuse picture, probably because there is a combination of lesions. The examiner should then concentrate on the features that are understood and compatible with a known syndrome.

Most difficult are those patients who exaggerate or simulate their symptoms. They pretend to have problems in the hope of persuading the examiner or of making people believe their story. The examiner should look for inherent unlikely details and inconsistencies in the history and functional examination, which will enable a positive diagnosis of 'psychogenic pain' or 'malingering' to be made.

Symptoms

Immediately after the accident the patient is stunned and confused, and complains mainly of any head problem that is present. A feeling of discomfort in the neck, often associated with some degree of nausea, may develop. Although not usually reported, up to 60% of patients have evidence of concussion with momentary loss of consciousness. Examination in a hospital emergency department may not reveal any positive signs and discharge follows. In the following hours and days, a cluster of symptoms may then develop: soreness, tenderness and swelling in the anterior neck region, stiffness and restriction of movement at the neck, headache, visual and auditory disturbances, dizziness, concentration and memory disturbances, pain in the upper thorax, scapular area, shoulder and arms, and numbness or paraesthesia in the upper limbs, accompanied by a feeling of heaviness and weakness. Most symptoms gradually disappear but the majority of patients are left with pain in the neck, radiating to the scapular area or to the shoulder region.

The examiner should enquire about the mechanism and velocity of the trauma so that a judgement can be made about the severity of the injury and so that a prognosis can be reached.[31] Other important information relates to the time interval between the accident and the onset of symptoms, which often is 2–3 days.[32] Many patients have associated low back pain[33] and this is noted and assessed later.

Important information can be obtained by asking the patient to describe exactly the localization of the symptoms: central or bilateral symptoms suggest a condition lying at the midline, whereas unilateral symptoms may stem from either a central or a unilateral condition.

The examiner also concentrates on the inherent likelihood of a certain type of lesion: for example, multisegmental pain in disc lesions, segmental pain in facet lesions, and local pain in muscular lesions. When a patient continuously presents inconsistencies or an improbable combination of symptoms and this is later confirmed during the functional examination, examiners should be on their guard.

Signs

Inspection of the position of the patient's neck may indicate an acute condition: for example, when a torticollis-like picture is found or when muscle spasm is present.

When the head is fixed in flexion with central or bilateral pain, a posterocentral disc displacement is clearly present.

Articular signs – pain on movement with or without limitation – suggest involvement of the intervertebral joint or the facet joints, certainly when movement is shown to be restricted. Discodural or discoradicular interactions are considered when articular signs are accompanied by dural or radicular signs (see p. 145). When there is no dural or nerve root involvement, the possibility of a condition involving the facet joint exists; the pain is purely unilateral, i.e. when either a convergent or a divergent pattern is found. Limitation of movement indicates that the joints are responsible.

End-range pain is typical of ligamentous or muscular conditions, the latter also giving rise to positive resisted movements (see p. 169).

Because combined lesions are not at all uncommon, the clinical picture may become difficult and therefore hard to interpret. It should be looked at in the light of the anatomical findings and compared to the clinical pictures known to occur in the cervical spine (see Ch. 8).

Natural history

The natural history of WAD is difficult to predict. An extensive study in Switzerland found that 30% of patients still complained of symptoms, even 4–7 years after the accident.[34] Another study showed that increasing age and the severity of the initial neck pain were predictors of lasting symptoms after 6 months.[35] Objective neurological signs and degenerative changes on radiographs or MRI (spondylosis, diminution of the diameter of the spinal canal) may be associated with a poor prognosis.

In 30–40% the condition needs up to 1 year to recover, ache diminishing quite considerably in the first 2 months. The pain usually disappears but, when no treatment is given, movements may remain restricted.

Chronicity

Not all patients will develop chronic symptoms after whiplash injury. In most instances it is a benign, self-limiting condition. All patients destined to recover will do so in the first 2–3 months after trauma. Those who do not may claim symptoms for several more years. Recent studies have indicated that between 14 and 42% of patients develop chronic symptoms and that about 10% have permanent difficulties,[36–42] although patients may improve even after many years.[43] A predisposing factor for chronicity could be pre-existing spondylosis.[44]

The QTF study clearly demonstrates that the most common whiplash injury – without bony or cord lesions – is essentially a benign and self-limiting condition. A small number of patients are refractory and responsible for the enormous costs incurred by the injury: about 50% of all costs are spent on an eighth of the total number of victims.

Of the 1 000 000 whiplash injuries per year in the USA, the majority of patients become asymptomatic after a limited number of weeks or months. According to certain statistics, 20–40% continue to have invalidaty symptoms for several years.

In those countries where the entity of 'chronic pain' resulting from rear-end collisions is not known and consequently there is no fear of long-term disability leading to indemnity and litigation, symptoms after whiplash injury are self-limiting, lasting for only a short period of time, and there is no evolution towards the chronic stage.[45]

Therapeutic approach

There is still some discussion about how to approach patients with lasting symptoms from WAD. It is difficult to find objective signs and physicians are therefore divided into two groups: those who believe the patients and those who think in terms of psychogenic pain, personality and chronicity because of concerns about financial compensation.

There is some consensus about the possibility of encouraging the patient to develop an active and positive attitude towards the problem. In those patients who do not present with intensive neck pain immediately following the accident, who do not show clinical signs of cord or root compression and in whom routine radiographs show no bony abnormalities, all further extensive and expensive examinations should be avoided.

Patients should be encouraged to remain active and functional. In the acute phase analgesics and anti-inflammatories should be administered but only temporarily. A collar should not be used for more than a few days and should be replaced by early mobilization of the cervical spine. The patient should be taught how to perform active movements, and prolonged physiotherapy must be avoided in order not to push the patient into a passive disabled attitude.

Such an approach is also recommended by the QTF in order to avoid prolonged disability.[46] An early return to work is advocated as one of the best measures to avoid chronicity. Immobilizing measures such as bed rest and collars are best avoided.

Specific treatment

In those cases where the diagnosis is clear, the condition must be treated properly along with the lesion.

When a discal pattern is found and thus a discodural or discoradicular interaction is present, manipulation is performed immediately because it is unwise to leave the displacement untreated; posterocentral protrusions draw out osteophytes fairly quickly, and this may lead to a situation in which extension or one rotation becomes permanently blocked. Because a posterocentral protrusion is present, great care must be taken in the choice of techniques and operation; rotation movements are avoided and the therapist must be experienced. In acute cases with gross deviation, manipulation is performed every day. Traction in the direction of the deviation, followed by pure traction manipulations without articular movement, quickly leads to full recovery. The more moderate and long-standing cases require more treatments, performed once or twice a week over a few weeks. The techniques for posterocentral

protrusions are used: straight pull, lateral flexion, anteroposterior gliding and traction with leverage (see p. 201).

Facet joint lesions can be treated either with steroid infiltration or with deep transverse massage; in more chronic cases slow stretching is used (see p. 201).

Ligamentous lesions are best treated with deep transverse massage, unless there are adhesions which can be manipulatively broken. Muscular lesions respond to deep transverse friction or infiltration with local anaesthetic. If a combination of ligament and muscle damage exists, the muscle should be treated first.

Proper treatment and re-examination on a regular basis are the guarantees of a maximal therapeutic result.

Access the complete reference list online at **www.orthopaedicmedicineonline.com**

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Treatment of the cervical spine

11

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Before treatment of any kind is undertaken, a correct diagnosis should be made and indications and contraindications carefully considered.

Most discodural and discoradicular interactions are approached by either manipulation or traction. Prophylactic measures may then help to avoid recurrence. In non-reducible discoradicular lesions, nerve root blocks or epidural injections can be used or surgery may prove necessary. Simply waiting for spontaneous cure may also be an option. Non-discogenic disorders may respond to mobilization, manipulation or deep transverse massage. On occasion infiltration and partial immobilization may be required and in exceptional cases surgery is called for.

Treatment of discodural and discoradicular interactions

The purpose of treatment in these disorders is to stop the conflict between the displaced discal fragment and the involved sensitive structure. Because the disc displacement not only affects the dura mater or the nerve root but also disturbs the function of the intervertebral joint, it is logical to try to influence the disc first. Reducible discs can be managed by manipulation or continuous traction, the choice depending on the nature of the displacement: annular displacements are treated by manipulation, nuclear ones by traction. In those instances where, for some reason, reduction has become impossible, measures are taken to sedate the pain during the period of spontaneous recovery. Surgery is called for when the pain becomes intolerable or when a posterocentral protrusion threatens the spinal cord.

Manipulation

Controversy

Manipulation of the cervical spine has become very controversial and its popularity depends on the school of thought of the practitioner. In more orthodox medicine, manipulation as a treatment for spinal conditions is hardly ever used. At the other end of the spectrum are chiropractors whose main treatment is chiefly manipulative. Osteopathy, the McKenzie school and manual therapy are somewhat in between these extremes. Also Cyriax placed his method somewhere in the middle, using manipulation in some well-defined instances – about half of all mechanical spinal conditions – and avoiding it in all the rest.

In light of numerous reports of complications after manipulation of the cervical spine (see below), different schools have

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published in their own class of journal, and manipulation is either defended or rejected as a useful therapeutic measure. Different schools sometimes have totally opposing approaches to the many spinal disorders and arrive at different diagnoses. They then adapt their treatment to what they think is the cause of the symptoms and may therefore manipulate for different reasons. Osteopaths and manual therapists use manipulative techniques to restore range in vertebral segments where they have found loss of mobility; chiropractors try to readjust the spine to correct a perceived misalignment between vertebrae; in Cyriax's orthopaedic medicine and in the McKenzie approach, manipulation is used to eliminate an internal derangement caused by a disc displacement.

Cervical manipulations can cause severe neurologic complications, which are both exceedingly rare and generally unpredictable.[1] More than 200 cases of more or less serious complications after manipulative treatment of the neck have been reported,[2] but it is suggested that they are probably under-reported.[3,4] Many authors have therefore become very cautious in accepting the suitability of this modality. The main arguments against manipulation are the possible hazards to the vertebrobasilar system and the spinal cord that are usually irreversible.[5–10] Therapists who are familiar with manipulative techniques continue to use them because they consider the risks to be minimal. However, such conclusions can only be made after comparative scientific studies have proven that the benefits of manipulation outweigh the risks.

Dangers of manipulation

Treatment decisions cannot be based on effectiveness data alone. Another important factor is safety, obviously. Weighing the risks of spinal manipulation against its benefits is therefore an exercise that should be done constantly. Every act in medicine, whether diagnostic or therapeutic, carries a risk of complications; an injection may result in an anaphylactic reaction, the use of anti-inflammatory agents may cause internal bleeding, and surgical procedures may lead to other, serious complications. It cannot be denied that spinal manipulation too, particularly when performed on the upper spine, is sometimes associated with adverse effects, which are usually mild to moderate. However, serious complications, such as vertebral artery dissection followed by stroke or death, myelopathy and epidural haematoma, have also been described. Mild side effects are very common. Surveys have shown that between one-quarter and one-half of all patients reported at least one post-manipulative reaction. The most common were headache, stiffness, local discomfort and fatigue.[11,12] Most of these reactions began within 4 hours and generally disappeared within 24 hours of receiving spinal manipulation.[13] The frequency of serious adverse events is difficult to estimate, but according to several publications it varies between 5 strokes/100 000 manipulations to 1.46 serious adverse events/10 000 000 manipulations and 2.68 deaths/10 000 000 manipulations.[14–18]

The complication that is feared the most is dissection of the vertebral artery, leading to infarction of the brain, cerebellum or brainstem, so-called Wallenberg's syndrome (see online chapter *Headache and vertigo of cervical origin*).

Vertebral artery dissection

Arterial dissection is an uncommon vascular wall condition that typically involves a tear at some point in the artery lining and the formation of an intimal flap, which allows blood to penetrate into the muscular portion of the vessel wall. Blood flowing between the layers of the torn blood vessel then causes the layers to separate from each other, resulting in arterial narrowing or even complete obstruction of the lumen.

Furthermore, pulsatile pressure damages the muscular layer, resulting in a further splitting or dissection, extending in the direction of blood flow.[19] The accrued blood soon develops into a thrombus and deforms the intima, pushing it into the arterial lumen. Blood flow in the cervical arteries can be obstructed either directly by the haematoma or indirectly by the detached emboli that move distally and obstruct the progressively smaller vessels in the brain, resulting in a stroke (Fig. 11.1).[20]

The cervical arteries are innervated with pain-sensitive nerve fibres that may generate neck pain and headache when provoked. Several studies have shown that pain is typically the first symptom associated with vertebral artery dissection (VAD), and a recent study reported that 8% of the patients presented with head or neck pain as their only symptom.[21] Pain related to VAD frequently occurs suddenly and is of severe intensity, involving mostly the ipsilateral occipitocervical area. These symptoms may or may not be followed by ischaemic involvement in the brain, cerebellum or brainstem. The interval of time between the initial pain of VAD and the ischaemic symptoms is quite variable, with reports ranging from almost immediately to several weeks.

Although the pathophysiology of a dissecting vertebral artery is well understood, the underlying cause of intimal tears remains uncertain. Most experts link VAD to trauma of varying

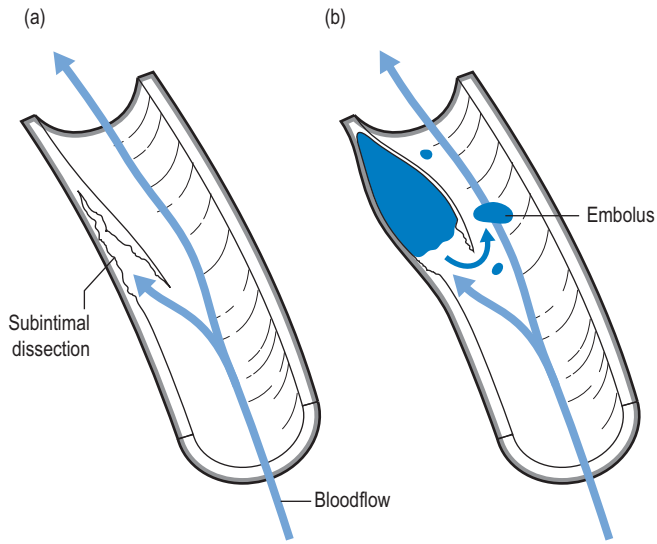


Fig 11.1 • (a) An initial tear in the artery lining allows blood to penetrate into the muscular portion of the vessel wall and causes the layers to separate from each other, forming a subintimal dissection. (b) A thrombus is formed and emboli may detach and travel distally to obstruct progressively smaller vessels in the brain.

degrees of severity and maintain that, because tearing occurs, previous trauma was necessarily involved.[22] A retrospective analysis of 80 patients with vertebrobasilar ischaemia determined to be caused by frank neck trauma reported that 70 of the cases were related to motor vehicle accidents.[23] However, VADs more commonly occur after very minor trauma and even after everyday activities that most people would consider to be non-traumatic. Examples of such trivial 'trauma' include countless everyday activities that involve head and neck movement, such as reversing a vehicle, coughing, vomiting, unusual sleeping positions, having one's hair washed at a beauty salon, and rhythmic movement of the head and neck to music.[24–26] One article listed 68 activities that have been implicated in the development of VAD.[27] Another article reviewing 606 cases of VAD reported that 371 or 61% were spontaneous. The remaining 39% were associated with trivial or other trauma, which included manipulation in 9% of the total number of cases.[28]

So far it remains unclear what the exact precipitating cause of the development of a VAD might be. Given that many VADs are not related to trauma or even abrupt head movements, one must wonder whether a clear cause-and-effect relationship can be established in cases that involve mechanical triggers. It has been suggested that a mechanical trigger is only one of multiple components and that an underlying arterial abnormality is the predisposing factor to dissection.[29,30] This opinion is based on several observations: first, many VADs are not related to trauma and simply occur spontaneously; second, VAD patients frequently have coexisting physiological abnormalities, such as hypertension, recent infection, migraine headache and several others; third, the average person is exposed to trivial events involving the neck on a daily basis, yet most people do not develop VAD.

For the individual therapist who wants to exclude potential disasters, it is extremely important to determine the group of patients at risk and the kinds of manœuvre that are dangerous. Judging from the existing literature, it seems that at this time, neither is possible. Cerebrovascular accidents after manipulation appear to be unpredictable and should be considered an inherent, idiosyncratic and rare complication of this treatment approach.[31] In a retrospective review, Haldeman and co-workers studied 64 medicolegal cases of stroke associated with cervical spine manipulation. The strokes occurred at any point during the course of treatment. Certain patients reported onset of symptoms immediately after the first treatment, while in others the dissection occurred after multiple manipulations. There was no apparent dose–response relationship to these complications. These strokes were noted following any form of standard cervical manipulation technique including rotation, extension, lateral flexion and non-force and neutral position manipulations. The results of this study suggest that stroke, particularly vertebrobasilar dissection, should be considered a random and unpredictable complication of any neck movement, including cervical manipulation. It may occur at any point in the course of treatment with virtually any method of cervical manipulation. The sudden onset of acute and unusual neck and/or head pain may represent a dissection in progress and be the reason a patient seeks manipulative therapy that then serves as the final insult to the vessel, leading to ischaemia.[32] This puts another perspective on the relationship between cervical spinal manipulation and VAD: is VAD caused by the manipulation or is the patient manipulated because of incipient VAD?[33]

Although the risk of serious complications cannot be denied, it should be put into context. The incidence of VAD in relation to cervical manipulation remains very low. One Canadian survey, for instance, reported 23 cases of VAD after cervical manipulation over a 10-year period, representing a rate of 1/584 638 manipulations.[34] Other publications estimate the incidence of VAD to be 1 in a million.[35,36] Furthermore, several authors have made a comparison between the occurrence of injury following manipulation and the complications of other treatments for cervical disorders. The incidence of a serious complication such as a gastrointestinal event after the use of non-steroidal anti-inflammatory drugs is 1 in 1000.[37] After surgical procedures to the cervical spine, 1.6% of complications occur. These figures can be used to argue that risks from other causes such as drug therapy can be between 100 and 400 times greater than after cervical manipulation.[38]

Premanipulative testing

During recent decades, tests have been described to detect vertebrobasilar insufficiency in order to identify those patients who may be at risk of serious post-manipulation complications. These tests are based on the assessment that blood flow to the contralateral vertebral artery is decreased when the cervical spine is rotated and extended to one side.[39,40] This is because, during rotation, the contralateral vertebral artery slides forwards and down, causing it to narrow. However, in normal individuals, collateral vascular supply by means of the circle of Willis is sufficient to assure blood flow and to prevent symptoms (see online chapter *Headache and vertigo of cervical origin*).[41,42]

Mainly under the influence of the Australian Physiotherapy Association, performing a vertebral artery test routinely prior to manipulation has become widely accepted.[43–45] It is believed that the test could determine tolerance to cervical extension and rotation or could differentiate between dizziness caused by vertebrobasilar insufficiency and dizziness caused by other conditions, such as benign paroxysmal positional vertigo and inner ear pathology.[46,47]

Although prudent premanipulative testing seems to reduce the level of risk,[48] both its validity and safety have recently been questioned.[49] Premanipulative testing should not be considered as the ultimate safety precaution; studies have shown a high likelihood of obtaining false negative results, suggesting that the validity of the test is poor.[50–53] Furthermore, it can even be argued that examination procedures are more dangerous than therapeutic manipulation, because the sustained posture required for the test exposes the patient to greater risk than a quick, high-velocity manipulation.[54–56]

The doctor or physiotherapist who intends to manipulate should always be aware that even tests during examination carry a risk and that there will always be a factor of unpredictability even when all premanipulative tests are negative and

even if the patient responded positively to earlier manipulations. Screening for manipulative risks should not rely on the outcome of provocation tests but on the whole clinical picture that emerges after thorough history taking and clinical examination. In this perspective, a recent study that compared the results of a simple questionnaire to a duplex Doppler ultrasound demonstrated excellent sensitivity (1.00) and good specificity (0.78).[57] The questionnaire consisted of the following: 'Do you avoid looking up as if into a high cabinet shelf because doing so causes neurological symptoms such as: visual problems, dizziness, unsteadiness, confusion, headaches and symptoms in the extremities?' The same question was posed in relation to turning the head to the right and to the left, as if reversing a car. Review of the literature, especially of the many case histories that have been described, also leads to the conclusion that a number of serious complications could have been prevented had practitioners recognized warning signs which should have led them to exclude manipulation.[58–60] One review mentions faulty diagnosis, insufficient clinical knowledge or examination, inaccuracy as a result of routine, overconfidence, bad technique and therapeutic obstinacy as the main causes of complications.[61]

In recent years another view of the relationship between cervical manipulative therapy and VAD has emerged. The relationship was usually seen as simple cause and effect, in which manipulation causes a dissection in certain susceptible individuals. Recent evidence, however, suggests that the relationship is not causal, and that patients with VAD often have initial neck symptoms which cause them to seek care; they then have a stroke some time later, independent of the treatment. This new understanding has shifted the focus for the therapist from one of attempting to 'screen' for 'risk of complication with manipulation' to one of recognizing the patient who may have a VAD so that early diagnosis and intervention can be pursued.[62] Again, this recognition can only be achieved by looking at the complete clinical picture and by following the proper diagnostic procedures (Box 11.1).

Box 11.1

Protocol for premanipulative testing

Before manipulation, check the following items:

- Have I taken a proper history and done a thorough standardized functional examination?
- Have I correctly interpreted the examination?
- Have I been able to reach a positive diagnosis of a discodural or discoradicular interaction?
- Have I checked on all possible warning signs?
- Have I checked on all possible contraindications?
- Have I found a clear indication?
- Have I a chance of success?
- Have I done premanipulative testing?
- Have I recalled Cyriax's advice?
- Have I mastered the manipulative technique?
- Do I know the manipulative procedure?
- Do I know the manipulation strategy?

Dangers of not manipulating

The dangers of *not* carrying out manipulation in subjects who need it are as important to consider as the dangers of performing it. Many discodural interactions, especially those causing unilateral or bilateral cervicoscapular pain, have no tendency to spontaneous cure. Unless manipulative reduction is achieved, the patient is left in pain that continues intermittently and that could have been relieved by a few sessions of treatment. Delay is also harmful because there may come a point when a protrusion, originally suitable for manipulation, becomes irreducible or dangerous.

A posterocentral disc displacement that is left untreated exerts constant pressure against the posterior longitudinal ligament. It may slowly become larger or give rise to an osteophyte in the spinal canal as a consequence of ligamentous traction. When the bulge enlarges it may eventually compress the spinal cord as well as the anterior spinal artery (see p. 165). The symptoms and signs elicited may finally become irreversible. For all these reasons it is unwise to leave an early minor disc displacement unreduced.

Evidence: results

Everyone who undertakes manipulation sees good and sometimes spectacular results in daily practice. This is somewhat in contradiction with the results that often emerge from randomized trials.[63,64] The discrepancy is mainly caused by the fact that, in most of the studies, subgroups of patients are not well defined. Prospective randomized trials that take more care with inclusion and exclusion criteria usually result in a stronger trend favouring manipulation. Koes and colleagues, for example, performed a randomized trial on back and neck pain and found promising results for manual therapy and physical therapy in subgroup analyses of patients with neck pain.[65,66] Hoving *et al* conducted a randomized, controlled trial of manipulation, physical therapy, and continued care by a doctor, which confirmed the superiority of manual therapy over physical therapy and continued care.[67] Other authors studied the cost-effectiveness of manipulative treatment. The cost-effectiveness ratios and the cost-utility ratios showed that manipulation was less costly and more effective than physiotherapy or general practitioner care. The manual therapy group showed a significantly faster improvement than the others, with a total cost of less than one-third of that of physiotherapy and general practitioner care.[68] Other randomized clinical trials in patients with mechanical neck pain confirmed clinically and statistically significant short- and long-term improvements in pain, disability and patient-perceived recovery when treated with manipulation.[69–71]

Precautions

In orthopaedic medicine the utmost precautions are taken to avoid any possible complication. Therefore the therapist must follow a strict routine (see Box 11.1). First of all, there should be a firm clinical diagnosis, if necessary confirmed with

technical investigation. Warning signs and contraindications are heeded. Manipulation is only considered if there is an unambiguous indication for it. Prognosis is determined and, if provisionally positive, manipulation is begun with strict methods. The manipulator will only proceed when sure of personal skill. During each manipulative session, constant reassessment is made and the decision as to whether or not to continue is totally dependent on the response obtained so far.

Proper history taking followed by a thorough standardized functional examination

The diagnosis of discodural or discoradicular interaction is made on clinical grounds and therefore a detailed history is necessary, which takes time. The complaints are recorded chronologically from the first appearance of symptoms up to the present. All possible symptoms are noted and a record is made of their behaviour and relationship to activity, movement and/or posture. Functional examination is always complete and no tests are omitted. Movements are examined for pain and range, and muscle strength is ascertained. Neurological features are tested in detail.

Proper interpretation of the examination (clinical reasoning)

The results of the examination, both positive and negative, are then interpreted in the light of anatomical reality (see p. 181). Correlation between history and functional examination is sought (inherent likelihoods): does the behaviour of the pain (onset and evolution, referred versus local) and the paraesthesia (development, presence and pattern) match the clinical findings? Examination features are related to possible articular, root or cord origin. Articular signs are mirrored by specific patterns of limitation. Root involvement may be obvious from motor or sensory deficit or disturbances of reflexes. Evidence of cord involvement is revealed by particular patterns of neurological presentation.

Diagnosis: discodural or discoradicular interaction

These interactions are diagnosed when the whole clinical picture is compatible with that of disc displacement – symptoms and signs are found that incriminate one or several of the following structures:

- *The dura mater and/or dural nerve root investment.* Multisegmental pain and tenderness show the dura mater to be affected; pure segmental pain points towards the investment of the nerve root. Scapular pain on coughing is considered to be a dural symptom.
- *The intervertebral joint.* An asymmetrical pattern of pain and/or limitation ('partial articular pattern') indicates internal derangement in the intervertebral joint. Other features are pain on movement and/or posture, twinges and, in more acute examples, deviation of the head.
- *The nerve root parenchyma.* Segmental (dermatomal) paraesthesia may accompany the root pain. Neurological deficit (motor, sensory and reflexes) is often present.

Box 11.2

Warning signs in cervical disorders

History

- Gradually increasing pain
- Expanding pain
- Bilateral arm pain
- Arm pain in a patient under 35
- Arm pain for more than 6 months

Examination

- Full articular pattern
- Muscle spasm
- Resisted neck movements that are painful and weak
- Side flexion away from the painful side as the only painful movement
- Limited scapular elevation
- Horner's syndrome
- Hoarse voice
- T1 palsy
- Excessive loss of power
- Involvement of two or three nerve roots
- Muscle weakness without root pain
- Wrong time sequence

- *The spinal cord.* Multisegmental paraesthesia in the hands and/or feet is provoked by neck flexion; other signs of motor and/or sensory tract involvement may also be found.

Exclusion of warning signs

During history taking and functional examination, the examiner must remain alert and constantly aware so as not to overlook possible warning signs (Box 11.2). The presence of any one of these red flags indicates a non-mechanical lesion and is an *absolute bar to any active treatment*. The patient should be referred for further investigations immediately.

Exclusion of contraindications

It is crucial to know when a manipulation is unsafe. In this regard, data collected from history and clinical examination are far more important than the outcome of one or more premanipulative tests (see p. 183). Great caution should therefore be observed in detecting potential contraindications during history taking and functional examination (Box 11.3).

Absolute contraindications

The following conditions are absolute contraindications to manipulative treatment: compression of the spinal cord, basilar ischaemia, drop attacks, rheumatoid arthritis, anticoagulant therapy, ankylosing spondylitis and an adherent dura mater.

Compression of the cord

The moment that there is clinical evidence of an upper motor neurone lesion, manipulation must be abandoned. Symptoms are: paraesthesia in the hands and/or the feet influenced by

Box 11.3

Cervical manipulation for disc lesions:

Contraindications

Absolute

- Compression of the spinal cord
- Basilar ischaemia
- Drop attacks
- Rheumatoid arthritis
- Anticoagulant therapy
- Ankylosing spondylitis
- Adherent dura mater

Relative

- Posterocentral disc protrusion
- Acute torticollis in young patients
- Gross deformity
- Manipulation increases root pain

Futile

- No protrusion
- Root pain lasting too long
- Root pain with neurological deficit
- Root pain with swift progression
- Root pain with primary posterolateral development
- Unfavourable neck signs
- Nuclear disc protrusion

neck flexion.[72] Signs are: positive plantar reflex, positive Hoffman sign, spasticity and incoordination.

Basilar ischaemia

A patient who complains of vertigo or dizziness on neck movements or on changing posture should not be manipulated, until further investigations with Doppler ultrasound and/or magnetic resonance imaging (MRI) have shown no vascular lesions. Even then, only techniques under strong traction and with a minimum of articular movement (straight pull and traction with leverage) should be used.

Drop attacks

Drop attacks, whatever their possible cause, constitute an absolute bar to manipulation. The examiner is not only alerted by the history but also can be warned by the end-feel during clinical examination or any attempt at manipulative treatment.

Rheumatoid arthritis

This condition can lead to ligamentous laxity at the upper cervical joints, which creates an absolute contraindication to manipulation.[73] The typical soggy end-feel puts the examiner on his/her guard.[74,75]

Anticoagulant therapy

It is unwise to manipulate a patient who is on anticoagulant therapy because of the danger of an intraspinal haematoma.[76] Only if the therapy can be stopped for the duration of treatment is manipulation possible.

Ankylosing spondylitis

In the inflammatory stage of ankylosing spondylitis or in the unlikely event of a patient with this disorder developing a disc lesion, manipulation is not at all safe, especially in the cervical spine, where luxations, fractures and cord compression have been described.[77,78]

Adherent dura mater

The dura mater may become adherent to the cervical or thoracic vertebrae. This becomes apparent during an attempt to manipulate, even in patients who otherwise seem suitable. When traction is applied, the patient has paraesthesia in the hands. Traction must be released immediately and manipulation abandoned.

Relative contraindications

Other conditions – posterocentral discodural interaction, acute torticollis in young patients, gross deformity, brachial pain during an attempt to manipulate – are not complete restrictions to manipulation but form a contraindication to certain techniques.

Posterocentral disc protrusions

Rotation techniques are contraindicated. The larger the protrusion seems to be, the more the manipulator resorts to techniques without articular movement. Techniques are used under considerable traction, the effects of which help to reduce the fragment of disc (see p. 196).

Acute torticollis in patients under 30 years

Muscular guarding renders manipulation under traction in the direction limited by spasm impossible. Because the condition is the result of nuclear prolapse, the very restricted rotation and lateral flexion initially present are increased by gently sustained pressure. Restoration of movement is thus achieved (see p. 196).

Gross deformity

Significant deviations of the cervical spine, either in side flexion or in flexion, make manipulation under traction impossible. Before the usual techniques can be used, the manipulator must bring the patient's head back to the neutral position. This happens after repeated tractions (whether or not with manipulative thrust) in the line of the deformity (see p. 196).

Manipulation causing brachial pain

The slightest increase in root pain during initial traction shows that the technique chosen is not suitable, because it clearly pushes the protrusion further against the nerve root.

Circumstances in which manipulation is futile

It is inappropriate to manipulate when a disc protrusion is not present, when the root pain has lasted for too long or when neurological deficit has supervened.

Protrusion absent at the time of presentation

It is possible for the patient to give a history indicative of disc trouble but protrusion is not currently present, in which case clinical examination is negative. Manipulation during such a symptom-free period is futile. The patient should wait until the condition relapses or, in the presence of an unstable fragment, measures should be taken to prevent recurrences. It is also inappropriate to use manipulation, as chiropractors often

do, as a prophylactic measure. This approach cannot prevent the disc from shifting again at any time.

Root pain lasting longer than 2 months

Experience has shown that root pain that persists for more than 2 months seldom responds to manipulation. The worst is over, and spontaneous recovery is to be expected within 1 or 2 months. However, in the exceptional circumstance of discogenic root pain lasting longer than 6 months, the apparently delayed mechanism of spontaneous cure can be restarted by manipulation.

Root pain with neurological deficit

The presence of a neurological deficit shows that the discoradicular interaction is too intense. The disc fragment cannot therefore be replaced but a vain attempt to do so is not dangerous. An occasional case with very minimal deficit may, in fact, respond.

Root pain with swift progression

The patient develops cervicoscapular pain, followed by root pain and paraesthesia the next day. The protrusion moves very quickly from a posterocentral position to a fully posterolateral one, and this course cannot be stopped. Neurological deficit may be expected very quickly.

Root pain with primary posterolateral development

When root pain is caused by a unilateral disc protrusion, it does not respond to manipulative reduction because, as in the lumbar spine, the displacement is nuclear.

Unfavourable neck signs

When some neck movements elicit or influence the root pain, the chances that manipulation will work are very small.

Nuclear disc prolapse

These cases are characterized by an elastic recoil, felt during a manipulative attempt and through the discrepancy between active and passive rotation during clinical examination. Acute cases have to be reduced by gradual forcing in the restricted directions. Chronic circumstances need continuous traction.

Recognition of a clear indication

It is not enough to have excluded warning signs and contraindications. It is just as important to make sure that a clear indication is present: a posterocentral or posterolateral disc protrusion causing a discodural or discoradicular interaction (Box 11.4).

Box 11.4

Cervical manipulation for disc lesions:

Indications

- Posterocentral discodural interaction with unilateral cervicoscapular pain
- Posterocentral discodural interaction with central neck pain or bilateral cervicoscapular pain
- Posterocentral discoradicular interaction with bilateral root pain
- Posterolateral discoradicular interaction with unilateral root pain, without neurological deficit

Posterocentral discodural interaction with unilateral cervicoscapular pain

Acute torticollis with side flexion deformity

Acute torticollis constitutes a good indication for manipulation and the patient may be helped within a few sessions. Although the course of this condition runs towards spontaneous cure, treatment helps from the outset to diminish the pain.

A distinction is made between torticollis in the young (under 30 years old), which is usually of the nuclear type, and that in those over the age of 30, who suffer a cartilaginous displacement (see p. 196).

Unilateral cervicoscapular pain

Symptoms of this nature are almost always the consequence of an annular displacement. They can be treated with manipulation at any time and, no matter how long the protrusion had existed, the response is usually good. Full recovery is to be expected after 1–3 sessions. Protrusions at the mid-cervical spine (C2–C3 and C3–C4) with pain felt at one side of the upper neck tend to be more difficult to reduce.

Posterocentral discodural interaction with central neck pain or bilateral cervicoscapular pain

Central or bilateral neckache with or without radiation to the trapezii or scapular areas, but without symptoms of spinal cord compression, can be manipulated, provided the techniques are adapted to the specific situation. The manipulator has to realize that a posterocentral displacement may threaten the spinal cord and therefore great care is needed.

Acute torticollis with flexion deformity

This situation is more delicate because the protrusion is large and can impinge against and threaten the spinal cord. This is a clear contraindication to the techniques usually used in the treatment of unilateral disc displacement (see below), i.e. rotation. Special techniques that avoid too much articular movement are used.

Chronic neckache

Central or bilateral pain can be treated with manipulation, provided that rotatory techniques are avoided.

Posterocentral discoradicular interaction with bilateral root pain

If root pain is thought to be the result of a disc protrusion, no time limit is set for the manipulation to succeed – unlike unilateral cases. Slight bilateral root pain preceded by paraesthesia in both hands tends to respond well, although the discal cause is not always certain.

Posterolateral discoradicular interaction with unilateral root pain, without neurological deficit

The success of manipulative treatment in such circumstances depends on the stage at which the patient is seen. Reduction by manipulation is possible only in the first 2 months of the development of this clinical scenario, as measured from the moment that the root pain sets in. Prognosis is based on the clinical picture: neck movements that elicit or influence the pain in the scapular area rather than down the arm show the protrusion to lie in contact with the dura mater, suggesting that reduction may still be possible; when cervical

movements increase the root pain rather than the scapular pain, the chance of success is very poor.

Once neurological deficit has appeared (muscular weakness, sensory loss and sluggish or absent reflexes) no further benefit can be expected from manipulative treatment, except in the following two situations:

- *When severe scapular pain has remained after the root pain began*: usually the scapular pain and the articular signs disappear or considerably diminish when brachial pain occurs. In the rare event that some movements remain limited and cause severe pain in the scapular area, especially at night, one manipulative session may abolish the scapular pain and restore a full painless range of movement. The root pain remains unaltered and continues its spontaneous course (see p. 157).
- *When root pain lasts for a considerable period*: most root pain progresses to full recovery in the course of 3–4 months – except C8 pain, which may take up to 6 months to recover. Occasionally a patient may be seen with root pain that has lasted longer than 6 months, even up to 1 or 2 years, and investigation has not shown a neuroma or other similar lesion. Manipulation will not immediately influence the root pain; however, a few days after manipulation, the pain starts to diminish and after a second treatment 2 weeks later all symptoms may disappear. This peculiar clinical phenomenon could be explained by the concept that, although manipulation shifts the discal rim back immediately, it takes some time for the swelling and inflammation to abate and thus for pain to diminish.[79,80]

Prognosis: criteria of reducibility – presence of favourable or unfavourable signs

Before manipulation is decided upon, the chances of success should be assessed by considering the following questions. By answering these four questions one can make a reasonable prognosis for a number of clinical situations (Box 11.5).[81]

How large is the protrusion?

Gross deviation indicates that a large fragment of disc is displaced. Reduction may take more sessions of manipulation. The more pronounced the articular signs, the larger the protrusion. However, this does not mean that reduction would be impossible, as, for example, in acute torticollis. Neurological deficit indicates that the shift is too large (prolapse or extrusion) to make reduction possible.

Is the protrusion central, unilateral or bilateral?

A unilateral protrusion usually responds well to ordinary manœuvres, i.e. rotation and side flexion movements under traction.

A central or bilateral protrusion is more difficult and forms a contraindication to rotational manipulation. Other manœuvres are possible, except in severe cases, in which strong traction is applied without any movement.

A central protrusion with spinal cord compression is potentially dangerous and any sign – positive plantar reflex, spasticity, incoordination, gross weakness – must therefore be considered as an absolute bar to manipulation (see p. 185).

Box 11.5

Criteria of reducibility

Protrusion is reducible in:

- Unilateral pain in the neck and/or scapular area without root pain or neurological deficit (one or two sessions are needed)
- Unilateral cervicoscapular pain with root pain but without neurological deficit and with 'favourable' neck signs (mostly reducible)
- Bilateral pain in the cervicoscapular area with 'favourable' neck signs and irradiation down the arms plus paraesthesia in hands and/or feet (50% chance of successful manipulation in 1–4 sessions)
- Unilateral scapular pain with root pain and slight paraesthesia existing for less than 1 month and with good neck signs (sometimes reducible)

Protrusion is irreducible in:

- Unilateral pain in the scapular area, with root pain but no neurological deficit, and with 'unfavourable' neck signs
- Bilateral pain in scapulae and arms and paraesthesia in hands and/or feet with 'favourable' neck signs and without neurological deficit (50% are irreducible)
- Unilateral root pain followed by scapular pain
- Unilateral scapular pain with root pain and slight paraesthesia with 'favourable' neck signs and existing for longer than 1 month but less than 6 months
- Unilateral scapular pain that goes on after the disappearance of root pain, with only one movement being painful
- Unilateral scapular pain and root pain with marked neurological deficit and 'unfavourable' neck signs
- Quick evolution from scapular pain to root pain with paraesthesia and 'favourable' neck signs
- Elastic recoil and limitation of active rotation
- Paraesthesia in hands and/or feet with cord signs

Unilateral scapular pain and root pain for >6 months with reasonable neck signs and in which any neurological deficit is recovering:

- May be manipulated to restart the mechanism of spontaneous recovery: two or three sessions at 2-week intervals suffice

Is the pain influenced by articular movements?

When movements affect scapular pain, this is considered as 'favourable' and manipulation has a fair chance of success. Circumstances in which neck movements augment root pain have to be regarded as 'unfavourable', as the result of manipulation is usually inferior (see p. 187).

How long has the protrusion been present?

Scapular pain or bilateral root pain can be manipulated at any time, unilateral root pain within the first 2 months.

Decision to manipulate: patient's consent, type of patient

The decision to manipulate should be taken after all the safety measures previously mentioned have been considered. Some authors propose asking for a patient's consent, perhaps even in writing. Though this is not standard procedure, it is clear that

patients should be informed about what treatment will be started. Simple, honest and easily understandable information about the risks should be included but only up to a certain point. Some patients are easily scared. If they are given too much information about the possible dangers, they will have difficulties in accepting the treatment or relaxing during the manipulative manœuvres.

The manipulator must, of course, take into account the patient's personality. It is clear that certain patients are not suitable for manipulation because they cannot relax. Others, for some reason, do not want to get better; these patients are not manipulated.

Manipulation technique

General remarks

Position of the patient

The patient lies face upwards on the couch with the occiput just beyond the upper edge. This enables the manipulator to hold the patient's neck either in slight extension or in neutral position during the manipulative manœuvres. Physiological lordosis is maintained and positively influences the direction in which the discal fragment moves. Dentures must not be removed, to avoid uncomfortable overclosure of the mouth.

The patient must try to relax during the entire manœuvre because any muscular tension may result in after-pain. Holding on to the edges of the couch (with the intention of resisting the traction) is not allowed, because this makes the muscles of neck and shoulders contract, so complicating manipulation.

Position of the assistant

An assistant is needed for fixation. Positions are chosen whereby the patient's body does not move when traction is applied to the head. There are a number of possibilities (Fig. 11.2).

Fixation at the patient's feet

This is the most common method of fixation. The assistant stands at the patient's feet, takes hold of the ankles and bends forwards to bring his/her shoulders over the patient's feet. When the assistant's arms are then adducted, the feet are firmly fixed between the assistant's body and upper arms.

Fixation at the patient's shoulder

This is needed during a side-flexion manipulation. The assistant stands at the patient's side, opposite to the direction in which the head will be moved, and passes one arm between the patient's arm and body until the hand lies under the shoulder; the other hand lies on top of the shoulder. The fingers are clasped. The assistant's foot is placed against the leg of the couch and the assistant leans well backwards to resist the traction.

Fixation at the patient's side

This fixation method is used during the lateral gliding technique. The assistant stands at the side of the patient, who is asked to move towards the assistant. With one hand the assistant grasps the patient's opposite upper arm, and with the other hand takes the lower arm. The patient's body is then pulled firmly against that of the assistant, who will thus be able to resist the movements executed at the patient's head.

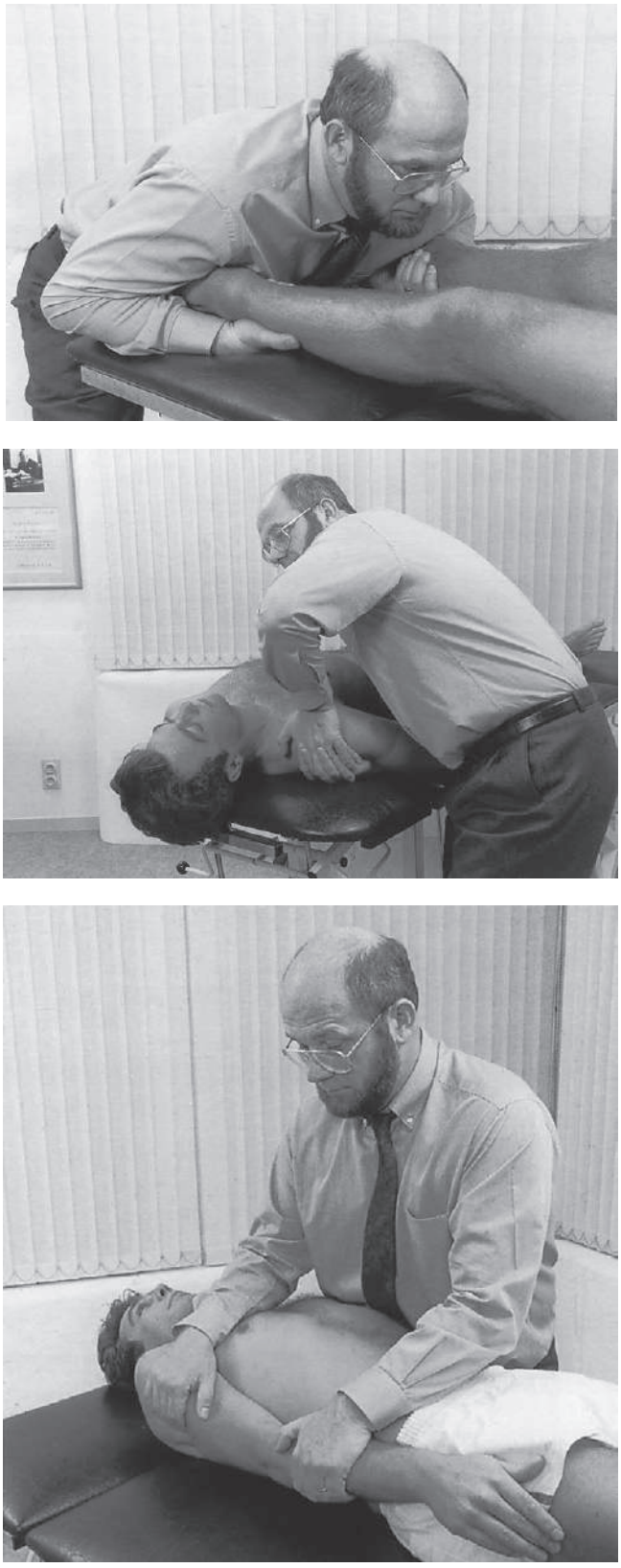


Fig 11.2 • Positions of the assistant for fixation: (a) at the patient's feet; (b) at the shoulder; (c) at the side.

Position of the manipulator

All techniques, except the lateral gliding manœuvre, are performed under *traction*. The maximum traction that is tolerable for the patient and allows relaxation is used. Traction is of vital importance because it has the following beneficial effects:

- Traction induces a subatmospheric pressure in the joint which results in a suction effect – a centripetal force.
- Compression on the pain-sensitive structures exerted by the protruded disc diminishes, with a resulting diminution or even disappearance of the pain. As an additional advantage, the patient will have no problems in relaxing muscles.
- As the muscles are well relaxed, the vertebrae can be distracted by a few millimetres so that the loose fragment has room to move.
- The facet joints are disengaged, which again makes movement easier.
- The posterior longitudinal ligament is taut and helps to exert a centripetal force on the disc.

All these factors combine to push the disc away from the spinal cord and the anterior spinal artery, which are thus protected.

In order to exert traction, the manipulator puts his/her feet against the legs of the couch, after taking hold of the patient's head. One leg is internally rotated and the other externally rotated, so that purchase is possible with the outer mid-tarsal area of one foot and the inner mid-tarsal area of the other. This position enables the manipulator to lean back and to use the body weight for traction.

Traction is built up gradually, which enables the operator to take up the slack when the vertebrae separate. When both feet are positioned against the couch and the manipulator moves away from the patient's head, by extending the knees and arms, maximal traction is obtained. This is not often necessary. The amount of traction depends on the type of patient. It is evident that much less traction will be needed in, for example, a woman of 45 kg than in a man of 95 kg. The traction is adapted to the patient's constitution and also to the ability of the patient to relax. Some patients are inclined to tense their musculature as soon as the head is pulled, others just relax under strong traction because it brings pain relief. The manipulator's experience is crucial in determining the amount of traction.

In order to become accustomed to how to apply traction, novices are advised in the beginning to put one foot further back. The traction force will be less. The foot is then gradually brought forwards until it reaches the couch (Fig. 11.3). The further the foot is brought forwards, the more traction is applied.

One advantage of using traction is that the operator's body, rather than hands, executes the traction element as well as the movement during taking up of the slack. The result is that both elements are much better controlled than if they were performed by the hands only.

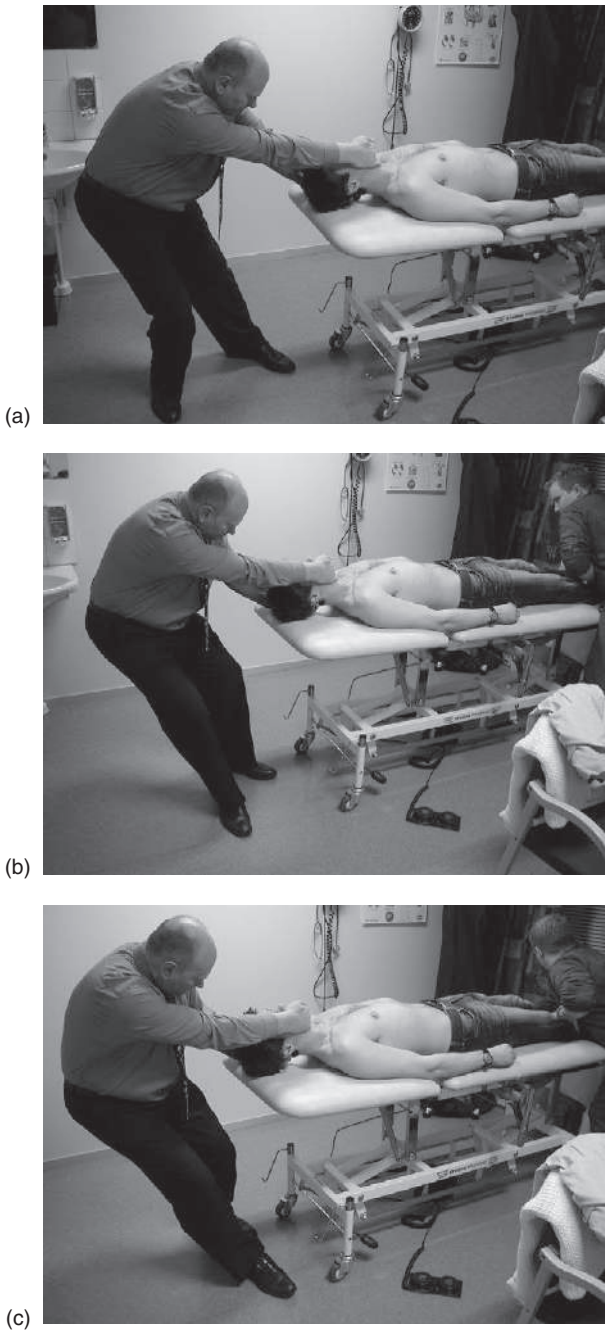


Fig 11.3 • Degrees of traction during manipulation: (a) slight traction, (b) moderate traction, (c) full traction.

Preparative massage to splenius and semispinalis capitis muscles at their mid-cervical extent

Deep transverse massage to these muscles can be used in those patients who have difficulty relaxing during manipulation. Massage will help to inhibit voluntary contraction.

The couch should have an opening near its head, through which the patient can breathe while face down. If this is not available, the patient's head is supported on a pillow.

The patient lies prone and the therapist stands level with the patient's head at the contralateral side. The fingers are placed on the affected area while the thumb supplies counter-pressure at the ipsilateral side of the neck (Fig. 11.4). Massage is imparted for 10 minutes by alternately flexing and extending the wrist, thus moving the fingers over the muscles.

Manipulation follows immediately.

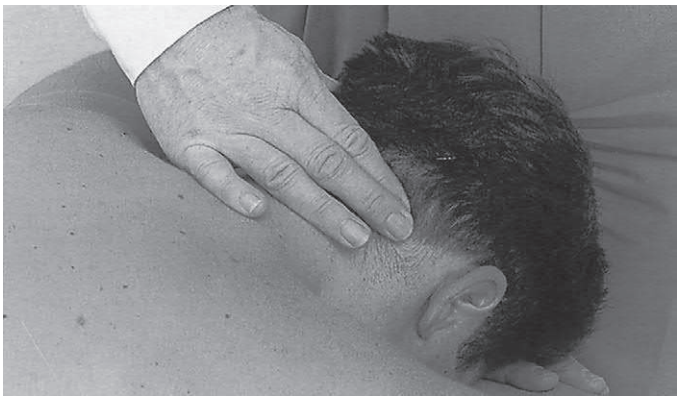


Fig 11.4 • Preparative massage to the splenius and semispinalis.

Techniques

Circumduction during traction

The patient lies supine on the couch, an assistant holding the feet.

After deciding which direction to turn the head in, the manipulator hooks one hand under the patient's chin. For rotation to the right the right hand is used, and the left hand for left rotation. The little finger is held in extension to avoid pressure on the trachea. The other hand grasps the occiput between thumb and fingers and supports the head. When the patient's head is grasped, pinching the chin or the occiput must be avoided. This could be very uncomfortable for the patient and might cause problems with relaxing. The manipulator now puts his feet against the legs of the couch, pointing them in the same direction as the rotation movement to be performed, and leans backwards. Moderate traction is exerted and during this, some slow rotary movements are executed without approaching the full range. This gives some idea of the state of the patient's neck. The patient now realizes what is to happen; confidence is established and the patient can relax.

During the manœuvre some 'cracks' may be felt. After a few movements traction is released.

Practitioner's checklist

- Use to accustom patient to techniques under traction
- Use to enable better relaxation
- Not a real manipulation but a preparative mobilization
- Moderate traction
- No impulse

Straight pull

The patient and assistant adopt the same position as for circumduction during traction. The manipulator hooks one hand under the patient's mandible and the other under the occiput, puts his feet against the couch and leans well backwards. The elbows are straightened as far as possible (Fig. 11.5).

After taking up the slack and assessing the end-feel, the therapist sharply pulls his shoulders backwards. This movement results in a quick manipulative thrust in line with the spine, during which a click may be felt. Traction is now gradually released.

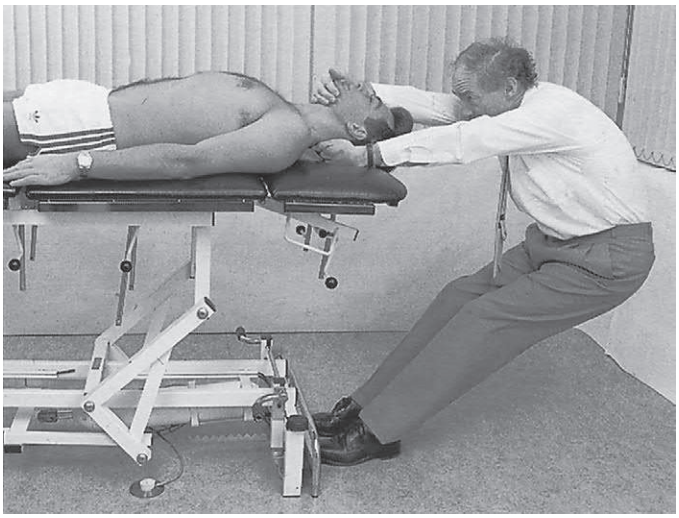


Fig 11.5 • Straight pull.

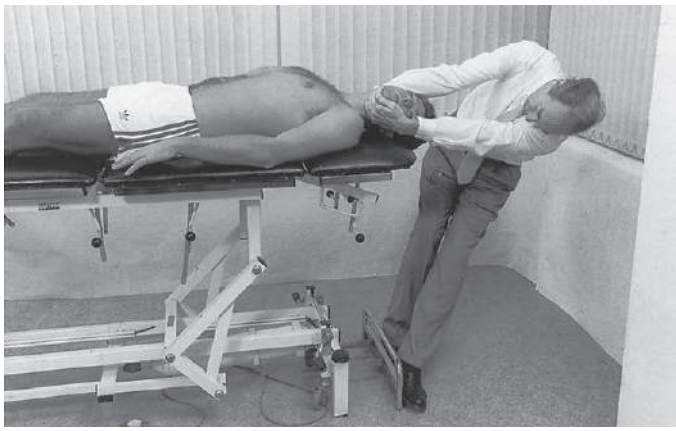
Practitioner's checklist

- Avoid neck flexion
- Use a comfortable grip
- Lean well backwards
- Equal traction by both hands

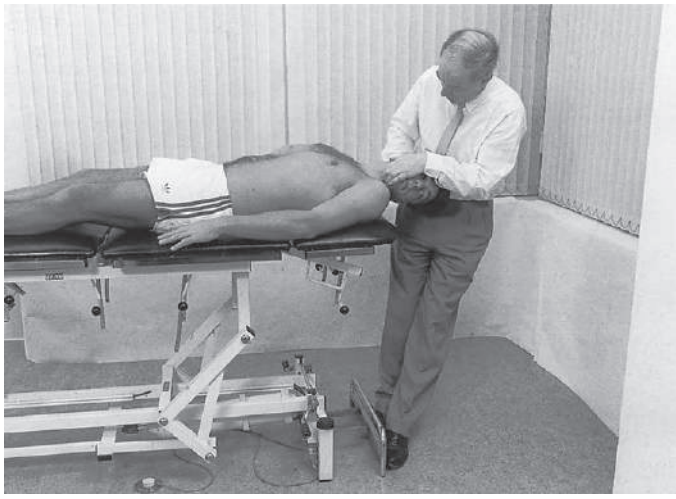
Rotation during traction

The patient and assistant take up the same position as described for the previous two manœuvres. The manipulator takes hold of the patient's head, places his feet against the couch and leans backwards to take up the slack (Fig. 11.6a). For a rotation to the right, the manipulator's right hand is at the patient's chin and the feet point in the same direction. The head is now slowly rotated to the right. This is not done with a hand movement but is the result of the manipulator side-flexing his trunk. The head is turned to half-range on the first occasion, then two-thirds and finally to the end of range. The degree of rotation depends on the response obtained from the patient, which is reassessed after each manœuvre. At full range the end-feel should be assessed and, when this is right, the impulse is given: a quick movement over a very small amplitude (1–2° only). In contrast to the previous trunk movement, this final thrust is performed with the hands only. The head is then brought back to the neutral position and traction is released.

The further the head is rotated, the more chance there is that one or more clicks will be felt. They are relevant when the movement seems to have improved afterwards. When no click is felt, nothing has happened. The end-feel at the end of the manipulative thrust tells the manipulator if further forcing is indicated.



(a)



(b)

Fig 11.6 • (a) Rotation during traction. (b) Full rotation during traction.

Practitioner's checklist

- Direction of rotation determines position of hands
- Feet point in the direction of rotation
- Equal traction during the entire manœuvre
- At the end of range, assess end-feel before manipulation

Full rotation during traction

When repetition of the previous manipulation ceases to afford any benefit, this technique may follow. The traction force will be somewhat less but it will be easier to achieve full rotation (Fig. 11.6b).

The patient and assistant maintain the same position.

For a rightwards rotation the manipulator puts the right hand on the patient's left cheek; the arm is held in pronation, the thenar eminence lies on the maxilla and the fingers are hooked under the chin. The other hand grasps the occiput as previously described for the circumduction during traction technique. After putting his feet against the couch, the operator leans backwards, thus applying traction, and turns the patient's head to full range.

When the end-feel is good, a quick manipulative thrust will be performed. The head is turned back to neutral and traction is relaxed.

Practitioner's checklist

- Thenar eminence on maxilla; avoid chin movement
- Pure rotation, no lateral flexion component
- Assess end-feel before manipulation
- Equal traction during the entire manœuvre

Side flexion during traction

The patient lies supine on the couch. The assistant stands level with the patient's shoulder on the painful side to apply counter-traction (Fig. 11.7) (see p. 189).

The manipulator puts one hand under the patient's occiput. The second metacarpophalangeal joint is applied against the posterior aspect of a facet joint at the mid-cervical area. The thumb is kept in extension to avoid pressure on the external carotid artery and the third, fourth and fifth fingers support the head. The other hand is hooked under the mandible in such a way that the forearm comes to lie anteriorly to the ear. The head will thus be prevented from rotating too much during side flexion (see Fig. 11.7b). The lateral aspect of the ipsilateral foot is now put against the nearer leg of the couch. The other leg is extended out and backwards. Traction in line with the spine is now achieved and this will be facilitated by bending the knee.

After taking up the slack the manipulator now swivels the body, thus bringing the patient's neck into side flexion with the help of the forearm. Simultaneously, counterpressure is applied by the other hand. When the movement comes to an end, the end-feel is assessed and the manipulation performed with a quick adduction movement of the contralateral shoulder. A click is felt, after which the head is brought back to the neutral position and traction released.

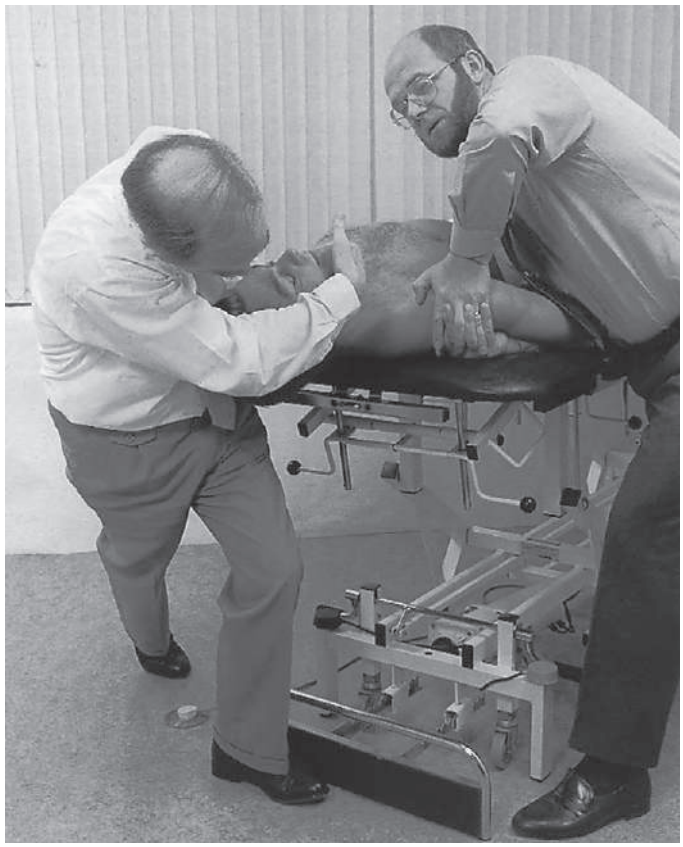
Practitioner's checklist

- Traction in line with the spine
- Avoid rotation
- Counterpressure during lateral flexion movement
- Assess end-feel before manipulation

Anteroposterior glide during traction

The patient lies supine on the couch, the head well over the edge and the shoulder level with it. An assistant holds the feet.

The manipulator stands at one side of the patient's head, facing it. One hand supports the head under the occiput and the other is positioned at the patient's chin using the following grip: the web between thumb and index finger is applied to the chin so that thumb and index finger lie on either side. The forearm is kept vertical. The flexed third, fourth and fifth fingers hook under the chin in order to enable traction. The manipulator now places his feet against the legs of the couch



(a)

(b)

Fig 11.7 • (a) Side flexion during traction. (b) Position of the hands for the side flexion technique.

and leans sideways, using his body weight for traction. After the slack is taken up, the head is gently glided anteriorly with the use of the hands (Fig. 11.8a). It is then glided posteriorly as far as it can go with a manipulative thrust, the result of a sudden flexion of both knees (Fig. 11.8b).

Practitioner's checklist

- Stand at one side of the patient's head
- The patient's head must be well over the edge of the couch
- Traction is obtained by leaning sideways
- Gentle anterior glide, followed by sudden posterior glide
- Avoid flexion–extension movement

Lateral gliding

The patient lies supine on the couch. The assistant stands at the patient's side and firmly fixes the patient's body (see p. 189).

The manipulator stands at the patient's head, feet apart. With the forearms supinated, the hands support the head and, with the thumbs kept horizontal along the cheeks, the thenar eminences are placed anteriorly to the ears on either side. Movement is now carried out without traction. By bringing the body weight over one leg and then back to the other as well as taking the patient's head along with the movement, the neck is moved sideways (Fig. 11.9). The position of the hands should enable the manipulator to maintain the patient's head in line with the body. Side-flexion movements should be avoided and the movement is purely horizontal.

Practitioner's checklist

- Avoid compression of the ears
- No traction
- Avoid side-flexion movements
- Avoid flexion
- Pure horizontal gliding

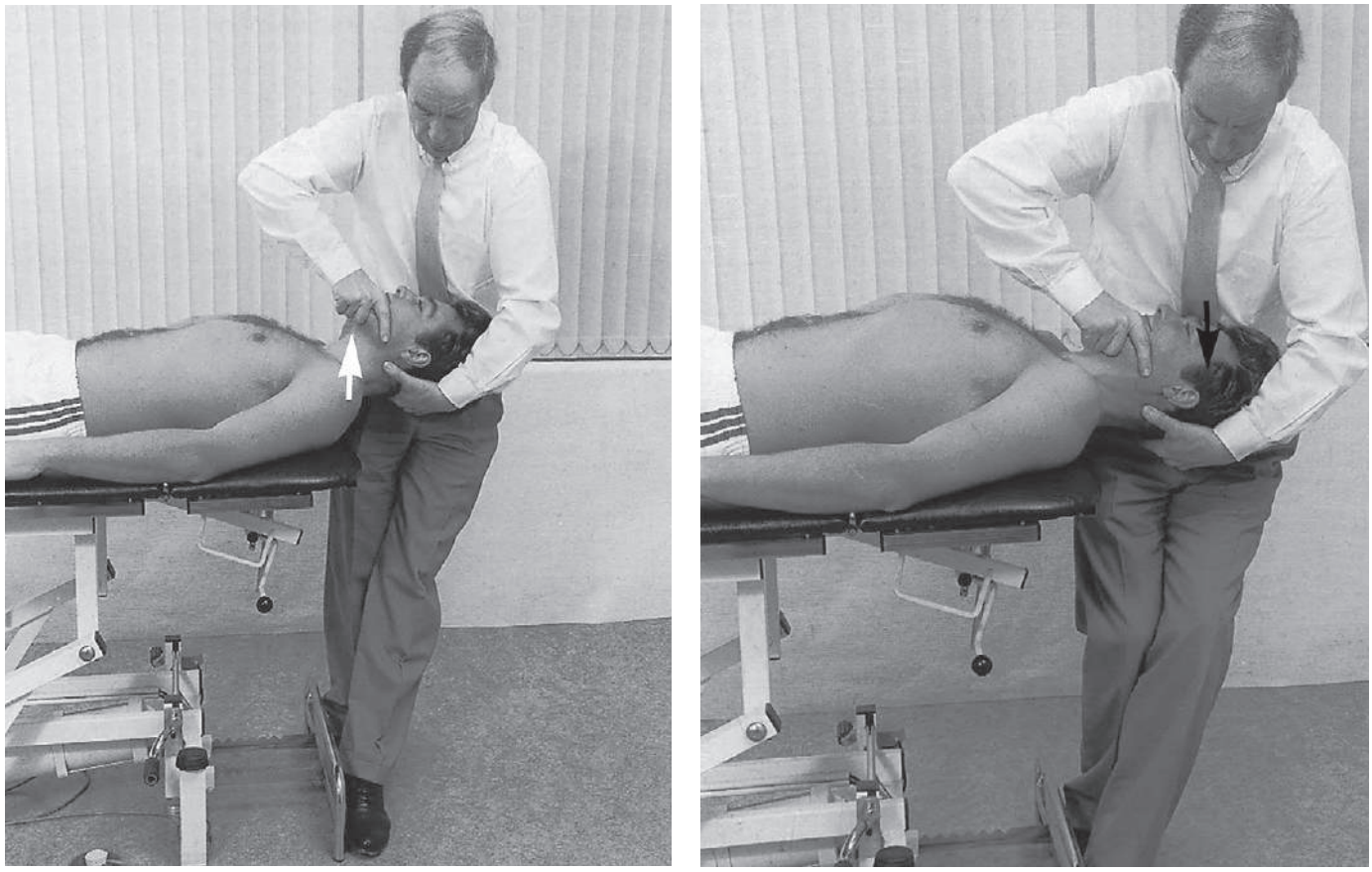
Traction with leverage

The patient lies supine on the couch with the occiput exactly level with the upper edge; an assistant holds the feet firmly so that the body does not move at all during the manipulative thrust.

The manipulator puts one hand, protected by a layer of sponge rubber 2–3 cm thick, under the patient's occiput. The patient's mandible is grasped with the other hand, holding the neck in slight flexion.

Before manipulating, great care is taken in positioning the patient's head in relation to the edge of the couch. An attempt is made to move the head gently further into extension so as to assess how far this movement can go. If extension can take place to a considerable degree, this is because the patient is too high on the couch; if there is no movement at all, this indicates that the patient is too low. It should be possible to place the patient's head in the neutral position where it comes to a stop.

The manipulator then puts the feet against the couch and leans right back until the whole or part of the body weight is sustained by the hand under the chin. Traction is not applied



(a) (b)

Fig 11.8 • Anteroposterior glide during traction: (a) starting position; (b) end position.

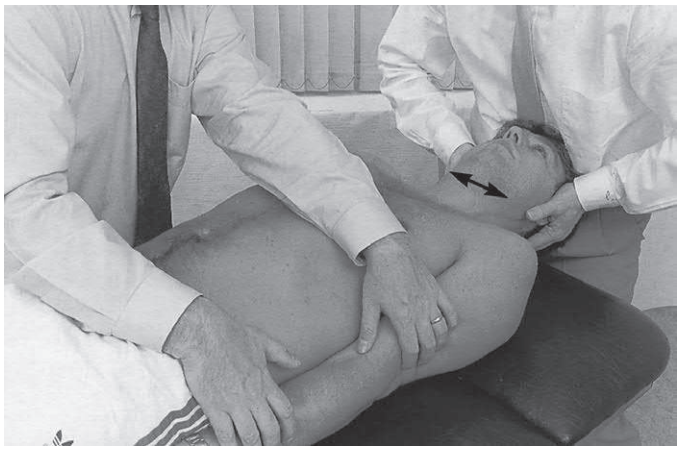


Fig 11.9 • Lateral glide.

Fig 11.10 • Traction with leverage.

via the hand under the occiput, which only steadies and is squeezed between head and couch to provide a fulcrum. The knees are now suddenly flexed, thus pulling the patient's neck with a jerk (Fig. 11.10). This results in a sudden increase of traction in the anterior spinal column from the foramen magnum downwards.

Practitioner's checklist

- Avoid flexion
- Careful positioning
- During manipulation: no movement, only traction

Manipulation procedure

Choice of technique

- *A preparative massage* to the muscles of the neck enhances relaxation and so is useful when a patient does not feel sufficiently relaxed (see p. 189).
- *Circumduction and straight pull* can be used as the first manœuvres, to accustom the patient to the traction element.
- *Rotation manipulation* is used for unilateral disc protrusions but is avoided in central displacements. Rotation is first performed in the most comfortable direction and then, if still necessary, in the least comfortable direction. It is executed to half-range first, then to three-quarters and only then to full range. When the other direction is chosen, a similar gentle start is again recommended.
- *Full rotation during traction* is only used when rotation caused improvement and only in the direction in which the result was obtained.
- *Side flexion* is mostly performed in the direction away from the painful side and very rarely towards the painful side – when, after a few sessions and after repeatedly trying the other techniques, a full result has not yet been obtained. When taking up the slack produces or increases arm pain, the manœuvre is abandoned.
- *Anteroposterior gliding* is indicated in unilateral disc displacements when the range of extension does not improve with rotation and/or lateral flexion techniques. In central protrusions it can be used in combination with straight pull and lateral flexion.
- *Lateral gliding* helps to get rid of possible post-manipulative soreness. When overcorrection is produced during a manipulation session – the unilateral pain changes sides – this manœuvre is performed.
- *Pure traction techniques* – straight pull and traction with leverage – are designed especially for the treatment of posterocentral disc protrusions. Articular movement is thus avoided.

Gentle start

Each time a new technique is chosen the manipulator starts gently. The intensity of a technique is only increased when the patient has responded satisfactorily to the previous technique.

Assessment of progress

After the use of each technique, the patient is asked to sit up and the result is assessed. A decision is then taken as to whether to continue in the same way or to change. Experience, the result of each particular manœuvre, the end-feel during exertion, the patient's age and estimation of tolerance all affect the types of manœuvre employed.

A change in symptoms or signs is interpreted as improvement when (a) intensity of pain has diminished; (b) range has increased; (c) fewer movements are painful; and (d) pain has centralized: for example, scapular pain instead of arm pain, trapezius pain instead of scapular pain, central pain instead of unilateral pain, or shortening of pain.

If one manœuvre has helped, it should be repeated until symptoms and signs no longer alter. Then another is tried.

If the manœuvre does not produce any positive results, it can be tried a second time with slightly more intensity. If improvement still does not occur, the manœuvre is abandoned and either another technique is chosen or the same technique is used in another direction. For example, if rotation to the right does not produce the expected result, either a lateral flexion technique or rotation to the left can be tried.

If a manœuvre makes the patient worse, it is not continued. The manipulator must then decide whether the worsening is the result of a wrong technique or of poor relaxation on the part of the patient, and whether the condition is in fact suitable for manipulation. If the technique is not appropriate, another can be chosen, perhaps with less movement and more traction. When two consecutive trials still increase the symptoms, manipulation is stopped. The patient is re-examined on the next occasion and a decision is taken as to whether to continue. Deciding is not always easy and great attention should therefore be paid to what the patient reports during and after each manœuvre, and in the hours that followed treatment. The moment there is the slightest doubt, manipulation is abandoned.

Manipulation strategy

Repetition of techniques

Acute conditions are best treated on a daily basis, while more chronic cases are manipulated 2–3 times a week. Manipulation is a short-term treatment, which means that, if no result is obtained after a few sessions, another approach is chosen.

Young patients can easily stand several (6–8) techniques in a session. Elderly patients receive the same treatment but only 1–3 techniques per session are used, and the interval between the treatments is longer – 1–3 times a week.

When the results of each manœuvre are only slight or minimal, manipulation is continued until the greatest possible result is obtained. When a significant improvement is suddenly noted, the session is stopped and the result reassessed on the next occasion.

The therapist should try to follow the sequence of manœuvres as recommended in the following section but should develop enough flexibility and creativity to work according to the results obtained in each individual case.

Course of the manipulative session

Posterocentral disc protrusion with unilateral cervicoscapular pain

Two types of case fall into this group.

Acute torticollis with side flexion deformity

In nuclear-type torticollis the following procedure is applied. The manœuvres are performed in a strict order, whereby manipulation in directions limited by the condition is avoided.

On the first day:

1. Traction manipulation (straight pull) in the direction of the deviation is performed, in which the head is gradually brought back to the neutral position. No other technique is used before the patient is able to maintain the head in the neutral position when seated upright.
2. Rotation manipulation under traction is then performed in the less restricted direction until this movement has become painless.
3. Lateral flexion manipulation under traction and towards the painless side should then restore a full painless range in that direction.
4. Progressive rotation, without traction, in the restricted direction, in which the head is gradually pushed to the point where pain begins and then immobilized in that position until the pain has disappeared; after this, further movement is undertaken and continued until a full range is achieved. The time taken for the pain to disappear at each stage may vary from a few minutes to 5 minutes or more, which means that up to 20 minutes may be necessary to restore the range of rotation.
5. Progressive lateral flexion, without traction, in the restricted direction is performed, in which the same slow procedure is followed until full side flexion is obtained. This movement usually improves more quickly than rotation; 5–10 minutes are required.
6. When the patient is reassessed after the whole procedure is finished, what has been achieved provides a basis for prognosis. When, after the first treatment, full painless range in all directions is attained, this indicates that by the next day the patient will be much improved or even cured.
7. The patient is then advised to repeat these progressive manœuvres every 2–3 hours and certainly before going to bed because there is a tendency to relapse. Instruction should also be given on what precautions to take to keep recurrence minimal; sleeping in the supine position is preferred. A very thin pillow should be used but support under the neck should be provided (e.g. a rolled towel with a diameter of 5–10 cm). Alternatively, if the patient sleeps on one side, a thick pillow should assure that the cervical spine lies in line with the thoracic spine. Sleeping prone should be avoided.

The patient is seen on a second day and, if considerably better, treatment then consists of manipulations for an ordinary unilateral disc protrusion (see below). When, on the contrary, a considerable relapse has taken place, the whole procedure is repeated. Full relief is usually obtained in 2–3 sessions (Fig. 11.11).

Annular-type torticollis in patients over 30 can be manipulated in the ordinary way (see below).

Unilateral cervicoscapular pain

These cases are almost always annular in nature and therefore respond very well to manipulative treatment. The following sequence is recommended:

1. Straight pull or circumduction under traction to familiarize the patient with strong traction techniques.

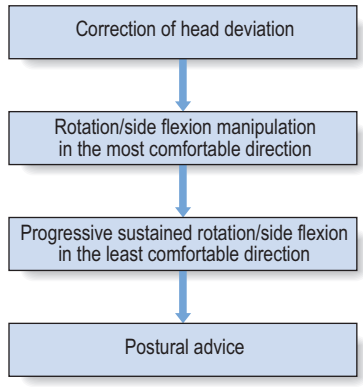


Fig 11.11 • Procedure in unilateral acute torticollis.

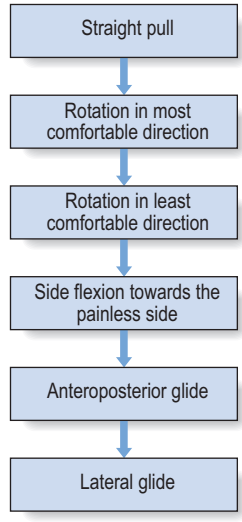


Fig 11.12 • Procedure in unilateral cervicoscapular or root pain.

2. Rotation under traction to less than full range in the less restricted or less painful direction.
3. Rotation under traction to full range in the same direction.
4. Rotation under traction to less than full range in the most restricted or most painful direction.
5. Rotation under traction to full range in the same direction.
6. Lateral flexion under traction towards the painless side.
7. Lateral glide (Fig. 11.12).

Posterocentral disc protrusion with central or bilateral cervicoscapular pain

This includes three types of case.

Acute torticollis with flexion deformity

The same procedure is used as for torticollis with deviation in lateral flexion, except that the traction in line with the deviation starts in flexion (enough time must be taken to bring the patient's head gradually back to the neutral position) and rotations are avoided. Side flexion manipulations under traction can be performed in both directions, after which the patient is put on his side and the limited extension very gradually forced to full range.

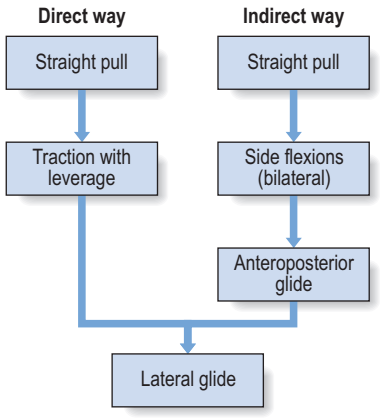


Fig 11.13 • Procedure in central or bilateral cervicoscapular pain.

Chronic neckache

A direct or an indirect approach can be taken.

Direct approach

1. Straight pull: strong traction manipulation in line with the spine.
2. Traction with leverage.
3. Lateral glide.

Indirect approach

1. Straight pull.
2. Lateral flexion under traction, which can be done bilaterally.
3. Anteroposterior glide.
4. Lateral glide.

The decision to use one approach or another depends on the severity of the clinical picture. In very mild cases it is easy to switch from one approach to the other.

The more pronounced the articular signs are and the more extension is limited, the more the manipulator will be inclined to choose the direct approach, following the principle of 'maximal traction with a minimum of articular movement'.

Signs of spinal cord involvement form an absolute bar to manipulation.

Bilateral root pain

The same procedure (Fig. 11.13) is used as in bilateral chronic neckache.

Posterolateral disc protrusion with unilateral root pain, without neurological deficit

The normal techniques are used:

1. Straight pull.
2. Rotation under traction in the less restricted or painful direction, first to less than full range and then to full range.
3. Rotation under traction in the most restricted or painful direction, first to less than full range and then to full range.
4. Lateral flexion under traction towards the painless side.
5. Lateral glide.

Before the impulses are given, the manipulator must make sure that taking up the slack does not influence the pain down the arm. If there is an effect, the technique is abandoned and another manœuvre is chosen. When two different techniques make the arm pain worse, the condition is not suitable for manipulation.

Traction

Adjuvant traction

Traction is a very important element of manipulation procedures because of its beneficial effects on the protruded fragment of disc (p. 190).

Traction as a treatment

For a long time, cervical traction has been regarded as an effective treatment for herniated cervical discs because it widens the intervertebral spaces[82] and exerts a centripetal force on the disc.[83,84] There is no doubt that traction induces pain relief and regression of herniated cervical discs.[85] A recent MRI study during 15 minutes of traction with a traction force of 18 kg also showed partial (62%) or complete regression (10%) of protruded cervical discs.[86] In spite of this, traction alone is not often used in orthopaedic medicine. The main reason is that clinical improvement is only achieved after 10 or more sessions – a result which could have been achieved much more quickly with ordinary manipulations.

However, traction is used as the primary treatment form in a small number of well-defined indications. It is most often given in the physiotherapist's room, but can – in some specific situations – be performed in bed. Usually the procedure is intermittent; on rare occasions traction is continuous.

Intermittent traction

Indications

There are four main indications for intermittent traction: in early nuclear protrusions, in minor posterocentral disc protrusions, to maintain reduction in unstable protrusions and as an alternative to manipulation.

Early nuclear disc protrusion

Differential diagnosis with an annular-type protrusion is not always easy and so it may happen that a patient is first manipulated but without success. On top of that, during manipulation an elastic rebound is felt at the end of range; when testing again after manipulation, full range cannot be obtained actively, though it was reached passively during manipulation. This type of displacement is a very good indication and responds quite well to daily traction in 3–6 sessions. However, long-standing cases do not benefit very much from this treatment and surgical removal may then be needed.

Minor posterocentral discodural interactions

Traction is a good alternative when manipulations are contraindicated (see p. 185). Even if the lesion is of the annular type,

Box 11.6

Intermittent traction: indications and contraindications

Indications

- Early nuclear disc protrusion
- Minor posterocentral disc protrusion
- To maintain reduction in unstable protrusion
- Instead of manipulation

Contraindications

- Paraesthesia induced by traction
- Brachial pain caused/increased by traction
- (*Annular disc protrusion*)

it may respond very well to traction, provided the patient is not too old and the articular signs are minor. Four to eight daily sessions usually produce good results.

To stabilize a recurrent disc

Two different situations may occur. The first is when the patient always relapses the day after manipulation. After 4–6 sessions the disc should have stabilized. If not, daily suspension at home is prescribed; 10 minutes' traction every morning may prevent the disc from protruding during the day.

Second, when the patient responds well to manipulation but continuously relapses a few weeks after the treatment, manipulation is followed by a week to 10 days' daily traction.

Instead of manipulation

Traction may be considered as an alternative for those patients who, for some reason, cannot be manipulated; 10–15 treatments may then be necessary.

Contraindications

These include the following:

Paraesthesia induced by traction

When traction provokes paraesthesia felt in the hands and/or feet, it must be stopped at once because this indicates involvement of the spinal cord.[87]

Brachial pain caused or increased by traction

When traction to the cervical spine elicits or increases root pain, the protrusion is clearly being pulled in the wrong direction – further against the nerve root.

Indications and contraindications are summarized in Box 11.6.

Technique

Traction can be applied with the patient sitting or lying supine (Figs 11.14 and 11.15). A leather collar is strapped under the patient's chin and occiput in such a way that the anterior portion clears the trachea while supporting the mandible. The collar is attached to a spreader and hangs by a cord passing over a pulley to the centre of the spreader. The therapist must make sure that the patient's ears are not squeezed. Traction is applied in the neutral position or with the head held in very slight flexion.

The physiotherapist stands by the patient and pulls gradually on the cord until the required force – usually between 8 and 15 kg – is obtained. The traction is continuous and the force maintained for 15–20 minutes.

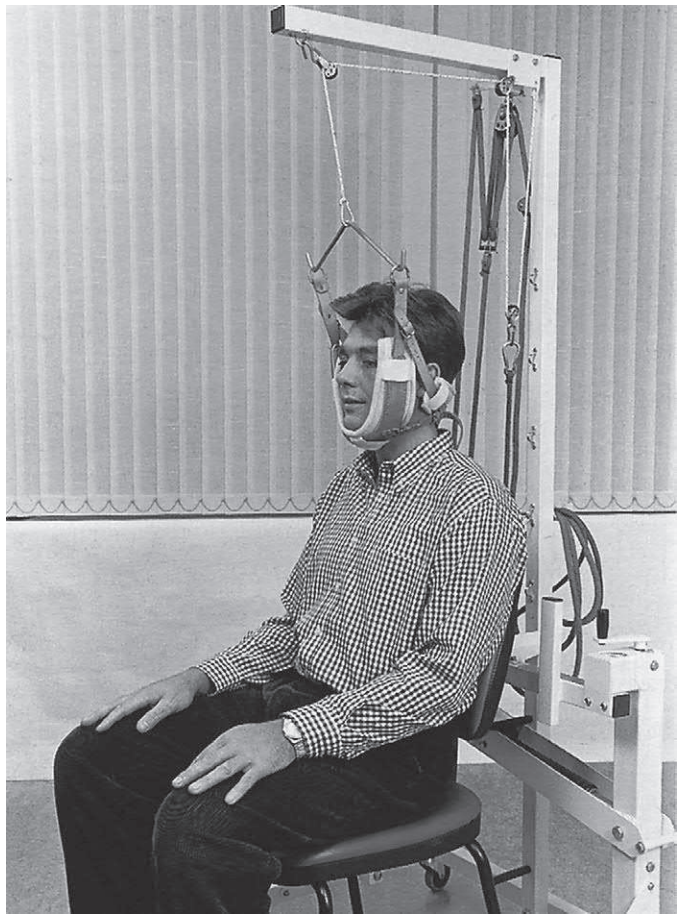


Fig 11.14 • Traction performed sitting (head suspension).

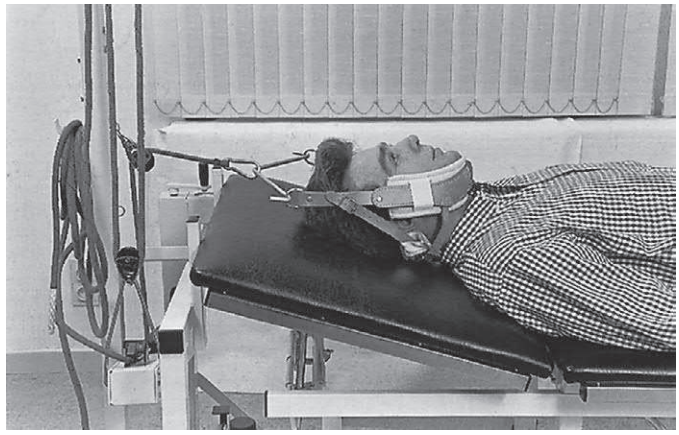


Fig 11.15 • Traction performed lying down.

Continuous traction in bed

This is used only when everything else has been tried and failed. Often it is the only alternative to surgery. The treatment has to be carried out in hospital because the patient requires full nursing care 24 hours a day. The traction (4–5 kg) is

maintained for the first 24 hours and then gradually diminished and released. The pain is expected to subside slowly and to have diminished after 7–12 days[88]; the patient may then be discharged from hospital.

Indications

There are three main indications for continuous traction.

Very severe root pain without neurological deficit

Those cases that do not respond to manipulation, head suspension or nerve block can be helped by traction in recumbency in the hope of quick diminution of the pain, which may otherwise take 3–4 months to abate.

Nuclear disc prolapse

Rarely, a young adult may be seen with a gross nuclear displacement: quite severe scapular pain is present with gross limitation of rotation in one direction. All other movements of the neck are normal. Manipulation does not work. During attempted manipulation the typical elastic recoil is felt – the head can be turned passively to full range but the active range remains very restricted. If head suspension fails, traction in bed is the only alternative.

Early posterocentral disc protrusion

Manipulation or intermittent traction is indicated and should produce good results. When these measures do not help, traction in bed is substituted.

Injection at a nerve root

Indications

The main indication for nerve root injection is root pain with neurological deficit. An injection is then the treatment of choice, especially when the root pain is too severe to await spontaneous cure.

Another indication is root pain without neurological deficit that lasts longer than 2 months, because no further improvement can be expected from manipulation. In recent root pain, nerve root infiltration is also called for if there is inadequate response to manipulation.

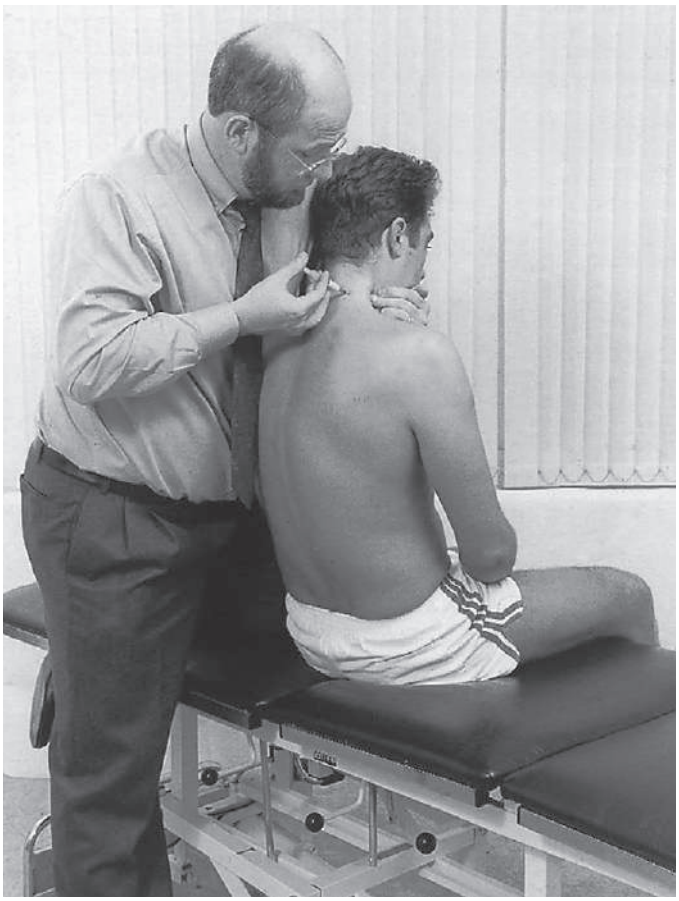
Technique

A 2 mL syringe filled with 20 mg of triamcinolone acetonide is fitted to a 4 cm needle.

The lateral edge of the corresponding transverse process is identified, just behind the posterior edge of the sternocleidomastoid muscle. The needle is inserted between the palpating finger and the midline, and aims at the identified transverse process (Fig. 11.16). It is inserted until it meets bone. Half a millilitre is injected there, and the needle is then moved laterally and slightly cranially until it slips over the edge and the rest of the injection is made here. During the injection the patient may feel radicular pain, which disappears after a few minutes. One to six injections at weekly intervals may be necessary, depending on the severity of symptoms.

Results

Radicular pain caused by a discoradicular interaction with or without neurological deficit usually benefits greatly from nerve root blocks. Most studies report a success rate of between 65 and 80% after one or two injections.[89–92] Bush *et al* performed a prospective study with independent clinical review on 68 patients presenting with cervical radiculopathy and neurological signs. The average duration of symptoms prior to presentation was 2 months (range 1–12 months). They received an



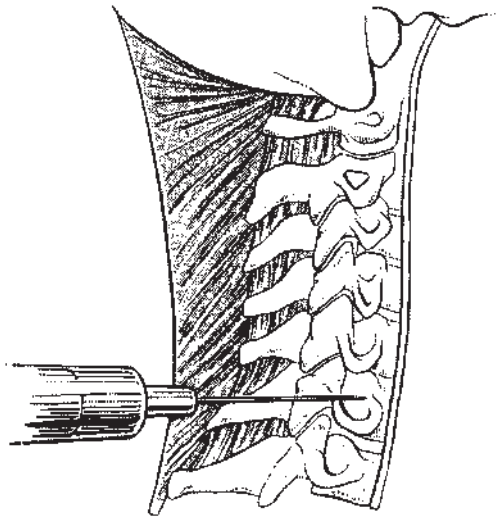


Fig 11.16 • Injection at a nerve root.

average of 2.5 periradicular corticosteroid injections per patient (range 1–6). Despite the fact that all 68 patients were potential surgical candidates, they all made a satisfactory recovery without the need for surgical intervention. Forty-eight patients (76%) did not experience any arm pain, and of the 15 patients (24%) who did, this improved from a score of 10 to an average of 2 (range 1–4) on a 10-point pain scale. The conclusion of the study was that patients with cervical radiculopathy make a satisfactory recovery with serial periradicular corticosteroid injections without the need for surgical intervention.[93]

Epidural injection

Epidural injections have been used to treat radicular pain from herniated discs, spinal stenosis, chemical discs, chronic pain secondary to post-cervical surgery syndrome, and chronic neck pain of discogenic origin. Epidural injections in the cervical spine are performed via either an interlaminar or a transforaminal approach. There are a number of studies supporting the benefit of cervical epidurals.[94,95] However, the evidence for cervical interlaminar epidural injections has been a subject of debate and at best has had only moderate success in managing cervical radiculopathy, while there is no evidence available for the management of axial neck pain, post-surgery syndrome or discogenic pain.[96–98] In our opinion, cervical epidurals are used far too often for conditions that are easily treated by simpler measures, such as manipulation and nerve root infiltrations. The injection is only called for in radicular pain that lasts longer than a year, has no tendency to spontaneous recovery and is not responding to manipulation (see p. 188).

The injection is always given in theatre conditions; access to the epidural space is obtained under sterile conditions with loss of resistance techniques under fluoroscopic visualization.[99] The epidural space is entered between C7 and T1, and 10 mL of either a steroid solution or a mixture of local anaesthetic and steroid is injected.

Collars

Partial or total immobilization of the neck should not be considered as a treatment for neck pain. It does not bring about reduction nor does it influence the normal spontaneous evolution of radicular pain. The role of a collar in non-traumatic cervical lesions is restricted to that of a prevention measure or a psychological support: it gives the patient a sense of security.[100]

Immobilizing collars are intended to prevent excessive movement that could exacerbate pain. Soft collars seem to have little effect on controlling neck movement[101,102] ; neither do hard collars (for example, the Philadelphia, NecLoc and Miami J collars) have the necessary effect, although they are more rigid.[103–105] Weight-relieving collars (for example, the Four Poster brace) diminish the weight of the head on the spine and can therefore be used in compression phenomena, such as cord compression, lateral recess stenosis or intractable neck pain.[106]

Prophylaxis

The best approach to a disc displacement is to try to reduce it, whereafter all symptoms disappear. However, because a disc is a cartilaginous structure and cannot heal, it may displace again at any time. Too many recurrences indicate that the disc is unstable and that further measures should be taken to stabilize it. First of all, the patient should avoid keeping the neck in flexion or fixed for a certain period of time. During the night a pillow should be used that enables the patient to hold the neck in physiological lordosis when lying supine, or in the neutral position when lying on the side.

When a collar is necessary, wearing it during the day (and if possible also at night) for several weeks or daily suspension at home may help to stabilize the reduced fragment.

Awaiting spontaneous recovery

Acute torticollis undergoes spontaneous cure in 1–2 weeks. The pain is constant and quite severe, and the head may be fixed in deviation, usually side flexion and occasionally flexion. After a few days the symptoms begin to diminish, but it may take 7–10 days for the pain to disappear completely.

Unilateral, central or bilateral neckache and cervicoscapular pain have no tendency to spontaneous recovery; nor has bilateral arm pain with or without paraesthesia in the hands.

Unilateral radicular pain recovers spontaneously in the course of several months, reckoned from the onset of radicular pain, not of scapular discomfort. When muscular paresis is not present, recovery may take 3–4 months. When a palsy is present, the pain disappears more speedily, usually in 2–3 months. Muscular power is restored within 3–6 months after symptoms have ceased. The only exception is a C8 root palsy, which may take up to 6 months to disappear and may occasionally leave a patient with a permanently weak thumb.

Time and patience should be employed to explain the mechanism of spontaneous recovery to the patient and to give a good prognosis (see p. 157). A continued interest should be taken, with regular review until symptoms have largely subsided so as to discourage the patient from seeking futile and expensive treatments elsewhere.

Surgery

Surgical intervention for neck pain and/or radicular pain is seldom required.[107] In the absence of neurological deficit, it has no better prognosis than the natural history of the process.[108] If the neurological deficit is caused by a posterolateral disc protrusion, natural history usually provides just as good long-term results as surgical interventions.[109,110] However, if the neurological deficit is caused by a bony outcrop in the lateral foramen, surgery will become inevitable. Surgery is also the treatment of choice in cervical canal stenosis, leading to myelopathy.

Technique

Currently, anterior cervical discectomy with fusion is the gold standard in the surgical treatment of cervical disc herniations.[111–113] Since the introduction of the technique in 1958 various anterior fusion methods have been developed.[114–116] The anterior approach has several advantages over posterior decompression, i.e. direct decompression of the spinal cord and roots, better alignment and a lower degree of invasiveness. Several modifications have been developed, but there is currently no consensus regarding the optimal technique.[117] Microsurgery, known as the posterior keyhole laminotomy-foraminotomy, is used mainly for single-level radiculopathy caused by osteophytic compression in the lateral recess.[118,119]

Treatment of non-discogenic lesions

The treatment techniques discussed here are: manipulation and capsular stretching, deep transverse friction and injection/infiltration.

Manipulation/capsular stretching

Indications

Migraine

Sometimes an incipient attack of migraine can be aborted with 30 seconds of strong traction on the neck. Although it is not clear what mechanism is responsible, Cyriax thought that stretching the carotid artery might come into play.

In middle-aged or elderly people, and especially in those who have had migraine from adolescence, manipulation can also be preventative. The techniques that are used are the same as for reduction of disc displacements: straight pull, rotation and side flexion under traction (see p. 189). When neck movements do not cause pain, one session – consisting of one manœuvre in all four directions – may suffice. Painful movements indicate that 2–3 treatments may be necessary before all discomfort ceases. This is a purely empirical finding, which Cyriax identified when patients treated for an ordinary disc lesion reported that, subsequent to manipulation, their attacks of migraine diminished or even disappeared.[120]

Capsuloligamentous disorders

Simple mobilizations without traction can be used to treat some soft tissue lesions in the neck.

Generalized arthrosis

Early arthrosis of the upper cervical joints which presents with upper neck pain, a moderate full articular pattern and a ligamentous end-feel responds well to a few sessions of *quick stretching* in rotation and side flexion to both sides.

In more advanced cases with gross symmetrical limitation and a hard end-feel, the treatment will consist of gradual *slow stretching* in all four directions. Up to four sessions may be necessary.

Facet arthrosis

Manipulation of the facet joint by gradual *capsular stretching* produces reasonable results. The range of movement will probably remain unchanged but the discomfort is largely abolished after 2–4 sessions, perhaps in combination with deep transverse friction (see p. 202) or with facet injection (see p. 203).

Ankylosing spondylitis

Painful stiffness of the neck can be temporarily offset by *slow stretching* in all directions. The manœuvre can be repeated as often as seems necessary – not during a flare-up of the condition – and such mobilizations are effective as long as bone-to-bone end-feel has not been reached.

Postconcussional syndrome

When diffuse capsular adhesions have formed about the occipitoatlantoaxial joints they can easily be ruptured by *quick stretch* manipulation in the direction that has been found to be painful at its extreme. Two to three treatments may be required.

Morning headache in the elderly

Symptoms can be lastingly eliminated by 1–2 sessions of *slow stretching*. There is no upper age limit to this approach. In very old patients only one movement is forced at a time, which means that the patient may have to attend several times before full recovery is obtained.

Indications and types of manipulation for non-disc lesions are listed Boxes 11.7 and 11.8.

Mobilization techniques without traction

Forced rotation

The patient lies supine on the couch.

The manipulator stands at the patient's head. If the neck is to be rotated to the right or left, the right or left thenar

Box 11.7

Cervical manipulation/capsular stretching for non-disc lesions:

Indications

- Migraine
- Postural vertigo
- Tinnitus
- Capsuloligamentous disorders
  - Generalized arthrosis
  - Facet arthrosis
  - Ankylosing spondylitis
  - Postconcussional syndrome
  - Morning headache in the elderly

Fig 11.17 • Forced rotation.

Fig 11.18 • Forced side flexion.

Box 11.8

Summary of types of manipulation for non-disc lesions

Under traction

- Migraine

Without traction

Quick stretch

- Early arthrosis
- Diffuse capsular adhesions after trauma

Slow stretch

- Advanced arthrosis
- Ankylosing spondylitis
- Arthrosis of a facet joint
- Morning headache

eminence is placed against the patient's left or right maxilla at the temporomandibular joint and the fingers are hooked around the chin. The other hand grasps the occiput and the head is held in slight extension. The head is turned to the end of range and movement is then forced by a simultaneous action of both hands in opposite directions.

Quick stretch results from a manipulative thrust over a very small amplitude at high velocity. Ligamentous adhesions may be felt (and heard) to break. This manœuvre is carried out once and a painless range ensues.

Slow stretch is obtained by maintaining a steady push at the extreme of range for as long as the patient can tolerate, usually between 30 and 40 seconds. Pressure is then very slowly released and the head gradually brought back to the neutral position (Fig. 11.17). The movement is then repeated once or twice, depending on the patient's age.

Forced side flexion

The patient lies supine on the couch and the manipulator stands at the patient's head. When side flexion is to be to the right, the head is supported under the occiput with the fingers of the left hand and the thenar eminence lies against the side of the head above the ear. The metacarpophalangeal joint of the index finger of the right hand is placed against the transverse process of the sixth cervical vertebra and the thumb is kept extended to avoid the patient's trachea. This hand serves as a fulcrum and helps to keep the movement localized to the cervical spine only.

The quick stretch manœuvre is a short manipulative thrust, the result of a simultaneous scissor movement of both hands in opposite directions.

During slow stretch, sustained procedure keeps the head side-flexed at the extreme of range (Fig. 11.18). Because the force has to be maintained for some time, this technique is quite tiring for the operator and therefore extra pressure can be applied with the knee against the elbow. The foot is therefore placed on a stool.

Deep transverse friction

Deep transverse friction can be given to the occipital muscular insertions and to the capsules of the facet joints.

Post-traumatic muscular lesions

Post-traumatic lesions at the occipital insertion of the semispinalis capitis and, rarely, the splenius capitis muscles give rise to occipital pain on resisted extension and resisted side flexion towards the affected side. These features can be treated with deep transverse massage performed 2–3 times a week. Chronic cases may need up to 20 sessions for recovery.

Technique

The patient lies prone on a couch with an opening near the head through which he can breathe. The physiotherapist sits on the painless side, level with the patient's neck. The head is steadied with one hand and the other imparts the massage. This is done with the index finger, reinforced by the middle

Fig 11.19 • Deep transverse friction to the occipital insertions of semispinalis or splenius capitis muscles.

finger, and counterpressure is taken with the thumb at the painless side. Tenderness is sought in the attachment of the semispinalis capitis between the superior and inferior nuchal lines. If the lesion lies in the splenius capitis, tenderness is found more laterally at the mastoid process.

The working forearm is held in slight supination, which enables the therapist to exert upwards pressure against the occipital bone, at the exact point of the lesion. The thumb is used as a fulcrum and massage imparted by a to-and-fro movement over the tender area (Fig. 11.19).

Facet arthrosis

Early arthrosis, usually localized at the C2–C3 or C3–C4 interapophyseal joint, can be treated with deep transverse friction. If the result is poor, either capsular stretching or infiltration of a steroid suspension is substituted.

Technique

The patient sits at the couch with the elbows resting on it and the chin supported by cupped hands. The physiotherapist, standing by the patient's painful side, palpates the area where the lesion is expected to lie. If the most tender spot corresponds to the facet joint considered to be responsible for the pain, massage is applied there. The articulation is found quite laterally, just behind the posterior border of the sternocleidomastoid muscle. Massage is performed with the flexed thumb and counterpressure with the fingers. The direction is oblique, so that, parallel to the joint line, the thumb passes downwards as it is drawn backwards by internally rotating and abducting the shoulder and flexing the wrist[121] (Fig. 11.20).

After 5 minutes' treatment, movements are tested again. When the correct spot has been located, there should be some improvement, in which case massage is continued for another 5–10 minutes. Lack of improvement indicates that the chosen level is not right and reassessment is carried out. The patient is treated 2–3 times a week, and 3–6 weeks of treatment may be required.

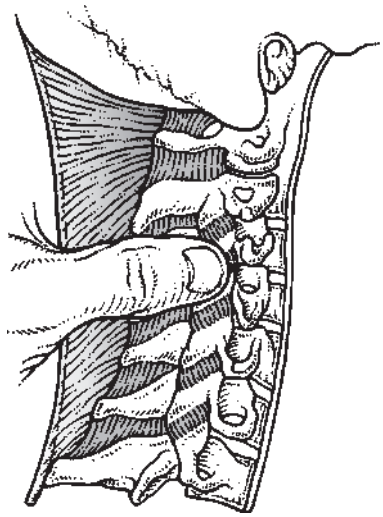


Fig 11.20 • Deep transverse massage (Troisier and Elroy technique).

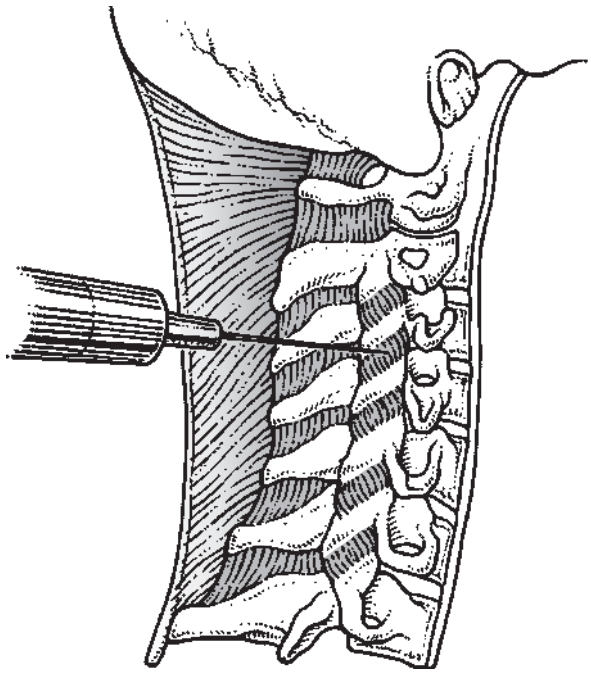


Fig 11.21 • Infiltration of a facet joint capsule.

Injection/infiltration

Early and more advanced arthrosis of an interapophyseal joint may respond to a steroid injection in the facet joint capsule. Unilateral localized pain at the mid-neck, which does not respond to manipulation, must be considered to originate from a facet joint and is most frequent at the C2–C3 and C3–C4 levels.

Intra-articular steroid is called for when ankylosing spondylitis or rheumatoid arthritis has affected the cervical spine and one or two particular facet joints only are painful.

Technique

The patient sits at the couch with the elbows resting on it and the chin supported by cupped hands. The operator stands behind the patient and palpates for the tender facet joint, which lies 2–2.5 cm from the midline. A 1 mL tuberculin syringe is filled with 10 mg of triamcinolone acetonide suspension and fitted with a thin needle 3 cm long. The needle is inserted until it strikes the lamina and is then moved until it traverses a tough ligamentous structure, the capsule, before it abuts against cartilage. This feeling assures the operator that he has reached the joint. A few drops are then infiltrated at several adjacent points, some intracapsular, some intra-articular. If the needle is thrust in too close to the midline it is possible to traverse the interlaminar space and enter the spinal canal. One to two infiltrations will render the patient pain-free for a considerable period (Fig. 11.21).

When a nerve root becomes compressed by an osteophytic outcrop and symptoms occur which are severe enough to concern the patient, infiltration with triamcinolone acetonide around the nerve root is tried. The technique for this is described on p. 199.

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12 Clinical examination of the shoulder

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Pain in the shoulder is, after low back pain, the most frequent complaint of orthopaedic patients. Despite the frequency of shoulder lesions – and the consequent pain and disability – much confusion still exists as to aetiology, terminology and treatment,[1] in contrast to the statement made by Cyriax:[2,3]

> The shoulder is the most rewarding joint in the whole body. As a rule, one can always come to a clear diagnosis, and, if treated in the proper way, most shoulder lesions seem to be curable. Moreover, it is the most suitable joint for the general practitioner, since almost no technical aids are required. A good history and full clinical examination, together with a detailed knowledge of the anatomy, suffices to solve the majority of the shoulder problems.

Complicated cases that are difficult to diagnose exactly are encountered. If clinical findings are difficult to interpret, the following general points should be of help.

First, it should be realized that double lesions do exist, clouding the diagnosis. For example, it is not uncommon to find supraspinatus tendinosis together with infraspinatus tendinosis or in association with subdeltoid bursitis. In these doubtful cases, a diagnostic infiltration of a local anaesthetic can be most helpful in isolating the second lesion.

On other occasions, patients present with a painful limitation of passive movement together with pain on resisted movements, and the question arises as to whether the problem is in an inert or a contractile structure. If there is capsular limitation on examination, the joint should be treated first. If resisted movements remain painful after the joint has been managed appropriately, then the tendons should be treated. This approach is the best one, because resisted movements very often become negative after arthritis has disappeared. The only explanation for the phenomenon is the close relationship between the capsule of the shoulder and the surrounding tendons.[4] It can easily be understood how tension on the contractile structures may influence the pain originating in arthritis. Therefore pain on resisted movement(s) in association with an articular pattern should not be interpreted as being caused by a simple tendinosis. Of course, a severe tendinosis can limit active movement, because of the pain. But passive movements are of full range with a normal end-feel, even though there might be severe pain at the end of movement.

An arthritis is an arthritis, a tendinosis is a tendinosis and both have to be treated as such. It is a common misbelief that, as long as steroid is injected somewhere in the shoulder area, it will spread and cure the lesion, no matter where the lesion lies or where it was injected.[5] In fact, if there is one region in the body which ought to be diagnosed and treated very specifically it is the shoulder. It is necessary to replace a vague diagnosis such as 'rotator cuff disease' or frozen shoulder by a precise one indicating exactly what is wrong.[6–8]

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Referred pain

Pain referred to the shoulder

Pain referred to the shoulder, and possibly further down the arm, can be caused by nerve root compression, mainly as the consequence of a posterolateral cervical disc protrusion. A good history is important; the pain has usually started in the neck, probably interscapularly, and has shifted laterally into the arm and hand. Very often the pain is worst at night and is accompanied by pins and needles and numbness. Most frequently the C7 nerve root is compressed, causing pain at the posterior aspect of the whole arm to the second, third and fourth fingers. Other nerve roots can also be involved in a cervical disc lesion but this is far less frequent. Referral to the appropriate dermatome is usual.

In a posterocentral cervical disc lesion with compression of the dura mater, the pain does not spread beyond the deltoid area. Therefore arm pain is not present.

One rare disorder causing pain down the arm is a cervical neuroma. The condition starts with pain in the arm, which progressively spreads proximally as the tumour increases in size.

Other possible causes of referred pain at the shoulder are visceral disorders. The diaphragm is largely developed from the third and fourth cervical segment, the heart from the eighth cervical to the fourth thoracic. Therefore both can give rise to pain in the shoulder and arm. Irritation of the diaphragm and of the phrenic nerve, for example by blood or air under the diaphragm, is another well-known source of acute shoulder pain.[9,10]

A pulmonary neoplasm at the base of the lung with involvement of the diaphragm can provoke pain in the shoulder area. The same may also happen with a tumour of the superior sulcus (Pancoast's tumour). The majority of these patients complain of shoulder pain and are often mistakenly thought to be suffering from a musculoskeletal lesion.[11,12]

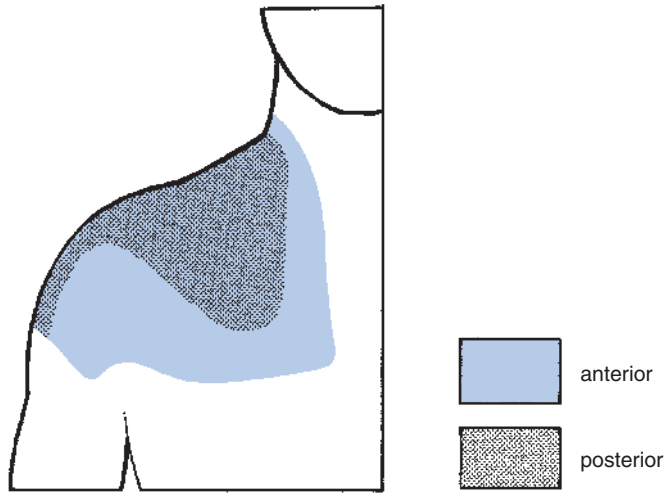


Fig 12.1 • The C4 dermatome.

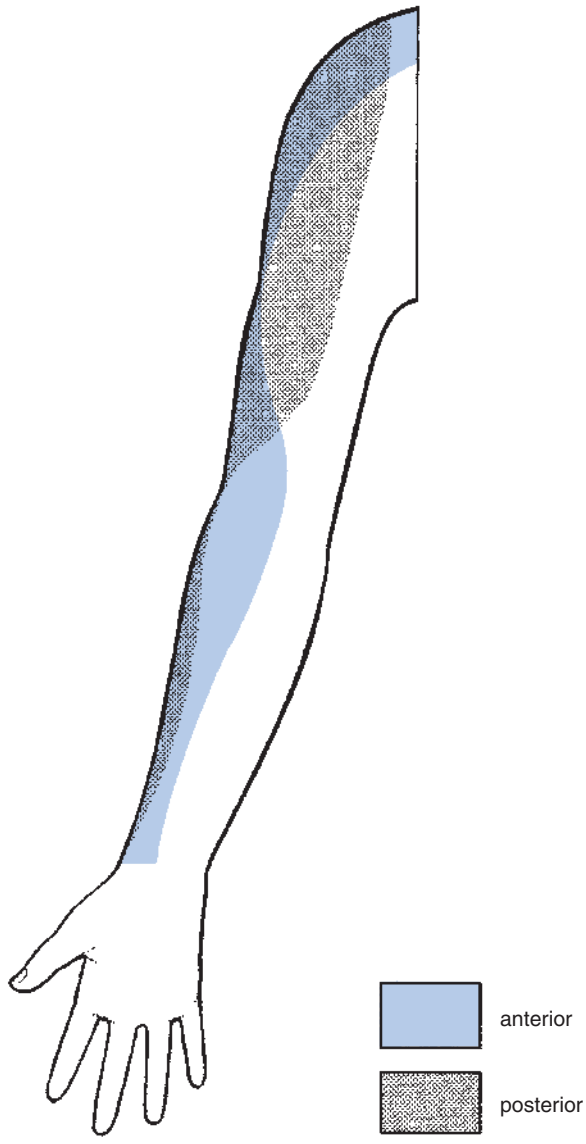


Fig 12.2 • The C5 dermatome.

Pain referred from the shoulder

Most structures around the shoulder are derived from the C5 segment. There is one important exception: the acromioclavicular joint, which is of C4 origin (Fig. 12.1). In acromioclavicular joint problems the pain is felt at the tip of the shoulder, with little spread. Exceptionally, when the lesion lies at the inferior acromioclavicular ligament, the pain can spread into the upper arm.

In a lesion of one of the other shoulder structures, such as in all types of tendinosis, arthritis and subdeltoid bursitis, the pain is not so much felt at the tip of the shoulder but rather starts in the deltoid region and may spread further down the radial aspect of the arm to the base of the thumb (C5 dermatome; Fig. 12.2). How far down the arm the pain is referred depends on the severity of the lesion: the more severe the inflammation, the further the pain will spread. In glenohumeral arthritis, the degree of pain reference is of particular interest

in following the healing process: as the patient improves, the inflammation decreases and the pain spreads less far.

History

The first question to be elucidated by the history is whether the pain in the arm is genuinely from a shoulder lesion or whether it is the consequence of a more proximal lesion, arising perhaps from the cervical spine. If the answer to this is not clear from the history, a preliminary examination, including tests of the cervical spine, shoulder and elbow, is necessary.

In arthritis of the shoulder, the history will be important to establish the stage (see Ch. 13). In other disorders it is of less significance.

The answers to a number of questions (summarized in Box 12.1) will be needed.

- *What is your age?* Age can be relevant in several disorders. It can be helpful in defining the exact type of arthritis. Traumatic arthritis will only be met after 40 years of age, arthritis from immobilization after the age of 60. Subdeltoid bursitis might be present between 15 and 65 years of age. Tendinosis can occur at any adult age.
- *What is the pain and does it radiate?* Pain starting in the deltoid area and spreading towards the wrist, along the radial aspect of the arm, is caused by a lesion that originates in C5. Such pain may be felt in the whole dermatome or only in part of it. The majority of the shoulder structures belong to the C5 segment. The acromioclavicular joint, a C4 structure, is the main exception. A patient who indicates the tip of the shoulder only as the site of pain suggests strongly that there is a lesion of the acromioclavicular joint. Whether the pain is caused by arthritis, bursitis or simple tendinosis will make no difference to where or how far distally the pain is felt. The distal spread of referred pain depends only on the degree of inflammation. It is routine to ask if the pain remains above the elbow or radiates below it – a matter of particular importance in arthritis.
- *Is there any pain in the arm at rest?* This gives information about the severity of the lesion: if spontaneous pain is present, there is a greater degree of inflammation than if pain is felt only on movement. Again, the answer to this question is one of the criteria for judging the stage of arthritis.
- *Can you lie on the affected side at night?* Pain when lying on the shoulder indicates more severe inflammation than just pain on exercise. Bursitis, tendinosis or arthritis may make it impossible for the patient to lie on the affected side at night. Consequently, this question is not of much help in defining the exact nature of the structure at fault. However, it is rather important in following the resolution of the disorder: as the condition improves, the pain at night diminishes and finally disappears.
- *Did the pain come on spontaneously or was there any particular reason for it, such as overactivity or an injury?* It is clear that overactivity may provoke tendinosis. In a ruptured tendon, however, one should not necessarily expect recent overuse. Overactivity can also cause arthritis in a joint that is already osteoarthrotic; this is just as true for the acromioclavicular joint as it is for the glenohumeral joint. In haemophilia, haemarthrosis usually comes on spontaneously; it is more common at the knee but may occur at the shoulder as well.
- *For how long have you had the pain?* If the pain has already been present for some months, an acute subdeltoid bursitis can be excluded because the full course of this condition is 6 weeks. In addition, onset is abrupt over a few days, sometimes only hours, as in an attack of gouty arthritis. Arm pain because of root compression by a cervical disc protrusion wears off in about 4 months. Long-standing pain can be the outcome of a chronic subdeltoid bursitis or a simple tendinosis. Both can last for years. Monoarticular steroid-sensitive arthritis can take up to 2 years to disappear spontaneously.
- *Are any other joints affected?* A more generalized inflammatory disorder is expected if other joints have been previously involved or are attacked at the same time. Indeed, the shoulder joint can be the seat of rheumatoid arthritis, lupus erythematosus and ankylosing spondylitis.
- *How is your general condition? Have you had any operations?* Recent unexplained loss of weight can be the first sign of a carcinoma. A primary tumour at the shoulder or metastases can be a local source of shoulder pain. A Pancoast's tumour of the lung often provokes pain in the shoulder area.

Box 12.1

Summary of history taking

- Age?
- Where is the pain? Does it radiate?
- Pain at rest or only on use?
- Can you lie on that side at night?
- How did the pain come on: spontaneously/overuse/injury?
- For how long have you had the pain?
- Are other joints affected?
- General condition? Any operations?

Inspection

The inspection starts with checking what position the head is held in and whether both shoulders are level. It is important to check for redness, swelling, muscular wasting or any deformity such as scapular winging. A step deformity at the upper lateral aspect of the shoulder is caused by an acromioclavicular dislocation, with the distal end of the clavicle lying superior to the acromion. Atrophy of the upper trapezius may indicate spinal accessory nerve palsy.[13] Atrophy of supraspinatus and/or infraspinatus is caused by either a supraspinous nerve palsy or long-standing rotator cuff lesions.[14] Effusion of more than 10–15 mL arising from the glenohumeral joint is normally

visible on inspection at the anterior centre of the humeral head. Local swelling may also be found in acute, haemorrhagic or chronic subdeltoid bursitis and in acromioclavicular joint cysts,[15] as well as in tumours.

Functional examination

The shoulder is an easy joint to examine. The intention is to obtain the maximum information from a minimum number of tests. Recent studies have shown high inter-rater and intra-rater reliability of the examination scheme presented.[16–18]

Preliminary examination

In most cases of shoulder–arm pain the history will reveal whether the pain originates from the shoulder itself or is of cervical origin. Sometimes, however, the examiner is not quite sure and will then use a quick survey of all structures between C1 and T2 to exclude other sources of pain in the upper quadrant. It is good practice to perform a preliminary examination of the upper quadrant in the following situations:

- There is or was neck pain
- There is or was trapezius pain
- The pain is only at the top of the shoulder and/or at the clavipectoral area
- The pain is in the arm but remains quite localized
- The pain in the arm is influenced by movements of the neck
- Coughing, sneezing or taking a deep breath increases the pain
- There is paraesthesia.

The preliminary examination of the upper quadrant is comprised of the following tests (Box 12.2):

1. Six active movements of the cervical spine – range of movement and/or painfulness; quick survey of the cervical spine
2. Active elevation of the shoulder girdle – range of movement and/or painfulness; quick survey of all the structures of the shoulder girdle
3. Resisted rotations of the cervical spine and resisted elevation of the scapulae; quick survey of nerve roots C1–C2–C3–C4
4. Active elevation of both arms – range and pain; quick survey of shoulder and shoulder girdle
5. Resisted movements of the upper limb – strength and pain; this is both a quick test for peripheral lesions at the elbow–arm–wrist and a neurological examination of roots C5–C6–C7–C8–T1 and of the peripheral nerves of the upper limb
6. Passive examination of the elbow; quick test of the elbow joint.

Any influence on the pain or any weakness will guide the examiner approximately towards the affected area, which is then examined thoroughly.

Box 12.2

Preliminary examination

Neck

Active movements

1. Flexion
2. Extension
3. Side flexion to the left
4. Side flexion to the right
5. Rotation to the left
6. Rotation to the right

Resisted movements

7. Rotation to the left (C1)
8. Rotation to the right (C1)

Scapula

9. Active elevation of both scapulae
10. Resisted elevation of both (C2–C4) scapulae

Shoulder

11. Active elevation of both arms
12. Resisted abduction (C5)

Elbow

Passive movements

13. Flexion
14. Extension

Resisted movements

15. Flexion (C5, C6)
16. Extension (C7)

Wrist

17. Resisted flexion (C7)
18. Resisted extension (C6)

Thumb

19. Resisted extension (C8)

Finger

20. Resisted adduction of the little finger (T1)

If the examination reveals that the lesion lies in the shoulder, the examiner will try to define in which particular structure the lesion is situated by carrying out a detailed shoulder examination; this is comprised of 12 basic tests (summarized in Box 12.3).

Basic functional examination of the shoulder

Clinical examination should not begin by palpation for local tenderness. This widespread habit is a common cause of misdiagnosis.

The basic shoulder examination consists of 12 tests. It is important always to perform every basic test and not to stop, even if the diagnosis appears clear after a limited number of tests. Stopping too soon can easily lead to an incomplete diagnosis. It is important to realize that, in mixed patterns of pain

on both passive and resisted movement(s), pain on resisted movements does not exclude a disorder in an inert structure, nor pain on passive movements a disorder in a contractile structure. These results may sometimes lead to diagnostic difficulties (see Ch. 4).

Accessory tests may be called for. They will only be performed if, after the basic functional examination, the diagnosis still remains unclear. After the basic examination is complete, at least a differential diagnosis should be in mind. To arrive at the final diagnosis, one or more accessory tests may be useful.

Box 12.3

Summary of the basic functional examination of the shoulder

Elevation

1. Active elevation of both arms
2. Passive elevation
3. Painful arc

Glenohumeral joint

4. Passive scapulohumeral abduction
5. Passive lateral rotation
6. Passive medial rotation

Resisted movements

7. Adduction
8. Abduction
9. Lateral rotation
10. Medial rotation
11. Flexion of the elbow
12. Extension of the elbow

Elevation of the arm

Active elevation

The patient is asked to raise both arms sideways above the head, as far as possible (Fig. 12.3a). The range of movement and the influences, if any, on pain are noted.

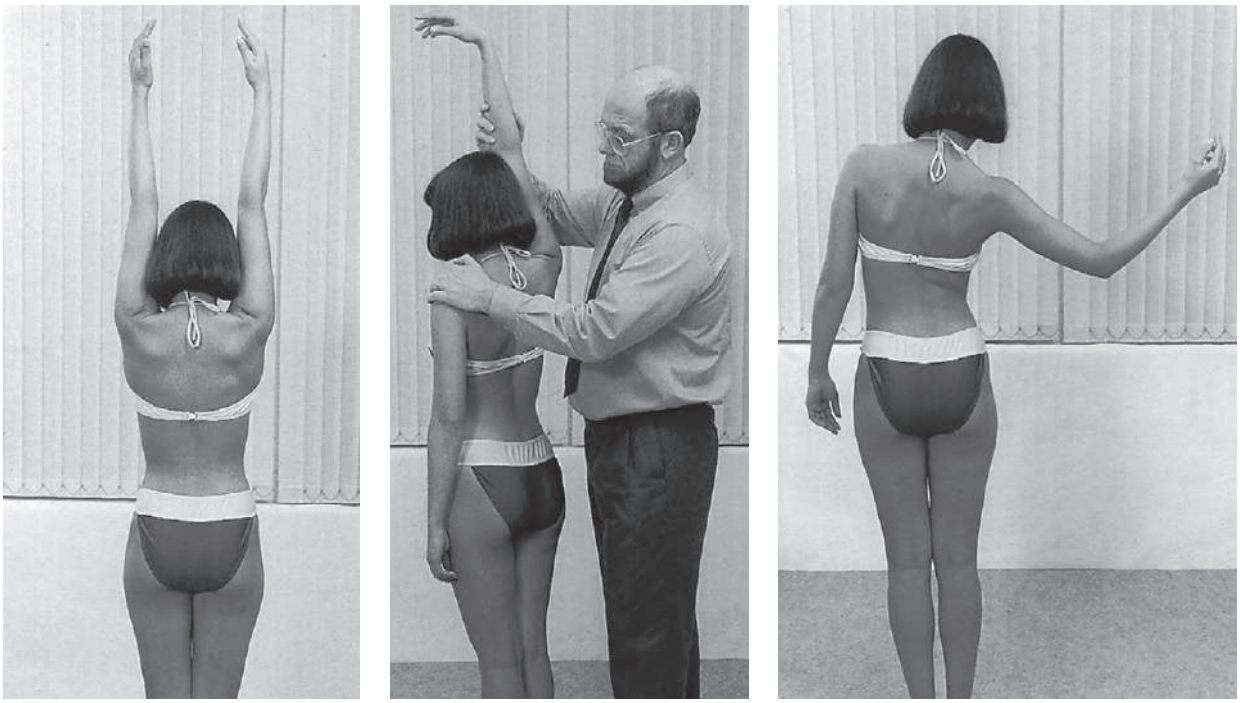
This very unselective and broad test investigates both inert and contractile structures, not only of one single joint, but also of all five 'joints' of the shoulder girdle (see online chapter *Clinical examination of the shoulder girdle*). Therefore, the result should always be interpreted in light of the other tests. As it gives a good idea of the patient's willingness to cooperate, it will also help in identifying a person who has no genuine lesion but is feigning illness.

Passive elevation

The examiner takes the patient's arm just proximal to the elbow, brings it upwards from the side and pushes it as far as it will go towards the head. At the same time counterpressure is applied over the contralateral shoulder, preventing the patient from side-flexing to the other side (Fig. 12.3b). Pain, range of motion and end-feel are noted. Because this movement comes to a halt when the axillary portion of the capsule is stretched, the normal end-feel is elastic.

Painful arc

The patient raises the arm, actively, in a frontal plane and concentrates on pain likely to occur at mid-range, being asked



(a) (b) (c)

Fig 12.3 • Elevation of the arm: (a) active elevation; (b) passive elevation; (c) painful arc.

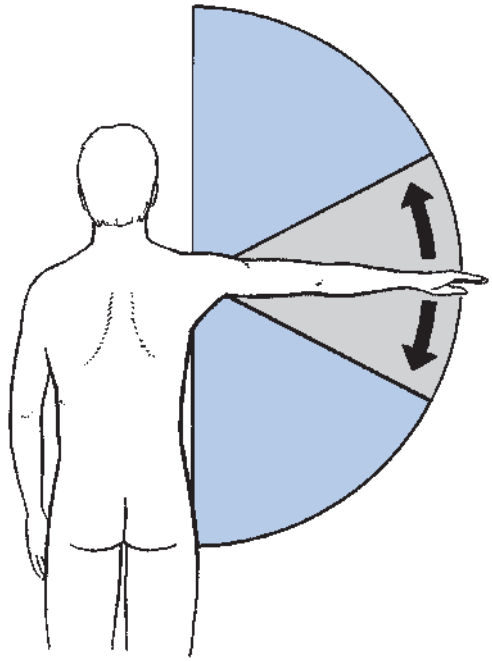


Fig 12.4 • Painful arc.

to indicate where pain starts and where it stops on further elevation (Fig. 12.3c).

A painful arc is defined as the symptom appearing somewhere around the halfway mark, with the arm near the horizontal and disappearing before the end of the movement (Fig. 12.4). This description holds even if pain is again present at the end-point. Some patients have an ascending arc, others a descending one; both are regarded as a real painful arc and no diagnostic distinction is made between them. Sometimes an arc becomes visible to the examiner, i.e. when the patient avoids the painful movement by bringing the arm towards the front of the body during elevation.

A painful arc is more likely to occur during active elevation rather than during passive movement because contraction of the abductor muscles pulls humerus and acromion closer to each other. It always implies a lesion in the subacromial area or its neighbourhood. Because of their localization between acromion and greater or lesser humeral tuberosity, the affected structures can be painfully pinched.

Three tests for the glenohumeral joint

Passive scapulohumeral abduction

The lower angle of the scapula is immobilized by the thumb and index. With the other hand, the examiner takes the patient's arm just above the elbow and lifts it up until the scapula starts to move (Fig. 12.5a). It is important for the patient not to assist this movement actively because then the scapula immediately starts to rotate, so making the movement a compound one involving several joints.

The normal range of scapulohumeral abduction is about 90°. Performed in the way described, only movement between humerus and scapula takes place. If the movement is impaired, the glenohumeral or the subacromial joint is at fault.

Passive lateral rotation

The examiner takes the patient's arm above the wrist, flexes the elbow to a right angle and pulls the arm with gentle pressure into full lateral rotation, meanwhile avoiding extension by holding the patient's elbow against the side of the abdomen. The trunk is immobilized by bringing the other hand around the patient's contralateral shoulder (Fig. 12.5b).

This movement comes to a stop by stretching the anterior portion of the capsule. Therefore the end-feel is elastic. The normal range is about 90°. Since individual differences exist, both sides should always be compared.

Besides the anterior portion of the joint capsule, other structures, such as the subcoracoid bursa, the acromioclavicular joint and the subscapularis tendon, are tested as well. Limitation of the movement is mainly found if something is wrong with the scapulohumeral joint itself; in this event, a harder end-feel is usually present.[19,20] A simple tendinosis of the subscapularis does not cause limitation of the movement but may render it very painful.

Passive medial rotation

With one hand still just above the patient's wrist and flexing the patient's elbow to 90°, the arm is brought into full medial rotation, without extension. The examiner's other hand is placed dorsally between the scapulae (Fig. 12.5c). The normal amplitude is about 90°. As before, this movement should be compared on both sides.

Occasionally a painful arc can be present on medial rotation. This has the same diagnostic value as a painful arc on elevation and bears the practical consequence that in order to test for real limitation of movement the examiner must persist to get beyond the painful arc.

Resisted movements

Resisted adduction

The patient is asked to pull the right arm towards the body as hard as possible. The examiner puts one hand around the elbow, and the other at the patient's ipsilateral side (Fig. 12.6a).

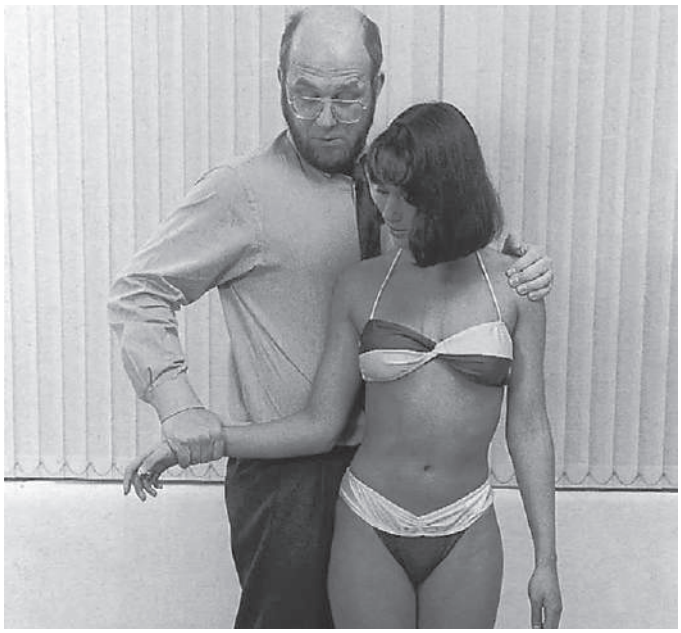
Resisted abduction

The test is performed with the arm hanging down, a few degrees of abduction being permitted. The examiner asks the patient to push his or her arm to the side, meanwhile applying counterpressure at the elbow. The examiner's other hand stabilizes the patient on the contralateral side (Fig. 12.6b).

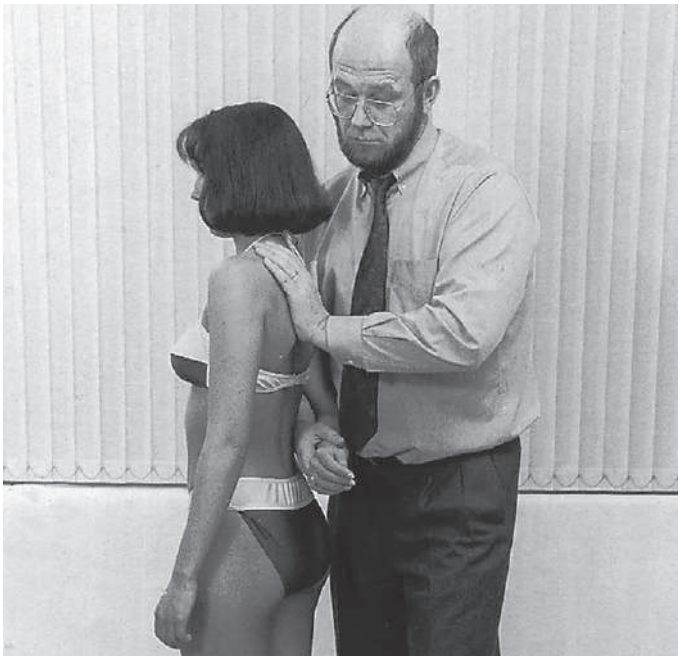
Resisted lateral rotation

The patient is asked to bend the elbow to a right angle and to push the forearm away from the body. To avoid any movement of the trunk, the other hand is put on the patient's contralateral shoulder (Fig. 12.7a). Counterpressure is applied just above the wrist and care must be taken to get two details right. First, the patient should keep the elbow against the body, so that there is no element of abduction. Second, extension of the elbow during lateral rotation is avoided. This is easily checked

(a)



(b)



(c)

Fig 12.5 • Three tests for the glenohumeral joint: (a) passive scapulohumeral abduction; (b) passive lateral rotation; (c) passive medial rotation.

if the examiner puts the little finger underneath the patient's wrist: extension at the elbow causes the digit to move down.

Resisted medial rotation

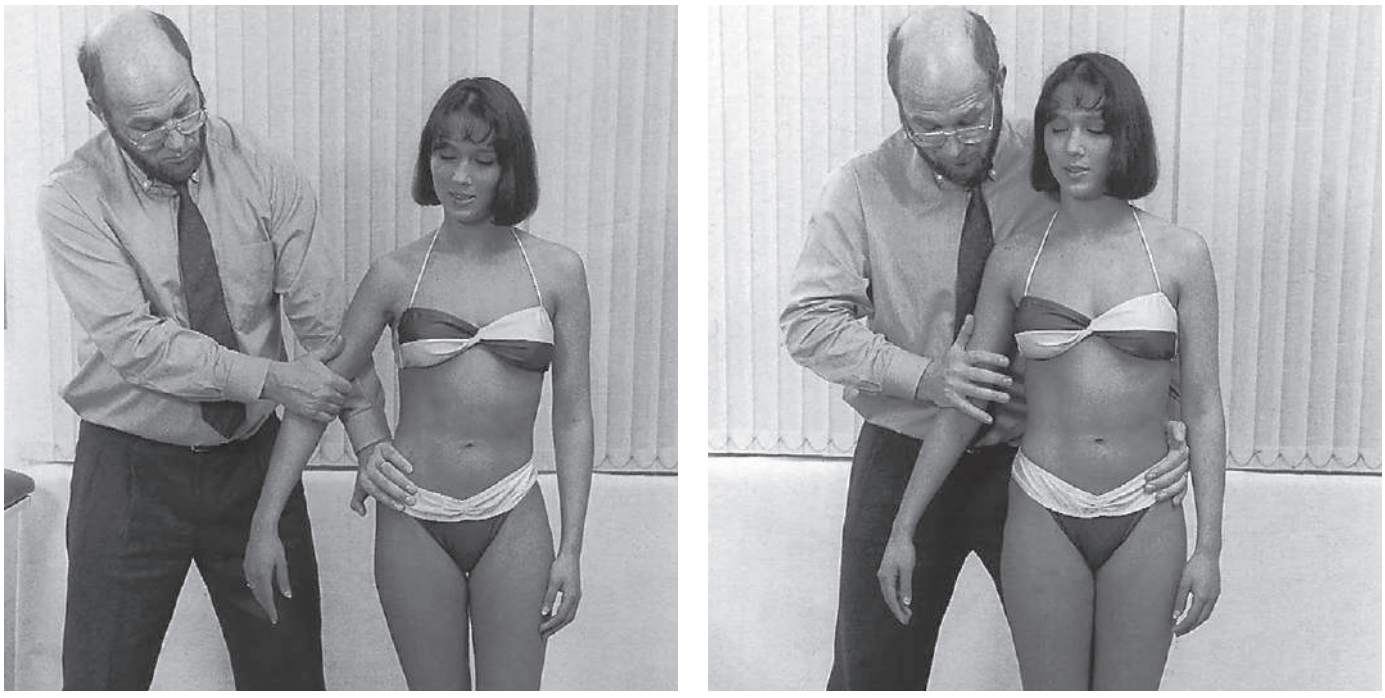
This is tested in the same position as resisted lateral rotation, but the patient's arm is held at the inner part of the wrist and the forearm is pulled towards the body (Fig. 12.7b).

Resisted elbow flexion

With the elbow still bent at a right angle and the forearm supinated, the forearm is pulled up. Counterpressure is applied to the distal part of the forearm just above the wrist. The other hand is placed on the patient's ipsilateral shoulder (Fig. 12.8a).

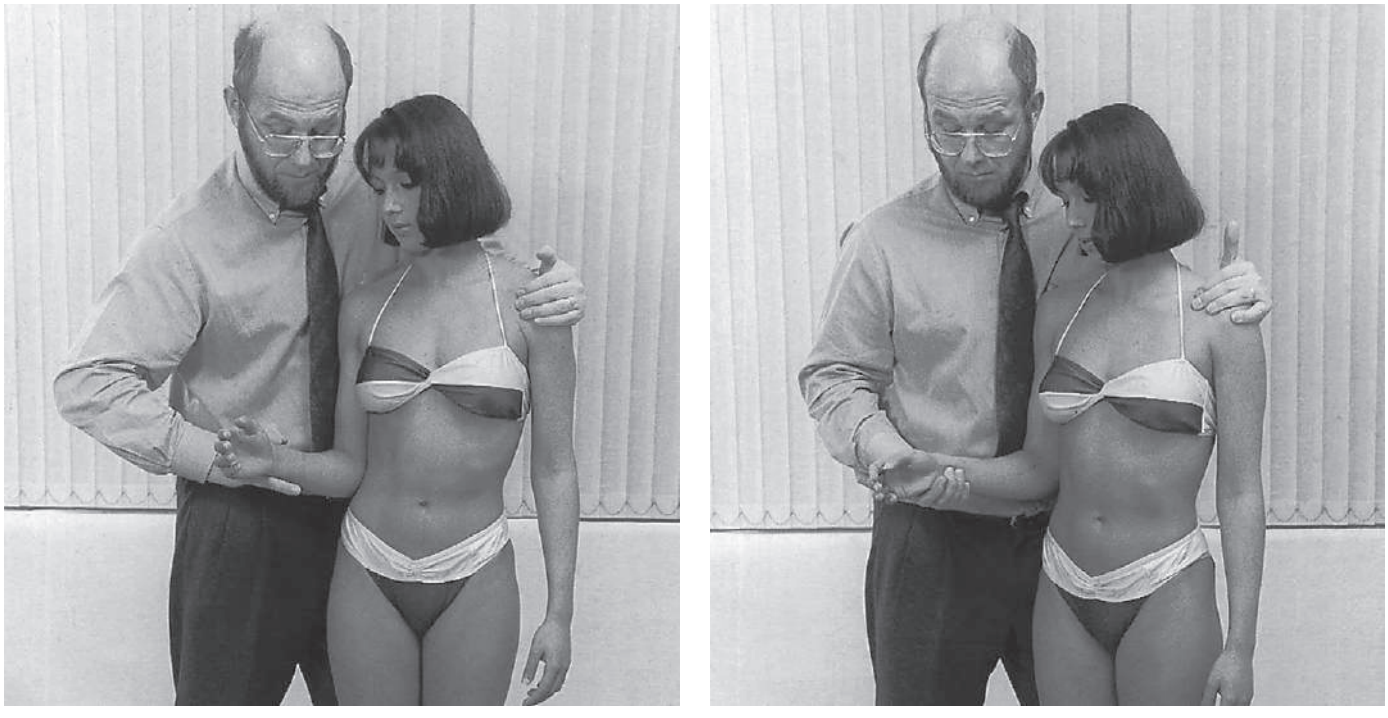
Resisted elbow extension

In the same position as for flexion, the patient attempts to extend the elbow. To be able to hold the patient's elbow at 90° of flexion, the examiner puts his or her own elbow on the iliac crest, the arm in an almost vertical position underneath the patient's wrist. The other hand rests on the patient's ipsilateral shoulder (Fig. 12.8b).



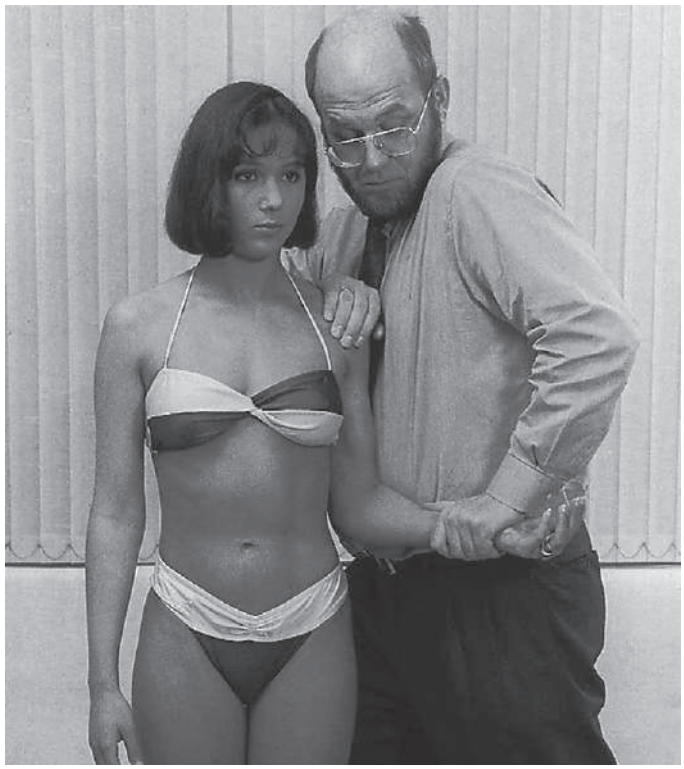
(a) (b)

Fig 12.6 • Resisted movements: (a) adduction; (b) abduction.



(a) (b)

Fig 12.7 • Resisted rotation: (a) lateral; (b) medial.



(a)

(b)

Fig 12.8 • Resisted flexion (a) and extension (b) of the elbow.

Palpation

General palpation for heat and swelling is done after the functional examination. These may be present in bacterial arthritis and in primary and secondary tumours of the humeral head, glenoid and acromion, and in acute and chronic subdeltoid bursitis.

Palpation for pain is only performed when the basic examination proves that the lesion lies within the reach of a finger. As already indicated, it always follows the clinical examination and never precedes it. Comparison between the two sides is essential. Palpation for tenderness is mainly done in acute and chronic subdeltoid bursitis and in a sprained superior ligament of the acromioclavicular joint.

Accessory tests

Sometimes the diagnosis is still not clear after the basic examination and a differential diagnosis has to be undertaken. At other times, the exact structure at fault has been identified by this stage of the examination but the precise localization of the lesion within that particular structure remains uncertain. In both cases, one or more accessory tests may be required (Box 12.4). Passive horizontal adduction is the only one which is explained here. The other tests are discussed, together with the corresponding disorders, in the following chapters.

Box 12.4

Summary of accessory tests of the shoulder

- Passive horizontal adduction
- Passive horizontal lateral rotation
- Apprehension test for anterior instability
- Apprehension test for posterior instability
- Load and shift manœuvre
- Sulcus sign
- Yergason's test
- Resisted horizontal adduction
- Resisted horizontal extension
- Resisted horizontal adduction with the arm forwards
- Resisted flexion
- Resisted extension
- Pressure against a wall
- Scapular adduction against resistance

Passive horizontal adduction

The patient's arm is brought horizontally in front of the body. At the end of the movement the elbow is pressed gently further towards the contralateral shoulder (Fig. 12.9). Twisting of the patient's trunk is prevented by the examiner bringing the other hand behind this shoulder.

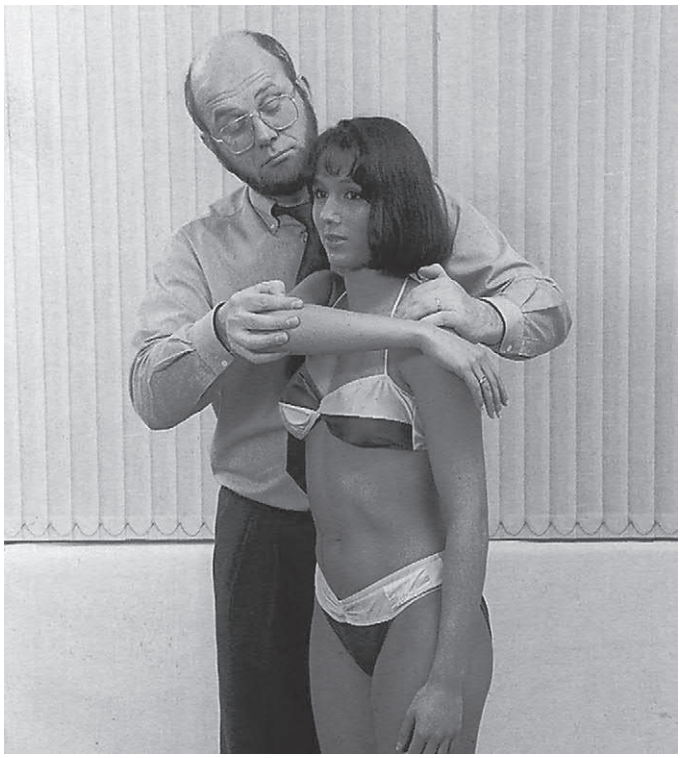


Fig 12.9 • Passive horizontal adduction.

The indications for this test are:

- Sprain of the acromioclavicular joint[21]
- Subcoracoid bursitis
- Subscapularis tendinosis.

Technical investigations

All technical investigations of the shoulder have in common the fact that they are performed to search for anatomical changes. This is in contrast with the objective of a clinical examination, which aims to detect functional deficiencies and/or pain. Anatomical changes that are not causing functional disturbances (remain asymptomatic) are very common in the shoulder region and their frequency increases with age.[22–24] Therefore, one should always interpret technical diagnoses with care and even reluctance, for it is perfectly possible that the lesion seen on imaging is in fact asymptomatic and another lesion that is not shown is at the root of the disability. A common mistake is the combination of a symptomatic glenohumeral arthritis that remains undiagnosed with sonography and an asymptomatic rotator cuff tear that is easily detected with the same technique. We therefore fully agree with Kessel, who states: 'An X- ray is best used to check a hypothesis which has been formed by history and clinical examination. One should eschew the temptation of a shortcut to the X-ray room or scanner.'[25]

Plain radiography

The main indications for radiography of the shoulder are evaluation of fractures and dislocations, imaging of bony disorders such as tumours and metastases, and identification of calcifications in or around tendons. Plain radiographs may also be helpful in the evaluation of anterior and posterior instability. Although ultrasonography is nowadays the most frequently used method to evaluate rotator cuff lesions, plain X-ray examination can be of use in the detection of accompanying appearances, such as changes in the coracoacromial arch, an unusual form or a spur off the acromion.[26] Radiography is also still advocated in long-standing massive cuff tears.[27,28]

Ultrasound scanning

Ultrasound scanning is mainly advocated for the detection of full or partial rotator cuff lesions. In experienced hands it can reveal not only the integrity of the rotator cuff but also the thickness of its various component tendons. Through careful positioning and through knowledge of the dynamic anatomy of the cuff, the experienced ultrasonographer can image selectively the upper and lower subscapularis, the biceps tendon, the anterior and the posterior supraspinatus, the infraspinatus and the teres minor. Ultrasonography has further advantages: it is non-invasive and safe, bilateral examinations are quickly performed, the shoulder can be examined dynamically and, above all, the procedure is inexpensive.[29]

One important disadvantage, however, is that the method has a long learning curve. The results are examiner-dependent and a good outcome can only be expected in experienced hands.[30] Specificity and sensitivity of as high as 98% and 91% respectively in comparison with surgical findings were claimed by Mack *et al*.[31] Others have found an overall sensitivity of 97% in diagnosing full cuff tears and of 91% in partial thickness tears.[32]

Arthrography

Single-contrast arthrography can be helpful in diagnosing complete tears of the rotator cuff, incomplete deep surface tears and instability problems.[33] It will show loss of axillary-fold space and diminished joint capacity in adhesive capsulitis, but because this disorder is so easy to detect clinically, arthrography should never be required. It is of no assistance in clarifying the differential diagnosis in cases of impingement, in the absence of a rotator cuff tear.

Arthroscopy

Arthroscopy is useful for both diagnosis and treatment.[34–39] It should be regarded as an adjuvant in those cases where the normal diagnostic aids are insufficient.[35] As it offers an excellent view of the glenoid, labrum and capsule, it is an excellent technique in repair of shoulder instability.[36]

However, confronted with the almost perfect results obtained through examination technique, the physician should never forget that full and partial tears of the rotator cuff exist in a substantial part of a normal asymptomatic population. Both cadaveric studies[37,38] and imaging studies[39,40] on asymptomatic individuals have demonstrated that cuff defects become increasingly common after the age of 40 and that most occur without substantial clinical manifestations.

Magnetic resonance imaging

Magnetic resonance imaging (MRI) has rapidly become a commonly used technique for shoulder evaluation, proving its utility in the evaluation of muscle, tendons, hyaline and fibrous cartilage, joint capsule, fat, bursae and bone marrow.[41] Today it is the most accurate non-invasive method available for imaging the rotator cuff.[42] The major advantages of this modality include its non-invasive nature, lack of ionizing radiation, excellent contrast and anatomic resolution, multiplanar imaging capability, and ability as a single imaging modality to evaluate simultaneously for a wide variety of pathologic processes.[43] One disadvantage is that small foci of soft tissue calcification may be missed on MRI.[44] As with all techniques that provide optimal visualization, one should always take into account the high rate of asymptomatic lesions and never rely simply on the outcome of the technique to make a diagnosis and start a treatment.[45–48]

Computed tomography

Computed tomography (CT) is equally as accurate as MRI for evaluation of the glenoid rim and labrum, the humeral head and the glenohumeral capsule. Nevertheless, MRI is to be preferred because it is more accurate in tendinopathies and no X-ray exposure occurs.[49] In shoulder instability, CT-arthrography seems to be the best diagnostic method.[50]

Bursoscopy

Bursoscopy can be performed under general or local anaesthesia. Although there are almost no indications for bursoscopy,[51] it may be of help in diagnosing lesions of the bursal part of the rotator cuff. At the same time, it offers visualization of the acromial roof.

Access the complete reference list online at **www.orthopaedicmedicineonline.com**

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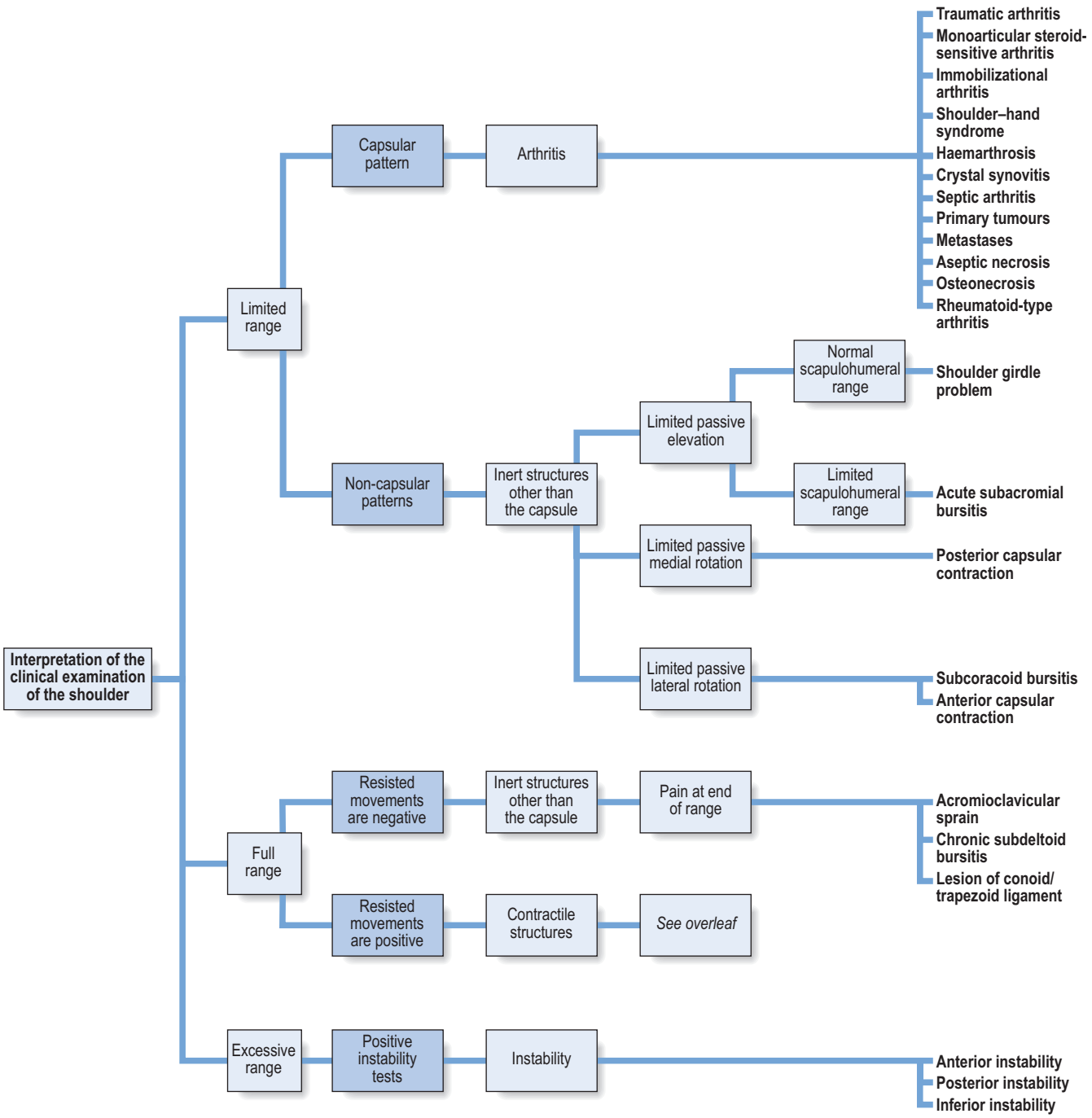
Interpretation of the clinical examination of the shoulder

13



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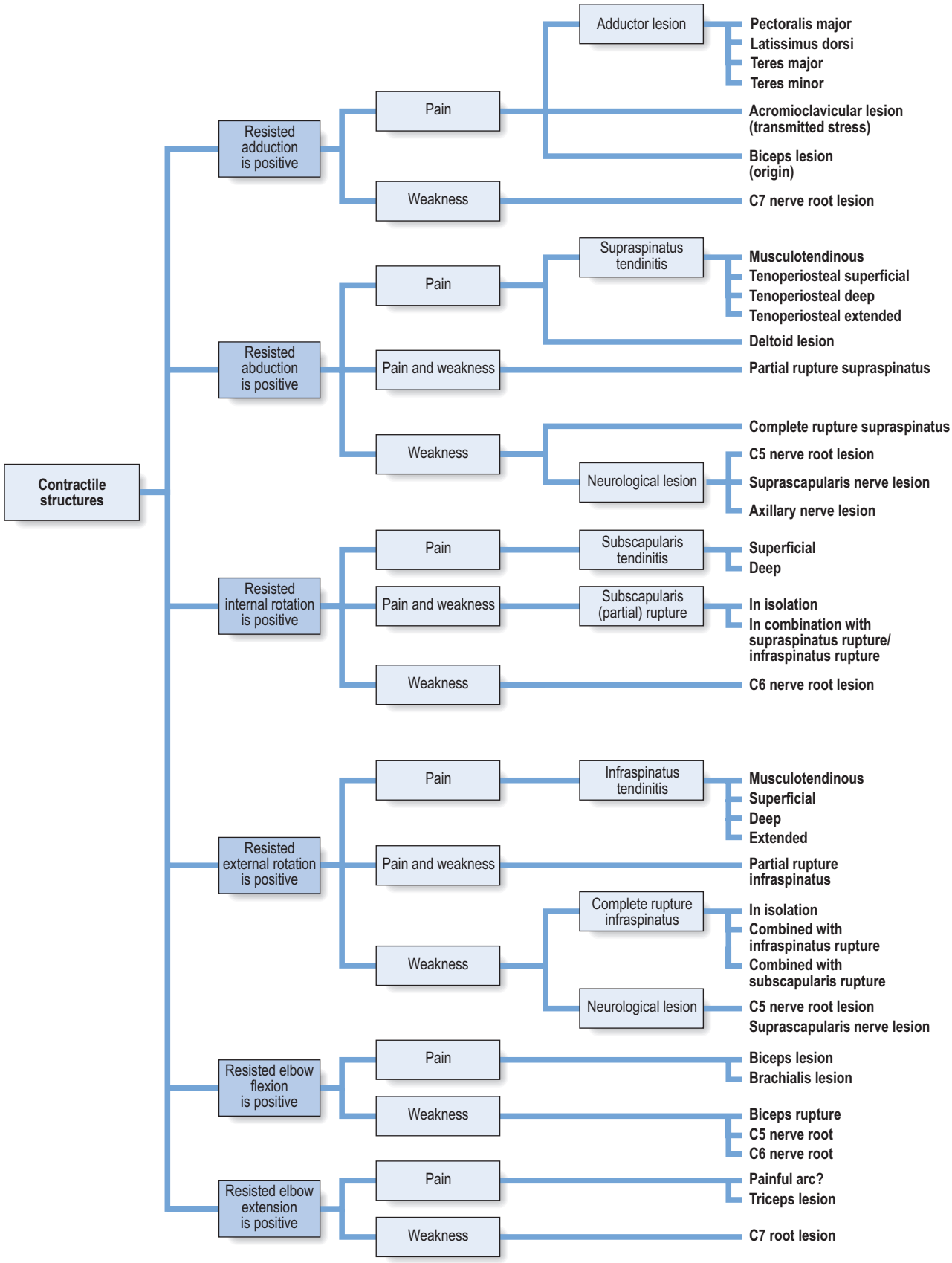
Contractile structures
Resisted adduction is positive
Pain
Adductor lesion
Pectoralis major
Latissimus dorsi
Teres major
Teres minor
Acromioclavicular lesion (transmitted stress)
Biceps lesion (origin)
Weakness
C7 nerve root lesion
Resisted abduction is positive
Pain
Supraspinatus tendinitis
Musculotendinous
Tenoperiosteal superficial
Tenoperiosteal deep
Tenoperiosteal extended
Deltoid lesion
Pain and weakness
Partial rupture supraspinatus
Weakness
Complete rupture supraspinatus
Neurological lesion
C5 nerve root lesion
Suprascapularis nerve lesion
Axillary nerve lesion
Resisted internal rotation is positive
Pain
Subscapularis tendinitis
Superficial
Deep
Pain and weakness
Subscapularis (partial) rupture
In isolation
In combination with supraspinatus rupture/ infraspinatus rupture
Weakness
C6 nerve root lesion
Resisted external rotation is positive
Pain
Infraspinatus tendinitis
Musculotendinous
Superficial
Deep
Extended
Pain and weakness
Partial rupture infraspinatus
Weakness
Complete rupture infraspinatus
In isolation
Combined with infraspinatus rupture
Combined with subscapularis rupture
Neurological lesion
C5 nerve root lesion
Suprascapularis nerve lesion
Resisted elbow flexion is positive
Pain
Biceps lesion
Brachialis lesion
Weakness
Biceps rupture
C5 nerve root
C6 nerve root
Resisted elbow extension is positive
Pain
Painful arc?
Triceps lesion
Weakness
C7 root lesion

Disorders of the inert structures

14

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Limited range of movement

Capsular pattern

Introduction

The capsular pattern at the shoulder joint is a proportional limitation of the three passive scapulohumeral movements. There is some limitation of abduction, more limitation of external rotation and less limitation of internal rotation.[1,2]

A capsular pattern always indicates a lesion of the capsule of the joint, whatever its nature may be.[3] It may be either an acute synovitis or a chronic organized reaction of the fibrous capsule.

In an acute inflammation of the synovia, the selective limitation of movement is caused by involuntary muscle spasm that protects the inflamed joint from further overstretching. In long-standing inflammation of the capsule, structural changes have set in. Intracapsular fibrosis and thickening of the capsule now cause mechanical obstruction of the movements. Several arthrographic[4,5] and magnetic resonance imaging (MRI) studies[6–9] have demonstrated that these adhesions form mainly at the axilla and the anterior portion of the capsule. This greater loss of inferior and anterior capsular elasticity explains the greater restriction of lateral rotation and abduction (the capsular pattern) (Fig. 14.1).

Staging

Although only one capsular pattern at the shoulder exists, it can present in many ways. A clear distinction must be made between the *degree of limitation*, the *stage* and the *phase*. Limitation of movement and stage do not always match.

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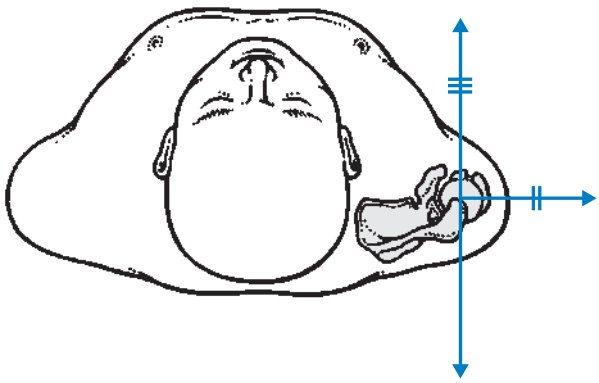


Fig 14.1 • The capsular pattern: for some limitation of abduction, there is more limitation of external rotation and less limitation of internal rotation.

Table 14.1 Capsular pattern: different degrees of limitation*

Severity of limitation	Lateral rotation	Scapulohumeral abduction	Medial rotation
Slight	30°	10°	Full range but painful
Medium	60–70°	45°	10–15°
Severe	90–100°	70–80°	15–25°

*From Cyriax[1]: p. 135.

The degree of the limitation is expressed in its magnitude; limitation can be slight, medium or gross, although it is always in the same (articular) proportion (Table 14.1).

The stage is a clinical estimation of the severity of synovial inflammation. The staging is based on four clinical criteria: pain at rest, nocturnal pain, the distal reference of the pain and the end-feel (Box 14.1). Three stages are considered. Stage I corresponds to a minor degree of inflammation: there is no pain at rest and no nocturnal pain, the pain does not spread beyond the elbow and the end-feel shows no protective muscle contraction. Stage III is the worst: the highly inflamed synovia leads to pain at rest and at night, the pain spreads beyond the elbow, and the end-feel shows protective muscle spasm. From a therapeutic point of view, stage is more important than degree of limitation, especially in post-traumatic arthritis and immobilizational arthritis.

The phase situates the arthritis on the timeline of the natural history. Classically three phases are considered: the painful phase, the progressive stiffening phase and the thawing phase (see p. 223).

During the initial phase of arthritis, when limitation has not yet set in, making a diagnosis can be very difficult. The only finding is pain at the end of all passive movements. It may then be helpful to remember that passive lateral rotation is the most painful movement, accompanied by a slightly abnormal end-feel. It is only after some time that an increasing limitation of lateral rotation sets in, subsequently followed by a diminishing range of both abduction and medial rotation.

Box 14.1

Staging

Stage I: minor inflammation

- No pain at rest
- No pain at night
- Pain remains above the elbow
- Normal or slightly hardened end-feel

Stage III: gross inflammation

- Pain at rest
- Pain at night
- Pain spreads below the elbow
- End-feel indicates muscle spasm

Stage II

- Shows a mixed result on the four clinical criteria (one or more of the criteria are slightly positive and the other ones negative). Example: a patient having pain below the elbow, but no pain at rest, who can lie on that side at night and has a more or less elastic end-feel. This is only one example of a stage II arthritis; all other combinations of the four criteria are possible

Conditions

Stiffness of the glenohumeral joint has classically been called 'frozen shoulder'.[10] Several investigators have attempted to propose a nomenclature to separate different types of shoulder stiffness.[11–13] The subgroup typing was based on both the severity of stiffness and the presence or absence of an associated cause. The group without an apparent background was further subdivided into either 'post-traumatic frozen shoulder', where an injury or a surgical intervention was at the root of the disorder, and 'primary frozen shoulder', where no causative precursor could be found.[14]

Cyriax[1] listed 13 different disorders leading to 'shoulder stiffness' with a capsular pattern (Box 14.2).

Whenever a capsular pattern is found, an attempt is made to categorize the lesion. Differentiation between the subgroups is achieved by history, clinical presentation and paraclinical investigations as follows:

- Try to detect an intrinsic aetiology, which may be either a general disease (rheumatoid type of arthritis) or a local condition: infection, gout, haemarthrosis or tumour.
- In the remaining subgroup where no intrinsic aetiology can be found and the only finding is of a progressive stiffening of the capsule, history will indicate whether the arthritis should be called post-traumatic, post-immobilization or primary.

Categorizing capsulitis of the shoulder in this manner is extremely important because different lesions have a different development and prognosis. Furthermore, categorization is of great use in deciding the course of treatment.

Box 14.2

Classification of 'frozen shoulder'/shoulder arthritis

Classification of 'frozen shoulder'	Cyriax's classification of shoulder arthritis
With apparent aetiology • Rheumatoid/infectious/crystalline • Osteoarthrosis Without apparent aetiology • Post-traumatic frozen shoulder • Primary frozen shoulder	• Traumatic arthritis • Immobilizational arthritis • Monoarticular 'steroid-sensitive' arthritis • Shoulder–hand syndrome • Rheumatoid-type arthritis • Haemarthrosis • Crystal synovitis • Septic arthritis • Primary tumours • Metastases • Aseptic necrosis • Osteoarthrosis • Neuropathic destructive arthropathy

Traumatic arthritis

This condition is almost never encountered in patients younger than 40 years. Because the risk of traumatic arthritis in youth is virtually zero, preventive measures after injury are unnecessary for the young.

Natural history

A capsular pattern may develop after glenohumeral (sub)luxations, contusions or surgical procedures to the shoulder.[15] Most often, however, injury need not have been severe and a traumatic arthritis may precipitate some days after the shoulder capsule sustained an indirect and sudden traction or, for example, after the joint bumped against a wall. Because it can take some weeks for the pain to become bad enough to force the patient to consult a physician, it is quite possible that such a minor accident may have been forgotten.

The evolution and natural history of traumatic arthritis are quite typical. It takes about a year for the lesion to heal spontaneously. During this process, three stages of about 4 months each are observed (Fig. 14.2).[16] In the first, 'painful phase', both pain and limitation of movement increase. In the second, 'progressive stiffness phase', pain diminishes but limitation remains the same. It is not until the beginning of the last 4 months that limitation begins to decrease (the resolution or 'thawing' phase), so that by one year movement is back to normal.[17] Several authors, however, have demonstrated a significant number of patients with a delayed thawing phase and one instance showed persistent stiffness for 6 years.[18–20] Sometimes elevation and lateral rotation may remain slightly restricted permanently.[21]

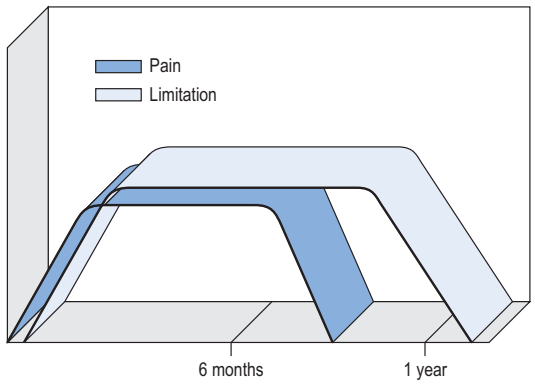


Fig 14.2 • Natural history of traumatic arthritis.

Painful phase

The onset of the arthritis is very characteristic. Immediately after the injury an ache develops that may wear off in 2 days or so but which re-appears a few days later, increasing progressively over the next few months. During the painful phase, the intensity of the arthritis typically evolves from stage I, through stage II to stage III.

In the first 2 weeks after onset, the patient complains of an ache mainly on activity. During this period, pain does not spread beyond the elbow and sleep on the affected side is possible. A subtle capsular pattern can be detected on functional examination: for example, limitation of some 20° on lateral rotation and 5° on abduction, together with a normal (but slightly painful) range on medial rotation. All these movements are also painful at the end of the achievable range. The end-feel may be slightly altered but not definitively abnormal. A clear stage I arthritis is present.

As the arthritis increases, stage II will be found at 2–6 weeks from the onset: pain may interfere with sleep, it spreads below the elbow or a spastic end-feel may be apparent on passive testing. After 2 months, the inflammation is at its worst. Now, with the features of stage III, the patient suffers from continuous pain, day and night, which spreads below the elbow. A limitation of about 80° on lateral rotation, 60° on abduction and 20° on medial rotation may be present. The end-feel is abrupt (hard or muscle spasm).

Progressive stiffness phase

Although the severity of the synovial inflammation may progressively diminish from the fourth month, the limitation in range remains the same for a few more months. With decreasing inflammation, pain at night and at rest gradually disappears but remains above the elbow. The end-feel changes from spastic to hard ligamentous (but still painful). The arthritis gradually returns to stage II and finally, after about 8 months, stage I.

Thawing phase

The final stage of the natural evolution is the resolution or the thawing phase, characterized by a slow and gradual gain in mobility. Usually a few months (4–6) may be required to achieve full functional motion. The joint is in stage III with moderate pain and a hard ligamentous end-feel.[22]

Treatment

The choice of treatment for post-traumatic arthritis should always be adjusted according to the duration and severity of symptoms. Treatment techniques should also be applied in the context of the patient's needs, risk factors and tolerance. Finally, the outcome of the treatment must always be related to the expected natural history of the disease, and treatment is only begun when it is expected to change the course of this natural history positively.[23,24]

Prophylaxis

The primary management for post-traumatic arthritis in the shoulder is prevention: to suppress the natural tendency of most patients to immobilize the painful joint until comfort returns. Therefore, a patient older than 40, seen shortly after a shoulder injury, should be encouraged to use the arm as normally as possible with movement to full range at least twice daily. When the patient has excessive pain, a therapist can undertake mobilization just to maintain normal movement at the glenohumeral joint. The joint should be only gently forced actively and passively, using the capsular stretching technique. In this way, arthritis with secondary limitation of movement will not have an opportunity to develop and the pain disappears. Once pain has ceased, treatment can be stopped.

Passive movements

If arthritis has set in, it is too late for prevention and treatment to the capsule should be given. Gentle but firm passive stretching exercises have proven effective in the relief of pain and recovery of range in motion in up to 90% of patients with capsular stiffness.[25–30] However, some studies report inadequate results with stretching and even exacerbation of the condition.[31,32]

Two main types of passive mobilization are used, depending on the degree of inflammation. As a consequence, clinical staging is the guide. Stages I and II with non-spastic end-feel (stage IIa) are cured by mobilization using the capsular stretching technique. Stages IIb (spastic end-feel) and III are treated by either capsular distraction, which is a less irritating type of mobilization, or by intra-articular injections with corticosteroid. Preference is for the steroid injections but, if the patient refuses injections or if use of steroid is contraindicated, distraction can be very useful.

Warning

Stretching manœuvres on a highly inflamed capsule exacerbate the condition. The following indications of a high degree of inflammation are therefore considered as contraindications:

- Arthritis in stage III or in stage II with a spastic end-feel.
- Wrong end-feel on first distraction attempt: when the therapist brings the patient's arm up and starts the manœuvre, attention should be paid to pain and end-feel. If it is possible to bring the elbow slightly further towards the couch without increasing pain too much or without provoking muscle spasm, it is a good sign that capsular stretching will succeed. In the opposite case, stretching should not be undertaken and intra-articular injections or the distraction technique should be used instead.
- After-pain lasting for more than 2 hours.

Capsular stretching

Technique

Before capsular stretching is begun, analgesic short-wave diathermy can be given for 10 minutes.

The patient lies supine and brings the ipsilateral hand on to the forehead. The therapist stands on the same side, facing the patient, and puts one hand on the sternum and the other on the elbow of the affected side. By pushing the elbow backwards towards the couch, the capsule of the joint is stretched (Fig. 14.3). In this way the inferior recess of the capsule, where

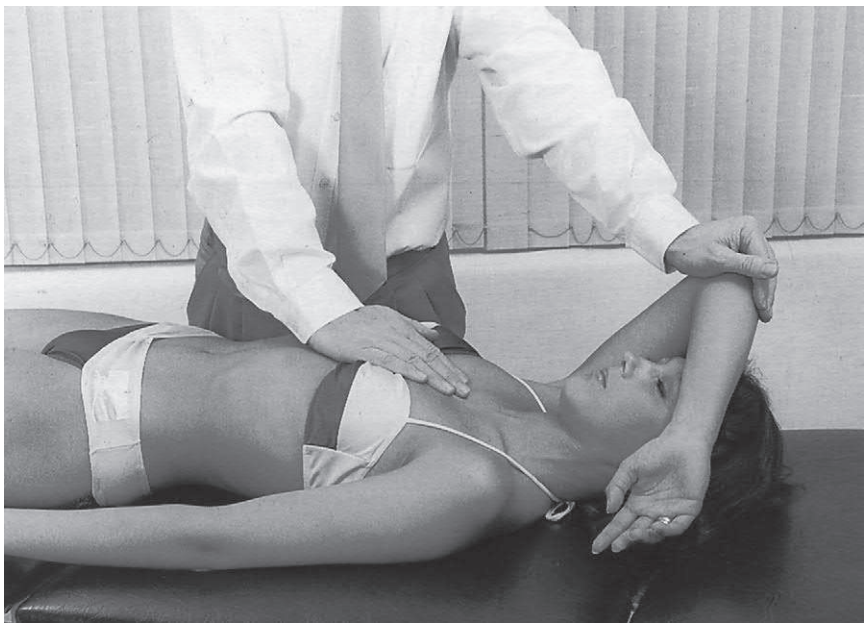


Fig 14.3 • Capsular stretching.

most of the adhesions lie, is elongated. The hand on the sternum prevents the patient from curving the trunk to avoid the stretch. The capsular stretching is done in elevation. As this is a combined movement, the rotations improve simultaneously with the increase in abduction range. If this does not happen, the shoulder must be stretched in lateral rotation as well.

Stretching is not manipulation and any tendency to 'jerk' should be avoided. Rather the manœuvre is carried out by exerting continuous pressure that is gently intensified for a few seconds, then slackened off slightly for a little while and increased again. This is repeated for as long as the patient can bear it and is followed by a full rest, in which the arm is brought down, avoiding pain by axial traction. Stretching should be repeated several times during one session and can be combined with hold–relax techniques.

The therapist should teach the patient which mobilizing exercises can be done at home in order to maintain the mobility that has been regained. These should be performed several times daily.

Force used during stretching

The stretching itself is applied with a reasonable amount of force, sufficient to provoke some discomfort at the time. But more important than the patient's sensation during the treatment is what is felt afterwards, which provides the information about the amount of force to be used.

If there is increased pain for the first 2 hours after the procedure, the correct amount of power has been used and future treatment must be identical. If pain is not increased, the stretch power has been insufficient and, at the next session, must be increased.

If the patient returns after 2 days still having increased pain attributable to the procedure the implication is not – as one might logically assume – that the stretching was too aggressive. Rather it indicates this shoulder cannot accept capsular stretching at all. Even if only stage I or IIa arthritis is present, and although all indications for stretching seem to be fulfilled, distraction or injections should be substituted.

Sequence and duration

These sessions are held three times a week for about 15–20 minutes each. Improvement is expected after 5–15 sessions, depending on the severity of the lesion.

Stretching is continued until the shoulder is back to normal (pain and range) or no further gain is achieved. The results are good, and passive capsular stretching has been proven to be a fast and safe mode of treatment for 'adhesive capsulitis' in stage I or IIa arthritis.[33–35]

Long-standing cases

Sometimes, in long-standing cases, it is possible to hear and feel adhesions rupture on stretching. Immediately afterwards, pain diminishes and mobility increases. Two or three of these ruptures may be necessary to restore a full range of movement.

Distraction

This technique consists of very gentle elongation of the joint capsule performed in such a way that the fibres are stretched longitudinally. It has been suggested that this inhibits nociceptive reflexes which result from long-standing stimulation of the nocisensors. These reflexes would be responsible for increased sympathetic activity giving rise to vasoconstriction of the vessels around the joint.[36]

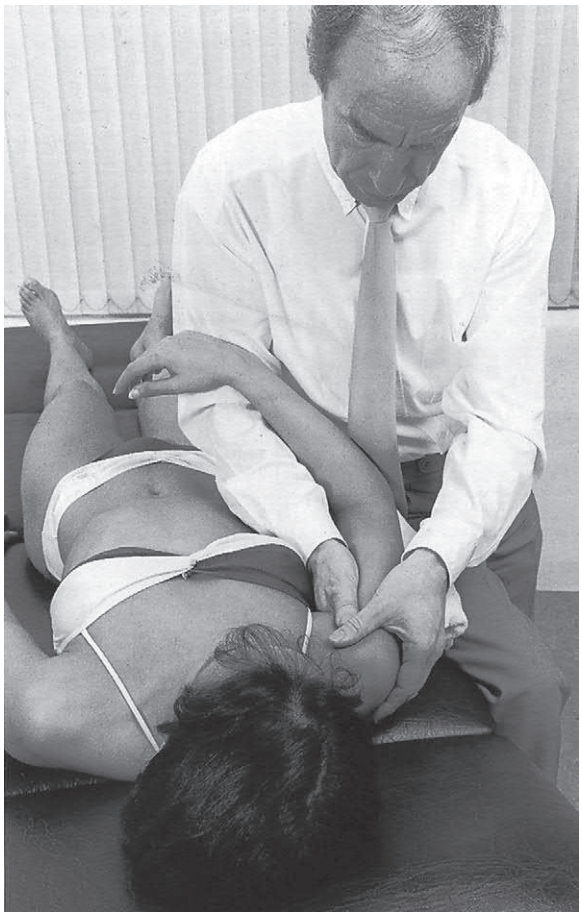


Fig 14.4 • Capsular distraction.

Indications

Patients with stage I or IIa arthritis in whom capsular stretching is contraindicated have an open choice between steroid or distraction.

Distraction may be used in patients with traumatic arthritis in stage IIb or III for whom steroids are refused, have been used without success or are contraindicated.

Technique

The patient lies supine, the arm along the side, with a small cushion beneath it for maximum comfort. The therapist sits at the patient's painful side and brings the ipsilateral hand deep into the axilla, the other one partially on the outer aspect of the shoulder, partially reinforcing the one in the axilla (Fig. 14.4). The ipsilateral hand will try to pull the head of the humerus out of the glenoid fossa. The direction of the pull is mainly lateral and slightly cranial and anterior.

Initially the manœuvre is done with the patient's arm in the most comfortable position. Once mobility has increased, some degree of lateral rotation and abduction can be added, so that the distraction is performed at the end of the possible range. At first, when there is a lot of pain, fine vibration can be additionally incorporated so as to stimulate the mechanoreceptors and inhibit the nocisensors, resulting in pain relief.

During the first sessions not much happens. It is only after a few sessions that the therapist feels a loosening of the patient's shoulder such that the humeral head is felt to leave the glenoid fossa.

This technique is performed with so little force that it is not at all painful during the session and is not followed by any after-pain.

Sequence

As with stretching, this technique is done three times a week for about 15–20 minutes each. Distraction is continued until the arthritis has regressed to stage II or IIa. Then normal capsular stretching can be performed.

Manipulation under anaesthesia

Manipulation under anaesthesia has been used for over a century. Some believe in its effectiveness,[36–38] while others have denounced its use because they think there is no change in the time course of the disease after the manipulation or they have seen too many complications. Ruptures of the subscapularis tendon, damage to the neurovascular structures, and fractures and dislocations have been reported after manipulation under anaesthesia.[39–43] That the method can cause iatrogenic damage was recently demonstrated by an arthroscopic study.[44] After manipulation, the anterior portion of the capsule was seen to be ruptured in 75% of the patients. Iatrogenic superior labrum lesions were observed in 15%, fresh partial tears of the subscapularis tendon in 10% and anterior labral detachments in 15%.

Manipulation should be considered only if all other treatment methods fail. In fact this type of treatment is very seldom needed as almost all capsular limitations can be resolved by either mobilization techniques or a sequence of intra-articular injections with triamcinolone.

Intra-articular injections

In an attempt to suppress the painful inflammatory response in post-traumatic capsulitis of the shoulder, intra-articular injections with corticosteroids have been used for decades. Studies that evaluate the response to intra-articular injections generally combine the injection with other treatment methods and rarely compare the efficacy of the injection alone. However, some studies could demonstrate improvement in pain scores and increase in range of motion after steroid injections alone.[45–49] Other investigators are very sceptical about the injections as their studies failed to demonstrate the benefit of the treatment.[50]

Cyriax could not initially find benefit in intra-articular hydrocortisone injections[51] but after he detected the advantages of a sequence of consecutive intra-articular injections with triamcinolone, he became a very enthusiastic advocate of intra-articular injections for capsulitis of the shoulder.[52]

Our experience is that traumatic arthritis responds very well to injections of 20 mg of triamcinolone, provided the treatment is given in stage III or IIb and the correct sequence is followed (see below).

Technique

The patient lies prone, the arm under the abdomen and the elbow flexed at a right angle. This position has two advantages: first, it brings the articular surface of the humeral head to point straight backwards, so creating a large target for the needle; second, the patient cannot move the arm.

The coracoid process is palpated in the infraclavicular fossa. The examiner puts the index finger here and places the thumb dorsally on the posterior angle where the scapular spine meets the acromion. A 4 cm needle is fitted on a 2 mL syringe filled with 20 mg of triamcinolone. It is inserted just below the thumb and aimed at the coracoid process. The same approach is used nowadays for arthroscopy via the posterior portal.[53,54] After about 2–3 cm the needle is stopped by the articular surface of the head of the humerus and there is a typical cartilaginous sensation. At this point, the needle lies intra-articularly. Just before the needle is arrested, tough resistance is felt on passing through the capsule (Fig. 14.5).

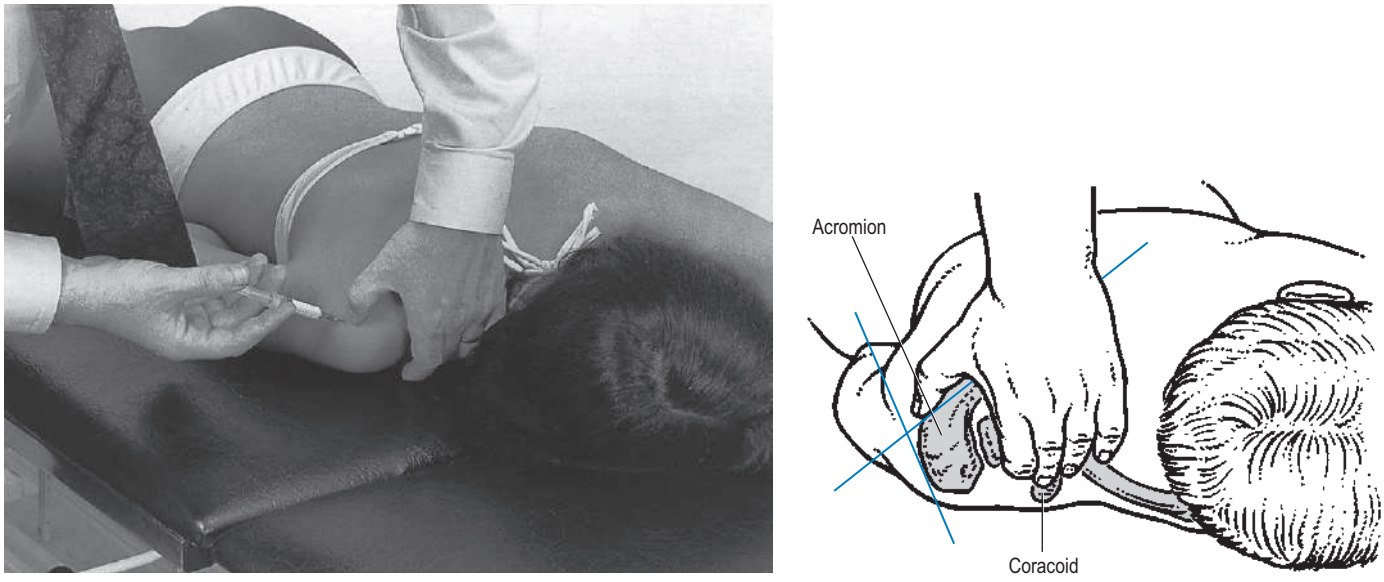


Fig 14.5 • Intra-articular injection.

With the needle in cartilaginous contact, 2 mL of triamcinolone are injected. A reasonable amount of resistance is encountered. Exceptionally, not even a single droplet can be injected, in which case the point of the needle is fully within the articular cartilage. Should this be the case, the needle should be withdrawn by about 2 mm while injection pressure is maintained. Once the tip of the needle leaves the cartilage, the steroid floats in. The injection is given in this position and is minimally painful.

Sequence

The aim is to keep the capsule continuously under anti-inflammatory influence until the inflammation has almost fully disappeared. Therefore the next injection must be given just before the effect of the previous one has worn off.

A practical scheme, with increasing intervals between the injections, is as follows:

- First injection: day 0.
- Second: after 1 week: day 7.
- Third: 10 days after the second injection: day 17.
- Fourth: 2 weeks after the third one: day 31.
- Fifth: 3 weeks later: day 52.
- Sixth: 4 weeks later: day 80.
- Seventh: 5 weeks later: day 115.
- Eighth: 6 weeks later: day 157.
- Ninth: 6 weeks later: day 199.

The usual sequence is that, after the first injection, the patient will have less pain, and from the third injection onwards the limitation of movement starts to decrease.

The injections are given until the arthritis reverts to stage I. Treatment can then be stopped and the arthritis will continue to resolve spontaneously. In general, about five injections are needed.

Exceptionally it happens that, even if the scheme is followed, a patient who is temporarily better after the last injection complains of increasing pain during the days before the next appointment. This means that the interval between injections was too long and should be reduced.

Sometimes the pain subsides progressively as expected but limitation of movement does not alter. In this event, capsular stretching should be tried and is started once the arthritis is at stage I. If stretching makes the pain relapse, it must be postponed for a few weeks and another injection given.

A summary of the treatment of traumatic arthritis is shown in Figure 14.6.

Complications and side-effects

Because there is a small but real risk of infection via intra-articular injections, it behoves the practitioner to prepare the skin and handle the procedure under surgical conditions.

It is also important to remember that steroid injections in diabetic patients, even given intra-articularly and in quite small doses, may cause blood glucose to fluctuate and also may incur a greater risk of infection.

Further side effects may include some flushing on the following day and perhaps some interference with the menstrual cycle because of hormonal effects.

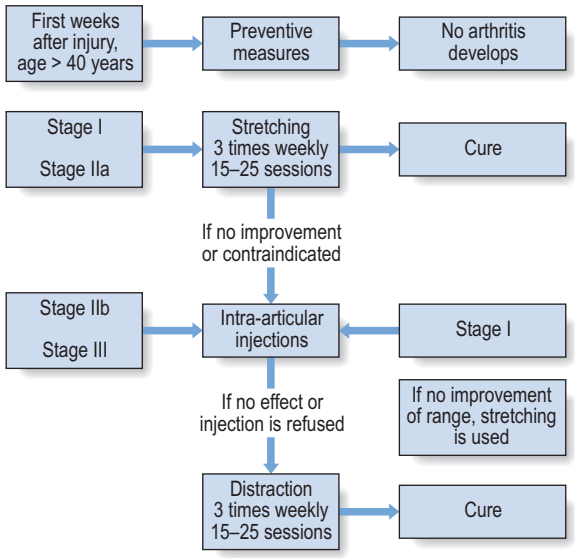


Fig 14.6 • Treatment of traumatic arthritis.

Other treatments

Hydraulic distension

Hydrodilatation, sometimes referred to as distension arthrography, has been proposed as a therapeutic procedure for glenohumeral joint contracture.[55] It is proposed that its benefits are derived from a combination of the anti-inflammatory effect of cortisone with the mechanical effect of joint distension, thereby reducing the stretch on pain receptors in the glenohumeral joint capsule and its periosteal attachments.[56] Hydrodilatation was first used in 1965 by Andren and Lundberg,[57] who reported variable results ranging from extremely effective to extremely painful. More recent studies have also cited variable results.[58–60] Several double-blind, prospective studies could not detect any significant differences between a regimen of hydrodilatation that included steroids compared with steroid injections alone.[61–64] As with manipulation under anaesthesia, this technique should be reserved for those few cases that do not respond to passive mobilization and injection with corticosteroids.

Arthroscopic release

Recent studies have demonstrated that arthroscopic capsulotomy may be an effective technique in the management of the frozen shoulder that does not respond to physiotherapy.[65–68] During standard shoulder arthroscopy, intra-articular cautery is used for complete division of the anterior–inferior capsule, the intra-articular portion of the subscapularis tendon and the middle glenohumeral, the superior glenohumeral and the coracohumeral ligaments.[69]

Immobilizational arthritis

In patients over 60, when the shoulder is immobilized, it is at risk of becoming stiff. The reasons for the initial immobilization may be multiple. Immobilizational arthritis is a well-known complication in hemiplegics.[70–73] During a prospective study, conducted by Bruckner and Nye, 25% of patients with

subarachnoid bleeding developed a frozen shoulder over an observation period of 6 months.[74] Other neurological conditions such as Parkinson's disease may precipitate capsular stiffening.[75,76] Immobilization of the arm for disorders such as a fracture of the elbow or the humerus is also reason enough to develop a post-traumatic arthritis.[77] For many years clinicians have associated ischaemic heart disease and shoulder arthritis,[78] which eventually develops as the result of immobilization after an infarction or surgery.

Natural history

The development and natural history of immobilizational arthritis cannot be distinguished from those of traumatic arthritis. Again, there are three phases in the progress of the disease. In the painful phase, both pain and limitation of movement increase. In the subsequent progressive stiffness phase, the pain diminishes but the limitation remains the same. Lastly limitation decreases during the final 'thawing' phase. Altogether, it takes about a year for the lesion to recover spontaneously and movement to return to normal.

Treatment

This condition should never be encountered. It is very important for primary care physicians and physiotherapists to realize that immobilized shoulders should be given gentle movements at least once a day, in order to prevent the development of an immobilizational shoulder arthritis. This simple advice was already given by Neviaser, who in 1945 wrote: 'I believe we can accept the fact that disuse and inactivity play a very important role in the etiology.'[79] In prevention, it is sufficient to maintain the normal range of movement from the very beginning of immobilization. If arthritis has set in by the time the patient is first seen, it should be managed in the same way as traumatic arthritis: stage I is treated with capsular stretching, stage III with a series of intra-articular injections of 20 mg of triamcinolone. Stage IIa can also be treated with stretching if the end-feel is right. If this is not the case, the patient should receive intra-articular injections.

Monoarticular 'steroid-sensitive' arthritis

A monoarticular arthritis that develops without apparent cause – neither trauma nor immobilization can be traced in the history – is called 'idiopathic frozen shoulder'[80] or monoarticular 'steroid-sensitive' arthritis. The latter term comes from Cyriax, who discovered that most cases of 'freezing arthritis' could be successfully treated with a series of intra-articular steroid injections.[81]

Pathophysiology

Although the exact cause of the capsular inflammation and the subsequent capsular fibrosis is not exactly known, recent investigators have focused on the inflammatory cellular changes and immunological response in the synovium and the capsule. Currently it is not known exactly what triggers the initial synovial inflammation. Some point to specific cytokines which may be involved in the early inflammatory stages of the disease.[82] Several references in the literature assume frozen shoulder to be an algoneurodystrophic process.[83,84] Others suggest that a proteinase may be involved in the pathogenesis of both a Dupuytren's contracture and a frozen shoulder.[85,86] Others have suggested that an area of focal necrosis in a degenerative tendon is the earliest lesion, followed later by a generalized chronic inflammatory reaction of the whole capsule and of the rotator cuff.[87]

Hannafin and colleagues have studied the histopathologic evolution of monoarticular shoulder arthritis. They found an initial hypervascular synovitis, provoking a progressive fibroblastic response in the adjacent capsule, finally leading to diffuse capsular fibroplasia, thickening and contracture.[88]

Incidence

The bulk of patients who present with primary monoarticular arthritis of the shoulder are between 45 and 60 years of age,[77,89] although the disease can be encountered at any age.[90] Approximately 70% of patients presenting with adhesive capsulitis are women.[91] The overall prevalence of the disorder is about 2%. In diabetics the figure is almost 11%.[92,93] Other studies show diabetes to be present in 25–38% of the patients suffering from adhesive capsulitis.[94,95] Some connection with hyper- or hypothyroidism has also been suggested, although the link between the disorders remains obscure.[96] Recently adhesive capsulitis has also been described in patients treated with highly active antiretroviral drugs.[97,98]

Natural history

The onset is spontaneous and involves only one shoulder at a time; sometimes the other shoulder may become involved within 5 years.[99]

In the spontaneous evolution of a monoarticular steroid-sensitive arthritis, four periods of about 6–9 months each are distinguished (Fig. 14.7).[100] Initially, the patient starts to feel pain at the shoulder for no apparent reason. This increases progressively, becomes continuous (although often worst at night) and starts to spread beyond the elbow. It causes many months of sleepless nights, a more than sufficient reason to start treatment at once. At the same time movement becomes

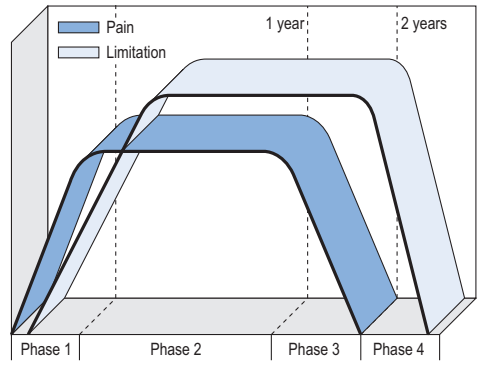


Fig 14.7 • The four phases of monoarticular steroid-sensitive arthritis.

progressively limited. During the second phase, the pain gets no worse but remains maximal for another 6 months. Limitation does not change for about 12 months. One year (sometimes even more) after the onset, the pain starts to diminish and it disappears almost fully at the end of this third phase. Restriction of movement, however, does not alter. Finally, in the fourth period, the limitation of movement gradually decreases. At this time, only some slight discomfort and a certain degree of stiffness remain, which usually disappear fully at the end of the 2 years. Exceptionally, a few degrees of restriction of elevation will be permanent.[101,102]

These clinical phases in the natural history of idiopathic capsulitis of the shoulder correspond roughly with the histopathologic phases identified by Hannafin:[88,103] first hypervascularization and inflammation of the synovia (first period), then progressive fibroblastic response of the capsule (second and third periods), and finally the remodelling of the capsule (fourth period).

Functional examination

The first phase is initially characterized only by pain, which occurs at the extreme of all passive movements. Almost no limitation will be present during the first few weeks. Arriving at a diagnosis may be difficult at this stage. As in traumatic arthritis, it is of help to know that the pain is referred in the C5 dermatome and that full passive lateral rotation is the most painful test. Also, the changing end-feel may be of clinical importance in making an early diagnosis. Later, as the pain increases, limitation of movements sets in. From now on, a clear capsular pattern is found that will have its maximum limitation by the end of the first period (6–9 months from the onset of the disorder). By then the end-feel will certainly be that of muscle spasm.

Both pain and limitation remain maximal during the entire second phase.

The third phase is characterized by a gradual decrease in pain. The end-feel progressively changes towards a hard one, indicating the loss of capsular elasticity. Limitation of movement, however, does not change yet.

During the final ('thawing') phase, the range of movement progressively increases to return to normal about 2 years after the onset. Some authors have stated, however, that it may take longer for the stiffness to disappear completely or that some degree of stiffness may remain.[104] In general, the duration of the recovery stage is related to the duration of the stiffness phase: the longer the stiffness phase, the longer the recovery phase.[105]

Staging

As in traumatic arthritis, three stages are distinguished in relation to the degree of inflammation (see p. 222): stage I is the slightest, while stage III is the worst (Table 14.2).

The stages are based on the following criteria:

- Does the pain spread beyond the elbow?
- Is there spontaneous pain?
- Can the patient lie on the affected side at night?
- What is the end-feel?

Table 14.2 Stages of monoarticular steroid-sensitive arthritis

	Stage I	Stage II	Stage III
Pain beyond elbow?	No	Yes	
Spontaneous pain?	No	Yes	
Can lie on the affected side at night?	Yes	Mixed pattern	No
End-feel?	Normal		Abrupt: hard or muscle spasm

Although the treatment is the same for stages I, II and III, the classification helps in following the natural development, gives an idea of the effect of the treatment, and indicates when to stop treatment.

Treatment

Because spontaneous recovery takes about 2 years and the patient suffers severely in the meantime, treatment is absolutely necessary.[106,107] As a rule, a series of intra-articular injections with triamcinolone are given, whatever the stage of the arthritis. Exceptionally, capsular distraction is used for those patients who do not want injections or when steroids are contraindicated. If distraction is used, it should be performed in the same way, frequency and duration as for traumatic arthritis (see p. 225). Injections for monoarticular steroid-sensitive arthritis are given in the same way (technique and interval) as for traumatic arthritis (see p. 226). They can be stopped once the patient can use the arm freely, the end-feel is back to normal and no relapse of pain occurs by 6 weeks after the previous injection. If some limitation of movement still exists by then, it usually disappears spontaneously during the following months.

It is rare for patients not to respond to injection therapy or to remain with a painless stiff shoulder.[108] In 1989 we did a prospective study on 54 patients with idiopathic arthritis of the shoulder. The youngest patient was 40, the oldest 71. On clinical examination all showed a clear capsular pattern and none had pain on any resisted movement. Laboratory tests were performed to exclude other rheumatoid types of arthritis and to check for a possible association with diabetes (only one diabetic patient was identified). Over 90% of the cases presented initially with stage II or stage III arthritis. All were treated by a series of intra-articular injections given at increasing intervals. The total number of injections given was between four and nine with an average of six (Fig. 14.8). After the first injection half the patients were less troubled at night. This figure increased to more than 90% after the third injection. Spontaneous pain decreased in the same way as the nocturnal pain but with some delay, being less severe and progressively less distantly referred. After eight injections, 98% of all patients had no pain (Fig. 14.9).

The range of movement of rotation and elevations began to increase after the first injection, though this was not very obvious clinically. An increased amplitude usually became clear after the third injection. The final conclusion of the study was that 98% of all patients recovered fully from their pain – only

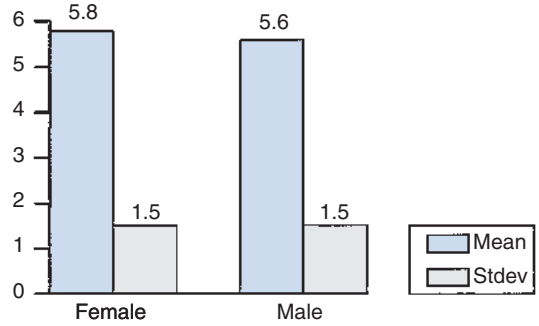


Fig 14.8 • Mean numbers of steroid injections given in a study of monoarticular steroid-sensitive arthritis (grey error bars are standard deviations (Stdev)).

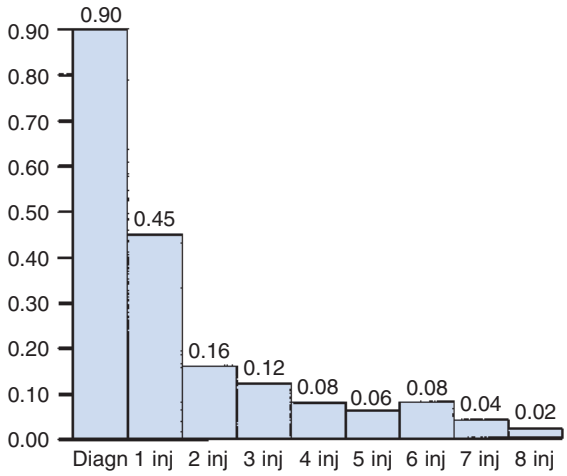


Fig 14.9 • Decay of nocturnal pain (% of total) in relation to the given number of injections.

one patient continued to have a painful shoulder. There was an 80% increase in range of movement after the seventh injection. The results of this study correspond roughly with what was found by others.[87,109]

Shoulder–hand syndrome

Shoulder–hand syndrome, first described in the 1950s, is a relatively rare clinical entity classified as a complex regional pain syndrome type 1 (CRPS1), or 'reflex sympathetic dystrophy'.[110] The condition consists essentially of a painful 'frozen shoulder' in combination with disability, swelling, and vasomotor or dystrophic changes in the ipsilateral hand. At onset, the hand is bluish and diffusely swollen. Later the wrist and fingers become stiffened (flexion contracture with limitation of extension) and the skin shiny and atrophic.[111] The shoulder involvement usually precedes, sometimes accompanies or rarely follows the changes in the hand. The pathophysiology is not completely clear but a predominant 'sympathetic' factor affecting the neural and vascular supply to the affected parts seems to be involved.[112,113] Cyriax considered the syndrome to be a type of monoarticular steroid-sensitive arthritis.[114] So far the exact cause has not been clarified, although some assume that emotional instability could be an important factor. The arthritis is treated in the same way as any other steroid-sensitive arthritis

Rheumatoid-type arthritis

Rheumatoid arthritis (RA) is an autoimmune disorder of unknown aetiology characterized by symmetric, erosive synovitis and sometimes multisystem involvement. Any joint can be involved, but the proximal interphalangeal and metacarpophalangeal joints of the hand and the wrist are preferential sites, as well as the metatarsophalangeal joints of the foot, the knee joint and the joints of the shoulder, ankle and hip. Symmetry is the hallmark of joint involvement. The synovium of bursae and tendon sheaths can also be affected. It gives rise to pain and stiffness, usually greatest in the morning. There is a marked capsular pattern with a spastic end-feel. Warmth and tenderness can be palpated over the joint.

Conventional radiography remains the standard imaging technique for joint studies in patients with suspected RA. The first radiological signs are osteoporosis and joint space narrowing. Later chondral erosions and small bone erosions at the joint margin are seen. Marginal and central erosions follow in advanced stages. Fibrous ankylosis, joint deformities (subluxations and dislocations), fractures and fragmentations are typical findings of more advanced RA.[115–117] RA is best treated systemically; local intra-articular injections are used only as a secondary aid.

Sometimes the shoulder is the seat of a *reactive type of arthritis* in which the inflammation is caused by an infection but in which no bacterial or viral agent can be isolated from the synovial fluid.[118]

Ankylosing spondylitis rarely starts in the peripheral joints but cases have been described with initial localization at shoulder or hip.[119,120] Particularly in the paediatric form of the disease (juvenile ankylosing spondylitis), peripheral joint involvement is more frequent and can precede, by many years, the onset of back features.[121] In its later course, signs and symptoms will be more localized in the spine and the sacroiliac joints. Arthritis at the shoulder from this disorder responds well to intra-articular steroids. The pain disappears fully but very often movement remains limited.

Reiter's disease seldom afflicts the shoulder joint. It is usually polyarticular in nature. Classically the triad urethritis–arthritis–conjunctivitis is present. Arthritis as a manifestation of psoriasis or lupus responds well to steroids, although a slight limitation of movement may remain.

Haemarthrosis

A patient complaining of severe pain immediately after an injury and showing a capsular pattern should always be suspected of having a haemarthrosis. In haemophilia, the haemarthrosis can develop spontaneously. It is more common at knee, elbow and ankle joints than in the shoulder.[122] Blood is very irritant to articular cartilage and so should be aspirated at once. If it is not, it will lead to full destruction of the joint over a the course of a few years.[123]

Crystal synovitis

Crystal synovitis at the shoulder from urate crystals (gout) is very rare.[124] This disorder should be considered when a capsular pattern comes on spontaneously in a few hours. It normally remains monoarticular but has often been preceded by earlier attacks in smaller joints (particularly in the metatarsophalangeal joint of the big toe). It disappears spontaneously in the course of a week and responds very well to colchicines or phenylbutazone. Diagnosis is mainly based on the presence of urate crystals in the synovial fluid.[125]

Pseudogout is the result of the presence of pyrophosphate crystals in the joint. The term 'chondrocalcinosis' is used if calcification in the hyaline cartilage of the joint is visible on radiography.[126] The knee is much more commonly affected than the shoulder.[127] Clinically, the presentation is spontaneous but is less acute in onset than gout. Crystals are also present in the synovial fluid and can be detected by high-resolution sonography.[128] Pseudogout resolves spontaneously in about 3–4 weeks.

Septic arthritis

Septic non-tuberculous arthritis

Septic arthritis can be provoked by direct inoculation of a bacterium into the joint, by haematogenous dissemination or from adjacent osteomyelitis. It is mainly seen in elderly people, often in connection with other predisposing factors such as diabetes, immune deficiency, malnutrition and alcoholism.[129,130] Some cases occur after mastectomy and radiotherapy for breast cancer.[131] A joint affected by a chronic arthritis, such as rheumatoid arthritis, is more likely to develop septic arthritis. It rarely occurs in the healthy elderly or in young adults. In children it may be a sequel to adjacent osteomyelitis. Acute septic arthritis may also present as an iatrogenic infection following joint arthroscopy, joint aspiration or local corticosteroid joint injection.[132–134] In many cases *Staphylococcus aureus* is the causative agent.[135] Sometimes a streptococcus or *Escherichia coli* is present, and even a gonococcal infection may be found.

The history is that of an acute and very painful shoulder, which after a few days becomes warm and red. A previous injection is sometimes mentioned. Usually the patient is very ill with high temperature, nausea and toxaemia. In rare cases, fever may be absent.

Inspection may show a swelling, which is often due to a subcutaneous abscess that communicates with the joint. On testing the shoulder, a very pronounced capsular pattern is found. In the initial stage, radiology is diagnostically irrelevant. As the condition develops further, periarticular osteoporosis, diminution of the joint space and finally joint destruction are found. Biological features, such as raised erythrocyte sedimentation rate and increased leukocyte counts, are suggestive but not confirmative. A diagnostic (and evacuating) aspiration of the joint, with a wide-bore needle (>20 gauge), usually shows over 100 000 leukocytes/mm^3, more than 90% being of the polymorphonuclear type. Sometimes the bacterial agent can be isolated.[136]

Treatment and prognosis

Septic arthritis of the shoulder is more difficult to treat than septic arthritis at any other joint. It is a very severe disorder and death is not uncommon.[137] The condition is normally managed with systemic antibiotics and daily evacuation of the pus. Also the combination of arthroscopic irrigation debridement and systemic antibiotic therapy is often used.[138] Sometimes open surgical drainage is necessary.

The erythrocyte sedimentation rate is a useful monitor of adequate treatment.[139] Very often the long-term result is significant limitation of movement at the glenohumeral joint because of bone destruction.

Tuberculosis of the glenohumeral joint

In tuberculosis of the shoulder joint, the clinical picture is far less pronounced than in septic arthritis and is slower in progression. Clinical diagnosis can be very difficult and is made only late in the course of the disease. A capsular pattern is found, often in association with severe muscle atrophy. Aspiration of the joint, followed by direct microscopic examination and culture, together with radiology (severe osteoporosis, narrowing of the joint space and erosions), are of help in diagnosis. The treatment is the same as for septic arthritis but specific antitubercular agents are administered.

Primary tumours

Primary tumours at the shoulder are mainly encountered in the young and may occur in acute leukaemia[140] or be due to sarcoma.[141] The tumour often presents insidiously. In the beginning it is characterized by localized, non-mechanical pain. From the moment that the tumour incites a synovial response, a painful capsular pattern at the shoulder will gradually develop.[142,143] In a younger patient, this should always arouse the suspicion of a primary tumour. Even the slightest limitation in a young patient is a formal indication for further careful exploration of this area by techniques such as radiography, computed tomography (CT) or MRI.

Warning

Spontaneously developing limitation of shoulder movement in a young patient should prompt suspicion of a primary tumour.

Metastases

Metastases can be localized either in the humeral head or at the glenoid. Rapidly increasing pain around the shoulder, radiating into the arm, and increasingly restricted shoulder movements in a patient with deteriorating general health are strongly suggestive of a secondary neoplasm. Sometimes a previous operation for a primary tumour is mentioned.

Localized warmth is usually the first sign, later followed by a very pronounced capsular pattern, with much pain and

limitation because both joint and muscles are affected.[144] Moreover, the resisted movements are extremely weak and painful. Visible muscular wasting is present. A radiograph or a bone scan can help confirm the diagnosis.

Warning

A gross capsular pattern, together with painful muscular weakness and wasting coming on over a short period of time, should prompt suspicion of metastases.

Aseptic necrosis

The humeral head remains the second most common site of osteonecrosis after the femoral head. While similarities in aetiology and pathogenesis exist, the anatomy and function of the glenohumeral joint are vastly different, which, consequently, results in delayed diagnosis and treatment. First of all, the glenohumeral joint is not exposed to the same weight-bearing forces as the hip joint. Additionally, the glenohumeral articulation is less conforming than the hip joint, and restricted shoulder motion can be compensated by surrounding joints. This allows for maintenance of shoulder function, even with advanced disease. Finally, the proximal humerus has an extensive anastomotic arterial supply, mitigating the effect of a loss of any single arterial inflow.

As in the hip, osteonecrosis of the shoulder results from disruption of the osseous arterial inflow or the venous outflow. The cause is either traumatic (fractures of the proximal humerus have been associated with an osteonecrosis rate ranging from 15 to 30%),[145] non-traumatic or idiopathic. Non-traumatic cases may be the result of haemoglobinopathies,[146] radiation of the joint or diving accidents.[147] High doses of systemic corticosteroids represent the most commonly reported iatrogenic cause of osteonecrosis.[148,149]

Often, the initial signs and symptoms are subtle and may include vague diffuse shoulder pain and difficulty sleeping. This pain deteriorates with disease progression and may be tolerable until the later stages. Determining the presence of risk factors for osteonecrosis, including prior steroid exposure, other medical conditions or alcohol abuse, may sometimes provide the only clue to the disease. Also, the patient's age at presentation may offer a hint, as these patients are generally younger than those with primary osteoarthritis.[150] The clinical picture, particularly early in the disease process, may be that of a slight capsular pattern. Also symptoms of locking, popping or a painful click may indicate the presence of loose osteochondral fragments.[151] With disease progression, significant limitations in motion of a non-capsular type – secondary to joint incongruity – and an increase in pain will become more evident.

Technetium bone scanning and MRI can detect aseptic necrosis in the early stage.[152,153] Later on, the whole joint is destroyed and the disease can be visualized on plain radiography.

Treatment consists of core decompression in the early cases.[154,155] In very severe cases, shoulder arthroplasty may be indicated.[156]

Osteoarthrosis

A number of different processes can destroy the glenohumeral joint surface. If no apparent reason for the development of osteoarthrosis can be found, it is termed *primary degenerative joint disease*. This is characterized by a triad of anterior capsular contracture, posterior wear of glenoid and subchondral bone, and posterior humeral subluxation.[157] Primary arthrosis does not usually evoke much pain. Indeed, a patient with an arthrotic shoulder, in the absence of any capsular inflammation, complains merely of painless crepitus on movement. During and after exertion there may be a vague ache, which usually disappears after a few hours. There is a capsular pattern with a hard but almost painless end-feel. With shoulder movement, crepitus may be detected on palpation. However, an osteoarthrotic joint is much more liable to develop arthritis, which can be the result of only a slight injury or some unusual activity. Once arthritis has set in, the limited movements also become painful. The diagnosis of primary arthrosis at the shoulder should be made on clinical grounds and should not be based solely on the radiograph, as it is quite possible to have no arthrosis on clinical examination but signs of it present on the radiograph.

In contrast, *secondary degenerative joint disease* may be much more painful and disabling. It occurs when previous injury, surgery or another condition affects the joint surface and causes degeneration. Chronic glenohumeral subluxations often lead to severe osteoarthrosis. The condition also develops when a chronic and massive tear of the rotator cuff subjects the uncovered humeral articular cartilage to compression against the undersurface of the coracoacromial arch. The resulting arthrosis is then called 'cuff arthropathy'.[158] In this case, clinical examination will reveal total rupture of the supraspinatus in combination with limited movement in a capsular way. Also, in the end-stage of avascular necrosis of the shoulder, the irregular head destroys glenoid articular cartilage, which results in secondary degenerative joint disease.[159]

Treatment

For the primary arthrosis, not very much need be done. Limitation of movement cannot be altered either by capsular mobilization or by intra-articular injections. If a traumatic arthritis or an immobilizational arthritis supervenes, intra-articular injections have no effect. The only remaining treatment is capsular stretching, which can only be executed if the arthritis is in stage I or stage II.

In secondary degenerative joint disease of the shoulder, considerable pain and functional impairment can result, for which there is no option other than surgery.

Neuropathic destructive arthropathy

Neuropathic osteoarthropathy, also known as Charcot neuroarthropathy, is a chronic, degenerative arthropathy and is associated with decreased sensory innervation.[160] There are numerous causes of neuropathic osteoarthropathy, the three most common being diabetes, syphilis and syringomyelia. Diabetic patients tend to have involvement of the joints of the

foot and ankle, whereas larger joints such as the knee are commonly affected in patients with syphilis. Patients with syringomyelia tend toward involvement of the shoulder and elbow.[161] Syringomyelia is a disorder involving a fluid-containing cavity (syrinx) within the spinal cord. These cavities commonly occur in the lower cervical and upper thoracic segments, and the distension may propagate proximally. Syringomyelia may have congenital, traumatic, infectious, degenerative, vascular or tumour-related causes.[162,163] The joint and the subchondral bone are destroyed because of the loss of the trophic and protective effects of its nerve supply. In neuropathic destructive arthropathy, a gross but painless capsular pattern with a very hard bone-to-bone end-feel is found. The complete clinical picture is slow to develop. By the time the painless capsular pattern and bony end-feel are found, the underlying condition is usually already known from other neurological signs, such as muscular weakness and atrophy in the upper limbs occurring over a short period. Radiography provides the key to the diagnosis.[164]

Table 14.3 summarizes shoulder lesions that present with a capsular pattern.

Table 14.3 Shoulder lesions presenting with a capsular pattern

Type	Disorder	Signs/symptoms
Monoarticular arthritis		
Acute onset	Gout	Urate crystals in aspirate
	Pseudogout	Calcium pyrophosphate crystals Radiographic signs
	Septic arthritis	Severely ill Shoulder very painful, warm, red and swollen
	Haemarthrosis	Haemophilia Injury
Slow onset	Traumatic arthritis	Patients over 40 Trauma
	Immobilization arthritis	Patients over 60 Immobilization of the arm
	Monoarticular steroid-sensitive arthritis	Spontaneous onset Negative blood tests
	Osteoarthrosis	Hard end-feel Painless crepitus
	Shoulder–hand syndrome	Hand bluish and diffusely swollen Distal dystrophy and stiffness
	Neuropathic destructive arthropathies	Painless, sometimes bilateral Bony-block end-feel Neurological signs
	Metastases	Local warmth Muscular wasting Extreme pain and weakness on all resisted movements
	Primary tumour	Young person developing a painful stiff shoulder in a short period of time
	Aseptic necrosis	Moderate or severe pain Only a few signs
Polyarticular arthritis		
Symmetrical distribution	Rheumatoid arthritis	
	Arthritis due to systemic lupus erythematosus	
Asymmetrical distribution	Ankylosing spondylitis	
Unspecified distribution	Psoriatic arthritis	

Non-capsular patterns

Three main groups are distinguished: limitation of active elevation, limitation of passive lateral rotation and limitation of passive medial rotation.

Limitation of active elevation

To recall: the term 'elevation' is used to indicate the upward movement of the arm in a sagittal plane. Full elevation assumes a normal range of movement of the shoulder girdle and a normal range of 'abduction' at the glenohumeral joint. The latter is the upward movement in a sagittal plane of the humerus in relation to a fixed scapula.

A limitation of active elevation may result from either inert or contractile structures of both shoulder and shoulder girdle. If there is a full range of passive movement, together with pain and/or weakness during resisted movements of neck, shoulder girdle or humerus, the lesion belongs to a contractile structure. If both active and passive elevation is limited, the problem must lie in the inert structures. Passive scapulohumeral abduction will then differentiate between a lesion of the shoulder joint and a disorder that causes limitation of scapular movement (Fig. 14.10).

Both passive elevation and passive scapulohumeral abduction limited

Acute subdeltoid bursitis

This is one of the most painful disorders in orthopaedic medicine. It has a swift onset and, untreated, recovers spontaneously in about 6 weeks. According to Cyriax,[1] there is some tendency for recurrence within 5 years at one or both shoulders.

For no apparent reason, pain starts and increases progressively to reach a maximum in about 3 days (Fig. 14.11), by which time it is very severe and may radiate throughout the entire C5 dermatome. The slightest movement of the shoulder is unbearable and, even if the arm is kept totally immobile, the pain is very pronounced, leading to sleepless nights.

On presentation, the patient typically supports the arm with the other hand. Lack of sleep and the severe suffering can show clearly on the patient's face. The pain can be so excruciating that the patient refuses to move the elbow away from the body.

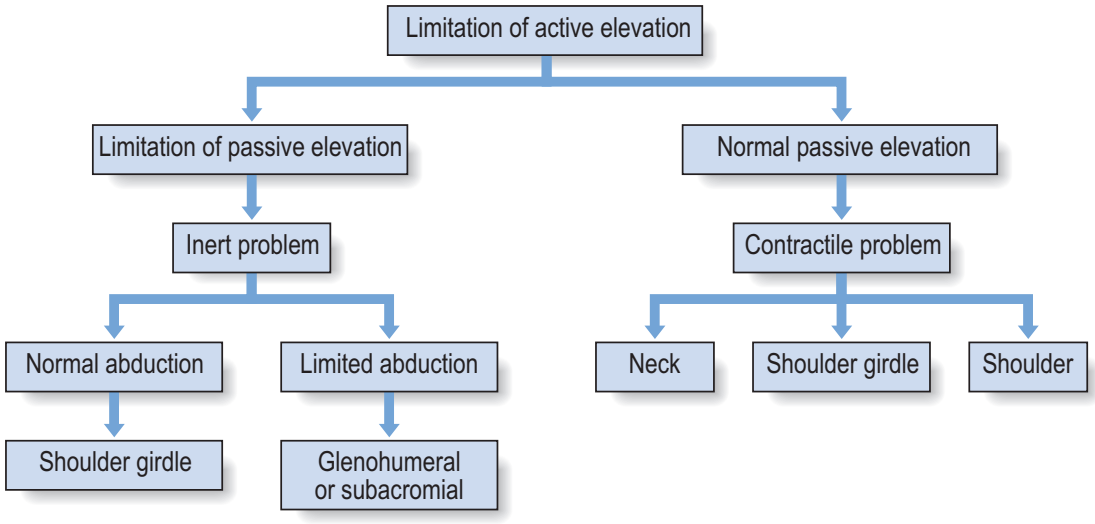


Fig 14.10 • Differential diagnosis of limitation of active elevation of the shoulder.

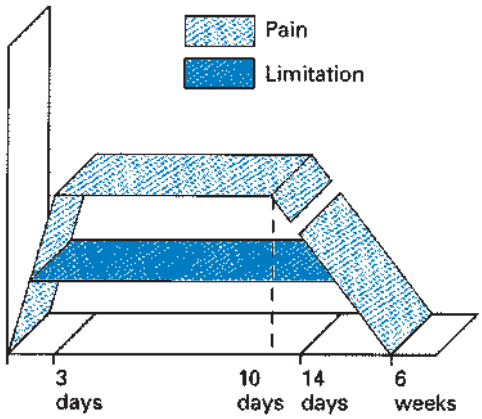


Fig 14.11 • Natural history of acute subdeltoid bursitis.

Active and passive elevation are therefore hardly possible and an 'empty end-feel' is found on passive elevation; the movement is stopped by the patient, who begs the examiner not to proceed, despite being possible as tissue resistance is not encountered. Marked limitation of passive scapulohumeral abduction is also present.

Other passive movements are also painful, sometimes only slightly limited but in a clearly non-capsular way; in acute subdeltoid bursitis, passive elevation and abduction are most restricted, whereas in arthritis it is passive lateral rotation that is most reduced. It is quite natural in such a painful disorder for some resisted movements, such as abduction and lateral rotation, to be painful too.

If the patient presents at the very beginning (first day after the onset), a painful arc on elevation may be found. However, once the inflammation has become severe, this disappears because it is no longer possible to go beyond the point of painful impingement. After 7–10 days of severe pain, it starts progressively to wear off so that at the end of 3–4 weeks only an ache remains. At this point, active elevation is still limited to about half-range. After 4–6 weeks the pain has disappeared totally and range of movement has returned to normal. During the last week, when full recovery is almost complete, a painful arc may reappear.

The clinical examination is followed by palpation of the superficial part of the bursa, which is very tender and sometimes even swollen. It should be noted that in acute bursitis not only the palpable (subdeltoid part) but also the subacromial bursa are involved.

Differential diagnosis

The clinical presence of this type of bursitis, in which a striking non-capsular pattern emerges, is so typical that differential diagnosis should not offer much in the way of difficulty. The only condition that has similar symptoms and signs is a septic bursitis. Subacromial septic bursitis also presents with acute shoulder pain and a gross non-capsular pattern of limitation. Signs that arouse suspicion are: swelling, redness, warmth and systemic signs such as fever and an elevated white blood cell count.[165] Septic bursitis should be suspected in patients with disease states associated with immunosuppression, patients with systemic infection, and patients who have received a corticosteroid injection.[166]

Other conditions with a swift onset can also be taken into consideration:

- Gouty arthritis: this has a very abrupt onset but lasts only 3–7 days. On clinical examination a capsular pattern is found.
- Septic arthritis: although very similar to acute bursitis – swift onset and very painful – this is easily differentiated by the accompanying symptoms of fever, general illness and local warmth and by the presence of a pronounced capsular pattern.
- Pathological fracture: mainly as a result of metastases, a pathological fracture is usually preceded by severe pain of weeks' or months' duration, together with limitation of capsular movement. Severe exacerbation of pain comes on immediately when fracture occurs.
- Dislocation of the shoulder: a history of an injury or of previous attacks is present. Deformation on inspection and radiography provides the key.

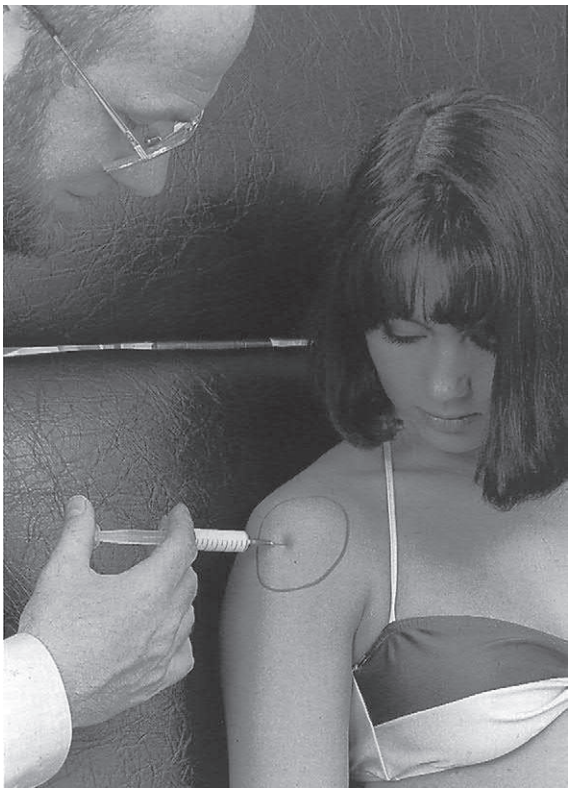


Fig 14.12 • Infiltration of the superficial part of the subdeltoid bursa.

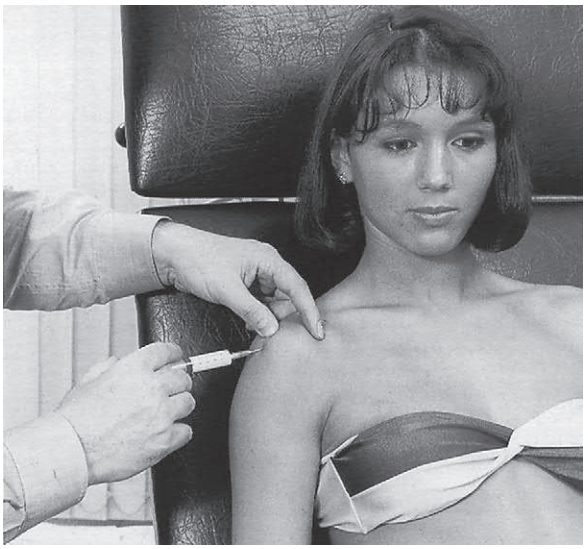


Fig 14.13 • Infiltration of the deep part of the subdeltoid bursa.

Treatment

During the first 10 days after onset, infiltration of the entire bursa with triamcinolone is most helpful. If done correctly, this is one of the most successful treatments in orthopaedic medicine; in almost all patients, full cure is achieved in less than 5 days. In patients who have not presented within 10 days or those who refuse steroids, a 'figure-of-eight' bandage at night, fixing the arm to the body, avoids any involuntary movement during sleep. In the day, the arm is carried in a sling for as long as the pain so warrants.

Giving an infiltration into such a severely inflamed bursa is extremely painful unless a strong local anaesthetic is added. Two syringes are used: one with a needle of about 3 cm, used for the superficial part of the bursa, and one with a 5 cm needle, used for the deep part of the bursa. Both syringes contain 1 mL of triamcinolone, 40 mg/mL mixed with 4 mL of prilocaine 2%. The technique used is the same as for chronic subdeltoid bursitis, but in acute bursitis, both subacromial and subdeltoid parts of the bursa must be treated (Figs 14.12 and 14.13). Gentle palpation of the entire subdeltoid part determines the area of tenderness, which is usually much larger than in chronic subdeltoid bursitis. The subdeltoid area is mapped and infiltrated thoroughly. The second infiltration is then given via a lateral approach under the acromial arch. Special care must be taken to deposit some of the product all over the tender part of the subdeltoid bursa and the entire subacromial bursa.

Immediately after the infiltrations, the pain is somewhat relieved and thereafter the patient improves gradually with time. Usually after-pain is not felt.

The patient should rest the arm and return 2 days later to be re-examined. If some pain still remains and elevation is not full and painless by then, some part of the bursa must have been missed. The remaining inflammation – either at the subdeltoid or the subacromial section of the bursa – is again localized and thoroughly infiltrated. Maximally, half the amount if infiltrate used at the first attempt is now used. Recurrences are not usually seen.

Special cases

There are two special types of subdeltoid bursitis:

- *Acute bursitis with calcified deposit*: The clinical picture is almost the same as in ordinary acute bursitis but spontaneous recovery does not take so long, although the tendency for recurrence is higher.[167] The treatment is the same. In order to diminish the tendency of recurrence, it is worth trying to dissolve the calcification by repeated infiltrations of procaine, after the acute attack has been dealt with. For this purpose, 5 mL of procaine 2% is infiltrated at weekly intervals for 3 or 4 weeks, each time infiltrating the part of the bursa where the calcification lies (Fig. 14.14).[168,169]
- *Haemorrhagic subdeltoid bursitis*: This occurs only in elderly patients. It usually comes on spontaneously or may accompany a tendinous rupture. The patient complains of moderate pain and swelling. On clinical examination, some limitation of movement is found which is mainly the result of fluid and not of pain. As in acute subdeltoid bursitis, a non-capsular pattern is present. On palpation, pain can be elicited and fluctuation detected. Treatment consists of repeated aspirations at weekly intervals. Infiltrations are not required. If blood keeps reaccumulating, a haemangioma has to be considered.

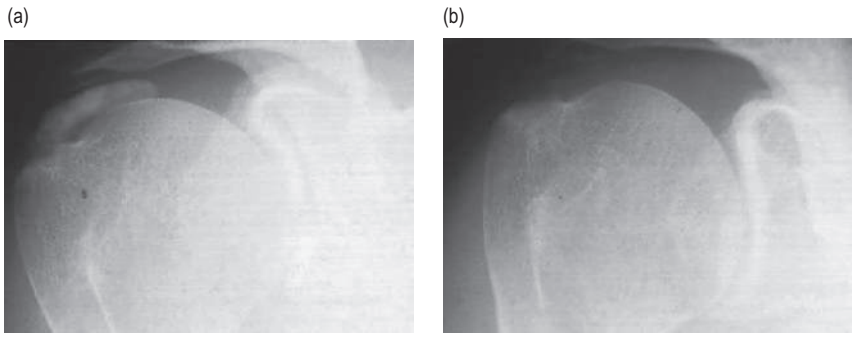


Fig 14.14 • Acute bursitis with calcified deposit before (a) and after (b) treatment.

Psychogenic limitation

Mental problems may sometimes be expressed in terms of physical behaviour. It should not be a surprise that psychogenic symptoms are often localized at the shoulder because the shoulder joint is closely connected with emotional tone: the outstretched arm is a symbol of pleasure and welcome; the arm held into the side expresses rejection.

The diagnosis 'functional' limitation of elevation is quite simple to make: there is a marked limitation of both active and passive elevation, the end-feel being that of an active, voluntary muscular contraction. However, active and passive elevation of the shoulder girdle (shrugging of the shoulders) is completely normal and the passive, scapulohumeral abduction is as limited as is passive elevation of the arm.

The patient does not realize that, even if the shoulder joint is ankylosed, mobility of the scapula permits 60° tilting and the arm must be capable of this amount of elevation unless the scapula has also become fixed. Hence, detection of a non-physical problem is simple if, in a patient with normal scapular mobility and normal elasticity of the pectoralis major muscle (see below), the range of voluntary and passive elevation is contrasted with the range of passive abduction at the scapulohumeral joint.

Passive elevation limited and passive scapulohumeral abduction normal

This indicates that the glenohumeral joint is intact but that the disorder is due to a malfunction of structures belonging to the shoulder girdle. These lesions are discussed more extensively in the online chapter *Interpretation of the clinical examination of the shoulder girdle*.

Contracture after breast surgery

Breast-conserving therapy has become a safe option for the primary surgical treatment of early breast cancer. Nevertheless, there are cases where this type of treatment is impossible. In these cases, mastectomy followed by immediate reconstruction is one type of alternative treatment. Another step in breast cancer surgery is management of the axilla, which may involve complete axillary lymph node dissection. After such a major operation, a limitation of about 30–60° on both active and passive elevation of the arm can occur, from loss of elasticity of the pectoralis minor muscle as a consequence of scarring tissues.[170,171] The scapula–humeral range remains normal. Pain is absent.

Pulmonary tumour

An apical tumour of the lung (Pancoast tumour) is often initially misdiagnosed as a shoulder problem.[172,173] Tumours of the base of the lung may also give rise to pain referred to the base of the shoulder (C4) in that they may irritate the diaphragm. Once a neoplasm of the lung involves the thoracic cage, muscle spasm of the pectorales muscles ensues and causes a limitation of both passive and active elevation: the arm cannot be raised beyond the horizontal. Trying to go any further is very painful and is stopped by muscular spasm. Pain and/or limitation are not found at the glenohumeral joint and the scapula is fully mobile. In addition, there is pain on resisted adduction and medial rotation of the arm. Care should be taken not to misinterpret these signs as being a psychogenic limitation.

Warning

The end-feel of muscle spasm always means that a serious disorder is present and should alert the examiner. A plain radiograph of the lungs must be taken at once.

Contracture of the costocoracoid fascia

This disorder often causes diagnostic difficulties. Initially there is only pectoroscapular pain on full elevation of the arm. Later on, when the pain becomes more or less constant, a slight limitation of about 5–10% of elevation can be detected. Passive movement at the glenohumeral joint is normal. The possibility of a shortened costocoracoid fascia is brought to mind when a small but painful limitation of active and passive scapular elevation is detected but resisted movements of the scapula prove painless. Forward movement of the scapula is slightly painful; backward movement is negative.

A shortening of the costocoracoid fascia may be caused by a neoplastic invasion, healed apical tuberculosis or an injury. The contracture may also develop without apparent reason. It should be differentiated from other subclavicular disorders, such as a lesion of the subclavius muscle, sprain of the conoid and trapezoid ligaments, or subcoracoid bursitis (see online chapter *Interpretation of the clinical examination of the shoulder girdle*).

Ankylosed acromioclavicular or sternoclavicular joint

This is encountered in advanced cases of ankylosing spondylitis, in arthrosis or in rheumatoid arthritis. It leads to a severe

limitation of elevation of the arm, which cannot be raised actively or passively beyond the horizontal because scapular rotation is limited. Clinical examination reveals full scapulohumeral abduction but total absence of scapular elevation and rotation.

Active elevation limited and passive elevation normal

Fracture of the first rib

A stress fracture rather than trauma is usually the cause. In that a fractured rib heals spontaneously in about 2 months, the condition is considered only with pain of recent onset. The patient complains of unilateral pain located under the scapula, behind the clavicle or at the root of the neck.[174] The lesion is characterized by neck, scapular and arm signs. Typically, both active and passive side flexion of the neck to the painless side increase the pain because this pulls on the fractured rib via the scalene muscles. As a consequence, resisted side flexion towards the pain is also painful. All scapular movements – active, passive and resisted – are also more or less painful. When the patient is asked to raise the arm actively beyond the horizontal, he or she cannot do so, whereas passive elevation is of full range but only slightly painful.[175] Radiography confirms the diagnosis.

Clay-shoveller's fracture

This rare condition is a traction fracture of a spinous process in the lower cervical or upper thoracic area.[176,177] At this level, the trapezius, rhomboid and posterior serratus superior muscles are attached to the spinous processes. A fracture is usually the result of heavy work. Less commonly it is encountered in athletes. The patient normally feels a sudden sharp pain followed by local tenderness. Although neck movements are almost painless, the patient can hardly move either arm actively into slight elevation. A very pronounced limitation of active elevation of about 150° is found but passive elevation remains normal. Both active scapular elevation and resisted scapular elevation are also painful. There is local tenderness and, on the radiograph, avulsion of the seventh cervical or first thoracic spinous process is seen. The lesion heals spontaneously in 5–6 weeks. Immobilization is unnecessary.

Long thoracic nerve palsy

A lesion of the long thoracic nerve resulting in a palsy of the serratus anterior muscle may follow local invasive procedures on the anterolateral aspect of the thorax,[178] local trauma or a traction injury to the nerve.[179,180] However, in the majority of cases no cause can be established. The usual onset is with unilateral scapular pain which continues day and night for about 3 weeks. During this period the arm becomes weak and heavy. In rare cases pain is totally absent, the patient complaining only of fatigue of the arm.[181]

The clinical examination is more specific and provides the key to the exact diagnosis. On inspection, winging of the scapula may be present. Clinical examination shows a painless limitation of active arm elevation of about 45–90°.[182] Passive movements are of full range. Neck, scapular or arm movements have no influence on the pain. Weakness is present within

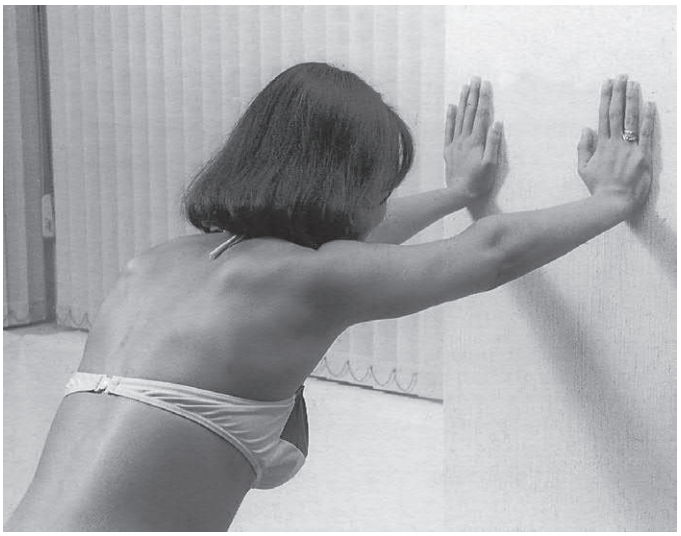


Fig 14.15 • Test for mononeuritis of the long thoracic nerve.

the serratus anterior muscle and can easily be detected by the following test: the patient is asked to push against a wall with the arms stretched out horizontally in front of the body (Fig. 14.15). This pushes the medial border of the scapula further away from the thoracic cage when the movement is not countered by a contraction of the serratus anterior muscle. The abnormal movement is seen as a considerable winging of the scapula.

The natural history is spontaneous recovery from the pain in about 3 weeks, and full recovery of normal muscle function usually occurs in an average of 9 months.[183]

Accessory nerve palsy

The spinal accessory nerve is the sole motor nerve of the trapezius. A palsy may result from a crush injury to the nerve[184,185] after a forceful blow to the neck. Often the lesion is posttraumatic, after cervical lymph node biopsy or other surgical procedures.[186,187] Sometimes no clear cause for the neuritis can be detected and in such a case the diagnosis may be quite difficult.

There is a severe and continuous unilateral scapular ache with spontaneous onset, lasting for about 3 weeks. The patient then starts to complain of weakness in the arm, which may last for months.

Inspection elicits an asymmetrical neckline with drooping of the affected shoulder. This may be accompanied with slight lateral displacement and winging of the scapula.[188] Elevation is between 15° and 30° limited. Winging is accentuated during arm elevation and disappears during forward flexion of the arm due to the action of the serratus anterior muscle.[189] Again, passive elevation is full-range and painless. Passive and resisted movements of the shoulder are completely normal. During resisted external rotation, winging of the scapula once again becomes visible.[190]

The diagnosis can be confirmed by testing the strength of the trapezius muscle. The patient is asked to pull both scapulae together while counterpressure is given at the medial side of

Fig 14.16 • Test for mononeuritis of the accessory nerve.

the inferior angle (Fig. 14.16). In accessory nerve neuritis, the scapula can be pushed away easily.

In an idiopathic mononeuritis the pain disappears after about 3 weeks but spontaneous recovery of motor function may take about 4–8 months.[191]

C5 full root palsy

This is usually the result of slowly but progressively increasing compression of the C5 nerve root by an osteophyte in the fourth intervertebral foramen. It may finally result in a painless inability to raise the arm actively because of pronounced weakness of the supraspinatus and the deltoid muscles. Other C5 muscles, such as the infraspinatus and the biceps, are, of course, also weak. A C5 palsy may also result from a traction injury, which is usually caused by a sudden depression of the entire shoulder girdle, in combination with a simultaneous and forceful side flexion of the neck in the opposite direction (see online chapter *Nerve lesions and entrapment neuropathies of the upper limb*).

C7 full root palsy

A severe C7 palsy may cause weakness of shoulder adduction. Often, a slight limitation of active elevation is also noticed. The diagnosis is obvious when a palsy of the triceps and/or the flexors of the wrist is also found.

Total rupture of the supraspinatus

In full rupture of the supraspinatus tendon the patient is unable to elevate the arm actively. Passive elevation is of full range with a severe painful arc. Resisted abduction is weak and painless (see p. 257).

Limitation of passive lateral rotation

Only a few lesions cause an isolated limitation of lateral rotation of the arm. History, end-feel and accessory tests differentiate between an anterior capsular contracture and a subcoracoid bursitis.

Anterior capsular contracture

An isolated contracture of the anterior capsule results from trauma or develops gradually after rupture of the infraspinatus tendon.

Previous shoulder injury such as subluxation is one cause. Usually the whole joint suffers from the traumatic impact and a traumatic arthritis results. Exceptionally, only the anterior portion of the capsule bears the impact of the injury. A localized synovitis and subsequent capsular contracture follow.

An anterior capsular contracture may also result from complete rupture of the infraspinatus tendon. Because the teres minor muscle is a weak and incomplete lateral rotator, it does not, on its own, bring the arm in full lateral rotation, which finally leads to a loss of the normal elasticity of the anterior portion of the capsule.

The condition is characterized by a painful limitation of passive lateral rotation together with an abnormal end-feel. In the beginning the end-feel is that of muscle spasm; later, it changes to typically hard.

Treatment

It is important for all patients with a rupture of the infraspinatus tendon to exercise on a regular basis to keep the shoulder mobile, as a means of prevention. This can be achieved through sporting activities or through passive mobilizations which bring the shoulder into full lateral rotation. Patients who have already developed a contracture of the anterior capsule are best treated by capsular stretching. This can be done in the same way as for shoulder arthritis but stretching in lateral rotation must also be included (Fig. 14.17).

Technique: capsular stretching in lateral rotation

The patient lies supine, the arm abducted to about 45°, and the elbow bent to 90°. The therapist stands at the affected side and takes the arm in both hands, just proximal to the elbow; the contralateral forearm pushes the patient's forearm down just above the wrist. This results in stretching of the anterior part of the joint capsule into lateral rotation. The amount of force used, the duration and the sequence are the same as for stretching in arthritis.

Subcoracoid bursitis

This rare condition comes on for no apparent reason and provokes unilateral pectoral pain. On clinical examination, a painful limitation of lateral rotation is the main finding. The limitation is due to passive stretching of the pectoralis major muscle over the inflamed bursa. If the lateral rotation is performed again, this time disregarding the patient's pain, the range of lateral rotation will be found to have increased. The limitation will even completely disappear during a lateral rotation with the upper arm abducted to the horizontal

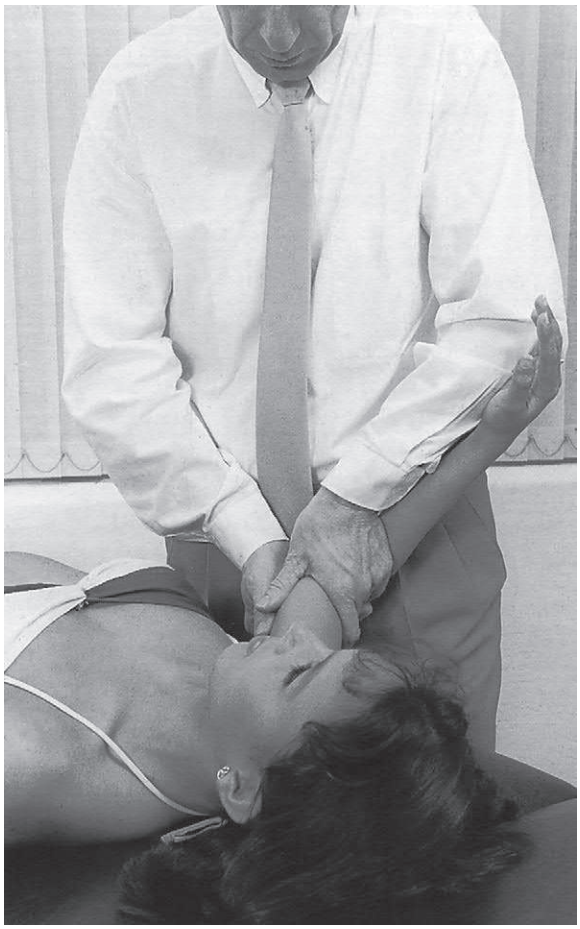


Fig 14.17 • Capsular stretching in lateral rotation.

Fig 14.18 • Passive horizontal lateral rotation.

(Fig. 14.18). Although still painful, it is no longer limited since, in this position, the pectoralis major compresses the inflamed bursa less.[192]

The main *differential diagnosis* is an anterior capsular contracture. Here, the limitation does not depend on the position of the upper arm and the movement always comes to a stop at the same point in the range. Another accessory test that differentiates between a capsular contracture and a subcoracoid bursitis is passive horizontal adduction in front of the chest, which pinches the subcoracoid bursa painfully between scapula and upper arm (see p. 217).

Other differential diagnoses that must be considered are subscapularis tendinitis, a lesion of the pectoralis major, sprain of the trapezoid–conoid ligament and early glenohumeral arthritis. In the former two conditions, passive lateral rotation, although painful, is not limited and resisted medial rotation causes pain. For a lesion of the pectoralis major, resisted adduction is also painful. A sprain of the trapezoid–conoid ligament does not give rise to limitation of movement; only pain at the extremes of all passive tests is present and passive horizontal adduction is painless.

At the very beginning of a glenohumeral arthritis (idiopathic or traumatic), it is possible to find only a slight painful limitation on passive lateral rotation, together with some pain on full passive elevation and medial rotation. The end-feel may be more or less normal. On passive lateral rotation with the arm abducted to the horizontal, the same limitation is found.

Treatment

The condition can be treated only by infiltration with steroid.

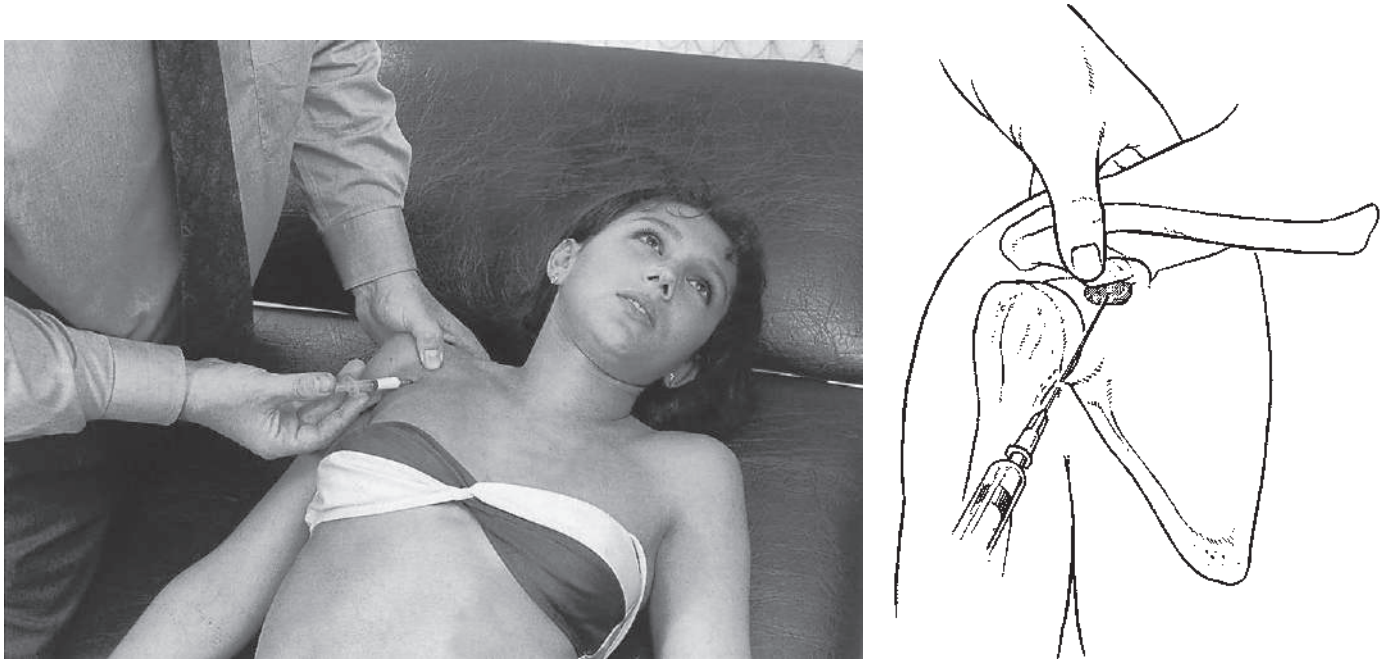
Technique

The patient is put in the half-lying position, and asked to adduct the scapula and shrug the shoulders. Adduction brings the coracoid process into prominence and shrugging the shoulders takes it away from the top of the lung. Next the tip of the coracoid process is palpated and a point chosen about 2 cm below it. A 5 cm needle is inserted here, pointing in a cranial–medial–dorsal direction and aiming at the base of the coracoid bone (Fig. 14.19a). After it hits the bone, it is withdrawn by about 1 cm and 2 mL of triamcinolone is infiltrated here over several withdrawals and reinsertions (Fig. 14.19b). The infiltration is repeated at weekly intervals until full relief is obtained. Three infiltrations usually suffice.

Limitation of passive medial rotation

Isolated limitation of the medial rotation is very rare. A discrete painful loss of internal rotation is sometimes seen in combination with a lesion of the upper/posterior rotator cuff (supraspinatus–infraspinatus). This limitation is most prominent if the internal rotation is performed in 90° of abduction and is thought to be caused by a thickened posterior capsule.[193] The limitation usually disappears spontaneously after the tendinous lesion has been healed.

A summary of the non-capsular limitation patterns at the shoulder is shown in Figure 14.20.



(a) (b)

Fig 14.19 • Infiltration of the subcoracoid bursa.

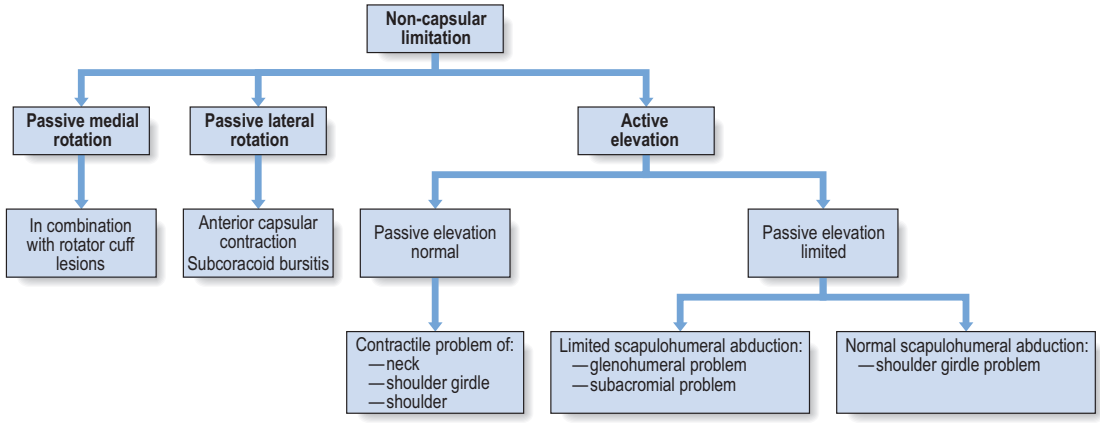


Fig 14.20 • Summary of the non-capsular limitation patterns at the shoulder.

Full range of movement

Disorders of inert structures may be characterized by a full range of passive movements and a normal end-feel. However, passive movements are painful at the end of range or at half-range (painful arc). Resisted movements from the basic functional examination are, of course, negative – painless and strong. Only a few potential lesions correspond to this pattern. They are: a sprain of the acromioclavicular ligaments, a sprain of the coracoclavicular ligaments and chronic subacromial bursitis.

Acromioclavicular sprain

The acromioclavicular joint is stabilized by capsular ligaments which reinforce the thin capsule all around, and by extracapsular (coracoclavicular) ligaments. Vertical stability of the joint is controlled mainly by the coracoclavicular ligaments, whereas horizontal stability is controlled by the acromioclavicular ligaments.[194]

An acromioclavicular injury is most commonly the result of a fall on to the point of the shoulder with the arm at the side, as often occurs in football, hockey, alpine skiing[195] or judo.[196,197] In this position, the acromion is driven medially and

downwards in relation to the distal end of the clavicle, the latter being fixed through interlocking of the sternoclavicular ligaments.[198] Less often the trauma is a fall on the elbow or on the outstretched arm. In this event, a cranial force is exerted on the acromion.

Finally, on occasion, the sprain is the result of too much tension on the acromioclavicular ligaments from overuse, as can be seen in swimmers, weightlifters and bodybuilders.[199] In an osteoarthrotic joint, ordinary daily work may cause acromioclavicular sprain.

Symptoms

Pain is felt at the shoulder during and after activity. Lying on the affected side is usually painful. In that the acromioclavicular joint is derived from the C4 segment, and the C4 dermatome is rather small, little referred pain is to be expected. As a rule, when the patient is asked to point out the exact site of the pain, he or she generally puts a finger right on the acromioclavicular joint. Exceptionally the pain spreads beyond the lateral acromial rim or upwards to the trapezius, which usually indicates involvement of the inferior capsular ligament. In this event, differentiation from a case of chronic subdeltoid bursitis may be difficult and often necessitates a diagnostic infiltration with local anaesthetic.

Functional examination

There is pain at full range on passive elevation and passive lateral and medial rotation. Limitation of movement is not found. Resisted movements are usually painless, although exceptionally pain may be present on resisted adduction or abduction as a result of transmitted stress on the acromioclavicular ligaments.

Logically one would expect all scapular movements to hurt as well, because they all put strain on the acromioclavicular joint. Strangely enough, these tests are mostly negative in that far less stress is put on the acromioclavicular joint during active and passive scapular movements than on using the arm as a lever on passive arm movements. Sometimes a painful arc is present, locating the lesion in the inferior acromioclavicular ligament.

With this type of clinical pattern, an accessory and useful differential diagnostic test should be done: passive horizontal adduction across the front of the chest (see p. 217). This is the most painful movement when the acromioclavicular joint is affected and may even be limited in a severe sprain of the posterior part of the acromioclavicular ligaments. After the diagnosis has been established clinically, the joint line is palpated for local tenderness. When palpation is painful, it establishes the superior ligament at the site of the lesion. The inferior ligament is obviously beyond the reach of the fingers and cannot be palpated but, as previously described, if it is involved, a painful arc is usually present. Sometimes there is a combined lesion of both superior and inferior ligaments. In this event, palpation of the joint line is positive and a painful arc is also found.

Most commonly, a sprain to the joint occurs without ligamentous damage and no displacement can be palpated. If the traumatic force had sufficient magnitude to disrupt the acromioclavicular ligaments, (sub)luxation of the acromioclavicular joint becomes visible. Acromioclavicular sprains and dislocations are classified on the integrity of the acromioclavicular and coracoclavicular ligaments. Classically there are three grades of acromioclavicular dislocation (Fig. 14.21).[200–202] Recently grades IV, V and VI acromioclavicular dislocations have been added to the classification system.[203] In type IV injuries, the clavicle is grossly displaced posteriorly into the trapezius muscle; type V is a severe vertical separation of the clavicle; and in type VI the clavicle is dislocated inferiorly into either a subacromial or a subcoracoid position.

Differential diagnosis

An uncomplicated (grade I) sprain of the acromioclavicular joint is sometimes difficult to differentiate from chronic subdeltoid bursitis and early glenohumeral arthritis.

In chronic subdeltoid bursitis, a very similar clinical picture (all passive movements are painful at full range, together with a painful arc) may be seen. The pain in bursitis is usually

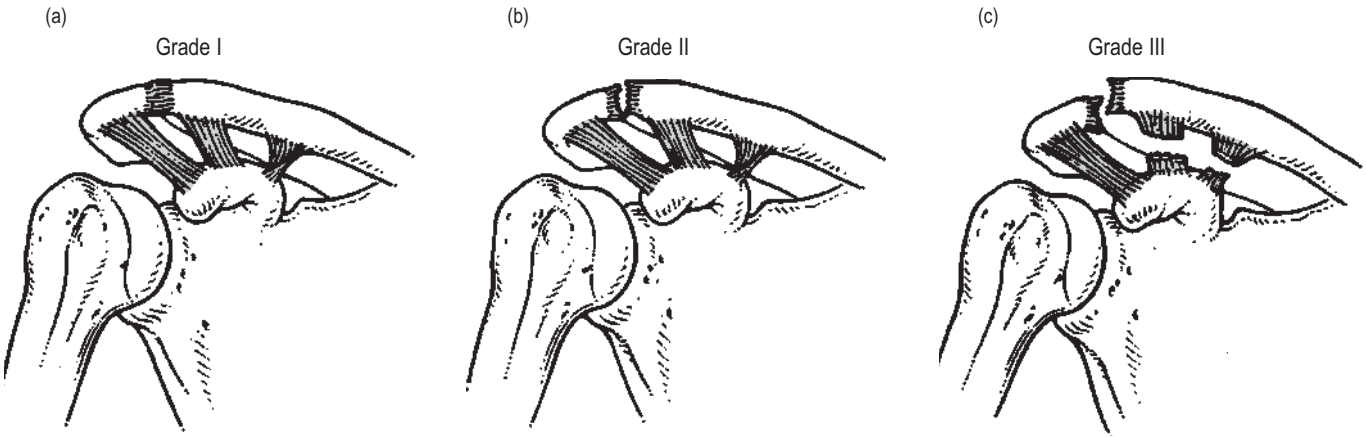


Fig 14.21 • Acromioclavicular joint lesions: (a) grade I: a small lesion of the acromioclavicular ligament without displacement; (b) grade II: rupture of the acromioclavicular ligament with cranial displacement of the clavicle, of less than half its width; (c) grade III: a a rupture of the coracoclavicular ligaments and a full dislocation of the clavicle.

felt in the deltoid area and spreads further down the arm in the C5 dermatome. There is usually no history of trauma. Painful passive horizontal adduction is less pronounced than in acromioclavicular sprain. If the superficial part of the bursa is at fault, palpation is positive; if the deep part is affected, certainty can only be obtained by a diagnostic infiltration of local anaesthetic.

The differential diagnosis from early arthritis of the shoulder is difficult when no notable limitation of movement is present. The patient complains of pain at the shoulder, radiating down the arm. On functional examination, pain is present at the end of all passive movements. The most painful movement is full passive lateral rotation, whereas in a sprain of the acromioclavicular joint it is passive horizontal adduction. In arthritis the end-feel on lateral rotation is slightly harder than normal.

Treatment

Sprain without displacement (grade I)

As adhesions do not form in a sprain of the acromioclavicular joint, it is best treated by relative rest. The only additional measure needed is to stop the inflammation. This can be achieved by deep friction (superior ligament) or by a local infiltration of steroid (superior and inferior ligaments). No matter how long the lesion has existed, the treatment remains the same.

Rest on its own is usually not enough. It gives the patient a false impression of healing. The pain disappears only temporarily until normal activity is restarted, when it recurs.

When sprain is the result of repeated stress on the acromioclavicular ligaments caused by specific activities, the lesion may recur. In this event the patient must avoid these activities in the future.

Technique: palpation

The patient sits with the arm in the neutral position and the back resting against the couch. The posterior angle of the scapular spine is palpated first. The finger is then brought to the lateral edge of the acromion and more medially on the flat upper surface of the acromion. A hard bony rim is felt, sticking out at approximately 2 cm medial to the lateral acromial border. This is the outer end of the clavicle, which is always slightly elevated. The joint line lies just lateral to it.

Palpation may be difficult in stout patients, or in elderly people in whom a rim of osteophytes at the acromial part of the acromioclavicular joint can be mistaken for the outer clavicular end. In these cases, some extra landmarks can be used. When the anterior edge of the acromion and clavicle is palpated, a depression is felt at the level of the acromioclavicular joint line. The anterior depression can be slightly opened if an assistant pulls the arm into full lateral rotation. If traction is applied to the arm in a distal direction, the palpating fingers on the acromioclavicular joint can usually feel the movement between clavicle and acromion.

Technique: infiltration of the superficial ligament

After exact delineation of the painful spot, a 2.5 cm needle is fitted to a syringe containing 1 mL of triamcinolone. If the deep ligament is also affected (painful arc), 2 mL is used.

The patient sits in the same position as previously described for palpation. The needle is obliquely inserted at the centre of the painful area (Fig. 14.22a). The aim is to infiltrate the whole of the tender area, at both sides of the joint line, via a series of partial withdrawals and reinsertions of the needle while depositing a few droplets into the ligament each time. A typical ligamentous resistance is encountered. It should be noted that the ligament lies superficial to the osseous structures and therefore the needle should not be inserted deeply but must have its tip in bony contact.

Technique: infiltration of the deep ligament

The same type of needle and amount of steroid are used. After the joint line has been identified, the needle is inserted from above, halfway between the anterior and posterior margins of the acromioclavicular joint. It may be useful to have an

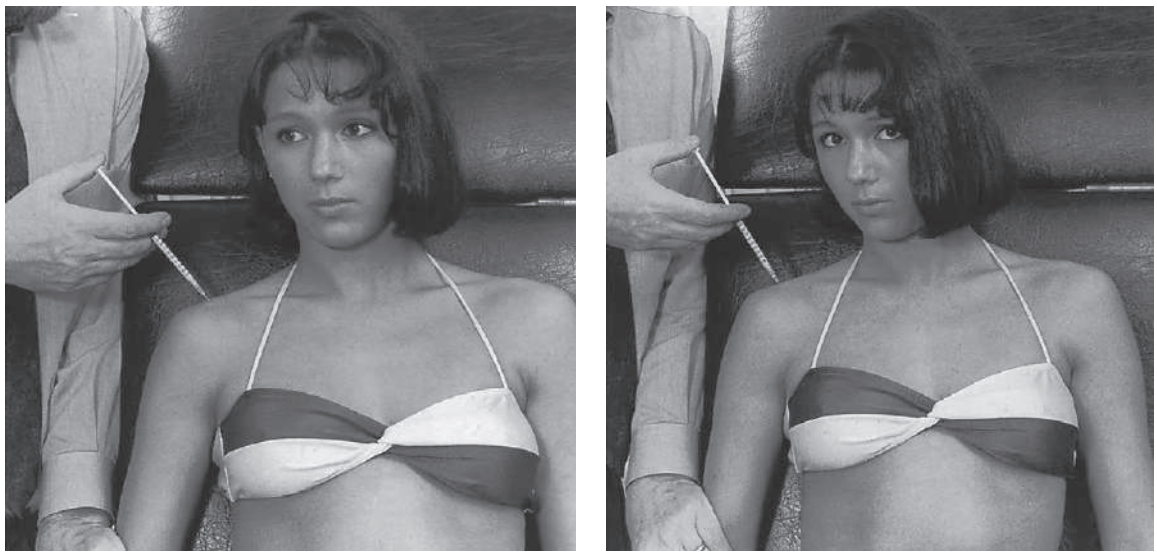


Fig 14.22 • Infiltration of the (a) superficial and (b) deep (lower) ligament of the acromioclavicular joint.

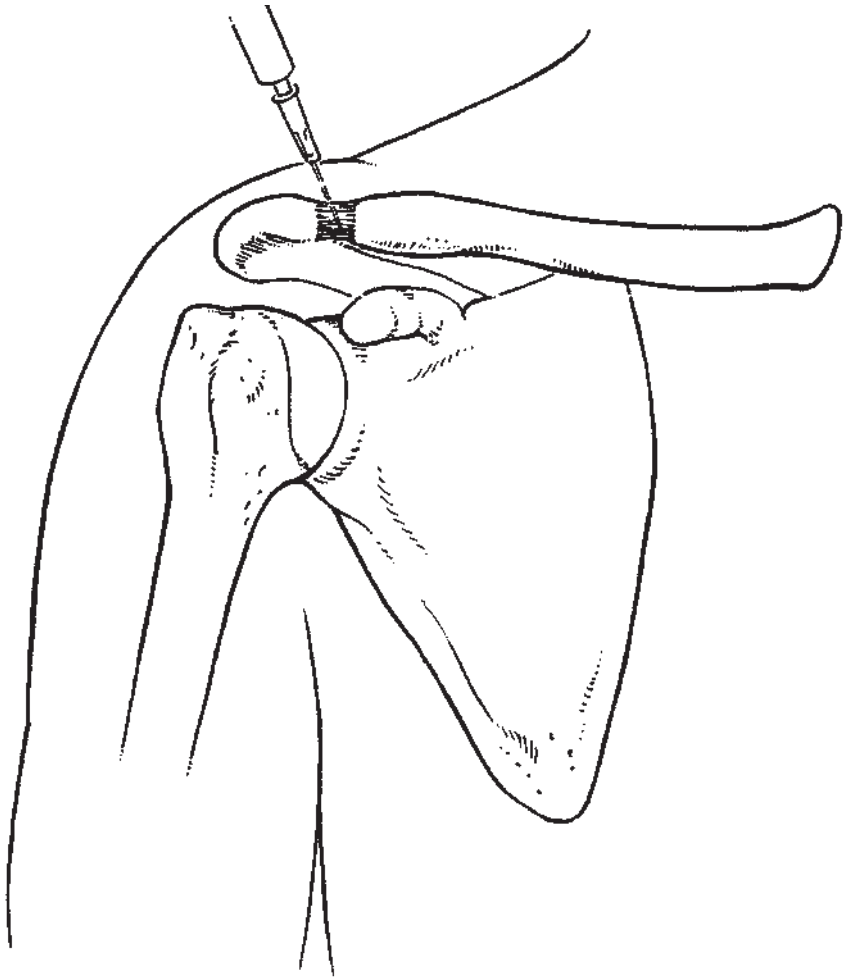


Fig 14.23 • Infiltration of the acromioclavicular joint: deep ligament.

assistant who brings the arm of the patient into full lateral rotation, so as to open the acromioclavicular joint space as widely as possible. The needle is normally inserted in a craniolateral to caudomedial direction (Figs 14.22b and 14.23). First the superficial ligament is encountered, next the meniscus and finally the deep ligament. They all offer the same resistance. The deep ligament lies about 2 cm from the surface. The needle must be inserted almost to its full length until ligamentous resistance is felt. The structure is then infiltrated fanwise over its full length.

Follow-up

The patient rests the arm for 1 week and is reassessed. If the tests are still positive, a further infiltration is done. One or two infiltrations usually suffice.

Technique: friction

This applies only to the superficial ligament. The patient adopts the same position as for palpation. The therapist stands behind, level with the affected shoulder. Friction is given with the index finger of the ipsilateral hand reinforced by the middle finger (Fig. 14.24). The fingers are placed exactly on the tender fibres of the superficial ligament at the joint line. Counterpressure is applied with the thumb at the back of the shoulder (see Fig. 14.24), placed vertically under the fingers. It is best to keep the index finger rather flat in order to treat the whole lesion at once. Because of the orientation of the fibres, friction is applied in an anteroposterior direction. As usual, the therapist alternates active and passive phases: the active moment is when the finger is pulled backwards towards the body, starting with the fingertip at the anterior portion of the ligament.

Friction is carried out three times a week for 20 minutes each session. Cure is normally obtained after 10–15 sessions.

Treatment of recurrences

Some cases tend to recur. In this event, sclerosant infiltrations may be useful. The technique used is the same as for steroid infiltration: 0.75 mL of P2G (phenol solution) mixed with 0.25 mL of xylocaine 2% is used per ligament and repeated twice at weekly intervals. The patient should be warned to expect severe after-pain for about 4 days.

Sprain with moderate displacement (grades II and III)

In all but the most severe dislocations, treatment consists of a short period of standard sling immobilization and early functional rehabilitation. Many studies have reported good functional results in spite of residual deformity in patients treated by this manner of 'skilful neglect'.[204–207] Treatment consists of a standard sling until the acute inflammation has subsided. If the pain and inflammation persist, a steroid infiltration can be given into the ligamentous remnants in order to suppress the inflammation. After 10 days the patient is allowed to mobilize

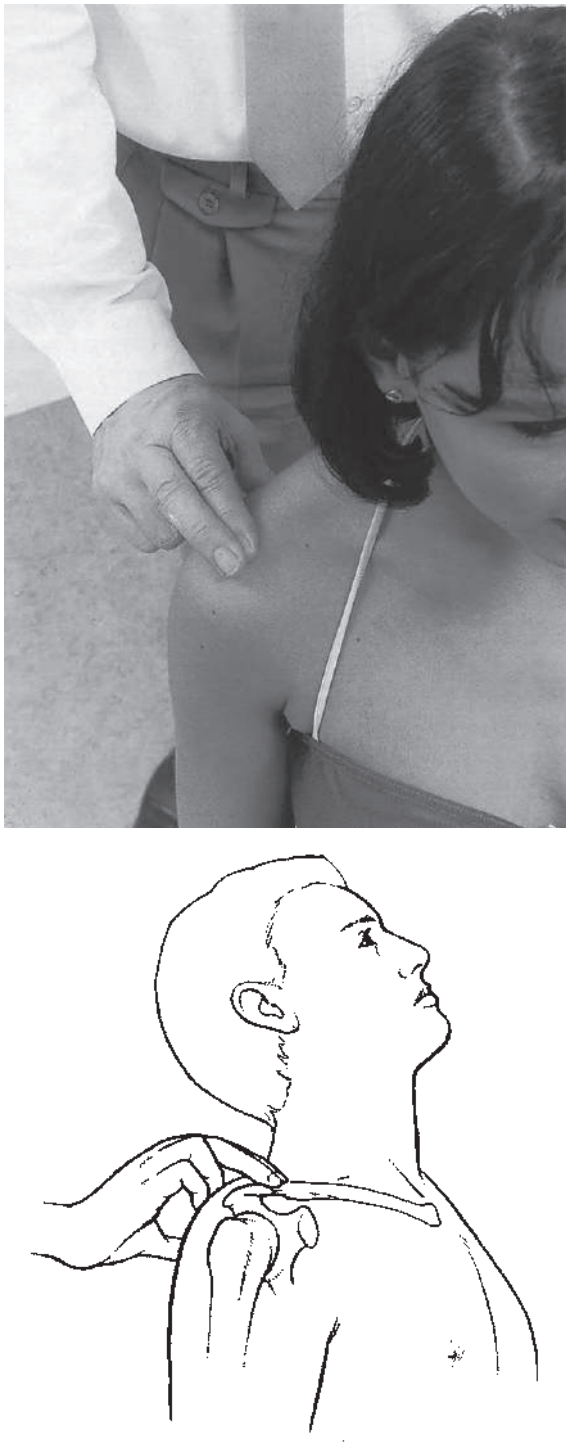


Fig 14.24 • Deep friction to the superficial acromioclavicular ligament.

the shoulder and is referred to a rehabilitation programme to strengthen the muscles of shoulder and shoulder girdle. Return to work or to sport is advised as soon as the shoulder can tolerate it. Several studies have shown that non-operatively treated patients with grade III acromioclavicular separations return to work earlier and with a lower complication rate than patients who have had surgery.[208,209]

Treatment of grade IV, V and VI injuries

Because of the severe displacement of the distal clavicle, surgical repair is advised.[210]

Special cases

Atraumatic osteolysis of the distal clavicle

Atraumatic osteolysis of the distal clavicle in athletes is a stress failure syndrome of the distal clavicle.[211] It is characterized by symptomatic resorption of bone over a period of weeks to many months. The origin is uncertain but the condition is usually related to strenuous physical activity.[212] There is never a history of any major injury to the acromioclavicular joint. It occurs principally in young athletes who have a long history of intense strength training.[213] Sporting endeavours with repeated episodes of significant trauma to the shoulder, or when the participant suffers repeated falls on to the point of the shoulder, have also been identified as precipitating causes of osteolysis.[214]

History and clinical findings are the same as in ordinary grade I sprain. As symptoms become more established, there is tenderness of the entire joint and some swelling can be palpated. Often a joint effusion can be aspirated.[215]

Diagnosis is by plain X-ray examination. Classically the radiographic changes are divided into three phases: lytic, reparative and 'burnt-out'. Optimal visualization of the acromioclavicular joint is only provided by taking an anteroposterior film with the X-ray beam tilted in a 25–30° cephalic direction.[216]

The natural history of the condition seems to run a self-limiting course of 1–2 years. Symptomatic treatment consists of modification of training activities and local infiltration of the acromioclavicular ligaments with triamcinolone.[217] Operative treatment is the exception and consists of resection of the distal clavicle,[218] which is usually performed arthroscopically.[219] The reported results are fair to good.[220]

Arthrosis of the acromioclavicular joint

Generally, a joint that is already arthrotic is more susceptible to the effects of exertion. This also applies to the acromioclavicular joint. The arthrosis as such normally gives rise to a vague ache in the C4 dermatome for some hours after activity. The pain usually wears off spontaneously. Sometimes it persists and, if this is so, the clinical picture is that of a grade I acromioclavicular sprain. Cure is easily achieved by steroid infiltration. If relapse occurs, sclerosant infiltration of the ligaments can be tried. If this is unsuccessful, the patient should avoid the precipitating activity for the rest of his life.

In ankylosing spondylitis, arthrosis of the acromioclavicular joint is very common and total ankylosis is often seen.[221]

Sprained coracoclavicular ligaments

History and examination

A sprain of the conoid and trapezoid ligaments is a particular hazard in sports such as squash or tennis, where the preliminary movement to a 'smash' may require the arm to be pulled far back.[222] It is also sometimes the result of a clavicular fracture.

(a)

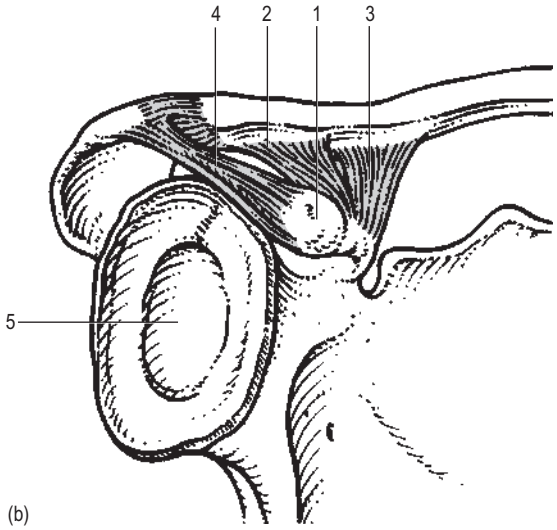


Fig 14.25 • (a) Infiltration of the clavicular insertion of the coracoclavicular ligament. (b) Anatomy: 1, coracoid process; 2, conoid ligament; 3, trapezoid ligament; 4, coracohumeral ligament; 5, glenoid labrum.

The clinical picture may be difficult to interpret. Pain is felt in the mid-clavicular area at the extreme of all passive arm and scapular movements. No limitation is found and resisted movements are painless. If the disorder is suspected, forced lateral rotation with the arm in horizontal abduction must be added to the clinical examination (see p. 229). This is usually the most painful test.

The lesion may be found at the superior side of the coracoid process or at the insertion on the inferior aspect of the clavicle. Differentiation between the two locations is made by palpation, which is best done with approximated scapulae. Careful comparison with the painless side should always be done because the coracoid process is always somewhat tender to the touch. In cases of doubt, a diagnostic local anaesthetic infiltration must be performed.

Differential diagnosis

Differential diagnosis is necessary, and includes the following disorders:

- *Sprained acromioclavicular joint*: the pain is felt more laterally at the tip of the shoulder. Passive horizontal adduction is the most painful test.
- *Subcoracoid bursitis*: this usually gives rise to a limitation of passive lateral rotation, which disappears when the test is repeated with the arm abducted to the horizontal.
- *Sprain of the subclavius muscle*: resisted shoulder depression is painful.
- *Subscapularis tendinitis*: the pain is felt more laterally, at the level of the axillary line. Resisted medial rotation is painful, as is passive lateral rotation.

Treatment

Because the coracoid attachment is deeply situated and the clavicular insertion is beyond the reach of the finger, both sites are treated by infiltration with steroid.

Technique: infiltration of the coracoid insertion

If tenderness is found at the coracoid process, 2 mL of triamcinolone are infiltrated at the superior aspect with the tip of the needle in bony contact. The typical ligamentous resistance must be felt. The whole amount is infiltrated drop-wise at, for example, 5–10 different places.

Technique: infiltration of the clavicular insertion

If palpation is painless, the problem lies at the clavicular insertion. A 3 cm needle is fitted to a syringe containing 2 mL of triamcinolone. The needle is inserted about 2 cm distal to the centre of the ligamentous insertion at the clavicle (Fig. 14.25). It is then moved further in until it meets bone. When ligamentous resistance is felt, the product is infiltrated drop by drop, starting just medial to the line of the acromioclavicular joint and over about 3 cm towards the midline until ligamentous resistance is no longer felt. During the whole procedure, the tip of the needle stays in bony contact. In some cases it may be of help to have the arm in full elevation, which brings the inferior aspect of the clavicle to lie anteriorly.

Follow-up

The patient is reassessed 1 week later and reinfiltrated if necessary. Results are fairly good as long as the activity that caused the lesion is avoided.

Chronic subdeltoid bursitis

A chronic subdeltoid bursitis is not the late result of an acute subdeltoid bursitis; it is chronic from the onset. It is therefore a clinical entity in itself, being far less painful than acute bursitis. Local inflammation and fibrotic adhesion formation between the acromial and tendinous parts of the bursa are the anatomical substrates of the lesion.[223] Also fluid may collect between the bursal walls. However, in the majority of cases, the lesion is localized with only one part of the bursa being affected.[224] Recently an increased amount of substance P was demonstrated in the subacromial bursal wall of patients with signs and symptoms that were attributed to chronic subdeltoid bursitis.[225]

History

The condition may affect all ages. It comes on spontaneously, or after injury complicates rheumatoid arthritis. Untreated, it does not show a great tendency for spontaneous healing and it can even persist for life. It is also not exceptional for those who have been cured by proper treatment to suffer a recurrence.

The pain is usually localized in the deltoid area but can spread further down the arm in the C5 dermatome. Sometimes it is felt only on activity, while at other times it is felt mainly at rest or even continuously, day and night. Consequently, the pain of chronic bursitis is not readily differentiated from C5 pain caused by other shoulder disorders.

Functional examination

The main difficulty with chronic subdeltoid bursitis remains the heterogeneity of the clinical pattern. Sometimes there is a mixed clinical picture of pain on some passive movements and pain on some resisted movements, with or without painful arc. Cyriax considered this pattern to be an 'incomprehensible bursitis'. It is quite possible that such a case is, in fact, a more evolved form of rotator cuff failure: a tendinous lesion of the bursal surface of the cuff tendons in combination with reactive inflammation of the bursa (see p. 250).

Most typically, chronic subdeltoid bursitis is characterized by a painful arc in the absence of any limitation of movement. The arc is sometimes the only positive clinical finding, can be very pronounced and is usually the most painful test. Often, in addition to this, the extremes of all passive movements also hurt.

Exceptionally, chronic subdeltoid bursitis may provoke limitation of movement in a non-capsular way. Limitation of either passive scapulohumeral abduction or passive internal rotation is present. All resisted movements are painless or equally painful.

Although the list given in Box 14.3 is not exhaustive, it describes one of the clinical pictures corresponding to a chronic subdeltoid bursitis.

Box 14.3

Clinical patterns of chronic subdeltoid bursitis

Most frequent patterns

- Painful arc only
- Pronounced painful arc
- Pain at the extremes of all passive movements
- Limitation of abduction or medial rotation
- All resisted movements painless or equally painful

'Incomprehensible bursitis'

- Pain on full passive lateral and/or medial rotation
- Pain at the end of all passive movements
- Painful arc
- Resisted abduction and lateral rotation painful
- Full passive lateral and medial rotation painful
- Painful arc
- Varying pattern of pain on resisted movements, which are sometimes transiently painful

If subdeltoid bursitis is suspected, palpation of the superficial part always follows the functional tests. For this the patient sits on a couch, the arm in the neutral position and the hand resting on the thigh for maximal relaxation. The whole deltoid area must be palpated and no single point overlooked. Palpation starts posteriorly below the outer end of the spine of the scapula and is continued laterally and anteriorly below the acromial edge. It is a good habit to start palpation away from the expected site of the lesion. It should always be done on both sides. Sometimes a local swelling or a small effusion is present.[226,227]

It must be stressed that the diagnosis always remains doubtful until it is confirmed by infiltration with local anaesthetic. Sonography can be used in the diagnosis of bursitis but the results should always be considered in relation to the clinical assessment, as asymptomatic 'bursitis' may be as prevalent as asymptomatic rotator cuff tears.[228–230] Naranjo *et al* identified sonographic signs of subdeltoid bursitis in 29% of asymptomatic shoulders,[231] while an MRI study identified changes consistent with subacromial bursitis in 100% of asymptomatic patients who had undergone rotator cuff repair.[232]

Differential diagnosis

The diagnosis of chronic subdeltoid bursitis is not always obvious and there are several other disorders of both inert and contractile structures which must be differentiated.

Sprain of the acromioclavicular joint

If the upper ligament is at fault, there is localization of the pain at the tip of the shoulder, lack of pain reference down the arm, and pain on palpation of the acromioclavicular joint line.

The situation is more complicated with a sprain of the inferior ligament. In this case, the pain may spread further into the deltoid area and is impossible to differentiate from the pain of

subdeltoid bursitis. A point in favour of the acromioclavicular joint is severe pain on passive horizontal adduction, a test which is usually less pronounced in bursitis. Nevertheless, the diagnosis should always be confirmed by an infiltration with local anaesthetic.[233]

Tendinitis

As, in chronic subdeltoid bursitis, one or more resisted movements may be positive, together with pain on passive movements and a painful arc, a disorder of a contractile structure must be eliminated. Most often, differentiation from supra- or infraspinatus tendinitis is needed, and less frequently the subscapularis structure. Differentiation is usually done by repeating the resisted movement in the supine position, because this relaxes most other structures except the one which is elicited. If the muscle is at fault, the resisted test remains positive to the same degree; in bursitis, the pain usually diminishes or disappears totally when repeated in the lying position. If resisted abduction is painful, it may be of help to repeat the test not only with the patient lying down but also with longitudinal traction. If this renders the test less positive, bursitis is most likely.

Subcoracoid bursitis

In subcoracoid bursitis the pain is more localized in the outer infraclavicular area and does not radiate into the arm. A slight limitation of passive lateral rotation is present, disappearing when the test is repeated with the arm abducted to the horizontal (see p. 239).

Sprain of the subclavius muscle

This provokes pain in the same area as in subcoracoid bursitis but resisted shoulder depression is painful.

Sprain of the coracoclavicular ligaments

Pain is felt in the mid-infraclavicular area. The most painful movement is passive lateral rotation in 90° horizontal abduction.

Aseptic necrosis

In its early stage, aseptic necrosis may give rise to limitation of a non-capsular type. The discrepancy between symptoms and signs is striking: a great deal of pain and only slight influence of the arm movements. Diagnostic infiltration in the subdeltoid bursa can be helpful; in cases of doubt, technetium scanning should be obtained.

Treatment

As a rule, only one part of the bursa is affected and palpation will reveal which part must be dealt with. If local tenderness is found on palpating the superficial part, this must be treated; if no local tenderness can be detected, the subacromial part must be at fault.

An infiltration of 10 mL of 0.5% procaine solution is administered. Procaine has the advantage in this disorder of being both diagnostic and curative. If no lasting benefit follows the first infiltration, procaine should be substituted by steroid in a subsequent infiltration.

Fig 14.26 • Infiltration of the superficial part of the subdeltoid bursa.

Technique: infiltration of the superficial part of the bursa

If local tenderness is found on palpation, the superficial part must be treated. The patient sits on a high couch with the arm in neutral position. The tender part of the superficial bursa is marked (Fig. 14.26). A 3 cm needle is fitted to a 10 mL syringe filled with a 0.5% procaine solution. The needle is inserted at the centre of the tender area and thrust in until it hits the bone. On withdrawal, a little of the procaine is injected. This manœuvre is repeated several times until all the procaine is divided over the whole lesion.

Technique: infiltration of the subacromial part of the bursa

If palpation is negative, the subacromial part of the bursa is at fault. To infiltrate this portion, the lateral edge of the acromion is first localized. A thin needle, 5 cm in length, fitted to a 10 mL syringe filled with 0.5% procaine, is inserted just underneath the middle of the outer acromial edge and in a slightly cranial direction (Fig. 14.27). It is inserted to its full length, meeting hardly any resistance. If the needle does encounter resistance, either the coracoacromial ligament or the capsulotendinous structures have been contacted and pain is felt. The needle should be slightly withdrawn and the direction adjusted. Occasionally an effusion is encountered in the bursa and must be evacuated by aspiration before injection of local anaesthetic.

Once the needle is correctly placed, the infiltration is given while withdrawing and reinserting it about four or five times. Fanwise infiltration is used in order to reach the whole of the subacromial bursa.

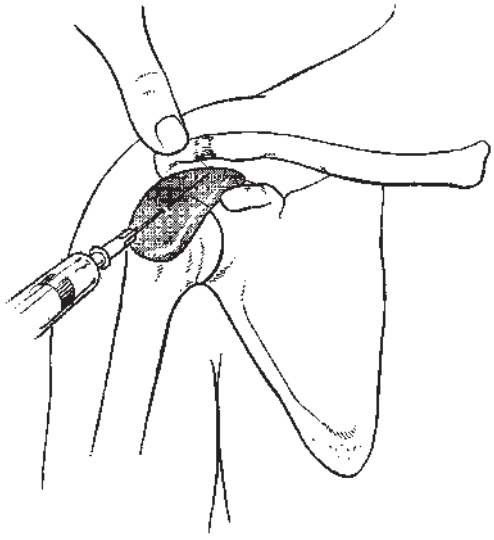


Fig 14.27 • Infiltration of the deep part of the subdeltoid bursa.

Follow-up

The patient is re-evaluated 1 week later. If there has been an improvement but not full recovery, the infiltration is repeated. Usually, three infiltrations are sufficient for full cure. If no improvement is obtained after the first infiltration, 5 mL of steroid should be substituted.

Some patients suffering from chronic subdeltoid bursitis never fully recover or tend to have frequent recurrences. They usually present one of the less comprehensible clinical pictures. This may be the consequence of a minor rupture in one of the rotator cuff structures, leading to the formation of adhesions and self-perpetuating inflammation. Definitive cure for this is often difficult to obtain. In such cases, the infiltrations may be repeated on a regular basis at increasing intervals until full cure is obtained and no relapse occurs.

Crepitating bursitis

After a previous bursitis with effusion, some patients may have crepitus on movements of the arm, which does not cause pain, merely a vague discomfort. No treatment is known.

Excessive range of movement: instability of the shoulder

See online chapter *Excessive range of movement: instability of the shoulder*.

Access the pathology of excessive range of movement of the shoulder and the complete reference list online at **www.orthopaedicmedicineonline.com**

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15

Disorders of the contractile structures

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Introduction

Functionally, shoulder muscles are of two types: stabilizing muscles and effector muscles. Stabilizing muscles (A, Fig. 15.1) are relatively small, with insertion tendons that lie close to, or even in, the substance of the fibrous capsule. Therefore they are not capable of causing significant shoulder movement but rather maintain the humeral head in the glenoid fossa. These stabilizing muscles are called the rotator cuff and include supra- and infraspinatus, teres minor and subscapularis. They all originate from the scapula, run partly under the acromial roof and insert on the humeral tubercles.

Effector muscles (B, Fig. 15.1) are much larger, with tendon insertions at a greater distance from the joint. Consequently, they produce powerful movements and are not primarily involved in stabilization. They are the deltoid complex, the pectoralis major, the latissimus dorsi and the teres major.

Although the standard clinical examination tests both muscle groups, the great majority of the positive findings point towards lesions of the rotator cuff, because lesions of the large effector muscles are extremely rare.

Rotator cuff

Rotator cuff disorder is one of the commonest afflictions of the shoulder and is a major cause of impairment of health in the young as well as in older individuals.[1]

Lesions of the rotator cuff should be recognized as different from those of other tendons in the body for a variety of reasons. The tendons of the rotator cuff blend intimately with each other and with the capsule. The insertion of the tendons, as a continuous cuff around the humeral head, permits the cuff muscles to provide an almost infinite variety of movements to

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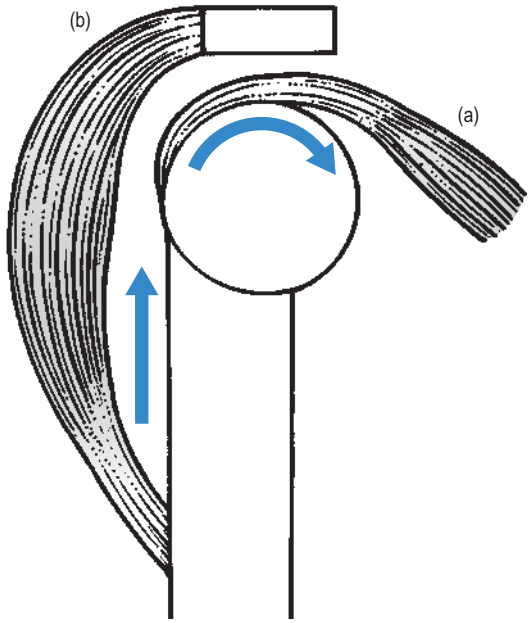


Fig 15.1 • A, Rotator cuff (stabilizing) muscles; B, large effector muscles.

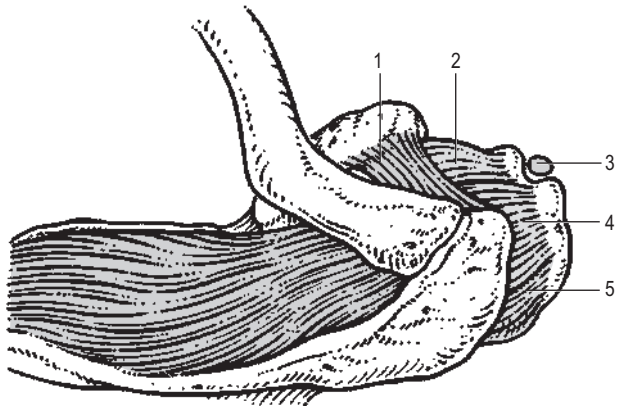


Fig 15.2 • Rotator cuff from above: 1, coracoacromial ligament; 2, subscapularis tendon; 3, biceps tendon; 4, supraspinatus tendon; 5, infraspinatus tendon.

Box 15.1

Functions of the rotator cuff

1. Rotate the humerus
2. Compress the humeral head
3. Provide muscular balance

rotate the head and to oppose unwanted movements generated by the larger effector muscles.[2] In addition, the long head of the biceps may be considered a functional part of the rotator cuff because tension in the tendon helps to compress the humeral head into the glenoid (Fig. 15.2).

Apart from their primary function (to rotate the humerus with respect to the scapula), the cuff muscles have two other actions: they compress the head of the humerus into the glenoid fossa and provide muscular balance (Box 15.1). The latter is mainly performed through eccentric contraction of the muscle. Both functions are extensively discussed in the online chapter *Excessive range of movement: instability of the shoulder*.

Pathology

Pathological changes associated with rotator cuff tendinopathy features are variable.

Inflammatory tendinitis is a reversible process, associated with an inflammatory infiltrate, increased vascularity and hyperaemic changes within the rotator cuff tendon.[3]

Partial rotator cuff tears may develop within the substance of the tendon on either the acromial (bursal) or articular surface of the tendon.[4] Most of the lesions occur near to the tendon insertion side. Full thickness tears are often initiated by a partial tear. They can be associated with a traumatic event or can progress with normal daily use of the arm.

Changes in the rotator cuff may also involve calcifying tendinopathy and rotator cuff arthropathy (degenerative glenohumeral osteoarthrotic disease associated with chronic massive rotator cuff tears).

The pathogenesis of rotator cuff disease has been associated with three factors: age-related degeneration, impingement and microvascular blood supply.

The primary cause of tendon degeneration is age.[5] Changes in the rotator cuff include diminution of fibrocartilage at the cuff insertion, diminution of vascularity, fragmentation of the tendon and disruptions of the attachment to the bone.[6,7]

Changes in the coracoacromial arch have been described in association with cuff disease and it is quite clear from both cadaver and clinical data that individuals with full thickness rotator cuff tears have changes in the acromial shape, with spur formation on the undersurface of the acromion and/or hypertrophy of the acromioclavicular joint.[8–11] Although these data indicate a strong association between the presence of cuff tears and alterations of acromial contour,[12–14] it is still unclear whether the change in acromial shape is the cause or the result of the cuff defect or if both are consequences of ageing.[15] Recent studies suggest that acromial deformity is usually developmental. Most acromial 'hooks' develop within the acromial ligament as traction spurs (analogous to the traction spur in the plantar ligament at its attachment to the calcaneus – see Fig. 15.11). The traction results from loading of the ligament by the cuff, which is increased when superior instability and cuff degeneration are present.[16,17]

Changes in the microvascular supply to the rotator cuff also have a possible role in the pathogenesis of rotator cuff lesions.[18] A hypovascular region exists at the 'critical zone' of the supraspinatus (the deep surface of the anterior insertion).[19,20] Microangiographic studies demonstrate that inadequate vascular supply to this critical zone is present in the adducted position of the arm.[21] Furthermore, microvascular supply changes within the thickness of the tendon: the acromial part has much better vascularity than the articular part.[22–24]

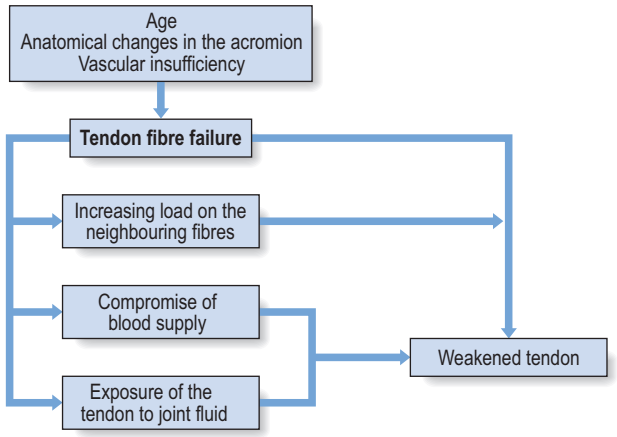


Fig 15.3 • Tendon fibre failure.

It is likely that a combination of age, anatomical changes and vascular insufficiency is responsible for rotator cuff 'failure'.[25] Throughout life the cuff is subjected to various adverse factors such as traction, compression, contusion, subacromial abrasion, inflammation and age-related degeneration. A lesion may start where the loads are the greatest and the vascular supply the lowest, i.e. at the deep surface of the anterior insertion of the supraspinatus.[26] Each fibre rupture then generates other adverse effects: it increases the load on the neighbouring fibres, it compromises the blood supply of the tendon fibres by distorting the microcirculation and it exposes increasing amounts of the tendon to joint fluid that contains lytic enzymes. The cuff is gradually weakened and at increased risk of further failure. With subsequent loading episodes the pattern repeats itself, rendering the cuff weaker and progressively more susceptible to additional failure (Fig. 15.3).[27]

Incidence

The incidence of rotator cuff tears has been studied both in cadaveric studies and in living subjects and found to range from 5 to 80%. All the studies show a strong relationship with age: rotator cuff tears are rare before age 40 and common after age 60.[28] However, almost all of the reported cadaver studies failed to correlate cuff disorder with a history of clinical symptoms (Table 15.1). Some of the most important studies in living subjects have concerned the prevalence of cuff lesions in asymptomatic patients (Table 15.2). They all demonstrated a high prevalence of tears of the rotator cuff in asymptomatic individuals, an increasing frequency with advancing age and compatibility with normal, painless functional activity.

It must be concluded from these studies that rotator cuff lesions are a natural correlate of ageing. They should be regarded as 'normal' degenerative attrition, not necessarily causing pain and functional impairment. This realization poses substantial questions about the anatomical diagnosis (magnetic resonance imaging (MRI) and sonography) of shoulder pain and of the indications for cuff surgery. Once again, it must be stressed that diagnosis and treatment should be based on clinical findings and not on the results of imaging.

Table 15.1 Incidence of rotator cuff tears in cadaver dissections

Authors	Incidence (%)	Remarks
Keyes (1933)[29]	19	
Wilson (1943)[30]	20–22	
Grant and Smith (1948)[31]	37	Increasing incidence with age
Cotton and Rideaout (1963)[32]	20	
Uhtoff *et al* (1986)[33]	30	
Ozaki *et al* (1988)[34]	12 (complete)	None in cadavers <40 years 40% in cadavers >60 years
Kummer and Zukerman (1995)[35]	17	6% in cadavers <60 years 30% in cadavers >60 years
Jerosch *et al* (1991)[36]	28.3 (partial) 31 (complete)	More common in women
Fukada *et al* (1987)[37]	13 (partial)	30% in cadavers >40 years No tears in cadavers <40 years
Sakurai *et al* (1998)[38]	61.5 (partial)	
Jiang *et al* (2002)[39] % partial and full thickness tears	11.5 (massive rotator cuff tears)	Average age 76.3 years

Diagnosis and treatment

Given the high prevalence rate of asymptomatic macroscopic rotator cuff lesions, it is unwise to rely solely on imaging techniques (sonography, computed tomography (CT), MRI) for a diagnosis.[49] The examiner should be wary of the common belief that what is found on technical investigation is always relevant to the cause of the patient's pain.[50] Diagnosis should first of all be made functionally; paraclinical and technical investigations have a secondary function only. During resisted movements, pain and weakness, separately or in combination with each other, are sought. If pain only is found, this points to an uncomplicated tendinitis or to a partial thickness lesion. A combination of pain and weakness brings a partial (full thickness) tendinous rupture to mind. Painless weakness is usually the outcome of a massive tear of a rotator cuff tendon or of a neurological problem. It is worth emphasizing once again that, when performing resisted movements at the shoulder, it is very important to use the correct technique, as has been explained earlier (see p. 214). If this is neglected, misdiagnosis can easily result.[51]

Treatment options are also determined only by the outcome of the clinical examination and not by the extensiveness of the (anatomical) lesion. Asymptomatic cuff lesions, for instance, in which the shoulder does not bother the patient but imaging studies document a partial or even full thickness lesion in the cuff tendon, should not receive treatment. However, if the rotator cuff lesion is symptomatic, it will usually not heal by itself because rotator cuff tendinitis is not self-limiting.[52]

Table 15.2 Incidence of rotator cuff tears in asymptomatic patients

Authors	Incidence (%)	Remarks
Petterson (1942)[40]	20 (partial and full)	50% in subjects 55–85 years of age
Milgrom *et al* (1995)[41]	50 (tears after age 50 years)	Rotator cuff lesions are a normal correlate of age; they often present without clinical symptoms
Sher *et al* (1995)[42]	15 (full), 20 (partial)	Significant increase of tears with age; defects are compatible with normal, painless, functional activity
Yamaguchi (2006)[43]	35.5% prevalence of a full thickness tear on the painless side	Sonography of 588 consecutive patients with unilateral shoulder pain
Tempelhof (1999)[44]	Rotator cuff tears in 23% 13% age 50–59 years 20% age 60–69 years 31% age 70–79 years 51% age >80 years	Ultrasonographic study of 411 asymptomatic volunteers
Shibany (2004)[45]	Complete rupture of the supraspinatus tendon in 6%	Ultrasound examination of 212 asymptomatic shoulders at 56–83 years (mean: 67 years)
Kim (2009)[46]	0% age 40–49 years 10% age 50–59 years 20% age 60–69 years 40.7% >70 years	Ultrasound and MRI investigation of asymptomatic individuals
Moosmayer (2009)[47]	Full thickness tears in 7.6% 15% age 70–79 years	420 asymptomatic volunteers aged 50–79 years
Yamamoto (2010)[48]	16.9% asymptomatic 20.7% total group	Prevalence of tears among the 683 residents of a mountain village in Japan

Initial treatment for a symptomatic cuff lesion should always be conservative, no matter what the result of imaging may be. Non-operative treatment consists of either deep friction transversely to the affected tendon fibres or local infiltration with small amounts of triamcinolone at the tenoperiosteal junction of the affected tendon. In recurrent cases it is wise to add functional exercises for strength and proprioception (see online chapter *Excessive range of movement: instability of the shoulder*).[53]

The effectiveness and safety of steroids for the treatment rotator cuff disease remains the subject of much controversy. Repeated injections with steroids are believed to produce tendon atrophy or to reduce the ability of damaged tendon to repair itself. Animal studies suggest that corticosteroids damage the ultrastructure of collagen molecules[54] and reduce collagen density, as well as inhibiting the reparative properties of tendon by inhibiting tendon cell migration and synovial fibroblast proliferation.[55] This has been shown experimentally to weaken collagen fibres and precipitate tendon ruptures.[56–58] In human subjects, repeated injections have been correlated with softening of the rotator cuff substance[59] and an inferior result of surgical repair.[60] Other studies, however, failed to find a deleterious long-term effect of corticosteroid injections in animal tendons,[61,62] and a recent case-controlled study suggests that corticosteroid use in patients with 'subacromial impingement' should not be considered a causative factor in rotator cuff tears.[63] Although rotator cuff disorders are generally believed to benefit from steroid injections, evidence for the efficacy of the injections is difficult to demonstrate. Most reviews have found conflicting results,[64] which can be explained mainly by the fact that very heterogenous populations with poorly designed subgroups were used.[65] In other studies that used a better anatomical classification, local triamcinolone infiltrations were shown to be superior to placebo[66–70] and to methylprednisolone[71] in reducing pain, improving active abduction and reducing functional limitation. The success rate of the infiltrations is further increased if precise diagnostic and infiltration techniques are used.[72] In a randomly allocated double-blind study, Hollingworth *et al* compared two different methods of corticosteroid injection. The method of anatomical injection after diagnosis by the technique of selective tissue tension gave a 60% success rate, compared with the method using tender or trigger point localization, which produced only 20% success.[73] Also, a recent meta-analysis concluded that injections of corticosteroids are effective for improvement for rotator cuff tendinitis for up to a 9-month period.[74]

We strongly believe in the beneficial effect of small-dose (10 mg) and targeted infiltrations of triamcinolone in the treatment of rotator cuff disorder. Potential hazards are minimal if a few necessary precautions are taken:

- The infiltration is always on the tenoperiosteal junction, never in the body of the tendon.
- The arm should be rested for 2 weeks following the infiltration.
- No more than three consecutive infiltrations should be given.

The operative treatment of rotator cuff lesions without rupture is acromioplasty: a wedge-shaped piece of bone is resected from the anterior surface of the acromion, along with the entire attachment of the coracoacromial ligament. The operative treatment of rotator cuff tear is cuff repair.

Calcifying tendinitis

Calcium deposits may form in the tendons of the rotator cuff. The aetiology is still a matter of speculation but it is generally accepted that degeneration precedes calcification. The incidence of calcification ranges from 3 to 20%[75,76] and in most instances the lesion is completely asymptomatic.[77] The highest incidence occurs in those aged between 31 and 50,[78,79] and calcification is absent in elderly patients. The disease is usually self-limiting with a variable natural course: 80% of calcific lesions show spontaneous resorption over a period of 3 years.[79] If the lesion causes symptoms, these are treated in the same way as uncomplicated tendinitis of the rotator cuff. The treatment of choice is infiltration with triamcinolone. If the pain reappears after initially successful treatment, the calcium deposits can be dissolved with weekly infiltrations of procaine

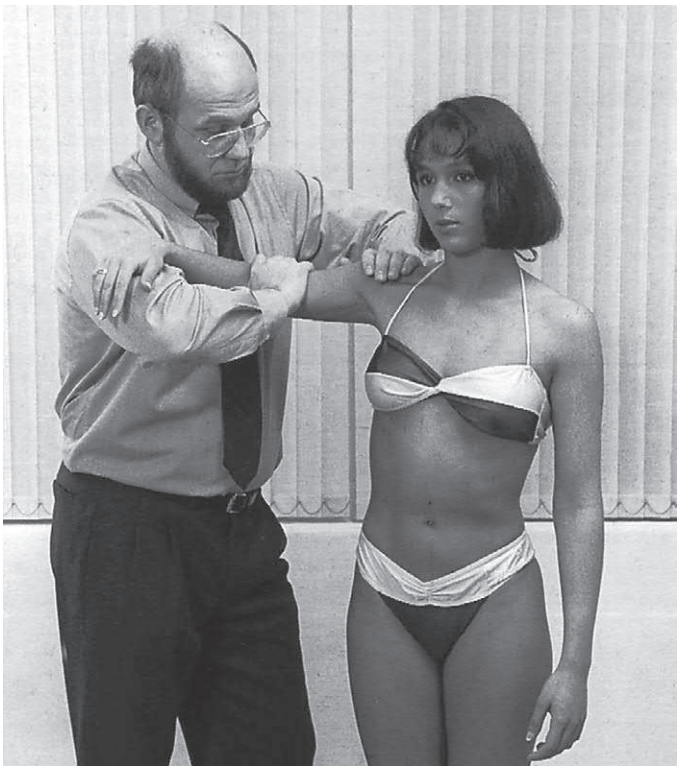


Fig 15.4 • Resisted horizontal adduction.

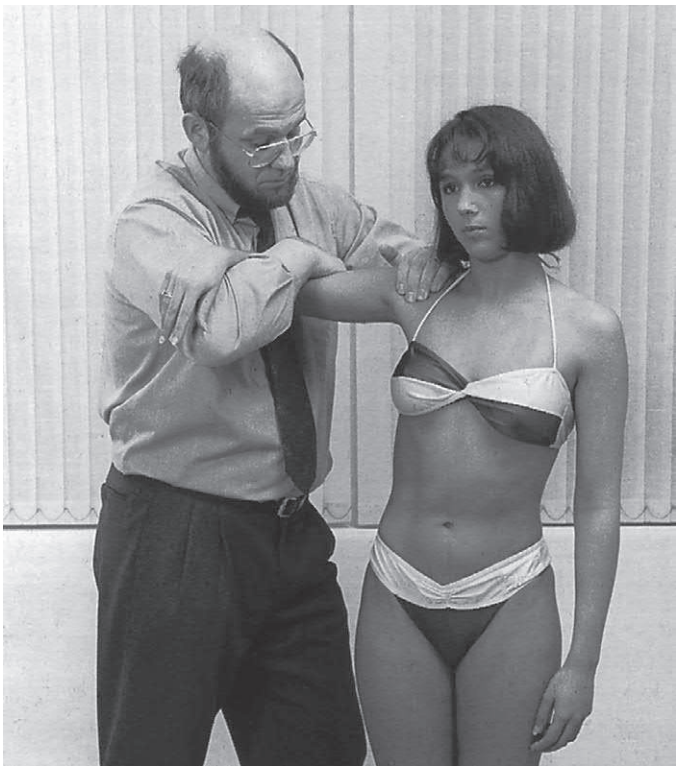


Fig 15.5 • Resisted horizontal extension.

0.5%.[80] Surgical intervention is seldom necessary but if it is indicated, most calcifications can easily be removed by arthroscopic procedures.[81] In recent years extracorporeal shock wave therapy has been proposed as an alternative to operative treatment.[82,83]

Resisted abduction

Pain

Pain on resisted abduction is the consequence of a lesion of either the deltoid or the supraspinatus muscle.

Deltoid muscle

The deltoid is very seldom at fault, although a lesion can sometimes occur as a result of direct injury. The lesion usually lies in the muscle belly.

Disorders of the deltoid cannot provoke a painful arc, because no part of it can be trapped between two osseous structures. Therefore the presence of a painful arc excludes a deltoid lesion. If any doubt exists, two accessory tests are used:

- *Resisted horizontal adduction* (Fig. 15.4): the examiner stands at the painful side and stabilizes this shoulder with one hand. With the other hand, he grasps the patient's arm just above the elbow and brings it into horizontal abduction. The patient is now asked to push the arm forwards against the examiner's hand. This tests the anterior portion of the deltoid.
- *Resisted horizontal extension* (Fig. 15.5): this is the exact opposite of the previous test. The patient is asked to push backwards, the examiner applying counterpressure in an anterior direction. The posterior fibres of the deltoid are tested in this movement.

Treatment

A lesion in the muscle belly of the deltoid responds equally well to deep transverse friction or to infiltration with 20–30 mL of procaine 0.5% once a week, for about 3 weeks. Friction is given on alternate days until full cure is achieved.

Supraspinatus

Clinical experience shows that the most frequent reason for pain on resisted abduction is tendinitis of the supraspinatus muscle, which is by far the most common tendinous lesion at the shoulder. The tendon may be affected at four different sites; these each give rise to a slightly different clinical picture but are all characterized by a common major finding, which is pain on resisted abduction.

Most of the lesions occur at the tenoperiosteal insertion into the greater tuberosity. Inflammation and partial tears may develop within the substance of the tendon on the acromial surface (bursal side) or the deep surface (articular side of the tendon).[84] If situated on the deep part, pain on full passive elevation is also present. This is believed to be caused by the abutment of the deep surface of cuff insertion against the glenoid rim at the extremes of motion (Fig. 15.6).[85] If situated on the bursal part of the tendon, a painful arc is found. Cyriax regarded this as the most common cause of a painful arc on

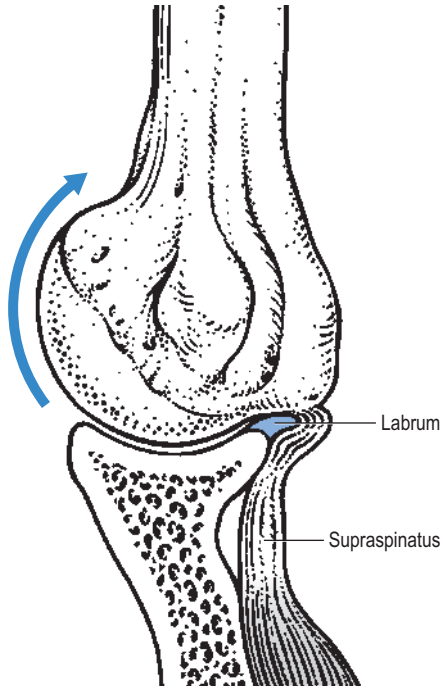


Fig 15.6 • On full passive elevation the undersurface of the supraspinatus tendon may become squeezed against the upper labral rim.

shoulder elevation.[86] A third possibility is a lesion that involves both superficial and deep aspects tenoperiosteally. In this case, pain on full passive elevation is present, together with a painful arc (Fig. 15.7).

The lesion sometimes lies at the musculotendinous junction, just beneath the acromion. In this case, the only sign is pain on resisted abduction. Because this type of lesion is rare, its presence should always be confirmed by a diagnostic infiltration with local anaesthetic.

Figure 15.8 summarizes the differential diagnosis of painful resisted abduction.

Treatment of tenoperiosteal lesions

The lesions situated at the tenoperiosteal insertion can be treated either by deep friction or by infiltration with steroid. Friction takes longer but has a more definite effect; steroids work quicker but there may be a tendency to recurrence.

Localization by palpation

The main problem in treatment is finding the structure involved. Although the tendon lies quite superficially, many have difficulty in localizing it. It cannot be easily localized with the arm in the neutral position at the side. Although the greater tuberosity points laterally in this position, part of the insertion can be covered by the outer rim of the acromion. Moreover, all structures here feel the same on palpation. Therefore, it is

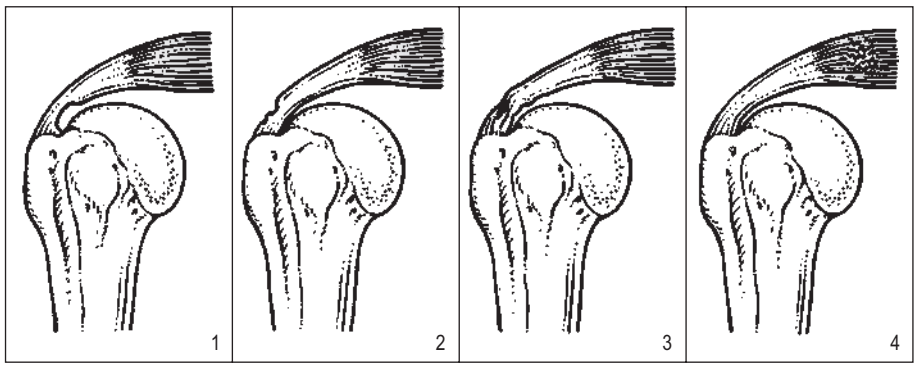


Fig 15.7 • Accessory signs indicate the exact localization of the supraspinatus lesion: 1, pain at the end of passive elevation; 2, painful arc; 3, painful arc and pain on full passive elevation; 4, no localizing sign.

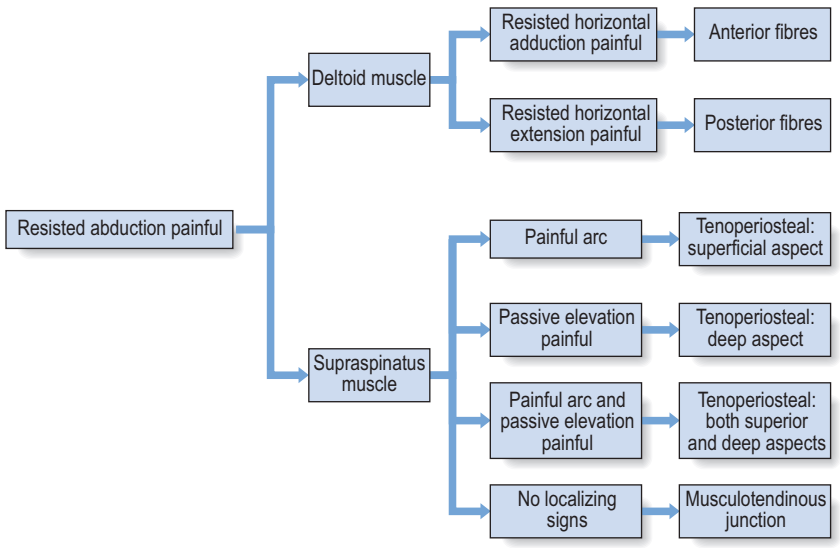
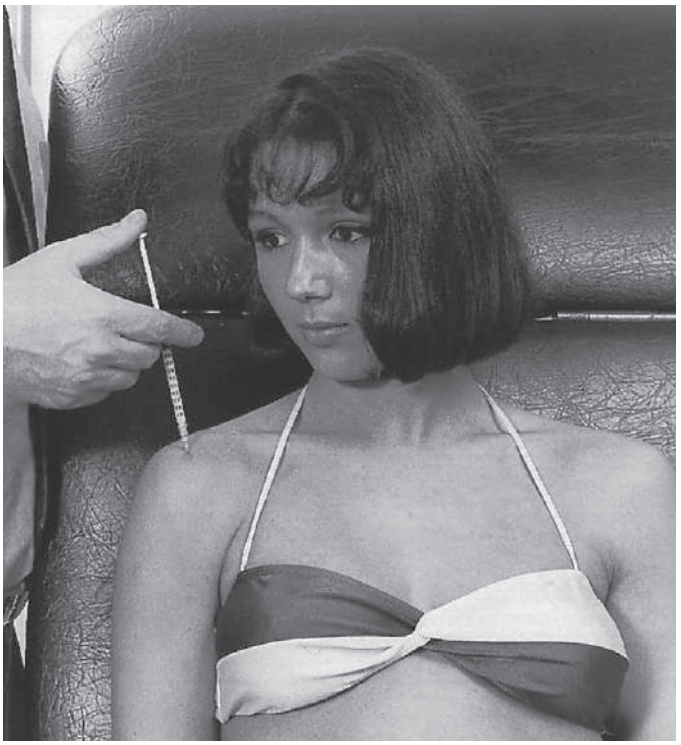


Fig 15.8 • Differential diagnosis of painful resisted abduction.

Fig 15.9 • Position for localization of a tenoperiosteal lesion of the supraspinatus.



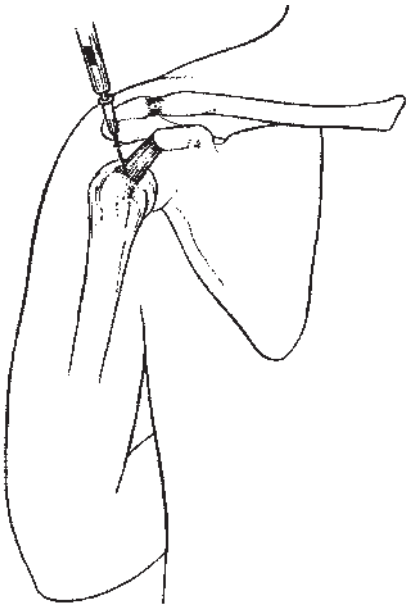


Fig 15.10 • Infiltration of a tenoperiosteal lesion of the supraspinatus.

better to bring the upper arm into full medial rotation by asking the patient to put the lower arm behind the back, with the elbow bent to 90° (Fig. 15.9). In this position the tenoperiosteal insertion lies anterior to the acromion.[87,88] The fibres at the insertion are now situated in a sagittal plane because the tendon curves around the base of the coracoid process as a result of the medial rotation.

First, bony landmarks are defined. Starting at the posterior acromial angle, the lateral rim of the acromion is localized. The finger then moves over to the anterior acromial border, until the acromioclavicular joint is met. The supraspinatus insertion lies lateral to this joint line, just anteriorly to the acromion.

Next, the infraclavicular fossa is palpated. This is best started medially, at the level of the coracoid process. The finger is moved laterally, retaining close contact with the anterior border of the clavicle and acromion. Initially, the finger digs smoothly into the deltoid muscle, and continues to do so until the tenoperiosteal insertion of the supraspinatus is reached. Then a much tougher resistance is felt under the finger, which does not sink in deeply. The supraspinatus insertion has a width of about 1.0–1.5 cm. It is important to define the medial edge of this insertion accurately, since it permits the localization of the rest of it. The lateral edge is more difficult to palpate because the fibres merge with the anterior of the infraspinatus tendon.

Technique: infiltration of the supraspinatus

A tuberculin syringe, filled with 1 mL of triamcinolone, is fitted to a 2.5 cm needle. The patient sits in the same position as for palpation, the arm behind the back. After the insertion has been precisely located, the needle is thrust in vertically downwards at its centre (Fig. 15.10). The needle glides in smoothly initially, until it encounters the tenoperiosteal

junction, at which point the typical tendinous resistance is felt. When the needle is thrust in a little further, it is arrested against the bone. Then 1 mL of triamcinolone is infiltrated at 5–10 different places over an area of 1 cm^2, in close bony contact. During the whole infiltration, a typical counter-pressure is felt.

After-pain is seldom severe and wears off spontaneously. The arm should be rested for about 2 weeks, and reassessment follows after 2 weeks. If the clinical examination is still positive, a second infiltration is given. One to three infiltrations are usually curative.

Treatment that leads to good but temporary results is a common experience in supraspinatus tendinitis. After one or two infiltrations the pain has disappeared but recurs a few months later. The patient should then be sent for standard radiography of the shoulder. If some intratendinous calcification is confirmed, 4–5 weekly infiltrations with 5 mL of procaine 2% are administered. In most cases this is sufficient to dissolve the calcium deposits and to alleviate the pain. If no calcification is visible, the patient must be referred to the therapist for deep transverse friction because it is unwise to infiltrate the tendon repeatedly, even with small doses such as 10 mg of triamcinolone. In cases of recurrent tendinitis it is also good practice to look for an underlying cause. This may be a small multidirectional or superior instability or an anatomical divergence in the acromial roof that causes recurrent impingement. The diagnosis and treatment of the former have been discussed earlier (see online chapter *Excessive range of movement: instability of the shoulder*). The diagnosis of the latter is through a lateral X-ray that visualizes the so-called 'supraspinatus outlet' – the space between the coracoacromial roof and head of the humerus (Fig. 15.11). If there is an anatomical divergence in the supraspinatus outlet, and the tendinitis tends to recur despite proper treatment, surgical decompression (deletion of anterior and inferior parts of the roof) is highly recommended.[89]

The treatment of supraspinatus tendinitis is summarized in Figure 15.12.

Technique: deep friction to the supraspinatus

Patients who opt for the longer but more certain way are best treated by friction. The same goes for athletes and in cases of recurrence after previous infiltrations with steroids. Friction works better on the superficial part rather than on deeper lesions.

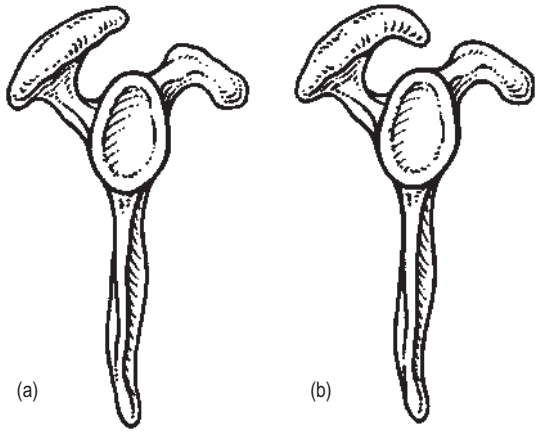


Fig 15.11 • Supraspinatus outlet: (a) normal acromion; (b) hooked acromion.

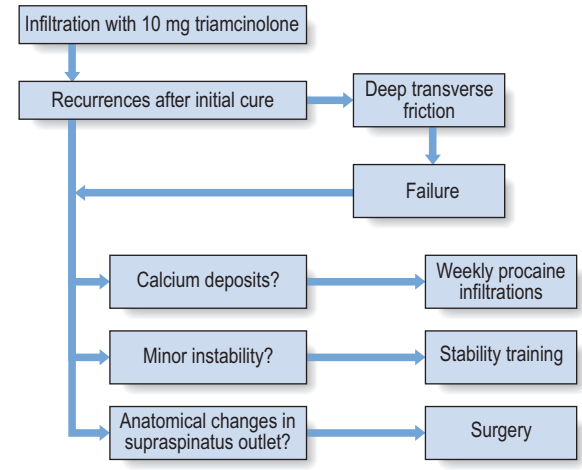


Fig 15.12 • Treatment of supraspinatus tendinitis.

The patient adopts the same position as for infiltration: seated with the back against the couch, the arm behind the back. The therapist stands laterally on the patient's painful side. The index finger of the ipsilateral hand, reinforced by the middle finger, is placed at the medial edge of the insertion. The hand is meanwhile stabilized by the thumb placed against the lateral aspect of the upper arm, almost vertically under the index finger (Fig. 15.13). When the index finger is pulled outwards over the tendon, pressure is applied. This is the active phase of the friction. The pressure is directed caudally, not towards the clavicle – easily obtained if the stabilizing thumb is placed rather low on the upper arm, the nail of the finger that applies friction thus pointing upwards.

Friction is given three times a week for about 20 minutes each time. Normally, 10 sessions suffice. The patient should rest the arm during the whole course of treatment. Improvement can be expected after about five sessions.

Treatment of musculotendinous lesions

The only effective treatment is deep transverse friction, but before this is undertaken the diagnosis must always be confirmed by an infiltration of local anaesthetic.

Technique: deep friction to musculotendinous lesions

The patient sits on a chair with the arm abducted sideways to the horizontal, the elbow and forearm resting on a couch. In this position the musculotendinous junction lies in the supraspinous fossa at the angle between the scapular spine and the acromion, just posterior to the clavicle. The therapist stands at the pain-free side facing the shoulder. The ipsilateral middle finger reinforced by the index finger is placed deeply into the scapuloacromial angle, holding the slightly bent finger parallel to the muscle (Fig. 15.14). Friction is given by pronation–supination movements of the lower arm, the active moment being in supination.

Friction is applied for about 15 minutes. Cure is expected after about 10 sessions.

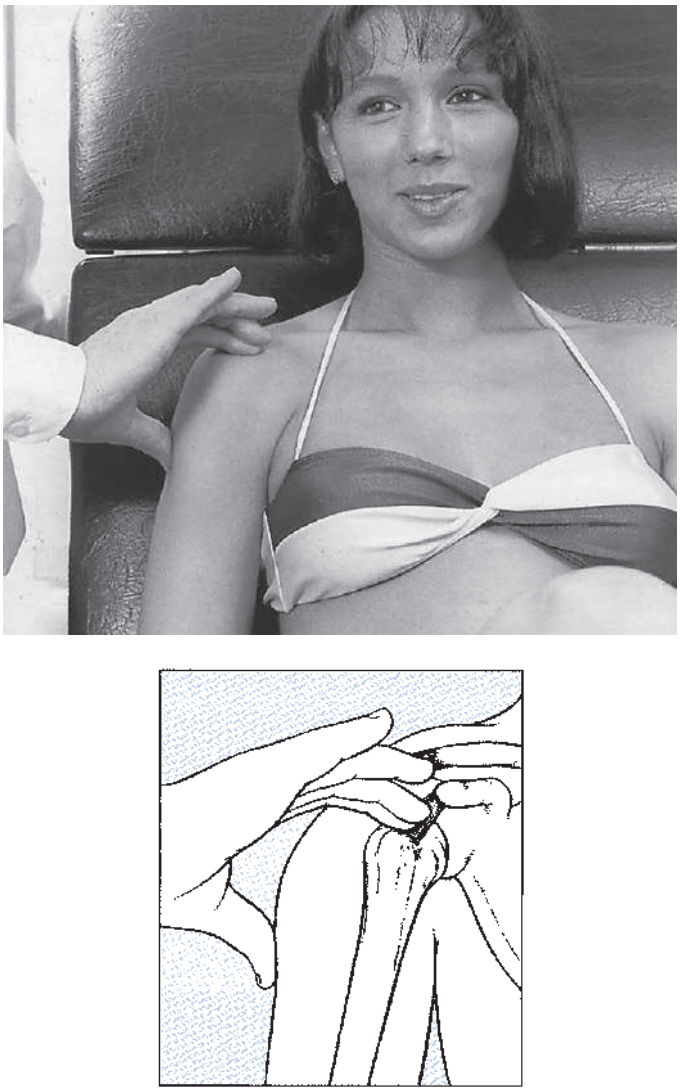


Fig 15.13 • Treatment of a tenoperiosteal lesion of the supraspinatus: deep friction.

Painful weakness

If resisted abduction is found to be both weak and painful, a partial rupture of the supraspinatus tendon is most likely.[90]

Ruptures of the rotator cuff occur most frequently at the supraspinatus tendon. A full-thickness defect usually starts at the critical zone (articular side of the anterior part near to the bicipital groove) and may propagate either in the direction of the infraspinatus or towards the subscapularis tendons.[91,92]

The pain felt in a partial rupture of the supraspinatus is the same as in uncomplicated tendinitis. The patient usually does not mention any weakness; this is found only during clinical examination on resisted abduction, which is also painful. To rule out weakness caused by pain alone, resisted abduction is tested again after infiltration of local anaesthetic. The only differential diagnosis is metastatic infiltration of the acromion – a very rare disorder. Here the pain is localized. On clinical examination a painful arc is found, together with pain and gross weakness on resisted abduction. Imaging techniques confirm the diagnosis.

Treatment

Conservative treatment is reasonable for most partial ruptures of the supraspinatus tendon.[93] Treatment is relief of pain, achieved by infiltration of 1 mL of triamcinolone at the tenoperiosteal insertion and into the most distal part of the tendon. The same position and technique are used as for an uncomplicated tendinitis.

Infiltration of a partially ruptured tendon is not without danger. Disappearance of inflammation and pain usually removes the natural reserve towards movement and load. A weakened tendon in combination with increased load must inevitably lead to further ruptures and disaster. Before any decision to infiltrate is made, the patient must be warned about the dangers. Furthermore, if complete rest cannot be fully guaranteed, the therapist must stop infiltrations and refer the patient for deep transverse friction.

If the treatment leads to good but only temporary results, the same measures should be taken as in uncomplicated but recurrent supraspinatus tendinitis.

Painless weakness

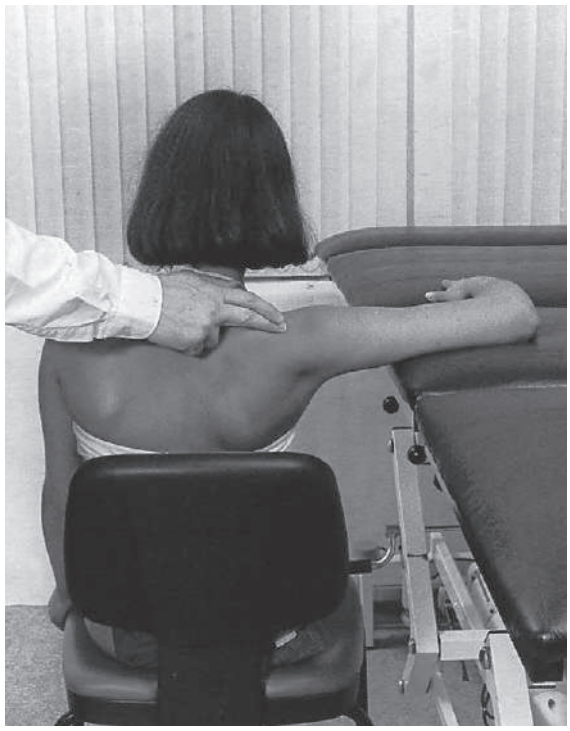
A painless inability to abduct the shoulder actively can be caused by either a complete rupture of the supraspinatus tendon or a neurological lesion.

Total rupture of the supraspinatus tendon

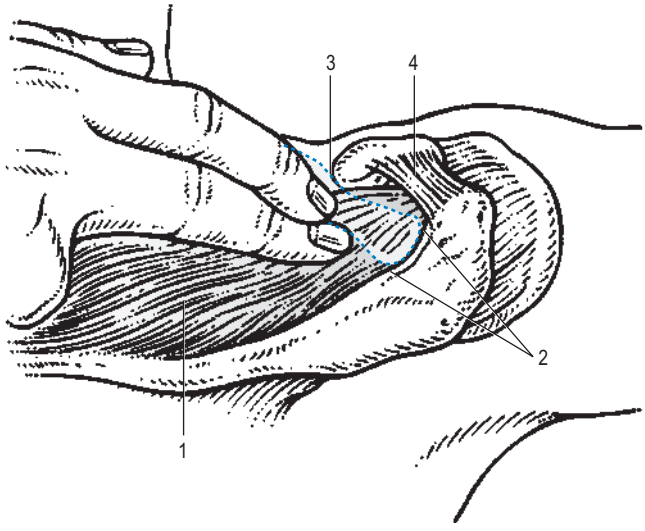
The supraspinatus muscle initiates active elevation of the arm and is active during the entire arc of abduction. It is responsible for about 50% of the torque and can abduct the joint without action of the deltoid.[94] Therefore a total rupture of the supraspinatus tendon presents as painless weakness on resisted abduction.

In massive tears of the supraspinatus, the patient is unable to abduct the arm actively. This is caused not only by loss of muscular power but also by loss of the passive stabilizing effect of the tendon.

Previously it was believed that the arm could not be abducted actively by the deltoid muscle alone if some contraction of the supraspinatus did not initiate the movement.[95] However, studies in which the suprascapular nerve and the axillary nerve were blocked have shown that both the supraspinatus and the deltoid muscles are capable of initiating elevation of the arm, in both sagittal and coronal planes.[96–98] This is not in accord with what is found clinically. When the supraspinatus tendon is massively ruptured, the patient is unable to initiate active scapulohumeral abduction. Starting from a position of 0° (the arm hanging by the side), the deltoid muscle only pulls the humerus upwards. Elevation is then only produced by rotation of the scapula in relation to the thorax and not by any movement between scapula and humerus. This is explained by the superior displacement of the humerus that occurs during active contraction of the deltoid in the absence of an intact



(a)



(b)

Fig 15.14 • (a) Deep friction to a musculotendinous lesion of the supraspinatus. (b) Anatomy: 1, supraspinatus muscle; 2, acromioclavicular joint; 3, clavicle (transparent); 4, coracoacromial ligament.

supraspinatus tendon.[99,100] In a normal situation the rotator cuff muscles form a supplementary musculotendinous glenoid which, in conjunction with the osseous glenoid, holds the humeral head stable. Experimental sections of the supraspinatus tendon also show the humeral head to move in a cranial direction until the superior humeral load is applied directly to the acromion (Fig. 15.15).[101] This phenomenon has been referred to as 'the spacer effect'[102] and is one of the most significant plain radiographic signs of massive cuff deficiency.[103]

Symptoms and signs

The lesion usually affects middle-aged and elderly people. In patients under 40 years of age a rupture is usually acute and results from indirect trauma, such as a fall on the outstretched hand.[104,105] In most cases the rupture is due to a chronic failure of the tendon: repeated failure of small groups of fibres leads to progressive weakness of the supraspinatus, making it increasingly susceptible to damage from lesser loads.[106,107] The observation that major cuff defects may occur without recognized injury has led to the concept of 'creeping tendon failure'.[108]

An acute trauma is usually accompanied by a sharp pain and followed by complete inability to raise the arm actively. The pain remains severe for the first few days and diminishes progressively, later becoming bearable without drugs but still sufficient to interfere with normal activities.

On clinical examination active elevation is limited to about 30°, which is fully accounted for by scapular rotation: no active humeroscapular movement is noted. A very pronounced painful arc is present when the elevation is performed passively. On muscular testing there is a complete but painless weakness of resisted abduction. After a few weeks the supraspinatus becomes atrophic; the deltoid maintains its normal size and strength. Although the patient cannot actively elevate the arm from 0°, active elevation becomes possible if the arm is passively moved through the first 30°.

Frequently, because the patient is unable to move the arm actively, an immobilizational arthritis will set in after a few months and a capsular pattern emerges (see p. 228).

Treatment

Because most total ruptures occur insidiously in the elderly, surgical correction to restore normal function will not be the first option.[109] Some defects cannot be repaired simply because they only offer 'rotten cloth to sew' (McLaughlin).[110] Many defects do not need to be repaired because they exist without causing much in the way of clinical symptoms.[111] However, surgery should always be considered in a younger patient with a significant acute tear in a previously normal shoulder,[112,113] and in the patient with a chronic tear associated with significant symptoms and not responding to conservative treatment. Fair functional results have been observed using open[114,115] and arthroscopic techniques,[116] but it is important to bear in mind the fact that repair does not restore the quality of the tendinous tissue. Reported recurrence rates after rotator cuff repair range between 15% and 90%.[117,118] Because rotator cuff integrity is important to its function,[119] long-term results of repair are better in younger patients with acute tears.[120,121]

For most supraspinatus ruptures the treatment of choice is conservative. The primary aim is to get rid of the pain. To that end, an infiltration with 10 mg of triamcinolone should be given at the remnants of the insertion into the humeral tuberosity to abolish the painful arc, following the same procedure as for uncomplicated supraspinatus tendinitis. The therapist should try to reach the inflamed tissue because infiltrating the gap is useless. Therefore the typical counterpressure on syringe and needle should be felt during the entire

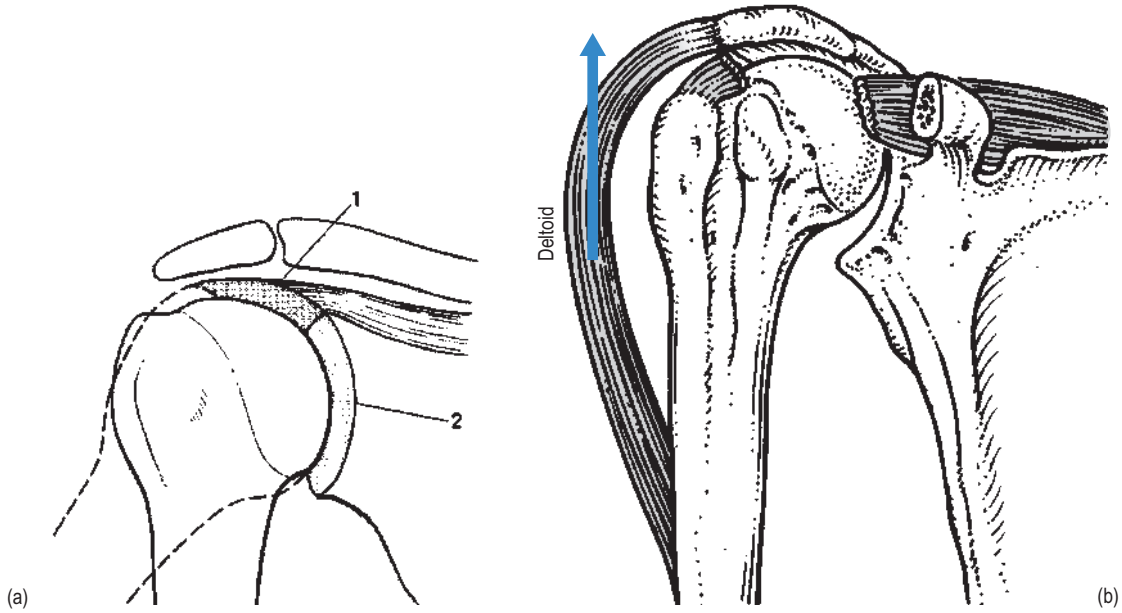


Fig 15.15 • (a) The glenoid: 1, tendinous; 2, osseous. (b) Spacer effect: massive rupture of the supraspinatus leads to a cranial subluxation of the humeral head during deltoid contraction.

procedure. Once the painful arc has gone, patients should be encouraged to use the arm normally. To lift the arm up, they should initiate elevation via a swinging movement of the trunk so that the arm is thrown laterally until it reaches the point where the deltoid muscle becomes effective. It is also wise to prescribe functional exercises for the antagonists of the deltoid and the remaining rotator cuff muscles. In spite of the slight disability, the function of the joint is usually good and the patient should be capable of doing light work.[122–124] Outcome studies report good to fair results in 60–90% of conservatively treated supraspinatus ruptures.[125–127]

Neurological lesions

Several neurological disorders may provoke painless weakness on resisted abduction.

Axillary nerve palsy

This may follow a subluxation of the humeral head. The axillary nerve innervates the deltoid muscle. Differential diagnosis is based on testing the strength of both the deltoid and infraspinatus muscles. Isolated painless weakness of abduction is found. A painful arc is not present, which differentiates this disorder from a supraspinatus rupture. Active elevation is difficult but possible (see online chapter *Nerve lesions and entrapment neuropathies of the upper limb*).

Suprascapular nerve palsy

This is most often the result of idiopathic neuritis, and seldom of an injury. The suprascapular nerve innervates both supra- and infraspinatus muscles. Weakness of abduction and lateral rotation is present. This disorder can be differentiated from a combined supra- and infraspinatus rupture by the absence of a painful arc (see online chapter *Nerve lesions and entrapment neuropathies of the upper limb*).

Fifth cervical root palsy

The C5 root innervates both spinatus muscles, the deltoid and the biceps brachii. Weakness on abduction and lateral rotation of the arm and on flexion of the elbow is present (see p. 155).

See (Fig. 15.16) for a summary of resisted abduction.

Resisted adduction

Pain

A painful but strong resisted adduction movement of the arm may be caused by a lesion of one of the adductor muscles, a sprain of the acromioclavicular joint or tendinitis of the long head of the biceps.

It may happen that isometric movements indirectly provoke pain in structures other than the ones that are supposed to be tested. Cyriax called this phenomenon 'transmitted stress'. During strong resisted adduction with the arm hanging alongside the body, the strong actions of the latissimus dorsi and pectoralis major pull indirectly on the acromioclavicular joint. Resisted adduction may therefore also provoke local shoulder pain in a chronic strain of the acromioclavicular joint. The pain will then be localized within the C4 dermatome and other passive tests will point to the acromioclavicular joint (see p. 241).

Pain on resisted adduction of the arm may rarely be caused by a tendinitis of the long head of the biceps at the glenoid origin. Cyriax arrived at this conclusion after finding that resisted adduction proved to be painful with the elbow in extension but not if it was kept in flexion. As a consequence, the structure at fault must overlie both shoulder and elbow

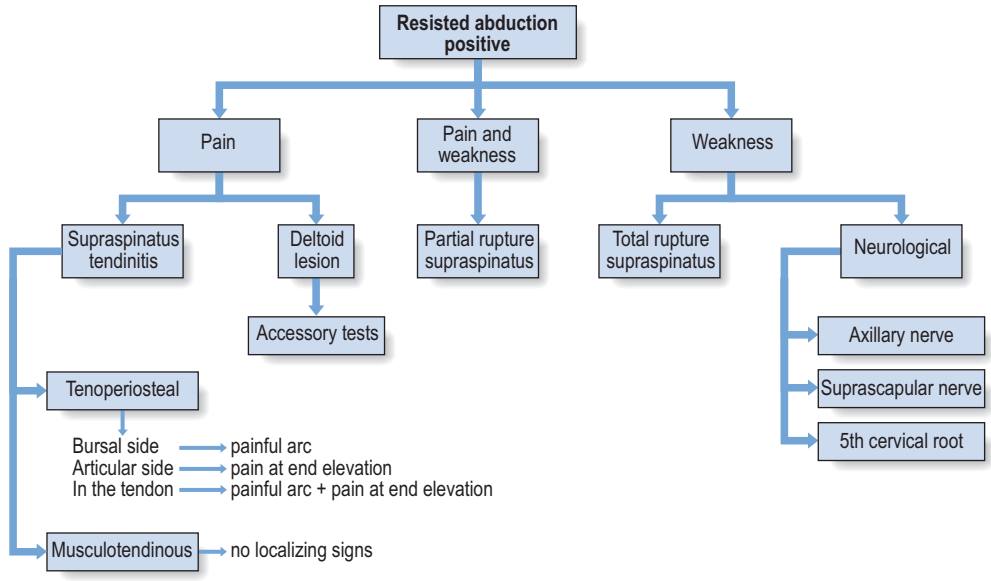


Fig 15.16 • Resisted abduction positive.

(see p. 269). The pain is mainly felt under the acromioclavicular joint.

More often, however, a positive test will indicate a lesion of one of the adductors: pectoralis major, latissimus dorsi, teres major and teres minor.[128] An important element in distinguishing between them is the localization of the pain. Pain in the thorax anteriorly suggests the pectoralis major and posterolaterally the other three muscles. Axillary pain may be the result of a problem in the pectoralis major, teres major or latissimus dorsi.

The four adductors can be more accurately differentiated by additional tests. Because the pectoralis major, latissimus dorsi and teres major are medial rotators and the teres minor a lateral rotator, resisted medial and lateral rotation of the arm is the next test to perform in order to arrive at an exact diagnosis.

The pectoralis major muscle can be tested on its own by resisted horizontal adduction with the arm forwards (Fig. 15.17a). The examiner stands on the painful side. One hand is placed on the patient's shoulder. With the other hand, the patient's arm is grasped just above the elbow and brought anteriorly to the horizontal. The patient is now asked to exert further medial force against the examiner's hand. The same can be achieved, but with loss of assessment of muscle strength, when the patient pushes both hands against each other holding both arms horizontally in front of the body.

Further differentiation is done by a resisted extension movement (Fig. 15.17b), which involves the latissimus dorsi, teres major and teres minor muscles. The patient tries to move the arm backwards with the arm hanging down and the examiner applying anteriorly directed counterpressure.

The only further differentiation required is between a lesion of the latissimus dorsi and the teres major. Because the latissimus dorsi also causes side flexion of the trunk and depression of the scapula, while the teres major does not, testing resisted side flexion towards the painful side and resisted scapular depression may be helpful.

Because lesions of the teres minor and teres major are extremely rare, these structures are not discussed in detail.

Pectoralis major

Lesions of the pectoralis major may be the result of overuse or of direct injury.

The lesion usually lies in the muscle belly, either in its lateral lower portion or in the fibres just below the outer portion of the clavicle (Cyriax:[86] p. 145). In rare instances, the pain is felt at the shoulder and radiates down the inner aspect of the arm. The problem must then be sought at the insertion at the crest of the greater tubercle.

It should be noted that in severe lesions of the pectoralis major, full passive elevation of the arm and resisted adduction and medial rotation are positive. In minor lesions, resisted medial rotation is sometimes negative.

The functional tests are followed by palpation, best performed with the patient's arm slightly abducted and the hand resting on the iliac crest.

Differential diagnosis is a rib fracture. In fracture in the anterior thorax, the same tests as in pectoralis major strain are painful. Additionally, all movements of the thoracic spine influencing the site of fracture are painful as well, as is deep inspiration.

Treatment

Infiltration of the muscle belly with procaine usually has a good therapeutic effect. Two or three infiltrations suffice. If not, or if the lesion lies at the insertion, infiltration can be replaced by deep transverse friction.

Technique: infiltration of the pectoralis major

The patient adopts the half-lying position and puts the hand on the affected side on the iliac crest. A 4 cm needle is fitted to a syringe filled with 10–20 mL of procaine 0.5%. After accurate palpation, the lesion is held between thumb and index finger and the needle is introduced between them obliquely

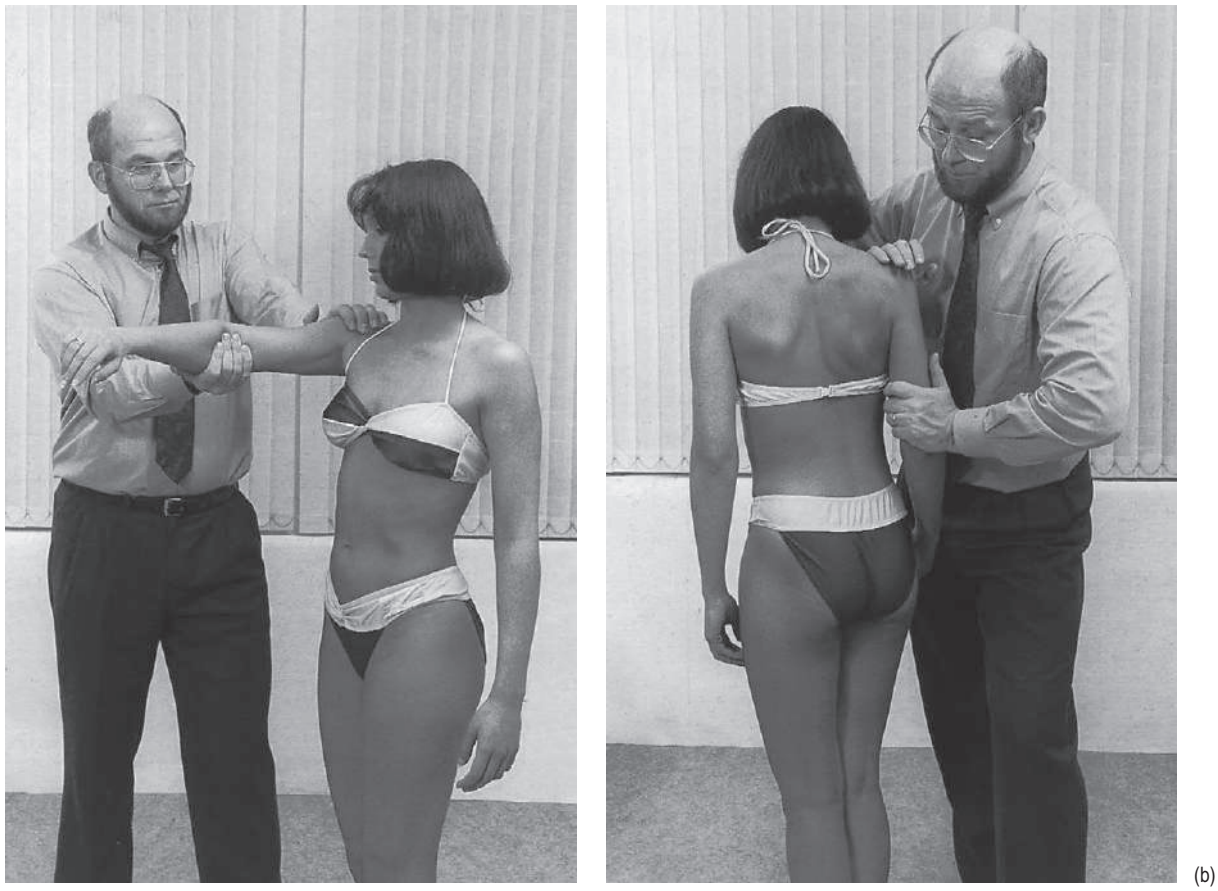


Fig 15.17 • Resisted horizontal adduction with the arm forward (a) and resisted extension (b).

and to its full length. The finger and thumb are kept in this position during the infiltration, to feel whether the product is deposited at exactly the right place (Fig. 15.18). The injection is given while withdrawing the needle. The syringe is emptied gradually, over about five withdrawals and reinsertions.

Technique: deep transverse friction to the pectoralis major

Palpation will demonstrate whether the lesion lies in the belly or the insertion of the muscle and so determine the friction method.

- *To the muscle belly*: massage is best given with the patient in the half-lying position and the hand resting on the iliac crest, so that the upper arm is somewhat abducted. This brings the pectoralis major more into prominence. The therapist sits facing the patient at the painful side. With the ipsilateral hand, the painful spot is taken between fingers and thumb and pulled laterally while pressure is applied (Fig. 15.19). This is the active moment of the friction. Massage is given for 20 minutes. Cure is normally obtained after 10 sessions.
- *To the insertion*: if palpation shows the lesion to lie at the insertion, deep friction is the only treatment. First the bicipital groove is localized, which is best done by putting the finger proximally between the minor and major

Fig 15.18 • Infiltration of the pectoralis major belly.

Fig 15.19 • Deep transverse friction to the pectoralis major belly.

humeral tubercles and in the long axis of the arm. By rotating the arm alternately medially and laterally, the groove can be identified. The therapist sits facing the patient at the painful side and places the contralateral thumb between the fibres of the deltoid muscle at the outer aspect of the bicipital groove. Counterpressure is applied over the dorsal aspect of the arm by the other fingers. Friction is given by pronation–supination movements.

Latissimus dorsi

Lesions of the latissimus dorsi are sometimes met with in gymnasts and water-skiers but are rare. They are usually found laterally at the uppermost part of the muscle belly. On clinical examination, full passive elevation of the arm and resisted adduction and medial rotation are painful.

Treatment

The responses to infiltrations with procaine and deep friction are equally good. The infiltration technique is the same as for the pectoralis.

Fig 15.20 • Deep transverse friction to the latissimus dorsi.

Technique: deep transverse friction to the latissimus dorsi

The patient lies prone, the hand on the iliac crest. The therapist faces the patient's painful side. The tender area is taken between thumb and fingers of the ipsilateral hand, and friction is performed by pulling the hand laterally (Fig. 15.20).

Painful weakness

Ruptures of the pectoralis major follow extreme muscle tension or direct trauma, or a combination of both. They have been reported in gymnastics or after a fall.[129,130] The lesion is more common in weightlifting and is caused in particular by a bench press.[131] The rupture usually occurs at the lateral fibres when the bar is at its lowest (loading of the fibres that are maximally stretched).[132,133]

In an acute injury there is sharp and burning pain, sometimes accompanied by significant swelling and ecchymosis. Passive elevation of the arm is very painful and somewhat limited. Resisted adduction and medial rotation of the arm are painful[134] and weak.

The functional tests are followed by palpation for tenderness and for a gap, and are best performed with the patient's arm slightly abducted and the hand resting on the iliac crest (see Fig. 15.19).

Treatment consists of surgical repair. Results are usually good to excellent.[135]

Painless weakness

A severe C7 root palsy can provoke painless weakness of adduction (see p. 156). Resisted extension of the elbow is also remarkably weak. The triceps reflex is sluggish or absent, together with some weakness on flexion of the wrist, more

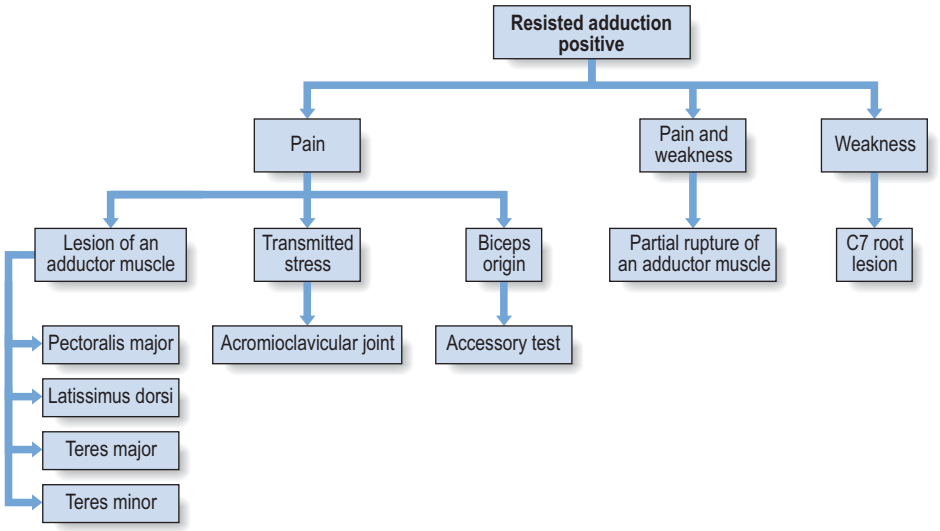


Fig 15.21 • Resisted adduction positive.

rarely on extension or on both. Numbness of the second and third fingers is usually present.

See Fig. 15.21 for a summary of resisted adduction.

Resisted lateral rotation

The main lateral rotators of the shoulder are the infraspinatus and teres minor. The former is by far the stronger of the two. The teres minor also adducts, so that resisted adduction offers the key to differential diagnosis.

Pain

If both resisted lateral rotation and resisted adduction are painful, the teres minor is at fault, although this is extremely uncommon. In contrast, when resisted lateral rotation is painful but other resisted movements are normal, the lesion lies in the infraspinatus.

Infraspinatus

There are some localizing signs of lesions of the infraspinatus. A painful arc indicates that the lesion lies at the superficial part of the tenoperiosteal insertion. If full passive elevation causes pain, the deep part of the tenoperiosteal insertion is affected (Fig. 15.22). If, apart from resisted lateral rotation, no other tests are painful, the lesion is in the body of the tendon or at the musculotendinous junction.[136,137] Palpation will disclose the exact location of the lesion.

Treatment

A lesion at the tenoperiosteal insertion of the infraspinatus can be treated either by deep friction or by infiltration with steroid. The general remarks made for the supraspinatus also apply here. The superficial part responds better to friction than does the deep aspect. It takes longer to achieve a cure by friction than by steroid infiltration but recurrence is less frequent. The body of the tendon is only treatable by deep friction.

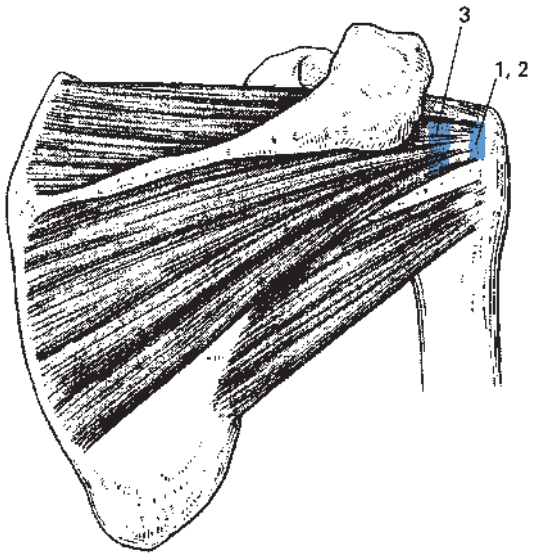


Fig 15.22 • Lesions of the infraspinatus; 1, superficial aspect tenoperiosteal insertion; 2, deep aspect tenoperiosteal insertion; 3, body of tendon.

Palpation

The patient lies prone, resting on the elbows, the shoulders vertically above them. The elbows are flexed to 90°. The upper arm of the affected side is brought into slight lateral rotation by asking the patient to hold the edge of the couch. In this position the whole infraspinatus structure is within the reach of the finger. In order to tighten the tendon and to bring it further laterally from underneath the acromion, the patient moves the shoulder slightly over towards the painful side.

First the spine of the scapula is palpated, then the posterior angle of the acromion and the lateral acromial edge. In the infraspinal fossa, the muscle belly of the infraspinatus is located and, when the fingers are moved further towards the greater tuberosity, the musculotendinous junction, the tendon and finally the tenoperiosteal insertion are located. The latter is

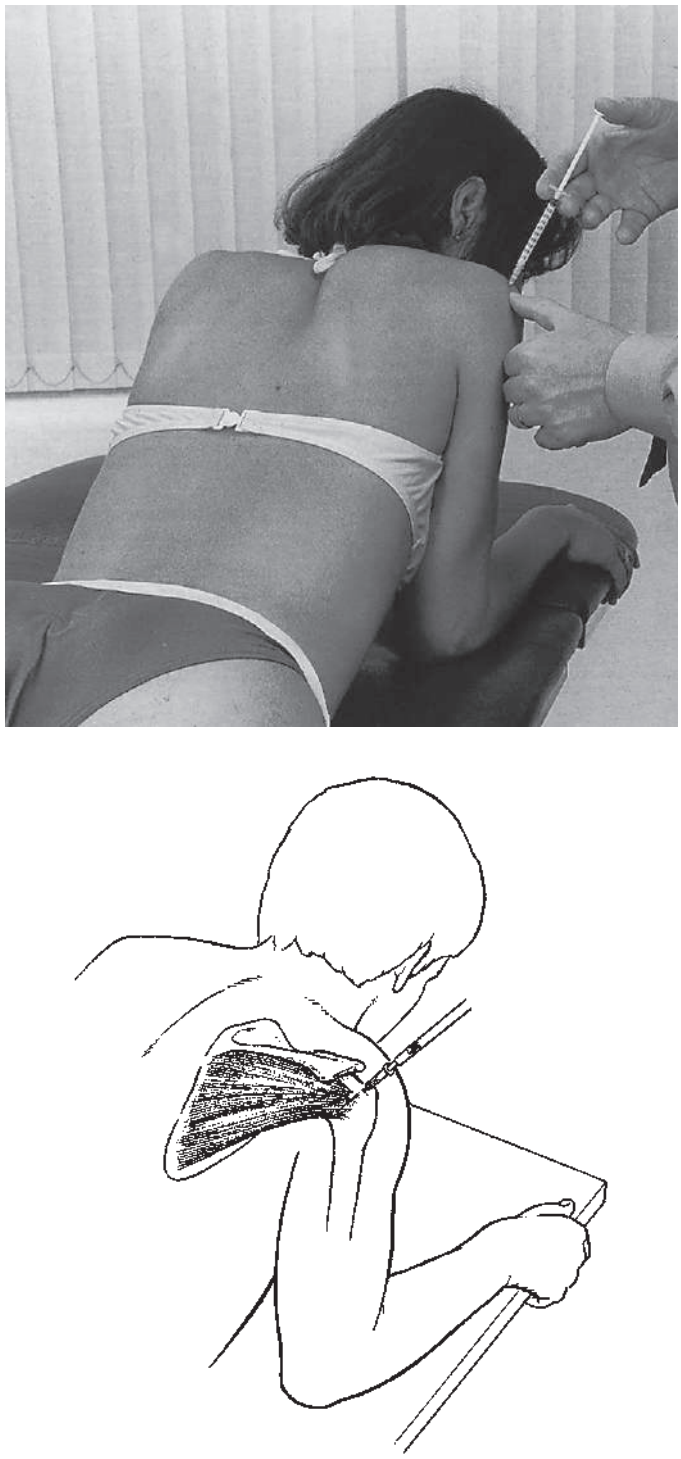


Fig 15.23 • Infiltration of the infraspinatus.

about 2 cm wide and lies partly lateral to the outer rim of the acromion and partly lateral behind the acromial angle.

Technique: infiltration of the infraspinatus tenoperiosteally

A 1 mL syringe is filled with 10 mg of triamcinolone and fitted to a 3 cm long needle. After the landmarks have been located, the needle is introduced in an anterocaudal direction, aiming at the tenoperiosteal insertion, which is reached after passing through soft tissue for about 2 cm (Fig. 15.23). Just before bone is reached, the tough tendinous resistance of the insertion is felt. The total amount is now injected in 5–10 slightly different sites. At each partial injection, strong counterpressure is felt.

The patient should rest the arm for about 2 weeks and is then re-examined. If pain remains, a second infiltration is given. Two infiltrations normally suffice.

Technique: deep friction to the infraspinatus

Deep friction is indicated in athletes, if infiltration has been unsuccessful or when the body of the tendon is affected.

The patient lies in the same position as for infiltration. The therapist stands at the painful side. The thumb of the ipsilateral hand is placed on the lesion, while counterpressure is applied with the other fingers at the front of the shoulder (Fig. 15.24). The body of the tendon feels like a cord, whereas the tenoperiosteal insertion feels more like a flat resistance just covering the underlying bone.

Friction is undertaken in a caudocranial direction by pronation–supination movements of the arm. The active moment is on supination. To avoid blisters, a piece of cotton wool between thumb and skin can be used: indeed, it may be necessary because in this position the skin is stretched, which allows the finger to glide over it too easily. Firm skin contact must be maintained.

Friction is given for 20 minutes, three times a week, and is usually curative after 10–15 sessions.

It may be difficult for the patient to hold this position for 20 minutes, so a break in treatment and sitting up for a few minutes should be allowed.

A specific case: abduction and lateral rotation against resistance are both painful

Both resisted abduction and lateral rotation are frequently painful on clinical examination and often there is a painful arc. This clinical pattern usually causes differential diagnostic difficulties that are not always easy to solve (Fig. 15.25).

No painful arc

If a painful arc is not present and both resisted abduction and lateral rotation are equally painful, the question is whether a supraspinatus or an infraspinatus tendinitis is present or whether both structures are inflamed. The test of resisted lateral rotation is first repeated, asking the patient to pay special attention to holding the elbow against the body when pushing the forearm sideways. Abducting the arm at the same time as resisted lateral rotation is performed could render the latter falsely painful when supraspinatus tendinitis is present. If the patient has difficulty in performing the test correctly, a sheet of paper should be placed between the elbow and the body during the test.

If both tests are still painful when correctly performed, they should be repeated in a supine position, which fully relaxes all the shoulder structures not intended to be tested. If one test becomes painless, the other one remaining painful, full value is given to the latter. For example, if resisted lateral rotation remains painful and resisted abduction becomes negative, the infraspinatus is at fault. If, on testing in the supine position, both tests remain painful, it is more likely that the

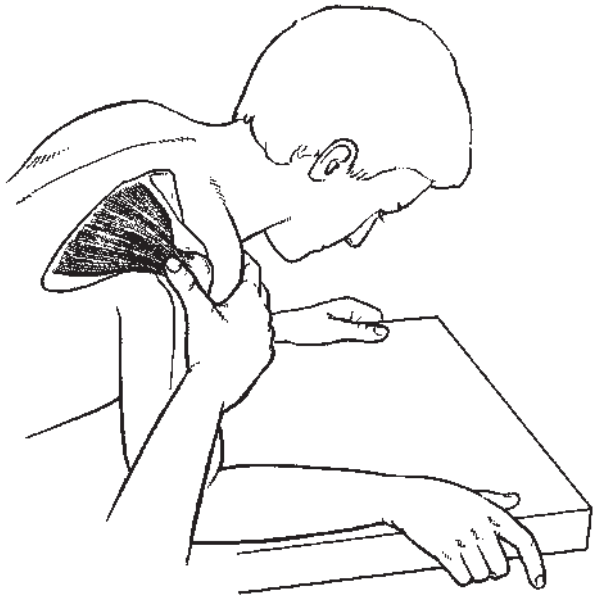


Fig 15.24 • Deep friction to the infraspinatus.

supraspinatus is at fault because this structure is more frequently affected. To obtain diagnostic certainty an infiltration with local anaesthetic is necessary and is the only way to determine whether the supraspinatus, infraspinatus or both together are affected.

Painful arc

If a painful arc is also found, then the following lesions may be present:

- Chronic subdeltoid bursitis
- Supraspinatus tendinitis

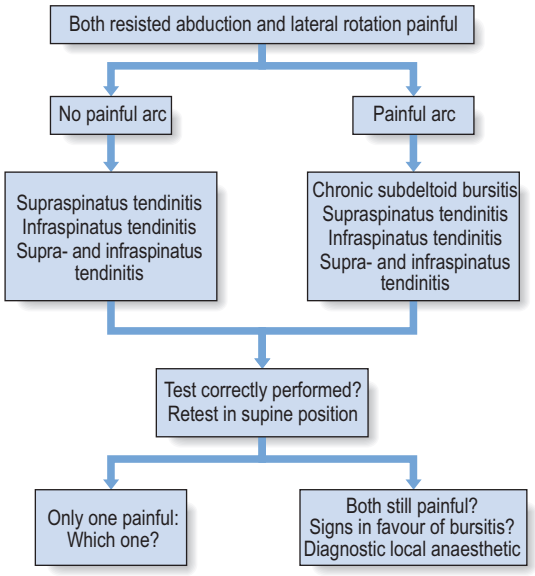


Fig 15.25 • Differentiation between subdeltoid bursitis and infra- and supraspinatus tendinitis.

- Infraspinatus tendinitis
- Tendinitis of both supraspinatus and infraspinatus.

If a painful arc is present, the tests should be repeated standing and lying.

Signs in favour of chronic bursitis

Presence of these signs makes it more likely that chronic bursitis is present:

- Very pronounced painful arc
- All passive movements painful at full range
- Tests are less painful in a supine position
- Resisted movements become less painful with axial traction, which prevents the humeral head approaching the coracoacromial roof
- Tenderness of the superficial part of the subdeltoid bursa on palpation
- Infiltration of the deep part of the bursa with local anaesthetic abolishes the pain.

Signs in favour of supraspinatus and infraspinatus lesions

Presence of these signs means it is more likely that tendinitis is the problem:

- Less pronounced painful arc
- Testing standing and lying are similarly painful
- Testing resisted abduction under axial traction is equally painful
- Tender spot in the tendon or tenoperiosteally
- Infiltration of the suspected tendon with a local anaesthetic diminishes the pain.

Painful weakness

Painful weakness on resisted lateral rotation is caused by a partial rupture of the infraspinatus tendon. The treatment

is the same as for uncomplicated tendinitis at the insertion. The same remarks as for the treatment of a partial rupture of the supraspinatus also apply here: infiltrations are not given in athletes but rather only if the patient can guarantee complete rest.

Painless weakness

If resisted lateral rotation is painlessly weak, differential diagnosis between the following lesions must be made.

Muscular disorder: total rupture of the infraspinatus tendon

This usually happens in patients over 50 years old who have suffered from overstrain or trauma, very often a fall.

Infraspinatus tears may present in isolation or in combination with a partial or total rupture of the supraspinatus tendon. The infraspinatus defect then propagates in a posterior direction from an established full thickness tear in the supraspinatus.[138]

As in supraspinatus tears, the lesion usually develops in patients over 50 years of age and is often asymptomatic (see p. 145). The lesion results from a combination of wear and tear and repetitive minitrauma. Sometimes the tear occurs suddenly during an acute overload or a fall.

Acute symptoms will be a painful arc on active and passive elevation and pain on full lateral rotation. Resisted lateral rotation is extremely weak but painless. The painful arc is caused by impingement of the tendon remnants between the greater tuberosity and the coracoacromial roof; the pain at the end of lateral rotation is caused by squeezing against the bulging posterior labrum. After some time (often many months), the inflammation decreases and the pain disappears. Resisted lateral rotation remains weak.

Chronic tears are characterized by marked and visible atrophy in the infraspinatus fossa. Secondary to the permanent loss of the infraspinatus, the patient can no longer bring the arm into full lateral rotation. This reduces the elasticity of the anterior capsule of the shoulder and results in a permanent isolated limitation of external rotation. The impaired external rotation leads to the typical 'hornblower's sign': bringing the hand to the mouth is only possible in internal rotation of the arm with accompanying excessive elevation of the elbow. Hornblower's sign has a 100% sensitivity and 93% specificity for irreparable degeneration of the infraspinatus.[139,140]

Treatment

When a painful arc is present, an infiltration is given in the tendinous remnants. For this, 1 mL of triamcinolone is infiltrated at the tenoperiosteal insertion, using the same technique as for uncomplicated tendinitis. It is repeated after about 2 weeks if the painful arc has not fully disappeared.

Abolishing the pain is only half the treatment. To prevent later limitation of lateral rotation, older patients are instructed to move the arm regularly into full lateral rotation with the help of the other arm or by twisting the trunk to the contralateral side while the forearm is stabilized against a fixed object.

Surgical treatment should be considered in younger patients with acute traumatic ruptures.

Neurological disorders

C5 root palsy

Resisted abduction of the arm and flexion of the elbow are also weak (see p. 155).

Suprascapular nerve palsy (see online chapter *Nerve lesions and entrapment neuropathies of the upper limb*)

Suprascapular nerve entrapment is usually an acquired neuropathy secondary to compression of the nerve in the bony suprascapular notch. Chronic mechanical irritation may be caused by overhead activities such as raquet sports, lifting and volleyball. Direct trauma, compression (e.g. backpacking) or a fracture of the scapula may also cause the lesion.[141] Sometimes it is idiopathic.

Symptoms are constant pain in the trapezius and scapular area for about 3 weeks, followed by functional disturbances in the arm (difficulties in performing overhead activities of the arm). Clinical examination reveals normal movement of neck, scapula and shoulder. Resisted movements show some painless weakness of abduction (although the supraspinatus is paralysed, the deltoid remains fully active) and gross but painless weakness of lateral rotation (the infraspinatus is completely paralysed).

Spontaneous cure is the rule in an idiopathic 'neuritis'. The pain disappears within 3 weeks, weakness within 6–12 months.[142]

During the last few decades, there have been several reports of isolated infraspinatus muscle paralysis in volleyball players. The reported incidence in high-level volleyball players is between 25 and 45%.[143,144]. Usually the finding is accidental and the disorder without any complaint: no pain, no loss of function, and even no reduced efficiency in playing volleyball.[145] The most plausible pathomechanism for this suprascapular neuropathy is traction or stretching of the nerve.[146] This traction injury may occur when repetitive overhead activities result in local nerve strain that exceeds the passive tolerance of the nerve.[147,148] In one study the players with suprascapular nerve entrapment displayed significantly greater shoulder mobility than those without.[149]

Neuralgic amyotrophy

This usually affects young males and starts with central neck pain, later spreading towards both shoulders and arms. Finally it leaves one arm and remains only in the other one. The pain is very severe and may last for about 6 months. Right from the onset, muscular involvement is visible. Often wasting is present together with a total palsy, not just weakness. It affects muscles of both sides, irrespective of segmental origin. Paralysis of the infraspinatus and the triceps, extensors of the fingers or extensors of the thumb is a common pattern (Cyriax:[86] p. 90) (see p. online chapter *Nerve lesions and entrapment neuropathies of the upper limb*).

Myopathy

Bilateral painless weakness of the upper limbs can be the result of myopathy affecting serratus anterior, deltoid and both

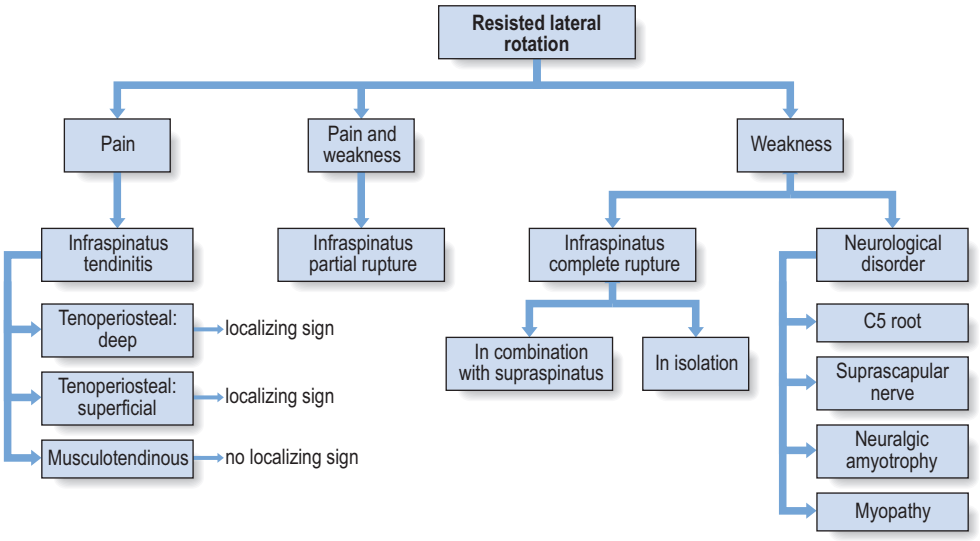


Fig 15.26 • Resisted lateral rotation.

spinatus muscles. If this is the case, then pronounced weakness and wasting of both spinatus muscles and of the serratus anterior muscle are present. Because of the muscular weakness there is sometimes some degree of immobilizational arthritis.

See Figure 15.26 for a summary of resisted lateral rotation.

Resisted medial rotation

Pain

Four different structures are tested on resisted medial rotation: subscapularis, pectoralis major, latissimus dorsi and teres major. The latter three are adductors as well and have been discussed previously (see above). If resisted adduction is painless, the lesion must lie in the subscapularis, the insertion of which is large and flat.

Subscapularis

The upper portion of the subscapularis inserts through a collagen-rich tendon into the lesser tuberosity. The rest (about 40%) of the tendon inserts into the humerus below the lesser tubercle and medial to the bicipital groove. Here the insertion is directly from muscle to bone.[150]

Tendinitis of the subscapularis can be localized tenoperiosteally in the cranial or caudal part of the tendon. Lesions in the muscle belly or in the tendon itself do not seem to occur. A lesion in the cranial part causes a painful arc because of impingement under the coracoacromial arch.[151] If the caudal portion is affected, the accessory test of passive horizontal adduction is painful as a result of pinching of the lesion between the lesser tuberosity and the coracoid process. Full active and passive elevation and full lateral rotation may be painful as well, because they stretch the subscapularis. Passive lateral rotation can even be so painful that it seems limited. To exclude arthritis, the examiner must gently try to bring the arm to full range, so as to confirm that limitation does not exist.

Treatment

Tendinitis of the subscapularis can be treated by friction or infiltration. Friction is a rather painful and difficult technique and so preference is often given to infiltration.

Technique: anatomical localization by palpation

The patient sits on the couch, the hand of the affected side resting on the thigh and the arm rotated slightly laterally. The bicipital groove is now located at the anterolateral aspect of the arm. This is best done at the proximal part, between the two tuberosities. In this position the lesser tuberosity points anteriorly, the greater tuberosity laterally. The examiner places one thumb longitudinally between the tuberosities. Using the other hand, the patient's arm is moved into lateral and medial rotation. On lateral rotation the lesser tuberosity contacts the thumb in the groove, while on medial rotation it is the greater tuberosity which comes in contact with it.

Once the lesser tuberosity has been identified, the finger is placed at the anteromedial aspect of it and, further distally, at the lesser tubercular crest. This is where the insertion of the subscapularis is found: partly on the lesser tuberosity, partly on its crest. The total width is about 3 cm.

The insertion does not offer any particular sensation to the palpating finger: it feels as hard as bone. Depending on the localizing sign (painful arc or passive horizontal adduction positive), the proximal or the distal half of the structure must be dealt with. Before any treatment is started, the affected part is carefully palpated to localize the most painful region. Palpation vaguely around the insertion must be avoided because this area is always tender.

Technique: infiltration of the subscapularis

A 3 cm needle fitted to a syringe filled with 1 mL of triamcinolone is inserted at the subscapularis insertion in the middle of the most painful part, and directed towards the bone (Fig. 15.27). Just before the humerus is reached, tendinous resistance is felt. As always, the infiltration is given in droplets. This time the needle is moved along a line in a craniocaudal direction over about 1.5 cm so as to include the whole lesion.

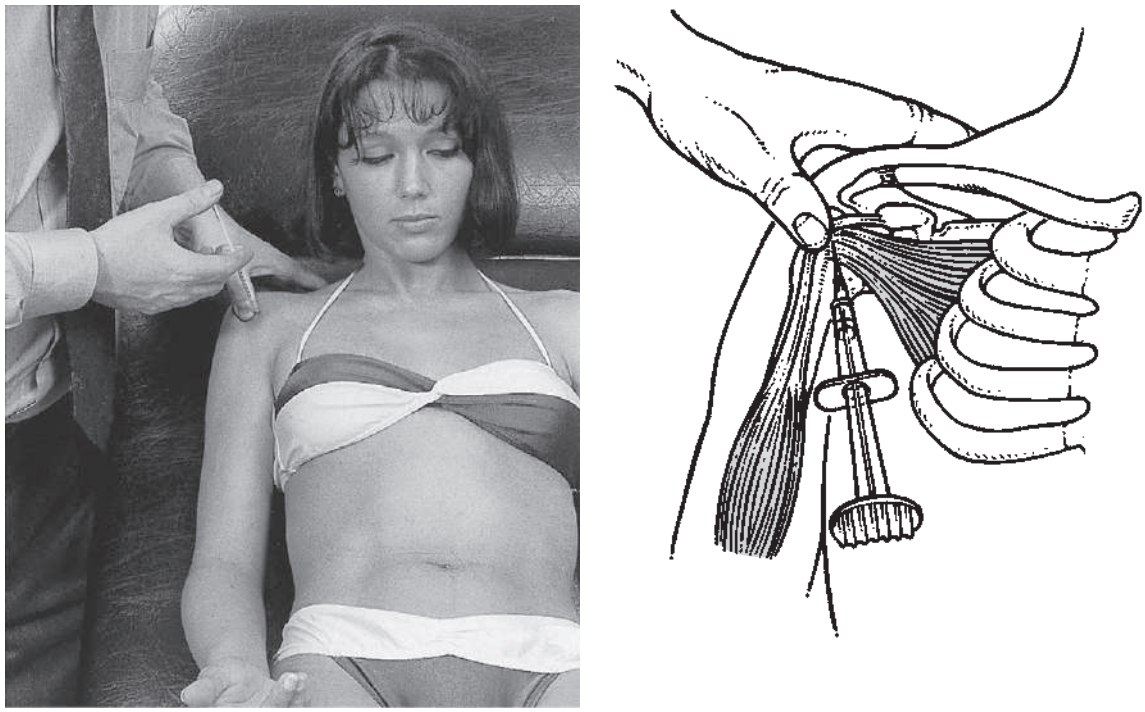


Fig 15.27 • Infiltration of the subscapularis.

Counterpressure is experienced during the infiltration. Care must be taken not to move the needle too far laterally, so as to avoid infiltrating the tendon of the long head of biceps, which could lead to its early degeneration.

The patient is told to avoid using the arm for about 2 weeks and then reassessed. If full cure is not attained, infiltration is repeated. Results are usually very good, as long as the correct point has been dealt with. Recurrences are less frequent than in supra- or infraspinatus tendinitis.

Technique: deep friction to the subscapularis

This form of treatment is mainly used in recurrences or in patients who refuse steroids.

The patient is brought into the half-lying position, the arm in slight lateral rotation. Palpation is done in the same way as for infiltration.

The therapist stands on the affected side and faces the patient. The contralateral hand is used. Beginning at the coracoid process, the thumb is brought just underneath it and the other fingers curved laterally around the arm, on to the back of the shoulder. The thumb is now pulled laterally while pressure is applied in a posterior direction. During this movement two tendinous structures are momentarily felt slipping under the thumb; these are the coracobrachialis muscle and the short head of the biceps. By moving the thumb yet further laterally, while maintaining some dorsal pressure, the anteromedial edge of the deltoid is reached and is pulled to the side. Finally the lesser tuberosity is contacted (Fig. 15.28a). The hand is now turned into a vertical position (Fig. 15.28b), still keeping the deltoid to the side.

Friction is given by a flexion–extension movement at the wrist, the active moment being when the thumb moves upwards. During the whole procedure the deltoid is kept to the side. This is a painful, difficult and, for the therapist, tiring technique. Progress towards cure tends to be slow and it may take about 1 month's friction before the patient starts to get better. Only two-thirds of patients can be fully cured in this way and, if no improvement has occurred after 1 month of friction, it is useless to continue and an infiltration should be given.

Painless weakness

Rupture of the subscapularis tendon

Tears of the subscapularis tendon occur either in isolation or as a propagation of a supraspinatus tear.[152] The former usually is the result of a hyperextension–external rotation trauma or follows an anterior dislocation of the shoulder.[153] The lower part of the muscle is mostly affected, where the insertion is more or less directly from muscle to bone. In the latter, the lesion is more proximal at the lesser tubercle, the distal part of the tendon remaining unaffected.[154,155] In traumatic rupture there may also be an excessive range of passive lateral rotation. Weakness of internal rotation can also be demonstrated by the 'lift-off' signs described by Gerber and Krushell: the patient places the arm in internal rotation with the dorsum of the hand on the sacroiliac joint. If the patient is unable to rotate the arm internally any further and lift the hand off the pelvis, incompetence of the subscapularis is suspected.[156]

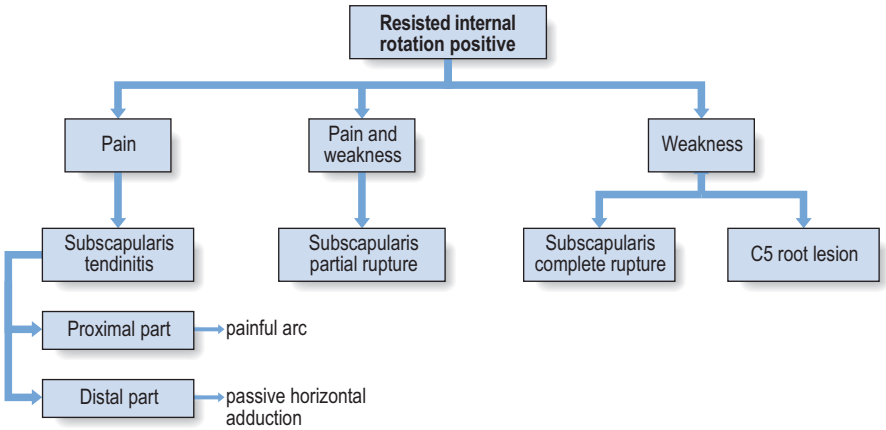
Treatment

Isolated traumatic tears of the subscapularis are best treated with surgery.[157] The surgical outcomes are less favorable when other tendons are involved.[158] If the condition remains painful, an infiltration with corticosteroid into the remnants at the tenoperiosteal insertion can be helpful.

(a) (b)

Fig 15.28 • Deep friction to the subscapularis. (a) Preparative position of the hand. (b) Position of the hand for friction.

Fig 15.29 • Resisted internal rotation positive.



Neurological lesion

Internal rotation is controlled by C5 and C6

In painless weakness of internal rotation, a lesion of these nerve roots should be ruled out.

See Figure 15.29 for a summary of resisted internal rotation.

Resisted elbow flexion

An examination of the shoulder is incomplete until the biceps has been tested. The muscle mainly moves the elbow but, as it crosses the shoulder joint, it can be the cause of pain around the shoulder as well. For this reason, resisted flexion of the elbow must always be included when the shoulder is assessed.

Pain

A positive test implicates the biceps. If pain is felt in the shoulder area, it is unlikely that the brachialis muscle is at fault, because a lesion in this muscle is more likely to cause pain around the elbow. The diagnosis can be confirmed by testing resisted supination of the forearm with the elbow flexed. If this is positive, the brachialis muscle is excluded.

The classification of biceps lesions can be divided into tendinosis of the biceps and biceps instability. The former may be an impingement lesion or occurs in isolation as a primary lesion of the biceps tendon.[159]

Lesions of the biceps can lie at different levels. Only those that can provoke pain at the shoulder are discussed here. These are tendinitis of the long tendon at the glenoid insertion, in its intra-articular course and at the bicipital sulcus (Fig. 15.30). Lesions of the muscle belly and further down are discussed in Chapter 19.

Lesion of the long bicipital tendon at the glenoid origin

Strain on the glenoid from the working biceps is greatest when the arm is in overhead abduction and lesions are usually caused in throwing athletes by traction injuries.[160,161] The pain is usually felt underneath the acromioclavicular joint but may radiate in the C5 dermatome. Besides pain on resisted flexion and supination of the elbow, resisted adduction of the arm will

also show positive. When the resisted adduction is tested again, this time with the elbow flexed, it becomes painless. This phenomenon is explained by the constant-length phenomenon on a structure spanning two joints (see Ch. 4): the degree of stress on the painful tissue in one joint depends on the position in which the adjacent joint is held.

Treatment

The condition can usually be treated successfully with one or two infiltrations of 20 mg of triamcinolone.

Technique: infiltration of the long bicipital tendon

The condition can be treated by infiltration only. The patient lies supine, the arm in 90° elevation, and the elbow flexed at a right angle. An assistant now turns the arm into 45° lateral rotation. In this position, the bicipital groove lies on a line extended from the anterior margin of the acromion.

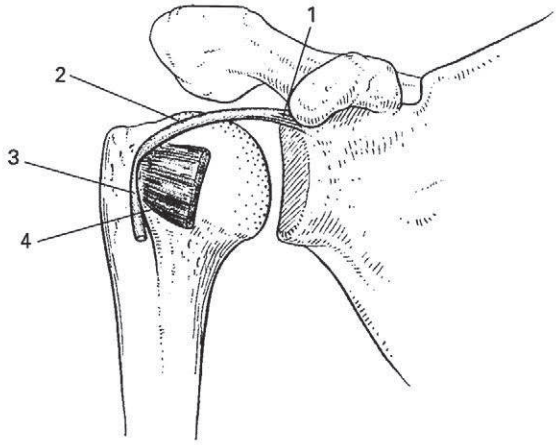


Fig 15.30 • Lesions of the biceps at the shoulder: 1, at the glenoid insertion; 2, at the intra-articular course of the tendon; 3, at the tendon in the sulcus; 4, subscapularis muscle.

A 2 mL syringe is filled with 20 mg of triamcinolone and a 4 cm needle fitted to it. One thumb is placed just beyond the acromion at the groove. The needle is inserted just distally to the thumb parallel to the bicipital sulcus and aiming at the glenoid (Fig. 15.31). It is then moved further in between the humeral tuberosities until it strikes the glenoid. A tough resistance is felt just before touching the bone. The steroid is infiltrated here in drops so that the whole insertion is treated. During the infiltration some counterpressure is felt.

The patient is re-examined after 2 weeks and, if necessary, the infiltration is repeated.

Intra-articular lesion of the long head of biceps

Pain on resisted flexion and supination of the elbow is found, together with a painful arc, which shows that the lesion lies at the intra-articular part of the tendon. As this part of the tendon lies intra-articularly, an intra-articular injection with 2 mL of steroid is required.

Lesion in the sulcus of the long head of biceps

The only positive sign is pain on resisted flexion and supination felt at the upper part of the arm. There is a full range of movement and a painful arc is absent. Palpation localizes the lesion in the sulcus.

Technique: deep friction to the long head

This lesion responds well to deep friction. The patient adopts the half-lying position, the palm of the hand on the thigh and the elbow at the side. The greater tuberosity then points laterally, the lesser tuberosity anteriorly. The sulcus lies in between the tubercles. In order to locate the groove precisely, the first part of the procedure for palpation for subscapularis tendinitis is performed (see above).

The therapist sits at the side, facing the patient. The patient's arm is put into a degree of lateral rotation. The thumb of the

Fig 15.31 • Infiltration of the long bicipital tendon at the glenoid.

Fig 15.32 • Deep friction to the long head of the biceps.

contralateral hand is put flat in the sulcus, the tip pointing cranially and the radial side in contact with the medial aspect of the sulcus. In this position the thumb lies on the medial aspect of the tendon. The other fingers apply counterpressure at the back of the arm. Pressure towards the bone is applied with the thumb.

With the other hand the therapist grasps the patient's lower arm, the elbow bent to 90°, and brings the arm into medial rotation until the radial side of the frictioning thumb comes in contact with the outer edge of the sulcus (Fig. 15.32). During this movement the tendon glides under the thumb and friction is achieved. The arm is now brought back into lateral rotation while releasing the pressure on the tendon (passive moment).

Treatment is carried out for 20 minutes, three times a week. Two to four sessions should be sufficient.

Alternative technique

Instead of the patient's arm being rotated, pronation–supination movements of the therapist's contralateral arm have the same effect. The active movement is on supination.

Snapping the long head of biceps

Biceps instability is seen most commonly in throwing athletes. Motion is often accompanied by a palpable snap or pop at a certain position in the arc of rotation.[162]

According to Slätis and Aalto, a rupture of the intertubercular transverse ligament allows for no appreciable medial or lateral movement of the tendon.[159] The key structure which guides the tendon in its groove is the medial portion of the coracohumeral ligament, which inserts at the lesser tubercle. It fills the space between the upper margin of the subscapularis and the anterior border of the supraspinatus. If it is transected, the long tendon of the biceps can easily be displaced medially.

Instability test

Dislocation of the biceps is tested with the biceps instability test of Abbott and Saunders.[163] The arm, abducted to 90° and fully rotated externally, is slowly brought to the side and rotated slightly internally. A palpable and even audible and sometimes painful click is noted as the biceps tendon, now forced against the lesser tuberosity, becomes subluxated or dislocated from the groove.

Rupture of the biceps tendon

Classically, the rupture takes place at the proximal part of the long head of the tendon. It is usually the result of degeneration and occurs spontaneously, provoking almost no discomfort. Distal ruptures are infrequent and are more often encountered in younger patients, usually as a result of local trauma, such as a direct blow.

Patients are usually male and over 50 years of age. Sometimes a loud crack is heard at the moment of rupture, with some transient pain in the upper arm. The most significant sign is the prominence that persists from the distal displacement of the muscle belly, best visible on contraction. On testing, no appreciable loss of muscle power is found.

Usually treatment is not required, although, in cases of less than 6 weeks' standing, surgical correction may be successful.[164]

Painless weakness

Fifth cervical root palsy

Painless weakness is found on resisted abduction and lateral rotation of the arm and on resisted flexion of the elbow.

Sixth cervical root palsy

Weakness is found on resisted flexion of the elbow (biceps and brachialis), resisted supination (supinator brevis), resisted extension and radial deviation of the wrist (extensor carpi radialis longus and brevis).

Resisted elbow extension

Pain

If this movement causes pain at the shoulder, it is logical to think of a lesion of the triceps, although this is very uncommon. On contraction of the triceps, the head of the humerus moves towards the acromion. If a lesion lies between one of the tuberosities and the coracoacromial roof, it can be pinched. Therefore pain on resisted extension of the elbow usually means the same as a painful arc. To exclude a lesion of the triceps definitively, palpation of the muscle can be used. If it is positive, it should always be followed by diagnostic infiltration with local anaesthetic because local tenderness is often present, even in normal circumstances.

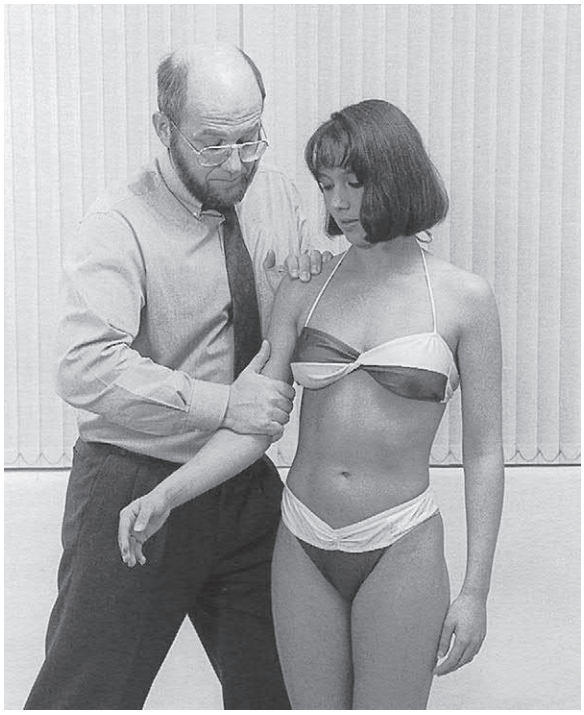


Fig 15.33 • Resisted flexion.

Painless weakness

Weakness on extension of the elbow is mainly the result of C7 root palsy caused by a C6 cervical disc lesion. Spontaneous pain is felt at the dorsal aspect of the arm and is increased by movements of the neck. Resisted extension of the elbow is painless. The latissimus dorsi (adduction of the arm) and the radial flexor, seldom the extensors, of the wrist may also be reduced in power. Theoretically the triceps reflex should be impaired, but in practice it seldom is.

Resisted flexion of the arm

The patient is asked to push the arm forwards against the examiner's hand, the latter applying counterpressure in the opposite direction (Fig. 15.33). Pain on this movement alone is the outcome of a lesion of the coracobrachialis muscle, which is rare. Although resisted adduction should also be painful, in practice it is not.

The lesion normally lies at the upper part of the coracobrachialis. Thus it can give rise to pain felt in the upper outer chest, radiating towards the inner side of the arm. It is treated by deep transverse friction.

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16

Clinical examination of the elbow

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Referred pain

One of the rules of referred pain is that the further distally the lesion lies, the more accurately the patient can localize it. It is therefore reasonable to start off with the clinical examination of the elbow and, only when this seems to be negative, to check the cervical spine, the shoulder girdle and the shoulder.

Pain referred to the elbow

Pain referred to the elbow from structures that lie higher up the limb is one possibility. The localization of the pain indicates the segment in which the lesion lies: the lateral aspect of the elbow is formed by the C5 dermatome, the anterior aspect by C6, the inner aspect by T1–T2 and posteriorly by C7.

Pain referred from the elbow

Pain referred from the elbow is uncommon. A lesion at the origin of the extensor carpi radialis brevis may give rise to pain radiating down the posterior aspect of the forearm to the hand as far as the third and fourth fingers. A lesion at the distal bicipital insertion radiates down the proximal and palmar aspect of the forearm. Proximal reference of elbow pain is very rare.

History

The history is not very important in elbow problems but some questions should be asked.

- *Where is the pain?* The location of the pain is usually closely related to the site of the lesion. When the patient indicates exactly where the symptoms are felt, all causes that cannot produce pain in that area are automatically excluded.
- *How did it all start?* Did the symptoms start spontaneously or has there been any trauma; if so, what type? If the onset was spontaneous, did it begin suddenly or gradually, or as the result of a particular activity?
- *What was the evolution?* Was there any change in the location, intensity or frequency of the painful episodes? Did the pain spread and, if so, where to? This may indicate the dermatome and, in consequence, the segment in which the lesion must be sought.
- *Is there any functional loss?*
- *Has the elbow ever been swollen?* If the swelling came on after trauma, how soon did it appear? Immediate general effusion is probably the result of a haemarthrosis; gradually increasing swelling usually indicates the presence of synovial fluid. Spontaneous swelling may be the result of an impacted loose body or a rheumatoid condition.

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Localized swelling may occur in bursitis or in some exceptional cases of tennis elbow.

- *What influences the pain?* Is the pain constantly present, or does it come on during or after either general or specific activity? In an arthrotic or arthritic joint the maintenance of a particular posture at the extreme of the possible range may become very painful. Release from this position is usually very uncomfortable. 'Twinges' when picking up objects (e.g. a telephone or a coffee pot) with an outstretched elbow is a well-known symptom in tennis elbow.
- *Are any other joints involved?* In rheumatoid-type conditions other joints may be affected.

Inspection

Inspection may reveal diffuse or local swelling: arthritis causes a more generalized effusion, whereas other lesions (bursitis and tendinitis) may give rise to more localized swelling. There may also be redness of the skin or muscular atrophy.

Functional examination

The examination consists of 10 tests: four passive movements and six resisted movements.

Passive movements

The passive movements (Fig. 16.1) are used to examine the inert structures: the joint, the capsule, the capsular ligaments and the bursae. It is also clear that, by passively testing the elbow, one also indirectly stretches or pinches muscular and tendinous structures.

The range of movement is ascertained and the end-feel noted.

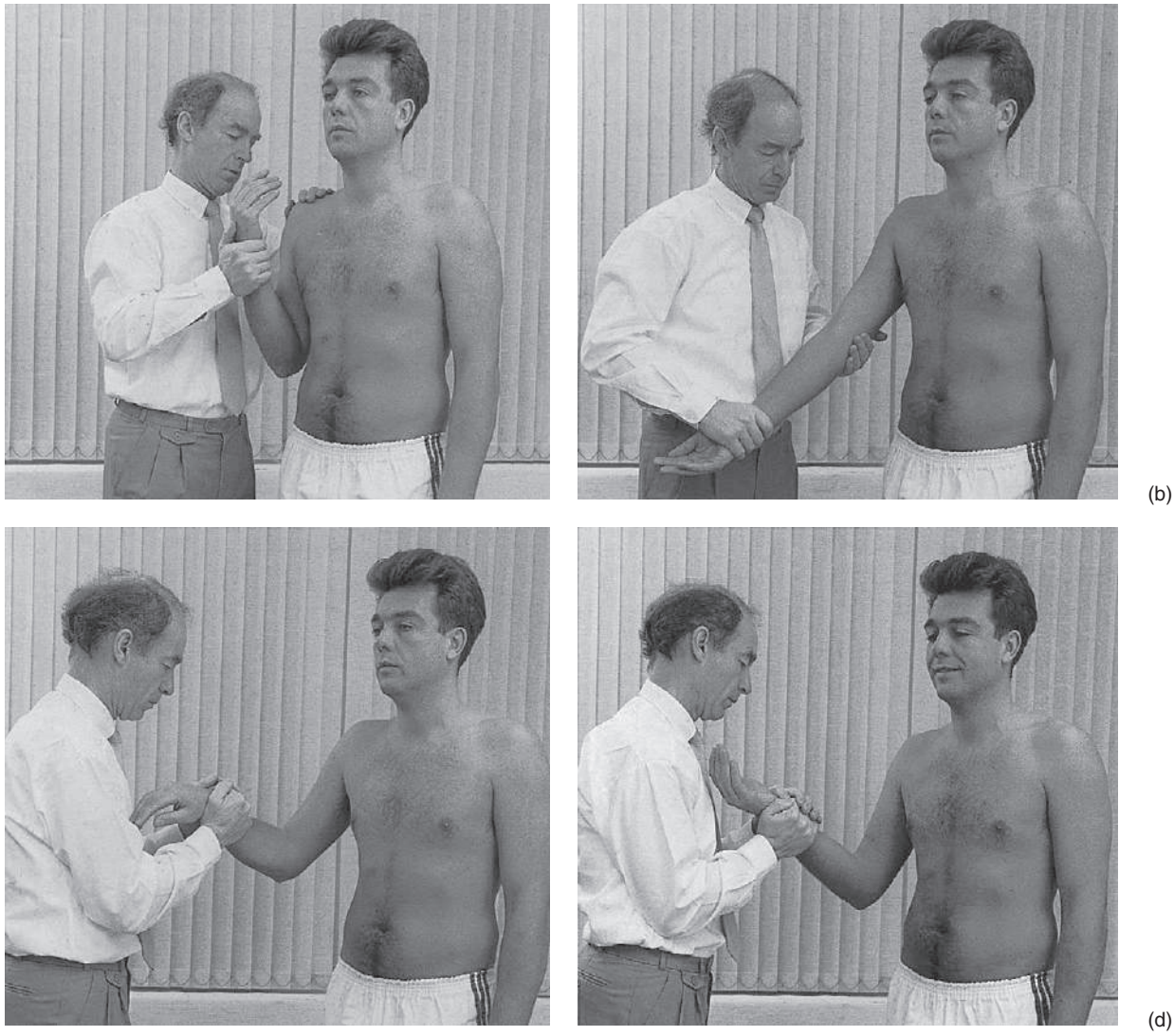


Fig 16.1 • Passive movements: (a) flexion, (b) extension, (c) pronation, (d) supination.

The four passive tests give the examiner an idea of the behaviour of the inert structures around the elbow. The pattern that is formed suggests a lesion of either the capsular type – arthritis, in the joint between forearm and humerus or in the upper radioulnar joint – or the non-capsular type.

Passive flexion

The examiner places the contralateral hand at the dorsal aspect of the patient's shoulder to prevent the body from trying to move backwards in order to escape from the pain. The ipsilateral hand takes hold of the patient's forearm just proximally to the wrist and moves the joint as far into flexion as possible (Fig. 16.1a).

The normal end-feel is one of tissue approximation as the muscles of the forearm come in contact with the brachial muscles at an angle of about 160°.

Passive extension

With the contralateral hand, the examiner takes hold of the patient's upper arm at the level of the olecranon. The ipsilateral hand is put at the distal end of the patient's forearm. Both hands move in opposite directions so as to extend the elbow (Fig. 16.1b).

The normal end-feel in passive extension is hard and bony, caused by the olecranon coming into contact with the posterior aspect of the humerus, and by tightening of the anterior capsule of the joint. As the forearm now lies in line with the upper arm, the angle of extension is 0°. In instances of over-extension, the angle may become 5–10°.

Passive pronation

The elbow is bent to a right angle. The examiner stands in front of the patient and grasps the distal forearm just proximal to the wrist with both hands. The heel of the contralateral hand is placed at the palmar aspect of the ulna, the fingers of the other hand at the dorsal aspect of the radius. A simultaneous movement of both hands presses the wrist into full pronation (Fig. 16.1c).

The extreme of movement is characterized by an elastic, capsular end-feel as the 85° range is approached. Together with passive supination, this movement tests the integrity of the upper radioulnar joint. Pain at the end of the range may also be a localizing sign in tendinitis of the insertion of the bicipital tendon on to the radial tuberosity, because of pinching of the tendon between the radial tuberosity and the ulna.

Passive supination

The position of the examiner's hands is slightly changed: the heel of the ipsilateral hand applies pressure at the dorsal aspect of the ulna and the fingers of the other hand pull at the palmar aspect of the radius. The forearm is twisted into supination as far as it goes (Fig. 16.1d).

Normally the capsular end-feel is noted at about 90°.

Resisted movements

The same four movements are repeated but against isometric resistance to examine the contractile structures (Fig. 16.2).

Resisted flexion

The patient holds the forearm in supination. The examiner puts the contralateral hand on top of the patient's shoulder to prevent it from moving upwards during the contraction. The other hand is placed on the distal forearm with the examiner's forearm held vertically, to prevent the patient's forearm from moving as the flexor muscles are contracted (Fig. 16.2a).

The muscles thus tested are the brachial biceps, the brachialis and the brachioradialis. The brachialis is a strong flexor and the brachioradialis is clinically unimportant.

Resisted extension

Again the contralateral hand is placed on top of the patient's shoulder. The other one is placed vertically under the patient's forearm and prevents the arm from moving downwards (Fig. 16.2b). If necessary, in a very strong patient, the examiner's own elbow may be supported on the thigh.

The muscles that are tested with this movement are the triceps and, secondarily, the anconeus muscle.

Pain at the shoulder during this movement has the same applications as painful arc (see online chapter *Disorders associated with a painful arc*).

Resisted pronation

The patient's forearm is held in the neutral position between pronation and supination. In order to prevent any movement during the resisted movement, the examiner's hands should be placed as follows: the ipsilateral hand, held in supination, is placed under the patient's distal forearm; the contralateral hand, held in pronation, is placed on top. The patient is asked to perform a pronation movement and resistance is supplied by the heels of both hands (Fig. 16.2c).

This movement mainly tests the pronator teres muscle but will also be painful in the presence of golfer's elbow.

Resisted supination

The hands are placed in a slightly different position from the previous test so that there is resistance against the palmar aspect of the distal ulna and the dorsal aspect of the distal radius (Fig. 16.2d).

In this test, the brachial biceps is tested with respect to its secondary function, and also the supinator brevis muscle.

Resisted tests of the flexors and extensors of the wrist

There are two muscle groups – *the flexors and extensors of the wrist* – that control wrist movements but lie at the elbow and therefore can give rise to symptoms around this joint. They should be systematically tested in the elbow examination.

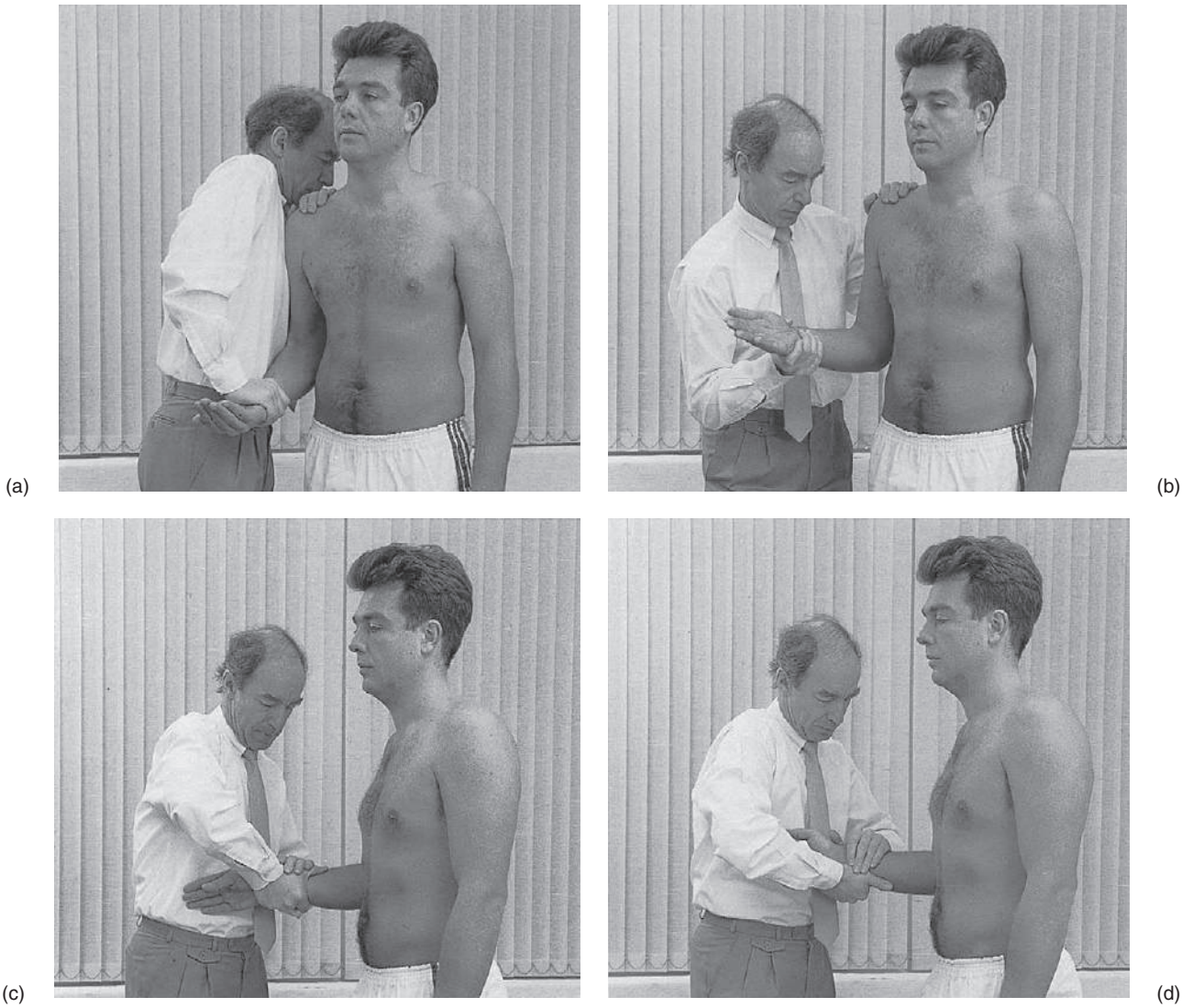


Fig 16.2 • Resisted movements: (a) flexion, (b) extension, (c) pronation, (d) supination.

The patient's elbow is held in extension, so as to put maximum stress on these structures (Fig. 16.3).

Resisted flexion of the wrist

The patient's hand is held palm downwards. The examiner passes the contralateral arm under that of the patient and grasps the forearm just proximally to the wrist in order to fix the upper limb. The upper arm lies under the patient's elbow and holds it in full extension. The hand of the other arm is now brought into the palm of the patient's hand and resists the patient's attempt to flex the wrist (Fig. 16.3a).

The following muscles are tested: flexor carpi radialis, flexor carpi ulnaris, flexor digitorum superficialis and flexor digitorum profundus.

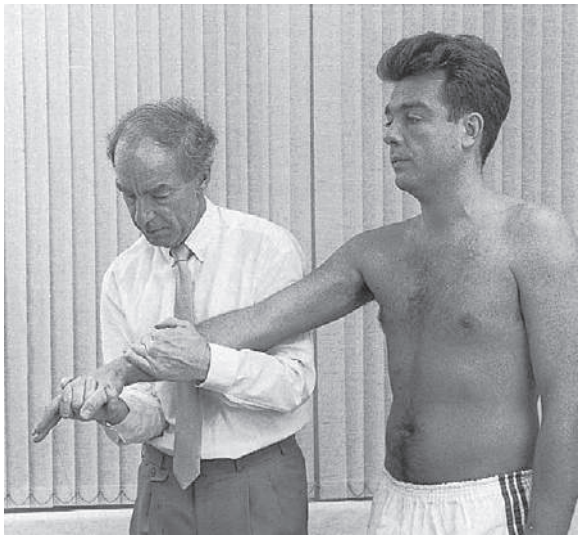
Resisted extension of the wrist

The patient remains in the same position as in the previous test. The examiner puts the hand on top of the patient's hand to resist an attempt to execute an extension movement (Fig. 16.3b).

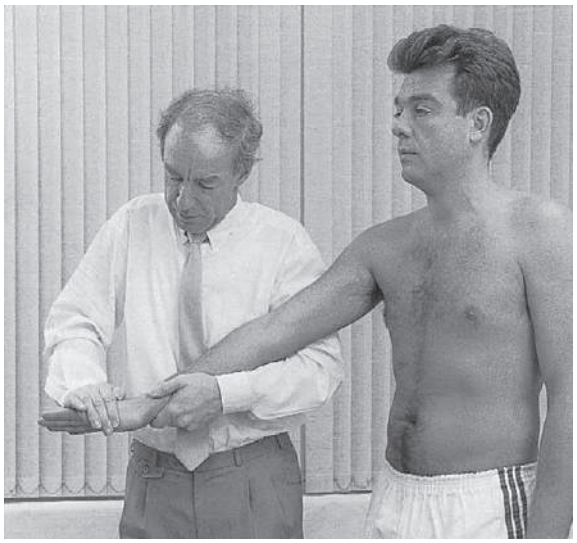
The muscles tested are the extensors of wrist and fingers: extensor carpi radialis longus and brevis, extensor carpi ulnaris, extensor indicis proprius, extensor digitorum communis and extensor digiti minimi.

Palpation

After the clinical examination (and only if it is likely to supply more information), the elbow is palpated.



(a)



(b)

Fig 16.3 • Resisted movements: (a) flexion and (b) extension of the wrist.

The elbow joint can be palpated either at rest or during movement, in order to detect warmth, swelling, synovial thickening (palpation laterally over the head of the radius), crepitus or clicking. The muscle or tendon at fault is palpated to define the exact site of the lesion.

Accessory tests

Resisted extension of the wrist with the fingers actively flexed

The patient is asked to flex the fingers actively by pressing the fingertips into the palm of the hand. Resisted extension of the wrist is now executed, as already described (Fig. 16.4).

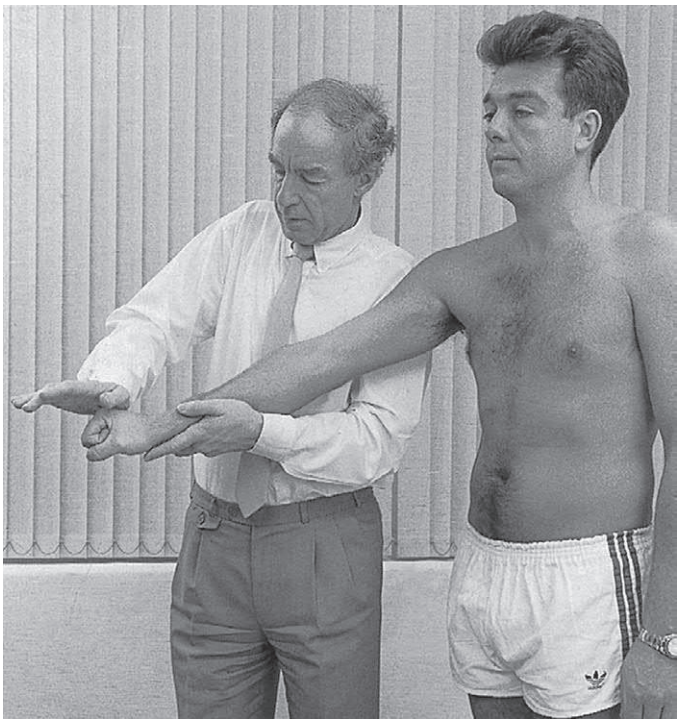


Fig 16.4 • Accessory test: resisted extension of the wrist with the fingers actively flexed.

Resisted radial and ulnar deviation

With the elbow in extension and the wrist in the neutral position between flexion and extension, radial and ulnar deviation are tested against resistance (Fig. 16.5). These tests differentiate between a lesion of the radial extensors or flexors of the wrist or of the ulnar extensors or flexors.

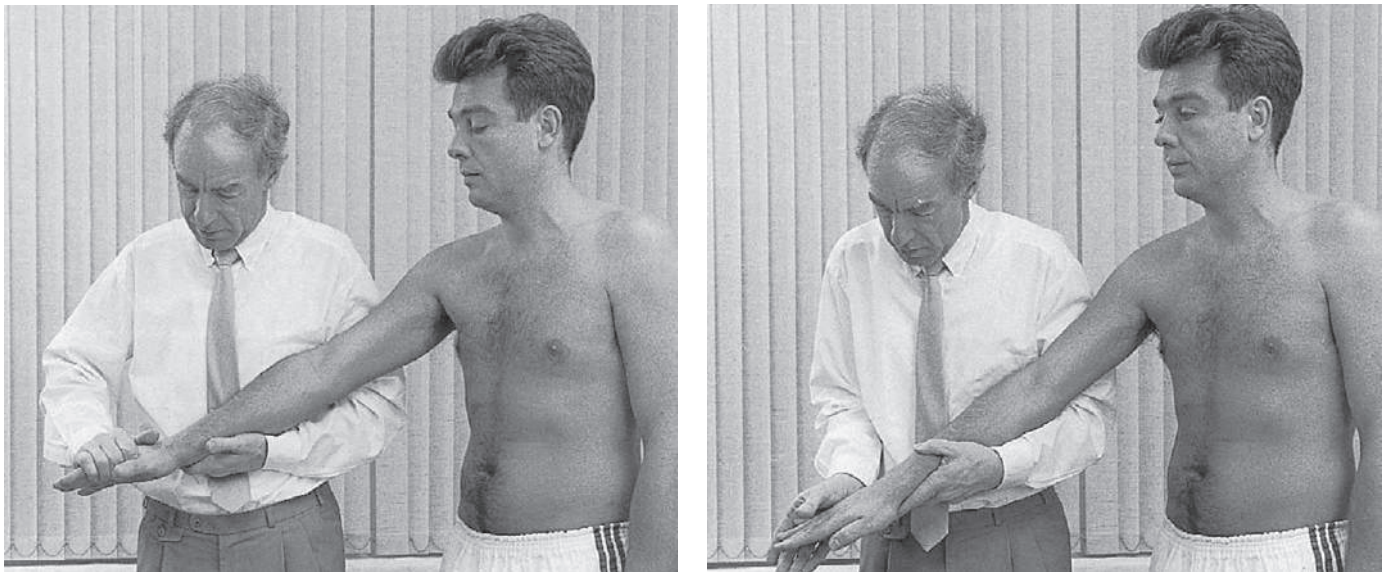
Resisted extension and flexion of the fingers

When a lesion of the finger extensors has been diagnosed, the examiner may test resisted extension of each finger in turn (Fig. 16.6a) to find out which tendon is at fault.

If a finger flexor is affected, resisted flexion of each finger (Fig. 16.6b) may disclose the exact tendon.

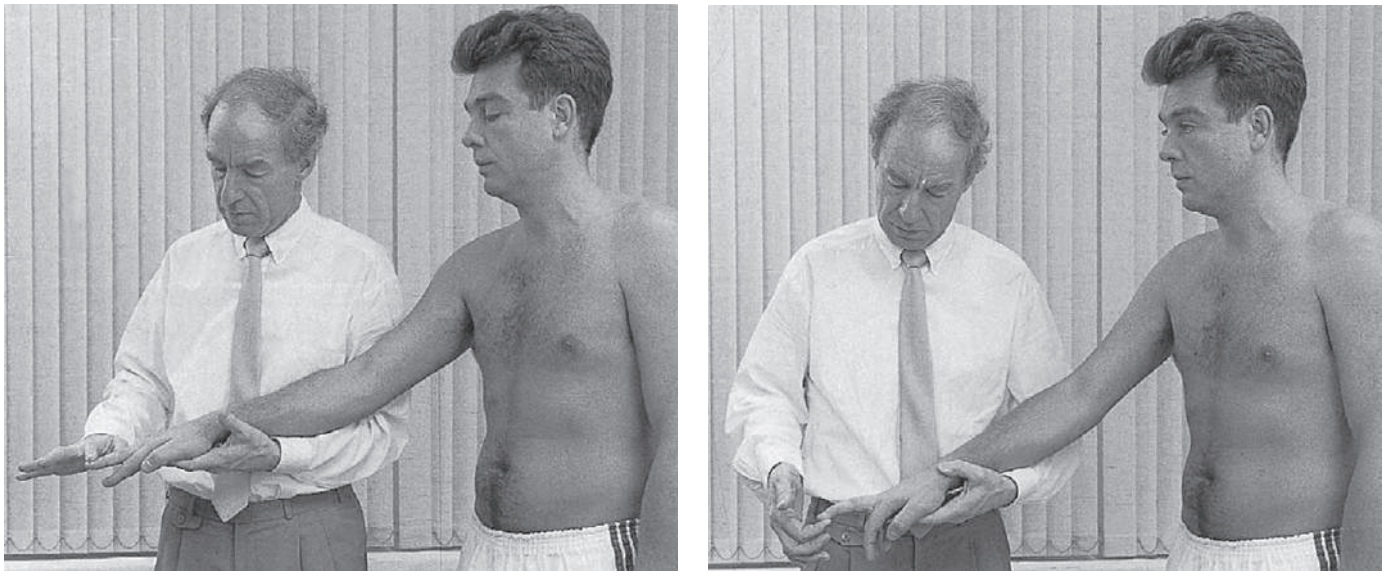
Resisted supination in extension

In order to differentiate between lesions of the brachial biceps and the supinator brevis muscle, this test should be performed. When the arm is brought into extension, the action of the biceps is diminished considerably but the supinator contracts normally.



(a) (b)

Fig 16.5 • Accessory tests: resisted (a) radial and (b) ulnar deviation.



(a) (b)

Fig 16.6 • Accessory tests: resisted (a) extension and (b) flexion of the fingers.

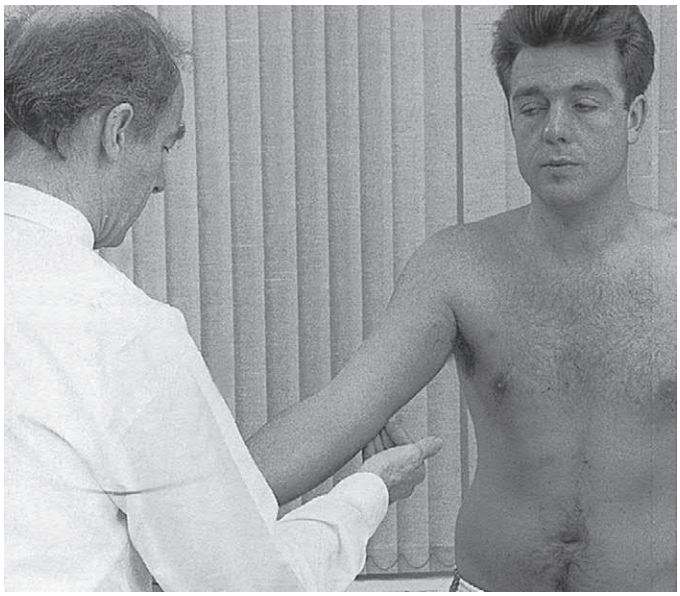


Fig 16.7 • Accessory test: Tinel's sign.

Box 16.1

Summary of clinical examination

1. History
2. Inspection
3. Functional examination

Passive tests
- Flexion
- Extension
- Pronation
- Supination

Resisted tests
- Flexion
- Extension
- Pronation
- Supination
- Flexion wrist
- Extension wrist

4. Palpation
5. Accessory tests

Tinel's sign

Percussion to the ulnar nerve in the groove between the olecranon and the medial epicondyle (Fig. 16.7) gives rise to distal paraesthesia in the territory of the ulnar nerve – in the forearm and the hand – Tinel's sign. This test can be used to assess the progress of regeneration of the sensory fibres of the nerve. The most distal point where the pins and needles are felt indicates the limit of regeneration.

The clinical examination is summarized in Box 16.1.

Interpretation of the clinical examination of the elbow

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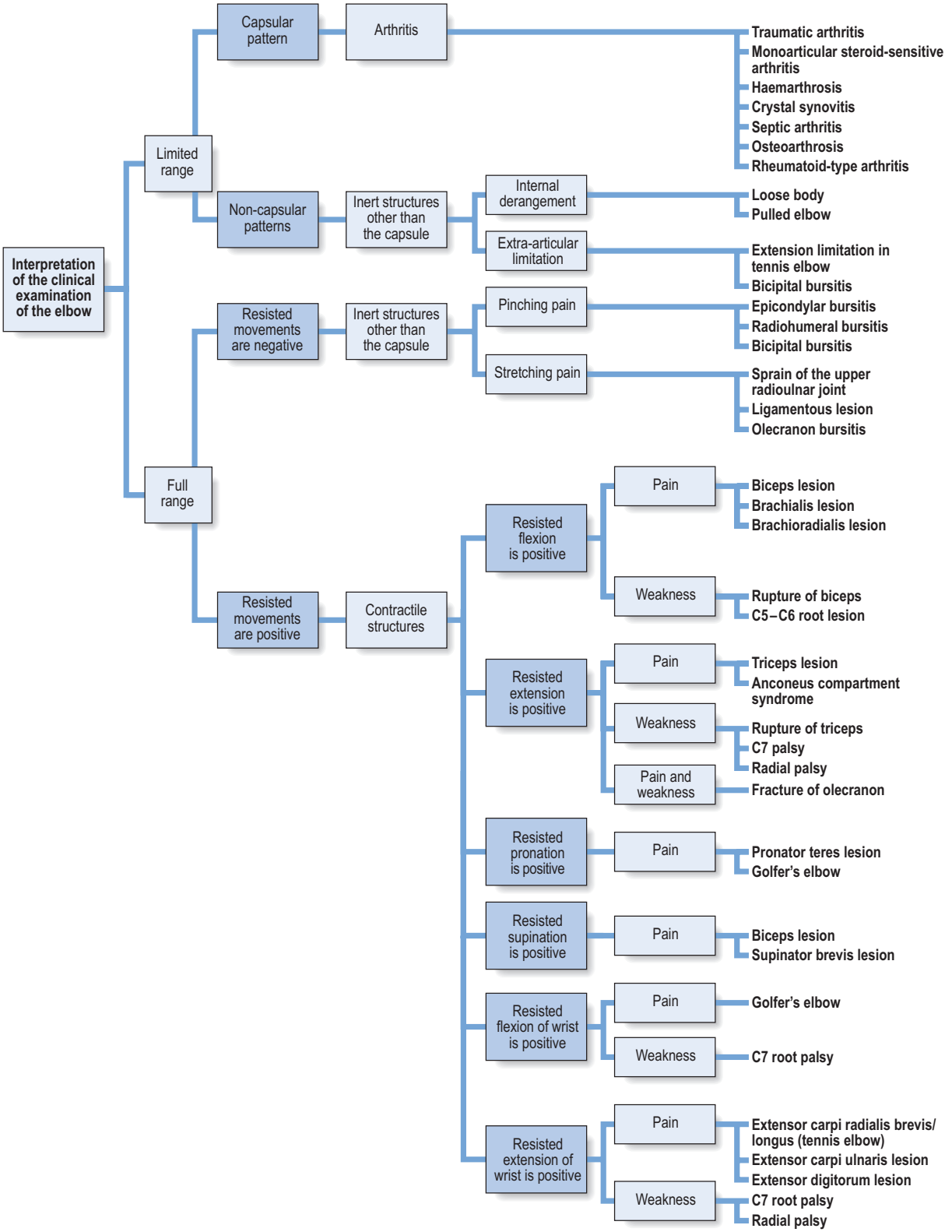

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18

Disorders of the inert structures

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Limited range of movement

Capsular pattern

The capsular pattern at the elbow is characterized by limitation of flexion and extension (Fig. 18.1), flexion usually being more limited than extension, although equal limitation of both movements does occur. Rotations remain full and painless except in advanced arthritis, in which they can be painful at the end of the range.

The following conditions are the most common.

Traumatic arthritis

A traumatic arthritis presents as an isolated condition at the joint between humerus and ulna; hence there is isolated impairment of flexion and extension. The arthritis can be the result of either trauma (not necessarily severe) or – in middle-aged or elderly patients – overuse of the joint.

The patient usually states that, immediately following an injury or the day after doing heavy work or exertion, the elbow hurts diffusely. The pain is quite constant, and there is stiffness and difficulty in moving the joint.

Clinical examination shows a capsular pattern: flexion and extension movements are markedly limited and painful; rotations are of full range and painless.

On palpation, some swelling may be detected. If the swelling came on immediately after an accident, it is probably caused by blood and this should be aspirated at once. If not, the effusion is secondary and disappears as soon as the arthritis subsides. Also, a positive fat pad sign on a lateral radiograph – a response to distension of the joint capsule – is indicative of intra-articular fluid.[1,2]

There are two situations that are worthy of attention.

Fracture of the olecranon

The olecranon lies superficially and is therefore very vulnerable. Injury to the elbow, and especially a fall on a bent elbow, may result in fracture of the olecranon. It is, of course, tender to the touch and marked articular signs are found on examination: warmth, swelling and limitation of passive movement in the capsular pattern.[3,4] Resisted movements are also positive in that isometric extension, an action of the triceps muscle, is painful and weak (see also p. 453). Radiography confirms the fracture and its type: it is mostly displaced but stable, and then requires surgery. When it is not displaced, immobilization suffices.[5]

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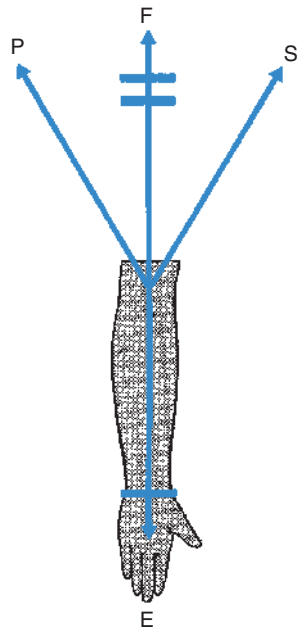


Fig 18.1 • Capsular pattern. F, Flexion; E, extension; P, pronation; S, supination.

Fracture of the head of the radius

Radial head fractures account for about 30% of all elbow fractures and mostly occur as a result of falling on to an outstretched hand.[6] They are most common in females.[7]

Should pronation and supination both hurt in an acute traumatic arthritis at the elbow, the head of the radius is almost certainly fractured. A radiograph confirms the diagnosis.

Treatment options for radial head fractures include conservative treatment, excision, open reduction–internal fixation, and arthroplasty, all depending on the type of fracture and the degree of displacement.[8,9]

Warning

Traumatic arthritis in combination with pain on pro/supination and/or painful weakness during isometric testing is suggestive of an intra-articular fracture.

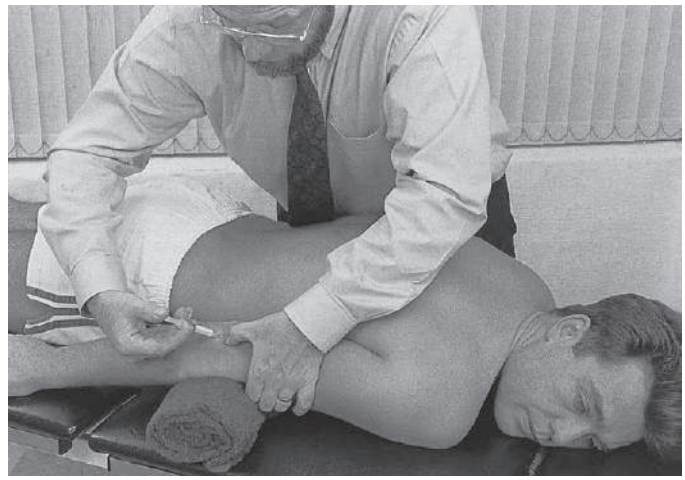
Treatment

Traumatic arthritis recovers spontaneously but may take several months. Treatment with massage and/or exercises only irritates the joint and is therefore contraindicated.

The treatment of choice consists of two intra-articular injections of 20 mg of triamcinolone acetonide. The injection is given when the patient is first seen. The arm is kept in a sling for a couple of days. A week later a second injection is given and the joint should have recovered in 2 weeks' time.

Technique: intra-articular injection

The patient lies prone on a couch with a small pillow under the elbow. The arm is held by the side with the forearm fully supinated. In this position the joint lines between humerus and radius and the radial side of the olecranon are both easily felt. A 2 mL syringe is filled with triamcinolone acetonide 10 mg/mL and a 2 cm thin needle is fitted. The needle is inserted at the joint line and aimed slightly obliquely under the olecranon (Fig. 18.2).



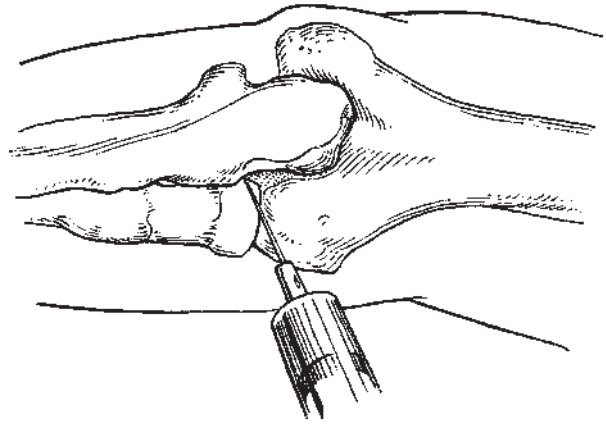


Fig 18.2 • Intra-articular injection.

Rest in flexion

Another valuable treatment is rest in flexion. It can be used in those patients who cannot tolerate an injection. As soon as the patient is seen, the elbow is immobilized in as much flexion as possible by means of a collar-and-cuff bandage. Every day the elbow is flexed more, until full movement can be achieved; thereafter it is held in this position for 2 weeks. The elbow is then rested in slightly less flexion. Three days later the joint is re-examined and, if the range of flexion is still full, the forearm is allowed to extend a little further. Some 6 weeks later, the patient reaches the stage in which the arm can be worn in a sling. After 2 or 3 months, movements of the elbow should be full and painless. Another static progressive splinting method by means of a turnbuckle splint has proved to be useful in the treatment of long-standing post-traumatic stiffness of the elbow.[10,11]

Arthrosis

This condition may come on spontaneously in late middle age[12] and is often bilateral. It may also occur as the result of a fracture or dislocation.[13] Intra-articular distal humerus fractures, for example, are most often associated with the development of degenerative joint disease over time.[14] Repeated minor injuries[15,16] or a loose body in the joint may also account for early arthritic changes.[17]

The patient, most often a male,[18] complains that, after he uses his elbow excessively, the joint aches slightly. He may also find the inability to fully straighten the arm inconvenient.

On examination a slight capsular pattern is found with a hard end-feel on flexion. The end-feel on extension is also hard but this is because of capsular contracture and is not the normal bone-to-bone end-feel. There may be coarse crepitus. The radiograph may show the arthrotic changes, although a positive radiograph can also be compatible with full range and painless function at the joint.

The differential diagnosis is neuropathic arthropathy,[19] which presents with gross painless limitation of movement in the capsular pattern.

An arthrotic joint does not of itself give rise to the sort of symptoms that warrant treatment. What may happen is that, on top of the arthrosis, a traumatic arthritis develops as the result of overuse. This can be treated as outlined earlier. Loose bodies are quite common and may also complicate the arthrosis (see below).

Monoarticular steroid-sensitive arthritis

A seronegative arthritis may occur, confined to one elbow joint. The pain is spontaneous in onset and the elbow very soon starts to swell.

A marked capsular pattern is found and, in due course, some limitation of pronation and supination may occur. In the acute and subacute stages, the end-feel is of muscle spasm. The joint is warm to the touch and synovial thickening can be found on palpation over the head of the radius laterally. In advanced cases muscle atrophy may also be seen. If, after some years, crepitus is present, it is of the silky type.

On radiography, decalcification and later erosion of cartilage may be visible.

Treatment with intra-articular triamcinolone suspension (see earlier) is symptomatically very effective in monoarticular steroid-sensitive arthritis; it stops the pain but produces hardly any change in the amplitude of movements. The patient may be pain-free for many months, whereupon the injection can be repeated without fear of steroid arthropathy because the elbow is not a weight-bearing joint.

Crystal synovitis

Gout (uric acid crystals) and pseudogout (calcium pyrophosphate crystals) seldom affect the elbow joint during a first attack (in only 4.5% of gout cases)[20] but more frequently cause acute olecranon bursitis.[21] As disease develops, polyarticular attacks may occur, in which case the elbow joint is affected in 30% of cases.

The sudden unprovoked onset and the shiny red appearance of the joint are characteristic.

The following diagnostic criteria may be useful in gout. When uric acid crystals are found in the synovial fluid during microscopic, chemical or histological examination, or tophi are seen on the ears, the diagnosis is certain, as it also is when two of the following four criteria are present: history of a typical attack of gout at the big toe; history of two typical attacks of gout at another joint; clinical picture of tophus; remission within 48 hours of the acute attack after the administration of colchicine or phenylbutazone.[22]

Haemarthrosis

Haemarthrosis may occur after injury to the joint, especially an intra-articular fracture or a direct contusion of the joint capsule, or, less commonly, in haemophilic patients. Bleeding into the joint leads to gross swelling and a marked capsular pattern.[23] Aspiration must be carried out immediately to avoid destruction of cartilage.

Rheumatoid-type arthritis

Polyarticular rheumatoid arthritis may affect the elbow joint. Apart from the gross swelling, rapidly ensuing limitation of extension is typical.[24] The elbow is also one of the sites of predilection, together with the knee and shoulder, for chondromatosis. Pigmented villonodular synovitis occurs most commonly at the knee, followed by elbow and ankle.[25]

In these and the other rheumatoid-type arthritides, systemic medication is required, and sometimes even a surgical approach. Good results have been reported with total elbow arthroplasty.[26–33]

Septic arthritis

A bacterial infection of the elbow joint is always very serious. It may not only lead to total destruction of the joint but may also be life-threatening.[34]

It can be the result of an open injury to the joint (e.g. open fracture), penetration of a foreign body (e.g. rose or bramble thorns during gardening or fruit picking) or direct inoculation of a bacterium during intra-articular injection, especially injection of a steroid suspension. It can also be caused by haematogenous dissemination from focal infections: dental abscess, cystitis, urethritis, skin infections. These causes are very dependent on the patient's resistance to infections: patients with diabetes, renal failure or a deficient immune system (e.g. rheumatoid arthritis) are more likely to suffer from haematogenous dissemination followed by a septic arthritis.[35–37]

Symptoms start suddenly and are easily recognized. The joint shows the signs of a hyperacute inflammation: a great deal of pain, gross swelling, redness, warmth and gross limitation of movement in the capsular pattern. There are also general

symptoms, such as high fever, a toxic appearance, pallor, loss of appetite and rigors.

Treatment consists of systemic antibiotic therapy and daily local aspiration[38] and drainage by arthroscopy.[39]

Tuberculous arthritis

Elbow tuberculosis is a rare disease which accounts for 1–3% of all cases of osteoarticular tuberculosis.[40] The diagnosis is very difficult to make because of the insidious onset with mild and non-specific local or systemic symptoms. The radiological findings are also non-specific in the early stage. Tuberculosis of the elbow is therefore easily misdiagnosed as degenerative arthritis or rheumatoid arthritis.[41] Several months after the onset of symptoms, there is pain at night, and examination reveals a gross capsular pattern and a spastic end-feel. The radiograph reveals periarticular osteopenia, bone erosion and joint space narrowing.[42] On haematological testing, the erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) are elevated.[43]

The gold standard for the diagnosis of tuberculous arthritis is identification of *Mycobacterium tuberculosis* either directly or after culture of the synovial fluid.[44]

Non-capsular pattern

Limitation of flexion or extension in isolation

Loose body in the joint

A loose body in the elbow joint is not uncommon and may hinder normal movements. It prevents the joint either from moving into full flexion, leaving extension free (Fig. 18.3), or from moving into full extension, leaving flexion free (Fig. 18.4). It then changes the hard end-feel of extension into a rather soft one.

Three different clinical pictures can be considered, depending on the age group in which they appear: adolescents, adults or the elderly.

In adolescence

A loose body is a common cause of elbow trouble in adolescents and is the only non-traumatic cause of arthrosis encountered in a young person. The condition does not occur before the age of 14 and usually results from osteochondritis dissecans, mostly on the humeral capitellum,[45,46] or an intra-articular chip fracture, conditions that may lead to exfoliation of one or more fragments of bone covered by articular cartilage.[47] Osteochondritis dissecans is not uncommon in young female gymnasts with hyperextension and valgus of the elbow.[48–50] It also affects young pitchers or athletes involved in high-demand, repetitive overhead activities.[51,52]

The history is quite typical: the young person states that the elbow joint locks suddenly, usually in flexion. It is impossible to straighten the elbow to full range because of the pain. In a few days, the condition gradually subsides. One or more of these attacks may have occurred in the past, with pain-free episodes in between.

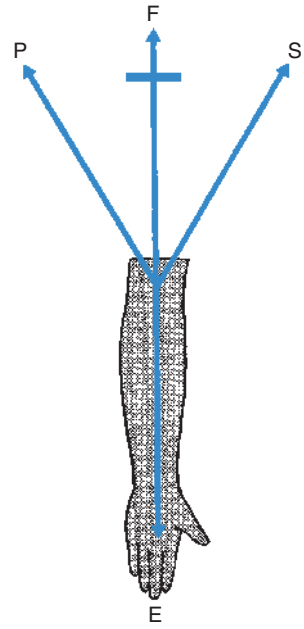


Fig 18.3 • Non-capsular pattern with limitation of flexion.

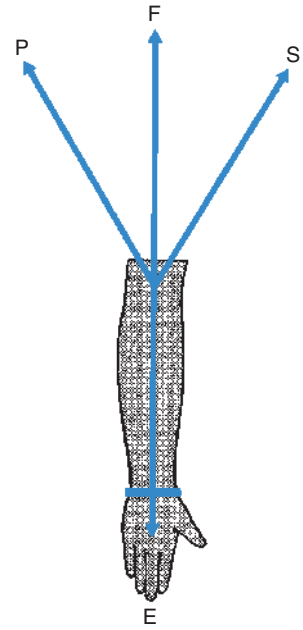


Fig 18.4 • Non-capsular pattern with limitation of extension.

Clinical examination during an attack shows a non-capsular pattern: limitation of passive extension with a soft end-feel. During a pain-free period, examination is negative; the diagnosis is suggested by the typical history.

Diagnosis can be confirmed by anteroposterior radiography performed with the elbow in 45° of flexion,[53] because in this

position the X-ray beam is almost parallel to the gap between the fragment and the underlying capitellar bone. Recently, magnetic resonance imaging (MRI) has been suggested for assessing osteochondritis dissecans,[54] and sonography also seems to be effective.[55,56]

If the patient is seen during an attack, manipulative reduction can be carried out. However, arthroscopic[57,58] or surgical[59] removal of the loose piece(s) should always be advised for the following reasons: as the loose body has an osseous nucleus and still lies within its nutrient synovial fluid, it may grow and the condition may worsen at each attack. It is also important to realize that the loose body is considerably larger than it may seem on a radiograph, as it is covered with radiotranslucent cartilage. If surgery is not performed, loose bodies may finally cause gross arthrosis.[60] Limitation of extension may considerably hinder activities and should therefore be prevented.

In a normal joint in adulthood

The cause of loose body formation in adults is usually traumatic, the injury having chipped off one or more pieces of cartilage. Middle-aged patients who develop a loose body may have multiple cartilaginous fragments in the joint that do not show on radiography; these do not tend to cause arthrosis.

The history is not as typical as it is in young people; the patient does complain of attacks of pain at the elbow, during which exertion increases the pain, but gradual unlocking in the following days does not occur; hence the importance of the clinical examination. Unless these patients are properly examined, they are thought to suffer from tennis elbow.

A very clear non-capsular pattern is found with limitation of either flexion or extension, depending on the position of the fragment. If the loose body lies in the triangle formed by the humeral capitellum, the head of the radius and the base of the coronoid process of the ulna, extension is slightly limited, while flexion remains full and painless. The end-feel on passive extension is soft. When the loose piece of cartilage lies anteriorly, flexion is quite limited, the fragment catching between the anterior aspect of the humerus and the tip of the coronoid process (Fig. 18.5).

A loose body that limits extension can usually be reduced. Manipulation under strong traction shifts the loose piece of cartilage to a position at the back of the joint. It then no longer blocks movement, which becomes normal again. The manipulation can be repeated each time derangement occurs. Nothing else should be done, unless recurrence is very frequent, in which case removal during arthroscopy would be a possibility.[61,62]

A loose body that limits flexion cannot be reduced by manipulation, but limitation of flexion, unless gross, is not a major concern. The alternatives in this case are: arthroscopic or surgical removal or nothing, depending on the patient's age, preference and functional disablement.[63] If nothing is done, there is some danger that the loose body will become embedded and be responsible for a permanent limitation of flexion.

Technique: manipulative reduction of a loose body

The patient sits on a couch. The arm is abducted to the horizontal and the elbow bent to a right angle. An assistant grasps

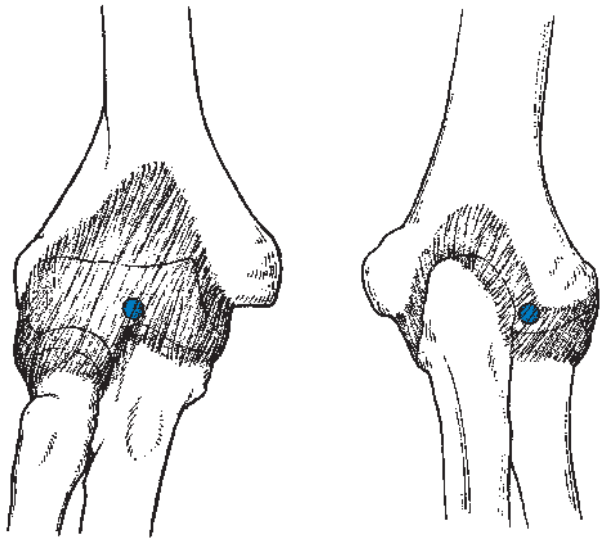


Fig 18.5 • Location of a loose body in a normal joint. Left, loose body in the anterior part of the joint limiting flexion. Right, loose body in the posterior part of the joint limiting extension.

the patient's arm just proximal to the elbow with both hands and fixes the arm against the couch. A second assistant steadies the patient's thorax with a hand on the lower ribs; the other hand holds the shoulder on the affected side against the couch. When assistants are not available, the patient's body and arm can be fixed with straps.

In this manipulation different elements are incorporated: traction, movement from flexion towards extension, and rotation movements, either pronation or supination (Figs 18.6 and 18.7).

The manipulator puts the contralateral foot against that of the first assistant vertically below the patient's elbow. This is the fixed point around which pivoting takes place as the elbow is moved from flexion towards extension. The patient's lower forearm just proximal to the wrist is then grasped with both hands. The position of the hands is slightly different, depending on the chosen rotation (see Fig. 18.6). The use of the manipulator's body weight is then able to exert the traction required to distract the joint. Pivoting on the foot, movement is gradually made from flexion towards extension while the patient's forearm is moved in either pronation or supination through the full range. At the last moment as extension is approached, the manipulator's trunk is side-flexed away from the patient to exert maximal traction. Extension movement is not performed beyond the degree of limitation; if this should happen, it would, of course, result in traumatic arthritis of the joint.

After each attempt, extension is re-examined. The chosen rotation is continued for as long as the signs improve. When no further improvement is found, the alternative rotation can be tried. Treatment should be stopped when the end-feel has become normal – i.e. hard.

One does not expect to reach a painless range of movement during the manipulation session but the change in end-feel

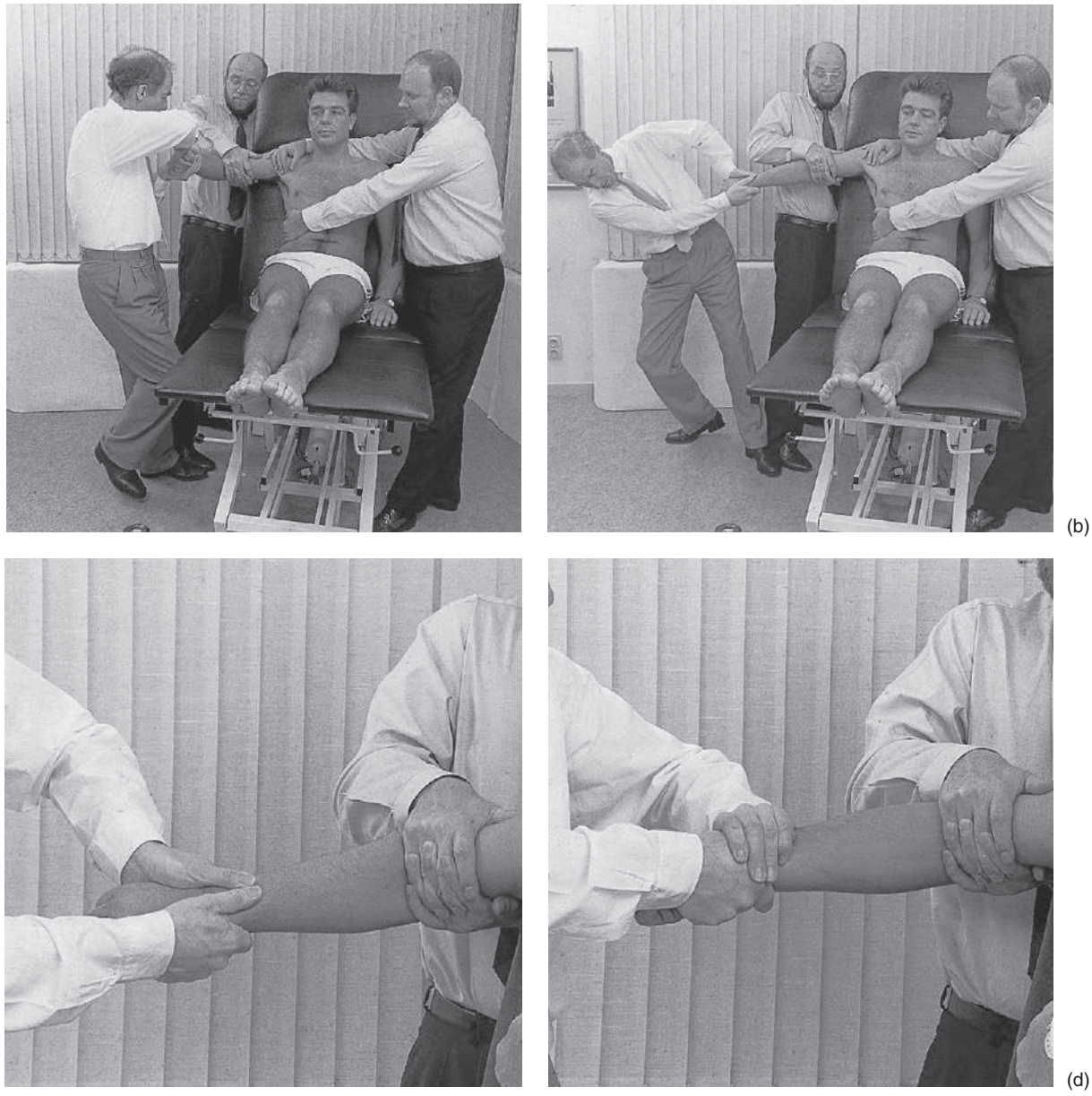


Fig 18.6 • Manipulative reduction of a loose body. (a) Starting position; (b) end position using the pronation technique (easiest and most commonly used). (c) Detail of hand position for this technique. (d) Detail of hand position for the supination technique (more difficult and less often necessary). This technique is not shown in a general view. Use straps to hold the patient when assistants are not available.

indicates that the loose fragment has moved. The slight traumatic reaction consequent on the original subluxation of the loose body will subside in the days following. The patient is seen after a couple of days and, if necessary, the manipulation is repeated.

In an arthrotic joint in middle or old age

Again the history is very indicative. The patient is middle-aged or elderly and possibly mentions a slight aching in the elbow as the result of the arthrosis. In addition, attacks of pain are reported that last about a week. The symptoms come on gradually (over the course of a few hours) and disappear even more gradually (over the course of some days).

Between attacks, the elbow presents a capsular pattern with a hard end-feel at the end of the achievable range, the result of the arthrosis. During the attacks the limitation becomes of the non-capsular type; it has a soft end-feel and is particularly restricted.

The fragments are osseous and visible on radiographs. However, it is possible to have loose bodies in the joint without having attacks of internal derangement.

Manipulative treatment can be performed during the attacks, although it is not strictly necessary because the condition subsides spontaneously. It is enough to explain the mechanism to the patient. Alternatively, arthroscopic removal can be considered.[64,65]

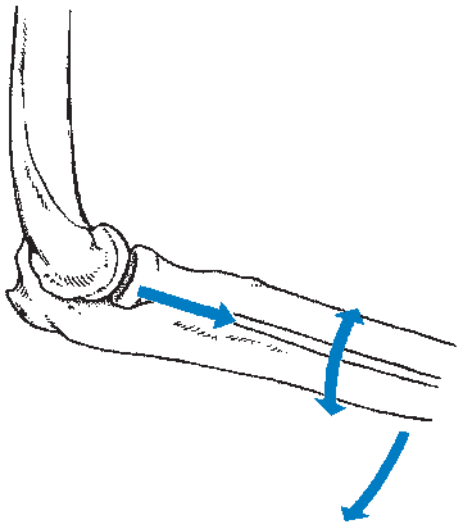


Fig 18.7 • The movements in manipulative reduction of a loose body. The illustration shows the three elements of the technique: traction, movement from flexion towards extension, and rotation.

Limitation of extension in tennis elbow

In some cases of tennis elbow, a slight limitation of passive extension may be found with a softish end-feel. The primary signs of tennis elbow will, of course, suggest the diagnosis (see p. 304).

Acute limitation of supination and extension in children

Radial head subluxation also known as pulled elbow, dislocated elbow or nursemaid's elbow, is one of the most common upper extremity injuries in young children under 8 years of age. The injury usually occurs when forceful longitudinal traction is applied to an extended and pronated arm,[66] as in lifting a child by the forearm or pulling the forearm of a resisting child. This manœuvre is immediately followed by pain and limitation of movement; the elbow is held flexed at about 90° and in pronation – hence the French name, 'la pronation douloureuse'.[67]

A 'pulled elbow' is actually a displacement of the annular ligament between the capitellum of the distal humerus and the radial head. The latter is pulled distally through the annular ligament, which becomes displaced from its normal position covering the radial head, into the radiohumeral joint (Fig. 18.8).[68]

The subsequent disturbance of the lower radioulnar joint is responsible for the limitation of supination. Ultrasonography of the radiohumeral joint shows that the distance between the radial head and the capitellum is increased, probably because of the interposition of the annular ligament into the radiohumeral joint.[69] A radiograph of the wrist shows a distal shift of the radius compared to the ulna, which is restored after manipulation.[70]

Reduction is easy. Sometimes spontaneous reduction occurs just by bringing the forearm into supination and flexion, as in examining for passive flexion of the elbow.[71,72] When this does not happen, manipulation is performed.[73]

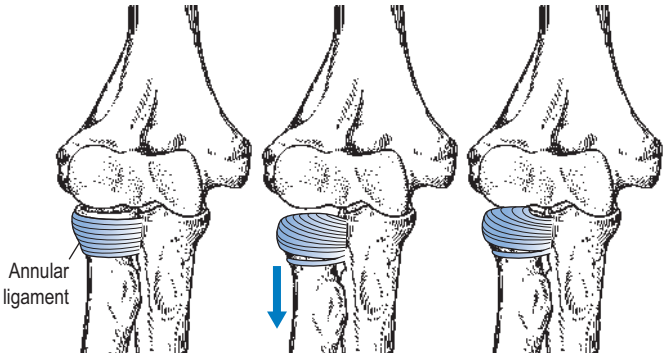


Fig 18.8 • Annular ligament displacement between the capitellum of the humerus and the radial head.

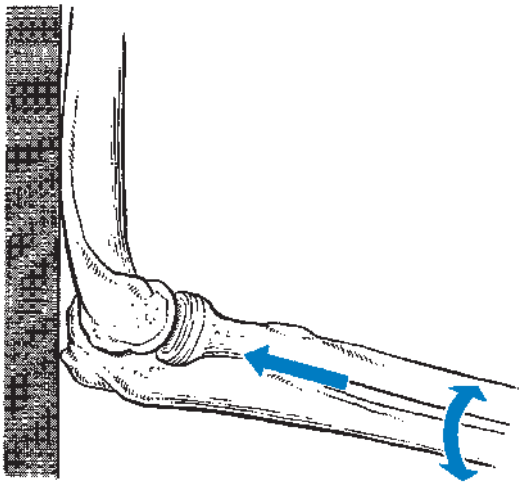


Fig 18.9 • Manipulative reduction of the 'pulled elbow'. This illustration shows the two elements incorporated in the technique: compression and rotation.

Technique: reduction of a 'pulled elbow'

The child is asked to stand against a wall. The upper arm is abducted and the elbow flexed to a right angle. The manipulator grasps the child's lower forearm with the ipsilateral hand and pushes the radius upwards towards the humerus by pressing the elbow against the wall. In the mean time, the forearm is rapidly rotated to and fro to the end of the range in either direction (Fig. 18.9). Suddenly, on full supination, the radial head reduces with a palpable click.

Limitation of pronation

The bicipitoradial bursa is located at the insertion of the distal biceps tendon. In supination it surrounds the biceps tendon. In pronation, the radial tuberosity rotates posteriorly, which compresses the bicipitoradial bursa between the biceps tendon and the radial cortex (Fig. 18.10).[74]

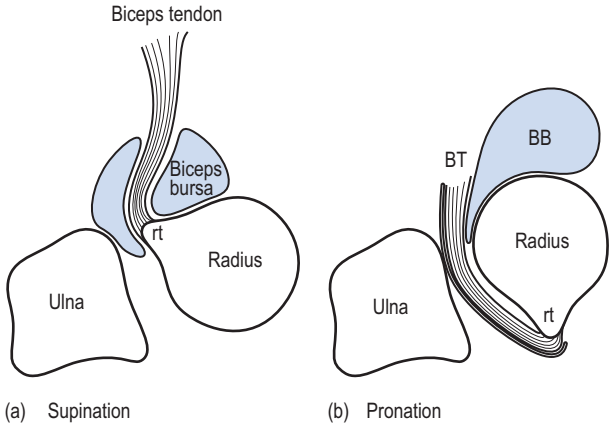


Fig 18.10 • During pronation, the bursa is compressed between the radius and the radial shaft. BB, bicipitoradial bursitis; BT, biceps tendon; rt, radial tuberosity.

When inflamed, the bursa may enlarge and produce symptoms and signs.[75] Classically, patients with cubital bursitis present with a mass in the cubital fossa and pain at the anterior aspect of the elbow,[76,77] together with symptoms and signs resulting from compression of one of the branches of the radial nerve.[78] In our experience, most cases of bicipital bursitis present only with cubital pain and a painful limitation of the pronation with a soft end-feel. The condition can be treated with infiltration of 20 mg of triamcinolone suspension. The same technique is used as for bicipital tendinitis but the infiltration is done somewhat more proximally between the tendon and the bone of the radius (see p. 299).

Full range of movement

Pain on full passive supination

Occasionally the proximal radioulnar joint may become sprained as the result of overuse. As the pain is felt at the outer side of the elbow, the patient may be misdiagnosed as suffering from tennis elbow. On examination, however, full but painful passive supination is the only positive finding. This lesion has no tendency to spontaneous cure.

Treatment consists of one or two intra-articular injections with 20 mg of triamcinolone suspension. The second injection is given 2 weeks after the first. The patient is cured after a few weeks. As this joint lies within the common joint capsule, the same technique as described earlier is used.

Pain on full passive pronation

This sign usually accompanies the primary signs of a bicipital tendinitis (see below) and indicates that the lesion lies at its attachment to the radial tuberosity. In the unusual circumstances in which bicipital tendinitis is absent, pain on passive pronation should be regarded as a bicipitoradial bursitis.[79]

Ligamentous lesions

Ulnar collateral ligament

Partial or total rupture of the ulnar collateral ligament resulting from overhand throwing has been described extensively in athletes.[80–82] Most affected individuals are high-level baseball pitchers, tennis players and javelin throwers. This condition is often complicated by irritation of the ulnar nerve.[83] Usually the lesion results from chronic overuse of the elbow leading to ligamentous insufficiency, even in the absence of a singular catastrophic episode of ligament failure.[84]

Commonly athletes note a history of recurrent elbow pain after or during throwing, without a specific injury.[85] On physical examination there may be a capsular pattern if the lesion is acute. In chronic lesions, the standard clinical examination will be entirely negative and the joint must be tested for valgus instability. For this purpose the moving valgus stress test has been found to be extremely useful, given its high degree of sensitivity and specificity.[86] The patient lies supine with the arm abducted and externally rotated. The elbow is in neutral rotation.[87] The examiner then applies and maintains a constant moderate valgus torque to the fully flexed elbow while gradually extending it. The test is positive if the medial elbow pain is reproduced within the arc between 120 and 70°.[88]

Surgical intervention may be necessary when conservative treatment fails and in a patient who wants to return to highly competitive sports.[89,90]

In young throwing athletes, repetitive high valgus stress and pull by the common flexor tendon may result in subtle stress fracture of the medial epicondyle epiphysis. It manifests with pain at the medial aspect of the elbow and diminished throwing distance and effectiveness. The medial epicondyle is very tender to the touch and a slight capsular pattern may be found. This lesion is called 'medial epicondylar stress lesion' or 'little league elbow'.[91–93]

Radial collateral ligament

A lesion of the radial collateral ligament occurs rarely as the result of repetitive isolated varus stress but mostly after complete elbow dislocation.[94] It may lead to posterolateral rotatory instability, giving rise to symptoms such as clicking, snapping and locking.[63] Tests, such as the lateral pivot shift or posterolateral rotatory drawer test, have been devised for confirming the diagnosis.[95] In recurrent problems, surgical reconstruction or repair seems to give good results.[96]

Bursitis

Olecranon bursitis

Because the bursa is superficial it is very vulnerable. The lesion can be provoked by repetitive direct pressure – for example, leaning the elbow on a table – or by falling heavily on to the bent elbow. It is often associated with occupational or sports trauma, or systemic conditions, such as rheumatoid arthritis, gout, tuberculosis and rheumatic diseases (e.g. chondrocalcinosis, xanthomatosis).[97,98]

The symptom is pain at the posterior aspect of the elbow. The signs depend on the type of bursitis present. In acute cases due to trauma or infection, there is swelling over the olecranon which varies in size from a slight distension to an egg-sized swelling. A variable amount of heat and redness is also found. In more chronic cases, slight swelling is the only sign. The functional examination is usually negative, although full passive flexion may cause some discomfort, described as 'tight'. Palpation reveals tenderness over the tip of the olecranon. In chronic lesions palpable thickening of the bursal wall may be present.

Differential diagnosis must be made between a traumatic bursitis and an infected bursa. Septic bursitis presents with heat and redness of the skin and pain on resisted extension of the elbow.[97,98] Analysis of the bursal fluid obtained through aspiration may be helpful.[99]

Sometimes aspiration alone will not prevent the fluid from reaccumulating because of continued movements of the elbow. Adding non-steroidal anti-inflammatory drugs will then hasten symptomatic improvement. Arthroscopic excision can be used when conservative treatment fails.[100] Septic bursitis should be treated immediately with antibiotic therapy and/or drainage. Sometimes, in the most stubborn cases, surgical excision is necessary.[101]

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19

Disorders of the contractile structures

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Unlike the shoulder, combined lesions of the elbow are quite rare. Therefore positive resisted movements usually indicate a lesion of one contractile structure only.

Resisted flexion of the elbow

Pain

The biceps and brachialis muscles are tested during resisted flexion, which should be performed with the elbow bent at a right angle – the neutral position between flexion and extension and also the one in which elbow flexion strength is maximal[1,2] – and the forearm held in active supination. This is the best position to test the biceps muscle,[3] which is more often strained than the brachialis.

Electromyographic studies confirm decreased activity of the biceps when flexion is performed in pronation.[4–6]

Biceps muscle

If the biceps muscle is at fault, resisted supination will also be painful. There are four possible localizations, indicated by the site of the pain (Fig. 19.1).

Pain at the shoulder

If resisted flexion and resisted supination elicit pain at the shoulder, the lesion should be sought in the upper extent of the long head of biceps, either in the bicipital groove, or higher up in its intra-articular course, or even at its attachment to the glenoid labrum.

Rupture of the long head of biceps is one of the commonest tendinous ruptures. It causes scarcely any symptoms, except the appearance of a round ball of muscle at the lower part of the arm when the patient flexes the elbow. Resisted flexion remains painless and strong, the brachialis muscle still functioning as the strongest flexor of the elbow – only 8% of elbow flexion strength and approximately 21% of forearm supination strength is lost.[7] Treatment is not required.

Pain at the mid-arm

The lesion may lie in the muscle belly itself, usually in the lower half. It is very often the result of a single excessive strain. The patient complains of anterior brachial pain during flexion movements. The symptoms may continue indefinitely, especially in those patients who continue exerting their arm. Even when the patient is prepared to take relative rest, spontaneous cure may require 2 years to occur.

A lesion in the belly of the biceps presents with two particular features: (a) it usually lies at the posterior aspect of the

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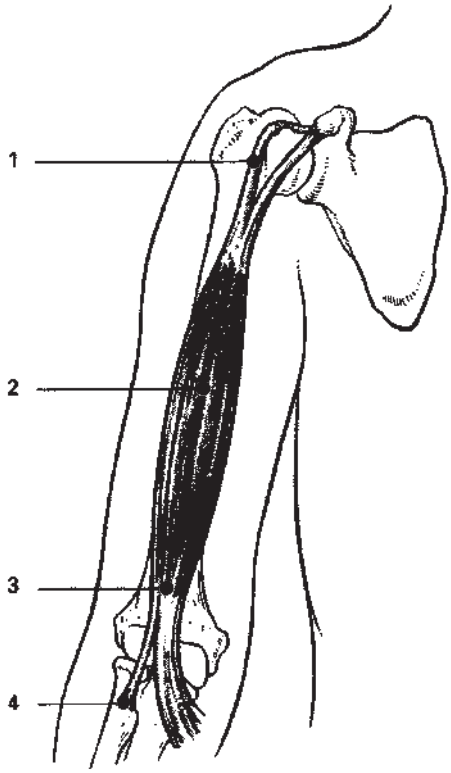


Fig 19.1 • Sites of lesions in the biceps: 1, tendon in bicipital groove; 2, muscle belly; 3, musculotendinous junction; 4, tenoperiosteal insertion.

muscle belly – therefore the tender spot must be palpated with a pinching grip; (b) the site is not always especially tender to the touch – it may therefore be difficult to find the exact localization by palpation. Diagnostic infiltration with a local anaesthetic may then be very helpful and, in cases of recent onset, the infiltration may even prove curative. Even if this is not the case, it shows exactly where the lesion lies so that the therapist knows where to give deep transverse friction, a treatment which is curative in a few weeks, except in very chronic cases – lesions which have lasted for more than 5 years may sometimes prove intractable.

Technique: deep friction to the biceps belly

The patient is in a half-lying position and the forearm rests on the thigh, so that the muscle belly is well relaxed.

The therapist grasps the muscle belly at the point of the lesion with the ipsilateral hand in a pinching grip, holding the fingers well flexed, which enables the posterior aspect of the muscle to be reached (Fig. 19.2). The hand is then pulled anteriorly. This results in a transverse friction to the muscular fibres, which can be felt to pass under the fingers. The hand is then brought back to the starting position without losing contact with the patient's skin. The manœuvre thus has two phases: an active phase, when the hand is pulled anteriorly, and a passive phase, when the hand is brought backwards again.

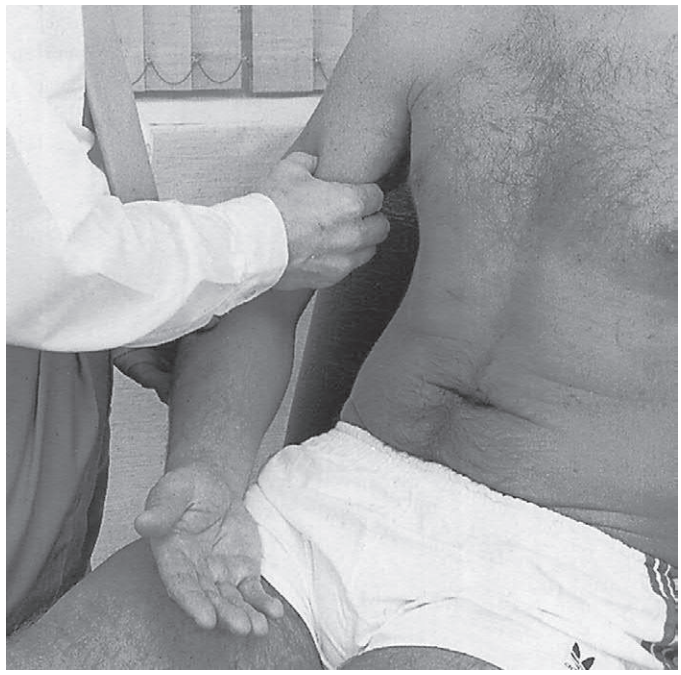


Fig 19.2 • Deep friction to the biceps muscle belly.

Pain at the lower arm

Palpation with a pinching grip shows the lesion to lie at the musculotendinous junction but again it is only possible to be sure of this after diagnostic infiltration with a local anaesthetic. Tendinitis at the body of the lower tendon of the brachial biceps does not occur.

Deep transverse friction is, in our experience, the only treatment. Without proper treatment the lesion may cause pain indefinitely, having occurred at a point where the natural mobility of muscle is restricted by the presence of tendinous strands.

Technique

The same technique is used as described earlier. The position of the hand and fingers, as well as the amplitude of the movement, are adapted to the volume of the structure at the musculotendinous junction.

Pain at the elbow

When pain is felt at the anterior aspect of the elbow, probably with some radiation down the front of the forearm as far as the wrist, a lesion at the tenoperiosteal insertion to the radial tuberosity should be suspected.

Apart from the usual signs on examination, a localizing sign is present – pain at full passive pronation. This is the result of the bicipital insertion being pinched between the radial tuberosity and the shaft of the ulna as the radius turns over the ulna during pronation.[8] Palpation is not necessary and would even be negative, as tenderness cannot be elicited by probing with a finger.

The treatment of choice is infiltration with triamcinolone. Because it is difficult to infiltrate the entire lesion accurately in the absence of localizing tenderness, up to three infiltrations may be necessary.

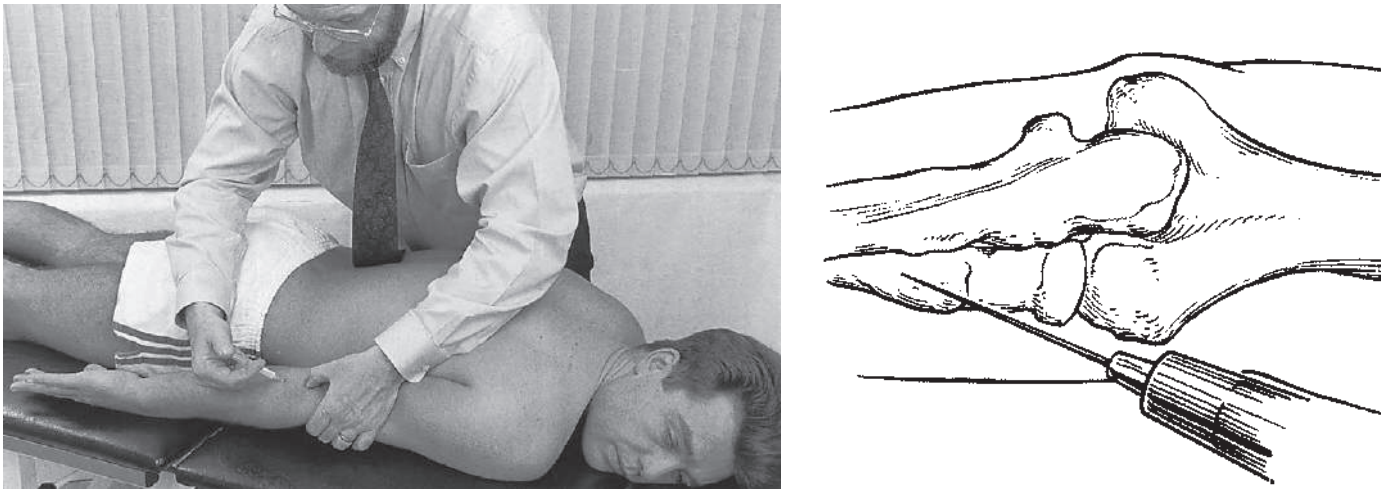


Fig 19.3 • Infiltration of the distal insertion of the biceps.

The alternative treatment is deep transverse friction. Cure takes about 2 weeks, and the treatment is quite painful, in that the lesion lies deeply and is difficult to reach.

Technique: infiltration of the distal insertion of the biceps

The patient lies prone on the couch. The upper arm is passively held in external rotation by the side. The forearm is put in full pronation, which turns the radius so that the radial tuberosity, where the insertion lies, points upwards.

The joint line between humerus and radius is palpated. The posterior edge of the ulna is easily felt. A point is chosen 2–3 cm further distally from the joint line and just laterally to the ulna. A 2 mL syringe is filled with triamcinolone and a thin needle, 2 cm long, is fitted. The needle is inserted at the chosen point and pushed vertically downwards until it hits the shaft of the radius, either directly or through a tendinous structure (Fig. 19.3). In the former case, the needle has to be partly withdrawn and reinserted in a slightly different direction in order to feel the tendinous resistance. Then a series of droplets is infiltrated at the tendinous insertion according to the usual infiltration technique.

The patient should be warned of moderate after-pain and to avoid flexion and supination movements for a week. Follow-up is after 2 weeks and, if the tests are not completely negative, a second infiltration is given. In most cases, one or two infiltrations suffice.

Fig 19.4 • Deep friction to the distal insertion of the biceps.

Technique: friction to the distal insertion of the biceps

The patient sits on the couch with the elbow bent at a right angle and supinated. The therapist sits and with the contralateral hand palpates for the radial tuberosity, searching the radiohumeral joint line and descending 2 cm along the shaft of the radius. At that level the thumb is moved anteriorly to identify the tuberosity deeply under the muscles of the forearm. The thumb is held well flexed and placed medially to the biceps tendon. The fingers apply counterpressure at the dorsal aspect of the elbow.

The ipsilateral hand, which grasps the patient's forearm about the wrist, is used to perform a pronation movement. The forearm is pronated until the biceps tendon passes under the therapist's thumb. The tendon now lies medial to it. Friction is thus performed by this indirect movement. During this part of the movement, the pressure on the tendon is augmented (active phase). The forearm is then brought back to the supinated position, while the pressure is released (passive phase). These repeated movements result in a transverse friction (Fig. 19.4), which is continued for 15 minutes.

The therapist must make sure that neither the median nerve nor the brachial or radial arteries are compressed during the manœuvres.

Brachialis muscle

Resisted flexion of the elbow is painful, certainly with the forearm in pronation, the position in which the biceps does not work. Resisted supination is negative.

Again, the tender area is difficult to find but usually lies in the distal third of the muscle belly, just dorsal to the bicipital tendon. It is therefore not impossible for passive flexion to hurt too because this pinches the injured part of the muscle. Diagnostic infiltration may prove necessary.

Treatment consists of 2 or 3 weeks' deep transverse friction.

Technique: deep friction

The same technique is used as for the brachial biceps muscle. The distal part of the brachialis muscle can be palpated just dorsally to the bicipital tendon. The muscle is well relaxed and friction performed with a pinching grip.

During treatment, the therapist should make sure that no nervous (paraesthesia) or arterial (paleness of fingers) symptoms arise, which would indicate that either the ulnar nerve or the brachial artery is being contacted. In such a case, the position of the hand must be altered.

The brachialis muscle and heterotopic ossification

Myositis ossificans is a benign condition, characterized by heterotopic bone formation, which occurs after injury to muscle fibres, connective tissue, blood vessels and underlying periosteum.[9] It occurs most often in males aged between 15 and 30 years and presents with the following triad of symptoms: pain in the affected muscle, a palpable mass and a flexion contracture.[10] The history of severe contusion is helpful in the early evaluation of these patients, before radiographic changes are evident.[11]

When, after an injury, usually fracture,[12] pain in the elbow region increases and the range of movement gradually decreases,[13] this may indicate the development of myositis ossificans. An increasing firm mass in the brachialis muscle is mostly palpable in the middle or distal segment of the arm and becomes visible on the radiograph in the course of a few weeks.[14] A technetium bone scan is clearly positive.[15] When myositis ossificans has fully developed, only a little movement to either side of the right angle is possible and even rotations are markedly limited.

Warning

Osteogenic sarcoma may also present with the formation of a mass invading the muscle.[16] The characteristic radiographic appearance of the sarcoma (presence of periosteal elevation and cortical destruction) provides the diagnosis.

Myositis ossificans does not respond to conservative treatment and may disappear spontaneously over the course of 2 years. Lipscomb *et al*[17] have stated that myositis ossificans in the upper extremity is very likely to resorb completely. If not, surgical excision may be performed, but only when the lesion is completely ossified, because removal of immature bone may cause extensive local recurrence.[18]

Because some authors attribute the occurrence of myositis to previous treatments, massage and mobilization to the brachialis muscle should only be given when there is a full passive range of movement at the elbow joint and radiographic evaluation shows no heterotopic bone formation.

Brachioradialis muscle

We have never encountered a lesion of the brachioradialis muscle but a case has been described by Barbaix.[19] The patient, following exertion, experienced supracondylar pain at the radial aspect of the elbow occurring during activities which included elbow flexion and simultaneous rotations. The pain could be elicited on passive extension, which was slightly limited, and on resisted flexion of the elbow. Accessory examination showed resisted supination with the forearm in full pronation more painful with a flexed elbow than with an extended one. Diagnosis was confirmed with echography and the patient responded well to infiltration with triamcinolone acetonide.

These clinical findings are compatible with an earlier electromyographic study showing electrical activity with flexion, especially with the forearm rotated in pronation.[20]

Weakness

Neurological conditions

Weakness of elbow flexion usually results from a cervical root palsy, either C5 or C6. In palsy of the fifth cervical root, resisted abduction and external rotation of the shoulder are also weak. In sixth cervical root palsy, resisted extension of the wrist is also weak.

Rupture

A distal biceps tendon rupture is usually at the tendinous insertion and sometimes accompanies an avulsion of the radial tuberosity.[21] The lesion is reported increasingly in the current literature.[22–26] The mechanism of injury is most commonly an eccentric load applied to a flexed elbow. On physical examination there may be swelling, haematoma and a 'Popeye' deformity of the upper arm. However, the normal contour of the distal biceps may still appear to be intact if the lacertus fibrosus is not torn.[27] Range of motion is usually full unless there is severe swelling. Strength testing reveals weakness in forearm supination and, to a lesser extent, elbow flexion. The biceps squeeze test and the hook test are two physical examination manœuvres that have been shown to be sensitive and specific in diagnosing distal biceps tendon ruptures. The biceps squeeze test involves compression of the biceps muscle belly, which results in forearm supination if the tendon is intact.[28] In the hook test, with the elbow actively flexed and supinated, an index finger should be able to 'hook' under a cord like structure in the antecubital fossa if the tendon is intact.[29]

The treatment of choice is surgery: anatomic repair of distal biceps tendon rupture provides consistently good results and early anatomic reconstruction can restore strength and endurance to the elbow.[30–32]

Resisted extension of the elbow

Pain

Triceps muscle

Lesions of the triceps muscle are rare. Three possible localizations can be differentiated by palpation (Fig. 19.5): the musculotendinous junction, which is the most common site; the body of the tendon; and the tenoperiosteal junction at the olecranon.

For all the sites, treatment consists of deep transverse friction. The tenoperiosteal junction responds equally to deep transverse friction and infiltration.

Technique: infiltration at the tenoperiosteal junction

The patient lies prone with the arm slightly flexed by the side. A small pillow is placed under the elbow.

A 1 mL syringe is filled with triamcinolone acetonide and a 2 cm needle is fitted. The tip of the olecranon is palpated and the tender point located. The needle is inserted 1 cm proximal to it and directed towards the olecranon (Fig. 19.6). Tendinous resistance should be felt before hitting bone. Infiltration is then carefully performed by several withdrawals and reinsertions of the needle and maintaining bony contact.

Technique: friction to the musculotendinous junction, body of tendon and tenoperiosteal junction

The patient sits next to the couch with the arm rested, the elbow held in 90° flexion. The therapist sits at a right angle to the patient's arm. With the contralateral hand the patient's elbow is grasped, while the other hand stabilizes the patient's forearm. The tender spot in the triceps tendon is palpated and then the index finger, reinforced by the middle finger, is placed at the medial side of the tendon. Counterpressure with the thumb is then applied at the lateral aspect of the proximal part of the forearm (Fig. 19.7). The therapist abducts the arm and then brings it back into the side. This is the active phase and is followed by relaxation while bringing the arm back into abduction. Such repeated movement results in a transverse friction to and fro over the lesion.

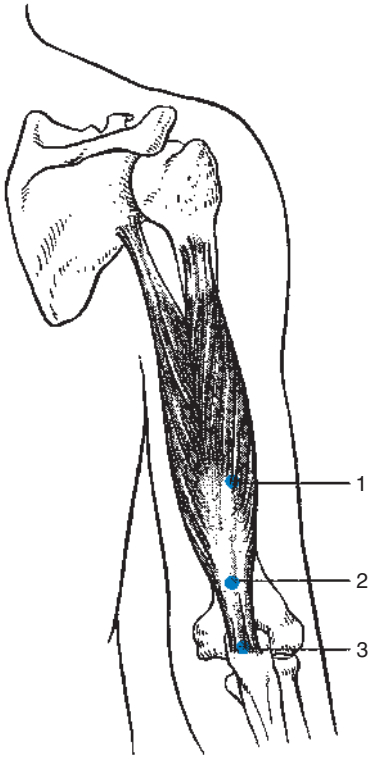


Fig 19.5 • Sites of lesions of the triceps: 1, musculotendinous junction; 2, body of the tendon; 3, tenoperiosteal insertion.

Resisted extension of the elbow and painful arc

Another possibility is that resisted extension of the elbow elicits pain at the shoulder in the C5 distribution. This has the same significance as a painful arc at the shoulder. As a result of contraction of the triceps muscle, the humerus is pulled upwards towards the acromion. A tender structure, lying between the head of the humerus and the roof of the joint, especially an inflamed subacromial bursa, may be painfully squeezed by this movement.

Anconeus compartment syndrome

Abrahamsson *et al*[33] reported a case of lateral elbow pain caused by a chronic compartment syndrome of the anconeus muscle. The symptoms were lateral elbow pain and muscular dysfunction during heavy work. The pain was reproduced by repeated extension of the elbow and pronation of the forearm. There was a slightly bulging mass and pronounced tenderness of the anconeus muscle, which were successfully treated by fasciotomy.

Weakness

Painful weakness

Total or partial rupture of the triceps tendon

This has been reported as a rare injury.[34,35] The most common form is an avulsion from the osseous tendon insertion.[36] Injury

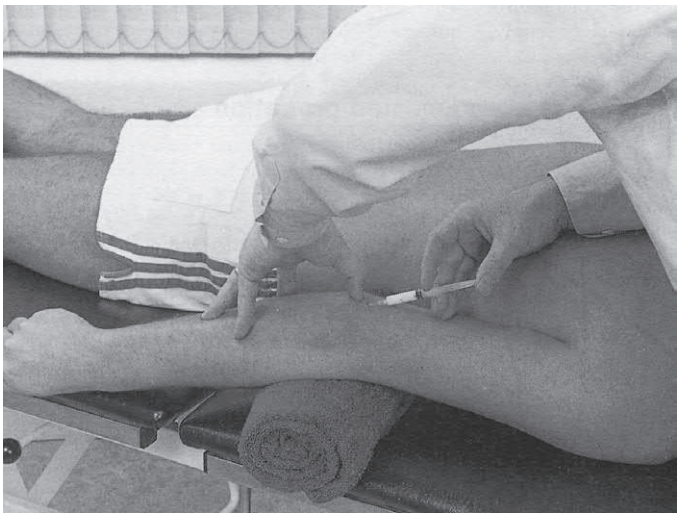


Fig 19.6 • Infiltration at the tenoperiosteal junction of the triceps.

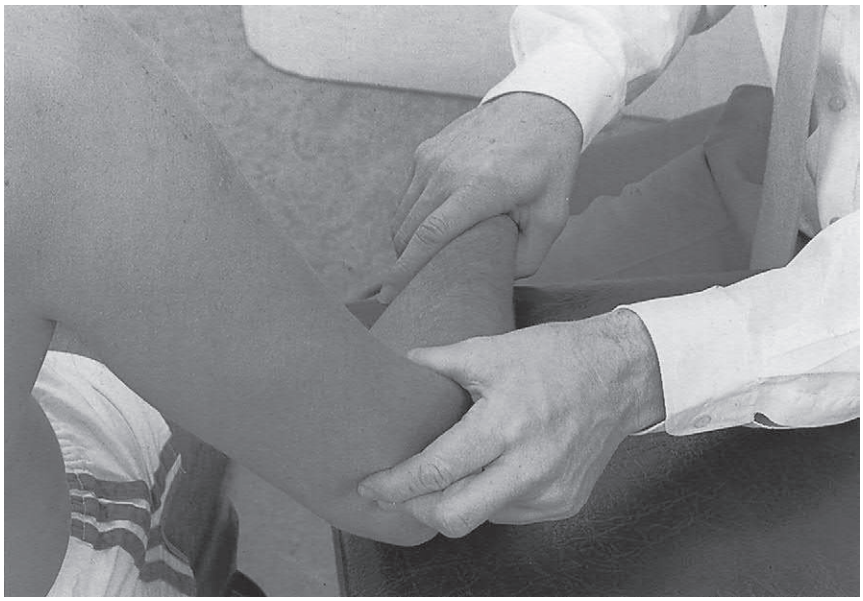


Fig 19.7 • Deep friction to the triceps.

at the musculotendinous junction occurs less often.[37] The most common mechanism of injury is a deceleration stress superimposed on a contracted triceps muscle.[38] A palpable gap, with complete weakness of arm extension, is pathognomonic of triceps rupture. In many cases, however, the palpable defect is not noted initially because of swelling and ecchymosis.[39] Early surgical repair of the injury is the treatment of choice and gives predictably good results.[40,41] Sometimes the same mechanism of injury causes a triceps avulsion fracture. Apart from the same symptoms and signs, the lateral radiograph will show the presence of flecks of avulsed osseous material from the olecranon ('flake sign'), which is almost pathognomonic of this lesion.[42]

Fracture of the olecranon

If the pain is the result of an injury and resisted extension of the elbow is painful and weak, the possibility of a fracture of the olecranon must be considered. Local tenderness and marked articular signs at the elbow joint will also be present: warmth, swelling and limitation of passive movements in the capsular pattern. In most cases, conservative treatment is sufficient.[43]

Painless weakness

A radial palsy and a C7 root palsy are the most common of the other neurological disorders.

Radial palsy

Apart from known traumatic causes, a radial palsy is quite often the result of pressure from a crutch or sleeping with the inner side of the arm pressed against a hard edge ('Saturday-night paralysis'). In this case, resisted extension of the wrist is also very weak and may even result in wrist-drop. The condition is painless and recovers spontaneously over the course of 3–6 months. Some severe cases require neurosurgical repair (see online chapter *Nerve lesions and entrapment neuropathies of the upper limb*).

C7 root palsy

This is the most common cause of a weakness of elbow extension and usually results from a C6 disc protrusion, compressing the C7 nerve root. The history points towards a neck problem, and cervical examination is positive. The triceps muscle may be found to be weak in isolation or in conjunction with weakness of the flexor muscles of the wrist and, in severe cases, the adductors of the shoulder.

Resisted supination

Painful resisted supination usually occurs in conjunction with pain on resisted flexion, in which case the lesion lies in the biceps muscle. If resisted flexion is not positive, the supinator brevis muscle must be at fault. The latter condition is quite rare.

To confirm the diagnosis, the test is repeated with the elbow held actively in full extension – a position which inhibits the function of the biceps. The muscle is not found to be particularly tender and therefore an infiltration with a local anaesthetic should be given (Fig. 19.8). This may effect a cure, but treatment with deep transverse friction (Fig. 19.9) is usually necessary. It is successful in 1 or 2 weeks.

Technique: friction to the supinator brevis

The patient sits beside the couch. The elbow is bent at a right angle, pronated and placed on the couch. The therapist sits at a right angle to the patient's arm and palpates for the shafts of radius and ulna. The tender spot is found in between the two bones. The supinator, being a flat muscle, cannot be felt on palpation, so anatomical knowledge is required to be able to palpate in that region. The therapist then places the thumb on the most tender area and applies counterpressure with the fingers at the medial aspect of the forearm. The friction is performed by an oblique movement perpendicular to the course of the muscular fibres, a movement that starts proximally on the radius and ends more distally on the ulna.

Fig 19.8 • Infiltration of the supinator brevis.

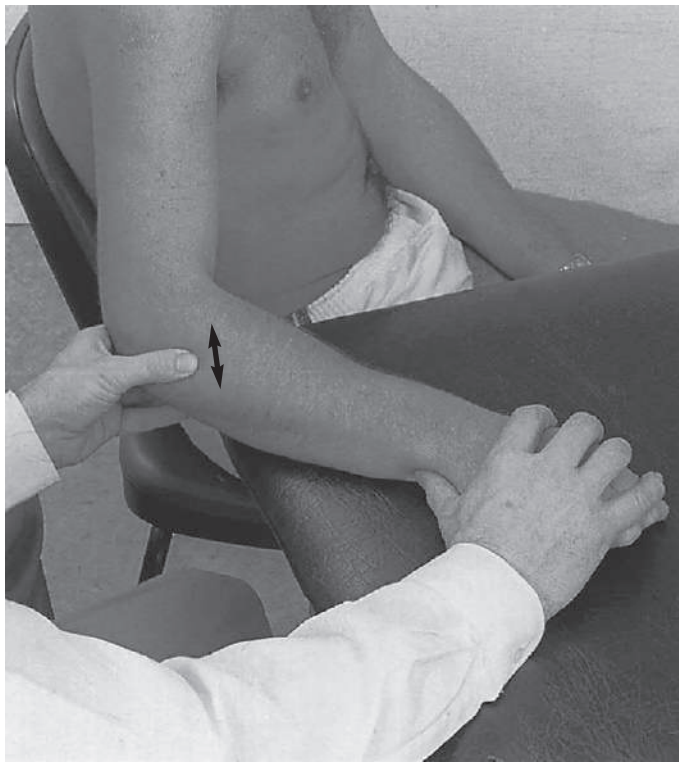


Fig 19.9 • Deep friction to the supinator brevis.

Resisted pronation

Pain on resisted pronation usually indicates golfer's elbow – a lesion in the common flexor tendon – in which case, resisted flexion of the wrist must also be painful. The fact that the pronator teres muscle partly originates from this tendinous complex at the medial epicondyle explains why resisted pronation may be positive (see golfer's elbow).

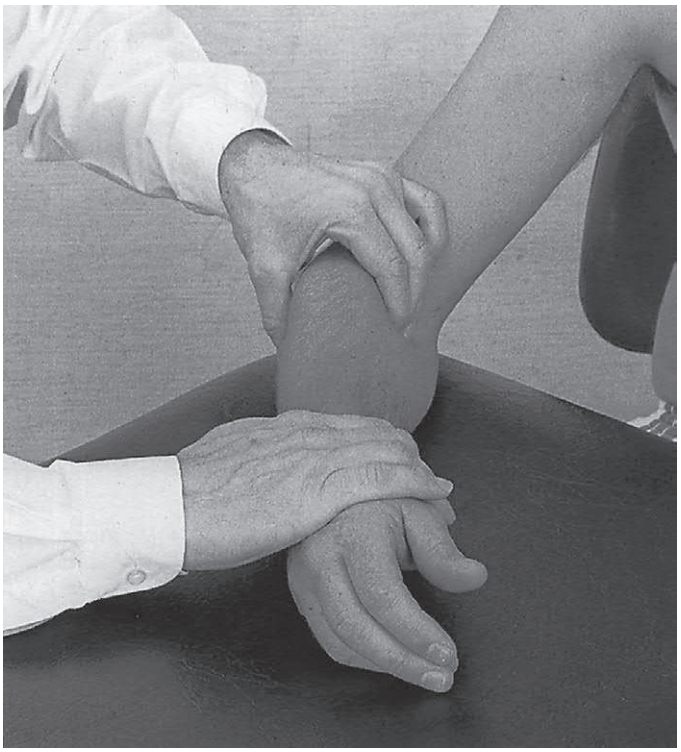


Fig 19.10 • Deep friction to the pronator teres.

Very occasionally, the lesion lies in the pronator teres muscle itself. Palpation discloses tenderness in the muscle belly, usually at its midpoint. In this unusual lesion, treatment consists of a few sessions of deep transverse friction.

Technique: friction to the pronator teres muscle

The patient sits with the arm rested on the couch. The therapist sits at a right angle to the patient's arm. The elbow is held in 90° flexion and the forearm in neutral position between pronation and supination. The therapist palpates at the anterior aspect of the forearm for the muscle belly, which may be facilitated by an active contraction by the patient. The fingertips of two or three fingers of the contralateral hand are then placed on the tender area and counterpressure is applied with the thumb at the lateral aspect of the forearm. The forearm is stabilized with the other hand (Fig. 19.10). The friction movement is begun underneath the muscle and the fingers are pulled over the muscle towards the therapist. This is the active phase. The fingers are then brought back to the previous position (passive phase). The sequence is repeated.

Pain on resisted extension of the wrist

Tennis elbow

The term 'tennis elbow' has been used to describe a multiplicity of conditions of the elbow.

Renton,[44] in 1830, was the first to describe a patient with pain along the outer forearm which increased on using the

hand. In 1873, Runge, a German physician, wrote an article on 'writer's cramp'. He distinguished the condition from a case of 2 years' inability to write, associated with tenderness on the lateral condyle of the humerus. Rest and electrotherapy had no effect and he then cauterized the skin over the tender area, after which the patient became well. He thought the cramp was the result of traumatic inflammation of the periosteum in this location, originally caused by a forcible supination effort and made chronic by the continual pull of the extensor muscles attached to the lateral condyle.[45] Morris (1882)[46] compared a condition named 'lawn-tennis arm' to rider's sprain – a lesion of the adductor longus muscle at the hip – and Major also called the condition 'lawn-tennis elbow'.[47] An annotation in *The Lancet* (1885) drew attention to the number of people suffering from 'tennis elbow', whose plaintive letters had recently appeared in the lay press. From then on, the term 'tennis elbow' has been used to describe symptoms at the outer aspect of the elbow. Remak (1894)[48] and Bernhardt (1896)[49] agreed that it was a periosteal tear from occupational overuse of the extensor muscles that arise from the lateral condyle. Couderé (1896)[50] called it a 'ruptured epicondylar tendon', Féré (1897)[51] 'épicondylalgie', Franke (1910)[52] 'epicondylitis'. In 1921, Schmidt[53] suggested superficial epicondylar bursitis, thereby incriminating the bursa described by Schreger in 1825.[54] In 1922, Osgood[55] and, in 1932, Carp[56] believed the lesion to lie in the radiohumeral bursa, and extra-articular bursa under the extensor tendons, which was first described by Monro in 1788.[57]

In 1936, Cyriax[58] compiled a list of no fewer than 26 different types of lesion to which the name tennis elbow had been given by 91 authors during the previous 63 years. He presented a synopsis of opinion as to the cause of tennis elbow and stated that a condition, the symptoms and signs of which are as constant as those of tennis elbow, may well be supposed to have but one background and, as a corollary, but one type of treatment.

An enormous number of articles on tennis elbow have been published in the ensuing years, describing different pathological entities such as radiohumeral bursitis, radiohumeral synovitis, irritation of the synovial fringe, degeneration of the annular and collateral ligaments, fibrillation of the articular cartilage, osteoarthrosis of the radiohumeral joint, osteochondritis dissecans, aseptic necrosis of the radial head, stenosis of the orbicular ligament, chondromalacia of the radial head and capitellum, calcific tendinitis of the extensor muscles, myofasciitis, traumatic periostitis of the lateral epicondyle, and enthesopathy of the lateral epicondyle of the humerus, among others.[59–68]

A new concept was introduced in 1972 when Roles and Maudsley described the 'radial tunnel syndrome'[69] (see online chapter *Nerve lesions and entrapment neuropathies of the upper limb*).

The most encompassing classification is perhaps that of O'Neill *et al*,[70] who state that 'tennis elbow or lateral epicondylitis consists clinically of pain localized in the region of the lateral aspect of the elbow with tenderness in the vicinity of the lateral epicondyle'. This vague description illustrates how unsure most authors are about the pathogenesis of tennis elbow.

Most authors consider tennis elbow as a local condition in relation to the lateral epicondyle of the humerus. However, some authors have described this condition as resulting from a cervical problem and they claim curative results with treatment to the neck. Gunn *et al*[71] considered a tennis elbow to be caused by a reflex localization of pain from radiculopathy at the cervical spine. Several authors agreed with this point of view.[72–74] Weh[75] believes that the idea of a local clinical assessment of tennis elbow should be broadened to an overall vision of a functional chain consisting of cervical spine, shoulder and elbow, in which local reflex inhibition of muscles innervated by the seventh nerve root and a limitation of movement of the cervical spine are causative elements. It is quite certain that elbow pain may have a cervical origin. This should not be designated 'tennis elbow' but instead 'referred elbow pain' from the cervical spine. If clinical examination shows the elbow pain to be evoked by movements at the elbow, then the lesion must be sought there. In contrast, if cervical examination is positive, this means that cervical movements must be the cause of pain in the elbow region.

We agree with those who consider tennis elbow as a lesion in the radial wrist extensors at the elbow. This is a logical deduction from what is found at clinical examination of the elbow, the primary sign being pain on resisted extension of the wrist.

Tennis elbow is characterized by pain on resisted extension of the wrist.

Terminology

'Tennis elbow' is technically a misnomer, since it most often occurs in non-tennis players: less than 5% of the patients play tennis.[76–78] Even in a population of 500 tennis players complaining of lateral elbow pain, Grachaw and Pelletier found tennis elbow present in only 39.7%.[79] This accords with Krämer's findings.[80] Neither is the term 'epicondylitis' right: at least 10% of the lesions do not lie at the epicondyle itself. Furthermore, the suffix '-itis' indicates an inflammatory process, but histological analysis shows only disruption of the collagen bundles with vascular and fibroblast proliferation and without inflammation. Therefore the term 'tendinosis' should be preferred over 'tendinitis'.[81]

Of all the patients seen in a general medical practice, 0.5% suffer from 'tennis elbow'.[82] The condition is therefore important enough to try to define it properly. The lesion lies at the elbow, in the structures controlling wrist extension and radial deviation (extensores carpi radiales). Theoretically, the lesion could also lie in the ulnar deviators or in the extensors of the fingers but this is much less common and should be separately named: for example, lesion of the extensor carpi ulnaris muscle, lesions of the extensor digitorum muscle.

Pathological features

It is generally believed that tennis elbow is an overuse phenomenon caused by repeated contractions of the radial extensors

of the wrist,[61,83] and especially of the extensor carpi radialis brevis.[84] Post-traumatic causes have also been described.[85] In the early stages, when the tissue attempts repair, and often just as the tear begins to unite, continued muscular contraction pulls the healing surfaces apart once more. This results in the formation of a painful scar with self-perpetuating inflammation (Cyriax[86]: p. 178). Cyriax's view was confirmed by Coonrad and Hooper in 1973.[76]

More recent observations during surgery have led Nirschl to classify this condition mostly under the heading 'tendinosis' rather than tendinitis: surgical inspection shows a typically 'grayish color and homogenous and generally edematous and friable tissue' indicating angiofibroblastic invasion – fibroblasts and vascular granulation-like tissue.[87–89] Inflammatory cells are not usually found.[90]

History

The patient is usually between 30 and 60 years,[91,92] and seldom under 20 or over 70. As the lesion is the result of overuse, the patient does not notice the pain the moment a tennis elbow develops but rather a few days later. A pain is described starting at the outer aspect of the elbow and radiating down the posterior aspect of the forearm, very often as far as the dorsum of the hand. Pain may be felt in the long and ring fingers. It is evoked by grasping, pinching and lifting – the movements that involve extension of the wrist and in which the wrist extensors merely act as stabilizers of the joint.

In a few weeks, the symptoms develop to the point that even light objects are dropped as the result of sudden painful twinges. Patients may be encountered who have constant pain, which may be worst at night and followed by waking in the morning with the elbow feeling stiff.

Functional examination

Nine out of the 10 tests of the elbow are negative and one is positive: resisted extension of the wrist. It should be remembered that the elbow must be held in full extension in order to avoid a false-negative result.

Tennis elbow = 9 tests negative + 1 test (resisted extension of the wrist) positive.

Our experience has shown that, if resisted extension of the wrist causes such a sharp twinge that the patient has to drop the hand, this points to a lesion at the tenoperiosteal junction. Resisted extension of the wrist, with and without the fingers held actively flexed, is then compared. Active flexion of the fingers inhibits the action of the finger extensors. In most instances both versions will be equally positive, which shows the wrist extensors to be at fault. Rarely, resisted extension of the wrist with the fingers held actively flexed will be negative, which implicates the finger extensors. If one or more finger extensors are strained, resisted extension of the different fingers will show which tendon is involved.

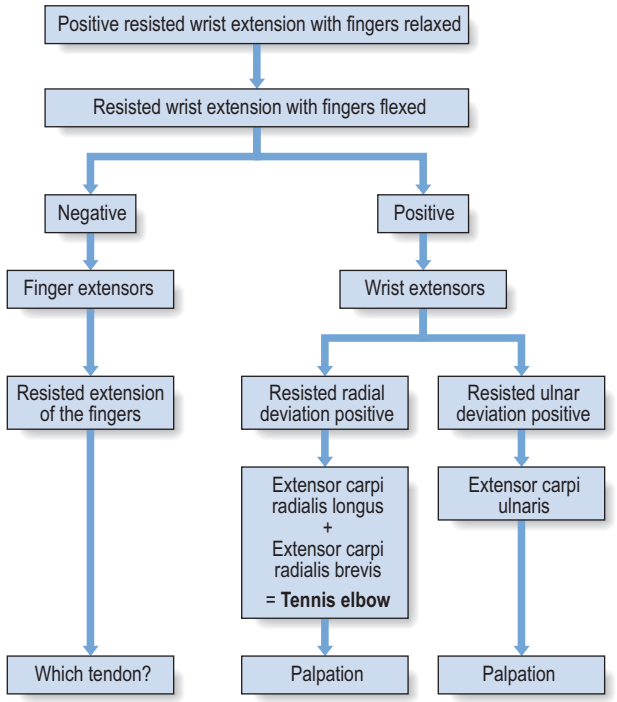


Fig 19.11 • Clinical examination of tennis elbow.

The next approach is to differentiate the radial extensors from the ulnar extensor using two accessory tests. Resisted radial deviation is compared with resisted ulnar deviation, the wrist being held in the neutral position between flexion and extension. In the rare circumstance that resisted ulnar deviation is painful, the lesion lies in the extensor carpi ulnaris muscle. Much more frequently, resisted radial deviation is painful, which indicates a lesion in either the extensor carpi radialis longus or the extensor carpi radialis brevis – the structures involved in tennis elbow (Fig. 19.11).

Palpation

The exact location is revealed by palpation. The palpatory manœuvres must be confined to the structures at fault: the extensor carpi radialis longus and brevis. In that the lateral aspect of the elbow is very often tender to the touch, even in those without a lesion, palpation is sometimes misleading. One elbow should be compared with the other, as should possible locations. In corpulent women, it may be difficult to define the lateral epicondyle. The palpation techniques are identical to the friction techniques (see Treatment, below) although it might sometimes be necessary to put the elbow further into extension to be able to reach the tender spot.

Practice shows that a tennis elbow has four different localizations (Fig. 19.12). Double lesions, however, are not at all uncommon. On palpation from proximal to distal, a tender spot can be found at one of the following places:

- *just proximal to the epicondyle* along the supracondylar ridge at the anterior aspect – i.e. the origin of the extensor carpi radialis longus. This structure feels like a muscular rather than a tendinous tissue (type I).

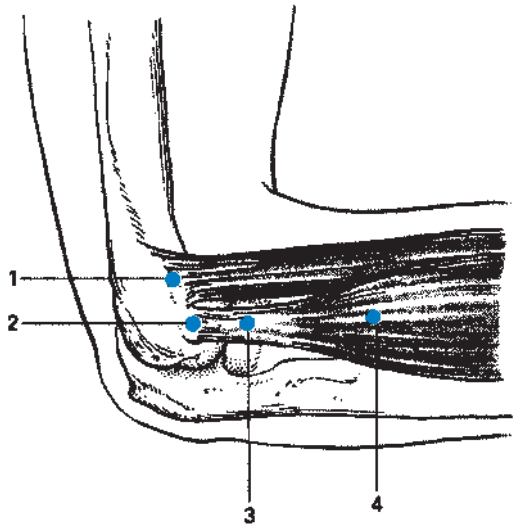


Fig 19.12 • Localizations of tennis elbow: 1, supracondylar (type I); 2, tenoperiosteal (type II); 3, tendinous (type III); 4, muscular (type IV).

- *at the anterior aspect of the lateral epicondyle* – the tenoperiosteal origin of the extensor carpi radialis brevis. In this instance palpation must be precisely at the origin of the tendon itself. Palpation of the lateral or posterior aspect of the epicondyle may also be positive, which is merely 'associated' tenderness. This disappears as soon as the lesion is cured. The tenoperiosteal variety at the epicondyle itself is by far the most common localization of tennis elbow (type II).
- *level with the radiohumeral joint line* and over the head of the radius – the body of the tendon of the extensor carpi radialis brevis, lying in between other tendinous structures (type III).
- *level with the neck of the radius* and a few centimetres distally – the upper part of the muscle bellies of extensor carpi radialis longus and brevis (type IV).

Experience shows that type I occurs in 1% of cases, type II in 90%, type III in 1% and type IV in 8% (Cyriax[86]: p. 178).

Treatment of type I (supracondylar) tennis elbow

Treatment of this type is quite simple. It responds very well to deep transverse friction and should be cured in 3–6 treatments, performed two or three times a week. Infiltrations are futile.

Technique: deep friction

The patient sits with the arm resting on the couch. The elbow is held in 90° flexion and in supination. The therapist sits lateral to the patient and uses the contralateral hand, with the tip of the thumb placed on the anterior aspect of the humerus just proximally to the lateral epicondyle. The thumb is kept well flexed and is held perpendicular to the shaft of the humerus.

The fingers apply counterpressure at the posterior aspect of the arm (Fig. 19.13).

Friction is imparted by an up-and-down movement of the entire hand, resulting in a movement of the thumb parallel to the axis of the humerus. The manœuvre has two phases: an active phase during the downwards movement and a passive phase when the hand is brought upwards again.

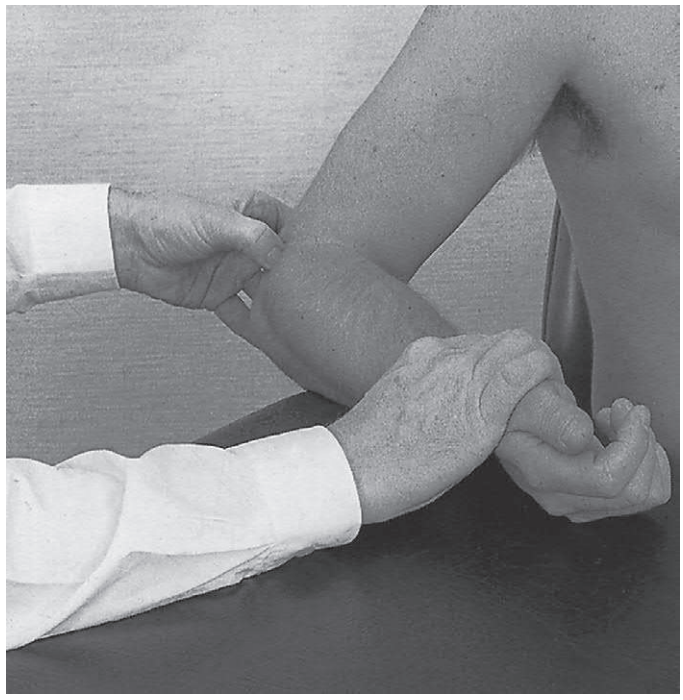


Fig 19.13 • Deep friction in supracondylar tennis elbow.

Treatment of type II (tenoperiosteal) tennis elbow

Treatment of the tenoperiosteal type of tennis elbow varies from very easy to very complicated. The choice of treatment depends on different factors, such as age, activities and duration of the symptoms. As the condition tends to resolve spontaneously, this possibility should be presented to the patient. Active treatment consists of infiltration with triamcinolone and Mills's manipulation. Recurrent or refractory cases may need other approaches, including infiltration with a sclerosant solution, percutaneous tenotomy and partial immobilization – taping and bracing (see Treatment procedures, p. 312).

Natural evolution

The tenoperiosteal variety is the only type of tennis elbow that has a tendency to spontaneous cure[93] (Fig. 19.14); this may take a year in patients under 60 and up to 2 years when the patient is older than this. After full spontaneous recovery, a second attack is rare. However, infiltration of any steroid solution inhibits the mechanism of spontaneous cure. The patient should be told about the possibility of spontaneous recovery, certainly if symptoms have been present for some time. Waiting, meanwhile sparing the elbow as much as possible, may be the preferred option. Cyriax[86] (his p. 179) explains the mechanism of spontaneous recovery as follows: as the result of the many contractions of the extensors of the wrist, which put tension on the healing breach, the gap between the two edges gradually widens. Finally the two surfaces cease to lie in apposition and the tension on the scar ceases. The gap now fills with

fibrous tissue. The painful scar is now embedded and heals with permanent lengthening of some tendinous fibres. In consequence, the strain no longer falls on that part of the tendon. Recurrence is prevented by this structural alteration. Because several muscles are attached here, slight permanent lengthening of the section of tendon relevant to only one muscle does not weaken the power to control the wrist.

Infiltration with triamcinolone suspension

Until 1952 there was no alternative to deep transverse friction in the treatment of tendinous lesions. For many years a means of reducing a localized area of unwanted traumatic inflammation had been sought. In 1952, Cyriax and Troisier[94] suggested the use of steroid infiltration as a treatment for tennis elbow. After the infiltration, the scar remains in place but the inflammation ceases. Because the steroid is a suspension of insoluble particles, its action is confined to the structure which is infiltrated without much effect on or spread to the adjacent structures.

In the following decades, steroid infiltration gained a bad reputation. Some authors therefore advise that the injection be given in such a way that the solution will bathe the underlying tendon rather than being injected into the tendon itself.[95] This technique may easily lead to the development of lipoatrophy and depigmentation of the skin. All these side effects can be avoided by using the smallest possible amount of weak steroid and precise infiltration into the tendon itself.

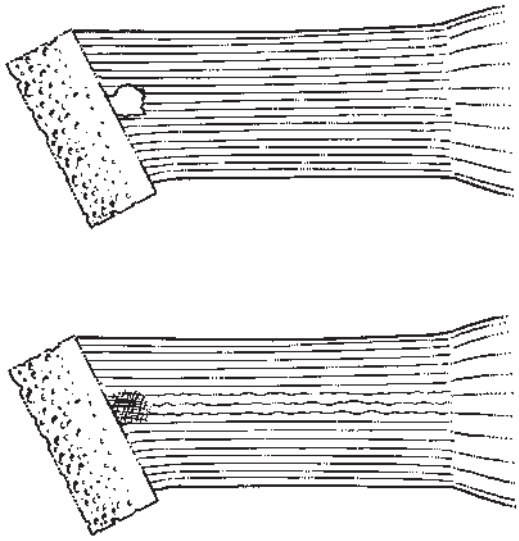


Fig 19.14 • Mechanism of spontaneous cure of tenoperiosteal tennis elbow.

Technique: infiltration

The patient sits with the elbow resting on the couch. The joint is flexed at a right angle and held in full supination. The boundaries of the lesion are now meticulously defined by palpation.

A 1 mL tuberculin syringe is filled with triamcinolone suspension and fitted with a thin needle 2 cm in length. The thumb is placed at the tender spot. The needle is thrust in vertically until it pierces the tendinous insertion and hits bone (Fig. 19.15). As one droplet is infiltrated, the appearance of a tiny bulge is felt for with the palpating thumb, confirming that the correct spot has been found. By half-withdrawing and reinserting the needle at slightly different angles, the entire lesion, which must be envisaged as a three-dimensional object, is infiltrated. Each time a droplet is injected, its exact position should be determined using the thumb. Considerable pressure sometimes has to be exerted on the syringe.

The patient is warned against possible severe discomfort for 24 hours, which may be possible to reduce by the application

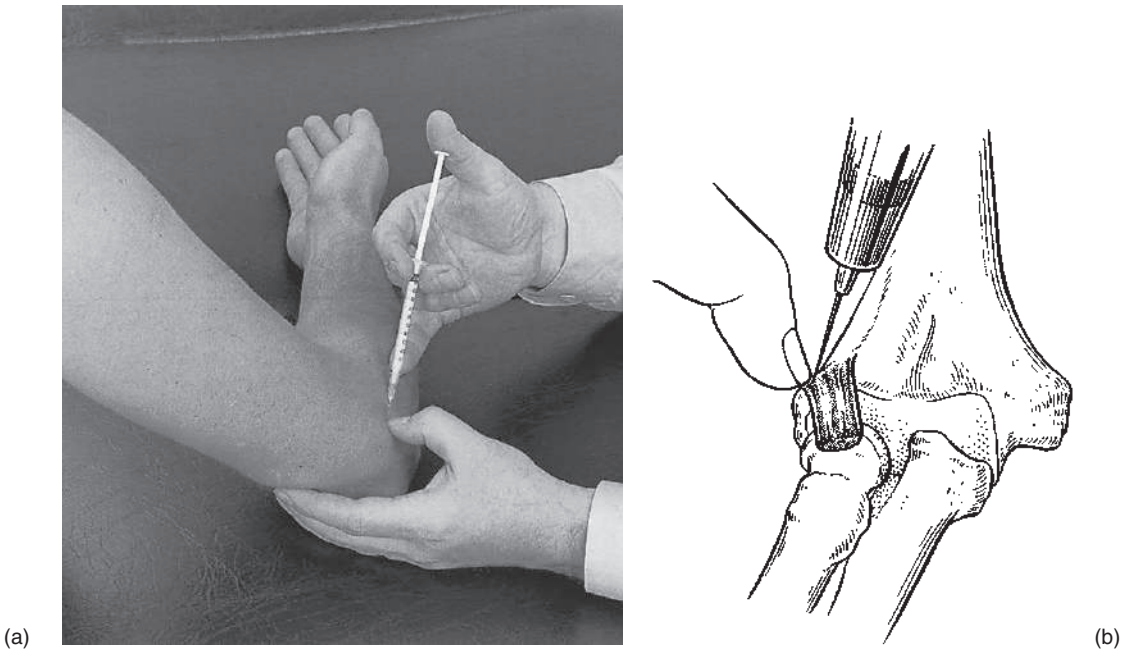


Fig 19.15 • Infiltration in tenoperiosteal tennis elbow. Viewed (a) from behind and (b) from the front.

of ice. Complete rest should be taken for a week and relative rest for another week. Follow-up is after 2 weeks with careful retesting, even if cure appears to have been achieved. If the examination is negative, no more need be done. If the elbow still aches or if the clinical examination is not 100% negative, infiltration should be repeated.

Results

Our personal experience is that 1–3 steroid infiltrations give full relief in all cases of uncomplicated tennis elbow. However, only two-thirds of patients remain asymptomatic. The rest will relapse within 2–6 months. Although renewed infiltrations may be of most benefit, there will remain some refractory cases that are difficult to treat (see p. 310). The prerequisites for success are: accurate diagnosis and localization of the lesion, correct infiltration technique, and a completely negative test on re-examination. A similar success rate was found by Troisier in 1991[96]: in a series of 257 patients, infiltration with corticosteroids gave full relief in all cases but the recurrence rate was 66.7%, although 67% of these recurred only once or twice.

Over the last few decades the effectiveness of steroid infiltration has been investigated via multiple controlled trials. All showed that steroid injections produced quick and significant pain relief in the short term, but that long-term outcomes are less clear. Hay *et al* performed a multicentre pragmatic randomized controlled trial of local corticosteroid injection and naproxen in 164 patients. They came to the conclusion that early local corticosteroid injection is much more effective in the first weeks than treatment with naproxen or placebo, but that the outcome at 1 year was the same in all groups.[97] This confirms the findings of Assendelft *et al*[98] and Verhaar *et al*.[99]

In another randomized clinical trial, Smidt *et al* claimed that the long-term outcomes of steroid injections might be poor due to the high number of relapses.[100] The study by Tonks *et al* was very positive for steroid injections but did not follow up the patients beyond 7 weeks,[101] while Altay *et al* reported that local steroid injection produced excellent pain relief at 1 year.[102] In a prospective randomized study Dogramaci *et al* compared three different methods of injection: a local injection of triamcinolone, a peppering infiltration of local anaesthetic and a peppering infiltration of triamcinolone. The peppering infiltrations with triamcinolone produced superior clinical results with cure in 84% of cases, whereas corticosteroid injection was only effective in 36% and peppering with lidocaine alone gave good results in 48%.[103]

Mills's manipulation

Several authors, such as Cyriax, Mills, Mennell, Stoddard and Kaltenborn, have proposed manipulation as a treatment for tennis elbow.[58,104–108] Although it is based on different pathological premises, they found that manipulative treatment could lead to good results. Manipulation, preceded by deep transverse friction, was Cyriax's treatment of choice before infiltration with steroids was found to be effective in 1952. He intended to stretch the extensor carpi radialis brevis tendon maximally in order to try to pull apart the two surfaces of the painful scar. This would thus imitate and speed up the mechanism of spontaneous recovery (see p. 307).

Several manipulations have been compared and it seems that Mills's manipulation, as described by Cyriax and for the indications which he describes, has the greatest potential to stretch the affected tendon with the least potential for harm to the joint.[109] Mills was an orthopaedic surgeon who described his manipulation technique in 1928 with the intention of re-aligning a torn section of the annular ligament, which he regarded as out of place.[104] Cyriax later discontinued the use of his own initial manipulation method and advocated the use of his modification of Mills's manœuvre.

The manipulation is preceded by deep transverse friction because of its desensitizing and softening effect, which makes the actual manœuvre less painful and therefore more tolerable. Friction alone is ineffective. This second type of tennis elbow is the only tenoperiosteal tear in the body that is not treated by deep transverse friction alone, and the only one for which manipulation has any effect. Some authors claim good results with stretching of the wrist extensors as home exercise following the treatment.[110]

Contraindication

Mills's manipulation should not be performed when extension of the elbow is limited, either as the result of an existing capsular pattern (arthritis or arthrosis) or as the result of a limitation of extension in a non-capsular pattern (loose body disorder), because of the danger of provoking a traumatic arthritis. If the extension seems limited but is so because of pain alone, a careful manipulation may be tried and, if this does not set off an irritation or reaction of the joint, there is no danger in doing this at each treatment session.

Technique: preparative deep friction

The patient sits with the elbow flexed to 90° and in full supination. The therapist sits facing the patient's arm and grasps the elbow with the contralateral hand. The fingers apply counter-pressure at the medial aspect of the elbow.

The thumb is well flexed and the thumb tip placed on the tender spot at the lateral edge of the epicondyle. The thumb is then brought over the anterior aspect of the epicondyle and imparts friction by moving medially over the bone (Fig. 19.16). The amplitude of the friction is only a few millimetres. Pressure is exerted vertically downwards. The therapist should start gently, because of the painfulness of the condition, and gradually increase the pressure.

After 10–15 minutes' friction the manipulation follows.

Technique: manipulation

The patient sits on a chair and the therapist stands behind. The patient's arm is abducted and also put in internal rotation. The forearm is then fully pronated and the wrist flexed. The patient's hand is grasped with the ipsilateral hand by bringing the thumb between the patient's thumb and index finger, so that the therapist's thumb is on the patient's palm. Consequently the fingers come to lie at the dorsum of the hand. This position enables the therapist to maintain full flexion at the patient's wrist at the moment of manipulation. The contralateral hand is then placed on top of the olecranon (Fig. 19.17). By moving both hands in opposite directions, the elbow is extended and the extensor muscles are thus stretched.

The therapist focuses attention on the patient's hand, which should be forced into full flexion. As the manœuvre proceeds,

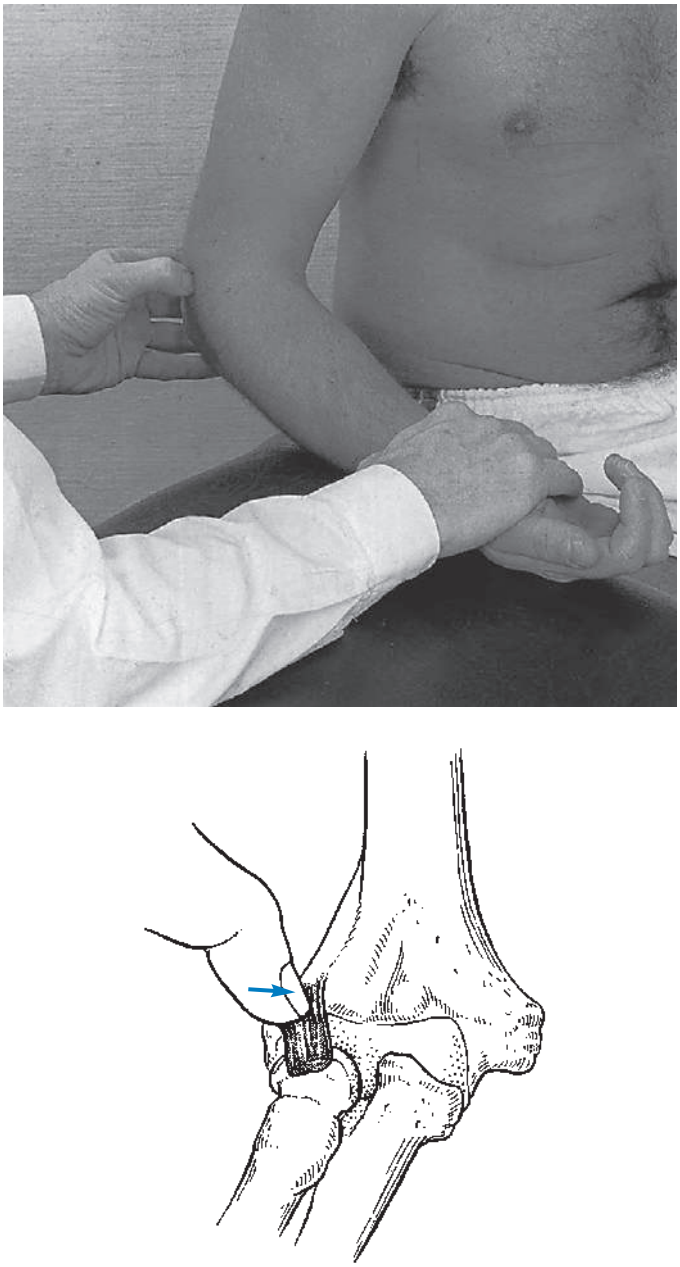


Fig 19.16 • Preparative friction prior to Mills's manipulation.

the body weight is brought to the other leg, so that the body moves away from the elbow. In this position the therapist is able to keep the patient's elbow and wrist in the required position.

A quick thrust towards elbow extension (Fig. 19.18) is then performed by moving both hands in opposite directions. A crack is very often heard, indicating the rupturing of some tendinous fibres. This manipulation is carried out once at each attendance. It usually takes 8–15 sessions for the patient to be cured but some cases require 20–30 treatments and not every tenoperiosteal tennis elbow responds well.

Troisier[96] reported a series of 131 cases in which good and excellent results were achieved in 63% by deep transverse friction and manipulation. A recent randomized clinical trial compared the effectiveness of deep transverse friction massage with Mills's manipulation versus phonophoresis with supervised exercise. Whereas both groups improved significantly from the initiation of treatment, a between-group comparison revealed significantly greater improvements regarding pain, pain-free grip and functional status for the experimental group compared to the control group.[111]

Refractory tennis elbow

The great majority of patients with the commonest variety of tennis elbow can be cured by the two procedures described. If the lesion seems refractory or if it frequently recurs, less usual measures can be used.

Infiltration with a sclerosant solution

The intention is no longer to reduce inflammation of the scar but to provoke the formation of dense adhesive scarring, which engulfs the original scar, using the irritant effect of hypertonic dextrose. The beneficial result may also stem from the powerful destructive effect of phenol on the small unmyelinated pain fibres.[112]

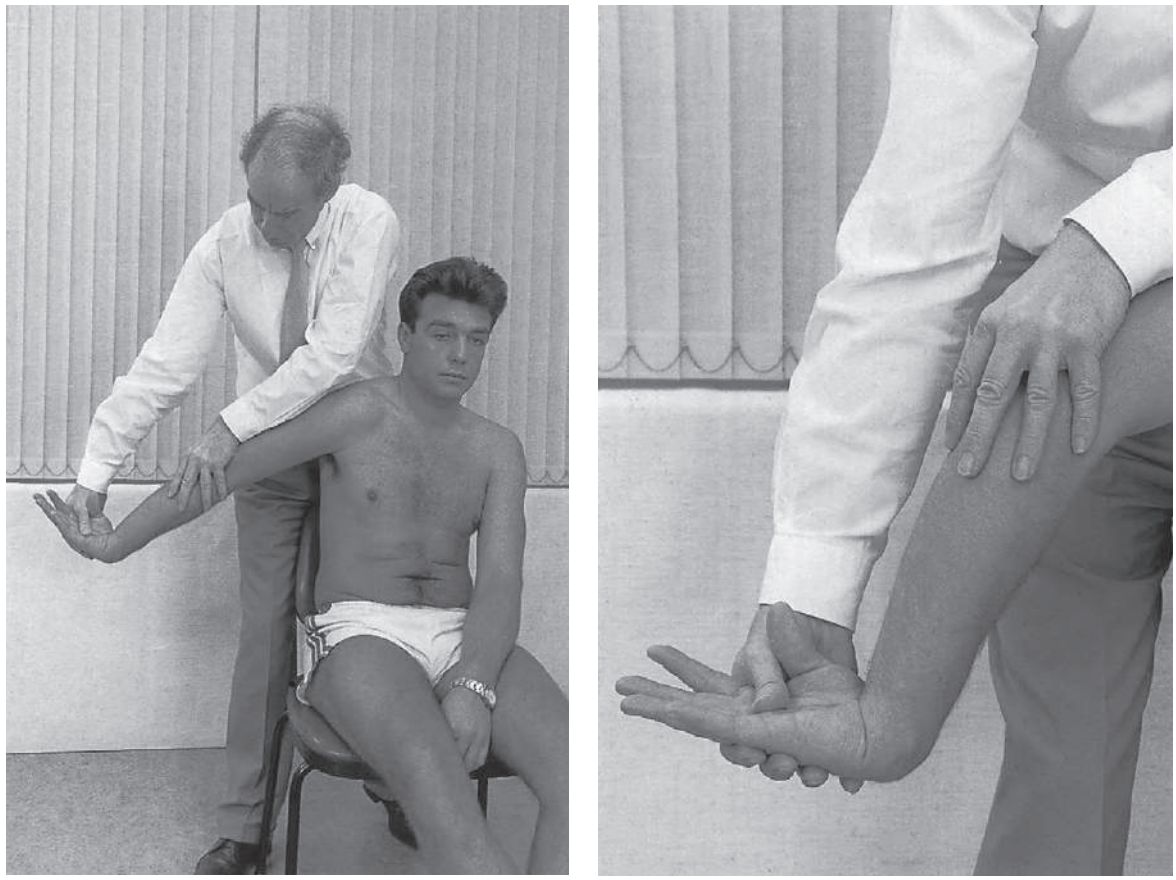
After-pain is considerable and may last several days, after which the condition gradually improves. The effect may only be expected after 2–3 weeks. Symptoms should disappear after one infiltration; if not, a second is unlikely to help.

Recent studies have demonstrated that 1–2 injections with a sclerosant solution effectively decreased elbow pain and improved strength testing in subjects with refractory tennis elbow compared to control injections.[113,114]

Tenotomy

If all other means have been tried without lasting effect, percutaneous tenotomy under local anaesthesia is worth consideration. This is a very simple operation, performed on an ambulant patient. The extensor carpi radialis brevis tendon at the anterior aspect of the lateral epicondyle is divided across its whole width. It is immediately followed by Mills's manipulation to ensure complete separation of the two cut ends. This treatment again imitates the mechanism of spontaneous cure. A study by Daubinet of 18 tennis elbow patients who did not respond to conservative treatment showed the effectiveness of insertion–tenotomy of the extensor carpi radialis brevis.[115] A prospective long-term follow-up study by Verhaar *et al* claims gratifying results and a low rate of complications with lateral extensor release as a simple outpatient procedure. They consider it to be the operation of choice for the treatment of long-standing tennis elbow.[116] Dunkow *et al* conducted a prospective randomized controlled trial of 47 patients with tennis elbow, who underwent either a formal open release or a percutaneous tenotomy. They concluded that the percutaneous procedure is a quicker and simpler procedure to undertake and produces significantly better results.[117] Other authors also claim good to excellent results with percutaneous release of the common extensor origin for long-standing symptoms.[118–122]

If tenotomy fails, one unexpected outcome is that treatment can be started again from the very beginning. The lesion, which hitherto showed no lasting response to infiltration with triamcinolone, may become amenable and will be helped with one or two further infiltrations.



(a) (b)

Fig 19.17 • (a) Mills's manipulation for tenoperiosteal tennis elbow. (b) Detail of hand position.

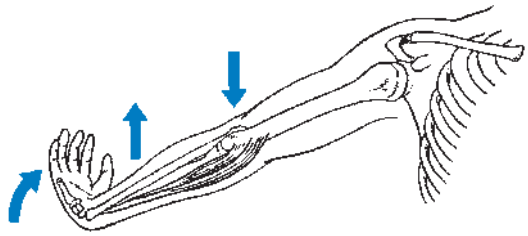


Fig 19.18 • Mills's manipulation for tenoperiosteal tennis elbow.

Technique: tenotomy[123]

The exact spot is palpated. Using the same infiltration technique as for treatment with steroids, the whole tender area is infiltrated with 1 mL of a 2% xylocaine solution. Some of the fluid is reserved to inject subcutaneously in order to render the skin anaesthetic. After a minute, resisted extension of the wrist is retested. If the correct spot has been chosen, this movement should be completely painless.

A small, thin, double-edged tenotome is then thrust through the skin into the dense tendinous tissue in the same way the needle was introduced – vertically. The tenotome is moved further down until it touches bone (Fig. 19.19). By moving the tip of the tenotome up and down along the bone of the epicondyle in a perpendicular way, the tendon of the extensor carpi radialis brevis is divided transversely across its entire width. The small lancet is then removed and the bleeding, if present, stopped by pressure.

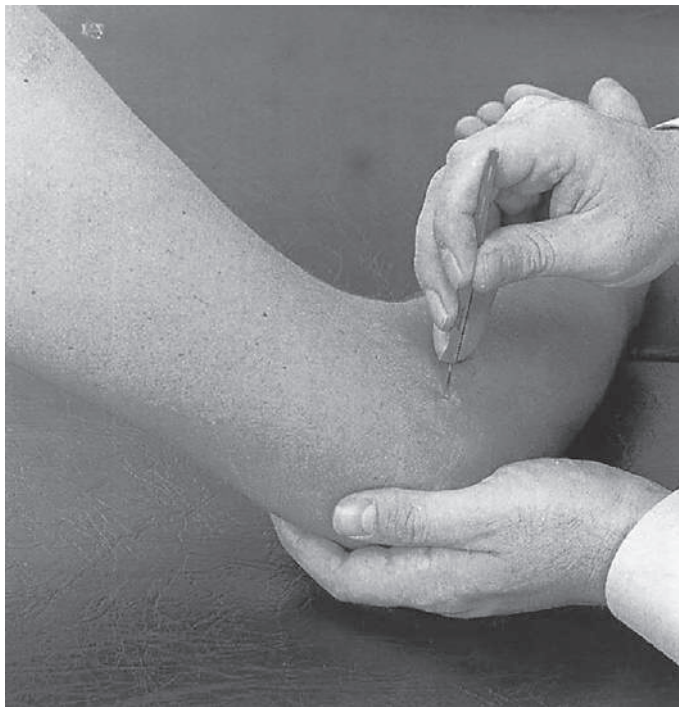
Mills's manipulation is then carried out to ensure complete separation of the two cut ends. No further treatment is needed. Suture is not necessary as the opening in the skin is only a few millimetres long.

The patient should be warned of serious after-pain, which may last for a few days. The elbow should not be exerted for a week and follow-up is after 2 weeks. Most of the traumatic arthritis, which sometimes results after a tenotomy, will have disappeared by then. If this is not the case, an intra-articular injection with 20 mg of triamcinolone acetonide should be given.

If the operation is not successful and resisted extension of the wrist remains painful after, say, a month, a further infiltration with 10 mg of triamcinolone acetonide should be given at the tender spot, which is usually localized at both cut ends.

Extracorporeal shock wave therapy

Attempts are made with extracorporeal shock wave therapy (ESWT) to treat resistant cases. The patient receives 500–3000 shock waves of 0.08–0.12 mJ/mm^2 three times at weekly intervals. Some authors consider this a useful conservative alternative in the treatment of tennis elbow.[124–126] However, more recent randomized clinical studies comparing different



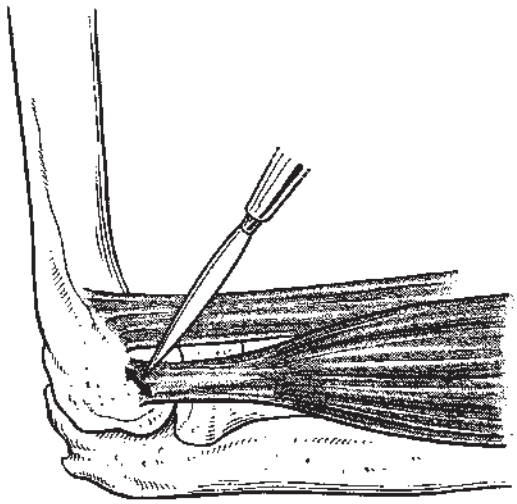


Fig 19.19 • Tenotomy.

extracorporeal shock wave regimens with placebo did not show strong statistical evidence in favour of ESWT.[127–129]

Bracing and taping

Through the years, tape and braces have been used to treat injuries suffered by athletes. In the early stages of the use of external support systems, physicians and certified athletic trainers used mostly trial and error and intuition to provide support for the injured area. Nowadays, interest in this aspect of preventive sports medicine has increased, and sound scientific principles have been used to improve the design and effectiveness of external support systems.[130,131]

At the elbow, a constricting band can be applied to the proximal part of the forearm (Fig. 19.20). It acts as a

Fig 19.20 • Counterforce brace for tennis elbow.

counterforce brace, constraining full muscular expansion and therefore decreasing intrinsic muscular force to the sensitive area in the forearm extensors. The intent is to cause the extensor mechanism on the lateral side of the elbow to work at the point where the band is attached, rather than at its point of origin on the lateral condyle. We consider the use of a counterbrace to be a valuable method for preventing recurrence because it causes improvement in wrist extension and grip strength with positive biomechanical effects.[132–135]

Special circumstances

There are two rare types of lateral elbow pain that are worth considering here.

Tenoperiosteal lesion of the extensor carpi radialis brevis with limitation of movement

This is encountered from time to time. If the limitation is in the capsular pattern, involvement of the capsule is suggested. This possibility will have been confirmed recently by arthroscopic examination.[136] If this is the case, the capsule should be treated first. If the limitation follows a non-capsular pattern, it may be caused either by a loose body limiting mostly extension, or by a cyst. The loose body can be manipulated. A cyst gives rise to a few degrees' limitation of passive extension of the elbow. The end-feel is softish, sometimes even a bit springy. Palpation usually reveals a cystic swelling under the origin of the tendon.

This type of tennis elbow has been shown to be refractory to any of the treatments described above. When a thick needle is thrust in, cystic fluid can be aspirated. Immediately afterwards, passive extension becomes full-range. A triamcinolone solution is now infiltrated, 1 mL at the origin of the tendon and 1 mL at a deeper site where the cyst has been detected. The patient is seen again after 2 weeks and, if necessary, treatment is repeated.

Radial tunnel syndrome

Some authors consider the so-called radial tunnel syndrome to be a type of tennis elbow (see online chapter *Nerve lesions and entrapment neuropathies of the upper limb*).

Treatment procedure for type II tennis elbow

There are three therapeutic options for a type II tennis elbow (Fig. 19.21):

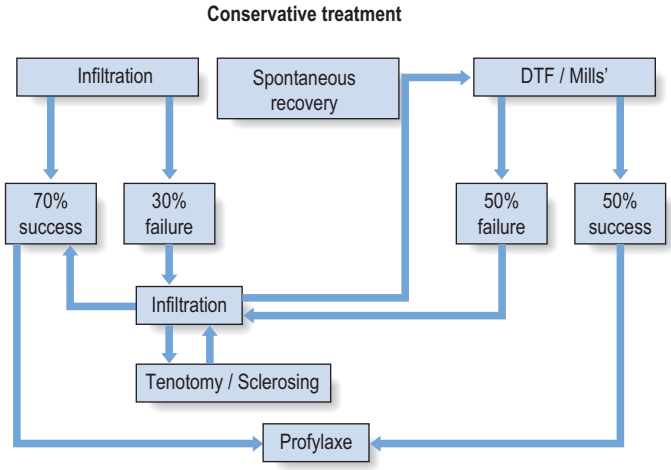


Fig 19.21 • Treatment procedure for type II tennis elbow.

1. *Waiting for spontaneous cure*. Tennis elbow is a self-limiting disorder that is usually spontaneously cured in 1–2 years. Therefore, in a mild case of lateral epicondylitis that is present for several months, it is worthwhile waiting before giving active treatment, and advising the patient to wear a counterbrace during activities.[137]
2. *Treatment with triamcinolone infiltrations*. Immediate relief is always obtained, provided the diagnosis and the applied technique are correct. However, this does not mean that all cured patients with type II tennis elbow remain well. The recurrence rate is high if the procedure (repeated reassessment and treatment), together with good secondary prophylaxis, is not properly followed. With a good infiltration technique and consequent procedure, complete and lasting cure is obtained in more than 70% of cases. If the condition recurs a few months after complete cure, triamcinolone infiltrations can be applied again. If relapses are very frequent, the case must be regarded as refractory and another treatment form should be considered: sclerosing injections or tenotomy.
3. *Mills's manipulation*. This form of treatment has a success rate of about 50%. However, there are no recurrences after complete cure. Mills's manipulation and triamcinolone infiltrations are interchangeable but are never applied at the same time.

Treatment of type III (tendinous) tennis elbow

Deep transverse friction is the better treatment in this case and should be successful in six sessions, performed 2–3 times a week.

Technique: deep friction

The patient sits at the couch with the arm resting on it. The elbow is held in 135° extension and in nearly full pronation by the ipsilateral hand. The tendon of extensor carpi radialis brevis now overlies the head of the radius.

The therapist takes hold of the patient's elbow with the contralateral hand. Counterpressure is applied at the medial aspect of the elbow with the fingers. With the flexed thumb, palpation is carried out at the tender spot. The therapist starts with the thumb at the medial aspect of the tendon and the friction is applied by pulling the thumb towards him, so that it slips over the tendon (Fig. 19.22). The thumb is then relaxed and the same movement started again.

The therapist must make sure that the amplitude of the friction movement is not diminished or stopped by the hand coming in contact with the couch. The patient's forearm is therefore best supported with a cushion, which leaves the elbow free.

Treatment of type IV (muscular) tennis elbow

The treatment of choice is infiltration with 10 mL of a local anaesthetic in the upper part of the belly of the extensor carpi radialis muscles. Two to four weekly infiltrations usually give lasting relief (Fig. 19.23).

Deep transverse friction is an alternative treatment; it is difficult, painful and not always curative. It is performed with a pinching grip (Fig. 19.24).

Technique: infiltration of the extensor carpi radialis muscle belly

The patient sits with the elbow resting on the couch. The elbow is flexed at a right angle and supinated. A 10 mL syringe is filled with 0.5% procaine and a 5 cm, thin needle is fitted. The tender spot is held pinched up by the therapist's thumb and index finger. The needle is inserted obliquely through the belly of the brachioradialis muscle until the point strikes the radius. It is then slightly withdrawn to lie between the fingertips (see Fig. 19.23). The solution is now infiltrated fanwise with different withdrawals and reinsertions so that the entire lesion is covered.

After 5 minutes, resisted extension of the wrist is retested. This movement should no longer hurt; if it does, the lesion was not entirely reached. Infiltration should be repeated at the patient's next appointment and concentrated on the areas that were missed.

The patient must be warned that a temporary weakness of hand extension is possible because infiltration of a local anaesthetic may paralyse the deep branches of the posterior interosseous nerve.

Lesion of the extensor carpi ulnaris muscle

Occasionally, resisted extension and resisted ulnar deviation of the wrist are painful at the outer aspect of the elbow. This indicates a lesion of the extensor carpi ulnaris muscle, usually at its tenoperiosteal origin at the posterior aspect of the lateral epicondyle of the humerus (Fig. 19.25) or, more rarely, at the olecranon. Palpation will, of course, disclose the exact site of the lesion.

Treatment consists of either infiltration with triamcinolone suspension or deep transverse friction.

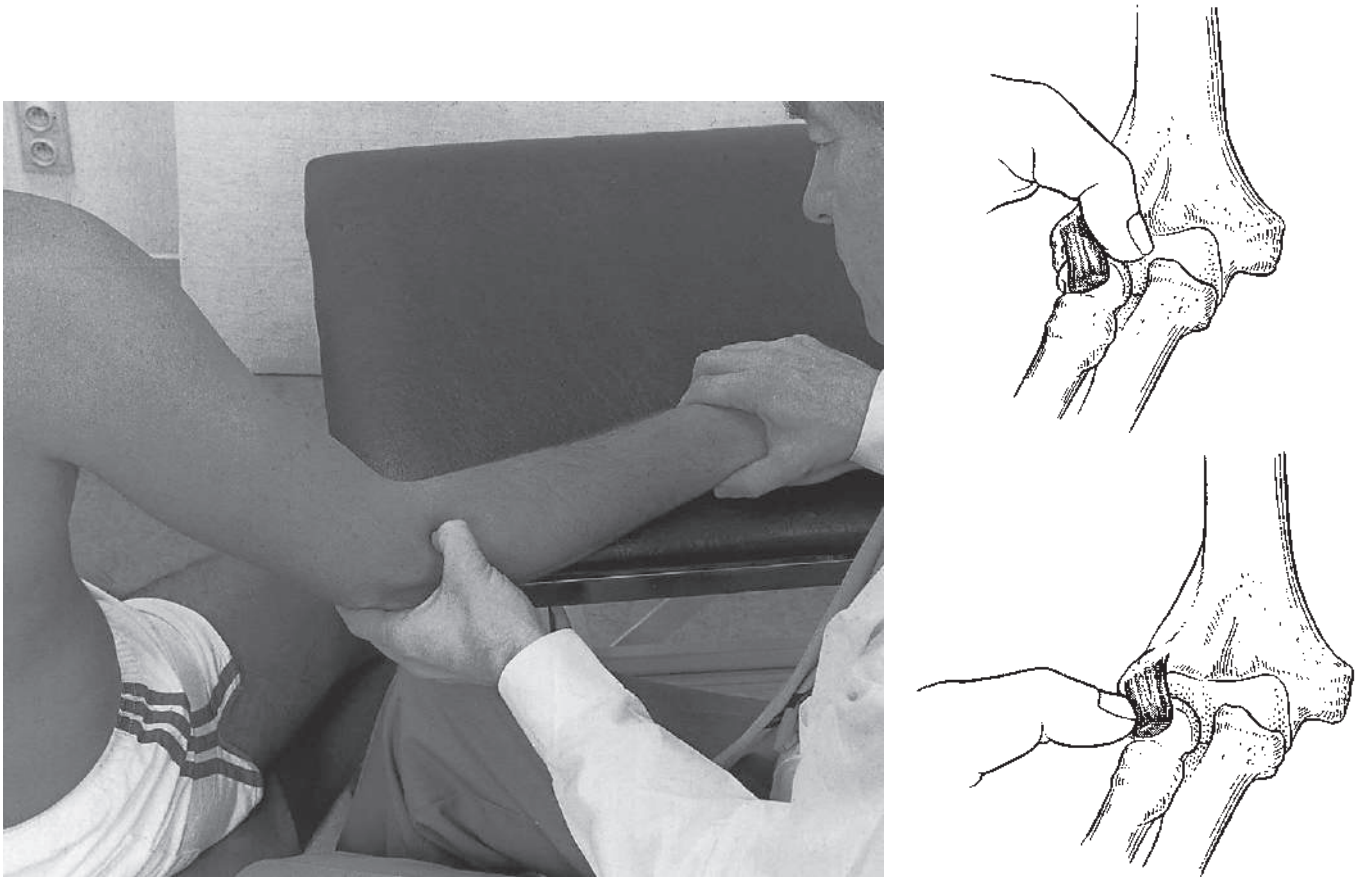


Fig 19.22 • Deep friction in tendinous tennis elbow: (a) starting position; (b) end position.

Lesion of an extensor digitorum muscle

Lesions of the extensors of the fingers do occur but are quite uncommon. The diagnosis is made too often and is probably the result of a misinterpretation of the clinical examination – resisted extension of the fingers, and especially of the middle finger, is often found to be positive in tennis elbow and apparently is a consequence of the close relationship of the extensors of wrist and fingers in the region of the elbow. When a lesion of the extensor digitorum does occur, resisted extension of the wrist with the fingers held actively flexed should be negative.

Treatment consists of either infiltration with triamcinolone (origin) or deep transverse friction.

Weakness on resisted extension of the wrist

Painless weakness of extension of the wrist can be the result of a radial palsy, a C5 disc protrusion compressing the C6 nerve root or, rarely, a C6 disc protrusion compressing the C7 nerve root.

Painful weakness also occurs at the moment that a painful twinge is elicited in type II tennis elbow.

Resisted flexion of the wrist

Pain

Resisted flexion of the wrist (with the elbow held in extension) causing pain at the medial aspect of the elbow indicates a lesion of the common flexor tendon, which originates mainly from the medial epicondyle. The lesion is known as medial epicondylitis or *golfer's elbow*. The disorder is diagnosed 7–10 times less often than lateral epicondylitis.[138] Although golfer's elbow may affect patients from 12 to 80 years of age, it is most commonly seen in the 40–50-year age group.[78] The condition has been attributed to repetitive forearm pronation and wrist flexion.[139,140] As in tennis elbow, it follows a series of repeated (often occupational) strains,[141] rather than a single injury. Histological analysis shows pathological changes in the common flexor tendon: micro-tearing with disruption of the collagen bundles, vascular and fibroblast proliferation, focal hyaline degeneration and an incomplete reparative process. As in tennis elbow, these changes have been called 'angiofibroblastic hyperplasia' and, as there is no evidence of any inflammatory process, the term 'tendinosis' is preferred over 'tendinitis'.[142]

Fig 19.24 • Deep friction to the extensor carpi radialis muscle bellies.

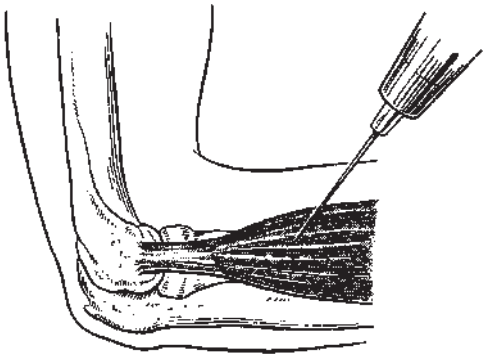


Fig 19.23 • Infiltration of the extensor carpi radialis muscle bellies.

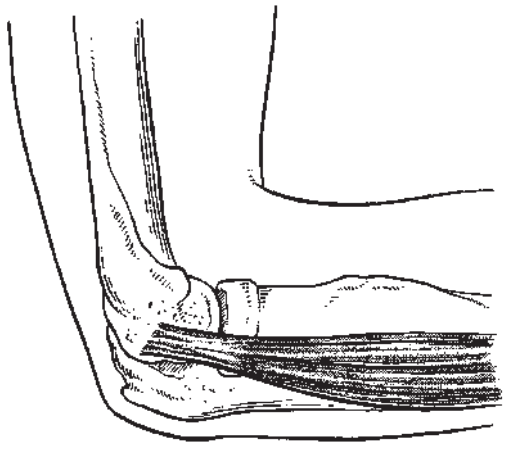


Fig 19.25 • The extensor carpi ulnaris muscle.

The patient, aged between 40 and 60 years, describes pain at the medial aspect of the elbow that may radiate down the upper part of the forearm at the ulnar side. The pain is provoked by palmar flexion of wrist and/or fingers. Resisted pronation of the elbow is often positive as well, because the pronator teres muscle partly originates from the common flexor tendon. Rarely, pain is only evoked by resisted pronation and/or resisted flexion of one or two fingers. This may occur in very localized lesions and depends on which part of common flexor tendon is affected.

Palpation of the medial epicondyle is necessary to find the exact spot, which is usually very localized. There are two possible sites (Fig. 19.26):

- *The tenoperiosteal variety*: the tender spot is found at the anterior aspect of the medial epicondyle of the humerus at the origin of the common flexor tendon.
- *The musculotendinous variety*: the tender spot is 5 mm farther down, level with the inferior edge of the epicondyle. This is the musculotendinous junction (the common tendon diverging immediately below its origin into the muscle belly).

Occasionally, and only in long-standing cases, calcification may be seen on radiography. As this is merely a radiological finding without clinical significance, it may be ignored and does not influence the therapeutic approach.

Treatment

Tenoperiosteal junction

The treatment of choice is one or two infiltrations with 1 mL of triamcinolone suspension (Fig. 19.27). The alternative is deep transverse friction (Fig. 19.28). Very often, 6–12 sessions, 2–3 times a week, are needed. The results of surgical treatment are poor.[143,144]

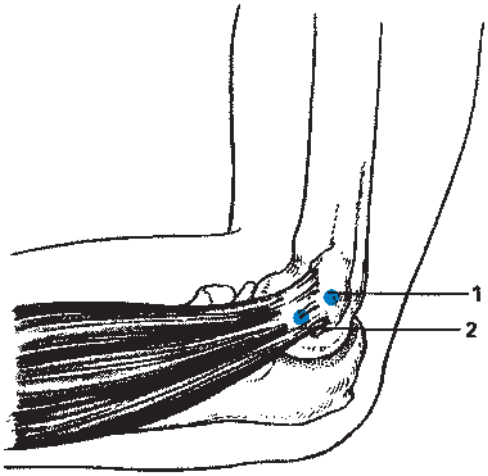


Fig 19.26 • Sites of lesions in the common flexor tendon: 1, tenoperiosteal; 2, musculotendinous.

Technique: infiltration

The patient lies on the couch with the upper arm abducted and externally rotated so that the elbow lies on the lap of the therapist, who sits next to the patient. The elbow is held in almost full extension and in full supination. The tender spot is sought at the anterior aspect of the medial epicondyle. A 1 mL tuberculin syringe is fitted with a fine 2 cm needle. The needle is thrust vertically downwards until the resistance of the tendon is felt and bony contact made. The whole of the affected area is infiltrated by means of a series of withdrawals and reinsertions so as to fill a three-dimensional area. The thumb continuously palpates to make sure that the whole area has been covered.

Technique: deep friction

The patient lies in the same position as for the infiltration, again with the elbow held in full extension and supination by the therapist's ipsilateral arm and hand, of which the thumb palpates for the most prominent point at the medial side. This

Fig 19.27 • Infiltration of the tenoperiosteal junction.

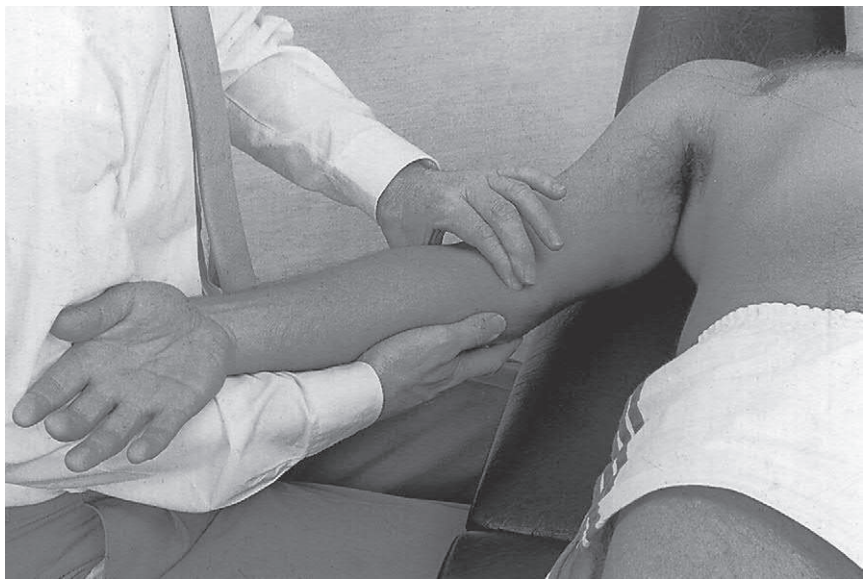


Fig 19.28 • Deep friction to the tenoperiosteal junction.

is the medial epicondyle. At its anterior aspect the origin of the common flexor tendon can be found.

Transverse friction is performed with the index finger of the other hand, reinforced with the middle finger. Counterpressure is applied with the thumb at the lateral aspect of the elbow. Friction is produced by movement of the entire arm, which results in a flexion–extension movement at the wrist.

Musculotendinous junction

Deep transverse friction is performed 2–3 times a week. It is the only successful treatment and usually leads to recovery in 4–8 sessions.

Technique: deep friction

When the tenoperiosteal junction is found on palpation, the finger is then moved 5 mm distally. The ulnar border of the index finger now lies in contact with the distal border of the epicondyle. The structure feels like a soft, muscular and round mass. The index finger now moves to the medial aspect of the muscle and the transverse friction movement is performed by pulling the hand towards the therapist's body.

The elbow may be slightly flexed. This relaxes the musculotendinous junction, which can be penetrated more deeply.

Refractory golfer's elbow

Sclerosant infiltration

Medial epicondylitis that does not respond properly to the usual approach can be infiltrated with sclerosant solution (PDG) in order to create dense scarring which engulfs the original scar so that muscular contraction force is no longer transmitted.

Counterforce brace

A medial counterforce brace can be applied. It helps to diminish the forces acting on the origin of the common flexor tendon.

Weakness

Weakness of flexion of the wrist is usually the result of a C6 disc protrusion, compressing the C7 nerve root, and occurs in combination with weakness of extension of the elbow and occasionally of adduction of the shoulder.

Access the complete reference list online at **www.orthopaedicmedicineonline.com**

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20

Clinical examination of the wrist, thumb and hand

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Referred pain

When a patient refers to the wrist as the site of symptoms, an area is indicated in which there are many structures that could be responsible. The region that patients call the wrist contains:

- The distal radioulnar joint
- The wrist joint
- The tendons that control the wrist
- The trapezium–first metacarpal joint
- The tendons that control the thumb
- The tendons that control the fingers
- The intrinsic muscles of the hand.

When a patient feels pain proximal to the distal radioulnar joint, this is usually described as being in the forearm. The same applies distally, where pain is described as being in the fingers.

Pain is usually the result of a local lesion, although more proximal causes, such as cervical, shoulder girdle and shoulder disorders, as well as problems at the elbow, may refer pain to the distal part of the upper limb. However, in a lesion in the distal part of the limb, the patient is able to point accurately to the exact site of the lesion and it is only when the clinical examination of the wrist and hand is negative that the lesion should be sought more proximally.

Paraesthesia, being a 'nerve symptom', may result either from lesions higher up in the segment (e. g. cervical spine or thoracic outlet) or from local lesions (e. g. ulnar nerve problem or carpal tunnel syndrome).

History

The history is seldom distinctive and therefore examination must be relied on for diagnosis. However, some questions are important.

- *What is the problem?* The patient describes the symptoms that are experienced in the wrist, thumb, hand or fingers. They are variable and may include pain, paraesthesia, numbness and weakness.
- *How did it start? Was there an injury?* Especially when a capsular pattern is found during the examination, the possibility of a fracture must be considered.
- *Or did it start after particular exertion?* Most ligamentous, muscular or tendinous lesions result from overuse. The symptoms may have started *spontaneously*, as usually happens in arthrotic or arthritic disorders.
- *How has the problem developed?* Because of the distal localization of the lesion, its evolution can only be judged in terms of the intensity of the symptoms, or by their appearance or disappearance, but not by reference of the symptoms.

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Inspection

The typical articular deformities of arthrotic or arthritic changes in the joints are well known.

Local swelling may be found. In dorsal subluxation of a carpal bone, the bony projection is visible when the wrist is held in flexion. A cyst on the dorsal aspect may mimic such a subluxation. The same applies to mal-united fractures, where bony outcrops may be visible on inspection. Palpation or disappearance of a fluid collection after puncture will help to reveal the difference.

Another important aspect is generalized swelling. Swelling coming on quite soon after a trauma – for example, a fall – is highly suggestive of fracture of a carpal bone. Spontaneous swelling occurs in rheumatoid arthritis and is quite often bilateral. In long-standing rheumatoid arthritis, multiple large ganglia may also occur.

In arthrosis at the trapezium–first metacarpal joint, the thumb is often visibly fixed in adduction; osteophytes can be seen and felt.

There may be changes in the colour of the hands, which may suggest a circulatory disorder – for example, Raynaud's syndrome, or a cervical rib pressing on the subclavian artery or vein.

Functional examination

Many different structures – inert and contractile – lie close together and have to be examined. It should be clear that by passive testing, which is meant to examine the inert structures, either stretching or pinching may elicit symptoms. In some instances, painful movement of a contractile structure is provoked – for example, of a tendon within its sheath.

The wrist, thumb and hand are examined using 21 tests (Box 20.1).

The distal radioulnar joint

The two movements described below test the integrity of the distal radioulnar joint. Painful supination is also a localizing sign in tendinitis of the extensor carpi ulnaris in the groove at the distal part of the ulna. The normal end-feel of both movements is capsular (elastic).

Passive pronation

The patient holds the elbow in 90° flexion. The examiner grasps the patient's forearm just proximally to the wrist with both hands. The heel of the contralateral hand is placed on the palmar aspect of the ulna, the fingers of the other hand at the dorsal aspect of the radius. Pronation is performed by a simultaneous movement of both hands (Fig. 20.1a).

Box 20.1

Tests for the wrist, thumb and hand

The distal radioulnar joint
- 2 passive movements

The wrist
- 4 passive movements
- 4 resisted movements

The thumb
- 1 passive movement
- 4 resisted movements

The hand
- 6 resisted movements of the fingers

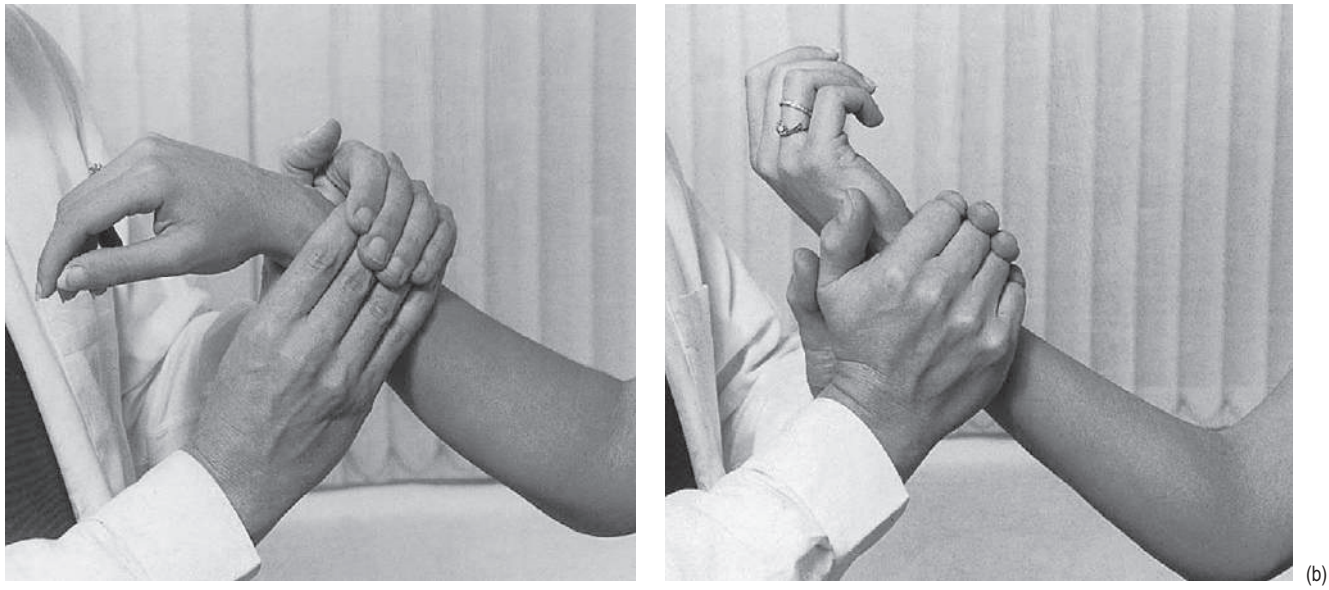


Fig 20.1 • Passive pronation (a) and supination (b).

Passive supination

The examiner changes hand position and puts the fingers of the contralateral hand on to the palmar aspect of the radius and the heel of the other hand on the dorsal aspect of the ulna. Supination is again performed by a movement of both hands (Fig. 20.1b).

The wrist

Passive movements (Fig. 20.2)

The normal end-feel of flexion and extension is capsular (elastic). If these movements are positive, the examiner must be able to tell whether the condition is of the capsular or the non-capsular type (see Ch. 23).

Passive flexion

The patient holds the elbow flexed at a right angle. The examiner takes hold of the patient's forearm with the contralateral hand, in order to achieve good fixation. With the other hand, the patient's hand is grasped and the wrist is flexed to the end of its range. The movement stretches the structures on the dorsal aspect of the wrist (ligaments, tendons) and pinches some structures on the palmar aspect (Fig. 20.2a).

Passive extension

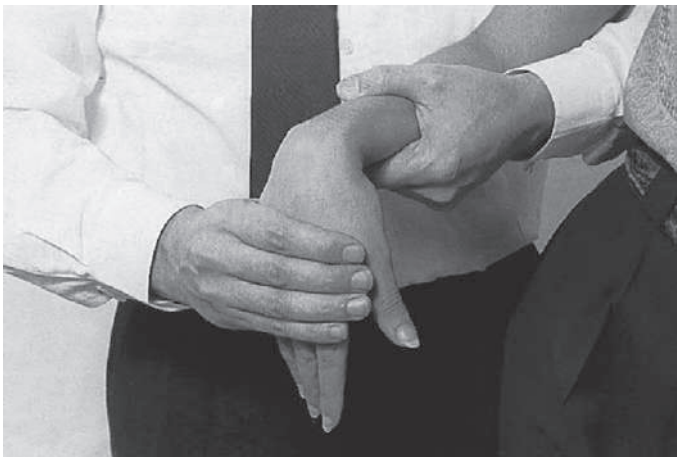
Using the same grip, the examiner can bring the wrist into extension. Extension stretches the palmar tissues and pinches other tissues dorsally (Fig. 20.2b).

Passive radial deviation

The examiner brings the patient's hand into radial deviation, which stretches the structures at the ulnar side of the wrist – the ulnar collateral ligament and the extensor carpi ulnaris (Fig. 20.2c).

Passive ulnar deviation

The examiner brings the patient's hand into ulnar deviation, stretching the structures at the radial side of the wrist – the radial collateral ligament and the tendons in the first tunnel (the extensor pollicis brevis and abductor pollicis longus) (Fig. 20.2d).



(a)

(b)

(c)

(d)

Fig 20.2 • Passive movements of the wrist: (a) flexion; (b) extension; (c) radial deviation; (d) ulnar deviation.

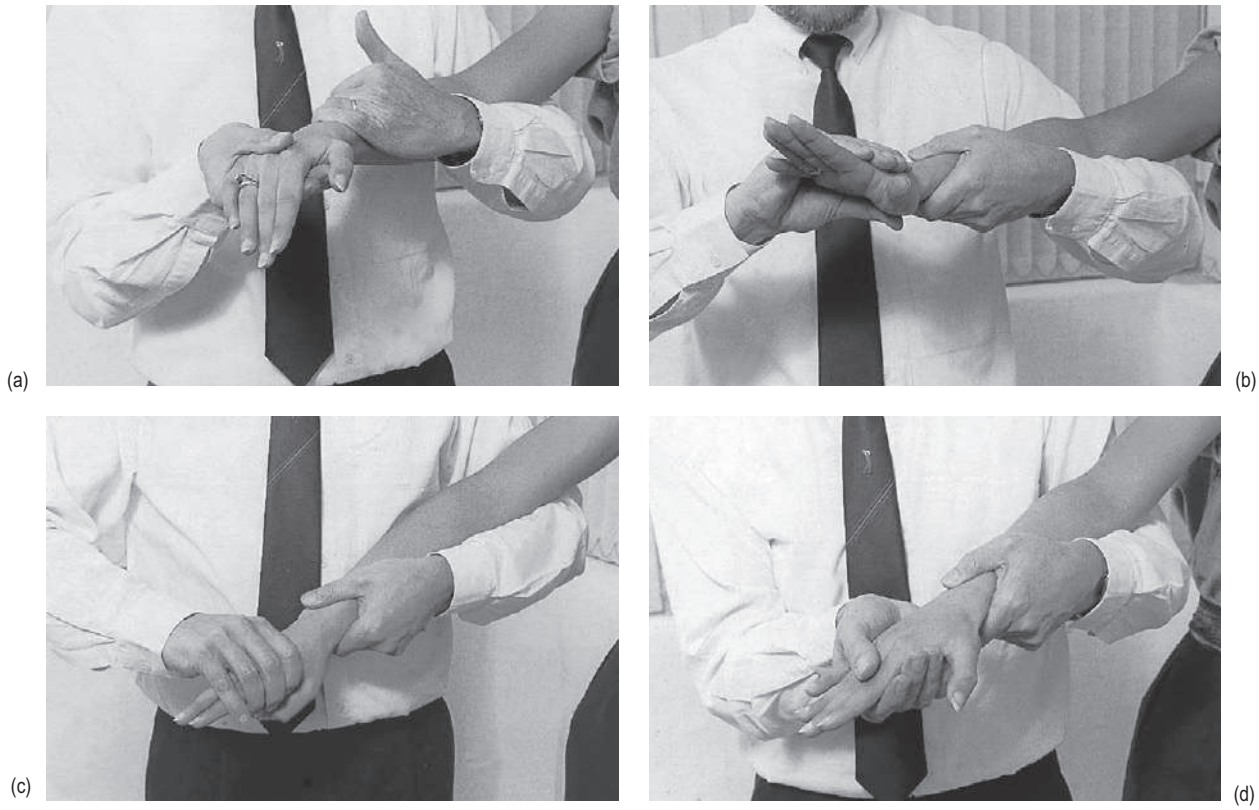


Fig 20.3 • Resisted movements of the wrist: (a) flexion; (b) extension; (c) radial deviation; (d) ulnar deviation.

Resisted movements

For the resisted tests (Fig. 20.3) the patient's elbow is kept in extension. This puts more stress on the contractile structures of the wrist and makes it possible to detect even the slightest tendinous and muscular lesions.

Resisted flexion

The patient's hand is held in the neutral position. The examiner's contralateral arm is passed under the patient's in order to hold the patient's elbow in extension and fixes the forearm with the hand. The other hand is placed under the patient's hand and creates resistance at the palmar aspect.

This test examines the flexors of the wrist and fingers – the flexor carpi radialis, flexor carpi ulnaris, flexor digitorum superficialis and profundus (Fig. 20.3a).

Resisted extension

Fixation of the patient's forearm is as in the previous test. The examiner creates resistance at the dorsal aspect of the patient's hand.

This tests the extensors of the wrist and fingers – the extensor carpi radialis longus, extensor carpi radialis brevis, extensor carpi ulnaris, extensor digitorum communis, extensor indicis proprius and extensor digiti minimi (Fig. 20.3b).

Resisted radial deviation

Resistance is applied at the radial aspect of the patient's hand – the thumb is not involved – and tests the radial deviators of the wrist – the extensor carpi radialis longus and brevis and flexor carpi radialis (Fig. 20.3c).

Resisted ulnar deviation

Resistance to ulnar deviation is applied at the ulnar aspect of the hand and tests the ulnar deviators of the wrist – the extensor carpi ulnaris and flexor carpi ulnaris (Fig. 20.3d).

The thumb

Passive movement

Backwards movement during extension

The patient flexes the elbow to a right angle and presents the hand with the palm upwards. The examiner takes hold of it with the ipsilateral hand and fixes it. With the other hand the thumb is brought into extension. Then the thumb is moved backwards so as to stretch the anterior part of the capsule of the trapezium–first metacarpal joint (Fig. 20.4).

Resisted movements (Fig. 20.5)

Resisted extension

The patient presents the hand with the thumb upwards and it is fixed by the examiner's contralateral hand. Resistance is then applied with the thumb of the other hand at the dorsal aspect of the distal phalanx. Extension tests the extensors of the

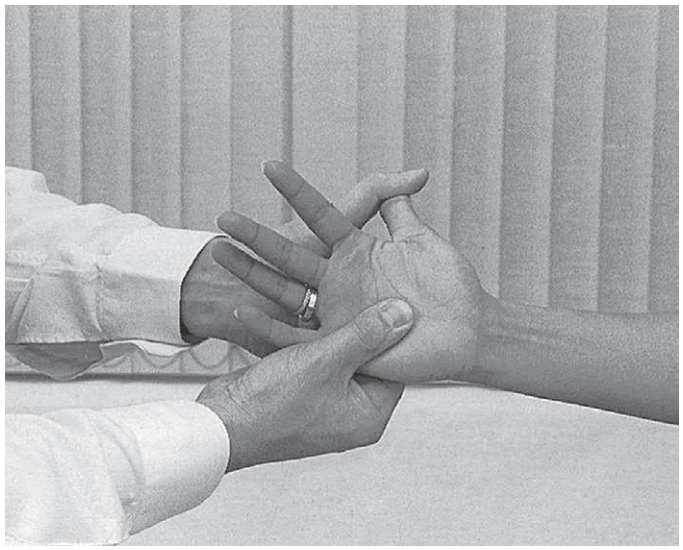


Fig 20.4 • Passive movement of the thumb.

thumb – the extensor pollicis longus and extensor pollicis brevis (Fig. 20.5a).

Resisted flexion

Resistance is applied to the palmar aspect of the distal phalanx of the patient's thumb and tests the flexors of the thumb – the flexor pollicis longus and brevis (Fig. 20.5b).

Resisted abduction

Resistance is applied to the distal part of the first metacarpal bone and tests the abductors of the thumb – the abductor pollicis longus and brevis (Fig. 20.5c).

Resisted adduction

This movement tests the adductor of the thumb – the adductor pollicis (Fig. 20.5d).

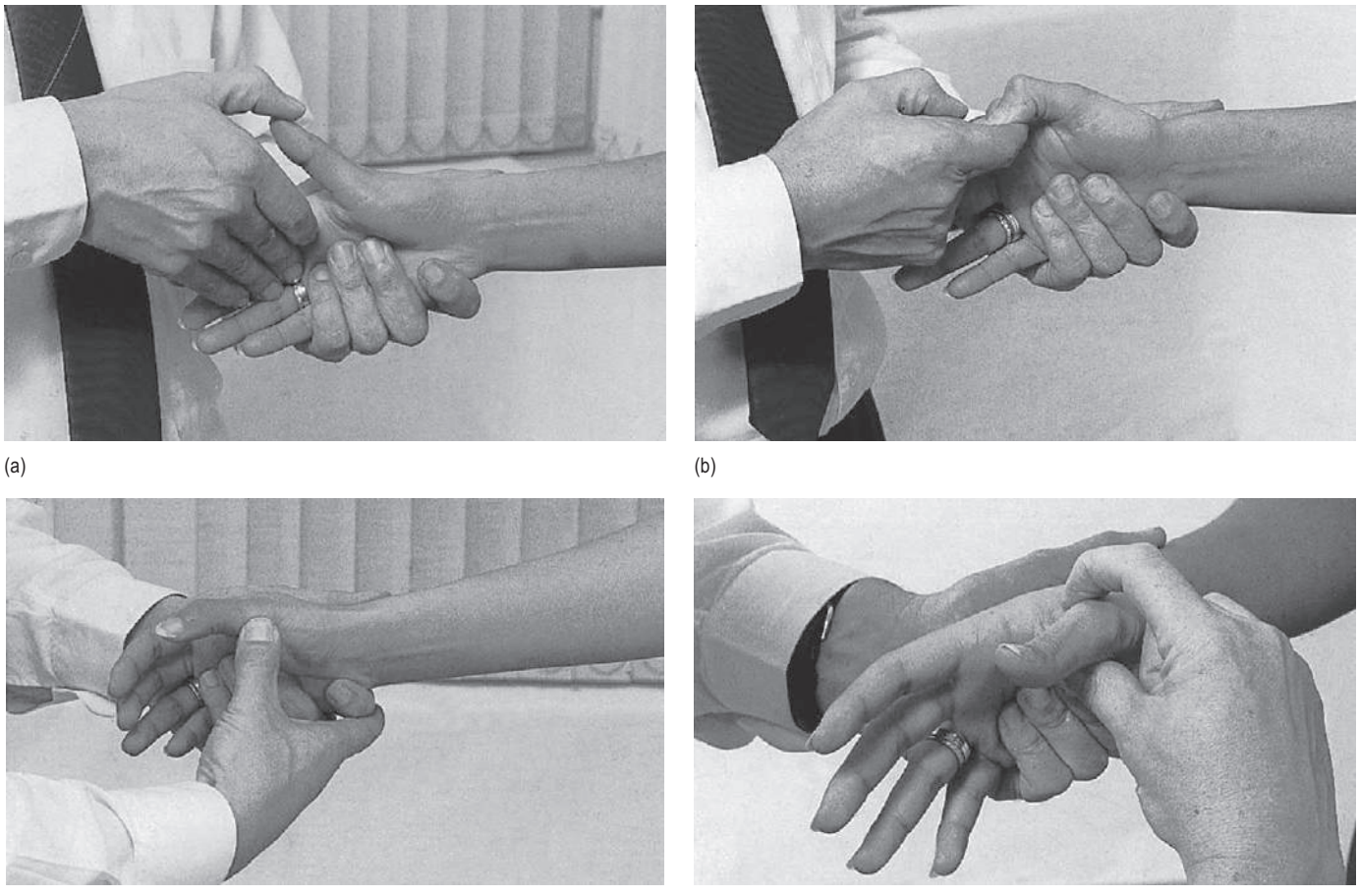


Fig 20.5 • Resisted movements of the thumb: (a) extension; (b) flexion; (c) abduction; (d) adduction.

The intrinsic muscles of the hand: dorsal and palmar interossei

Squeezing with the index and middle fingers

The patient presents the hand in the horizontal position with the dorsal aspect upwards. The examiner puts an index finger between the patient's index (II) and middle (III) fingers and asks the patient to squeeze it, so testing the palmar interosseus of the index finger and the dorsal radial interosseus of the middle finger (Fig. 20.6a).

Squeezing with the middle and ring fingers

The examiner's finger is squeezed between the patient's middle (III) and ring (IV) fingers. The examiner carries out the test as before, with the finger between the patient's middle and ring fingers, so testing the dorsal ulnar interosseus of the middle finger and the palmar interosseus of the ring finger (Fig. 20.6b).

Squeezing with the ring and little fingers

The examiner's finger is squeezed between the patient's ring (IV) and little (V) fingers. The dorsal interosseus of the ring finger and the palmar interosseus of the little finger are tested (Fig. 20.6c).

Resisted separation of the index and middle fingers

The examiner resists the separation of the patient's fingers at the distal phalanges, which tests the dorsal interosseus of the index finger and the dorsal ulnar interosseus of the middle finger (Fig. 20.7a).

Resisted separation of the middle and ring fingers

This assesses the dorsal radial interosseus of the middle finger and the dorsal interosseus of the ring finger (Fig. 20.7b).

Resisted separation of the ring and little fingers

This tests the palmar interosseus of the ring finger and the abductor of the little finger in the hypothenar, abductor digiti minimi (Fig. 20.7c).

Palpation

Palpation with the joints at rest helps to find the exact localization of lesions in ligaments, tendons or muscles. Palpation is also performed for warmth, swelling and synovial thickening.

Palpation during movement may reveal crepitus. Fine creaking during movement of a tendon in its sheath indicates roughening of the gliding surfaces, the result of overuse. This is quite common in the tendons or muscle bellies of the structures that pass through the first and third tunnels: namely, the abductor and extensors of the wrist. Coarser crepitus can indicate tuberculosis or advanced rheumatoid disease.

Accessory tests

These tests (Fig. 20.8) are not part of the standard examination. They are only done when there is a need for more information about the patient's problem.

(a)

(b)

(c)

Fig 20.6 • Squeezing the fingers: (a) II–III; (b) III–IV; (c) IV–V.

Resisted extension of the wrist with the fingers held actively flexed

The patient is asked to flex the fingers and to press the fingertips in the palm of the hand. In this position resisted extension of the wrist is repeated (Fig. 20.8a).

Resisted extension of each finger separately

Resistance is applied at the distal phalanx (Fig. 20.8b).

(a) (b) (c)

Fig 20.7 • Resisted separation of the fingers: (a) II–III; (b) III–IV; (c) IV–V.

Resisted flexion of each finger separately

Resistance is applied at the distal phalanx (Fig. 20.8c).

Tests for carpal tunnel syndrome

These are described in the online chapter *Nerve lesions and entrapment neuropathies of the upper limb*.

The clinical examination of the wrist, thumb and hand is summarized in Box 20.2.

Examination of the fingers

This examination (summarized in Box 20.3) is performed only when the patient clearly presents with a problem in the fingers.

Passive movements

Passive movements are performed at the metacarpophalangeal joints of thumb and fingers, at the interphalangeal joint of the thumb, and at the proximal and distal interphalangeal joints of the fingers. There are four tests for each joint:

- *Flexion*: with one hand the examiner fixes the proximal bone and with the other at the distal bone the joint is brought into flexion.
- *Extension*: with one hand the examiner fixes the proximal bone and with the other at the distal bone the joint is brought back to the starting position. Extension is usually not possible, except in very mobile or hypermobile individuals.
- *Rotation in one direction*: with one hand the examiner fixes the proximal bone. The other takes hold of the distal bone, brings the joint into slight flexion and rotates it in one direction. Very little movement is possible.
- *Rotation in the opposite direction*: the same manœuvre is performed but this time the rotation goes in the opposite direction.

There are two tests for the collateral ligaments:

- *Radial deviation*: the proximal bone is fixed with one hand; with the other the joint is kept in extension and forced into radial deviation, thereby using the distal bone as a lever.
- *Ulnar deviation:* the same technique is used but now the joint is forced into ulnar deviation.

Resisted movements

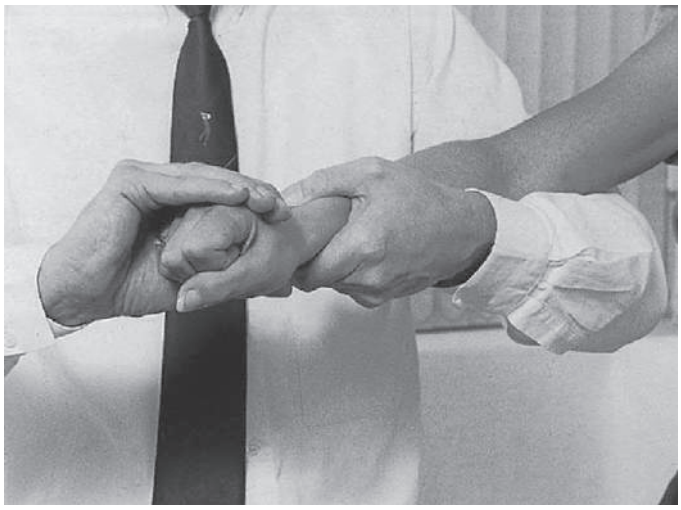
Resisted movements test the tendinous structures in the fingers – the long flexor and extensor tendons. There are two tests; in both, resistance is applied at the distal phalanx.

- *Flexion*: with one hand the examiner takes hold of the patient's wrist and hand; the other is used to supply resistance against the patient's attempt to flex the finger. Resistance is supplied at the palmar aspect of the distal phalanx.
- *Extension*: the examiner again fixes the patient's wrist and hand. With the other hand at the distal phalanx the patient's attempt to extend the finger is resisted.

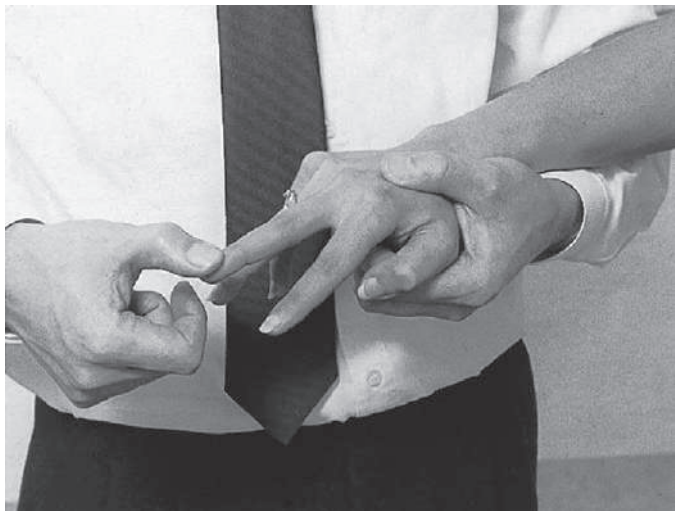
Palpation

Palpation can be done at the level of the different joints and seeks to detect:

- *Fluid* which may be found in joint or ligamentous disorders.
- *Synovial thickening* which accompanies rheumatoid-type arthritis. It is best palpated at the level of the collateral ligaments.



(a)



(b)

(c)

Fig 20.8 • Accessory tests: (a) resisted extension of the wrist, with fingers flexed; (b) resisted extension of one finger; (c) resisted flexion of one finger.

Box 20.2

Summary of the clinical examination of the wrist, thumb and hand

History

Inspection

Functional examination

- Lower radioulnar joint
  1. Passive pronation
  2. Passive supination
- Wrist joint
  3. Passive flexion of the wrist
  4. Passive extension of the wrist
  5. Passive radial deviation of the wrist
  6. Passive ulnar deviation of the wrist
- Wrist muscles and tendons
  7. Resisted flexion of the wrist
  8. Resisted extension of the wrist
  9. Resisted radial deviation of the wrist
  10. Resisted ulnar deviation of the wrist
- Trapezium – first metacarpal joint
  11. Passive backward movement during extension
- Thumb muscles and tendons
  12. Resisted extension of the thumb
  13. Resisted flexion of the thumb
  14. Resisted abduction of the thumb
  15. Resisted adduction of the thumb
- Intrinsic hand muscles
  16. Squeeze II and III
  17. Squeeze III and IV
  18. Squeeze IV and V
  19. Spread II and III
  20. Spread III and IV
  21. Spread IV and V

Palpation

Accessory tests

Box 20.3

Summary of the functional examination of the fingers

Passive movements

1. Passive flexion
2. Passive extension
3. Passive rotation in one direction
4. Passive rotation in the opposite direction
5. Passive radial deviation
6. Passive ulnar deviation

Resisted movements

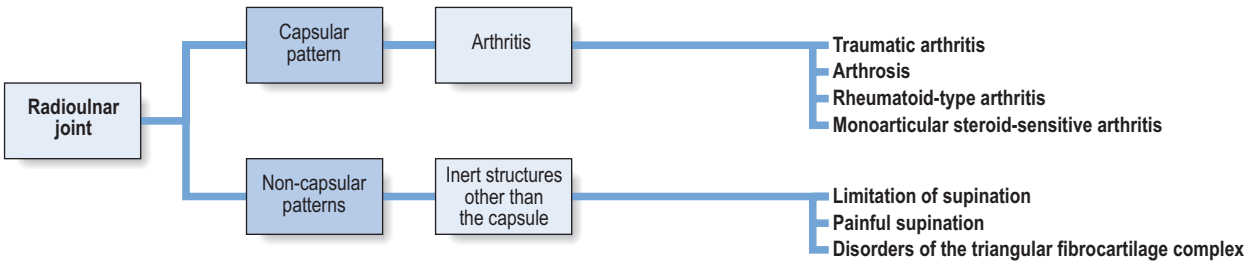
7. Resisted flexion
8. Resisted extension

Palpation

9. For fluid
10. For synovial thickening

Interpretation of the clinical examination of the wrist, thumb and hand

21



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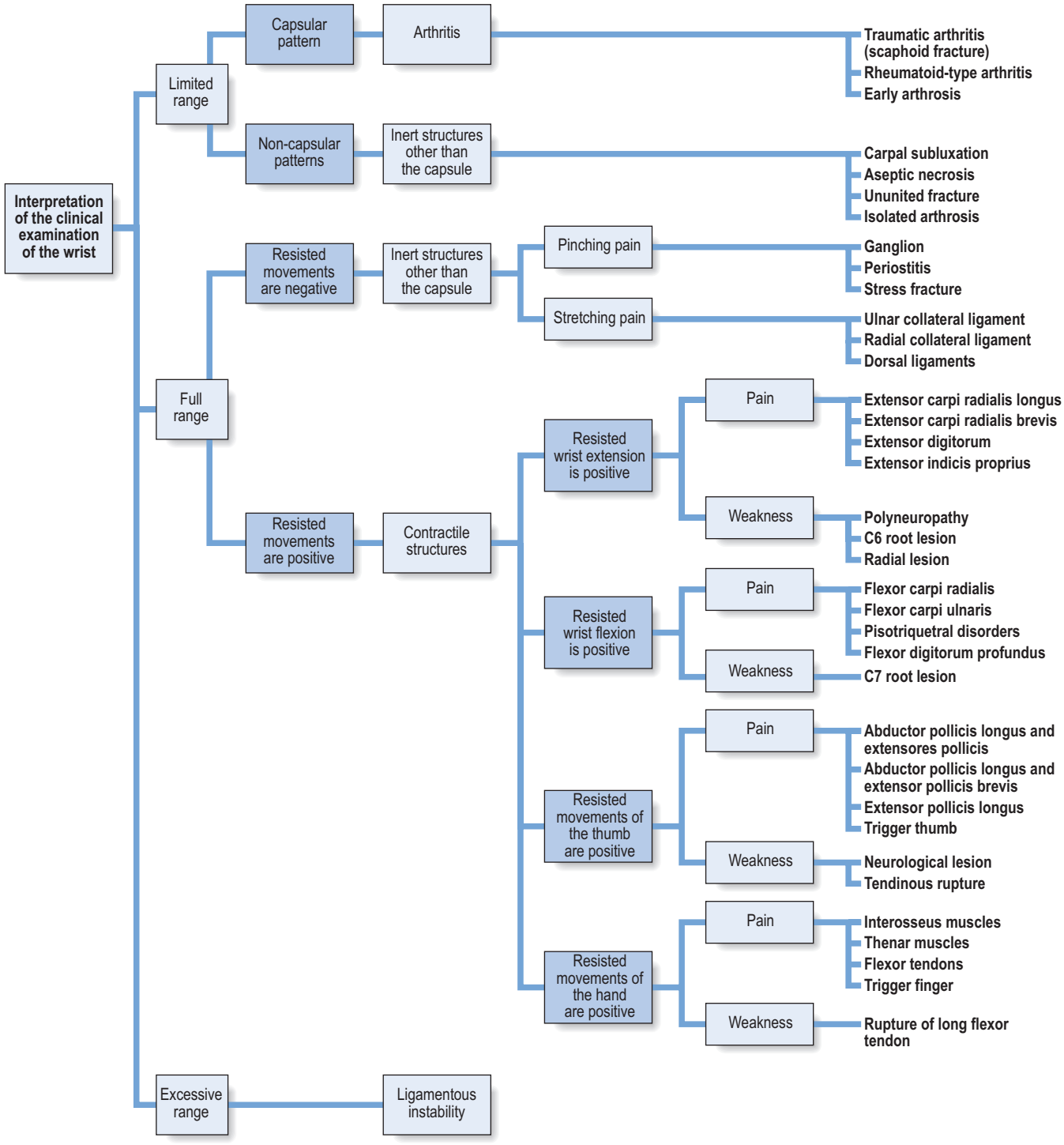
Interpretation of the clinical examination of the wrist
Limited range
Capsular pattern
Arthritis
Traumatic arthritis (scaphoid fracture)
Rheumatoid-type arthritis
Early arthrosis
Non-capsular patterns
Inert structures other than the capsule
Carpal subluxation
Aseptic necrosis
Ununited fracture
Isolated arthrosis
Full range
Resisted movements are negative
Inert structures other than the capsule
Pinching pain
Ganglion
Periostitis
Stress fracture
Stretching pain
Ulnar collateral ligament
Radial collateral ligament
Dorsal ligaments
Resisted movements are positive
Contractile structures
Resisted wrist extension is positive
Pain
Extensor carpi radialis longus
Extensor carpi radialis brevis
Extensor digitorum
Extensor indicis proprius
Weakness
Polyneuropathy
C6 root lesion
Radial lesion
Resisted wrist flexion is positive
Pain
Flexor carpi radialis
Flexor carpi ulnaris
Pisotriquetral disorders
Flexor digitorum profundus
Weakness
C7 root lesion
Resisted movements of the thumb are positive
Pain
Abductor pollicis longus and extensores pollicis
Abductor pollicis longus and extensor pollicis brevis
Extensor pollicis longus
Trigger thumb
Weakness
Neurological lesion
Tendinous rupture
Resisted movements of the hand are positive
Pain
Interosseus muscles
Thenar muscles
Flexor tendons
Trigger finger
Weakness
Rupture of long flexor tendon
Excessive range
Ligamentous instability

Disorders of the lower radioulnar joint

22

CHAPTER CONTENTS

Disorders of the inert structures 329
- Capsular pattern 329
- Traumatic arthritis 329
- Arthrosis 329
- Monoarticular steroid-sensitive arthritis 329
- Rheumatoid arthritis 329
- Non-capsular pattern 330
- Limited supination 330
- Painful supination 330
- Limited pronation 330
- Disorders of the triangular fibrocartilage complex 330

Disorders of the contractile structures 331

Disorders of the inert structures

Pain felt at the wrist during pronation and supination movements of the forearm inculpates the distal radioulnar joint. The source can be the joint capsule,[1] the ligaments[2–4] or the articular disc.

Capsular pattern

The capsular pattern of the lower radioulnar joint presents with pain at the end of range of the two movements (pronation and supination, Fig. 22.1) and indicates arthritis. Usually there is only pain at end-range but sometimes there may be equal limitation, or slightly more limitation of supination than of pronation.

Traumatic arthritis

Traumatic arthritis is usually not the result of a single injury but of repeated and excessive pronation/supination movements. There is constant pain, aggravated by movement. Swelling may be seen at the ulnar side of the distal forearm. Treatment consists of one or two intra-articular injections of 10 mg of triamcinolone acetonide.

Arthrosis

When a fracture of the distal part of the radius fails to unite properly, arthrosis at the distal radioulnar joint may follow. Mal-union of the distal part of the ulna does not give rise to persistent problems[5] and painless ulnar styloid non-union is a frequent incidental radiographic finding.[6]

The patient feels discomfort with movement of the joint. On examination, the extremes of both rotations are uncomfortable. The condition may be helped by strapping the distal part of the forearm; if this does not help, intra-articular triamcinolone may be tried.

Monoarticular steroid-sensitive arthritis

Arthritis may develop without a provable cause such as rheumatoid or traumatic arthritis. Furthermore, the condition will worsen when attempts are made to mobilize the joint. Such a condition responds particularly well to intra-articular triamcinolone.[7]

Rheumatoid arthritis

Rheumatoid arthritis (RA) involves the wrist in up to 95% of cases. The distal radioulnar joint is affected in 31–75% of these patients and is frequently the first compartment of the wrist involved,[8] often bilaterally.[9] Triamcinolone suspension injected intra-articularly once or twice a year may keep the joint free from symptoms.[10]

Long-standing rheumatoid arthritis results in ligamentous laxity. At the distal radioulnar joint this leads to the so-called

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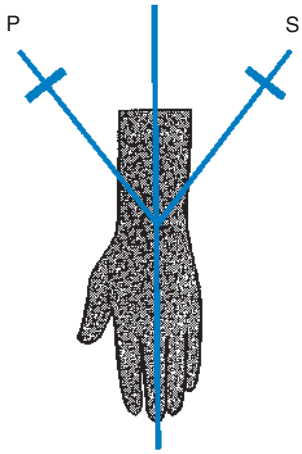


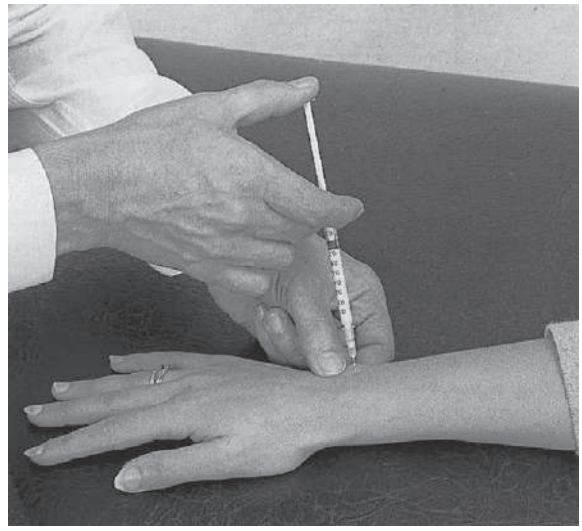
Fig 22.1 • The capsular pattern of the lower radioulnar joint.

'caput ulnae syndrome': dorsal subluxation of the distal part of the ulna, supination of the carpus on the forearm, and palmar dislocation of the tendon of the extensor carpi ulnaris.[11–14]

Technique: intra-articular injection

The patient sits at the couch with the arm lying in pronation. A 1 mL syringe filled with triamcinolone acetonide and fitted with a 2 cm needle is used. The joint line, which is very short, is identified just radially to the head of the ulna. Gliding movements between the ulna and radius may help to find it. As the extensor digiti minimi tendon lies just dorsal to the joint line, care must be taken to avoid puncturing it (Fig. 22.2).

The needle is inserted vertically downwards at the midpoint of the joint line, about 5 mm proximal to the lower edge of the ulna. It is thrust down and will hit bone at about 1.5 cm. It is then manœuvred in an oblique direction towards the radius, until it slips beyond it without resistance. The injection is then carried out.



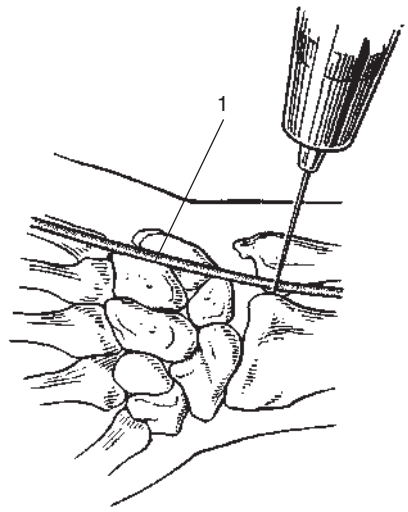


Fig 22.2 • Injection of the radioulnar joint: 1, extensor digiti minimi.

Non-capsular pattern

Limited supination

After a mal-united Colles' fracture, shortening of the radius may be responsible for an irreversible limitation of supination only, with the end-feel of a bony block.[15,16] The movement may be painful in recent cases but should become painless in due course.

A dorsal dislocation of the ulna also presents with a block to supination and a visible dorsoulnar prominence. The mechanism for dorsal subluxation and dislocation is extreme pronation and extension, which pull the ulnar head out through the dorsal capsule. Triangular fibrocartilage complex avulsion and attenuation of the palmar radioulnar ligament will allow this dislocation.[17]

Painful supination

In tenosynovitis of the extensor carpi ulnaris, passive supination may be painful at the end of the range. This is a localizing sign, indicating that the lesion lies in the groove at the base of the ulna. The tenosynovitis will, of course, be diagnosed by interpreting resisted movements at the wrist (see p. 345).

Limited pronation

A block to pronation is present in palmar dislocations of the ulna. On inspection–palpation there will be a volar–ulnar prominence and a palpable radial sigmoid notch.[18]

Disorders of the triangular fibrocartilage complex

During the last few decades it has become obvious that triangular fibrocartilage complex (TFCC) tears are a common source of ulnar-sided wrist pain. The TFCC plays an important role in load bearing across the wrist, as well as in distal radioulnar joint stabilization.

Palmer devised a classification system of TFCC tears in 1989.[19] The main division is between traumatic type I and atraumatic (degenerative) type II tears. The traumatic conditions (type I) follow hyperpronation or axial load-and-distraction injury to the ulnar part of the wrist (e.g. fall on an outstretched extremity[20]) and include perforation and avulsion[21] with or without fracture.[22] Type IA (***A***vascular articular disc) tears are the most common. The other type I tears are peripheral in nature: type IB (***B***ase of the styloid) tears; type IC (**C**arpal detachment) tears; type ID (detachment from the ra***D***ius). The degenerative disorders (type II) result from chronic injuries after repetitive loading on the ulnar side of the wrist.[23] They vary from triangular fibrocartilage wearing to chondromalacia and ligament perforation.[24] Degenerative changes in the TFCC often accompany those in the distal radioulnar joint.[25]

TFCC disorders result in ulnar-sided wrist pain.[26] Uncomplicated cases show a capsular pattern at the radioulnar joint. Complicated cases may present with some subluxation of the joint (limitation of pronation or supination). A provocative test for TFCC lesions, the ulnar grind test, has been described. It involves dorsiflexion of the wrist, axial load, and ulnar deviation or rotation. If this manœuvre reproduces the patient's pain, a TFCC tear should be suspected.[27] Another quick and highly sensitive test to evaluate tears of the TFCC is the 'press test', which axially loads the wrist in ulnar deviation as the patient pushes him- or herself up from a seated position.[28] The best place to palpate the TFCC is between the tendons of the extensor and flexor carpi ulnaris, distal to the styloid and proximal to the pisiform. In this soft spot of the wrist, there are no other structures than the TFCC.[29] Acceptable methods to confirm the clinical diagnosis are magnetic resonance imaging (MRI)[30] and high-resolution ultrasonography.[31]

The treatment depends on type and degree of the lesion. Most symptomatic lesions respond very well to relative rest and one or two intra-articular injections into the distal radioulnar joint. Surgery is the treatment of choice when gross instability occurs. Instability is found when the ligamentous components of the TFCC proper – the dorsal and palmar radioulnar ligaments – are torn.[32] Early surgery is then preferred.[33,34] Chronic disorders of the TFCC, often combined with instability, require arthroscopic[35–37] or open repair,[38] including ulnar shortening.[39,40] The results are good.[41]

Disorders of the contractile structures

Resisted pronation and supination are not tested in the standard functional examination, because they are not relevant at this level. However, resisted pronation can be performed as an accessory test in order to examine the pronator quadratus muscle. This being said, a lesion of this structure has never been described and does not seem to exist. Resisted pronation movement also tests the common flexor tendon (in the case of golfer's elbow) and the pronator teres muscle, but in lesions of these two structures, pain is felt near the elbow (see Ch. 19).

Resisted supination does not test any structure in the wrist region, only those at the elbow – the brachial biceps and supinator brevis.

Access the complete reference list online at **www.orthopaedicmedicineonline.com**

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Disorders of the wrist

23

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Lesions at the wrist often have a traumatic cause – either a single injury or overuse – and are localized. Usually the patient can locate the lesion precisely because wrist and hand disorders seldom cause referred pain. If the symptoms appear spontaneously and are diffuse over the entire wrist, arthritis, usually of the rheumatoid type, is very likely. Further data obtained from the history include pain at rest, pins and needles, twinges and functional loss. The interpretation of the functional examination is classic and encompasses inert structures (capsular, non-capsular and ligamentous lesions) and contractile structures.

Disorders of the inert structures

Limited range: capsular pattern

The capsular pattern is an equal degree of limitation of flexion and extension (Fig. 23.1). In advanced arthritis, fixation of the wrist in the neutral position takes place.

Traumatic arthritis

The discovery of a capsular pattern following a trauma should always induce further examination. Uncomplicated traumatic arthritis is rare and the finding of a post-traumatic capsular pattern is highly suggestive of a fracture. The radiographic picture is unreliable because it may take up to several weeks for radiological evidence of a fracture to be conclusive.[1] This delay may be responsible for serious complications because failure to diagnose and immobilize carpal fractures often leads to non-union and avascular necrosis.[2,3] However, the clinical picture is so evident that the diagnosis should not be missed: any case of traumatic arthritis of the wrist lasting for longer than 2 days should be considered as a fracture.

Warning

A capsular pattern lasting for more than 2 days after trauma makes fracture, either of a carpal bone or of the epiphysis of the radius, a strong possibility.

History and examination

The history is that of an injury, varying from a simple thrust to a fall onto the outstretched and dorsiflexed hand. There is

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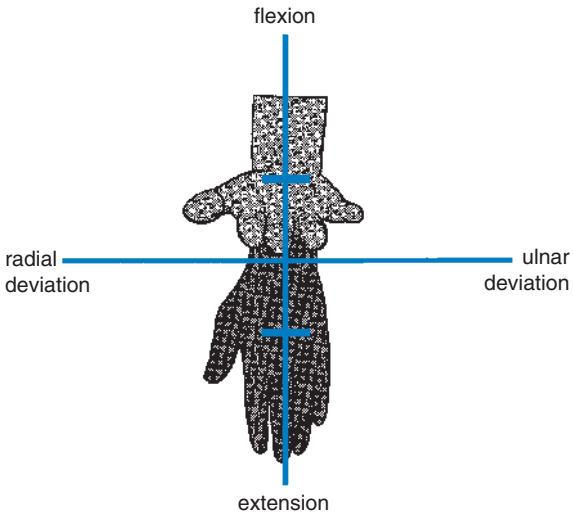


Fig 23.1 • The capsular pattern at the wrist.

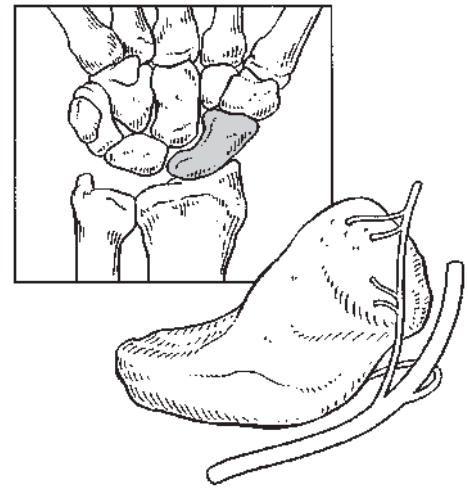


Fig 23.2 • Vascular supply to the scaphoid.

instant and severe pain that prevents hand movement. Initially the pain is localized but spreads over the entire wrist later on.

Inspection shows swelling of the wrist. On examination, a capsular pattern is found: passive flexion and extension are very painful and limited. The end-feel is that of a vibrant twang caused by a protective muscle spasm. Passive deviation of the wrist towards the painful side also increases the pain – a compression phenomenon that provides further evidence of a bony lesion. Careful palpation of the bones reveals localized tenderness. Most often the scaphoid bone is involved.[4] In this case, tenderness will be elicited in the anatomical snuffbox.[5] This is often described as a classic sign of scaphoid fracture and has a reported sensitivity of 90% but a low specificity of 40%.[6] The scaphoid compression test also has a high sensitivity but a better specificity for scaphoid fractures. This test is performed by grasping the thumb and applying an axially directed compressive force along the line of the thumb metacarpal; wrist pain indicates a positive result.[7]

The scaphoid bone spans the joint between the proximal and distal rows of carpal bones and is most vulnerable: scaphoid fracture comprises between 51 and 78% of all carpal fractures.[8–11] Young males and persons between 10 and 19 years of age are at highest risk for scaphoid fracture.[12–14]

Scaphoid fractures occur most easily during loading of a hyperextended wrist, possibly in combination with ulnar deviation. This movement tightens the palmar ligaments (the radiocapitate and radioscaphoid ligaments), so stabilizing the scaphoid against the radius. As the proximal pole is strongly stabilized, further loading and extension of the wrist causes bending forces on the distal pole, which may result in fracture.[15,16]

Fractures may occur at any level in the scaphoid but the more proximal the fracture, the greater the chance of avascular necrosis of the proximal fragment.[17,18] The main reason for this is the special nature of the scaphoid's blood supply.[19] The blood supply to the scaphoid can be divided into extraosseous and intraosseous sources. The extraosseous blood supply is primarily derived from a branch of the radial artery, the artery to the distal ridge of the scaphoid.[20] The branches of this vessel enter the scaphoid through a foramen at the dorsal ridge at the level of the wrist (Fig. 23.2). These vessels then run proximally and palmarly within the medullary chamber, forming the intraosseous supply to the proximal pole.[21] Since vascularity of the proximal pole is limited and dependent on intraosseous flow, acute proximal pole fractures have a potentially prolonged healing period, averaging 3–6 months, and there is higher incidence of non-union.

Because of the danger of avascular necrosis developing, it is important to recognize a scaphoid fracture as quickly as possible. Acute scaphoid fractures are often missed on initial plain radiographs. Therefore an initial negative radiograph does not alter the clinical diagnosis. When clinical suspicion of a scaphoid fracture is high and plain films are negative, the traditional recommendation is for these patients to be immobilized in a thumb spica splint or cast, with repeat radiographs after about 2 weeks.[22]

Warning

It is important to diagnose a scaphoid fracture as soon as possible in order to avoid avascular necrosis. In post-traumatic arthritis lasting longer than 24 hours, the condition should be regarded as a fracture until the contrary has been established.

Alternative imaging modalities for diagnosis include bone scans and magnetic resonance imaging (MRI). A bone scan reportedly shows focally increased uptake within 72 hours and is very sensitive for detecting a fracture, but may not be extremely specific.[23–25] MRI has been reported to have 95–100% sensitivity and almost 100% specificity for scaphoid fractures.[26–28] Additional advantages of MRI include its potential to assess vascularity of the proximal scaphoid pole when a fracture is present.[29]

Treatment

Treatment consists of immediate immobilization of the wrist, which is protective and can decrease the incidence of non-union and avascular necrosis.[30] The best position remains unclear. Several dissection studies have come to different conclusions.[31–34] Immobilization seems to be best performed with a thumb–spica cast, holding the thumb in the palmar abducted position and the wrist in the mid-position but with an element of deviation towards the site of the fracture, which, for the scaphoid, would be radial deviation. This helps to press the fractured surfaces together and stimulates the healing process.[15] The time needed for a scaphoid fracture to heal is variable, from a minimum of 6 weeks to as long as 9 months,[35] but 90% of fresh fractures heal with adequate treatment.[30]

Fractures of the other carpal bones can occur. Frequently, multiple radiographic views and/or follow-up studies with tomograms or bone scans may be necessary for a definitive diagnosis.[36] Displaced fractures may require open reduction and internal fixation.[37,38]

Rheumatoid arthritis

The wrist can become affected by any type of rheumatic disorder, such as systemic lupus erythematosus, progressive systemic sclerosis, psoriatic arthritis, gout and pseudogout, ankylosing spondylitis and sarcoidosis, but rheumatoid arthritis is one of the commonest, frequently affecting both wrists[39] and often following involvement of the fingers. In the acute stage a capsular pattern is found on examination, together with swelling, warmth and synovial thickening. At this stage MRI can be a useful technique for reaching a diagnosis.[40] Recently joint ultrasound has also proved to be an important tool to evaluate synovitis of the wrist.[41] In addition to pain, the more chronic cases present with gross limitation of movement. At this stage, there is no longer any warmth and even the swelling has diminished; diagnosis is easily made on conventional radiographs.

In the acute stage, the patient can be treated locally by immobilization of the wrist for some weeks. In the subacute and chronic stages, triamcinolone is a much better approach. One infiltration of 20 mg of triamcinolone acetonide is given at all the different areas of synovial thickening and/or tenderness, which are first precisely identified by palpation. Although treatment is very painful, the results are spectacular and symptoms at the wrist tend not to recur.

Arthrosis

Arthrosis may come on spontaneously as age advances or may be secondary to severe injury or overuse of the wrist (e.g. professional causes).

The patient complains of stiffness and of some discomfort on certain activities. On examination a moderate capsular pattern is found, with a hard end-feel and crepitus on passive testing.

The radiograph shows osteophytes, sclerosis of the bony margins and diminished joint spaces. It is important to realize that minor arthrosis, visible on radiograph, does not always interfere with the function of the wrist.

Because the disability is usually minor in arthrosis, treatment is seldom necessary. In rare instances where fairly heavy loads are imposed on the joint, arthrodesis may be warranted.[42]

Limited range: non-capsular pattern

Carpal subluxation

Subluxation of a carpal bone, most often the capitate, occurs quite frequently. The diagnosis is purely clinical and, once the condition is recognized, it can be dealt with successfully by conservative management. Recurrence is not at all uncommon and may ultimately cause problems.

History and examination

The patient complains of pain on certain movements at the dorsal aspect of the wrist, especially when weight is put on an extended wrist. The symptoms may come on spontaneously or as the result of a minor injury in flexion.

On examination, signs of internal derangement are found (Fig. 23.3): one movement, most often extension, is limited with – certainly in acute cases – an end-feel of muscle spasm. Other movements are full-range, although often painful – the result of overstretching the irritated ligaments (see p. 34). The bony subluxation may be visible and palpable if the wrist is held in flexion. There is local tenderness in the ligaments.

The radiograph is negative: the edge of the subluxated bone appears merged with the others and cannot be visualized separately on the radiograph, nor can its position be measured against the superimposed margins of the other bones (Cyriax:[43] p. 184).

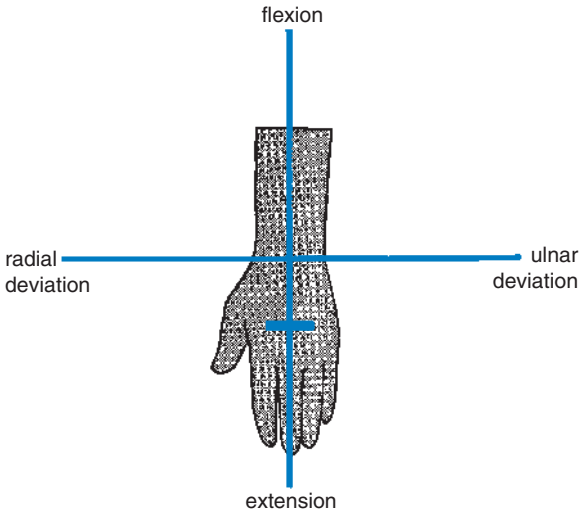


Fig 23.3 • The non-capsular pattern in carpal subluxation.

The clinical examination supplies the diagnosis: the joint is blocked in one direction only, (which is typical of internal derangement) and there is a visible displacement on inspection. Instant reduction during manipulation confirms the tentative diagnosis.

Palmar subluxation, mostly of the lunate bone, is very exceptional. It gives rise to (slight) limitation of flexion and may compress a palmar branch of the median nerve, causing paraesthesia in the corresponding territory (see online chapter *Nerve lesions and entrapment neuropathies of the upper limb*).

Differential diagnosis

Limitation of wrist extension does not necessarily indicate carpal subluxation and differential diagnosis should be made from the following disorders:

- *A ganglion* at the dorsum of the wrist usually feels softer on palpation. It does not respond to manipulative treatment but can be diagnosed and treated by puncture.
- *Kienböck's disease, un-united fracture and isolated arthrosis* have a different history and can easily be detected on radiography (see p. 337).
- *A carpal boss* (see p. 338).

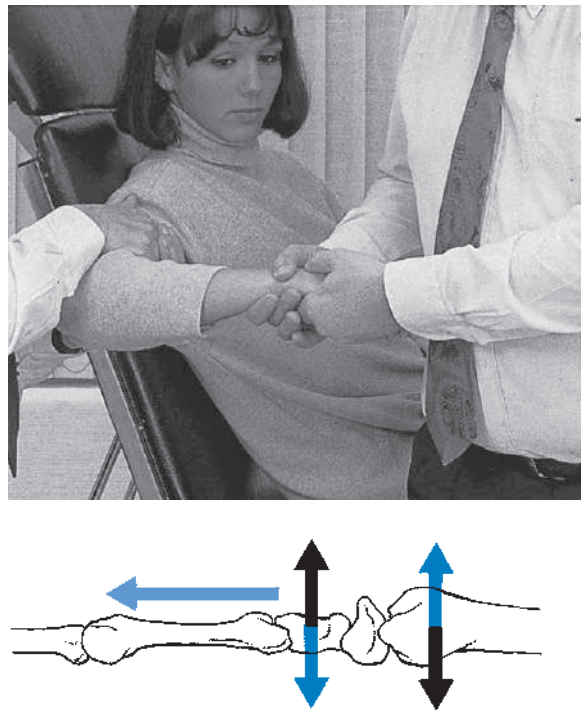


Fig 23.4 • Basic manipulation for carpal subluxation.

Treatment

Treatment consists of manipulative reduction, which restores extension. Because of the accompanying sprain of the dorsal ligaments, flexion often remains painful. A few sessions of deep transverse friction will resolve this problem. If the condition recurs, manipulation is repeated. Frequent recurrences indicate instability and should be treated with infiltration of a sclerosant solution into the ligaments attached to the subluxated bone.

Basic technique: manipulation for carpal subluxation

The patient sits on a couch, the arm held in abduction against the back of the couch. An assistant grasps the patient's upper arm just proximally to the elbow and holds it firmly against the couch. One foot is placed forwards for the manipulator's benefit: the latter will place a foot against it.

The manipulator stands level with the patient's hip, between the couch and the patient's arm, and places the ipsilateral foot against the assistant's foot; later, this will enable the manipulator to lean sideways and use the body weight for the pull required to obtain traction on the wrist joint. With the ipsilateral hand, the patient's forearm is grasped just proximally to the radiocarpal joint, with the thumb on the dorsal aspect of the forearm. With the contralateral hand, the patient's wrist is held with the thumb dorsally on the subluxated bone.

The aim of the manipulation is to perform a gliding movement between the rows of carpal bones during traction. The technique is executed as follows. The manipulator leans sideways but makes sure that the pull is only carried out with the distal hand, so as to obtain traction on the wrist. After the slack has been taken up, both hands are moved vertically up and down in opposite directions (Fig. 23.4). The little finger of the contralateral hand is kept in the palm of the patient's hand to control movement; the manœuvre should result in a pure gliding movement and flexion–extension should be avoided.

This gliding movement is performed several times in one manœuvre. The movements of the wrist are then re-examined and the manipulation repeated until a full range of extension has been restored or until no further benefit results. If there is no immediate result, the attempt should not be abandoned too quickly. It may sometimes help to slightly change the position of the patient's hand during the manœuvre and to build in a bit more flexion, extension or radial or ulnar deviation.

When this technique does not lead to full recovery, the next manœuvre is tried.

Second technique: manipulation for carpal subluxation

This technique is never used first but is introduced only when the bone is partially reduced. The aim is to perform a squeezing of the wrist during traction.

The patient, assistant and manipulator maintain the same positions as in the previous technique. The manipulator's distal hand is now moved slightly downwards, so that it grasps the patient's hand at the level of the bases of the metacarpal bones. Using the other hand, the patient's wrist is approached from above and encircled with thumb and index finger. The metacarpophalangeal joint of the index finger is placed on the subluxated bone. The manipulator's forearm is held vertically (Fig. 23.5). The manipulator leans sideways, applying traction with the distal hand and, having taken up the slack, executes a short, sharp squeezing manœuvre.

Although the manipulative thrust is a pure squeezing, there is an involuntary downwards movement of the hand, which helps reduction. This technique can be repeated several times.

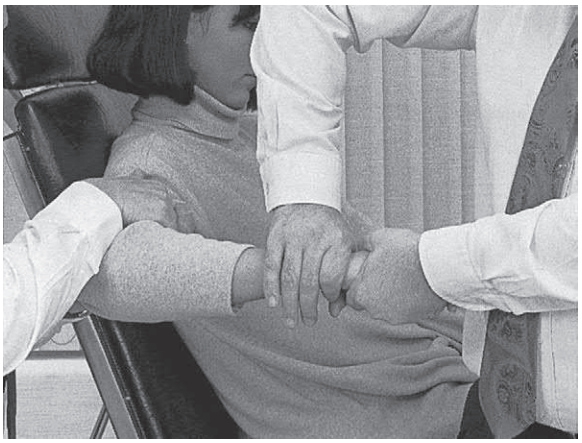


Fig 23.5 • Second manipulative technique for carpal subluxation.

Fig 23.6 • Alternative manipulation for carpal subluxation.

Alternative technique: manipulation for carpal subluxation

The patient stands next to the couch, with the forearm, stabilized by the other hand, placed on the couch, the distal part level with the edge. The manipulator stands in front of the patient and grasps the hand in such a way that one thumb, reinforced with the other, is placed dorsally on the subluxated bone, while one index finger, reinforced by the other, is placed on the palmar aspect of it. The little fingers are placed in the palm of the patient's hand, in order to prevent flexion–extension movements during the manipulation (Fig. 23.6).

Traction is applied with both hands and, after having taken up the slack, an anteroposterior glide is performed with emphasis on downward movement. Several movements are performed and the patient is re-examined after each manœuvre.

Other joint problems

The four following disorders, all visible on radiograph, give rise to limited extension only.

Aseptic necrosis of the lunate bone (Kienböck's disease)

Avascular necrosis of the lunate was first described by Peste in 1843.[44] The condition was then forgotten until 1910, when Kienböck recorded lunatomalacia, which he assumed had a vascular traumatic aetiology.[45] Despite recognition of this disease entity for the past 100 years, its cause is still debated. Some investigators relate it to a stress fracture that leads to devascularization of the major segment of the lunate. Other authors consider the avascular necrosis to be the result of interruption of the vascularization of the lunate bone following excessive shear forces caused by acute trauma or repeated microtrauma.[46] This would happen more easily in those lunate bones that are supplied by a single volar or dorsal blood vessel.[47] Others suggest that a vascular non-traumatic process, with a minor infarction pattern in the proximal subchondral area, may be nearer to the truth.[48]

Avascular necrosis usually affects the dominant hands of males between 20 and 40 years of age.[49,50] The symptoms come on spontaneously or as the result of minor injury, and usually interfere very significantly with work-related activities. The initial symptoms are pain and stiffness, which may vary from moderate to severe and incapacitating. The patient may also mention reduced strength in the hand.[51]

Clinical examination in the initial stage shows a capsular pattern with muscle spasm, due to the accompanying synovitis. Later, painful limitation of passive dorsiflexion becomes obvious, together with pain at the end of range of the other movements – a clear non-capsular pattern.

The classification of Kienböck's disease is based on its roentgenological appearance.[52,49,53]:

- Stage 1 consists of small fracture lines – bone scintigraphy[54] or MRI[55] will be required for diagnosis.
- Stage 2 is rarification along the fracture line, usually on the volar pole.
- Stage 3 shows sclerosis of the bone dorsal to the fracture site.
- Stage 4 shows sclerosis of the bone dorsal to the fracture site, and collapse and secondary fracture with loss of architectural integrity of the lunate.
- Stage 5 shows secondary arthritic changes of the radius.

Treatment methods extend from immobilization to revascularization surgery on the affected bone. However, there is still no gold standard for the treatment of Kienböck's disease.[56] A recent systematic review showed that, to date, there are insufficient data to determine whether the outcomes of any intervention are superior to placebo or the natural history of the disease.[57]

Avascular necrosis in other carpal bones has been described but is rare.[58,59]

Un-united fracture

This leads to pseudoarthrosis and also gives rise to limitation of extension.

Isolated arthrosis

This comes on some years after an un-united fracture, especially of the scaphoid bone. The projecting osteophytes can be seen and felt. Extension is very limited but the condition is not necessarily painful.

Carpal boss

This bony prominence at the base of the second and third metacarpals adjacent to the capitate and trapezoid bones may represent degenerative osteophyte formation and/or the presence of an os styloideum, an accessory ossification centre that occurs during embryonic development. The symptoms of a carpal boss may result from an overlying ganglion or bursitis, an exterior tendon slipping over this bony prominence, or from osteoarthritic changes at this site. Diagnosis is by X-ray.[60]

Full range

Although passive movements of the wrist show a full range of movement, they can be painful at the end of range, showing that a structure is either stretched (ligament) or pinched (impaction syndromes).

Ligamentous lesions

Ligamentous injuries range from minor sprains with no instability (which are discussed here) to complete rupture with gross instability (see p. 341).[61]

Sprain of the ulnar collateral ligament

This disorder is the result of an injury causing a fracture, either a Colles' fracture or fracture of the styloid process of the ulna. The fracture has healed but pain remains at the ulnar aspect of the wrist. On inspection, the deformity of the radius is visible and passive radial deviation causes pain at the ulnar aspect of the wrist. The symptoms can be quickly relieved by an infiltration of triamcinolone.

Technique: infiltration

A 1 mL syringe, filled with 10 mg of triamcinolone acetonide, and a 2 cm needle are used. The patient's arm rests in pronation on the couch. The hand is brought into slight radial deviation. The tender spot is identified, and the infiltration given with bony contact and ligamentous resistance (Fig. 23.7).

Sprain of the radial collateral ligament

This rare disorder results in pain felt at the radial aspect of the wrist during passive ulnar deviation. A more common cause of these symptoms is a tendinous lesion in the extensor pollicis brevis and abductor pollicis longus tendons (see p. 354). Differential diagnosis is easy because, in tendinitis, resisted movements are also positive.

Radial collateral sprain responds well to treatment with either one infiltration of triamcinolone or several sessions of deep transverse friction.

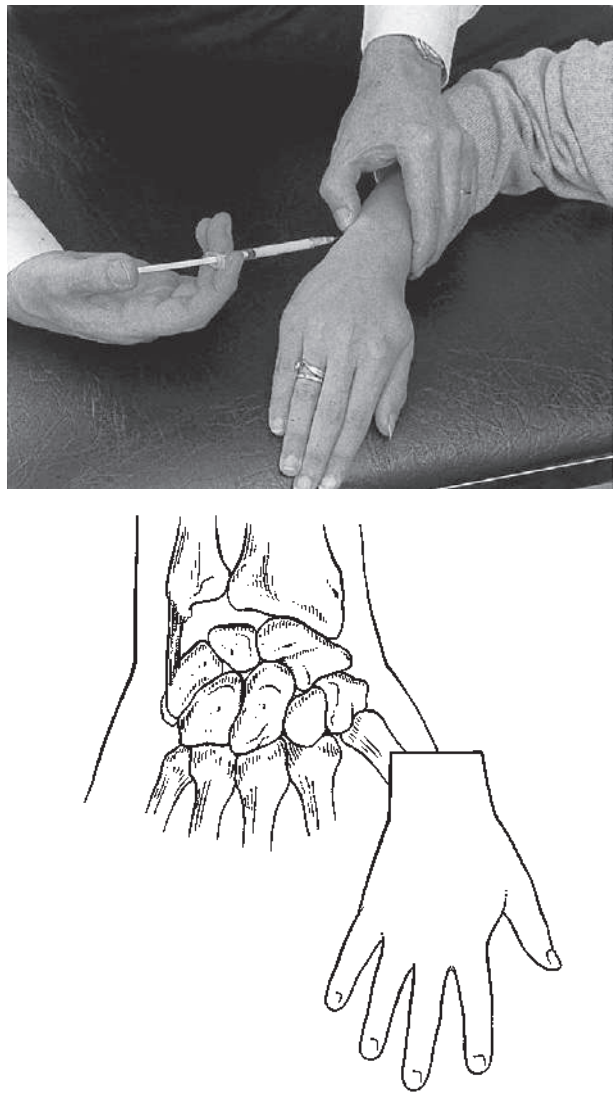


Fig 23.7 • Infiltration of the ulnar collateral ligament.

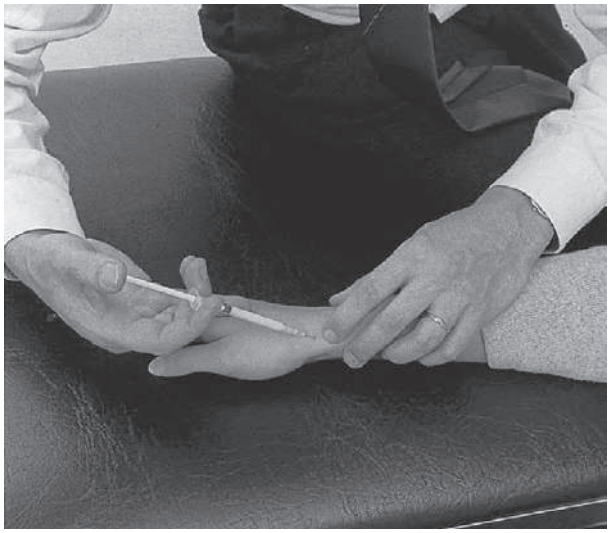
Technique: infiltration

The patient sits with the forearm in the neutral position and the hand on the couch. The wrist is brought into slight ulnar deviation. The tender spot is palpated and 10 mg of triamcinolone acetonide is infiltrated using a 2 cm needle (Fig. 23.8). During infiltration, ligamentous resistance and bony contact should be felt. Care must be taken to avoid puncture of the radial artery.

Sprain of the dorsal ligaments

The most common minor lesions are usually the result of overuse but may also occur when long-standing carpal subluxation causes irritation of the overstretched ligaments.

Pain is felt at the dorsal aspect of the wrist on full passive flexion. Other tests are negative. Careful palpation reveals the exact site of the lesion. Most of the lesions are found in the lunate–capitate ligament but occasionally also in the radiolunate, capitate–third metacarpal or ulnar–triquetral ligament.



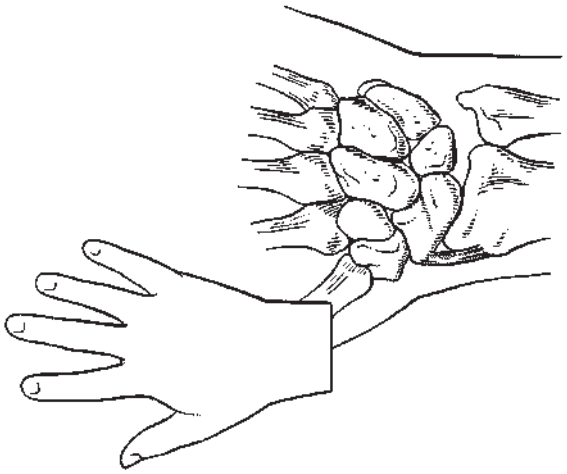


Fig 23.8 • Infiltration of the radial collateral ligament: (a) the intercarpal ligaments; (b) the radiocarpal or carpometacarpal ligaments.

Treatment consists of several sessions of deep transverse friction, which should lead to complete cure, independently of how long symptoms have been present. In the rare case in which it is not clear whether the symptoms are caused by a pure ligamentous sprain or by irritation of the ligaments from carpal subluxation, manipulation may be tried. If it proves ineffective, deep transverse friction is substituted. Even in the chronic stage of a dorsal ligamentous lesion at the wrist, the only effective treatment is deep transverse friction. Other treatments, such as steroid injections, immobilization or operation, are of no use. In order to avoid permanent problems, and because these lesions are often multiple, friction must be given to all the sprained ligaments. Palpation should therefore be performed carefully until all the tender spots have been found.

Technique: deep transverse friction

The patient sits with the hand over the edge of the couch. The therapist sits facing the patient's wrist, which is flexed with one hand within the limits of pain. Friction is imparted with the other hand. The direction depends on the course of the ligamentous fibres: the radiocarpal and carpometacarpal ligaments are aligned in the same longitudinal axis as the forearm, while the intercarpal ligaments have a more or less transverse direction (Fig. 23.9). For a radiocarpal or carpometacarpal ligament, the direction of the friction is transverse on the ulnar–radial axis. Friction is given with either the index finger, reinforced with the middle finger (Fig. 23.10), or with the thumb. As the lesion is very localized, the therapist must make

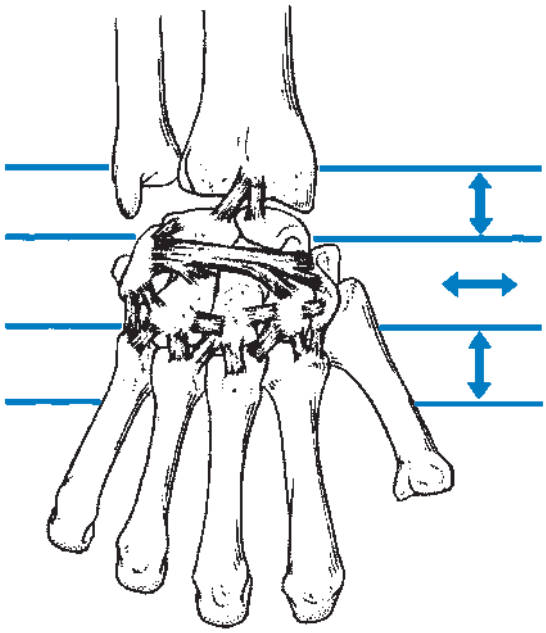


Fig 23.9 • The dorsal ligaments. The arrows emphasize the direction of the ligamentous fibres at the joint lines.

Fig 23.10 • Friction to the dorsal carpal ligaments.

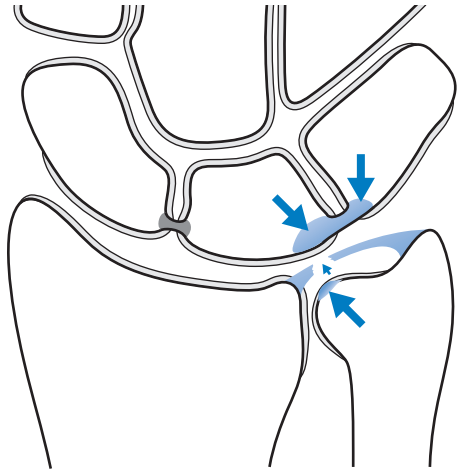


Fig 23.11 • Possible pathology in ulnar impaction syndrome: periostitis of the ulnar head, the ulnar side of the lunate bone and the radial side of the triquetral bone (arrows); lesions of the triangular fibrocartilage complex (small arrowhead).

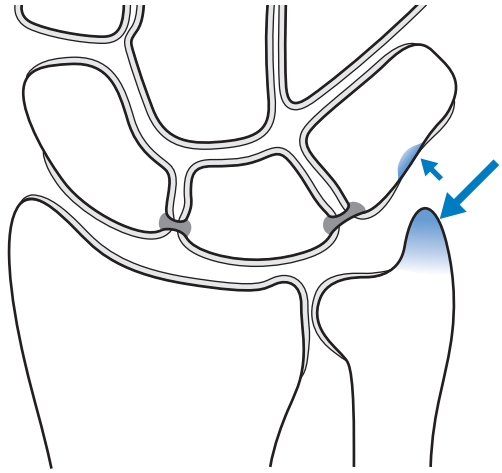


Fig 23.12 • The pathological conditions that characterize ulnar styloid impaction syndrome: periostitis of the proximal pole of the triquetral bone (short arrow) and on the styloid process (long arrow).

sure that the finger remains deep between the extensor tendons and does not pass over them.

An intercarpal ligament is given friction with the thumb and in a proximo–distal direction (see Fig. 23.9).

Wrist impaction syndromes

Dorsal and/or ulnar-sided wrist pain may be caused by periosteal bruising that results from impaction between the carpus and the radioulnar border.

Ulnar impaction syndrome

Ulnar impaction syndrome, also known as ulnar abutment or ulnocarpal loading, is a degenerative condition characterized by ulnar wrist pain related to excessive load bearing across the ulnar aspect of the wrist. Chronic impaction between the ulnar head and ulnar carpus results in periostitis and degeneration of the lunate bone, triquetral bone and distal ulnar head, and degenerative tears of the triangular fibrocartilage complex (Fig. 23.11).[62]

The clinical manifestation of ulnar impaction syndrome generally consists of chronic or subacute ulnar wrist pain, often exacerbated by activity such as loading in extension and relieved by rest. Functional examination shows dorsal pain on passive extension. If the triangular fibrocartilage complex is involved, there will also be pain on forearm rotations.

Radiographic changes include subchondral sclerosis and cystic changes in the ulnar head, ulnar aspect of the proximal lunate bone, and proximal radial aspect of the triquetral bone. MRI is helpful in detecting occult disease in the early stage.[63]

Treatment will depend on the severity of the lesions. Periostitis responds well to local infiltration with 10 mg of triamcinolone acetonide, provided that further irritation is avoided. This can be done by avoiding the extremes of wrist movement in the loaded position or by the application of a splint or a cast. In more advanced cases or in lesions of the triangular fibrocartilage complex, arthroscopy and surgery will be needed.[64,65]

Ulnar styloid impaction syndrome

This ulnar-sided wrist pain is caused by impaction between an excessively long ulnar styloid process and the triquetral bone.[66] The ulnar styloid process is a continuation of the prominent subcutaneous ridge of the shaft of the ulna, which projects distally towards the triquetral bone for a variable distance (2–6 mm).[67] A single or repetitive impaction between the tip of the ulnar styloid process and the triquetral bone results in contusion, which leads to periostitis of the opposing surfaces. If a single-event trauma is forceful enough, fracture of the dorsal triquetral bone may occur.[68]

The diagnosis of this condition is made on the basis of pinching pain during ulnar deviation and radiographic evidence of an excessively long ulnar styloid process. MRI may show chondromalacia of the ulnar styloid process and proximal triquetral bone (Fig. 23.12).[69]

Treatment consists of a local infiltration with 10 mg of triamcinolone. In advanced cases, resection of all but the two most proximal millimetres of the styloid process is the treatment of choice.

Radial impaction syndrome

Pain at the dorsal aspect of the wrist may occur as the result of repetitive extension movements during weight bearing, as frequently happens in gymnastics or other high-energy sports; this may lead to periostitis at the distal epiphysis of the radius or the proximal carpal bones,[70,71] described as 'wrist impingement' syndrome[72] or 'stress reaction'.[73] The pain is elicited each time the patient puts weight on an extended wrist.

On examination, passive extension of the wrist is found to be full but painful, all other movements being negative. Careful

palpation shows the lesion to lie at the inferior border of the radius or at the scaphoid and lunate bones.

Periostitis responds well to local infiltration with 10 mg of triamcinolone acetonide, provided that further irritation is avoided. This can be done by avoiding the extremes of wrist movement in the loaded position or by the application of a splint or a cast, if necessary. Surgery can be performed in recurrent cases or to avoid long-term complications.[74]

Repetitive compressive loading may result in stress fractures of the distal part of the radius, the scaphoid and the capitate bones. Scintigraphy and MRI are diagnostic and immobilization in a cast is therapeutic.[75,76]

Ganglion

A ganglion is a fluid-filled cavity that originates from the joint capsule or tendon sheath. It is very common at the wrist. It occurs dorsally, most often over the scapholunate joint, as well as at the palmar aspect, usually at the trapezioscaphoid joint.[77] Its aetiology has been debated for many years and various hypotheses have been presented: retention cyst, herniation of tendon or capsular synovia, bursal transformation, neoplasia, or mucinous degeneration of fibrous tissue, the last of these being the most widely accepted theory.[78]

The main complaint is the presence of a swelling that was not there before. It may sometimes be painful either at rest or during movement. The size may vary and so does its consistency: it can be either very soft or quite hard, giving the impression of a bony structure. When the ganglion grows, the neighbouring tissues may become compressed and there may be pressure on nervous tissue as well (see online chapter *Nerve lesions and entrapment neuropathies of the upper limb*).

Many techniques have been used to treat this condition, varying from crushing the ganglion (e.g. pressure with the thumbs or a firm slap with a book) to aspiration and infiltration with a steroid or sclerosant solution. These measures usually lead to temporary cure but recurrences are frequent because the cause has not been treated. More consistent results are obtained by surgical removal, although the risks associated with surgery may be more undesirable than the minor symptoms associated with the presence of a ganglion.[79]

Excessive range

Ligamentous instability

According to Ekenstam, instability of the wrist is 'a condition of altered joint kinematics in which one or several carpal bones are permitted abnormal patterns of motion as a result of bony abnormalities, ligamentous lesions or joint laxity'.[80]

Carpal instability remains a very controversial topic. Since 1943, when Gilford *et al*[81] first mentioned instability of the wrist, many authors have worked on the subject. They have developed several hypotheses and proposed different methods of classification. This variability indicates the difficulty in reaching a consensus on such a vague topic as 'instability' in the complex area that is the wrist. There is still a lack of standardization in evaluation methods and in the findings and radiographic criteria. Arthroscopy, however, has helped to provide a clearer picture of the possible underlying mechanisms.[82]

Aetiology

Wrist instability is generally believed to be caused by post-traumatic stretching or rupture of one or several radiocarpal or intercarpal ligaments. The injury is usually hyperextension combined with radial or ulnar deviation and/or pronation or supination.[83]According to some authors, however, it can also be the result of anatomical changes and malalignment subsequent to radial or carpal fracture. Other possible causes of ligamentous laxity are degenerative and inflammatory conditions (i.e. crystal synovitis, rheumatoid arthritis), as well as iatrogenic causes.[84]

Epidemiology and classification

Epidemiological data on carpal instability are still not available, although wrist injuries occur in 28.6% of all accidents.[85] Several methods of classification have been described.[86]

Taleisnik[87,88] has proposed a theory based on the columnar concept of Navarro.[89] This concept recognizes three vertical columns in the wrist: (a) the central (flexion/extension) column contains two blocks – one on the lunate bone and one on the trapezium, trapezoid, capitate and hamate bones; (b) the radial column comprising the mobile scaphoid bone; and (c) the ulnar (rotating) column ,which includes the triquetral bone and the pisiform. Taleisnik therefore classifies wrist instability as being radial, ulnar or proximal.[90]

This concept does not mention the transverse or perilunar patterns (stages I–IV), which were later described by Mayfield *et al*,[91] or the more recently discussed mid-carpal instability, proximal row instability and miscellaneous instability.

The Mayo Clinic proposes a classification, based on the work of Amadio, Cooney, Dobyns and Linscheid.[92–96]

The Lichtman classification[97] deviates from the columnar wrist concept and the consequent longitudinal classification system. It is based on the 'carpal ring concept' – the distal and proximal rows are joined by two links: on the radial side the mobile scaphotrapezial joint and on the ulnar side the rotatory triquetrohamate joint. This concept includes the perilunate and mid-carpal instabilities.

Other classification systems – for example, the Barton classification – have also been proposed.

We consider Saffar's classification[86] to be a clinical and useful approach.

1. Static or dynamic instabilities

   Dynamic instability only shows when, during movement of the wrist or under specific loading, symptoms can be elicited. Static instability is fixed, cannot be corrected by the patient and shows an abnormal position of the bone.

2. Location of instability

   Proximal row instability is the most frequent type of instability:

(a) Scapholunate: there is a malalignment between the scaphoid and the lunate. This condition may lead to arthrotic changes.

(b) Triquetrolunate is the result of a complete or partial rupture of the triquetrolunate ligament.

(c) Or both.

Mid-carpal instability:

(a) Ligament attenuation or tear after light trauma in hyperlax persons: this results in a malalignment between the proximal and middle row of carpal bones.

(b) Ligament tear after rotational injury: there is a rupture or elongation of the ligaments that stabilize the mid-carpal joint.

Proximal instability (radiocarpal) with the two Taleisnik stages:

(a) Stage 1: ulnar gliding of the entire carpus

(b) Stage 2: ulnar gliding of the entire carpus with scapholunate separation.

Clinical diagnosis

Although post-traumatic wrist pain occurs frequently, instability does not. The patient is examined thoroughly, and only when the history and the physical findings are compatible with carpal instability should the diagnosis be made.

The patient has a history of past trauma with or without fracture or of past inflammatory disease. There is wrist pain mostly during or after activity, which may be accompanied by a feeling of weakness or of snapping during movement. Information about the type of injury should be obtained but the patient often does not recall such features.[98] The localization of the pain is also very informative and points towards specific possibilities. The passive movements are full or even excessive in range and, although they are performed very gently, they may cause pain at the end of the movement.

Provocative tests can be performed:[99]

1. For scapholunate instability

- The scaphoid bell sign: the examiner grasps the patient's scaphoid bone between thumb and index finger and controls its movement while the patient is actively moving from radial to ulnar deviation. The movement of the scaphoid should be smooth. If this is not the case, the test is considered positive.
- Watson's test:[100,101] the examiner puts the patient's hand in ulnar deviation. With the other hand the scaphoid bone is grasped between thumb (on the tubercle) and index finger. The hand is then brought into radial deviation and resists flexion movement of the scaphoid by the thumb. In instability this results in the proximal pole shifting backwards, which can be felt at the dorsal aspect (Fig. 23.13).
- Lane's test (the scaphoid shift test):[102] the examiner assesses the anteroposterior gliding of the scaphoid.

2. For triquetrolunate instability

- Kleinman's shear test:[103] the examiner grasps the patient's distal forearm and puts the fingers on the most distal and dorsal aspect of the ulna. The thumb is placed on the pisiform bone. The ulna is so stabilized and a posterior pressure exerted on the pisiform. Pain may be produced (Fig. 23.14).
- Linscheid's test:[104] the examiner grasps the patient's distal forearm and puts the finger on the most distal and radial aspect of the radius. The thumb is placed at the ulnar aspect on the triquetral bone (Fig. 23.15).

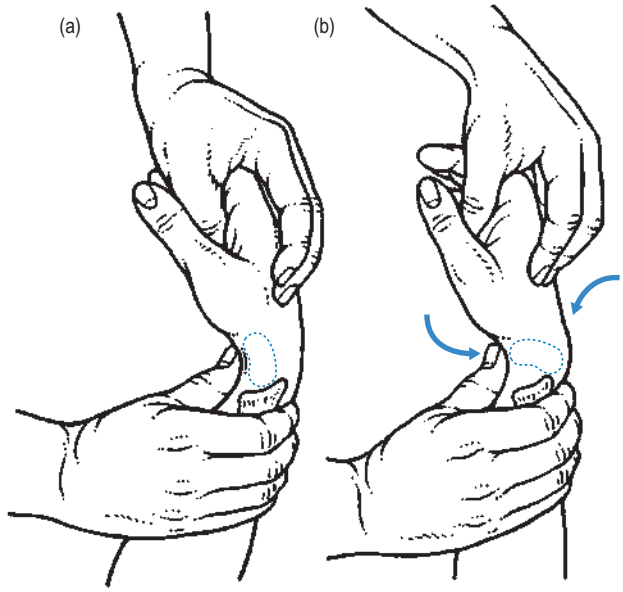


Fig 23.13 • Watson's instability test: pressure is exerted on the palmar aspect of the scaphoid during movement from ulnar to radial deviation.

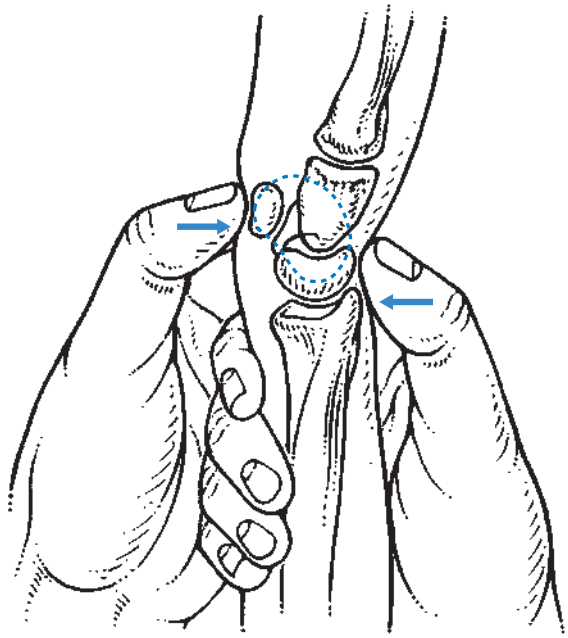


Fig 23.14 • Kleinman's test: the dorsal aspect of the wrist is stabilized and pressure exerted on the pisiform bone in a dorsal direction.

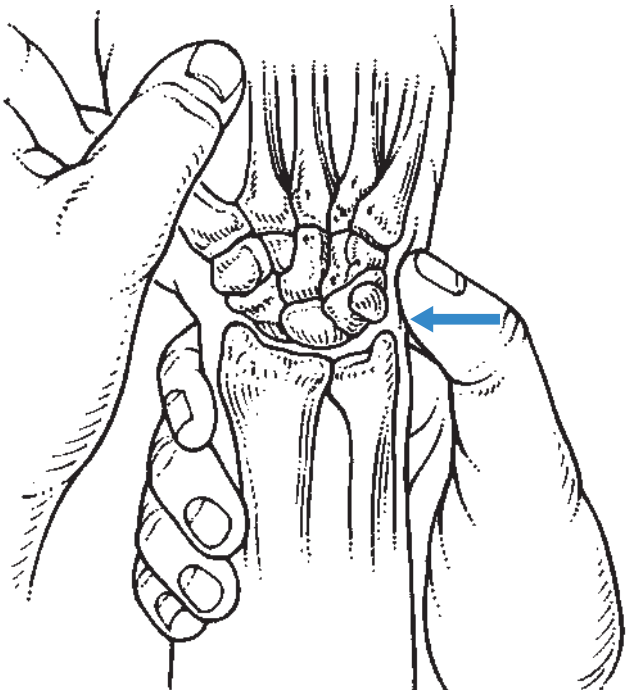


Fig 23.15 • Linscheid's test: the radius is stabilized and pressure is exerted on the triquetral bone in a radial direction.

The radius is stabilized and pressure exerted laterally on the triquetrum. Pain may be produced.

3. For mid-carpal instability
   - Mid-carpal shift test:[105] the examiner exerts axial compression on the wrist via the hand and asks the patient to move the wrist actively. A painful click may be produced. The click will not be present when pressure is brought to bear on the pisiform or during active contraction of the hypothenar muscles.
   - Apprehension test:[106] when the capitate bone is glided in a posterior direction, pain and/or apprehension are produced.
   - Metacarpal gliding:[107] the wrist is placed in ulnar deviation and the metacarpals glided in a palmar direction. Pain may be elicited.
4. For radiocarpal instability
   - Medial radiocarpal drawer test:[108] pressure is exerted on the posterior aspect of the triquetral bone and counterpressure is on the radius. The test is positive when painful.
   - Ulnar sag: the ulnocarpal sag has increased.

Technical investigations may include arthrography, arthroscopy, computed tomography (CT), MRI and scintigraphy.

Treatment

Treatment is still controversial and is very much surgeon-, physician- or therapist-related. Therapy varies from conservative treatment to a range of different surgical techniques. Asymptomatic or slightly symptomatic cases are usually treated conservatively, including immobilization, anti-inflammatory drugs and gradual physiotherapy. Clearly symptomatic, persistent or recurrent cases are often treated surgically. Therapeutic evidence should be looked for in the years to come.

The disorders of the inert structures of the wrist are summarized in Box 23.1.

Box 23.1

Summary of disorders of the inert structures of the wrist

Limited range

Capsular pattern
- Traumatic arthritis
- Rheumatoid arthritis
- Arthrosis

Non-capsular patterns
- Carpal subluxation
- Other joint problems

Full range
- Ligamentous lesions
- Impaction syndromes
- Ganglion

Excessive range
- Ligamentous instability

Disorders of the contractile structures

Tendinitis and tenosynovitis of the wrist are often encountered. Their frequency is the consequence of the long course of the tendons over the wrist, the role played by these tendons in the mobilization and stabilization of the wrist, and overuse of the hand in daily life, as well as in professional and sporting activities.

Resisted extension

Pain

Pain on resisted extension indicates a lesion of either the wrist or the finger extensors. The movement hurts near the wrist, which shows that the distal part of either muscle group is implicated.

Differentiation between wrist and finger extensors can be made by testing extension first with the fingers relaxed and then actively flexed, which inhibits the action of the finger extensors. When an extensor of the wrist is at fault, it is possible to differentiate between the radial extensors and the ulnar extensor by testing resisted radial and ulnar deviation (Fig. 23.16).

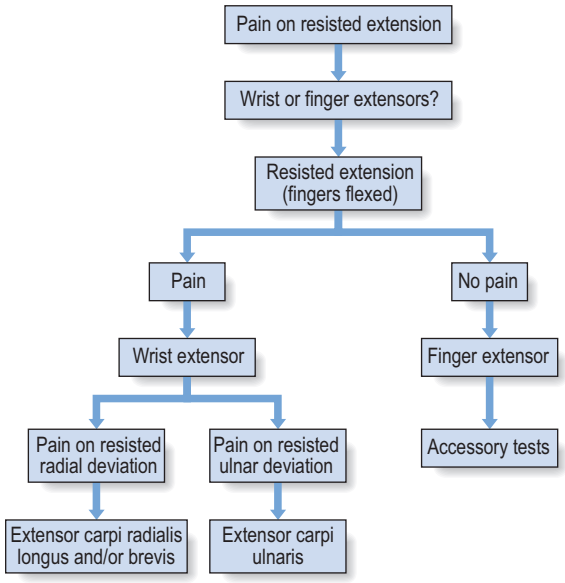


Fig 23.16 • Differentiation between the wrist and finger extensors.

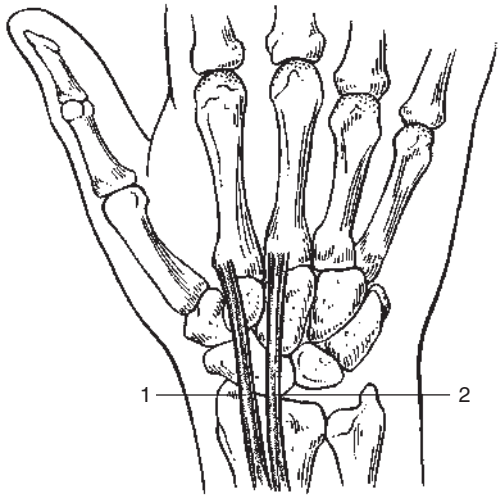


Fig 23.17 • The extensor carpi radialis longus (1) and brevis (2).

Extensor carpi radialis longus and/or brevis

A lesion of the extensor carpi radialis longus and/or brevis (Fig. 23.17) is usually the result of overuse.[109,110] The patient complains of local pain during activity.

Resisted extension and radial deviation of the wrist are painful at the dorsal and radial aspect of the joint. Full passive flexion may also cause pain but is usually negative.

On palpation (in full flexion) two different localizations can be found. Tenderness is elicited at the insertion of the tendons at the base of the second (longus) and/or third (brevis) metacarpal bones. Sometimes the lesion lies in the distal part of the tendon(s), level with the carpus.

Treatment consists of either deep transverse friction three times a week, in which case full recovery should be obtained within 2 weeks, or one or two infiltrations with triamcinolone suspension, provided the lesion is at the tenoperiosteal junction.

For the duration of treatment, the patient should avoid exertion that causes pain. Exercises, passive movements, immobilization and surgery are not indicated.

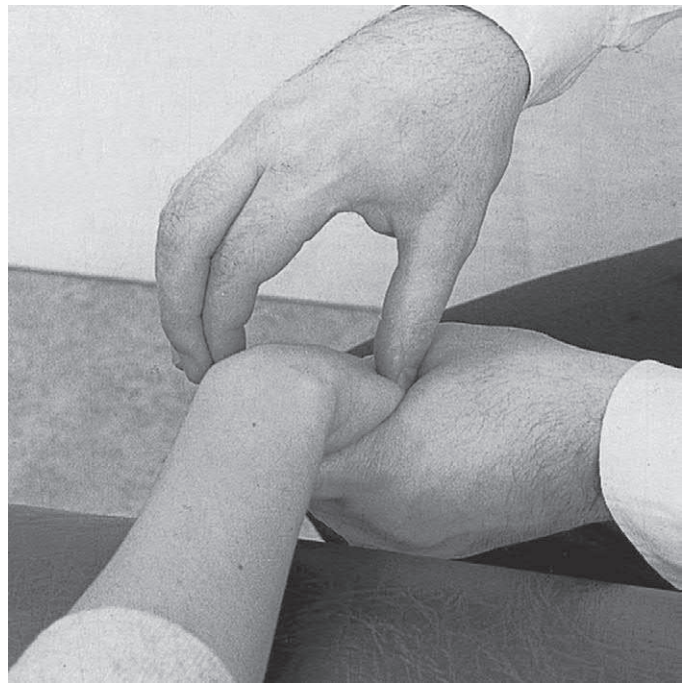


Fig 23.18 • Friction to the extensor carpi radialis longus and brevis.

Technique: deep transverse friction

The patient sits with the arm resting on the couch, the hand lying over its edge. The therapist sits lateral to the patient's hand. To stretch the tendon, the patient's wrist is flexed but this should not provoke pain. The index finger of the other hand, reinforced with the middle finger, is placed on the tender spot. Counterpressure is applied with the thumb at the ulnar aspect of the wrist (Fig. 23.18). The friction starts at the radial side of the tendon and, when the finger has passed over the tendon, the movement restarts. This results in a to-and-fro movement, the active phase being the movement towards the therapist.

Technique: infiltration

This treatment is only applied to insertion tendinitis on the base of the second and/or third metacarpal bones. Ten milligrams of triamcinolone acetonide are infiltrated at the tender area, using a 2 cm needle. The infiltration is given when tendinous resistance is felt and the needle is in contact with the bone. If the palpating finger is kept on the tender spot during the whole procedure (Fig. 23.19), the exact location of the infiltration can be confirmed.

Extensor carpi ulnaris

The tendon is the second most common site of tendinitis in the upper limb.[111,112] A tendinitis of the extensor carpi ulnaris

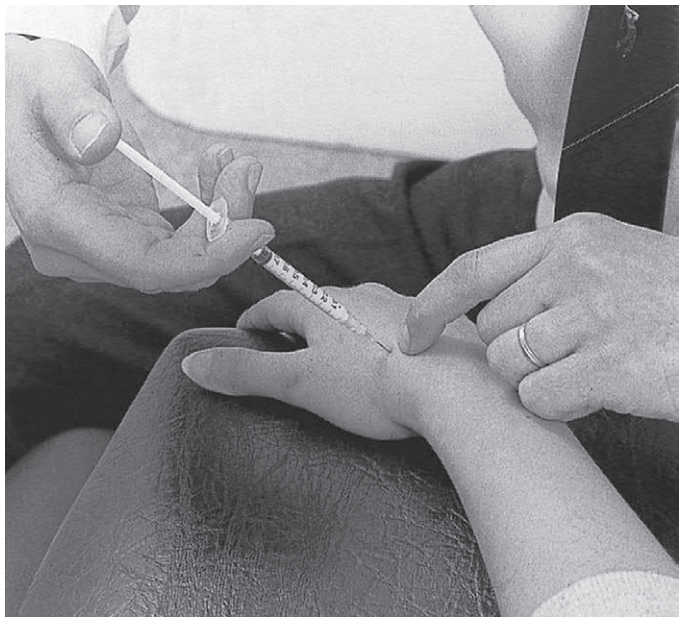


Fig 23.19 • Infiltration of the extensor carpi radialis longus and brevis.

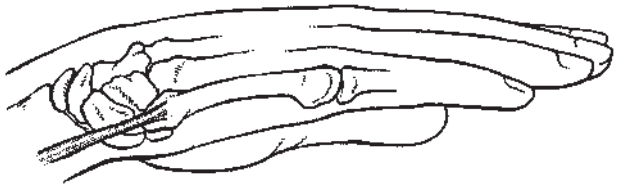


Fig 23.20 • The extensor carpi ulnaris.

(Fig. 23.20) usually develops after a strain or overuse. A tenosynovitis is a rheumatoid manifestation.

On examination, pain at the dorsal and ulnar aspect of the wrist is elicited by testing resisted extension and resisted ulnar deviation. Passive radial deviation may also cause pain at the ulnar side. Palpation is performed with the hand in radial deviation. In rheumatoid tenosynovitis the structure is warm to the touch and swollen or nodular. In an overuse disorder three possible localizations can be palpated. They are (in order of frequency): the tenoperiosteal junction at the base of the fifth metacarpal; the tendon body between the triquetral and the head of the ulna; and the tendon at the groove of the ulna. In the latter, pain is also elicited at the extreme of passive supination. This puzzling localizing sign is explained by Bowers as follows:

> 'The arrangement of the fibrous septa about the ECU [extensor carpi ulnaris] creates an angular approach of the tendon to its insertion in the position of full supination. This angle results in an ulnar translocation stress on the tendon sheath during ECU contraction, particularly with the forearm in supination and the wrist ulnarly deviated.'[113]

Fig 23.21 • Friction to the extensor carpi ulnaris.

In mechanical tendinitis, either 2 weeks of deep transverse friction or one or two infiltrations with triamcinolone suspension will effect a cure. Infiltration is performed at the tenoperiosteal junction or between tendon and sheath. Mobilization, exercises or immobilization do not afford any help. Surgery is seldom necessary. Rheumatoid tenosynovitis is treated by infiltration of a steroid suspension between the tendon and its sheath.

Post-traumatic rupture of the subsheath or of the extensor retinaculum may result in recurrent subluxation of the tendon. The patient feels a sudden snap on certain movements – for example, active supination with the wrist slightly flexed.[114] During the clinical examination, an attempt can be made to reproduce the instability using isometric stress in supination. Treatment is surgical reconstruction.[115]

Technique: deep transverse friction

The patient's pronated arm rests on the couch. The therapist, sitting at the medial aspect of the patient's arm, stretches the tendon by bringing the patient's hand into radial deviation. Depending on the extent of the tenderness, friction is applied with the fingertips of one, two or three fingers, or the index finger is reinforced by the middle finger (Fig. 23.21). Counterpressure is applied to the radial and distal aspect of the forearm. The finger is placed at the palmar aspect of the tendon and then moved upwards over the tendon. Repetition of this movement results in up-and-down friction.

Technique: infiltration at the tenoperiosteal junction

The patient's forearm rests on the couch in the same position as for friction. Ten milligrams of triamcinolone acetonide are infiltrated with a 2 cm needle at the insertion of tendon at the fifth metacarpal bone, while one finger of the other hand ensures that the agent is placed at the correct spot (Fig. 23.22).

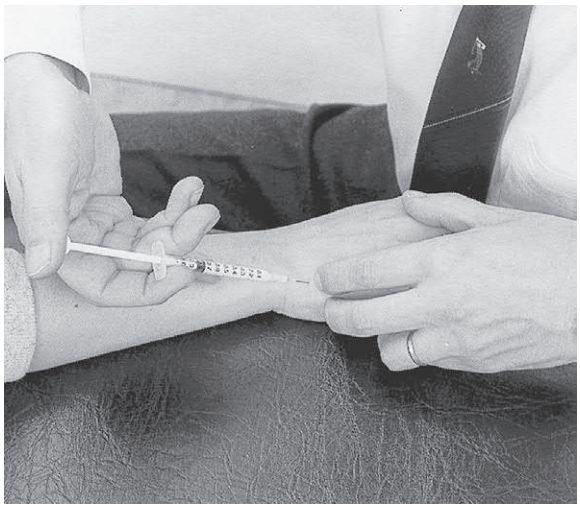


Fig 23.22 • Infiltration of the extensor carpi ulnaris.

The extensor carpi ulnaris and its related structures

This structure is closely connected to the distal radioulnar joint (DRUJ) and to the triangular fibrocartilage complex (TFCC). The distinct subsheath blends proximally with the capsule of the DRUJ and distally with the TFCC, and explains the phenomenon of 'transmitted stress' which is particularly present at this level. Resisted extension and ulnar deviation of the wrist may be positive in lesions of the inert structures (DRUJ and TFCC). The reverse also holds: passive supination may hurt in a lesion of the extensor carpi ulnaris (see p. 345). The phenomenon can be reduced to a minimum by making sure that all examination tests are performed in a starting position which is technically correct (see Ch. 20).

Extensor indicis proprius

Lesions of the extensor indicis proprius are rare and result from overuse.

Pain at the dorsal and radial aspect of the wrist is felt on resisted extension with the fingers relaxed but disappears when the fingers are held actively flexed. The lesion becomes obvious when each finger is separately extended against resistance. Palpation shows the lesion to lie at the carpal extent of the tendon. Crepitus during movement is occasionally felt.

A few sessions of deep transverse friction are curative.

Extensor digitorum

Tenosynovitis, either mechanical or rheumatoid, may occur at the level of the carpus – where the tendons lie in a common sheath – and gives rise to pain felt locally at the dorsum of the wrist. Resisted extension of the fingers provokes this symptom, as does passive flexion, which stretches the tendon. Rheumatoid tenosynovitis is characterized by a discrepancy between (pronounced) symptoms and (moderate) signs. Palpation shows the exact location and, in rheumatoid conditions, reveals swelling as well. Crepitus accompanies mechanical tenosynovitis.

The mechanical type responds to either infiltration with 10 mg of triamcinolone acetonide between the tendon and tendon sheath, or a number of sessions of deep transverse friction. In the rheumatoid type, triamcinolone injection is the only effective treatment.

Weakness

Painless weakness on resisted extension of the wrist should lead to a full neurological examination of the upper limbs. Combination with weakness of other muscle groups helps to determine the causative lesion. Although other conditions may cause painless weakness, those described below are the most common.

Bilateral weakness

If examination shows that only wrist extension is weak, lead poisoning is a possibility. Involvement of motor function in the peripheral nervous system is one of the main features, in addition to those arising from haematopoietic, central nervous, gastrointestinal and renal involvement.[116]

If lead poisoning is not the cause, either carcinoma of the bronchus or one of the general neurological diseases should be suspected.

Unilateral weakness

Unilateral weakness of wrist extension may result from a lesion of either a nerve root or a peripheral nerve. Combination with weakness of other muscles provides more information: the cause lies at the C6–C8 nerve roots or at the radial nerve.

- C6 *root palsy*: in a C6 root palsy, flexion of the elbow is weak as well.
- C7 *root palsy*: weakness of extension of the wrist is exceptional in C7 root palsy but extension of the elbow and flexion of the wrist are normally found to be weak.
- C8 *root palsy*: this is suspected when the extensor carpi ulnaris and flexor carpi ulnaris are weak. Consequently, the hand deviates radially when resisted extension of the wrist is tested. Extension and adduction of the thumb are also weak.
- *Radial palsy*: pressure on the radial nerve in the arm gives rise to radial palsy. The causes may be pressure from a crutch in the axilla, the edge of a chair or, in acute circumstances, fracture of the humerus at mid-shaft (see online chapter *Nerve lesions and entrapment neuropathies of the upper limb*).

Resisted flexion

Pain

The first question that arises is: is there a lesion of either the flexors of the wrist or the flexors of the fingers, and, if a wrist flexor is at fault, is it the radial or the ulnar tendon? Radial and ulnar deviation, tested against resistance, as well as resisted

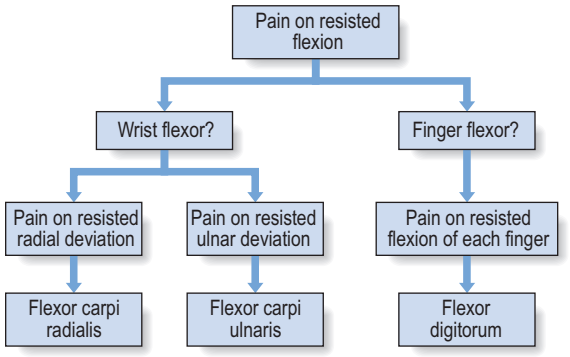


Fig 23.23 • Differentiation between the wrist and finger flexors.

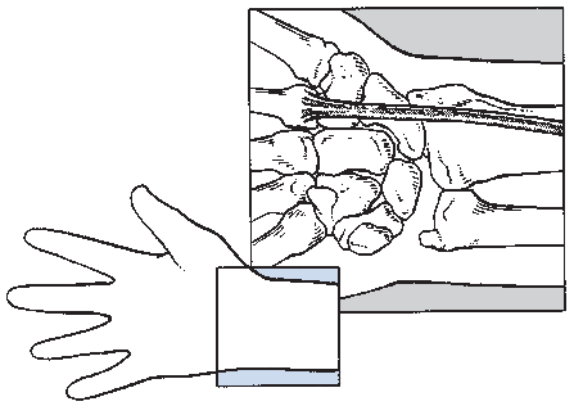


Fig 23.24 • The flexor carpi radialis.

flexion of each finger in turn, should define the affected structure (Fig. 23.23).

Flexor carpi radialis

Lesions of the flexor carpi radialis (Fig. 23.24) are not very common[117] but typically occur in women (75% of cases)[118] and are the outcome of overuse, either recreational or occupational. Pain is felt at the palmar aspect of the wrist during movements such as grasping and lifting with the fingers outstretched.

On examination, resisted flexion and resisted radial deviation of the wrist are painful. Tenderness can be palpated either over the whole distal extent of the tendon or at the insertion of the tendon at the base of the second metacarpal bone.[119]

Tenoperiosteal lesions respond well to both infiltration with triamcinolone acetonide and deep transverse friction, although friction through the thenar musculature is not easy, and is quite uncomfortable and time-consuming. Tendinous lesions are better treated with friction, as the lesion is usually too extensive.

Technique: deep transverse friction

The patient sits with the supinated arm lying on the couch and the hand over its edge. The therapist faces the patient and extends the patient's wrist with the ipsilateral hand. With the fingers of the other hand, friction is imparted to either the

Fig 23.25 • Friction to the flexor carpi radialis.

body of tendon or the insertion on the second metacarpal bone (Fig. 23.25). Friction is started at the ulnar aspect of the tendon and, during the active phase, the fingers are moved to the radial aspect.

Technique: infiltration

The patient sits and places the supinated forearm on a high couch, the wrist slightly extended. A 1 mL syringe is filled with 10 mg triamcinolone acetonide and fitted with a 2 cm needle. The tendon and its insertion at the base of the second metacarpal bone are difficult to identify through the muscles of the thumb. Therefore, the base of the second metacarpal is palpated at the dorsum by the index finger while the thumb presses on the palmar side. The needle is inserted through the thenar muscles and directed towards the palpating index finger (Fig. 23.26). Before it touches bone, the resistance of a tendinous structure is felt. Infiltration is then performed.

Flexor carpi ulnaris

Tendinitis is more common at the ulnar flexor tendon (Fig. 23.27) than at the radial flexor tendon because of the greater mobility at the ulnar side.[120] This lesion usually arises after a single excessive strain.

Usually, the patient can precisely locate the symptoms to the palmar and ulnar aspect of the wrist. Pain is elicited during resisted flexion and resisted ulnar deviations of the wrist. Palpation discloses the exact site of the lesion: distal to the pisiform bone – at the tenoperiosteal junction at the fifth metacarpal bone – or proximal to the pisiform bone in the body of the tendon.[121]

Both deep transverse friction (4–6 treatments) and infiltration with triamcinolone suspension are effective.

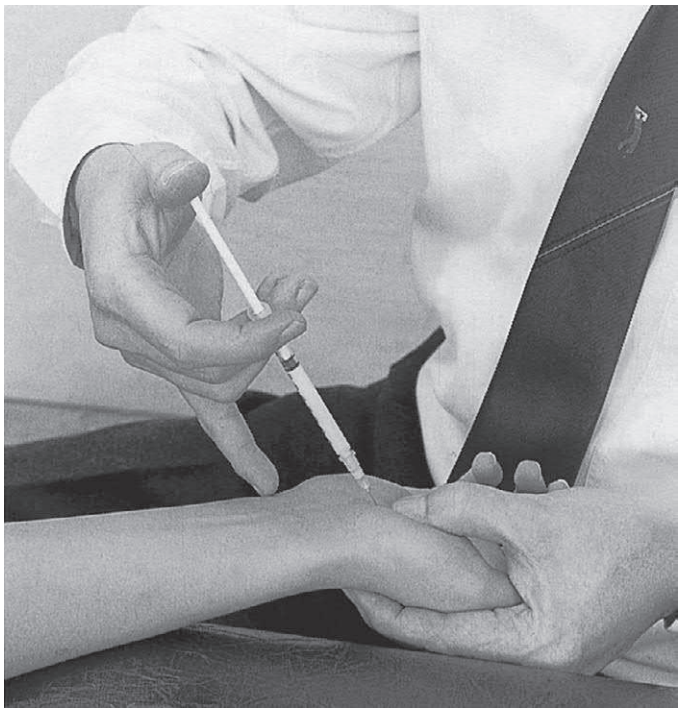


Fig 23.26 • Infiltration of the flexor carpi radialis.

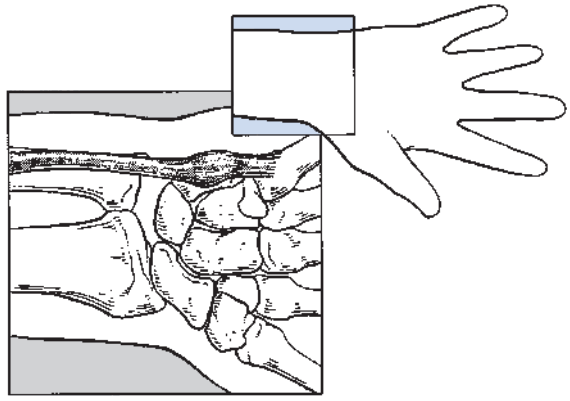


Fig 23.27 • The flexor carpi ulnaris.

Technique: deep transverse friction

The patient sits with the arm held in supination and the hand hanging over the edge of the couch. The therapist, facing the patient, extends the patient's wrist with the contralateral hand and imparts friction with the thumb of the other hand. Counterpressure is given at the dorsal aspect of the patient's wrist (Fig. 23.28). Friction starts at the radial aspect of the tendon and finishes at the ulnar aspect.

Technique: infiltration

This technique is particularly useful for a lesion at the insertion of the tendon onto the fifth metacarpal bone. The patient's hand is brought into the same position as for friction. A 1 mL syringe, filled with 10 mg of triamcinolone acetonide and fitted with a 2 cm needle, is used. The tender area is located by palpation. The operator keeps the thumb on the painful spot during the whole procedure. After the needle is inserted, tendinous resistance should be felt before a series of droplets are deposited in contact with the bone (Fig. 23.29).

Fig 23.28 • Friction to the flexor carpi ulnaris.

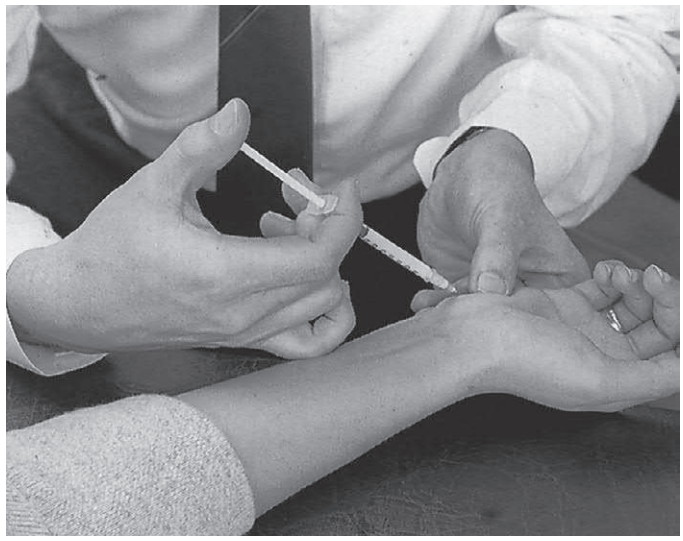


Fig 23.29 • Infiltration of the flexor carpi ulnaris.

Pisotriquetral disorders

Disorders of the joint between the pisiform and the palmar surface of the triquetrum may occur and, because of the attachment of the tendon to the pisiform bone, give rise to the same signs as in flexor carpi ulnaris tendinitis.[122] Disorders at the joint have been described as pisotriquetral arthritis[123] and pisotriquetral instability.[124] Direct pressure on the pisiform elicits pain. Side-to-side passive motion of the pisiform on the triquetrum will also lead to pain and crepitus. If pisotriquetral disorder is suggested, intra-articular steroid is required.[125] In recurrent cases, surgical removal of the pisiform may be necessary.[126,127]

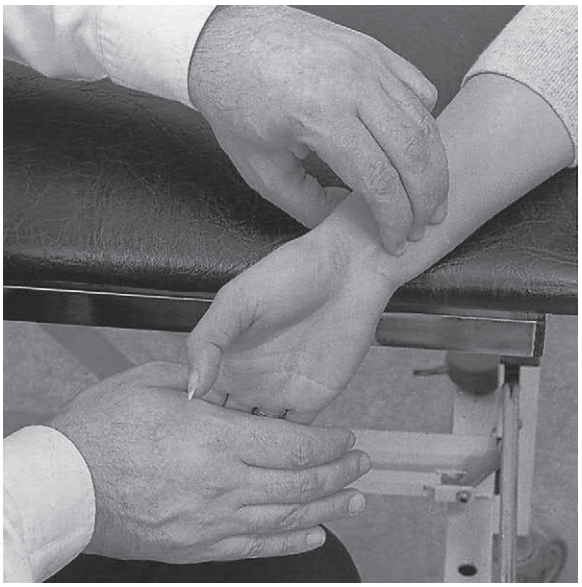


Fig 23.30 • Friction to the flexor digitorum profundus.

Flexor digitorum profundus

A lesion of the flexor digitorum profundus is either a mechanical tenosynovitis or a rheumatoid tenovaginitis. The former results from overuse. The pain is brought on by testing resisted flexion of the fingers. The lesion lies over an area 3–4 cm above the level of the carpus on the palmar aspect of the lower forearm. It is treated by deep transverse friction (see below) or by injection of a steroid suspension between the tendons.

A rheumatoid tenovaginitis is usually confined to one flexor tendon near the carpus. Early in its progress, diffuse swelling and local warmth on the palmar aspect of the forearm are the only signs.[128] Later, tenderness of the tendon may be found over a greater extent than that which occurs in overuse. After a few weeks, the swelling becomes nodular and local warmth persists.

The symptoms abate after one or two infiltrations with triamcinolone.

Swelling on a digital flexor tendon may sometimes lead to compression of the median nerve distal to the carpal tunnel, thus causing a partial syndrome (see the online chapter *Nerve lesions and entrapment neuropathies of the upper limb*).

Technique: deep transverse friction (mechanical tenosynovitis)

The patient sits at the couch with the forearm supinated, the hand hanging over the edge of the couch. The therapist approaches the patient's arm from the radial side. With the ipsilateral hand, the wrist and fingers are brought into extension. The friction is applied with two or three fingertips of the other hand. Counterpressure is maintained with the thumb at the radial aspect of the patient's forearm (Fig. 23.30).

Friction starts at the ulnar side of the tendons and ends at the radial aspect, the therapist pulling the fingers towards him- or herself.

Box 23.2

Summary of disorders of the contractile structures

Pain

Extension

- Extensor carpi radialis longus/brevis
- Extensor carpi ulnaris
- Extensor indicis proprius
- Extensor digitorum

Flexion

- Flexor carpi radialis
- Flexor carpi ulnaris
- Flexor digitorum profundus

Weakness

Extension – bilateral

- Lead poisoning
- Bronchus carcinoma
- Other neurological conditions

Extension – unilateral

- Nerve root: C6–C8
- Radial nerve

Flexion

- Nerve root: C7, C8
- Other neurological conditions

Weakness

Weakness of wrist flexion is frequently the result of a C7 or C8 nerve root lesion, as well as more general neurological conditions:

- C7 *root palsy*: in addition, there is marked weakness of extension of the elbow; the triceps jerk is seldom affected.
- C8 *root palsy*: both the extensor carpi ulnaris and the flexor carpi ulnaris are weak. The result is that the wrist moves radially when resisted flexion is tested. Resisted extension and adduction of the thumb are also weak.

Disorders of the contractile structures are summarized in Box 23.2.

Access the complete reference list online at **www.orthopaedicmedicineonline.com**

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Disorders of the thumb

24

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Disorders affecting the base of the thumb may cause pain over the radial aspect of the wrist. This is why the trapezium–first metacarpal joint and its neighbouring tendinous structures are tested in examination of the wrist.

Disorders of the inert structures

The only relevant passive test for the first carpometacarpal joint is backward movement during extension. Pain and/or limitation indicate a capsular lesion. In the majority of cases, it is the anterior aspect of the capsule that is affected and the joint is found to be tender anteriorly.

The three following conditions are quite common.

Rheumatoid arthritis

Both first carpometacarpal joints are frequently affected in rheumatoid arthritis and respond well to intra-articular triamcinolone. Surgery is seldom necessary but, if so, resection arthroplasty is preferred.[1]

Traumatic arthritis

The patient describes an injury to the thumb, usually overstretching. Discomfort and pain are experienced over the thenar area and the radial side of the wrist. The backward movement during extension elicits pain and is limited. The radiograph is negative.[1]

Spontaneous recovery may take many months. The condition can be treated either by intra-articular triamcinolone or by 2 weeks' treatment with deep transverse friction. Friction is given on alternate days and directed to the anterior and anterolateral capsuloligamentous structures.

Arthrosis

Arthrosis of the trapeziometacarpal joint, or 'rhizarthrosis', is very common. It occurs most frequently in middle-aged or postmenopausal women[2,3] and affects at least 1 in 3 women over 65 and a quarter of men over 75.[4] Rhizarthrosis is often bilateral and is sometimes found in association with arthrosis at the distal interphalangeal joints of the fingers.[5] The aetiology is still very unclear. There is evidence that ligament laxity[6] and trapeziometacarpal subluxation are important early events in the development of thumb arthrosis.[7]

The main symptoms are pain in the dorsoradial and thenar area of the hand and a loss of manual ability and grip strength. In the beginning the pain is cyclic and is felt only during particular activities. As the disease progresses, pain may become constant and may even be present at night. In the final stage, when there is gross joint destruction and subluxation, pain eases but weakness of grip and inability to pinch remain.

Inspection often reveals a dorsoradial prominence of the thumb metacarpal base secondary to subluxation and osteophyte formation. Later on, adduction and Z-deformity of the thumb develop[8]: the first metacarpal is displaced radially and dorsally and the metacarpophalangeal joint is in a hyperextended position. The thenar muscles are atrophic.

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On examination, the combined extension–abduction movement is limited and extremely painful. Crepitus may be felt, especially when the joint is axially compressed and then circumducted – the 'grind' test.[9,10]

The radiographic changes in trapeziometacarpal osteoarthritis are classified into four stages, ranging from mild joint narrowing and subchondral sclerosis to complete joint destruction with cystic changes and bone sclerosis.[11] The final stage shows clear signs of sclerosis of the bone, gross osteophytes and a metacarpal that is displaced radially and dorsally. However, care must be taken to base the diagnosis not only on radiographic evidence, but also on symptoms and the physical examination. Most people with radiographic evidence of degeneration of the trapeziometacarpal joint remain asymptomatic and, when questioned, only 28% will admit to pain.[12]

Treatment

The treatment that is chosen depends on the stage of the arthrosis and the functional disability of the joint (Table 24.1). Classically, conservative treatment of osteoarthritis of the trapeziometacarpal joint includes analgesics, joint protection, strengthening exercises of the intrinsic and extrinsic muscles of the thumb, and splints.[13] Surgical management is recommended to relieve intractable pain.

In early arthrosis, deep transverse friction to the anterior and lateral aspect of the capsule can cause the pain to cease but does not influence mobility.

Later in the course of the condition, intra-articular triamcinolone can be tried. It usually has a temporary result only. An open label trial found that steroids had no benefit on carpometacarpal pain at 26 weeks,[14] while a randomized controlled trial evaluating steroids against placebo injection showed that steroids had no benefit in moderate to severe trapeziometacarpal osteoarthritis at 24 weeks.[15] However, the long-term results of triamcinolone are more effective when a traumatic arthritis has supervened.

During the last few decades, intra-articular injections with hyaluronic acid have been promoted as a valuable alternative to intra-articular injections with steroids.[16] A few open label trials[17,18] found that hyaluronic acid reduced pain and improved grip strength during 6-month follow-up, and two randomized controlled trials stated 'non-inferiority' of hyaluronic acid compared with steroids for pain relief at 26 weeks.[19,20]

Indications for surgical intervention in trapeziometacarpal arthritis are similar to those for arthroplasty of most joints: persistent pain, decreased function, instability, and failure of conservative management.[21,22] Surgical options vary with the stage and nature of the disease. In the early stages, trapeziometacarpal ligament reconstruction and tendon interposition procedures[23–27] have been shown to provide good symptomatic relief. For severe or late-stage disease, some have advocated arthrodesis of the trapeziometacarpal joint, assuming that there is good mobility of the joints proximal and distal.[28–30] Arthroplasty techniques have ranged from simple partial or complete trapeziectomy to various implant and ligament interposition and reconstructions.[31,32] These techniques have been generally indicated for stage II or greater disease once conservative management has failed.

Table 24.1 Summary of treatment of capsular disorders

Deep friction	Intra-articular injection	Surgery
–	Rheumatoid arthritis	Rheumatoid arthritis
Traumatic arthritis	Traumatic arthritis	–
Early arthrosis	Moderate arthrosis	Severe arthrosis

Technique: intra-articular injection

The patient lies supine on a high couch. The physician takes the patient's outstretched hand on to his or her knee, with the thumb uppermost. With one hand the joint is palpated in the anatomical snuffbox. Caution is necessary not to mistake the edge between the metacarpal bone and an osteophyte for the joint line. With the other hand, slight traction (arrow in Fig. 24.1) and ulnar deviation are needed to open the joint. If the patient relaxes properly, the joint line can be identified by a small gap, which is marked with the nail of the palpating thumb.

A 1 mL syringe, filled with 10 mg triamcinolone acetonide, is fitted with a thin needle, 2 cm long. Traction is applied again. The needle enters at the marked line just proximal to the first metacarpal on the extensor surface. Care must be taken to avoid the radial artery and the extensor pollicis tendons. To avoid the radial artery, the needle should enter towards the dorsal (ulnar) side of the extensor pollicis brevis tendon. The needle is then directed at an angle of 60° to the horizontal. At about 1 cm, the tip is felt entering the joint capsule. If it strikes bone at less than 1 cm, the position is not intra-articular and should be adjusted until the tip is felt entering the joint. The injection is then given.[33]

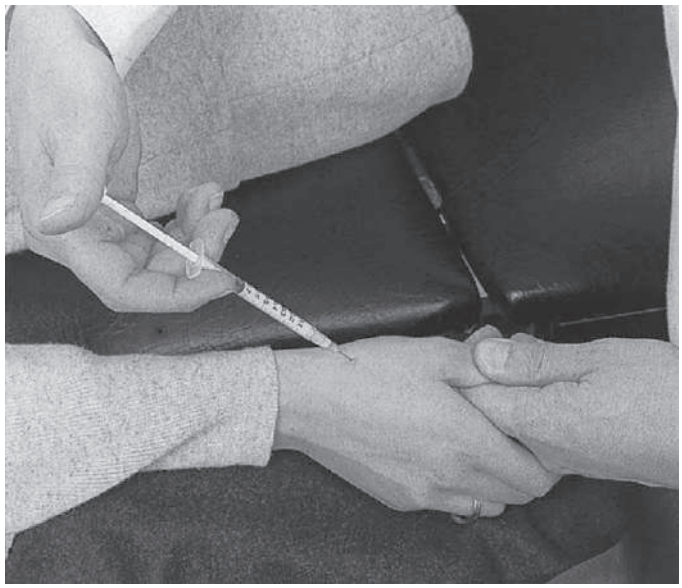
The injection is stopped when considerable resistance is encountered, which may happen after 0.5 mL has entered the joint. The patient should be warned of after-pain, which may last for up to 24 hours. Exertion should be avoided for a few days, and review carried out after 2 weeks. One to two injections should suffice.

Technique: deep transverse friction to the anterior capsule

The patient sits with the supinated arm resting on the couch, the hand hanging over its edge. The therapist extends the patient's wrist and brings the dorsum of the ipsilateral hand in contact with the dorsum of the patient's hand; with the thumb the patient's thumb is directed into extension and a slightly backward position, so as to stretch the anterior capsule. Pain should be avoided.

The joint line between the trapezium and the first metacarpal is identified. It should not be confused with the joint line between the scaphoid and the trapezium, which lies just a centimetre more proximal.

With the thumb of the other hand, friction is performed parallel to the joint line. Counterpressure is taken by the fingertips on the proximal interphalangeal joints of the supporting hand (Fig. 24.2a).



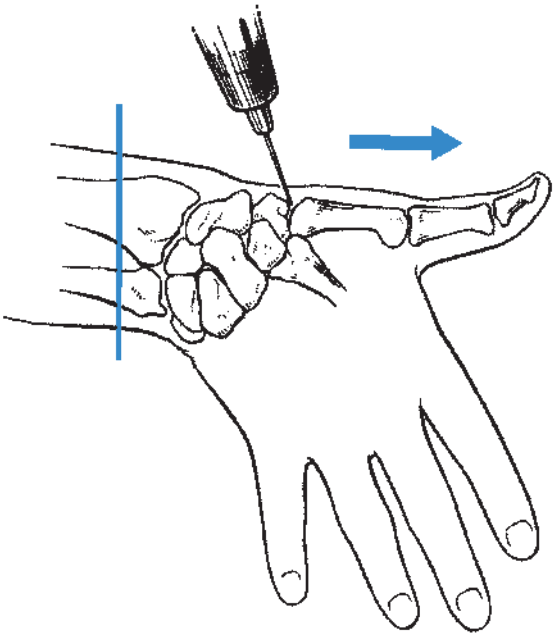


Fig 24.1 • Intra-articular injection.

Technique: deep transverse friction to the anterolateral capsule

The patient's forearm is brought into neutral position. With the contralateral hand, the therapist brings the patient's wrist into ulnar deviation and the thumb into flexion, which stretches the capsule.

Friction is imparted at the joint line and parallel to it with the thumb of the other hand. Counterpressure is given with the fingers on the ulnar aspect of the patient's hand (Fig. 24.2b). It is important to ensure that the thumb remains palmar to the extensor pollicis brevis tendon, in that the lesion lies at the anterolateral aspect of the joint.

Friction is given 2–3 times a week for about 2–3 weeks. The results are good in traumatic arthritis and early arthrosis.

Disorders of the contractile structures

Pain

Resisted extension

This movement seldom causes pain in isolation. Because the extensor brevis and abductor longus tendons lie together in one tendon sheath, in the majority cases resisted abduction is also painful.

Abductor pollicis longus, and extensor pollicis brevis (first tendon sheath)

Intersection syndrome

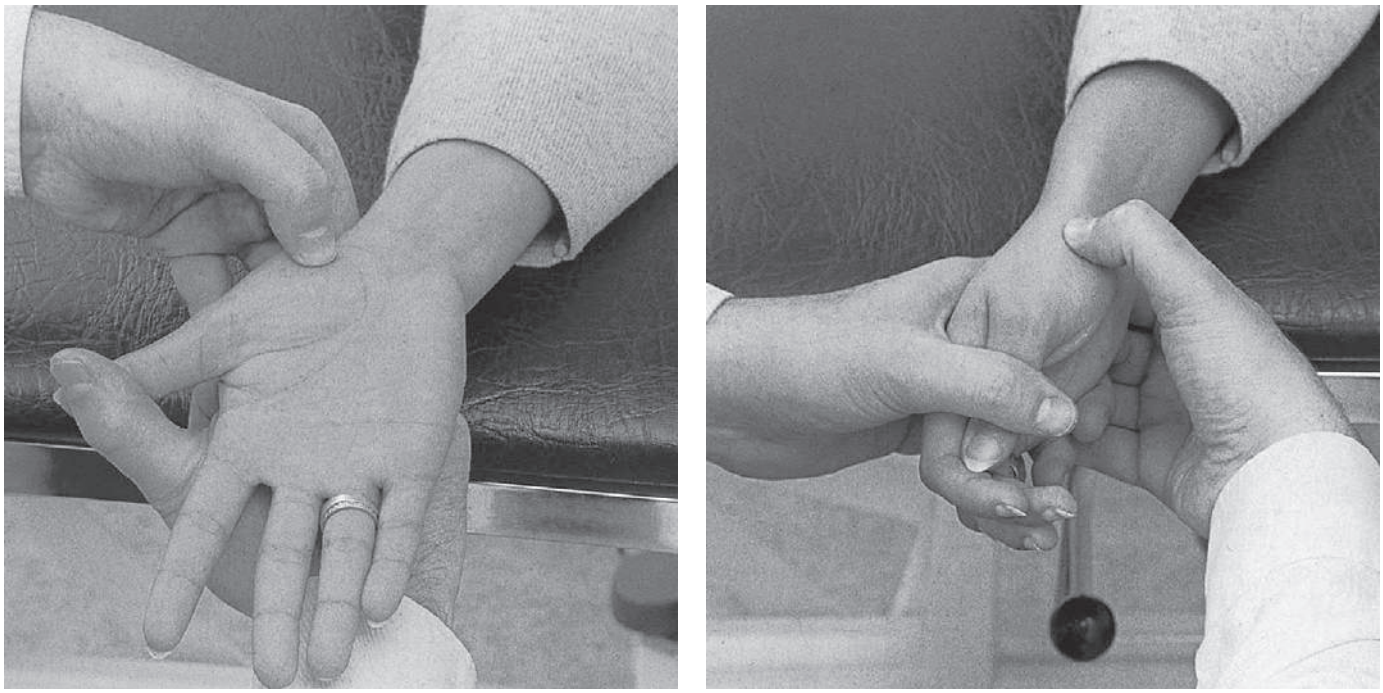
Intersection syndrome is a specific painful disorder of the forearm that is relatively common but sometimes not correctly diagnosed clinically. It has also been referred to in the literature by the terms 'peritendinitis crepitans', 'oarsmen's wrist', 'crossover syndrome', 'subcutaneous perimyositis', 'squeaker's wrist', 'bugaboo forearm' or 'abductor pollicis longus syndrome'.[34] Dobyns *et al* introduced the term 'intersection syndrome', an anatomical designation related to the area in which the musculotendinous junctions of the first extensor compartment tendons (abductor pollicis longus and extensor pollicis brevis tendons) intersect the second extensor compartment tendons (extensor carpi radialis longus and extensor carpi radialis brevis tendons), at an angle of approximately 60° (Fig. 24.3).[35]

The most plausible pathophysiology is that of a peritendinitis at the intersection of the two tendinous compartments, which spreads upwards to the musculotendinous junction. The lesion may also lie somewhat more proximally in the muscle bellies; hence the name, 'myosynovitis'.[36] This is shown by magnetic resonance imaging (MRI) findings demonstrating the presence of peritendinous oedema concentrically surrounding the second and the first extensor compartments, beginning at the point of crossover, 4–8 cm proximal to the Lister tubercle and extending proximally.[37]

The lesion always results from occupational overuse or after unusual effort. It is often associated with sports-related activities, such as rowing,[38] canoeing, playing raquet sports, horse riding and skiing.[39,40]

The patient mentions crepitus during wrist movements. On examination, resisted extension and resisted abduction of the thumb are painful. Some passive wrist movements can also be painful. This puzzling phenomenon can be explained by the fact that discomfort is increased not only by movements that stretch the tendons (e.g. passive flexion of the thumb and ulnar deviation of the wrist), but also by every movement that pushes the tendon into the tendon sheath (e.g. thumb extension, radial deviation and flexion or extension of the wrist).

On palpation, tenderness and swelling are found in a region about 4–8 cm proximal to the Lister tubercle, where the first and second extensor compartment tendons cross. In cases of



(a) (b)

Fig 24.2 • Friction to the anterior (a) and anterolateral capsule (b).

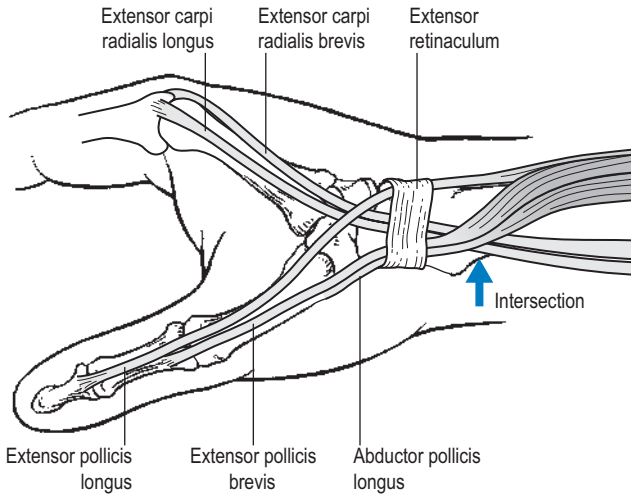


Fig 24.3 • Intersection syndrome: the lesions lies at the intersection of compartments one and two, between 4 and 8 cm proximal of Lister's tubercle.

recent onset, a silky crepitus during active wrist movements can be palpated.

The differential diagnosis is made from suppurative inflammation of the tendon sheaths, de Quervain's tenosynovitis (see below), early chronic evolutive polyarthritis, arthrosis of the trapezium–first metacarpal joint of the thumb, lesions of the radial collateral ligament, and tendinosis of the extensor carpi radialis longus and/or brevis.

After an initial stage involving a great deal of pain and disability, the condition may evolve towards a more chronic state. Spontaneous cure may take many months and only occurs when the patient gives the wrist complete rest. Treatment with non-steroidal anti-inflammatory drugs, immobilization and infiltration of steroid or local anaesthetic does not always lead to swift, full and permanent recovery. Deep transverse friction, however (three times a week, over 2 weeks), is extremely successful in this condition, to such a degree that all other treatments must be considered obsolete. This view was confirmed by Paton in 1978,[41] who had used deep transverse friction since 1947 without failure. Bisschop describes 62 cases – 48 men and 14 women – that he treated between 1975 and 1982. The onset was recent (less than 6 weeks) in 55 cases and chronic (2.6 months on average) in 7. Forty-eight of the patients received other treatments, with poor results: steroid infiltration in 16 cases; local anaesthetic infiltration in 4; plaster immobilization in 13 (average 1.8 weeks); partial immobilization with tape in 2; ice friction in 4; physiotherapy in 9. Deep transverse friction – performed three times a week for 15 minutes – led to complete recovery in all but one case, with an average of 6.7 treatments, and 39 patients improved from the first treatment onwards.[42]

Technique: deep transverse friction

The patient's forearm rests in pronation on the couch, the hand over its edge. With the contralateral hand the therapist brings the patient's wrist and thumb into flexion (Fig. 24.4). This stretches the tendons. The other hand takes hold of the patient's wrist; the thumb then lies flat on the site of the lesion.

Movement starts with the thumb at the ulnar side of the tendons. By supination of the arm, the thumb passes over the tendons to the radial aspect. Repeated movement results in a to-and-fro movement.

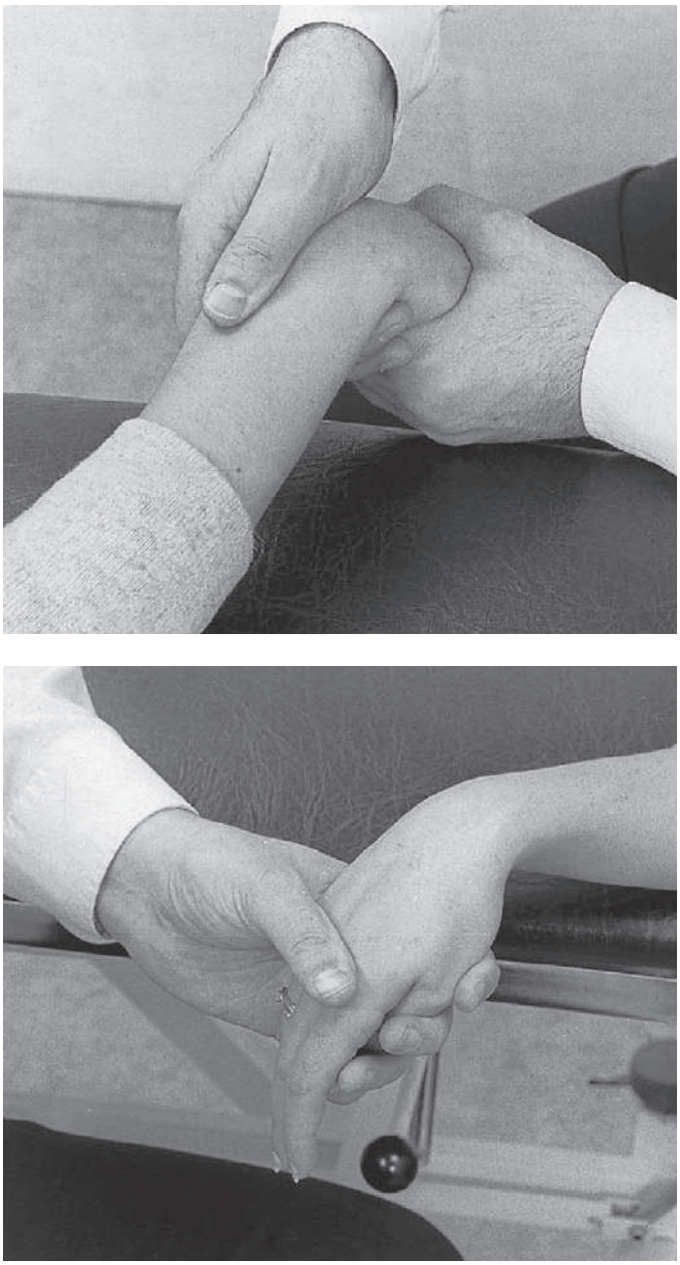


Fig 24.4 • (a) Friction to the abductor pollicis longus, and extensor pollicis longus and brevis; (b) detail of hand fixation.

Tenovaginitis of the first compartment

This is a lesion of the common tendon sheath of the abductor pollicis longus and extensor pollicis brevis tendons at the wrist. Two conditions can be distinguished – mechanical and rheumatoid – that require the same treatment.

Mechanical tenovaginitis

Fritz de Quervain, a Swiss physician, is given credit for first describing this condition in a report of five cases in 1895.[43] The disorder has since been known as de Quervain's disease, tenovaginitis stenosans or styloiditis radii.[44] Although the term 'stenosing tenosynovitis' is frequently used, the pathophysiology of de Quervain's disease does not involve inflammation. On histopathological examination, de Quervain's disease is not characterized by inflammation, but by thickening of the tendon sheath and most notably by the accumulation of mucopolysaccharide, an indicator of myxoid degeneration.[45] Therefore de Quervain's disease should be seen as a result of intrinsic, degenerative mechanisms rather than extrinsic, inflammatory ones. The term 'styloiditis radii' is also a misnomer as the lesion is not bony or periosteal.

Incidence of de Quervain's disease has risen considerably in recent decades.[46] It occurs mostly in women, with an average age of 47, and almost never appears before the age of 30.[47] A significant association was noted in patients with de Quervain's disease after pregnancy.[48] The cause is presumed to be endocrine in origin and similar to the carpal tunnel syndrome described during pregnancy and the lactating postpartum period.[49]

Very often de Quervain's disease comes on spontaneously, but it can also result from overuse. Swift repeated movement with exertion of considerable strength is a possible cause.[50]

Symptoms consist of pain and/or tenderness at the radial styloid, sometimes radiating down to the thumb and up the lower forearm. Often there is localized swelling over the distal part of the radius. The patient finds the symptoms very disabling, preventing the hand from being used properly. Triggering may complicate the more severe forms[51] and demonstrates a more recalcitrant course when treated non-operatively.[52]

On examination, resisted extension and abduction of the thumb are painful. Passive movements of wrist and thumb also cause pain, as they slide the tendon up and down within its irritated sheath, so setting up painful friction. Very occasionally, resisted radial deviation of the wrist also hurts because the thumb tendons assist this movement. Finkelstein's test (deviating the wrist to the ulnar side, while the patient makes a fist with the thumb inside the fingers) reproduces the symptoms.[53] Some consider this pathognomonic.[54,55] Because the lesion is a tenovaginitis, crepitus is always absent but localized swelling can often be palpated.

Palpation must be performed over a wide area, as there are three possible localizations for the lesion[56]: at the tenoperiosteal insertion of the abductor pollicis longus into the base of the first metacarpal bone; at the level of the carpus; and at the groove on the lower extremity of the radius (Fig. 24.5).

It is important not to be misled by the localization of tenderness. Very often, the styloid process of the radius is more tender than the tendon itself, which has led to the lesion being described as styloiditis radii. This is a misnomer, since it gives the impression that the lesion is bony and lies at the styloid process. That this is not so can be shown during clinical examination and the progress of treatment: pain increases when resisted movements of the thumb are tested and disappears as the result of treatment directed to the tendons. Styloiditis radii does not exist and is another example of the phenomenon that Cyriax called 'associated tenderness'.

Spontaneous recovery may take 3–4 years. However, treatment is quite simple. The lesion responds remarkably well to one or only occasionally two injections with triamcinolone suspension,[57] provided that the injection is correctly placed between tendon and tendon sheath. The effectiveness of injection therapy is often attributed to the anti-inflammatory effects of corticosteroids but the exact mechanism of action remains unclear. Reviews of the effectiveness of corticosteroid injection

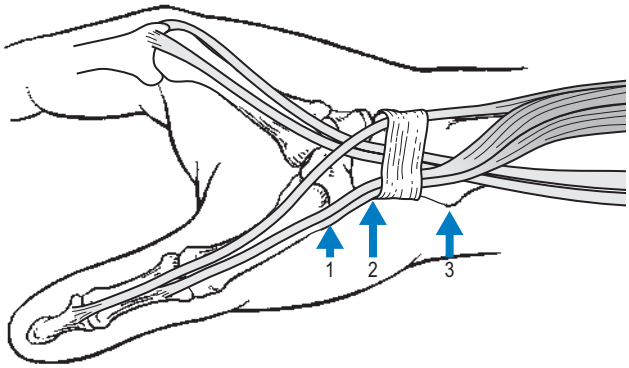


Fig 24.5 • Three possible localizations for mechanical tenovaginitis in the first compartment: 1, insertion of abductor pollicis longus; 2, carpus; 3, distal radius.

for de Quervain's disease showed success rates between 78 and 89%.[58–61] In a controlled, prospective, double-blind study by Zingas *et al*, the accuracy of infiltration in the first extensor compartment was defined and correlated with clinical relief of the disorder. The authors concluded that only those patients in whom the steroid solution really entered the tendon sheath were cured.[62] Failure to inject may therefore be caused by misplacement of the needle or by anatomical variations of the tendon sheaths. Anatomical[63] and ultrasonographic[64] studies confirm the observations during surgery[65] that the first extensor compartment may contain a septum separating the extensor pollicis brevis tendon from the abductor pollicis longus. During a study of variants of the tendon of the abductor pollicis longus in 110 upper extremities, Melling *et al* found an intertendinous septum in 30%.[66] This was recently confirmed by Gousheh.[67] Leslie *et al* found that most wrists for which injection fails have a separate extensor pollicis brevis compartment at surgical release. They recommend a second, more dorsal injection in patients when the first has failed.[68]

Other treatments, such as deep transverse friction, ointments and immobilization, should be regarded as obsolete. Decompressive surgery is only used in those rare cases that do not respond to conservative treatment.[69] Simple decompression of both tendons and partial resection of the extensor ligament give excellent long-term results.[70] For those patients who have a septum in the first extensor compartment, Yuasa and Kiyoshige suggest decompressing only the extensor pollicis brevis subcompartment.[71]

Rheumatoid tenosynovitis

In this condition, there is a striking discrepancy between symptoms and signs. Contrary to a mechanical disorder, which shows little swelling and gross disability, rheumatoid tenosynovitis presents with marked thickening of the tendon sheath but not a great deal of pain.

The condition responds well to one or two injections with triamcinolone suspension. There is no tendency to recurrence.

Technique: injection

The patient lies supine on a high couch, the forearm halfway between pronation and supination and supported on the operator's thigh. The wrist is held in ulnar deviation and the thumb slightly flexed. The tendons are easily identified when the patient is asked to extend and abduct the thumb actively. The border between the two tendons is identified and marked. The common sheath containing the extensor pollicis brevis and the abductor pollicis longus tendons is pinched between thumb and index finger.

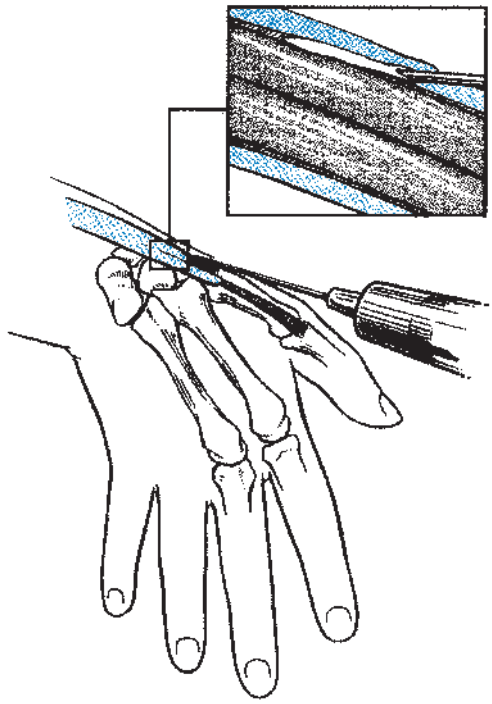


Fig 24.6 • Infiltration of the abductor pollicis longus and extensor pollicis brevis.

A 1 mL syringe is filled with 10 mg triamcinolone acetonide and fitted with the thinnest possible needle. A point is chosen just proximal to the base of the first metacarpal bone and the needle is thrust in almost horizontally between the two tendons so that the tip comes to lie in the common tendon sheath. When the fluid is forced in, the palpating fingers feel a small, sausage-shaped swelling along the course of the tendons (Fig. 24.6); 0.5–1 mL of fluid can be injected before counterpressure is experienced on the barrel of the syringe.

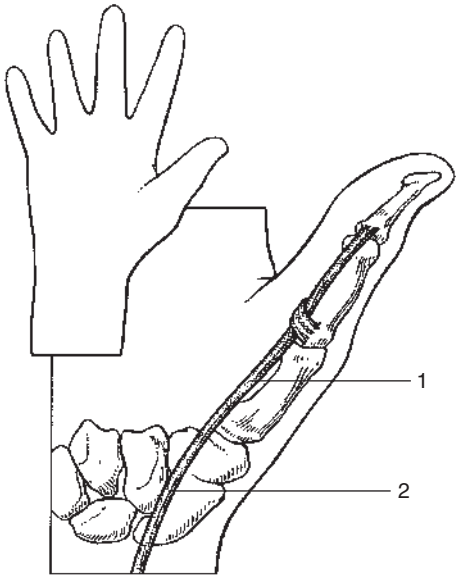


Fig 24.7 • The flexor pollicis longus: 1, level of the first metacarpal; 2, level of the carpus.

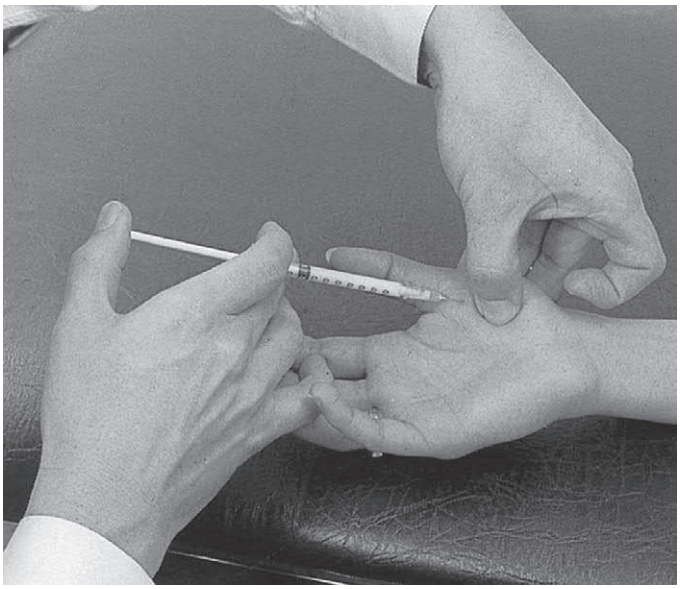


Fig 24.8 • Infiltration of the flexor pollicis longus.

Because of the risk of lipodystrophy from subcutaneous injection, care must be taken that fluid does not flow back into the subcutaneous tissue when the needle is withdrawn.

Extensor pollicis longus

Pain on resisted extension of the thumb is rare and is the consequence of a lesion of the tendon of the extensor pollicis at its carpal extent. Overuse ('drummer boy palsy'), forced wrist extension, direct trauma or distal fractures of the radius may all precipitate an extensor pollicis longus tendinitis.[72] Pain is felt over the dorsal aspect of the wrist and can be reproduced by testing resisted extension of the thumb.

Treatment consists of deep transverse friction and the condition takes about 2 weeks to cure.

Resisted flexion

Flexor pollicis longus

Tenosynovitis of the flexor pollicis longus may present at two different sites which can be differentiated by palpation: at the level of the first metacarpal (Fig. 24.7), where it responds well to an infiltration of triamcinolone but not to deep transverse friction; and at the level of the carpus, where crepitus may be present. Deep transverse friction is then effective.

Technique: infiltration

The patient sits with the hand resting on the couch. When the patient is asked to flex the thumb against resistance, the tendon can be palpated and the lesion thus precisely located. The tender spot along the first metacarpal shaft is identified. A syringe filled with 10 mg of triamcinolone suspension fitted with the thinnest possible 2 cm needle is used. The needle is inserted at the level of the tenderness and drug is infiltrated using several withdrawals and reinsertions along the inflamed tendon (Fig. 24.8).

Fig 24.9 • Friction to the flexor pollicis longus.

Technique: friction

The patient sits with the hand on the couch. The therapist sits in front of the patient's hand. With the contralateral hand, the wrist and thumb are brought into extension. With the thumb of the other hand, the tender spot is identified, which is deep in between the tendons of flexor carpi radialis and palmaris longus, level with the carpus. The tendon can be felt when the patient is asked to perform flexion and extension of the thumb. Counterpressure is applied with the fingers at the dorsum of the patient's wrist (Fig. 24.9). Friction is started at the radial side of the tendon and ended at the ulnar side. The movement

is performed by a pronation–supination movement of the forearm.

Trigger thumb

A tendinous node frequently forms on the flexor pollicis longus and becomes engaged in the tendon sheath. The mechanism is as follows: the nodule catches the proximal end of the fibrous sheath at the carpometacarpal pulley with flexion of the thumb, causing the symptoms of trigger thumb with extension. Initially, patients experience intermittent pain, swelling and triggering of the involved digit. In the most severe state, the digit becomes locked in the flexed position, after which the patient has to extend the joint passively with the help of the other hand. A snap and pain may accompany this.[73] This typical history suggests the diagnosis, because resisted movements of the thumb do not elicit pain. The tender node can be palpated, just proximal to the head of the first metacarpal bone, and is felt to move when the thumb is flexed and extended.

Spontaneous cure may occur but takes several months.[74] One infiltration with triamcinolone is usually curative.[75] When the condition recurs, simple surgery to split the tendon sheath longitudinally will give lasting relief from symptoms.[76]

Technique: infiltration

The patient lies supine on the couch and the operator sits adjacent. The patient's hand is placed on the operator's thigh, palm upwards. A tuberculin syringe is filled with 1 mL of a 10 mg/mL triamcinolone solution and attached to a thin needle, 2 cm long. With one finger the tender node, which lies just proximal to the metacarpophalangeal joint, is identified. The needle is inserted about 1 cm distally and directed toward the palpating finger (Fig. 24.10). Half of the solution is infiltrated around, the rest into the nodule.

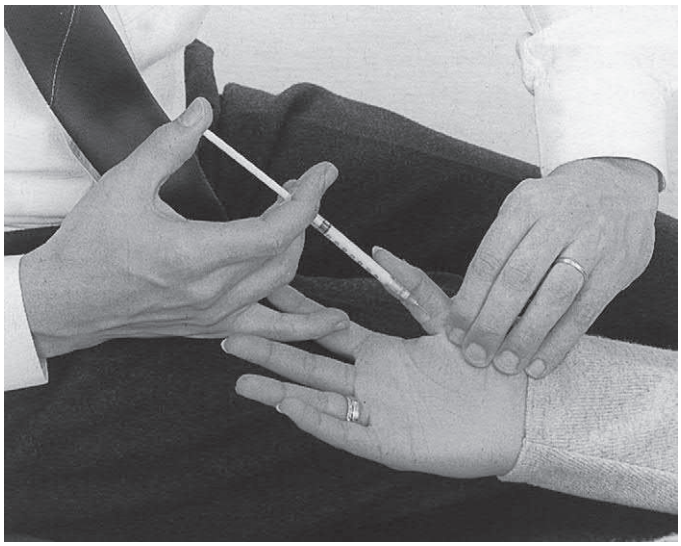


Fig 24.10 • Infiltration of the flexor pollicis longus in trigger thumb.

The patient should be warned that there may be considerable after-pain. The result is evaluated after 2 weeks; a second infiltration is needed only rarely.

Weakness

Weakness of one or several muscles of the thumb may follow either rupture of a tendon or a nerve lesion.

Rupture

A tendinous rupture is easily detected by clinical examination.

Rupture of the extensor pollicis longus tendon occurs rarely after a fracture at the distal extremity of the radius (Colles' fracture); it usually happens some weeks later as the result of the tendon being frayed by a bony callus.[77,78] It is also seen in patients with advanced rheumatoid arthritis.

Surgical repair is the appropriate treatment and, in acute cases, this should be done with some urgency to prevent a second rupture, which often follows.[79,80]

Nerve lesions

Nerve lesions causing weakness of the thumb may lie at different levels (Table 24.2).

- *C8 nerve root*: compression of the C8 nerve root, usually caused by a C7 disc protrusion, leads to weakness of adduction and extension of the thumb. This is accompanied by weakened ulnar deviation of the wrist.
- *Brachial plexus*: when the lower trunk of the brachial plexus becomes affected at the thoracic outlet – for example, as the result of compression by a cervical rib – there may be marked wasting of the abductor pollicis brevis, which does not necessarily cause weakness of abduction of the thumb. In advanced cases, weakness of the muscles innervated by the median and ulnar nerves may be detected.

Table 24.2 Nerve lesions and weakness of the thumb

Weakness	Muscle	Nerve
Extension	Extensor pollicis longus Extensor pollicis brevis	Radial Radial
Flexion	Flexor pollicis longus Flexor pollicis brevis	Anterior interosseous Ulnar/(median)
Abduction	Abductor pollicis longus Abductor pollicis brevis	Posterior interosseous Median
Adduction	Adductor pollicis	Ulnar
Opposition	Opponens pollicis	Median

Box 24.1

Summary of disorders of the thumb

Disorders of the joint capsule

- Rheumatoid arthritis
- Traumatic arthritis
- Arthrosis

Disorders of the contractile structures

Pain

Resisted extension

- Abductor pollicis longus
- Extensor pollicis longus/brevis
- Abductor pollicis longus
- Extensor pollicis brevis
- Extensor pollicis longus

Resisted flexion

- Flexor pollicis longus
- Trigger thumb

Weakness

- Rupture
- Nerve lesions

- *Median nerve*: long-standing compression of the median nerve in the carpal tunnel may lead to slight weakness and wasting of the thenar muscles.
- *Posterior interosseous nerve*: a lesion of the posterior interosseous nerve at the elbow results in weakness of thumb abduction and finger extension (see online chapter *Nerve lesions and entrapment neuropathies of the upper limb*).
- *Ulnar nerve*: entrapment of the ulnar nerve in the hand gives rise to weakness of thumb adduction.

Disorders of the thumb are summarized in Box 24.1.

Access the complete reference list online at **www.orthopaedicmedicineonline.com**

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Clinical examination of the thoracic spine

25

CHAPTER CONTENTS

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Introduction

Thoracic or abdominal wall pain is a common complaint and poses a major diagnostic challenge to physician and therapist.

The pain is often referred from visceral disorders, although the frequency of musculoskeletal lesions of the thorax and the abdomen should not be underestimated. A physician not familiar with the musculoskeletal disorders of the region could be tempted to ascribe unexplained pain to vague lesions such as intercostal neuralgia, neuritis, cardiac neurosis, pleurodynia or rib syndromes. Again, lack of an exact diagnosis leads to inadequate and unsuccessful treatment. The absence of a precise (orthopaedic) diagnosis can, to a certain extent, be a consequence of the complexity of the region itself. However, another important reason is the lack of an appropriate clinical approach to this part of the body. Thorough examination should not be restricted to the routine visceral examination (e.g. auscultation, percussion and palpation) but also must include proper orthopaedic and neurological tests. Although a large number of reliable technical investigations for detecting all types of visceral disorders are available, the same cannot be said when it comes to musculoskeletal disorders, for which technical investigations are often of limited diagnostic value.

Clinically, the thoracic region is approached in a different way from the cervical or the lumbar spine because it behaves differently in many aspects.

Principal differences between the thoracic and the lumbar and cervical spines

Visceral versus musculoskeletal pain

Because referred pain from visceral problems can mimic pain of musculoskeletal origin and vice versa, the first step towards diagnosis must always be to differentiate these two categories. The character of the pain is usually of little help in the differential diagnosis because it has the same features in both. Pain referred from heart, lungs and intestines is usually poorly localized and vaguely delineated, and is referred to a segmental or multisegmental distribution. The behaviour of the pain may also mislead the examiner. One of the main characteristics of pain in lesions of moving parts is that it is brought on by posture and movement. This is also the case in thoracic lesions: if the patient's symptoms depend on activity rather than on visceral function, a cause originating from moving parts should be considered. However, it is important to keep in mind the

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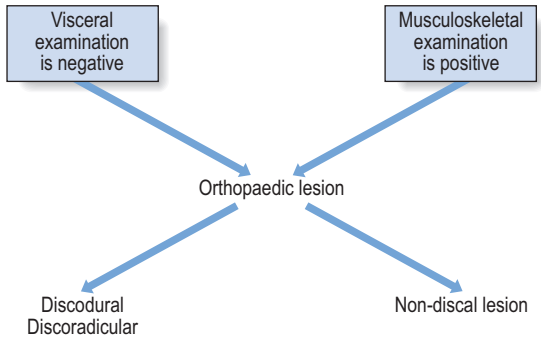


Fig 25.1 • Routine for differentiation between thoracic and abdominal wall pain.

fact that posture, physical activity, a deep breath or a cough may also influence visceral pain in the thorax or abdomen.

The best method of differentiating is to work in two complementary ways: exclusion of any visceral disorder through a thorough internal check-up, together with positive confirmation of a provisional orthopaedic diagnosis (Fig. 25.1). This routine will also safeguard against unnecessary technical investigations and delays in diagnosis and treatment.[1–3]

Discal lesions

Discodural and discoradicular interactions are well-known causes of cervical and lumbar pain.

In a discodural lesion, a shifted component of the disc impinges on the dura and causes pain that has multisegmental characteristics (crossing the midline and occupying several dermatomes). Discodural conflicts are characterized by two sets of symptoms and signs: articular and dural (see Ch. 33).

In a discoradicular lesion, the subluxated disc component impinges on the nerve root and its dural sleeve. The pain and paraesthesia that result are strictly segmental. Discoradicular conflicts are characterized by three sets of symptoms and signs: articular, root and cord.

Disc lesions also commonly occur in the thoracic spine but often show characteristics that are quite different from those found at the lumbar and cervical spines.

- *The articular signs are subtle*: a discodural interaction at the cervical or lumbar level usually presents with a clear partial articular pattern: some movements hurt or are limited and others do not, always in an asymmetrical way. This is not so in the thoracic spine. Because of the rigidity of the thorax, such an obvious pattern is seldom found. Very often, only one of the six passive movements, usually a rotation, is positive and then only slightly so. Therefore diagnosis in the thoracic spine is more tentative and may have to be based on smaller, subtler abnormalities.
- *Neurological deficit is seldom encountered in a thoracic disc lesion*: whereas some degree of neurological deficit is a common finding in cervical or lumbar posterolateral disc lesions, muscular weakness is rarely detectable in thoracic discoradicular lesions. Also, disturbance of sensation is very rare. This absence of neurological signs is probably the outcome of the location of the nerve root in the intervertebral foramen, where it lies mainly behind the lower aspect of the vertebral body and less behind the disc (see online chapter *Applied anatomy of the thorax and abdomen*).
- *There is no tendency to spontaneous recovery*: in the lumbar and cervical spines there is usually a spontaneous cure for root pain, which seldom lasts longer than 4 months at the cervical level and 12 months at the lumbar level. At the thoracic level, no such tendency exists and constant root pain can persist for many years.
- *Protrusions can usually be reduced*: although thoracic disc lesions are more difficult to diagnose, they are easily and effectively cured. Protrusions – no matter how long they have been present, or whether they are posterocentral or posterolateral, or soft or hard – can usually be reduced by 1–3 sessions of manipulations. Unlike at the lumbar or cervical levels, time is not a criterion for reducibility. Hence a disc displacement may well prove reducible after constant root pain, even of several years' standing. Traction is seldom required because the protrusions are usually of the annular type.

Non-discal lesions

In the thorax, non-discal musculoskeletal lesions are also encountered frequently. To name but a few: ribs, rib joints, cartilage, intercostal and abdominal muscles. This is in contrast to the cervical and lumbar spines, where lesions of the disc are the principal cause of dysfunction and pain.

Referred pain

Both musculoskeletal and visceral lesions can be the source of pain referred to the thoracic and/or abdominal wall.

Pain referred from musculoskeletal structures

Dura mater and nerve roots

Pain originating from the dura mater is referred in a multisegmental way: it crosses the midline and may cover several consecutive dermatomes (see p. 18). A possible explanation for this phenomenon may lie in its multisegmental origin, which is reflected in the considerable overlap between the fibres of the consecutive sinuvertebral nerves innervating its anterior aspect.[4] Recent research has demonstrated that dural pain may spread over eight segments with considerable overlap between adjacent and contralateral dura mater.[5] This may be an explanation for the fact that lower cervical discodural conflicts may produce pain that spreads into the upper thoracic level (Fig. 25.2a) or that lumbar dural pain causes pain in the lower thoracic region (Fig. 25.2b).

Pain originating from a nerve root sleeve has a strict segmental reference and is restricted to the borders of the dermatome.

Thoracic disc lesions may thus cause referred pain in the thorax, not only as the result of extrasegmental reference in

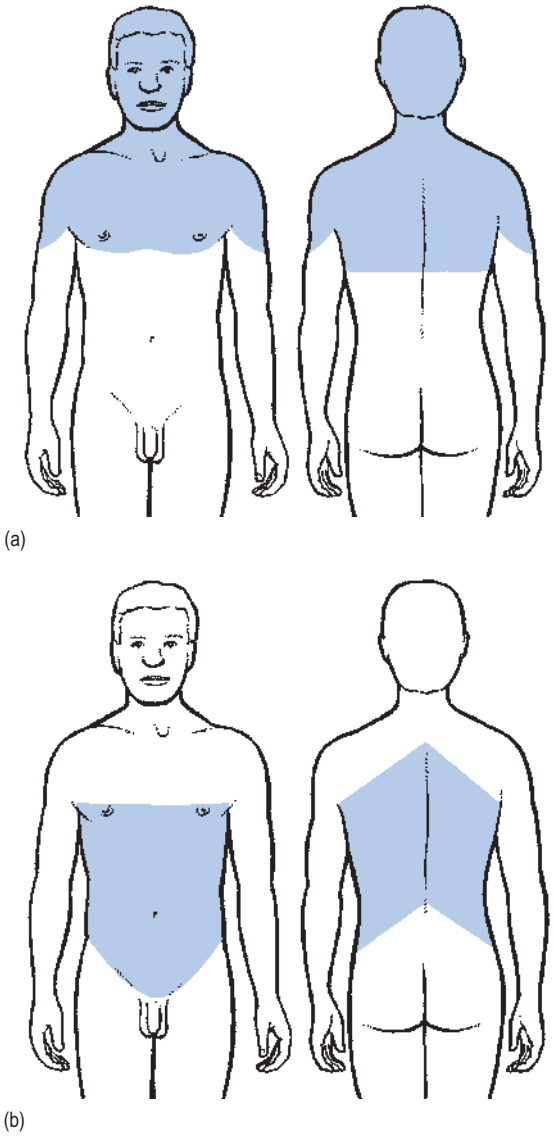


Fig 25.2 • Extrasegmental pain: (a) cervical origin; (b) thoracic origin.

the case of discodural contact, but also when a discoradicular interaction has been created. However, cervical and lumbar discal lesions may also be the origin of thoracic referred pain.

Cervical disc lesions

Some cervical disc lesions may cause pain in the thoracic region.

Cervical discodural interactions

Low cervical discodural interactions very often result in unilateral interscapular pain, usually felt above T6 and spread over several dermatomes. The pain is also often referred to the sternum and the precordial region. Exceptionally, the pain may be felt only in the anterior chest, so misleading almost all clinicians. It is important to remember that extrasegmentally referred pain of cervical origin never spreads into the upper limb (see p. 18).

Cervical discoradicular interactions

A posterolateral disc protrusion compressing the C5, C6, C7 or C8 nerve root gives rise to unilateral root pain characterized mainly by a sharp pain down the upper limb. There may also be some degree of scapular pain, especially in C7 lesions, but this is usually not very severe (see p. 156).

The C4 nerve root gives rise to pain in the trapezius area, the infraclavicular chest and the scapular region above the scapular spine.

Thoracic disc lesions

Thoracic discodural and discoradicular interactions are common causes of referred pain in the thoracic and abdominal region.

Thoracic discodural interactions

It is important to note that extrasegmental pain from a postero-central thoracic disc protrusion usually remains in the trunk itself, where it can spread anteriorly and/or posteriorly over several segments (see Fig. 25.2b). It seldom spreads into the neck or into the buttocks. The pain is usually unilateral and spreads over several segments. Exceptionally it is felt centrally at the spine, radiating bilaterally towards the sides.

Thoracic discoradicular interactions

A posterolateral impingement on the two upper thoracic roots produces pain felt in the arm (Fig. 25.3). If the T1 nerve root is involved, pain may be referred to the ulnar side of the forearm, whereas a T2 nerve root compression gives rise to pain felt over the inner aspect of the arm from the elbow to the axilla, at the anterior aspect of the upper thorax around the clavicle and at the posterior upper thorax around the scapular spine. Clinically the upper two thoracic segments belong to the cervical spine and are thus most easily examined with the cervical segments.

If the 3rd–12th root is compressed, pain spreads unilaterally as a band around the thorax, sometimes reaching anteriorly as far as the sternum (see Fig. 25.3).

The following landmarks may be helpful in determining which root is involved:

- If pain is felt around the nipple, the T5 nerve root is at fault.
- Because the epigastrium belongs to the T7 and T8 segments, pain present here arises from structures of the same origin.
- Pain at the umbilicus and in the iliac fossa may point to a lesion of the T9, T10 and T11 nerve roots.
- If the T11 or T12 thoracic root is compressed, pain may be referred to the groin or even further down to the testicle.

Lumbar disc lesions

Lumbar discodural interactions

Extrasegmentally referred pain from a lumbar discodural interaction is usually felt in the lumbar area and the abdomen, sometimes radiating into the lower limbs. The pain seldom spreads into the lower thorax.

Lumbar discoradicular interactions

Segmentally referred pain from L1, L2 and L3 nerve roots may be felt in the side and in the groin.

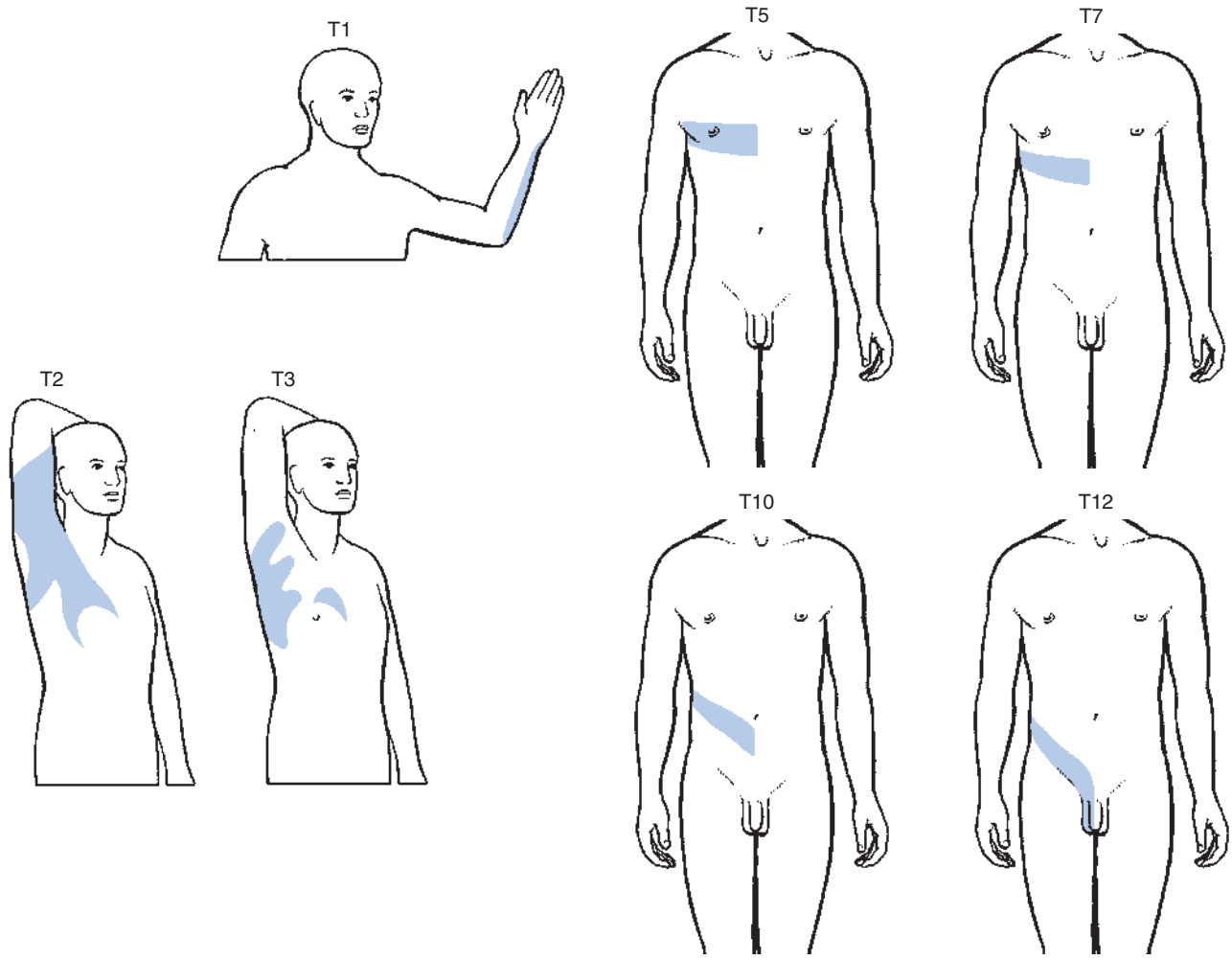


Fig 25.3 • Segmental referred pain of thoracic origin.

Nerves

Neuritis

Neuritis of the spinal accessory nerve, the long thoracic nerve and the suprascapular nerve can provoke unilateral pain at the base of the neck and over the scapula.

Herpes zoster

In that the thorax is often the seat of a herpes zoster infection, unilateral spontaneous pain of recent onset always calls for a careful inspection of the skin for erythema and grouped vesicles.

Bones

Osseous structures usually do not give rise to much in the way of referred pain; the pain remains typically local. Intense though localized pain is a warning sign and the following conditions should be considered:

- *Traumatic fracture of a vertebral body*: severe central pain is to be expected for about 2–6 weeks. For the first week there is often girdle pain. Thereafter it gradually disappears. If uncomplicated, spontaneous cure is to be expected within 12 weeks.
- *Vertebral tumours*: all types of bony tumour of the vertebrae, primary or metastatic, as well as infections, initially provoke local pain in the centre of the back.
- *Contusion or fracture of a rib*: the patient can point exactly to the tender spot. The same is the case in malignant invasion.
- *Disorders of the sternum*: traumatic disorders and tumours of the sternum may also give rise to local sternal pain.

Joints and ligaments

Ligaments and joints consistently obey the rules of referred pain, which means that the deeper the location of the affected structure and the closer its position to the midline, the more referred pain is to be expected. On the other hand, the further the lesion lies from the spinal axis and the more superficial it is, the more accurate will be its localization by the pain it provokes.

- *Manubriosternal and sternoclavicular joints*: as these are superficially located, the pain is felt locally.
- *Costochondral and chondrosternal joints* (Tietze's syndrome and costochondritis): the patient is able to indicate the site of the lesion accurately.

- *Intervertebral facet joints*: these give rise to unilateral paravertebral pain, felt deeply and locally, but not going further lateral than the medial edge of the scapula. If several joints are affected at the same time, as may be the case in ankylosing spondylitis, the pain spreads more in a craniocaudal direction than mediolaterally; the opposite is true for a disc protrusion.
- *Costovertebral and costotransverse joints*: the pain is felt unilaterally between the vertebral column and the scapula.
- *Anterior longitudinal ligament*: when this ligament is affected, pain is usually located anteriorly behind the sternum.
- *Posterior longitudinal ligament*: involvement of this ligament causes pain in the back, felt centrally between the scapulae.
- *Disorders of the costocoracoid fascia or the trapezoid and conoid ligaments* (see online chapter *Disorders of the inert structures*): the pain is usually felt in the infraclavicular fossa.

Muscular lesions

Muscular sprains of the intercostal muscles, the abdominals and the muscles of the shoulder girdle usually provoke well-localized pain at the site of the lesion.

Pain referred from visceral structures

The heart

Pain arising from disorders of the heart can be referred to dermatomes C8–T4 because the heart is derived largely from these segments. Therefore pain can radiate towards the tip of the shoulder, to the anterior chest and to the corresponding region of the back. It may also be referred towards the ulnar side of both upper limbs, though referral to the left side is more common (Fig. 25.4a). Pain from the pericardium always arises from the parietal surface because this is the only part which has a sensory innervation.

The aorta

Pain from the aorta, as in dissecting aneurysm, may be felt behind the sternum or in the abdomen, depending on the exact level of the disorder. It often radiates into the back and may expand as the lesion progresses.

The lungs

The lungs are insensitive. Therefore pain is caused either by the parietal pleura, as in pleurisy, or by invasion of the chest wall by a tumour.

The parietal pleura

Pleuritic pain is caused by an inflammation of the parietal pleura (pleurisy). Though the visceral pleura does not contain any nociceptors, the parietal pleura is innervated by somatic nerves that sense pain when the parietal pleura is inflamed. Parietal pleurae of the outer rib cage and lateral aspect of each hemidiaphragm are innervated by intercostal nerves. Pain is localized to the cutaneous distribution of those nerves. The phrenic nerve supplies innervations to the central part of each hemidiaphragm which is a C4 structure, with pain reference to the trapezius area.[6]

The oesophagus

Disorders of the oesophagus (T4–T6) usually give rise to pain felt at any part of the sternum, often radiating between the scapulae into the back (Fig. 25.4b).

The diaphragm

The central part of the diaphragm is mainly derived from the third, fourth and sometimes, although rarely, the fifth cervical segments. Pain from irritation of the central part of the diaphragm is felt at the tip of the shoulders and the base of the neck. Pain from the peripheral part is felt more locally in the lower thorax and in the upper abdomen at the costal margin (Fig. 25.4c).

The stomach and duodenum

Pain from stomach and duodenum (T6–T10) is most commonly felt in the epigastrium and upper abdomen, sometimes substernally and exceptionally in the lower thoracic part of the back (Fig. 25.4d).

The liver, gallbladder and bile ducts

The liver is derived from the right side of T7–T9. The gallbladder and bile ducts are of right T6–T10 origin. Pain is felt in the right hypochondrium and may radiate towards the inferior angle of the right scapula (T7–T9) (Fig. 25.4e).

The pancreas

Patients suffering from a pancreatic disorder complain of upper abdominal pain often referred to the back at T8 (Fig. 25.4f).

The spleen

Pain arising from disorders of the spleen is usually felt in the left hypochondrium, sometimes in the left side at low thoracic level (T7–T10).

The small intestine, appendix and colon

Problems in the small intestine (T9–T10) give rise to pain felt around the umbilicus. In disorders of the colon (T10–S5), pain is usually felt in the neighbourhood of the lesion. The appendix is a T10–L1 structure.

The kidneys and ureters

Disorders of kidney and ureters (T10–L1) give rise to pain felt posteriorly in the side, at and just below the lower ribs, and at the anterolateral aspect of the abdomen. The pain often radiates towards the testicles or the labia (Fig. 25.4g).[7]

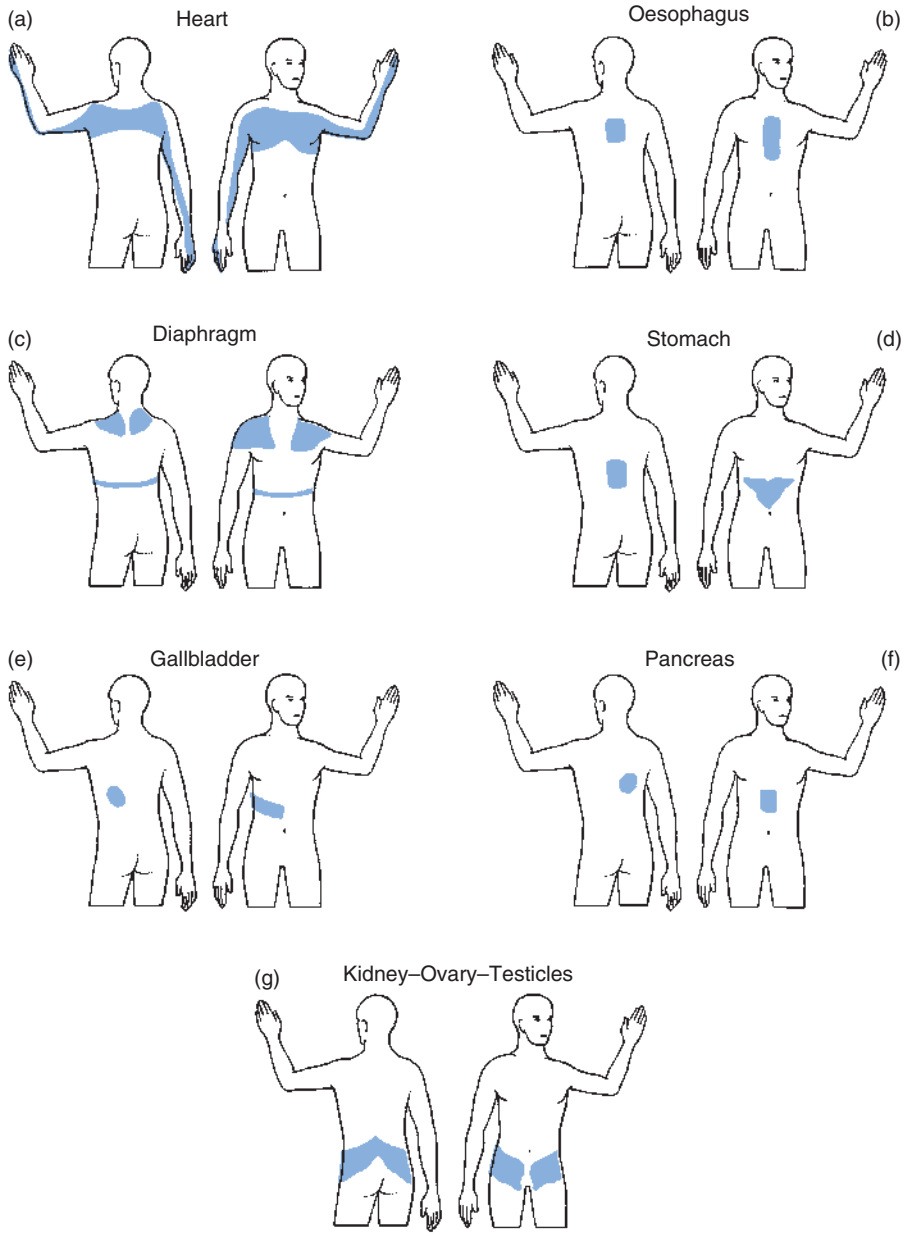


Fig 25.4 • Pain referred from visceral structures: (a) heart; (b) oesophagus; (c) diaphragm; (d) stomach; (e) gallbladder; (f) pancreas; (g) kidney/ovary/testicles.

Reproductive system

Disorders of the ovaries (T11–L1) may result in unilateral low abdominal pain, sometimes felt in the periumbilical area. Testicular problems (T11–L1) give rise to scrotal pain, sometimes radiating into the groin and to the side.

See Box 25.1 for a summary of referred pain in the thorax and abdomen.

History

In dealing with pain in the thorax or abdomen, the main goal of history taking is to differentiate between musculoskeletal and visceral problems. As already pointed out, a relationship between the patient's symptoms and positions or activities does not necessarily exclude a visceral disorder, because in several visceral disorders the same relationship exists.

The history is kept simple and clear. Detailed information is elicited about pain, paraesthesia and the intake of anticoagulants or the presence of bleeding disorders.

Pain

What made the pain come on?

If the patient mentions an injury to the chest, a bony problem of the ribs, sternum or vertebra is likely. In a pathological fracture sudden pain may follow a very trivial movement.

Box 25.1

Summary of referred pain in the thorax and abdomen

Pain of musculoskeletal origin

Dura mater and nerve roots

- Cervical disc lesions
- Thoracic disc lesions
- Lumbar disc lesions

Nerves

- Neuritis
  - Spinal accessory nerve
  - Long thoracic nerve
  - Suprascapular nerve
- Herpes zoster

Bones

- Vertebrae
  - Fracture
  - Bony tumours
  - Infections
- Ribs
  - Contusion and fracture
  - Malignant invasion of a rib
- Sternum
  - Fracture
  - Tumours

Joints and ligaments

- Manubriosternal and sternoclavicular joints
- Costochondral and chondrosternal joints
- Intervertebral facet joints
- Costovertebral and costotransverse joints
- Anterior and posterior longitudinal ligaments
- Costocoracoid and coracoclavicular ligaments

Muscular lesions

- Intercostals
- Pectoralis major
- Pectoralis minor
- Subclavius
- Latissimus dorsi
- Serratus posterior inferior
- Abdominals

Pain of a visceral nature

- Heart
- Aorta
- Pleura and lungs
- Oesophagus
- Diaphragm
- Stomach and duodenum
- Gallbladder and bile ducts
- Pancreas
- Spleen
- Small intestine and colon
- Kidney and ureters
- Genitals

When the pain has come on without trauma, it is of interest to know what the patient was doing at the time and in which position the body was held. Thoracic disc lesions are, just like lumbar ones, most often the result of a combined flexion–rotation movement. However, in disc lesions a history of such provocation cannot always be obtained, the patient stating that the pain started without any specific activity or posture.

Pain that came on after a forceful movement of trunk or arms – such as during sporting activities – may be from a muscular lesion.

Arthritis of the costovertebral, costotransverse or facet joints begins spontaneously. When it is the result of ankylosing spondylitis, pain and stiffness often occur in phases and are usually worst in the early morning.

Warning

Pain of spontaneous onset, increasing in intensity and constantly expanding, should be suspected of being caused by a tumour.

Where was the pain at the beginning, where did it spread or shift to, and where is it now?

When the pain is felt between the scapulae above T6, a cervical disc problem is most likely. In such cases, clinical examination of the cervical spine should be done initially. If this is negative, clinical examination of the thorax follows.

A thoracic disc lesion usually gives rise to discomfort felt centrally or unilaterally in the posterior thorax. Here, as in the cervical spine, the pain may be felt centrally at first and then shift more to the side. A shifting pain suggests a shifting (disc) lesion.

A posterocentral protrusion interfering with the dura normally results in unilateral extrasegmental referred pain, usually felt in the anterior chest. However, it can also give rise to abdominal pain, discomfort in the groin and even lumbar pain. Sometimes bilateral extrasegmental pain is felt posteriorly and over several segments on both sides.

An acute 'thoracic' lumbago may exceptionally cause pain felt only at the sternum. This is a most misleading phenomenon, as it is not very logical to think of a disc problem in a patient with an acute sternal ache not preceded by any posterior thoracic pain. The possibility of such reference should always be borne in mind.

In posterolateral disc protrusions, pain is felt unilaterally and is referred to one segment only. It is mainly felt posteriorly and at the side but sometimes also anteriorly. This type of protrusion usually follows a posterocentral displacement that has subsequently shifted more laterally. In this case, root pain is preceded by a period of extrasegmental referred dural pain. In the rare event of a primary posterolateral protrusion, the disc fragment moves directly in a posterolateral direction. From the onset, pain is felt to the side, radiating segmentally anteriorly and not preceded by extrasegmental pain.

Pain which increases and expands all the time is from a 'growing' disorder, usually a tumour. Older patients who are symptom-free on first waking in the morning but complain of

central posterior thoracic pain that starts after some hours and increases throughout the day, are likely to be suffering from posterior bulging of the whole content of an intervertebral disc as a consequence of excessive thoracic kyphosis.

Pain felt at the base of the neck is sometimes the result of a problem at the first costovertebral joint, the sternoclavicular joint or a fracture of the first rib.

Pain at the sternum is seldom the result of a musculoskeletal disorder. Most often it has a visceral origin. The same is true for pain felt in the abdomen: muscular lesions do exist but are rare. They usually give rise to well-localized pain.

Is the pain influenced by coughing, sneezing or deep inspiration?

When a Valsalva manœuvre or a deep breath provokes or increases the cervical or lumbar pain, it is generally interpreted as a dural symptom, from interference with the dura mater by a protruded disc or a tumour. In the thoracic area, it is not so simple. Many disorders other than problems of the dura may give rise to the same symptom. Deep inspiration may increase pain because of one of a number of visceral disorders or other musculoskeletal problems. Tumours of the respiratory tract, pleurisy, lung embolism, pneumothorax and even pericarditis can all give rise to pain increased by coughing or respiration. The same may be found in non-discal musculoskeletal problems of the ribs and sternum. Fractures and contusions of the ribs, a sprained intercostal muscle and a fracture of the sternum are all in the same class. Consequently, the influence of respiration on symptoms at the thoracic level is only regarded as a dural symptom once there is clinical certainty of disc protrusion. In thoracic disc protrusions it is more usual for deep inspiration, rather than a cough, to exacerbate the symptoms.

Paraesthesia

Protrusion of a disc in the thoracic region can produce pins and needles in two ways. First, it may be the result of compression of the spinal cord, which is characterized by extrasegmental pins and needles felt in both feet and typically increased or provoked by flexion of the neck. Cord compression and paraesthesia can also be caused by intra- and extraspinal tumours, intraspinal haemorrhage or a vertebral fracture. Second, paraesthesia can be stem from posterolateral protrusion compressing a nerve root. For example, pins and needles may be felt in the groin from compression of the T12 nerve root. In this event, the features are always localized and limited to the corresponding dermatome.

Both causes are rare, and when paraesthesia is present, other conditions, such as neuropathy due to generalized disorders (diabetes, pernicious anaemia and multiple sclerosis), must always been considered first.

Anticoagulant treatment and bleeding disorders

The use of anticoagulants is always an absolute contraindication to manipulation of the spine, because it can lead to uncontrollable intraspinal bleeding. The same applies in congenital or acquired disorders of blood coagulation.

Inspection and palpation

On inspection, the curvature of the thoracic spine is noted. Scoliosis and hyperkyphosis may be detected.

Scoliosis

A deformity in the frontal plane is named scoliosis. If present, it is important to determine whether it is located at the lumbar or thoracic level. Thoracic scoliosis can often be voluntarily corrected by the patient. In this event, it is postural and has no specific pathological implications. In structural scoliosis, the deformity cannot be corrected by muscular activity. There is often associated shoulder asymmetry too. In this event, the scapula at the convex side is usually more prominent and the arm of this side touches the hip, but the other arm hangs further away from the body.

Kyphosis

A hyperkyphotic thoracic spine with a flattened lumbar spine may suggest ankylosing spondylitis. An excessive low thoracic and high lumbar kyphosis can be the result of Scheuermann's disease or osteoporosis.

Sometimes a localized angular kyphosis is found as the result of a collapsed vertebral body, usually due to pathological or traumatic fracture or resulting from adolescent osteochondrosis. It is best felt by gliding the fingers over the spinous processes in a craniocaudal direction. It is more difficult to detect at the thorax than elsewhere because of the general kyphotic shape of the thoracic spine.

Functional examination

Clinical routine differs significantly according to the level of the pain (Fig. 25.5). For a number of reasons, pain felt above the T6 level (mid-scapular) demands a preceding clinical examination of the cervical spine and the shoulder girdle. First, the upper two thoracic vertebrae, anatomically belonging to the thoracic spine but clinically part of the cervical spine, are tested in the clinical examination of the cervical spine. Second, it was demonstrated previously that cervical discodural interactions often provoke extrasegmentally referred pain in the upper half of the thorax. Third, numerous lesions of the thoracic apex and the shoulder girdle, although causing pain in the cranial aspect of the thorax, are only detected by proper examination of the cervical spine and/or the shoulder girdle: a tumour of the apex of the lung involving the T1 nerve root is only detected by cervical tests; neuritis of the suprascapular nerve causes pain in the supraspinatus fossa and is detected by a combined weakness of supra- and infraspinatus muscles. A fracture of the first rib provokes upper thoracic pain but the diagnosis can be missed if the examiner proceeds immediately

with examination of the thoracic spine. For all these reasons, in patients with upper thoracic pain, a full cervical examination followed by examination of the shoulder girdle must be done first. Only when these are negative should a thorough examination of the thoracic spine follow.

If the patient has pain below T6, a cervical or shoulder girdle problem is unlikely and attention is immediately directed to the thoracic spine.

Functional examination of the thoracic spine consists of a large set of basic tests. The examination is performed with the patient standing, sitting and lying prone. During the procedure, dural, articular, muscular and cord signs are sought. Sometimes, when particular symptoms or signs from the basic examination warrant, the procedure is completed by specific accessory tests.

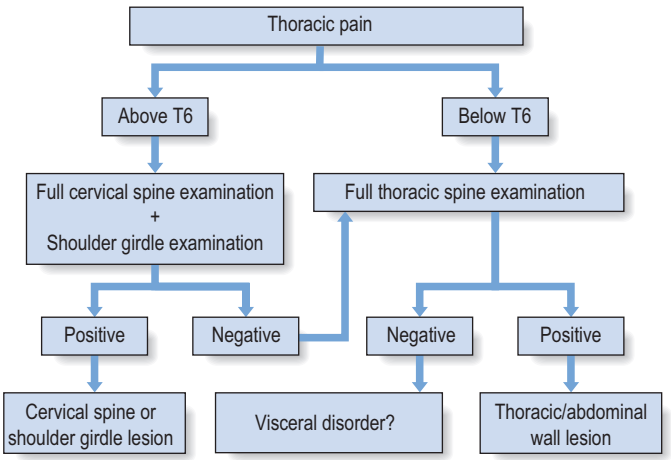


Fig 25.5 • Examination strategy in thoracic pain.

Standing

Dural tests

It is believed that deep inspiration, neck flexion and some scapular movements may indirectly stretch the dura mater.

Taking a deep breath (Fig. 25.6a)

The patient is asked to take a deep breath and to state if the pain increases. A positive test is regarded as a dural sign only when, after the rest of the examination is performed, a disc lesion seems to be present. In such a case, stretching of the dura mater via the intercostal nerve roots is interfered with.

Flexion of the neck (Fig. 25.6b)

The patient is asked to bend the head actively forward. This may increase the pain or provoke paraesthesia.

In thoracic spine problems, pain during active neck flexion is basically regarded as a dural sign because flexion stretches the dura mater from above. However, pain on neck flexion as the result of impaired dural mobility does not necessarily mean a disc lesion is present. Indeed, any kind of intraspinal space-occupying lesion that interferes with the dura, such as a tumour, may provoke pain on neck flexion. A problem with one of the posterior ligaments or posterior paravertebral muscles may also cause pain on neck flexion.

Sometimes a patient feels a sudden sensation on neck flexion, resembling an electrical discharge going down his back and occasionally even spreading towards both arms and legs. Sometimes it also occurs on extension of the neck. This is known as Lhermitte's sign and was previously regarded as pathognomonic for disorders of the cord at the cervical level. In later reports it has been suggested that problems of the thoracic cord may cause the same sign. This can be caused by

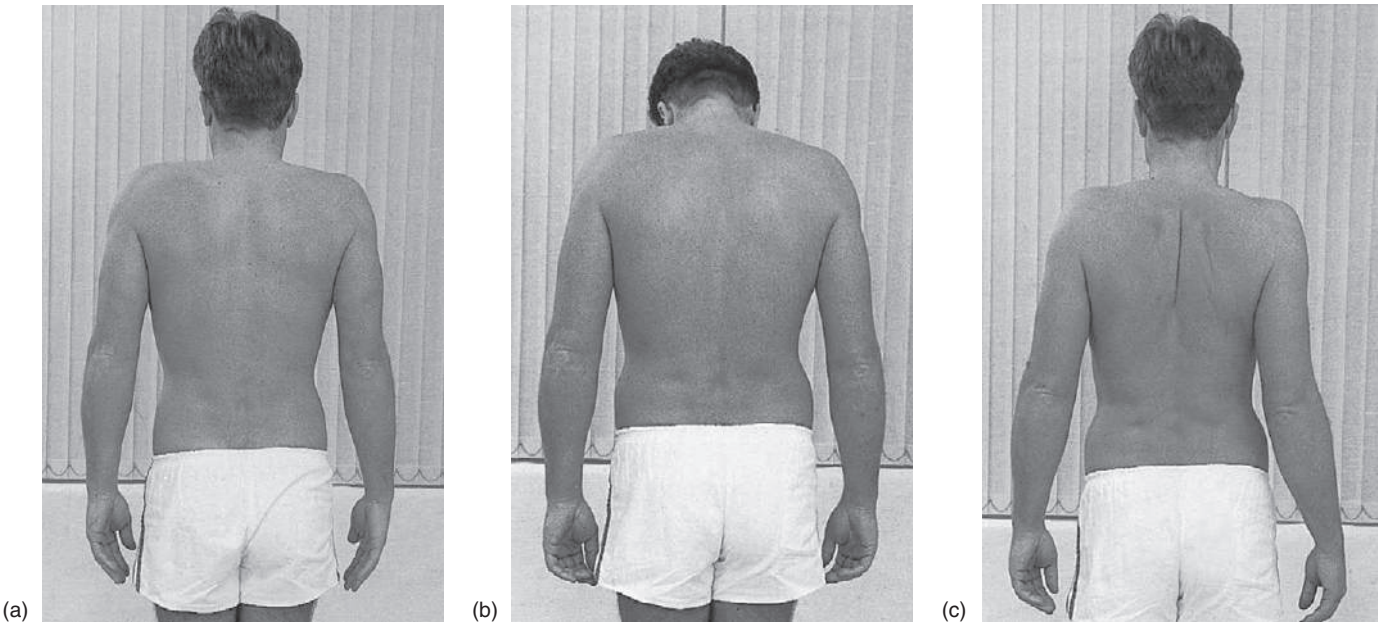


Fig 25.6 • Dural tests at the thoracic level: (a) taking a deep breath; (b) flexion of the neck; (c) backward movement of the scapulae.

multiple sclerosis, a tumour of the cord, disc lesions, tuberculosis, spondylosis, arachnoiditis or radiation myelopathy.[8]

Neck flexion may also provoke or increase pins and needles. If these are felt in one or both lower limbs, this draws attention to compression of the spinal cord at the thoracic level, which is most commonly the result of a disc lesion or a tumour.

Shoulder movements: upwards, forwards and backwards

The patient is now asked to shrug both shoulders and to bring them forwards and backwards.

These tests are basically active movements of the structures of the shoulder girdle. Therefore some will be positive when a disorder of one of those structures is present (see online chapter *Applied anatomy of the shoulder girdle*).

However, if one or all of these movements elicits pain, it is most frequently the result of a thoracic disc lesion, because they all (to a greater or lesser degree) stretch the dura mater at the thoracic level, which is elongated in a cranial direction via the T1 and T2 nerve roots (Cyriax[9]: p. 202). The most sensitive test is scapular approximation (shoulders backwards, Fig. 25.6c).

Active trunk movements

The patient is now asked to perform six active movements of the trunk. These involve both the thoracic and the lumbar spine. Differentiation is made by the level of the pain, as indicated by the patient, and on extension pressure at the end of the examination.

On flexion, extension and side flexion to both sides the range of movement is greatest in the lumbar spine, rather than the thorax. Rotation movements, in contrast, occur only slightly at the lumbar level and involve mostly the thoracic spine.

The movements performed are:

- Anteflexion (Fig. 25.7a)
- Extension (Fig. 25.7b)
- Left side flexion (Fig. 25.7c)
- Right side flexion (Fig. 25.7d)
- Left rotation (Fig. 25.7e)
- Right rotation (Fig. 25.7f).

The patient performs these movements actively. Pain and limitation are noted. In structural scoliosis the associated hump persists and is accentuated on forward bending.

While performing rotations, the patient keeps the head in neutral position in relation to the shoulders, to avoid cervical rotation.

Conclusions from active trunk movements

In principle, the six movements described so far are articular. However, because they are performed actively, they involve some contractile structures as well. Passive and resisted movements (see below) provide the key in differential diagnosis between inert and contractile structures. After these tests, the examiner should know whether an articular pattern is present or not.

The articular pattern of the thoracic spine is an equal degree of pain and limitation of both side flexions and of both rotations, together with a larger limitation of extension and little or no limitation of anteflexion (Fig. 25.8). It resembles the pattern for the cervical spine. If this is found, a disorder of the entire segment of motion, such as in ankylosing spondylitis or osteoarthrosis, is present.

Any other combination of abnormal tests is regarded as a partial articular pattern (Fig. 25.9). Such a combination could be, for instance, pain on one rotation or one side flexion together with one rotation, or one side flexion and extension, or three, four or five of the six movements being positive in producing pain or limitation. As long as abnormal tests are present in a non-symmetrical way, the pattern is regarded as being partial articular.

In all types of disc lesion a partial articular pattern is expected. In the thoracic spine, a common finding is that only one out of the six articular movements is positive – usually one of the rotations. Differentiation must always be made from a facet joint lesion or a muscular lesion, in which a partial articular pattern is also found. In the latter, some of the resisted movements are more painful.

Warning

When side flexion away from the painful side (Fig. 25.10) is the only painful and limited movement, this always indicates a severe extra-articular lesion such as a pulmonary or abdominal tumour or a spinal neurofibroma.

Sitting

The examination is now continued by looking at passive and resisted rotations to both sides, with the patient sitting. In the seated position Babinski's reflex is also elicited.

Passive tests

- Passive left rotation (Fig. 25.11a)
- Passive right rotation (Fig. 25.11b).

The patient crosses both arms in front of the chest. The knees are held between the examiner's legs to immobilize the pelvis. The patient's trunk is now twisted towards the left and the right by the examiner. Pain, range of movement and end-feel are noted.

The normal end-feel is elastic. A hard end-feel is typical of ankylosing spondylitis or advanced arthrosis. Both an empty end-feel and muscle spasm suggest a severe disorder: neoplasm, fracture and infectious disorders.

Rarely, pain is present at half range, disappearing when rotation continues. This is known as a painful arc and was regarded by Cyriax[9] as pathognomonic for a disc lesion when combined with a partial articular pattern.

At the end of both passive rotations the patient is asked to bend the head actively forwards. If this movement further increases the pain, it is regarded as a dural sign if the rest of the examination suggests that a disc lesion is present.

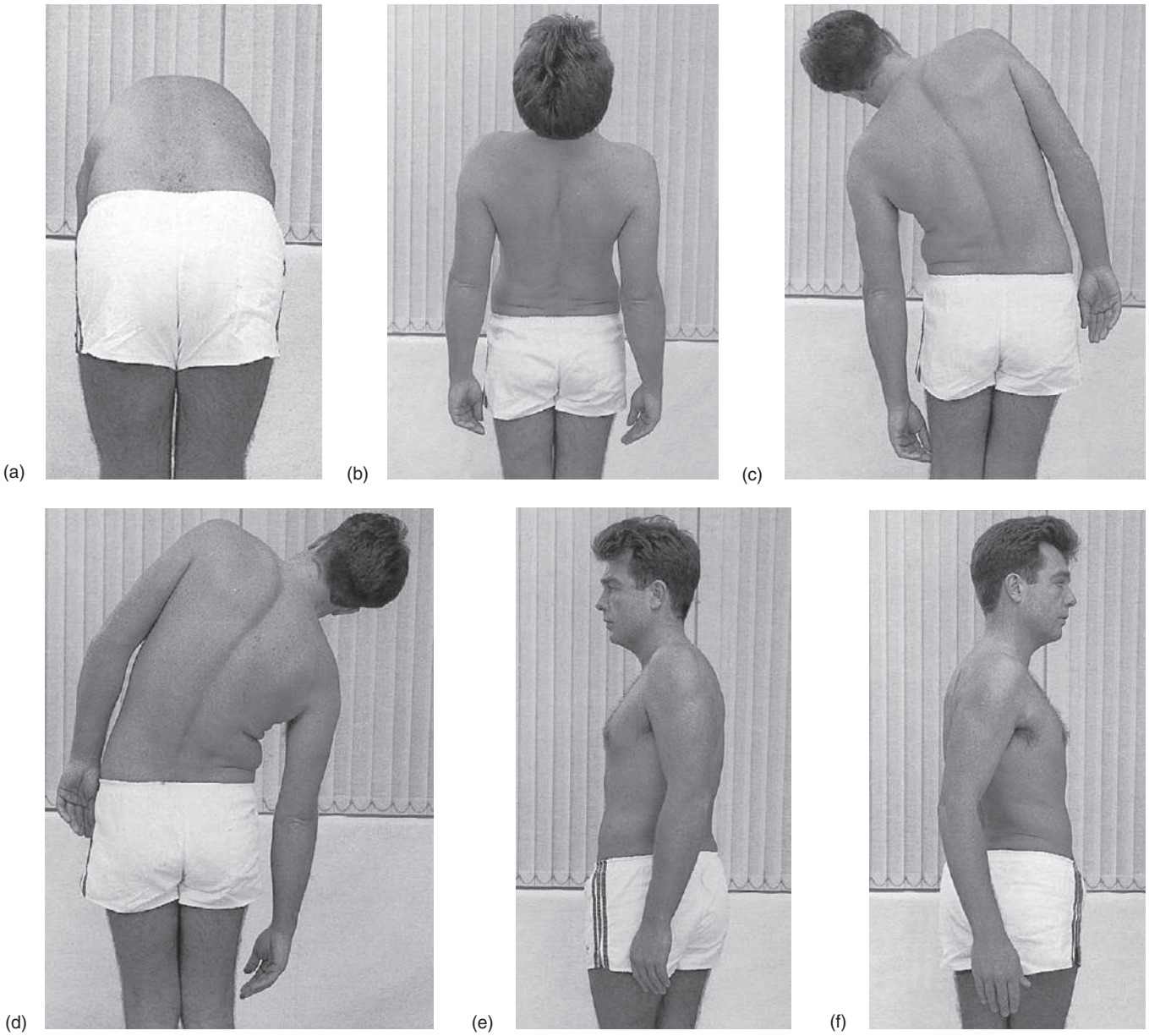


Fig 25.7 • Active trunk movements: (a) anteflexion; (b) extension; (c) left side flexion; (d) right side flexion; (e) left rotation; (f) right rotation.

Resisted tests

In the same position as used for the passive tests, isometric contractions are done. The patient is asked to twist the trunk to the left (Fig. 25.12a) and to the right (Fig. 25.12b) while the examiner applies counterpressure at both shoulders, so that the patient is kept immobile. Pain and weakness are noted. Because muscular lesions do occasionally occur at this level, these tests must always be performed.

The results of both resisted and passive rotations are carefully compared. In a disc lesion, passive rotations are more painful than resisted ones. Given that resisted movements are more painful, a muscular problem is most likely, unless a psychogenic problem or a rib fracture is present. In both events, accessory tests should follow (see below).

Cord sign: plantar reflex

The examiner glides a relatively sharp instrument along the lateral aspect of the sole, starting at the heel and moving forwards and medially towards the big toe (Fig. 25.13). Normally the toes either do not move at all or they all uniformly go into flexion. This test is pathological if the patient spreads the toes apart and the big toe moves into extension. A positive test indicates interruption of the descending motor fibres. If there is the slightest doubt about interference with the spinal cord, a full neurological examination of the lower limbs must be carried out. This includes all reflexes in the lower limbs and abdomen, resisted movements of the thigh and leg musculature, control of coordination, testing for numbness and temperature sensitivity, and the straight leg raising test.

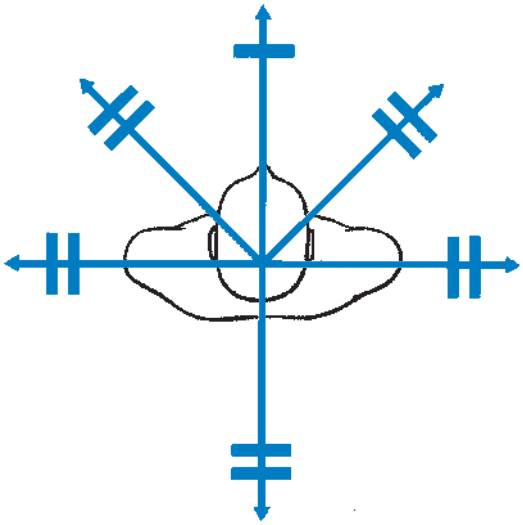


Fig 25.8 • The full articular pattern.

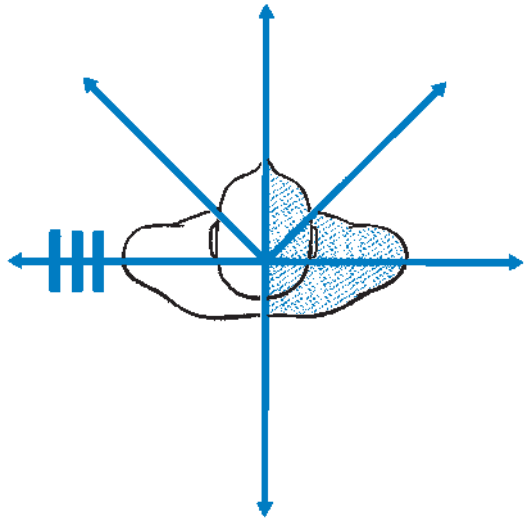


Fig 25.10 • Pain and limitation on side flexion away from the painful (coloured) side is a warning sign for serious disorders.

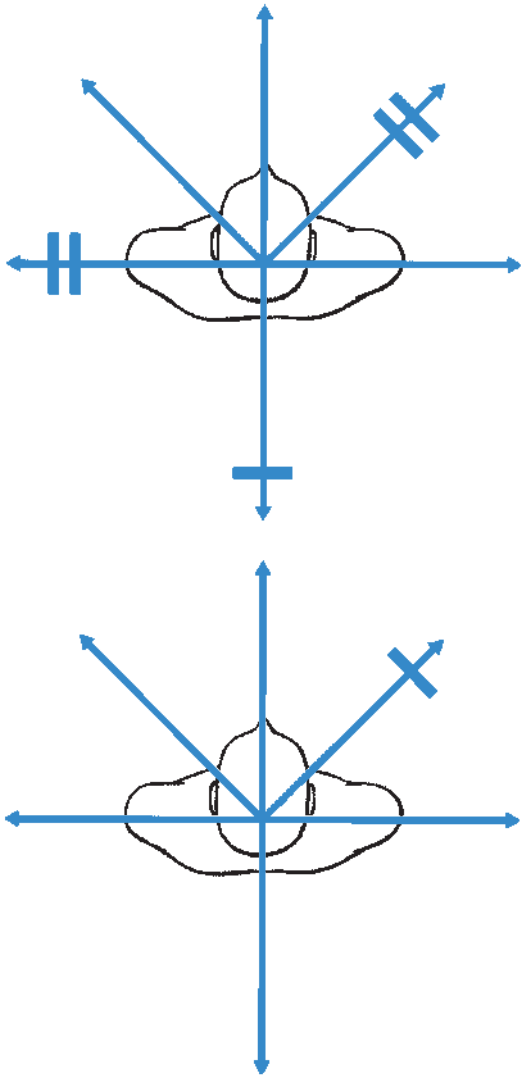


Fig 25.9 • Examples of partial articular patterns.

Lying prone

Location of affected level by passive extension thrust

The patient lies prone and a hyperextension thrust is given over every thoracic spinous process to locate the painful level. To do this, the hand is placed obliquely, with the fifth metacarpal bone on the spinous process (Fig. 25.14). Identification of the exact level is important, because some manipulations for thoracic disc protrusions are performed specifically at the level at fault.

During extension pressure, attention is also paid to the type of end-feel. Normally it is elastic. Muscle spasm is a warning of more severe disorders.

Accessory tests

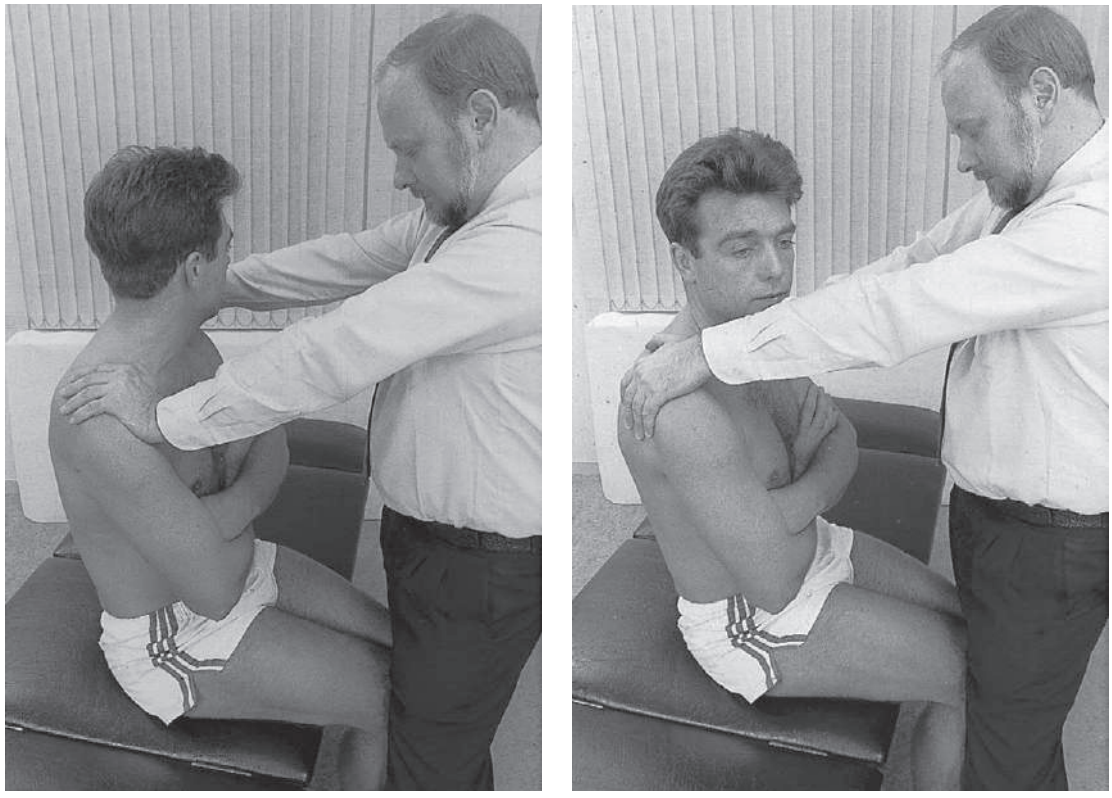
To reach a diagnosis, the basic clinical examination normally suffices. In circumstances that remain unclear or when a muscular problem is suspected, accessory movements must be carried out.

Stretching the T1 nerve root

The patient is asked to lift the arm sideways from the horizontal. The hand is now put in the neck by flexing the elbow (Fig. 25.15). This movement stretches the T1 nerve root via the ulnar nerve, which may provoke pain between the scapulae or down the arm when the mobility of the T1 nerve root is impaired. The test is useful for differentiating between a problem of the cervical spine and one of the upper thorax which interferes with the dura or the T1 nerve root: if it is painful, a thoracic problem is more likely.

(a) (b)

Fig 25.11 • Passive rotation: (a) left; (b) right. At the end of each movement the patient is asked to bend the head forwards.



(a) (b)

Fig 25.12 • Resisted rotation: (a) left; (b) right.

Fig 25.13 • Testing the plantar reflex.

Resisted movements and extension of the trunk

To gain more information on a muscular lesion, the following resisted movements should be performed.

Resisted side flexion (Fig. 25.16)
The patient stands, with the feet slightly apart. The examiner places himself at the patient's painless side, hips against each other, and puts the arm around the patient's farther shoulder. The patient is now asked to bend sideways away from the examiner. By holding the patient's shoulder, side flexion of the trunk is resisted.

Resisted flexion (Fig. 25.17)
With the patient sitting down, the examiner places one hand on the proximal part of the sternum and the other on the patient's knees. The patient tries to bend forwards against resistance exerted by the examiner. This is a test for all the flexors of the abdomen and of the hip.

Extension of the trunk

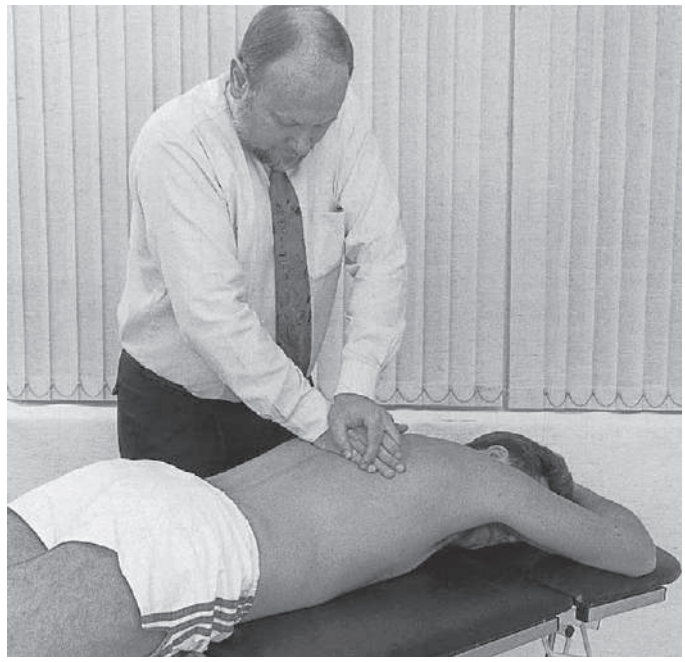
This movement is performed in three different ways.

Resisted extension (Fig. 25.18a)
Resisted extension is best done with the patient prone. Counterpressure is applied at the proximal part of the thorax and at the posterior aspect of the knees.

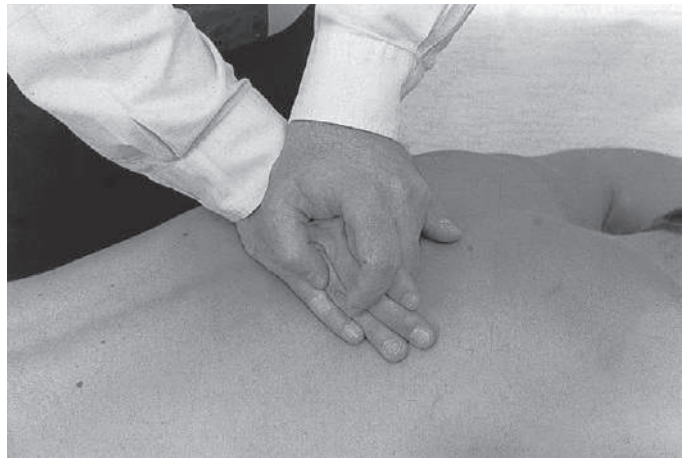
Active extension (Fig. 25.18b)
For active extension the patient remains prone and is asked to lay both hands on the sacrum and lift the trunk off the couch actively by use of the paravertebral muscles.

Passive extension (Fig. 25.18c)
For passive extension the patient pushes the body up off the couch by means of the arms. The pelvis must stay down on the couch.

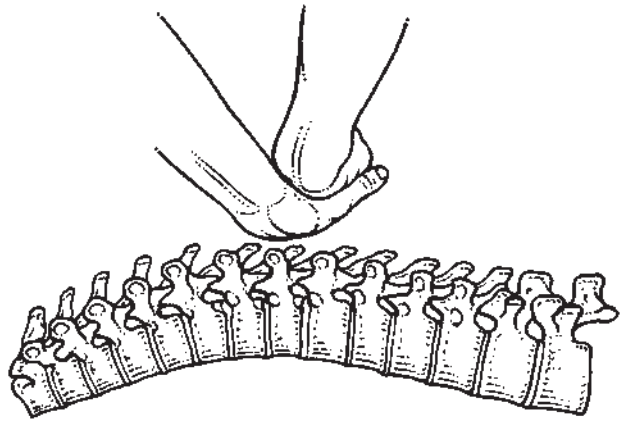
The results of extension movements are carefully compared with each other. In a muscular lesion, active and resisted extension is painful but resisted extension is the most distressing. In a lesion of an inert structure, active and passive extension is painful but the latter provokes the most pain.



(a)



(b)



(c)

Fig 25.14 • Passive extension thrust.

Testing the long thoracic nerve

The patient pushes against a wall with the arms stretched out horizontally in front (Fig. 25.19). If the medial edge of the scapula moves away from the thorax to produce a winged appearance, a disorder of the long thoracic nerve is present.

Oscillation of a rib

The examiner stands on the pain-free side and places one hand distally on the thorax, with the fifth metacarpal bone exactly on the suspected rib. The other hand rests with the pisiform bone on the contralateral transverse process of the corresponding vertebra (Fig. 25.20). Oscillations are now given by the hand resting on the rib. At the same time, the other hand is used to prevent rotation of the vertebra by pressing simultaneously on the transverse process. These oscillations influence mainly the costovertebral and costotransverse joints. When there is inflammation, pain will be provoked; when ankylosing spondylitis is present, the movement will be less elastic.

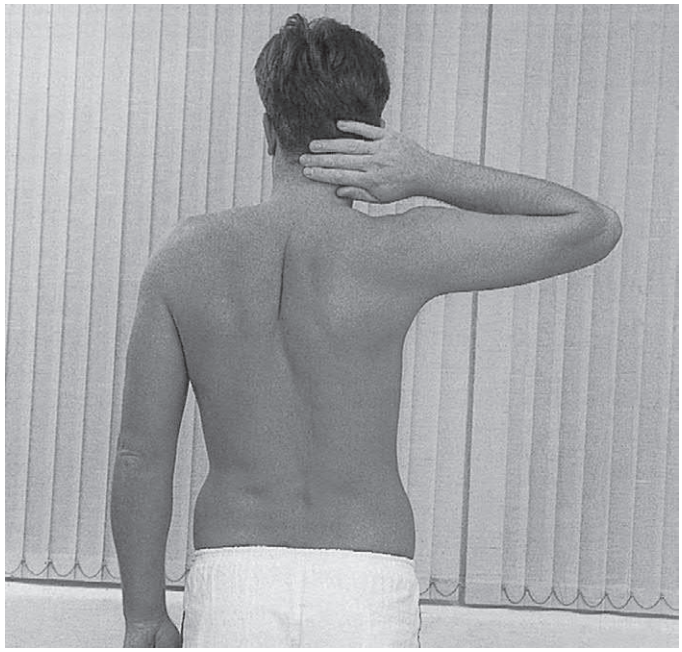


Fig 25.15 • Stretching the T1 nerve root.

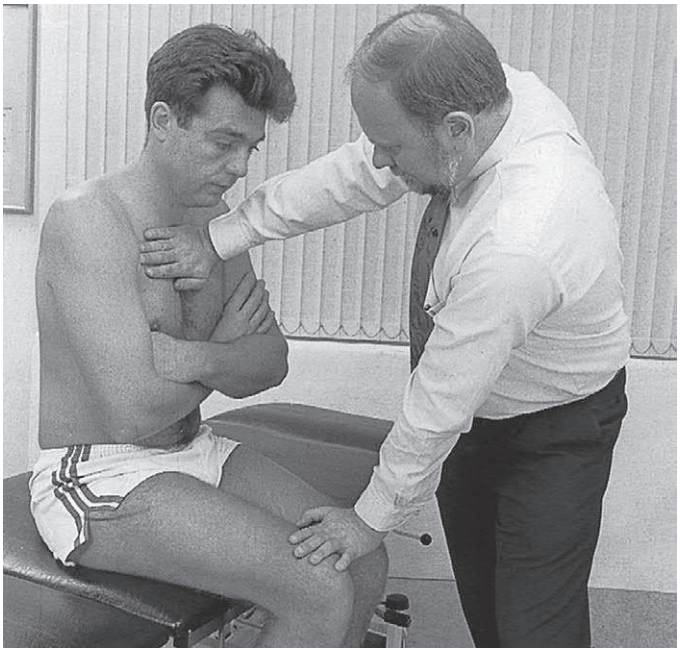
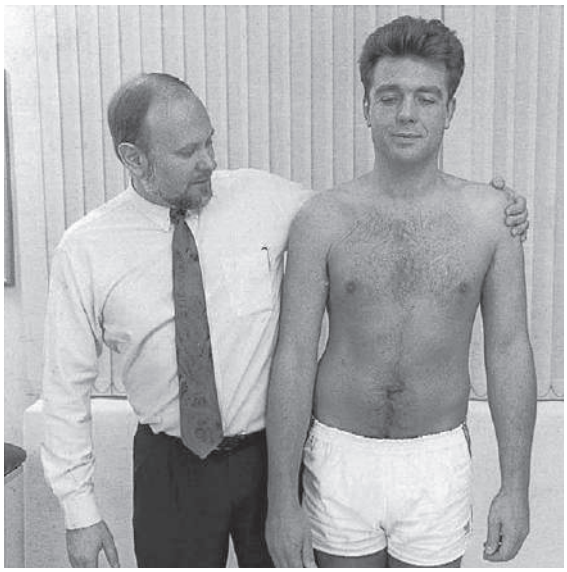


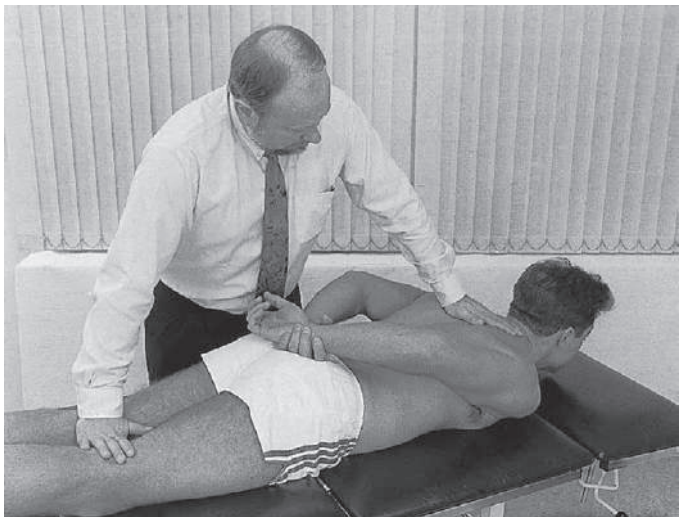
Fig 25.17 • Resisted flexion.

(a)



(b)

Fig 25.16 • Resisted side flexion: (a) right; (b) left.



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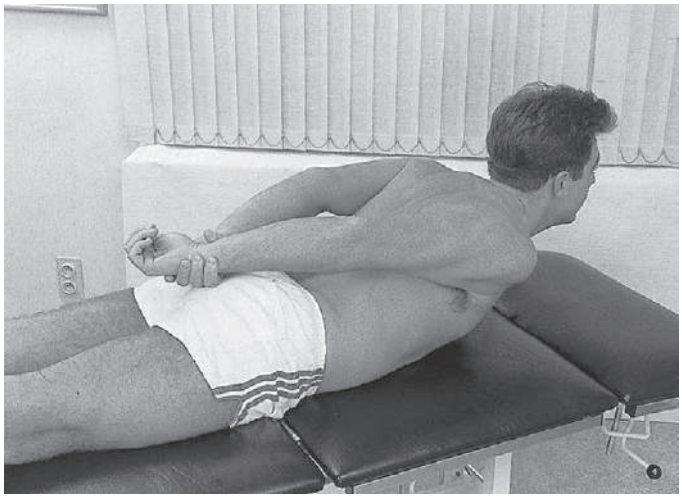

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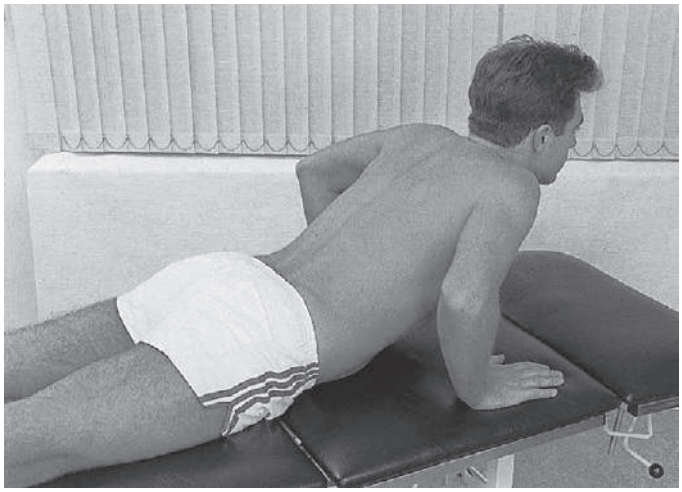


(c)

Fig 25.18 • Extension of the trunk is performed in three different ways: (a) resisted; (b) active; (c) passive.

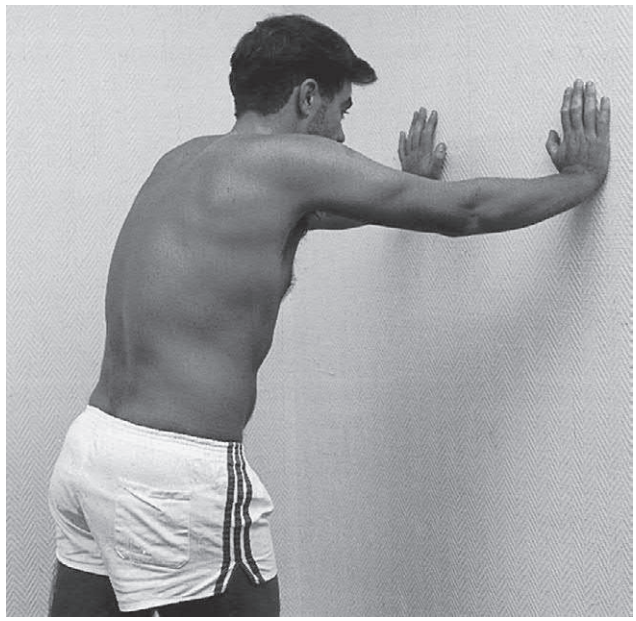


Fig 25.19 • Testing the long thoracic nerve.

Neurological examination

A full neurological examination must be carried out when compression of the spinal cord or a neurological disorder is suspected.

The following accessory tests should be performed.

Beevor's sign

The patient lies supine, crosses the arms in front of the chest and is asked to raise the trunk slightly off the couch. The examiner pays attention to the umbilicus, which should not move during this test. Any movement in a cranial or caudal direction or to the side may point towards a denervation of the contralateral muscles.

Cremasteric reflex (in men)

When the pointed end of a reflex hammer is glided over the medial aspect of the thigh, the ipsilateral half of the scrotum moves upwards via contraction of the cremaster muscle. Absence of this reflex may point towards a lesion of the spinal cord.

Full neurological examination of the lower limbs

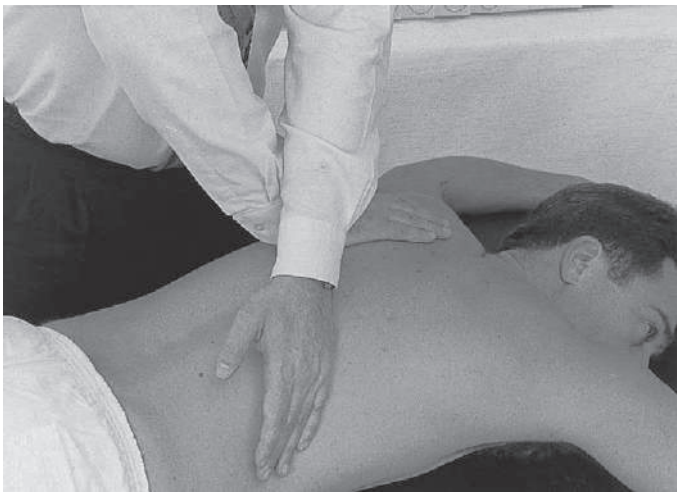
This is described in Table 25.1.

Oppenheim's sign

This may confirm a positive Babinski's sign. When the fingers are slid downwards along the tibia, no movement of the toes should occur. In cord compression, the big toe extends while the others spread. However, this test is less reliable than a Babinski's sign.

Palpation

If a muscular lesion is suspected, the affected structure should be palpated.



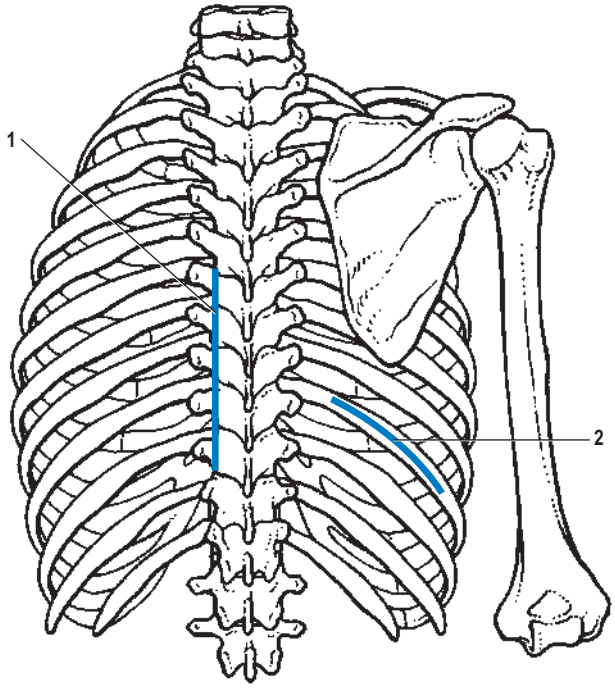


Fig 25.20 • Oscillation of a rib: 1, position of fifth metacarpal right hand; 2, position of fifth metacarpal left hand.

Palpation is also necessary to differentiate between a lesion of an intercostal muscle and a true rib problem.

In a disc lesion, cutaneous anaesthesia must always be checked. If present in the territory of one nerve root, a posterolateral protrusion is likely; bilateral numbness suggests compression of the spinal cord instead.

The clinical examination of the thoracic spine is summarized in Box 25.2.

Technical investigations

Plain radiographs are useful in confirming osseous lesions and in the evaluation of degree and development of scoliosis.

Table 25.1 Full neurological examination of the lower limbs

Tests	Nerve root
Inspection of gait	
Motor tests	
Resisted flexion of hip	L2–L3
Resisted extension of knee	L3
Resisted dorsiflexion of foot	L4
Resisted extension of big toe	L4–L5
Resisted eversion of foot	L5–S1
Resisted flexion of knee	S1–S2
Squeezing the buttocks	S1–S2
Raising on tiptoe	S1–S2
Reflexes	
Patellar reflex	L3
Achilles tendon reflex	S1
Sensitivity	
Temperature	
Numbness	

During the last decades the use of computed tomography (CT) in combination with myelography and magnetic resonance imaging (MRI) has greatly increased the ability to visualize thoracic spine disorders accurately. MRI is the best way to define the specific abnormality, as well as the effect on the adjacent spinal cord. CT after myelography may be useful as well, especially in those patients in whom there is involvement of the posterior ligamentous and osseous structures of the thoracic spinal canal.[10]

However, the superior resolution of the available imaging methods has also made the incidental detection of asymptomatic thoracic disc abnormalities more common.[11] As with the lumbar and the cervical spine, it has become evident that the correlation between gross anatomical findings on MRI and clinical signs and symptoms detected by the clinician may be lacking. A significant proportion of the population has disc disease as depicted on imaging studies, yet many have no clinical findings at all.[12,13] The relative frequency of asymptomatic thoracic herniated nucleus pulposus has been documented in several studies.[14,15] Wood *et al* reviewed MRI studies of the thoracic spines of 90 asymptomatic individuals to determine the prevalence of abnormal anatomical findings: 66 (73%) had positive anatomical findings at one level or more, including herniation of a disc in 33 (37%), bulging of a disc in 48 (53%), an annular tear in 52 (58%), deformation of the spinal cord in 26 (29%) and Scheuermann endplate irregularities or kyphosis in 34 (38%).[16]

Awwad *et al* retrospectively reviewed postmyelography CT scans of 433 patients and identified 68 asymptomatic thoracic

Box 25.2

Summary of the clinical examination of the thoracic spine

History

Pain

What made the pain come on?

- Injury?
  - Bony problem
- Spontaneous onset?
  - Disc lesion
  - Arthritis
  - Tumour
- Forceful activity?
  - Muscular lesion

Where was the pain at the beginning/where did it spread or shift to/where is it now?

- Interscapular above T6?
  - Cervical problem
  - Shoulder girdle
  - Thoracic lesion
- Interscapular below T6?
  - Thoracic lesion
- Base of the neck?
  - Costovertebral
  - First rib
  - Sternoclavicular
- Shifting pain?
  - Disc
- Increasing or expanding pain?
  - Tumour

Is the pain influenced by coughing, sneezing or a deep inspiration?

Paraesthesia

- Multisegmental/lower limbs?
  - Cord compression
- Segmental?
  - Root compression
- Undefined?
  - Other neurological disorder

Anticoagulant treatment and bleeding disorders

Inspection

Functional examination

Standing

- 3 dural tests
  - Taking a deep breath
  - Flexion of the neck
  - Shoulders backwards
- 6 active trunk movements
  - Anteflexion
  - Extension
  - Left side flexion
  - Right side flexion
  - Left rotation
  - Right rotation

Sitting

- 2 passive tests
  - Passive left rotation (+ neck flexion)
  - Passive right rotation (+ neck flexion)
- 2 resisted tests
  - Resisted left rotation
  - Resisted right rotation
- Cord sign
  - Plantar reflex

Lying prone

- Location of the affected level by passive extension thrusts

Accessory tests

- Stretching the T1 nerve root
- Resisted movements of the trunk
- Testing the long thoracic nerve
- Oscillation of a rib
- Neurological examination
- Palpation

herniated discs. After comparing the imaging characteristics with a series of five symptomatic thoracic herniated discs, the authors were unable to identify any features that could reliably classify a herniated disc as asymptomatic or symptomatic.[17]

All these studies clearly demonstrate that thoracic disc herniations shown by MRI may not be related to patients' symptoms. The diagnosis 'symptomatic thoracic disc lesion' is therefore primarily a clinical one.

Access the complete reference list online at www.orthopaedicmedicineonline.com

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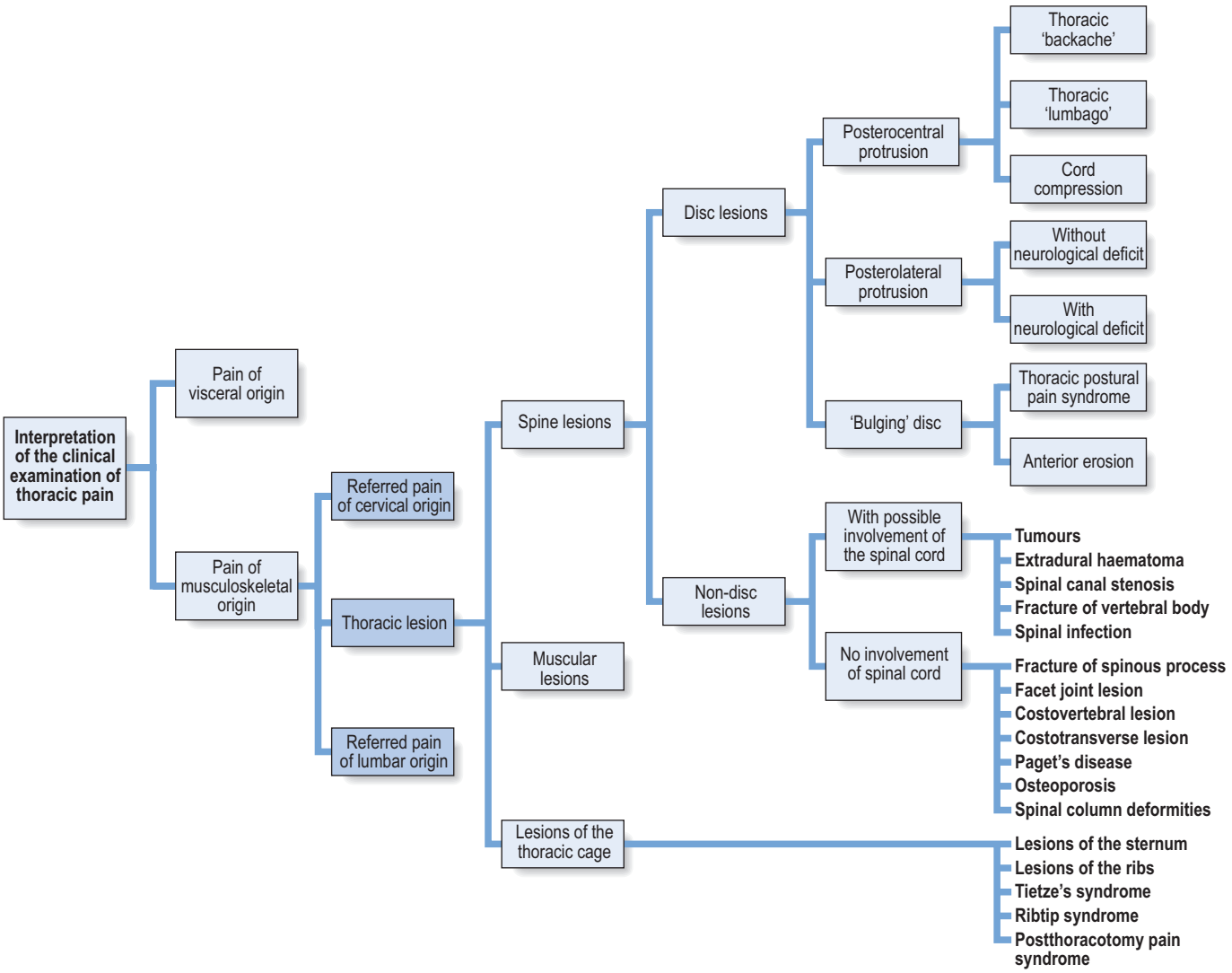
Interpretation of the clinical examination of the thoracic spine



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Disorders of the thoracic spine: disc lesions

CHAPTER CONTENTS

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Although the spine is anatomically part of the thoracic cage, we prefer to discuss the thoracic disorders in two main categories – spinal lesions (this chapter and Ch. 28) and lesions of the thoracic cage and abdomen (see online chapter *Disorders of the thoracic cage and abdomen*). Thoracic ankylosing spondylitis is discussed separately (Ch. 29). This is done to standardize the discussion of the spine throughout, in the hope that a better clinical understanding may result.

Introduction

Cervical and lumbar disc lesions are widely accepted as common causes of pain. For the thoracic spine, the situation is different. Although thoracic disc lesions giving rise to compression of the spinal cord are well recognized,[1–5] disc protrusion resulting in pain without causing neurological signs is poorly documented.[6] The incidence of thoracic disc lesions affecting the spinal cord is about one case per million people per year,[3,7] usually affecting adults, although cases have been reported in children as young as 12.[8] The existence of minor thoracic disc lesions provoking pain in the absence of cord compression was first established by Hochman, who removed a disc protrusion at T8–T9 in a 67-year-old lady with continuous unilateral pain in the thorax.[9] Neurological signs were not present. The diagnosis was established by computed tomography (CT).

The incidence of minor thoracic disc lesions is much higher. Degenerative changes of the thoracic spine are observed in approximately half of asymptomatic subjects and 30% have a posterior disc protrusion.[10] A recent magnetic resonance imaging (MRI) study found a prevalence of thoracic disc herniations of 37% in asymptomatic subjects, disc bulging in 53% and annular tears in 58%.[11] Another study of asymptomatic patients identified impressive disc protrusions in no less than 16%.[12] An unexpectedly high prevalence of thoracic disc herniation (14.5%) was also demonstrated in the thoracic spines of a group of 48 oncology patients examined by MRI.[13] Although these relatively high figures do not correspond to the real clinical situation, we believe that symptomatic thoracic disc protrusions are far more common than is generally accepted and agree with Krämer,[14] who estimated the frequency of thoracic disc lesions to be about 2% of all symptomatic disc lesions. Recent studies also confirmed the incidence of symptomatic thoracic disc prolapses as being between 0.15% and 4% of all intervertebral disc prolapses.[15,16]

However, the clinical diagnosis is often not made and the patients are frequently classified as suffering from intercostal neuralgia, neuritis, cardiac neurosis or pleurodynia. If left untreated, the pain can persist for many years, causing continuing morbidity. Nevertheless, the majority of such lesions can easily be reduced in a few manipulative sessions, fully relieving the patient's pain.

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Although more frequently present than commonly believed, thoracic disc protrusions are clinically far less common than those in the lumbar spine because of the greater rigidity of the thoracic spine. This is partly a result of the stabilizing effect of the rib cage on the thoracic spine and partly due to the thoracic intervertebral discs, which are thinner on account of a less voluminous nucleus pulposus.[17] Therefore extension and flexion movements are of a smaller range in the thoracic spine.

Minor thoracic disc lesions occur most often between T4 and T8. Those with cord compression are usually found in the lower half of the thorax.[6,18] About 70% lie between T9 and T12, the commonest level (29%) being T11. A logical explanation for this could be that the lower segments have an increased mobility due to free ribs at these levels.[19] Another reason could be that the cord has a critical vascular supply at this level.[20]

Clinical presentation

It is hypothesized that disc degenerations and disc displacements are of themselves painless events because the disc is almost completely without nociceptive structures. Clinical syndromes originate only when a subluxated fragment of disc tissue impinges on the sensitive dura mater or on the dural nerve root sleeve. This clinical hypothesis is extensively discussed in the lumbar section of this book (see Chs 31 and 33).

Disc displacements (protrusions and prolapses) are either soft (nuclear) or hard (annular), and may have a posterocentral or posterolateral localization.

Posterocentral protrusions compressing the dura mater may provoke multisegmental pain, which is mainly referred into the posterior thorax but may also spread into the anterior chest, the abdomen or the lumbar area.[21] The pain is never referred down the arm. When a posterocentral displacement increases, cord compression can result.

Posterolateral protrusions interfering with the dural sleeve around the nerve root result in pain that is segmentally referred into the corresponding dermatome. A more massive posterolateral protrusion may compress the ganglion or the nerve root fibres, resulting in motor and/or sensory disturbances in the innervation area of the root.

When the T1 nerve root is compressed by a disc lesion, pain is referred to the inner side of the arm between elbow and wrist. A T2 nerve root impingement creates pain referred towards the clavicle and to the scapular spine and down the inner side of the upper arm. The corresponding dermatomes of the T3–T8 nerve roots follow the intercostal spaces, ending at the lower margin of the thoracic cage. The dermatomes of T9–T11 include a part of the abdomen, and T11 also includes part of the groin (see Fig. 25.3).[22,23]

Thoracic disc protrusions may give rise to four different clinical presentations: chronic thoracic backache, acute thoracic lumbago, thoracic root pain and spinal cord compression (Cyriax:[24] pp. 202–205).

Each clinical pattern corresponds to a specific type of disc lesion. Besides dural and articular signs and symptoms, elements which may indicate compression of the spinal cord must always be sought.

Symptoms and signs

The clinical findings in symptomatic thoracic disc displacements are analogous to the lumbar and cervical disc syndromes. Again, both dural and articular signs and symptoms can be identified (see Ch. 52).

Dural symptoms

Increasing the intra-abdominal pressure by deep inspiration, coughing or sneezing may aggravate the pain in all types of symptomatic disc displacements. A deep breath usually has more effect than does a cough – the opposite is true for the lumbar spine – and is the consequence of traction exerted on the dura mater via the intercostal nerves. This dural symptom is of major importance in the lumbar spine but is less helpful in the thorax. Indeed, pain from other disorders of a musculoskeletal or visceral nature may also be under the influence of respiratory movements. Therefore these symptoms are nonspecific at the thoracic level and can be interpreted only as suggesting that the dura is involved when the rest of the history and examination indicates a disc lesion.

Articular symptoms

Pain in disc lesions is caused by certain positions and movements, and disappears with others. Often activities in prolonged flexion or rotation movements provoke or increase the pain.

Dural signs

In disc lesions interfering with the normal mobility of the dura mater or of the dural nerve sleeve, movements that stretch the dura pull in harder against the protrusion and increase compression and pain. The movements regarded as dural signs are:

- Pain on neck flexion
- Pain on movements of the scapulae, most commonly on scapular approximation, which stretches the dura via the T1 and T2 nerve roots.

Articular signs

A disc lesion usually affects only a part of the intervertebral joint. Therefore, certain movements cause biomechanical changes that result in forcing the protrusion against the dura. Consequently, it is to be expected that on clinical examination only some of the active movements will increase the discodural interactions while other movements will be without effect and therefore painless. This results in a partial articular pattern, which is an absolute condition for the diagnosis of a disc protrusion, although it is not pathognomonic.

Examples of non-articular patterns are illustrated in Figure 27.1.

Depending on the degree of the compression, the partial articular pattern is more or less pronounced: more tests are painful and more severe pain is present. However, frequently, only one movement is painful, most commonly one of the rotations.

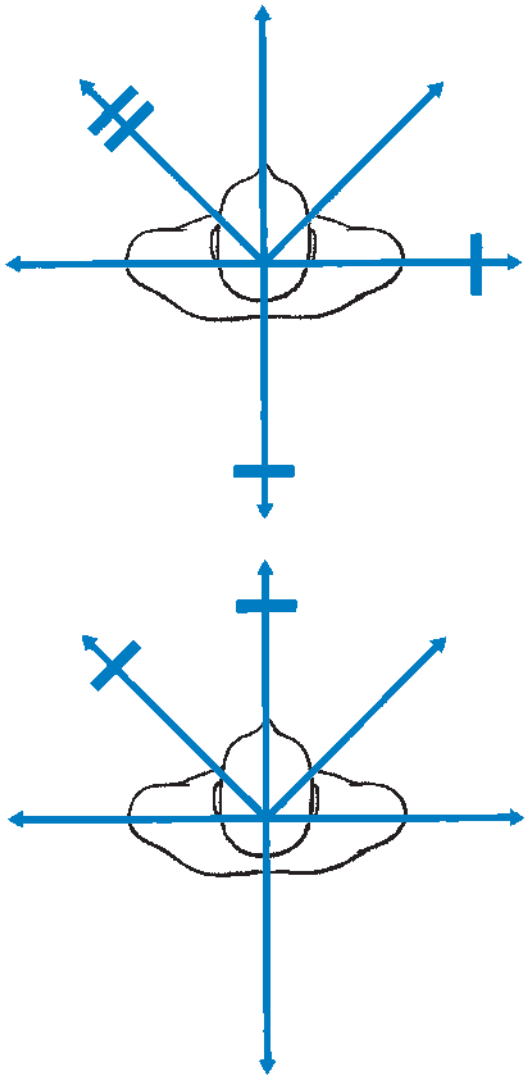


Fig 27.1 • Examples of partial articular patterns.

Often both articular and dural signs are present, although the latter are sometimes absent.

Exceptionally, a combination of a partial articular pattern with pain on one or more resisted movements is found. In this event, the question that arises is whether there is a muscular problem or a lesion of an inert structure. In the latter, pain on passive movements is more severe whereas, in the former, resisted movements are more painful.

Pain and limitation on side flexion towards the painless side as the only positive movement does not match the pattern of a disc lesion. Other disorders, such as a pulmonary or abdominal tumour with invasion of the thoracoabdominal wall, must be considered. An intraspinal tumour – for example, a neurofibroma – is also possible (see online chapter *Disorders of the thoracic spine and their treatment*).

Warning

If pain and limitation on side flexion away from the painful side is the only abnormal movement (Fig. 27.2), a tumour should be suspected.

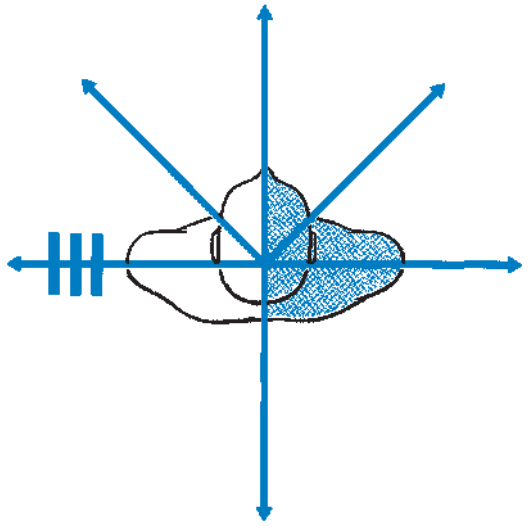


Fig 27.2 • Pain and limitation only on side flexion away from the painful (coloured) side is a warning sign for serious disorders.

Table 27.1 Articular and dural symptoms and signs in thoracic disc lesions

	Symptoms	Signs
Articular	Particular movements or postures increase the pain; others ease	Existence of a partial articular pattern
Dural	Pain on deep breath	Pain on neck flexion Pain on scapular movements

Symptoms and signs of cord compression

In thoracic disc lesions, careful attention must always be paid to abnormal neurological elements that may indicate compression of the spinal cord: pins and needles in both feet, disturbed coordination of lower limbs and positive Babinski's sign (see Table 27.1 and pp. 165–168).

Clinical types of thoracic disc protrusion

Symptomatic disc displacements in the thoracic spine may give rise to four different clinical syndromes: acute thoracic 'lumbago', chronic thoracic backache or 'dorsalgia', thoracic root pain and spinal cord compression (Figs 27.3–27.5).[24] Each syndrome corresponds to a specific type of disc lesion.

It is obvious that, besides dural and articular signs and symptoms, elements that may indicate compression of the spinal cord must always be sought.

Thoracic backache

A small annular posterocentral disc displacement causes a unilateral discodural interaction. The subsequent unilateral

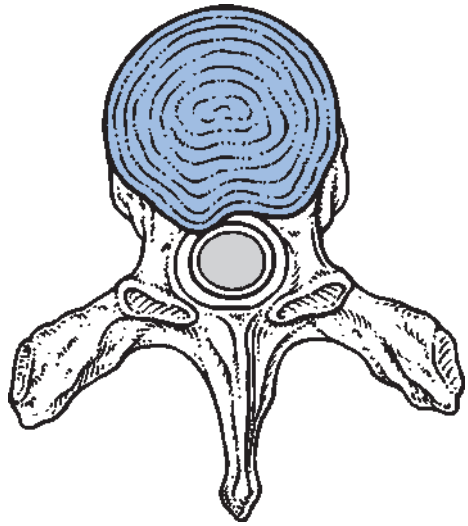


Fig 27.3 • Thoracic disc displacement: small posterior displacement with dural contact – nuclear 'lumbago' or dorsalgia.

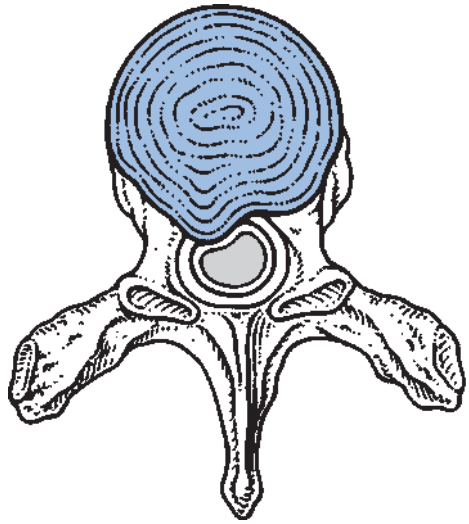


Fig 27.4 • Thoracic disc displacement: massive posterior displacement – cord compression.

interscapular pain is of moderate severity and is usually brought on by prolonged activities or postures in anteflexion. The pain gets worse during or after particular movements or positions and wears off on rest, but seldom disappears completely. In chronic thoracic backache there is no spontaneous recovery and untreated pain can persist for many years.

Because the pain is usually not severe, the articular signs are not always very impressive. However, no matter how subtle the outcome of the tests, some 'asymmetry' in the clinical picture will always be present. It is, for instance, not unexceptional to find only one articular movement that is slightly positive, very often one of the rotations.

This non-articular pattern, in the absence of pain on resisted movements, can usually be interpreted as the outcome of a disc lesion. A further argument in favour of this diagnosis is

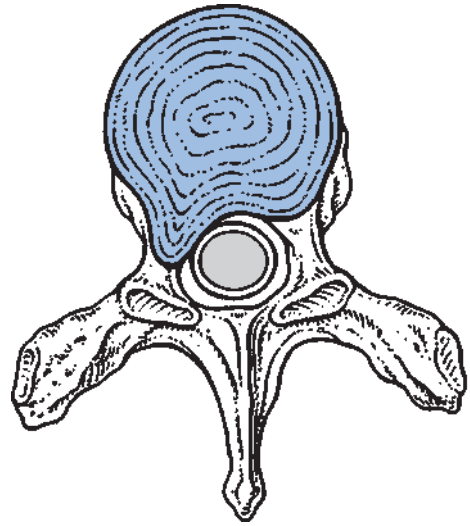


Fig 27.5 • Thoracic disc displacement: posterolateral displacement with radicular compression.

when neck flexion increases the pain on full passive rotation of the trunk (dural sign).

If both posterior pain and anterior thoracic pain are present, the posterior pain is often influenced by articular movements and the anterior pain is increased by neck flexion.

Disc lesions at level T1 and T2 may give rise to diagnostic difficulties in that very little spinal mobility exists at this level and articular movements may have no influence at all on the pain, so none is positive. The pain is often provoked only by flexion of the neck, which then suggests a cervical disc lesion. When this pattern is found, a differential diagnosis between both types of disc protrusion must be made. Stretching the T1 root and performing all three scapular movements provides the key: they may provoke or increase the pain in a high thoracic disc lesion but usually have no influence on a cervical disc protrusion.

In chronic thoracic backache there is no spontaneous recovery and untreated pain can persist for many years.

All cases of thoracic backache from a disc protrusion, in the absence of any contraindication, should be treated by manipulations, which are usually quickly successful. If this fails, traction must be tried. If there is frequent recurrence, sclerosant infiltrations into the different ligaments and facet capsules must be given to increase spinal stability.

Special case: self-reducing disc lesion

As the term implies, the disc undergoes spontaneous reduction. Patients who suffer from this condition usually sit for most of the day. On waking up in the morning they are symptom-free but, after they sit for some hours, pain starts in the mid-thorax and gets progressively worse. On lying down, the pain gradually eases off. The time needed for the pain to disappear fully depends on the degree of displacement and is initially from 10 to 15 minutes. Later, or if the patient sits for a longer period, it may take an hour or more. Pain may be absent when prolonged sitting is not part of the daily routine – for example, at weekends.

In this condition the disc gradually dehydrates as the result of the prolonged sitting position.[25] Simultaneously, the imposed kyphosis pushes the whole intra-articular content of the disc posteriorly, compressing the dura mater and resulting in thoracic backache. On lying down, the effects of hyperkyphosis and gravity are largely diminished and the disc shifts spontaneously back into its original position. These patients should avoid prolonged anteflexion. Manipulative reduction is useless but sclerosant infiltrations may be helpful.

Acute thoracic lumbago

Due to a combined flexion–rotation movement of the trunk, the patient becomes suddenly immobilized by a sharp posterior pain in the thorax, with the trunk fixed in flexion. The condition is similar to acute lumbago at the lumbar spine. In both, the underlying cause is a posterocentral disc protrusion. Pain is usually felt posteriorly in the centre of the back, radiating unilaterally or bilaterally around the chest. In high thoracic lesions, the pain may even reach the sternum; in low thoracic lesions, the pain is sometimes referred to the abdomen.

A deep breath is very painful and normally hurts more than a cough. Sudden onset in the absence of injury in a middle-aged person, together with pain on deep inspiration and on neck flexion, is very suggestive of such a disc lesion.

A partial articular pattern is present in which three, four or five movements are painful, and in severe instances all movements cause pain, but still in an asymmetrical way. The symptoms and signs are thus much more pronounced than in thoracic backache.

If the patient stays in bed for some days, spontaneous resolution occurs over about 2 weeks. Cure can be obtained much more quickly by manipulative reduction. Exceptionally the pain can be so severe that the patient can hardly stand up or needs a great deal of time to turn around on the couch. Such patients should remain in bed until they have improved to such a point that normal manipulations become possible. A manipulative attempt should not be made during the initial period because it is unbearable. Sometimes special oscillatory techniques can be tried.

Recurrence may occur but the pain is not necessarily always felt at the same side.

Special case: sternal lumbago

This is a rare disorder that often causes diagnostic difficulties. A high or mid-thoracic posterocentral protrusion coming on suddenly can give rise to agonizing pain felt only anteriorly over the sternum or in the epigastrium, without back pain. Differential diagnosis with an acute myocardial infarction has to be made but is not always simple. The effect of deep inspiration can be helpful: it increases the pain of a disc lesion but usually does not affect that originating from the myocardium.

Thoracic root pain

As in the cervical and lumbar spines, two types of root pain can be encountered: a primary and a secondary posterolateral protrusion. The latter is more common. In the first type, the protrusion is deviated in a posterolateral direction from the onset; in the second, there is first a posterocentral protrusion that later shifts laterally, analogous to a lumbar disc lesion.

- *In a primary posterolateral protrusion*, segmental pain is felt from the start at the lateral aspect of the thorax and often radiates unilaterally to the front of the chest or the abdomen. The absence of pain in the back may lead to the discogenic origin being overlooked.
- *In a secondary posterolateral protrusion*, an extrasegmental posterocentral or posterior unilateral pain is initially present, which then moves more to the side and sometimes towards the anterior thorax or abdomen – meanwhile becoming segmentally referred – a sequence of symptoms that strongly suggests a secondary posterolateral disc lesion.

Both types of root compression give rise to segmental referred pain. This has a unilateral band-shaped distribution that follows the intercostal nerves. At the thoracic level a posterolateral protrusion seldom gives rise to pins and needles. If present, they follow the same segmental distribution as the pain. As the T12 dermatome spreads into the lower abdomen, interference with this nerve root can result in pain and occasionally pins and needles in the groin and/or the testicles.[26]

On functional examination, a partial articular pattern is usually found. Occasionally, the patient feels nothing on articular movements but flexion of the neck provokes a sharp unilateral sternal pain, sometimes accompanied by pins and needles in the same place. As flexion of the neck also stretches the nerve roots via the dura, one of these can be pulled further against a protrusion, resulting in a sudden pain.

Neurological signs are seldom encountered and, if present, are always difficult to diagnose. As the majority of the thoracic nerve roots cannot be stretched by movements of arms or trunk, an analogue of straight leg raising does not exist. Moreover, motor deficit, except for a T1 lesion, cannot be tested. The only feature that is occasionally found is numbness. When present, it is of little help in determining the exact level of the protrusion because the dermatomes overlap. Only numbness in the groin draws attention to a nerve root palsy – T12. In rare instances, an area of hyperaesthesia is felt in the front of the chest.

A T1 root palsy is detected during the clinical examination of the cervical spine. It is seldom the result of a disc protrusion but usually the outcome of a serious disorder such as a superior sulcus tumour of the lung, a neurofibroma or vertebral metastases. Although T1–T2 discoradicular compressions with neurological deficit have been reported,[27–29] it should be kept in mind that if a neurological deficit of T1 is present, more severe disorders should always be excluded first (see see online chapter *Disorders of the thoracic spine and their treatment*).

Thoracic disc lesions compressing a nerve root do not usually resolve spontaneously, although there are a few reports of spontaneous regression at a lower thoracic level.[30,31] However, posterolateral thoracic disc protrusions which cause root pain remain reducible by manipulation, no matter how long they have existed. Where manipulation has failed or where neurological deficit is present, a sinuvertebral block should be given.

Compression of the spinal cord

The spinal cord is most vulnerable at the lower thoracic levels, between T9 and T12,[29,32] because the spinal canal is at its narrowest there and the vascularization is at its most critical.[33] It has been suggested that signs of cord compression do not always stem from pressure on the cord itself, but rather are the result of interference with the blood supply.[3–5]

Osteophytes narrowing the spinal canal are an extra contributing factor.[34] Previous injury to the thoracic spine can also play a role in the later development of cord compression, although this circumstance is rare.

History

The most commonly observed chronological sequence in cord compression is pain, followed by sensory disturbance, motor weakness and finally visceral dysfunction. All of these features may be present in any combination.

Pain

Initially almost all patients complain of pain. It is never particularly severe, often has a vague band-shaped distribution, and may sometimes disappear completely.[35–38] It is usually localized in the back, although it may radiate into the pelvis or groin and down the legs. Occasionally, patients complain of subumbilical pain.[1] The quality varies from a constant, dull and burning pain to – exceptionally – a lancinating, cramping and spasmodic pain.

Pins and needles

These are the most common symptom. They are usually felt in one or both feet, sometimes radiating into the legs, and are often provoked or increased by flexion of the neck.

Numbness

Later in the course, unilateral or bilateral numbness may set in and may be accompanied by a motor palsy. The numbness is more a diminution of normal sensation than a complete loss of sensitivity. It often starts at the big toe and is accompanied by a subjective sensation of coldness.[3]

Weakness

Because of interference with motor function, patients often complain of difficulty on walking, which sometimes presents as subjective weakness of the legs with unsteadiness or stiffness; it may, however, truly interfere with gait, making the patient stagger or even making walking impossible. It is important to note that the patient's subjective complaint of motor weakness is not always confirmable clinically.

Visceral symptoms

Very often visceral symptoms involving the urinary tract or bowel are also mentioned. Urinary symptoms may vary from difficulty in starting, to urgency, change in frequency, incontinence, incomplete emptying and even complete urinary retention. Occasionally, patients complain of impotence or of decreased sensation during intercourse.

Bowel problems are of the same nature as those related to the urinary tract, with constipation quite frequently present.

Functional examination

The functional examination normally confirms what is already expected from the history. The most characteristic signs are found on neurological examination; the articular signs are of secondary importance.

Some or all of the following neurological signs may be present:

- Disturbed coordination with spastic gait.
- Increased muscle tone, with the affected muscles not limited to one myotome.[39] Occasionally, weakness of the lower abdominal muscles can be demonstrated, when the umbilicus is seen to move as the patient attempts to sit up.[1] This is known as Beevor's sign.
- Weakness and/or atrophy of some lower limb muscles.
- Hyperreactive patellar or Achilles tendon reflexes with ankle clonus.
- Occasionally, absent tendon reflexes, particularly and inevitably when a flaccid type of paraplegia is present. The abdominal reflexes are often absent or diminished, most commonly in both lower quadrants. All these signs may be unilateral or bilateral.
- Positive Babinski's and Oppenheim's signs.
- Absence of the cremasteric reflex.
- Numbness.
- Limitation of straight leg raising, sometimes bilateral.[2]
- Occasionally, a Brown–Séquard syndrome in cord compression.[6] It is characterized by an ipsilateral flaccid segmental palsy, together with an ipsilateral spastic palsy below the lesion, with ipsilateral anaesthesia and loss of proprioception and loss of appreciation of the vibration of a tuning fork. Contralateral discrimination of pain sensation (analgesia) and thermoanaesthesia may be present and are both sited below the lesion.[40,41]

Although many of these tests are not included in the basic examination, they should be undertaken when the slightest suspicion of compression of the spinal cord exists.

Sometimes the neurological disturbances are found to extend proximal to the territory of the level of compression. This may be the outcome of interference with the blood supply of the anterior spinal artery rather than compression on the cord as such.[3–5]

Differential diagnosis

Differentiation from other compressive disorders of the spinal cord, such as neoplasm, infection, vertebral fracture and epidural haematoma, dissecting aneurysm and diseases of the cord (multiple sclerosis, Guillain–Barré syndrome and amyotrophic lateral sclerosis), is not always easy and requires further clinical reasoning and more technical investigations.

Technical investigations

The clinical description given above necessitates further imaging investigations. A plain radiograph sometimes shows

calcification in the disc that is at fault.[1,6,29,42] However, other authors, including Cyriax, consider calcification, together with the accompanying narrowing of the intervertebral joint space, to be non-specific.[43]

A myelogram usually indicates the level of the lesion with certainty, although special projections may be needed.[44]

Today, MRI is the imaging method of choice in the investigation of the thoracic spinal canal.[45] It provides a good-quality image over the entire length of the spine and can assess the morphology of the discs and cord. It is non-invasive, has comparable sensitivity to conventional myelography in visualizing lumbar nerve roots, and allows overall assessment of the spinal canal even in the presence of cerebrospinal fluid block.[46,47]

Treatment

The only treatment in cord compression is surgery.

Warning

Summary of warning signs: these are two-fold.

1. Presence of neurological signs.
   First exclude: neoplastic process
   infectious process
   fracture
   If negative: consider disc protrusion
2. Side flexion away from the painful side is the only painful and limited movement:
   No disc lesion is present but a tumour is most likely

Treatment

Almost all thoracic disc protrusions can be reduced by manipulation in 3–5 sessions. If manipulation does not succeed after, say, three sessions, the diagnosis should be reconsidered and, if a disc lesion is confirmed, traction can be tried. Traction can also be used for thoracic postural pain syndrome and for lateral recess stenosis in the thorax (see later). Other treatments, discussed later, include sinuvertebral blocks for persistent root pain or for root pain with neurological deficit, surgery, bed rest and prevention of recurrence.

Manipulation

Indications

All actual and symptomatic thoracic disc protrusions should be manipulated, in the absence of contraindications.

Unlike in the cervical and the lumbar spine, the type of onset is of less importance for treatment of the thoracic spine, since the great majority of thoracic disc protrusions are annular. Consequently, manipulations should be tried first. If these fail, and if the diagnosis of a disc lesion is confirmed, a nuclear protrusion should be considered. The time that has elapsed from the onset of pain can also be disregarded, because thoracic disc protrusions remain reducible regardless of their duration. Although T1 and T2 disc lesions will come to light in the cervical examination (rather than the thoracic examination), when a disc protrusion is present it should be treated using manipulative techniques designed for the thoracic spine.

Indications for and contraindications to manipulation are summarized in Box 27.1.

Box 27.1

Indications for and contraindications to manipulation

Indications

- All symptomatic disc displacements in the absence of contraindications

Contraindications

Relative

- Absence of actual symptomatic disc displacement
- Self-reducing disc lesion
- Monoradicular neurological deficit

Absolute

- Signs and/or symptoms of cord compression
- Patients with bleeding disorders
- Patients on anticoagulant treatment

Contraindications

Relative contraindications

Disc protrusion is not present

It is obvious that, in the absence of a disc protrusion, manipulation is inappropriate and could even do harm by displacing a disc fragment.

Self-reducing disc lesions

In self-reducing disc displacements, no manipulative attempt must be undertaken because reduction occurs on lying down. However, measures for prevention of recurrences must be taken. A series of infiltrations with sclerosant solution is then the treatment of choice.

Segmental neurological deficit

Neurological deficit indicates that the protrusion is too large to be reduced. Therefore no manipulative attempt is made if monoradicular numbness and/or motor palsy is found. Should this be present, the patient can be treated by an infiltration around the nerve root.

Central protrusion

This occurs mainly in young patients with a large thoracic kyphosis, is nuclear in origin and posterocentrally localized, and causes bilateral radiation of pain. The treatment of choice is sustained traction.

Absolute contraindications

Cord compression

Signs and symptoms of compression on the spinal cord, such as a positive Babinski's sign, weakness of the lower limbs or spastic paresis, are absolute contraindications to manipulation. Such patients should be operated on at once.

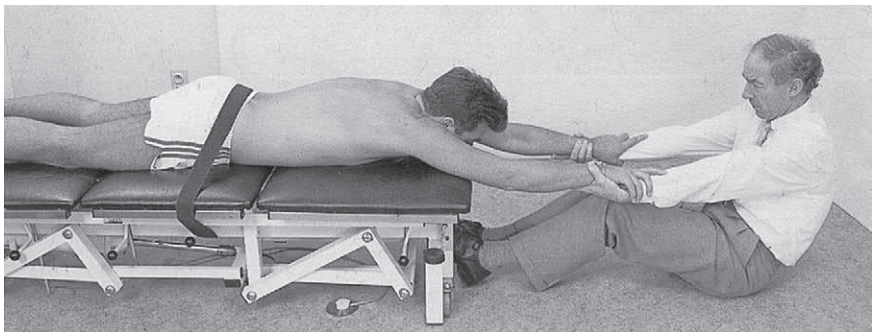


Fig 27.6 • Traction given by one assistant, using a belt.

Fig 27.7 • Traction is applied via the head if the lesion is situated above T6.

Sometimes patients with cord compression are encountered who complain of pins and needles only in the legs and feet, increased or provoked by flexion of the neck. On clinical examination, further evidence of cord compression is not found. The implication is that the protrusion interferes minimally with the cord; nevertheless, manipulations could easily increase the problem and are absolutely contraindicated. This does not apply to sustained traction, which can be tried in cases in which the clinical features have not been present for too long.

Bleeding disorders

Patients who suffer from bleeding disorders, either inherited or acquired, or who are on anticoagulants should never be subject to manipulation because this could provoke an intraspinal haematoma. If a patient on anticoagulants suffers from a disc lesion, the anticoagulants must be stopped over a period of days (should the condition for which treatment is prescribed permit) until blood coagulation has returned to normal. Manipulation can then be performed safely.

Techniques

Thoracic manipulations are always performed under strong traction. Three main types of procedure are considered: extension techniques in which extension is always present, sometimes together with a rotation; rotation techniques, in which rotation is the only element and extension is not incorporated; a third type, the 'high thoracic technique', used only in upper thoracic disc lesions.

All extension manipulations of the thoracic spine are specific, i.e. they are performed only at the two vertebrae on either side of the disc protrusion.

Principle of strong traction

No one can foresee with absolute certainty the direction in which the protruded fragment will move during a manipulation. Theoretically, it could be displaced further towards the spinal cord. To avoid this and for a number of other beneficial effects (see p. 256), traction is always incorporated in thoracic manipulation.

Traction is normally provided by one assistant who sits beyond the patient's head and takes hold of either the head or the hands. Stabilization of the pelvis is provided either by a second assistant sitting at the patient's legs and holding the ankles or by a fixation belt around the pelvis (Fig. 27.6). Whether traction is given via the head or the arms depends on the level of the lesion and the patient's comfort. As traction on the arms opens the intervertebral joints only from T6 downwards, traction must be given via the head in disc protrusions above T6 (Fig. 27.7).

If traction via the arms provokes pain anywhere in the upper limb, it is replaced by traction on the head.

> Disc protrusion above T6: traction via the head.
> Disc protrusion below T6: traction via the hands.

Traction via the head is provided by putting one hand under the patient's chin and the other below the occiput, with the head and neck always kept in the neutral position (see Fig. 27.7). Both hands pull equally hard.

Traction via the arms is achieved by the patient and assistant taking each other's wrists in their hands.

Before any manipulation is done the assistant must first create traction because very often slack in a belt must be taken up and adjustment may be needed.

The traction provided should always be given with maximum force in order to open the joint space as far as possible. Because maximum traction can only be maintained for a few seconds, it is only given after the manipulator has taken up all the slack of the manœuvre to be performed. The different steps of the manipulation, including traction, are coordinated by the manipulator.

Although exerted with as much strength as possible, traction is not a manipulation in itself. In other words, the assistant does not add a jerk to the pull during the manipulation but only maintains the pull maximally until the manipulation is complete.

Amount of force

Each manipulation must be performed with a reasonable amount of force. This depends on both the manipulator's and patient's physical size and the level of the protrusion. If a large examiner is dealing with a lightly built patient, the whole of the former's strength will not be used, but if a lightly built manipulator has to manipulate a heavier person, the maximum effort may be needed to achieve good results.

Because the upper half of the thorax is the most rigid, disc protrusions at this level require more forceful manipulation than for the lower half. Therefore a lesion below T6 can be treated with a less vigorous impulse.

All manipulation must be performed using the body. The most common mistake occurs when the manipulator uses his arms rather than his body. This leads to a loss of the slack already taken up and results in increased amplitude of manipulation, which may be dangerous because the manipulator has no control over the force used.

Extension techniques in prone position

Unless the patient is 60 years old or more, extension techniques are used initially. All extension manipulations are specific in that they are performed directly on the vertebrae between which the lesion lies.

Identifying the level of the lesion

The two most painful consecutive spinous processes are identified by extension pressure. The protrusion normally lies between their corresponding vertebrae. If only one spinous process is found to be tender, the disc at fault is usually the one lying just below. This being so, the extension manipulations are undertaken between the vertebra with the tender spinous process and the one immediately below.

Sometimes all spinous processes are equally tender on extension pressure. It can then be useful to take two consecutive spinous processes between the fingertips one by one and to try to rotate them in the opposite direction towards each other. This is done at all levels and the two that prove most painful are indicative of the level of the lesion.

From this point on, in describing the different extension techniques, the phrase 'two most tender spinous processes' is used. It is important to understand that this always means the two most tender on extension pressure and not just on palpation for local tenderness.

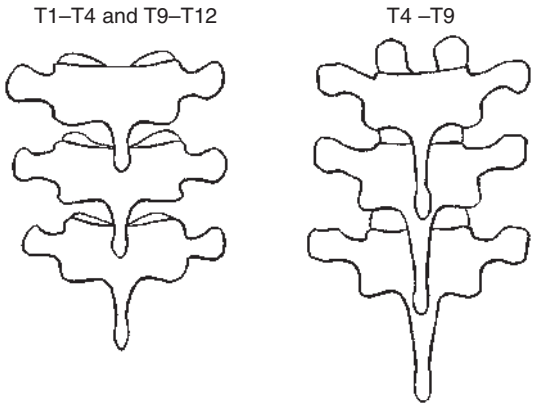


Fig 27.8 • Relationship between the level of the spinous processes and their corresponding transverse processes.

Some extension techniques are executed on the transverse processes. To be able to perform these manipulations at the right level, it is essential to have a good knowledge of the relationship between the spinous processes and their corresponding transverse processes, because this varies with the level (Fig. 27.8).

- *T1–T4 and T9–T12:* there is a difference of one level between the consecutive vertebrae, i.e. the transverse process of one vertebra lies level with the spinous process of the vertebra above. For example, the transverse process of T3 lies level with the spinous process of T2.
- *T4–T9:* there is a difference of 1½ levels. The transverse process lies level with the interspinal line between the spinous processes of the first and the second vertebrae above (Fig. 27.9). The transverse process of T8, for example, lies between the spinous processes of T6–T7, that of T9 between T7–T8.

Direction of rotation

This is important in manipulations that include an element of rotation. Manipulation is first undertaken in the direction of the less painful rotation. If this is only partly or completely unhelpful or makes the patient worse, rotation in the other direction follows.

The direction of the rotation is always defined by the direction in which the anterior part of the upper vertebra rotates. So when the upper vertebra rotates to the left, the movement is defined as a left rotation (Fig. 27.10).

Technique I: central pressure (Fig. 27.11)

The patient lies prone on a low couch. The two most tender spinous processes are marked. The examiner stands to the side, facing the patient, and places one hand, reinforced by the other, on the lower of the two processes. Because the thoracic spinous processes cover each other like tiles on a roof, it is logical to place the hands on the lower one in order to achieve as much movement as possible between the vertebrae. If the hand is placed on the upper vertebra, the movement is assured by the bony contact between the spinous processes; when the hand is placed on the lower vertebra, it is pushed away from the upper vertebra, resulting in increased range of motion.

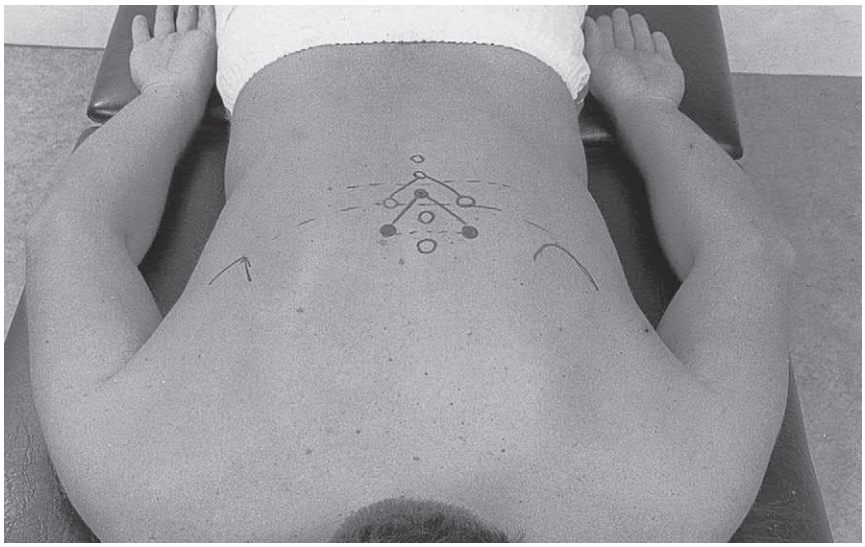


Fig 27.9 • The 1½-level difference between the spinous and transverse processes, which is present in T4–T9.

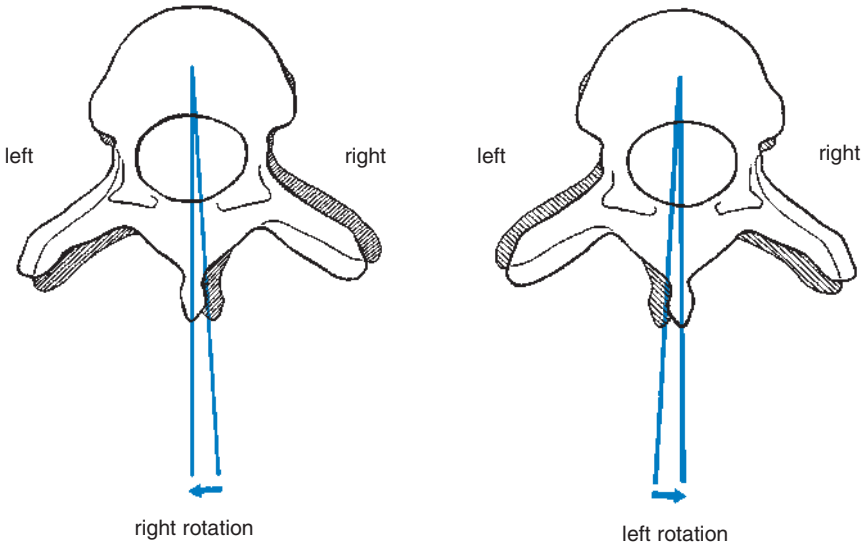


Fig 27.10 • Direction of rotation.

The hand makes contact with the spinous process via the fifth metacarpal bone and, to make the manœuvre less painful, the hand is placed slightly oblique so that the hypothenar muscles lie between the bones of the hand and the patient's spine.

The assistants adopt the positions already described.

The manipulator leans over the patient, the shoulders vertically above the hands, elbows straight, and feet a distance away from or close to the couch, depending on the effort to be used. All the slack is taken up by use of the body weight and is maintained until the final manipulative thrust is given. The patient is now asked to relax fully by taking a deep breath, followed by a maximal expiration. The moment the patient starts to breathe out, the manipulator tells the assistants to pull. Waiting until the end of expiration and under maximal traction, the final extension thrust is given by the manipulator's body, transmitted to the patient via the arm and hands.

Technique II: unilateral pressure (Fig. 27.12)

This manipulation is performed unilaterally on the transverse process.

The patient lies prone and the two most painful spinous processes and their corresponding transverse processes are marked. Vertical pressure is exerted on the transverse process of the lower of the two vertebrae.

The manipulator stands on the contralateral side of the patient: on the left for a right rotation. One hand, reinforced by the other, is placed with the pisiform bone on the transverse process. To make good bony contact, the paravertebral muscles are first moved to the side. This is achieved by pushing the muscles away from the vertebra with the base of the lower hand. One assistant holds the patient's ankles, while the other grasps the head or hands. With the elbows straight, the manipulator leans over the patient and brings the body weight perpendicular over the spine, taking up all the slack. The patient

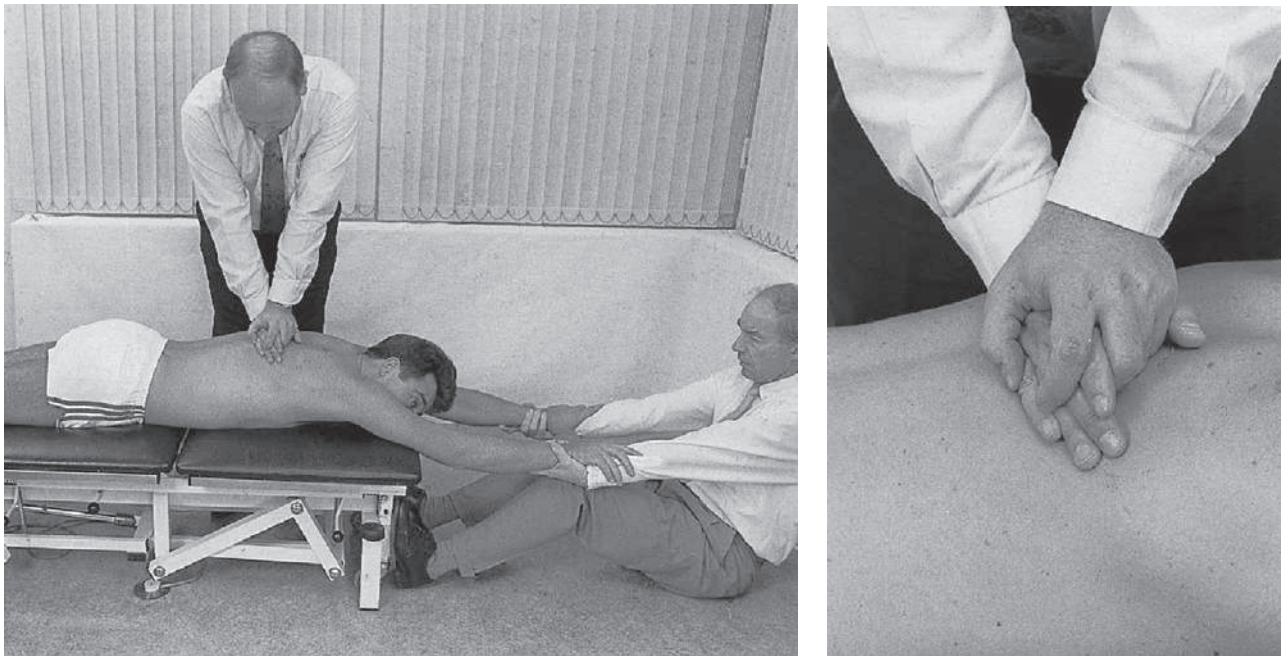


Fig 27.11 • Central pressure.

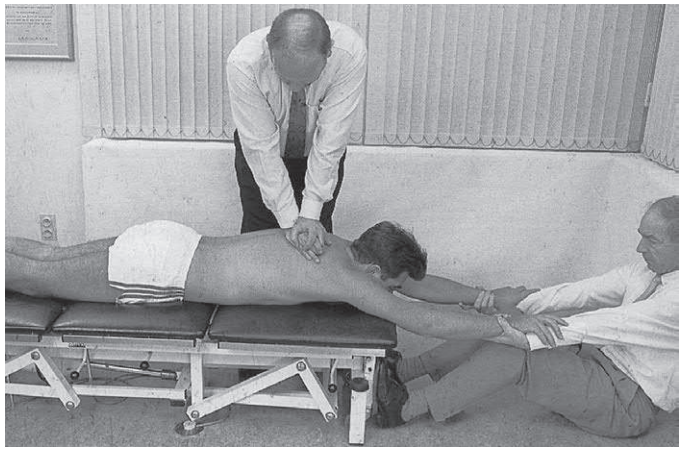
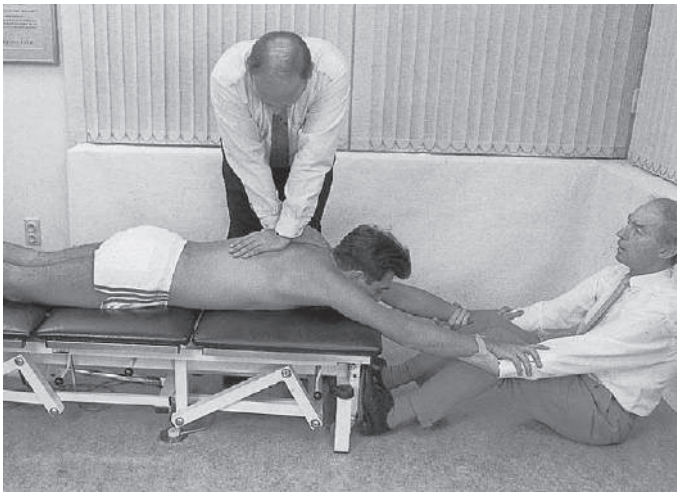


Fig 27.12 • Unilateral pressure.

is now asked to relax, take a deep breath in and then expire fully. The assistants are told to pull. After a couple of seconds of traction, the manipulator gives the final jerk vertically downwards. This causes a rotation and an extension movement.

Technique III: using crossed hands (Fig. 27.13)
Although a strong rotation element is present in this manipulation, it is still regarded as an extension technique.

The patient lies prone on a low couch. The manipulator can choose to stand on either side of the patient. The two most tender spinous processes and their corresponding transverse processes are marked. The rotation found during the clinical examination to be the less painful is performed first. If this is without benefit, the opposite rotation is performed. One hand is put on the transverse process of one vertebra, the other hand on the transverse process of the second vertebra on the opposite side. Care should be taken to cross the hands fully. The



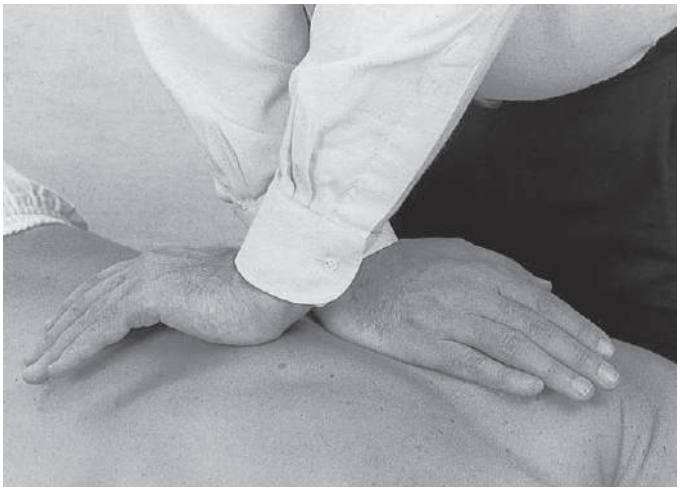


Fig 27.13 • Using crossed hands.

hand closer to the manipulator is placed on the transverse process via the pisiform bone. The other hand is in contact via the base of the trapezium–first metacarpal joint. Good bony contact is essential and is achieved by first pushing the paravertebral muscles away from the spine. The trapezium–first metacarpal joint of the one hand and the pisiform of the other are first placed just to the side of the spinous process. Both hands are now twisted into ulnar deviation, so pushing the muscles further to the side, and are then brought back closer to the midline but still remain on the transverse processes.

With the elbows straight, the manipulator leans over the patient and brings both shoulders vertically above the hands. As a result, all the slack is taken up. The assistants provide traction and the final jerk from the manipulator's body follows.

Extension technique in supine position

The patient lies supine just near the edge of the couch and places both hands behind the neck, the fingers covering the upper thoracic spinal processes. The elbows are placed well forwards and close together. The manipulator stands on the right-hand side facing the patient. By grasping the patient's left shoulder in the right hand and both elbows on the left hand (Fig. 27.14a), the manipulator flexes the patient's neck and trunk and rolls the upper body inwards (Fig. 27.14b).

Then a fist is made with the middle, ring and little fingers of the right hand – thumb and index finger are left out. This fist is now brought into firm contact with the lower vertebra of the segment being manipulated – the thenar eminence against the left and the middle phalanx of the flexed middle finger against the right transverse process. In this way the spinous process of the lower vertebra lies in the groove between these two eminences (Fig. 27.14c).

Now the patient is lowered back again until the manipulator's hand is wedged between the patient and the couch. In order to achieve full control over the movement, the patient's elbows are firmly held against the manipulator's sternum (Fig. 27.14d). Leaning well over the patient and using the weight of the trunk, the manipulator obtains considerable separation at the intervertebral joint. At the moment when the limit of tissue tension is felt (and the patient relaxes as fully as possible), the manipulator pushes the body forwards to apply a certain amount of overpressure. At that moment a 'click' or 'snap' is nearly always heard and felt, and the result of the manipulation is then assessed.

Variations

- If the patient is not able to reach the upper thoracic spine with the hands because of stiffness or a painful shoulder, the technique may be varied by crossing the arms fully over the chest. The right hand clasps the left shoulder, the left hand the right shoulder.
- Some degree of rotation can be added if the thenar eminence and the middle phalanx of the middle finger are positioned on two consecutive transverse processes and not on those of the same vertebra.

Techniques for upper thoracic disc lesions

Rotation techniques for upper thoracic disc lesions (Fig. 27.15)

This manœuvre is suitable only for upper thoracic disc lesions. It is best performed on a couch of medium height. The less painful rotation is done first.

The patient lies prone. For a right rotation, the manipulator stands facing the patient on the left-hand side. The right arm of the patient lies fully medially rotated on his back, the elbow flexed. One assistant exerts traction via the patient's head and also rotates it slightly to the right. In this way, the cervical spine is kept in neutral position during the whole manœuvre. The other assistant holds both of the patient's feet.

The manipulator hooks his left arm underneath the patient's furthest shoulder so that the proximal part of the lower arm can lift the patient's shoulder off the couch. The right hand is placed on the dorsal aspect of the ribs as high and as close to the thoracic spine as possible. The fingers of both hands are now clasped together. Both hands rest on the ribs, just lateral to the spine and no further down than mid-thorax. The manipulator now bends the trunk to the right, meanwhile pulling the patient's right shoulder off the couch via the left lower arm. The rest of the spine is held down on the couch by both hands pressing on the ribs. The assistants are asked to apply traction and the manipulator suddenly increases the side flexion. With this final movement, extension and rotation at the upper thoracic spine are increased. The rigidity of the upper thorax allows movement over only a small range.

Longitudinal traction for upper thoracic disc lesions (Fig. 27.16)

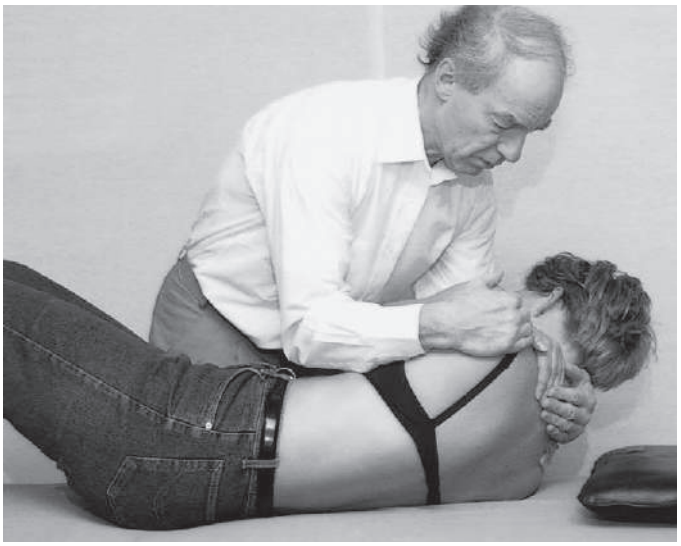
The patient stands or sits with the back to the manipulator. A sitting position is preferred if the patient is taller or heavier in weight than the manipulator. The patient is then asked to grasp the hands behind the neck, allowing the elbows to drop forwards.

The manipulator threads the arms in front of the patient's axillae and grasps both wrists. The patient is then asked to bend the head, so allowing the elbows to drop further forwards and to lean backwards against the chest of the manipulator. The latter performs a preliminary lift and at the moment the patient allows the trunk to sag, suddenly extends the knees. This results in a longitudinal traction and separation particular to the upper thoracic joints. A 'click' or 'snap' is felt and the result then assessed.

Some degree of extension may be added to this technique. The manipulator achieves this by adopting a slightly modified starting position: turning the trunk slightly to one side allows positioning of the lower ribs against the spine at the level at which manipulation is required.

Warning

Beware of forcefully flexing the neck of the patient during the upward thrust.



(a)

(d)

(b)

(e)

(c)

Fig 27.14 • Extension technique in supine position (a, b, c). Variation for upper lumbar level (d). Position for thenar and middle finger (e).

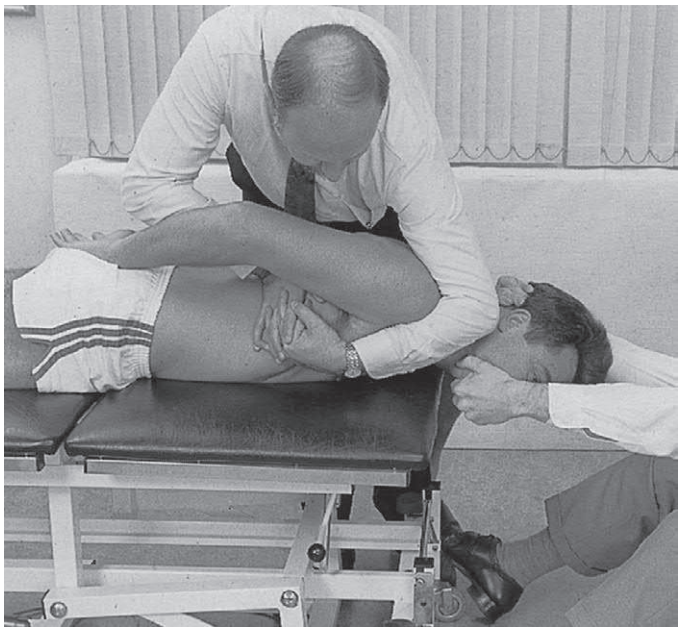


Fig 27.15 • Technique for upper thoracic disc lesions.

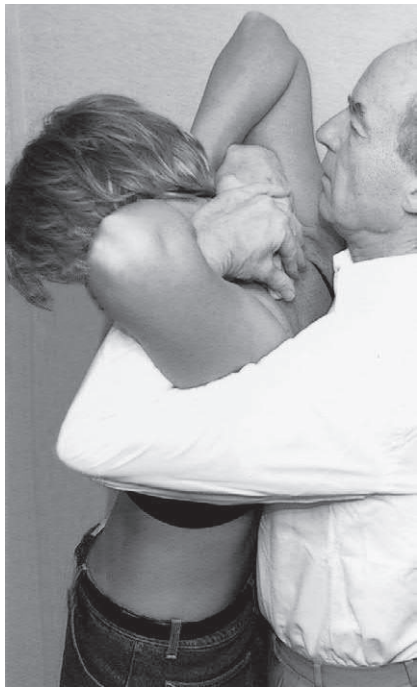


Fig 27.16 • Longitudinal traction for upper thoracic disc lesions.

Choice of manœuvre

The different techniques are used in a specific sequence, depending on the patient's age, the level of the protrusion and the immediate result of the preceding manipulation.

After each manœuvre the patient is re-examined and, if improved, the same manœuvre is repeated several times with increasing strength until the patient has completely recovered or until no further improvement is obtained. In the latter event, another type of manipulation follows. If the patient does not improve or even becomes worse, the same manipulation is not carried out a second time but is immediately replaced by another. In such cases, the same type of technique is performed, but in the opposite direction. If this manipulation also aggravates the lesion, manipulation must be stopped and the diagnosis reconsidered.

Improvement is indicated by pain, which is perceived over a small area and/or which becomes less severe. Another important sign is centralization of the pain: pain that first spreads far distally or laterally, but becomes more centrally localized, is regarded as an improvement.[48,49] Manipulations are stopped as soon as the pain has disappeared and the clinical examination becomes negative. A total number of 5 (elderly) to 10 (younger patients) manipulations are executed during one session. These can be repeated daily, although for the elderly it is advisable to allow 3 or 4 days in between. It may take 3–5 sessions before the patient has fully recovered. A certain amount of after-pain is sometimes present. If there is a recurrence, the patient should return as soon as possible to be manipulated again.

Usually the extension techniques are used first. In patients over 60 years of age, however, extension techniques may fracture a rib and are therefore never used. The same applies if the extension pressure on the spinous processes performed at the end of the clinical examination is very painful. In such a case, oscillatory techniques should then be used (see below).

Warning

Extension techniques should never be used in patients over 60 years of age or when there is excessive pain on extension pressure.

Failure of manipulative reduction

Ninety-five percent of thoracic disc lesions are reduced in 3–5 sessions. If, after 3–5 manipulative sessions, relief of symptoms is not obtained, it should be accepted that either the diagnosis is wrong or the disc lesion is not suitable for manipulation. The latter may be the result of too large a protrusion, as shown by the presence of neurological deficit, or may occur when a nuclear disc lesion is present (Box 27.2).

Oscillatory techniques

Some cases respond better to oscillations. These consist of gentle high-frequency mobilizations at 2–3 vibrations per second. Oscillations should be given for 10–15 minutes daily and are performed as either central or unilateral pressure to the thoracic spine.[50]

Indications

There are three groups of indications:

- Patients who present with a great deal of discomfort but with very minor articular signs on clinical examination.
- Patients with acute thoracic lumbago who are in such pain that they cannot tolerate normal manipulations. Oscillatory techniques can be used until the pain is

Box 27.2

Unsuccessful manipulations

Wrong diagnosis: no disc protrusion?

- Facet joint?
- Tumour?
- Muscular lesion?
- Osseous lesion?
- Ligamentous lesion?
- Visceral disorder?

Disc protrusion confirmed

- Check for neurological deficit
  1. If neurological deficit is present:
     - Stop manipulation
     - Give sinuvertebral block
  2. If no neurological deficit is present, consider nuclear protrusion:
     - Stop manipulation
     - Try traction

reduced to a level at which normal manipulations can be started.

- Patients who cannot tolerate the extension or rotation techniques.

Sustained traction

In some patients with thoracic disc protrusion, reduction by means of traction may be needed. However, traction may be technically impossible in patients suffering from orthopnoea, asthma or hiatus hernia or who have recently undergone thoracic or abdominal surgery (see Ch. 40).[51]

Indications

- *Central protrusions*: it can be dangerous to manipulate very central protrusions, as there is a possibility of causing compression of the spinal cord. Central protrusions are present mainly in those with a marked thoracic kyphosis, either postural or after a wedge fracture. These patients complain of central pain radiating to both sides.
- *Failure of manipulation*: patients suffering from a disc lesion in whom manipulative attempts have failed or who have been made worse, should receive traction unless nerve root compression with neurological deficit is present. There is usually a history of gradual onset, although this is not pathognomonic in the thoracic spine for a nuclear protrusion.
- *Symptoms of cord compression in the absence of signs*: patients suffering from a thoracic disc protrusion with pins and needles in both feet can undergo cautious traction if no other symptoms or signs of cord compression are present.
- *Thoracic postural pain syndrome*: these patients can be helped by daily sustained traction with increasing traction-free intervals (see online chapter *Disorders of the thoracic spine and their treatment*).
- *Disc lesions at a very kyphotic thoracic joint.*
- *Disc lesions adjacent to a wedge fracture of a vertebral body.*
- *Anterior and lateral erosion* (see online chapter *Disorders of the thoracic spine and their treatment*).
- *Lateral recess stenosis* (see online chapter *Disorders of the thoracic spine and their treatment*).

Technique

Traction is given daily over 30–45 minutes. Thoracic discs above T9 are treated with cervical traction. For these, the rules of cervical traction should be observed (see Ch. 11).

For thoracic protrusions below T9, traction is given using the lumbar technique (see Ch. 40). An intensity of 35 kg (small person) to 70 kg (heavy, well-built person) is used. Obviously, the thoracic belt should be placed cranial to the level of the lesion. The initial traction sessions are used as the manipulator's guide for positioning the patient and for the strength to be used.

Results

Improvement normally begins after 6–10 sessions, whereas full cure may require up to 20 sessions.

Sinuvertebral nerve block

In persistant root pain or in pain associated with neurological deficit, a sinuvertebral nerve block is the treatment of choice. This should only be performed under radiological control.

Bed rest

If both manipulation and traction have failed and no indication for surgical intervention is present, the only remaining alternative is prolonged rest in bed in the hope that removing the influence of gravity on the spine may allow the protrusion to reduce spontaneously.

Surgery: removal of protruded discs

The main indication is early cord compression by a progressive disc lesion.

Laminectomies done before 1960 were dangerous interventions with disappointing results.[52,53] It was found that almost all serious complications occurred among those having midline protrusions at levels T10–T11.[53] This is probably the outcome of the shape of the spinal canal, which is quite narrow at this level. The posterior approach used required some displacement of the cord in a very confined space, with consequent considerable risk to the blood supply.

Since 1960 a transthoracic lateral approach has been used and in recent years more progress has been made with new thoracoscopic microsurgical techniques.[54,55] They seem to give better results and fewer complications.[56] Recently,

percutaneous laser disc decompression – intervertebral discs are treated by reduction of intradiscal pressure through laser energy – has also been promoted as a valuable method in treating recalcitrant thoracic disc lesions.[57]

Prevention of recurrence

Postural prophylaxis

Reduction of a thoracic disc protrusion normally does not cause much in the way of difficulties but recurrence is not uncommon and stability is often difficult to achieve. This is obviously because of the normal thoracic kyphosis which results in a posteriorly directed force acting on the disc.

All the rules on prophylaxis for the lumbar spine are also applicable here (see Ch. 40). However, it is much more difficult and sometimes even impossible to put them into practice. For example, it is impossible to obtain the equivalent of lumbar lordosis because even the most flexible person cannot get beyond a straight line in full extension. The most hazardous movements are those that include combined flexion and rotation elements. Lifting a weight at the same time makes them even more dangerous. Therefore patients should avoid rotating their trunk but must turn their body around using their legs and should bend their knees to lift.

Sitting for a prolonged period in a kyphotic posture must also be avoided.

Ligamentous sclerosis

In those patients in whom the risk of recurrence is high (thoracic hyperkyphosis), or when recurrence is frequent, local reinforcement is needed for the supraspinal and interspinal ligaments and for the capsules of the facet joints of the vertebrae between which the protrusion lies. This can be achieved by infiltrating a sclerosant solution into these structures, which leads to proliferation of fibroblasts and formation of new collagen.[58] The final result is increased stability of the disc fragment because the two vertebrae become less mobile. Before this procedure is undertaken, full reduction must be achieved.

Occasionally, the same technique can be employed for arthritis of the facet joint or for ligamentous lesions. In these cases triamcinolone is used.

The sclerosant infiltrations are given at weekly intervals and the greatest effect is normally obtained after 3 weeks; exceptionally it may take up to 2 months. Usually, one full treatment suffices; if there is further recurrence, it can be repeated.

Technique: infiltration of the interspinal and supraspinal ligaments (Fig. 27.17)

A 5 mL syringe is filled with 3 mL of sclerosant solution and 1 mL of lidocaine 2%, and a 4 cm needle is fitted to it; 2 mL of this solution are used for the interspinal and supraspinal ligaments, the rest for the facet joints.

The relevant spinous processes are marked and the needle is inserted halfway in between them at the midline. At about 1 cm deep it is changed into an almost horizontal position, pointing cranially and aiming at the inferior part of the upper spinous process. Once it hits bone, 0.5 mL of the solution is infiltrated dropwise into the supraspinal ligament and the same

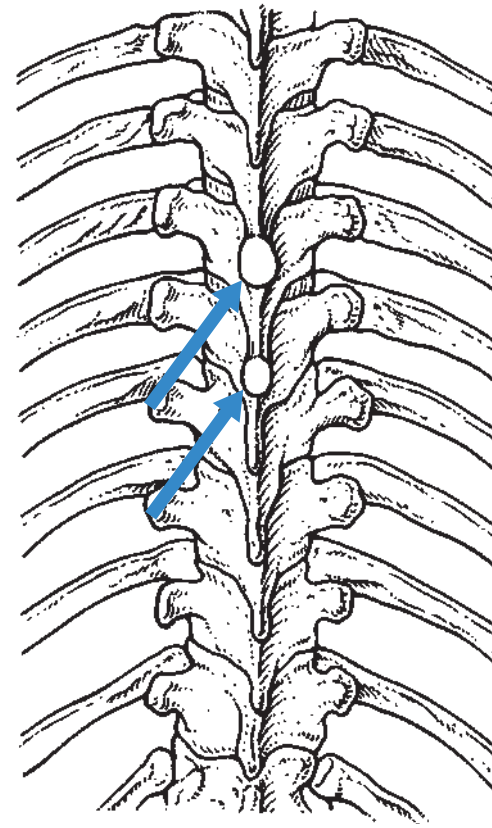


Fig 27.17 • Infiltration of the interspinal and supraspinal ligaments.

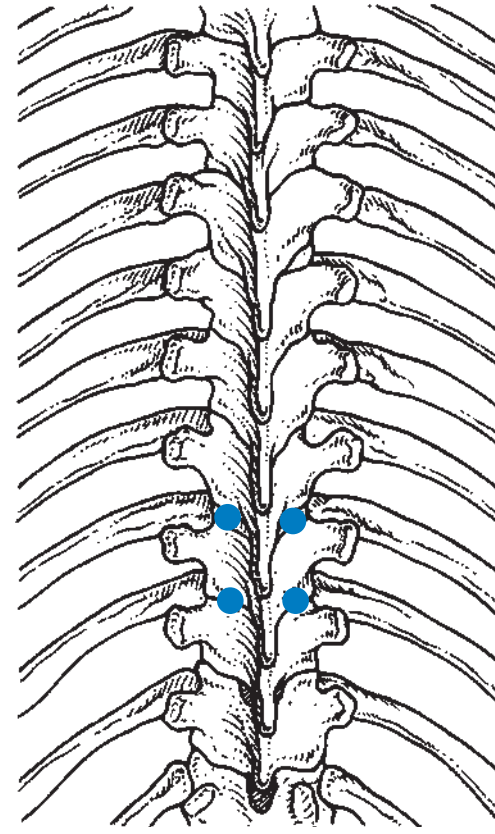


Fig 27.18 • Infiltration of the capsule of the facet joint.

quantity a little deeper down in the interspinal ligament. Infiltration is only given with the tip of the needle in bony contact. The needle is then half-withdrawn and brought into a more vertical position towards the insertions of the ligaments on the upper aspect of the lower spinous process, which are infiltrated in the same way. When the supraspinal ligament is infiltrated, it offers tough resistance, whereas infiltration of the interspinal ligament is much easier.

Technique: infiltration of the facet joint (Fig. 27.18)
A 4 cm needle is used. The patient remains prone and the transverse processes corresponding to the most painful consecutive spinous processes are marked. The needle is inserted at about 1.5 cm from the midline halfway in between the two transverse processes. It is thrust in vertically downwards until it touches the articular process at about 3 cm depth. Capsular resistance to the needle tip is sought by minor repositioning of the needle. The infiltration is given mainly into the capsule with the tip of the needle always in bony contact and considerable resistance is usually felt during the whole infiltration. This is done at both sides using 1 mL of the solution at each side.

The interspinous and supraspinous ligaments and the facet joints are infiltrated at the same time, repeated three times at weekly intervals.

Access the complete reference list online at **www.orthopaedicmedicineonline.com**

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28

Disorders of the thoracic spine: non-disc lesions

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Signs 404
Pathology: see the online content

Introduction

Non-disc lesions of the thoracic spine comprise the following disorders:

- Tumours
- Extradural haematoma
- Spinal cord herniation
- Spinal canal stenosis
- Vertebral fracture
- Chest deformities
- Fracture of a transverse process
- Spinal infections
- Lateral recess stenosis
- Localized lesion of costovertebral and costotransverse joints
- Localized lesion of a facet joint
- Paget's disease.

Very often, the signs and symptoms of the non-discal lesions of the thoracic spine resemble those of discodural disorders.

Some of the lesions mentioned are serious and even life-threatening. Therefore examiners must be on their guard and should immediately request further investigation whenever there is doubt about the exact diagnosis.

Warning symptoms and signs that warrant the examiner's attention are listed here and are summarized in Box 28.1.

Warning symptoms and signs

Symptoms

Progressively increasing pain

In disc lesions the pain may increase over a short period or come and go. In expanding lesions the pain tends to increase continuously. This may occur quickly over days (extradural haematoma), weeks to months (malignant diseases) or even longer periods (benign tumours).

Expanding pain

Pain that not only increases in intensity but also expands to occupy a larger area is very likely to be caused by an expanding lesion, most commonly a malignant disease or an extradural haematoma.

Continuous pain, not changed by posture or activity

Although this symptom is less alarming than the previous ones, a mechanical disorder is unlikely in the absence of variation of symptoms on movement. Nevertheless, the situation in the thoracic spine is slightly different from the rest of the spine because its rigidity sometimes results in a lack of change in pain with activity. Pain that is worse at night is seldom caused by a disc lesion, and is more suggestive of a rheumatic disorder, infection or trauma.

Increasing postoperative thoracic backache

Surgery correctly indicated and performed, but which is still followed by continuous pain, demands a fresh diagnostic evaluation. A clear relationship between what is found on further investigations and the patient's symptoms must be established. Local infection and a tumour must be excluded.

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Box 28.1

Summary of warning signs

History

- Progressive increasing pain
- Expanding pain
- Continuous pain, not changed by posture or activity
- Increasing postoperative thoracic backache
- First symptoms and signs in people over 50 years of age
- Central pain radiating bilaterally and spreading anteriorly

Examination

- Gross limitation of both side flexions and rotations
- Gross limitation of extension
- Pain and limitation of side flexion away from the painful side as the only positive finding
- Flexion with a rigid thoracic segment
- Presence of one of following neurological signs:
  - Signs of involvement of multiple nerve roots
  - Band-shaped area of numbness
  - All signs of cord compression
  - Muscle spasm
  - Local paravertebral mass

First symptoms and signs in the thoracic area in patients over 50 years of age

Because both intra- and extraspinal tumours occur mainly over the age of 50, symptoms that first appear beyond middle age arouse suspicion. Moreover, disc lesions become less frequent with increasing age because of increasing stiffness of the spine.

Central pain radiating bilaterally around the thorax and spreading anteriorly towards the epigastrium

Although a posterocentral disc protrusion may provoke this type of pain, a tumour or an extradural haematoma should always be excluded.[1]

Signs

The following may draw attention to non-discal lesions.

Gross limitation of both side flexions and rotations

In older patients it is normal for these movements to be diminished in range, although the limitation should not be major. In younger patients gross limitation is never normal; if present, it usually points to a non-mechanical disorder, such as ankylosing spondylitis or a tumour.

Gross limitation of extension

In an ordinary disc lesion it is usually anteflexion or one of the rotations that is most painful and limited. If active extension is severely limited and painful, attention is drawn to the possibility of a fracture of a vertebral body, an infection or a tumour. There is only one type of thoracic disc lesion in which severe limitation of active extension can occur: acute thoracic lumbago. In acute thoracic lumbago, other movements are, of course, also limited and/or painful.

Pain and limitation of side flexion away from the painful side as the only positive finding

This pattern suggests that a lesion is being stretched to such an extent that the pain becomes so severe such that the movement is arrested involuntarily. This may indicate a neoplasm in the lung or abdomen, or a thoracic neurofibroma.

Flexion with a rigid thoracic segment

Absence of movement at the thoracic level indicates a disorder other than a disc lesion. Ankylosing spondylitis is a possibility, as are advanced osteoarthrosis, fracture, tumour or a vertebral infection.

Neurological signs

The presence of one of the following neurological signs should always put examiners on their guard.

- Signs of involvement of multiple nerve roots
- A band-shaped area of numbness related to one or more dermatomes
- All signs of spinal cord involvement[2–4] (see p. 390).

A large space-occupying process, or a large disc protrusion which cannot and may not be manipulated, is very likely and a myelogram or magnetic resonance imaging (MRI) should be ordered immediately.

Muscle spasm

If one of the passive movements is stopped by an involuntary muscular contraction, a serious disorder should be suspected.

Local paravertebral mass

A local paravertebral mass may be caused by extraspinal invasion of a tumour.[5]

Access the pathology of the non-mechanical disorders of the thoracic spine and the complete reference list online at **www.orthopaedicmedicineonline.com**

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Ankylosing spondylitis of the thorax

29

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About 0.1% of the total population has ankylosing spondylitis (AS). It affects mainly the attachment of ligaments to the axial skeleton. Solitary involvement of the thoracic spine and cage is seldom encountered because this is normally part of a generalized ankylosing spondylitis. Only 2–5% of all patients have chest pain as an initial symptom.[1,2]

The condition usually starts in the sacroiliac joints and extends upwards to the spine, often to the thoracolumbar junction first, and then later to the lumbar, thoracic and cervical spines. Although involvement of the sacroiliac joints may remain silent, spinal localization of AS without sacroiliitis is very rare.[3]

Clinical findings

Ankylosing spondylitis of the thoracic spine

Patients complain of a chronic stiff back, especially early in the morning and easing with activity. Periods of worsening pain and stiffness come on spontaneously and are not provoked by activity or exercise. During the pain-free periods, the patient is effectively normal. Only slight radiation to the sides is present. Because several levels are usually involved and little referred pain is present, the pain is distributed in the vertical axis. All these are important differences from disc lesions, in which pain is usually referred horizontally, is worse during the daytime and is brought on by activity.

The findings on inspection depend on the duration of the disorder. Initially, a rigid lumbar segment is present, which later becomes flat, together with a slight accentuation of the thoracic kyphosis. This develops further to a thoracic hyperkyphosis with hyperextension of the upper cervical spine and flexion of the hip.[4] Chest expansion may be diminished.

On functional examination, a clear capsular pattern is present with an equal amount of pain and limitation of both side flexions and rotations, more pain and limitation on extension, and only slight discomfort on flexion. The end-feel on rotation and extension is typically hard. The range of rotation has a significant negative correlation with the duration of the disease.[5] Pain provoked by pressure on the spinous processes is usually much more severe than in an ordinary disc lesion.[6] At a later stage, the vertebrae are much more vulnerable to fractures, because of loss of normal capsular and ligamentous elasticity.

The capsular pattern with hard end-feel, chronic prolonged morning stiffness and pain with periods of spontaneous exacerbations, together with other localizations of the same

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disorder, all in young patients (mainly 18–30 years), strongly indicates AS.

A radiographic examination should be done at once and must always include the sacroiliac joints.

Ankylosing spondylitis of the anterior longitudinal ligament

Involvement of the anterior longitudinal ligament gives rise to pain felt only at the sternum or in the epigastrium, without central dorsal pain. The pain has the same characteristics as in other localizations of AS at the thoracic spine.

Ankylosing spondylitis of the thoracic facet joints

The apophyseal joints are usually involved, together with the intervertebral joints. The former are mainly responsible for bilateral paravertebral pain. AS may lead to total fusion of the facet joints.

Ankylosing spondylitis of the costovertebral joints

Costovertebral and costotransverse joints are often affected in AS. Computed tomography (CT) scans usually show the classic lesions: erosions, sclerosis, joint widening and bridging.[7] It is accepted that these changes provide the anatomical basis for the understanding of the thoracic pain in AS patients.[8] Although the pain is usually rather dull, it may sometimes be severe, even mimicking renal colic.[9] Chest expansion is diminished. Therefore chest expansion should always be assessed when AS is suspected. It is measured level with the nipples. The normal difference between inspiration and expiration is at least 7 cm; less than 4 cm is regarded as abnormal.[10] Surprisingly enough, reduced chest expansion seldom interferes with normal lung and heart function, because of normal diaphragmatic mobility.[11] If it does, chronic cor pulmonale is to be expected, with shortness of breath. The latter may also be the result of upper lobe fibrosis from involvement of the lung by AS.

Ankylosing spondylitis of the manubriosternal joint

This joint is affected in 50% of all cases.[12] Forced inspiration may be painful. Palpation reveals a precise area of local tenderness together with swelling of the ligaments.

Further investigations

Laboratory tests

The erythrocyte sedimentation rate may be elevated in active disease. A mild anaemia may be present. HLA-B27, although not diagnostic as such, is present in over 90% of cases, whereas in a normal population it is only found in 8%.[6]

Radiography

Sacroiliac joint

Early confirmation of AS is based mainly on radiography of the sacroiliac joints. A single anteroposterior pelvic radiograph is sufficient. AS is characterized by loss of distinctness of the subchondral bone in the iliac portion of the joint, which shows as loss of outline and pseudo-widening. Later, subchondral sclerosis and joint bridges may develop, ultimately with total fusion of the joint.

Spinal changes

Plain radiography of the spine may show the following changes:

- *Erosion*: loss of bone cortex at the corner of the vertebral body, called the 'shiny corner sign' or 'Romanus lesion'.[13]
- *Squaring* of the vertebral body: another characteristic feature of AS. It is caused by a combination of corner erosions and periosteal new bone formation along the anterior aspect of the vertebral body.
- *Syndesmophyte* formation: refers to the process in which ossification of the outer fibres of the annulus fibrosus leads to bridging of the corners of one vertebra to another.
- *Ossification* of the adjacent paravertebral connective tissue fibres: posterior interspinous ligament ossification, combined with linking of the spinous process, produces an appearance of a solid vertical dense line in the midline on frontal radiographs.
- The *apophyseal and costovertebral joints*: these are frequently affected by erosions and eventually undergo fusion.
- *A bamboo spine*: results from complete fusion of the vertebral bodies by syndesmophytes and other related ossified areas.

Manubriosternal joint

The earliest information that can be gained on AS is sometimes a radiograph of the manubriosternal joint. Normally, the joint surfaces of the manubriosternal joint are flat but in AS they may become biconcave.[14]

Differential diagnosis

Diffuse idiopathic skeletal hyperostosis

Diffuse idiopathic skeletal hyperostosis (DISH) is a condition characterized by calcification and ossification of ligaments, mainly of the thoracic spine. This condition was described by Forestier over 50 years ago and was termed senile ankylosing hyperostosis.[15] It affects middle-aged and elderly persons and is often asymptomatic, or is associated with mild dorsolumbar pain and/or some restriction of spinal mobility. Prevalence

studies based on the radiological characteristics have shown that between 2.4 and 5.4% of those over 40 years of age have DISH, as well as 11.2% of those over 70.[16]

The diagnosis of DISH is usually based on the definition suggested by Resnick and Niwayama[17]:

- The presence of flowing ossification along the anterolateral aspect of at least four contiguous vertebral bodies. The anterior margins of the vertebral bodies are usually distinct and separated from the hyperostosis by a vertical linear radiolucent zone.[18]
- The presence of relative preservation of the intervertebral disc height in the involved vertebral segment and the absence of radiographic changes of degenerative disc disease.
- The absence of apophyseal joint bone ankylosis and sacroiliac joint sclerosis, erosion and fusion.

Unlike in AS, there is no association with HLA-B27.

Osteoarthritis

In osteoarthritis, the disc spaces are narrowed. Osteophytes arise from the anterior and lateral margins of the vertebral bodies and have a horizontal, beak-like orientation. There are also obvious signs of osteoarthritis of the facet joints and costovertebral joints.

Treatment

The aim of treatment is to keep pain and stiffness under control, to maintain mobility and a straight spine, and to preserve as satisfactory a respiratory excursion as possible.

The basic treatment is administration of anti-inflammatory drugs and a daily exercise programme. Exercises, sports and swimming are strongly recommended.

Significant involvement of the costovertebral joints may affect their mobility, and care should be taken to prevent this. Deep breathing should be practised regularly and can be assisted by a therapist. Mobilization exercises to increase joint movement on inspiration and expiration must also be performed.

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30

Introduction to the lumbar spine

There is much confusion about the meaning of commonly used words such as backache, sciatica and lumbago. In this book they are defined as follows:

- *Backache*: pain in the lower back, central, unilateral or bilateral with or without radiation to the gluteal region and iliac crest.
- *Lumbago*: acute and severe 'backache', causing twinges on attempted movement together with some degree of fixation and/or trunk deviation. The pain reference is wider and sometimes involves the legs in an extrasegmental distribution.
- *Sciatica*: pain in the leg, radiating segmentally to L4, L5, S1 and S2. Pain felt along the anterior aspect of the thigh, as a result of L3 root compression, is not true 'sciatica', although patients may describe it as sciatica.

During recent decades, syndromes related to the lower back have been identified by many epidemiological studies. As a result, the incidence of low back pain, and its socioeconomic impact, are well known.[1] Low back pain is so common that only a minority of individuals escape it. Eighty percent of the general population will at some time suffer from low back pain and 20% are suffering at any given time.[2,3] In Western countries, the 1-year incidence rate for backache is between 24 and 36%.[4–6] In the United States, a study of Veteran Affairs outpatients found a 3-year incidence rate of 67%.[7] Knepel reported that in general practice every tenth patient had back complaints.[8]

Backache and sciatica have also become an increasing socioeconomic problem in industrialized countries: back disorders account for between 1 and 2% of all working days lost[9–13] and for 12.5% of all sickness absence days.[14] They are the most common cause of disability among younger adults in the USA.[15] The number of working days per year lost because of low back pain is 1400 per 1000 workers in the USA,[16] and 2600 per 1000 workers in some British factories.[17,18] In the United Kingdom, low back pain was the largest single cause of absence from work in 1988 and 1989 and accounted for 12.5% of all sick days and over £11 billion in direct and indirect costs in 2000.[19] The cost of treatment and compensation for low back syndromes is enormous and increases every year. In 1976, in the USA, the total cost of spinal disorders was approximately $14 billion. By 1983, this figure had risen to $20 billion,[20] and by 1991 to more than $50 billion[21]; for 1998, estimates and patterns of direct healthcare expenditures among individuals with back pain in the USA had reached $90.7 billion.[22]

Backache and sciatica are complaints rather than specific diagnoses. Although most of these symptoms stem directly or indirectly from lesions of the intervertebral discs, it is widely accepted that low back pain spans a group of disorders with varying causes.

As many as 80% of all cases of low back syndromes relate to the lumbar intervertebral discs; the posterior support structures (facets, ligaments, laminae and fasciae) are directly responsible for less than 20% of cases of back disorders.[23] The evidence for this statement is derived from anatomical and imaging studies, but Cyriax[24] came to the same conclusion almost 50 years ago, purely on careful clinical observations: 'In my experience lumbar disc lesions are responsible for more continuing – yet avoidable – annoyance, frustration, semi-invalidism, general misery and bad temper than any other tissue in the body. In our view, lumbar disc lesions are responsible for well over 90% of all organic symptoms attributable to the back.'

It is remarkable that, in disorders that affect both individuals and society so much, there is so little agreement about possible pathogenesis and pathological entities. Despite the advanced technology available for diagnosis and treatment, the number of patients suffering from backache and sciatica continues to increase.[25] It has even been suggested by both the lay press and professionals that the epidemic increase in disability is partly caused by unnecessary technical investigations and too much surgical treatment.[26]

The main reason for ineffective diagnosis and treatment is that patients are submitted to technical investigations and tests before careful clinical assessment has been made. A basic

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knowledge of anatomy, histology and behaviour of the tissues involved is necessary to be able to understand a patient's symptoms and to interpret the signs found during the clinical examination. These skills are not widely taught nowadays. Students are trained in the interpretation of discograms, myelograms and magnetic resonance images (MRI), but they do not learn how to take a history and make a clinical examination effectively nor how to interpret the clinical picture that emerges from the findings, how this can be translated into a diagnosis and what is the natural history of the detected disorder.

As for other parts of the body, the information gained from the history and physical examination is the first and most important requisite for correct diagnosis. If the clinical assessment is properly done, technical investigations add little information and scarcely improve diagnostic precision. If clinical assessment is omitted, the results of radiographs, myelograms, discograms, computed tomography (CT) scans and MRI will pose more questions than they solve, simply because these investigations, although very sensitive, are not sufficiently specific to guarantee that the detected lesions are indeed the source of the pain. Indeed, CT investigations have a false-positive rate of 15.5% and a false-negative rate of 40%; MRI has a false-positive rate of 13.2% and a false-negative rate of 35.7%.[27] These rates are far too high for imaging alone to be decisive in the diagnosis of an individual patient's problem. In spite of this, many surgeons base their decision to carry out surgery solely on the outcome of these investigations.

With a good understanding of the applied anatomy and a clear insight into the way that tissues behave, it is possible to translate the symptoms and signs found during clinical assessment into a deduction about the anatomical changes that have taken place in a patient's back. The true art of clinical investigation is to organize all the clinical data in such a way that the typical patterns are easily recognized. There are, in fact, only a few symptom patterns and patients' descriptions are frequently strikingly similar.

The *dura mater* is the most important pathway by which an intervertebral disc produces pain. It is the link between the macroscopic changes at the posterior aspect of the intervertebral joint and the symptoms: backache or lumbago result when the dural sac is irritated; sciatica occurs when the dural investment around the spinal nerve is pinched. This 'dural concept', in which both disc and dura have a particular role, accounts for most of the low back syndromes, and is discussed in the chapter on the dural concept (Ch. 33).

Changes in the disc can also have consequences for the *posterior support structures* and result in other signs and different findings. We discuss these in the chapter on the ligamentous concept (Ch. 34).

Increasing degeneration of the spine usually remains painless and is, in fact, a normal event which does not signify any particular disease. Sometimes increasing degeneration can account for acquired *stenosis of the vertebral canal or the lateral recess*. The resulting symptoms and signs are discussed in the chapter on the stenotic concept (Ch. 35).

There are also a number of pain syndromes not related to mechanical problems, such as spondylolisthesis, infectious and rheumatological diseases, and diseases of the osseous structures. These are discussed in the chapter on *non-mechanical spinal disorders* (Ch. 39).

The treatment administered for lumbago, backache and sciatica depends on the diagnosis, i.e. on the data obtained during history taking and clinical examination. A 'standard' treatment for backache does not exist; treatment procedures depend entirely on the underlying disorder. For example, if backache is caused by a small posterior displacement of the intervertebral disc, irritating the dura mater, attempts are made to shift the protruded fragment back into place, either by manipulation or by traction. If this proves impossible, attempts are made to desensitize the dura with an epidural injection. If the protrusion is unstable, and the patient has a history of recurrent back trouble, prophylactic measures against further disc displacements should be taken. If backache is caused by a sprained posterior ligament or an inflamed facet joint, local sclerosing injections or infiltrations with triamcinolone should be used.

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31

Applied anatomy of the lumbar spine

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Human posture

The human spine is a self-supporting construction of skeleton, cartilage, ligaments and muscles. Erect, there are four sagittal curves, which are the result of man's evolution from quadruped to biped. This began in Africa 3 million years ago with *Homo australo-pithecus*, which had a pelvis strong enough to support an erect posture. After a further 1.5 million years, the definitive erect posture had been adopted – *Homo erectus*.[1] The four curves resulted: cervical lordosis, thoracic kyphosis, lumbar lordosis and sacrococcygeal kyphosis. This S form seems to be a compromise between the static and the dynamic qualities of the spine[2]; theoretical considerations suggest that the S form is the shape an elastic bar adopts when it is subjected to axial compression.[3]

The phylogenetic evolution from the large thoracolumbar kyphotic spine of a quadruped into two kyphotic and lordotic curves is also reflected in the spine's ontogeny. In intrauterine life and during the first 5 months after birth, the spinal curves are absent and there is only one slight kyphosis of the whole spine. At 13 months the lumbar spine is straight, at 3 years some lumbar lordosis is present, and by 8 years the lumbar spine has attained its normal adult posture (Fig. 31.1).[4]

As a result of the lumbar lordosis, the intervertebral space is larger in front than behind, which has some consequences for the intervertebral disc. It is clear that axial pressure exerted on the disc in a lordotic spine will result in a forward strain on the disc, whereas increased axial pressure during trunk flexion will force the disc backwards. A backward shift of the disc is a very undesirable event because nerve roots and dura mater emerge at the posterior aspect of the vertebral column. Indeed, these very sensitive structures lie at the weakest and most unprotected part of the column, and it is good posture, rather than any ligamentous device, that protects them against a posterior or posterolateral shift of the disc. Maintaining normal physiological lordosis is therefore important in the prophylaxis of low back syndromes.

The development of the erect posture is recent and it seems that, apart from the compensatory lordosis, not much in the way of structural adaptation has taken place. Comparative anatomical evidence suggests that the spine has evolved as a hanging structure between the anterior and the posterior parts of the body. However, during development from quadruped to biped, the function of the spine had to alter completely (Table 31.1); this has serious consequences. In an upright position, the spine is submitted to axial load, which probably leads to

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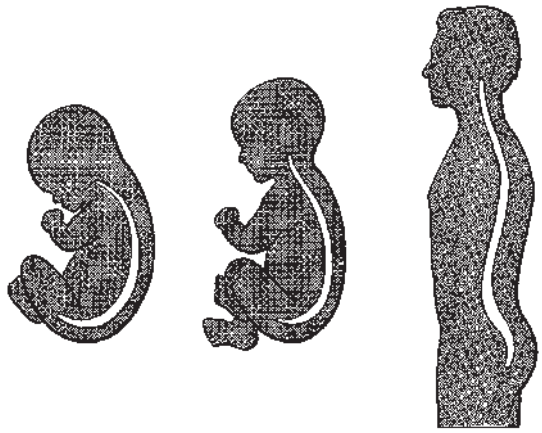


Fig 31.1 • Development of the lumbar lordosis.

Table 31.1 Comparison of the quadruped and biped lumbar spines

	Quadruped	Biped
Structure	Horizontal	Vertical
Load	Horizontal	Axial
Curve	Slight kyphosis	Lordosis
Strength	Against extension	Against flexion
Strong structures	Anterior	(Posterior?)

the premature disc degeneration from which humans are apt to suffer. In the upright position, the lumbar spine has to resist flexion, whereas the quadruped spine has to resist extension because it is structurally undesirable for a 'bridge' to sag in the middle. The change to an upright position, however, has not yet been followed by anatomical adaptation, and the human spine has an anatomy that more readily withstands extension than flexion: the anterior part of the annulus fibrosus is stronger and thicker than the posterior, and the anterior longitudinal ligament is almost twice as thick and broad as the posterior.[5]

Conclusion: the spine was originally designed as a horizontal, slightly kyphotic, hanging structure, strong in resistance to extension. Once the erect posture was achieved, the spine became submitted to axial and flexion stresses. Slight lordosis prevents lumbar discs from shifting backwards and keeps them away from painful and vital structures, such as dura and nerve roots.

Vertebrae

Embryologically, the lower half of a vertebra and the upper half of the one below it originate from the same segment. Between them is the disc, which is partly a remnant of the notochord.

Vertebral bodies

Each vertebral body is more or less a cylinder with a thin cortical shell which surrounds cancellous bone. From L1 to L5, the posterior aspect changes from slightly concave to slightly convex, and the diameter of the cylinder increases gradually because of the increasing loads each body has to carry. At the upper and lower surfaces, two distinct areas can be seen: each is a peripheral ring of compact bone – surrounding and slightly above the level of the flat and rough central zone – which originates from the apophysis and fuses with the vertebral body at the age of about 16. The central zone – the bony endplate – shows many perforations, through which blood vessels can reach the disc. A layer of cartilage covers this central zone, which is limited by the peripheral ring. This is the cartilaginous endplate, forming the transition between the cortical bone and the rest of the intervertebral disc. A sagittal cut through the vertebral body shows the endplates to be slightly concave, which consequently gives the disc a convex form.[6]

Pedicles

The two pedicles originate posteriorly and attach to the cranial half of the body. Together with the broad and flat lamina, they form the vertebral arch. From L1 to L5, the pedicles become shorter and broader, and are more lateral. This narrows the anteroposterior diameter and widens the transverse diameter of the vertebral canal from above downwards. Together with the increasing convexity of the posterior aspect of the vertebral body, these changes in the position of the pedicles alter the shape of the normal bony spinal canal from an ellipse at L1 to a triangle at L3 and more or less a trefoil at L5 (Fig. 31.2).

Laminae

Each lamina is flat and broad, blending in centrally with the similarly configured spinal process, which projects directly backwards from the lamina. The two transverse processes project laterally and slightly dorsally from the pediculolaminar junction. The superior and inferior articular processes originate directly from the lamina.

The part of the lamina between the superior and inferior articular processes is called the 'pars interlaminaris'. It runs obliquely from the lateral border of the lamina to its upper medial border. This portion of the lamina is subjected to considerable bending forces, as it lies at the junction between the vertically oriented lamina and the horizontally oriented pedicle. This 'interlaminar part' will therefore be susceptible to fatigue fractures or stress fractures (spondylolysis) (see Ch. 39).[7]

Intervertebral discs

Two adjacent vertebral bodies are linked by an intervertebral disc. Together with the corresponding facet joints, they form the 'functional unit of Junghans' (Fig. 31.3).[8]

The disc consists of an annulus fibrosus, a nucleus pulposus and two cartilaginous endplates. The distinction between annulus and nucleus can only be made in youth, because the consistency of the disc becomes more uniform in the elderly.

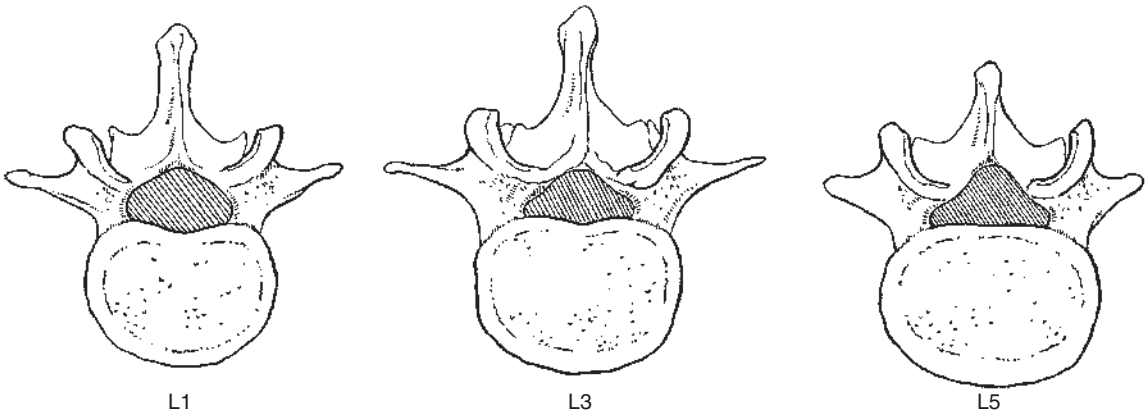


Fig 31.2 • L1, L3 and L5 from above, showing the changes in the diameter of the spinal canal.

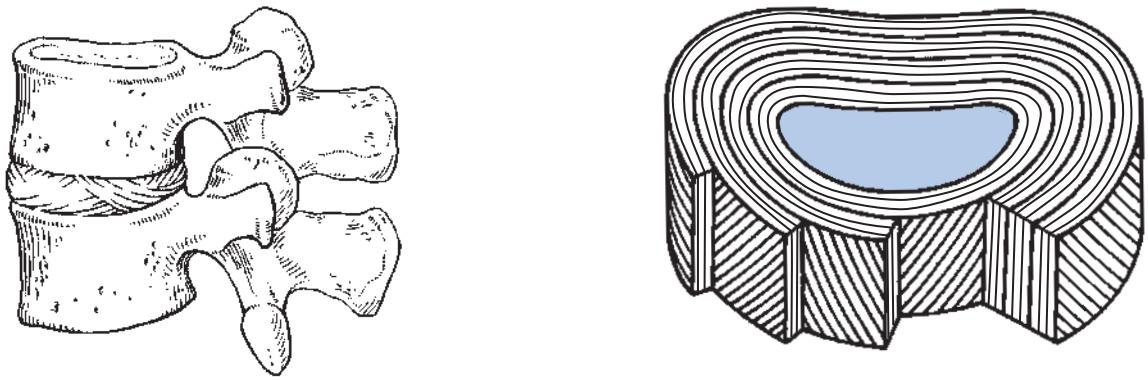


Fig 31.3 • Lateral view of two vertebrae: the 'functional unit'.

For this reason, nuclear disc protrusions are rare after the age of 70. From a clinical point of view, it is important to consider the disc as one integrated unit, the normal function of which depends largely on the integrity of all the elements. That means that damage to one component will create adverse reactions in the others.

Endplates

An upper and a lower *cartilaginous endplate* (each about 0.6–1 mm thick) cover the superior and inferior aspects of the disc. They are plates of cartilage that bind the disc to their respective vertebral bodies. Each endplate covers almost the entire surface of the adjacent vertebral body; only a narrow rim of bone, called the ring apophysis, around the perimeter of the vertebral body is left uncovered by cartilage. That portion of the vertebral body to which the cartilaginous endplate is applied is referred to as the vertebral endplate. The endplate covers the nucleus pulposus in its entirety; peripherally it fails to cover the entire extent of the annulus fibrosus.[9] The collagen fibrils of the inner lamellae of the annulus enter the endplate and merge with it, resulting in all aspects of the nucleus being enclosed by a fibrous capsule.[10]

The endplate permits diffusion and provides the main source of nutrition for the disc.[11,12] Up to the age of 8 years, the cartilaginous endplates are penetrated by blood vessels

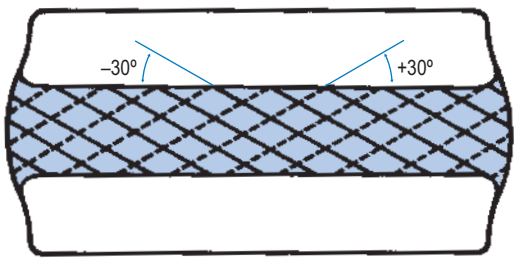


Fig 31.4 • Lamellar construction of the annulus fibrosus.

which pass into the peripheral layers of nucleus and annulus. Thereafter, the disc's nutrition is achieved by diffusion through the endplate. The hyaline endplate is also the last part of the disc to wear through during severe disc degeneration.[13]

Annulus fibrosus

This is made up of 15–25 concentric fibrocartilaginous sheets or 'lamellae' (Fig. 31.4), each formed by parallel fibres, running obliquely at a 30° angle between the vertebral bodies.[14] Because the fibres of two consecutive layers are oriented in opposite directions, they cross each other at an angle of approximately 120°.[15] This arrangement of the annular fibres gives the normal

disc great strength against shearing and rotational stresses,[16] while angular movements remain perfectly possible.[17,18] The outermost fibres are attached directly to bone, around the ring apophysis, and for that reason they are referred to as the ligamentous portion of the annulus fibrosus. The inner third merges with the cartilaginous endplate and is referred to as the capsular portion of the annulus fibrosus (Fig. 31.5).

Nucleus pulposus

This consists of a gelatinous substance, made of a meshwork of collagen fibrils suspended in a mucoprotein base which contains mucopolysaccharides and water.[19] With advancing age, the amount of mucopolysaccharides diminishes, as does that of the water they bind. A young nucleus is 85% water, whereas it is only 65% water in the elderly.[20] These biological changes are mirrored in the macroscopic aspects of the nucleus. In the second and third decades the nucleus is clear, firm and gelatinous but subsequently it becomes drier and more friable. In the elderly, the nucleus has the texture of thickened cream cheese, and is dry, brownish and friable.

At birth the nucleus pulposus occupies the centre of the intervertebral space. As the anterior part of the vertebral body grows faster than the posterior part, the nucleus comes to lie more posteriorly. Consequently, the anterior part of the annulus will have thicker and stronger fibres,[21] which means that the annulus gives better protection against anterior than posterior displacements of the nucleus; this is disadvantageous with respect to the contiguous nerve roots and dura.

Cartilage is devoid of nerves and it has been conventional to draw the same conclusions about the disc. However, over the last few decades, there has been a great deal of research on the possibility that there is some innervation. The presence of free nerve endings has been demonstrated as far as one-third of the way into cadaveric annuli fibrosi,[22] and as far as halfway into annuli fibrosi obtained during posterior fusion operations.[23] Other research has shown a few nervous elements in the periphery of the annulus fibrosus.[24,25]

More recent studies have demonstrated mechanoreceptors to be present in the outer two or three lamellae of the human intervertebral disc and the anterior longitudinal ligament.[26] Although the presence of substance P – generally accepted as an important nociceptive neurotransmitter – has so far not been demonstrated in human intervertebral discs,[27] other neuropeptides have.[28,29] The exact relationship between the existence of small nerve endings in the outer layers of the disc and back pain therefore still remains controversial.

The lack of blood supply to the intervertebral disc has been shown by microangiographic studies.[30] There is some vascularization of the vertebral borders of the disc in children but by the age of 8 years all cartilaginous penetration by blood vessels has disappeared. Vascular buds in the bony endplate remain during adulthood as a vascular bed under the cartilaginous endplate, and diffusion from these through the endplates remains the main nutritional pathway for the disc during adult life,[31,32] although some nutrition via contact with the intimate anterior and posterior longitudinal ligaments is also possible (Fig. 31.6).[33] The disc is thus the largest non-vascular structure in the body, which causes difficulties in healing and regeneration after damage.

Functions of the disc

The primary function of the disc is to join the vertebrae and allow movement between them. The other functions are typical of the erect spine: a shock absorber; a load distributor; and a separator of the posterior facets to maintain the size of the intervertebral foramen.

Behaviour of the disc

The disc as an osmotic system

The main structural components of the intervertebral disc are collagen, proteoglycans (PGs) and water. The water is not free

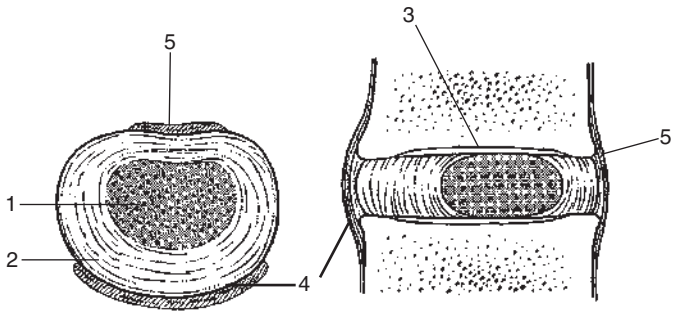


Fig 31.5 • The intervertebral disc: 1, nucleus; 2, annulus; 3, cartilaginous endplate; 4, anterior longitudinal ligament; 5, posterior longitudinal ligament.

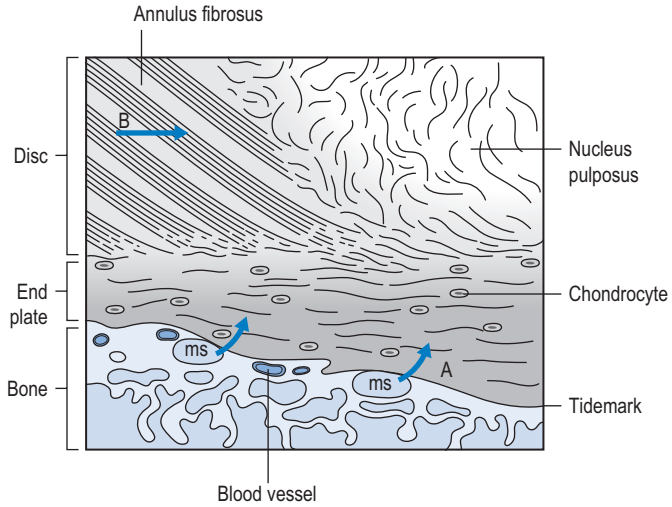


Fig 31.6 • The vertebral endplate, consisting of a hyaline cartilage layer, loosely bonded to a plate of perforated cortical bone. The two routes of metabolite transportation are: A, through the marrow spaces (ms) of the endplate; B, through the annulus. Reproduced from Adams M, Bogduk N, Burton K, Dolan P. *The Biomechanics of Back Pain*, 2nd edn. Churchill Livingstone, Edinburgh, 2006: Fig 7.15 (p. 90).

but is bound by the PGs,[34] which, because of their pronounced osmotic properties, maintain the hydration and turgor of the disc. The proportion of the three constituents varies across the disc. Fluid and PG concentrations are highest in the nucleus and lowest in the annulus, whereas the reverse is true for collagen.

Proteoglycans are complex chemical structures, existing as monomer subunits and aggregates. The former are made up of a central protein molecule with an attached long-chain glucosaminoglycan; the latter consist of monomers, attached to a long hyaluronic acid filament (see Ch. 3). The synthesis of PGs is performed by the cartilage cells and is a continuous process, demanding a well-balanced metabolism.[35] For its nutrition, the disc, devoid as it is of any penetrating vascular structure, depends entirely on diffusion through the central portion of the endplates and the outer annulus (see Fig. 31.6).[11,36] Consequently, the disc is vulnerable and changes in its composition are inevitable as age advances. Although the total collagen content remains fairly constant during adult life,[37] the PG concentration falls.[38,39] The result is that the osmotic properties and the turgor of the disc will also decline as age advances (see p. 438).[40]

Proteoglycans play a key role in the osmotic system of the intervertebral joint, which incorporates the nucleus, annulus and cartilaginous endplates, and also the cancellous bone of the vertebrae. Two compartments – nucleus and paravertebral tissues – are separated by the semipermeable barrier formed by the cartilaginous endplate and the annulus fibrosus, which permits the transport of small molecules only: water, ions and substances of low molecular weight. Diffusion tests with dye demonstrate that only substances with a molecular weight under 400 can pass the disc tissue barrier.[41] The PGs of the inner compartment take up water, until the hydrostatic pressure that results is in balance with the physical tension that arises from the tensile forces of the annulus and the loads applied by muscles, ligaments and gravity (Fig. 31.7).

At this point, there is no net fluid loss or gain. If the external stress is increased – say, by an increase in load – the balance will be disturbed and fluid is expressed from the nucleus (Fig. 31.8a). This loss of fluid has two consequences: tensile stress in the collagen network falls and the concentration of PGs in the nucleus and thus the osmotic pressure rise. In other words, loss of fluid increases the internal swelling pressure, until the latter has risen to the physical stress and a new balance is achieved.[43] The reverse happens when the external load decreases: the internal osmotic pressure is momentarily higher than the external load and fluid is attracted (Fig. 31.8b). The concentration of PGs and the swelling pressure decrease until external and internal pressures again reach an equilibrium.

Conclusion: the fluid content of the disc is not an intrinsic property of the tissue but depends on changes in the external load. Fluid flow is caused by pressure changes on the disc: increased load causes fluid to be expelled, whereas low pressure allows PGs in the disc to absorb fluid from the surrounding tissues.[44]

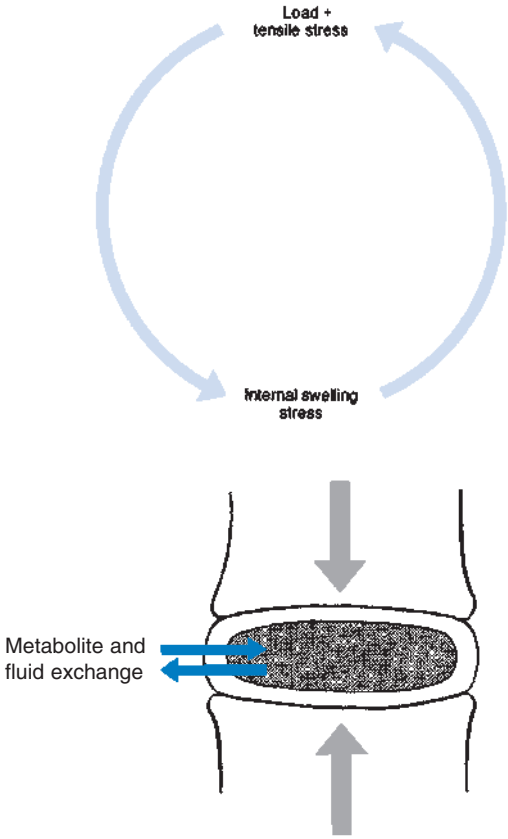


Fig 31.7 • The intervertebral disc as an osmotic system, as described by Krämer.[42]

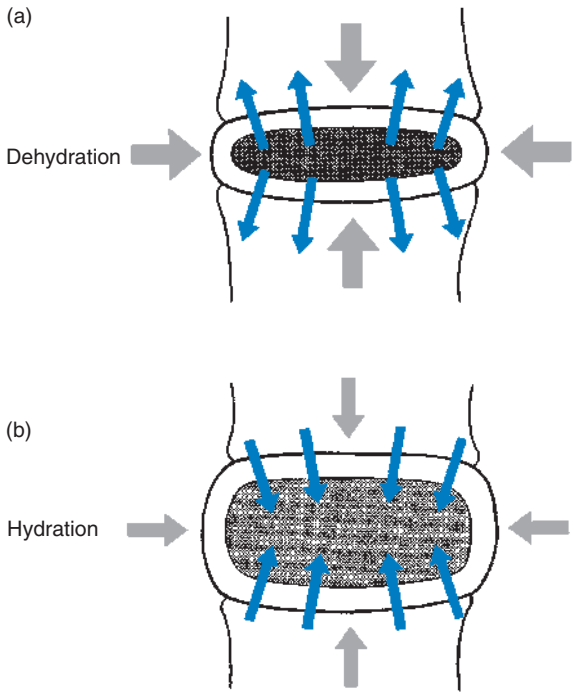


Fig 31.8 • (a) Increase in external stress causes dehydration of the nucleus; (b) decrease in external stress causes hydration of the nucleus.

Influence of the external load on hydration of the disc

Using diffusion techniques with dye and with radioactive substances, Krämer was able to show that in normal non-degenerated discs there is an extravasation of fluid when a load of more than 80 kPa is applied. Absorption takes place when the load is lower than 80 kPa.[45]

From 1966 on, Nachemson and co-workers demonstrated the relationship between body posture and intradiscal pressure by intravital recordings.[46–51] They demonstrated that the pressure in an L3 disc of a healthy individual, weighing 70 kg, is 30 kPa in a supine lying position. Standing and walking around sets up a pressure between 70 and 85 kPa, whereas sitting raises the pressure to 100 kPa and slightly bending forwards to 120 kPa. Lifting a 20 kg object with a bent back and straight legs increases the intradiscal pressure to a surprising 340 kPa.

These findings suggest the dehydration–hydration point, found by Krämer to be around the standing and walking position (Fig. 31.9). The supine position causes hydration of the disc, whereas sitting, bending or lifting squeezes fluid out of the disc.

Since the transport processes in the disc depend largely on fluid flow, continuous change in intradiscal pressure could be of utmost importance for the nutrition of the disc.[53] Load and de-load acts as a pump, and transports water and metabolites from and to the intervertebral disc.[54] In order to protect discs against early degeneration, it is therefore important to keep the intradiscal pressure as low as possible during daily activities. This can be achieved by adopting a slight lordosis at the lumbar spine which protects the disc against excessive pressure. Also, regular changes in position continuously alter the intradiscal pressure, so causing a nutritional fluid flow to and from the disc.[55] The prophylactic measures derived from these findings will be discussed in the section on back schools (see p. 582).

Biomechanical properties of the disc

The fibroelastic annulus provides the disc with hydraulic properties and provides resistance to tensile forces. Retained by this fibroelastic mesh, the nucleus pulposus acts like a fluid-filled balloon. During load, it distributes the axial pressure equally over the cartilage plates and the annulus fibrosus (Fig. 31.10). The annular fibres are under a constant slight stretch, because of the turgor of the nucleus. McNab[56] compares the annulus with a coiled spring that pulls the vertebral bodies together against the turgor of the nucleus. If the load is axial and symmetrical, the nucleus pulposus distributes the force to all sides and therefore perpendicularly on the stretched annular fibres. In this position the disc is very strong and, during high compressive loads, outward herniation of the nucleus is not seen but there is a collapse of the cartilaginous endplates.[57,58]

Asymmetrical loading, however, simultaneously involves tension, compression and shear stresses at different locations in the disc. Bending results in a tensile stress on the convex side and a compressive stress on the concave side: that under tension stretches, while that under compression bulges.[59,60] Tensile stresses on the convex side are increased by the migration of the nucleus. During such asymmetrical loading, the nucleus pulposus is pushed away from the area of compression, following the simple mathematical parallelogram of forces (Fig. 31.11). This means that, in bending forwards, the nucleus will move posteriorly, and therefore greater stress will fall on the posterior annular fibres, which are already subjected to a strong tensile stress.

The posterior migration of the nucleus pulposus in bending has been demonstrated experimentally by putting a metal pin

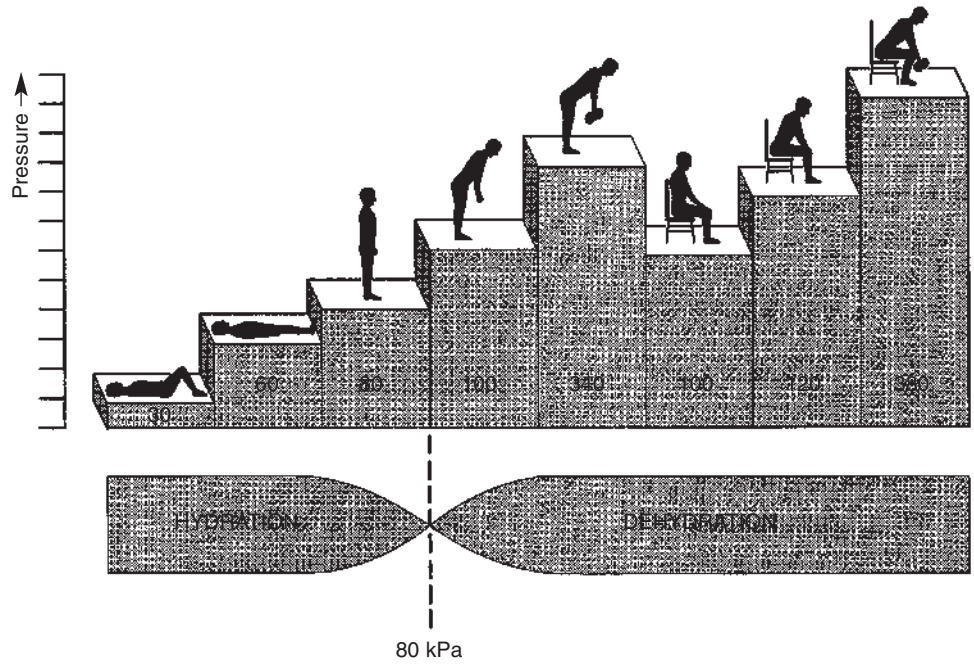


Fig 31.9 • Intradiscal pressure is related to position.[52]

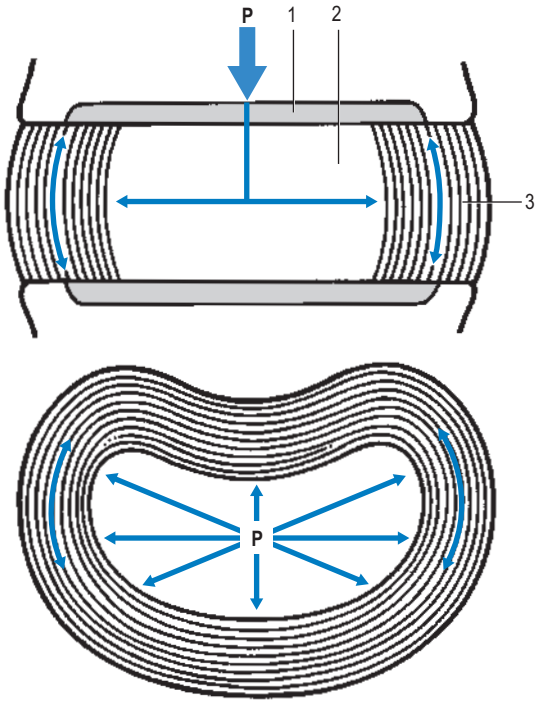


Fig 31.10 • During an axial and symmetrical load, the nucleus pulposus distributes the force to all sides and therefore perpendicularly on the stretched annular fibres. P, load; 1, endplates; 2, nucleus; 3, annular fibres.

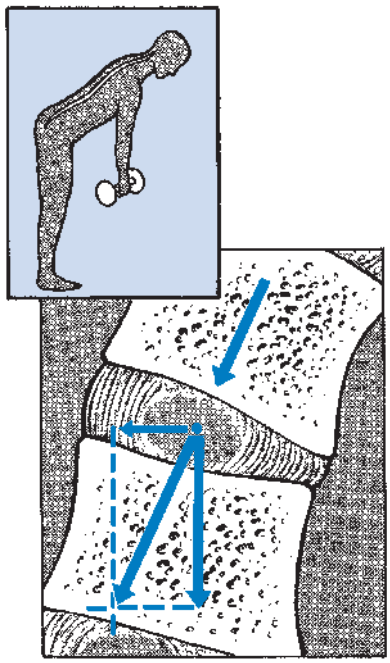


Fig 31.11 • Asymmetrical loading.

in the nucleus pulposus.[61] On continuous forward bending, the nucleus migrated backwards at a speed of 0.6 mm/min during the first 3 minutes and this continued very slowly during the next hour. After bending ceased, the nucleus regained its original position only very slowly. These findings have been confirmed by discography[62] and by magnetic resonance imaging (MRI) studies.[63–65] Biomechanical studies, conducted *in vitro*, have also demonstrated that the normal nucleus moves posteriorly in kyphosis and anteriorly in lordosis.[66–68]

The weak zone of the disc

Several anatomical, biochemical and biomechanical properties make the posterior aspect of the disc the most critical and vulnerable part of the whole intervertebral joint.[69,70]

- The posterior annular fibres are sparser and thinner than the anterior.
- Because the area available for diffusion is smaller posteriorly than anteriorly, the posterior part of the nuclear–annular boundary receives less nutrition and again the posterior part of the disc is the most strained part.[71]
- The posterior longitudinal ligament affords only weak reinforcement, whereas the anterior fibres are strengthened by the powerful anterior longitudinal ligament.
- Because of the special mechanical arrangements of the annular fibres, the tangential tensile strain on the posterior annular fibres is 4–5 times the applied external load.[72]

All these elements explain the predominance of the posterior part of the disc in the development of weakening, radiating ruptures and posterior nuclear displacements. This is unfortunate, because most nociceptive tissues responsible for backache and sciatica (nerve roots and dura mater) emerge just beyond the posterior aspect of the disc.

In order to prevent early degeneration and internal derangement, the erect body has developed only one adequate defence system: namely, a slightly lordotic lumbar posture. Cyriax was the first clinician to point out the importance of the lumbar lordosis. Long before biomechanical experiments, such as those of Nachemson, and purely on clinical findings, he demonstrated the importance of correct posture in the avoidance of backache and sciatica.[73] This is the physiological lumbar lordosis, which diminishes the intradiscal pressures and protects the disc against backward displacements of the nucleus pulposus. 'Keep your back hollow' is still the best advice against recurrent discogenic backache, even more so in recent decades because of the more sedentary jobs many people have. A sitting position not only increases the intradiscal pressure but also forces the lumbar spine into kyphosis by a backwards inclination of the sitting pelvis.[74] Increased sedentary work is probably one of the reasons for the rising rate of lumbar syndromes.

Facet joints

The joints between the lower and upper articular processes are called zygapophyseal joints, apophyseal joints or 'facet' joints. They are true synovial joints, comprised of cartilaginous

articular surfaces, synovial fluid, synovial tissue and a joint capsule (Fig. 31.12). The superior articular surface is slightly concave and faces medially and posteriorly. The convex inferior articular surface points laterally and slightly anteriorly. In general terms, there is a change from a relatively sagittal orientation at L1–L3, to a more coronal orientation at L5 and S1 (Fig. 31.13).[75,76]

Unlike the disc, the facet joints normally do not bear weight and during normal loads they are not subjected to compression strain.[72] In degenerative fragmentation of the disc, however, intervertebral height diminishes and the articular surfaces are subjected to abnormal loading, setting up spondylarthrosis.[77] The main function of the facet joints is to guide lumbar movements and keep the vertebrae in line during flexion–extension and lateral flexion. Because of the more sagittal slope of the articular surfaces, very little rotation takes place at the four upper lumbar levels. More distally, at the lumbosacral level, the joint line has a more coronal plane, which makes rotational movements potentially possible, but these are limited by the iliolumbar ligaments (see p. 424).[78] The total range of rotation in the lumbar spine is therefore very limited, although not completely zero.[79]

The capsule of the joints is well developed and thick and elastic at the dorsal, superior and inferior aspects. At rest, the fibres run slightly diagonally from lateral–caudal to medial–cranial. As the articular excursion is about 0.5 cm at each level, the capsule must have a considerable laxity to follow the points of insertion during flexion. It therefore possesses capsular recesses of varying size, at the superior and inferior poles of the joint, which gives the joint the appearance of a dumb-bell during arthrographic examinations.[80] In extension the posterior capsule can become pinched between the apex of the inferior facet and the lamina below. In order to prevent this, some fibres of the multifidus blend with the posterior capsular fibres and keep the capsule taut.[81] The ventral aspect of the capsule is an extension of the ligamentum flavum. It is very thin[82] and may rupture during intra-articular injections.[83]

During flexion, the inferior articular process slides upwards on the superior articular process. The lower part of the latter loses contact and becomes exposed. Similarly, the lower part of the inferior articular process becomes exposed ventrally. In order to protect these exposed surfaces, and to maintain a film of synovial fluid over the articular cartilages, the facet joints are endowed with small intra-articular 'meniscoids'.[84–86] These small fibro-adipose crescent wedges have a base attached to the joint capsule and an apex that projects into the capsular pouches.[87] Stretching of the capsule during flexion makes them disappear. Some believe that these fibro-adipose enlargements could become pinched between the articular surfaces, constituting a probable source of backache.[88–91]

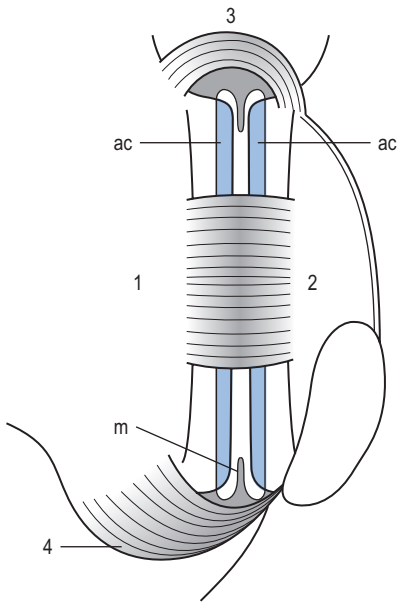


Fig 31.12 • Lateral view of the facet joint with the capsule partly removed. 1, Inferior articular process; 2, superior articular process; 3, superior capsule; 4, inferior capsule; ac, articular cartilage; m, meniscoid.

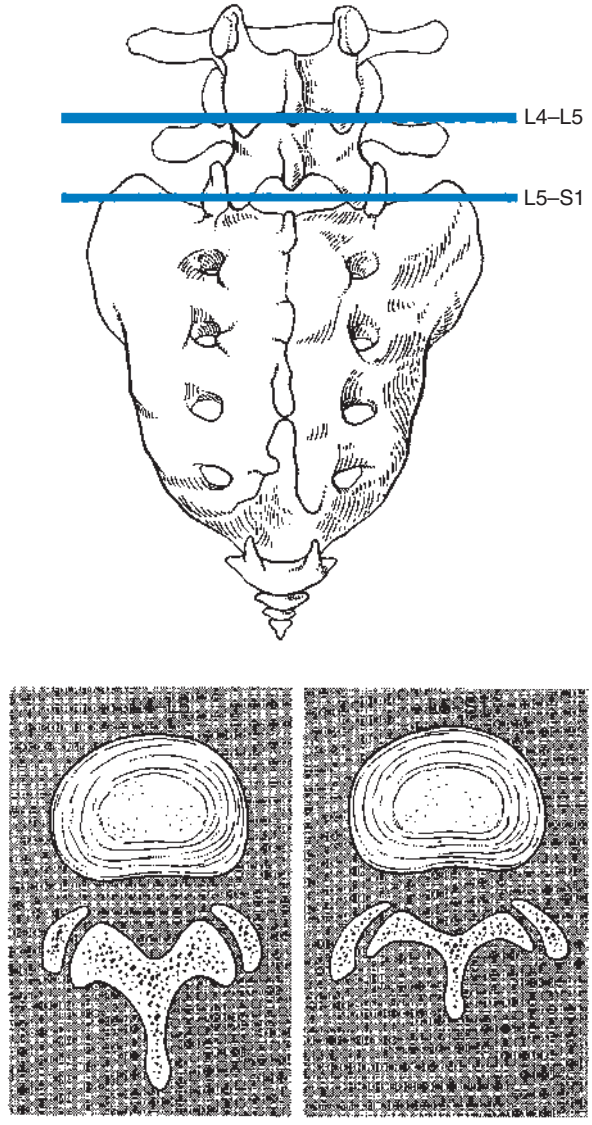


Fig 31.13 • Facet joints at L4–L5 and L5–S1.

The facet joints are innervated by fibres of the medial branch of the dorsal root. The same nerve supplies the inferior aspect of the capsule and the superior aspect of the joint below.[92]

Ligaments

The broad, thick *anterior longitudinal ligament* (Fig. 31.14) originates from the anterior and basilar aspect of the occiput and ends at the upper and anterior part of the sacrum. It consists of fibres of different lengths: some extend over 4–5 vertebral bodies; the short fibres attach firmly to the fibres of the outermost annular layers and the periosteum of two adjacent vertebrae.

The posterior longitudinal ligament (Fig. 31.15) is smaller and thinner than its anterior counterpart: 1.4 cm wide (versus 2 cm in the anterior ligament) and 1.3 mm thick (versus 2 mm). This is another fact in favour of the theory that the lumbar spine was originally designed to be a horizontal hanging structure: to withstand extension strains, the back had to be stronger anteriorly than posteriorly.[5] The posterior longitudinal ligament is narrow at the level of the vertebral bodies, and gives lateral expansions to the annulus fibrosus at the level of the disc, which bestow on it a denticulated appearance.[93]

Although the posterior ligament is rather narrow, it is important in preventing disc protrusion.[94] Its resistance is the main factor in restricting posterior prolapse and accounts for the regular occurrence of spontaneous reduction in lumbago. This characteristic is also exploited in manipulative reduction, when a small central disc displacement is moved anteriorly when the ligament is tightened. The fact that the ligament occupies only the midline of the vertebral column is one of the predetermining factors in the progression of sciatica: as a central protrusion enlarges, it tends to move in the direction of least resistance – lateral to the ligament. Once free from ligamentous resistance, it further enlarges and starts to compress the nerve root. This anatomical evolution is mirrored by the change in the clinical picture: a central backache is replaced by a unilateral sciatica.

The ligamentum flavum (Fig. 31.16) connects two consecutive laminae and has a very elastic structure with an elastin content of more than 80%.[82] The lateral extensions form the anterior capsule of the facet joints and run further laterally to connect the posterior and inferior borders of the pedicle above with the posterior and superior borders of the pedicle below. These lateral fibres form a portion of the foraminal ring and the lateral recess.[95,96]

The interspinous ligament (see Fig. 31.14) lies deeply between two consecutive spinal processes. Unlike the longitudinal ligaments, it is not a continuous fibrous band but consists of loose tissue,[97] with the fibres running obliquely from posterosuperior to anteroinferior.[98] This particular direction may give the ligament a function over a larger range of intervertebral motion than if the fibres were vertical.[99] The ligament is also bifid, which allows the fibres to buckle laterally to both sides when the spinous processes approach each other during extension.[97]

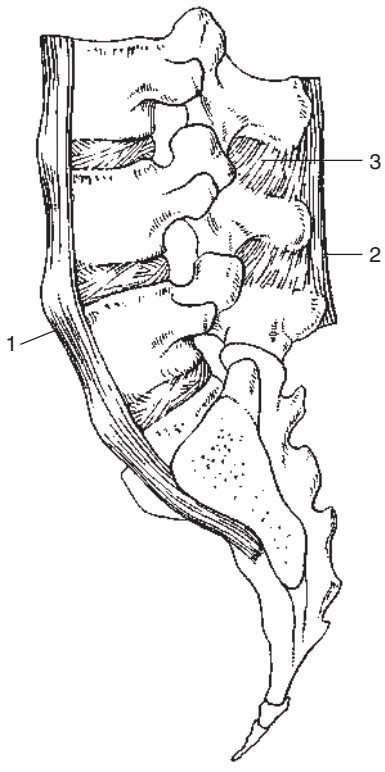


Fig 31.14 • Anterior longitudinal (1) and supraspinous and interspinous ligaments (2 and 3, respectively).

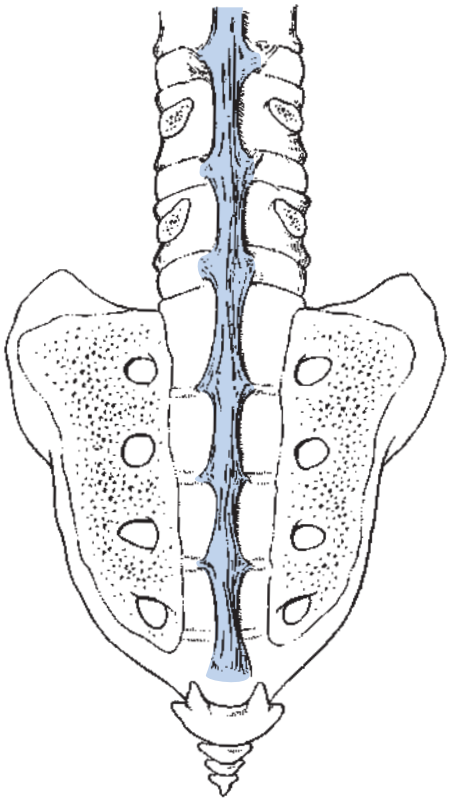


Fig 31.15 • Posterior longitudinal ligament.

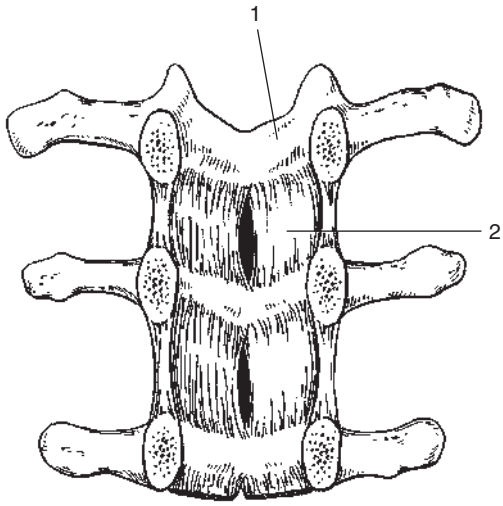


Fig 31.16 • Lamina (1) and ligamentum flavum (2).

The supraspinous ligament is broad, thick and cord-like. It joins the tips of two adjacent spinous processes, and merges with the insertions of the lumbodorsal muscles. Some authors consider the supraspinous ligament as not being a true ligament, as it seems to consist largely of tendinous fibres, derived from the back muscles.[100] The effect of the supraspinous ligaments on the stability of the lumbar spine should not be underestimated.[101] Because the ligament is positioned further away from the axis of rotation and due to its attachments to the thoracolumbar fascia,[102] it will have more effect in resisting flexion than all the other dorsal ligaments. Pearcy[103] showed that the distance between the tips of the spinous processes increases during full flexion by 360% at L3–L4 and 129% at L5–S1. By contrast, the posterior longitudinal ligament only increases by 55% at L3–L4 and 34% at L5–S1. This demonstrates the limiting effect of the ligament on the increasing posterior disc height during stooping. The importance of a strong supraspinous ligament in the prophylaxis of recurrent disc protrusions will be discussed later.

The intertransverse ligaments are thin membraneous structures joining two adjacent transverse processes. They are intimately connected to the deep musculature of the back.

The iliolumbar ligaments (Fig. 31.17) are thought to be related to the upright posture.[104] They do not exist at birth but develop gradually from the epimysium of the quadratus lumborum muscle in the first decade of life to attain full differentiation only in the second decade.[105] The ligament consists of an anterior and a posterior part.[106–108] The anterior band of the iliolumbar ligament is a well-developed, broad band. Its fibres originate from the anterior–inferior part of the L5 transverse process from as far medially as the body of the L5 vertebra to the tip of the transverse process, and expand as a wide fan before inserting on the anterior part of the iliac tuberosity. The posterior band of the iliolumbar ligament originates from the apex of the L5 transverse process and is thinner than the anterior. It inserts on the iliac crest, behind the origin of the quadratus lumborum.[108]

The iliolumbar ligaments play an important role in the stability of the lumbosacral junction by restricting both side flexion and rotational movement at the L5–S1 joint and forward sliding of L5 on the sacrum.[104,109,110] One clinical consequence of this is that posterolateral disc protrusions at the L5–S1 level will not be followed by large lateral flexions of L5 on the sacrum. Marked adaptive deformity will therefore be absent here. Consequently, a large lateral tilt in a patient with acute backache means a displacement at L3–L4 or L4–L5, since these intervertebral joints can open up more easily. Also, stabilization of the lumbosacral junction by the strong iliolumbar ligaments may explain the fact that L5–S1 pars defects are more stable than L4–L5 lesions (see spondylolisthesis, Ch. 58).[111,112]

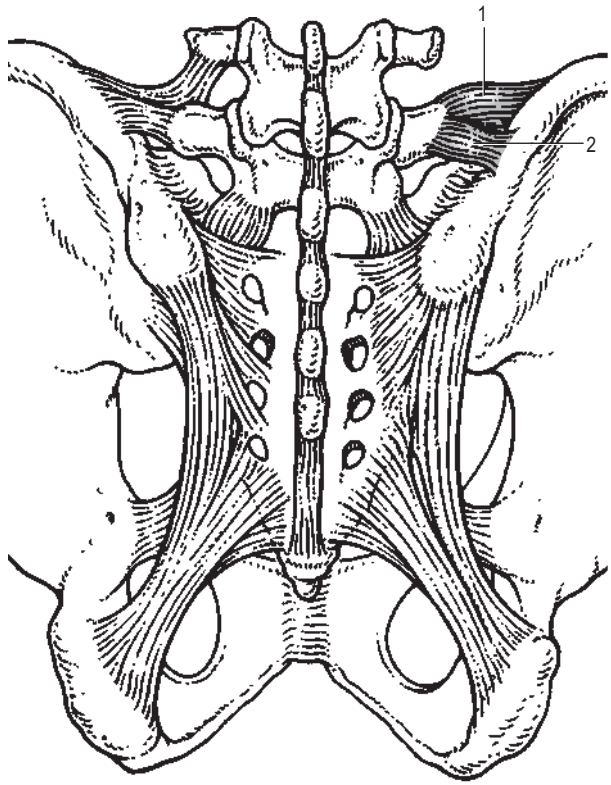


Fig 31.17 • Iliolumbar ligaments: 1, anterior band; 2, posterior band.

Muscles and fasciae

The spine is unstable without the support of the muscles that power the trunk and position the spinal segments.[113] Back muscles can be divided in four functional groups: flexors, extensors, lateral flexors and rotators (Fig. 31.18).

The extensors are arranged in three layers: the most superficial is the strong erector spinae or sacrospinalis muscle. Its origin is in the erector spinae aponeurosis, a broad sheet of tendinous fibres attached to the iliac crest, the median and lateral sacral crests and the spinous processes of the sacrum and lumbar spine.[114] The middle layer is the multifidus. The fibres of the multifidus are centred on each of the lumbar spinous processes. From each spinal process, fibres radiate inferiorly to insert on the lamina, one, two or three levels

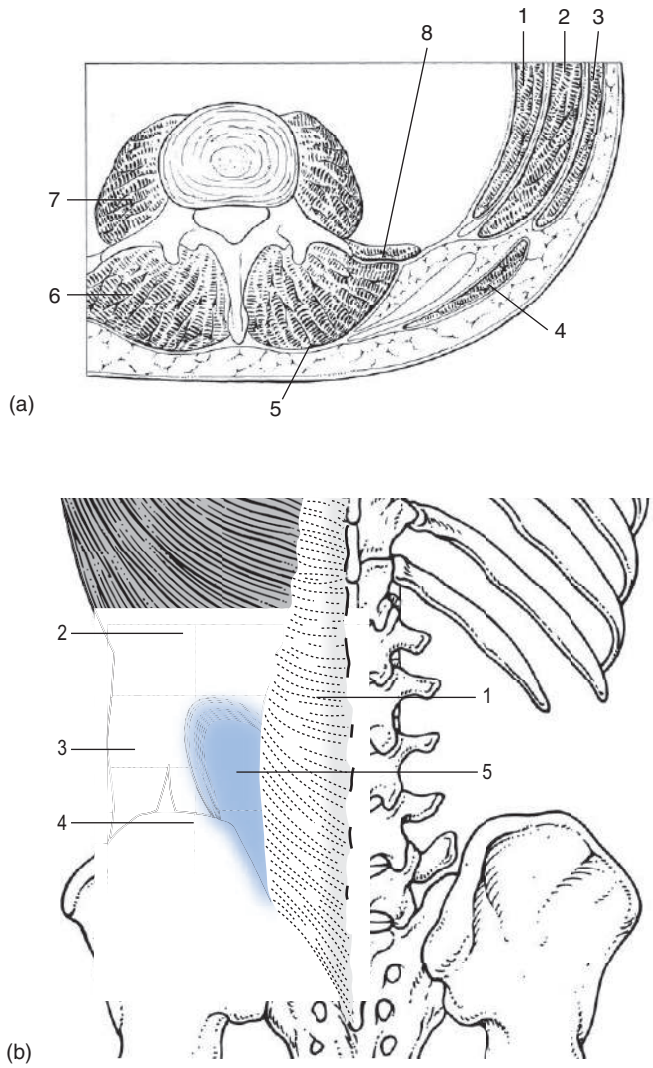


Fig 31.18 • (a) Muscles of the lumbar spine: 1, transversus abdominis; 2, internal oblique; 3, external oblique; 4, latissimus dorsi; 5, lumbar fascia; 6, erector spinae; 7, psoas; 8, quadratus lumborum. (b) Posterior layer of the thoracolumbar fascia: 1, thoracolumbar fascia; 2, fascia latissimus dorsi; 3, fascia of external oblique; 4, posterior superior iliac spine; 5, lateral raphe.

below. The arrangement of the fibres is such that it pulls downwards on each spinal process, thereby causing the vertebra of origin to extend.[115] The third layer is made up of small muscles arranged from level to level, which not only have an extension function but are also rotators and lateral flexors.

The extensor muscles are enveloped by the *thoracolumbar fascia* (Fig. 31.18b), which in turn consists of three layers. The anterior layer is quite thin and covers the anterior surface of the quadratus lumborum. Medially, it is attached to the anterior surfaces of the lumbar transverse processes, and in the intertransverse space it merges with the intertransverse ligaments. The middle layer lies behind the quadratus lumborum muscle. Medially, it also continues into the intertransverse ligament to attach to the lateral border of the lamina. The posterior layer covers the back muscles. It arises from the lumbar spinous processes and from the supraspinous ligaments to envelop the back muscles and blend with the other layers of the thoracolumbar fascia along the lateral border of the iliocostalis lumborum. The union of the fasciae is quite dense and forms a strong raphe (the lateral raphe[116]) which fuses with the fibres of transversus abdominis, internal oblique and latissimus dorsi muscles. The lateral raphe further inserts at the posterior segment of the iliac crest and the posterior superior iliac spine.[117]

The flexors of the lumbar spine consist of an intrinsic (psoas and iliacus) and an extrinsic group (abdominal wall muscles).

Lateral flexors and rotators are the internal and external oblique, the intertransverse and quadratus lumborum muscles. It is important to remember that pure lateral flexion is brought about only by the quadratus lumborum.

Spinal canal

The spinal canal is made up of the canals of individual vertebrae so that bony segments alternate with intervertebral and articular segments. The shape of the transverse section changes from round at L1 to triangular at L3 and slightly trefoil at L5 (see Fig. 31.2).[118]

The margins of the canal are formed by an anterior wall and a posterior wall, connected through pedicles and intervertebral foramina.

The anterior wall consists of the alternating posterior aspects of the vertebral bodies and the annulus of the intervertebral discs. In the midline these structures are covered by the posterior longitudinal ligament, which widens over each intervertebral disc.

The posterior wall is formed by the uppermost portions of the laminae and the ligamenta flava. Because the superoinferior dimensions of the laminae tend to decrease at the L4 and L5 levels, the ligamenta flava consequently occupy a greater percentage of the posterior wall at these levels.[95] The posterolateral borders of the posterior wall are formed by the anterior capsule of the facet joint and the superior articular process, which is located well anterior of the articulating inferior articular process.

The spinal canal contains the dural tube, the spinal nerves and the epidural tissue.

Dura mater

The dura mater is a thick membranous sac, attached cranially around the greater foramen of the occiput, where its fibres blend with the inner periosteum of the skull, and anchored distally to the dorsal surface of the distal sacrum by the filum terminale. The latter descends to the coccyx, where its fibres merge with the connective tissue of the sacroiliac ligaments.[119] The dural sac itself ends blind, usually at S2. There is an inconstant dural attachment, the 'Hofmann complex',[120] made up of bands of connective tissue and loosely joining the anterior dura to the vertebral column (Fig. 31.19). Ventral meningovertebral ligaments pass from the ventral surface of the dura to the posterior longitudinal ligament. They are variable in structure and may present either as tight bands, bifurcations

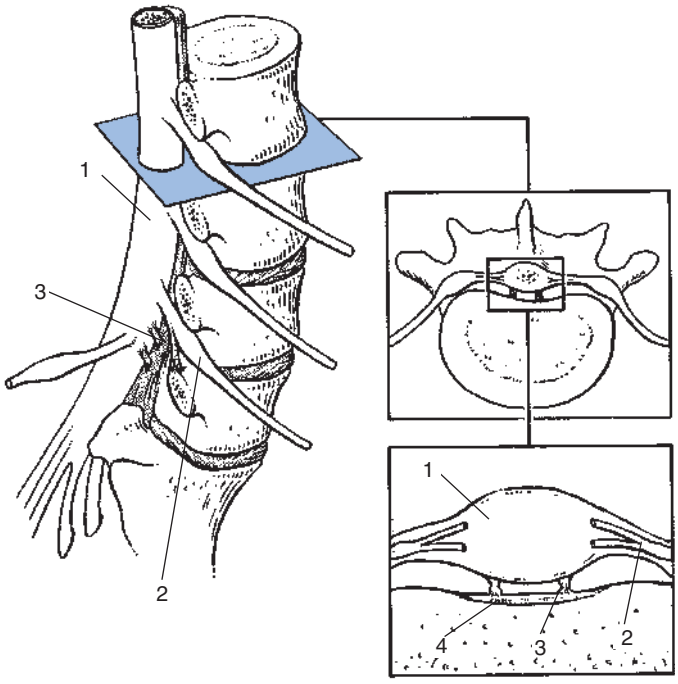


Fig 31.19 • 1, Dura mater; 2, nerve root in the nerve root sleeve; 3, meningovertebral ligaments; 4, posterior longitudinal ligament.

in a Y shape or paramedian bands.[121–123] Others reported on more lateral ligaments, passing from the lateral surface of the dural sac and blending with the periosteum of the pedicles.[124–127]

At the lumbar level, the dura contains the distal end of the spinal cord (conus medullaris, ending at L1), the cauda equina and the spinal nerves, all floating and buffered in the cerebrospinal fluid. The lumbar roots have an intra- and extrathecal course. Emerging in pairs from the spinal cord, they pass freely through the subarachnoid space before leaving the dura mater. In their extrathecal course and down to the intervertebral foramen, they remain covered by a dural investment. At the L1 and L2 levels, the nerves exit from the dural sac almost at a right angle and pass across the lower border of the vertebra to reach the intervertebral foramen above the disc. From L2 downwards, the nerves leave the dura slightly more proximally than the foramen through which they will pass, thus having a more and more oblique direction and an increasing length within the spinal canal. The practical implications of this oblique course of the roots are discussed later.

The dura mater has two characteristics that are of cardinal clinical importance: mobility and sensitivity.

Dural mobility

During spinal movements, the canal is subject to variations in its length and shape. It is obvious that all variations in dimensions of the vertebral canal will influence its contents.

The vertebral canal lengthens considerably during flexion: O'Connell[128] showed by radiological measurements that in full flexion the length of the cervical canal increases by 3 cm, compared with its neutral position. The dura mater, a structure situated in the vertebral canal but anchored at the top and at the bottom, will consequently move in the spinal canal. Breig[129] suggests that the dura mater unfolds and stretches. Other authors have found a gliding of the dural sac in relation to the spinal canal during flexion and extension.[130–133] Using gas myelography, Decker[134] showed that the dura moved towards the front of the canal during flexion: like a rubber band, it shifts towards a position of less tension, and is pulled against the anterior wall. Klein[135] demonstrated an upward displacement of the dura by more than 5 mm at L3 level during full flexion of the spine.

Straight leg raising can put considerable traction on the dural sac. During this manœuvre the L4, L5, S1 and S2 nerve roots are dragged downwards and forwards (Fig. 31.20). At the level of the intervertebral foramen, the degree of downward movement is between 1 and 4 mm.[136–138] As the root is connected through its dural investment with the distal part of the dura, the latter will also be involved in the downward movement. Therefore, straight leg raising drags on the dura mater and pulls it caudally, laterally and forwards.[135,139]

During neck flexion and straight leg raising, the dura thus moves slightly in relation to the anterior wall of the spinal canal, despite some loose attachments between the posterior longitudinal ligament and the dural sac. Anatomical changes at the anterior walls – for instance, a disc protrusion bulging dorsally into the canal – compress the dura. Conversely it can be pulled against this protrusion, whether from below during straight leg raising or from above during neck flexion. The observation that the dura is mobile thus has considerable clinical significance, since an increase of lumbar pain during neck flexion or during straight leg raising implicates the dura mater as the source. In fact, these signs have been accepted for decades as being positive in meningeal irritation (Kernig's sign and neck retraction), but this mechanism of dural pain was not elucidated until Cyriax's paper was published in 1945.[140] In the differential diagnosis of lumbar pain syndromes, 'dural signs' are extremely important in distinguishing a lesion in which the anterior part of the dura mater is involved (disc displacements) from possible lesions at the posterior wall (facet joints and ligaments).

Dural sensitivity

Clinical experiments have shown that the anterior part of the dura is sensitive to both mechanical and chemical stimulation.[141,142] Back pain is also well known in the context of neurological diseases in which the dura becomes inflamed[143] or compressed.[144] Further evidence for dural pain comes from neurosurgical studies that report relief of postlaminectomy pain after resection of the nerves to the dura.[145]

From the 1950s on, numerous neuroanatomical studies have been conducted that describe the innervation of the dural tube.[146] Several authors have shown that the ventral half of the dura mater is supplied by small branches of the sinuvertebral nerve.[147,148] Further work has confirmed that the innervation is from the sinuvertebral nerves and is confined to the anterior part of the dura only.[149,150]

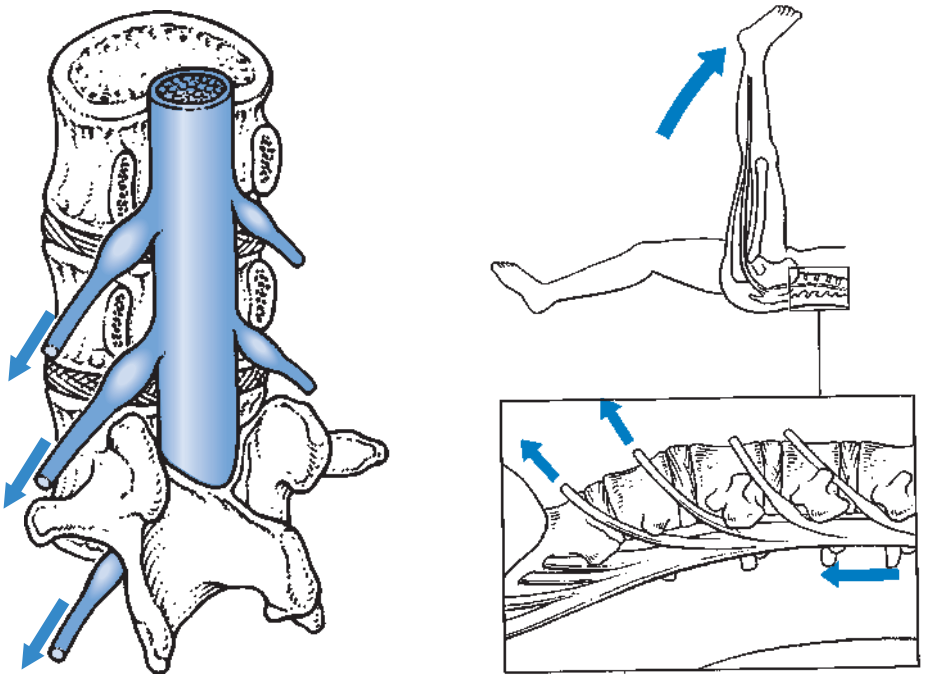


Fig 31.20 • Movement of nerve root and dura mater during straight leg raising.

During the last decade, immunohistochemical studies clearly demonstrated a significant number of free nerve endings, containing substance P, calcitonin-generated peptides and other neurotransmitters contributing to nociception.[151,152] All these findings have been confirmed and extended recently so that the present concept is of a dense, longitudinally orientated nerve plexus in the ventral spinal dura, extending over up to eight segments, showing a great deal of overlap between adjacent levels and crossing the midline.[153,154] The anterior part of the dura mater is thus innervated by a mesh of nerve fibres which belong to different and consecutive sinuvertebral nerves (Fig. 31.21). This probably explains the phenomenon of 'dural pain', which is a pattern of large and broad reference of pain covering different dermatomes, commonly found in low back syndromes. The patient then describes lumbar pain, radiating to the abdomen or up to the chest, to the groin or to the front of both legs.[155]

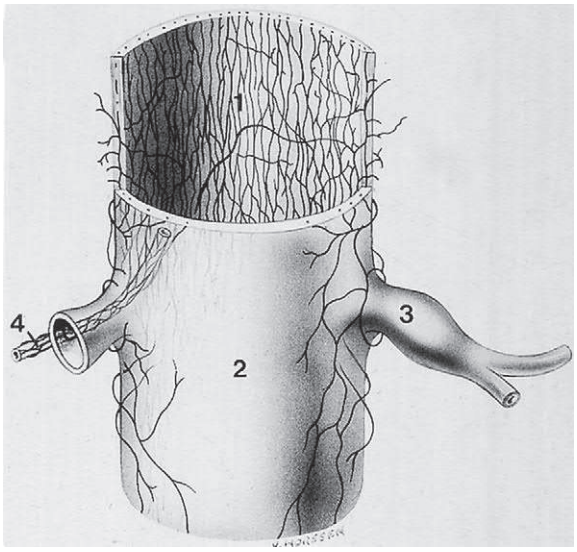


Fig 31.21 • The anterior part of the dura mater is innervated by a mesh of nerve fibres belonging to different and consecutive sinuvertebral nerves. 1, anterior part of the dura; 2, posterior part of the dura; 3, nerve root; 4, sinuvertebral nerve. Reproduced with permission from Groen.[154]

Nerve roots

Definition

The spinal cord terminates at the level of T12–L1. Consequently the lower lumbar and sacral nerve roots must run within the vertebral canal. The motor (ventral) and dorsal (sensory) rootlets that take their origin in an uninterrupted series of attachments at the ventrolateral and the dorsolateral aspects of the cord, run freely downwards through the subarachnoid space of the dural sac.

The rootlets that form one 'nerve root' are gathered into pairs before they leave the dural sac. They do so by taking with them an extension of dura mater and arachnoid mater, referred to as a 'dural sleeve'. The pair of roots covered by dura mater is called the *intraspinal, intrathecal part* of the spinal nerve.

The pairs of spinal roots join at the level of the foramen. Immediately proximal to its junction with the ventral root, the dorsal root forms an enlargement – the dorsal root ganglion – which contains the cell bodies of the sensory fibres in the dorsal

root. Distal to the junction at the foramen, the dura mater merges with the epineurium of the spinal nerve. From here the *extraspinal part* of the spinal nerve begins.[125]

Boundaries

The entire course of the intraspinal part of the spinal nerve is enveloped by the *radicular canal*[156] or *spinal nerve root canal*.[157] The term 'lateral recess' has been applied to the bony boundaries of this radicular canal.[158]

The radicular canal is a small, cone-shaped, osteofibrous space, which begins at the point where the nerve root leaves the dural sac and ends at the lateral border of the intervertebral foramen. It thus shelters the complete extrathecal nerve root in its dural sheath. The direction of the canal is caudal, lateral and slightly anterior. The anterior wall is formed by the posterior aspects of the vertebral body and intervertebral disc, both partly covered by the posterior longitudinal ligament. The posterior wall is the ligamentum flavum, the lamina and the corresponding superior articular facet. The medial wall is the dura mater. The lateral aspect of the radicular canal is formed by the internal aspect of the pedicle and is continuous with the intervertebral foramen.

The length of the radicular canal increases from L3 to S1, so making the L5 and S1 roots more liable to compression. The L3 nerve root travels behind the inferior aspect of the vertebral body and the L3 disc. The L4 nerve root crosses the whole vertebral body to leave the spinal canal at the upper aspect of the L4 disc. The L5 nerve root emerges at the inferior aspect of the fourth lumbar disc and crosses the fifth vertebral body to exit at the upper aspect of the L5 disc (Fig. 31.22).[159,160] Further clinical applications of this downward direction of the nerve roots are:

- At L4 level, a disc protrusion can pinch the fourth root, the fifth root or, with a larger protrusion, both roots.
- At L5 level, a disc can compress the fifth root, the first sacral root or both.
- Root L5 can be compressed by an L4 or an L5 disc.

It is, however, important to remember that aberrant courses and anastomoses exist between the lumbar nerve roots,[161] which may be present in about 4% of the population.[162]

The intervertebral foramen[163] is the point where the spinal nerve emerges from the canal (Fig. 31.23). It is located in a sagittal plane, so it can be demonstrated perfectly on a plain lateral radiograph. The foramen is limited cranially by the upper pedicle and caudally by the pedicle below. The anterior wall corresponds to the posterior aspect of the vertebral body and the disc. The posterior wall of the intervertebral foramen is formed by the articular facets. The size of the foramen increases from T12–L1 to L4–L5, but the foramen L5–S1 is the smallest of all and is located slightly more anteriorly.

Anatomy

The radicular canal contains the intraspinal extrathecal nerve root. The nerve root consists of a sheath (dural sleeve) and fibres. Each structure has a specific behaviour and function,

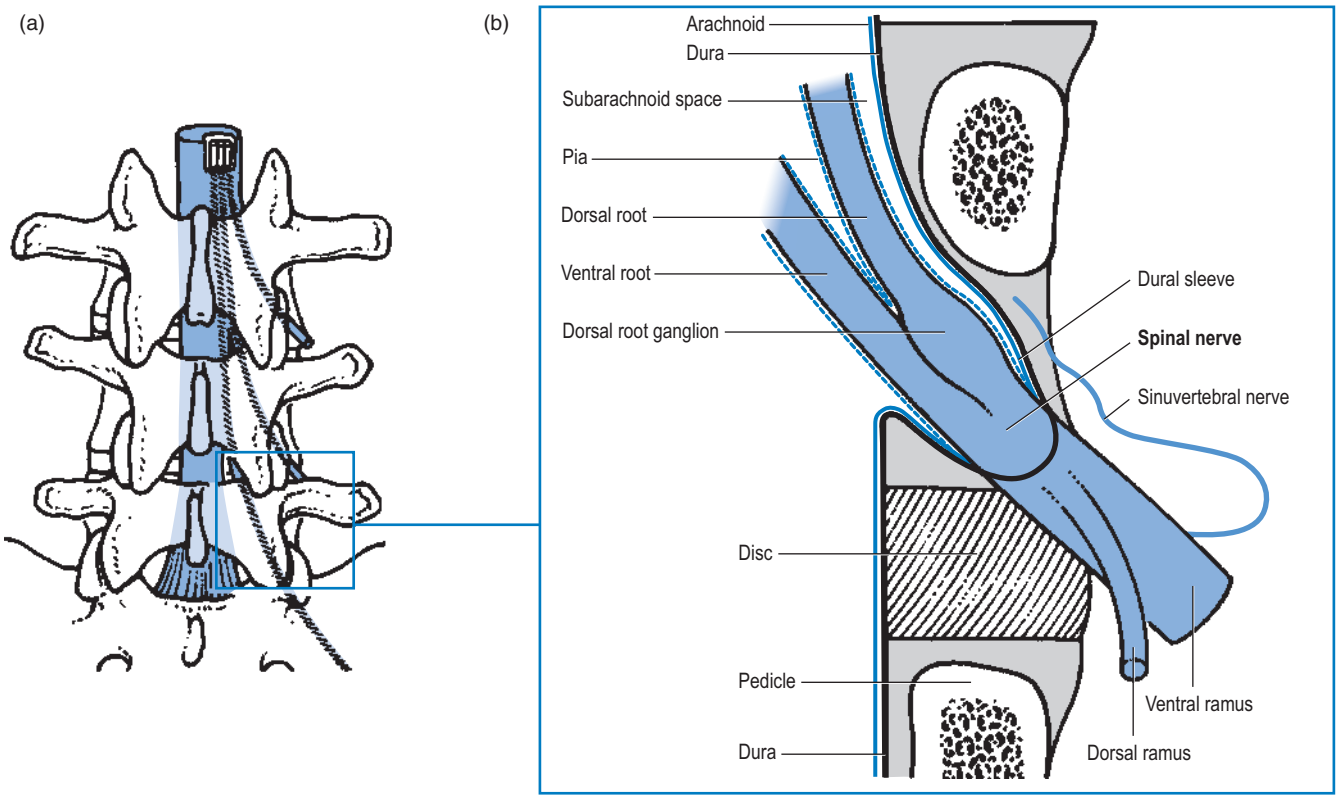


Fig 31.22 • (a) Course of the lumbar nerve roots; (b) anatomy of the nerve root.

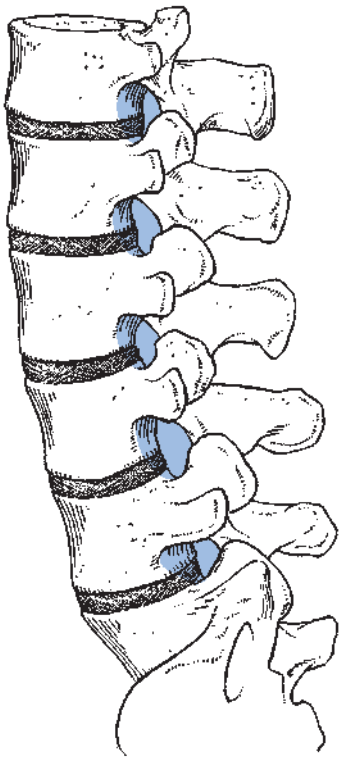


Fig 31.23 • Intervertebral foramina.

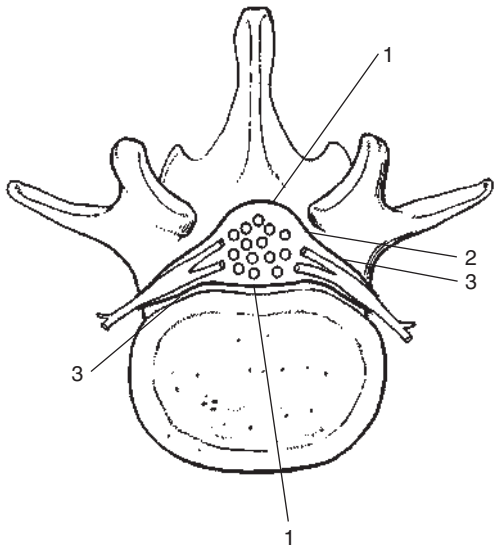


Fig 31.24 • The relationship between the nerve root and dural sheath: 1, dura mater; 2, dural pouch; 3, dural sleeve of nerve root.

Box 31.1

Nerve root behaviour

Sheath	Fibres
Responsible for:	Responsible for:
• Segmental pain	• Paraesthesia
• Mobility	• Conductivity

responsible for typical symptoms and clinical signs (Box 31.1). This has some clinical consequences: slight pressure and inflammation only involve the sleeve and provoke pain and impaired mobility. More substantial compression of the root will also affect the nerve fibres, which leads to paraesthesia and loss of function.

The dural sheath

The dural sheath (Fig. 31.24) starts as a funnel-shaped pouch, enclosing the anterior and posterior roots at their exit from the dural sac. The dural nerve root sleeve proper is formed at the end of this short pouch and continues distally to the foramen, where it merges with the connective tissue sheath of the ganglion and the spinal nerve. The dural investment of the nerve root therefore does not extend beyond the lateral border of the vertebral foramen. In this sleeve, the anterior and posterior roots no longer lie free but are firmly bound to the dural sleeve by the arachnoid membrane. In other words, the subarachnoid space forms a bilaminar tube within the root sleeve as a whole.[164]

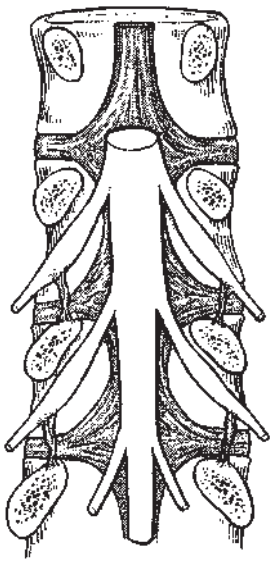


Fig 31.25 • The lateral root ligament.

At the foramen, the epidural tissue becomes more condensed and forms a loose ligamentous fixation of the epineural sheath to the bony boundaries of the intervertebral foramen.[165,166] A stronger ligament (the so-called 'lateral root ligament'; Fig. 31.25), connecting the epineural sheath to the pedicle, has also been described.[167] It has been suggested that the fixation of the dural sleeve, together with the anterior attachment of the dura to the posterior longitudinal ligament, could be of some importance in the mechanism of sciatica.[168] Simple mechanical analysis suggests that pressure applied to the nerve root by a disc protrusion is determined by the extent of the dural ligament fixation rather than by compression of the root against the posterior wall.[169]

The dural investment of the nerve root is sensitive and mobile, like the dural sac.

Mobility

Although the intervertebral foramen represents a point of relative fixation of the nerve, some caudal migration of the latter remains possible.[170] Distal traction on the sciatic nerve and lumbosacral plexus thus pulls the nerve root downwards and drags on the dural sheath and the dura. This occurs during straight leg raising, when the nerve roots of L4, L5, S1 and S2 are moved downwards at the level of the intervertebral foramen.[136,171,172] The main range of motion of the S1 root is 4 mm, of L5 3 mm and of L4 1.5 mm. Straight leg raising does not pull directly on the L3 root. This structure can only be moved caudally during knee flexion in the prone position, which stretches the femoral nerve.[173] It is not possible to test the mobility of the S3 and S4 roots because they do not reach the lower limb.

Because of the downward and anterior direction of the nerve roots and the relative fixation of the dural investment at the anterior wall, a downward movement of the nerve always involves anterior displacement, which pulls the root against the posterolateral aspect of disc and vertebra. Restriction of nerve root mobility therefore always means anterior compression of the root. Internal rotation of the hip during straight leg raising adds more tension to the lumbosacral plexus and nerve roots.[174] To clinicians this is not surprising, because it is common to see patients with considerable limitation of straight leg raising actively rotating their hips laterally when it is performed, thus protecting the inflamed root against further traction.

Cyriax drew attention to two interesting phenomena in relation to the mobility of the nerve root sheath: namely, the existence of a painful arc and the aggravation of the pain during neck flexion.[175]

- It is a common clinical finding that patients with sciatica show momentary pain during straight leg raising: there is pain only in a certain sector of movement (usually between 45 and 60°). The most acceptable explanation for this curious sign is that a small discal bulge exists over which the root slips and thereafter the rest of the movement is painless. This painful arc during straight leg raising always implies a small disc displacement and is a good indication that reduction by manipulation or traction is possible.
- The dural sheath can also be stretched from above. As we have seen previously (in the section on dural mobility), the dura can slip upwards during neck flexion. If pain brought on by straight leg raising is aggravated by neck flexion, the tissue thus stretched must run in a continuous line from the lumbosacral plexus to the neck. Only the dura mater and its continuations, the dural investments, can possibly be stretched from above and below at the same time.

Sensitivity of the dural investment

Dural root sheaths are innervated by the sinuvertebral nerve,[149] and each sheath receives branches from the nerve of the corresponding side and level only. In contrast to the anterior aspect of the dural sac, anastomoses between branches of adjacent sinuvertebral nerves do not exist. Pain originating from the dural sheath is therefore strictly segmental and follows the corresponding dermatomes in the limb.[176]

Compression of the spinal nerve beyond the intervertebral foramen does not generate pain but only pins and needles, numbness and paresis. This is the case when a disc protrusion has passed very laterally, when the fifth lumbar nerve is compressed between a corporotransverse ligament and the ala of the sacrum,[56] or in some spondylolytic compressions of the nerve root. Experience during the performance of a sinuvertebral block also confirms the insensitivity of the nerve root fibres. When the needle, just before touching the posterior aspect of the vertebral body, brushes against the nerve root, no pain but a sharp 'electric' shock results. As the dural investment of the root ends at the same level, it must be concluded that the latter is responsible for the radicular pain in sciatica.[175]

Nerve root

The structure of the nerve root differs from that of the peripheral nerves in three ways: the epineurium is less abundant, the fasciculi do not branch and the perineurium is missing. Thus, compared with a peripheral nerve, the parenchyma of the nerve root is more susceptible to injury, by either mechanical or chemical irritation.[177]

Irritation of the parenchyma leads to *paraesthesia*. Unlike 'radicular' pain, which is merely a symptom of compression of the dural sheath, pins and needles indicate that the nerve fibres are irritated as well. Paraesthesia is thus a symptom of direct involvement of the nerve root. Further irritation and destruction of the neural fibres leads to interference with conduction, resulting in a *motor and/or sensory deficit*. The fact that the motor and sensory components of the nerve root remain completely separated during the course of the nerve root along the radicular canal has some clinical consequences: it is possible for a nerve root compression to cause a pure motor paresis or a pure sensory deficit. If pressure is exerted from above, sensory impairment may result, whereas an impingement from below can induce a motor paresis. A larger protrusion, pressing between two roots, can result in a motor palsy of the root above, together with a sensory deficit of the nerve root below (Fig. 31.26).

Controversy still exists over the mechanism of nerve root compression by a protruded disc. Inman and Saunders[178] stated that the nerve root is rarely 'compressed' between anterior and

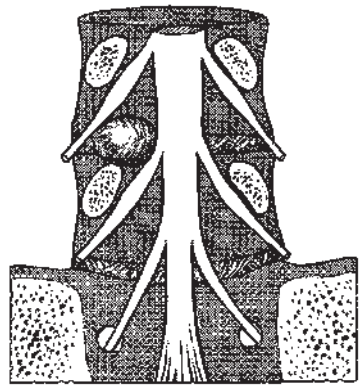


Fig 31.26 • Protrusion pressing between two roots.

posterior walls, but is merely brought under tension by the disc herniation. Others have observed that the extrathecal, intraspinal nerve root is relatively fixed to the anterior wall and the intervertebral foramen by the dural ligamentous complex and the foraminal complex (see p. 429).[171,179] Therefore, this particular part of the root cannot easily slip away from a disc protrusion and is tethered over it, and a pressure-induced nerve lesion can develop.[168]

These anatomical findings probably help to explain why the magnitude of signs and symptoms in sciatica does not necessarily correspond to the magnitude of the disc protrusion, and also why many asymptomatic protrusions exist. The amount of interference with conduction is related to the degree of the compressing force, which in turn depends not only on the magnitude of the protrusion but also on the tightness of the dural fixation to anterior wall and intervertebral foramen.

The involvement of nerve fibres is tested during clinical examination: resisted movements and the reflexes test the integrity of the motor fibres, while cutaneous analgesia indicates loss of sensory conduction. Interference with conduction suggests that an attempt to achieve reduction by manipulation or traction will fail. In general, a disc lesion affects only one nerve root and the neural effects are rather subtle. As described above, combinations of sensory and motor effects or their independent existence may occur. It is also possible for two roots to be pinched by one disc protrusion, which can be the case at the L4 level where a combined fourth–fifth palsy can arise, probably resulting in drop foot, or at the L5 level where a combined fifth lumbar–first sacral deficit can occur.

Massive pressure may finally cause ischaemic root atrophy and then complete loss of sensitivity of the sheath. Reflex hamstring contraction to protect the nerve root no longer takes place and straight leg raising becomes full-range, despite the massive disc protrusion and the complete lesion.

Conclusion

For clinical purposes it is as well to divide the components of the nerve root into an external aspect (the sheath), which is mobile and is responsible for pain, and an internal aspect (the nerve fibres), which serves conduction only. This helps to distinguish the symptoms and signs of each, so permitting proper assessment of the location of a lesion, the magnitude of compression and the degree of functional incapacity (Box 31.2).

In nerve root compressions by a displaced disc, the development of symptoms and signs allows the anatomical changes in the radicular canal to be followed: slight pressure will only involve the sheath of the root (Fig. 31.27), giving rise to pain in the corresponding dermatome and probably impaired mobility, reflected by alterations in straight leg raising. Greater pressure will result in pressure on nerve fibres, reflected in paraesthesia at the distal end of the dermatome. Clinical examination will now reveal not only interference of mobility but also impaired conduction – a sensory deficit and/or loss of motor power. Greater pressure causes root atrophy, which results in loss of sensitivity of the dural sheath and gives a painless straight leg raising test. At the same time the sensory deficit and motor palsy become complete (Fig. 31.28).

Box 31.2

Neurological deficit at each level

L1 Compression of the L1 root produces neither paraesthesia nor muscle weakness and cutaneous analgesia is only found below the inner half of the inguinal ligament

L2 Involvement of the L2 root causes paraesthesia and analgesia over the anterior aspect of the thigh, from the groin to the patella. Muscle weakness is found in the psoas

L3 Interference of conduction in the L3 root causes paraesthesia at the anterior aspect of the leg from the distal third of the thigh, over the knee and the lower leg, down to the ankle. Cutaneous analgesia extends from the patella along the front and the inner aspects of the leg, and ends just above the ankle. The weak muscles are psoas and quadriceps and the knee jerk is sluggish or absent

L4 Compression of L4 has the following clinical signs: paraesthesia at the outer leg and the big toe, sensory deficit at the lateral aspect of the lower leg, over the foot up to the big toe, and weakness of the extensor hallucis and the tibialis anterior muscles

L5 Involvement of the L5 root results in paraesthesia at the outer leg, the front of the foot and the big and two adjacent toes, and cutaneous analgesia of the outer leg, the dorsum of the foot and the inner three toes. Weakness is found at the extensor hallucis longus, the peroneal and gluteus medius muscles

S1 Compression of the S1 nerve root shows the following signs: paraesthesia at the two outer toes, numbness at the calf, the heel and the lateral aspect of the foot. The weak muscles are the calf muscles, the hamstring, the gluteus maximus and the peronei

S2 Involvement of the S2 root results in paraesthesia at the heel and cutaneous analgesia at the posterior aspect of the thigh, the calf and the heel. The calf muscles, the hamstrings and the gluteal mass are weak

S3 Neural deficits cannot be detected in S3 lesions

S4 Parenchymatous lesions of the S4 root result in paraesthesia in the perineum, vagina or penis, anal analgesia, and functional disorders of the bladder and rectum, usually incontinence

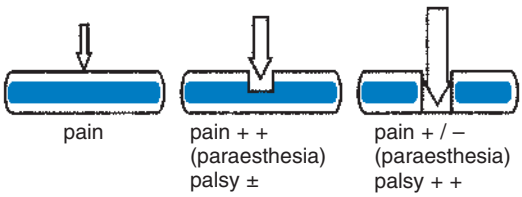


Fig 31.27 • Symptoms and signs of a compressed nerve root vary according to the intensity of compression.

Epidural space

The virtual space between the dural sac, the dural sheaths of the nerve roots, and the spinal canal is the epidural space. This space is quite narrow because the dural sac lies very close to the boundaries of the vertebral canal and is filled with a network of loose connective tissue, fat, arteries and a dense network of veins.[180]

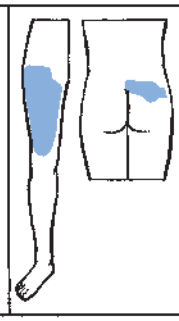
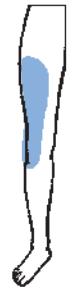
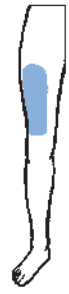
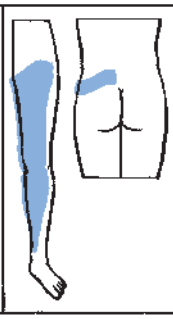
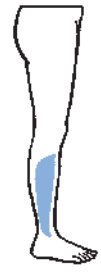
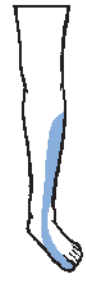
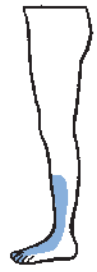
Sheath			Nerve fibres			
	Symptom	Sign	Symptom	Signs		
Root	Pain	Mobility	Paraesthesia	Motor deficit	Sensory deficit	Reflex disturbances
L1		None	None	None	Inner half inguinal ligament	None
L2		None (femoral stretch)		Psoas		None
L3		Femoral stretch		Psoas quadriceps		Knee jerk
L4		SLR		Tibialis anterior, Extensor hallucis longus		Knee jerk
L5		SLR		Extensor hallucis longus, Peronei, Gluteus medius		Ankle jerk

Fig 31.28 • Symptoms and signs of nerve root compression at each level. SLR, straight leg raising.

Continued

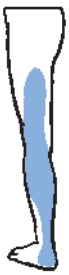
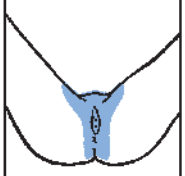
	Sheath		Nerve fibres			
	Symptom	Sign	Symptom	Signs		
Root	Pain	Mobility	Paraesthesia	Motor deficit	Sensory deficit	Reflex disturbances
S1		SLR		Peronei, Calf muscles, Hamstrings, Gluteal muscles		Ankle jerk
S2		SLR		Calf muscles, Hamstrings, Gluteal muscles		Ankle jerk
S3		None	None	None	None	None
S4		None	Perineum	Sphincters	Perineum	None

Fig 31.28 Symptoms and signs of nerve root compression at each level (continued). SLR, straight leg raising.

The sinuvertebral nerve is in the anterior half of the epidural space. The venous system is extensive and valveless, with multiple cross-connections. Batson[181] has described retrograde venous flow from the lower pelvis to the lumbosacral spine, which probably provides the route for metastases and infections spreading from the pelvic organs to the spine.

Innervation

The spine is innervated by the sinuvertebral nerve and the posterior primary ramus. All the tissues lying posterior to the plane of the intervertebral foramina at each level (i.e. the facet,

the vertebral arch, the related tendinous and aponeurotic attachments and the flaval and interspinous ligaments) are innervated from the posterior primary rami. Those anterior to the intervertebral foramina (longitudinal ligaments, anterior dura and dural sleeves) are supplied by branches of the sinuvertebral nerves (Wyke, cited by Cyriax[182]).

Sinuvertebral nerve

The sinuvertebral nerve was first described by Luschka in 1850.[183] It emerges from the anterior aspect of the spinal nerve, distal to the nerve ganglion, and receives some sympathetic branches from the ramus communicans[22] (Fig 31.29). In the fetus the nerve is composed of several filaments which may become bound together during later life, to form the adult sinuvertebral nerve.[146] The composite nerve is between 0.5 and 1 mm thick,[184] passes through the intervertebral foramen and points upwards around the base of the pedicle, to pass along the cranial side of the corresponding disc to reach the medial aspect of the posterior longitudinal ligament. Here it divides into ascending, descending and transverse branches, which anastomose with the sinuvertebral nerves of the contralateral side and with those from adjacent levels. Therefore, instead of a recognizable nerve trunk, the sinuvertebral nerve is represented by a network of overlapping fine filaments from different levels and from both sides (Fig. 31.30).[185]

Branches of the sinuvertebral nerve supply the vertebral body, the outermost layers of the annulus fibrosus, the posterior longitudinal ligament, the anterior aspect of the dural sac and the dural investments around the nerve roots.[186] Branches of the sinuvertebral nerve also surround the blood vessels of the vertebral canal. The posterior aspect of the dura is devoid of nerve endings. There is still disagreement as to whether the ligamentum flavum and the lamina are innervated by the sinuvertebral nerve.

Posterior primary ramus

Distally from the intervertebral foramen, the spinal nerve divides into a large anterior branch and a smaller posterior ramus (Fig. 31.31). The latter divides almost immediately into a medial and a lateral branch,[187] although a smaller intermediate branch has also been identified.[92]

The medial branch descends posteriorly to the transverse process, where it lies in a groove formed by the junction of the superior articular and transverse processes. A strong fibrous

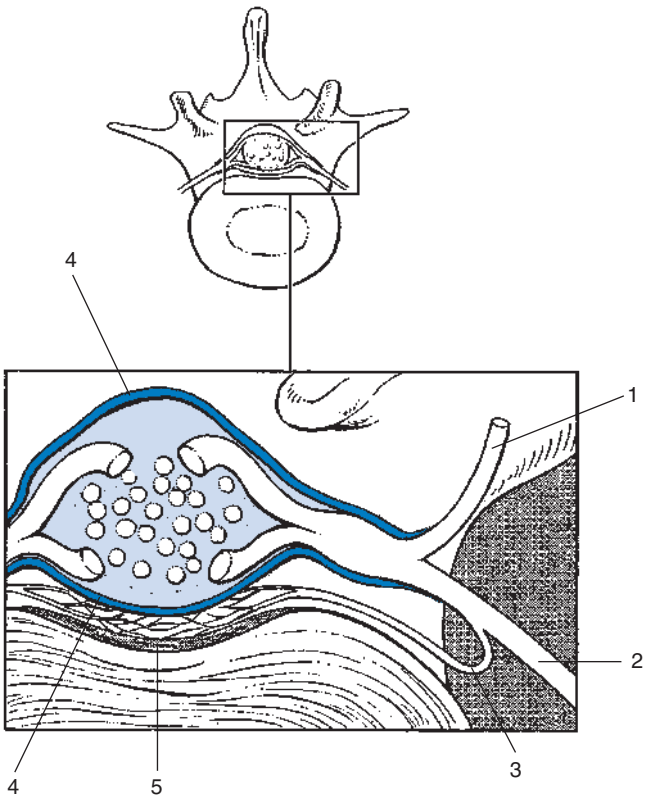


Fig 31.29 • Sinuvertebral nerve: 1, posterior ramus; 2, anterior ramus; 3, sinuvertebral nerve; 4, dura mater; 5, posterior longitudinal ligament.

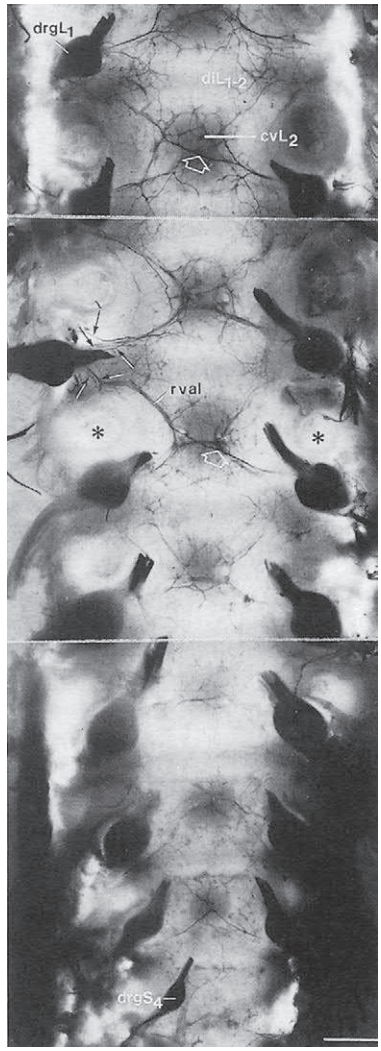


Fig 31.30 • Dural nerve branches. *Cut pedicle of a vertebral arch; cv, vertebral body; di, intervertebral disc; drg, spinal ganglion; rv, ventral ramus of spinal nerve. Reproduced with permission from Groen.[154]

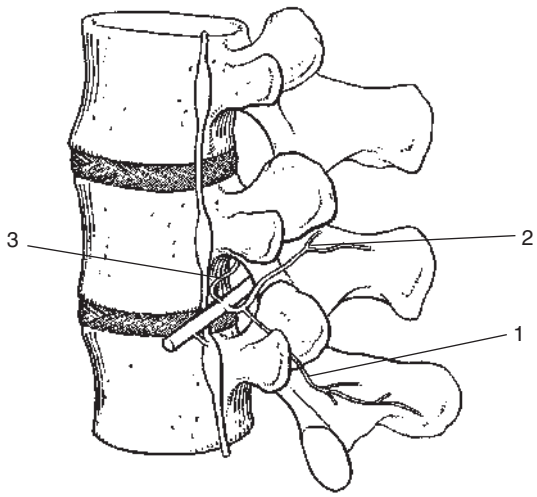


Fig 31.31 • Posterior primary ramus: 1, medial branch; 2, lateral branch; 3, sinuvertebral nerve.

band transforms this osseous groove into an osteofibrous tunnel. At this level a branch innervates the inferior part of the articular capsule of the facet joint. The nerve continues its course caudally on the lamina, to supply the dorsal muscles and the superior part of the articular capsule of the facet joint of the level below.[188]

Each medial branch thus supplies the facet joints above and below its course. Consequently, each facet joint is innervated by two consecutive medial branches.[189,190]

The lateral branch of the posterior ramus emerges between the deep layer of the lumbodorsal fascia and the lateral edge of the lamina. It supplies the muscles and the fascia. The lateral branches of the ramus posterior have cutaneous nerves and reach distally as far as the greater trochanter.[148]

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32

Ageing of the lumbar spine

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Introduction

The human spine functions in a reverse way to that for which it was originally developed: instead of a hanging bridge connecting the anterior to the posterior limbs of a quadruped, it has become a vertical column of a biped, fundamentally changing the biomechanics and imposing larger loads on the lower lumbar discs. Humans' upright posture has long been blamed as the main cause of the development of early disc degeneration: 'intervertebral disc disease is the price we pay for the gift of the erect posture.'[1]

Disc degeneration in animals is very unusual.[2] Neither have localized bulging and Schmorl's nodules ever been observed in quadrupeds.[3] However, a quadruped forced to adopt an erect position quickly develops structural changes in the annulus fibrosus and nucleus pulposus.[4–6] Large disc herniations at the lumbosacral level also occur in 25% of bipedal rats.[7] It seems obvious, therefore, that the upright position and the consequent increase in static loading of the disc[8] are the main causes of the early and progressive development of degeneration in the human lumbar spine.

Degenerative changes in the intervertebral discs are so common that they can be regarded as normal physiological processes. Therefore we do not use the term intervertebral disc 'disease' but prefer to speak of intervertebral disc 'ageing'.

Degeneration of the lumbar discs starts very early in life. Clear microscopic signs of degeneration have been demonstrated in a 4-year-old.[9] Several reports of prolapsed discs in children and adolescents show that major disc degeneration appears at or before puberty[10–14] and it has been maintained that after the age of 30 years there is no lumbar disc which does not show some degenerative changes.[15] There seems to be no way to escape this degeneration because it affects all, whatever their weight, physical build or athleticism.[16]

Ageing of the disc

Mechanism

Both the erect posture, which imposes heavier mechanical stresses on the spine, and the decreased regenerative capacity of the disc make it very vulnerable. As has been described (see Ch. 31), the disc depends for its nutrition on diffusion through the endplates and annulus, which can only take place as an outcome of change in intradiscal pressure within a physical range (Fig. 32.1). Consequently, both excessive forces and immobilization will have deleterious effects on disc tissue.[17] A modern, mostly sedentary lifestyle, for example, puts continuous increased

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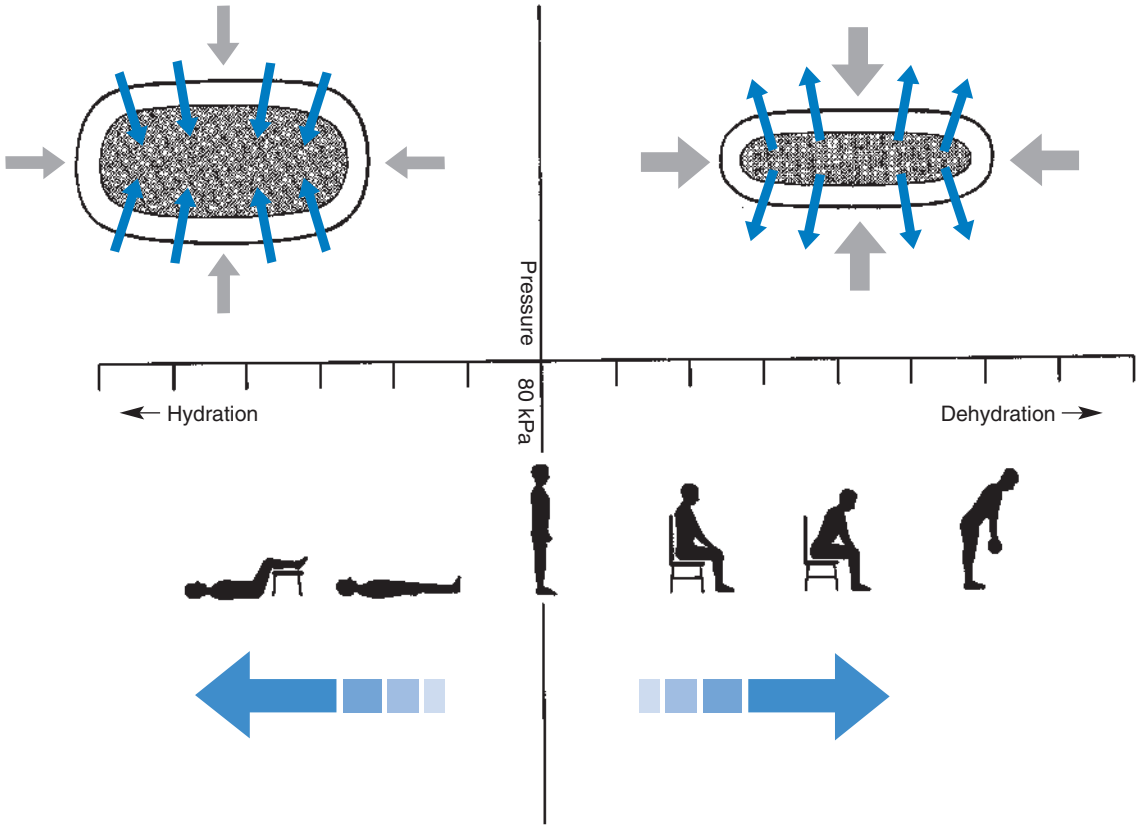

Fig 32.1 • Hydration–dehydration changes with load and consequently with activity and posture.

pressure on the disc; it is then constantly kept low in water content, which prevents the transport of nutrients. As the cells are undernourished, the fibrils and ground substance become damaged and their chemical composition changes.[18] Enzymatic depolymerization of collagen and mucopolysaccharide causes a decrease in weight and content that results in a drop in oncotic pressure, which leads to further loss of water.[19] The drying out of the disc is the most characteristic sign of intervertebral degeneration. The loss of hydrostatic pressure in an ageing disc is also reflected in the change in daily variations in body height: in adolescence the difference in body height between morning and night is 2%, but this drops to 0.2% in adults.[20] The mature disc is thus permanently dried out and lacks the osmotic elasticity of the young disc.

Loss of water-binding capacity is also responsible for the decrease of disc turgor and loss of endurance. The disc does not sustain the normal loads and the hydration–dehydration curve shifts in the direction of further dehydration (Fig. 32.2). A vicious circle of further degeneration, further decrease in molecular weight and further fall in water-binding capacity is set up (Fig. 32.3).[21]

With advancing age, the permeability of the vertebral endplates also decreases. The accompanying calcification compromises the nutrient supply to the adjacent nucleus, so enhancing further disc degeneration.[22,23] The early stage of the changes in vertebral bone structure is first seen on magnetic resonance imaging (MRI).[24] Vertebral endplate sclerosis later becomes radiographically visible.[25]

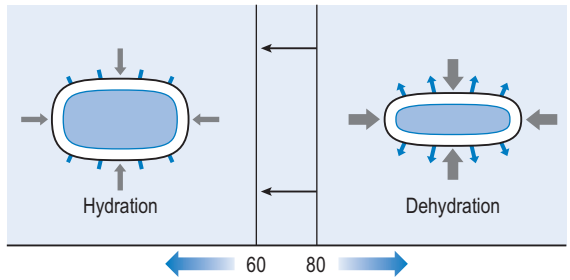

Fig 32.2 • Loss of water-binding capacity leads to loss of endurance; the disc cannot sustain normal loads and the hydration–dehydration curve shifts further in the direction of dehydration.

Another consequence of the decrease of oncotic pressure is loss of disc height and narrowing of the intervertebral space, which in turn influences the function of the facet joints and the anatomical relationships of the intervertebral foramina.

Structural changes

The intervertebral disc is characterized by a tension-resisting annulus fibrosus and a compression-resisting nucleus pulposus. As the nucleus pulposus dries out, it becomes less able to exert fluid pressure. The annular fibres, no longer under constant tension in relation to the nucleus, bear a greater share of vertical load and are subjected to bulk deformations and shear

strains.[26] This leads to tears in the annular fibres.[27] The posterior annulus, especially the area between annulus and nucleus, is subjected to most of the mechanical strain.[28] As this posterior boundary zone is also most exposed to nutrient deficiency,[29] it will be the first to wear through, so that small radial fissures appear.[30,31] Further softening and loosening of annular tissue leads to expansion of these fissures and the formation of cavities and cysts, which are radiographically seen as air pockets.[32] Nuclear displacements through these fissures will lead to a further decrease in disc height. As a result, the intervertebral joint becomes unstable and is subjected to more bulk deformation and more shear strain. Ruptures permit further migration of the disc material in the direction of least resistance (mostly backwards). The result is intradiscal displacements, bulging beyond the vertebral borders and disc prolapses (see Ch. 33).

Ageing of the disc also involves structural changes in the endplate. Between the ages of 20 and 65, the endplate becomes thinner and cell death occurs in the superficial layer of the cartilage.[33] The vascular channels in the subchondral bone of the endplate are gradually occluded, finally leading to complete endplate sclerosis. As the endplate plays an important role in the nutrition of the disc, changes in it will contribute to further degeneration of the whole intervertebral disc. Also, the strength of the vertebral endplate decreases with age, which renders it more liable to (micro)fractures. Microscopic defects may be followed by invasion of the disc by young blood vessels and connective tissue cells.[34] The latter undergo metaplasia into cartilage cells and granulation tissue, and adult connective tissue also forms.[35] This 'invading' connective tissue can spread out into the disc, create zones of decreased resistance and cause further weakness and instability. It has even been hypothesized that this 'new' proliferative tissue plays a part in disc herniations.[36]

The macroscopic degeneration of the disc was divided into four grades by Friberg and Hirsch[37] and later by Nachemson.[38] This scheme for assessing the gross morphological changes in the lumbar intervertebral discs has recently been improved by Thompson *et al*,[39] who consider five morphological grades (Table 32.1 and Fig. 32.4).

Macroscopic grades of disc degeneration have been correlated with age, sex and spinal level for 600 lumbar intervertebral discs from 273 cadavers in a review by Miller *et al*.[40] Their conclusion was that, by the age of 50 years, 97% of all lumbar discs exhibit degeneration and that the L3–L4 and L4–L5 levels usually contain the most severely degenerated discs. Another study demonstrated that there is disc degeneration in at least one lumbar level in 35% of subjects between 20 and 39 years of age, and that all subjects 60–80 years of age show disc degeneration.[41]

These figures establish that disc degeneration is a normal age-dependent pathophysiological process; it therefore cannot be regarded as a 'disease' but is a normal biological event from which no one will escape.[42]

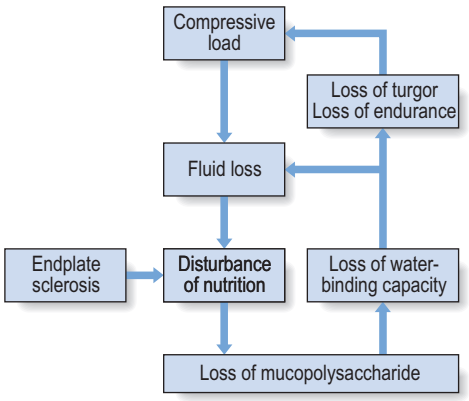

Fig 32.3 • Vicious circle of disc degeneration.

Disc displacements

Fissures in the annulus fibrosus will inevitably lead to changes in the biomechanical behaviour of nucleus and annulus, which can then result in intradiscal nuclear displacements, protrusions and prolapses (Fig. 32.5).

The nucleus and inner annulus do not contain free nerve endings. Displacements of these structures are therefore painless, except when they irritate other more sensitive tissues, such as the outer annulus, the dura and the nerve root sleeves.

Table 32.1 Description of the morphological grades

Grade	Nucleus	Annulus	Endplate	Vertebral body
I	Bulging gel	Discrete fibrous lamellae	Hyaline, uniformly thick	Margins rounded
II	White fibrous tissue peripherally	Mucinous material between lamellae	Thickness irregular	Margins pointed
III	Consolidated fibrous tissue	Extensive mucinous infiltration; loss of annular–nuclear demarcation	Focal defects in cartilage	Early chondrophytes or osteophytes at margins
IV	Horizontal clefts parallel to endplate	Focal disruptions	Fibrocartilage extending from subchondral bone; irregularity and focal sclerosis in subchondral bone	Osteophytes less than 2 mm
V	Clefts extend through nucleus and annulus		Diffuse sclerosis	Osteophytes greater than 2 mm

Reproduced with pernission from Thompson *et al*.[39]

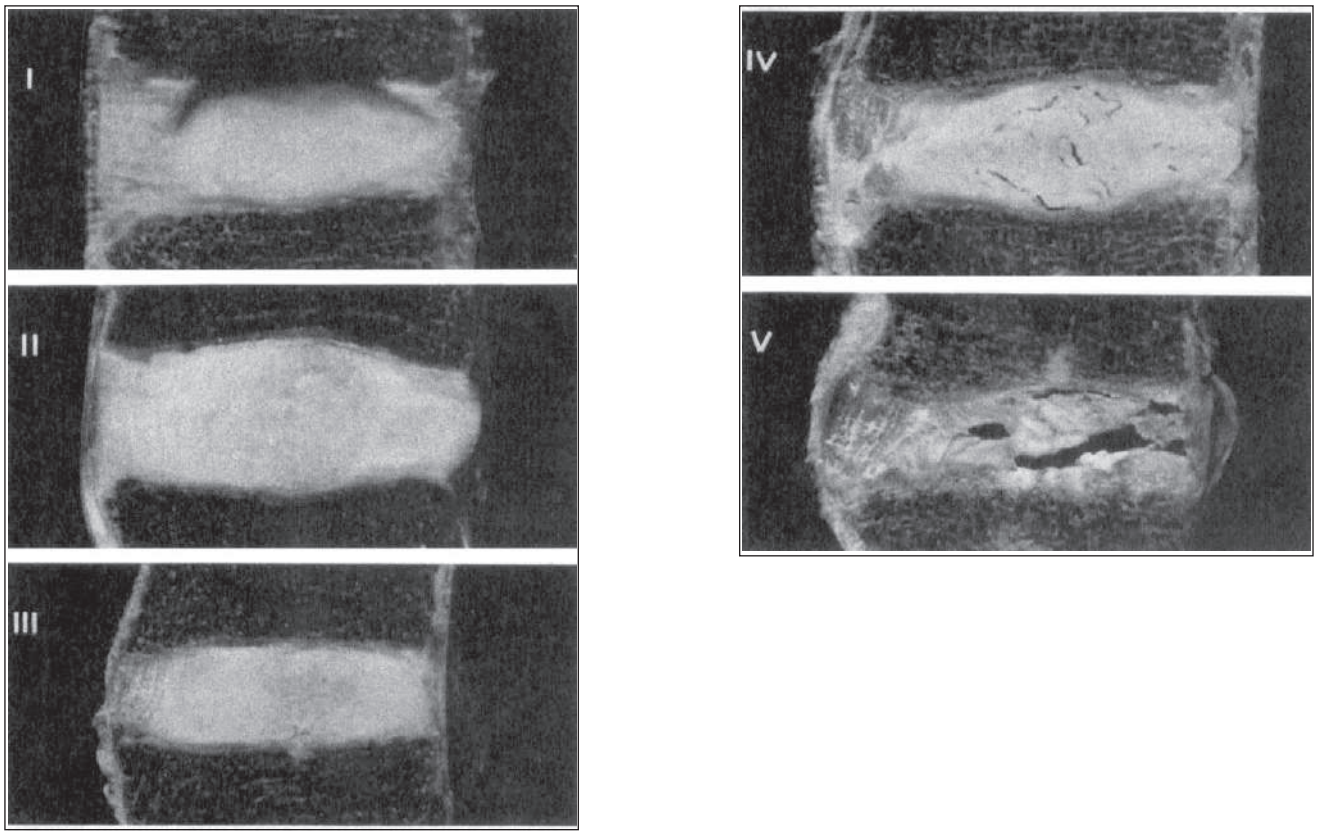

Fig 32.4 • The appearance of a typical disc of each grade. From Thompson *et al*,[39] with permission.

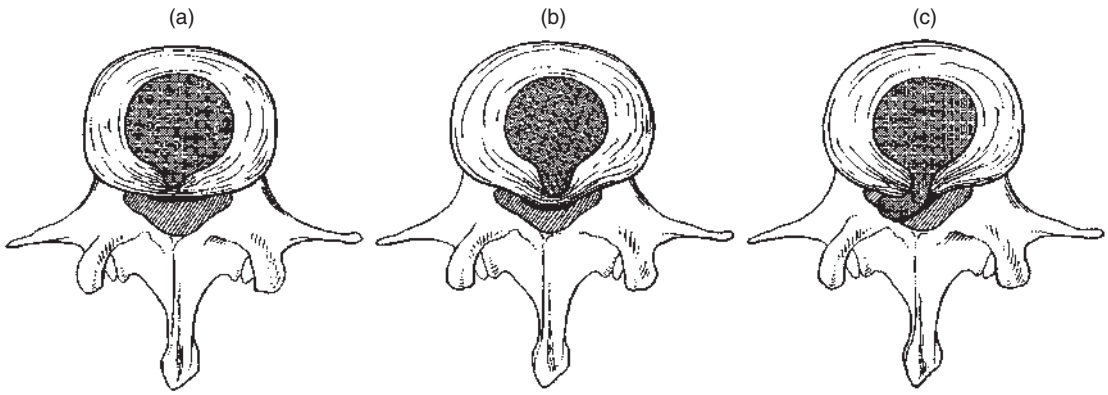

Fig 32.5 • Disc displacement: (a) displaced tissue within the disc; (b) bulging of the intervertebral disc without perforation of the annulus fibrosus; (c) herniation of intervertebral disc tissue through the annulus fibrosus into the spinal canal (prolapse).

Therefore it is not the size of the migration that is important but the degree of irritation of the pain-sensitive tissues. Because imaging techniques (computed tomography (CT), CT discogram and MRI) can only detect the size and localization of the bulges, and not always their effect on surrounding tissues, many more disc displacements are detected during imaging than actually cause symptoms.[43,44] More than 50% of asymptomatic patients over 40 years of age have either alterations in or displacements of disc.[44] Single or multilevel disc bulging is visible on the MRIs of between 28 and 85% of the adult population who do not have activity-limiting low back pain.[45–48]

Cyriax[49] listed five different directions in which discal tissue can move: posterior, posterolateral, anterior, vertical and circular. Each type of displacement occurs during a specific period in the degeneration of the disc. Posterior and posterolateral displacements are typical in the third, fourth and fifth decades. Anterior displacement resulting in osteochondrosis is a disorder of teenagers, whereas anterior displacement in combination with disc resorption is a lesion of the elderly and is seldom

seen below the age of 50. It is surprising to find that low back syndromes (lumbago, backache, sciatica) are age-related,[50,51] whereas the incidence of fissures, ruptures and degeneration of the disc increases linearly during the whole of life. Extensive pressure within the nucleus pulposus is higher in individuals between 30 and 50 years of age than in the elderly (Fig. 32.6).[52] The conclusion is that posterior displacement of the disc will be more likely to appear when increased intradiscal pressure coincides with some damage to the posterior annular fibres. In other words, the combination of intradiscal pressure and lowered resistance of the annulus fibrosus is the main biomechanical factor contributing to protrusion–prolapse. When the expansive forces of the nucleus pulposus decrease and, because of the loss of water, the disc gradually deflates, the tendency to displacement diminishes.[53,54] Other factors protecting the ageing disc against displacements are increasing stiffness of the posterior ligaments, which limit spinal mobility, and the formation of osteophytes, which distribute the load over a larger area.

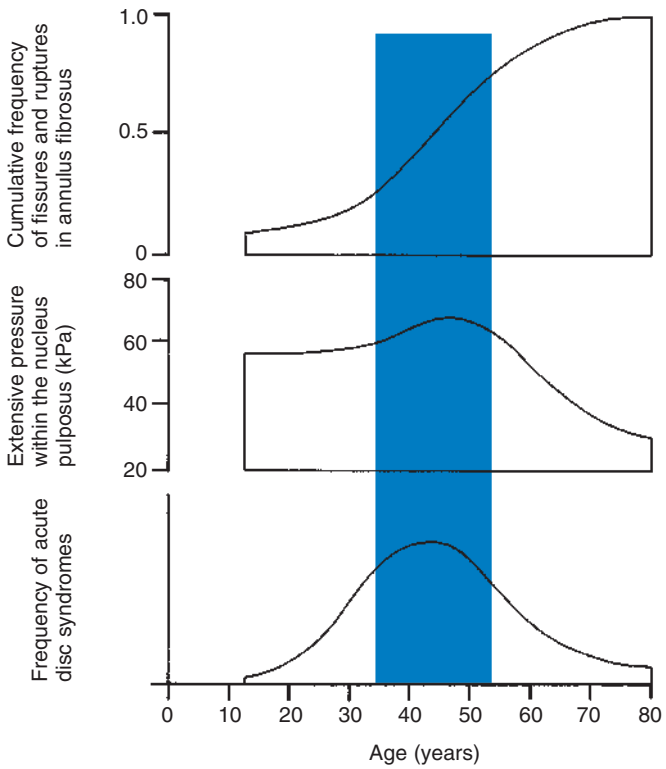

Fig 32.6 • The relationship of age to acute disc syndromes. From Krämer J. *Intervertebral Disc Diseases, Causes, Diagnosis, Treatment and Prophylaxis*, 1981, Elsevier, Stuttgart, with permission.

Posterior displacement

The posterior boundary zone between the annulus and the nucleus is the first part to develop areas of degenerative change and fissures (see p. 438). In sitting and forward bending, the disc is asymmetrically loaded, with a compression strain anteriorly and a tensile stress dorsally. Because of the increase of intradiscal pressure, the nucleus pulposus is pushed dorsally and applies more stress to the weakened posterior fibres, which are already subjected to the strong tensile stress imposed by the flexion.[55]

Posterior migration of disc tissue beyond the posterior limits of the intervertebral joint space causes tension on the outer annulus and the posterior longitudinal ligament. Irritation of ligament, dura mater and the Hofmann complex combines to produce *backache* (Fig. 32.7a). The patient's history usually suggests the type and localization of the displacement and the degree of dural irritation: progressively increasing backache usually stems from a nuclear displacement. A sudden shift indicates that the material that has moved is of harder, annular consistency. If the bulge irritates the dura centrally, the pain is central, probably with some bilateral gluteal radiation. If it lies more to one side, unilateral pain and pain in one buttock result.

Further irritation of the dura mater results in a clinical picture that is called *acute lumbago* (Fig. 32.7b). In a cartilaginous displacement, the patient states that bending, lifting or standing, after sitting for a while, resulted in acute and severe pain. Alternatively, the pain and disability have come on gradually, some hours after a period of stooping or sitting, which suggests protrusion of part of the nucleus. In both events, a constant ache is experienced which forces a pain-avoiding

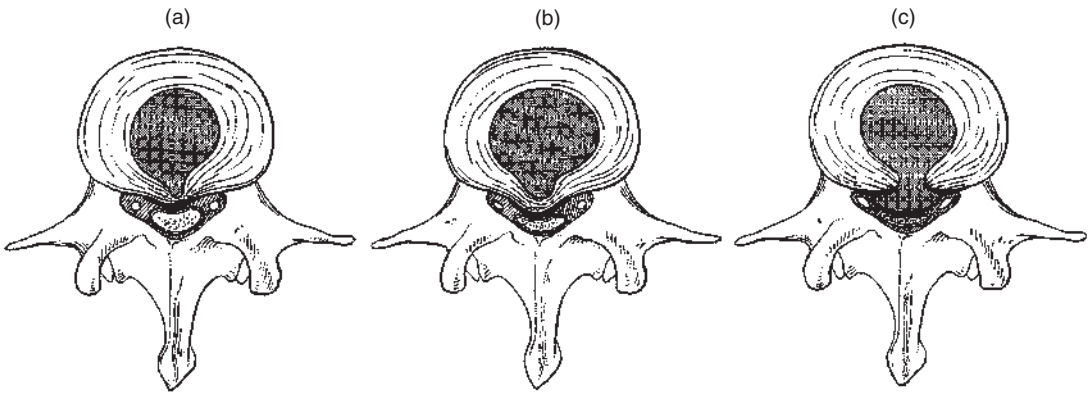

Fig 32.7 • Posterior disc displacement in (a) backache; (b) acute lumbago; (c) massive posterior protrusion (S4 or cauda equina syndrome).

position, usually in flexion and/or lateral deviation. The pain is agonizing and radiates to one or both legs in an extrasegmental way. Dural symptoms (pain on coughing and sneezing) are positive, as are dural signs (limited straight leg raising and lumbar pain on full neck flexion), indicating that the dura is under constant and severe stress.

In backache and lumbago, the posterior displacement puts the posterior longitudinal ligament under tension, which causes counterpressure on the bulge. This explains why acute lumbago usually ceases after a lapse of time, especially when the intradiscal pressure is reduced by lying down and avoiding sitting or bending.

A *massive posterior protrusion* may cause a rupture of the posterior longitudinal ligament. The entire cauda equina will then be compressed between the protrusion and the laminae. This not only squeezes the nerve roots on both sides but also causes compression and atrophy of the centrally localized S3 and S4 roots, with disturbance of bladder and rectal function (the S4 syndrome or cauda equina syndrome) (Fig. 32.7c).[56,57]

Posterolateral displacement

Root pain results from pressure of the prolapsed or protruded disc against the dural sleeve of the nerve root and subsequent irritation of the latter (Fig. 32.8). This may be primary or secondary.

Secondary posterolateral protrusion

This is more common. The root pain starts after the patient has suffered a number of attacks of lumbago or backache. After the most recent attack, the counterpressure exerted by the posterior longitudinal ligament has not resulted in the usual reduction of the bulge but rather in a lateral shift which impinges on the nerve root. The back pain will have passed, to be replaced by pain in the limb. According to the severity of irritation, which is determined not only by the degree and consistency of the bulge but also by the tightness of the 'tethering ligaments', the size of spinal canal and the degree of inflammatory response, the symptoms and signs are related to impairment of mobility of the dural sleeve (pain and limitation of straight leg raising) alone, or interference with conduction (paraesthesia, numbness and motor deficit).

Primary posterolateral protrusion

In this case, the pain usually begins in the calf and the posterior thigh; the absence of previous backache indicates that the dura has not been previously compressed. This condition usually affects young adults (aged 18–35 years). The signs are radicular, with pain and paraesthesia in the affected dermatome and limitation of straight leg raising. Signs of impaired conduction at the affected root are exceptional.

Anterior displacement in adolescents

Because of its high oncotic and hydrostatic pressure, the young disc sometimes invades the vertebral endplates. This occurs particularly in the regions where the endplates have low resistance against increased anterior loading.[58,59] The nucleus pulposus may then move forwards between the cartilaginous endplate and the bone of the vertebral body, causing injury to the growth zone of the vertebra (Fig. 32.9). The result is a developmental deformity of the anterior part of the vertebra. During the course of this, protrusion may separate a small triangle of bone at the anterior corner of the vertebral body (limbus vertebra).[60] If the osteochondrosis involves several consecutive vertebrae, a kyphotic deformity results (Scheuermann's disease).[61]

Such deformities occur only during the period of spinal growth and are asymptomatic. Care must therefore be taken not to overemphasize the significance of incidental radiological findings.[62]

Anterior displacement in the elderly

Tears at the anterior and anterolateral part of the disc lead to both anterior and anterolateral herniation. Anterior displacement of the disc causes dissection of the anterior longitudinal ligament from its bony attachments, which stimulates bony outgrowths along the anterior and anterolateral aspects of the vertebral body. These first appear in a horizontal direction and then vertically, following the course of the anterior longitudinal ligament (see p. 423). In advanced cases, the whole disc is displaced anteriorly. On a lateral radiograph this can be seen

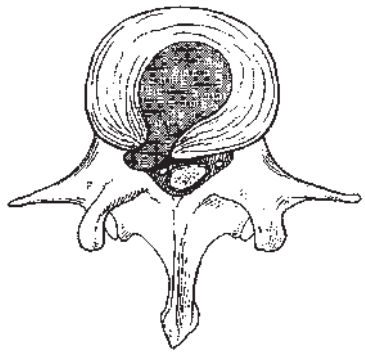

Fig 32.8 • Posterolateral disc displacement.

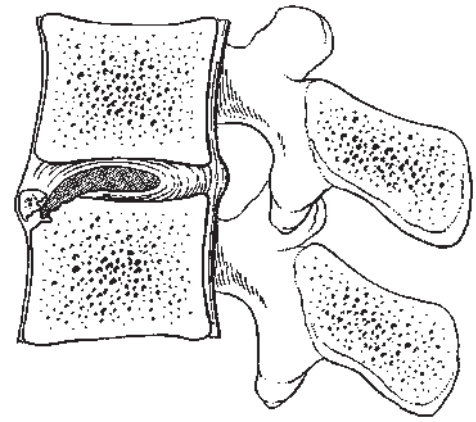

Fig 32.9 • Intravertebral herniation in adolescence.

as two 'beaks' containing the disc, while the intervertebral space is so narrowed that the vertebral bodies come to lie in apposition.[63] Cyriax called this situation the 'mushroom' phenomenon, because of the characteristic appearance of the osteophytes, surrounding a 'mushroom' – the anteriorly displaced disc (Fig. 32.10).[64] The formation of the protrusion itself is completely painless but the gross narrowing of the intervertebral space, the folding of the posterior longitudinal ligament and the enlarged arthrotic posterior joints can cause considerable narrowing of the spinal canal and the lateral recess, which may result in compression of dura or nerve root when the spine is subjected to an axial load.

Vertical displacement

There are two types of vertical displacement: Schmorl's node and biconvex disc.

A herniation of the nucleus through the cartilaginous endplate is known as Schmorl's nodes (Fig. 32.11),[65] although it was first described by von Luschka in 1858.[66] After the nuclear material has invaded the cancellous bone, a reactive osseous shell forms, visible on a radiograph from the age of 17; this does not alter over the years. The disorder is very common at the lower thoracic and upper lumbar levels. Schmorl reported an incidence of 13% on radiographic examination of an adult and asymptomatic population but postmortem studies reported nodes in more than 75% of ordinary individuals.[67]

Biconvex discs appear as the result of traumatic or degenerative changes in the cartilaginous or bony endplates. Osteoporosis or osteomalacia causes a weakening of the bone and the disc balloons between two adjacent and slightly collapsed vertebrae.[68]

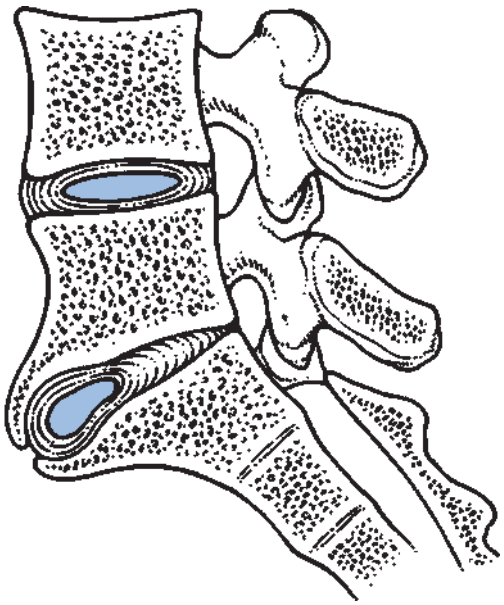

Fig 32.10 • Anterior displacement in the elderly: the 'mushroom' phenomenon.

Circular displacement

As a result of degeneration, weakening and drying out, the disc may bulge out all the way round the joint. The outward pressure pulls on the ligaments and lifts the periosteum off the bone. Formation of osteophytes at the anterior and lateral aspects of the vertebra results. These bony outcrops encircle the damaged disc. Mechanically, the presence of osteophytes is beneficial because they increase the weight-bearing surface and decrease the load per unit area.[69] They also decrease mobility. Both effects augment the stability of the joint.

Ageing of the surrounding tissues

Effects on the ligaments

The adjacent vertebral endplates develop poorly demarcated sclerotic densities, probably as the result of changed load distribution on the disc.[30] However, the most commonly observed radiographical manifestation of disc degeneration is osteophyte formation in the vertebral bodies.

The first sign of degeneration and instability of the disc is the formation of small *traction spurs*[70] which project horizontally to about 2 mm above and below the vertebral body (Fig. 32.12a). Their formation is explained as follows: decrease in turgor in the disc and the increasing anteroposterior instability which results cause the outermost fibres of the annulus, firmly attached to the undersurface of the epiphyseal ring, to exert traction on the periosteum. A horizontal lifting of the periosteum results in the formation of traction spurs.[71]

Spondylitic spurs (Fig. 32.12b) or claw spurs are very common in the elderly and there is a positive correlation between osteophyte size and patient age.[72] They have been reported to occur most frequently at L3–L4 in men who carry out heavy physical activity and in obese patients.[73] The claws form as the result of subperiosteal ossification at the anterior and anterolateral aspect of the vertebrae and their aetiology is considered to be follows: a softened and dehydrated disc bulges easily under the influence of external load and lifts the

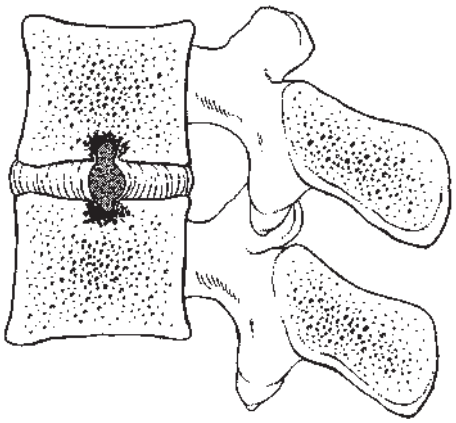

Fig 32.11 • Vertical displacement: Schmorl's nodes.

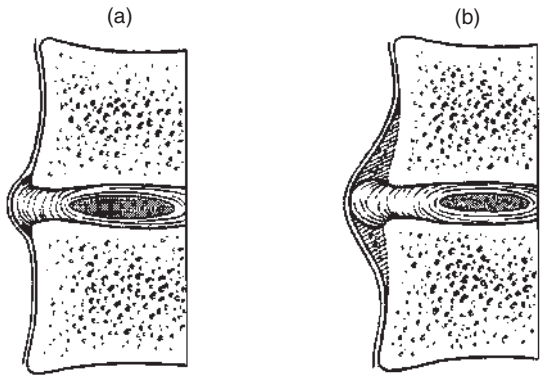

Fig 32.12 • Traction spur (a) and spondylitic spur (b).

longitudinal ligament and the periosteum from the vertebral margin. Osseous reaction takes place, provoking the formation of new bone in a beak-like protuberance.[74] This process can be viewed as if the vertebral body were trying to expand its articular surface area. By distributing axial loads over a wider area, the vertebral body lessens the stress applied to the annulus fibrosus and the spinal column becomes more stabilized.[75]

Effects on the facet joints

The disc and facet joints form a 'three-joint complex'. Degenerative changes affecting the disc are followed by increasing changes in the posterior joints. Arthrosis of a facet joint is thus always secondary to changes in mechanical load, induced by disc degeneration.[76,77] Like disc degeneration, facet arthrosis is a universal finding in the human lumbar spine. Evidence of arthrosis begins early, with more than one-half of adults younger than 30 years demonstrating arthritic changes in the facets. The most common arthritic level appears to be L4–L5.[78]

Initially, alterations in the height and volume of the disc result in positional changes of the facet joints. Axial compression on a degenerated and narrowed disc causes subluxation of the posterior joints: they move telescopically in relation to each other and slide past the normal range of movement, sometimes to such a degree that the tip of the inferior articular facet impinges on the adjacent lamina.[79]

A height reduction in the functional unit results in changes in the orientation of capsular fibres: they become stretched during axial loading, which gives the capsule a load-bearing function in the upright position.[80] The apophyseal joints resist about 16% of the intervertebral compressive forces in the erect standing posture if the disc is thinned.[81]

Another consequence of disc degeneration is that flexion–extension movement in the functional unit becomes disturbed. In a normal disc with adequate turgor, the instantaneous axis of rotation remains more or less in the region of the nucleus. In full flexion, it moves immediately anterior to the nucleus and in full extension it lies posterior to it.[82] In a normal joint, the vertebral bodies thus rock over the nucleus and the facet joints serve only to guide the movement. When disc degeneration is present, the axis of movement becomes irregular and completely unpredictable: sliding and anteroposterior

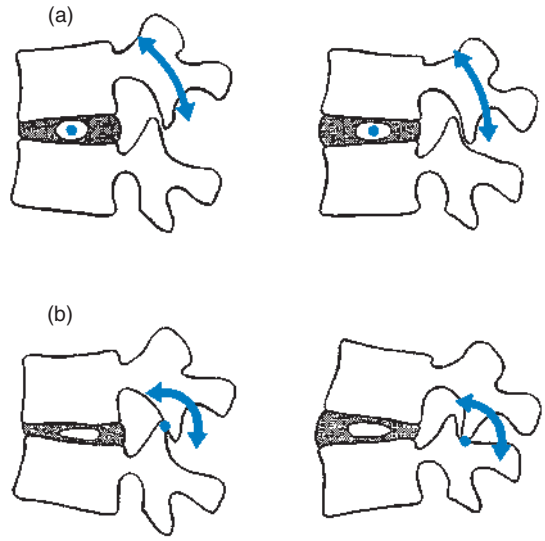

Fig 32.13 • In a normal functional unit the vertebral bodies roll over the nucleus and the facet joints only guide the movement (a). When disc degeneration is present, the facet joints are subject to rocking movements, resulting in compression and distraction (b).

movements, or tilting of the upper vertebra during flexion, become possible. Alternatively, the axis of rotation remains constant in position through or behind the posterior joints.[83] Then the facet joints not only guide and steady motion but are also subjected to a rocking movement, which results in compression of the articular surfaces and distraction of the capsule (Fig. 32.13).

These abnormal stresses cause laxity of the facet joints and the posterior ligaments. Together with the increasing laxity of the disc, the final result is instability of the functional unit, which in turn favours the development of internal derangement at the intervertebral disc.[84] This instability of the three-joint complex most commonly appears during a particular period in the cycle of degeneration, which normally falls between the third and fifth decades, when backache is most likely to occur.[50]

At the facet joints, the repeated pressure results in erosion and thinning of the cartilage. Where cartilage is lost, fibro-fatty intra-articular inclusions may increase in size to fill the space vacated by the cartilage.[85] Other regions may exhibit swelling, with subsequent formation of subperiosteal osteophytes and periarticular fibrosis.[86] Osteoarthrotic changes in the apophyseal joints finally result in gross enlargements of both inferior and superior facets, giving them a bulbous aspect. All these degenerative changes cause a substantial decrease in motion of the facet joints and, together with the stabilizing changes in the disc, they provide, at the end of the degeneration cycle, the ultimate stabilization of the functional unit.

Effects on the spinal canal and intervertebral foramen

Because of the change in pressure and volume of the lumbar disc, the position of the intervertebral joints changes so that

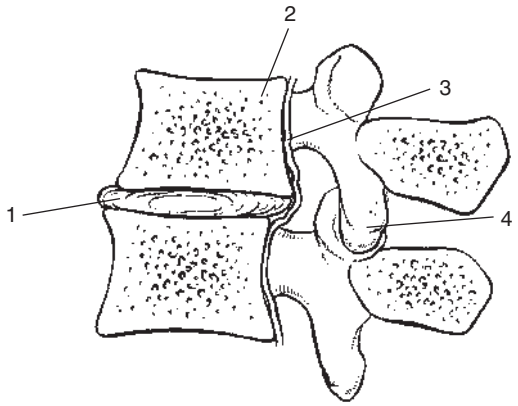

Fig 32.14 • Effects on the spinal canal and the lateral recess. 1, The vertebral bodies approach; 2, owing to the inclination of the joint surfaces, the upper vertebra is displaced backwards and produces a retrospondylolisthesis; 3, the posterior longitudinal ligament buckles; 4, the facet joints become hypertrophied and develop a bulbous aspect.

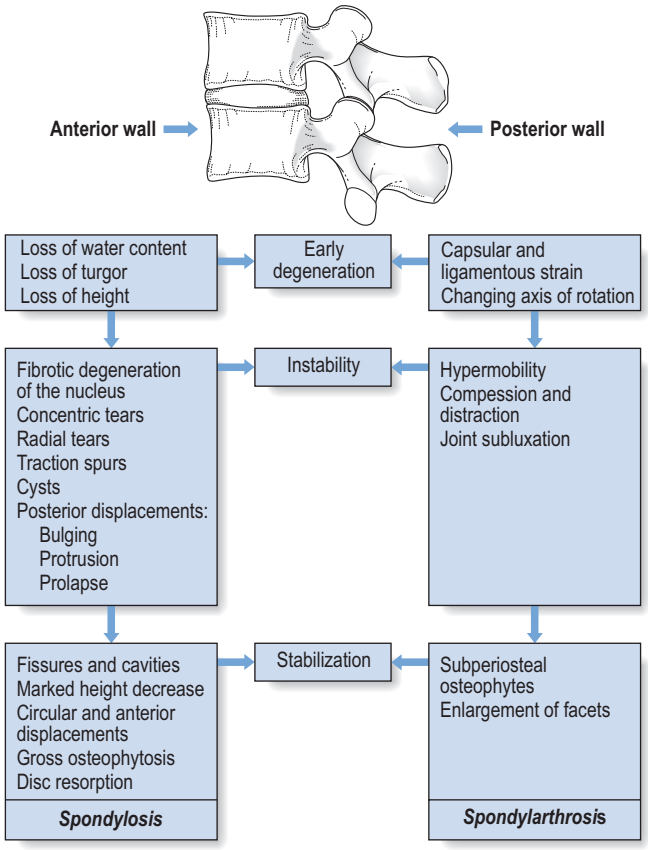

Fig 32.15 • Natural history of degeneration of the lumbar spine.

the articular processes override each other. As the vertebral bodies approach each other and because of the inclination of the joint surfaces, the upper vertebra moves backwards (retrospondylolisthesis) to the intervertebral foramen (Fig. 32.14). With increasing approximation of the vertebrae, both the ligamentum flavum and the posterior longitudinal ligament may buckle, so further narrowing the lumbar canal.[87] Further, hypertrophied and bulbous arthrotic apophyseal joints commonly project into the intervertebral foramina, where they may add to compression on the dural sac and nerve roots.[76]

The natural history of degeneration of the lumbar spine is summarized in Figure 32.15.

Radiological changes

The direct radiological signs of disc degeneration include disc space narrowing, gas within the disc space, vertebral sclerosis and osteophytes. Radiological signs of apophyseal joint degeneration are the same as seen in other degenerative joint diseases elsewhere in the body (joint space narrowing, sclerosis, osteophytosis and subluxation).

An ageing disc loses height because of a loss of water and altered biomechanics. Consequently, narrowing of the intervertebral disc space is the first radiographic sign of lumbar degeneration.

The presence of air pockets, located in discrete cavities within the nucleus, is evidence of long-standing disc degeneration.[88] The mechanism by which the gas is produced has been ascribed to a vacuum phenomenon.[89,90]

Alterations of adjacent vertebral bodies frequently accompany disc degeneration. Increased and poorly demarcated bone density of the adjoining vertebral endplates is clearly visible on plain radiographs and is often referred to as 'discitis'.

Two types of osteophyte are commonly seen. *Traction spurs*, which project horizontally from the corner of the vertebral body, are located about 5 mm from the vertebral margin (see Fig. 32.12a). They are believed to be caused by degenerative disc disease, allowing abnormal motion between the vertebrae. Traction spurs are therefore thought to be associated with segmental instability. *Claw osteophytes* are larger and arise from the margin of the vertebral body. These bone formations are thought to be a response to increased mechanical loading which arises in deficient discs (see p. 443).

The radiological signs of degeneration in the apophyseal joints include joint space narrowing, sclerosis of the subchondral bone and osteophyte formation. Increasing narrowing of the intervertebral space also provokes some subluxation of the zygapophyseal joints. On a lateral view, the tip of the superior articular facet is then seen to extend more than a few millimetres beyond the posteroinferior border of the vertebral body above.[91] Further damage to the posterior joints results in retrolisthesis. Oblique projections of the lumbar spine can detect more malalignment of the articular surfaces and the onset of arthrotic deformity.

The end result of disc degeneration is radiologically characterized by an almost complete loss of disc, which is now represented only by a thin intervertebral space between the dense and sclerotic vertebral endplates.[92] Gross beak-like osteophytes are seen at the anterior aspect of the vertebrae. The intervertebral foramen is narrowed, the facet joints are subluxated and enlarged, and retrolisthesis may be visible (Table 32.2).

Table 32.2 Radiological changes in apophyseal and intervertebral joints

	Apophyseal joint	Intervertebral joint
Early changes	Joint space narrowing Beginning subluxation	Intervertebral disc space narrowing Traction spurs Beginning endplate sclerosis
Late changes	Joint space narrowing Subchondral sclerosis Osteophyte formation Enlargement of articular facets Joint subluxation	Considerable disc space narrowing Air pockets (vacuum phenomenon) Endplate sclerosis Claw osteophytes Retrolisthesis

Relationship between degeneration and symptoms

Although disc degeneration and its radiographic appearances progressively increase with age, it cannot be too strongly emphasized that low back pain and disability do not. It is well known that backache peaks in the middle working years of life (40 years),[93–96] which establishes that disc degeneration as such does not produce symptoms *per se*. It is therefore unwise to rely on radiographic appearances for the diagnosis of lumbar disorders. It has been shown repeatedly in controlled studies that there is absolutely no relationship between clinical symptoms and the radiographic changes of degeneration.[97–104] To take but one example, a prospective study on 536 patients by Sanders *et al* compared the radiological findings in patients with backache and disablement (n = 270) with those in an asymptomatic control group (n = 266): there was not the slightest radiographic difference between the two. Their conclusion was that radiography afforded little or no help in the diagnosis of mechanical lumbar disorders[105] and remains a very poor method to indicate past, present or future low back pain.[106]

In contrast, the practitioner must constantly be aware of the problems that can be induced by the interpretation that the clinician, the radiologist or the patient places on a radiograph. The results of radiographic examinations should never be given to the patient *as a diagnosis*. Since there is no evidence of a correlation between the radiographic appearances and the actual complaints, it is vital to be extremely careful in discussing the results of radiology with the patient. Telling the patient that the back shows 'a marked degree of arthrosis' may mean incurability to the patient, and may conjure up a picture of a hopelessly crumbling spine. The radiograph, instead of helping the patient, will increase the disability. Many patients become depressed when they hear that the back is 'worn out'.

Symptoms are produced by a more complex pathway than simple and uncomplicated arthrosis. Making a specific diagnosis and designing a specific treatment are only possible if symptoms and signs are meticulously assessed. The interpretation of the data obtained is based on knowledge of the mechanical behaviour of the tissues involved and the mechanism of pain production. In this way, it is possible to classify activity-related spinal disorders under three major concepts: the dural, the ligamentous and the stenotic (Chs 33–35). These three appear in a specific age group, have a certain correlation with the natural ageing of the lumbar spine and are clinically distinct. Classification of a particular patient's backache into one of these types thus depends on the information obtained from the history and clinical examination, and not on interpretation of radiographs.

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33

The dural concept

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Hypothesis

The dural concept was first defined by James Cyriax in 1945.[1] His hypothesis was that lumbago and backache originate when a subluxated fragment of disc tissue impinges on the sensitive dura mater. This concept – lumbar pain may be of dural origin – is based on two premises:

- Disc degeneration and disc displacements are of themselves painless events.
- The dura mater is sensitive and translates deformations of the posterior border of the disc into pain.

Clinical evidence for insensibility of the disc

The most important evidence for the first premise (insensibility of the disc) is the poor correlation between obvious disc lesions and lumbar pain:

- Data obtained from postmortem studies show the existence of large, symptomless disc protrusions in almost 40% of the cadavers.[2]
- Several controlled studies have failed to show a relationship between radiological changes seen in disc degeneration and the existence of clinical syndromes.[3–5]
- Myelograms in asymptomatic patients show defects in 37% of cases,[6] and the incidence of asymptomatic disc herniations demonstrated by computed tomography (CT) in subjects over 40 years of age is more than 50%.[7]
- Recent magnetic resonance imaging (MRI) studies[8–11] have demonstrated anew the high incidence of disc degeneration and displacement in an asymptomatic group of patients.

These observations permit the conclusion that degenerated or displaced disc tissue is not itself the source of lumbar pain, which only appears when other, more sensitive structures are involved.

Clinical evidence for sensibility of the dura mater

The second premise was deduced from clinical observations on the natural course of backache and sciatica.

One of the most striking clinical features to support the pain-mediating role of the dura is the chronological evolution from backache to sciatica. Almost every instance of sciatica starts with a period of central or unilateral backache, but once leg pain supervenes, the backache usually disappears. Since the work by Mixter and Barr,[12] it has been widely accepted that most radicular pain is caused by a disc protrusion compressing the dural investment around the nerve root and, if this is so, it is logical to argue that the earlier backache was brought about by the same disc lesion. If sciatica is referred pain from the dural sleeve, by analogy the prior backache must have originated from the dura mater. Sciatic pain is thus only the final

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stage of a progression. A small posterior protrusion, bulging out of the intervertebral joint, lifts the posterior longitudinal ligament, touches the dura mater and causes backache. Kept under control by the posterior ligament, the bulge can recede, resulting in spontaneous recovery, or stay unaltered, causing chronic backache. If it increases, however, the counterpressure exerted by the stretched posterior longitudinal ligament pushes it laterally. No longer subject to any resistance, it immediately swells and compresses the nerve root. At the same time, pressure against the dural tube is released and backache ceases (see p. 459).

Another proof of the role of the dura in lumbar pain syndromes is the effect of diagnostic local anaesthesia. A weak solution of procaine, induced via the sacral hiatus into the epidural space, and thus forced between the dural tube and the boundaries of the neural canal, causes contact anaesthesia of the dura mater (see p. 556). Because procaine 0.5% is too weak to penetrate the ligaments or the dural membrane, it acts as a surface anaesthetic, thus only desensitizing the structures with which it comes into contact. If the patient had backache before the injection, and anaesthesia affords temporary relief of symptoms and signs, the dura is most likely to be the source of pain. In all cases of acute lumbago and in most cases of acute or recurrent backache, epidural local anaesthesia immediately abates the pain, thus strongly suggesting a dural origin.

During the last few decades, numerous neuroanatomical studies have shown that the ventral half of the dura mater is supplied by small branches of the sinuvertebral nerve.[13,14] Immunohistochemical studies further demonstrate a significant number of free nerve endings in the dura that contain substance P, calcitonin gene-related peptides and other neurotransmitters contributing to nociception.[15,16]

The mechanism of dural pain is dual

The original concept was quite simple: a subluxated (but of itself painless) component of the disc impinges on the dura or the dural sleeves of the nerve roots. These pain-sensitive structures translate the anatomical changes into back pain or root pain, respectively (Fig. 33.1).

However, recent anatomical and biochemical studies have slightly changed this original concept:

- *The outer border of the disc is innervated*. Although earlier anatomical studies demonstrated the disc to be totally deprived of innervation,[17] more recent research could detect sparse nerve fibres and free nerve endings in the three outer lamellae of the annulus fibrosus,[18–20] penetrating to a maximum depth of 0.9 mm into the annulus. This means that, except at the surface, a normal intervertebral disc remains almost without innervation.
- *Dura mater attachments exist between the anterior part of the dura and the posterior longitudinal ligament* (Fig. 33.2). Recent anatomical and MRI studies have demonstrated that the dura mater is not totally disconnected from the vertebral column but also attached to the posterior longitudinal ligament by connective tissue, consisting of ventral and lateral fibrous bands.[21–24] Although these ligaments are sufficient to allow for

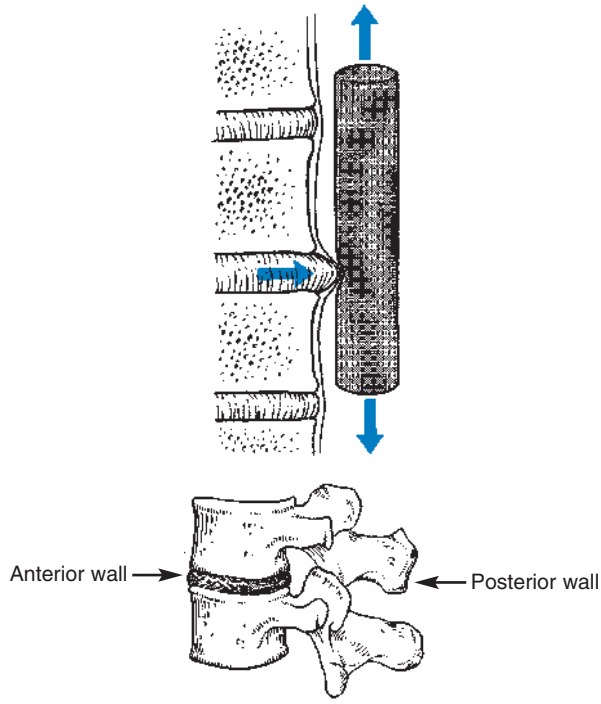


Fig 33.1 • Interaction between displaced disc tissue and the dura mater.

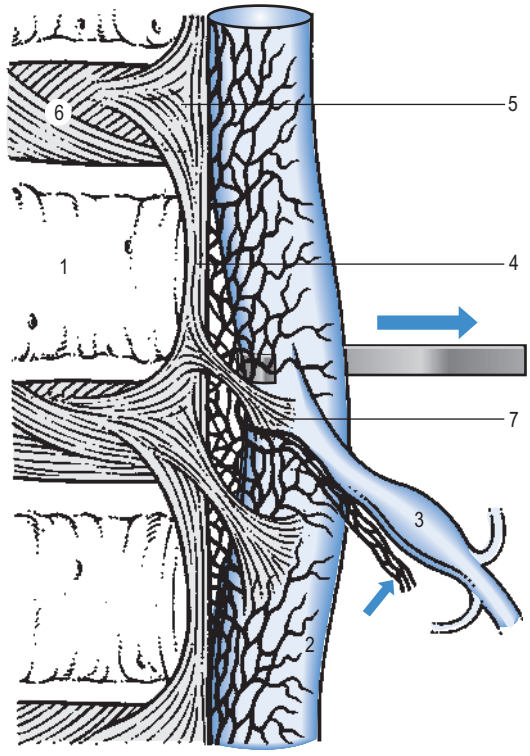


Fig 33.2 • Dura mater attachments: 1, vertebra; 2, dura mater; 3, nerve root; 4, posterior longitudinal ligament; 5, lateral expansion of posterior longitudinal ligament; 6, intervertebral disc; 7, dural ligaments. Small arrow: sinuvertebral nerve, anterior to the nerve root (3).

displacement of the dural sac during movement, they could act to place traction on the dural sac in the event of nuclear bulge or herniation.[25]

- Pain is not only mechanical: inflammatory mechanisms are also involved. Apart from being stimulated mechanically, nociceptors in dura mater may also be activated chemically. An increasing number of experimental studies suggest that disc lesions and/or displacements may induce sufficient chemical changes to irritate the dura mater and to elicit dural pain.[26–31]

Although these new anatomical and histochemical developments have improved understanding of the discodural relation, the original (Cyriax) concept has not changed. From a clinical point of view, the mechanism of discodural interactions is still that of a conflict between an inert and mostly painless structure (nucleus and inner part of the annulus) and a pain-sensitive duroligamentous complex (outermost rim of annulus, posterior longitudinal ligament, dura mater and dural ligaments), all innervated by the sinuvertebral nerves. Discodural pain therefore has a multisegmental behaviour with a broad reference that involves multiple dermatomes and crosses the midline. The interaction is not just a mechanical impingement of discal tissue on the dura mater but also involves inflammatory reactions of the pain-sensitive tissue.

This hypothesis has important clinical consequences. Because the mechanism is dual, the symptoms and the physical signs are also dual. Therefore both 'discal' (articular) signs and symptoms and 'dural' signs and symptoms should be looked for during history taking and functional examination.

- Articular signs and symptoms are those that are related to the mechanical behaviour of the disc: certain postures and movements create biomechanical changes, which force the protrusion against the dura mater.
- Dural signs and symptoms are those that are related to the increase of dural irritation: traction exerted from a distance (straight leg raising and neck flexion) pulls on the inflamed dura or, via the dural ligaments, on the posterior longitudinal ligament or outer annular rim. Also, a sudden increase in spinal fluid pressure pushes the dura against the protrusion (painful coughing and sneezing).

The duality is also important in drawing up a therapeutic strategy. The first measure is to attempt to alleviate the pain by removing the subluxated disc from contact with the dura, which can be achieved by manipulation or traction. If this fails, attempts can be made to desensitize the dura by epidural local anaesthesia.

The dural concept in the natural history of the ageing disc

One of the factors involved in the dural concept is a subluxated portion of the disc and so the biomechanical conditions to allow such a displacement must be present. First, there must be some degeneration of the disc, leading to weakness of the annular fibres and to radiating fissures. These changes are present very early in the degeneration cycle and, for a number of biomechanical and biochemical reasons, occur most frequently at the rear side of the disc. Second, repeated wear and tear, together with shearing forces and slight decrease of disc height, creates some ligamentous laxity which results in an instability of the whole 'motion segment'. Third, through enzymatic depolymerization of macromolecules in the disc, the oncotic pressure temporarily rises.[32] This means that during a particular period of life (between the ages of 20 and 50 years), the osmotic pressure within the nucleus pulposus increases.

Raised intradiscal pressure together with increased segmental laxity is the perfect foundation for disc displacement. A kyphosis imposed on such a predisposed intervertebral joint not only increases the intradiscal pressure but also tends to shift disc material backwards in the direction of the convexity.[33] The intensity of the contact between disc and dura determines whether lumbago or backache will result. When the protrusion is more posterolateral, the dural investment and the content of the nerve root, rather than the dural tube, are compressed, with the symptomatic outcome of root pain.

Further degeneration of the disc results in its 'deflation' and a decline of intradiscal pressure. Decrease in disc height leads to reactive changes at the intervertebral joint and at the posterior structures, which stiffen and stabilize the segment and so diminish the tendency for disc displacements during the later stages of ageing of the spine (Fig. 33.3).

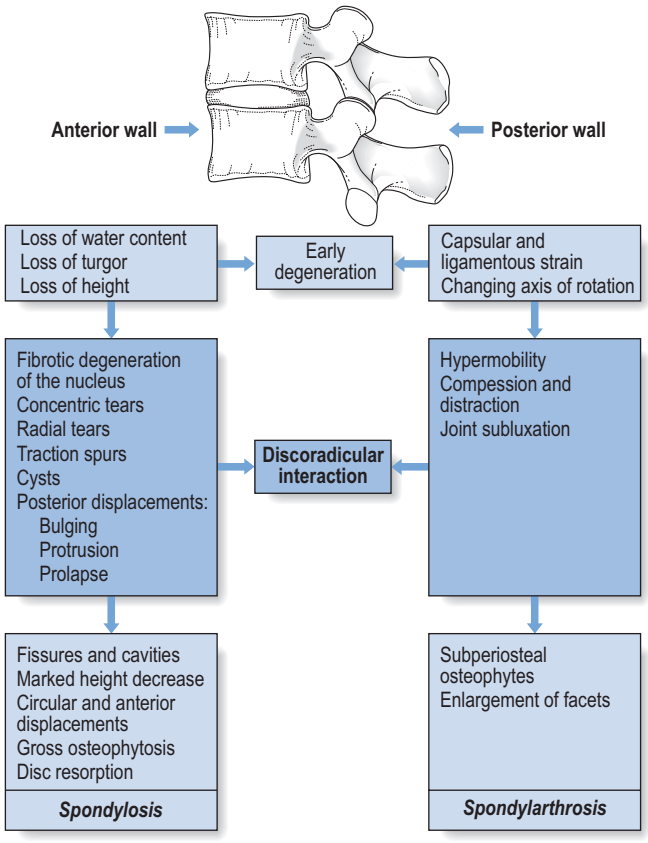


Fig 33.3 • Position of discodural interactions in the degeneration of the lumbar spine.

Conclusion: acute lumbar clinical syndromes of dural origin occur during a specific period of life. This has been established by epidemiological studies, which place the peak incidence between the ages of 30 and 60 years.[34,35]

Clinical syndromes

Lumbago

The most striking example of a discodural interaction is acute lumbago – a sudden attack of severe and incapacitating backache, with obvious limitation of movement, together with gross dural signs and symptoms, summarized in Box 33.1.

History

The history is typical and depends largely on the composition of the protruded disc tissue: annular, nuclear or combined.

Annular lumbago

The patient states that, during some trivial activity, a sudden 'snap' was felt and agonizing pain in the back immediately followed. Very often, this acute event has occurred during a simple movement: coming up after bending, rising from a chair or picking up a light object. Initially, the pain is central and spreads bilaterally over the lower lumbar area and the buttocks. Later, it often tends to radiate more and more unilaterally. Although centralized in the lumbar and/or gluteal area, it spreads to the groin and abdomen, downwards to one or both legs as far as the ankles, or upwards in the trunk as far as the inferior aspect of the scapulae (Fig. 33.4).

Box 33.1

Summary of lumbago

Definition	a sudden attack of severe and incapacitating backache, caused by a large posterior shift of disc material, with dural contact	
Acute onset	Annular	
Slow onset	Nuclear	
	Articular	**Dural**
Symptoms	Twinges Pain on sitting/bending	Extrasegmental pain Pain on coughing/sneezing
Signs	Deviation: forwards sideways Gross partial articular pattern	Pain on neck flexion Limited straight leg raising
Treatment	Manipulation Mobilizations Relative rest	Epidural injections

Differential diagnosis from radicular pain must be made if the ache spreads to one leg only. In such a 'pseudoradicular' dural pain, the distribution is vague, covers several dermatomes and never spreads into the feet. The lumbar or gluteal component also remains more pronounced than the vague and poorly localized referred pain in the limb.

A typical statement in acute lumbago is that the pain is aggravated by sitting and bending forwards, the latter even being impossible. Also, changing from sitting to standing or raising the trunk after lying down for some time is extremely painful and often takes considerable time. The most characteristic sensations in acute lumbago are sharp *twinges*. For fear of these, lumbar movements are executed very cautiously and in 'slow motion'.

The spine is held in the position of least pain by reflex spasm of the trunk muscles and every attempt to straighten the back is associated with severe twinges; the patient walks with the trunk largely immobile, leaning forwards or sideways, keeping the hips and knees slightly bent. Coughing and sneezing are extremely painful and some patients find that even taking a deep breath increases their pain.

Usually, the patient retires to bed, which is probably the only place where a greater or lesser degree of freedom from pain can be assured. Characteristically the 'psoas position' is adopted: supine, with hips and knees flexed to 90°. The pain eases gradually and in most cases all symptoms have gone after a few days or weeks.

An attack of acute annular lumbago is caused by posterior subluxation of part of the annular rim, pressing the posterior longitudinal ligament against the dura mater (Fig. 33.5a). It is obvious that a history of sudden pain, immediately followed by a 'locking' in flexion, indicates some internal derangement,

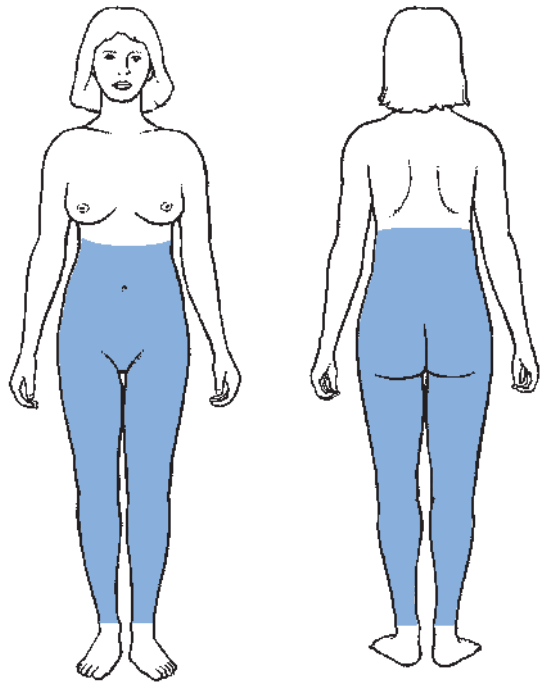


Fig 33.4 • Possible reference of dural pain caused by an L5 protrusion.

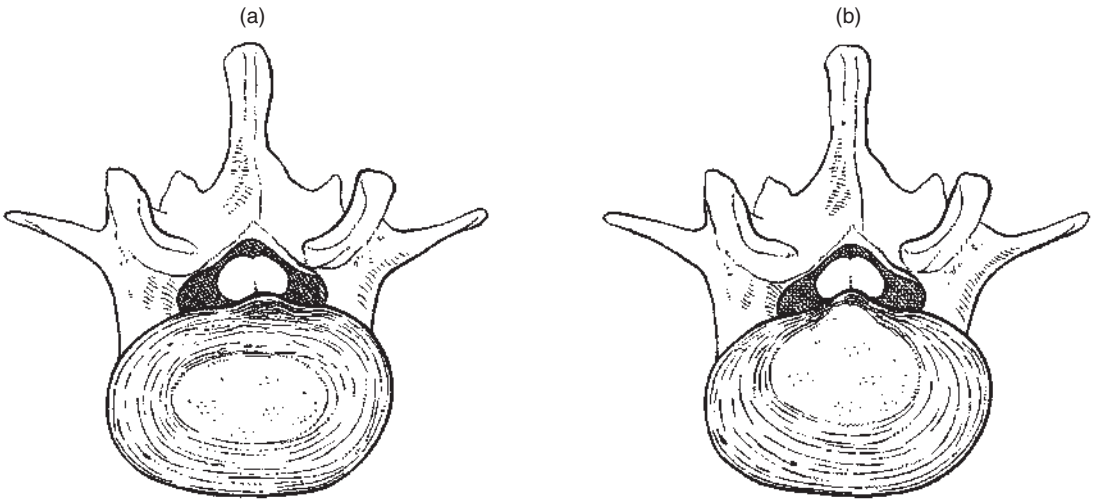


Fig 33.5 • (a) Annular and (b) nuclear lumbago.

just as a sudden pain in the knee, followed by inability to straighten it, indicates subluxation of a meniscus. The dural extrasegmental reference of the pain, together with pain on coughing and sneezing, implicates the dura mater and therefore excludes locking of the posterior facet joints. In displacement at the back of the intervertebral joint, the lumbar spine is held in flexion because extension squeezes the protrusion, which in turn increases the painful pressure on the dura. In order to keep the protrusion away from the dura and as immobile as possible, the patient adopts a flexed position. Muscle spasm prevents any further movement at the lumbar spine.

Nuclear lumbago

The pain, although equally incapacitating, does not appear suddenly but gradually increases over the course of a number of hours or days. Alternatively, after heavy work involving much stooping and lifting or sitting for an unusual length of time in an uncomfortable position, slight backache is felt but is initially regarded as trivial. However, by the next morning the backache is sufficiently severe to make getting out of bed impossible. The pain radiates in a way that is typical of dural involvement (see Fig. 33.4). The patient is immobilized in flexion or side flexion and every attempt to straighten the back is followed by an agonizing twinge in the lumbar area and the buttocks. Sometimes even simple neck flexion is impossible or coughing or sneezing creates a twinge. As in an attack of annular lumbago, the patient has to go to bed to cope with the pain. As a rule the pain eases after a few days or weeks.

In gradually increasing lumbago, the protrusion presumably consists of soft and pulpy nuclear material, oozing slowly backwards. This typically happens during the maintenance of a kyphotic posture (sitting, bending or lifting). The displaced nuclear material gradually presses more and more against the outermost layers of the annulus and the posterior longitudinal ligament and makes them protrude (Fig. 33.5b). This provokes dural irritation, resulting in the typical dural pain in lumbar area, buttocks and limbs.

Mixed lumbago

Sometimes there is a sudden attack of acute low back pain, increasing slowly during the subsequent few hours or days. This indicates that the protrusion is probably mixed, consisting of an annular crack, with some pulpy material oozing backwards between its edges.

The distinction between an annular and a nuclear protrusion is extremely important, for both treatment and prophylaxis. A hard fragment reacts very well to manipulation, whereas a soft protrusion is more difficult to reduce. In the maintenance of reduction and in prophylaxis, a patient with a history of recurrent annular protrusions has to be constantly on guard during specific movements (bending and lifting) but a nuclear protrusion only reappears after prolonged loading of a joint in flexion.

Clinical examination

During clinical examination the following are important.

Inspection

Deviation towards flexion is noted and the sacrospinalis muscles are seen and felt to be in contraction to maintain the adaptive posture. Because the flexed position places the upper trunk in front of the centre of gravity, the muscles contract to prevent further forward toppling. Lumbago is not caused by muscle spasm – as was maintained by some authorities for many years[36] – but is the result of a disorder at the posterior aspect of the intervertebral joint.

A lateral shift associated with acute lumbago is a common clinical event, undoubtedly associated with a disc protrusion.[37] The lateral shift can be either towards the dominant side of pain (ipsilateral) or away from the side of the pain (contralateral). The majority of affected patients have a contralateral shift.[38] Occasionally, the shift may change from side to side, which has been termed an alternating scoliosis. The lateral shift is explained as avoidance of compression or irritation of

the dura mater, either actively or reflexively through muscle spasm.[39,40]

When gross lateral deviation is present, a lesion at the fourth or third lumbar level should be suspected. Because of the stabilizing effect of the iliolumbar ligaments, fifth lumbar protrusions very seldom result in gross lateral deviation.

Alternating deviation is pathognomonic for central protrusions at the fourth lumbar level. At a given moment, the patient is deviated to the left but, after performing some lumbar movement, deviates to the right. This curious phenomenon is explained by the dura slipping to one or other side of a midline protrusion.

Spinal movements

There is gross but unequal degree of limitation in the four directions (the partial articular pattern), which indicates that one part of the joint is more affected than the others.

Extension

As a rule, extension is considerably limited (Fig. 33.6) on account of the posterior displacement of the disc causing a block at the back of the joint.

Side flexion

In one direction, side flexion is considerably more limited than in the other and is usually associated with visible lateral deviation on inspection. If side flexion towards the pain is more limited, manipulation seems to be less effective but, if pain is felt more on the side away from which the patient bends, manipulative reduction usually succeeds.

Alternatively, there is a gross painful arc when the trunk passes the vertical, on swinging from one side to the other. Both side flexions are then painless at the end of range. This phenomenon indicates a central bulge at the fourth lumbar level and corresponds with the alternating deviation seen on inspection.

Flexion

This is extremely painful in cases of acute lumbago and is usually the last movement to free up after a manipulative session.

Sometimes deviation becomes visible during flexion, despite its absence in the standing position, or an evident lateral tilt in the upright position disappears during flexion.

Dural tests

Neck flexion

In lumbago, neck flexion often hurts in the lower back, which proves the involvement of the dura mater in the origin of the pain[41] (see p. 502).

Straight leg raising (SLR)

We have discussed the evidence that SLR is a dural sign (see p. 427) and, just as neck flexion stretches the dura from above, so SLR stretches it from below. A lesion resulting in such a gross discodural interaction as acute lumbago, therefore, *must* influence SLR. Acute lumbago with full and painless SLR should make the clinician reluctant to accept the diagnosis of a displaced disc. If the acute pain in the back is so severe that the patient cannot move out of bed, but dural symptoms and signs – including a positive SLR – are absent, gross bony lesions such as osteomyelitis or metastases should be considered (see Ch. 39).

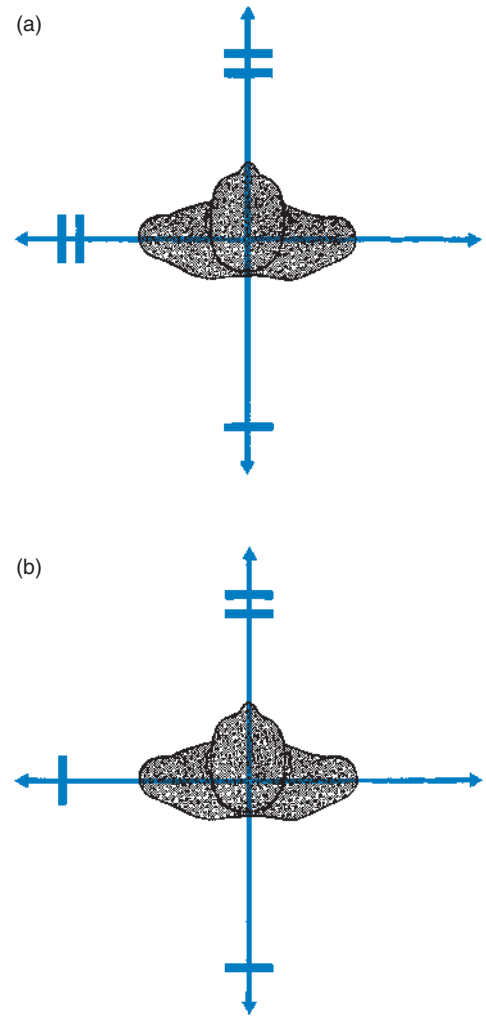


Fig 33.6 • Two examples of gross partial articular patterns. (a) Gross limitation of flexion and left side flexion, slight limitation of extension, normal right side flexion. (b) Gross limitation of flexion, slight limitation of extension and left side flexion.

Lumbago usually causes bilateral limitation of SLR: because the bulge and the dura mater both lie centrally, raising both legs pulls on the dura equally. In unilateral lumbago there will often be more limitation of SLR on the painful side. Occasionally this may be reversed, when the crossed SLR phenomenon is present (see p. 498).

The degree of limitation of SLR is an indication of the intensity of the discodural interaction.[42] In hyperacute lumbago, any attempt to move the straight leg upwards results in considerable pain, whereas in more moderate lumbago the SLR is limited at 45–60°. During recovery, when the reduction is almost complete, SLR will probably only be painful at the end of range or show a painful arc at mid-range. The progress of SLR is therefore a very sensitive clinical index in following the position of the protrusion during manipulation.

Tests of conduction

Neither muscle weakness nor cutaneous analgesia is present in cases of acute lumbago. Because protrusion is more or less central, nerve roots are not involved. Care should be taken, however, not to miss a compression of the fourth sacral root. Because it lies centrally, partly protected by the posterior longitudinal ligament, a central protrusion can endanger it, especially if the protrusion overstretches the ligament. Physical findings are non-existent and the diagnosis is made entirely on the history. If pain deep in the sacral area, pain and paraesthesia in the penis, vagina or rectum, numbness in the saddle area or problems with continence are mentioned, damage to the fourth sacral root should be considered and the patient immediately referred for further assessment.[43] A fourth sacral lesion occurs at a level proximal to the posterior ganglion and permanent interference with bladder function can result if decompression is not carried out.[44] Therefore its onset, however slight, is an indication for laminectomy. Cyriax[45] (his p. 284) recommends operation even when bladder function is returning after the attack of lumbago, because there is no guarantee that lasting incontinence may not follow the next attack.

Natural history

With, without or despite treatment, most cases of acute lumbago recover spontaneously and completely within 2–6 weeks (Dixon[46]; Chöler, cited by Nachemson[47]; Spitzer[48]). The tension in the posterior longitudinal ligament exerts counter-pressure on the bulge, which moves gradually anteriorly, until compression of the dura mater ceases and symptoms disappear. However, as cartilage has little tendency to reunite, a fragment that has moved backwards once will sooner or later move again, which implies that, although complete recovery after an attack of acute lumbago is the rule, recurrences are to be anticipated.[49]

Sometimes, however, a disc protrusion will not recede completely and chronic backache results. Although the patient largely recovers and most of the symptoms disappear, a continuous lumbar ache remains, especially during or after particular movements or in particular positions.

Alternatively, the lumbago disappears but there is simultaneous onset of root pain. As has already been discussed (see p. 442), the protrusion has moved from the centre to one side.

Treatment

Most cases of acute lumbago recover without treatment. MacNab put it well when he remarked that 'The physician must constantly remind himself that even if he elected to treat the patient by rubbing peanut butter on each buttock, in the balance of probabilities, the patient would get well fairly quickly.'[50] However, keeping the intradiscal pressure as low as possible will, of course, ease symptoms and hasten the reduction of the bulge. It is therefore wise to adopt the supine lying position from time to time, with the knees and the hips flexed to a right angle[51]; this decreases the load on the disc to about 30 kPa[52] (the 'psoas position'; Fig. 33.7). It is also sensible to avoid movements and positions that cause high intradiscal pressures, such as sitting or bending (Nachemson[53]: p. 708).

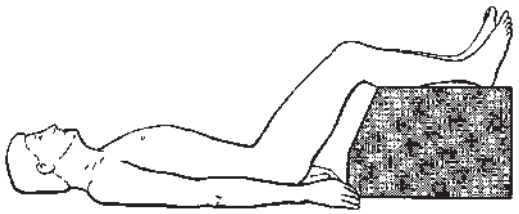


Fig 33.7 • The 'psoas position'.

Standard textbooks almost unanimously recommend bed rest as the first line of treatment for acute lumbago.[54,55] However, it has never been proven that complete and continuous bed rest decreases the time of recovery. In a comparative trial, statistically significant differences between bed rest, early mobilization and no treatment have not been found, although results tended to favour early mobilization.[56] Others have reported little difference between mobilization and rest,[57] and 2 days in bed was found to be even better than 7 days; put the other way around, 7 days in bed was more harmful than 2 days.[58,59] Also, a recent Cochrane review concluded that there is no difference in effect between advice to stay in bed and advice to stay active.[60] Therefore bed rest is only necessary if bed is the only place where the patient is comfortable. If, after a couple of days, walking around is possible without a considerable increase in pain, such a regime should be followed. Furthermore, a patient should never be forced to stay in bed against his or her will.

Annular lumbago

If acute lumbago is of the annular type, there is a good chance of early and complete reduction over the course of one or two manipulative sessions; instead of awaiting spontaneous recovery over 2 or more weeks, the patient can return to work from the second or third day.

Nuclear lumbago

In nuclear types of acute lumbago, classic manipulative reduction usually fails. A slow onset of symptoms usually indicates that the protrusion is too soft to be pushed back. Manipulation is also apt to fail in lateral deviation away from the painful side. A good alternative, then, is a sustained manual stretching technique. Positioning the patient in increasing but still comfortable lordosis (McKenzie technique) is another alternative in treating acute nuclear lumbago.[61,62]

Although effective in chronic nuclear backache, where the dural symptoms are much milder, traction should *never* be used in acute nuclear lumbago. Experience shows that, if traction is applied on a patient who mentions the presence of 'twinges', considerable worsening of the condition for several days may be expected. The reason for this is not completely understood. Presumably the size of the bulge increases when the hydrostatic and osmotic conditions within the disc change during traction (see p. 420).

Hyperacute lumbago

If the lumbago is really hyperacute, which means that the dural symptoms are so intense that repeated and agonizing twinges force the patient to lie motionless, any attempt at manipulative reduction is unthinkable. It is obvious that manipulation cannot be done when the patient can hardly move or when it takes some minutes to roll from a prone to supine-lying position on the examination couch. In these cases, the only alternative to several weeks of bed rest is epidural local anaesthesia, which affords immediate and complete relief of symptoms over 1 or 2 hours. Curiously enough, and although the anaesthesia only works for 2 hours, there is lasting relief from the next day on. The injection probably has some long-term effect on the inflamed dura, rendering it less sensitive. Once the immobilizing twinges have been abolished, the patient is capable of getting up and travelling for manipulative reduction of the residual displacement. This combination of epidural local anaesthesia and manipulation is rapidly successful in nearly all cases of hyperacute lumbago.[63]

Backache

About 80% of all cases of low back pain relate directly to the intervertebral discs.[64] Discodural backache presents a typical complex of symptoms and signs, both articular and dural. The mechanism of pain and dysfunction is exactly the same as described in lumbago, but the signs and symptoms are less acute because the discodural interaction is more moderate.

Mechanism

In a predisposed and slightly degenerated disc, a small posterior displacement occurs when the biomechanical factors are favourable: increased load in a flexed spine shifts the disc material backwards (towards the convexity). Contact with the dura mater then creates the pain.

If the displaced disc material is nuclear, the onset of the symptoms will be slow; if it is annular, the pain will appear suddenly. Because, in contrast to lumbago, the protrusion is rather small, the dura will not be stimulated continuously and therefore dural irritation remains moderate; the twinges and gross deviation typical of lumbago are absent. Sometimes dural contact occurs during particular movements of the spine only when an increase in load pushes the bulge in the direction of the dural tube. The protrusion may not be large enough to interfere with the dura when the latter is moved forwards during neck flexion or straight leg raising, and therefore dural signs are not always present. This explains why, in moderate backache, there can be articular signs only, even though the mechanism of pain is that of a discodural interaction.

History

The pain is usually located in the back, unilaterally or centrally, at the sacral region or in one buttock. Because the pain is of dural origin, it can spread to the iliac crest, the trochanteric area and the groin, although seldom beyond the gluteal folds. Occasionally it may be vaguely situated in the posterior or anterior aspect of the thigh.

Depending on which part of the dura is irritated, the pain is central or unilateral or shifts in location.[65] Shifting pain is a common history and indicates that the lesion has moved from one side of the intervertebral joint to the other. Shifting pain in the back is one of the most characteristic phenomena in discodural backache. An alternating ache in the buttock, however, suggests sacroiliac arthritis rather than a disc problem (see Ch. 41).

The localization of pain not only varies according to the site of compression of the dural tube but is also determined by the intensity of the stimulation. One of the rules of referred pain is that the stronger the stimulus, the further the pain will be referred. This has some practical bearings when it comes to evaluation of therapy: when the pain has originally been located in a buttock but, during a manipulative session, tends towards the centre and becomes paravertebral, this implies that the pain stimulus has been reduced and discodural contact is now less pronounced than it was. 'Centralization' of the pain is thus a good predictor of a successful outcome.[66,67] The reverse change – pain moving more and more distally – indicates that the situation has worsened.

The onset of the complaints is very important from a clinical point of view but cannot always be recalled. Especially in long-standing cases, the patient may have difficulty in remembering if the symptoms appeared suddenly – as would occur with an annular lesion – or if disability was progressive – a nuclear displacement. In an *annular* protrusion, the patient may describe a sudden sensation of something giving way in the back, which may have been accompanied by an audible click or 'thud' in the lower back, associated with a twinge of pain. From that moment the back has ached during particular positions and movements. The typical history of a *nuclear* protrusion is that the initial symptoms were slight, with little disability, the patient easily being able to continue normal activities, probably with only minor backache. The same evening, however, after sitting down to eat, read or relax, considerably increased discomfort will have been felt and the following day worsening will have become apparent from the moment an attempt was made to get out of bed.

Relation between posture/movement and pain is also important. In minor disc lesions, the ache probably depends entirely on the level of exertion. Any work involving stooping, lifting or sitting for too long is followed by pain, which may, however, be almost or completely absent at rest. In more advanced instances, particular positions are very painful or even impossible. It is obvious that contact will increase in positions and activities that increase intradiscal pressure and thus discodural contact. Bending forwards and lifting result in higher pressure than standing erect. To most patients with backache, walking around is more comfortable than sitting, because the latter imposes more load on the disc.[68] Sitting without support causes yet more load and consequently more pain than does sitting with a reclined back rest.[53]

Pain is influenced not only by position but also by particular movements. It is striking that in backache caused by disc problems one of the most consistent complaints is a temporary

increase in pain when the patient changes position. The aggravation of pain on standing after sitting for some time or the momentary increase in pain on sitting after walking is typical of a discodural interaction. Turning in bed and putting on footwear in the morning is often mentioned as being associated with an increase in pain. Dural symptoms, such as pain on coughing and sneezing, are often present.

A paradoxical symptom complex is sometimes encountered. The dynamics of the disc, described above (see p. 420), suggest that intradiscal pressure should decrease and any bulge become less prominent when the patient lies down. Yet some patients have more pain during and after bed rest, wake during the night and have to get out of bed before dawn. The explanation is probably an increase in swelling when the external load diminishes. Diurnal changes in disc hydration and pressure have been demonstrated both *in vitro*[69] and *in vivo*,[70,71] and it is estimated that around 25% of the disc fluid is expressed and re-absorbed during each diurnal cycle.[72] A small posterior bulge that becomes more hydrated swells to increase dural contact. This phenomenon is also mirrored in the diurnal changes in the range of the SLR; comparison of the range of SLR after recumbency and after 2 hours erect shows an increase in range of 10% or more.[73]

The characteristics of discodural pain are summarized in Box 33.2.

Clinical examination

Inspection

Lateral deviation or flexion deformity is present only in more marked cases of backache. As in lumbago, the lateral tilt may be either away from or towards the painful side. Some protective muscle spasm may be seen or felt.

In mild examples, inspection reveals nothing special; nor does the patient mention any pain in the neutral position.

Spinal movements

A partial articular pattern is present on the four active movements. The degree of limitation is unequal in different directions (Fig. 33.8); if there is no limitation, some movements are painful at their extremes and some not. All these findings are typical of internal derangement – some movements increase the annular or nuclear bulge, so increasing dural contact, while others reduce it.

Box 33.2

Characteristics of pain in discodural conflicts

- Dural reference
- Dural symptoms: on coughing, sneezing and pressing
- Shifting pain
- Pain increases during sitting/bending and when position changes
- (Pain worse in the morning)

Extension

This is painful in the centre of the lumbosacral area and, if the protrusion lies centrally, is also limited. Sometimes the lumbar spine is seen to shift slightly laterally during extension.

Side flexion

Usually this is unequally limited (Fig. 33.8a). Alternatively there is pain at the end of one side flexion only, the other being full-range and painless. If side flexion away from the pain is the more difficult to achieve, manipulative reduction will almost certainly succeed, but in the reverse situation quick and lasting relief by manipulation is more uncertain. Sometimes there is a painful arc (Fig. 33.8d): momentary pain is experienced on moving the trunk from one side to the other. The arc may be quite extensive and is only overcome with considerable effort. Therefore the patient should be encouraged to continue movement and not stop the moment pain is felt; otherwise, the presence of an arc could be missed. Sometimes both side flexions are full and painless. This does not eliminate a disc protrusion but probably indicates that it is too small and too centrally localized to come in contact with the dura during side flexions. Only extension or flexion will then influence the pain and asymmetry is probably only shown by some momentary deviation, a painful arc during flexion or unilateral localization.

Forward bending

As in acute lumbago, forward bending is usually the most painful and limited movement, because it not only acts on the intervertebral joint but also drags the dura forwards in the direction of the protrusion. During flexion the trunk may deviate, although in the upright position it was straight. The reverse may also happen: a lateral tilt in the erect position is lost during flexion. Sometimes the deviation is momentary: the spine shifts away from, then back towards, the midline as the movement proceeds. There may also be an alternating deviation: one way in forward flexion and the other way when the direction of movement is reversed.

Deviations, whether in the upright or flexed position or alternating, are all defensive mechanisms to avoid pain: the dura mater has to be held to one side or the other of the projection. Therefore the clinical finding of deviation, of whatever type, strongly suggests disc protrusion.

It is also possible for flexion to be of full range and only painful at the extreme. Or pain is only provoked when, at the end of range, active neck flexion is added.

Painful arc

Frequently, a painful arc is encountered, with a transient pain somewhere at mid-range (Fig. 33.8c, d). Alternatively, slight deviation may be seen at the midpoint of flexion. Careful observation is needed to detect this visible arc, of which the patient is usually unaware. A painful arc during flexion can be associated with a partial articular pattern but it can also be an isolated finding. It *always* means that a small fragment of disc tissue impinges momentarily against the dura mater. At the beginning of flexion, an increase in both intradiscal pressure and convexity of the posterior aspect of the intervertebral joint provokes discodural contact (Fig. 33.9). Flexion beyond the horizontal imposes more distraction than compression on the

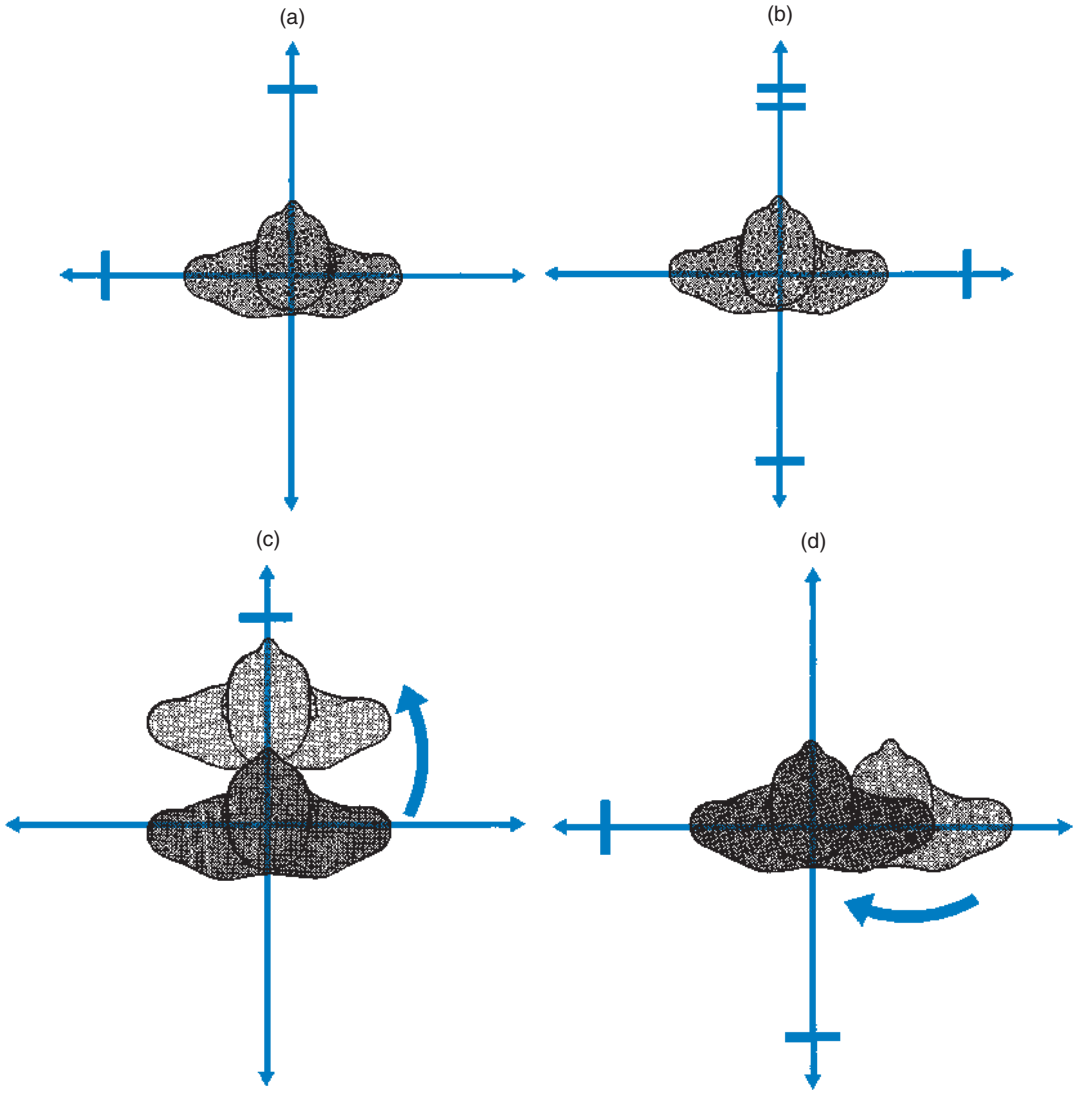


Fig 33.8 • Example of partial articular patterns. The lower figures are associated with a painful arc: (c) during flexion; (d) during side flexion.

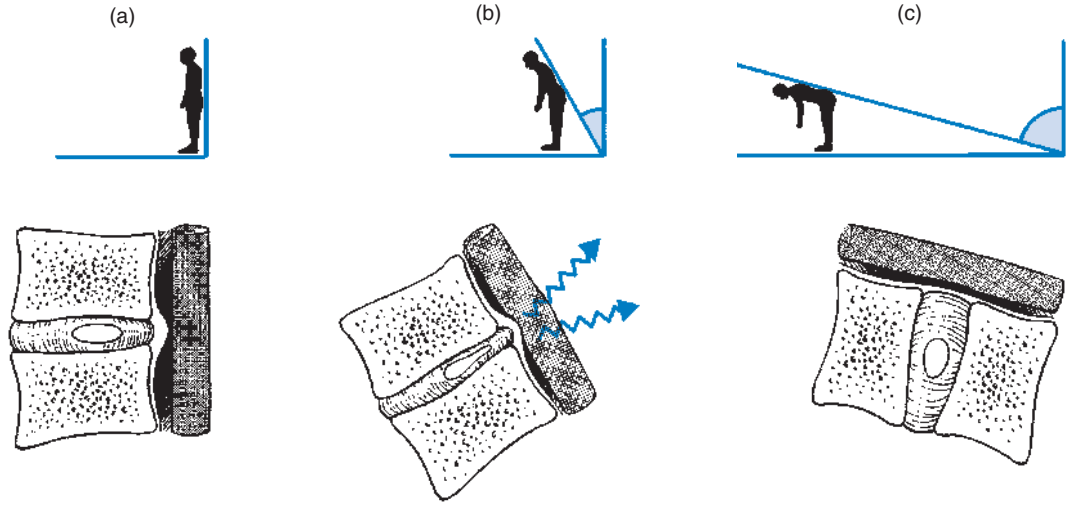


Fig 33.9 • Painful arc: (a) erect position, (b) 30° of flexion, (c) 60° of flexion.

intervertebral joint. The backward pressure on the disc then decreases and is replaced by a more centripetal force on the disc, which is supported by the tightening of the posterior longitudinal ligament. The small posterior displacement is then removed from contact with the dura and pain ceases.

Dural signs

Any hindrance to the normal mobility of the dura leads to a limitation of SLR or to pain during neck flexion. This is the normal finding in acute lumbago. In discodural backache SLR may be painful or limited but, should this not be the case, the disc cannot be automatically eliminated as a cause of symptoms. Especially in mild examples, displacement can be small and the impact of the bulge on the dura not continuous. In a supine-lying position the protrusion may not be large enough to cause interference with dural mobility, and so SLR remains negative. Contact between disc and dura then only originates when a momentary increase of intradiscal pressure moves the disc backwards, which happens during forward flexion while standing. In the course of manipulative treatment it is a common finding that SLR becomes negative before reduction is complete. Lumbar movements, tested while the patient stands, then still provoke pain because, in the erect position, the joint is subjected to axial pressure, which causes increased posterior bulging of the disc.

Root signs

In uncomplicated backache there are no root signs.

Natural history of discodural backache

It is extremely difficult to predict the natural history and therefore unwise to tell the patient that backache will very soon abate. Although it is true that most episodes are self-limiting,[74] the disability often becomes chronic.[75] Though an acute bout shows some tendency to spontaneous cure,[76] recent research shows that the course of back pain is merely episodic, with repeated recurrences following an acute episode.[77–79] A substantial minority of patients may not even experience resolution of their pain and disability, and suffer for years from chronic lower lumbar pain.[80–83]

Particular types of backache

Bruised dura

Sometimes a patient complains of a constant ache in the back, which is unaltered by position or movement. The onset takes the form of an attack of acute lumbago. Resolution usually took place within 2 weeks, except for persistent constant backache over months or years. Coughing and sneezing may aggravate the pain but other movements definitely do not; there may be increased pain during the night or in the morning.

Clinical examination reveals absolutely nothing: there is a full range of spinal movements and dural signs are absent.

One possible explanation for this type of backache is 'bruising' of the dura mater.[84] The acute lumbago has induced inflammation of the dura mater. Although the disc displacement has receded after some time, the dura has remained inflamed, which results in continuous pain. Obviously this type of backache – chronic pain unaltered by posture or exertion and with a negative clinical examination – can also exist as the result of pain referred to the back from other (visceral) structures.

When a bruised dura is suspected, an epidural injection with procaine 0.5% is indicated. A positive diagnostic response not only settles the differential diagnostic question but is also the treatment, because about half of those with backache attributed to a bruised dura are permanently cured by one injection.

Nocturnal or morning backache

Backache may be confined to night-time. The patient can do everything normally undertaken during the day, without the slightest discomfort, but every morning is woken in the early hours because of increasing and severe backache which forces the patient to get out of bed. The pain quickly eases, and once the patient has been upright for, say, half an hour, the disability has totally disappeared. A pain-free day follows, in spite of the level of exertion. Alternatively, unbearable backache after 2 hours of sleep forces the sufferer out of bed. The pain abates quickly and resumption of sleep is possible. During the day there is not the slightest discomfort, even on heavy work.

Clinical examination during the day is negative: there is a full range of movement and dural signs are absent.

This nocturnal backache often occurs in middle-aged people. It is best explained by an increase in intradiscal oncotic pressure at an early stage of degeneration. When the external load is diminished in the horizontal position, there is a considerable increase in water content. Expansion forces the disc against the pain-sensitive dura. Resumption of the upright position raises the hydrostatic pressure, water is extruded and the disc deflates, which alleviates the tension on the dura mater so that pain disappears.[72]

Because the pain is of dural origin, epidural injection is the treatment of choice and succeeds in about 70% of cases.[63] Should the injection fail, ligamentous sclerosis is used, in order to stabilize the lower lumbar segments.

Nuclear self-reducing disc

Sometimes the history obtained is the converse of that described in the previous section. Waking is comfortably pain-free and exertion does not cause symptoms. Backache starts after some hours upright, increases slowly as the day goes on, being worst in the evening, and varies in intensity according to activities. Lying down abolishes the pain, which has completely gone by the next morning.

Clinical examination in the morning is negative with a full range of movement and absence of dural signs. By the evening a partial articular pattern and pain on SLR are present. Clearly there has been protrusion of nuclear material as the result of axial loading of the spine. Recumbency during the night then results in spontaneous reduction.

Because this history indicates a reducible but unstable disc protrusion, treatment should be directed towards stabilization

of the intervertebral joint. This can be achieved by sclerosing injections, which usually give good results.

Treatment

Before specific treatment is given, a few questions must be answered:

- Is the backache caused by an activity-related spinal disorder?
- If this is the case, is the disorder a discodural interaction or not?

When there is a clear combination of both articular and dural symptoms and signs, the answer is obvious. In moderate discodural backache, however, when the patient presents with articular signs only, it may be more difficult to make a certain diagnosis of internal derangement. However, a partial articular pattern always indicates a disc lesion, except in a few cases (see Ch. 39). A deviation, whether in the upright position or in flexion, signifies a protrusion. Also, the presence of a painful arc, whether during side or forward flexion, is the sign of a small posterior bulge.

The therapeutic approach to discodural backache differs from patient to patient and depends largely on the data obtained from the history and clinical examination. Although most cases of backache are caused by an impingement of a subluxated disc against the dura mater, the size, structure, position, level and stability of the bulge differ considerably. Treatment therefore must be selective. A single definitive treatment for discodural low back pain does not exist. If a subluxated fragment of disc is believed to be the cause of the pain, further questions should be posed:

- What is the level of the lesion and what is the size and composition of the bulge?
- Is the subluxated fragment an acute and occasional event, or does the patient have recurrent attacks of backache? How long does the disability last? Does the pain disappear completely between bouts or is there a continuous ache?
- What is the degree of pain and how much is the dura inflamed?
- What attitude does the patient have towards the problem?
- Does the patient want to get better? Is there any compensation claim or does the patient show clear evidence of psychological disorder? (see online chapter *Psychogenic pain*).

If there is proof of a disc lesion and the patient is well motivated, conservative treatment consists of:

- Reduction of the displacement
- Maintenance of reduction
- Desensitization of the dura (in acute or gross inflammation of the dura, it is sometimes better to desensitize it in order to abate the pain instead of trying to move the disc back into place).

Reduction

Reduction is achieved by manipulation or traction. When a slight displacement of disc tissue is believed to be responsible for the symptoms, the obvious treatment is to restore its anatomical position.

If the displacement is annular, the treatment of choice is manipulative reduction. A nuclear displacement is an indication for sustained traction. Although the two techniques are, to some extent, interchangeable, some protrusions prove irreducible by traction, yet reducible by manipulation and vice versa. It is obvious that a protrusion composed of hard annular material will respond better to manipulation but that a soft nuclear bulge requires traction. Cyriax said[45]: 'You can hit a nail with a hammer, but treacle must be sucked.' If sufficient data on the onset of the complaints cannot be obtained and the choice of treatment is in doubt, manipulation should be tried first. If it fails, the patient should attend for traction from the next day on. If considerable improvement is achieved by manipulation but, despite further attempts, a residual displacement cannot be reduced, traction should be substituted so as to complete the process.

Manipulation

The indication for manipulation is a posterior annular displacement. Signs that favour the use of manipulation are a small painful arc or a small lateral deviation during flexion. Reduction is usually easy to achieve in patients whose pain is greatest when they bend away from the painful side. The chance of success of manual reduction is also greater in elderly patients.

The manipulation techniques used in this book tighten the posterior longitudinal ligament and create space at the posterior aspect of the joint. The increased distance between the two vertebral borders gives the fragment room to move, and the force exerted by the posterior ligament pushes it back in place.

Displacements at the third and fourth levels respond best to rotatory manœuvres, whereas at the fifth level extension manœuvres are usually more effective. Rotatory manœuvres are also chosen if deviation of any kind exists. Elderly patients tolerate prone extension techniques better than rotational techniques.

The simple and easy-to-learn manipulation measures are usually speedily effective. Overall, acute backache is relieved by one session of manipulation in 46–57% of patients.[85,86] As a rule, small annular displacements are cured by a single manipulative session. In larger protrusions, 2–4 sessions may be required. When the patient presents with a marked lateral deviation, up to four manipulation sessions are sometimes needed. Manipulation is successful after a small number of sessions or not at all. Hence, if the patient does not improve almost immediately and lastingly, it is unwise to continue treatment and daily traction should be used instead.

Young patients tolerate daily manipulation when repeated sessions are required. In the elderly, however, it is wise to manipulate on alternate days, for fear of increasing ligamentous pain.

Sustained daily traction

The indication for traction is a patient under 60 years old, who describes slow onset of pain in the back. Patients with a discodural conflict, in whom the consistency of the protrusion is uncertain and who do not improve immediately after

manipulation, should also be treated with daily traction. Experience also teaches us that, when trunk side flexion towards the painful side increases the pain, traction will succeed better than manipulation.

The intention of traction is to create negative pressure in the disc[87] and to tauten the posterior longitudinal ligament, which exerts a centripetal force on the nuclear material.[88] This results in a gradual reduction of the bulge and a release from the dura mater.

Maintenance of reduction

Once the displacement is reduced and the patient asymptomatic, the question of specific prophylaxis arises.

If the history is that of a nuclear protrusion, the patient should be careful about posture, especially during prolonged sitting or bending. Maintaining the spine in slight lordosis is beneficial and helps to prevent posterior bulging of nuclear material.

Occasional attacks of annular low back pain – say, once a year – can be managed by a good manipulator, provided one is available. More frequent recurrences call for attention to active prevention. The necessity for lowering intradiscal pressure and keeping the back hollow during daily activities should be explained during 'back school' sessions, where the patient is instructed in sitting, standing, bending and lifting. However, the back school is prophylactic and not therapeutic, and patients should never attend before they are completely asymptomatic.

Strengthening the muscles of the trunk does not increase the stability of the disc. Back muscles do not directly control the intervertebral content and in consequence stability will not depend on their strength but on the position in which they keep the body. Exercise and strengthening of the abdominal and sacrospinalis muscles are therefore futile and may make backache worse because intradiscal pressure increases significantly with prone-lying extension exercises[89] and sit-up and curl-up exercises.[90,91]

In recurrent backache, where the disc is unstable and the patient suffers repeated attacks despite maintaining good posture, sclerosing injections should be given to the posterior ligaments. The purpose is to induce inflammation at the inter- and supraspinous ligaments, the posterior capsule of the facet joints and the deep aspect of the lumbar fascia, at the fourth and fifth lumbar levels. In response to the inflammation, fibrous tissue and tissue contracture in the injected ligaments occur. Permanent shortening of the injected structures then decreases the mobility and increases the stability of the intervertebral joint. About 80% of patients treated by sclerosing injections benefit from them. Another indication for these stabilizing injections is nuclear self-reducing disc protrusion, for which no other treatment is effective. They are also indicated when nocturnal or morning backache does not respond to epidural local anaesthesia.

Epidural injection

In discodural interactions in which inflammation of the dura is more important than the actual disc protrusion, the treatment of choice is epidural injection of a local anaesthetic. This is the case in nocturnal backache, for instance, where a discal swelling only intermittently presses against the dura. When backache is brought on by a 'bruised dura', epidural local anaesthesia is also used.

When backache is clearly the result of a low lumbar disc displacement but proves refractory to both manipulation and traction, the next step is epidural injection. If it is impossible to correct the discal bulge, it is logical to try to desensitize the dural tube.

Definition, symptoms, signs and treatment of discodural backache are summarized in Box 33.3.

Sciatica

Since Mixter and Bar published their classic paper in 1934,[12] it has been generally acknowledged that lateral disc displacements are the main source of radicular pain. Pressure of the protruded disc against the nerve root causes mechanical nerve fibre deformation and changes in the nerve root circulation, which result in pain and functional changes.

Mechanism

A posterior disc displacement (Fig. 33.10a) usually remains more or less under the physical influence of the posterior longitudinal ligament. The resistance of the ligament keeps the bulge in place or tends to push the protrusion forwards again, as happens during the spontaneous or manipulative reduction of a disc in acute lumbago: the pressure exerted by the ligament is higher than the intradiscal pressure and moves the bulge gradually forwards.

Sometimes, however, when intradiscal pressure remains high, the displaced tissue is pushed more and more laterally towards the posterolateral edge of the disc – a zone of lesser resistance (Fig. 33.10b).[92,93] Moved laterally, and freed from the counterpressure of the strong central part of the posterior longitudinal ligament, the bulge enlarges, lifts or ruptures the lateral ligamentous expansion and herniates into the lateral compartment of the epidural space where it compresses the nerve root. This is the typical development of a secondary posterolateral protrusion leading to a classic attack of sciatica.

For a good understanding of the clinical picture, it is important to remember that the severity of the symptoms depends not only on the mass of protruded disc material[94] but also on other factors (Fig. 33.11). Among these, the distension within the mass – in other words, the softness or hardness of the bulge – plays an important role.[95,96] Furthermore, the relative fixation of the root to the bony elements of the intervertebral foramen can determine the degree of traction on it.[97–99] Finally the degree of inflammation of the nerve is a significant element in producing symptoms.[100] There can be a direct chemical injury to the nerve root,[101–105] or extra- and intraneural swelling,[106,107] with further compression.[108] Many experts have emphasized that pain is provoked mainly when the nerve root is the site of a chronic irritation,[109–111] and experimental confirmation of this has been obtained by inflating Fogarthy balloon catheters

Box 33.3

Summary of discodural backache

Definition

- Backache, sometimes with dural reference in buttocks or legs, which is caused by a posterior shift of disc material constantly or intermittently pressing against the dura mater

Symptoms

- Acute, chronic or recurrent
- Onset indicates the type of protrusion:
  - Acute onset: annular protrusion
  - Slow and gradual onset: nuclear protrusion
  - Acute onset, with slow worsening: mixed protrusion
- Pain is unilateral, central, bilateral or alternating (extrasegmental pain reference in buttocks and legs is possible)
- Pain is increased by particular movements: as a rule sitting, coming upright after sitting, and getting out of bed are the most painful
- Dural symptoms may be present
- Particular types of backache
  - Continuous pain, 'bruised dura'?
  - Nocturnal/morning backache
  - Self-reducing disc

Signs

- Articular
  - Partial articular pattern
  - Deviation: in upright position, in flexion or alternating
  - Momentary deviation during flexion
  - Painful arc
- Dural (not always present)
  - Neck flexion?
  - Positive straight leg raising
  - Painful arc during straight leg raising

Treatment

- Manipulation
  - Annular lesions
  - Small nuclear lesions
- Traction
  - Nuclear lesions
  - Disc lesions unaltered after manipulation
- Epidural injections
  - Intractable backache
  - Nocturnal and morning backache
  - Bruised dura
- Back school: only as a prophylactic measure
- Sclerosing injections
  - Recurrent discodural conflict
  - Self-reducing nuclear protrusions
  - Nocturnal and morning backache

placed around the roots, which produce sciatica only if the pressure is maintained long enough to set up an inflammatory reaction.[112] During operations using progressive local anaesthesia, sciatica can be produced only by direct pressure or stretch on an inflamed nerve root, whereas pressure on a normal root is painless.[65]

It should thus be clear that the severity of sciatic symptoms and signs is a function of the magnitude of the mass, the intensity of the discoradicular contact and the inflammatory responses around the nerve root.[113] For all these reasons, *sciatica is not simply the existence of a bulge*, demonstrable on CT. Judging the severity of sciatica therefore depends only on data obtained from the history and clinical examination. Technical investigations usually add little information.

Symptoms and signs, the consequence of the involvement of the nerve root in the pathological changes, are articular, dural and nervous. Nerve root sleeve and nerve root fibres give rise to two different sets of clinical findings: dural and parenchymal.

The dural sleeve

Symptom: segmental pain

The dural investment is first to be compressed and inflamed, and causes the appearance of radicular pain. Unlike extrasegmental pain from pressure on the dura, pain stemming from the dural root sleeve follows exactly the rules of segmentally referred pain[114] (see Ch. 1). However, an inexperienced examiner may sometimes find it difficult to differentiate segmental radicular pain from extrasegmental dural pain. The following features may then be of value. First, dural pain is never felt beyond the ankle, whereas radicular pain of L4, L5, S1 and S2 origin usually spreads to the foot and the toes. Second, dural pain is not restricted to precise dermatomes of which the patient can give an accurate description but is felt vaguely over a large area. The patient will therefore be more imprecise in describing the area.

Sign: alterations in mobility

The dural sheath of the nerve root moves in relation to the neighbouring structures. As the course of the nerve root is downwards and slightly anterior and the nerve root is loosely bound to the pedicle below by the lateral root ligament, it will be caught against the posterolateral aspect of the disc when downward traction is exerted[115] (Fig. 33.12).

During SLR, the L4, L5, S1 and S2 roots undergo a downward and anterior excursion of 2–4 mm at the level of the intervertebral foramen[116] (see p. 430). It is obvious that, in large posterolateral disc protrusions, the mobility of these roots is impaired and SLR painfully limited. Sometimes a painful arc rather than limitation is observed: pain appears during the movement but disappears when the leg is raised higher – an indication of a small projection, which the nerve catches against and then slips over.[117] Such a momentary pain is an encouraging sign for conservative treatment.

The third lumbar root continues into the femoral nerve, which remains relaxed during SLR. Therefore lack of pain with

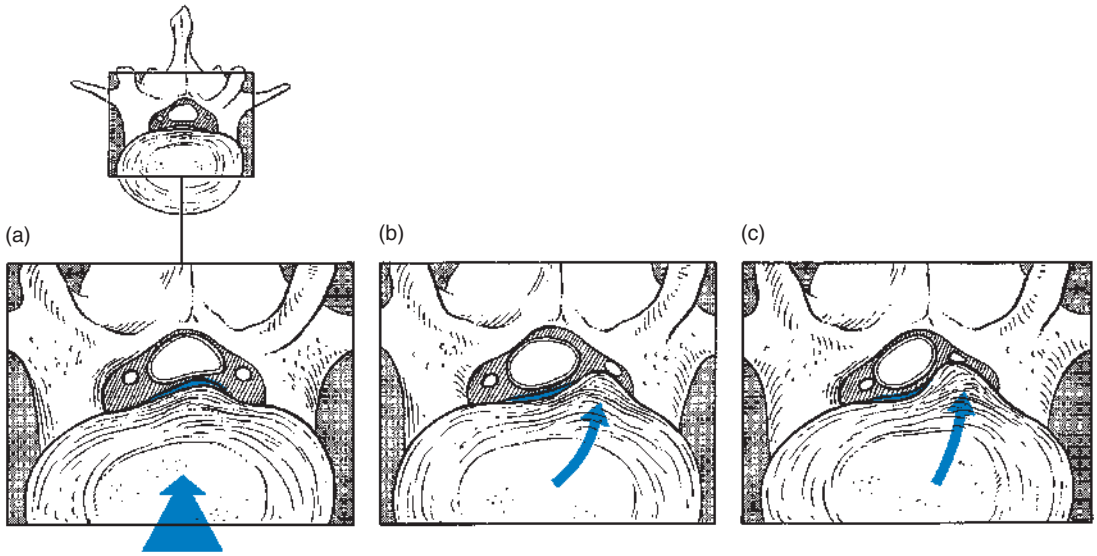


Fig 33.10 • Mechanisms of sciatica (see text for detail).

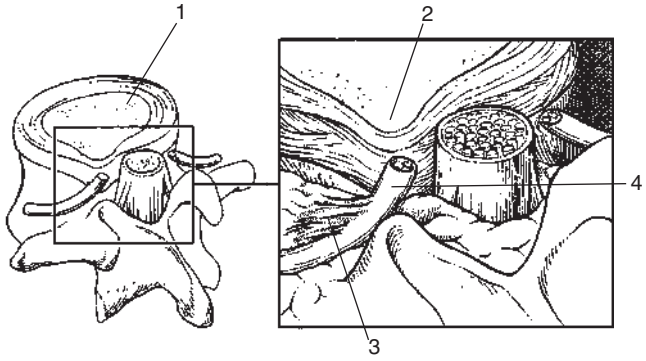


Fig 33.11 • Factors in the production of symptoms and signs in sciatica: 1, in hardness; 2, size of the bulge; 3, relative fixation of the nerve root; 4, inflammation of the nerve root.

this manœuvre is not an indication that the L3 root is intact. A better test is knee flexion in the prone position.[118,119]

There are no clinical tests for the mobility of the S3 and S4 roots.

The parenchyma

Mechanical factors are mainly responsible for intraneural blood flow and formation of intraneural oedema, which in turn causes structural damage to the nerve fibres. It has also been suggested that breakdown products from the degenerating nucleus pulposus may leak through the root and induce a 'chemical radiculitis'[100,120] and that autoimmune mechanisms play a role in the inflammatory tissue reactions seen around degenerating discs.[121,122] The details of disturbances in nerve tissue during discoradicular interaction are not yet fully understood; however, their clinical consequences and functional changes are clear.[123] On the one hand, hyperexcitability of the fibres results in paraesthesia[124] and muscle fasciculations.[125] On the other hand, there is loss of nerve function – muscle weakness, sensory deficit and reflex changes.

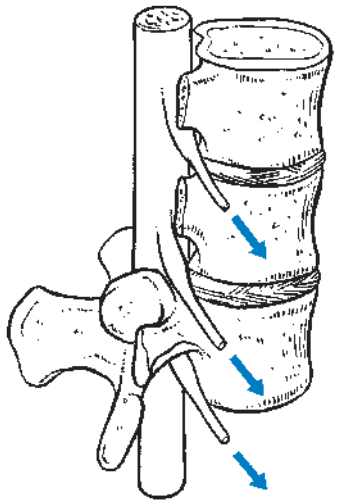


Fig 33.12 • The nerve root drags anteriorly on the disc bulge.

Symptom: paraesthesia

Pins and needles only appear as the result of hyperexcitability of nervous tissue. They are therefore pathognomonic for peripheral nerve lesions. In nerve root compression they are strictly limited to the respective dermatome and occupy an area at its distal extremity. As a rule, paraesthesiae tend to disappear when numbness begins – hyperexcitability ceases when pressure has induced a sensory deficit (see Ch. 2).

Signs: sensory deficit, motor deficit and reflex changes

Sensory and motor deficit are not always easy to detect, and both legs should be carefully compared. Motor deficit usually remains moderate and most patients are unaware of loss of function. Nevertheless, weakness has important therapeutic and prognostic consequences, and it must be sought in each muscle group.

On account of the obliquity of the nerve roots and the fact that the sensory and motor rootlets have separate courses within the dural sleeve, it is possible for one protrusion to compress one root, half of a root, two roots or the halves of two consecutive roots (see Fig. 33.16 below). This is particularly so in a lesion at the fifth lumbar level, where the same protrusion compresses the motor rootlet of L5 and the S1 sensory fibres.

The root signs in discoradicular interactions (see Fig. 33.17 below) are as follows (see reference 126 and Cyriax[45]: pp. 283–286):

- *L1:* cutaneous analgesia at, and just below the inner half of the inguinal ligament.
- *L2:* cutaneous analgesia from the groin to the patella, and weakness of the psoas muscle.
- *L3:* cutaneous analgesia over the anterior aspect of the leg, from the patella to the ankles, weakness of the psoas and quadriceps muscles and a sluggish or absent knee jerk.
- *L4:* cutaneous analgesia over the outer ankle, dorsum and inner aspect of the foot and big toe, and weak tibialis anterior and extensor hallucis muscles.
- *L5:* cutaneous analgesia over the outer leg, the dorsum of the foot and the inner three toes, weak extensor hallucis and peroneal muscles; the ankle jerk may be absent or sluggish.
- *S1:* cutaneous analgesia of the posterolateral aspect of the leg, behind the lateral ankle, the lateral aspect of the foot and the two outer toes; the peronei, calf hamstrings and gluteus medius muscles may be weak; the ankle jerk is sluggish.
- *S2:* cutaneous analgesia of the dorsum of the leg and the heel, and weakness of the calf, hamstrings and gluteus medius muscles.
- *S3:* cutaneous analgesia at the inner aspect of the thigh; no muscle weakness.
- *S4:* numbness of the saddle area and dysfunction of bladder and rectum.

History

Secondary posterolateral protrusion

This is 'classic sciatica'. The patient is usually between 20 and 50 years of age. A number of attacks of backache or lumbago have taken place. Again, a sudden or increasing backache occurs, which tends to become unilateral. Then the pain shifts towards one aspect of the limb, where it occupies one particular dermatome; as a rule, the backache ceases when pain in the limb begins.

The exact *localization* of the pain is of considerable help in diagnosis. Pain at the groin and the front of the thigh may indicate a second or third nerve root compression. When anterior pain spreads down towards the ankles, the third nerve root is at fault. Pain at the lateral aspect of the leg and crossing the dorsum of the foot is caused by a fourth or fifth disc lesion. The differentiation between these is not always easy. If the big toe only is affected, both roots may be responsible, but if in addition the second and third toes hurt, the lesion is of the fifth lumbar root. Pain at the lateral aspect of the leg and foot, reaching the two outer toes, indicates a first sacral lesion, whereas a second sacral root pain occupies the dorsal aspect of the thigh, calf and heel.

After a while, and in addition to the increasing root pain, the patient will report *paraesthesia* at the distal aspect of the respective dermatome. Pins and needles not only indicate that nerve fibres are being compressed, which immediately excludes other, non-radicular sources of the segmental pain, but also provide a better pointer to which nerve root is at fault.

Later, the patient will mention *numbness and weakness* of the leg or foot. As weakness increases to a maximum, the pain ceases – the root has atrophied.

The symptoms caused by a *posterolateral disc displacement have a striking similarity.*[127] The history is vital in the diagnosis of sciatica and is probably the most important diagnostic technique. The onset and development of symptoms, their relation to posture and exertion, the exact localization of the pain and the presence of paraesthesia and numbness are extremely important features in diagnosis and decision making for both treatment and prophylaxis. The pain often increases on sitting, and eases in recumbency, especially when the patient adopts the 'psoas position' – supine, with the hips and knees flexed (see Fig. 33.7). In severe instances, however, when the continuous pressure has induced a considerable inflammation of the dural sheath, the pain may be continuous, sometimes increasing at night. As a rule, standing is better than sitting, but sometimes walking can be difficult, especially if nerve root mobility is impaired in such a way that moving the affected leg forwards during the swing phase drags on the sciatic nerve. The patient then walks with an adaptive gait. In lesions of the third lumbar root, pain increases on standing or reclining and eases only in sitting, because the latter is the only position which relaxes the tension on the femoral nerve and the third lumbar root. These patients often prefer to sleep sitting up. In discoradicular interactions the symptoms are usually worse in the morning, probably as a result of the increased swelling pressure in the disc (Krämer[32]: pp. 17–21). In an active patient, the pain decreases somewhat around midday and increases again by the evening. Coughing and sneezing may cause pain in the gluteal area or in the limb.

Symptom sequence

The progression from central bulge to posterolateral protrusion, with pressure on the nerve root sleeve and subsequently the nerve root fibres, is reflected in the sequence of symptoms. Initial backache is followed by radicular pain, then paraesthesia and sensory and motor deficits. As the last two increase, the nerve root sheath becomes insensitive and sciatic pain abates. A large protrusion which causes nerve atrophy may produce complete loss of function: the patient may then be spontaneously and subjectively better but anatomically worse. However, most patients recover from the palsy without lasting loss of power, especially if only one root is paretic.

For diagnostic, therapeutic and prognostic reasons it is very important to differentiate between radicular pain and dural pain in the limb. In acute lumbago or severe backache, this is usually not very difficult, since the referred pain in the leg is not as severe as the lumbar or gluteal pain itself. Also, when

the patient presents with a clearly delineated, severe ache in the leg, it is easy to recognize the segmental pain of root compression. When, however, as happens occasionally, only referred dural pain is present, distinguishing this from segmental pain can be more difficult. The typical example is pain in the groin, which can stem from dural reference, segmental reference from L1–L2 or segmental reference from T12. Pain in the buttocks also causes diagnostic problems sometimes: in one or both buttocks it is usually of dural origin, especially if the pain is restricted to the upper buttock and is not as severe as the back pain. Pain in one lower buttock is only rarely dural and is more often a segmental reference from S2.

Although the sequence of symptoms as set out above is always present in primary posterolateral protrusions, it is not always as typical as described. Pre-existing backache may never have occurred but backache and sciatica have originated almost simultaneously. Careful enquiry, however, may show that, 2 days or so before the onset of the sciatic pain, there had been vague sacral aching after prolonged sitting or difficulties on bending. Alternatively, acute backache may have changed almost immediately into leg ache. The short or insignificant period of backache will not be mentioned unless specific enquiry is made. Sometimes the pain in the back does not disappear when the root pain comes on and this is particularly so in elderly patients. In these cases, the symptoms can go on indefinitely, which is not the case in 'classic sciatica'. Especially when the back aches more than the limb, this type of sciatica shows little tendency to spontaneous cure.

Primary posterolateral protrusion

In this type of discoradicular interaction, the pain is radicular from the beginning and there has been no previous backache. The lesion is nuclear and only affects young patients between the ages of 18 and 35 years. The protrusion is usually at the L5–S1 joint, where it compresses the S1 root.

The history is typical. A young patient states that a calf aches when sitting is prolonged. Alternatively, the pain may be at the lateral side of the knee and the leg, but seldom spreads to the foot. Very occasionally, the onset is with numbness in the heel, later spreading to an ache in the calf and thigh. The moment the patient stands up, the pain disappears. Previous backache has not occurred and the patient usually does not associate the pain in the calf or knee with a disorder in the back; however, a cough or sneeze hurts in the leg. The ache gets slowly worse over a period of months, during which it spreads upwards to the posterior aspect of the thigh. By the time the pain has reached the buttock, it may be constant except in bed. Lumbar flexion and SLR gradually decrease. In the end, even extending the knee becomes painful, which forces the patient to adopt a waddling gait with flexed knee.[128]

The diagnosis of a primary origin of the posterolateral protrusion is important with respect to treatment. Because a primary posterolateral protrusion is always nuclear, manipulations do not influence the symptoms.

'Bruised' dural sleeve

Sometimes the root pain is constant, unaltered by posture or exertion. Usually the patient has a history of a typical discoradicular pain from which recovery has been largely complete. However, the pain failed to disappear completely and is now more or less constant, although less so than before. Alternatively, the patient may have had a discectomy, which improved his or her condition considerably but not to the point of cure. Sometimes the pain is bilateral, which strongly suggests spondylolisthesis. Clinical examination reveals nothing but a full range of movement.

A possible explanation for this unusual pain syndrome is probably a persisting inflammation of the root sleeve, resulting from a past disc lesion that has undergone spontaneous reduction or has been surgically removed. Although there is no more discal contact, the sleeve remains irritated.[94]

Epidural injection with local anaesthesia is necessary to determine the diagnosis and often abolishes the pain permanently.

Differential diagnosis

Sciatica has to be differentiated not only from dural extrasegmental pain in the limb (see earlier) but also from segmental pain not caused by discodural interactions. These disorders will be discussed in detail in a separate chapter, but a few salient points are listed here:

- Sciatica in the elderly is more often caused by a lateral recess stenosis and, especially if the pain appears during standing or walking, the existence of a narrow radicular canal should be suspected (see Ch. 35).[129]
- Bilateral sciatica is seldom caused by one disc lesion, unless there is a massive protrusion of the disc with rupture of the posterior longitudinal ligament. Evidence of an S4 lesion will also be present (see above). Another, although uncommon, possibility is the presence of two posterolateral protrusions, one at L5 and another at L4 on the other side. Alternatively, one disc has developed two posterolateral disc protrusions, one at each side of the posterior longitudinal ligament. In bilateral sciatica in younger patients, spondylolisthesis should be considered; in elderly patients, suspect spinal or lateral recess stenosis.
- Alternating sciatica is rarely caused by a disc lesion but suggests the sacroiliac arthritis of an early ankylosing spondylitis.
- Increasing backache together with worsening sciatica indicates a serious disorder, especially if the pain does not vary with exertion but steadily gets worse, irrespective of posture or exertion (see Ch. 39).

Clinical examination

Inspection

A lateral pelvic tilt or a deviation may be present. As in lumbago, the deviation can be towards or away from the painful side, depending on the position of the protrusion. If the latter occurs lateral to the nerve root, there is a lateral shift towards the painless side, which reduces contact with the root (Fig. 33.13). If the protrusion is located at the 'axilla' between

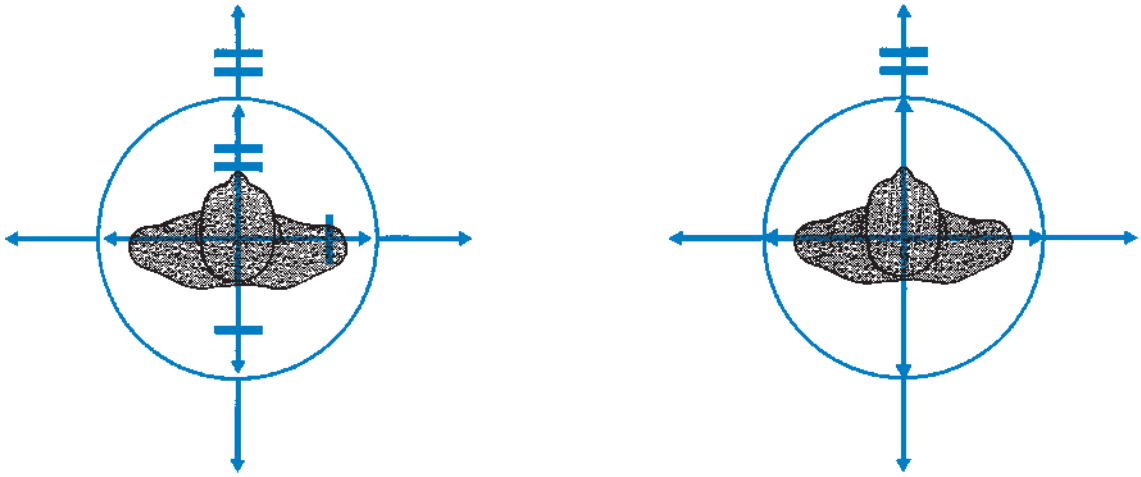


Fig 33.13 • Common patterns in sciatica. Arrows within the circle indicate pain in the back/buttock. Arrows outside the circle symbolize pain in the leg.

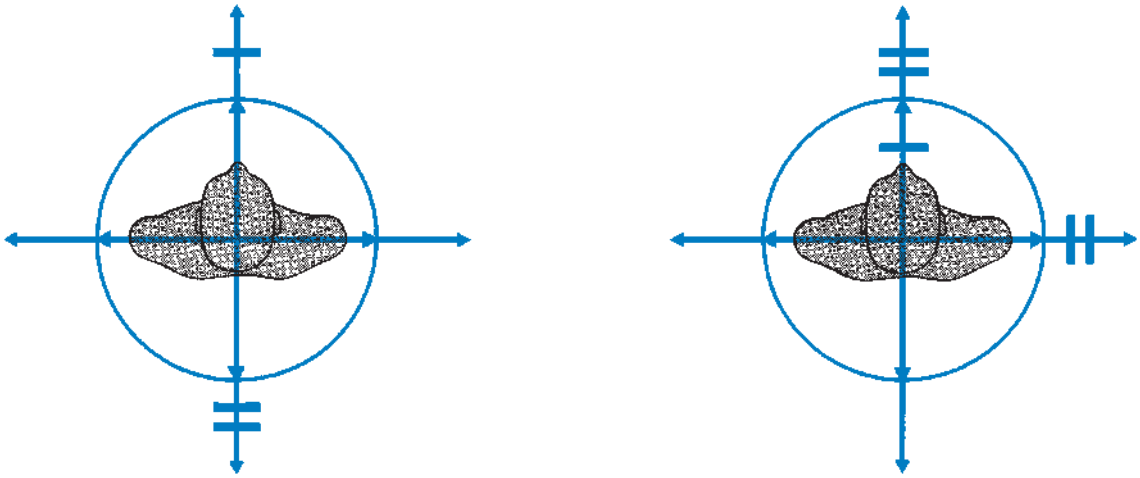


Fig 33.14 • Unfavourable patterns in sciatica.

the dura and dural sleeve of the root, the spine is deviated towards the painful side in an attempt to decrease pressure on the root.

It is rare to see patients with sciatica adopting the flexed posture so common in acute lumbago. Extension is not possible and every attempt to straighten the back is followed by severe pain at the back of the leg. This form of sciatica is very difficult to treat conservatively and most patients result in having a discectomy.

In compression of the L3 root, the patient may adopt a specific posture: slight flexion of the trunk and flexion of the hip. Patients with acute hip lesions position themselves similarly and a clinical distinction between the two must be made.

Spinal movements

In sciatica, as in lumbago or backache, articular signs indicating a partial blocking of the joint may be present. It is important to remember, however, that flexion is not only an articular sign but also a test of the mobility of the root and, except in L3 lesions is usually limited because of increasing pain in the limb. The classic pattern in sciatica – the consequence of disc lesions – is thus a severe and increasing leg ache during flexion, together with pain felt in the lower back or upper buttock during one or two of the other lumbar movements. If there is discoradicular contact only and the dural tube remains untouched during lumbar movements, pain will be felt in the limb only on flexion. This is typically the case in a primary posterolateral protrusion, where flexion is the only painful and limited movement.

Sometimes an increasing lateral tilt is seen during flexion. Again, the deviation can be towards or away from the painful side, depending on the position of the protrusion.

If side flexion or extension hurts in the leg instead of at the lumbar or gluteal area, manipulation nearly always fails, especially if the patient is less than 60 years old (Fig. 33.14).

A full and painless range of flexion does not imply the absence of a disc lesion – it is possible for even a large protrusion not to cause limitation of flexion. In L3 compressions, for instance, the nerve is relaxed during flexion and therefore this

movement can be painless. In severe compression, in which root atrophy has developed, flexion is again of full range and painless. The patient has lost the pain and the lumbar movements have returned to their normal degree but the considerable weakness of some muscles is evidence of the gross posterolateral disc lesion.

Root tests

Testing the mobility of the root

Straight leg raising examines the mobility of the nerve root sleeves of L4 and S2, whereas prone-lying knee flexion tests that of the L3 root. It is important to remember that each nerve root is incompletely fixed by a ligamentous band, running from the sheath of the nerve root to the inferior pedicle of the respective foramen.[99,130,131] During SLR, the sciatic nerve is pulled downwards and the root dragged forwards. Because of its fixation, the nerve root cannot slip away and it is caught against any space-occupying lesion at the front of the canal.[98] In contrast, compression of the nerve root from above or from behind does not result in a decrease in root mobility. The anterior and relatively fixed position of the root protects it from a posterior compression when SLR is performed. This observation is extremely important in the differential diagnosis of radicular pain. Lateral recess stenosis or hypertrophy of the facets causes compression from behind (posterior wall lesions) and does not influence the mobility of the root. Thus, SLR (or femoral stretch) specifically tests the mobility between the nerve root and the posterior aspect of the intervertebral joint (anterior wall).

However, limitation of nerve root mobility is not pathognomonic of a disc lesion,[132,133] as the specificity of the SLR test is about 90%.[134]

- Any space-occupying lesion at the anterior aspect of the nerve root canal which interferes with the anterior aspect of the nerve root will cause the same clinical feature. Such is the case in neuromas and tumours, for instance, which cause as much limitation of SLR as do disc lesions.
- Lesions in the buttock can also produce significant limitation of SLR. The combination of a limitation of SLR with serious limitation of flexion of the hip immediately draws attention to such lesions (see Section 12).
- Lesions of the hamstrings and sacroiliac joints also cause pain at the extreme of SLR, as the result of direct tension being exerted on tender structures.

On the other hand, full and painless SLR does not exclude a disc lesion[135,136]:

- Lesions at L1, L2 and L3 are not detected by SLR because the sciatic nerve does not directly pull on the roots of these levels. However, the L3 root can be subject to some traction at the extreme of SLR because of the downward pull on the dura mater, exerted from the nerve roots below.
- Small posterolateral protrusions are sometimes not large enough to impinge on the dural sleeve during mobilization of the roots in the supine position. In contrast, trunk flexion with the patient erect can provoke pain in the leg because the joint is now compressed by the body weight and the bulge is squeezed in the direction of the root. There is therefore no inconsistency in a patient being unable to bend fully forwards and yet having a full and painless SLR.
- In root atrophy, SLR is also of full range and painless.
- When sciatica causes gross limitation of extension of the trunk, SLR is also often of full range and painless, although at laminectomy a large disc protrusion may be seen. These cases of sciatica are resistant to conservative treatment. It has been suggested that the nerve root emerges here a little higher up in the foramen and therefore is not affected during SLR or bending.

For these reasons it must never be assumed that a disc lesion *cannot* be present simply *because* SLR is full-range and painless. Tests for dural mobility must always be interpreted in the context of other clinical findings and SLR as an isolated test has no diagnostic significance.

However, *a painful arc during SLR is pathognomonic of a disc lesion* and also indicates that the lesion is so small that the nerve root is only temporarily intercepted. A painful arc is an encouraging sign that manipulative reduction will be successful.

Sometimes SLR on the painless side causes pain in the other limb and sometimes may even be limited. This phenomenon – crossed SLR – is encountered more frequently at the L4–L5 level,[137] and indicates axillary protrusion: the downward movement of the dura mater drags the medial aspect of the root against the protrusion (Fig. 33.15).

When neurological deficit is not present, the degree of restriction of SLR is proportional to the pressure exerted on the nerve root. The course of SLR over time is then the best objective criterion by which the development of sciatica can be followed. However, this situation changes when conduction becomes impaired, and then the degree of interference with this affords the best measure of the size of protrusion.[45]

Testing root conduction

Once the compression of nerve fibres is such that it causes deformation, neurological deficit becomes clinically detectable. From this stage onwards, the magnitude of the protrusion is no longer determined by the limitation of root mobility but is reflected in the degree of deficit. Sensory and motor conduction and reflex changes must be carefully tested, since the existence of neurological deficit has both diagnostic and therapeutic value: diagnostic because it indicates pressure on the nerve root(s), and is a standard for the degree of discoradicular interaction; therapeutic because signs of interference with conduction mean that an attempt at reduction is no longer indicated.

Clinical examination of the conduction of the nerve roots must be thorough. Because the lesion is often incomplete, most of patients are unaware of any loss of power or sensitivity, except when complete root atrophy is present.

Because of the oblique course of the nerve roots, a disc lesion can compress one single root or two consecutive roots. It is also possible for compression to affect just the upper part of the root and cause sensory deficit, whereas pressure from below will result in motor palsy. A large protrusion can

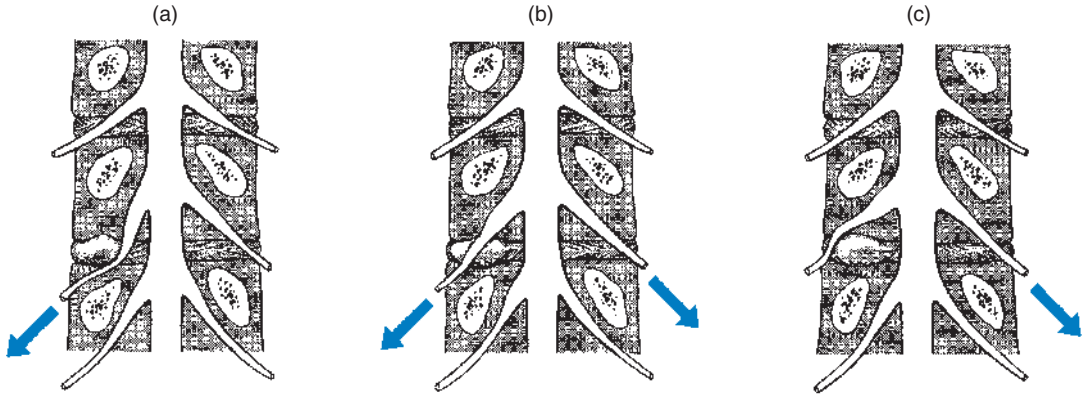


Fig 33.15 • Straight leg raising: (a) unilateral limitation; (b) bilateral limitation; (c) crossed limitation.

compress two consecutive roots, or the motor fibres of one root and the sensory part of the root below (Fig. 33.16). A fourth–fifth root compression, resulting in a permanent drop foot, can stem from a large protrusion at the fourth level. A fifth–first sacral compression can occur at the fifth level.

Combined third–fourth palsies are extremely rare, and seem to occur only in congenital anomalies of the nerve roots.[138,139] Also triple palsies are not possible in single disc lesions. Because L2 disc lesions are extremely rare, an L2 palsy (psoas) always suggests a non-discogenic lesion. Also, bilateral palsies are scarcely ever caused by disc lesions; hence neoplasm should be suspected when there is bilateral weakness. Serious lesions should also be suspected if total loss of power is present, as it is unusual for a disc lesion to cause a complete palsy.

The power of all the key muscles is tested and alterations in skin sensitivity are sought. The latter are subjective and may at times be very difficult to assess. It is also vital to test identical areas in both limbs, at the same time or consecutively.

Sometimes, in severe sciatica, the affected leg is found to be colder than the other. Attention may be drawn to this by the patients and confirmation obtained during the clinical examination or by thermography.[140–142] In our experience, a cold limb only occurs in combination with neurological deficit.

Ankle and knee jerks sometimes disappear earlier than the muscle power or skin sensitivity (Fig. 33.17). Loss of ankle jerk is permanent in about half of the cases, whereas the knee jerk recovers more often. It is a curious phenomenon that both ankle jerks occasionally disappear during a unilateral sciatica. Bilateral loss of ankle jerk should therefore not be a cause for concern.

Natural history

The majority of patients suffering from a discoradicular interaction heal spontaneously without surgery. Although low back pain can continue for years, sciatica usually has a natural history of spontaneous improvement, even if there is clinical evidence of weakness or radiological evidence of disc extrusion. Despite an abundant literature that proves the contrary, there is still a belief among doctors and patients alike that a herniated disc should be treated operatively. Especially if the signs and

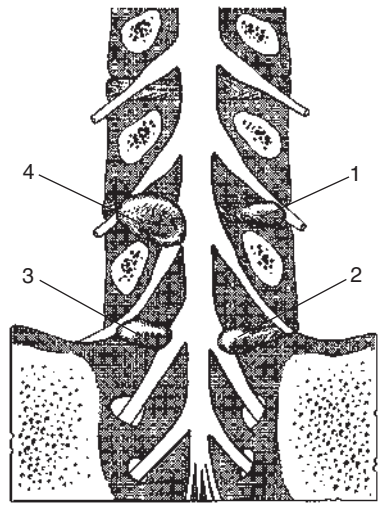
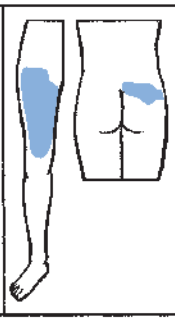
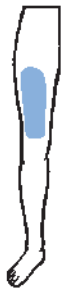
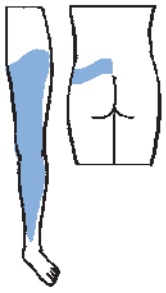
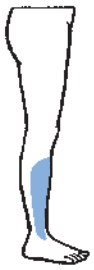
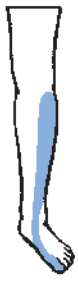
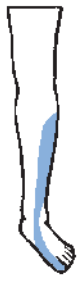


Fig 33.16 • Compression of the root at the same level (1), the root below (2), two halves of two roots (3) and the whole of two roots (4).

symptoms have not improved after a few weeks of bed rest, and if the diagnosis is confirmed by a positive CT or MRI, surgical intervention is recommended.[135] This opinion is not supported by studies, however, which show equally good or better results after conservative treatment.[143] As soon as the early 1970s, studies found no difference between the final results of surgical and non-surgical therapy after 7–10 years of observation.[144–146] These conclusions have been confirmed by more recent work which found that conservative treatment has as good a result as the operative approach after 1 and 2 years of follow-up.[147–150,1,32] Obviously, a better knowledge of the natural history of discal herniations and of the mechanisms leading to changes in the extruded discal tissue would be of great help in planning the therapeutic procedure.[151]

As a rule, root pain takes 6–12 months to recover at the L4 and L5 levels, but at the L3 level it is usually faster. The process seems to start from the moment that the protrusion has moved laterally beyond the edge of the posterior longitudinal ligament. It is a striking clinical fact that chronic backache can go on forever but that, once it has changed into localized root

Sheath				Nerve fibres		
	Symptom	Sign	Symptom	Signs		
Root	Pain	Mobility	Paraesthesia	Motor deficit	Sensory deficit	Reflex disturbances
L1		None	None	None	Inner half inguinal ligament	None
L2		None (femoral stretch)		Psoas		None
L3		Femoral stretch		Psoas quadriceps		Knee jerk
L4		SLR		Tibialis anterior, Extensor hallucis longus		Knee jerk
L5		SLR		Extensor hallucis longus, Peronei, Gluteus medius		Ankle jerk

Continued

Fig 33.17 • Radicular signs and symptoms in sciatica.

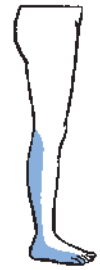
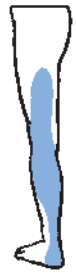
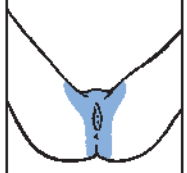
Sheath				Nerve fibres		
	Symptom	Sign	Symptom	Signs		
Root	Pain	Mobility	Paraesthesia	Motor deficit	Sensory deficit	Reflex disturbances
S1		SLR		Peronei, Calf muscles, Hamstrings, Gluteal muscles		Ankle jerk
S2		SLR		Calf muscles, Hamstrings, Gluteal muscles		Ankle jerk
S3		None	None	None	None	None
S4		None	Perineum	Sphincters	Perineum	None

Fig 33.17 Radicular signs and symptoms in sciatica (continued).

pain, the evolution to spontaneous recovery has started. The prognosis of spontaneous recovery from root pain is related to the date of onset. If backache or gluteal pain continues unchanged – which indicates that the bulge is still in dural contact – spontaneous relief cannot be expected. Also, in elderly patients, spontaneous recovery from root pain commonly does not occur, probably as the result of combination with lateral recess stenosis. In bilateral root pain, spontaneous recovery does not often take place and symptoms can continue unchanged for many years.

The different mechanisms that result in spontaneous recovery in sciatica, enumerated by Cyriax (his pp. 233–234),[45] are spontaneous reduction, erosion of the posteroinferior aspect of the vertebral body, bulge shrinkage and root atrophy. The most important mechanisms are bulge shrinkage and root atrophy.[152]

Spontaneous reduction of the bulge

This accounts for most of the recoveries in lumbago and acute backache but does not play an important part in the natural history of sciatica. Because the bulge lies lateral to the ligament, centripetal force is not very great and the protrusion has no tendency to return forwards. For the same reason, manipulative treatment in sciatica is not as successful as in lumbago or backache.

Erosion of the posteroinferior aspect of the vertebral body

As described by Young, this is probably not a very important mechanism in recovery from sciatica.[153] However, there have also been recent reports of disc herniation eroding bone and thus effectively creating more space and less pressure.[154–157] It is considered likely that the defect is caused by a purely mechanical effect.[156,157]

Bulge shrinkage

The protrusion slowly shrivels away over the course of a few weeks or months, and this probably accounts for the slow and progressive spontaneous recovery from uncomplicated sciatica without neurological deficit.[158,159] CT and MRI studies have demonstrated that a high proportion of intervertebral disc herniations have the potential to resolve spontaneously.[160,161] The largest herniations appear to be the most likely to undergo a significant decrease in size. The presence of large herniations and/or disc extrusions should therefore not be considered as indications for surgery.[162–165] On the contrary, MRI reports confirmed that the more degenerate the disc and the larger the initial herniation, the more the size of the herniated fragment decreased.[166–169] There also seems to be a higher incidence of diminution of lateral hernias, compared to central hernias,[170,171] and the further the herniated nucleus pulposus migrated, the more rapid decrease in size could be observed,[172,173] with full regression of an extruded fragment in all cases.[174]

The precise mechanism is not totally understood, but one plausible explanation could be that the dissolution of disc material is accelerated when the latter enlarges and becomes deprived of the nutrient influence of the endplates and the posterior longitudinal ligament.[175,176] Loss of water content then deflates the protrusion, which decreases the pressure on the nerve root. Additionally, cellular infiltration of the epidural space promotes phagocytosis[177–179] of the offending nuclear material, which is transformed into scar tissue.[180] Later on, inflammation[108] and the resultant venous congestion[181] decrease, which in turn further reduces pressure on the root. The spontaneous shrinkage of the protruded material is probably comparable with the disc shrinkage induced by chemonucleolysis (see p. 590).[182,183]

Root atrophy

As ischaemic root atrophy becomes complete, the pain abates quickly and the patient experiences symptomatic improvement. It is obvious that this situation does not represent a neurophysiological recovery – initially the patient is anatomically worse. Recovery from the palsy may take 6–18 months.

The clinical picture is as follows. A patient with sciatica suddenly experiences an increase in pain. After a certain length of time (hours to days), the pain ceases and the skin in the respective dermatome becomes numb. From this time, there may be some weakness in the foot or the leg. Examination shows full range of SLR but complete root dysfunction, both motor and sensory. In root atrophy, there seems to be a relationship between the degree of pain relief and the neurological deficit: the more marked the neurological weakness, the quicker the pain disappears. Neurophysiological recovery is usually slow and not always complete – the atrophy may lead to some slight permanent weakness if two consecutive roots are paretic. A large posterolateral protrusion at the fourth level, for instance, compressing both L4 and L5, may occasionally result in permanent foot drop. In general, however, and when only one nerve root is involved, complete return of strength within 1 year is the rule. The spontaneous recovery of neurological deficit has been studied in monoradicular weakness: in all cases, full recovery was complete in an average of 7 months; when there was multiradicular weakness, only 13% recovered fully.[184] The present author re-examined 42 patients with a monoradicular deficit due to a discoradicular interaction, 1–4 years after they had recovered from their sciatic pain; all were completely rehabilitated and muscular weakness could not be detected. Some cutaneous analgesia may be permanent: for instance, the outer side of the foot stays numb after an S1 palsy, or the dorsum of the foot after an L4 root palsy. Some permanent sensory dysfunction remains in about 35% of patients after 10 years.[145] In about half, the ankle jerk will not recover but the knee jerk usually does.

The speed of recovery from neurological deficits is very variable and difficult to predict. Usually, a nerve root recovers slowly over 6–12 months, but it can recover with inexplicable rapidity, sometimes within 2–4 weeks and before the pain has ceased completely. This cannot be explained by a simple regrowth of the axons – as regrowth of nerve takes place at a rate of about 1.5 mm a day – and it has been suggested that there might be a peripheral reinnervation of the muscle from intact nerve endings.[185,186]

It is important to remember that myelograms[130] and CT can remain positive for up to 15 months after the pain has disappeared. The same phenomenon has been reported after successful treatment with chymopapain.[187] It is therefore unwise to rely on CT for evaluation of the course of sciatica, and again the clinical facts are more important than the radiological appearances.

Because most patients are unaware of loss of motor function, the statement that there is 'some loss of power' must immediately be followed by explanation and reassurance. Too many operations are carried out because there is slight weakness and the patient has been told that there is a risk of persistent lameness. To date, there is no evidence that surgically

treated patients recover more quickly or better from a neurological deficit than do those treated conservatively. Slight or moderate weakness is therefore not an indication for operation. However, if there is evidence of an incipient drop foot or the third and fourth sacral roots are threatened, surgery should be recommended immediately.

Once there has been a spontaneous recovery from sciatica, whether by erosion, shrinkage or atrophy, there is no likelihood of *recurrence of sciatica at the same level*. All the mechanisms of spontaneous cure seem to encourage some stabilization at the joint and therefore recurrence is not the rule. This does not imply that there might not be some chronic or recurrent backache because of a fresh lesion at another level or other mechanisms (ligamentous laxity, and posterior wall problems – see Ch. 34). However, and as a rule, patients who have recovered without surgical treatment do not need to take more care than others. They can therefore continue their normal lifestyle and perform any sports they used to do before the episode. This contrasts strongly with the attitude to be taken to those who have had a laminectomy. The tendency to recurrence then prohibits heavy work, and even with care a constant or intermittent ache may make them aware of their back. Seventy percent of patients who have undergone surgery still complain of backache and 45% of sciatica 4–17 years after the intervention and 37% continue to receive some form of treatment.[188,189] The incidence of re-operation ranges from 17 to 23%.[190–193] The decision to intervene surgically should therefore not be taken lightly and not until all possible non-operative management, including epidural local anaesthesia, has been tried. Even in an 'unrelenting' case, with tolerable *root* pain, the patient should be made aware of the chance of spontaneous recovery and encouraged to wait at least 8–12 months before opting for operation. With such a conservative approach, very few patients will need an operation. Our personal experience is that such an attitude is appropriate, provided pain remains reasonably controlled.

Treatment

The possibility of spontaneous resolution must influence any evaluation of treatment. Placebo treatment can be effective; for instance, in randomized trials, a placebo for chymopapain injection gave relief in 42–60%.[194–197]

Apart from awaiting a spontaneous cure or referring the patient for surgical treatment, there are two different strategies for solving discoradicular problems. The first is reduction of the protrusion. If it is not too large, not too laterally placed and not too long-standing, a trial of reduction should always be undertaken. If reduction is not possible, attention should be given to the second component of the interaction – the nerve root. A caudal epidural injection should then be given in an attempt to reduce some of the inflammatory reactions. Should this injection fail, nerve root infiltration can be tried.

As for the treatment of discodural interactions, there is no clear-cut overall treatment for sciatica (Fig. 33.18). As the anatomical basis of sciatica differs completely from one patient to another, treatment will always be chosen in relation to the symptoms and signs: 'Sciatica has many faces, and treatment should always be selective' (James Cyriax).

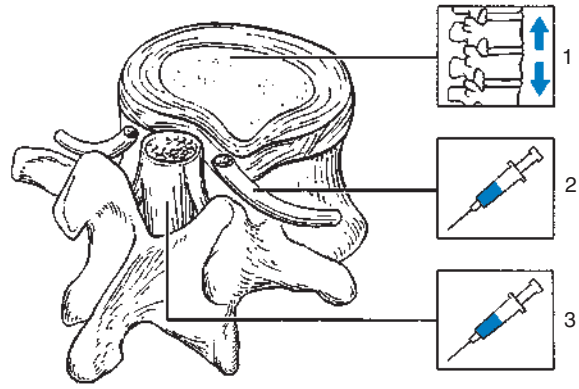


Fig 33.18 • Impact of the different therapies for discodural conflict: 1, repositioning; 2, nerve root infiltration; 3, epidural local anaesthetic.

Repositioning the disc by manipulation or traction

A few patients with sciatic pain can be treated by manipulation or traction. Only those protrusions that are not too large or too long-standing will have a reasonable chance of quick improvement with traction or manipulation, i.e. patients with recent root pain (less than 6 months' duration) and without neurological deficit. There are, however, a few exceptions. In elderly patients (over the age of 60), there is no time limit for manipulation, especially in those who still have low back pain after the appearance of the sciatica. Also, in recurrent sciatica, where the history indicates that the motor deficit stems from a previous attack, the present bout can sometimes be alleviated quickly by manipulation.

The choice between manipulation and traction is made on the data obtained from the history and clinical examination. If these indicate a soft nuclear displacement, traction should be applied. If the features point towards a hard annular protrusion, manipulation should be undertaken. In elderly patients traction is of no use, and in young patients suffering from primary posterolateral protrusions, manipulation will always fail. If neither the patient's age nor the symptoms indicate the consistency of the displacement, manipulative reduction should be undertaken first. If there is immediate improvement, such treatment can be continued. If there is no response, traction is substituted.

Only 30% of patients suffering from sciatica can be treated successfully by manipulation or traction, which means that for the majority another strategy must be considered.

Epidural local anaesthesia

If *impaired conduction (motor or sensory)* or *increasing nocturnal pain* shows that the discodural contact is intense, attempted manipulative reduction will almost certainly fail. The disorder should be treated by epidural anaesthesia. This also applies to *laterally situated protrusions*. Because the protrusion has moved lateral to the posterior longitudinal ligament, the ligament no longer pushes on the displaced bulge during manipulation or traction. Therefore if there is root pain only, manual

reduction will almost certainly fail. In contrast, the more that backache accompanies the sciatica, the greater the chance of relief by reduction.

If root pain is *long-standing*, an attempt at manipulative reduction will always fail. It is generally agreed that after 6 months of radicular pain, attempts at manipulative reduction are in vain.

Epidural local anaesthesia is also the treatment of choice in *sciatica that is recovering*; the patient is symptomatically over the worst and leg pain has largely subsided with bed rest.

Root pain without physical signs, the *'bruised' root*, is also treated by epidural injection. The injection initially given for diagnostic purposes often permanently abolishes the pain too.

The mechanism of caudal epidural injection is still a matter of debate. Probably the fluid has some hydrostatic effects – epidural injections with 50% of 0.5% procaine produce a hydrostatic pressure that removes the dural tube and the nerve roots from the bulge.[198] The effect is not just temporary but persists for the next few weeks. Another explanation for the results obtained after procaine injections in sciatica is that they might influence the chemical mediators of inflammation. Procaine seems to produce better results than lidocaine, perhaps because of the higher pH of procaine (6.5), which may have an influence on the chemical radiculitis.[100,199,200]

Nerve root infiltrations

Nerve root infiltration is an alternative way of dealing with the painful inflammation caused by a discoradicular interaction and is used when the induction of epidural local anaesthesia has been unsuccessful. If signs and symptoms are unaltered 1 or 2 weeks after the epidural injection, the next approach to the problem is to introduce 20 mg of triamcinolone around the affected nerve root. The main difficulty, however, is to decide at what level to inject, especially in the case of fifth lumbar root pain, where the protrusion can lie at either the fourth or the fifth level.

In general, *elderly patients* respond better to nerve root infiltrations than to epidural local anaesthesia. Also, lesions of the *second or third lumbar nerve root* are treated preferentially by a local nerve block because epidurals seem to have only a moderate effect.[201]

Discoradicular sciatica is summarized in Box 33.4.

Box 33.4

Summary of discoradicular sciatica

Definition

- Leg pain, radiating segmentally and caused by a posterolateral shift of disc material, compressing against the nerve root

Symptoms

- Onset: primary posterolateral or secondary posterolateral
- Segmental pain
- Segmental paraesthesia
- Weakness/sensory disturbances

Signs

- Partial articular pattern
- Impaired mobility of the nerve root (SLR and L3 stretch)
- Sensory deficit
- Motor deficit
- Reflex changes

Spontaneous development

- Spontaneous reduction
- Erosion
- Shrinkage (absorption)
- Root atrophy

Treatment

- Reposition (manipulation/traction)
- Desensitization (epidural injection/nerve root block)
- Await spontaneous recovery
- Surgery

Surgery

See Chapter 40.

Access the complete reference list online at www.orthopaedicmedicineonline.com

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34

The ligamentous concept

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Introduction

Lesions of the posterior arch (posterior ligaments and facet joints) have long been considered an important source of low back pain. However, the classic work of Mixter and Barr in 1934, which focused attention on the disc, overshadowed the importance of posterior ligamentous lesions. Disc lesions must be considered to be the main source of backache and sciatica but ligamentous problems remain a possible basis for lumbar symptoms. In lesions of the lumbar spine, discs take the strain first but it is obvious that any change in height and mechanical properties of the disc will also influence the posterior ligamentous structures. There is anatomical evidence for free nerve endings in the posterior ligaments and the capsule of the facet joint.[1–3] In addition, a number of investigations have shown that low back pain can be produced by direct stimulation of facet joints and ligaments.[4–6]

Although ligamentous pain is difficult to prove by technical investigations, it is possible to identify it on clinical grounds.[7–10] The ultimate proof of a posterior arch lesion is the improvement of pain and dysfunction after diagnostic infiltration with local anaesthesia.

The structures discussed in this chapter are the supra- and interspinous ligaments, the facet joints, and the intertransverse and the iliolumbar ligaments. The behaviour of the sacroiliac, sacrospinal and sacrotuberous ligaments shows some similarity to that of the lumbar ligaments; however, their disorders and treatment are detailed in Chapter 43.

Mechanism of ligamentous pain

Most of the stabilizing support for the lumbar spine in standing, sitting and flexion–extension is determined by the tension of the ligaments rather than the strength of the paravertebral muscles (Wyke[11]: Ch. 11; Stokes and Frymoyer[12]). Postural strain, therefore, will affect the nociceptors in the capsules of the facet joints and the ligaments of the posterior arch, which happens when prolonged or increased postural pressure falls on normal tissues, or when abnormal (traumatized, inflamed or deformed) ligaments are subjected to normal postural stress. The first – prolonged or increased static loading of normal and healthy ligamentous tissues – is the 'postural' syndrome. The second – symptoms arising from abnormal (degenerated or inflamed) tissues subjected to normal mechanical stress – is the 'dysfunction' syndrome.

Postural syndrome

Pathological changes in the structures responsible for the pain are not present. Only the maintenance of a stress on a normal tissue creates this type of pain – an example of what has been termed 'the bent finger' syndrome; if a finger is bent backwards, sooner or later pain will appear.[9] If sufficient force is applied for long enough, mechanical deformation of the sensitive structures in the involved tissues induces pain. It is this lumbar aching that everyone experiences when a particular posture is maintained for a long period. Pain increases in intensity the longer the time spent in the position, but the moment another position is adopted the pain will gradually disappear (Box 34.1).

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Dysfunction syndrome

Although, in the postural syndrome, the structures are normal and pain is initially produced by subjecting these structures to increased mechanical stresses, it is not unlikely that, sooner or later, damage to the tissues involved will follow. The ligaments can become elongated or inflamed, which results in pain of chemical origin and also in structural changes. In this new situation, pain will be provoked by stress on structures which have become pathological. It is this progression which produces the dysfunction syndrome, defined as lumbar pain resulting from normal mechanical stress on pathological ligaments (Box 34.2).

Box 34.1

Characteristics of postural syndrome[1–3]

- Intermittent pain, induced by the maintenance of positions for a prolonged time and abolished by posture correction or movement
- Time factor is important in differentiation from dysfunction pain; some time must pass before pain becomes apparent: the longer the posture is held, the greater the pain
- Spinal movements are thus full-range and painless: the stress applied during lumbar tests is not maintained long enough to induce pain

Box 34.2

Characteristics of dysfunction syndrome

- Intermittent pain, immediately produced during a particular position and abolished when the stress is released
- Some lumbar movements are painful at the end of range

Postural syndrome

Postural pain appears when normal ligaments are subjected to abnormal mechanical stresses. This happens with inappropriate spinal loading – poor sitting or prolonged bent positions. Alternatively, abnormal mechanical stresses can originate when, as a result of decreasing intervertebral height, too great a load is applied posteriorly to the spine, a situation which can occur during a particular period in the ageing spine. The loss of turgor in the disc and the decrease in intervertebral space will first allow the posterior ligaments to become lax, causing some postural strain. Further diminution in disc height results in structural changes. At the posterior facets, the joint surfaces override and simulate hyperextension (Fig. 34.1). In this position, considerably more weight falls on the facet joints and the posterior capsule becomes overstretched. Two types of fibre orientation have been described in the capsular fibres of the facet joint.[13] Type I capsules have the fibres running diagonally from lateral–caudal to medial– cranial; in type II, the direction is horizontal between the points of insertion on the lower and upper articular processes. Especially in type II, axial loading of the spine results in considerable stretching of the fibres as the upper articular process slides downwards over the lower. Pain may then result.

A similar mechanism probably also accounts for ligamentous pain following disc excision and could also explain back pain after chemonucleolysis, which causes a sudden decrease in disc height.[14]

Postural pain in the ageing spine

Postural pain typically occurs in young patients (30 years of age or less). During this period, and because of the onset of the decrease in disc height, some instability and laxity of the posterior segment may occur. Then an unfavourable position imposes a high and continuous axial load on the spine, so stretching the ligaments and the capsules of the facet joints. In addition, continuing dehydration of the disc causes further

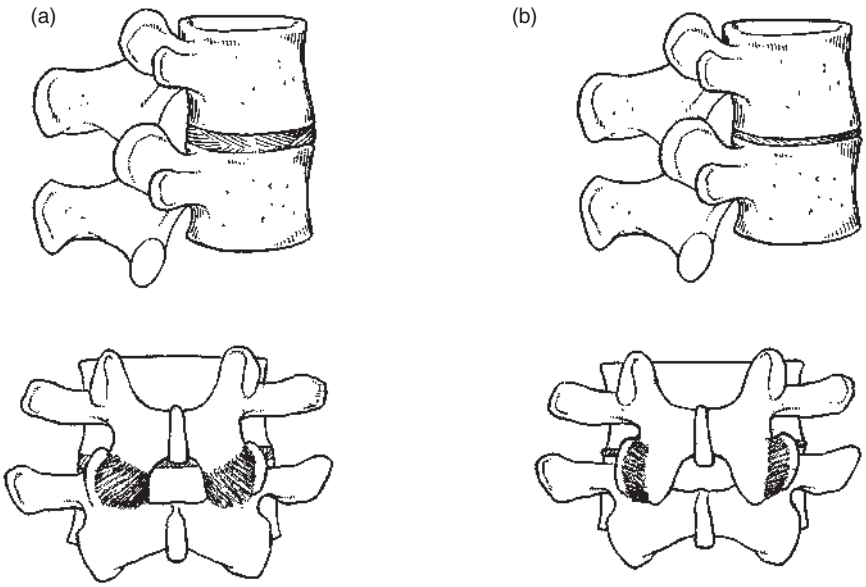

Fig 34.1 • Decrease in intervertebral space may result in stretching of the facet joint capsule. Normal (a) and decreased (b) intervertebral space.

narrowing of the intervertebral space, which in turn adds more tension in the ligaments.

Continuing degeneration produces a stiff spine because of periarticular fibrosis and enlargement of the facets. As a result, postural pain normally disappears as these changes advance after middle age (see Fig. 34.3 below).

History

The localization of the pathological changes in the posterior part of the lumbar spine means that dural symptoms are absent.

The patient is typically young and female, and has diffuse backache, with bilateral radiation over the iliac crests and the sacroiliac joints (Fig. 34.2); pain is never referred below the upper buttocks. When postural pain originates from the sacroiliac ligaments, however, pain reference in the S1 and S2 dermatomes can be encountered (see Ch. 43).

The ache usually starts after being in one position for a considerable length of time – sitting or standing – and the intensity of the pain and the duration of the position are related. Barbor[8] described postural ligamentous pain as the 'theatre, cocktail party' syndrome, because these are characteristic examples of prolonged sitting or standing which produce low back pain. Lying down – for instance, prone – often leads to increased pain, and walking can be painful, especially if the patient is merely strolling slowly. In contrast and surprisingly, to someone who is not familiar with the syndrome, the patient states that during activity and sports he or she is absolutely pain-free. Postural pain (Fig. 34.3) is, as its name implies, a result of *positions not movements*.

Clinical examination

The clinical examination is found to be quite normal: there is a full range of movement without the slightest pain. Dural and nerve root signs are absent.

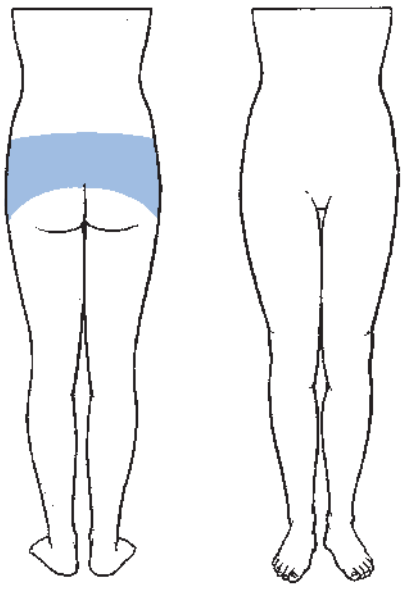

Fig 34.2 • Pain localization in lumbar postural syndrome.

Differential diagnosis

A patient seeking help for a lumbar problem, who presents with a clear and detailed history but has no signs at all on clinical examination, must be considered to have a genuine lesion and not a psychogenic disorder. The differential diagnosis is then between a postural lumbar syndrome, a bruised dura mater, a self-reducing disc protrusion or visceral pain referred to the back. In all these instances the clinical examination is of no help and differentiation relies entirely on the history. In a bruised dura or referred visceral pain, the pain is independent of position. A bruised dura mater can be confirmed by epidural injection. If visceral disease is suspected, appropriate investigations should be made. A self-reducing disc is not always easy to differentiate from postural pain but, because the treatment is the same (sclerosing injections), the question is theoretical.

Treatment

Classically, self-treatment and especially prophylaxis are recommended. The patient should be informed about the pain mechanism and taught how to avoid constant static postures. If prolonged sitting is unavoidable, attention should be directed to a proper posture and good choice of furniture. Standing should involve regular movement of the body weight from one leg to the other. All such information and training can be given

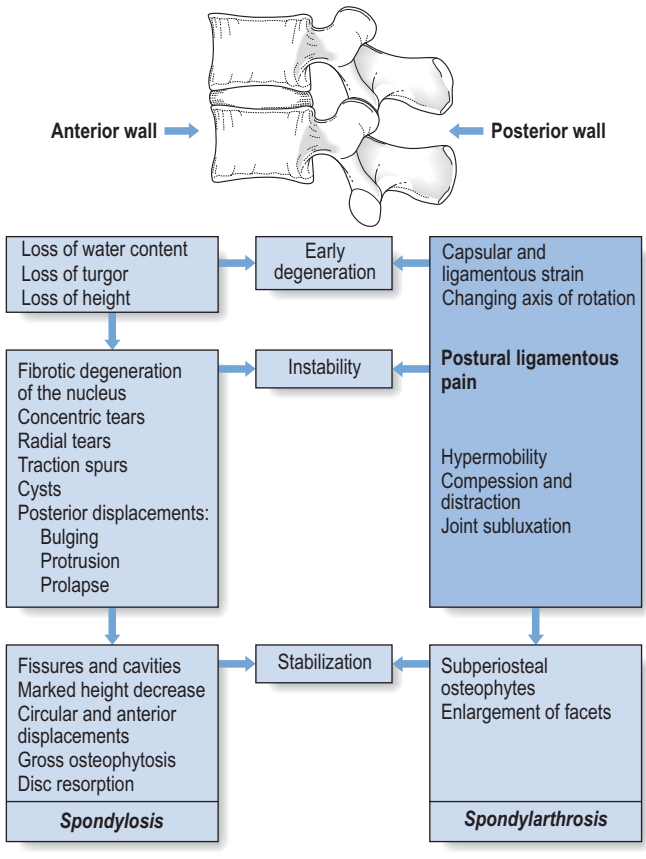

Fig 34.3 • The ligamentous concept in the ageing of the spine.

during a 'back school' programme (see p. 588). Contrary to general belief,[15] it is unnecessary to give patients with postural back pain a programme of strengthening exercises. Strong muscles will not prevent pain provoked by static mechanical stresses.

Though these prophylactic measures should always be taken, it is our personal experience that the results are poor and that only a few patients improve. Most patients with postural backache have their condition for years. Some learn to cope with the disability, until the pain gradually disappears as the result of progressive fibrosis and sclerosis of the posterior elements. Others become chronically disabled and frustrated. For these, one excellent treatment remains – induction of artificial sclerosis in the ligamentous structures.

Chemical sclerosis was used to treat inguinal hernias between the wars and the resulting dense fibrosis of the tissues was noted by Hackett. He adapted the method for the ligamentous periosteal junctions of the posterior lumbar arch as a treatment for chronic low back pain,[4] and others followed.[16,17] The initial solution used was zinc sulphate and carbolic acid but a bewildering variety of other materials ensued, including various soap derivatives and psyllium seed oil. Not surprisingly, considerable side effects were experienced: three instances of paralysis[18,19] and two deaths after injection into the subarachnoid space.[20] Dextrose–phenol–glycerol solution, originally developed for the treatment of varicose veins, had a good safety record[21] and was introduced into spinal use by Ongley in the late 1950s.[10] The mixture provokes an effective inflammatory response, which leads to fibroblast proliferation and new collagen production (O. Troisier, cited by Cyriax[22]: p. 339). At the periosteal junctions of the ligaments, the fibrosis results in an increase in girth of the ligaments, with contraction and subsequent pain relief. As some of the phenol is injected at or around the medial and lateral branches of the posterior ramus, a direct effect on the nerves may also occur[23] and could explain the rapid relief (sometimes from the day or days after the injections) in some patients.

Over 3 consecutive weeks, 3 mL of the solution mixed with 1 mL of lidocaine 2% is infiltrated into the different lumbar ligaments at the ligamentoperiosteal junction, in the following sequence. In the first week, the injection is made at the interspinous and supraspinous ligaments of L4–L5 and L5–S1, and at the iliac insertions of the iliolumbar ligaments. In the second, the posterior aspects of the apophyseal joints of L4 and L5 are infiltrated at both sides. In the third, injection is into the lateral aspects of the laminae of L4 and L5, where the ligamentum flavum blends with the medial aspects of the deeper layer of the lumbar fascia.

In chronic postural backache, the results of these injections are fair. In our experience, about 70% of patients suffering from a postural syndrome become pain-free after 6–8 weeks – the time required to induce sufficient sclerosis. The experience of others is similar.[24] Two randomized studies have shown the effectiveness of the treatment in a group of patients suffering from chronic postural low back pain for an average period of 10 years.[10,25]

Postural syndrome is summarized in Box 34.3.

Box 34.3

Summary of postural syndrome

Symptoms

- Vague lumbar pain produced by *maintenance* of position
- Relationship between duration of posture and degree of pain
- Pain is abolished by change of position
- Pain is not referred below upper buttocks
- Dural symptoms are absent

Signs

- None: full range of movement, no pain

Treatment

- Self-treatment and prophylaxis: back school
- Sclerosing injections

Posterior dysfunction syndrome

As already outlined, posterior dysfunction syndrome is the appearance of pain when posterior structures affected by pathological change are subjected to mechanical stresses.

The posterior structures involved are the facet joints, the supraspinous and interspinous ligaments, and the iliolumbar ligaments. Disordered muscles are a great rarity, easily distinguished because they are contractile. Ligamentous lesions of the sacroiliac joint will be discussed in Chapter 43.

Facet joints

Arthropathy of the facet joints has been regarded as an important source of low back pain for some time. In 1911, Goldthwait maintained that disease of the facet joint was the chief source of backache.[26] By 1933, the term 'facet joint syndrome' had been introduced.[27] In the 1960s and 1970s, the question as to whether back pain could arise from a facet joint problem was vigorously debated in the medical literature.[28–30] Opponents of the idea use the following arguments: first, there is no sensory innervation in the synovial tissue of the articular capsule[31,32]; second, the frequency of grossly disordered joints seen on random radiographs of asymptomatic patients suggests that it is unlikely that minor disorders would cause pain.[33] To this end, Cyriax[22] listed lesions of the facet joints known to cause no problems:

- Gross overriding of the articulating surfaces of the facet joints, occurring as the result of disc resorption in elderly people.
- Gross osteoarthritis of the facets, as seen in more than 50% of people above the age of 45[34–38]; this is an age-dependent and body mass index (BMI)- and gender-independent phenomenon, most frequently observed at two caudal levels, L4–L5 and L5–S1.[39]

- The angulation that occurs after a wedge fracture of the vertebral body.
- Retrolisthesis, when the inferior articular process shifts backwards on the superior articular process of the vertebra below.

During the last few decades, however, the balance of the controversy has somewhat tipped towards the conviction that facet joints can be a primary source of low back pain. First, there is an anatomical basis: in contrast to the insensitive synovia, the capsule of the facet joint is richly innervated by nociceptors which become activated when the capsule is stretched or pinched.[40] In both pain patients and volunteers, chemical or mechanical stimulation of the facet joints and their nerve supply has been shown to elicit back and/or leg pain.[41–43] During spine surgery performed under local anaesthesia, lumbar facet capsule stimulation elicits significant pain in approximately 20% of patients.[44] Finally, in a substantial percentage of patients with chronic lower back pain, there is a considerable degree of pain relief after diagnostic injections of the joints with local anaesthetic.[45–50] However, some studies have demonstrated extravasation into the epidural space following rupture of the joint capsule if large volumes of anaesthetic agent are used. This may result in an unintentional epidural block, explaining the good diagnostic results.[51,52]

Potential causes of the 'facet joint syndrome'

Each facet joint receives dual innervation from medial branches arising from the posterior primary rami at the same level and one level above.[53] However, free nerve endings have been found in the capsule only and not in the articular cartilage or the synovial tissue.[11,33]

Inflammation could produce pain, as happens during a traumatic arthritis, and it has also been suggested that pain is caused by impingement of a synovial fold between the opposing facets.[54,55] Also, inflammatory arthritides, such as rheumatoid arthritis, ankylosing spondylitis and reactive arthritis, may affect the facet joints.[56,57] It is not known if advanced osteoarthrosis as such could be the source of a facet syndrome but it is probable that reduced spinal mobility can sometimes predispose to a sprain of the fibrous capsule.[58] In degenerative spondylolisthesis, a disorder that causes the whole upper vertebra, including the neural arch and processes, to slip relative to the lower vertebra, the capsules of the facet joints come under permanent traction. This may account for the increased incidence of backache seen in patients with degenerative spondylolisthesis.[59] A sprain may also follow excessive strain directly on the posterior arch, which sometimes happens as the result of an unintentional twist, usually in extension. Extension of the lumbar spine may be limited by impaction of the inferior articular process on the lamina below. If that happens on one side only, continued application of the extension movement will force the segment towards rotation around the impacted articular process, which draws the inferior articular process of the contralateral facet joint further backwards.[60] This may result in sprain of the capsule.[61] A similar lesion may result from excessive rotation; rotation is usually limited by the impaction of the facet joint opposite the direction of the movement (normally, the axis of rotation is situated in the posterior third of the disc). If the torque continues, a new axis of rotation will be located in the impacted joint and the contralateral joint will be drawn backwards (Fig. 34.4).[62] Although most post-traumatic inflammation subsides spontaneously in the course of a couple of days or weeks, it is conceivable that a chronic ligamentous sprain and lasting pain might occasionally result.

History

The patient presents after hurting his or her back during a particular movement, frequently hyperextension but alternatively hyperextension accompanied by side flexion.[63] The pain is strictly unilateral and usually localized, sometimes with slight reference to groin, upper buttock or trochanteric area (Fig. 34.5).[64,65] Because a facet joint is a lateral structure, it

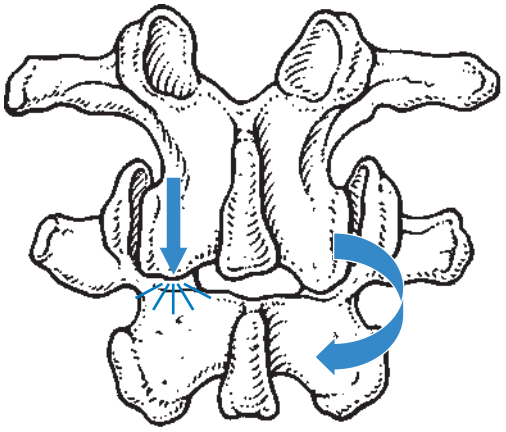

Fig 34.4 • When extension is arrested at one facet only, the contralateral inferior articular process is further forced backwards into radiation, resulting in capsular sprain.

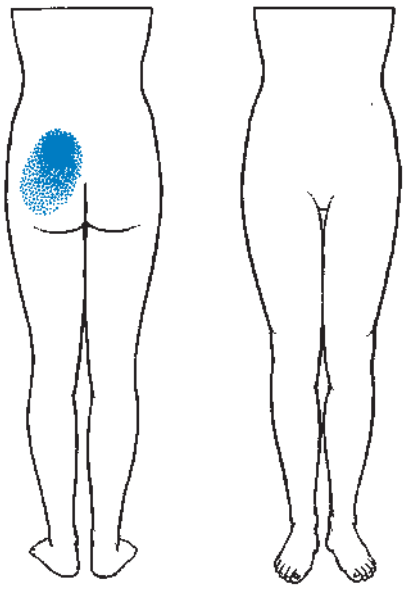

Fig 34.5 • Reference of pain in facet joint lesions.

cannot give rise to central pain.[66] However, if there is a bilateral lesion, pain can be bilateral.

There is absolutely no change in pain localization, whereas in disc lesions the movement of pain is an important diagnostic element: it can be felt on one side and move to the other, or can pass from the paravertebral to the gluteal area, or root pain can follow backache. In facet joint lesions, however, the pain is always felt at the same spot.

Dural symptoms, such as a painful cough or sneeze, are absent.[67] Pain usually appears during extension, but standing and lying prone can also be painful.[58,68]

Sitting usually does not cause problems because the spine is kept in mid-position, so putting little strain on the facet joints.

Clinical examination

The patient stands straight. Usually there is a full range of movement, though extension may be slightly limited. Movements that cause pain at the end of range follow a typical, convergent pattern (Fig. 34.6): for instance, when a left facet joint is at fault, left-sided flexion and extension are painful.[69,70] Exceptionally, a divergent pattern can also indicate a facet joint lesion: for a left-side lesion, flexion and side flexion to the right then provoke pain. Sometimes the pain can be provoked only by a combination of extension with side flexion.[47]

A painful arc is always absent, as are dural signs (straight leg raising and neck flexion), typical of discodural interaction; there are no root signs.[71] Although some believe that pain on palpation over the facet joint is one of the diagnostic features of the lesion,[72,73] palpation of the paravertebral region is non-specific because the tenderness so elicited often results from referral, typical of dural pain.

Because facet joint lesions are rare, the diagnosis is never made without the confirmation of local anaesthetic injection. The technique is set out below but it is important to remember that the results of the block are not always reliable. First, only small amounts of anaesthetic (no more than 1 mL at the dorsal aspect of the capsule) must be used to avoid unintentional epidural injection, which nullifies the test.[51,52] Second, numerous studies have documented that the infiltration is associated with a high false-positive rate, ranging from 25 to 40%.[74–76]

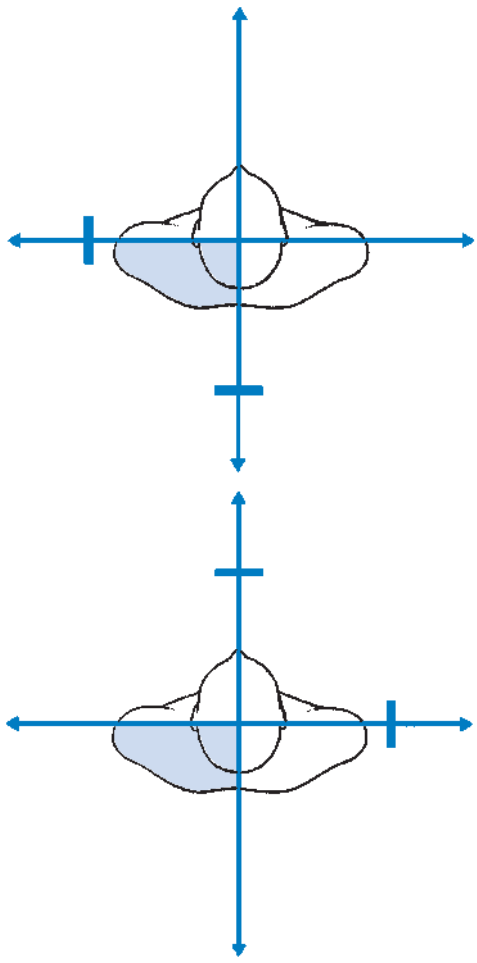

Fig 34.6 • Convergent and divergent patterns

Treatment

Once the diagnosis is made, treatment is infiltration of the posterior aspect of the joint capsule with 10 mg of triamcinolone. In long-standing cases, we prefer to use phenol solution, since the triamcinolone often affords only temporary relief. Others have reported good results after intra-articular injections of a mixture of lidocaine and a corticosteroid suspension,[77,37,78] and pain relief is equally good with intra-articular and periarticular injections, indicating that the pain may be of capsular origin, rather than synovial inflammation.[79] Good outcomes have also been described after injection with phenol aimed at denervating the joint[23] and after radiofrequency 'denervation'.[80,81]

Surgery is occasionally performed to treat facet arthropathy despite a lack of evidence supporting fusion for degenerative spinal disorders.[82,83]

A summary of facet joint lesions is given in Box 34.4.

Iliolumbar ligaments

The iliolumbar ligament arises from the tip and lower parts of the transverse process of the fifth lumbar vertebra. It splits

Box 34.4

Summary of facet joint lesions

Presentation

- Localized and unilateral lumbar pain

Clinical examination

- Full range of movement with convergent or divergent pain
- Dural and radicular signs are absent

Treatment

- Infiltration of the posterior capsule with a corticosteroid suspension or phenol

into an anterior and a posterior band to insert at the anterior and posterior aspect of the top of the iliac crest (see p. 424).[84,85] It plays an important role in the stabilization of the lumbosacral junction. Both anterior and posterior bands restrain flexion of L5 on the sacrum.[86] Side flexion is controlled by the contralateral band and extension by the anterior bands. The iliolumbar ligaments are also thought to be important in maintaining torsional stability of the lumbosacral junction.[87] Because of the specific orientation of the facets of the lumbosacral junction, some rotation is permitted between L5 and S1, in contrast to the lack of rotation at the more superior lumbar joints, and it seems reasonable to speculate that the iliolumbar ligaments fulfil the same function as the articular stabilization of the higher level.[88,89] The stabilizing and anchoring function of the ligaments also modifies the outcome of acute disc displacements. At the L5–S1 level, the strong ligament prevents gross lateral deviations, as is usually seen in L3–L4 or L4–L5 protrusions. However, it is possible for the ligaments to become stretched during long-standing disc displacements. The patient may then suffer from persistent and chronic pain after the primary lesion has been adequately treated by manipulation, traction or surgery.[90,91]

Alternatively, iliolumbar strain can originate from sudden or repeated overstretching, as in forced rotation during flexion.[92] Our personal experience includes instances in soccer players, apparently as the result of repeated lumbosacral torsions; the differential diagnosis of groin pain in such circumstances includes strain of the iliolumbar ligaments.

History

The patient suffers unilateral or bilateral localized pain at the lower lumbar area. The pain may spread along the iliac crest to the groin (Fig. 34.7).[93,94]

The symptoms may develop following injury, or persist after prior lumbago in which dural pain and gross disability have resolved but intermittent pain, appearing during particular movements or after maintaining a particular posture for a certain length of time, remains. As in facet joint lesions, the pain always remains to one or both sides and is never central. There is no history of changing or moving pain, and dural symptoms are completely absent.

Clinical examination

No deviation is visible in the standing position. There is a full range of movement. Side flexion away from the painful side can cause pain at the upper sacroiliac region or in the groin (Fig. 34.8). Sometimes there is also pain at the end of flexion or extension. There are no dural or nerve root signs.

Routine clinical examination is sometimes entirely negative and assessment of the sacroiliac joint and hip fails to reveal any abnormality. An additional provocative test can then be performed: the patient is positioned supine, and the hip is flexed to less than a right angle and adducted until the examiner reaches the ligamentous end-feel. Internal rotation together with simultaneous axial pressure on the femur will then exert a pull on the iliolumbar ligament. This test is *not specific* for iliolumbar lesions but will also be positive if a hip lesion is present. Care should therefore be taken to perform this test only in those cases in which an iliolumbar lesion is already suspected and other sources of sacroiliac or groin pain are excluded.

Treatment

Treatment consists of three weekly infiltrations with sclerosing solution at the insertion of the ligament at the upper aspect of the posterior superior iliac spine (see p. 582).[95] Improvement usually starts some weeks after the infiltration and, if relief is gained, it is lasting.

Supraspinous and interspinous ligaments

In vivo measurements taken from lateral radiographs of lumbar spines showed that the interspinous distance extends up to

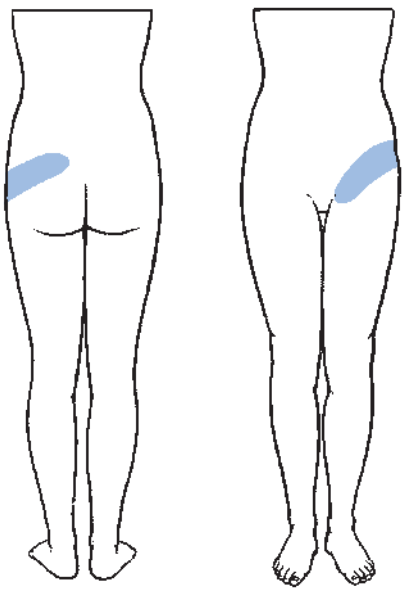

Fig 34.7 • Pain reference in iliolumbar sprain.

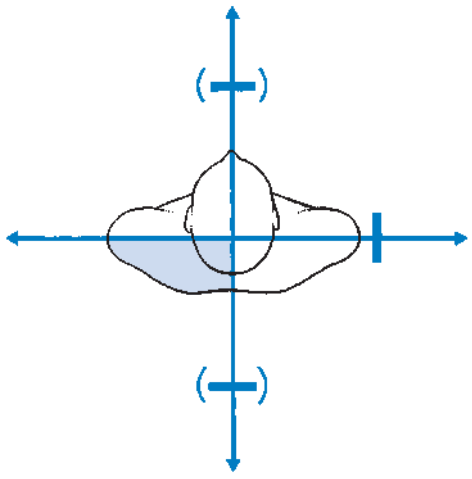

Fig 34.8 • Patterns in iliolumbar lesions.

almost four times during full flexion,[96] which could imply that the interspinous ligament is lax in the upright position and becomes taut only in the extremes of flexion. However, the orientation of the fibres in this ligament, which is obliquely from posterosuperior to anteroinferior, allows it to be active over a large range of motions during flexion.[97,98] Overstretching of the ligaments is therefore very rare, and it is doubtful whether this could be a cause of backache.

Autopsy studies of a large number of subjects have shown that, in subjects over 20 years, over 20% have ruptures of one or more of their supraspinous ligaments, occurring mostly at L4–L5 and L5–S1 segments.[99] These figures strongly suggest that ruptures of the supraspinous ligaments, defined by Newman as 'sprung back',[100] rarely are, of themselves, the cause of low back pain. It is important to realize that, in disc degeneration, an intact supraspinous ligament probably plays an important role in the prevention of posterior displacements. In such circumstances, the posterior fibres of the annulus fibrosus and the posterior longitudinal ligament no longer limit forward flexion because the instantaneous axis of rotation is not at the nucleus but more posteriorly, behind the posterior longitudinal ligament (Fig. 34.9).[101] Flexion of the spine is then controlled by the supraspinous ligament (see p. 422). Although a 'sprung back' is certainly not the primary cause of pain, it can add to segmental instability and recurrent disc displacements. For this reason, sclerosis of the interspinous and supraspinous ligaments is part of the treatment of recurrent disc displacements (see p. 579).

During full extension, the interspinous distances decrease to between 2 and 4 mm and the processes may meet as the bifid ligament buckles laterally on both sides.[102] The ligament is not trapped between the processes and, if pain originates in 'kissing spines', it is due to irritation of the periosteum or an adventitial bursa between the abutting spinous processes.[103,104]

History

There is localized and central pain (Fig. 34.10a), which started after an unintentional hyperextension strain – for instance, bending backwards during gymnastics or diving.[105] The pain does not spread over a large area. The patient states that standing upright and backward bending cause discomfort. There are no dural symptoms.

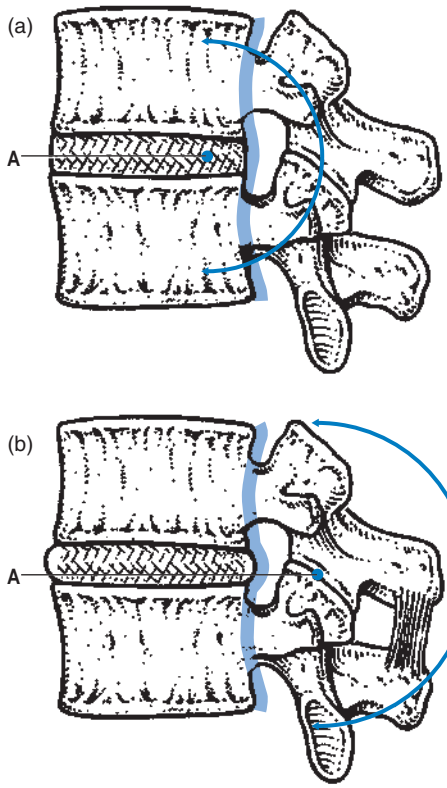

Fig 34.9 • Axes of rotation: (a) normal segment – posterior fibres of annulus and posterior longitudinal ligament limit forward flexion; (b) degenerated disc – flexion of the spine is controlled by the supraspinous ligament. A, instantaneous axis of rotation.

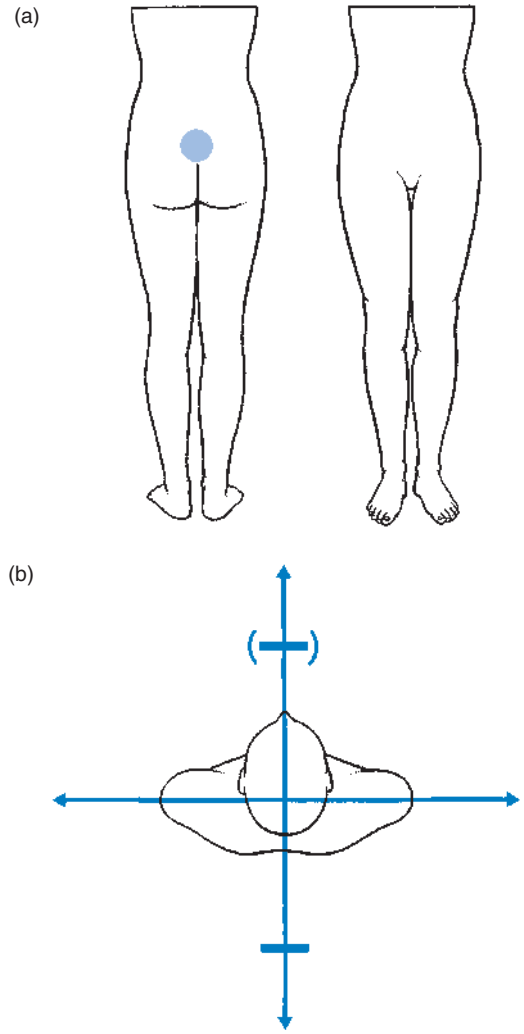

Fig 34.10 • Lesions of the supra- and interspinous ligaments: (a) central pain; (b) pain on backwards bending.

Clinical examination

Extension causes local pain (Fig. 34.10b). Sometimes the end of flexion is also painful. Side flexions are usually free. The lesion is very localized and the patient often indicates the site with one finger. A tender spot, usually at the tips of two consecutive spinal processes, can be palpated. There are no dural or root signs.

Treatment

Treatment consists of local infiltration of triamcinolone, which affords good results. No recurrences need be anticipated if the lesion has resulted from a single hyperextension episode.

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35

The stenotic concept

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Introduction

Compression of lumbosacral nerve roots by osteoarthritic changes in the lumbar spine has been recognized since 1900, when Sachs and Fraenkel[1] published the case of a Russian tailor suffering from intermittent paresis of the legs, which improved markedly after removal of the thickened, enlarged laminae of T11 and T12. Further reports on the consequences of lumbar osteoarthrosis on the function of nerve roots followed.[2,3] Patients suffering from root pain in whom the only operative finding was a marked thickening of the ligamentum flavum were also recognized,[4–6] and excision of the ligaments produced considerable relief of symptoms. A hypertrophied ligamentum flavum as one possible cause for backache and sciatica was suggested both in 1937[7] and 1945.[8] By 1945, congenital narrowing of the bony canal was recognized as responsible for pressure on the cauda equina[8] and, in the same decade, for nerve entrapment at the foraminal level.[9] Overhanging facets were also postulated to be a cause of nerve root irritation.[10] The definitive account of developmental narrowing of the lumbar vertebral canal causing radicular symptoms was given in the 1950s.[11,12] In recent decades there have been many publications on the different types of 'spinal stenosis', and a consensus has been reached on the classification and pathophysiology of the different types of this lesion.[13–17]

Definitions

Definition of stenosis of the vertebral canal or the nerve root canal

- The diameter(s) of the canal is (are) narrowed to a degree that may cause compression of nerve roots in the cauda equina, lateral recess or one of the foramina.
- Narrowing may be developmental or acquired and segmental or generalized.

- *Idiopathic developmental stenosis* of the bony lumbar vertebral canal is a genetic disturbance that reveals its pathological effect only when growth is complete.[18] The narrowing is uniform over almost the whole length of the lumbar bony canal. A mid-sagittal diameter of 10 mm or less is an *absolute stenosis* – the narrowing is capable of producing signs of compression of the neural content in the absence of other compressive agents. A *relative stenosis* has a diameter of 10–12 mm and can only lead to

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compression if other vertebral deformities, such as spondylitis or posterior vertebral osteophytes, compound the narrowing.

- *Acquired spinal stenosis* is the consequence of arthrotic changes, disc herniations, postsurgical or post-traumatic lesions, or bone diseases, including tumours.
- *Degenerative stenosis*, as its name implies, follows degenerative changes in the spine. Such narrowing is segmental and most marked between disc and posterior articular processes. Between the stenotic regions, the diameters of the vertebral canal are usually normal.
- *Iatrogenic stenosis* is the name sometimes given to stenosis resulting from surgical procedures that have caused considerable peridural fibrosis.[19]
- *Lateral recess stenosis* indicates a narrowing of the vertebral canal in its lateral portion (the lateral recess). The condition should be differentiated from narrowing of the entire vertebral canal (*lumbar stenosis*) caused by either a short sagittal or transverse diameter, or by a combination of both. The two cause different clinical syndromes: lumbar stenosis is related to both legs, often involving several dermatomes; the main symptom is neurogenic intermittent claudication. Lateral recess stenosis is characterized by root pain, confined to one dermatome only.

Incidence

The frequency of lumbar spinal stenosis is low. A recent series of 443 lumbar spines yielded only 6 patients in 100 with relative stenosis and none with absolute stenosis.[20] Lateral recess stenosis is more common. In the same series, a trefoil vertebral canal was found in 15% of the examined vertebrae and the same incidence was found in an earlier study.[21]

Spinal stenosis

Pathological changes

In developmental stenosis, the laminae, pedicles and posterior articular processes are increased in size (Fig. 35.1).

Symptoms only arise in the third or fourth decade. The reason for this is that the bony stenosis is mostly relative, and degenerative changes are required to create narrowing that effectively compresses the content of the canal. Bulky bone overgrowth may well be tolerated as long as it lies in a normally developed spine.

Degenerative stenosis results from gross anatomical changes alone (hypertrophy of the articular facets, osteophytosis, thickening of the ligamenta flava) or in combination with degenerative spondylolisthesis.

The latter is a vertebral displacement with an intact neural arch. Although the exact mechanism is not fully understood, the combination of disc degeneration,[22] general joint laxity,[23] increased mechanical stress[24] and structural abnormalities of laminae and facet[25] may precipitate forward displacement of

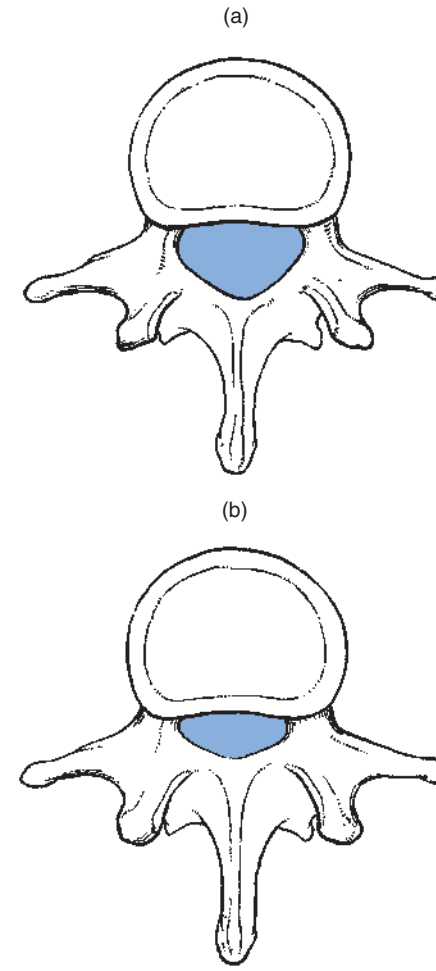

Fig 35.1 • (a) Normal spinal canal; (b) narrowed spinal canal in developmental stenosis.

the whole vertebra. The common denominator in the development of degenerative vertebral slippage is loss of the normal coronal orientation of the facet joints. Several studies showed a significant increase in sagittal facet joint orientation in patients with degenerative spondylolisthesis, compared with a normal population.[26–28] Association with narrowing of the intervertebral joint space and development of osteoarthrosis of the facets then leads to forward vertebral subluxation.[29] Later on, buckling and hypertrophy of the ligamentum flavum may supervene.[30] This results in stenosis between the lamina and the posterior border of the underlying vertebra (Figs 35.2 and 35.3).[31,32] The condition occurs mostly after the age of 50 years, is 4–6 times more common in women than in men, and most frequently affects the L4–L5 level.[33,34] The displacement is usually moderate, less than or equal to one-third of the anteroposterior diameter of the superior border of the adjacent vertebral body.[35] The displacement does not progress continuously but is arrested by further degenerative changes of the intervertebral and facet joints.[36]

The mechanisms that underlie the clinical symptoms in spinal stenosis are complex and poorly understood. There is pressure not only on the dural sac and nerve roots but also on the adjacent blood supply. Therefore, arterial obstruction,

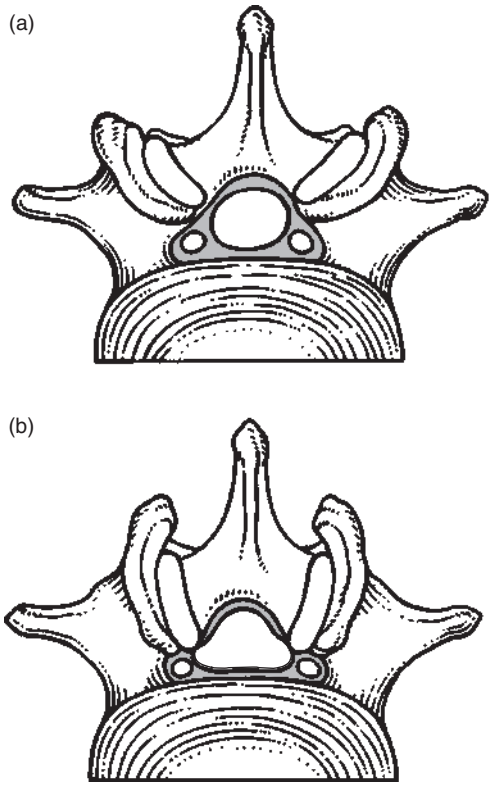

Fig 35.2 • (a) Normal position of upper and lower articular processes; (b) position of lower articular processes in degenerative spondylolisthesis.

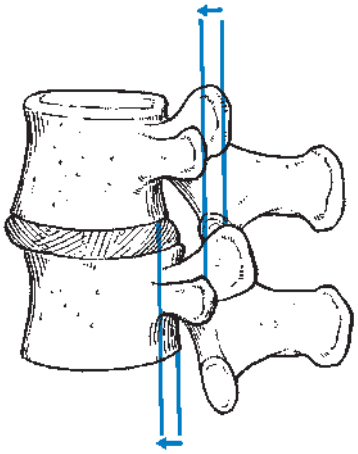

Fig 35.3 • Stenosis between the lamina and the posterior border of the underlying vertebra in degenerative spondylolisthesis.

venous hypertension and pressure–traction on nerve roots, dura and sinuvertebral nerves are all believed to be of importance.[13,37–40]

Symptoms

In patients with degenerative spondylolisthesis, back pain is the most common complaint and is probably caused by overstretching of the facet capsules. Often the pain is episodic or chronic for many years. Patients usually report that their symptoms vary as a function of imposed posture and pain frequently worsens over the course of the day.[41] The most significant symptom of lumbar stenosis is neurogenic intermittent claudication, or 'pseudoclaudication'.[42–44] As in true intermittent claudication, the pain is brought on by walking and relieved by rest. However, pseudoclaudication is believed to be secondary to mechanical compression of the spinal content, whereas intermittent claudication is the result of vascular insufficiency (Table 35.1).[45] The two conditions can be distinguished by their symptoms.

Table 35.1 Differences between neurogenic and vascular claudication

	Neurogenic	Vascular
Localization	Vague, including the back	Mostly in the calf
Paraesthesia	Present	Absent
Walking	Worse	Worse
Standing still	Worse	Better
Bending	Better	No change
Cycling	No change	Worse
Lying prone	Worse	No change

In neurogenic claudication, leg pain is usually bilateral, poorly localized and associated with paraesthesia and numbness. Although the pain is usually provoked during walking, it does not disappear on standing still, which can even aggravate the situation.[46] The pain is relieved only by adopting a stooped posture or by sitting.[47,48] The main characteristic of neurogenic claudication is thus that it is generated by posture rather than by exercise. This postural mechanism explains why a patient may have the same symptoms during recumbency in the prone position and does not usually experience symptoms while riding a bike.[49–51] At its most extreme, patients may report the need to sleep in the fetal position to relieve leg symptoms.

With extreme stenosis, interference with bladder and bowel control can occur but, unlike the acute and often devastating bladder and bowel symptoms of cauda equina syndrome in lumbar disk herniation, spinal stenosis often has an insidious and subtle presentation.[52]

Signs

The patient may have difficulty in standing erect and adopts a 'simian stance' with flattened lumbar lordosis and hips and knees slightly flexed.[53] Extension is limited and may provoke pain in the legs, especially after the extended posture is maintained for a certain length of time. There is symmetrical and painless limitation of both side flexions. As a rule, flexion does not provoke symptoms in the legs but rather relieves the leg pain.[54–56] This can be explained by the fact that extension significantly decreases both mid-sagittal and subarticular sagittal diameter, whereas flexion has the opposite effect.[57,58] Straight leg raising is usually normal, as are reflexes. Because

the lesion involves different levels and the compression of the roots is intermittent, there is often no demonstrable sensory deficit or muscle weakness. If there is some objective weakness, it is normally situated in the muscles supplied by the fifth lumbar and first sacral nerves.[16]

Radiography

Plain radiographs are sufficient to suggest the possibility of a narrow canal. Short pedicles and narrow intervertebral foramina are clearly demonstrable on a suitable lateral projection (Fig. 35.4).[59] Hypertrophy of facets, disc resorption, retrolisthesis and degenerative spondylolisthesis can also be seen easily on the same views.[60,61]

On the plain anteroposterior projections (Fig. 35.5), hypertrophy of the pedicles (A), hypertrophic articular processes (D), small interlaminar spaces (C) and some sagittalization of the articular facets may be seen (B).[62,63]

However, it is as well to remember that these radiographic findings are non-specific and can also be seen in asymptomatic individuals. In patients with clinical features, there is no correlation between symptoms and signs and the severity of the radiographic appearances.[64] They are helpful only in supporting the clinical diagnosis.

In a symptomatic patient, the evaluation of spinal stenosis can best be made by magnetic resonance imaging (MRI), which has largely replaced computed tomography (CT) and CT-myelography.[65] MRI is a non-invasive technique that can define the typical factors contributing to the symptoms of spinal stenosis: significant constriction of the cauda equina associated with a diminished cross-sectional area of the vertebral canal, apparent thickening and buckling of the ligamentum flavum, and hypertrophy of adjacent facet joints.[66]

Treatment

The natural course of spinal stenosis can vary, but in most patients it is a relatively stable disorder, with severe disability and neurological deficits developing over time and not rapidly. A recent survey reported that, in 80% of patients treated conservatively, symptoms did not worsen over 4 years.[67] For patients with moderate or severe symptoms, different conservative and surgical treatment modalities are recommended.

Non-operative, conservative treatment should be used first and includes relative rest and non-steroidal anti-inflammatory drugs.[68–70] Back school instruction is given in activities of daily living and pelvic tilting exercises to reduce lumbar lordosis (see p. 588).[71,72] Epidural injections may be tried but reports of their efficiency are mixed.[73] Some data suggest that epidural injection of corticosteroids relieves leg pain for a limited time but has no effect on functional status or the need for surgery after 1 year.[74]

If the patient does not respond to conservative treatment or if there is progressive deterioration, surgery is required. Decompression of the stenotic area is the usual procedure.[75,76] Some advocate spinal fusion after the decompression, in order to avoid postsurgical instability.[77,78] The surgical outcome is good to excellent in 45–80% of patients operated on.[79–84] Patients with a preoperative duration of symptoms of less than 4 years, with no preoperative back pain and without previous back surgery tend to have a better outcome.[85–88]

Lateral recess stenosis

There are many terms used to describe stenosis at the lateral recess: lateral recess stenosis, subarticular stenosis, superior

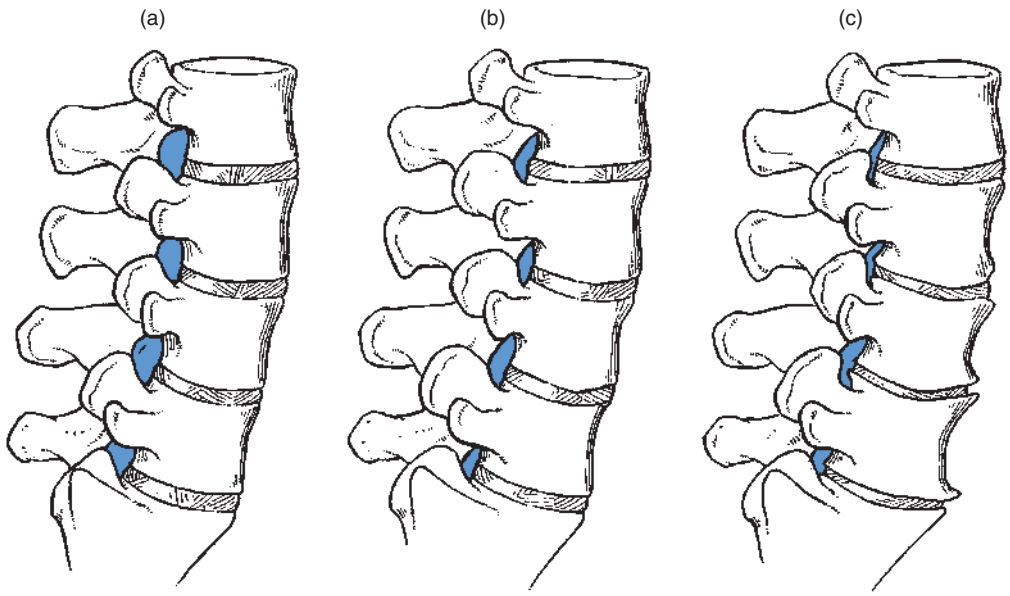

Fig 35.4 • Interpretation of radiographic appearances of spinal stenosis (lateral projection): (a) normal intervertebral foramina; (b) developmental narrowing of the foramina; (c) degenerative narrowing of the intervertebral foramina.

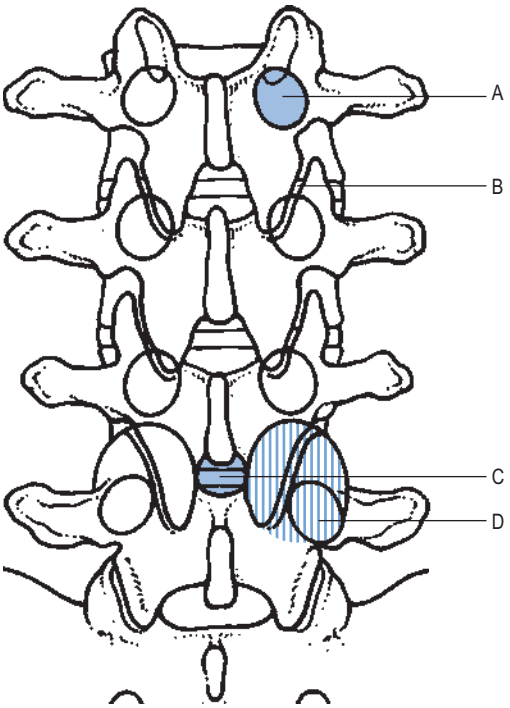

Fig 35.5 • Interpretation of radiographic appearances of spinal stenosis (frontal projection): see text.

facet syndrome, intervertebral foramen stenosis, lateral gutter stenosis and radicular canal stenosis.

The radicular canal is at the lateral aspect of the spinal canal. It begins at the point where the nerve root sheath emerges from the dural sac and ends at the intervertebral foramen. Its posterior border is formed by the ligamentum flavum, superior articular process and lamina. The anterior border is the posterior aspect of vertebral body and disc, both covered by the posterior longitudinal ligament. The dural sac forms the medial wall and the internal aspect of the pedicle the lateral wall.[89] The radicular canal can be classified into three zones (Fig. 35.6): the entrance zone is medial and anterior to the superior articular process; the midzone is located under the pars interarticularis of the lamina and below the pedicle; the exit zone is the area surrounding the intervertebral foramen.[89]

Pathological changes

The radicular canal may be narrowed by different mechanisms but it usually is a combination of advanced intervertebral disc degeneration, associated with marked narrowing of the intervertebral space and increasing arthrotic hypertrophy of the articular processes, which causes the entrapment of a nerve root.

Compression of the nerve root can be the result of different mechanisms: subarticular entrapment, pedicular kinking, or foraminal impingement due to posterior joint subluxation (MacNab[90]: pp. 98–104). Cyriax[91] believed that the main reason for the compression lies in the fact that the posterior longitudinal ligament and the outer layers of the degenerated disc bulge in the upright position, so impinging on the nearby

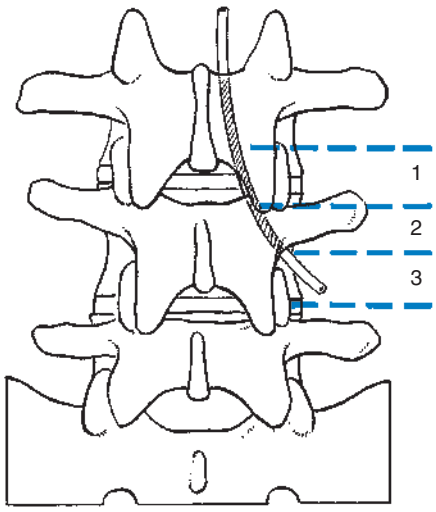

Fig 35.6 • The radicular canal: 1, entrance zone; 2, midzone; 3, exit zone.

nerve root. Recently, stenosis as a result of surgical procedures has become more frequent.

Subarticular entrapment (Fig. 35.7)

This type of stenosis is related to the entrance zone. Emerging from the dural sac, the nerve root passes between the posterior aspect of disc and vertebral body and the anteromedial aspect of the superior articular facet of the same vertebra. Hypertrophic osteoarthrosis of the facet joint, especially involving the superior articular process, may considerably diminish the distance between both borders and thus compress the nerve root,[92] although there may be an additional compression by a thickened and folded posterior longitudinal ligament[91,93] (this is suggested by the observation that the pain is not constant but dependent on posture[94]).

The mechanism is as follows: increasing degeneration and anterior migration of discal tissue cause diminution of the intervertebral space and eventually lead to disc resorption and formation of gross anterior osteophytes. Consequently, the posterior longitudinal ligament, which spanned a joint 1 cm high, is now too long and will therefore bulge, especially in the standing or lordotic position.[95] The thickened and buckling ligament can then exert pressure against the nerve root, especially if the recess is narrowed by the hypertrophic superior facet. Pain in the upright posture which disappears on sitting and bending is better explained in this way than by postulating a static subarticular entrapment. Cyriax called this mechanism the mushroom phenomenon, because of the characteristic radiographic appearances of a long-standing anterior shift of disc material, which results in a marked narrowing of the joint space and gross beak-like osteophytes (Fig. 35.8).

Pedicular kinking

This is a midzone stenosis. The mechanism is as follows: disc degeneration causes considerable narrowing of the intervertebral space and the upper vertebral body descends. The pedicle may then press on the nerve root (Fig. 35.9), especially if

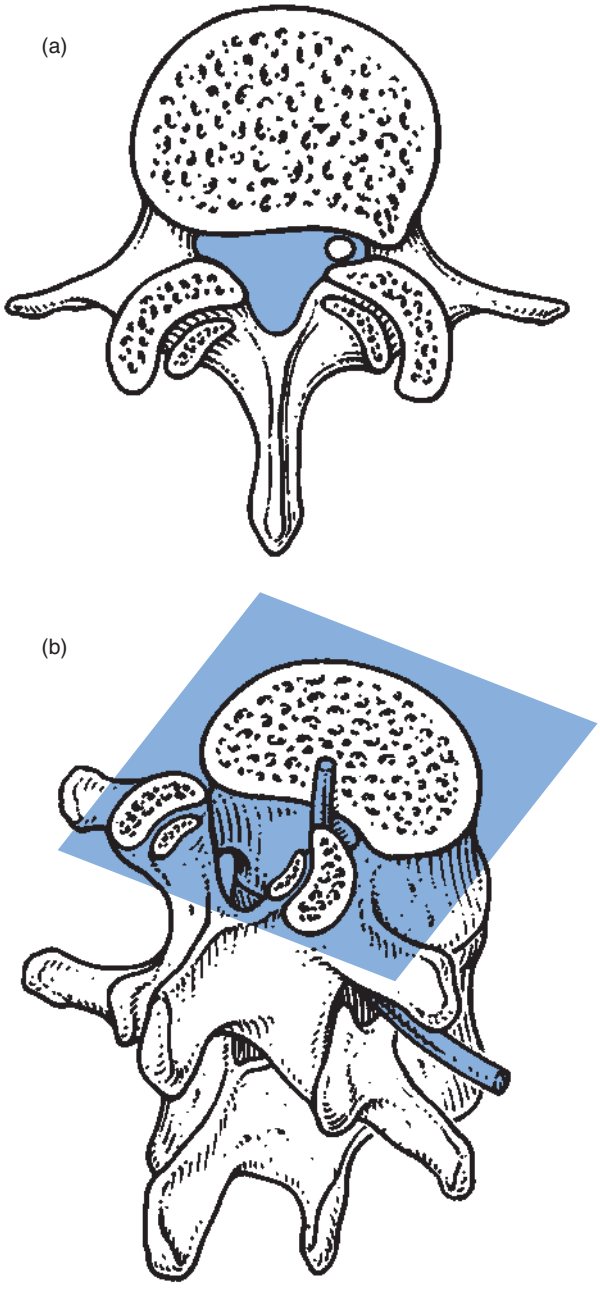

Fig 35.7 • Subarticular entrapment.

the narrowing is asymmetrical, and cause inflammation and oedema, which will give rise to symptoms. An alternative source of the symptoms is that the nerve root is trapped in the gutter formed by the pedicle and the posterolateral aspect of the degenerated and bulging disc.

Foraminal encroachment

This occurs at the exit zone in the intervertebral foramen. *In vitro* anatomical and biomechanical studies of normal motion segments showed that the intervertebral foramen of the lumbar spine narrows significantly during extension and ipsilateral bending, and increases during flexion.[96] With disc degeneration

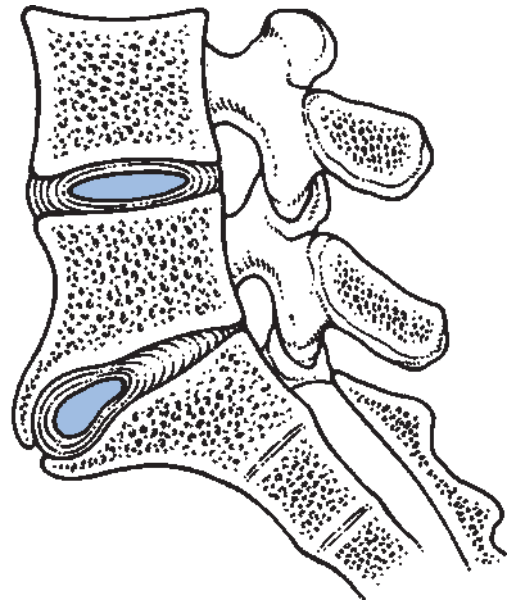

Fig 35.8 • The mushroom phenomenon: the buckling posterior longitudinal ligament causes pressure against the nerve root.

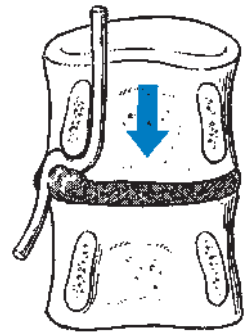

Fig 35.9 • Pedicular kinking.

and subsequent loss of intervertebral height, the facet joints are forced into a permanent extension position: they move telescopically in relation to each other with the inferior facet moving downwards. Because of the inclination of the joints, a downward movement is accompanied by a backward one and a small retrolisthesis is produced. This further narrows the foramen, and the nerve root, which lies in close relationship to the tip of the superior articular process of the underlying vertebra, may become compressed between the tip of the subluxated (and eventually enlarged) superior facet, and the pedicle or body of the vertebra above (Fig. 35.10).

Postsurgical fibrosis and stenosis

Surgical procedures on the spine can cause the formation of scar tissue around the dura and the nerve roots – a so-called laminectomy membrane.[97] After laminectomy the incidence is 9%; it is approximately the same after dorsal fusion.[19] Removal of a herniated disc, which decreases the intervertebral space, may also aggravate a pre-existing stenosis that has not been recognized at operation.[98]

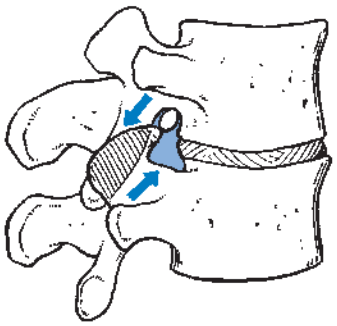

Fig 35.10 • Foraminal encroachment.

Lateral compression of L5

Another, more exceptional cause of radicular entrapment is found at L5. A strong fibrous band – the corporotransverse ligament – runs from the undersurface of the fifth transverse process to the side of the vertebral body (MacNab[90]: p. 53). The L5 nerve root can become trapped between this and the ala of the sacrum if there is a marked downward and forward drop of L5 – for example, in degenerative spondylolisthesis at L5–S1.[99] Compression can also occur in elderly patients with degenerative lumbar scoliosis, between the ala of the sacrum and the transverse process – the 'far out' syndrome.[100,101] It has also been suggested that enlarged lumbosacral ligaments can cause extraforaminal compression of the L5 root.[102]

Mechanism of compression

In order to comprehend the mechanism that causes symptoms of radicular compression in a lateral recess stenosis, it is vital to understand that any impingement on the nerve root is intermittent and related to dynamic changes in the recess during changes in posture and movement. Most compression occurs when the canal is at its most narrow diameter; relief of the symptoms may be expected when the diameter increases. During an axial load on a degenerated spine, the lateral recess decreases in size[103]: the posterior longitudinal ligament folds and buckles in a posterior direction, the superior articular process moves forwards and upwards in relation to the vertebra above, and the pedicle tends to push downwards on the nerve root. Extension and, to a lesser degree, side flexion towards the painful side will further narrow the space, and therefore cause further compression on the root.[104,105] In contrast, compression is reduced during flexion, which stretches the posterior longitudinal ligament and moves the superior articular process away from the foramen.[106]

Symptoms of root compression are caused not only by direct compression of the boundaries but also by inflammation and oedema in and around the nerve root.[107,108] Swelling within an already confined space further increases the degree of compression.[109]

Lateral recess stenosis and natural history of the ageing spine

Lateral root entrapment is characteristic of elderly patients with disc degeneration, marked intervertebral narrowing and spondylarthrosis of the posterior wall (Fig. 35.11).

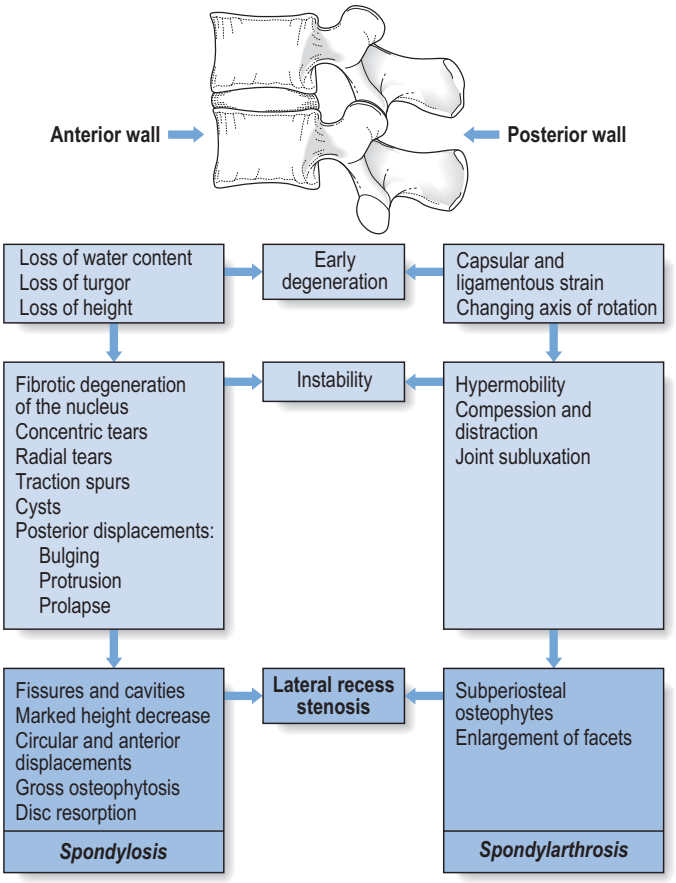

Fig 35.11 • The stenotic concept in the ageing spine.

Symptoms

The patient is typically middle-aged or elderly (onset is seldom before the age of 50 years, and most patients are over 70).[110] The complaint is of unilateral sciatica that comes on during standing and walking. Sometimes, but rather exceptionally, the root pain is bilateral. The pain does not usually start immediately on standing, only after some minutes or on walking. In the latter instance, claudication must be excluded: in vascular claudication the pain rapidly abates on standing still but in lateral recess stenosis it may persist until the patient sits. After some time, numbness and pins and needles in the foot may develop. Also bending forwards *immediately* relieves the pain, whereas extension movements increase it. There is sometimes nocturnal pain, caused by an increased lordosis in the prone position.[111]

Unlike in radicular pain caused by a herniated disc, there is no change in symptoms over time: neither degree nor localization changes, even over years, although the patient may progress in experiencing pain with less precipitating cause and incapacity increases.

There are no dural symptoms.

Signs

There is a striking contrast between the clear and severe symptoms and the almost complete absence of signs.

At presentation there is commonly restriction of the spinal extension, which is caused merely by the underlying spondylarthrosis. That apart, spinal movements are quite normal and flexion particularly is of full range. Standing upright for inspection of the back sometimes brings on the familiar pain in the leg and bending forwards causes it to disappear almost immediately. Occasionally, increasing leg ache and paraesthesia are felt during extension or on side flexion towards the painful side.

Straight leg raising is normal and painless and, as a rule, there is no weakness or sensory deficit. In long-standing cases, there may be slight neurological deficit: a sluggish ankle jerk or a slight weakness of the toe extensors.

However, in most cases of lateral recess stenosis, the clinical examination is completely normal, which means that – as is usual in this and allied problems – the diagnosis is made entirely on the history: an elderly patient with root pain that comes on in the erect position and immediately disappears on lying, sitting or bending forwards.

Radiography

Various authors have noted a lack of correlation between radiographically detected stenosis and the presence and/or absence and intensity of symptoms and signs.[112,113] Difficulties associated with finding such correlations include the presence of a large number of patients with radiographic changes compatible with the morphological diagnosis of lateral recess stenosis and a complete lack of symptoms, variations in canal size throughout the population and lack of an accepted system for quantifying the degree of narrowing.[114,115] One should also never forget that the extent of narrowing is dynamic and likely to change with the posture of the patient (extension significantly decreases the canal area, whereas flexion has the opposite effect). Therefore, a static image of the canal dimensions may not be predictive of a patient's symptoms. However, once a clinical diagnosis has been made, radiography can determine the affected level and is therefore a good guide in planning treatment of the lesion.

The characteristic changes on a plain lateral view are narrowing of the intervertebral space, sometimes gross, beak-like anterior osteophytes (mushroom) and subluxation of the facet joint, with anterior and superior movement of the superior articular process. On an anteroposterior view there is narrowing of the interlaminar space, which is a characteristic feature of lateral recess stenosis; the interlaminar space is encroached if there is an overgrowth of posterior facets or an abnormal configuration of the laminae.[62,116,117]

Myelograms may be difficult to interpret but areas of subarticular narrowing are very clearly demonstrated by CT.[118] In recent decades, however, MRI has become the gold standard as the diagnostic tool in lateral recess stenosis.[119] On the basis of sagittal MRI, four grades of lumbar foraminal stenosis can be distinguished[120]: grade 0 refers to the absence of foraminal stenosis; grade 1 refers to mild foraminal stenosis with some perineural fat obliteration; grade 2 refers to moderate foraminal stenosis showing perineural fat obliteration but no morphological changes; and grade 3 refers to severe foraminal stenosis showing nerve root collapse or morphological change.

Natural history

The natural course of lateral recess stenosis is likely to become chronic. Although symptoms tend to fluctuate considerably over time, they get worse as the years go by. Without treatment, severe disability and neurological deficits develop gradually over a longer period.[121]

Treatment

Occasionally, symptoms can be improved by reducing lumbar lordosis. The patient is thus instructed in the correct posture for activities of daily living (see p. 588).

Failure of response to such a programme is an indication for nerve root infiltration with 20 mg triamcinolone (see p. 588). The chief difficulty is determining where the lesion is. When there is doubt, the injection can be given at the most likely level and, if improvement does not take place after 2 weeks, it is repeated at the other level. Locating the lesion is particularly difficult after laminectomy and several attempts, at different locations, may be necessary.

It may seem surprising that infiltration around the nerve root and the sinuvertebral nerve often causes permanent relief in lateral recess stenosis.[122,123] Also, prolonged relief of radicular pain after injection of a local anaesthetic around the nerve root has been reported.[124–126] These excellent results can be explained by the reduction of chronic irritation in ligaments and perineural tissues.[106] Triamcinolone, injected at the appropriate point, suppresses inflammation and thus reduces perineural swelling. As a result, relative narrowing of the canal is alleviated and the vicious circle of anatomical stenosis and inflammation is broken. The good and permanent results sometimes seen after infiltration of a local anaesthetic alone are explained by a chemical effect on the C fibres of the dorsal root.[126]

When nerve root infiltration does not afford permanent or semipermanent cure, surgery is indicated; it consists of decompression of the nerve root. Sometimes excision of an osteophyte by fenestration may be sufficient,[127] but more usually laminectomy with partial excision of the enlarged facets is necessary.[128] In recent years, interspinous implants ('spacers') have been used in the treatment of lumbar spinal stenosis. The rationale behind this minimally invasive intervention is that of de-lordosing the segment and thus widening the spinal canal in the upright position.[129]

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Clinical examination of the lumbar spine

36

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History

Introduction

Assessment of backache and/or sciatica is never easy. Despite the increasing accessibility of highly sophisticated technical aids, diagnosis of lumbar problems still relies on the principles of history and clinical examination.

Taking the history is by far the most significant tool in the diagnostic procedure. Whereas examination techniques – both clinical and paraclinical – give current information only, the history also puts the evolution of the disease in the picture. History not only is the record of past and present suffering but also constitutes the basis of future treatment, prevention and prognosis. Furthermore, it also gives information about the degree of disablement the problem has produced and will produce.

For these reasons, a careful history, detailed and in chronological sequence, cannot be taken in a hurry. The examiner must make time to listen and have the patience to unravel complicated stories. Most patients have difficulty organizing their story or remembering every detail. Sometimes they are even unable to give precise responses to simple questions, and mix up past and present symptoms, pain and disablement, and physical, emotional and social disturbances. For a satisfactory diagnosis, however, it is essential to obtain a description of the past and present pain in meticulous detail. Therefore the examiner should develop a specific technique of questioning which is chronological and precise. The answers (both positive and negative) form a pattern that is related to knowledge of applied anatomy, biomechanics and pathogenesis. Taking a history thus translates the patients' subjective complaints into an anatomical and biomechanical context which correlates with one of the well-known syndromes.

The importance of the chronological order in which the symptoms present is illustrated by the following (simple) case

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report: a patient states that he developed a sudden backache, which was followed a few days later by severe pain in the left calf. Two days later he noticed numbness of the outer two toes and difficulty standing on tiptoe. By that time the backache had ceased. This is the story of an S1 root compression with neurological deficit, almost certainly caused by a large disc protrusion at the level of L5–S1. This chronological description pinpoints not only a diagnosis but also the therapy: manipulative treatment will be of no help and the patient should either be treated by epidural local anaesthesia or surgery, or await spontaneous recovery.

In practice, the patient's symptoms are not always as obvious as in this idealized case but it does give an idea of the value of a good history. After the history has been taken, the examiner should have a fair idea of the diagnosis or at least be able to distinguish activity-related backache from non-activity-related, referred or functional pain.

Localization of the symptoms

Symptoms are grouped under the headings lumbago, backache and sciatica. These terms are used as follows:

- *Lumbago*: a sudden attack of severe low back pain, causing some degree of fixation and twinges on attempted movement.
- *Backache*: discomfort in the lower back.
- *Sciatica*: pain that radiates strictly from the buttock to the posterior thigh and calf. It is restricted to a specific dermatome (L4, L5, S1 or S2) and may be accompanied by paraesthesia and motor and/or sensory deficit. In practice, however, the term is used inaccurately if pain and paraesthesia are felt in the anterior part of the thigh and/or lower leg (L2–L3).

Pathogenesis

In lumbar spine problems, the mechanism of causation is usually reflected in the behaviour of the pain. Localization of the symptoms, their evolution and the relation to activity and posture differ according to the tissue involved. Pain in the lumbar and pelvic–gluteal area is usually of local origin but may also be *referred* from intra-abdominal or pelvic lesions. Sometimes lumbar pain is devoid of any organic basis and is then labelled as *non-organic* or '*functional*'. Local organic disorders may or may not be related to activity. The former are called *activity-related spinal disorders*, the latter *non-activity-related spinal disorders* (Box 36.1).

Activity-related spinal disorders (Ch. 38)

These are caused by a mechanical dysfunction: discodural and discoradicular lesions, capsular or ligamentous lesions, and spinal stenosis.

Discodural interactions may cause all three major syndromes: lumbago, backache and sciatica. Both articular and dural symptoms are present and have a strong tendency to evolve over time: because the discal displacement seldom remains stable, localization and intensity of the symptoms vary with localization and intensity of the dural impingement.

Lesions of the facet joints are characterized by localized pain. There is absolutely no change in localization. Dural and root symptoms are also absent.

Ligamentous pain typically occurs in relatively young people. It is created by prolonged or increased postural stress and abolished by correction of posture. In long-standing cases, movements also become painful at full range. In disorders of the lumbar ligaments, pain is always intermittent and vague and never referred below the upper buttocks. Lesions of the iliolumbar or sacroiliac ligaments, however, may give rise to slight reference of pain in the groin and the back of the upper thigh, respectively. Again, neither dural nor root symptoms are present.

In central spinal stenosis, chronic and vague lumbo-sciatica is brought on by walking or standing and relieved by stooping or sitting. Pain is often associated with feelings of numbness and weakness in both legs. These patients are never under 30 years old and more often are over 60.

In stenosis of the lateral spinal recess, a middle-aged or elderly patient complains of unilateral sciatica coming on during standing and walking. Sitting or bending forwards alleviates the pain immediately. As in central spinal stenosis, dural symptoms and signs are absent, as are root signs.

Non-activity-related spinal disorders (Ch. 39)

These include:

- *Inflammatory diseases*, both septic and rheumatological.
- *Osseous disorders*, such as osteoporosis, fractures or tumours.
- *Acquired defects* of the vertebral arch.
- *Intraspinal lesions*, such as neuroma, metastases and cysts.

In ankylosing spondylitis, pain may vary in an unexpected way. One day the patient awakes without any discomfort and is able to do any kind of heavy work. The next week, the patient may

Box 36.1

Origin of low back pain

Organic disorders

Activity-related spinal disorders

- Discodural and discoradicular disorders
- Capsuloligamentous disorders
- Stenotic disorders

Non-activity-related spinal disorders

- Inflammation (septic and rheumatic)
- Osseous disorders
- Acquired defects
- Tumours
- Metabolic disorders

Pain referred to the back

- Visceral disorders

Non-organic disorders

wake up early with a painful back and disablement persists for the rest of the day. This differs markedly from the patient with discodural backaches, in whom pain starts on getting up or when an attempt is made to put on socks or tights. The back thus aches during certain movements or positions, whereas others ease the pain.

In rheumatoid conditions, the pain is typically experienced most severely in the morning and improves throughout the day.

In malignant disease, pain is unremitting and worse at night. Lumbar pain increases steadily even after root pain has set in; its distribution is not confined to a single dermatome.

Pain referred to the back

Pain in the back that is completely unrelated to movement or posture and displays hardly any temporal pattern suggests a referred source from intra-abdominal or pelvic lesions, such as those of the aorta, and the genitourinary and gastrointestinal tracts. In the initial stages of these diseases, the history may signal the disease via warning signs. Some of these are briefly discussed here.

Warning

- Pain in the upper lumbar region suggests the possibility of aortic aneurysm, neoplasm, caries or ankylosing spondylitis, or may result from visceral disease.
- Steadily increasing lumbar pain, especially in elderly patients, also occurs in malignant disease.
- Gradually expanding and increasing pain is associated with a lesion that is increasing in size: for example, neoplasm or neuroma. A good example of this is a patient with backache, followed by sciatica in which the pain in the back worsens instead of diminishes.
- Continuous pain, not altered by movement and posture, is ominous; it is important to remember, however, that 'endless' pain may also be present in psychogenic disorders.

Problem solving

While taking the history, the examiner endeavours to find an answer to the following questions:

- Is this an organic or non-organic lesion?
- Do the symptoms point to activity-related disorders?
- If so, is it a disc lesion?
- What sort of disc lesion is present?
- What other type of lesion is more in accordance with the symptoms?
- What type of person is the patient? Is it obvious that the degree of pain and effect on daily activities tally with appearance and behaviour?

Because low back pain is most often caused by a soft tissue lesion and so is frequently attributed to disc disorders, the history serves in the first place to verify whether this is the case. 'All discs are alike, all other lesions are different' is Cyriax's statement, which has been proved true in orthopaedic practice. Therefore, in disc displacements of all types, confirmation of the facts detailed in Chapter 33 is expected.

The history also provides an opportunity to interpret the credibility of the patient's story, again by looking for 'unlikelihoods' – facts that do not correlate with one of the well-known syndromes. If, during the history, the slightest doubt arises, the clinician should be on the alert. More 'unlikelihoods' should then be sought in the patient's story or during the following clinical examination. In contrast, when patients state exactly what is expected and normal for a comparable case, there is certainly no reason to doubt their reliability; nor will a search for possible psychoneurotic components be necessary. Patients devoid of a physical cause will rarely escape detection if the history is properly taken. In these patients, none of the well-known physical patterns emerges – the rules and facts of referred pain do not fit. They do not so much describe their symptoms as the degree of suffering. They fail to supply relevant answers and, if the examiner insists, questions are often resented.

During the history the interviewer should obtain specific data on the following:

- Age and daily activities
- Symptoms:
  - Pain
  - Paraesthesia
  - Influence of posture, movements or coughing/sneezing
  - Bowel or bladder problems/S4 root
- Patient's reaction to these symptoms.

Age and activities of daily living

Disc lesions causing backache and sciatica are most common between the ages of 20 and 50 years. Over 60, the frequency decreases. Under the age of 20, discodural interactions are rare, although not impossible.

Sciatica caused by a posterolateral disc protrusion can be expected from adolescence to old age.

In elderly patients, lateral recess stenosis is to be more frequently expected as the cause of root pain (Table 36.1). Also, degenerative spinal stenosis is a disease that occurs predominantly in the elderly.

Spondylolisthesis can provoke posterior ligamentous pain in the young. Postural ligamentous pain is also more frequent in young patients with a standing job.

Ankylosing spondylitis typically provokes alternating sciatica between 15 and 35 years of age. It is 4–9 times more frequent in men.[1]

Table 36.1 Age-dependent disorders

Age (years)	Disorder
15	Spondylolisthesis
15–35	Ankylosing spondylitis
20–50	Disc lesions
Elderly	Spinal and lateral recess stenosis

The activities undertaken as part of the patient's profession, hobbies and sports will give additional information that is extremely important in judging the actual functional incapacity and in designing a treatment strategy. Most patients do not suffer from pain, rather from the disability the pain provokes. It is obvious that discodural backache will produce more disability in a truck driver who has to sit for the whole day than in a patient who does light and varying work. For some, normal activities are unrestricted but their favourite sport is impossible, and this is a major concern.

Profession and activities are also important in relation to treatment, recurrences and prophylaxis. If a bricklayer gets lumbago every second year, his back can be judged to be reasonably stable and manipulation will help him sufficiently each time there is a new attack. If, by contrast, an office worker has attacks of lumbago five times or more a year, these repeated events indicate that the back is very unstable. Although manipulation may solve the problem for a short time, it is obvious that stronger prophylactic measures will be necessary; successful manipulation should be followed by sclerosing injections, back school and/or a good lumbar support.

Routine of history taking

In disorders of the lower back, symptoms can diversify. The clinician must try to obtain a clear impression not only of present discomfort but also of former events (see Box 36.4). Pain is the most common and important symptom and is usually what forces the patient to seek medical help. Other symptoms are not always mentioned spontaneously but should be asked about: the presence of paraesthesia, numbness, a cold foot or incontinence.

Symptoms are usually presented by the patient in a very disorganized way. The interviewer then tries to create 'order in the dis-order'. The best approach is chronological, the patient being asked about events leading up to the onset of the symptoms and then recounting chronologically what has happened since.

Pain

All the different aspects of pain should be investigated: localization, onset, evolution and duration of the perceived 'current' pain; influence of movement and posture; and the presence of dural symptoms. It is also very useful to obtain information on the same factors in previous attacks (Box 36.2).

In disorders of the lower back, pain may be experienced as backache, as gluteal pain with or without reference to one or both legs, or as typical root pain.

Current pain

Side and level

Patients are first asked if they feel any pain at the present time and to point to its location. The method chosen may give information on emotional status. A stable patient generally places the palm of the hand at the site of maximal pain and moves it across the body to demonstrate the route of radiation. A psychologically unstable patient never touches the painful area but only points it out vaguely with the thumb.[2]

Back pain may be felt *centrally, unilaterally or bilaterally*. Central pain can never be referred from a unilateral structure – for example, a facet joint or a sacroiliac joint. Bilateral pain also hardly ever has a central origin. Unilateral pain in one buttock is typical of a discodural problem. Sometimes the sacroiliac joint is responsible but a strained muscle is a rarity.

Bilateral, vague leg pain is usually dural pain. Segmental pain in both legs can sometimes be caused by two protrusions. However, bilateral root pain is more often the result of spondylolisthesis, spinal stenosis, lateral recess stenosis or metastases. Bilateral osteoarthritis of the hip joints and intermittent claudication due to thrombosis in the iliac arteries may also create pain in both legs.

The level of the pain is also important. In backache with dural reference, pain is usually situated in the lower lumbosacral region, and may radiate downwards to one or both buttocks.

If the patient points to the upper lumbar area, the investigator should immediately be on the alert. Malignant diseases in the lower back have a great preference for this area (see p. 536).

In sacral, coccygeal or perineal pain and numbness, compression of the S4 root is a real risk and constitutes an absolute contraindication to manipulation.

Box 36.2

Pain: important reminders

Current pain

Localization

- Side: unilateral, bilateral or central?
- Level: upper lumbar, lower lumbar, gluteal, leg?

Onset

- When did the pain start? Days, weeks, months or years ago?
- How did the pain start? Suddenly, gradually?

Evolution

- Relationship between back pain and leg pain?

Relationship between pain and activity

- What posture or movement makes the pain worse?
- Is coughing painful?
- What eases the pain?
- Is there any nocturnal pain?

Previous pain episodes

- Earliest manifestation?
- Localization?
- Frequency of attacks?
- Pain-free intervals?
- Previous treatment?

In sciatica, unilateral pain in the relevant dermatome results from pressure on the dural sleeve of one of the lower lumbar nerve roots. It is important, though not always easy, to distinguish radicular pain from dural pain. The latter is extrasegmentally referred and therefore experienced over a larger area, not restricted to one dermatome. It may even spread upwards to the chest or down both thighs, sometimes reaching the ankles. By contrast, the segmental pain of root compression is easily recognized when a patient presents with a severe ache in the leg clearly situated within the borders of the respective dermatome. The difference between radicular and dural pain is extremely important in both diagnosis and treatment. Every effort should therefore be made to obtain a precise description of the localization and characteristics of the pain. To a patient, a leg is a leg, and most are not precise about whether the pain is in the front or back of the thigh, whether it spreads beyond the knee and whether it is localized or generalized.

Pain in one lower buttock only is rarely dural; more commonly, it is a segmental reference from S2.

Onset of pain

Low back disorders may be acute, chronic or recurrent. The patient should identify the first time that the symptoms occurred. '*When did your back problem start?*' is thus an important question.

A long history of, say, 20 years of 'suffering' from backache requires further questioning on whether the problem is continuous or intermittent. It may be that there was just one attack of acute lumbago 20 years ago with a second attack 2 days ago; or a constant and daily ache may have been present over the whole period of 20 years; or the problem may be recurrent, incapacitating backache six times a year. Although in all three instances the history extends over a period of 20 years, it is obvious that the diagnostic and therapeutic approach will differ, as will the prophylactic measures to be taken.

Information about the duration of symptoms is also extremely important in cases of sciatica. There is no limit to the duration of radicular pain resulting from lateral recess stenosis but in discoradicular interactions a course terminating in spontaneous recovery is the rule. Once the protrusion has shifted to one side, symptoms tend to abate; the protrusion has settled itself outside the intervertebral joint and there it lacks nutrition and shrivels away. As a rule, the patient recovers within 12 months of the onset of radicular pain. However, this only applies in patients under 60 years of age.

The next question concerns the speed of onset: 'How did it start: was the onset sudden or gradual?'

Backache coming on over some hours, or even the morning after doing heavy work involving much stooping and lifting, suggests a soft disc lesion slowly increasing in size, i.e. a soft nuclear displacement. Cyriax used to say: 'Pulp oozes, cartilage subluxates in an instant.' In this type of discal displacement, traction is usually the treatment of choice, except in very acute cases where it is strongly contraindicated. An epidural injection is then the alternative.

Acute lumbago starting suddenly, at the moment the patient bent forwards or lifted a weight, is typical of a hard, annular disc displacement. In a case that is not too acute, manipulation is almost always successful in one or two sessions.

Course of pain

In most discoradicular interactions, pain typically starts in the back and sooner or later shifts into one leg. 'Shifting pain' of this nature strongly indicates a disc lesion. If backache gradually increases and after some time extends into one leg and finally involves the back and both legs, a progressive lesion such as a tumour is very likely and the examiner should be on the alert.

Pain starting in the leg, slowly getting worse over a period of months and finally spreading upwards to the posterior aspect of the thigh, is very suggestive of a primary posterolateral protrusion compressing the S1 root. The lesion occurs in young adults and is always nuclear.

Chronic sciatic pain in the elderly, extending over months to years, is typical of radicular pain from a narrowed lateral recess.

'Alternating pain' in the legs suggests bilateral sacroiliac arthritis, which is usually a manifestation of early ankylosing spondylitis. Less often, it indicates a disc lesion in a very unstable lumbar intervertebral joint.

Sequence of backache and root pain: which came first, backache or root pain?

Usually, backache is followed by root pain and ceases when the root pain begins. From that point, the mechanism of gradual spontaneous recovery starts. However, there are exceptions and there is no point in awaiting spontaneous recovery in patients over 60.

Root pain without previous backache, which is caused by a primary posterolateral protrusion, has been shown to be irreducible by manipulation. All displacements of this nature appear to be nuclear, moving posterolaterally from the onset.

What factors influence the symptoms?

In activity-related spinal disorders, it is obvious that there is a relationship between the symptoms and posture or exertion.

Posture and exertion

In a discodural interaction, the ache is increased by stooping, lifting, sitting or coming upright after sitting and is relieved during walking and in recumbency. However, there are some characteristic histories that are slightly different from the usual findings for disc lesions. For example, a patient between 20 and 40 years of age may awake without any pain and remain asymptomatic over the next few hours, even on exertion. During the day backache comes on, slowly getting worse. On going to bed, the pain ceases after an hour or so. This is the typical history of a 'self-reducing' disc lesion.

A patient may complain of pain in the lower back which comes on with prolonged standing. The ache gets worse and is finally followed by bilateral root pain, eventually with paraesthesia in both feet. Sitting or lying down results in cessation within a minute. In young patients this pattern suggests a spondylolisthesis. If the patient is elderly, stenosis of the spinal canal should be suspected.

In 'ligamentous postural' syndromes, pain is particularly increased by maintenance of a particular posture, whereas altering the position relieves the pain. Moreover, the longer the position is maintained, the more intense the pain becomes.

Barbor[3] described the discomfort of ligamentous pain as 'the theatre, cocktail party syndrome': it is impossible to sit at the theatre or stand at the cocktail party without low backache occurring. In contrast, the symptoms are relieved by activity. This syndrome is typically found in the young.

Coughing and sneezing

Another factor that may influence symptoms is raised intra-abdominal pressure during coughing and sneezing (Box 36.3). Pain in these circumstances may be a dural sign produced by sudden increased intradural pressure, which in turn causes sudden expansion of the dura pressed against the protrusion. Although it is very often related to a disc protrusion, it is clear that any *space-occupying lesion* in the lumbar spinal canal compressing the dura mater (e.g. a neuroma or malignant tumour) may evoke the same response. Often the patient will not mention it spontaneously, so the investigator must enquire about coughing and sneezing.

In disc lesions, coughing and sneezing normally increase the lumbar or gluteal pain. However, when they also increase the pain in the leg, manipulative reduction will almost certainly fail. Epidural injections should then be tried. A neuroma may evoke the same sign but the pain is usually felt more in the leg than in the back.

In active sacroiliitis, pain on coughing is felt in the buttock and sometimes radiates into the thigh. It results from the painful distraction of the joint caused by the momentarily increased intra-abdominal pressure.

'Early morning' pain

This wakes the patient and eases on getting up, after which it is possible to do fairly heavy work; the pattern is suggestive of ankylosing spondylitis. The pain is felt centrally in the whole lumbar region and varies from day to day.

Sometimes early morning pain is caused by a discodural interaction. The increased hydration of the disc during recumbency possibly exacerbates an existing small posterior bulge, which then slowly compresses the dura mater, so waking the patient before dawn. This type of disc lesion is best treated by epidural local anaesthesia.

It should be emphasized that these histories are quite different from the usual story for a patient with a small disc lesion who has a painful stiff back on getting up in the morning. Turning in bed is also mentioned as causing a twinge and is quite different from increasing pain in a recumbent position.

Root pain worsening during the night

This results from a large protrusion with a high degree of inflammation. For this reason, manipulation and traction will be of no help and the patient is best treated by an epidural injection.

Continuous pain

If lumbar movements or posture do not influence the pain, there is certainly no mechanical lesion and the condition may be the result of some other pathological disorder such as intra-abdominal or spinal malignancy or infection, a bruised dura or bruised nerve root sleeve.

Box 36.3

Low back pain disorders in which coughing produces symptoms

Intraspinal lesions

Intradural

- Neuroma

Extradural

- Disc protrusion
- Disc infections
- Metastasis
- Epidural abscess
- Haematoma

Extraspinal lesions

Sacroiliac joint

- Arthritis
- Strain

Duration of pain

Lumbago usually recovers spontaneously within a week because of the strong counterpressure exerted by the posterior longitudinal ligament, which gradually reduces the large postero-central displacement.

In backache, there is no predictable time limit: the protrusion is small and remains more or less in contact with the rest of the intervertebral content of disc, end-plates and ligaments, thus receiving sufficient nutrient supply to maintain it. Because of a decrease in intervertebral height, the counterpressure exerted by the posterior longitudinal ligament becomes less effective. As a result, the discal tissue may remain displaced for years, with or without remission. Spontaneous recovery probably never occurs, whereas reposition by manipulation or traction is simple and is indicated, regardless of how long the patient has had the problem.

In sciatica, the protrusion has slipped posterolaterally and becomes extra-articular. Consequently, the bulge is cut off from its nutrient supply. Slow but continuous shrinking sets in from the moment the backache ceases, and results in spontaneous remission of the sciatic pain within 8–12 months. Therefore treatments such as manipulation and traction are worth trying but only during the first 6 months. Once this time limit has passed, they are not relevant because the process of spontaneous recovery becomes more and more likely to succeed. Later recurrences at the same level are no longer likely. In major or long-standing sciatica, epidural local anaesthesia may relieve the pain during the period of spontaneous recovery. If this fails and pain remains unbearable, surgery is indicated.

Root pain that does not get better within a year or even gets worse after, say, 8 months is suggestive of conditions other than disc lesions, such as neuroma or lateral recess stenosis.

Previous attacks

Previous attacks should be related to age, localization, origin, evolution, influence of movement and posture. Also important are the frequency of attacks, as well as the type of treatment and its result.

Age

If a patient states that episodes of backache or lumbago have occurred since childhood, spondylolisthesis with a secondary disc lesion should be suspected.

Localization of symptoms

In disc lesions, the localization of symptoms is determined by the site of the pressure on the dura mater or nerve root. In a new episode, the pain may have similar localization. However, as a disc may easily shift and compress sensitive structures at other places, repeated attacks of backache caused by the same disc may provoke pain on different sides. Reports of former attacks, not located on the same side, point to a disc protrusion as the cause of the problem. A shifting pain means a shifting lesion, and only the disc is free to move from one side to another.

Pain changing from one buttock to the other is also seen in early ankylosing spondylitis with involvement of the sacroiliac joints.

In capsular and ligamentous disorders or in spinal stenosis, localization is fixed, and remains unchanged over a long period.

Cause

A disc is damaged by prolonged wear and tear but symptoms only become manifest at the moment of internal derangement. An annular crack can also be caused by a single injury, but if there is a time gap between the injury and the onset of pain it will usually be very difficult to prove the aetiological significance of the latter.

Evolution

Episodic backache for years does not suggest progressive, serious disease. Greater suspicion arises if elderly patients, for the first time in their life, get a backache that is progressive. When pain is continuous, it is therefore vital to know if it is getting better or worse, is unchanging or is variable.

Frequency of attacks

The frequency of previous attacks provides information about the stability of the disc; it should always be considered in relation to the patient's profession. If the frequency is less than once a year in a person doing heavy work every day, the implication is that the disc is relatively stable. Treatment other than reduction and back school instruction is not required. In contrast, in a patient who does a fairly light job, attacks of lumbago three or four times a year indicate an unstable disc. Reduction is then just one part of the solution. The back requires stabilization with a corset, sclerosing injections into the surrounding inert structures or even operative intervention.

Pain-free intervals

The degree of pain and disability in between attacks must be assessed. Do the symptoms disappear completely and can the patient do everything that is wished between the bouts of pain, or does the pain never completely recede? In the former, reduction has been complete and a manipulative attempt will probably succeed again; in the latter, either the disc has not been reduced completely or a second (ligamentous) lesion remains. In either event, continuing pain is a bad prognostic sign for manipulation or traction alone.

Previous treatment

It is also necessary to determine whether the symptoms disappeared spontaneously or as the result of some specific treatment.

Discodural interactions

The history may also serve to obtain an idea of the degree of discodural interactions.

Marked articular symptoms (twinges) and postural deviation

These are characteristic of intense discodural contact. The deformity is noticed by the patient or by others. The typical case is acute lumbago in which the patient is painfully locked in flexion by a large central protrusion at the posterior aspect of a lumbar intervertebral joint. Any attempt to extend the lumbar spine squeezes the protrusion further backwards and increases the already painful pressure on the dura mater. Adoption of the flexed position decreases the pressure exerted by the subluxated part of cartilage on the dura mater.

A large posterolateral protrusion is accompanied by some deviation of the lumbar spine in lateral flexion, so projecting one hip sideways. The patient is not able to move in the opposite direction. Such lateral deviation suggests a lesion at the fourth or perhaps the third lumbar level.

When the lumbar spine is fixed in flexion or in lateral flexion because of root pain, all conservative treatment is likely to fail and surgical management is indicated.

If a patient with acute lumbago states that twinges are felt on even the slightest movements, therapists should be on their guard. Although manipulation can be tried safely, it will not always be tolerated. Traction, however, should definitely be abandoned, because it makes matters worse, usually at the moment it is released. The safest and most effective treatment is epidural local anaesthesia. It almost always affords immediate relief, although the large displacement remains present, continuing the marked deviation and limiting joint movements. Manipulation, carried out from the next day on, is much better tolerated and usually gives good results.

Numbness and/or weakness

The patient states that the foot flops during walking or that standing on tiptoe is impossible: this suggests a large posterolateral protrusion not reducible by manipulation or traction.

Box 36.4 summarizes the routine of taking a pain history. Table 36.2 outlines some typical histories.

Paraesthesia

When the patient states that there are 'pins and needles', this is pathognomonic of pressure on or inflammation of the peripheral nervous system. In practice, the cause is pressure. These symptoms are extremely important but are often not mentioned by the patient, so the examiner must enquire about them.

Box 36.4

Summary of pain history

Location

- Central, unilateral, bilateral
- Level ('forbidden area', S4 dermatome)

Onset

- Low back/leg
- Sudden/gradual

Evolution

- Shifting pain
- Expanding pain
- Alternating pain
- Sequence of backache–root pain
- Usual evolution – primary posterolateral protrusion

Factors influencing pain

- Relation between symptoms and posture/activities
- Typical histories:
  - Self-reducing disc lesion, spondylolisthesis
  - Spinal stenosis
  - Ligamentous postural syndrome
- Pain on coughing, in back/leg
- Twinges
- Early morning pain/nocturnal pain
- No influence on movement or posture

Duration of pain

- No time limit for backache
- Spontaneous recovery in unilateral sciatica

Previous attacks

- Frequency
- Pain-free periods

Two different syndromes causing paraesthesia must be considered: nerve root compression and pressure on the spinal cord.

Pressure on a nerve root results in a typical set of symptoms: pain and paraesthesia, strictly related to the segment involved. Pressure on the dural sleeve of a nerve root causes severe segmental pain. Pins and needles indicate that the nerve fibres are irritated and they are always felt in the distal extremity of the dermatome. For this reason, it is vital to determine their exact areas; an accurate dermatomal distribution of paraesthesia is always a better pointer to the affected nerve root than is the pain itself.

In external compression of the nerve root, the sheath is compressed before the fibres and pain will therefore appear before paraesthesia. In discoradicular interactions, the sequence of segmental pain first, followed later by pins and needles and numbness, is therefore an 'inherent likelihood'. If the paraesthesia appears before the pain begins, other lesions such as a neuroma or tumour should be suspected. In lateral recess stenosis, pain and paraesthesia usually appear simultaneously. Also, the symptoms do not tend to change over months or years.

If the paraesthesia is painless, a lumbar disc protrusion is an unlikely cause. Multiple sclerosis, diabetes, pernicious anaemia or cord compression is more likely. In these circumstances, pins and needles are also more diffuse in both feet or in all four limbs. The symptoms extend beyond the borders of innervation of any root or peripheral nerve. In cord compression, neck flexion may also bring on pins and needles.

Danger to S4 nerve roots

These roots are situated in the midline of the spinal canal, well protected by the posterior longitudinal ligament. In a large posterocentral protrusion, this ligament is placed under increased pressure. Finally, the ligament may rupture and damage the S4 roots (cauda equina syndrome).

Because mobility tests for the fourth sacral roots do not exist, it is almost impossible to evaluate their function. The diagnosis of cauda equina syndrome should therefore be made entirely on the history. Patients typically present with a classic triad of (1) saddle anaesthesia, (2) bowel and/or bladder dysfunction, and (3) lower extremity weakness.[4] Some patients are timid and do not mention these symptoms, so it is important to ask about them in the three types of case in which a large posterocentral protrusion is to be suspected: acute lumbago, acute perineal pain and bilateral sciatica. It should be re-emphasized that manipulation is absolutely contraindicated; even traction is not at all safe if the slightest suspicion of compression of the fourth sacral roots arises. Prompt surgery is required and any delay results in substantial morbidity.[5]

The patient's reaction to the symptoms

History taking should also determine how far the patient is disabled by the symptoms. Some patients are stoical, while others react in a hypersensitive way. Before active therapy such as manipulation is instituted, the presence of pronounced psychological factors must be established. For this reason, patients should be encouraged to relate how far their daily activities are disturbed. Later on, during the clinical examination, it will be established objectively how bad the handicap really is. If disablement is severe for a minimal lesion, it is likely that psychological problems are responsible for the symptoms and should be treated first. Furthermore, it is unwise to manipulate or inject a patient who seems to demonstrate psychoneurotic behaviour or is involved in a compensation claim.

Inspection

One important feature of this part of the examination is gaining an idea of the degree of disability. The clinician should observe the patient from the moment he or she enters the consulting room. In particular the following are noted:

Table 36.2 Some typical histories

Diagnosis	Age	Pain localization	Dural symptoms	Posture	Exertion
Discodural backache	15–70	Lumbar gluteal	+	Sitting provokes Walking eases	Bending provokes
Postural syndrome	30 or younger	Lumbar	–	Provokes	Eases
Lateral recess stenosis	Elderly	Leg, unilateral segmental	–	Standing and walking provoke Sitting, lying or bending forwards eases	No influence
Sciatica	Usually 20–50	Leg, unilateral segmental	+	Sitting often provokes Supine lying often eases	Provokes
Ankylosing spondylitis, 'active stage'	15–35	Lumbar; less often unilateral gluteal	–	Often worst on waking	May aggravate pain already present
Spondylolisthesis	15–35	Lumbar, bilateral sciatica	–	Prolonged standing provokes	Largely unconnected with exertion
Spinal malignant disease	No particular age Increasing central backache in an elderly patient	(Upper) lumbar, legs; multisegmental distribution	+	No particular postures, worse at night	Muscle spasm markedly limits movements

- *How does the patient enter the room?* A posture deformity in flexion or a deformity with a lateral pelvic tilt, possibly a slight limp, may be seen.
- *How does the patient sit down and how comfortably/uncomfortably does he or she sit?*
- *How does the patient get up from the chair?* A patient with low back pain may splint the spine in order to avoid painful movements.
- *What is the facial expression?* Is it in accordance with the pain the patient seems to suffer?

Next, the patient undresses so that posture can be observed, especially the lower back, pelvis and lower extremities. This is best done in good and uniform light; light falling from a unilateral source will give unilateral shadows, which may give a false idea of shape and posture.

The shape of the normal trunk

The patient should be observed posteriorly and laterally. From the posterior aspect, the shoulders and pelvis should be level and equal, and the soft tissue structures on both sides should be symmetrical (Fig. 36.1a). The thoracic and lumbar vertebrae should be vertically aligned. The angles of the scapulae should be level with the seventh thoracic spinous process; the iliac crests should line up with the fourth lumbar vertebra. The lower extremities should share the body load and be in good alignment: the hip joints not adducted or abducted, knees not bowed or knock-kneed, feet parallel or toeing out slightly, and the calcaneal bones neither pronated nor supinated.

From the side (Fig. 36.1b), the thoracic kyphosis and lumbar lordosis are observed and should have a normal curve.

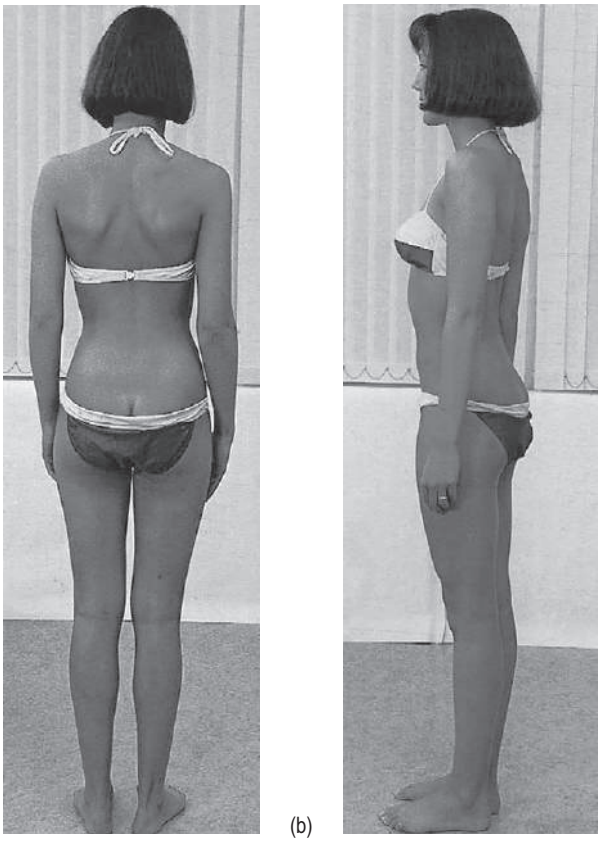


Fig 36.1 • The shape of the normal trunk.

The pelvis should be in the neutral position, i.e. the anterior superior iliac spines lie in the same vertical plane as the symphysis pubis. Hip, knee and ankle joints should be neither flexed nor hyperextended.

The pathological trunk

Posterior view

Many lumbar spinal disorders present with *asymmetrical posture*. This asymmetry may be in the vertical plane – the spinous processes do not align, or in a horizontal plane – the iliac crests, the anterior and posterior superior spines and the greater trochanters are not level in relation to each other. A pelvic tilt may be caused by anatomical changes above or below the greater trochanter, such as changes of the femoral head and neck or anatomical leg length discrepancy from growth disturbance. A lateral shift or list may have several causes.

Static scoliosis (Fig. 36.2a)

The origin of the list is a pelvic tilt due to a leg length difference; placing boards of various thicknesses under the foot of the shorter limb levels the pelvis, making the list disappear.

There is no clear evidence as to the significance of differences in leg length in the generation of spinal symptoms. If a platform under the shorter limb eases or even abolishes the pain while standing or on lumbar flexion or extension, a raised heel is advised. Some physicians recommend correction of any kind of leg length inequality. However, most investigators agree that mild leg length inequality of up to 15 mm is not a factor that contributes to low back pain.[6,7] Correction is therefore only of importance in recurrent attacks of lumbago and in the presence of a difference of more than 15 mm.

Sciatic scoliosis (Fig. 36.2b)

The lateral shift caused by mechanical dysfunction and muscle spasm in the lower lumbar spine is called sciatic scoliosis. It usually results from painful impingement of dura mater or nerve root. Most often, a shifted disc is responsible but it is good to remember that any space-occupying lesion in the vertebral canal can cause such an impingement.

In disc lesions, gross lateral deviation usually results from displacements at the L4 or L3 levels. Disc lesions at L5–S1 seldom result in marked lateral deviation because of the stabilizing action of the iliolumbar ligaments on the joint, although some pelvic tilt remains possible.

In lumbar disc displacements, six possible types of deviation (sciatic scoliosis) exist:

- *Towards the painful side*. This shows that the displacement is situated medially, i.e. at the axilla of the nerve root.
- *Away from the painful side*. In this case, the protrusion lies lateral to the nerve root, which is drawn away by the deviation of the trunk.
- *Alternating deviation*. This demonstrates that the dura mater slips from one side to the other of a small midline protrusion. It is also diagnostic of a protrusion at the fourth lumbar level.
- *Deviation on standing*, which disappears during flexion.
- *No deviation when standing erect but marked deviation on attempted trunk flexion*. This is often seen in root pain.
- *A momentary deviation when the trunk is flexed halfway*. The patient is seen to deviate suddenly at a particular moment during flexion, returning to a symmetrical posture as this point is passed. Usually pain is felt at the moment of deviation but occasionally it is not. This sign indicates that a fragment of disc alters its position at the back of the intervertebral joint and temporarily touches the dura mater.

Idiopathic scoliosis (Fig. 36.2c)

The curve of an idiopathic scoliosis, present since childhood, differs from a lateral shift associated with recent disc problems in that it is accompanied by a lower thoracic or lumbar rotation deformity. If this is not evident in the erect posture, it will become obvious during flexion.

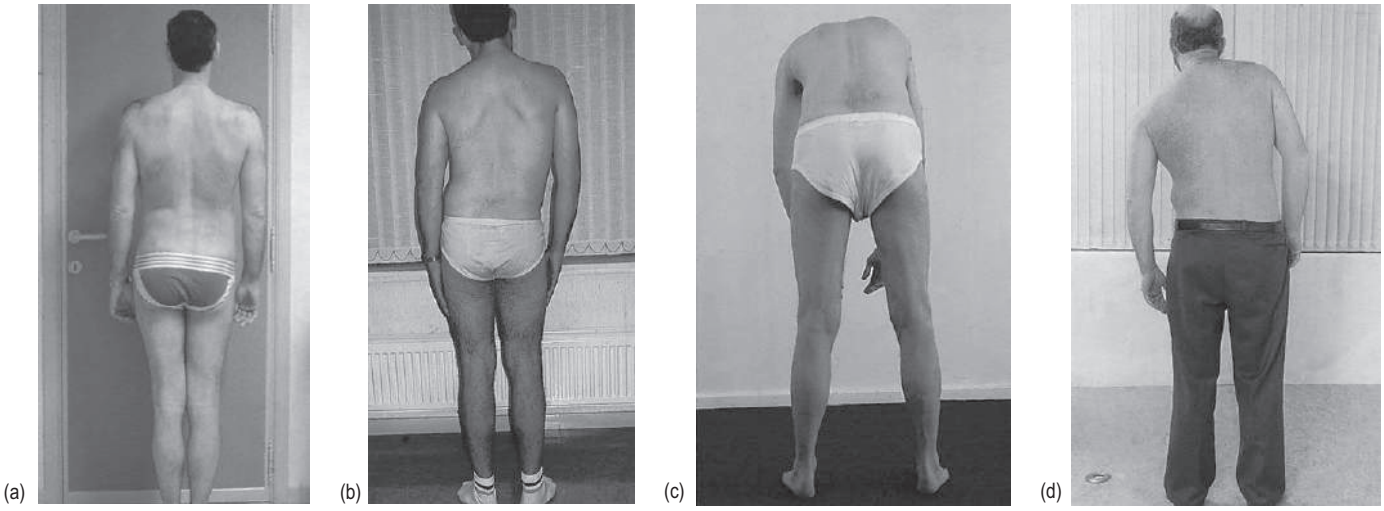


Fig 36.2 • Types of scoliosis: (a) static; (b) sciatic; (c) idiopathic; (d) psychogenic.

Psychogenic scoliosis (Fig. 36.2d)

In a psychogenic scoliosis, the wrong level is held fixed: although the pain is alleged to be lumbar, the patient holds neck, shoulders and thoracic spine in deviation whereas the lumbar spine remains vertical.

Lateral view

Increased lumbar lordosis

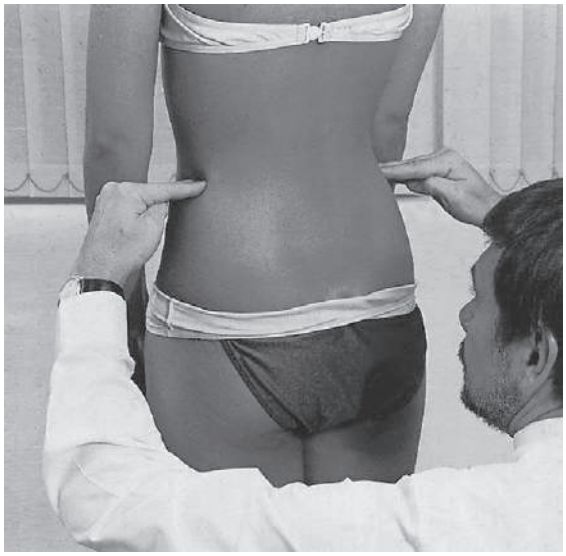
This often results from weak abdominal muscles and is then compensated by an increase in thoracic kyphosis. Lumbar lordosis may also compensate for a flexion deformity of the hip joint.

Excessive lordosis

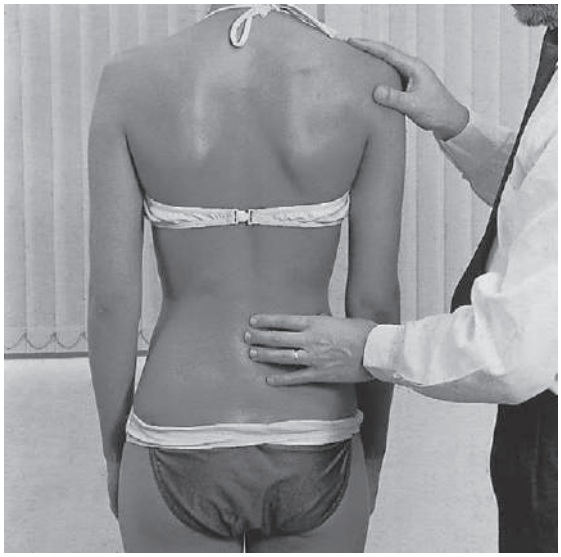
If this is not compensated by an equally excessive thoracic kyphosis, it is suggestive of spondylolisthesis. The whole spine lies in a plane anterior to the sacrum. This is characterized by a mid- or low-lumbar shelf at the spinous processes which, if not visible, can be palpated: when the hand slides gently downwards along the spinous processes, it engages the step at the fourth or fifth level (Fig. 36.3b). In concealed spondylolisthesis the shelf disappears during recumbency, and radiography in this position may not reveal the displacement.

Kyphotic posture

This is typical in acute lumbago. A large posterior projection accounts for a block at the back of the intervertebral joint; any attempt to straighten the back results in painful squeezing of the dura mater by the subluxated fragment. The patient stands in flexion deformity, with or without a lateral pelvic tilt. A discoradicular interaction at L3 may also force the spine into an antalgic kyphosis.



(a)



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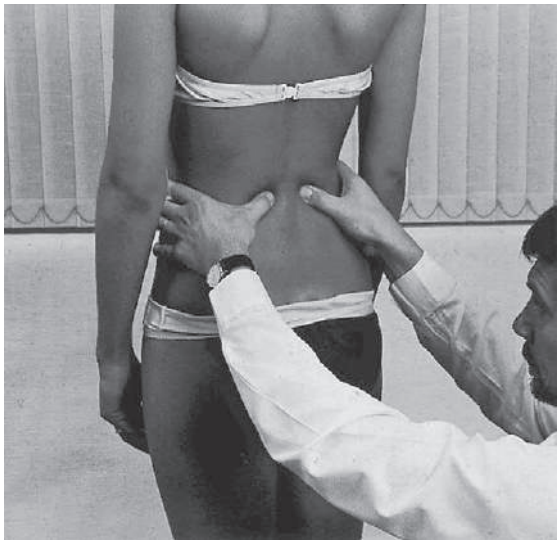


(c)

Fig 36.3 • Palpation of the iliac crests (a), shelf (b) and muscle spasm (c).

Exaggerated thoracic kyphosis

In young patients, this is characteristic of ankylosing spondylitis or adolescent osteochondrosis. In the elderly, it may indicate senile osteoporosis.

Angular kyphosis

This is caused either by gross thinning of two adjacent discs or by a wedge-shaped fracture of a vertebral body. The sign thus calls for a radiograph.

Flattened back

Patients with lumbar spinal stenosis or lateral recess stenosis usually present with a flattened back. They stay in a slightly stooped position, eliminating the normal lumbar lordosis.

Reduction of the space between the iliac crest and the thoracic cage

This indicates shortening of the thoracolumbar spine by disc-space narrowing at consecutive levels or marked osteoporosis.

Muscles

Wasting

Wasting of the paraspinal muscles is rare but may indicate chronic inflammatory disease, such as ankylosing spondylitis or tuberculosis, or point to poliomyelitis or a myopathy. It may also be seen after a previous spinal operation because of denervation.

Marked wasting of the calves, hamstrings or buttock occurs in fifth lumbar and first sacral root palsy.

In severe arthritis of the hip, the buttock, hamstrings and quadriceps will show visible wasting.

Spasm

Asymmetric spasm of the paraspinal or gluteal muscles, making them stand out compared to the normal side, is an ordinary finding in discodural or discoradicular problems, and is then accompanied by an adaptive posture in flexion or in side flexion. In mild cases, the difference in tension can be palpated (Fig. 36.3c). Muscle spasm, accompanied by visible flexion and/or lateral deformity, is also an unfavourable sign in sciatica. The protrusion nearly always proves irreducible.

Spasm of both sacrospinalis muscles, holding the lumbar spine in lordosis, may be suggestive of serious disease such as metastasis.

Skin and hair

A midline dimple or tufts of hair may suggest a variety of congenital, osseous or neurological disorders. In over 80% of all cases of occult spinal dysraphism, excess hair is present in the midline.

The colour of the skin may be an indication of vascular disorders. If the foot turns a dusky red on standing but blanches on elevation, advanced arterial obstruction is present. If this is associated with a painful limb, intermittent claudication is a real possibility.

Functional examination

Before the examination of lumbar movements is begun, the patient should be asked if there is any pain at this moment and to point out its site. If he or she indicates the upper lumbar/lower thoracic area, the examiner should be on the alert. Disc lesions at this spot are extremely rare but serious non-activity-related disorders are often situated here. Therefore the area is called the 'forbidden' area (Fig. 36.4).

In order to avoid missing important information, the examination must be performed in a practical and orderly routine. Tests are conducted in a standing position first, followed by lying supine and prone.

Examination standing

Procedure

Four active movements are examined while the examiner watches the patient from behind: backward bending, side bending to each side and forward bending completed at full range by neck flexion (Fig. 36.5).

Movements should be performed smoothly and gradually. Any deviation and/or restriction are noted and painfulness ascertained. As a movement is performed, the patient should tell the examiner when pain is felt and where. Momentary pain during the movement (painful arc) should not be missed and is pathognomonic for a disc lesion.

- *Extension* is recorded by noting the accentuation of the lumbar curve, as well as how far the patient can lean back before the pelvis tilts.
- *Lateral flexion* is measured by determining how far the patient can run the hand down the side of the leg. At full

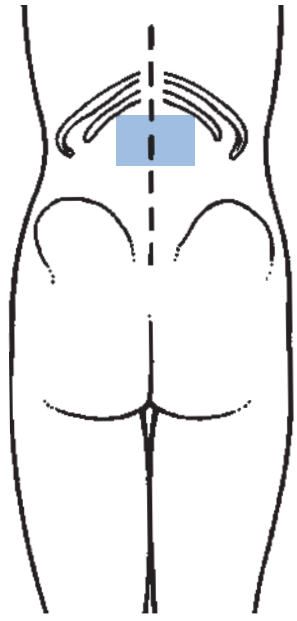


Fig 36.4 • The 'forbidden' area.

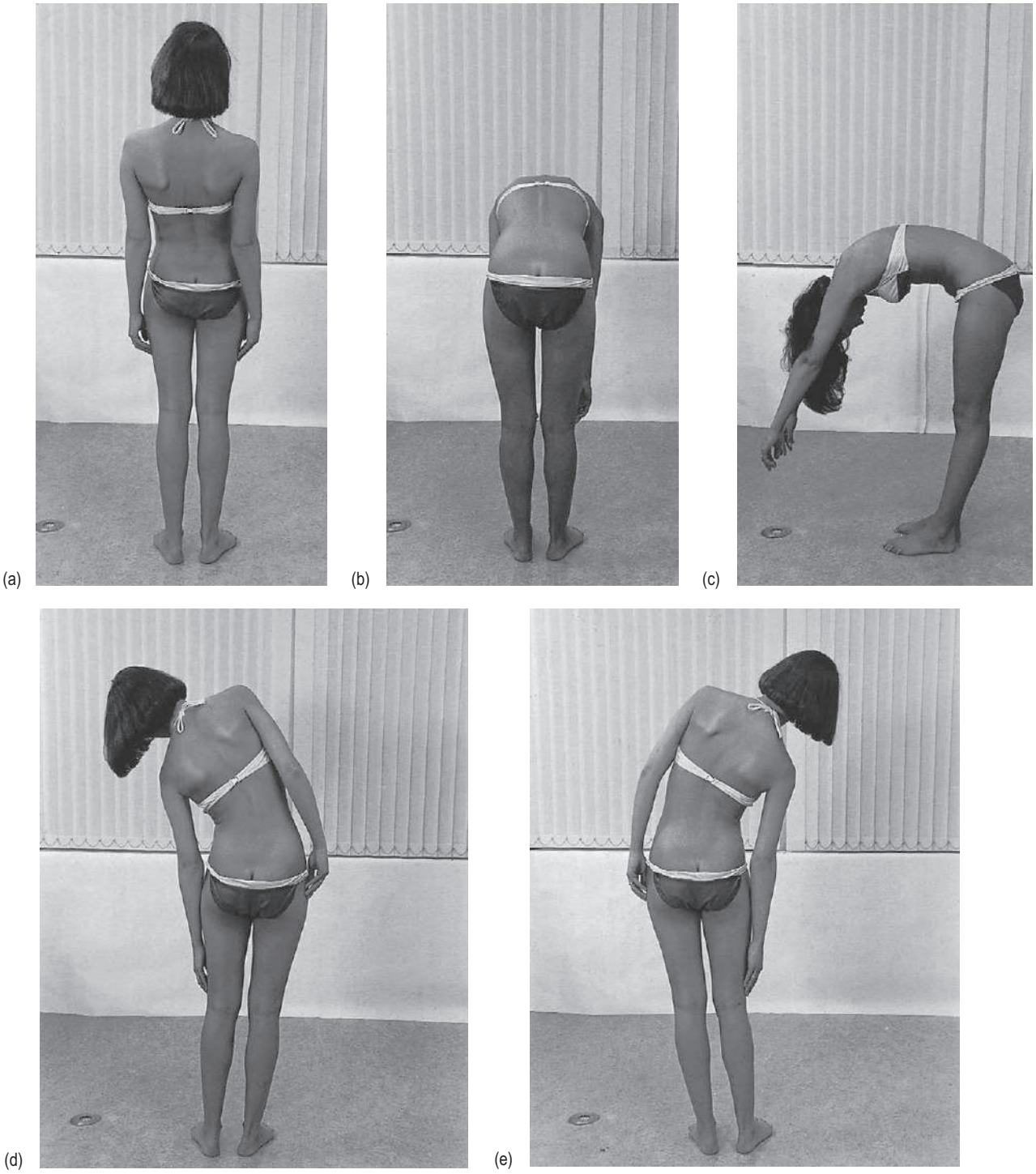


Fig 36.5 • Examination standing: (a) backward bending; (b, c) forward bending; (d, e) side bending.

range the lumbar spine should be curved uniformly in both directions. The patient is not allowed to bend forwards or backwards while performing the movement.

- *The range of forward flexion* is assessed by noting the distance of the fingertips from the floor. When complete body flexion has been attained, the lumbar spine is flattened or in young people even slightly convex. Forward bending is usually the most restricted and painful movement and may leave a persistent ache obscuring the responses to other movements. It is therefore preferable to examine this movement last. However, in ligamentous disorders and in stenosis of the spinal canal, bending forwards may be pain-free or may cause only minor discomfort.

The patient is also asked to flex the neck whenever forward bending becomes painful or at the full range of flexion. This movement stretches the dura by traction exerted from above. Pain provoked on neck flexion is a typical finding in backache and lumbago.

Findings

After the four lumbar movements have been tested, one of the following patterns may emerge:

- A partial articular pattern, with or without deviation.
- A painful arc of movement.
- End of range painful.
- Full articular pattern.
- Full range, no pain.

Partial articular pattern

This is very suggestive of internal derangement and strongly suggests a disc protrusion. One or more of the lumbar movements are painful, whereas the others are not, or are less painful (Fig. 36.6). If there is limitation of range, its degree is unequal and corresponds with the degree of pain.

The severity of the signs depends on the size of the displacement. The most striking example of the partial articular pattern is an attack of acute lumbago from a gross discodural interaction. Although all movements commonly hurt, pain and limitation on one movement will be more serious than in the opposite direction. A distinctive asymmetry is present.

Pain may be felt centrally or unilaterally, depending on the position of the protrusion. If the attack of lumbago is caused by a posterocentral displacement, flexion and extension are very painful and grossly restricted, whereas side flexion is only painful at the end of the range. In a gross unilateral protrusion, one side flexion may be completely blocked and painful, together with flexion and extension, whereas side flexion to the opposite side is not limited and causes only slight discomfort.

In backache caused by internal derangement, some movements are slightly limited or only painful at their extremes, and others are normal. Restriction of movement is not as striking as in acute lumbago.

Rarely, lesions of the posterior arch, i.e. posterior ligaments and capsules of the facet joints, cause a partial articular pattern but neither restriction nor a painful arc is to be expected here. Spinal deviation is also absent.

Painful arc

A painful arc may occur during or on the way back from forward bending or side bending. It always means that a fragment of disc shifts, jarring the dura mater momentarily via the posterior longitudinal ligament. Sometimes a painful arc exists when the trunk passes the vertical on swinging from one side to the other. The sign is usually associated with a partial articular pattern but it can also be an isolated finding.

Sometimes there is only a painless momentary deviation, which implies an arc unnoticed by the patient; a fragment of disc alters its position at the back of the intervertebral joint, without touching the dura mater.

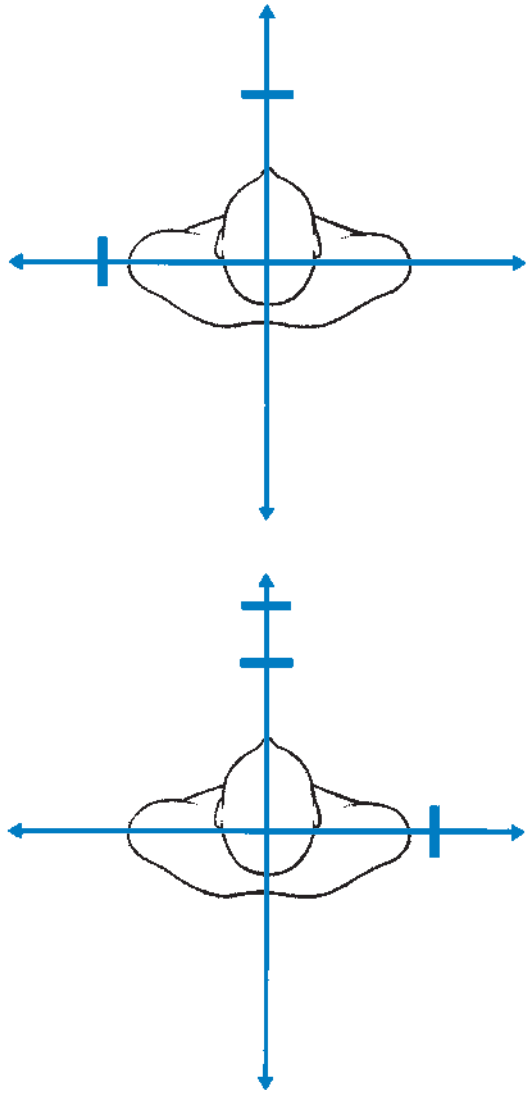


Fig 36.6 • Examples of partial articular patterns.

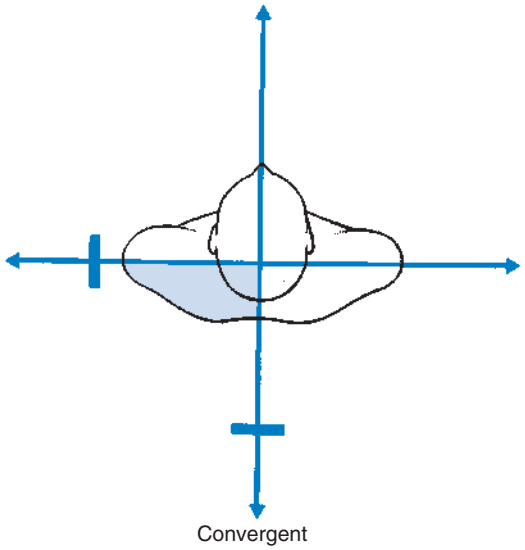
A painless click is a sign of abnormal articular displacement that is insufficient to irritate sensitive structures. It is not defined as an arc and its clinical significance is unclear.

The finding of a partial articular pattern together with a painful arc leads to three important conclusions:

- It is never psychogenic.
- It is pathognomonic of a disc protrusion – the dura comes into contact with the protrusion and slips over it.
- It indicates that the protrusion is small and reducible.

Pain at the end of movement

This is a common symptom in a small disc protrusion. However, it can also be the result of stretching an injured muscle or a sprained ligament or capsule. The discovery that resisted movement in the opposite direction is painless excludes the muscles. In a sprained ligament there is never a painful arc, and dural signs or root signs are absent. The movement that is supposed to stretch a ligament is also predictable: in sprain of the supraspinous and interspinous ligaments, full flexion is



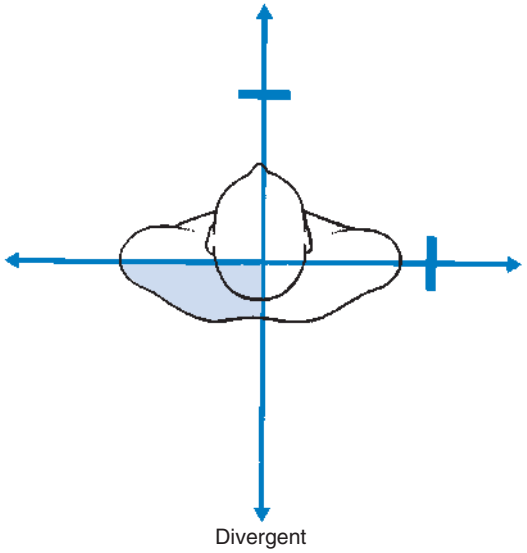


Fig 36.7 • Pain (coloured side) at the end of movement.

painful. If the iliolumbar ligaments are sprained on one side only, side flexion away from that side is painful, although there may also be pain on full flexion or on full extension.

In a capsular lesion of one of the apophyseal joints, movements also cause pain at the end of the range but now a convergent or divergent pattern is to be expected. This means that in a left-side joint, extension and side flexion to the left or flexion and side flexion to the right are painful (Fig. 36.7).

Full articular pattern (Fig. 36.8)

If all movements are painful and/or restricted in a uniform pattern, arthritis, arthrosis, fracture or malignant disease is suspected. In this respect, age and habitus are very important. Therefore gross limitation in every direction is quite normal in an elderly person but in adolescence it is usually a sign of a non-activity-related spinal disorder.

The typical example is a patient with ankylosing spondylitis who has a flat lumbar spine combined with bilateral limitation

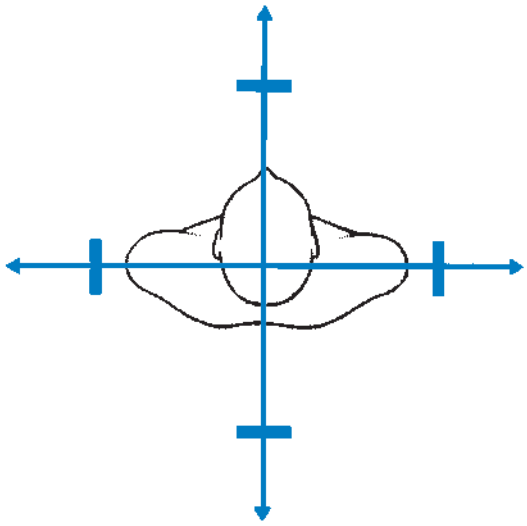


Fig 36.8 • Full articular pattern.

of side flexion and gross rigidity on forward flexion. The same limitations are present in tuberculosis of the disc or vertebra, malignant or benign tumour, Paget's disease and chronic osteomyelitis.

The finding of a full articular pattern is therefore often a warning sign and an indication for technical investigations.

Full range, without pain

Sometimes none of the four lumbar movements causes any discomfort. This may occur in the following circumstances.

- *History of a lumbar disc lesion but without displacement at the time of examination.* This is a well-known event in patients presenting with a self-reducing type of disc lesion. Every morning the patient awakes comfortable and is able to bend the back in every direction without any pain. After some hours the back begins to ache. If such a patient is seen early in the morning, all clinical tests are negative. Another example is the patient who is seen some days after an attack of acute lumbago. Because of spontaneous recovery, all symptoms may have been lost and no disc protrusion is present at the time of examination.
- *Pain referred to the back in the case of visceral disease.* If the history reveals that pain is not aggravated by activity or relieved by rest, a non-activity-related disorder should be suspected.
- *Ligamentous postural syndrome.* The pain is only provoked after standing or walking for a long time. Spinal movements are painless for the simple reason that the stress applied during the tests is not sufficient to induce pain.
- *Spondylolisthesis without a disc lesion.* This disorder resembles the ligamentous postural syndrome but patients may complain of bilateral sciatica as well. Inspection often shows a mid- or low-lumbar shelf.
- *'Bruised' dura mater or dural sleeve.* These patients have started with an ordinary attack of lumbago and/or sciatica.

There is a constant ache in the back or the limb, unaltered by movement or posture and most often worse at night. Epidural local anaesthesia abolishes the symptoms, which proves the dural origin.

- *Spinal stenosis*. The typical history is that of pain coming on during standing and walking. Lumbar movements, except perhaps extension, do not provoke the pain. If the patient is asked to stand for a while, pain arises in the back and limbs, disappearing again on flexion.

Interpretation

Each of the four movements may show some particularities that can have diagnostic importance. However, it should be emphasized again that a clinical diagnosis is only made on the patterns that emerge after all the tests have been performed.

Extension

The movement is initiated by contraction of the paravertebral muscles, whereas the iliopsoas and abdominal muscles relax smoothly to allow the movement to reach its extreme. The backward bending is usually limited to 20–30°. In order to stabilize the back, the patient can place both hands on the iliac crests while performing the test.

Painful limitation as part of a partial articular pattern

In acute lumbago, extension is usually completely blocked because of a large posterocentral protrusion. This limitation is part of a gross partial articular pattern. In sciatica, if trunk extension is considerably limited by severe pain shooting down the back of the limb, the prognosis is very poor and surgery is almost always indicated.

Painless limitation

In middle-aged or elderly people, painless limitation of extension results from osteophyte formation and/or diminished intervertebral joint space.

In long-standing ankylosing spondylitis, pain ceases when bony ankylosis is complete. Then not only extension is limited but also both side flexions.

Vertebral hyperostosis (Forestier disease) also leads to increasing painless stiffening of the spine.

Painful limitation and full articular pattern

Elderly patients with a spondylotic back show painless limitation of all four movements. However, if a disc lesion is superimposed, extension may also become painful. A similar picture may be seen in spinal stenosis: although there is a full articular pattern, pain and paraesthesia are only provoked by extension. Painful limitation of extension may also indicate ankylosing spondylitis. There is an obvious full articular pattern but only extension is painful.

In lateral recess stenosis, extension may provoke pain and/or paraesthesia in one leg only.

Pain on full range

In unilateral discodural backache, one common pattern is for extension to be of full range and painful centrally, whereas flexion causes unilateral lumbar or gluteal pain.

The L3 root is stretched on extension and relaxed on flexion. Therefore, in L3 root compression, extension is often painful whereas flexion produces relief.

In sciatica, extension sometimes causes pain in the lower limb instead of the back. If the patient is under the age of 60, manipulation will almost certainly fail.

Localized central pain on full extension may result from local periostitis at a spinal process.

Unilateral pain at the upper sacroiliac region or in the groin on full extension may result from a lesion of the iliolumbar ligaments.

In backache caused by a lesion of the capsule of a facet joint, a convergent pattern is often present: both extension and side flexion towards the pain produce pain at the end of range.

It is sometimes difficult to find the source of the problem if trunk extension creates pain in the buttock or the lower limb. When the pain is felt in one buttock only, its origin may be in the lumbar spine, the sacroiliac joint or the hip joint. When it is combined with segmental pain over the front of the thigh, the lesion must originate in the third lumbar segment: a third lumbar disc lesion or arthritis at the hip joint. They can be differentiated by performing an extension movement of the lumbar spine after flexing the hip to 90° – a position that avoids extension strain falling on the hip joint. If the pain is felt at the back of the thigh, the fifth lumbar and the sacroiliac joints are likely to be strained. Further investigation will then differentiate between these two locations.

Deviation

Sometimes the lumbar spine is seen to deviate slightly during extension so as to avoid pain. This involuntary manœuvre strongly suggests a disc lesion.

Side flexion

This movement is initiated by the paravertebral muscles, the psoas major and the external and internal oblique abdominal muscles on the same side. Contralateral muscles relax smoothly, controlling the movement. At the end of the range, the thorax and iliac crest approximate laterally. Side flexion accounts for approximately 15–20° on both sides.

Painful limitation of both side flexion movements

Cyriax stated that: 'All serious diseases of the lumbar spine result in limitation of the range of both side flexion movements.' The finding of this sign should be considered as a warning in young and middle-aged patients. Malignant and benign neoplasms, tuberculosis, chronic osteomyelitis, ankylosing spondylitis and fractures must be excluded.

Painless limitation of both side flexion movements

This is a normal finding in the elderly and is usually associated with spondylosis, or advanced osteoporosis, in which case extension and flexion are also seriously restricted.

Painful limitation of one side flexion movement

This usually results from a large unilateral protrusion. The joint is blocked at one side only. At the fourth or third lumbar level, these protrusions are usually associated with lateral deviation of the lumbar spine on standing.

If side flexion away from the symptomatic side is painful and limited, manipulative reduction is likely to have good results, although it may take special techniques and several sessions to produce a lasting cure.

In contrast, if side flexion towards the painful side hurts in a patient under the age of 60, manipulation often fails and traction is more likely to succeed. If this movement also causes pain in the lower limb instead of in the lumbar region or the upper buttock, manual reduction is almost impossible.

If gross limitation of side flexion away from the painful side is the only positive finding, a serious extra-articular lesion must again be suspected. Abdominal neoplasm or a neuroma at the lumbar or lower thoracic level commonly demonstrates this warning sign.

Pain at full range

Together with a partial articular pattern, this points towards internal derangement at an intervertebral joint. The absence of any limitation means that the protrusion is small and indicates that manipulation should succeed quickly.

Pain at the end of one side flexion is exceptionally caused by a muscular lesion, fracture or sprained ligament.

- *In a muscular lesion or fracture of a transverse process*, pain arises from stretching (bending to the contralateral side). Resisted side flexion in the opposite direction is also painful.
- *In unilateral posterior ligamentous dysfunction syndrome*, painful side bending towards the contralateral side suggests a lesion of either the iliolumbar ligament or the capsule of a facet joint. In the former, anteflexion and extension may also be painful. A facet joint lesion shows a divergent pattern: as well as side flexion, forward flexion is also painful at the end of range.

Painful arc

A painful arc during side flexion indicates a disc lesion, usually at the fourth lumbar level. An arc may be very subtle and present as a slight momentary pain when the patient moves the trunk from one side to the other. Sometimes the arc is quite extensive and can be missed if the patient is not encouraged to continue the movement when the pain appears.

Flexion

This is a complex movement that influences not only the lumbar spine and its neural contents but also the sacroiliac and hip joints. The movement is initiated by contraction of the iliopsoas and abdominal muscles. It then proceeds due to the force of gravity, the paravertebral muscles, gluteal muscles and hamstrings relaxing smoothly to allow the movement to be carried out to its extreme. At the end of the range, the vertebral column is stabilized only by the passive action of vertebral ligaments fixed to the bony pelvis.

Bending forwards causes pelvic rotation together with flexion of the lumbar spine. Normally, a smoothly graded ratio exists between the degree of pelvic rotation and that of lumbar flattening. This constitutes the 'lumbar–pelvic rhythm', which is difficult to quantify. However, at any phase of body flexion, the extent of lumbar curve flattening must be accompanied by a proportional degree of pelvic rotation around the transverse axis of the two hip joints. During these movements, a posterior shift of the hips in a horizontal plane takes place simultaneously, in order to maintain balance, an integral part of the pelvic portion of the lumbar–pelvic rhythm. The rhythm is disturbed if any of the component parts lacks function.[8]

Painful limitation

In an acute and severe discodural interaction, pain grossly limits trunk flexion because the weight of the body on forward bending further increases the size of the protrusion. In addition, this movement stretches the dura and draws it strongly forwards against the protrusion. Flexion of the neck performed at the moment of maximum lumbar flexion further stretches the dura from above and therefore increases the pain.

Flexion may also be limited by root pain. If this is the only sign, it usually indicates a primary posterolateral protrusion as found in young adults. The sign is then regarded as a root sign rather than an articular sign. Also, in secondary posterolateral protrusions, trunk flexion is limited by posterior leg pain. Here, however, this sign is associated with back or buttock pain on one or two other lumbar movements. Again, neck flexion may provoke or increase the pain in the limb, as it draws the nerve root structures more against a projection into the spinal canal.

Fixation in lordosis

During flexion, the lumbar spine may stay fixed in lordosis because of spasm of the sacrospinalis muscles. If both side-bending movements are also markedly limited, a serious non-activity-related disorder must be excluded.

Pain at the end of range

Central or unilateral pain in the low back on full flexion is a common articular sign found in most cases of backache and results from a small midline protrusion contacting the dura mater. It is usually accompanied by pain on some of the other spinal movements as part of a partial articular pattern. Rarely, localized central pain is caused by a sprained supra- or interspinous ligament. The only clinical finding then is pain at the end of flexion and extension.

If pain on full flexion is felt unilaterally at the level of the sacroiliac joint or the buttock, a small unilateral discodural conflict is likely. However, this must be differentiated from a lesion of the sacroiliac joint, hip joint or gluteal structures, all of which are also stretched at the end of flexion. A strained iliolumbar ligament is possible too. Side flexion away from the painful side is then also painful. An inflamed capsule of the facet joints may give rise to local unilateral pain, perhaps with slight reference to the upper buttock. Side bending away from the painful side is painful – the divergent pattern. It is important to note that, if the accessory movement of neck flexion, performed at the moment of full flexion, provokes or increases the pain in the back or buttock, all ligamentous, facet joint, sacroiliac or hip lesions can be excluded. This sign points to irritation of the dura mater.

Painless limitation

In elderly people, limitation of flexion, in combination with limitation of extension and both side movements, is normal and results from spondylosis. However, if a small lesion (disc or ligament) is superimposed on this condition, flexion is also painful. In L3 root compression, flexion is usually full and painless, because this movement relaxes the nerve.

Painful arc

A painful arc on flexion always means that a fragment of disc shifts, jarring the dura mater halfway through the movement.[9]

The pain disappears when the patient continues forward bending.

Lateral deviations

Lateral deviation of the spine during forward flexion also points to a disc lesion. It is caused by the way the spine accommodates a protrusion, preventing it from pressing on the dura mater or the dural investment of the nerve root. Usually the spine that stands upright symmetrically deviates on flexion. Sometimes the deviation that was present while standing disappears on bending forwards. In other patients, the deviation alternates, one way on bending forward and the other way on straightening up, the implication being that the dura mater has to be held to one or other side of a small projection.

Standing on tiptoe

The last test in the standing position is standing on tiptoe, which examines the strength of the calf muscles and thus the integrity of the S1/S2 segment. The patient is invited to perform the test, first on the good leg and then on the bad. The examiner steadies the patient with both hands, without taking any of the weight (Fig. 36.9). Inclining the body forwards and flexing the knee is evidence of weakness.

This test is best repeated several times in order to discover those cases with only slight weakness.

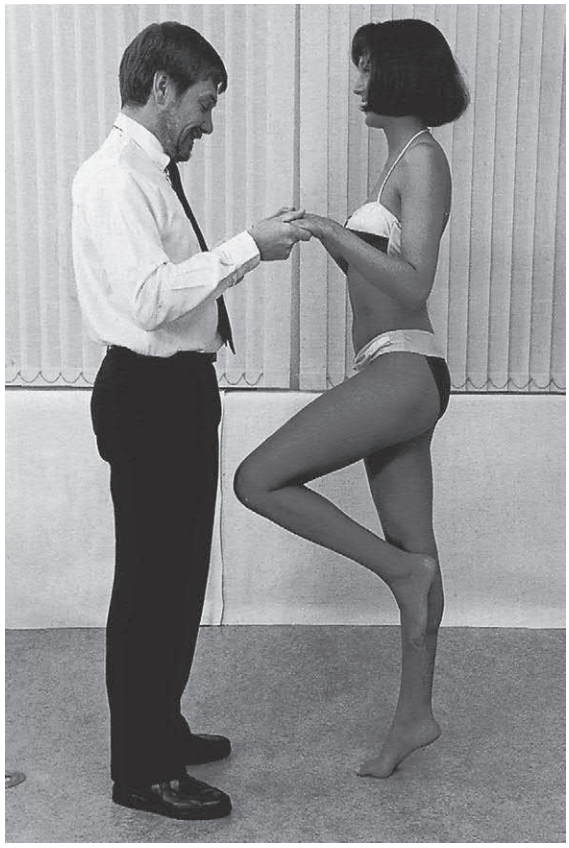


Fig 36.9 • Standing on tiptoe.

Warning

Standing

- Impossible to stand for a moment because of severe pain.
- Full articular pattern.
- Limitation of the range of both side flexion movements in young and middle-aged patients.
- Gross limitation of side flexion away from the painful side as the only positive finding.
- Fixation in lordosis during flexion together with marked limitation of both side flexion movements.

Examination supine

The patient is asked to lie supine. The way patients move to get on the couch should correspond to their previous performance and to the information gained from the history. In suspected exaggeration or 'malingering', careful observation of the patient's attitude can be informative. For example, turning from a sitting to a supine position places particular strain on the low back, and, especially in acute lumbago, patients can be expected to support themselves with their arms. Moving onto and off the couch easily means that the psoas muscles must have normal strength. If, before lying down, the patient is able to sit on the couch with the legs stretched out, straight leg raising must be of full range. Other discrepancies are discussed in Section 8.

Sacroiliac joints

Pain in the buttock most often results from disorders of the lumbar spine. However, pain from hip and sacroiliac disorders is referred to the same area. To exclude sacroiliac disorders, a specific test should be done to exert tension on the capsule and ligaments of the sacroiliac joint without affecting the lumbar spine or the hip joint. Distraction of the iliacs seems to be the best scanning test that fulfils this condition.[10] It is performed as described below.[11]

The examiner places the hands on the anterior superior spines of the ilium with the arms crossed (Fig. 36.10). Pressure is exerted in a downward and outward direction and should be evenly distributed to prevent moving the lumbar region.

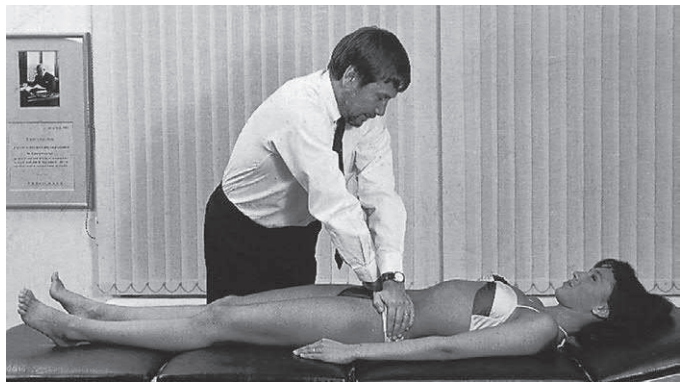


Fig 36.10 • Testing the sacroiliac joints.

In a positive test, a deep-seated unilateral ache is evoked at the gluteal and/or posterior crural area. In acute lumbar pain syndromes, it may be necessary to perform this test with the patient's forearm supporting the low lumbar area. In this way, the lumbar segments are better prevented from moving into flexion. This measure also prevents a tender part of the sacrum or of one of the posterior superior iliac spines from being pressed painfully against the couch. Confirmation of unilateral pain strongly indicates sacroiliitis or strain of the anterior sacroiliac ligaments. In contrast, if the patient states that the pain is felt centrally, it is clear that this is irrelevant, because it is impossible for a unilateral structure to refer pain centrally. Probably it has more to do with the appearance of referred tenderness at the dorsal part of the sacrum, which is pressed against the couch. Discomfort at the anterior superior iliac spines can also be ignored.

The distraction test at the sacroiliac joint has very high specificity and 100% sensitivity.[12] The test is extremely important in the clinical diagnosis of back pain and should never be omitted; there is almost nothing in the nature and extent of sacroiliac pain that distinguishes it from a disc protrusion compressing either the dura mater or the dural extent of the S1 and S2 nerve roots. The fact that the pain probably comes and goes irrespective of posture and exertion, or often changes sides, draws attention to the possibility of sacroiliac arthritis. To make matters more confusing, coughing also hurts because the increase in abdominal pressure painfully distracts the ilium from the sacrum. Also, routine clinical examination does not usually differentiate sacroiliac arthritis from a disc lesion: the lumbar movements may increase the pain a little at full range; flexion can be very painful and even limited; and straight leg raising may also prove to be painful. It should therefore not be surprising that the diagnosis is easily missed and that patients are often treated on the assumption that a disc lesion is present, which may even lead to unnecessary surgery. Moreover, a normal radiographic appearance of the sacroiliac joints does not always exclude arthritis, as symptoms may precede the radiological evidence by months or even years. It is therefore vital never to forget the sacroiliac distraction test during routine lumbar examination.

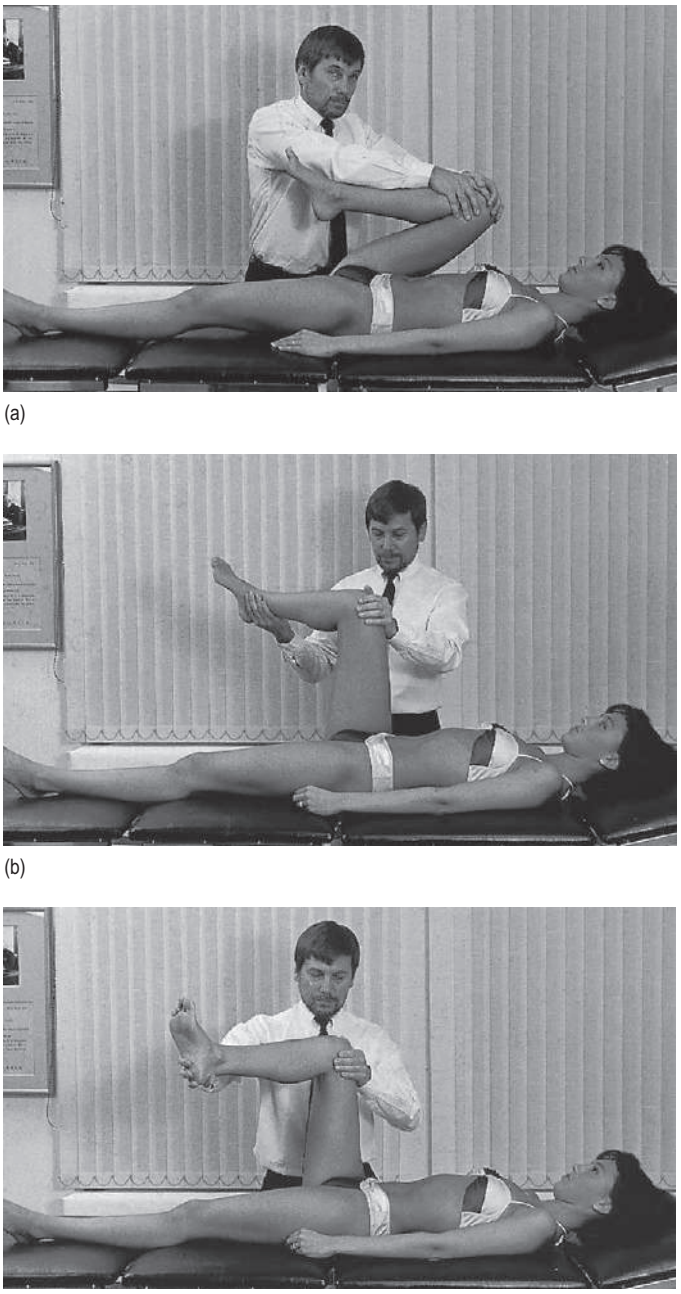


Fig 36.11 • Testing the hip joints: (a) flexion; (b) medial rotation; (c) lateral rotation.

Hip joints

After the sacroiliac joints are tested, three basic tests for the hip joints are performed. It may be important to differentiate between the lumbar spine and the hip joints, especially in the case of pain in one buttock and/or the anterior thigh.

Both sides, the uninvolved one first, are tested for range, end-feel and pain on flexion, lateral rotation and medial rotation.

The thigh is moved into flexion until it touches the abdomen (Fig. 36.11a). The movements of rotation are tested while the hip joint is held in 90° of flexion. One hand stabilizes the femur at the knee, while the other is placed at the distal end of the lower leg and performs the rotation movement (Fig. 36.11b, c).

In minor lumbar lesions, none of these movements usually hurts at the back. In a patient with severe lumbar pain, however, some of these tests can be slightly painful. Full hip flexion, for instance, may exert slight traction on the sciatic nerve roots. Also, full medial rotation stretches the sciatic nerve trunk and may cause an ache in the buttock.[13] The same applies to the sacroiliac joints. Moving the hip joint beyond full range involves the next link of the moving chain – the sacroiliac joint – and may evoke sacroiliac pain. Full lateral rotation at the hip stretches the anterior sacroiliac ligaments and full medial rotation has the same effect on the posterior ligaments (see Ch. 43).

Straight leg raising test

The straight leg raising (SLR) test is performed to estimate the mobility of the dura mater, as well as the dural investments of the nerve roots at the *fourth lumbar to second sacral segments*.

Historical note

This test was first presented by JJ Frost in 1881 with reference to his teacher, Ernest Charles Lasègue. Frost proposed the test as an aid in distinguishing hip from sciatic pain.[14] Later, a number of workers[13,15–17] demonstrated that this movement exerts tension on nerve roots and dural tube. The mobility of the nerve roots at the intervertebral foramen has also been investigated and shows a range of 2–8 mm on SLR.[18–20] Careful study[21] of subjects between 35 and 55 years of age showed a downward movement of 1.5 mm for the fourth lumbar root, 3 mm for the fifth lumbar root and 4 mm for the first sacral root. A more recent study on fresh cadavers showed the intrathecal movement of the lumbosacral roots induced by SLR of 70° to be 0.96 mm, 1.54 mm and 2.31 mm for roots L4, L5 and S1, respectively.[22]

Significance of the test

In a study of 50 consecutive surgical patients with clinical and radiographic evidence of lumbar disc herniation, it was shown that SLR was the most sensitive preoperative physical diagnostic sign (96%) for correlating intraoperative pathology of lumbar disc herniation.[23] The sign has also been helpful in diagnosing discodural backache.[24,25] However, the significance of neither the presence nor the absence of the signs should not be overestimated.[26,27] SLR as an isolated phenomenon has *no* diagnostic significance and must always be interpreted in association with other clinical findings. As can be seen in Box 36.5, limitation of the mobility of nerve roots and dural tube is not pathognomonic for a disc lesion.

Box 36.5

Straight leg raising (SLR)

The SLR test may be positive in:

Intraspinal lesions

Discogenic

- Protrusion

Non-discogenic

- Tumour
- Neuroma

Extraspinal lesions

- Sacroiliac joint lesions
- Major lesions at the buttock
- Major lesions at the hip joint
- Lesions of the hamstring muscle belly
- Non-organic disorders

Conversely, a completely painless SLR does not exclude a disc lesion. Circumstances that produce a negative test in discoradicular problems are as follows:

- Cases where the nerve root emerges a little higher up in the foramen and does not come into contact with the protrusion
- Protrusions at the second or third lumbar level and the third or fourth sacral nerve roots, which are not influenced by the manœuvre
- Minor protrusions that interfere only slightly with the dura mater and therefore do not influence its mobility.

Intraspinal space-occupying lesions

Any space-occupying lesion situated at the anterior wall of the vertebral canal may interfere with the dura mater and/or the nerve root structures and thus painfully limit movement. Most often this is a posterocentral or posterolateral disc displacement but it may also be a tumour, inflammation or haemorrhage.

The sign is absent in radicular disorders where pressure is exerted from behind, such as in a narrowed lateral recess or a hypertrophic apophyseal joint. Because the SLR drags the nerve roots downwards and forwards, they are moved away from the compression during the manœuvre.

Apparent acute lumbago and negative SLR

Examiners should be on their guard against apparent cases of acute lumbago in combination with a negative SLR. In these cases, it is non-mechanical disorders that should be suspected rather than a lower-level discodural conflict.

Disc lesions at the second or third lumbar level

In these lesions, SLR is usually of full range because the movement does not directly interfere with these roots.[18] However, full SLR may aggravate lumbar pain through traction exerted on the dura via the roots below.

Hamstring tightness

It is also possible for tightness of the hamstrings to limit the manœuvre, sometimes by up to 50°. By raising the leg on the other side, the same degree of limitation, caused by a similar tightness, will differentiate from the true reactive muscle spasm caused by dural irritation.

Painful disorders of the sacroiliac joint

Straight leg raising may also be painful, though not limited, in sacroiliac disorders. At full range, traction through the tightened hamstrings is exerted on the sacrotuberous and sacrospinous ligaments, and the anterior capsule of the joint.[28]

Major lesions at the buttock

Serious disorders at the buttock, such as osteomyelitis and metastasis at the ilium or upper femur, a fractured sacrum or chronic septic sacroiliac arthritis, also influence the SLR manœuvre. The combination with positive hip signs forms the 'sign of the buttock' (see p. 637).

Performing the test

Before testing, it should be assumed that there is at least 90° of flexion at the hip joint; otherwise conclusions cannot be drawn. Then the leg is lifted upwards from the anatomical position by supporting the foot at the calcaneus. To prevent

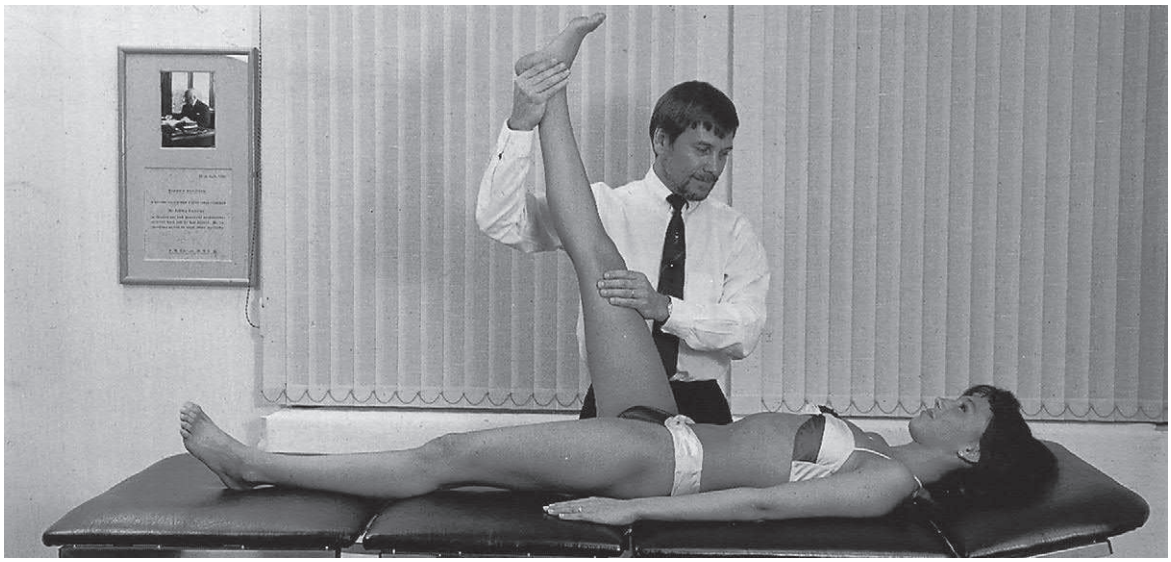


Fig 36.12 • Straight leg raising.

the knee from bending, the other hand is placed on its anterior aspect (Fig. 36.12). The patient should also not be allowed to rotate the pelvis forwards or to abduct and externally rotate the leg at the hip, in order to escape painful stretching.

The examiner should compare both sides for any differences in range, end-feel and degree of discomfort, starting with the painless side. The normal range varies greatly from person to person: due to tension in the hamstrings, stiff patients can only reach 60° whereas hypermobile people may show a range of more than 120°. In impaired mobility of the dura mater and/or one of the two lower lumbar and upper sacral nerve roots, involuntary spasm of the hamstring muscles abruptly prevents further movement to the full physiological range. At this point, the patient may state that pain in the back or leg is reproduced, a sign that the dura or the dural investments of the nerve roots are irritated. The examiner must not fail to force SLR gently, as long as this causes only slight pain and the hamstring muscles do not abruptly terminate the movement. Otherwise a painful arc may be missed or those uncommon cases in which pain begins at, for example 45° but the leg can be moved to 90° without increased discomfort.

The degree of limitation varies with the degree of the discodural or discoradicular interaction. However, this rule only holds as long as there is no parenchymatous involvement. As soon as there are detectable neurological sings, SLR becomes independent of the degree of discoradicular interaction. (See Box 36.6 for an outline of the six stages in the SLR test.)

Box 36.6

Stages in the straight leg raising test

Six stages can be distinguished and each used as a criterion to ascertain the size of the protrusion.

• Full and painless	This does not exclude disc protrusions. In the supine position these may be too small to make contact with the dura or the dural sleeve and thus these structures can move freely
• Pain on full range	A small protrusion is likely
• Painful arc	Suggestive of a small protrusion. The dura or nerve root slips over the projection
• Painful and limited without neurological deficit	The protrusion is larger, limiting the mobility of the dura or the dural sleeve of one of the lower lumbar or upper sacral nerve roots
• Painful and limited with neurological deficit	A large posterolateral protrusion is compressing a nerve root, impairing mobility and conduction. The severity of the palsy takes over from SLR as the criterion for the degree of interference
• Full and painless with neurological deficit	A large posterolateral protrusion has become maximal, compressing the nerve root so intensively that it has become ischaemic and atrophied

Painful arc (Fig. 36.13)

The patient feels a momentary pain on the way up and/or on the way down. A painful arc may be an isolated finding during SLR but is usually seen in combination with pain at full range. The finding is very important because it is pathognomonic for a disc lesion: the moving nerve root catches the protrusion momentarily and slips over it. It implies that the symptoms are not caused by a muscular or ligamentous strain, and that a psychogenic disorder can be excluded. Furthermore, such a protrusion must be small in size, and therefore manipulation is expected to succeed quickly.

Cross-leg straight leg raising test

This test is positive when moving the uninvolved leg reproduces the back or sciatic pain. This results from movement of the dura and the contralateral nerve, which is dragged downwards and medially.[29] It strongly suggests an axial localization of the protrusion and points to the fourth lumbar level[30] but

some[31,32] have failed to correlate the position of the disc protrusion in relation to the root at laminectomy. However, a very high incidence of sequestration or extrusion is seen at operation in patients with cross-leg pain (Fig. 36.14).[26,33,34]

Straining the contralateral sacroiliac joint

At full range the manœuvre may also strain the contralateral sacroiliac joint. After taking up all the slack in the ligaments on the uninvolved side, the sacrum and the iliac bone move together and the movement puts a rotational strain on the contralateral joint.

Straight leg raising with neck flexion

At the moment SLR becomes painful, the patient is asked to flex the neck, while keeping the trunk still. This often increases the pain by pulling on the dura mater from above, adding tension to the impaired dural structures.[35] This clear 'dural sign' excludes the possibility of a major sacroiliac buttock or hamstring lesion (Fig. 36.15).

Bragard's test

The presence of nerve irritability can also be confirmed with the manœuvre known as Bragard's test. The raised leg should be lowered until pain is relieved. In that position the foot is dorsiflexed, which causes a recurrence of pain as a result of stretching the sciatic nerve via the tibial nerve.[36,37]

The 'bowstring' sign

This is also suggested to be a very reliable test of root tension.[15] In this manœuvre, SLR is carried out until pain is reproduced. At this level, the knee is slightly flexed until pain abates. The patient's limb is rested on the examiner's shoulder and the patient's thumbs are placed in the popliteal fossa, over the sciatic nerve. If sudden firm pressure on the nerve gives rise to pain in the back or down the leg, the patient is almost certainly suffering from significant root tension.

Lumbago and straight leg raising

The degree of limitation corresponds to the degree of discodural contact.[22] In large posterocentral protrusions, SLR is limited bilaterally. Unilateral lumbago often restricts the

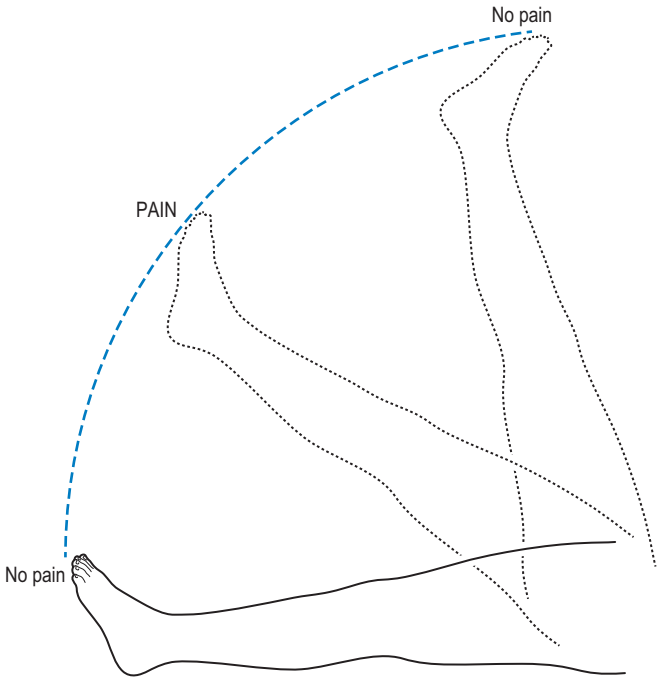


Fig 36.13 • Painful arc on straight leg raising. Redrawn from Cyriax J., *Textbook of Orthopaedic Medicine*, Volume 1, 1982, Harcourt Brace & Co Ltd, Elsevier Health Science Books, with permission.

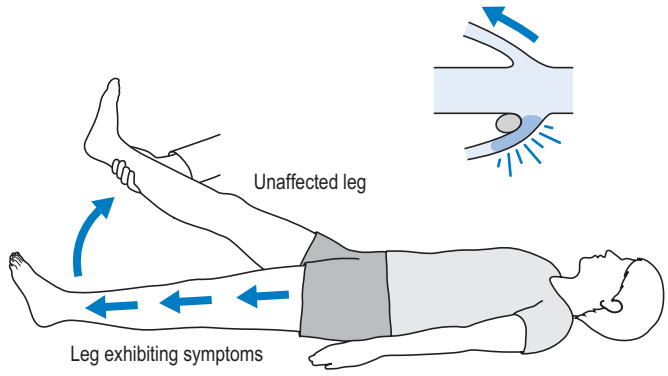


Fig 36.14 • Cross-leg straight leg raising: the localization of the protrusion is axial. Redrawn from Magee DJ. *Orthopaedic Physical Assessment*. 2008, Saunders, with permission.

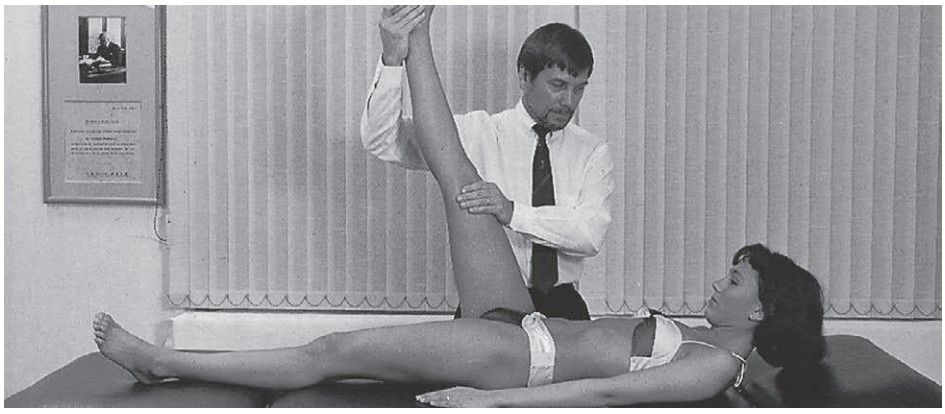


Fig 36.15 • Modified straight leg raising: with neck flexion.

manœuvre on the affected side only or to a greater degree on that side than on the other.

Any change in discodural contact alters the range of SLR instantly. Hence, the sign is very useful in assessing the effect of treatment. So, during a manipulation session, before starting traction, or during a period of bed rest, SLR is a good test for estimating progress without stressing the lesion. From the moment SLR becomes negative, active movement of the lumbar spine while standing becomes the new criterion for testing. In this position, intradiscal pressure is raised, which may cause renewed discodural contact.

Sciatica and straight leg raising

The SLR test is very useful to ascertain the degree of discoradicular compression.[23] If the root is not yet atrophic, the degree of restriction of SLR is proportional to the pressure exerted on the nerve root. However, from the point at which conduction becomes impaired, which is coupled with neurological signs, the degree of interference affords the new criterion of the size of the protrusion. Indeed, although the protrusion has become larger, restriction of SLR may not have altered or may even have returned to full range. In the latter case, the patient has developed an ischaemic root palsy. The protrusion has become maximal in size but, as a result of the ischaemia, the nerve root has lost its function, including that of pain conduction. Dural sleeve pain thus ceases and SLR returns to full range. The patient is subjectively better – pain-free – but the lesion is anatomically worse. The large protrusion will undoubtedly be seen on computed tomography (CT), although the SLR test has become negative (see Box 36.6).

Non-organic disorders and straight leg raising

The range of SLR must always be compared with the range of trunk flexion on standing. Because intradiscal pressure is higher on standing and bending forwards, restriction is expected to be greatest on this test or at least equal to SLR, in which the body weight is off the joint. The converse does not hold: many perfectly genuine disc lesions restrict trunk flexion but not SLR. Unless this difference is appreciated, patients with medicolegal claims may be treated unfairly.

However, the inconsistencies likely in psychogenic disorders should be recognized, in order to avoid treatment of a spinal lesion that does not exist. If there is any doubt, additional tests should be performed to establish these inconsistencies. For example, if the patient sits on the couch with the legs outstretched in front, discrepancy between the degree of alleged limitation of SLR and the degree of hip flexion needed to be able to sit on the couch confirms the suspicion (see online chapter *Psychogenic pain*).

Testing the integrity of spinal segmental innervation

This includes examination of muscle strength, sensation and reflexes of the entire lower extremity. Comparison should always be made with the contralateral side.

When signs of interference with nerve conduction are found, the degree of involvement should also be estimated. In discoradicular interactions, motor conduction may be reduced but there is seldom complete paralysis.

Positive root signs have both diagnostic and therapeutic value. Motor weakness is the most reliable localizing sign of nerve root involvement. Sensory changes, on the contrary, are rather subjective and easily affected by emotional state. Reflex changes may result from a previous episode of nerve root compression. As for treatment, disturbance of spinal segmental innervation indicates that the protrusion is too large and located too far laterally for attempts at manipulative reduction to succeed. For this reason, all tests of conduction should be repeated before each session of manipulation or attempted traction, especially if the patient does not respond in the expected way. It is possible that, during the interval between sessions, a root palsy develops, which makes further attempts at reduction futile.

A disc protrusion usually affects one root only. However, because of the obliquity of the lumbar nerve roots, a large protrusion may lie at the interval between two roots, catching the motor part of the upper root and the sensory part of the lower root. This clinical observation is seen in L4 protrusions affecting the fourth and fifth roots, as well as in L5 protrusions with palsy of the fifth lumbar and first sacral roots.

Impaired conduction of more than two roots is so rare that neoplasm should be suspected first. The same applies to bilateral root palsies.

Diffuse weakness of all muscle groups, particularly the psoas muscle, is highly suggestive of a psychological disorder.[2,30]

Tests of motor conduction

There are four tests in the supine position.

Resisted flexion of the hip

This tests the L2 and L3 nerve roots. It is performed with the hip joint flexed to 90° so as to eliminate activity of the rectus femoris as much as possible. Both hands are placed at the distal end of the thigh and the patient attempts to resist the strong force applied by the examiner (Fig. 36.16). At the same time, it is necessary to stabilize the ilium with one knee placed against the patient's ischial tuberosity.

If the attempted movement is weak and painful, neoplasm should be suspected. A second lumbar root palsy is hardly ever caused by a disc protrusion (1 in 1000 lumbar protrusions) and serious disease is more likely: for example, a neuroma or metastasis. The latter may be located vertebrally or at the upper femur.

In a third lumbar root palsy, resisted flexion of the hip is only slightly impaired but there is striking loss of power of the quadriceps.

Resisted dorsiflexion of the foot

This tests the L4 nerve root. The patient lies supine with the hips and knees extended. The patient holds the ankle in full dorsiflexion and should resist the full weight of the examiner's body (Fig. 36.17).

Resisted dorsiflexion of the big toe

This tests the L4 and L5 nerve roots. The examiner places the thumb on the nail bed of the great toe and the fingers on the ball of the foot. The patient is asked to resist the examiner's attempt to plantiflex the great toe (Fig. 36.18).

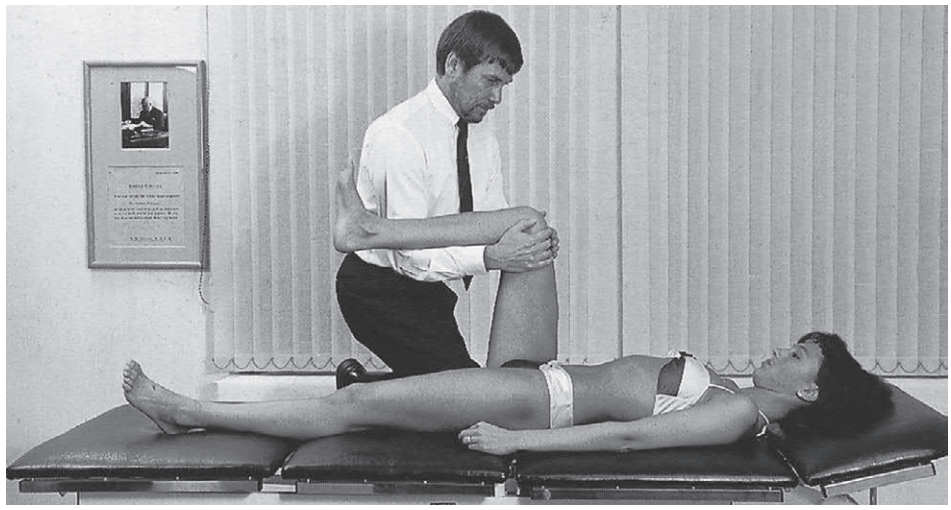


Fig 36.16 • Resisted flexion of the hip.

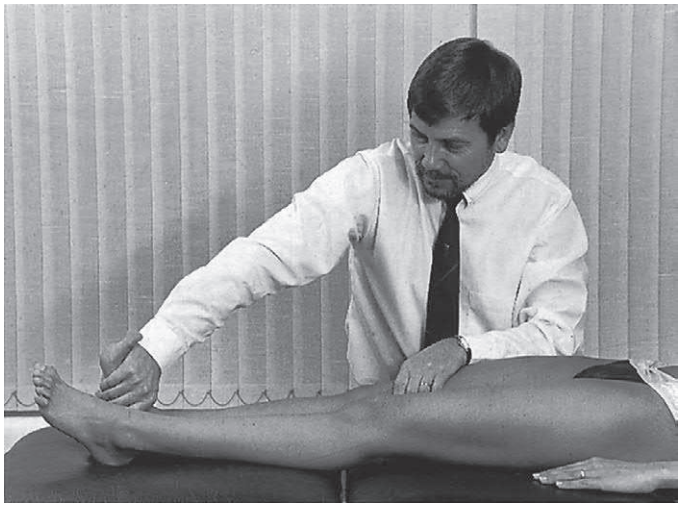


Fig 36.17 • Resisted dorsiflexion of the foot.

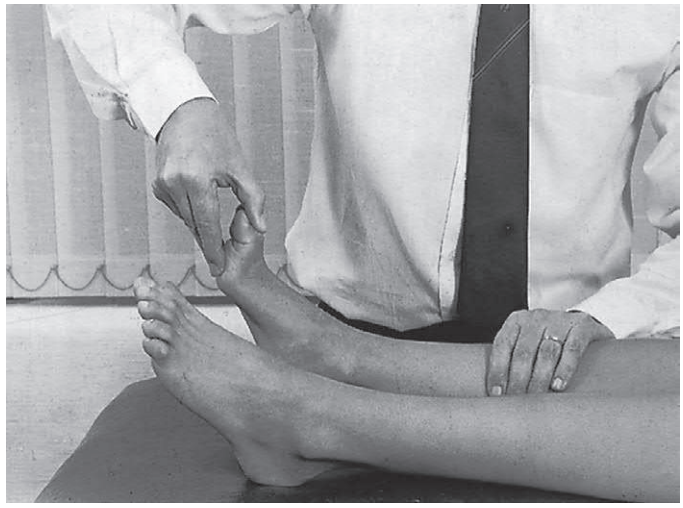


Fig 36.18 • Resisted dorsiflexion of the big toe.

Resisted eversion of the foot

This tests the L5 and S1 nerve roots. One hand stabilizes the ankle at the medial side, while the other hand is placed at the outer side of the forefoot. The patient is asked to resist the examiner's attempt to move the foot into dorsiflexion and inversion (Fig. 36.19). When weakness is present, the examiner needs to be aware of efforts to substitute the eversion movement by rotating the leg outwards at the hip.

It should be emphasized that, while these tests are being performed, the patient with normal motor nerve conduction will be able to resist the strongest power exerted by the examiner, except in dorsiflexion of the great toe, where the examiner will be stronger.

Tests of sensory conduction

These are performed next. The various areas are compared bilaterally at the same time (Fig. 36.20):

- Front of the thigh: L2.
- Front and inner side of the lower leg to just above the foot: L3.
- Big toe only: L4.
- Big toe and two adjacent toes: L5.
- Outer border of the foot, together with the two outer toes: S1.
- Sole of the heel: S2.

Disturbed sensory conduction in both legs is atypical in disc lesions but does occur in spondylolisthesis or neoplasm.

Knee reflex text

This may be diminished in lesions of the third lumbar root. Each knee is raised in turn with one hand and the ligamentum patellae struck with the reflex hammer (Fig. 36.21).

Testing the integrity of the spinal cord

The integrity of the spinal cord should be tested in all patients suspected of an upper motor neurone lesion. Clinical syndromes that suggest this are:

- Root palsy affecting more than one root, especially if this is bilateral.

Fig 36.19 • Resisted eversion of the foot.

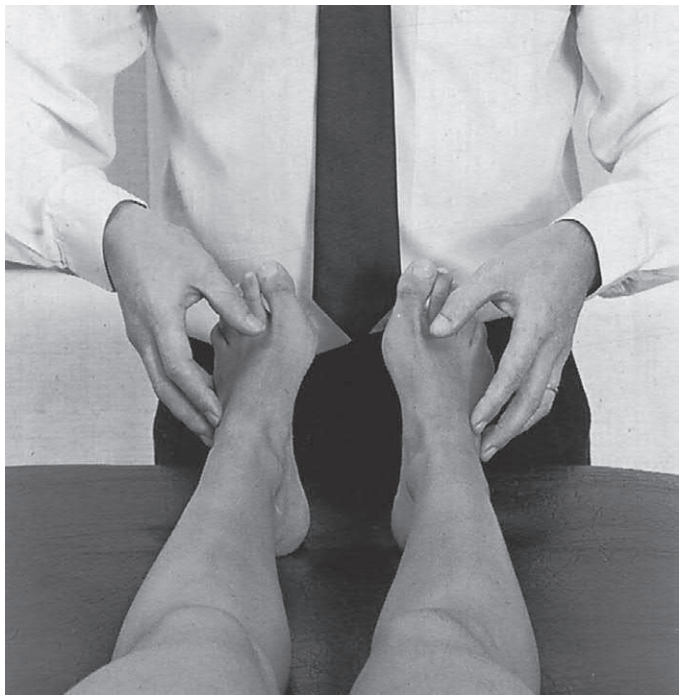


Fig 36.20 • Testing for sensory conduction.

- Backache in the upper lumbar area.
- Complaint of weakness in both legs.
- Paraesthesia in both feet.
- Backache with a spastic gait.

The reverse end of the reflex hammer is run firmly over the plantar surface of the foot from the calcaneus along the lateral

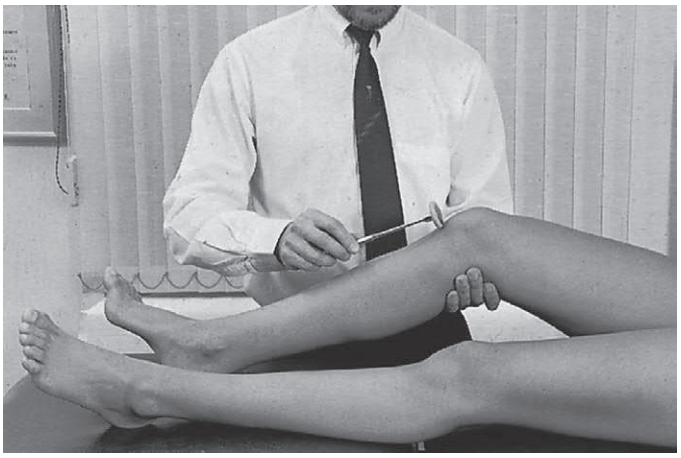


Fig 36.21 • Knee reflex test.

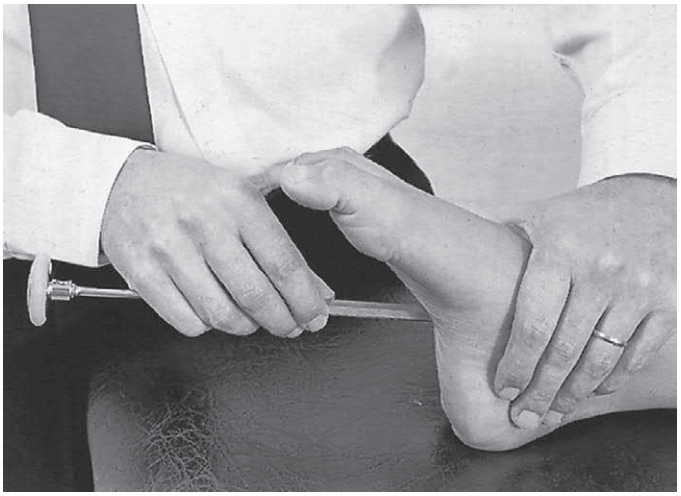


Fig 36.22 • Testing for Babinski's sign.

border to the forefoot, ending at the ball of the great toe (Fig. 36.22). In a positive reaction the great toe extends, while the other toes plantarflex and splay (positive Babinski's sign).

In a negative reaction the toes either do not move at all or flex uniformly (negative Babinski's sign).

Warning

Supine

- A positive sacroiliac distraction test
- Acute lumbago without any dural signs
- Buttock sign
- Discrepancy between trunk flexion and straight leg raise
- Signs of interference with conduction of more than one root
- L2 root palsy
- Bilateral nerve root palsy
- Complete paralysis
- A significantly warmer foot on the affected side

Examination of the circulation

This is optional and depends on the history and findings at inspection.

If intermittent claudication is suspected, the pulses of the femoral, posterior tibial and dorsalis pedis arteries should be felt (Fig. 36.23). If the pulse is diminished or absent at the femoral artery, the diagnosis is almost a certainty. Absence of a pulse at the ankle often exists without any vascular disorder.

In cases of claudication of the buttock, the internal iliac artery may be blocked on its own and the pulse of the femoral artery is normal (see Section 12).

Oedema in one foot is suggestive of venous thrombosis.

Local heat is found in patients with inflamed varices and in osteitis deformans of the tibia dorsalis. Neoplasm at the upper two lumbar levels may interfere with the sympathetic nerves; if so, the foot on the affected side is significantly warmer than the other.

Examination in the prone-lying position

This starts with the ankle reflex test.

Ankle reflex test

The foot is raised with one hand. Then all the slack of the plantiflexors is taken up by the little finger pushing the foot into dorsiflexion, before striking the Achilles tendon (Fig. 36.24).

This reflex is diminished or absent unilaterally in fifth lumbar and first or second sacral root palsy. It is well known that, once lost, this reflex does not return in about half the cases. Hence, in a new attack of lumbago, the absence of the ankle jerk does not confirm recently impaired root conduction.

Absence of the reflex on both sides may have no significance but is one of the findings in tabes dorsalis, malignant disease and spondylolisthesis, when the emerging nerve roots are involved bilaterally.

Passive knee flexion

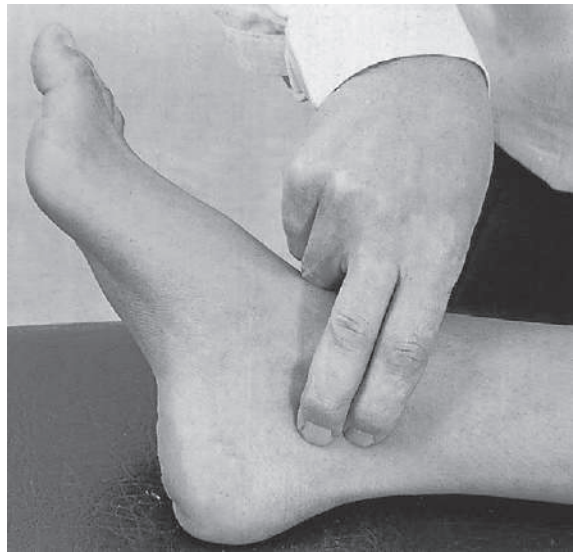
Next, passive knee flexion is performed to test the mobility of the third lumbar root (Fig. 36.25).[38]

In posterolateral disc protrusions at this level, flexion of the knee is painful at its extreme and occasionally limited in range. Pain is felt in the back and/or the anterior part of the upper leg, depending on whether the test provokes a discodural or a discoradicular interaction. Wasserman described the manœuvre in 1918. It was performed in soldiers with anterior thigh and leg pain where the SLR test was negative.[39]

A false-positive femoral stretch test has also been reported in osteoarthritic hip joints, diabetic neuropathy, anticoagulant medication, retroperitoneal haemorrhage and ruptured aortic aneurysm.[40,41]

Tightness of the rectus femoris may also influence this movement. The patient experiences anterior thigh pain, which

(a)



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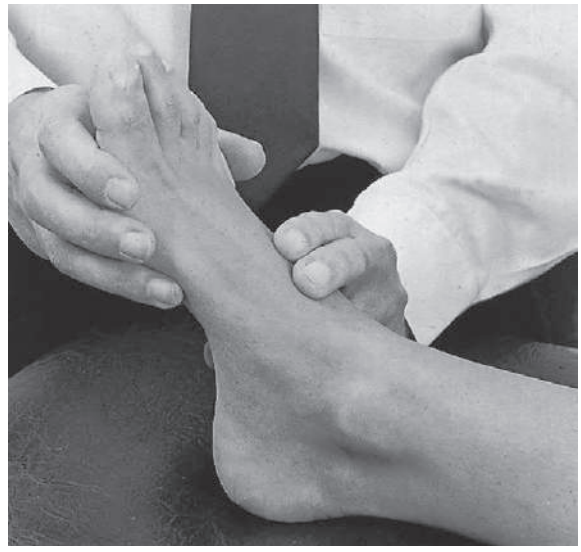


(c)

Fig 36.23 • Feeling the pulses of (a) femoral, (b) posterior tibial and (c) dorsalis pedis arteries.

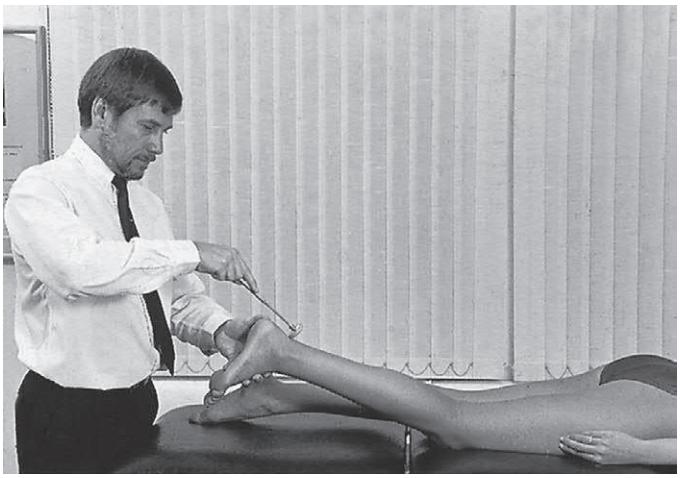


Fig 36.24 • Ankle reflex test.

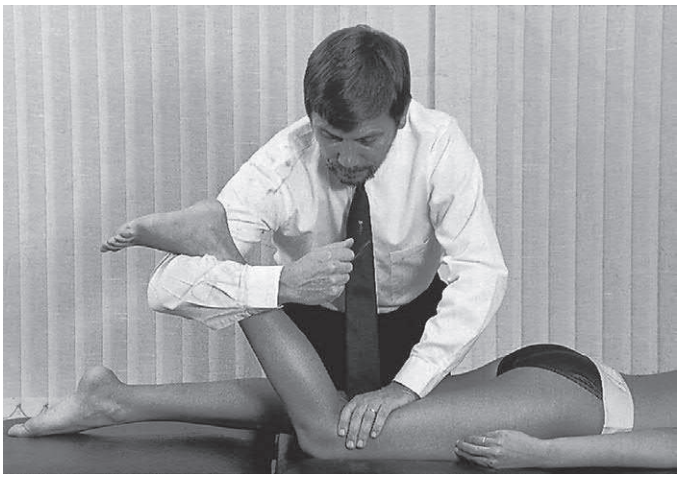


Fig 36.26 • Resisted extension of the knee.

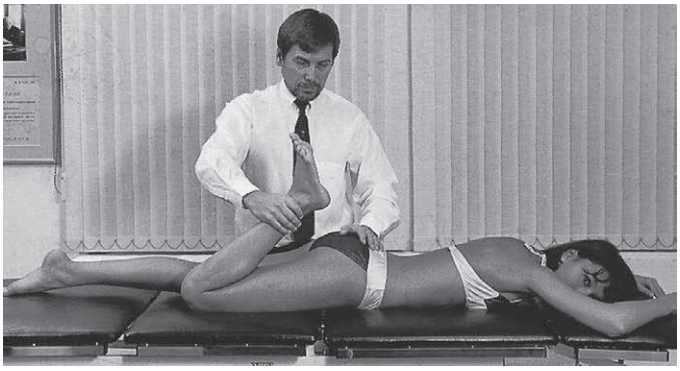


Fig 36.25 • Passive knee flexion.

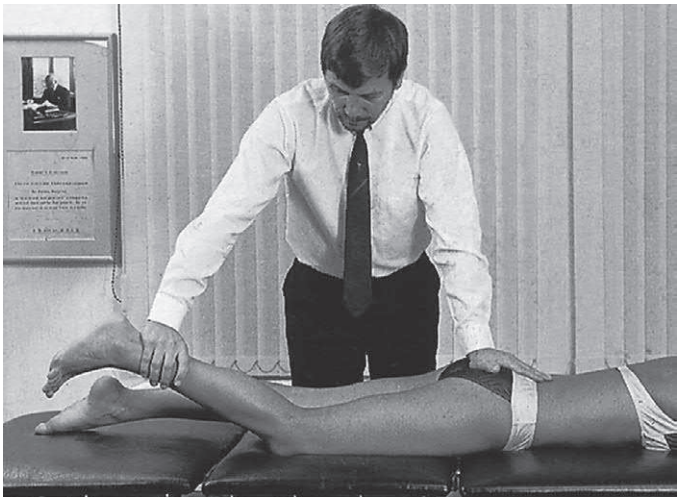


Fig 36.27 • Resisted flexion of the knee.

must be differentiated from the painful reaction in an L3 nerve root entrapment. If the non-painful side is flexed, the degree of pain and limitation can be compared and in this way distinguished from the defensive reflex muscle spasm in an L3 root involvement.

During this test, the pull of the rectus femoris on the anterior inferior spine of the ilium forces the lumbar spine into extension, which may also provoke lower lumbar pain.

Crossed femoral stretching test

This test is considered positive when flexion of the knee on the asymptomatic side reproduces the symptoms on the affected side. It is hypothesized to be a valid manœuvre to assist in the diagnosis of symptomatic disc herniation.[42] However, it is a far less constant sign, and in most third lumbar root lesions stretching is painful but not limited.

Testing motor conduction

There are three tests in the prone-lying position.

Resisted extension of the knee

This tests the L3 nerve root. The examiner tries to resist attempted extension with his or her flexed elbow, at the same time fixing the upper leg strongly just above the knee (Fig. 36.26). The normal patient is stronger than the examiner. Gross weakness goes together with weakness of the psoas, which is partly supplied by the same nerve root. If weakness is bilateral, spinal neoplasm or myopathy should be suspected.

Painful weakness indicates partial rupture of the quadriceps and, in more obvious instances, a fractured patella.

Resisted flexion of the knee

This tests the S1 and S2 nerve roots. The examiner resists attempted flexion at the same time as stabilizing the pelvis (Fig. 36.27). Normally, the examiner is *just* stronger than the patient. Weakness indicates a lesion of the first or second sacral root.

Painful weakness indicates partial rupture of one of the hamstrings.

Testing the buttock muscles

The S1 and S2 nerve roots are tested by asking the patient to contract the buttocks strongly (Fig. 36.28). Weakness is

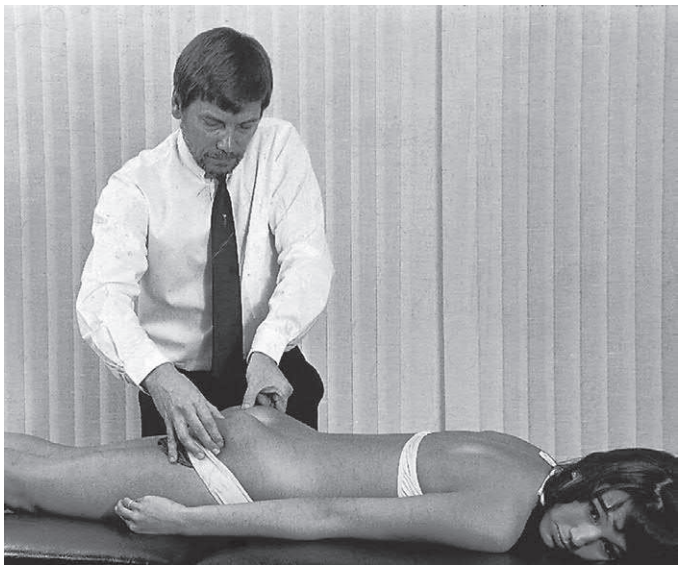


Fig 36.28 • Testing the buttock muscles.

demonstrated by decreased prominence on the affected side and loss of tone on palpation.

Palpation

To detect irregularities of the lumbar spinous processes

The index and middle fingers run quickly down the spine feeling for any *abnormal projections* (Fig. 36.29). If one is found, it may indicate wedging of a vertebral body or complete loss of two adjacent disc spaces. It should also prompt suspicion of bone erosion of a vertebral body (osteoporosis, tuberculous caries, secondary deposit or an old fracture), which requires radiography.

The finding of a *shelf*, most often at the interspace of L4–L5, or loss of a shelf palpable on examination in the standing position, indicates spondylolisthesis.

Pressure towards extension

Next a series of pressures towards extension are exerted to detect the level of the lesion. Starting at the sacrum, each lumbar segment is 'sprung' in turn, and it should be noted at which level pain and muscle guarding are most provoked (Fig. 36.30).

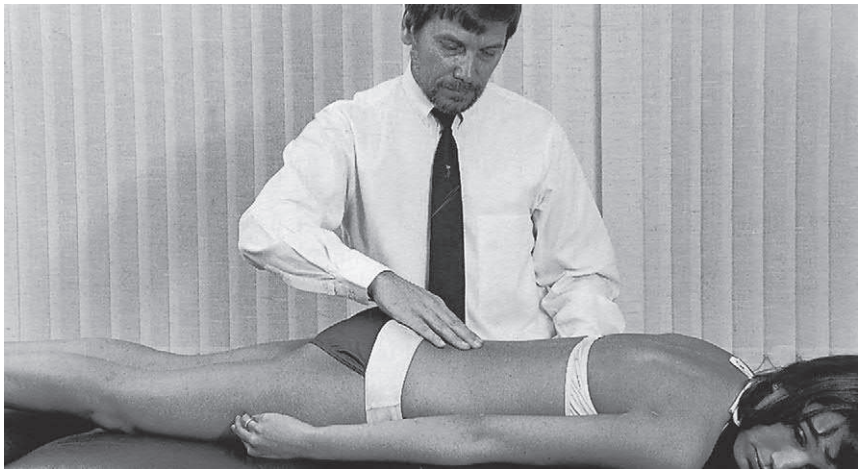


Fig 36.29 • Palpation for irregularities of the spinous processes.

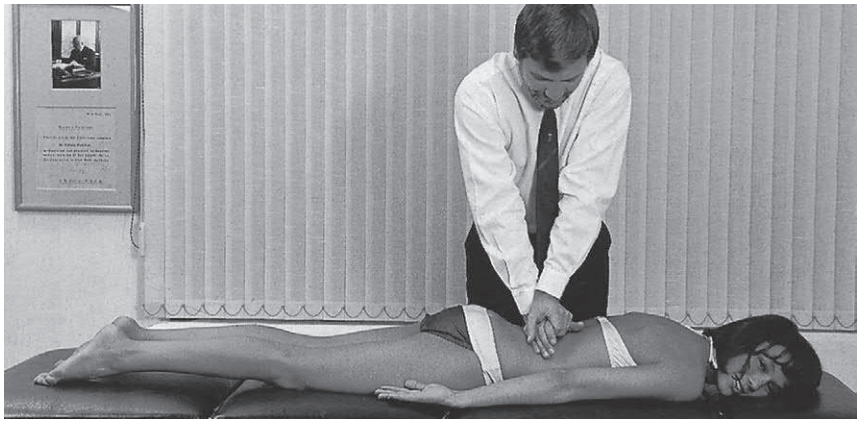


Fig 36.30 • Pressures towards extension.

In mechanical disorders, the expectation is that the painful level is in the *lower* lumbar area. If the *upper* lumbar area is the site of pain, the clinician must be on the alert. Serious disease is to be expected and further examination is indicated.

The test also serves to check the end-feel. In the young, the end-feel should be elastic, whereas in elderly persons it is hard because of spondylosis.

A hard end-feel in patients under 40 years old suggests ankylosing spondylitis.

Functional examination and palpation are summarized in Table 36.3.

Warning

Prone-lying

- An abnormal projection of one of the spinous processes.
- Pain and muscle guarding provoked by pressure towards extension in the upper lumbar area.
- A hard end-feel in patients under 40.

Accessory tests

Finally, the history will sometimes lead to the performance of resisted movements. These are particularly desirable when:

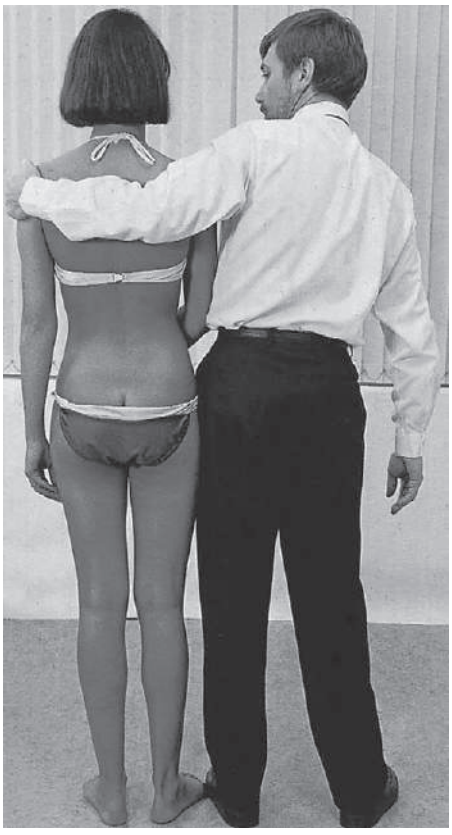
- *A fracture of the last rib or a transverse process is suspected*: local pain following unilateral injury to the lumbar spine. Pain is generated on side flexion away and resisted side flexion towards the painful site. Radiography confirms the diagnosis.
- *A muscle sprain is suspected*: at the lumbar region this scarcely ever occurs. It is only the combination of painful resisted extension with painless passive extension that directs attention to the muscle. However, in some (acute) disc lesions, resisted movements may also provoke pain because of increased compression of the joint.
- *The patient is suspected of psychogenic symptoms*: pain that is provoked by resisted movements is likely to occur here, because these patients tend to equate effort with pain.

Three resisted movements can be performed: (Fig. 36.31).

- Prone lying: resisted extension
- Standing: resisted side flexion.
- Side lying: resisted side flexion.

Trunk extension in the prone position is resisted by placing one hand on the upper thorax posteriorly, the other on the back of the knees.

On resisted side flexion with the patient standing, the examiner opposes the movement by applying his hip to the patient's, grasping the latter's far shoulder. Then the patient is asked to bend away from the examiner.



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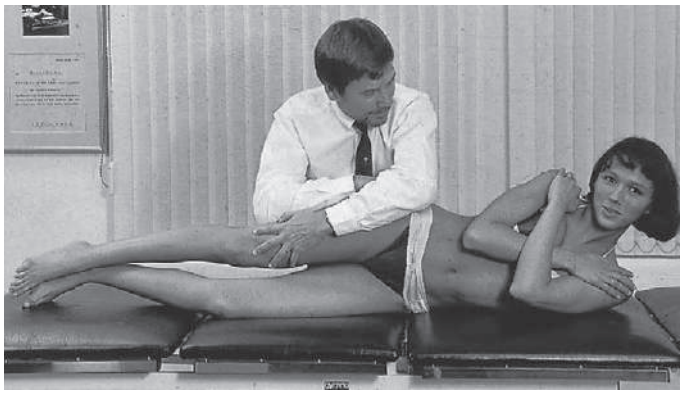

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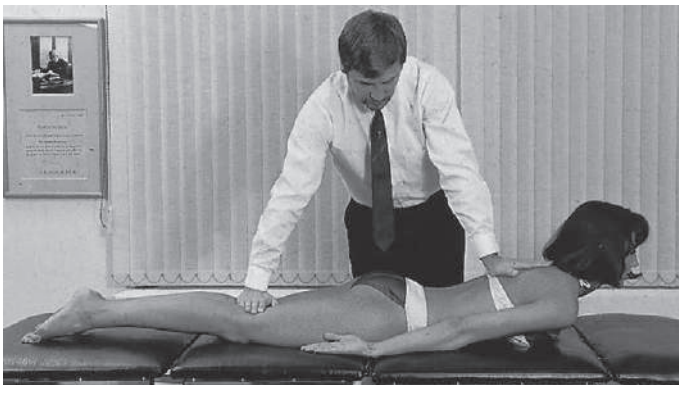

(c)

Fig 36.31 • Accessory tests: resisted side flexion (a), (b); resisted trunk extension (c).

Table 36.3 Summary of functional examination and palpation

	Test	Testing
Standing		
Inspection	Deviation	In the sagittal plane In the frontal plane (6 types)
	Pain	Level Side
	Irregularities	Bone Skin Muscle
Lumbar movements	Extension Side flexion Flexion (+ neck flexion)	Range, pain, painful arc, deviation Range, pain, painful arc, deviation Range, pain, painful arc, deviation
Motor conduction	Standing on tiptoe	S1 and S2
Supine		
Sacroiliac joint	Distraction	Inflammation of sacroiliac joint
Hip joint	Flexion External rotation Internal rotation	Lesions of the hip joint and buttock Lesions of the hip joint and buttock Lesions of the hip joint and buttock
Mobility of dura mater and nerve roots L4–S2	Straight leg raising	Range Pain Painful arc Crossed straight leg raising Neck flexion 'Buttock sign'
Motor conduction	Resisted flexion of hip joint Resisted dorsiflexion of foot Resisted extension of big toe Resisted eversion of foot	L2 and L3 L4 L4 and L5 L5 and S1
Sensory conduction	Front of thigh Front of thigh, inner side of lower leg Big toe Big toe and adjacent toes Outer border of foot and two outer toes Sole of heel	L2 and L3 L3 L4 L5 S1 S2
Knee reflex	Patellar tendon	L3
Plantar reflex	Plantar surface of foot	Spinal cord
Ankle reflex	Achilles tendon	L5, S1 and S2
Prone		
Mobility of L3 nerve root	Passive flexion of knee	Range Pain
Motor conduction	Resisted extension of knee Resisted flexion of knee Contraction of buttock muscles	L3 S1 and S2 S1 and S2
Palpation	Lumbar spinous processes	Irregularities
Extension pressure	Sacrum and lumbar vertebrae	Pain End-feel

In the side-lying position, with the body weight now off the joint, the patient crosses the arms in front of the chest; the examiner steadies the patient's thighs during this movement. Then the patient is asked to lift the thorax just off the couch.

Epidural local anaesthesia

Cases are sometimes met in which neither the history nor the physical signs clearly indicate one particular lesion. Although examination will show that the symptoms arise from a mechanical disorder, it may be uncertain whether a disc lesion, a disorder of the posterior structures or a sacroiliac lesion is present. The use of local anaesthesia may then be helpful.

A weak solution of procaine can be introduced epidurally via the sacral route. The solution desensitizes the dura mater and the dural investments of the nerve roots. In a discodural or discoradicular interaction, the pain will cease for the duration of the anaesthesia. In addition, epidural local anaesthesia induced for diagnostic purposes may also yield permanent improvement (see p. 566).

Alternatively, if a disorder of the posterior lumbar elements (facet or ligaments) is probable, local anaesthesia of the suspected structure should be performed. Five minutes after infiltration, the patient is asked to undertake the movements that were previously painful. If these no longer cause distress, the correct area has clearly been chosen and the diagnosis is confirmed. The infiltration must be precise, however, as false inferences may be drawn, a fact that is especially true for infiltration of the facet joints. It has recently been shown that, when relatively large volumes are injected into the facet joint, some extravasation occurs through the thin anterior capsule into the epidural space.[43] Facet arthrography confirms that epidural extravasation of dye takes place when more than 2 mL is injected in the facet joint.[44] More than this amount of local anaesthetic injected into a facet joint may thus result in an unintentional epidural block.

Technical investigations

Plain lumbar radiography

Most observations made on plain radiographs are of little or no value.[45,46] In particular, congenital anomalies, such as transitional vertebra, occult spina bifida and asymmetric facet orientation, are not clinically significant.[47]

It has also been repeatedly shown that there is no relationship between clinical symptoms and radiological changes associated with degeneration.[48–55] The poor diagnostic value of radiographs in patients with low back pain can also be appreciated from the observation that radiographs of the individual with symptoms remain unchanged over time, despite the fact that the symptoms come and go. Because radiographs do not show the position of cartilage, they are of no value in diagnosing current disc lesions either. Radiographs therefore remain a very poor method of indicating causes of past, present or future low back pain.[56]

It is common practice to order routine radiographs to reduce the risk of missing serious disorders. The possibility is in fact slight, and one series of 68 000 spinal radiographs found only 1 in 2500 with serious disorders not suspected clinically.[57] In contrast, it should be remembered that serious disease does not always show up immediately on a radiograph – about 30% of the osseous mass of a bone must be destroyed before a lesion is radiologically evident[58] – so that too much reliance on radiographic appearances can give a false feeling of security. In the short term, it is wiser and safer to rely on the history and the clinical examination: if symptoms and signs warrant (i.e. warning signs are found), the patient should be assumed to have a serious disease and, rather than manipulative treatment being undertaken, specific tests should be performed.

A further deterrent in radiographic evaluation of the lumbar spine, and one that it is important to remember, is that it is the single largest source of gonadal irradiation.[59] The total gonadal dose from a five-view lumbar spine examination is 75 millirads in men and 382 millirads in women[60] – unnecessary[61] oblique views are responsible for 65% of the irradiation dose.[62] Hall[63] estimated that the gonadal dose in women, when only a three-view examination is made, is equal to the dose of plain radiographs of the chest performed daily over a period of 6 years.

Radiographic 'labels' may confuse or bias patients and should never be transmitted to them as statements of disease because there is no evident correlation between radiographic appearances and the actual complaints. To patients, a statement such as 'your back shows a marked degree of arthrosis' means that they are incurable.[64] It implies a back that is crumbling like mouldy cheese: the situation is definite, incurable and hopeless. The diagnosis of 'osteoarthrosis' condemns the sufferer, and many patients become deeply depressed when they hear that the back is 'worn out'. An anxious or overconcerned patient will then suffer more from the idea that the back is beyond redemption and that no proper treatment for 'osteoarthrosis' exists, than he or she might from the back pain that is experienced. Technical investigation has become a problem rather than an aid. The radiograph does not help the patient; rather it may increase disability.[65]

Conclusion

Plain radiographs of the lumbar spine have a very limited value. They have a low specificity, require a high degree of gonadal irradiation in females, are relatively cost-ineffective[66] and carry a substantial risk of negative psychological repercussions in suggestible patients. The clinician should always be very careful and restrictive in using radiography. The results should be interpreted in the context of the normal ageing spine. A negative radiographic examination does not always exclude serious disease.

Other imaging studies

Ever since 1921, attempts have been made to increase the contrast in imaging between the various structures in the spine. Initially, gas was introduced into the subarachnoid space.[67] Next, positive contrast myelography with iodized oil solutions

was begun in 1922.[68] Gross toxic effects from this, including severe arachnoiditis and late meningeal disorders, led to the development of safer, water-soluble contrast agents.[69]

From the early 1940s, lumbar discs have been injected with contrast material in order to detect disc degenerations and disc ruptures.[70] However, discography has always been controversial. In the past decade, several authorities have seriously questioned its use: it is painful, expensive and without diagnostic value[71]; the sensitivity, specificity and predictive value are not as good as in myelography, CT and magnetic resonance imaging (MRI)[72]; and the risk of post-discography discitis is high.[73,74] Discographic studies are therefore considered obsolete.[75,76]

Recently, high-resolution CT and MRI have in many ways revolutionized the diagnosis of spinal disorders. These techniques not only visualize the bony anatomy and pathological features of the spine but also can confirm disc displacements or bony stenosis. They have therefore become the foundation of diagnostic imaging of the spine. Unfortunately, for a variety of reasons, the ability to visualize spinal disorders has not solved the diagnostic problems or the therapeutic dilemmas.

CT and MRI scans are highly sensitive but relatively unselective. In other words, these techniques have a very high prevalence of abnormal findings in images of asymptomatic individuals: postmortem studies show the existence of large, symptomless disc protrusions in almost 40% of cadavers[77]; myelograms in asymptomatic patients show defects in 37%[78]; and CT scans in subjects over 40 years of age show abnormality in more than 50%.[79] Numerous MRI studies have also demonstrated the high incidence of disc degeneration in asymptomatic patients.[80–86] Although additional imaging techniques are strongly indicated for the evaluation of patients presenting with warning symptoms and signs suggestive of neoplastic or infectious disorders, they have only limited value in the diagnosis of mechanical disorders of the lumbar spine.[87] It cannot be stressed enough that excessive reliance on diagnostic studies without precise clinical correlation can lead to erroneous (and disastrous) treatment. Diagnosis of spinal disorders depends on a detailed history and physical examination, as does treatment. The increased tendency that has developed over recent years to recommend surgery in the presence of a positive CT scan is a major error. Given the high number of asymptomatic disc protrusions, many patients will go forward to an unnecessary operation. Boden puts it well when he states: 'To get a MRI scan to see *if there is anything wrong with the spine* is usually the beginning of a very dangerous process.'[88] The presence of a disc protrusion and its size are unimportant; it is the impact of the protrusion on the surrounding pain-sensitive structures that determines management. Imaging cannot usually distinguish symptomatic from asymptomatic disc herniation, since it is usually unable to detect the degree of inflammation, the degree of pain or the functional impact on a nerve. Clinical examination can do so, provided it is intelligently interpreted.

Electrodiagnosis

The use of electromyography (EMG) was introduced 50 years ago.[89] Refinement of the technique, together with additional testing procedures (electrodiagnosis), now makes it possible to analyse and document nerve root dysfunction (level, degree and chronicity).[90] Though it is the only laboratory study that directly assesses the physiological integrity of the roots, the test will not be helpful in patients with so-called non-compressive radiculopathy[91]: the protrusion compresses only the dural nerve root sleeve and not the fibres.

Also, EMG examination is very time-dependent. Studies may be falsely negative if they are performed too early or too late in the course of sciatica. When it is done before sufficient fibrillation potentials have developed throughout the muscle, the result will be normal. This will also be the case in chronic radiculopathies, when muscles have been completely reinnervated.

In addition, the cause of the process leading to denervation cannot be determined. Furthermore, in severe compressive neuropathies, (serious) functional loss is easily detected during a proper clinical examination.

These factors mean that electrodiagnosis has very low specificity and sensitivity.[92] This diagnostic technique is therefore not important in lumbar disorders, except when objective documentation of the physiological integrity of the lumbar roots is required, which is sometimes the case when there are medicolegal implications.

Conclusion

- Additional imaging modalities are highly sensitive and relatively unselective.
- There is a high prevalence of abnormal findings on images of asymptomatic individuals.
- CT and MRI techniques are extremely important in the evaluation of patients with symptoms and signs of non-mechanical (neoplastic or infectious) disorders.
- In mechanical lesions, excessive reliance on diagnostic studies without precise clinical correlation can lead to erroneous (surgical) treatment.

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Lumbar instability

37

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Although lumbar instability is considered to be responsible for the majority of chronic or recurrent backaches, the word 'instability' is still poorly defined. A number of different definitions exist, but as yet there are no clear and validated clinical features by which instability might be diagnosed. It is also not clear how instability as such might set up pain and disability.

It is generally accepted that instability does not cause trouble in itself, but predisposes to other conditions such as (recurrent) disc displacements, strain of the posterior ligaments and the zygapophyseal joints, and nerve root entrapment.

Definitions

At the most simple level, instability is a lack of stability, a condition in which application of a small load causes an inordinately large, perhaps catastrophic displacement.[1] This is also the description given by the American Academy of Orthopaedic Surgeons, who state: 'Segmental instability is an abnormal response to applied loads, characterized by motion in motion segments beyond normal constraints.'[2]

A biomechanically more accurate definition of segmental instability, using a 'neutral zone' concept, has been proposed by Panjabi. The neutral zone concept is based on the observation that the total range of motion (ROM) of a spinal motion segment may be divided into two zones: a neutral zone and an elastic zone (Fig. 37.1).[3] The neutral zone is the initial portion of the ROM during which spinal motion is produced against minimal internal resistance. The elastic portion of the ROM is the portion nearer to the end-range of movement that is produced against substantial internal resistance. Segmental instability is thus defined as a decrease in the capacity of the stabilizing system of the spine to maintain the spinal neutral zones within physiological limits in order to prevent neurological deficit, major deformity and/or incapacitating pain.[4] This definition describes joints that, early in range and under minor loads, may exhibit excessive displacement.

The clinical definition of instability is: 'a condition in which the clinical status of a patient with low back problems evolves, with the least provocation, from the mildly symptomatic to the severe episode'.[5] Others consider instability to exist only when sudden aberrant motions such as a visible slip or catch are observed during active movements of the lumbar spine or when a change in the relative position of adjacent vertebrae is detected by palpation performed with the patient in a standing position versus palpation performed with the patient in a prone position.[6]

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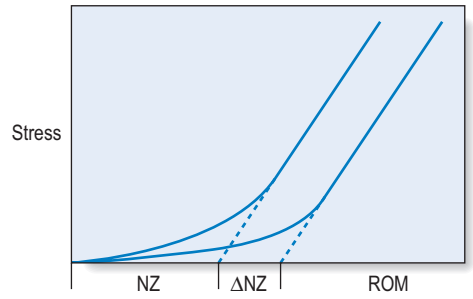

Fig 37.1 • In segmental instability, the stabilizing system is unable to maintain the spinal neutral zone (NZ) within physiological limits. ROM, range of motion.

Anatomy

In order to be clinically useful, the structures that are responsible for instability must be specified. The stabilizing system of the spine can be conceptualized as consisting of passive (inert) and active (contractile) parts and a neural control system.

Inert structures

The passive subsystem consists primarily of the vertebral bodies, discs, zygapophyseal joints and joint capsules, and spinal ligaments. The passive subsystem plays its most important stabilizing role in the elastic zone of spinal ROM (i.e. near end-range),[7] and numerous studies have been conducted that demonstrate the relative contributions of passive structures to segmental stability.

The posterior ligaments of the spine (interspinous and supraspinous ligaments), along with the zygapophyseal joints and joint capsules and the intervertebral discs, are the most important stabilizing structures when the spine moves into flexion.[8–11] End-range extension is stabilized primarily by the anterior longitudinal ligament, the anterior aspect of the annulus fibrosus and the zygapophyseal joints.[12,13] Rotational movements of the lumbar spine are stabilized mostly by the intervertebral discs, the zygapophyseal joints and, for the L4–L5 and L5–S1 segments, the iliolumbar ligaments too.[14]

Injury to the inert stabilizing system may have important implications for spinal stability. Intervertebral disc degeneration, weakening of the posterior longitudinal ligaments and early degeneration of the facet joints may increase the size of the neutral zone, increasing demands on the contractile subsystem to avoid the development of segmental instability.[3]

Contractile structures

The active subsystem of the spinal stabilizing system includes the spinal muscles and tendons and the thoracolumbar fascia; these contribute to stability in two ways. The first and lesser mechanism is to pull directly against the threatened displacement (which is, of course, not possible if the latter is a fragment of disc). The second, more important contribution is indirect: whenever the muscles contract, they exert compressive loads on the spine, which in turn achieves a stabilizing effect. In other words, by compressing joints in a neutral position, muscles may make it less easy for joints and discs to move.

During recent decades, a variety of studies have documented the stabilizing effect of muscles on the lumbar spine.[15–18] The lumbar erector spinae muscle group provides most of the extensor force required for many lifting tasks.[19] Rotation is produced primarily by the oblique abdominal muscles. The multifidus muscle seems to be able to exert some segmental control and is therefore proposed to function as a stabilizer during lifting and rotational movements of the lumbar spine.[20] The role of the oblique abdominal and transversus abdominis muscles in spinal stability has been the subject of much debate. The abdominals have been thought to play a stabilizing role, either by increasing intra-abdominal pressure or by creating tension in the lumbodorsal fascia.[21]

Box 37.1

Lumbar segmental instabilities: classification

1. Fractures and fracture dislocations
2. Infections involving anterior columns
3. Primary and metastatic neoplasms
4. Spondylolisthesis in children
5. Degenerative instabilities
6. (Progressive scoliosis in children)

Neuromuscular control

The neural control system may also play an important part in stabilization of the spine. Panjabi describes the stability system as being composed of an inert spinal column, the spinal muscles and a control unit.[22] In this model, changes in spinal balance resulting from position and load are monitored by transducers embedded in the ligaments that relay information to the control unit. When conditions that challenge spine stability are detected, the control unit activates the appropriate muscles to protect or restore stability, or to avoid instability. Evidence for this hypothesis is found in studies showing that patients with low back pain (LBP) often have persistent deficits in neuromuscular control.[23,24] This hypothesis was further supported by a recent electromyographic study demonstrating that a primary reflex arc exists from mechanoreceptors in the supraspinous ligament to the multifidus muscles. Such a reflex arc could be triggered by application of loads to the isolated supraspinous ligament, which in turn initiates activity of the multifidus muscles at the level of ligament deformation, as well as one level above or below.[25]

Classification of lumbar instability

The major categories of segmental instability are shown in Box 37.1.[26] Tumours, infections and trauma are beyond controversy. They produce mechanical weakening of the anterior

columns and can be diagnosed by medical imaging and by biopsy. Spondylolisthesis is a more controversial category. The condition is rarely progressive in teenagers or adults and can therefore be considered as stable in these age groups.[27] However, it has been suggested that concurrent severe disc degeneration at the level of listhesis may lead to progression of slip and convert an asymptomatic and stable lesion into a symptomatic one.[28]

More difficulties arise with respect to so-called 'degenerative instability'. The ageing of the lumbar spine has been discussed thoroughly (see Ch. 32). Grossly, it occurs in three sequential phases: dysfunction, instability and restabilization.[29]

During the early *phase of degeneration* (dysfunction), small annular tears and early nuclear degeneration appear in the disc, and ligamentous strains develop in the posterior ligaments and in the capsules of the zygapophyseal joints. The *unstable phase* includes reduction of disc height, gross morphological changes in the disc, and laxity of the spinal ligaments and facet joints. These changes lead to an increased and abnormal range of movement and to increased liability to disc displacements. During the *restabilization phase*, further physiological changes in the disc, such as increased collagen and decreased water content, together with the development of spinal osteophytes and gross osteoarthrosis of the zygapophyseal joints, result in increased stiffness of the spine and consequent stabilization (Fig. 37.2).

Biomechanical studies, both *in vivo* and *in vitro*, have confirmed this hypothesis: loss of stiffness, accompanied by annular tears or even nuclear disruption, has been reproduced in the laboratory by repetitive loading cycles which simulate normal human exposures.[30] In other experiments, load applications to degenerative segments have revealed loss of stiffness, sometimes with quite dramatic results.[31] However, difficulty remains in translating these anatomical and functional changes into clinical descriptions that could serve as a basis for diagnosis and treatment.

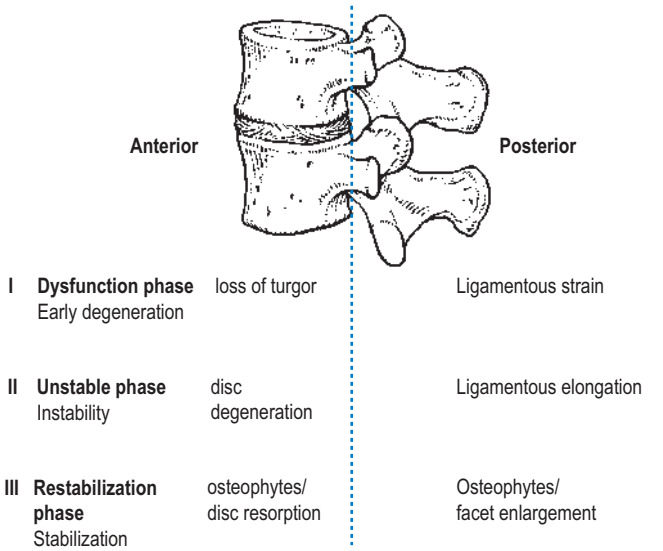

Fig 37.2 • The three sequential phases in the degenerative process of the lumbar spine.

A further classification system for degenerative lumbar instability (Box 37.2), based on a combination of history and radiographic findings, has been proposed.[32]

- *A primary instability* is one where there has been no prior intervention or treatment which might account for the development of the process.
- *A secondary instability* involves surgical destruction of one or more of the restraining elements of the spine. Secondary instabilities may develop after discectomies,[33] decompressive laminectomies, spinal fusions and chemonucleolysis.[34]

Rotational instability is still a hypothetical entity and so far normal radiological limits have not been identified.[35]

Translational instability is the most classic and best known of the primary degenerative instabilities. It is characterized by excessive anterior translation of a vertebra during flexion of the lumbar spine. At an early stage, it presents with disc space narrowing and traction spurs; later on, it represents degenerative spondylolisthesis. However, anterior translation is a normal component of flexion, and once again the difficulty that arises is setting a limit of normal translation. Many asymptomatic individuals exhibit anterior slips of more than 3 mm[36]; 4 mm of translation occurs in 20% of asymptomatic patients.[37]

Retrolisthesis develops when degeneration of the disc and the consequent decrease in disc height force the zygapophyseal joints into extension (see p. 442). Again, it has been shown that similar appearances occur in asymptomatic individuals.[38] Therefore, the simple detection of retrolisthesis on a radiograph is not an operational criterion for instability.

Instability and the clinical concept of mechanical lesions of the lumbar spine

Segmental instability is not painful in itself, and a patient with clear radiological signs of instability may be completely unaware

Box 37.2

Degenerative lumbar instabilities

Primary instabilities

- Axial rotational
- Translational
- Retrolisthetic
- (Scoliotic)

Secondary instabilities

- Post-disc excision
- Post-laminectomy
- Post-fusion
- Post-chemonucleolysis

of the condition. However, an unstable segment makes the spine more vulnerable to trauma; a forced and unguarded movement may be concentrated on the hypermobile segment and produce a posterior disc displacement. Repeated injuries may also lead to chronic irritation of posterior structures such as ligaments and zygapophyseal joints. An anterior or posterior shift of a vertebra may narrow the lateral recess to such a degree that the respective nerve roots become compressed.

Spinal instability is not a painful condition but may predispose to secondary lesions:

- Ligamentous sprain
- Recurrent discodural interactions
- Nerve root compression in a narrowed lateral recess.

Segmental instability and discodural interactions

It can be postulated that a hypermobile segment may predispose to recurrent disc displacements, leading to recurrent or chronic discodural interactions. Pain arises not from instability of the segment itself but from the instability of a fragment of disc lying within it.

The typical history is usually that of recurrent back pain, which begins either suddenly or gradually, depending on the consistency of the shifted fragment ('nuclear' or 'annular') (see Ch. 33, Dural concept). There are bouts of backache a few times a year, and between the attacks the patient is fit and the back is painless. However, the slightest sudden movement or unaccustomed posture leads to a new discal shift, resulting in a renewed discodural interaction and pain.

It is obvious that, in this case, not only should treatment address reduction of the displaced fragment of disc, but also treatment of the instability should be undertaken.

Segmental instability and ligamentous lesions

Postural ligamentous pain appears when normal ligaments are subjected to abnormal mechanical stresses (see Ch. 34, Ligamentous concept). This may occur during the dysfunction stage: some loss of turgor in the disc and the decrease in intervertebral joint space cause some laxity of the segment and an increase of the neutral zone. The facet joints override, with the upper articular processes sliding downwards over the lower. The joints adopt the extension position and the posterior capsules become overstretched. As instability proceeds, more tension is imposed on the ligaments and the facet joint capsules, leading to more postural ligamentous pain.

The patient is usually a young adult, who complains of diffuse backache with bilateral radiation over the lower back and the sacroiliac joints. The pain typically starts after maintaining a particular position for a long time and the intensity of the pain depends on the duration of this position. By contrast, there is absolutely no pain during activity or sports and all lumbar movements are free.

Segmental instability and the stenotic concept

Instability and the subsequent retrolisthesis may narrow the radicular canal and subsequently compress the nerve root (see Ch. 35, Stenotic concept). Usually, the process results from combined anterior pressure exerted by a buckled posterior longitudinal ligament and posterior compression of the superior articular process. The mechanism is as follows: considerable narrowing of the intervertebral disc space causes the posterior longitudinal ligament, which contains some fragments of remaining disc tissue, to bulge dorsally. This is especially the case in the standing or lordotic position. Because of the inclination of the facet joints, narrowing of the disc and increased laxity of the segment also result in retrolisthesis of the upper vertebra, which brings the nerve root in close contact with the tip of the anterior articular process of the vertebra below (Fig. 37.3).

The history is that of a middle-aged or elderly patient with unilateral or bilateral sciatica. The pain comes on during standing or walking and disappears on sitting or bending forwards.

Diagnosis of lumbar instability

The term 'segmental instability' is often misused and it has become fashionable to label any lumbar pain that is aggravated by movement as lumbar instability. The statement 'you suffer from lumbar instability' should be made sparingly, in that it is very hard to satisfy the criteria that justify use of this term. A diagnostic 'gold standard' for instability has not yet been identified.

Diagnosis is usually based on history, clinical examination, functional tests and imaging. Some elements can be found in the patient's history. It is believed that frequent recurrences of LBP precipitated by minimal perturbations, lateral shifts in prior episodes of LBP, short-term relief from manipulation and an improvement of symptoms with the use of a brace in previous episodes of LBP are confirmatory data for instability.[39]

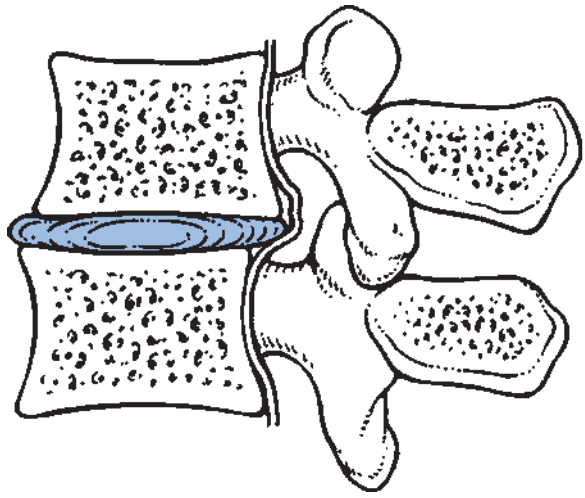

Fig 37.3 • Retrolisthesis narrows the nerve root canal.

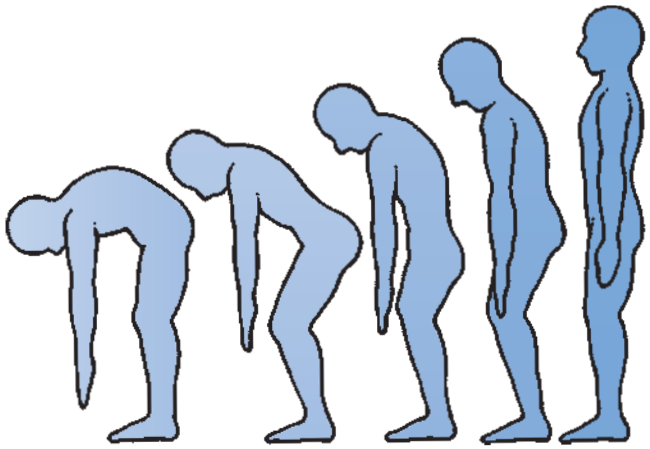

Fig 37.4 • Reversal of the normal spinal rhythm.

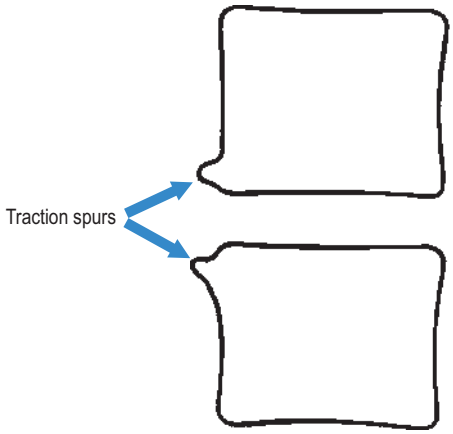

Fig 37.5 • Traction spurs.

Clinical observations

Some authors state that the palpation of increased mobility with passive intervertebral motion testing is indicative of instability.[40] The validity of these techniques, however, has never been demonstrated.[41] Others have proposed that aberrant motions such as the *instability catch* occurring during active ROM testing indicate instability.[6,42,43] The instability catch has been described as a sudden acceleration or deceleration of movement, or a movement occurring outside of the primary plane of motion (e.g. side bending or rotation occurring during flexion) and is proposed as an indication of segmental instability. However, this definition of an 'instability catch' is far too broad, because in the present description it also includes the common painful arc sign which indicates a momentary discodural interaction during movement (see p. 455).

In our opinion, MacNab's *reversal of the normal spinal rhythm* is much more characteristic of segmental instability.[44] In a normal lumbar–pelvic rhythm, there is a smoothly graded ratio between the degree of pelvic rotation and that of lumbar flattening. This rhythm may be disturbed in regaining the erect posture after forward flexion. In order to avoid putting an extension strain on the lumbar spine, the patient first slightly flexes the hips and knees in order to tuck the pelvis under the spine and then regains the erect position by straightening the legs (Fig. 37.4).

Radiological observations

Radiological measurements have been the most consistently reported method to establish instability, although again there is much controversy.

Disc space narrowing is a sign of questionable significance because this is a common age-related finding.

A second observation is the presence of traction spurs (Fig. 37.5), as described by MacNab.[45] The spur is considered to result from tensile stresses being applied to the outer annular fibres which attach to the vertebral body (see p. 444).[46]

The third observation is the presence of spinal malalignment. This radiological assessment is based on the early

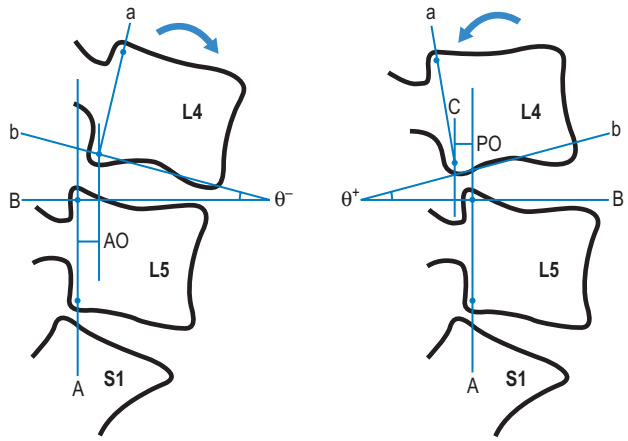

Fig 37.6 • Radiographic methods to evaluate anterior (AO) and posterior (PO) translation during flexion and extension (after Dupuis *et al*[50]).

observations of Knutsson, who defined instability as 3 mm or more of anterior translation measured between flexion and extension. However, as discussed earlier, there exists much debate about the upper limit of normal translations. Boden and Wiesel emphasized that any slip should be greater than 4 mm before instability could be considered.[36] Others concluded that a minimum of 4 mm of forward displacement was necessary at the L3–L4 and L4–L5 levels to define instability,[47] while at the L5–S1 level displacements of greater than 5 mm were necessary for accurate measurements (Fig. 37.6).[48,49] Furthermore, it has also been suggested that many instabilities do not occur at the extremes of flexion and extension, which is the usual technique utilized in routine radiographic studies.

Bracing

Lastly, it has been proposed that a trial of bracing should produce pain relief in a patient with instability. In general, the results have not been diagnostic, possibly because spinal braces usually produce little or no spinal immobilization.

Box 37.3

Diagnosis of lumbar segmental instability

History

- Chronic postural back pain and/or recurrent discodural interactions

Clinical examination

- Full range of movement
- No dural signs
- Reversal rhythm when regaining the erect posture from a flexed position

Radiography

- Traction spurs
- Anterior translation of more than 4 mm during functional radiographs

The diagnosis of segmental instability should be made sparingly. The features outlined in Box 37.3 may point to instability.

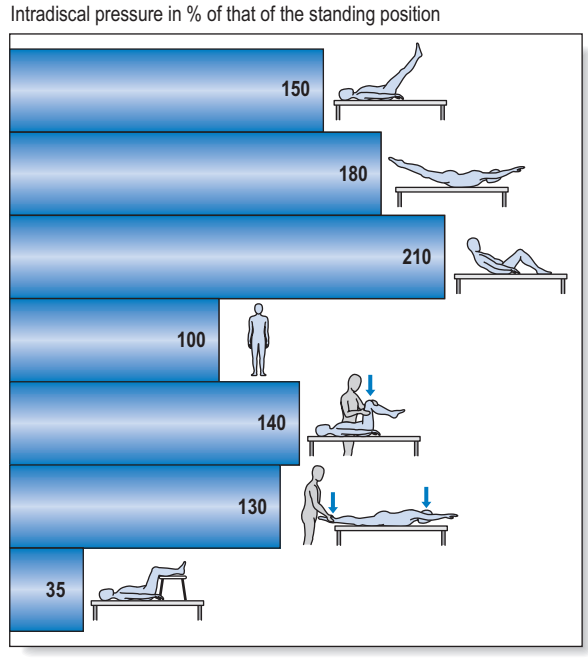

Fig 37.7 • Sit-up manœuvres may dangerously increase intradiscal pressure.

Treatment of lumbar instability

Patient education may be an important component in the treatment of patients with segmental instability. Education should, first of all, focus on avoiding loaded flexion movements, as they may create a posterior shift of the disc.[51] Patients should also be made aware of the importance of avoiding end-range positions of the lumbar spine because these overload the posterior passive stabilizing structures (see p. 583).

Physical therapy for segmental instability focuses on exercises designed (as is generally believed) to improve stability of the spine. During recent decades, all sorts of strengthening programmes have been designed for active stabilization of the unstable segment: the type of advocated treatment ranges from simple and intensive dynamic back extensor exercises to specific training of dynamic stability and segmental control of the spine.

As the lumbar erector spinae muscles are the primary source of extension torque for lifting tasks, strengthening of this muscle group has been advocated.[52] Intensive dynamic exercises for the extensors proved to be significantly superior to a regime of standard treatment of thermotherapy, massage and mild exercises in patients with recurrent LBP.[53–55] The abdominal muscles, particularly the transversus abdominis and oblique abdominals, have also been proposed as having an important role in stabilizing the spine by co-contracting in anticipation of an applied load. However, exercises proposed to address the abdominal muscles in an isolated manner usually involve some type of sit-up manœuvre that imposes dangerously high compressive and shear forces on the lumbar spine[56] and may provoke a posterior shift of the (unstable) disc (Fig. 37.7). Alternative techniques should therefore be applied when training these muscles.

Some authors strongly suggest that the transversus abdominis[57] and the multifidus muscles make a specific contribution to the stability of the lower spine,[58,59] and an exercise programme that proposes the retraining of the co-contraction pattern of the transversus abdominis and multifidus muscles has been described.[60] The exercise programme is based on training the patient to draw in the abdominal wall while isometrically contracting the multifidus muscle, and consists of three different levels:

- First, specific localized stabilization training is given. Lying prone, sitting and standing upright, the patient performs the isometric abdominal drawing-in manœuvre with co-contraction of the lumbar multifidus muscles.
- During the phase of general trunk stabilization, the co-contraction of the same muscles is carried out on all fours, and then elevating one arm forwards and/or the contralateral leg backwards, or on standing upright and elevating one arm forwards and/or bringing the contralateral leg backwards.
- Third, there is the stabilization training. Once accurate activation of the co-contraction pattern is achieved, training is given in functional movements, such as standing up from a sitting or lying position, bending forwards and backwards and turning. All daily activities are then integrated.

A significant result from a randomized trial has recently been reported comparing this exercise programme with one of general exercise (swimming, walking, gymnastic exercises) in a group of patients with chronic LBP.[61]

Despite the positive results with muscular training programmes, it remains difficult to understand how training of the lumbar and abdominal muscles can improve segmental

stability. Not only do the muscles (except for multifidus) have multisegmental attachments to the lumbar vertebrae, but also they are not very well oriented to resist displacements. Because they mainly run longitudinally, they can only resist sagittal rotation and are not able to resist anterior or posterior shears. However, whenever the muscles contract, and especially when they do this simultaneously, they exert a compressive load on the whole lumbar spine, as well as on the unstable segment. By compressing the joints, the muscles make it harder for the joints and for the intradiscal content to move.[62] The most important contribution of trained muscles to spinal stability may therefore be the creation of a rigid cylinder around the spine and increased stiffness.

It is important, however, for exercises to be prescribed as a means of prevention only after the actual problem – usually a discodural interaction – has been solved by manipulation, mobilization, traction or passive postural exercises.

Sclerosant treatment

Sclerosing injections given to the posterior ligaments are the conservative treatment of choice in segmental instability of the spine.

Therapy involves the injection of an irritant – phenol 2%, dextrose 25%, glycerol 15% – into the inter- and supraspinous ligaments, the posterior capsule of the facet joints and the deep part of the fascia lumborum at the affected level(s).

The infiltration produces a local inflammatory reaction, which is followed by increased proliferation of fibroblasts and the production of new collagen fibres. The final outcome is tightening, reinforcement and loss of normal elasticity of the connective tissue which decreases the mobility and increases the stability of the injected segments.[63–65]

The beneficial effect of this treatment method was recently shown in a double-blind controlled study that demonstrated a statistically significant difference between the active therapy group and those who received injections with a saline solution only.[66]

Technique

A series of infiltrations is made in all the dorsal ligaments at two consecutive motion segments (usually S1–L5–L4) and at the iliac insertions of the iliolumbar ligaments. Over 4 consecutive weeks, 3 mL of the solution, mixed with 1 mL of lidocaine 2%, is injected. The techniques are shown on page 918 but it is important to remember that, in order to steer clear of any vital structures, including those in the spinal canal, the injection should be made only when the tip of the needle touches bone.

- The first injection is at the interspinous and supraspinous ligaments.
- The second injection is given at the posterior capsules of the zygapophyseal joints.
- The third injection is given at the lateral aspects of the laminae, where the ligamentum flavum and the medial aspects of the deeper layer of the fascia lumborum merge.

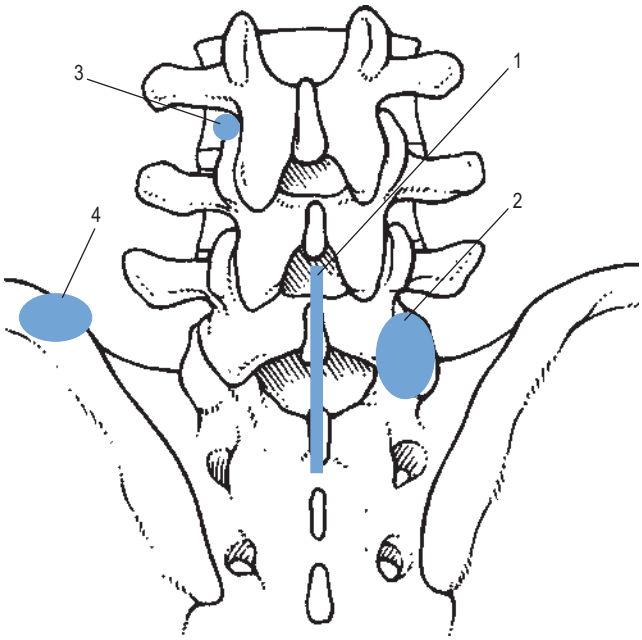

Fig 37.8 • Sclerosing injections in the treatment of segmental instability: 1, interspinous and supraspinous ligaments; 2, facet joint capsules; 3, deep layer of the fascia lumborum; 4, iliolumbar ligaments.

- The fourth injection aims at the insertion of the iliolumbar ligaments and the insertion of the thoracolumbar fascia on the posterior superior iliac spine (Fig. 37.8).

Surgery

The indications for spinal fusion in the treatment of degenerative instability are controversial. The basic problem lies, as discussed earlier, in the definition and the diagnosis of the disorder. However, despite the fact that indications for the procedure are uncertain, that costs and complication rates are higher than for other surgical procedures performed on the spine, and that long-term outcomes are uncertain, the rate of lumbar spinal fusion is increasing rapidly in the United States.[67,68] The rate of back surgery and especially of spinal fusion operations is at least 40% higher in the US than in any other country and is five times higher than in the UK.[69] Although there have been no randomized trials evaluating the effectiveness of lumbar fusion for spinal instability, the feeling remains that the operation should be reserved for patients with severe symptoms and radiographic evidence of excessive motion (greater than 5 mm translation or 10° of rotation) who fail to respond to a trial of non-surgical treatment.[70] The latter should consist of a combination of patient education, physical training and sclerosing injections.

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38

Mechanical disorders of the lumbar spine: differential diagnosis

Activity-related lumbar disorders have a multifactorial origin. Diagnostic precision is difficult, and imaging techniques usually have a relatively low specificity. Nevertheless, the clinician is required to make an accurate diagnosis, to choose an appropriate management strategy and to determine prognosis.

Therefore there is a need for a classification of spinal disorders based on simple clinical criteria. With the information gained from the history and examination, clinical syndromes can be defined and used as a basis for a classification which also embraces the concepts that have been described.

Syndromes
- Lumbago (Box 38.1)
- Backache (Box 38.2)
- Sciatica (Box 38.3).

Concepts (Fig. 38.1)
- Dural
- Ligamentous
- Stenotic.

Both syndromes and concepts have to be considered in the context of the normal changes in the ageing lumbar spine.

Box 38.1

Lumbago

Definition

- A sudden attack of severe and incapacitating backache

Mechanism

- Always caused by disc displacement, and thus comes entirely under the dural concept
- A large posterior shift of disc material compresses the dura mater: mechanism is dual; there is a discodural interaction

Symptoms

- Slow onset if the displacement is nuclear: *nuclear lumbago*
- Sudden onset if the shift is annular: *annular lumbago*
- *Articular*: twinges; severe pain during particular positions and movements, especially pain on sitting and on bending
- *Dural*: extrasegmental pain; pain on coughing and sneezing

Signs

- *Articular*: deviation; gross partial articular pattern
- *Dural*: painful neck flexion; limited straight leg raising

Natural history

- Spontaneous cure within 2 weeks in most cases

Treatment

- Hyperacute lumbago: epidural
- Annular lumbago: manipulation
- Nuclear lumbago: bed rest in psoas position; mobilizations–McKenzie techniques; no traction in the presence of 'twinges' or deviation

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Box 38.2

Backache

Definition

- Pain in the lumbar area, with or without radiation in a dural diffuse manner; in most cases pain does not radiate beyond the gluteal folds
- Pain can be acute or chronic, intermittent or constant

Mechanism

- *Acute and recurrent backache*: almost always caused by a discodural interaction, thus symptoms and signs are very similar to acute lumbago, although milder. Dural symptoms and signs are sometimes subtle or even absent. A clear non-capsular pattern or a painful arc during flexion is pathognomonic for a small central disc protrusion
- *Chronic backache*: caused by either a discodural interaction or a lesion of a posterior structure (facet or ligament)
- Differential diagnosis depends on the clinical picture

Symptoms

Discodural backache	Ligamentous postural syndrome	Ligamentous dysfunction syndrome
• Moment of onset is known • Acute: annular lesion • Gradual: nuclear lesion	• Pain comes on gradually • Moment of onset is known	• A hypertension trauma is often present
• Pain intensity is not constant but can fluctuate	• Pain appears during some postures; intensity depends on duration	• Pain is constant during the posture that provokes pain
• Pain is often unilateral but bilateral radiation is possible	• Bilateral and vague pain, seldom radiating beyond the glutei	• Localized and strictly unilateral pain, except in bilateral facet joint lesion • Central pain in lesion of the supra- and interspinous ligaments
• Localization changes: shifting pain	• No change in localization	• No change in localization
• Sometimes dural symptoms	• No dural symptoms	• No dural symptoms
• Twinges?	• Never twinges	• No twinges
• Pain is caused by movements	• Pain is caused by posture, increases with maintenance of posture and disappears during movement	• Pain is caused by posture and increases with maintenance of posture, sometimes with particular movements
• Sitting and bending are particularly painful	• Standing and strolling are particularly painful	• Movements and postures that increase the lumbar lordosis are painful

Signs

Discodural backache	Ligamentous postural syndrome	Ligamentous dysfunction syndrome
• Partial articular pattern	• Full range	• Full range, sometimes pain at the end of range
		• Facet lesions: convergent or divergent patterns
• Sometimes painful arc	• No painful arc	• No painful arc
• Sometimes positive dural signs; pain or limitation of straight leg raising; neck flexion increases the pain	• No dural signs	• No dural signs
• Improvement after manipulation/traction	• No improvement after manipulation/traction	• No improvement after manipulation/traction

Natural history

- Unpredictable: backache may recover spontaneously, but often does not. Chronic backache in particular shows no tendency to spontaneous cure

Treatment

Acute backache	Recurrent backache	Chronic backache
• Annular: manipulation and prevention (back school) • Nuclear: traction (epidurals)	• Annular: manipulation and sclerosing injections (or back school) • Nuclear: traction and sclerosing injections (or back school)	• Discodural: manipulation and traction • Bruised dura mater: epidurals • Self-reducing disc: back school and prevention; sclerosing injections • Postural ligamentous: sclerosing injections • Dysfunction of posterior structures: triamcinolone/sclerosing injections

Box 38.3

Sciatica

Definition

- Radicular pain resulting from compression of the dural investment of a nerve root
- Pain is limited to the dermatome of the root involved. If there is parenchymatous involvement, the pain is accompanied by paraesthesia, motor and/or sensory deficit

Mechanism

- Radicular compression can result either from a posterolateral disc herniation or from a narrowed lateral recess
- *Discoradicular* conflicts have a typical age of onset and typical natural history
- *Entrapment of the nerve root* in the lateral recess occurs in elderly patients; there is virtually no spontaneous evolution

Symptoms

Discoradicular interactions	Lateral recess stenosis
• Young to middle-aged patients	• Middle-aged to elderly patients
• Evolution in the pain localization	• No evolution of the symptoms

• Secondary posterolateral protrusions:
  • First backache
  • Then leg ache
	• No moving pain

• Primary posterolateral protrusions:
  • Pain starts in the calf
  • And moves upwards	

• Dural symptoms	• No dural symptoms
• Spontaneous recovery within 1 year in most cases	• No tendency to spontaneous recovery
• As a rule, sitting and bending are worst, although continuous pain is possible	• Pain on standing and walking, disappears during sitting and forward bending
	• Sometimes pain in prone-lying position

Signs

Discoradicular interactions	Lateral recess stenosis
• Partial articular pattern	• Full range or slight capsular pattern due to osteoarthrosis
• Limited flexion, sometimes with deviation	• Sometimes limited extension
	• Pain provocation after standing for a while
• Impaired nerve root mobility: • Positive straight leg raising or L3 stretch	• Normal mobility of the nerve roots; sometimes slight pain at end of straight leg raising
• Often signs of parenchymatous involvement/loss of motor and/or sensory functions, sluggish reflexes	• Seldom signs of parenchymatous involvement
• Diagnostic response after epidural local anaesthesia	• No diagnostic response after epidural local anaesthesia

Treatment

Discoradicular interactions	Lateral recess stenosis
• See p. 568	• Back school • Nerve root blocks • Surgery

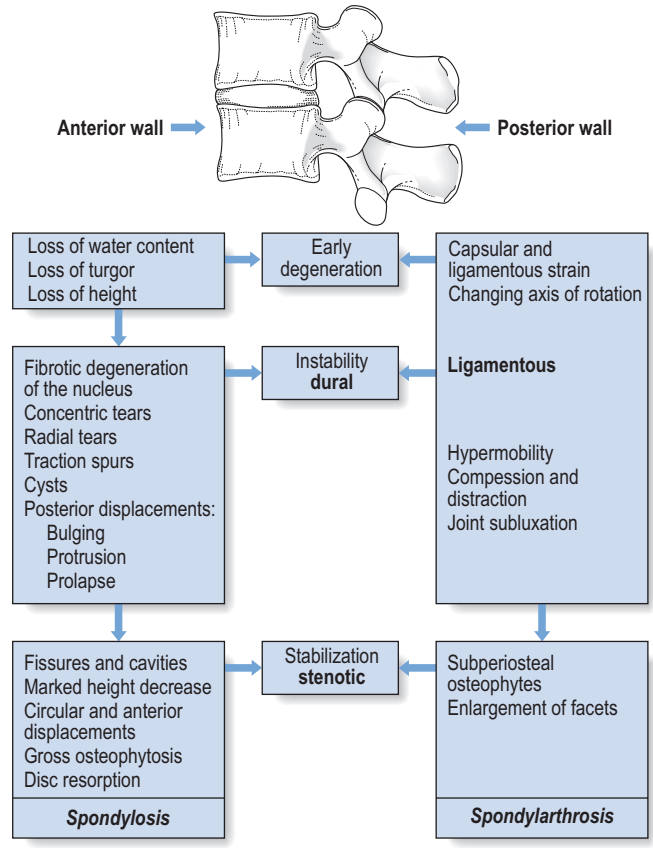

Fig 38.1 • The three clinical lumbar concepts in relation to the natural ageing of anterior and posterior walls of the vertebral column.

39

Non-mechanical disorders of the lumbar spine: warning signs

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Introduction

The majority of lumbar spine syndromes encountered in clinical practice result from mechanical – activity-related – disorders. They can be classified into dural, ligamentous and stenotic syndromes. Lumbar syndromes, however, can also stem from non-mechanical – non-activity-related – disorders affecting the spine. These are: inflammatory diseases, both septic and rheumatological; tumours and infiltrative lesions; metabolic disorders; and acquired defects in the neural arch. Finally, pain in the lower back, groin and pelvic area can be referred from visceral organs (see online chapter *Disorders of the thoracic cage and abdomen*). Pain in buttocks, groin and limb, as the result of reference from the sacroiliac and hip joints, although 'activity-related', does not have a spinal origin and is discussed thoroughly in the chapters on the hip joint and sacroiliac joint.

Although the occurrence of non-mechanical (non-activity-related) disorders is rare, it is important to differentiate them as quickly as possible from mechanical activity-related lesions. This is never easy, because these disorders frequently mimic other, more specific lumbar lesions. Sometimes the diagnosis is made radiologically but very often this is of no help, especially in the early stages of an inflammatory or neoplastic disease. A thorough history and clinical examination are what will first draw attention to the possibility of a non-activity-related disorder: the history may show an unusual localization or an atypical evolution of the pain; particular clinical signs may arouse suspicion. Most of all, however, it is the comparison between history and clinical examination, resulting in the existence of 'unlikelihoods', that focuses attention on serious spinal pathology such as vertebral fracture, malignancy, infection or inflammatory disease.[1]

Warning signs in backache and sciatica

Symptoms and signs that almost invariably point to non-mechanical disorders are termed 'warning signs' here. The finding of such signs indicates that the existence of a non-specific disorder in the lumbar spine is very likely. A patient presenting with a warning sign should never be considered to be suffering from a common mechanical disorder until the contrary has been proved. It is important, therefore, always to have confirmatory investigations carried out (radiography, bone scan, computed tomography (CT) and blood tests) to settle the diagnosis. It is also the duty of a physiotherapist who is asked to give active treatment (manipulation or traction) to a patient presenting with warning signs to report this to the referring doctor, and to send the patient back with a request for further thorough examination.[2]

Warning

Warning signs that may be detected during the history are:

- Significant trauma
- Deteriorating general health
- Pain in the 'forbidden area'
- Increasing, slowly worsening pain
- Expanding, rather than shifting, pain
- Continuous pain, unaltered by position or movement
- Sciatica with too long an evolution
- Bilateral sciatica
- Increasing postoperative backache

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Symptoms

Significant trauma

It is obvious that the statement of a significant trauma prior to the development of backache should be reason to ask for further imaging studies, especially if the patient is aged over 70 years and uses corticosteroids.[3,4]

Deteriorating general health

Unexplained weight loss, fever, feeling systematically unwell (tired, loss of appetite) and a previous history of cancer are all considered as potential symptoms of a serious illness.[5]

Pain in the 'forbidden area'

In the upper lumbar region pain is very seldom the result of a mechanical lesion. Disc lesions almost never occur at the first and second levels,[6] and even third lumbar lesions constitute only 5% of lumbar disorders.[7] Also, ligamentous lesions and recess stenosis do not seem to occur at these upper lumbar levels. Hence, if a patient has pain at the upper lumbar level – the 'forbidden area' (Fig. 39.1) – the suspicion is aroused that a non-mechanical lesion is present. Ankylosing spondylitis, neoplasm, tuberculosis, aortic thrombosis or reference from a viscus may then be possibilities (Cyriax[8]: p. 26).

Increasing pain

Lumbar pain steadily worsening over a number of weeks suggests malignant disease. This is especially true in elderly patients who have had central backache that has increased over a short period.

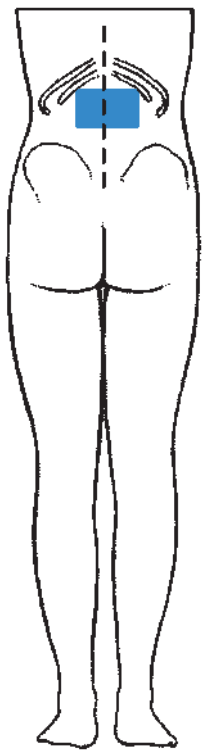

Fig 39.1 • The 'forbidden area'.

Expanding pain

A *moving pain* is a familiar symptom in disc lesions: the pain is central at first and becomes unilateral; or the backache changes sides; or there has been backache initially which has turned into leg ache after a time. Thus, in disc displacements, back pain may move to different localizations, or the backache eases when unilateral root pain comes on.

However, if the history is that of continuing backache, gradually *expanding* and worsening despite the appearance of root pain, a non-mechanical disorder should be suspected. In addition, a history of pain which first spreads to one dermatome but after some time also involves the neighbouring dermatomes should be considered to be the history of an increasing lesion, which almost never involves a disc. If pressure against the dura mater increases when pressure on the nerve root (or roots) sets in, the lesion responsible for the pain will not be a moving disc protrusion but a rapidly (neoplasm) or slowly (neuroma) increasing lesion.

Continuous pain

It is typical of mechanical disorders for postures and activities to have some influence on the pain: backache due to disc lesions is usually increased by sitting and bending and relieved by recumbency, while ligamentous pain has a typical postural nature and the radicular pain caused by a narrow lateral recess increases in the upright position and eases on sitting. When a patient's pain is more or less continuous and no posture can be found that relieves it, a serious spinal or extraspinal lesion should be suspected. Sometimes, however, a patient who is emotionally distraught or has hyperacute lumbago will claim that the pain is continuous. Further history taking will disclose that, although there is continuous disability, there may be some positions in which the pain eases somewhat, while severe twinges make other movements absolutely impossible. It is obvious that in these cases the pain is of mechanical origin.

Sciatica lasting too long

It is unusual for sciatica from a posterolateral disc protrusion to last longer than a year. The normal development is root pain which rapidly becomes worse and reaches a peak within 1–4 weeks. Severe symptoms then persist for a few weeks or months, thereafter improving. At the end of a year, nearly all patients have recovered. However, it is important to remember that patients over 60 years of age, especially those who still have some backache after root pain has appeared, do not always demonstrate a tendency to improve. Additionally, in cases of root compression caused by a narrowed lateral recess, the pain can remain present for months or years, without showing any tendency to worsen or improve.

If root pain continues to worsen after 9 months, the cause is almost certainly not a disc lesion, and a non-mechanical disorder, such as a neuroma or epidural cyst, is more likely. Rarely, sciatic pain that lasts longer than usual is caused by an adherent nerve root.

Bilateral sciatica

For the purposes of differential diagnosis, it is important not to confuse bilateral extrasegmental dural reference of pain with bilateral radicular pain. It is not difficult for an experienced examiner to distinguish between the two. Dural pain is dull, deep, diffuse and ill defined, and spreads to different dermatomes. Although dural pain often reaches the ankles, it never extends to the feet. Radicular pain is sharp and well localized, and stays within the borders of the dermatomes. The pain can reach the feet, except in the more exceptional cases of L1–L3 radicular pain, and can also be accompanied by distally localized paraesthesia and numbness.

If the patient presents with genuinely bilateral sciatica, a number of conditions must be taken into consideration:

- *Spondylolisthesis* can cause bilateral radicular pain, which presumably results from the forward movement of the listhetic vertebra, pulling the nerve roots painfully against the shelf formed by the stable vertebra below.
- *A disc lesion* resulting in bilateral sciatica is rare and should always be taken seriously because it probably means a massive protrusion, which poses a *risk to the S4 root*.[9] Bladder incontinence and numbness in the saddle area may then accompany the bilateral root pain. Rarely, a disc develops two protrusions, one at each side of the posterior longitudinal ligament; alternatively, two protrusions, one at the fourth level and one at the fifth, are present.
- *Bilateral lateral recess stenosis or a narrowed spinal canal* can also be the cause of bilateral sciatica. In the former, the typical history of increasing pain in the upright position is informative. In the latter, the patient mentions neurogenic claudication.
- *Malignant disease* is indicated by rapidly increasing bilateral sciatica, often spreading into the limbs in a distribution which corresponds to too many dermatomes.

Increasing backache after lumbar surgery

Intervertebral disc space infections most often follow surgical enucleation of a herniated disc.[10,11] After initial relief of the preoperative pain, severe and steadily increasing lumbar pain appears.

Signs

Warning

Clinical signs that constitute a warning are:

- Discrepancy between the marked articular signs and the absence of dural signs
- Gross limitation of both side flexions
- Gross limitation of side flexion away from the painful side as the only positive finding
- Flexion with a rigid lumbar segment
- Radicular pain and muscle spasm
- Discrepancy between pain and neurological deficit
- Involvement of multiple nerve roots
- Deficit of L1 and L2 nerve roots
- Buttock sign
- A warm foot on the affected side

Discrepancy between articular and dural signs

As acute lumbago is basically compression of the anterior part of the dural tube, dural signs should always be present. If a history of acute lumbago is described and marked articular signs are present but the patient has no dural signs at all, a disc lesion is unlikely and other more serious lesions should be considered, for instance:

- *In a pathological fracture* of a vertebra, resulting from a malignant disease or from senile osteoporosis, there is gross limitation of spinal movements but straight leg raising may remain normal and painless.
- *In ankylosing spondylitis*, an acute sprain of the stiffened lumbar joints will result in acute lumbago but dural signs are completely absent.
- *In chronic afebrile osteomyelitis* of a lumbar vertebral body, there is a gross contrast between the marked articular signs and the complete absence of dural signs.

Gross and bilateral limitation of side flexions

The range of side flexion diminishes with a patient's age. Some symmetrical limitation is therefore a normal finding in the elderly. However, bilateral limitation of these movements in younger or middle-aged patients is not normal and, if this sign is present, non-mechanical, and usually serious, diseases of the lumbar spine are strongly suggested. A lumbar localization of ankylosing spondylitis often presents with this sign, but so too can malignant or benign neoplasms, Paget's disease, chronic osteomyelitis and old fractures.

Gross limitation of side flexion away from the painful side

Gross limitation of side flexion away from the painful side (Fig. 39.2) is a common finding in acute disc lesions, in which it always appears in combination with other limited and painful

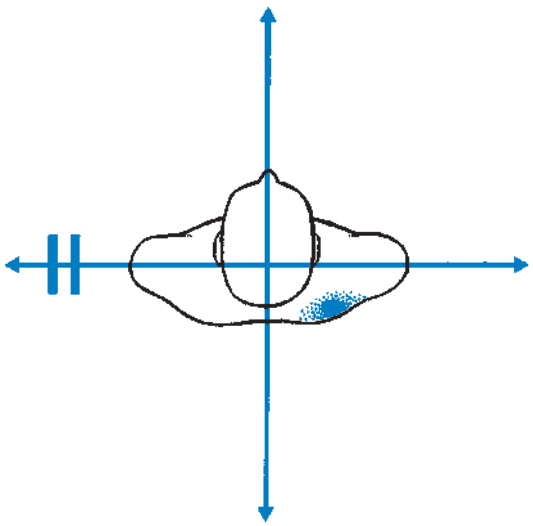

Fig 39.2 • Gross limitation of side flexion away from the painful side is a warning sign for serious disease.

movements, together forming the non-capsular pattern. However, when limitation of side flexion away from the painful side is the *only* positive lumbar feature, a disc lesion is never present and a serious extra-articular lesion must be suspected. This pattern suggests an abdominal neoplasm, usually carcinoma of colon or kidney, although a neuroma at the lumbar or lower thoracic level should also be considered.

Flexion with a rigid lumbar segment

Patients with serious disease of the lumbar spine flex from their hips; the lumbar spine is held in lordosis by spasm of the sacrospinalis muscle. The patient bends like an old-fashioned butler. This characteristic type of flexion – the lumbar spine held rigid and the body flexing as a whole at the hips – sometimes accompanies bilateral limitations of side flexion and must always be taken seriously. It is sometimes seen in acute lumbago caused by an ordinary disc lesion, but most often it indicates a spinal localization of ankylosing spondylitis or a more serious non-mechanical disorder of the spine. Care should be taken not to confuse this sign with a normal flexion range in a patient with marked kypholordosis. In such a case, the spine may stay horizontal at the end of the flexion, although the lumbar segment has undergone a considerable flexion movement.

Radicular pain and muscle spasm

In sciatica from a disc lesion, trunk flexion is limited because of pain felt in the limb, the patient being unable to stretch the sciatic nerve beyond a certain point. Usually, this limitation is associated with a degree of spasm of the sacrospinalis muscle. However, when side flexion or extension provokes radicular pain and also induces muscle spasm, more serious spinal diseases must be suspected, rather than a disc lesion.

Discrepancy between pain and neurological deficit

In disc lesions, some muscle weakness will be present only after a history of severe radicular pain. This does not mean that the patient must still have sciatic pain at the moment that paresis is detected. For instance, in root atrophy, radicular pain disappears the moment weakness becomes obvious. Although there is not a great deal of pain at this stage, the history is that of recent and severe sciatica.

If, by contrast, a patient presents with severe weakness without a record of severe pain in the limb, spinal metastases are likely. In disc lesions, it is very unusual to find complete paresis of a muscle except when two consecutive roots are involved, as sometimes happens in combined L4–L5 lesions at the fourth lumbar level and which leads to a drop foot.

Involvement of multiple nerve roots

Although one lumbar disc protrusion often compresses two adjacent nerve roots, triple palsies or bilateral palsies almost always result from metastases or from neuralgic amyotrophy.

Deficit of L1 and L2 roots

Disc lesions at the first and second lumbar levels are extremely rare; the estimated frequency is between 0.3 and 0.5%.[12–14] Also, lateral recess stenosis leading to muscular weakness does not occur at the upper lumbar levels. Therefore, if weakness of the psoas muscle is encountered, the initial diagnosis should never be a disc lesion but rather a serious non-mechanical disorder. In a neoplasm at the second lumbar level, a bilateral paresis is likely to appear. If unilateral weakness is accompanied by pain in the iliac fossa, brought on when the muscle contracts, a neoplasm at the iliac crest or in the pelvis is possible. If the weakness is accompanied by pain in the thigh, metastatic invasion of the upper femur is probable (see Ch. 48).

Presence of the 'sign of the buttock'

When the sign of the buttock is encountered, a serious lesion in the lumbopelvic area is always present (see Ch. 47). This can be a malignant deposit in the sacrum, iliac bone or femur, septic arthritis of the sacroiliac joint or a rectal abscess.

A warm foot on the affected side

In radicular pain caused by disc lesions, patients often complain that the foot and the leg on the affected side feel cold, which is often confirmed by palpation. If, by contrast, the affected side is warmer, neoplasm at the upper lumbar level should be suspected (Cyriax[8]: p. 292). The explanation is probably interference by the tumour with the sympathetic nerves at L1 and L2.

Access the pathology of the non-mechanical disorders of the lumbar spine and the complete reference list online at **www.orthopaedicmedicineonline.com**

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Treatment of the lumbar spine

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Introduction

Treatment of mechanical disorders of the lumbar spine should be selected according to the nature of the underlying problem. If this is neglected, an inadequate or inappropriate type of treatment may be given, disappointing the patient, the physician and the therapist.

A wide variety of non-surgical treatments are advocated in the management of low back pain. Back school instructions, bed rest, cryotherapy, medication, exercises, manipulation, traction, mobilization, local blocks, epidural infiltrations and spinal orthosis all claim to have their successes but until now, controlled studies of large, unselected populations have not demonstrated the superiority of any one of these measures,[1–7] which thus supports the view that there seems to be no definite evidence that any treatment for low back pain is much better than the placebo effect.[8,9] Some authors even suggest that the main effects of the therapies are produced not through the reversal of physical weaknesses targeted by the corresponding exercise method but rather through some 'central' effect, perhaps involving an adjustment of perception in relation to pain and disability.[10]

Obviously, these studies have reinforced therapeutic nihilism. As conservative measures are not proven to be effective, the patient is told to learn to live with the disability until it disappears spontaneously.[11]

However, in analysing the results, one striking factor emerges – the lack of a proper diagnosis. In current clinical trials on the effectiveness of treatment for lumbar disorders, the course of one symptom only (pain) is evaluated in randomized groups of patients. This is completely wrong. Controversies over treatment are usually the result of studies performed on widely differing lesions. A group of patients complaining of 'backache' is very heterogeneous. Even if, in most cases, the disc is responsible for the pain, the mechanism will differ considerably from patient to patient. Before any kind of randomization is done, the individual disorders in any group of patients should be clearly identified; treatment could then be given not for a symptom but for a well-defined condition.[12] We therefore believe that, before treatment of any kind is instituted, a clear diagnosis must be made and the physician must have a distinct idea of the underlying cause. Treatment can then be prescribed selectively, according to the type and the severity of the lesion. For example, a recent systematic review of randomized clinical trials on spinal manipulation could find no evidence of effectiveness, except in some subgroups of patients, using clearly delineated clinical inclusion criteria.[13]

The history and clinical examination almost always indicate the best method of treatment for an individual patient. It is

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our personal experience that, if conservative treatment techniques are employed intelligently, there is definite evidence that in each case one treatment is better than another or than the placebo effect. Also, only a few patients will remain wholly unrelieved, in which case surgery may be required. Even when surgery is indicated, the decision must be made on clinical grounds alone. An abnormality seen on imaging and not confirmed by clinical examination is not an indication for surgery.[14]

Before any form of treatment, conservative or surgical, is instituted, answers to the following questions must be obtained.

Is the pain caused by a spinal disorder?

It is important to exclude psychogenic pain and pain of visceral origin referred to the back.

Is the condition an 'activity-related' (mechanical) spinal disorder?

It is obvious that specific, non-mechanical disorders causing backache or sciatica – for example, rheumatic diseases, tumours and infections – need other types of treatment.

If the spinal disorder is activity-related (mechanical), to which 'concept' does it belong?

Is the disorder a discodural or discoradicular interaction?

In a clear combination of both articular and dural signs and symptoms, the answer will be obvious. In moderate discodural backache, though, where the patient presents with articular signs only, it may be more difficult to make the diagnosis of internal derangement with any certainty. However, a partial articular pattern, except in a few cases of ligamentous pain, always indicates a disc lesion. Deviation, whether in the upright position or on flexion, defines a protrusion. Also, the presence of a painful arc, whether during side flexion or forward flexion, is the signature of a small posterior or posterolateral bulge. If there is proof of a disc lesion, and the patient is motivated to take active treatment, conservative therapy consists of one or more of the following measures:

- *Reduction of the displacement*: achieved by manipulation or traction.
- *Maintenance of reduction*: obtained by sclerosing injections and/or back school instructions.
- *Desensitization of the dura mater*: acute or gross inflammation requires desensitization of the dura mater before manipulation or traction is attempted. Alternatively, if the dura mater remains inflamed after discodural contact has ceased, the treatment is epidural local anaesthesia.
- *Desensitization of the dural nerve root sleeve*: in intense discoradicular contact leading to some degree of parenchymatous involvement, attempts at reduction fail. De-inflammation and desensitization of the dural sleeve, while spontaneous recovery is awaited, is then a good and defensible therapeutic approach. Epidural local anaesthesia via the sacral canal is the technique of choice. Should this fail, a sinuvertebral and nerve root block can be substituted. Desensitization of the dural sleeve is also recommended when the discoradicular contact has lasted for some time or when the nerve root remains inflamed after the conflict has ceased (Fig. 40.1).

Is the disorder a ligamentous problem?

Ligamentous disorders are characterized by postural pain, absence of dural symptoms and a full range of movement.

Symptoms and signs make it possible to differentiate between a postural and a local dysfunctional syndrome. In the former, treatment consists of the induction of artificial sclerosis in the posterior ligamentous structures. The latter usually responds to local infiltration with triamcinolone or sclerosants.

Is the disorder a stenotic problem?

Spinal stenosis, suggested by the presence of 'neurogenic intermittent claudication', is very difficult to treat conservatively. Lateral recess stenosis, however, characterized by long-standing sciatic pain in an elderly patient, which appears during walking or standing and disappears during sitting or bending, responds extremely well to nerve root infiltrations with triamcinolone.

What sort of person is the patient?

The following questions must be addressed when devising a treatment plan:

- What attitude does the patient have towards the problem?
- Is there a desire to get better?
- Is there any compensation claim?
- Is there evidence of psychoneurosis?
- Will active and sometimes unpleasant forms of treatment be tolerated?

These questions must be addressed when devising a treatment plan.

If treatment is employed along these lines, only a few patients will remain unrelieved. This approach has also proved to be safe and, at the same time, is a meaningful, realistic and practical response to the enormous liability of lumbar disorders in terms of cost and needless human suffering. In 1986, the annual cost of back pain in the USA approached $81 billion.[15] By 1995, the total cost to society of low back pain was more than $100 billion[16] and the economic loss 101.8 million workdays.[17] In the UK, the direct healthcare cost of back pain in 1998 was estimated to be £1632 million. However, this figure is insignificant compared to the cost of the production losses related to it, which was £10668 million, making back pain one of the most costly conditions for which an economic analysis has been carried out.[18]

A substantial portion of low back care costs reflects expensive surgical therapy, when many patients could have been effectively treated if the above-mentioned less costly regimens had been tried first. Unfortunately, the average spinal surgeon is usually neither trained in nor knowledgeable about conservative spinal therapy. In fact, in some of the present state healthcare systems, surgery is routinely performed in almost all cases of low back pain (Burton[14]: p. 105). This has been confirmed

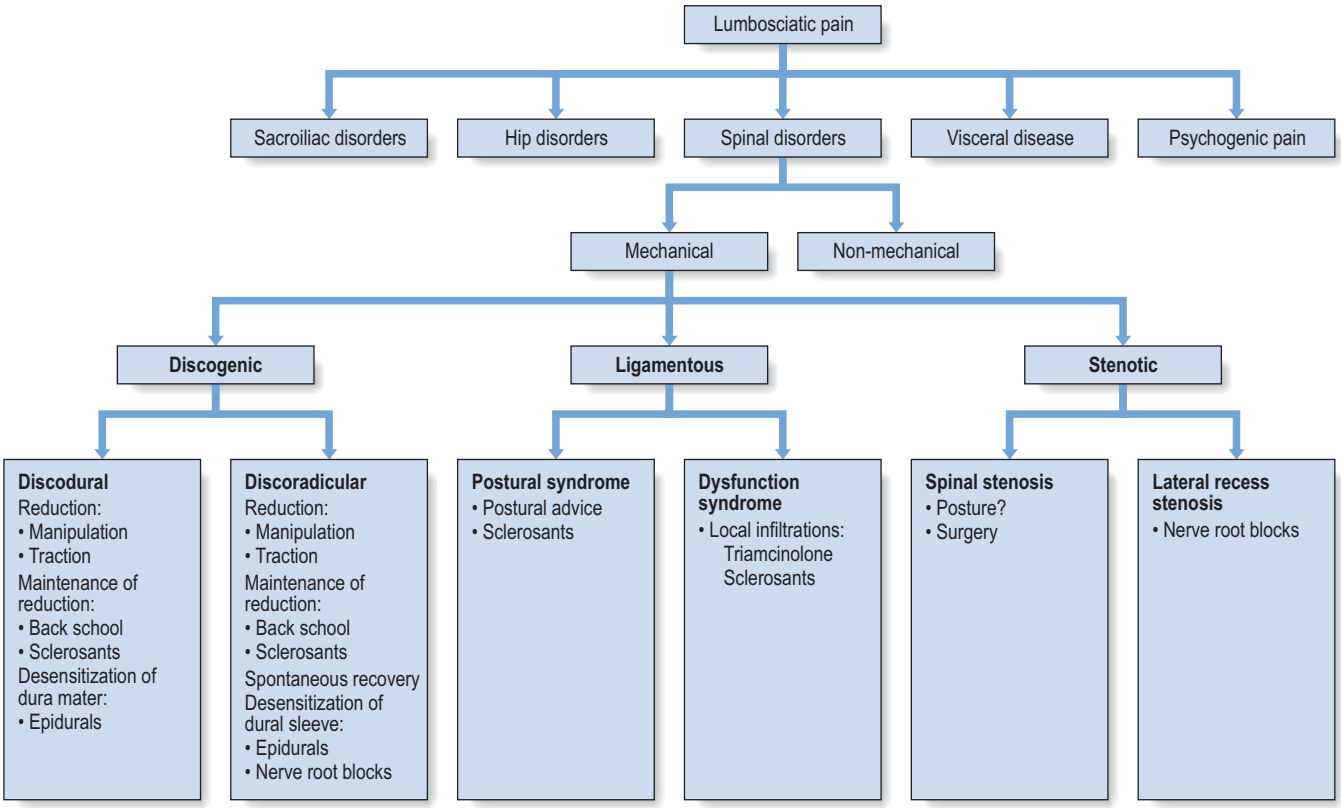


Fig 40.1 • Treatment scheme for lumbosciatic pain.

by Finneson,[19] who studied one series of 94 patients in whom back surgery had failed and found that in 81% the original surgery was not indicated.

Manipulation

Introduction

Manipulation and traction are no exceptions to the rule that all medicine can be traced back to Hippocrates (400 BC). His methods of treating back disorders were practised in the subsequent centuries by other famous physicians: for example, Apollonius, Galen, Avicenna, Ambroise Paré, Percival Pott, Sir James Paget and many others. In China, the technique of manipulation was fully established during the Tang Dynasty (AD 618–907). Illustrations show that the ancient methods of manipulation and apparatus traction did not differ very much from the methods of treatment practised nowadays in low back pain and sciatica (Fig. 40.2).

Manipulation and traction are methods of reducing pain and disability caused by internal derangement of intervertebral cartilaginous structures. This treatment undoubtedly stems from man's experience with those examples of lumbago, low back pain and sciatica that were relieved by a sudden twist or a fall. Schiötz and Cyriax[20] described several examples in which patients reported such a sudden relief of symptoms after an unintentional movement. One of our colleagues reported a patient who recovered from backache on falling down her basement cellar steps. Undoubtedly, such experiences have given rise to various, sometimes bizarre mechanical treatments in folk medicine, such as trampling on a patient's back, wrestling the back, striking the back with the weight of a steel bar or standing back to back 'weighing salt' (two persons stand back to back, hooking their arms at the elbow; each person bends forwards in turn, lifting the other from the floor). These measures seem non-specific and possibly harmful but Schiötz commented: 'It seems justified to infer that methods found effective by the natives of parts of the world as far apart as Norway, Mexico and the Pacific Islands over many, many centuries must be valid.' These primitive methods are also the models for more modern techniques of manipulation and traction taught and practised by trained doctors and therapists all over the world.

Although different methods and different theoretical concepts developed, this seemed not to matter much as all claimed their successes. Cyriax[21] was convinced that, in nearly all cases, a similar mechanism is at work – a dislocated fragment of disc returns to position or a protrusion is 'sucked back'. He also stated: 'Spinal manipulative methods of orthopaedic medicine are perfectly straightforward and possess an explicable intention directed to a factual lesion. They are based on the discovery of disc lesions as the primary cause of degenerative change and pain of spinal origin.'

It is hard to believe that, even nowadays, many doctors still refuse to recommend manipulation of the back as the treatment of choice in nearly all cases of acute low back pain and sciatica. Avoiding discussion of the topic of spinal manipulation is also unwise, since a significant number of their patients will

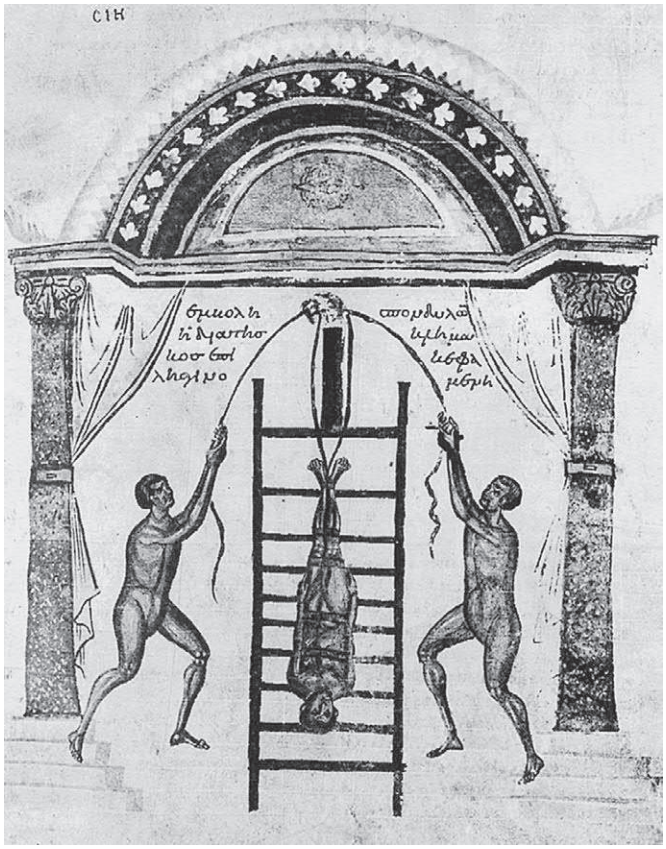


Fig 40.2 • Ladder treatment, 1st century BC. H. Schöme (ed) *Illustrierter Kommentar* [by Apollonius Citiensis] *zu der Hippokrateischen Schrift*. Teubner, Leipzig. (By courtesy of Wellcome Trustees.)

have received or will be considering this form of treatment: in the USA in 1980, 120 million surgery visits were made to chiropractors.[22]

An increasing number of controlled clinical trials on well-delineated subgroups have been published, which compare the results of manipulation to different forms of placebo therapy as well as to other forms of conservative management.[23–34] The positive effects of manipulation appear to occur either immediately after the manipulation session or within the first 4–6 weeks of treatment.[35–37]

Definition of manipulation

In orthopaedic medicine, spinal manipulation is defined as a method of conservative treatment by passive movements, carried out with a single thrust or sustained pressure, in order to return a displacement to its proper position (see Ch. 5, p. 95). During the thrust, an audible click is often produced. This may accompany immediate relief of symptoms and signs, which supports the precision of the diagnosis and selected therapy.

There are different schools of thought in manipulation, which derive from different attitudes towards spinal disorders. The theories on which manipulation is based include the reduction of disc protrusions,[38,39] the correction of posterior joint dysfunction,[40] the mobilization of blocked vertebral joints,[41–44] the reduction of nerve root compression,[45,46] the normalization of reflex activity[47–50] and the relaxation of muscles.[51]

The objective of the manipulative techniques discussed in this book is to alter the discodural or discoradicular interaction by moving a displaced cartilaginous rim away from sensitive structures. The method relies on two principles:

- The lumbar spine is positioned in such a way that the affected intervertebral joint opens, i.e. gives the loose fragment room to move.
- The manipulation is usually carried out during traction. This tautens the posterior longitudinal ligament and causes suction in the disc, so exerting a centripetal force (Fig. 40.3).

Most of our techniques are non-specific, long-lever manipulations: the force is exerted on a part of the body some distance away from the area where it is expected to have its beneficial effect. Leverage enables the manipulator to apply more force at the affected level. The normal joints are moved as far as they can go. The posterior longitudinal ligament becomes taut. The moment that resistance of the blocked joints and the taut ligament is felt, a quick additional thrust is given, to act at the affected level. Manipulation of the lumbar spine either is quickly successful or fails. If, after one or two manœuvres performed in a certain direction, signs and symptoms remain unaltered, another direction or another technique is tried. If these also prove ineffective, manipulative treatment is abandoned. If, by contrast, manipulation has led to reduction, both local and referred discomfort cease. Previously painful movements immediately become normal. So it is the patient, rather than the manipulator, who judges the effect of treatment.

Most other manipulative schools claim to work more selectively, i.e. on the affected level only. They claim to have developed the clinical skills to localize, by palpation, the exact site of the 'fixation' or 'locking'. Several studies have failed, however, to demonstrate the reliability of this approach.[52–57] We support McKenzie's[58] conclusion that demystification of spinal manipulative therapy is an urgent priority. Both chiropractice and osteopathy thrive by creating the impression that there is something complex and exclusive about the practice of passive end-range motion that only chiropractors or osteopaths can understand or have the skills to 'feel'. They generate the belief that, in order to become skilled in the understanding and delivery of spinal manipulative therapy, it is necessary to undergo 3 or 4 years of training.[59] This suggestion is undermined by the fact that the majority of lay manipulators in Britain have never had any tuition at all and yet have amassed many satisfied clients and also very rarely figure in actions for damages.[20]

The main advantages of the methods discussed in this book are, first, that they are much simpler but at least as effective as those advocated by chiropractors and osteopaths. Second, it takes only about 180 hours of tuition, provided that the student has completed medical or physiotherapy studies.

In orthopaedic medicine, the manœuvres are always intended to relieve the current cartilaginous displacements. This is in contradiction to other methods, where a protocol of regular or intermittent manipulation sessions is commonplace. The *type of displacement*, as well as the *patient*, has to be

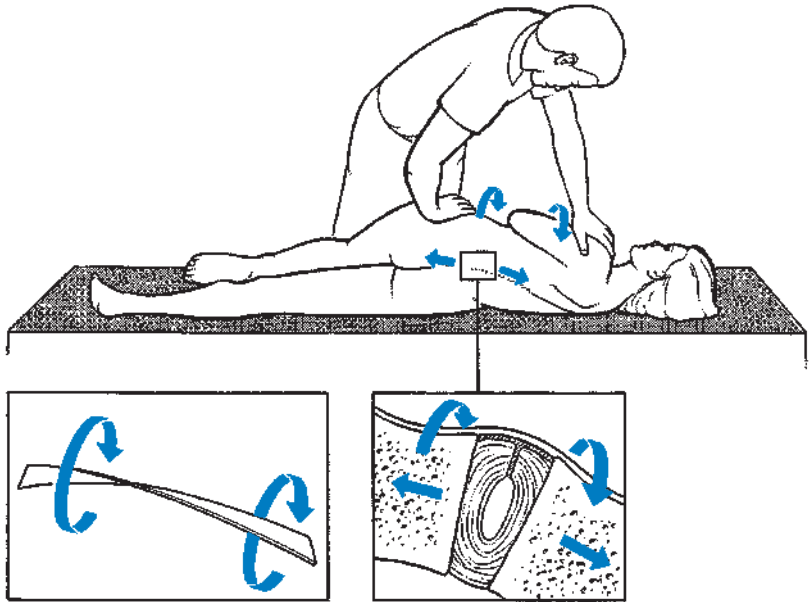


Fig 40.3 • Effects of long-lever manipulations. Positioning of the spine 'opens' the intervertebral space. A combination of traction and rotation produces a torque with tautening of the posterior longitudinal ligament and helicoidal traction on the lateral part of the annulus.

assessed before any kind of manipulative manœuvre is undertaken.

- *The displacement should be cartilaginous, not too large and not placed too far laterally*. Soft nuclear protrusions are seldom reduced by manipulation unless they are small and very recent, and the technique of manipulation is changed to sustained pressure. If the consistency of the displacement is not quite clear, with symptoms and signs pointing in opposite directions, it is worthwhile making one attempt at manipulation. During the first session, it is usually quickly apparent whether reduction by this means will prove feasible or not. If it fails, traction is substituted the next day. Reduction of cartilage displacements, together with full relief of symptoms and signs, has proved to be possible in two-thirds of all cases of backache and in one-third of all cases of sciatica.[60] Just about half of all lumbago cases are relieved in one treatment.[61,62]
- *The patient must be mentally stable and keen to get well*. If this psychogenic aspect is neglected, on some occasions a patient may be treated who claims to have been made worse by a type of therapy that is regarded in retrospect as unacceptable. Hence it is important to avoid these active methods of treatment when the patient's attitude appears to be more important than the minor mechanical disorder found on examination.

Indications for manipulation

History and clinical examination almost always supply sufficient information to select those cases suited to manipulation (see Box 40.3, below).

Acute annular lumbago

The attack is initiated by a click in the lower back, followed by sudden agonizing lumbar pain fixing the back in slight flexion or lateral deformity. The pain radiates in an extrasegmental way and there are marked dural signs and symptoms.

Reduction should always be attempted, except in hyperacute cases where the attempt proves impossible to bear. An epidural injection is then substituted and followed by manipulation the next day. If such an injection is refused, it is still possible for the patient to recover in about a fortnight with bed rest and the use of McKenzie's extension mobilizations[58] and anti-deviation techniques. The moment the process has ceased to be hyperacute, treatment by manipulation can be tried again.

Backache

Acute or recurrent backache that has started suddenly usually responds well to manipulative treatment. There are a number of symptoms and signs which indicate that manipulation is likely to be successful.

The description of a click and sudden pain in the back on bending forwards or on coming upright from a forward-bent or sitting position indicates displacement of a small cartilaginous fragment. Another 'favourable symptom' is the patient's age because, over the age of 60, nuclear protrusions no longer occur and a hard and mobile fragment of disc material is very likely.

'Favourable signs' are:

- A partial articular pattern in which some movements are only painful at extreme range: for example, flexion, extension and side flexion away from the painful side
- The existence of a painful arc with or without momentary deviation
- Absence of gross deviation caused by muscle spasm on standing or during as much flexion as the patient can tolerate
- Absence of gross limitation on movement – gross deviation or limitation of movement always requires several sessions of manipulation (Box 40.1).

Box 40.1

Symptoms and signs favouring manipulative treatment of backache

Favourable symptoms	Favourable signs

• Patient over 60 years
• Sudden onset of pain:
  ◦ On bending forward
  ◦ On coming up
	• Partial articular pattern
	• Side flexion away from the painful side hurts most
	• Painful arc with or without momentary deviation
	• Absence of gross deviation
	• Absence of gross limitation on movement

Box 40.2

Symptoms and signs favouring manipulative treatment of sciatica

Favourable symptoms	Favourable signs
• Backache still continues after root pain has begun • Root pain is recent	• Lumbar extension and side flexion hurt in the back but not in the limb • Absence of deviation or muscle spasm • Straight leg raising is only moderately limited, with absence of spasm of the hamstring muscles • Absence of neurological deficit

Box 40.3

Manipulation

Indications

- Acute annular lumbago
- Backache with favourable symptoms/signs
- Sciatica with favourable symptoms/signs
  - Mixed protrusions
  - Patient over 60 years

Contraindications

- Danger to S4 roots
- Anticoagulant therapy
- Aortic graft
- Last month of pregnancy
- Weakened body structures
- Muscle spasm
- Seriously neurotic patients

Not useful

- Too painful
- Too large a protrusion
- Too soft
- Too long a duration of root pain
- Compression phenomena
- After laminectomy, protrusion at the same level
- Unfavourable articular signs in:
  - Backache
  - Sciatica
- Primary posterolateral protrusion

However, some small protrusions do not respond well to manipulation. Patients under 60 years of age, in whom pain is greatest on pinching the lesion by side flexion towards the painful side, are usually 'unfavourable'. If such a manœuvre causes pain in the lower limb instead of the lumbar region or upper buttock, manipulation nearly always fails. A better response will be achieved with traction.

Sciatica

Reduction proves possible in about one-third of all patients with sciatica. Again, several symptoms and signs indicate those patients who can be expected to respond well (Box 40.2).

Patients with mixed protrusions

If neither the symptoms and signs nor the patient's age establish the consistency of the displaced fragment, manipulation should be tried first; it quickly becomes clear whether this is effective or not. In contrast, traction usually requires a week before efficacy can be determined.

Patients over the age of 60

The rules that determine a nuclear or an annular lesion are no longer applicable over the age of 60 years. The nucleus has become hard and dry, and will react correspondingly. Bed rest is wholly ineffective because there is no great difference in intradiscal pressure on lying and standing, and as the tension of the posterior longitudinal ligament lessens, it becomes elongated and loses its ability to apply a correcting centripetal force. Hence, all discodural or discoradicular interactions in this age group are best treated by manipulation, as this is the only way to achieve reduction.

However, for elderly patients, manipulative treatment should also be adapted. This means that only one or two manipulations are performed during a session. The interval between two sessions is also extended: say, to once a week. The intensity of each technique should not be changed, however, otherwise the centripetal forces acting on the joint at the moment of manipulative thrust are too small to influence a displacement.

Lumbar manipulations are not contraindicated in osteoporosis. However, some techniques are not used for fear of fracturing a bone.

Contraindications

These may be divided into circumstances in which manipulation is absolutely contraindicated and those in which manipulation is of no use, although not harmful to the patient (Box 40.3). Appropriate selection of patients and choice of techniques can avoid such serious complications as have been reported.[63,64]

Absolute contraindications

Danger to the fourth sacral roots[65,66]

Although these roots lie in the centre of the spinal canal, well protected by the posterior longitudinal ligament, they may be threatened by a massive central lumbar disc prolapse which has caused considerable bulging and possibly partial rupture of the posterior longitudinal ligament. Manipulation may rupture this ligament completely, causing extrusion of the entire disc.

The important symptoms of an S4 syndrome are rapid progression of bilateral sciatica and neurological symptoms in both legs. Pain and paraesthesia in the perineum, rectum, genitals or anus are other symptoms suggesting this menacing lesion. Finally, bladder weakness, causing frequency of micturition without a strong urge, loss of rectal tone and faecal incontinence result.

Acute lumbago and bilateral sciatica with compression of the nerve roots at the same level are examples of a large central protrusion in which bulging of the posterior longitudinal ligament is to be expected. Such a protrusion may also cause spinal claudication. These patients have symptoms during walking, immediately relieved by lying down.[67]

In all these conditions, a high-force rotational technique in the side-lying position could rupture the last protecting fibres of the posterior longitudinal ligament with massive extrusion of the entire disc.

Anticoagulant medication

Manipulation of patients on anticoagulant therapy may lead to an intraspinal haematoma.[68] A patient who has a clotting abnormality should also not be subjected to forceful manipulations.

Aortic graft

Although there have been no reports of damage at the junction of a graft in the aorta by manipulation, its presence is reasonably regarded as a contraindication to any forceful manipulation – rotations or extension thrusts.

Final month of pregnancy

During the final month of pregnancy, lumbar manipulation is impracticable. Bed rest or epidural local anaesthesia is an alternative possibility. There is no bar to manipulation during the first 4 months; after this, extension techniques which necessitate a prone-lying position are omitted.

Manipulation should not be employed at *any* time if there is any predisposition to miscarriage.

Weakened bone structure

This includes unstable fractures, severe osteoporosis, osteomyelitis, multiple myeloma and bony tumours.

Muscle spasm

Forced movements should never be used when there is obvious muscle spasm, which may indicate some kind of joint irritability, fracture or metastasis. Therefore, any sign of muscle spasm at the moment the lumbar spine is positioned and the manipulator starts to stress the affected intervertebral joint should be taken as a sign to stop the procedure immediately. Of course, such a warning sign should already have been detected by an adequate clinical examination.

Mental state

Patients with serious mental disturbance should not be treated by manipulation, even if there is an underlying disc protrusion which seems suitable for manipulative treatment. The symptoms largely exist in the patient's mind and indicate certain needs – compensation or protection – and it can be expected that manipulation will fail or even aggravate the disorder. In addition, an obsessional neurosis about vertebral displacement may result. This not only may make manipulation troublesome to undertake but also may cause the patient to blame the treatment for making matters worse.

Cases in which manipulation is not useful

Too much pain

In *hyperacute lumbago*, manipulation is impossible because of unbearable pain on any attempted movement. These patients are best treated by the immediate induction of epidural anaesthesia and manipulation is postponed until dural irritation has mostly ceased.

Too large a protrusion

There are two categories.

Protrusions causing impaired root conduction

All such protrusions are impossible to reduce by either manipulation or traction. If clinical signs of muscle weakness, cutaneous analgesia or reflex disturbances are present, the protrusion is too large (and located too far laterally) to be replaced.[38,69–73] Epidural local anaesthesia is the treatment of choice.

Sciatica with an excessive flexion or side flexion deformity

In such cases, an attempt to move in the contrary direction causes pain to shoot down the leg. Surgery is indicated.

Too soft a protrusion

Nuclear protrusions causing backache and sciatica do not respond to manipulation (except in small and very recent cases, and provided that the manipulation technique is changed to sustained pressure). The consistency of the protrusion is too soft to be influenced by a quick thrust. Traction is the treatment of choice. The history is rather typical and usually identifies this type of disc lesion.

Acute nuclear lumbago is also an example of a protrusion that is too soft to manipulate with a thrust. The history is of pain that began gradually, after much stooping and lifting, and became slowly worse over the next few hours. The following morning, the patient wakes unable to get out of bed because of severe lumbar pain. The patient is always under 60 years old and, although manipulation is indicated, it must be exerted by sustained pressure. If this makes the patient better, techniques should follow in the supine, side-lying and standing positions to correct a persisting lateral deviated position of the trunk (see pp. 554–556).

Alternatively, an epidural injection can be tried, again followed the next day by manœuvres to correct a lateral

deformity. However, if these measures all fail, *constant* pelvic traction, in a supine position and continued for some days, is called for, slowly changed to periodic half an hour daily traction. Extension mobilizations, as recommended by McKenzie,[58] have also been found to be effective; this treatment is based on McKenzie's hypothesis that flow or displacement of fluid, nucleus or sequestrum can occur within the intact annulus of the intervertebral disc as a result of prolonged or repetitive loading. This most commonly occurs with flexion loading. He recommends well-defined extension forces in order to reverse the direction of flow or displacement.

Too long a duration of root pain

In patients under 60 years, 6 months of root pain have been shown to be the limit for manipulative reduction. However, if backache persists together with root pain, manipulation should be tried because these patients often do not recover spontaneously.

Compression phenomena

Central stenosis, lateral recess stenosis and the 'self-reducing' disc protrusion do not respond to manipulative treatment. In stenosis, the underlying condition is the reason that attempted manipulation or traction fails.[71,74,75] The self-reducing disc protrusion, with symptoms at the end of the day only, may be reduced by manipulation but will prove to be transient anyway.[38]

After laminectomy

New protrusions, at the same level, are seldom successfully reduced by manipulation. Traction is often more effective.

Unfavourable articular signs in a patient under 60 years

In backache, side bending towards the painful side is an unfavourable sign for manipulation but this does not apply in lumbago.

If any movement other than flexion hurts in the lower limb instead of the back, manipulation is almost certain to fail.

If an attempted extension manipulation gives rise to root pain, the manœuvre should be discontinued, as the protrusion is pressed harder against the nerve root.

Primary posterolateral protrusions

These protrusions all consist of nuclear material and therefore cannot be reduced by manipulation (see p. 458).

Dangers of manipulation

Lumbar manipulation is quite safe.[63,64] The most frequently reported serious complication is further prolapse of a herniated disc, resulting in a cauda equina syndrome. However, the risk of spinal manipulation causing a cauda equina syndrome is estimated to be less than 1 per 3.7 million treatments.[65,76] Most of the incidents were described in patients undergoing manipulation under anaesthesia or chiropractic adjustments.[66] Long-lever, high-force rotation techniques in the side-lying position are regarded as responsible. This is only partly true: the underlying cause is the lack of adequate examination to rule out unsuitable disorders. If, in contrast, manipulative procedures are instituted after a *thorough* examination, those described in this book have never led to severe accidents. The main advantages of these manipulations are:

- A great deal of traction is used to exert a strong centripetal force on the intervertebral joint.
- Movements towards flexion, which can intensify potentially harmful centrifugal forces, are excluded.
- Each manœuvre is followed by a fresh assessment of dural, root and articular signs, which affords a clear pointer to what has happened inside the joint and what the next step should be.

Other complications, such as sprains of the costovertebral and costochondral junctions or fractures of a transverse process, are less serious and either the result of poor technique or inappropriate indications. Should they arise, spontaneous recovery is to be expected after a short period of, say, 4–8 weeks.

Side effects, remarks and precautions

If it becomes clear, after the history and clinical examination, that the orthopaedic problem is less important than the psychological one, the patient is best left untreated. Even if such a patient can be helped, the improvement will not persist. The moment the patient realizes that a cessation of symptoms may have adverse consequences, a postmanipulative mental crisis is to be expected, blaming the treatment.

Manipulation should cause only minor discomfort, which is due to stretching effects on soft tissue structures. In acute lumbago a more gentle start is often necessary, to assess patients' reaction and to gain their confidence.

Sometimes the patient leaves a session pain-free but, for the following 2 days, a rather strong reaction follows. However, examination during the next consultation shows that symptoms and signs have decreased or even disappeared. Therefore patients should be warned of some after-pain, which is due to muscular and/or capsular–ligamentous reactions. It disappears within 2–3 days and is unrelated to the lesion.

Elderly patients can be manipulated safely. However, the number of manœuvres during one session should be confined to, say, two or three. In these patients high-force, long-lever techniques should also be omitted.

Results

Acute lumbago

If there is no deviation on standing or in the maximal forward-bent position and the lumbago is of recent onset, 50% of patients will get well with one treatment.[61,62] Potter[73] noted that 93% of such patients either fully recovered or were much improved following manipulation.

Backache

Manipulation either works fast and leads to a swift disappearance of symptoms and signs, or has no effect at all. If there is no clear improvement after one or two sessions, this treatment form should be abandoned. Uncomplicated low back pain of

recent onset seems to be significantly more responsive to manipulation than are chronic cases.[29,73,74,77,78] The beneficial effect of manipulation is particularly significant in low back pain with dural signs.[79]

Sciatica

The success rate of manipulation in an uncomplicated discoradicular conflict is about 1 in 3 (J. Cyriax: personal communication, 1982). This result was confirmed by one report that claimed complete recovery in 18 out of 50 chronic cases with unilateral sciatica after one manipulative session.[60] Others found that 25–75% of their patients with uncomplicated sciatica recovered or improved considerably after spinal manipulations.[39,62,70,73,79]

Manipulation techniques

The manipulative techniques used in orthopaedic medicine can be divided into three groups:

- Rotation
- Extension
- Antideviation.

After a detailed description of each technique, the reader will find a 'practitioner's checklist' regarding choice of technique, assessment of progress, repetition of techniques and the course of a manipulative session.

Rotation techniques

Rotation strains have been shown to be very effective in reducing displacements at a low lumbar level. A session of manipulation therefore always starts with these manœuvres. First, a 'stretch' is performed, being the smallest rotation strain. The patient lies with the painful side uppermost in order to bring the joint surfaces apart on the side of the displacement. Then, if necessary, this technique is followed by stronger rotations, using the femur as a lever. However, the latter techniques are impracticable in patients with arthritis of the hip or in elderly patients in whom osteoporosis is suspected. If the displacement lies centrally, straight leg raising may indicate which side should be treated first. However, in the absence of any symptom or sign to indicate the side, either side can be treated and the manipulator proceeds by trial and error.

Five different rotation techniques are described, all of which are used frequently.

Stretch

The couch should be stable and adjustable to about 30 cm in height. Using a high couch makes it impossible to aid the distraction of body weight, which in turn decreases the effect of manipulation. The patient lies on the painless side. The upper thigh is flexed to a right angle with the underneath leg extended.

The manipulator stands behind the patient, level with the patient's waist. One hand is placed in front of the shoulder and rotates the thorax backwards and upwards as far as it will go. At the same time, the heel of the other hand, placed against the greater trochanter, rotates the pelvis forwards and downwards to the same extent. This brings the joint surfaces apart on the side of the displacement.

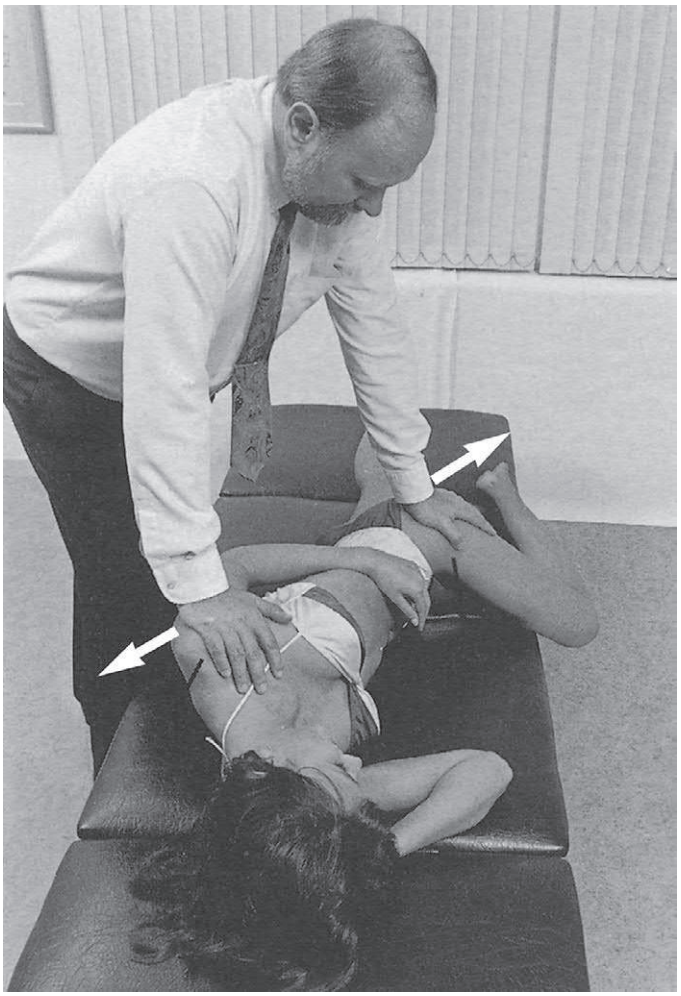


Fig 40.4 • Stretch.

By using the body weight and leaning well over the patient, the manipulator obtains considerable distraction at the lumbar joints. At the moment the limit of tissue tension is felt, the manipulator's body is pushed forwards on the vertically outstretched arms to apply overpressure (Fig. 40.4). At that moment a 'click' or 'snap' is nearly always heard and felt, after which the result of the manipulation is assessed.

Practitioner's checklist

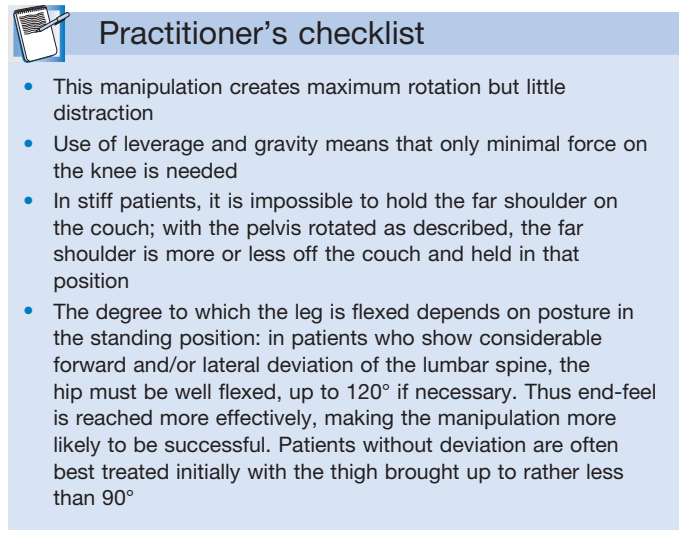
- Angles of rotation should be equal
- Hands positioned with fingers pointing in the direction of movement
- Elbows extended
- Equal amount of pressure on both hands
- Body weight over the patient but assess depending on build
- Manipulation is a stretch, not a rotation

Leg crossed over

The curved arrow symbolizes a 'leg crossed over'. The direction of the arrow, to the left, indicates that the patient's trunk is rotated to the left posteriorly. The letter R indicates that the patient lies on the right-hand side.

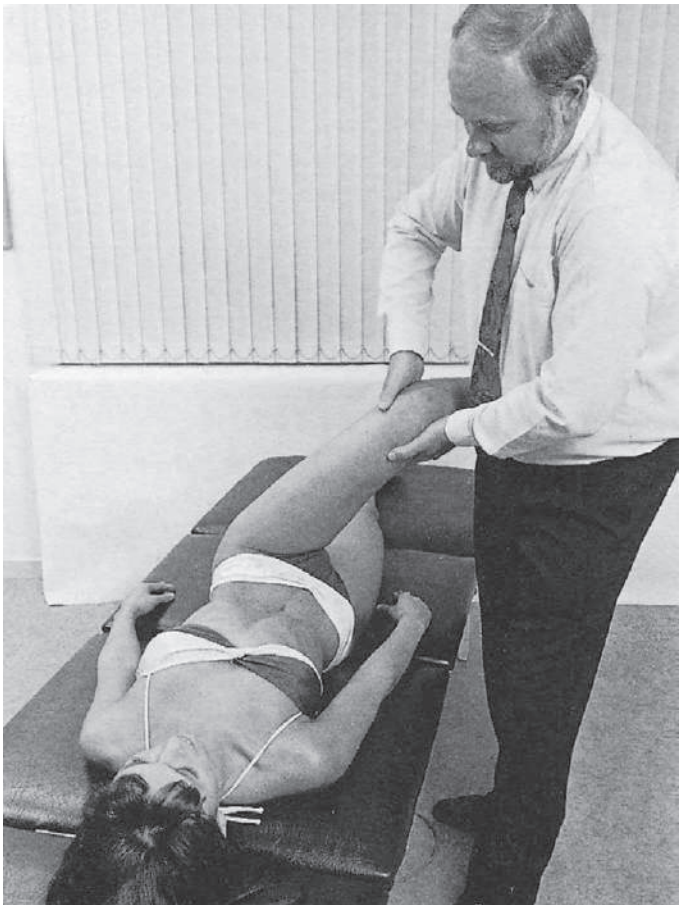
The couch should be stable and adjusted to about 60 cm in height. The patient lies supine about 20 cm or a hand's breadth from the edge of the couch.

The manipulator stands on the painless side, level with the patient's waist, facing the feet. With both hands flexing the thigh on the far side up to 90° and drawn forwards, the pelvis and lower back are rotated towards the operator. In this way, hip adduction is avoided. The ipsilateral knee of the manipulator is applied to the pelvis, if necessary, to prevent the patient from falling from the couch. Next, the contralateral forearm is turned into supination and the palm of the hand applied to the outer side of the knee. The other hand pushes the patient's far shoulder flat on the couch (Fig. 40.5). Then rotation of the

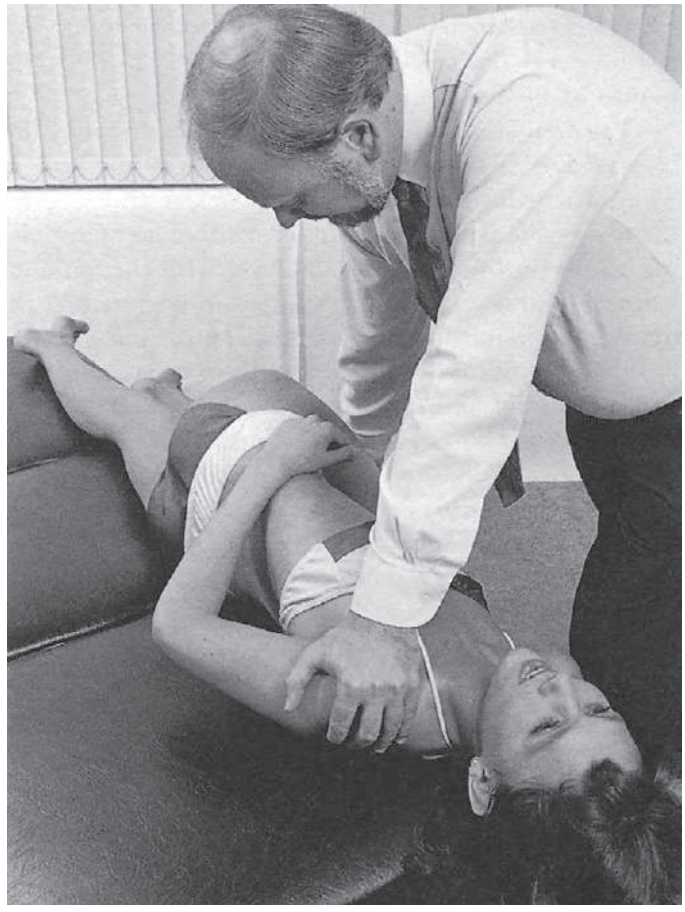


Practitioner's checklist

- This manipulation creates maximum rotation but little distraction
- Use of leverage and gravity means that only minimal force on the knee is needed
- In stiff patients, it is impossible to hold the far shoulder on the couch; with the pelvis rotated as described, the far shoulder is more or less off the couch and held in that position
- The degree to which the leg is flexed depends on posture in the standing position: in patients who show considerable forward and/or lateral deviation of the lumbar spine, the hip must be well flexed, up to 120° if necessary. Thus end-feel is reached more effectively, making the manipulation more likely to be successful. Patients without deviation are often best treated initially with the thigh brought up to rather less than 90°



(a)



(b)

Fig 40.5 • Leg crossed over: (a) with the leg forwards to rotate the pelvis; (b) shortly before manipulation.

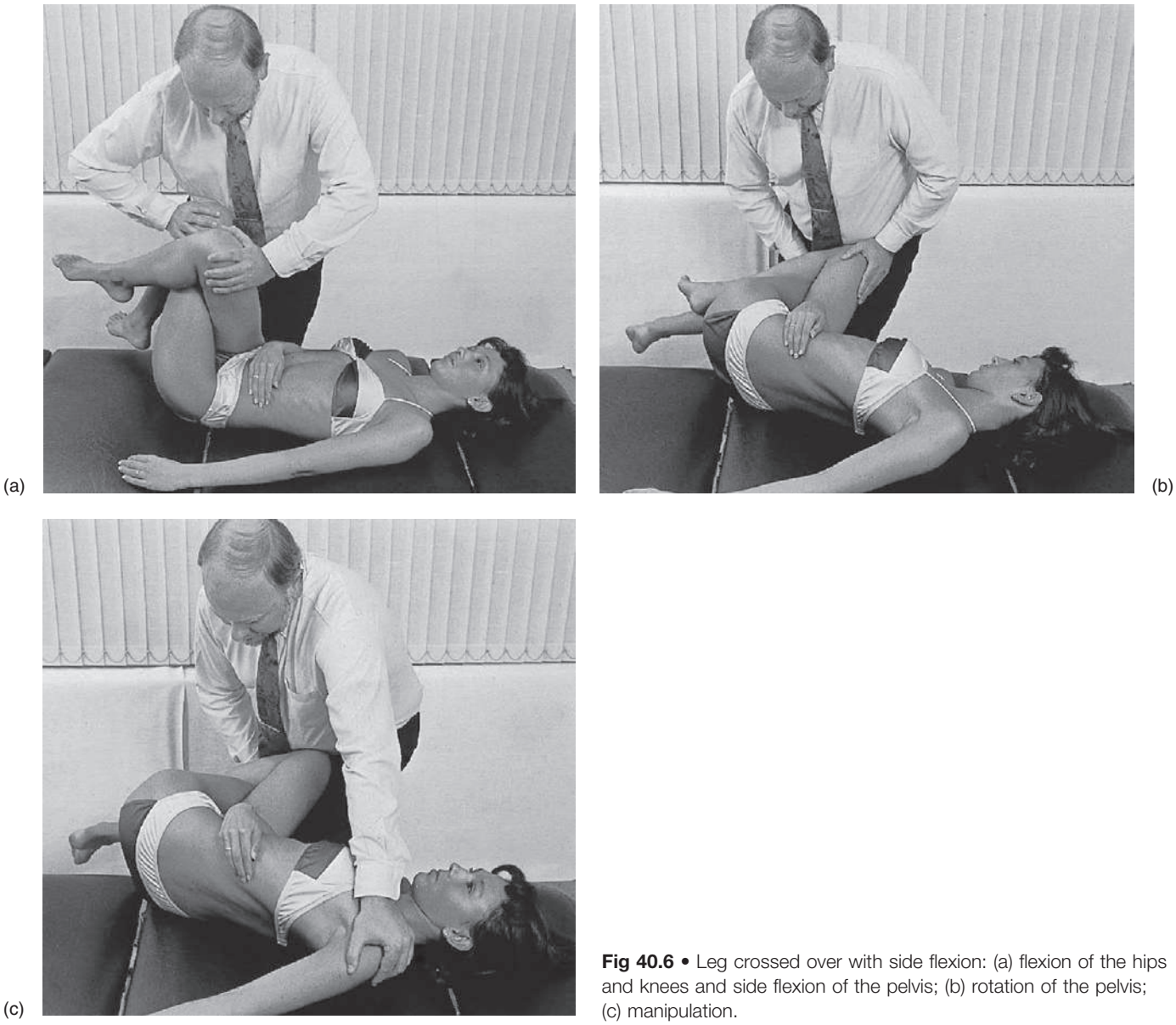


Fig 40.6 • Leg crossed over with side flexion: (a) flexion of the hips and knees and side flexion of the pelvis; (b) rotation of the pelvis; (c) manipulation.

pelvis is continued until tissue tension is felt to be maximal. At that moment, rotation is forcibly increased by pressing the patient's knee strongly and with high velocity towards the floor, using the thigh as a lever. At the same moment, the other hand maintains the position of the patient's far shoulder (if possible) flat on the couch.

Leg crossed over with side flexion

This manœuvre is a variation on the previous 'leg crossed over' technique and also achieves side flexion. The patient lies supine, both legs flexed and crossed, the leg on the painful side underneath. The manipulator stands on the painless side, level with the patient's waist. Holding the patient's knees in the hands, the manipulator moves the patient's hips into 90° of flexion. Then both legs are twisted, in order to tilt the pelvis laterally and open up the lumbar spine on the painful side. This position is maintained at full range. The hand that has been on the patient's uppermost knee is now freed to fix the far shoulder on the couch. The side-bent position of the lumbar spine is ensured by the manipulator's thorax and abdomen, which are used to engage the knee from the side. Next, rotation is stepped up slowly: under the influence of gravity, the legs turn in the direction of the floor until the limit of tissue tension is felt at the end of range. At that moment, the manipulator's thigh, engaging the uppermost knee from the side, has taken over to secure the side-bent position of the lumbar spine. Lastly, the hand at the knee is supinated to increase the manipulative force. Manipulation is performed by pressing the knee quickly downwards (Fig. 40.6). At the same moment, the other hand is used to maintain the position of the patient's far shoulder flat on the couch, if possible. Rotation is thus forced during side flexion.

Practitioner's checklist

- This manœuvre must not be performed in arthritis of the hip and in the elderly, where osteoporosis is to be expected
- Hip of the leg underneath should be flexed to 90°
- Lateral flexion should be maintained up to and including the final thrust
- Manipulation creates a quick but short downward thrust, without releasing the patient's far shoulder

Stretch Reverse stretch

Again, the couch is adjusted to about 30 cm in height. The patient lies on the pain-free side, close to the edge of the couch where the manipulator stands. The patient's upper hip is extended, the lower flexed to about 45° in order to stabilize this position. The upper arm hangs off the couch, the lower lies behind the back. The manipulator stands behind the patient, distal to the pelvis and facing the patient's head. The ipsilateral hand takes hold at the anterior iliac spine and twists the pelvis backwards as far as it will go. In this position, the manipulator's arm is fully pronated, with the hand placed against the anterior aspect of the anterior iliac spine, pushing the pelvis downwards and backwards. The other hand is placed against the scapula and pushes the thorax upwards and forwards (Fig. 40.7). Next, as the manipulator leans well over the patient, the joints are distracted by moving both hands in opposite directions, until tissue tension is felt to be maximal. Manipulation is performed by jerking the body downwards over the rigid arms. It is best to apply this overpressure at the moment of expiration.

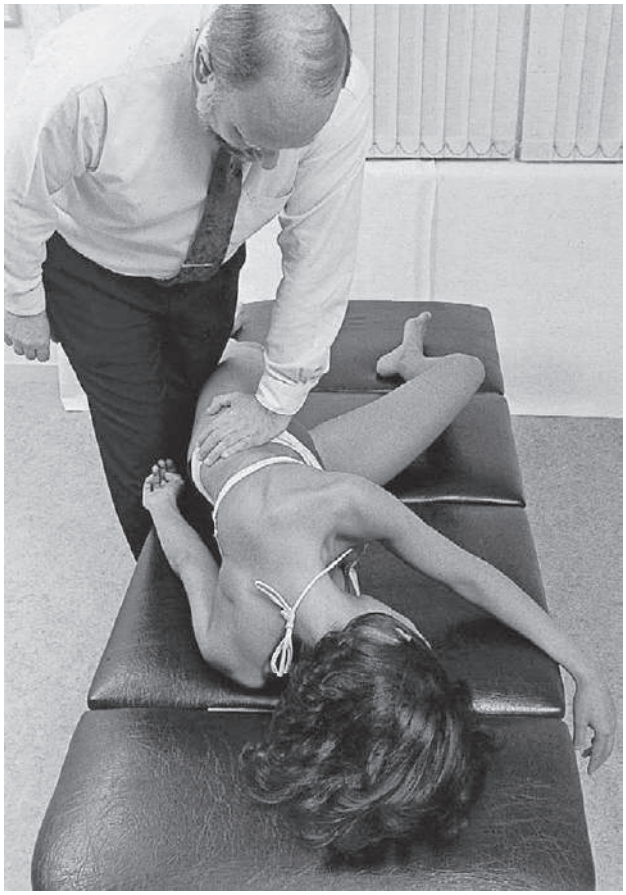
Practitioner's checklist

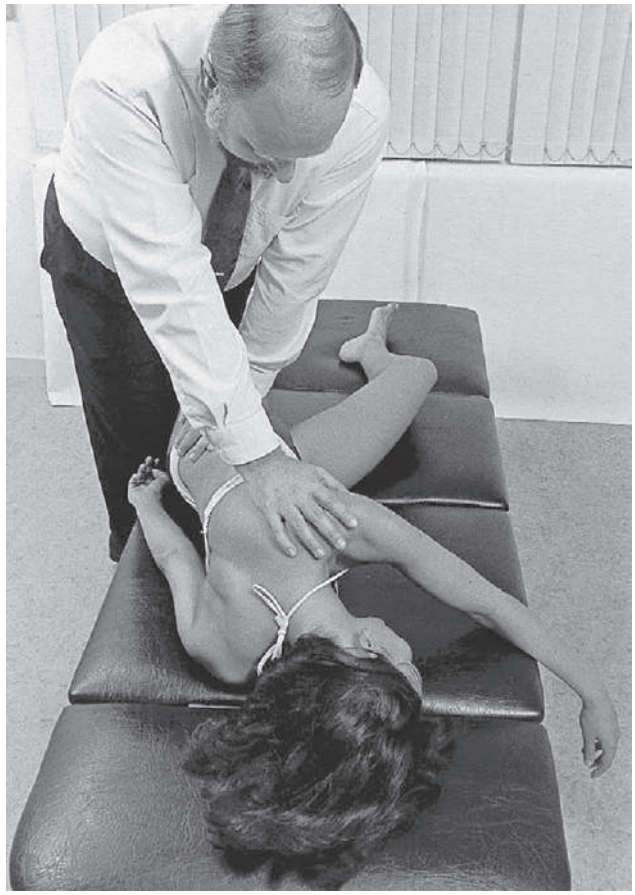
- Pelvis must be rotated well backwards; otherwise the manipulation compresses the pelvis instead of stretching the lumbar joints
- Angles of rotation should be equal
- Same amount of pressure is exerted by both hands
- Elbows are extended at the moment of the final thrust
- Body weight is brought well over the patient to reinforce the stretch but adapted according to the patient's constitution
- Manipulation is a stretch, not a rotation

Reverse rotation with thigh

The couch is adjusted to about 60 cm in height. The patient lies on the pain-free side, the upper leg extended and the lower hip flexed to 60°, with the lower arm behind the back. The manipulator stands behind the patient, level with the lumbar

(a)





(b)

Fig 40.7 • Reverse stretch: (a) the ipsilateral hand twists the pelvis backwards; (b) manipulation.

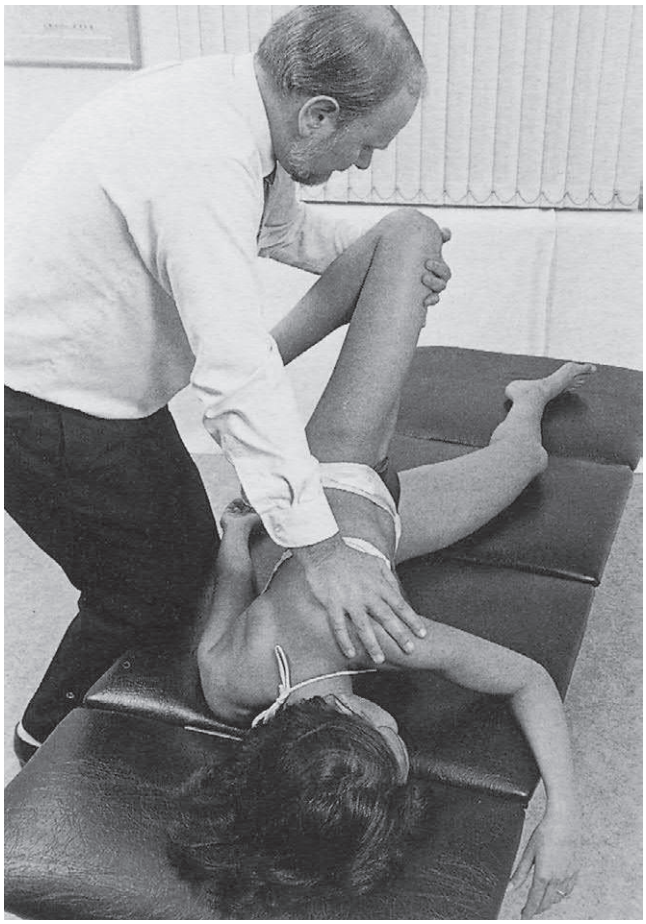


Fig 40.8 • Reverse rotation with thigh.

spine. The ipsilateral hand grasps the upper thigh at the knee and flexes the hip to 90°, abducting the thigh horizontally. As a result, the pelvis is twisted as far as it will go. The other hand is placed against the scapula and pushes the upper thorax to the couch (Fig. 40.8). While pressure is maintained on the thorax, the patient's upper thigh is now brought to 60° of flexion and full abduction. In some cases, it is also necessary to place a knee against the patient's lower buttock, to prevent the pelvis from slipping backwards. The moment that the manipulator feels the limit of tissue tension, manipulation is performed by a short, sharp rotation of the manipulator's body. This forces the arm at the thorax down, at the same time as it jerks the thigh backwards. Strong rotation and extension occur in the lumbar joints.

It is obvious that this manipulation must not be performed in the elderly or in patients with arthritis of the hip or osteoporosis.

Practitioner's checklist

- Suitable technique for lightly built manipulators
- Contraindicated in hip lesions, elderly patients and osteoporosis
- Upper thigh is in not less than 60° of flexion

Extension techniques

These techniques are very effective in small cartilaginous displacements that cause backache, especially in elderly patients and in those with persistent minor protrusions following incomplete reduction by a stretch in a rotated position. The techniques are milder than those performed with rotation strains and may substitute for the latter in osteoporosis. However, they affect one segment only.

During the manœuvre it is thought that interspinous pressure moves two adjacent vertebrae apart, so tightening the posterior longitudinal ligament and causing suction in the disc – a centripetal force which may reverse a displacement. The shape of the facets of L5–S1, which are more in a frontal plane, contributes to the better results achieved at this level than at the other lumbar segments. The impulse separates L5 from S1 in a cranial direction, instead of compressing the dorsal parts of the joint.

In acute lumbago and when extension pressure causes pain to shoot down the limb, extension techniques are contraindicated.

Deviation, as an expression of a large displacement, indicates that these techniques will almost certainly fail.

If a heavily built manipulator is dealing with a light patient, leaning on the patient's back using the whole weight of the body may give rise to strong resistance and therefore the amount of weight applied should be reduced.

Central pressure

The patient lies prone on a firm couch adjusted to about 30 cm height. The manipulator stands level with the lumbar spine, facing the patient, with the knees against the edge of the couch. One hand is placed with its ulnar border at the interspace of two adjacent spinous processes (normally between S1 and L5). The other reinforces it with the heel pressing on the radial and the thumb pressing on the dorsal and ulnar sides of the lower hand (Fig. 40.9). To prevent any contact with the iliac bones, it is useful to use the right hand, standing at the patient's left-hand side, and to turn this hand through about 45°. With the upper limbs extended and kept rigid, the manipulator leans well on to the patient's back and extends the knees, one after the other. From this moment the body weight presses fully on the patient's back and results in maximum tissue tension.

Practitioner's checklist

- Weight applied should be adapted when a heavily built manipulator deals with a light patient: one or even both knees remain in a flexed position and lean against the edge of the couch
- Lean well over the patient
- Arms extended and kept rigid
- If pressure of body weight shoots a pain down the limb, stop the manipulation

At the moment the patient relaxes and some extension has been achieved, the final thrust is given by bending the head and thorax abruptly forward. Usually a thud is felt or a click is heard if the manipulation is successful.

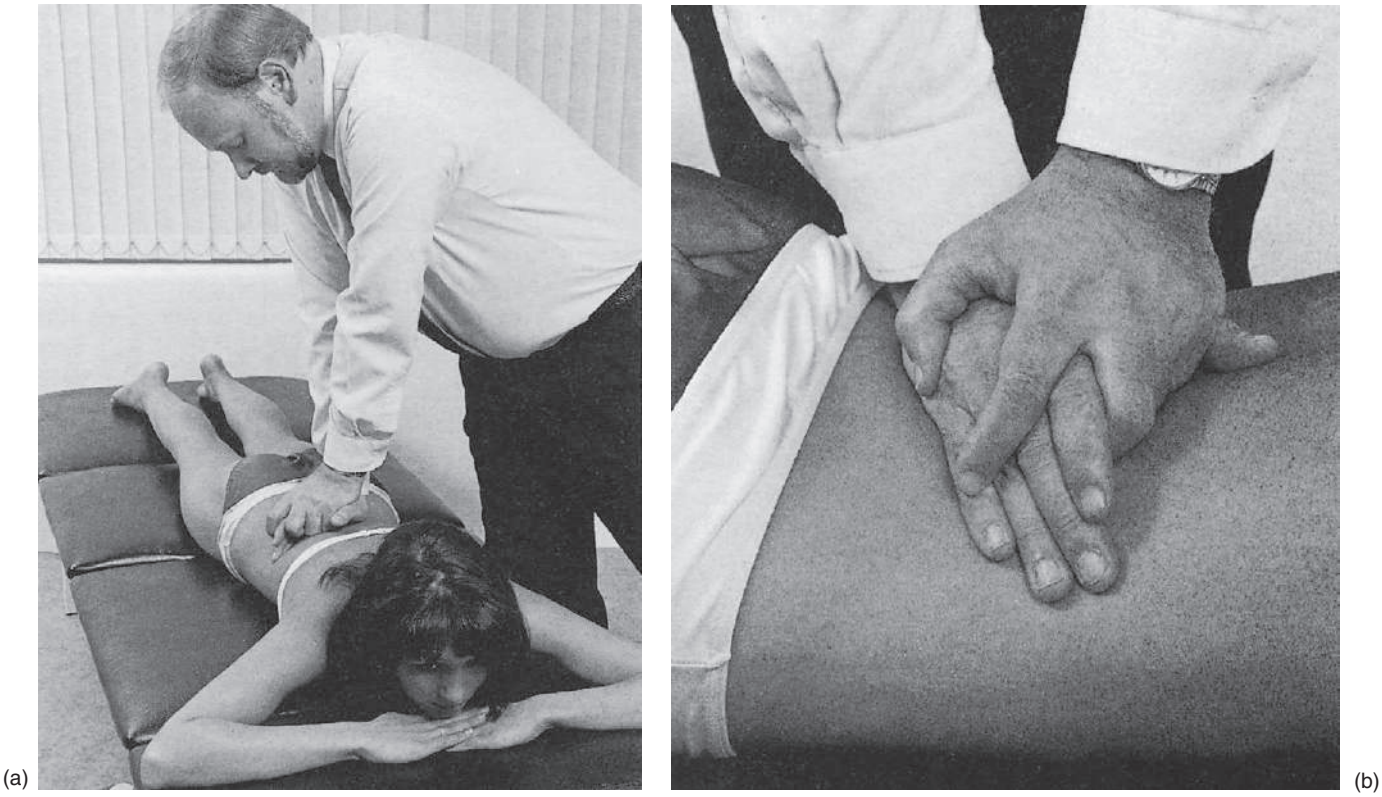


Fig 40.9 • (a, b) Central pressure; (c) the ulnar border of the hand is placed between two adjacent spinal processes.

Unilateral pressure

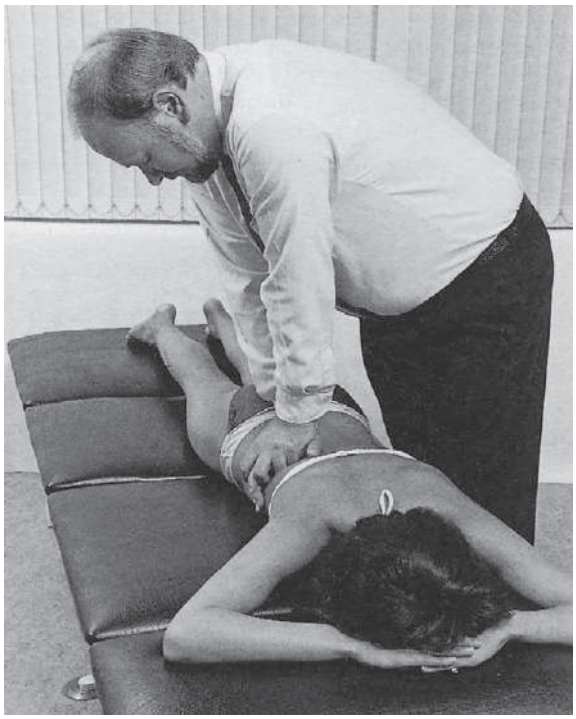
If repeated central pressure has neither fully relieved the patient nor made the problem worse, this technique is used immediately after central pressure.

The manipulator stands on the patient's painful side, although, if the pain is central, there will be no indication whether to start on the right or on the left. The wrist of the ipsilateral hand is extended and the prominent pisiform bone is used to exert localized and unilateral pressure at the base of the spinous process of L5 or L4. It is necessary to lean well over the patient, in order to press in a slightly oblique direction (Fig. 40.10). The other hand reinforces the pressure, using the heel to press on the manipulating hand. In order for the manipulator to stay well balanced, both legs are moved slowly backwards at the moment the body moves forwards. The knees or thighs should stay in contact with the edge of the couch.

Manipulation is all but identical to the previous technique, except that the thrust is now directed medially as well as downwards, which opens the joint on the painful side, at the same time also exerting some rotational stress and strong extension.

Practitioner's checklist

- Arms kept rigid
- The centre of gravity moves beyond the centre of the patient's body
- Knees or thighs stay in contact with the couch
- Final thrust at the moment the patient relaxes and all the slack in the tissue has been taken up
- If pressure of body weight shoots a pain down the limb, stop the manipulation



(a)

(b)

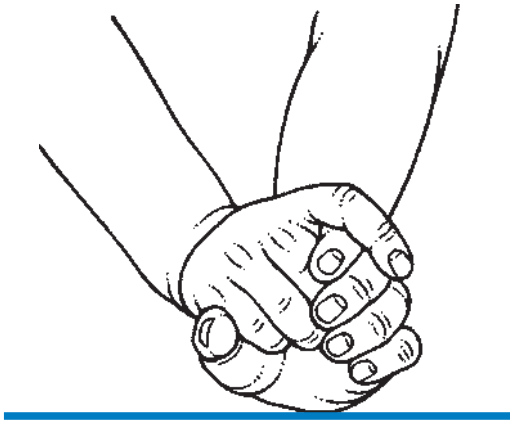


Fig 40.10 • (a) Unilateral pressure; (b) the prominent pisiform bone is placed at the base.

Unilateral pressure with thigh I

This is a much stronger technique, which follows the previous extension manœuvres but is undertaken only if partial reduction has been achieved and repetition affords no further improvement. In the absence of any benefit, it is unwise to continue with this technique or the next one.

The patient lies prone and near to the edge of a low couch. The manipulator stands on the pain-free side, level with the pelvis. With the ipsilateral hand, the front of the knee is grasped at the painful side around its lateral aspect. The ulnar border of the other hand is placed just above the posterior spine of the ilium. Then the hip is extended and strongly adducted by leaning heavily towards the patient's head (Fig. 40.11). This opens the joint on the side where the displacement lies. Manipulation is performed by a quick rotation of the manipulator's trunk towards the patient's head. In this way, the unilateral downward pressure of the lumbar hand and the upward pull of the hand on the knee are considerably intensified. This results in a combined movement of hyperextension, side flexion and rotation at the lower lumbar joints.

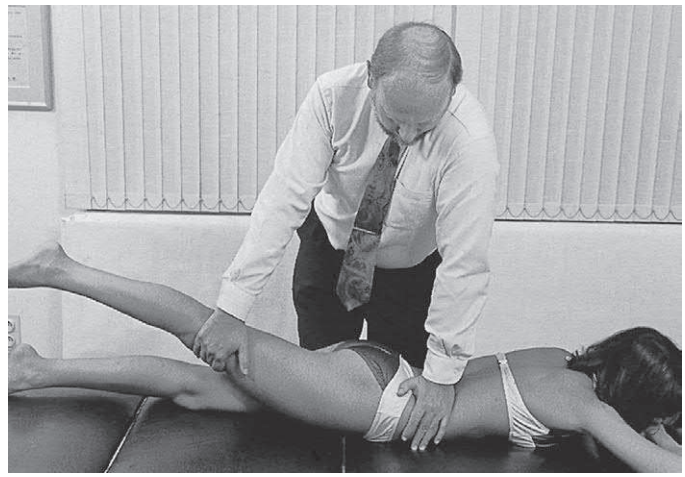


Fig 40.11 • Unilateral pressure with thigh I.

Practitioner's checklist

- Only use if the previous extension manœuvres have already achieved some reduction
- Not possible in a lesion of the hip joint
- Size of the patient should be comparable to that of the manipulator

Unilateral pressure with thigh II

With a heavily built patient, added force can be exerted by employing the knee; however, this technique should not be adopted if the previous extension strains have led nowhere.

The patient lies in the same position as in the previous technique. The manipulator stands on the painful side. With the contralateral hand, the front of the patient's knee is grasped around its medial aspect and the thigh is extended and adducted until the pelvis rises just off the couch. The palm of the other hand is placed on the sacrospinalis muscle covering the fourth and fifth lumbar levels on the painful side, with the forearm fully supinated (Fig. 40.12). The manipulative thrust is performed by pressing the ipsilateral knee with the hand at the same time as the patient's thigh is forced into full extension and adduction. A forced extension at the lower lumbar joints results.

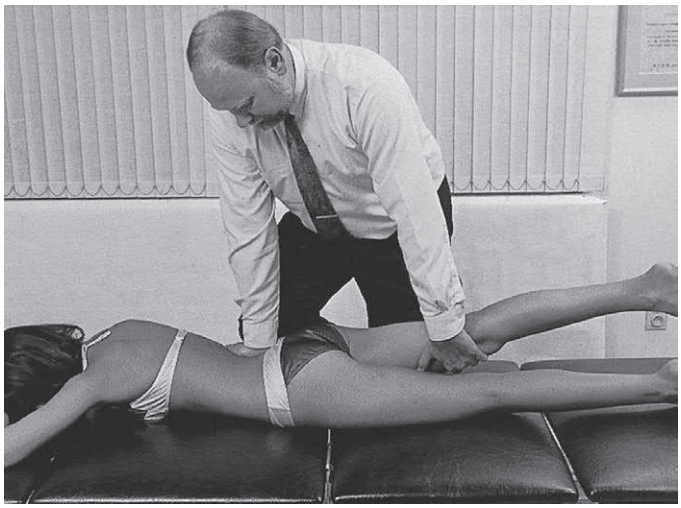


Fig 40.12 • Unilateral pressure with thigh II.

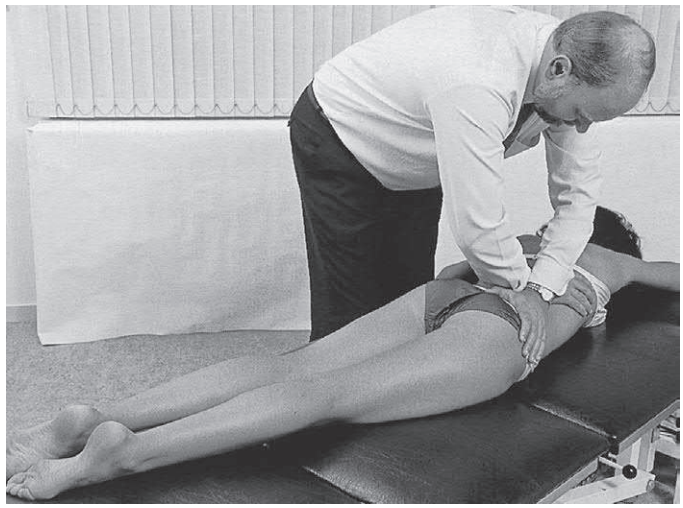


Fig 40.13 • Unilateral distraction.

Practitioner's checklist

- Use only if central and unilateral pressure techniques have achieved some reduction and the patient is heavily built
- Not possible in a lesion of the hip joint

Unilateral distraction

This technique is indicated if the previous manœuvres towards extension have helped but reduction has still not been fully achieved. It may also serve to remove a generalized ache that results from any manipulative manœuvre.

The patient lies prone and side-flexes the body to open the joint on the painful side as far as possible. The manipulator stands on the concave side, facing the patient, with the arms crossed and the elbows bent almost to a right angle. The heel of one hand is placed against the iliac crest, just lateral to the sacrospinalis muscle. The heel of the other hand is placed just under the lowest ribs (Fig. 40.13). To prevent the skin from being strained at the moment of manipulation, it is first pulled upwards with the lower hand, while the upper hand does the same downwards. Manipulation is now performed by repeated (10–20 times) forward movements of the trunk, keeping the elbows rigid. This forces the hands apart and imparts rhythmic further distraction, together with some extension at the lumbar level.

Antideviation techniques

These techniques are applied in backache and lumbago with an adapted posture, caused by posterocentral disc protrusions. The previous rotation and/or extension techniques will have already eased the pain, but when the patient stands for a few moments, the tilt of the trunk to one side quickly returns, as a result of persistent one-sided muscle spasm. On examination, side flexion towards the contralateral convex side, and sometimes extension, is still limited.

Three techniques can be used:

- Side bending in the supine position
- Rotation–distraction in side lying
- Side gliding, standing.

Side bending

The patient lies supine with both legs flexed and crossed, the leg on the concave side of the lumbar spine underneath. The manipulator stands on the convex side, level with the pelvis. With one hand the upper knee is pushed away, while the other is used to pull the lower knee towards the manipulator (Fig. 40.14). This simultaneous action tilts the pelvis and achieves full side flexion at the lumbar spine in the direction that was previously blocked. It is quickly repeated a number of times, whereafter the pressure is maintained for a few seconds. When there has been a previous nuclear protrusion, the extreme of range is better maintained for a minute or so. This position is consolidated either with the assistance of the manipulator's ipsilateral knee, pushing from a distal position against the patient's ischial tuberosity, or by using the contralateral knee to push from a lateral position against the patient's pelvis. The manipulation is repeated until the patient can keep the trunk in a neutral position on standing.

Rotation–distraction

The patient lies on the side of the lumbar convexity with the upper thigh flexed to about 60°, thereby rotating the pelvis to just over 90°. The manipulator stands in front of the patient, distal to the pelvis and facing the patient's head. The thigh of the uppermost lower limb is clasped between the manipulator's knees, just proximal to the patient's knee, to secure the position of the pelvis. Both hands are placed to one side of the upper thorax. Correction of the lateral tilt is achieved by pushing against the patient's thorax in an upward and backward direction (Fig. 40.15). This correcting force should be sustained for as long as the patient can endure it. The manipulator must stand well balanced to prevent the entire body weight

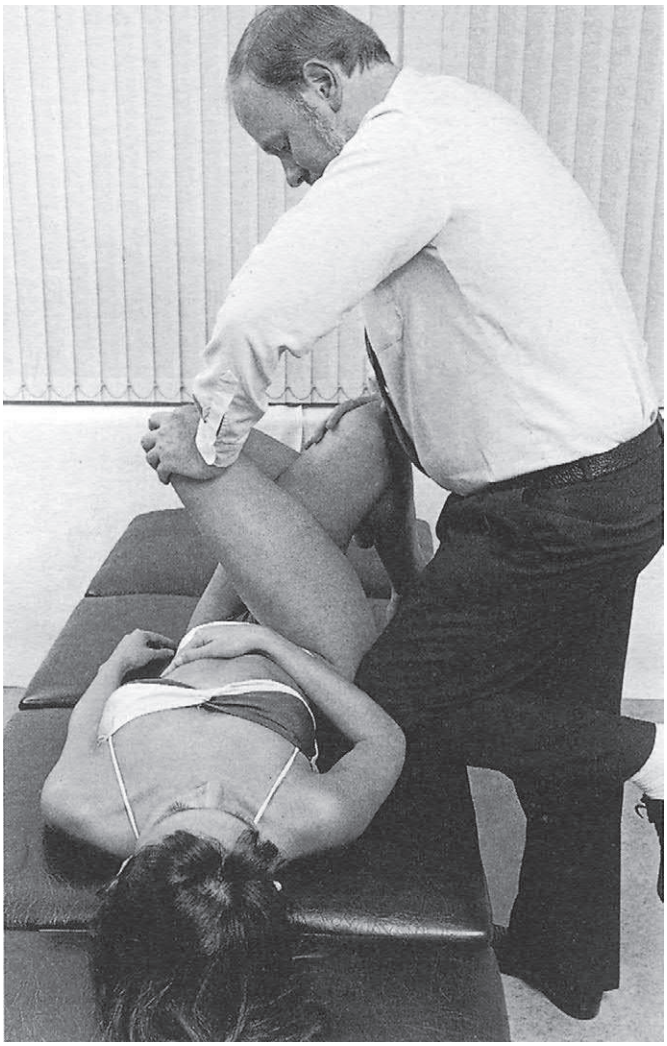


Fig 40.14 • Side bending.

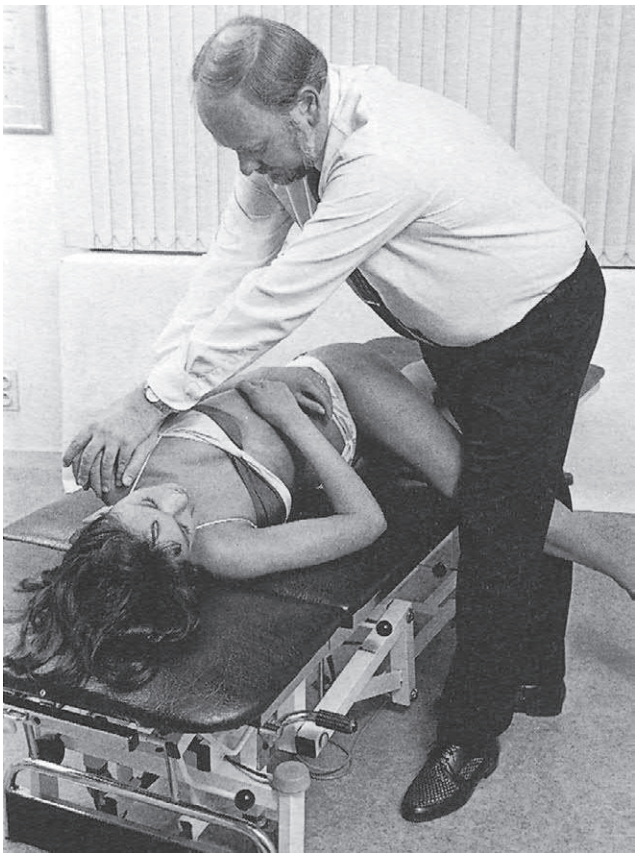


Fig 40.15 • Rotation–distraction.

from pressing on the patient. After some repetitions the patient is re-examined in the standing position.

The manœuvre is repeated until correction has been achieved or until repetition affords no further benefit.

Side gliding

The patient stands upright, the feet about 20 cm apart to provide a stable base, with the elbow held against the lower rib cage on the side of the lumbar concavity. The manipulator stands on the same side and presses the thorax against the patient's elbow, with the hands placed on the far side of the patient's pelvis.

Correction and even slight overcorrection is achieved slowly, by pressing the thorax against the patient's elbow, simultaneously pulling the pelvis from the far side towards the manipulator (Fig. 40.16). This pressure should be maintained for a couple of minutes and is repeated several times. It is essential that the movement is side-gliding rather than side-bending.

Once the spine is upright, an attempt is made to restore lordosis. To this end, the patient is brought into the corrected position again and asked to let the hips move forwards at the same time as the trunk bends backwards. In this way, the body stays well balanced all the time. This movement is repeated, until the range of extension is restored.

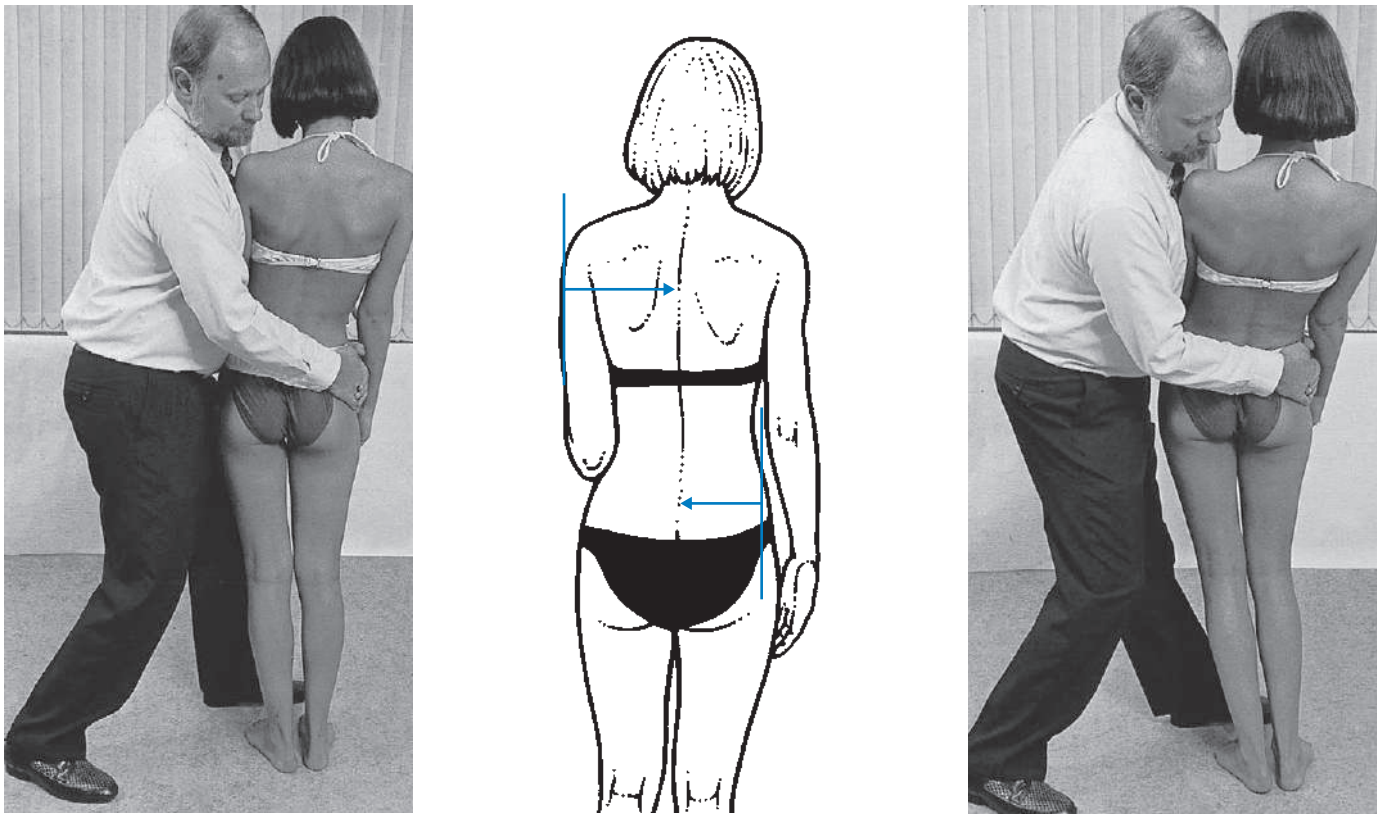
It will take at least 3–4 consecutive daily sessions to produce a lasting result. In addition, it is essential to instruct the patient in self-correction (Fig. 40.17).

Standing in front of a full-length mirror, one hand is placed against the lower lateral rib cage at the concave side of the lumbar tilt. The other hand is placed on the opposite lateral iliac crest. Then the patient performs the side-gliding movement of the pelvis in the restricted direction so as to correct the deformity. Once this has been achieved, a controlled extension movement is performed: the patient supports the trunk by placing the hands at the lower back and slowly bends backwards as far as is comfortable. These exercises should be repeated every hour.

Practitioner's checklist

- Correction involves side gliding, not side bending

The course of a manipulative session is summarized in Figure 40.18.



(a) (b) (c)

Fig 40.16 • Slide gliding: (a,b) correction; (c) extension.

Fig 40.17 • Self-correction.

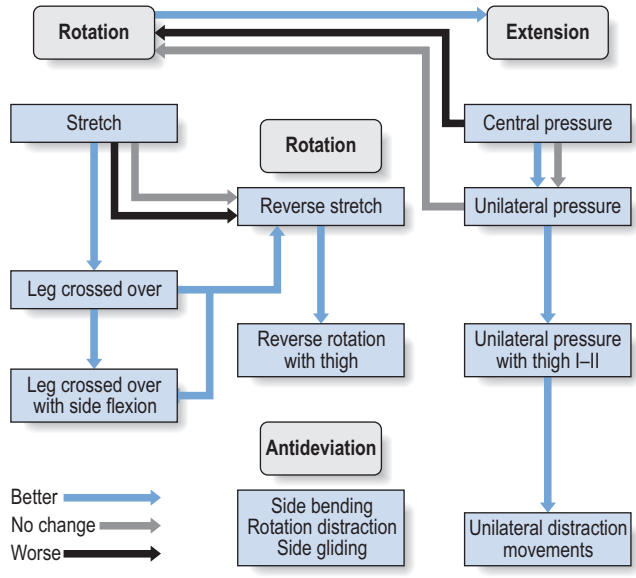


Fig 40.18 • Course of a manipulative session (see text for explanation).

Manipulation procedure

Choice of technique

- Stretch or reversed stretch in the side-lying position is usually the first technique to be tried, especially if the pain is unilateral.
- Extension techniques are chosen first in case of central pain and in elderly patients.
- Acute lumbago is unsuitable for extension techniques; rotation manœuvres usually give good results.
- Usually, L3–L4 protrusions respond better to rotation techniques.
- L5 protrusions may respond better to extension techniques, especially in elderly patients.
- In elderly patients it is also better to avoid long-lever techniques for fear of fracturing weakened bones.
- If rotation–stretch techniques lead to incomplete reduction in minor protrusions, one can move on to extension techniques.
- After the use of each technique, the result is assessed and a decision taken whether to continue with the same technique or to change. If one manœuvre has helped, it should be repeated until symptoms and signs no longer alter. Then another is tried. Experience, the result of each particular manœuvre, end-feel during exertion, the patient's age and estimation of tolerance all affect the types of manœuvre employed.

Assessment of progress (Box 40.4)

The physical signs are reassessed after each manœuvre. If there were dural signs, these are tested first: limitation of straight leg raising, before the manipulation, is tested afresh after the manœuvre. If there was pain on coughing, the patient is asked to cough once more and to describe how it feels.

It is only after the physical signs in the lying position have disappeared that lumbar movements are examined on standing to assess articular signs. In contrast, if it was only lumbar movements on standing that caused pain, the patient is asked to stand and bend backwards and sideways to judge whether there is any change in the degree of pain or in the amplitude of trunk movement. Because forward flexion movement is likely to increase any displacement, flexion of the trunk should not be tested before the manipulator can be sure that full reduction has been secured, i.e. all other tests have become negative.

Another important sign is 'centralization' of pain: a shift, after manipulation, to a more central position is regarded as an improvement.[58,80,81]

Assessment of outcome after each manœuvre assures the manipulator that:

- The displacement has moved.
- It has moved into the right direction.

Repetition of techniques

One manipulative session continues until symptoms and signs have been modified to the greatest extent possible. If a 'thud' is felt on performing an extension thrust and considerable improvement is noted, no more should be done until the next visit. If there is only a slight improvement, the same technique should be repeated until no further change occurs. In young patients, another technique can still be tried. However, 6–8 manœuvres are about the maximum a patient can tolerate per session. In elderly patients, it is better to stop after two or three and to continue on the next visit.

Sometimes a manœuvre may make a patient's symptoms worse. If this is the result of an extension technique, a rotation manœuvre can still be tried. If rotation makes it worse, the direction of rotation must be changed (reversed stretch instead of stretch, or vice versa). If these measures are unsuccessful, the question arises as to whether the patient is in fact suitable for further manipulative treatment. Manipulation is by no means free from danger if continued in spite of warning signals. Starting gently, paying attention to the patient's statements while being manipulated and re-examination after each manœuvre will avoid mistakes.

Box 40.4

Summary of improvement after manipulation

- Dural signs
  - Amplitude of straight leg raising
  - Pain on coughing
- Articular signs
  - Pain
  - Amplitude of lumbar movements
- Centralization of pain

Traction

Although there is still a great deal of controversy about the effectiveness of traction,[82] we still consider passive sustained stretching of the low back as the treatment of choice for nuclear, reducible disc protrusions causing backache and/or sciatica, unless there are specific contraindications.

Despite the poor design of most of the studies,[83] traction has been shown to be more effective than corsets, bed rest, hot packs and massages.[79,84–86]

Historical note

The Ancient Egyptians utilized the beneficial effect of axial traction.[87] An illustration of traction employed by the Spanish–Arabian physician Abu'L Qasim (1013–1106) of Cordoba is reproduced by Schiötz and Cyriax in their book on manipulation past and present.[20] In the same book, illustrations show the way in which traction was used by Hippocrates (400 BC) and Galen (AD 131–202). A 14th-century method of manipulation during traction is illustrated in Figure 40.19.

Nowadays, two methods of performing traction are practised. The sustained manner, as described in this book and first

suggested by Cyriax in 1950,[88] and several types of intermittent traction. Intermittent traction can be done either electrically, manually (by a therapist) or by the patient (autotraction). However, nearly all reported work has shown all types of intermittent traction to be *ineffective*.[89,90]

Effects of sustained traction

Several studies have investigated axial traction. It has been established that during sustained traction at least three effects result (Fig. 40.20).

The space between the vertebral bodies enlarges

This is an important precondition for a displacement to recede.

In young men, sustained traction of 60 kg, applied for 1 hour, results in an increased body length of 10–30 mm, which is thereafter lost at the rate of 4 mm/h.[92] In an excised lumbar spine, sustained traction of 10–30 kg increases each joint space by 1.5 mm.[93] Vertebral separation is greatest in those subjects with wide disc spaces and least where there is evidence of disc degeneration.[94]

The effect of lumbar sustained traction on stature has also been studied in 10 healthy young subjects; the investigators confirmed the significant increase in stature but also that this increase was over and above that known to occur when the load is taken off the spine by lying down.[95] The findings suggest that most of the vertebral separation takes place in the first 30 minutes. It has also been established that the enlargement between two consecutive lumbar endplates during normal traction is between 1.0 and 1.5 mm, which is 10–15% of the thickness of the disc.[96–98] Other studies demonstrate a widening of the lumbar intervertebral space of between 3 and 8 mm measured on radiographs of patients undergoing gravitational traction.[99,100]

The heavy lumbar paravertebral musculature normally exerts significant resistance to distraction. At least 30–35 kg of traction, not dissipated by friction, is required to influence the lumbar spine.[101] Other work has demonstrated that a traction force of at least 25% of the body weight is necessary to achieve distraction of the lumbar vertebrae against the inertia of muscular resistance of the body.[102] This supports an earlier study[103] in which any traction power less than 25% of body weight was regarded as a placebo.

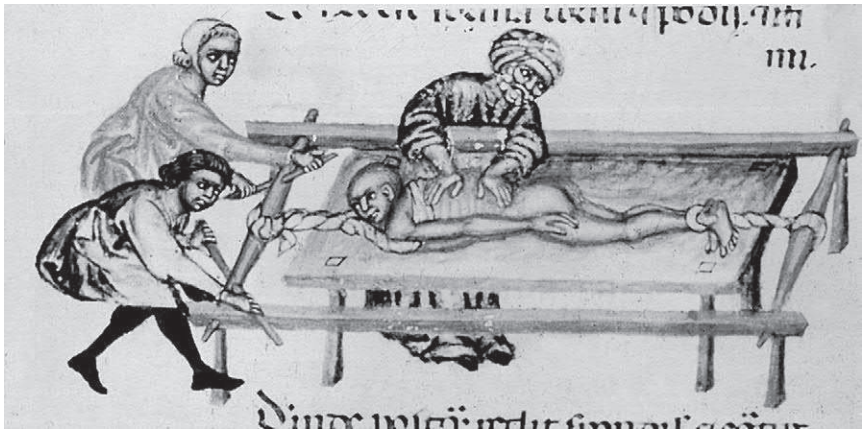


Fig 40.19 • Manipulation during traction in Albucasis's Surgery. Latin translation. Vienna, 14th century. (By courtesy of Wellcome Trustees.)

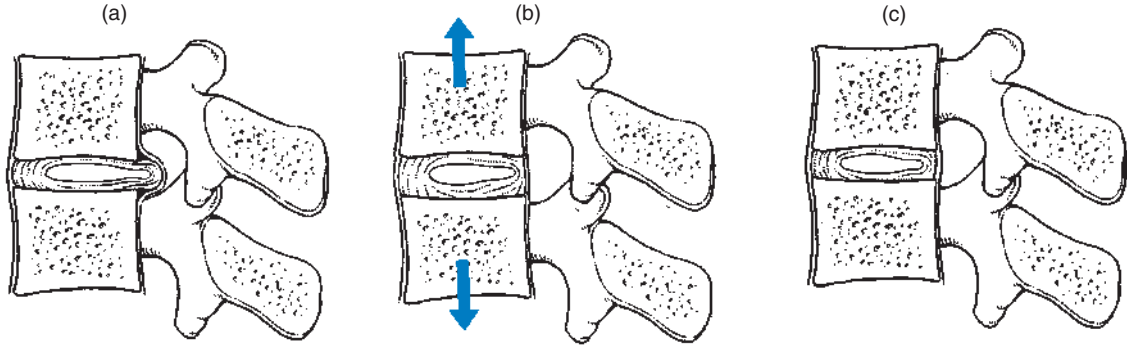


Fig 40.20 • Effects of sustained traction. (a) Before traction, nuclear protrusion posteriorly displaced. (b) During traction: the intervertebral space enlarges; the posterior longitudinal ligament is tautened; a negative intradiscal pressure is created. (c) After traction, reduction is maintained (after Mathews[91]).

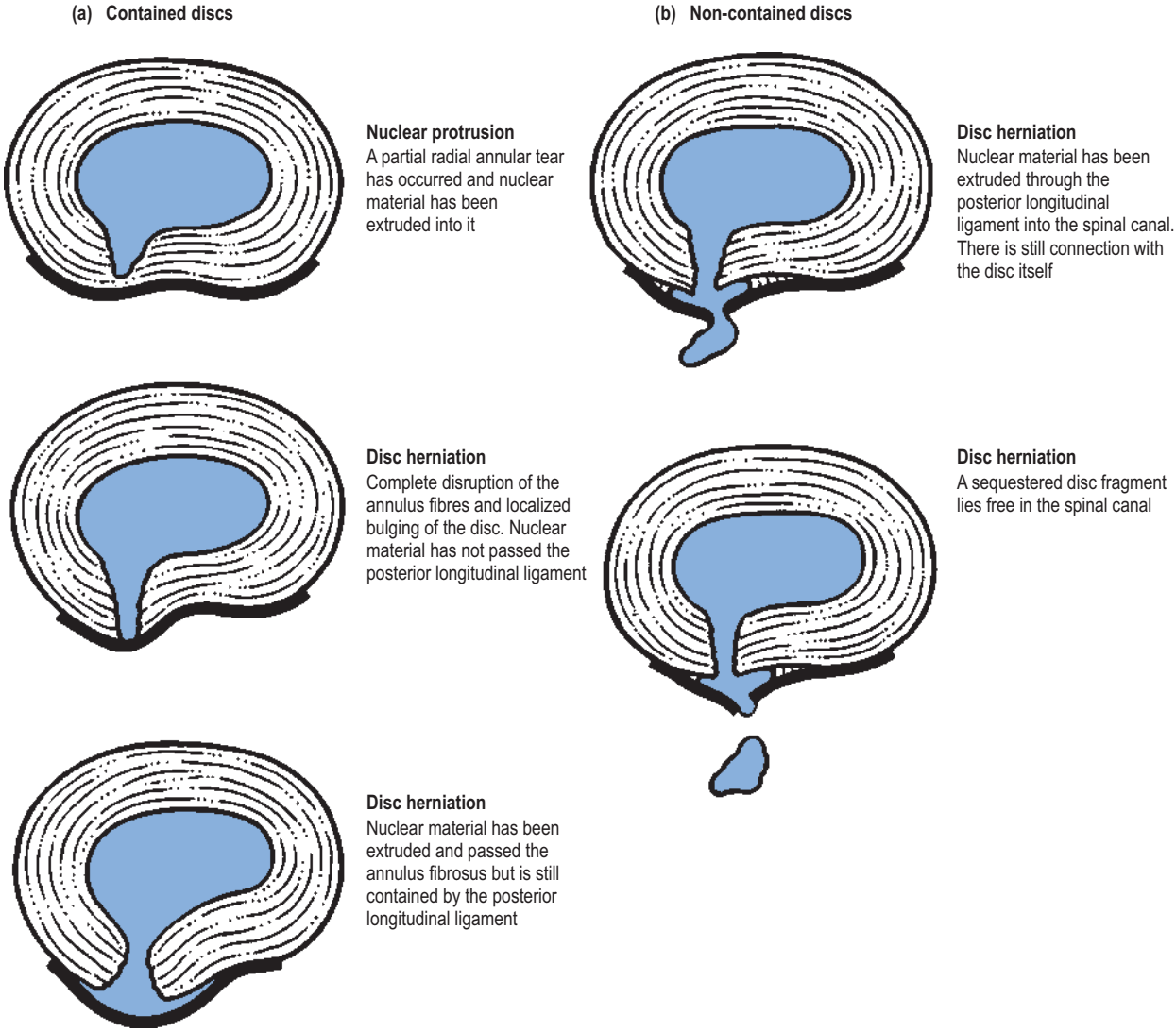


Fig 40.21 • (a) *Contained disc herniation*. Examples of displaced nuclear material that remains contained by annulus fibres and/or an intact posterior longitudinal ligament. Traction is indicated. (b) *Non-contained disc herniations*. Nuclear material has been extruded into the spinal canal. Traction cannot reduce the protruded material.

The posterior longitudinal ligament is tautened, exerting a centripetal force at the back of the joint

The increasing tension in this ligament is certainly of great therapeutic value, particularly if the protrusion is located anterior to, and remains in close contact with the ligament. Traction will therefore be less effective if the protrusion is laterally placed – a conclusion confirmed by computed tomography (CT) investigation of the effect of static horizontal traction on lumbar disc herniations: 'The clinical responses of the herniation to conservative treatment and the location of herniated nuclear material seem to be related. Traction is more effective on median and posterolateral herniation cases, and clinical improvement is evident in these cases, but traction is not very effective on lateral herniations.'[104] Also, re-entry of ruptured or sequestered disc material into the intervertebral disc is not possible (Fig. 40.21).

Suction draws the protrusion towards the centre of the joint

It is believed, on the basis of biomechanical calculations, that significant intradiscal negative pressure may be produced during sustained traction.[105] A traction load of 30 kg caused a lowering of the intradiscal pressure from 30 to 10 kp in the L3 intervertebral disc.[106] In another study, intradiscal pressure demonstrated an inverse relationship to the tension applied. Tension in the upper range was observed to decompress the nucleus pulposus significantly, to below 100 mm Hg.[107] Discography has established that the decrease in intradiscal pressure causes a suction effect with centripetal forces on the contents.[93] An interesting Chinese study investigated the changes in intradiscal pressure and intervertebral disc height on 31 prolapsed discs under traction. It was demonstrated that the intradiscal pressure decreased as the intervertebral distance increased in most cases under traction.[108,98]

Repair of the disc lesion

It has also been suggested[109,110] that, during episodes of disc decompression, nutrition is improved, reparative collagen is deposited and natural healing of annulus tears and fissures is promoted.

Sustained traction has the same effect on the intradiscal pressure as prolonged bed rest, but much more strongly. A few hours' traction achieves as much or more than bed rest for weeks. Although the latter can also bring about slow reduction of a nuclear protrusion, traction has the advantage of speed. Instead of simply avoiding the compression produced by the upright posture, it mechanically distracts the joint. In addition, the patient remains ambulant, which is far preferable to bed rest for several weeks – the latter not only is bad for morale but also increases the cost in lost working days and the payment of sickness benefit.

It is important to emphasize that this effect can only be achieved if traction brings about more in the way of reduction in half an hour than can be reversed during the rest of the day by new loading effects. To this end, traction should be sustained and must be given daily and as energetically as the patient can bear. Otherwise, it is merely a placebo.

There is increased motor activity of the sacrospinalis muscles on an electromyogram during traction, until the mechanoreceptors in the tendons are stimulated.[111] From that moment, motor activity is inhibited, the intervertebral joint takes the strain and reduction of the pulpy mass starts slowly. Electromyographic silence is reached after 3 minutes. This suggests that traction must be sustained. A study that measured the intradiscal pressure during 30 seconds of passive traction performed by two therapists and during 2 minutes' autotraction with 50 kg weight[112] has established that intradiscal pressure did not alter much in passive traction, whereas autotraction increased the pressure considerably. These findings strongly contrast with those of sustained traction, which makes it obvious that only the latter is able to diminish a nuclear protrusion in volume and return it to its normal position.

Indications for traction

Nuclear disc protrusions

Pulpy nuclear protrusions which remain contained and in contact with the posterior longitudinal ligament (see Fig. 40.21) are more effectively treated by traction just as hard annular protrusions are more readily treated by manipulation. Cyriax always said: 'You can hit a nail with a hammer, but treacle must be sucked.'

It is important to emphasize that signs of irreducibility, such as neurological deficit or gross lumbar deformity maintained by root pain, should be absent.

The typical clinical pattern of a lumbar nuclear protrusion is as follows. The patient is under 60 years of age. On bending forwards for a while, some aching in the back is initially felt, which gets slowly worse later in the day. The next morning, it is impossible to rise out of bed because of severe low back pain. Clinical examination shows a partial articular pattern and movements pinching the lesion, i.e. side flexion towards the painful side or extension, are often most painful.

This is quite different from the patient with an annular protrusion, who describes a sudden onset of lumbago on bending forwards, perhaps lifting a heavy object. This may happen at any age. Again, a partial articular pattern is present. A painful arc, with or without momentary deviation, suggests a small mobile annular fragment and is a further encouraging sign for manipulation.

The distinction between these different types of disc protrusion is not always as clear as in these examples. Nevertheless, for therapeutic reasons, it is important to differentiate between these mechanisms. The summary given in Table 40.1 may be helpful.

Indeterminate protrusions

If signs and symptoms do not indicate the consistency of the protrusion very precisely, manipulation is tried first because it may be effective much more quickly. If it fails or succeeds only partly, sustained traction is indicated. However, traction should not be given immediately after the manipulation but should be started the next day. There are two reasons for this:

- Painful twinges may occur after release if traction follows manipulation too quickly.
- It is not possible to judge the effect of the traction if it is combined with other forms of treatment at the same session.

First and second lumbar disc lesions

At this level, traction is regularly successful, whereas manipulation always fails. A disc lesion secondary to lower lumbar arthrodesis constitutes an exception: the fixation of the low

Table 40.1 Differences between nuclear and annular protrusions

	Nuclear protrusion	Annular protrusion
History		
Age (years)	<60	All ages
Onset	After a lot of stooping and lifting	During bending forwards and coming up again, abrupt displacement with a click, initiating acute lumbago
	Pain increasingly evoked by sitting in a kyphotic posture	
	Backache after exertion	Backache as soon as exertion starts
Clinical examination	Partial articular pattern	Partial articular pattern
	Pain on pinching the lesion in backache (not in lumbago or over the age of 60)	Pain on side bending away from the painful side

lumbar segment alters the biomechanical relationship in such a way that manipulation can often succeed.

Recurrence after laminectomy

Manipulation can be tried first but seldom proves successful if the protrusion is situated at the level of the operation. Traction is more often effective, although the prognosis remains less favourable than for those patients who have not had surgery.

Primary posterolateral protrusions

These protrusions may constitute a difficult therapeutic problem. Because they all consist of nuclear material, manipulation has no effect. The protrusion usually responds to daily traction but the tendency to relapse after reduction is considerable. Epidural local anaesthesia is a good alternative but only works when the protrusion is at its maximum size.

Considering the therapeutic approach in a primary posterolateral protrusion, Cyriax[21] (his p. 317) gives the following advice: '[S]uch a protrusion of a month or two's standing should be reduced by daily traction.' However, if relapse occurs after a successful reduction, or the protrusion has persisted for 3 or 4 months, it is better to leave it where it is, especially in a young patient with slight pain only, which is the common situation. Cyriax again: 'Spontaneous recovery usually takes nine months from the onset of root pain, and the strong tendency to recurrence is largely obviated by allowing the patient to get well of himself. [However] he should be kept under observation until the protrusion is stable at its maximum size, i.e. the range of straight-leg raising has stopped decreasing and is found unaltered at two examinations a fortnight apart. This is the moment for one or two inductions of epidural local anaesthesia which usually abolish the root pain in a few weeks.'

Backache together with bilateral long-standing limitation of straight leg raising

Those affected are mainly young adults with months or even years of backache associated with marked bilateral limitation of straight leg raising. Daily traction has proved to be successful but only if it is continued for about 3 months. It may take at least 1 month before any improvement is noted.

Pain with fourth sacral reference

Symptoms and especially signs of impaired conduction of the S4 root probably call for immediate surgical intervention. Nevertheless, if pain is the only symptom and paraesthesia, referred to the coccyx or the genital area, is absent, traction may first be attempted with caution. However, this measure is not entirely safe and should only be performed after discussion with the physician in charge.

Contraindications

Acute lumbago

Because lumbago with twinges is made so much worse for several days even by just one session of traction, traction is *absolutely* contraindicated. Even in patients with lumbago who have had no twinges recently, the first session of traction must be undertaken very cautiously. It is not during traction, but rather at the moment when tension is diminished, that the patient gets agonizing twinges, making it impossible to release the traction at that point. It may take the patient as much as 3 or 4 hours to rise from the couch. Cyriax advised a very slow diminution of the traction force. Then, once released, it can take a good 15 minutes for neck flexion to stop hurting and the range of straight leg raising to begin to increase. If twinges still persist on initiating movements, repeated rotation manipulations (leg crossed over) should be carried out, gently at first. If these fail, epidural local anaesthesia must be induced without delay.

Abdominal surgery or hiatus hernia

Patients who have had previous abdominal surgery or hiatus hernia may not tolerate abdominal compression.

Respiratory or cardiac insufficiency

The patient may not be able to lie down at all, let alone tolerate the harness.[113]

Respiratory irritation

In discodural interactions, pain may be evoked by coughing or sneezing as a result of the sudden increased expansion of the dura pressed against the protrusion. During traction, a powerful cough or sneeze is often painful too, making it impossible for the patient to relax. For this reason, respiratory problems should be dealt with first.

Painful reactions

If traction increases pain in the back or in the leg, the patient is unsuited to it. This often happens in chronic low back pain and sciatica, where the lumbar spine is fixed in flexion. Pain that increases as soon as the traction starts may indicate a worsening of the condition and is therefore reason enough to stop the treatment.[114]

A large protrusion

In gross lumbar deviation, any attempt to move in the contrary direction is aborted by sharp pain; traction is therefore not applicable. Discodural and discoradicular interactions with a high degree of inflammation (indicated by continuing pain or pain at night) are also best treated by epidural injections.

Mental state

Some patients cannot tolerate restrictions such as the harness used. The moment the thoracic harness is put on, they become very anxious and start to hyperventilate. In this case, it is better not to continue the treatment.

Cases in which traction is not useful

Although not really contraindicated, traction has no effect and should therefore not be applied in the following circumstances.

In patients with neurological deficit or gross lumbar deviation, the protrusion is larger than the pathway through which it emerged. Such material extruded through the outer rim of the annulus certainly cannot be reduced by traction or manipulation.

Box 40.5

Traction

Indications

- Contained nuclear protrusions
- Indeterminate protrusions
- First and second lumbar disc protrusions
- Recurrence after laminectomy
- Primary posterolateral protrusion
- Backache with bilateral limitation of straight leg raising
- Fourth sacral root pain (?)

Contraindications

- Acute lumbago
- Abdominal surgery
- Hiatus hernia
- Respiratory or cardiac insufficiency
- Cases in which traction increases the pain
- Psychogenic patients

Not useful

- Non-contained protrusion
- Large protrusion
- Long-standing clinical features

In the presence of a free protrusion or sequestered fragment, disc herniation traction will always fail.

In both these instances, an epidural injection may relieve the symptoms and, given adequate clinical and neurological justification, surgery may be indicated.

Long-standing clinical features

A primary posterolateral protrusion lasting longer than 3 months is better not treated by traction, especially in a young patient with only slight pain. There is a strong tendency to relapse after reduction, whereas spontaneous recovery takes place within 9 months of the onset of root pain.

A secondary posterolateral protrusion in a patient under 60 years old, causing root pain for more than 6 months, has passed the time limit for traction. The treatment of choice is epidural local anaesthesia. Spontaneous recovery is the rule and is to be expected within a few months.

Indications for and contraindications to traction, and cases in which it is not useful are summarized in Box 40.5.

Traction procedure

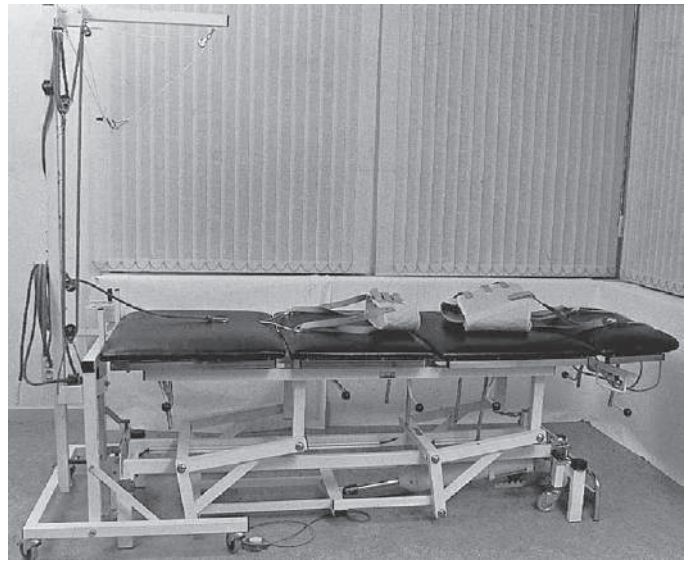
Traction apparatus

This consists of a couch and an electrically or manually operated machine, assembled at the foot of the couch.

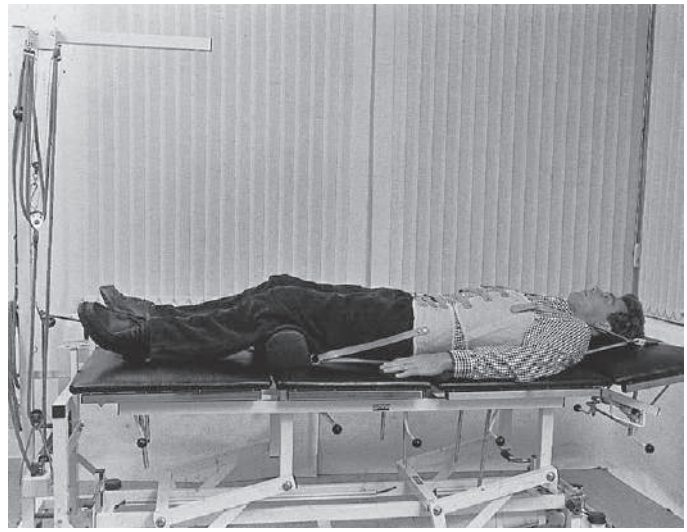
In the non-electrical form, a spring balance, attached to the traction rope, is necessary. It measures the amount of traction and ensures an even degree of traction. Whenever the pelvic belt slips slightly over the bench, the buffer mechanism of the spring balance intervenes to take up the slack and subsequently optimum force must be restored. The attendance of a therapist is consequently required, especially during the first 15 minutes of application, when traction force is regularly lost. In the electrical form, an electric device obviates this, although the therapist should remain within hearing distance because automatic machines are not 'fail-safe'.

There is mechanical equipment that has the advantages of the electrical appliance: constant traction force during the entire session (Fig. 40.22).

The couch should have an opening through which the patient can breathe when treated in a prone position. Frictional resistance in the system can be overcome by using a split-table model. However, the same results can be attained with an ordinary couch, although frictional resistance must be compensated for by a greater degree of traction force.



(a)



(b)

Fig 40.22 • (a) Mechanical traction apparatus and harnesses for continuous lumbar traction. (b) Patient in position for continuous lumbar traction.

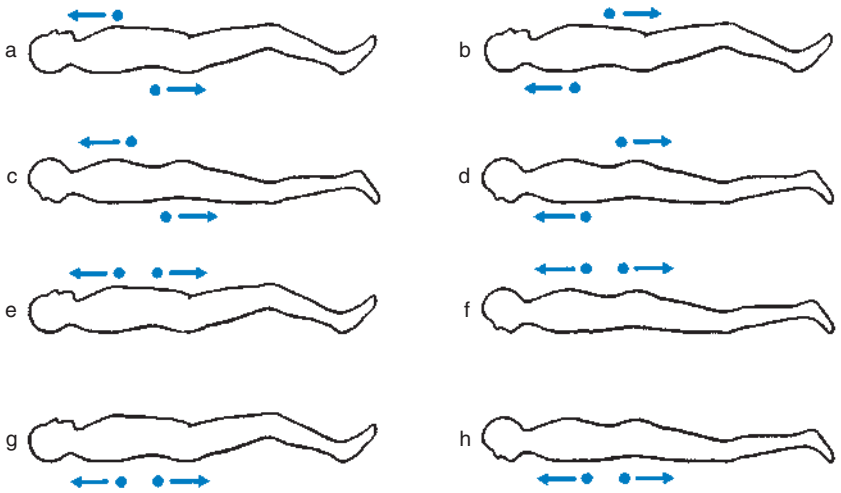


Fig 40.23 • Eight positions for lumbar traction (arrows indicate the position of the straps and direction of the pull).

Thoracic harness

The thoracic harness is tightened over a layer of thick foam rubber or a folded towel, placed outside the patient's clothing. This harness is placed as low down the thorax as possible, to confine the traction to the lumbar spine. Sometimes, though, patients cannot bear this position and fixation at a higher level is then required.

The harness anchors the patient at the head of the couch. The straps should clear the patient's trunk when applied above (not shown). For this reason, the upright, at the head of the couch, should be at least 30 cm high.

Pelvic harness

This is also placed over a folded towel or layer of foam rubber, and encircles the entire pelvis. This belt takes its purchase from the iliac crest, and connects to the traction rope.

For the same reason, in order to clear the patient's body, when applied above, the height of the *traction rope* should be adjusted to 30 cm.

Patient's posture

The possibilities are *prone* or *supine*. Cyriax suggests the position that the patient finds most comfortable in bed (provided that this is either prone or supine) as the best guide for the first treatment. If this is of little help, the degree of pain and/or limitation during flexion and extension should be assessed:

- *If both flexion and extension are painful* (or neither), the patient may lie supine or prone. One strap is situated posteriorly, and the other anteriorly. In this way, the articular surfaces stay parallel during distraction (Fig. 40.23a–d).
- *If flexion hurts* and extension is pain-free, traction is carried out with the lumbar spine in slight lordosis. The patient is positioned supine with a small pillow supporting the lumbar spine, or lies prone. In both positions, the straps of the harnesses are situated anteriorly (Fig. 40.23e,f).
- *If extension hurts* and flexion is pain-free, traction is carried out with the lumbar spine in slight kyphosis. The patient is positioned supine with the lower legs supported on a small bench and the knees bent upwards, or lies prone. In both positions, the straps of the harnesses are situated posteriorly (Fig. 40.23g,h).

Thus, eight different positions may be used. In practice, however, the positions regularly used are:

- Supine (seldom prone), in kyphosis, lordosis or neutral position
- 'Psoas position', with the hip and knee joints flexed to 90°. This position is particularly indicated in L3 root lesions.

Instructions to the patient

Before the session, eating a large meal should be avoided. The patient is informed about the underlying disorder and the therapeutic effects that can be obtained with sustained traction, and is also encouraged to stay as relaxed and as still as possible during the stretch. Coughing and sneezing should be avoided, because either may cause a twinge and after-pain. The patient is further assured that traction should never be painful while it is being applied; nor should it increase pain that already exists. Any discomfort should be reported at once. This explanation will allay any fears engendered by the similarity of the apparatus to a medieval torture rack.

Force and duration

Theoretically, the pull is as strong and lasts as long as the patient can tolerate. Usually, this will be 40–60 kg, applied for at least half an hour to be effective. In practice, the amount of traction is measured in relation to body weight. Traction with 60% of the body weight is considered to have the best ratio of effectiveness to tolerance.

Interval between treatments

Traction must be given daily, as the intention is to achieve greater reduction in half an hour than the patient can reverse

by weight bearing on the joint for the rest of the day. It is remarkable that this should be possible. Furthermore, traction is normally still effective with an interval over the weekend, provided that the patient relaxes on these days – lying down at regular intervals and not sitting for a longer time than is absolutely necessary. If these rules are neglected, the protrusion is likely to have returned to the same size by the time of the next visit. In an urgent case, traction can be performed more than once a day. Alternatively, one long stretch of up to an hour has proved effective in these circumstances. Passive back extension and prone lying can also be useful supplements.[58] If traction has not begun to have an effect after a few days, the question is whether to use a different position on the couch or a different position for the straps. If these alterations are made and the patient has not begun to improve after 2 weeks, traction should be abandoned. The physiotherapist should not despair too soon, however, as many patients only begin to improve during the second week. Obviously, if a patient is much better but not completely well after a fortnight, a third week's daily treatment is justified. In young adults with backache and long-standing bilateral limitation of straight leg raising, it may even take a month before any improvement is noted.

Procedure (Box 40.6)

(Re)examination

The patient must be (re)examined before each session. Symptoms and some physical signs – for example, lumbar movements and straight leg raising – are noted afresh and any alteration given due consideration. However, there is no point in examining a patient immediately after a session of traction: the transient changes, for better or for worse, detected at this point mean very little.

Box 40.6

Traction procedure

1. Inform the patient about the technique, the mechanism and the possible side effects.
2. Examine the patient each time before traction is applied.
3. Look for the most comfortable position.
4. Apply the straps firmly but not too tight.
5. Start the traction slowly, reaching 30–35 kg after 1 minute:
   First occasion: traction force may remain low.
   Further occasions: increase to 60% of the body weight.
6. Keep the applied traction force steady for about 30 minutes.
7. Release the traction force slowly – over 2–3 minutes.
8. Allow the patient a few minutes' rest before getting up.
9. Perform gentle active and passive movements of pelvis and hips before getting off the bench.
10. Patient's back should be kept straight on getting off the bench.
11. Instruct the patient about activities of daily living and allow him/her to walk a short distance before getting into a car.
12. Give traction on a daily basis for 2–3 weeks. Treatment must be abandoned if no improvement is experienced after seven or eight sessions.

Traction

The patient then lies down on the couch in the position that is considered to be most effective and the harnesses are applied evenly and firmly but not in such a way that the patient tenses up. Finally, traction is started slowly, reaching 30–35 kg within a minute. As the patient becomes accustomed to the pull, traction can be increased to reach the maximum that is tolerable. However, on the first occasion, the traction force should be low – say, 30 kg for a person of moderate body weight – and applied for no longer than 15–20 minutes. Care should be taken, particularly in acute lumbago, when twinges have only recently ceased. The therapist should always be within earshot and from time to time must directly observe the patient. This helps the patient to feel that the situation is being continuously monitored. It is still useful to ask the patient regularly about any discomfort because, although instructions will have been given about reporting discomfort, some patients still accept painful reactions. Anything more serious than the minor discomfort caused by the harnesses should not be disregarded.

Release of traction

When traction has been completed, particular care is exercised while the tension is being released and the harnesses loosened, for this is the time when twinges are most likely to occur. To prevent this unpleasant experience, release is slow – over, say, 2–3 minutes. The harnesses are then loosened carefully, the pelvic one first because this exerts least influence on the lumbar segments. The patient is also reminded of the need to remain quite still and to resist the temptation to take a very deep breath as release is completed. Five minutes' further rest on the couch before getting up is also advisable in that it gives the patient a chance to 'regain normal length' before compressing the joint by standing. Next, the low back is moved a little, first by flexing/extending each leg slowly in turn, then by tilting the pelvis. If this proves possible without twinges, the patient can get off the bench; this should be done keeping the back straight by rolling on to one side, putting the feet over the edge of the couch and rising sideways to a sitting and then a standing position. Any pain indicates that the patient spend another 5 minutes on the bench before trying to stand up again.

After-care

The patient is then shown how to put shoes on and how to sit in a car (see p. 583). If the lower back still feels stiff on leaving the room, it is better for the patient to take a short walk before getting into a vehicle. A lumbar support – for example, a rolled towel – is often useful to prevent the patient from sagging. In this way, the intradiscal pressure is kept as low as possible. However, during the whole period of treatment, the patient should be instructed to avoid sitting and stooping as much as possible.

Results

In most patients with discodural low back pain and sciatica, in whom manipulations have been insufficient, traction proves effective. The nuclear protrusions recede over the course of 2 or 3 weeks, while the patient is ambulant. First of all, the pain eases. Gradually, straight leg raising returns to full-range. Lastly, trunk movements cease to hurt. When the patient is

almost better, a painful arc often appears on straight leg raising.

One study measured the effects of lumbar traction with three different amounts of force (10%, 30% and 60% of body weight) on pain-free straight leg raise test of 10 patients suffering from sciatica caused by discoradicular interactions. The straight leg raise measurements were found to be significantly greater immediately following 30% and 60% of body-weight traction, as compared to 10% of body-weight traction.[115]

The effect of sustained traction on herniated nuclear material has also been investigated with CT. One study investigated the regression of displaced nuclear tissue during and after traction.[104] Patients were between 20 and 40 years of age. The duration of traction application was 40 minutes, with an applied load of 45 kg. Evaluation of the results showed a regression of herniated nuclear material in 78.5% of median herniations, 66.6% of posterolateral herniations and 57.1% of lateral herniations. Low back and leg pain decreased in all cases, except when the disc was fragmented or calcified. A recent, prospective, randomized, controlled study investigated the effects of continuous lumbar traction in patients with lumbar disc herniation on clinical findings and size of the herniated disc measured by CT.[116] They found a significant improvement in symptoms and signs, together with a decrease in size of the herniated disc material as measured by CT.

Alternative procedures for reducing nuclear protrusions

A nuclear protrusion occurs in a young to middle-aged population and is characterized by a gradual onset, and symptoms that develop with prolonged, mostly kyphotic, postures and after activities which usually include forward bending.

Reduction of such a disc displacement is gradual and often slow because it displaces under the influence of prolonged forces. Quick manipulative techniques, with short, high-velocity impulses, have no effect on the nucleus. Techniques such as continuous traction that allow the nucleus to ooze slowly back into place are then required. In our experience, traction is the treatment of choice for most patients with nuclear protrusions. However, some alternative techniques can also be used if the patient presents with contraindications to traction or the therapist has no traction equipment. We find the following techniques most effective in these cases: oscillatory and sustained Cyriax manipulations and McKenzie's reduction techniques.

Oscillatory and sustained manipulations

Most techniques, as described by Cyriax and regularly used for reduction of annular displacements, can be performed in a 'nuclear' way. The patient is positioned as described and the manipulator takes up the slack and probes for the articular end-feel. At this moment, instead of giving a sudden high-velocity thrust, the therapist proceeds to oscillation or sustained pressure.

Slight oscillations at the end of the possible range, lasting 10–15 seconds and frequently repeated, may be effective in small displacements with minor signs on examination. When symptoms and signs are more pronounced, the patient will probably respond better to more sustained pressure. The pressure is maintained for as long as the patient can bear, usually 20–45 seconds. The manipulator monitors the patient's respiration cycle and slightly increases the pressure during the expiration phase. In more difficult cases, these techniques can be combined with continuous traction.

Manipulations are continued as long as the patient improves, usually for a few sessions. When no further result is obtained, the patient is put in traction. When traction does not lead to full resolution but has improved the patient's condition, one can return to the manipulative procedures, which are then likely to give further relief.

McKenzie's reduction techniques

The thinking behind McKenzie's approach is based on Cyriax's disc theory. Being a physiotherapist, he has developed a prophylactic and therapeutic concept based primarily on self-applied repetitive exercises and/or positioning by the patient in order to reduce the nuclear displacement that leads to the 'derangement syndrome'. A classification – derangements 1–7 – is made according to the localization of the pain and the presence or absence of deviation, either in kyphosis or in scoliosis.[117,118]

The patient is taught how to apply progressively increasing mechanical forces that aim to cause centralization and subsequent diminution of pain. The therapist's intervention is only necessary when the results of self-treatment are insufficient. The choice of technique is based on the results obtained during repetitive movements – flexion, extension and side gliding – in the examination.[119] Those movements that reduce, centralize or abolish symptoms during testing are used as treatment procedures.[120] The reader is referred to McKenzie's books and articles for further details.[121–123]

The main principles for treating three of the most common discal conditions – posterocentral prolapse – are set out below. These conditions mostly respond to exercises based on the 'extension' principle.

Acute lumbago without deviation

The patient has central or bilateral pain in the back and/or both glutei, and may be treated with nuclear Cyriax manipulations (rotation techniques carried out in an oscillatory or sustained manner). If the McKenzie approach is chosen, it is the treatment of 'derangement 1' that is used.

The patient gradually moves from one position to another. Progression follows when the pain centralizes and/or diminishes.

Starting with 5 minutes of prone lying, the patient then progresses to lying prone in extension (resting on the elbows) for another 5 minutes. After a few minutes of relaxed prone lying, the patient is asked to do extension movements while lying down, in series of 10 motions. The patient is advised to repeat this procedure on a regular basis.

Acute lumbago with flexion deviation

This is 'derangement 2'. The patient again has central or bilateral pain but also deviation in flexion. The deviation should be corrected first. The treatment is started in prone lying, with the patient supported by a few pillows. Very gradually – every 5 minutes – a small pillow is removed until normal prone lying is possible. After a few minutes, the head of the table is raised, and then raised a bit more every 4–5 minutes until full extension – if possible – is obtained. Throughout this time, the pain should centralize and diminish. The patient is then gradually brought back to the neutral position. When 'derangement 2' has turned into 'derangement 1', in that the deviation has disappeared, treatment of derangement 1 (see above) can be pursued.

Acute lumbago with lateral deviation

The heavy nuclear lumbagos with lateral deviation are hard to manipulate as long as the lateral shift has not been corrected. Therefore the first technique is 'correction of the lateral shift' in standing. When the deviation can be corrected and even overcorrected in a prone-lying position, repetitive extension movements can be tried, possibly with the therapist's assistance. When these manœuvres lead to disappearance of the deviation, the situation has probably returned to 'derangement 1' and can be treated accordingly.

Reduction of a derangement is not the only feature and is not sufficient treatment. The patient should try to maintain the reduction by behaving according to the principles of back school ('keep your back hollow'). If normal function is impaired as a result of the derangement, the patient should be advised to exercise in order to obtain the full normal painless range of movement in all directions. Based on the movements that still influence the patient's symptoms, a treatment scheme is prescribed.

Injections

Epidural local anaesthesia

Introduction

Discodural or discoradicular syndromes are treated by manipulation, traction or epidural injection. The aim of manipulation or traction is to remove the displaced tissue from contact with the dura mater or the nerve root sleeves. These techniques are therefore suitable for reducible disc displacements. If, however, the protrusion cannot be moved or the dura mater is too inflamed, an epidural injection is the treatment of choice.

The aim of epidural injection is to deal with the second element of the interaction: the dura mater or nerve root sleeve. Because procaine 0.5% is used, only surface local anaesthesia is to be expected. The solution does not penetrate the ligaments, the dural sheath of the nerve root or myelinated nerve tissue. The only structures anaesthetized are the free nerve endings of the surfaces that the solution bathes: the dura mater (both tube and sleeves), the posterior surface of the posterior longitudinal ligament, and the anterior surfaces of the ligamentum flavum and facet joints. Fluid injected from the distal end of the epidural space (sacral hiatus) is forced upwards and passes between disc and dura. Cessation of symptoms after the injection shows that the fluid has intervened in the discodural interaction. Epidural local anaesthesia is therefore very helpful when the existence of a disc lesion is in doubt. The procedure can be used safely on outpatients. Often the injection is therapeutic; although procaine 0.5% has only a short anaesthetic effect, permanent benefit often results.

Intrasacral epidural injections were used in the treatment of sciatica long before the nature and mechanism of production of sciatic pain were understood. The technique was conceived by Sicard[124] and Cathelin[125] in 1901. The first reports of cures of sciatica with epidural anaesthesia came from Caussade and Queste in 1909[126] and Viner in 1925.[127] In 1930, Evans obtained complete and permanent cure in 61% of his patients after one or two injections. Although he used large amounts of procaine (up to 140 mL of a 1% solution), alarming symptoms occurred in one case only: cyanosis, opisthotonos, loss of consciousness and incontinence of urine and faeces followed the injection of 120 mL of procaine 2%, but 'consciousness returned within half an hour and sphincter control was re-established 12 hours later; recovery was complete.'[128]

Cyriax used the method from 1937, first diagnostically to determine whether the cause of backache and sciatica lay outside or inside the vertebral column, later as the treatment of choice for irreducible disc lesions.[129,130] In his series of 50 000 injections (50 mL of a procaine 0.5% solution was used), no disasters and only five mishaps were noted: one case of hypersensitivity, two cases of temporary paraplegia of the lower half of the body and two cases of chemical meningitis, all of whom recovered without lasting harm (Cyriax[21]: p. 325).

Since 1980, we have used the injection over 10 000 times on outpatients. There were only slight side effects, such as giddiness or headache, which usually did not last longer than half an hour. In one case, paraplegia of the legs developed. The patient recovered completely within 3 hours.

Indications for diagnostic injection

Procaine 0.5% is a surface local anaesthetic and the drug does not enter any structure; nor does it pass through the myelin barrier of larger nerves. Neither will the posterior longitudinal ligament, the facet joint capsule and the ligamentum flavum be penetrated. The only structures rendered anaesthetized are those bathed by the solution. Temporary relief of symptoms shows that the solution has been able to pass between the two interacting surfaces – disc and dura. Therefore the injection is a very helpful diagnostic procedure, in that a positive response is proof of an existing discodural interaction. In non-mechanical pain or in conditions such as uncomplicated spondylolisthesis, sacroiliac strain or arthritis, spinal or lateral recess stenosis and ligamentous or facet pain, the injection makes no difference to the pain.

In practice, a diagnostic epidural injection can be used in psychogenic backache, uncharacteristic backache or unusual referred pain.

Psychogenic pain

The history and functional examination usually clinch the diagnosis (see online chapter *Psychogenic pain*). However, even when a patient reacts in a neurotic way, a small underlying discodural interaction may also be present. The psychogenic signs overshadow the organic ones and make it difficult to arrive at a conclusion as to the nature of the lesion. A diagnostic epidural injection is then indicated.

Twenty minutes after the injection, clinical examination is performed again. A statement from the patient that the pain has gone identifies a minor discodural interaction. Should the patient wish to make the worst of the problem, the usual statement is that the infiltration has made the pain worse. Instead of straight leg raising being limited to 20°, a limitation of 45° will occur after the injection, which supports the psychogenic nature of the pain. If, after injection, the pain is unaltered, the question remains as to whether the patient is suffering from a psychogenic or a genuine non-discogenic disorder.

Uncharacteristic backache

There is a clear history of constant lumbar pain and symptoms do not vary with posture or exertion. Clinical examination shows a full and painless range of lumbar movements. The latter cause little or no ache, or only one movement slightly increases the pain. The question then arises as to whether the pain is of dural, ligamentous or non-mechanical origin. If it is abolished temporarily by the induction of local anaesthesia, dural origin is established. Often the injection is also therapeutic.

Referred pain

Diagnostic epidural anaesthesia may be needed in cases in which there is uncertainty as to whether the symptoms are local or referred. The typical example is pain in the buttock or thigh: if lumbar movements do not hurt and no abnormalities in the sacroiliac joint, hip or buttock are detectable, the question arises as to whether the pain has a dural or a local origin. The epidural injection immediately settles the diagnosis.

Medicolegal cases

Contradictory opinions often exist. The presence or absence of a discodural interaction can once again be determined by the use of local anaesthesia.

Indications for therapeutic injection

The injection is used as a treatment for those discodural interactions unsuitable for manipulation or traction, i.e. in those cases in which the bulge, for one reason or another, cannot be removed from the dura mater, or where the dura mater is too inflamed. This can be the case in all three syndromes: lumbago, backache and sciatica.

Lumbago

Hyperacute lumbago is an excellent indication for epidural injections. Because of the severe impingement of the protrusion on the dura mater and the extreme inflammatory reaction of the latter, the dural symptoms are so profound that the patient experiences severe twinges on the slightest movement. In such a case, the slightest attempt at manipulative reduction is aborted by severe muscle spasm and increasing pain. Traction is strongly contraindicated, as considerable worsening of the condition is to be expected. Epidural local anaesthesia, in combination with manipulations from the next day onwards, is then the only alternative to several weeks of bed rest.

Immediately after the injection, dural signs and symptoms disappear: a cough no longer hurts, and neck flexion and straight leg raising are of full range and painless. It has been suggested (Cyriax[21]: p. 320) that relief of pain leads to decrease of muscle spasm which, in combination with the recumbent position, allows rapid and spontaneous reduction of the bulge. It follows that, for the next 24 hours, a horizontal position should be maintained. The patient should also be *prohibited* from being driven home in a sitting position. He or she should return as they came – recumbent. By the next day, the symptoms have usually improved sufficiently for the patient to be able to get up and walk around, and it is even possible to travel to the therapist's surgery for further manipulative reduction. It is striking that the dural symptoms and signs do not usually return when the local anaesthesia has worn off. Once the immobilizing twinges have been abolished, the patient can be manipulated, which secures complete relief of the articular signs after 1–4 sessions.

Backache

Ordinary discodural backache responds very well to physical treatment: annular lesions are manipulated and nuclear displacements are treated by traction. Epidurals are reserved for those unusual cases which prove refractory to both manipulation and traction. Also, when manipulation and traction are technically not possible for one reason or another, epidurals are called for. There are also discodural interactions in which dural inflammation continues, despite total or partial reduction of the disc displacement; the approach will then be to desensitize the dural tube, in order to abolish the constant ache.

Intractable backache

Discodural backache is called 'intractable' when it proves to be refractory to both manipulation and traction. A distinction from recurrent backache should be made. Here the displacement can be reduced but remains unstable. In intractable backache, however, all attempts to replace the bulge fail. The next approach is to give a number (1–4) of epidural injections at weekly intervals, which very often abolish the permanent pain.

'Bruised dura'

After an attack of acute lumbago, the dura remains painfully inflamed, resulting in chronic backache that is unaltered by position or movement. The clinical examination reveals a full and painless range of lumbar movement.

Epidural local anaesthesia should be given at once. If inflammation lies at the origin of the symptoms, the pain disappears, not only for the duration of action of the anaesthetic but very often permanently too.

Early morning or nocturnal backache

The history is that of a patient who complains of being woken every morning by severe backache. Once the patient is out of bed, the pain quickly subsides. During the day, everything can be done without the slightest discomfort. Clinical examination reveals nothing but full and painless lumbar motion.

The pain mechanism in early morning backache is probably dural. The increased intradiscal oncotic pressure during the recumbent position causes expansion of the disc, which bulges against the dura mater.[131–133] In this condition, no physical treatment is possible but epidural local anaesthesia succeeds in about 70% of cases.

Pregnancy

During the final months of pregnancy, discodural backache cannot be treated by manipulations or tractions. The alternative is then epidural local anaesthesia.

Hypersensitivity

Sometimes the patient is too hypersensitive to accept active treatment, especially when the treatment methods are not totally devoid of unpleasant side effects. It is therefore not always wise to manipulate the back of a psychoneurotic patient. First of all, the patient is usually very anxious and not ready to accept (forceful) active treatment to the painful back. Second, if the patient does eventually agree to manipulation, one will feel that it has not really happened because of the increased muscle defence when slack is taken up.

Therefore, if the physician thinks that the personality of the patient is such that manipulations will not be tolerated, it is better to treat with epidural injections.

Sciatica

Only 30% of patients with sciatic pain are treated successfully by manipulation or traction. In practice, only those protrusions that are not too large or long-standing and do not involve much periradicular inflammation will respond.

When the bulge is too large (impairment of motor and/or sensory conduction) or too long-standing (more than 6 months), or there is considerable nocturnal pain, the treatment of choice is epidural local anaesthesia. Epidurals are also called for when sciatica is in the recovery phase or when a bruised dural sleeve results after the bulge has disappeared.

Discoradicular disorders with neurological deficit

Root pain together with signs of interference with conduction (motor and/or sensory) shows that the protrusion has reached such an extent that reduction is impossible. The induction of local anaesthesia via the sacral route is then strongly indicated and affords lasting relief in about 80% of cases. The mode by which improvement is secured is still a matter of discussion but it seems that the drug has a lasting desensitizing effect on the free nociceptive nerve endings in the dural sleeve. It has also been suggested that the fluid, which is forced between the dural sleeve and the bulge, separates both components with lasting effect, and thus ends their 'conflict'.

An epidural is the treatment of choice for root pain with muscle weakness or sensory deficit. As in other treatment methods recommended in this book, the injection is either quickly successful or not effective at all. The patient should therefore attend 1 week after the injection for the symptoms and signs to be re-evaluated. If the first injection has afforded no lasting benefit (objective and/or subjective), further epidural injections should be abandoned because there is almost no chance that they will amount to a cure. In contrast, if the first injection has afforded some benefit, further injections should be given, at intervals of 7–10 days, until the patient is completely pain-free. There is theoretically no limit to the number of epidural injections that can be administered but usually the patient needs 2–4, depending on the severity and the immediate diagnostic response after the first injection.

The diagnostic response after the first injection often has a predictive value for the number of injections required. If straight leg raising is full and painless, one more injection will probably be needed. If there is pain at the end of range, one or two more injections are required. If straight leg raising is improved but still somewhat limited, several weekly injections (up to six) may be necessary.

Sciatica with straight leg raising limited on the painless side usually requires more than the usual number of injections: after the first injection, the range on the good side is restored immediately, but it may take up to seven injections before the leg on the painful side can be raised painlessly to a full range.

Patients with severe neurological weakness usually lose their pain very quickly. After one or two injections they are relieved of the sciatic pain, although it may take some months before their strength recovers.

The course during the first few days following the injection is very difficult to predict. Many patients complain of increased pain during the days following the injection and then improve rapidly. Others see an immediate improvement, or improve over 2–3 days and then relapse. Sometimes the patient recovers slowly but gradually over the course of a week. The patient should therefore be warned that, during the first few days, no prognostic conclusions can be drawn from the symptoms and that, for a couple of days, it may be possible that pain is greater than before the injection. Symptoms and signs should only be assessed after 1 week. In long-standing root pain, it is even better to wait for 2 weeks, because considerable improvement often takes place during the second week after the injection.

A second injection is considered if either subjective or objective improvement is detected: for instance, the patient states that pain is diminished but during clinical assessment straight leg raising is as limited as before the injection; or there is a marked increase in straight leg raising but the patient insists that the pain is as severe as ever. Improvements in symptoms and signs usually run parallel, which simplifies the decision in favour of a second injection. However, if there is no lasting change in pain and/or signs, repetition of the procedure is pointless. It is then better to try a sinuvertebral block or to refer the patient for surgical intervention. Cases that are often refractory to epidural anaesthesia are those in which the patient stands deviated in flexion and away from the pain. If every attempt at extension or side flexion in the limited direction is

followed by severe pain shooting down the limb, surgery is almost unavoidable. The prognosis is also poor when the patient stands symmetrically but deviates markedly during forward flexion. Sciatica in the elderly and third lumbar disc lesions with root pain and deficit seldom respond to epidural injections. In both cases, we prefer to induce sinuvertebral blocks at the required level.

If the immediate diagnostic response after the injection is excellent but, after 1 week, the patient is in as bad a state as before, the injection can be repeated with some cortisone or triamcinolone added to it, which may produce permanent relief.

Sciatica without neurological signs

Clinical examination shows no clear signs of impaired conduction but data drawn from the history and clinical examination strongly indicate injection. This is the case in the following conditions:

- Root pain of longer than 6 months' standing
- Recovering root pain
- Recurrence within a year, following sciatica with neurological deficit
- Considerable nerve root inflammation
- 'Bruised dural sleeve'
- Primary posterolateral protrusion
- Failure of manipulation or traction.

Root pain of longer than 6 months' duration

The chances of reducing the bulge by manipulation or traction are very small. In contrast, the induction of local anaesthesia usually affords good results.

Normally, root pain caused by a disc lesion starts to recover spontaneously within 1 year. If this is not the case and pain and limitation of straight leg raising persist, one or two epidural injections should be given. It is as well to wait up to 2 weeks before the second injection. Cyriax explains the good results by mobilization of the nerve root during local anaesthesia and by lasting desensitization of the nerve root sleeve (Cyriax[21]: p. 323).

Recovering root pain

The patient has spent some days or weeks in bed and the sciatic pain is declining. Clinical examination shows limited straight leg raising but no weakness. Although this case seems to be an indication for manipulation or traction, epidural local anaesthesia usually affords better and quicker results.

Recurrent sciatica after recent root palsy

Occasionally, patients are encountered who see their sciatic pain disappear completely, whether as the result of root atrophy or after a couple of epidural injections, but then sustain another attack of sciatica within a year. This is most uncommon, as, in most patients, signs and symptoms do not recur on the same side and at the same level once they have disappeared. A small, recent protrusion probably underlies the 'new' pain. Therefore it could be argued that such a case would benefit from manipulation or traction. In practice, however, these measures seem to fail, whereas one or two epidural injections usually succeed.

Inflammation of the nerve root sheath

If the sciatic pain is constant and persists during recumbency, a considerable degree of inflammation around the nerve root sleeve should be suspected. Even though neurological signs are absent, manipulation or traction is best avoided, for fear of considerably aggravating the symptoms. The treatment of choice is epidural local anaesthesia. It is our experience that, in acute and intense nerve root inflammation, it is wise to add 40 mg of triamcinolone to the solution.

Bruised nerve root sleeve

The sciatic pain is more or less continuous and not dependent on posture or movement. The clinical examination shows nothing but a full range of movement, painless straight leg raising and normal conduction of the nerve roots. No alternative causes for the pain are present. The segmental pain may be caused by persisting inflammation of the dural nerve root sleeve after the bulge has undergone reduction, whether spontaneously or as the result of treatment. Although the nerve root lies free and has normal mobility, it remains inflamed and causes continuous sciatic pain.

Epidural local anaesthesia should be induced to settle the diagnosis. Very often, the pain is abolished not only for the time being but also permanently. Usually, one or two epidural injections are enough for complete cure.

Primary posterolateral protrusion (see p. 463)

The disorder can be treated by traction or by epidural local anaesthesia, depending on when the diagnosis is made. Traction very often succeeds but the recurrence rate is high. Epidural local anaesthesia affords no benefit at all when the problem is still developing. The injection should therefore only be given when signs and symptoms are at their worst. In practice, the patient should be re-examined every 2 weeks. The injection is given when the limitation of straight leg raising becomes more or less stable. One to three injections may then relieve the condition. Treatment of primary posterolateral protrusion is thus neither simple nor rapid. It should be explained to the patient that proper treatment can only be given at a specific moment during the development of the condition, which may require a wait of up to 4 months. As pain is never severe and never appears during the night, it is usually possible for the patient to avoid premature surgery.

Failure of manipulation or traction

The possibility that a displaced disc fragment causing a discoradicular interaction can be replaced by manipulation or traction is about 30% (J. Cyriax, personal communication, 1982). The reason is that the bulge is located beyond the edge of the posterior longitudinal ligament. The ligament is therefore not of great help in shifting the disc back into place, as is the case in manipulations for discodural interactions. However, manipulation and traction can always be tried first if no contraindications are present and the condition is suitable: root pain lasting no longer than 6 months, no neurological deficit and a moderate degree of inflammation. If there is no marked improvement after two or three manipulative sessions or after 10 sessions of traction, the patient should be referred for epidural local anaesthesia.

Indications for epidural local anaesthesia are summarized in Box 40.7.

Box 40.7

Indications for epidural local anaesthesia

Diagnostic

- Psychoneurosis
- Uncharacteristic backache
- Referred pain
- Medicolegal cases

Therapeutic

- Hyperacute lumbago (in combination with manipulation)
- Backache:
  - Intractable backache
  - 'Bruised dura'
  - Morning or nocturnal backache
  - Pregnancy
  - Psychoneurosis
- Sciatica:
  - Sciatica with neurological deficit
  - Sciatica without neurological deficit
    - Root pain of over 6 months' standing
    - Recovering root pain
    - Recurrence within the year, following sciatica with neurological deficit
    - Considerable nerve root inflammation
    - Bruised dural sleeve
    - Primary posterolateral protrusion
    - Failure of manipulation or traction

Contraindications

Sensitivity

Hypersensitivity to procaine is very exceptional. In a series of 50 000 epidurals, Cyriax encountered it once. The rarity of the condition does not imply that the physician can ignore it and the greatest care should be taken. If the patient mentions serious adverse reactions from previous local anaesthesia, it is better to test for sensitivity. If no special local or general reactions follow, the epidural injection can safely be given the next day.

Local sepsis

The existence of local skin sepsis around the area where the needle was introduced is an absolute contraindication. The introduction of bacteria into the neural canal is disastrous[134] and therefore the risk should not be taken. A previous local abscess or previous operations for a pilonidal sinus or anal fistula should also be considered to be contraindications.

Previous neurological infections

It has been argued that, after recovery from a febrile neurological infection, some septic adhesions may form at the lower end of the theca. Pressure on the epidural fluid could then reactivate the inflammation.

Anticoagulant therapy

The insertion of an epidural needle into the sacral canal very often causes a venous puncture. In the presence of anticoagulant therapy, bleeding could provoke serious haematoma, with the possible consequence of adhesions.[135]

Side effects and dangers of epidurals

Apart from hypersensitivity, which can usually be precluded before the injection, two main adverse reactions are possible: hypotension when the injection is given intravenously or too rapidly, and transient paraplegia if the solution leaks through the dura to cause spinal anaesthesia.

Hypotension

If certain precautions are taken, a drop in blood pressure should not be feared:

- Intravenous injection is avoided by testing for aspiration of blood after the needle has been inserted and following the injection of each 5 mL of fluid (see p. 573).
- The fluid is injected very slowly, at a rate of 5 mL/min. After each 5 mL, there should be a pause of 30 seconds to 1 minute.
- During the injection, a constant conversation is maintained with the patient. The slightest faltering in the voice is usually an indication of an incipient drop in blood pressure. The introduction of fluid is then temporarily stopped until the patient feels better.

Spinal anaesthesia

Cyriax described four cases of paralysis to the mid-thorax after the epidural induction of 50 mL procaine 0.5%. The solution had obviously transgressed the dural barrier and temporarily paralysed the lower limbs and the abdomen. Diaphragmatic breathing was retained, the patients did not need artificial ventilation and all recovered from their paraplegia within 2 hours (Cyriax[38]: p. 183). The present author encountered a similar instance of paraplegia up to the lower thoracic region. Weakness of the limbs became apparent during the injection, which was stopped after the introduction of about 20 mL of procaine 0.5%. It took 3 hours before strength in the limbs returned sufficiently for the patient to drive home.

The most likely explanation for the spinal anaesthesia is unintentional intrathecal injection: the tip of the needle pierces the dura mater during the procedure. Even if the tip does not remain intrathecally during the injection, enough of the solution can pass through the hole to give rise to complete sacral and lumbar root block.[136]

In order to avoid this complication, the injection should be given very slowly, and aspiration is made after each 5 mL, or after each unintentional movement by the patient. At each stopping point, the Achilles tendon reflex is tested, together with the force of the dorsiflexors of the toes on both sides. If weakness is discovered, the injection is stopped at once.

Technique

The aim of the technique (summarized in Box 40.8) is to introduce the fluid into the epidural space, a space between two concentric cylinders: the outer cylinder is the bony sacral and lumbar canal, the inner cylinder the dural tube with the dural nerve root sleeves. The space is filled with loose connective tissue, fat and venous plexuses.

The sacral canal ends at the lower end of the sacrum, where it is covered only by ligament, subcutaneous tissue and skin. The dura mater usually ends at the level of the second sacral vertebra. These anatomical peculiarities make the distal end of the sacral canal a suitable place to receive a needle, without fear of piercing the dura. Normally, the needle can be moved upwards over a distance of more than 6 cm before the tip reaches the distal end of the dural sac.[137]

Landmarks

The distal end of the sacral canal is formed by the distal basis of the sacrum, the two sacral cornua and the intercornual ligament. There is only subcutaneous fat between the borders of the sacral hiatus and the covering skin (Fig. 40.24).

The most important landmarks are therefore the *two cornua*, bordering the distal end of the sacrum. The palpation of these two bony prominences is not always easy. Some women have a thick layer of fat covering the sacrum, camouflaging the cornua. Sometimes the cornua are absent or asymmetrical. Sometimes they lie deeply between the buttocks or may appear higher up, where they can be palpated some centimetres proximal to the intergluteal line.

In order to palpate the cornua, the following instructions should be followed:

- The patient lies prone, the pelvis slightly tilted upwards by means of a small pillow placed under the symphysis.
- The legs are slightly spread and rotated internally (Fig. 40.25). This position moves the buttocks outwards and makes the sacrum and the hiatus more prominent. In this position the two cornua can sometimes be seen at the upper extent of the intergluteal line.

If the cornua are not visible, they can be located in the following way: both *inferior iliac spines* are palpated and a downwards-pointing equilateral triangle is drawn, the base of which is formed by the line connecting the two spines; the tip of the triangle lies at the sacral hiatus (Fig. 40.26).

Box 40.8

Sacral epidural injection: technique

- Position the patient.
- Check the landmarks.
- Desensitize the skin and the intercornual ligament with 1 mL lidocaine 2%.
- Introduce the spinal needle about 4 cm into the sacral canal.
- Remove the stylet and check for blood or cerebrospinal fluid.
- Inject slowly – no more than 5 mL/minute while maintaining conversation with the patient.
- Interrupt the injection after each 5 mL and:
  - Check needle placement
  - Check blood pressure
  - Check strength of dorsiflexors.
- After the injection the patient remains prone for about 10 minutes and supine for another 15 minutes.

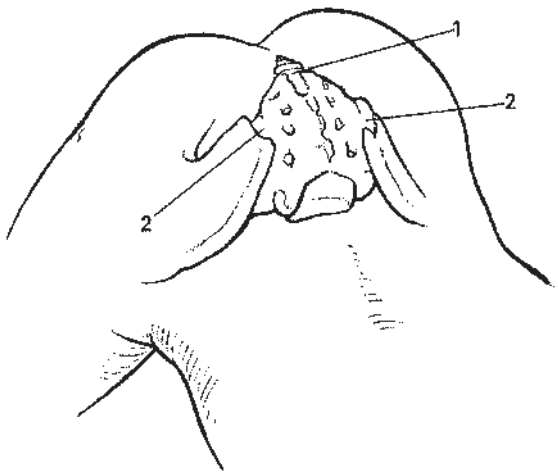


Fig 40.24 • The sacral hiatus (1) and inferior iliac spines (2).

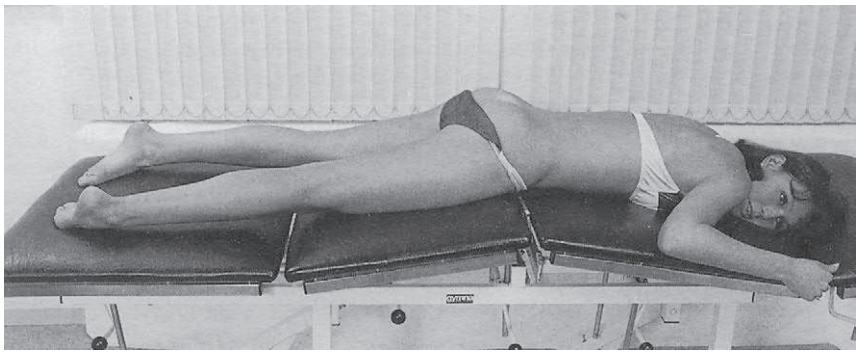


Fig 40.25 • Position of the patient for locating the cornua.

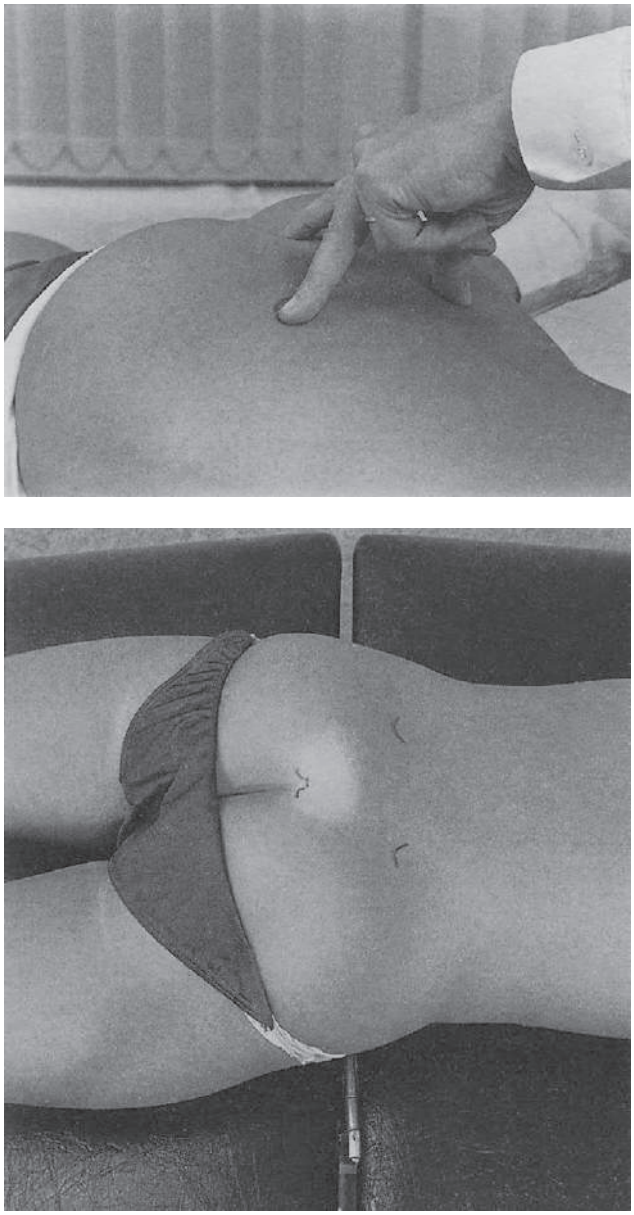


Fig 40.26 • The triangle used to locate the sacral hiatus.

Other important landmarks are the *sacral spinous processes* and the two *sacrospinal muscles* at the first sacral level.

The sacral spinous processes are palpated in order to estimate the curvature of the sacrum and the direction of the spinal canal. It is extremely important to assess the angle of the sacrum, in order to estimate the direction of the needle in the canal. Some people have a very flat sacrum with almost no curvature at all, while others have a very curved sacrum; occasionally, the sacrum is twisted or has a marked asymmetry. The sacrospinalis muscles are palpated to detect such asymmetry.

Preparation

A cambric roll is placed in the intergluteal fold to spread the buttocks and to protect the anus and vagina from irritation by the disinfectant. If hair is present, it is shaved.

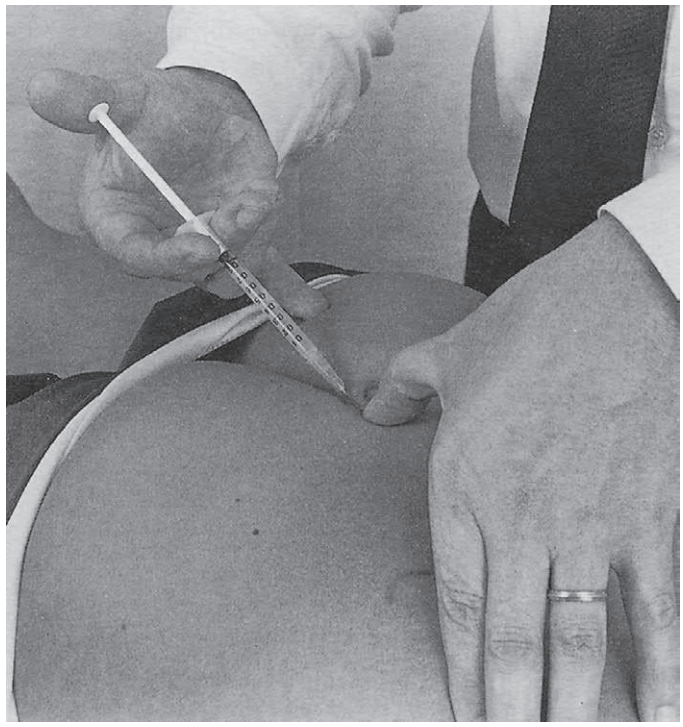


Fig 40.27 • Anaesthetizing the skin and subcutaneous tissue over the hiatus.

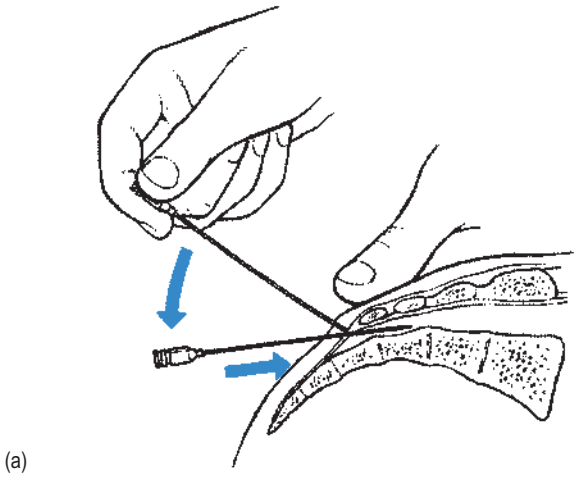
One thumb palpates for the cornua. If the patient cannot relax the gluteal muscles, or the cornua lie deeply, the buttocks are stretched outwards by an assistant. Care should be taken not to overstretch the skin over the cornua, for fear of obliterating the landmarks.

A small syringe, filled with 0.5–1.0 mL of a 2% lidocaine solution, is fitted to a 2.5 cm 25 G needle. This is used to anaesthetize the skin and the subcutaneous tissue over the hiatus, distal to the palpating thumb (Fig. 40.27). The needle also penetrates the intercornual ligament under the thumb. The angle at which the needle enters the hiatus is noted in order to serve as a guide for the introduction of the larger lumbar puncture needle. It is important not to use more than 1 mL solution, so as not to obliterate the bony landmarks.

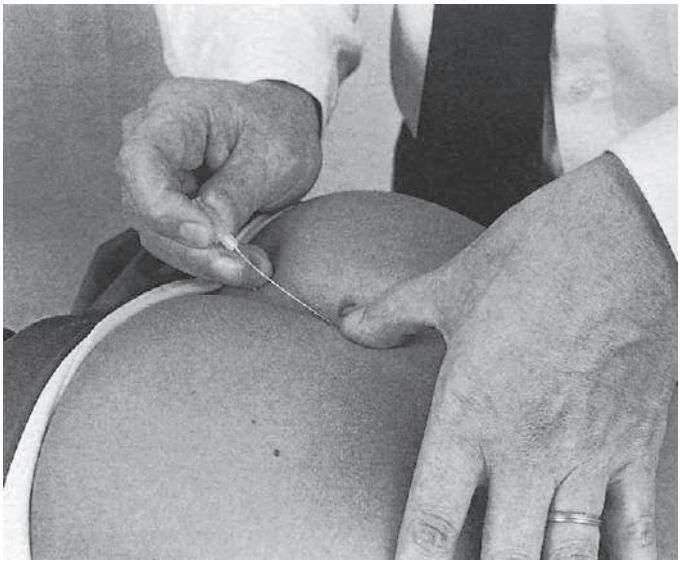
Introduction of the needle

The insertion is made with a lumbar puncture needle equipped with a stylet. In order to be manœuvred easily into the sacral canal, the needle must have some flexibility and therefore should be long: a 9 cm needle is perfect for this purpose (21 G × 9 cm). The stylet precludes the risk of introducing a piece of skin, which later could lead to the development of an inclusion dermoid cyst.

The needle is thrust in between the two cornua just distal to the palpating thumb. After piercing the thick intercornual ligament, it passes freely through the hiatus before it hits the bone of the sacral base. The needle is then partly withdrawn and angled slightly in order to conform with the obliquity of the sacral canal; this can be done by applying slight pressure with the palpating thumb on the skin above the needle (Fig. 40.28).



(a)



(b)

Fig 40.28 • Insertion of the lumbar puncture needle.

The needle is then moved further upwards over a distance of 3–5 cm. Normally, the tip remains well distal to the lower end of the dural sac, which is represented by the line between the two lower iliac processes.

Usually, the needle slips intrasacrally without any problem. However, the tip may meet bone, which indicates that the angle must be altered. Judging the angle of insertion of the needle is the most difficult part of the whole procedure. Important guides are previous palpation of the lower sacral spinous processes and the angle of the fine needle used for surface anaesthesia when it was pushed through the intercornual ligament. A very curved sacrum is difficult to approach, demanding puncture well distal to the bony ridge connecting the cornua. The needle is then aimed almost horizontally, in the direction of the patient's head. A patient with hyperlordosis usually has a flat, horizontal sacrum, which calls for an almost vertical insertion. A particular problem is posed by an overcurved bifid sacrum, where the sacral arches are not bony but consist of fibrous tissue. If the insertion is made too low down, the tip of the needle may slip through the ligamentous roof of the sacrum and come to lie in the fibrous tissue closing the defect. A sacrum with an intrasacral bony projection is an occasionally encountered difficulty. If the needle catches such a bony obstacle on its way up, it must be withdrawn a short distance and thrust in again at a slightly different angle. If this proves to be impossible, the tip of the needle should be left there and the injection made from this position, providing no palpable swelling at the hiatus appears as the fluid is administered.

When the needle is far enough into the sacral canal, the stylet is withdrawn. Care is taken to see that neither cerebrospinal fluid nor blood escapes.

The needle pierces the theca in only the occasional case in which the dural sac ends at an abnormally low level. In most, the dural sac terminates at the level of S2, which is considerably more proximal than the tip of the needle. If cerebrospinal fluid escapes, the needle should be removed immediately. It is a serious mistake to withdraw the needle only a little until it lies extradurally and then to continue with the injection, as enough of the procaine solution can pass through the hole to cause a spinal block. Hence, if the theca has been pierced, the whole procedure should be postponed until some days later, when the needle is not inserted so far proximally.

Often blood escapes when the stylet is withdrawn, which is not surprising, considering the number of epidural veins in the sacral canal. It is enough to move the tip of the needle into a position where it no longer penetrates the vein, and the injection can then be given without any danger. If the needle cannot be manœuvred into a position where blood ceases to escape, the injection should be postponed for 2 days.

The injection

A syringe containing 50 mL of procaine 0.5% is connected to the needle. Aspiration ensures that the needle has not penetrated either the dura or a vein. The solution is then injected very slowly at a rate of no more than 5 mL/min. Normally, not much tissue resistance is felt and the patient only experiences a slight ache in the sacral region. Usually the full volume of 50 mL is injected, but the amount can be altered according to the size of the patient and the localization of the lesion. Even in a large patient with a third lumbar lesion, 50 mL is always sufficient. Smaller amounts – 30–35 mL – are adequate in slightly built women, and 40 mL is enough for any patient with a lesion at the L5–S1 level.

If the fluid cannot be injected or the patient experiences considerable pain over the sacrum, subperiosteal localization of the tip of the needle should be suspected. Although the needle lies correctly intrasacrally, the tip remains under the periosteum, which obstructs free entrance of fluid (Fig. 40.29). In this case, it is usually sufficient to rotate the needle through 180°, so that the bevel faces towards the sacral canal. If introduction of the fluid is still obstructed, the needle should be withdrawn by a millimetre or two. Before the injection is tried again, aspiration should be performed anew to ascertain that neither the theca nor a vein has been punctured.

The physician keeps the palm of his or her free hand flat on the patient's sacrum during the whole injection. If the needle is inaccurately inserted and lies in the sacrospinalis muscle

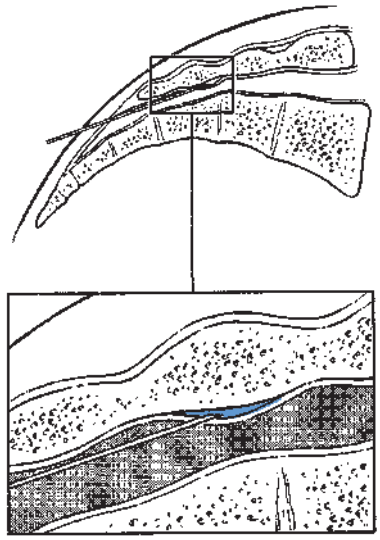


Fig 40.29 • If the fluid cannot be injected, subperiosteal localization of the tip of the needle should be suspected.

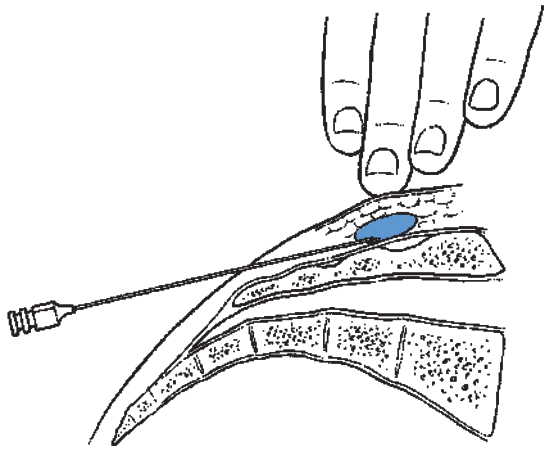


Fig 40.30 • Palpation of the sacrum during injection detects misplacement of the needle.

instead of in the sacral canal, a swelling will be felt under the palpating hand after the introduction of about 10 mL (Fig. 40.30). A misplaced needle is more common than expected. One study demonstrated correct needle placement in 85% of caudal epidural injections.[138] In another more recent study, successful injection placement was confirmed on subsequent fluoroscopic visualization in only 77% of patients.[139]

The hand on the sacrum is thus the main safeguard against a misplaced needle and it is therefore vital to palpate the two sacrospinalis muscles before the injection is started. Sometimes there is marked asymmetry in the two convexities of the sacrum. If this is not ascertained before the injection, an expanding bulge in one sacrospinalis muscle during injection will be more difficult to identify. Although an extrasacral injection causes no harm at all, it may lead to false diagnostic conclusions or an unjustified conclusion of therapeutic failure.

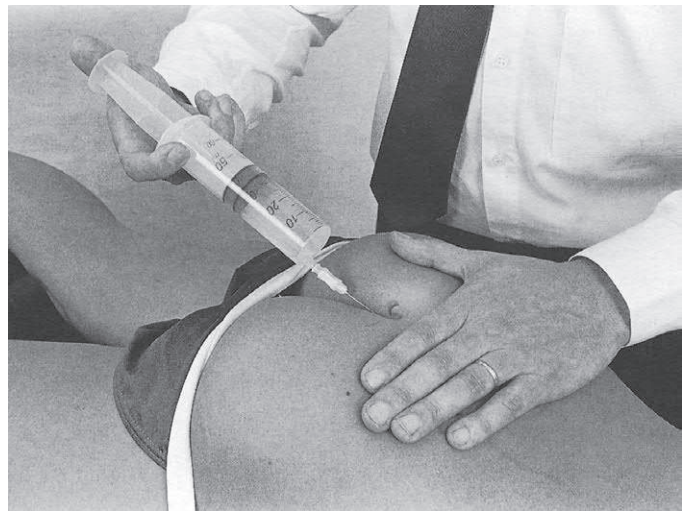


Fig 40.31 • The injection.

After each 5 mL is injected (Fig. 40.31), it is as well to interrupt the procedure and to test the strength of the dorsiflexors of the toes, in order to detect incipient paralysis. The strength of the foot muscles will quickly weaken if the solution passes the dural barrier. Aspiration is also performed each time the patient moves.

The physician should maintain a conversation with the patient for the whole duration of the injection. The patient describes any pain, locally or referred, as well as dizziness and headache. A stuttering voice is usually the first warning sign of an unwanted effect. If patients feel odd or their voice falters slightly, the injection is temporarily stopped until they feel better. If dizziness appears rapidly during the injection and continues for more than 5 minutes, even though no more fluid is introduced, the injection should be abandoned. Elderly people usually tolerate injection better than younger patients. The dizziness and the slight headache that follow too rapid an injection are explained by the rapid rise in cerebrospinal fluid pressure, which causes an abrupt drop in venous pressure in the sagittal sinus.[140]

The injection typically causes an unpleasant feeling of pressure at the sacrum, sometimes radiating to both buttocks or thighs. A patient with sciatica usually states that pain in the leg is reproduced when the fluid is injected, which probably results from increased pressure on the inflamed nerve root sheath.[141] Reproduction of root pain may start after the introduction of 5–10 mL if the patient suffers from a lesion at the fifth lumbar level, but if the lesion lies at L3 the pain will be felt only after 35–40 mL (Troisier[142]: p. 383).

Pain reproduction during an epidural injection is a good sign, in that it indicates that the fluid is placed epidurally and has reached the affected nerve root. The intensity of the pain depends on both the degree of inflammation and the speed of injection. If the injection is done too quickly, pressure on the inflamed nerve root may increase so rapidly that it causes unbearable pain, which should obviously be avoided. Because the injection may have to be repeated, it must be given in such a way that the patient does not fear it on the next occasion.

The physician therefore adjusts the injection according to the reaction of the patient.

After the injection

The patient remains prone for about 10 minutes and then turns into a supine-lying position, remaining there for at least 15 minutes and being kept under observation the whole time. Occasionally, a patient feels faint or has a slight fall in blood pressure a few minutes after the injection. If this occurs, it is usually sufficient to raise the legs. Sometimes there is a slight numbness over the sacrum and the saddle area, and in the supine position the pressure of the couch is not felt. The feet may become warm after the injection as a consequence of sympathetic block, which indicates that the fluid has reached the second lumbar level.

Within 15–20 minutes of the injection, the inflamed dura or dural nerve root sleeves should be more or less anaesthetized. The effect of the epidural is best tested by noting the change in range and pain on straight leg raising. Immediate improvement of straight leg raising has prognostic value, especially in sciatica. If full and painless straight leg raising is restored immediately after injection, one more injection will usually settle the matter. If there is only a little improvement in straight leg raising, which remains very uncomfortable or even limited, 2–4 more injections may be required. In sciatica with bilateral limitation of straight leg raising, the first epidural injection usually restores full and painless range of the leg on the unaffected side but the restriction on the painful side can remain unchanged; the range of straight leg raising on the affected side shows only slight improvement after the second injection and it may even take 5 more injections before a full and painless range is restored. Hence, it is probable that 5–6 epidurals are required to cure sciatica with bilateral limitation of straight leg raising.

The patient is allowed to get up after 20–30 minutes. For most patients this is not a problem; they can walk out and even drive a car safely. However, occasionally a patient may feel a little light-headed or have a slight and temporary disturbance of proprioception. Although muscle strength is completely normal, walking may be difficult. It has been suggested that this is merely the result of some loss of deep sensitivity.[143] Probably a tiny amount of the local anaesthetic has leaked through the nerve root sheath. The concentration is too low to have serious clinical effects[144] and symptoms disappear over the course of about 30 minutes. Patients who complain of dizziness or show some disturbances in gait after the injection should thus remain supine until their symptoms disappear completely. As it is very difficult to foresee how a patient will react to an epidural injection, and theoretically any patient may need to lie still for an hour or more, the technique must not be used if time is limited.

The patient is advised to take relative rest for the remainder of the day. From the next day on, normal daily activities can be resumed. Some patients complain of a slight headache but this does not last any longer than 24 hours.

The immediate result of the injection is very difficult to predict. Some patients with sciatica may have increased pain for 2–4 days and then improve rapidly, or see an initial improvement but relapse after a couple of days. Others have immediate and excruciating pain for 1–2 days and then gradually improve. Because the direct result of the injection is so variable, it is best not to evaluate the situation until a week has elapsed. In patients with long-standing sciatica, it is even better to wait 2 weeks because often improvement only takes place during the second week following the injection.

Follow-up

One to two weeks after the first injection, the patient attends for re-examination. In judging the results, both subjective and objective factors are taken into consideration. An improvement in either warrants a second injection. Although diminution of signs and symptoms usually runs parallel, straight leg raising is sometimes increased considerably, despite the patient declaring that there has been no improvement. Alternatively, there may be significantly less pain but nerve root mobility is as impaired as before the injection. In both situations, the condition is considered to be improved and a second injection is given (Box 40.9). However, if there has been no lasting improvement in symptoms or signs, continuation of the treatment with epidural local anaesthesia is pointless and other forms of treatment should be considered.

Further epidural injections at intervals of 1–2 weeks are given as long as there is progressive improvement. In hyperacute lumbago, one infiltration, followed by a series of daily manipulations, is usually enough for cure. Chronic backache usually requires 1–2 injections. In sciatica, up to six infiltrations may be required.

Sometimes, in spite of a positive diagnostic response, there is no appreciable improvement at the next attendance. It is then worth giving another epidural injection, this time with 40 mg of triamcinolone mixed in the procaine solution. The combination may prove successful when local anaesthesia alone has failed.

Box 40.9

Second injection on diminution of symptoms or signs

Symptoms

- Less pain
  - No more nocturnal pain
  - No more pain at rest
  - Pain has shifted upwards (e.g. gluteal instead of calf)
  - No more pain during coughing or sneezing
- More mobility in daily life

Signs

- Improvement in straight leg raising
- Improvement in lumbar mobility

40 mg of triamcinolone is added to the solution if injection with procaine helped only temporarily

Mechanism

The way epidural local anaesthesia works is not yet completely understood. A clinical response may be attributable to physical effects on disc, dura and neural structures or to pharmacological actions on the surfaces bathed by the fluid. It is also hypothesized that the fluid separates the dura from the disc and thus directly intervenes in the discodural interaction. Because of the increased pressure in the closed epidural space, the dural sac becomes compressed, narrows and is momentarily released from the disc. The epidural injection of a large volume of fluid thus has a mechanical influence on the dura mater. Instead of the disc being moved away from the nociceptive structures by manipulation or by traction, the dural tube or dural sleeves are separated from the disc by the amount of fluid injected in the epidural space, as has been experimentally demonstrated by introducing lipiodol into the dural sac and thereafter performing an epidural injection via the sacral route (Troisier[142]: pp. 390–391). It is likely that the separation of dura and disc not only is temporary but also causes a definite change in the relationship between disc and dura. Some support is lent to this view by the observation that the introduction of normal saline into the epidural space has a therapeutic benefit on its own.[145,146]

Further evidence for the separation hypothesis comes from a recent randomized double-blind study on epidural injections for postoperative lumbar spinal fibrosis. Forceful injections via the sacral hiatus of 125 mg prednisolone acetate plus 40 mL saline showed significantly greater improvement than injections via the same route with 125 mg prednisolone acetate alone.[147]

Although the hydraulic effect presumably contributes to the long-term results of caudal epidural injections, it is certainly not the only mechanism. Local anaesthetics have been described as providing short- to long-term symptomatic relief based on alteration of various mechanisms, including excess nociceptive process, excess release of neurotransmitters and nociceptive sensitization of the nervous system.[148–151]

Results

The first results of epidural local anaesthesia were published by Evans in 1930.[128] He obtained a 62% success rate in sciatica patients with limited straight leg raising. This was confirmed by other studies in subsequent decades.[152–154] Addition of steroids increased the success rate only slightly to 70%.[155,156,79] Cyriax (personal communication, 1982) obtained an overall success rate of 68% in patients with root pain and neurological signs treated with 50 mL of procaine 0.5%. Other studies describe excellent results in 60–83% of patients suffering from sciatica and treated with sacral epidurals with a mixture of local anaesthesia and methylprednisolone.[157,158] In a double-blind, placebo-controlled study, Bush and Hillier[159] compared the effect of 25 mL of procaine 0.5% containing 80 mg of triamcinolone acetonide with the introduction of 25 mL of normal saline in patients with sciatica. The active group showed significant pain relief and a significant increase in mobility at 4 weeks, which resulted in improved quality of life; at 1 year, subjective and objective measures were improved equally in both groups. More recently, a randomized, double-blind trial evaluated the effectiveness of caudal epidural injections with local anesthetic, with or without steroids, in managing chronic discogenic low back pain. Significant pain relief and functional status improvement were observed in 55% of the patients treated with local anaesthesia and 68% of the patients treated with anaesthesia and steroids.[160]

The present author performed a retrospective study of 94 consecutive patients treated with epidural local anaesthesia. A total of 208 injections were given without side effects and resulted in a full and painless range in 69 patients.[161] The findings were classified as follows.

Hyperacute lumbago

Five patients were pain-free within 5 days of combined treatment with epidural local anaesthesia followed by daily manipulations.

Chronic backache

Of 21 patients, there were 12 successes and 9 failures. This group was divided into three subgroups:

- Nine patients with a 'bruised dura mater' – dural symptoms, absence of dural and articular signs – produced 3 cures and 6 failures.
- Three patients with morning backache were all cured after 1–2 injections.
- Of nine patients with chronic intractable backache (i.e. manipulation or traction had failed to secure adequate relief), 3 were cured after the injection, 3 were pain-free after injection and subsequent manipulation, and there were 3 failures.

Root pain

There were 68 cases: 39 patients with and 23 without neurological deficit, and 6 patients suffering from postlaminectomy root pain.

- Nineteen of the 23 patients with root pain but no neurological deficit recovered and the treatment failed in 4.
- Of the 39 patients suffering from sciatica with neurological deficit, 31 recovered with 1–7 injections. To enhance an initially slow response, 40 mg of triamcinolone was added six times. The figures per affected nerve root are given in Table 40.2.
- The average limitation of straight leg raising was 35° (70–10°) at the beginning of treatment. The average duration of the treatment was 3 weeks (1–8 weeks).
- The number of epidurals given was as follows:
  7 patients – 1 injection; 9 patients – 2; 11 patients – 3; 2 patients – 4; 1 patient – 6; 1 patient – 7.
- Only 2 of the 6 postlaminectomy patients responded successfully to the epidural injection.

Nerve root blocks

An injection with local anaesthetic or triamcinolone at the lateral foramen and at the corresponding sinuvertebral nerve

Table 40.2 Result of epidural injection in sciatica with neurological deficit

Root	*n*	Success	Failure
L3	2	1	1
L4	4	1	3
L5	11	9	2
S1	15	14	1
S2	7	6	1
Total	39	31	8

may be required when a patient with a discoradicular interaction fails to benefit from the induction of epidural local anaesthesia. This injection is also indicated in root pain from a narrow lateral recess or in persisting root pain after laminectomy.

Indications

Failure of epidural injections

There are some cases that are suitable for epidural local anaesthesia and in which injection is followed by a good diagnostic response, but in which there is no improvement after 1 week. When repetition of the injection with 40 mg of triamcinolone added is also unsuccessful, the next step is to produce root block with local anaesthesia. If this affords immediate improvement of signs, 20 mg of triamcinolone is injected at the same level. All patients with a discodural interaction for whom epidural injection has been a failure should therefore receive a sinuvertebral block at their next attendance (Cyriax[21]: p. 353). Nerve root blocks are particularly indicated in radicular pain caused by far lateral or foraminal disc herniations. Foraminal injection of local anaesthetic and steroids afforded long-standing improvement in 80–90% of patients with severe lumbar radiculopathy secondary to foraminal and extraforaminal disc herniation.[162–166]

L2 and L3 root syndromes

Experience shows that L3 radicular pain from a disc lesion reacts more quickly and better to a root block than to epidural injections. Root block is also the treatment of choice in the very uncommon case of L2 pain due to an L1–L2 disc lesion.

Lateral recess stenosis

When root pain results from compression in a narrowed lateral recess, epidural local anaesthesia always proves a therapeutic failure.[88,167]

The introduction of a steroid suspension around the nerve root is very often successful and even local anaesthesia alone may produce a prolonged effect.[168–171] The fact that infiltration of a steroid suspension around the nerve root affords long-term relief (many patients are even permanently cured) suggests that radicular pain from a lateral recess stenosis is not simply caused by narrowing of the bony margins of the canal but stems from inflammation and pressure exerted by oedema and fibrosis in the perineural soft tissues.[172–174]

The main difficulty is distinguishing between the fourth and the fifth lumbar levels. Localizing symptoms cannot be provoked by clinical tests, so the triamcinolone injection is given at the likeliest level and, if there is no clear result after 1 week, repeated at the other level. If considerable benefit is achieved, one or two more injections at intervals of 2 weeks will produce complete recovery.

Patients with root pain after laminectomy

The postoperative spine with persistent or recurrent root pain is a major diagnostic and therapeutic problem. Differentiation should be made between a recurrent disc displacement, scar tissue formation and an overlooked lateral canal stenosis.[175] Myelography and CT, although valuable, cannot always establish the diagnosis.[176,177]

Epidural local anaesthesia sometimes succeeds, but more often scarring prevents the solution reaching the right spot. A second or third surgical intervention should only be considered as a last resort because, as Nachemson[4] has pointed out, the success rate drops dramatically after the first operation and second or subsequent procedures have only a 5% chance of success. Others have reported a similar pattern of success rates (8%[178] and 10%[179]) after a second operation.

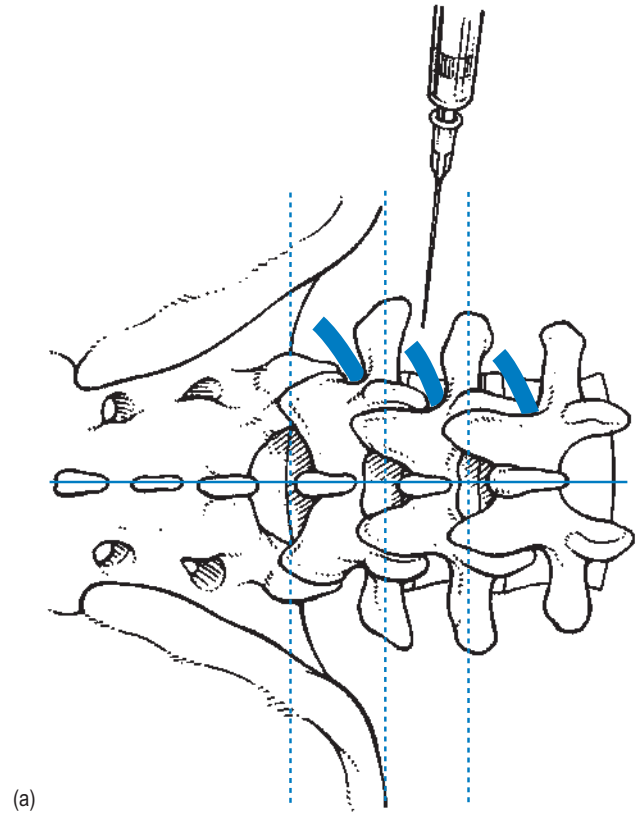
Sinuvertebral/root block can be a good alternative in persistent root pain after surgery. However, it is not always easy to identify the right point. Several attempts with a local anaesthetic may be required but, once the appropriate level has been found, 20 mg triamcinolone is injected at the same place during the next attendance. However, results of nerve root blocks in persistent root pain after back surgery are significantly poorer than in primary nerve root irritation.[180]

Technique for roots L2–L5

The patient lies prone with the arms alongside the body. A small pillow placed under the pelvis reduces the lumbar lordosis. This position relaxes the sacrospinal muscles and facilitates palpation of the bony landmarks.

The intervals between the spinous processes are marked. The line between the fourth and the fifth spinous processes usually coincides with the horizontal line joining the uppermost parts of the iliac crests (Fig. 40.32a). Care should be taken, however, not to press too much subcutaneous tissue between the palpating fingers and the iliac crest, which may be a particular problem in obese patients. Sometimes a patient presents with a high pelvis: the sacrum is relatively low in relation to the iliac crests. The line connecting the two iliac crests may then cross the fourth spinal process, or even lies at the L3–L4 level.

Another method of identification may be required. The levels of the palpated spinous processes and interspinous ligaments are then distinguished by using the posterior and inferior iliac spines as landmarks (Troisier[142]: p. 423): the tip of the first sacral spinous process invariably lies 2 cm above the line connecting the inferior borders of these spines. If there is any doubt, the landmarks are inferred from a radiograph of the pelvis and lower lumbar spine.



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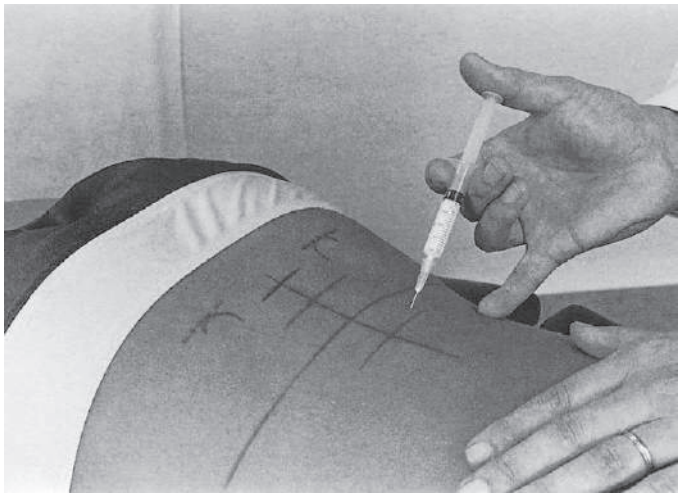


(b)

Fig 40.32 • Injection of the left L4 root.

The intervertebral foramen lies level with the spinous process. At the appropriate level, a point is thus chosen between two consecutive horizontal lines and about three fingers' breadth from the vertical midline (Fig. 40.32a).

A long, thin needle (usually a 7 cm needle will do but in obese patients 9 cm may be required) is fitted to a 2 mL syringe, filled either with procaine 2% or with 20 mg of triamcinolone. The needle is introduced at an angle of approximately 60° to the horizontal, aiming at the centre of the vertebral body (Fig. 40.32b). Normally, the needle goes in without meeting any particular resistance to its full length, where it is felt to meet the posterior aspect of the vertebral body. Sometimes the tough resistance of cartilage can be felt at full depth, which indicates that the needle has engaged against the disc.

If the needle hits bone or meets some ligamentous resistance at a depth of 3–5 cm, it has confronted the lateral aspect of the lamina or the posterior aspect of the facet joint. The needle must then be almost withdrawn and pushed in again in a straighter direction.

Occasionally, the transverse process is encountered and is indicated by bony contact at a depth of 5–6 cm. In this case, the needle is partly withdrawn and redirected slightly inferiorly.

If the needle touches the nerve root, the patient experiences a sudden, sharp electric 'shock' shooting down the leg. The sensation is only momentary and disappears immediately.

As soon as bone or cartilage is felt, aspiration is performed to make sure the tip of the needle is neither inside the theca nor in a blood vessel. If clear fluid is aspirated, injection with procaine should not be made, for fear of spinal anaesthesia. The needle should therefore be withdrawn immediately and the whole procedure postponed for 2 days. If blood flows back into the syringe, the tip of the needle can be slightly altered until it lies outside the vessel. If neither blood nor cerebrospinal fluid is aspirated, 1 mL is injected with the tip of the needle in contact with bone or cartilage, i.e. at the site of the sinuvertebral nerve. The needle is then drawn back 0.5–1.0 cm. Renewed aspiration is performed and the rest of the fluid injected at the level of the foramen and consequently around the nerve root. Normally, no particular tissue resistance is apparent. In a postlaminectomy patient, however, fibrosis may impede the injection.

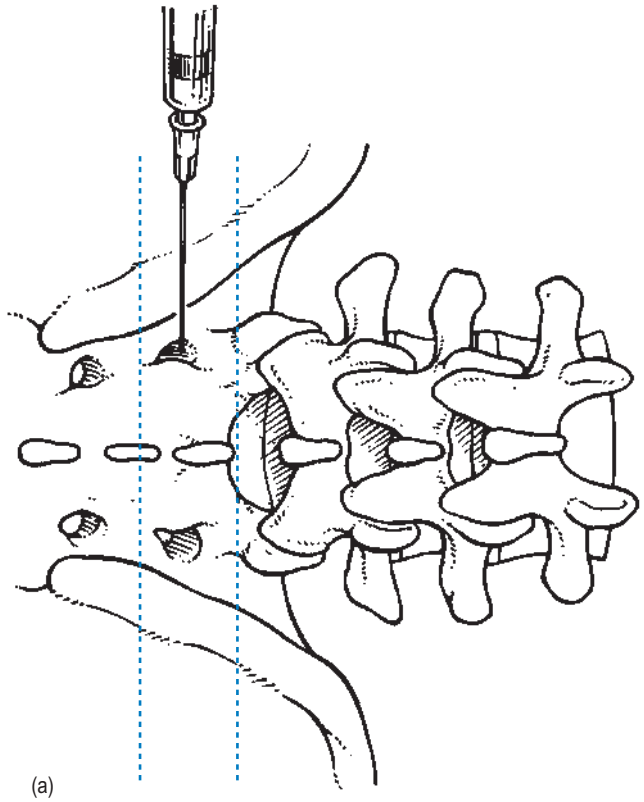
Usually, there is no discomfort in the back or down the limb. Tissue resistance and increasing root pain during injection indicate that the tip of the needle remains in the nerve root tissue or the spinal ganglion. The injection is not continued unless the needle has been withdrawn over a small distance because pressure necrosis can result.[181]

After the injection of local anaesthetic, the patient turns supine and nerve root mobility is tested afresh. If the correct point has been chosen, there will be a marked improvement in range and pain. If not, the injection is repeated, either the same day or at the next attendance, at one of the other lumbar levels. If an injection with triamcinolone solution has been made, pain relief should be evaluated after 1 week. Improvement of symptoms calls for a second injection at the same level. If no progression is noted, another level must be tried.

Technique for the first sacral root

The patient adopts a prone position with the arms alongside the body and the pelvis slightly tilted to relax the muscles and to reduce the lumbar lordosis. The edge of the ilium and the two inferior iliac processes are identified.

The first sacral foramen is a large aperture which lies at the level of the apex of the first sacral spinous process. The latter is located about 2 cm above a horizontal line connecting the two posterior and inferior iliac processes (Fig. 40.33a). The



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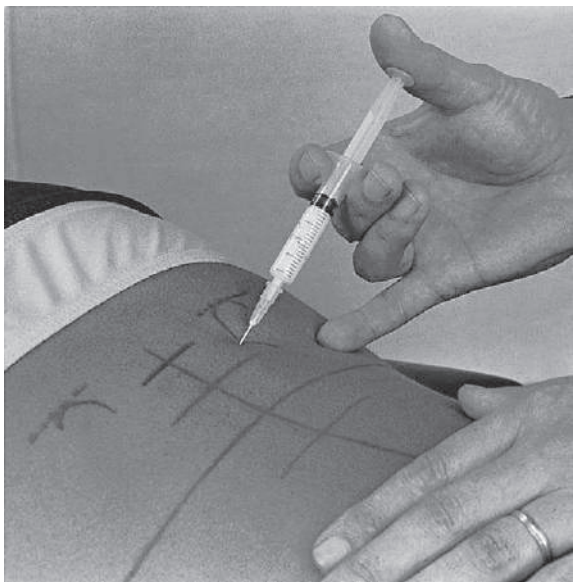


(b)

Fig 40.33 • Injection of the first sacral root.

first dorsal sacral foramen lies on this line, some 3 cm from the midline.[182]

A 7 cm needle with short bevel is fitted to a 2 mL syringe containing procaine 2% or 20 mg of triamcinolone. A point is chosen on the horizontal line as far from the midline as the ilium allows. The needle is then inserted in a 45° (oblique) direction, aiming at the dorsum of the sacrum (Fig. 40.33b). This oblique insertion is preferred because a transverse insertion could easily cross the whole sacrum, pierce the anterior foramen and possibly puncture the bowel. This would be disastrous because contamination would be transferred into the epidural space when the needle is withdrawn. The obliquely inserted needle usually meets the dorsum of the sacrum at a depth of 3–5 cm. It is then moved about in slightly different directions until the resistance of a tough ligament is felt. After the needle has penetrated this, no more resistance is felt and the needle passes freely for 1–2 cm into the epidural sacral space.

Injection can now be made after careful aspiration has been performed; aspiration is done because the point of a needle placed obliquely through the first sacral foramen into the sacrum can easily penetrate the dura. Cautious aspiration before injection is therefore vital. Cerebrospinal fluid flowing back into the syringe is an absolute contraindication to injection of local anaesthetic.

Injection of ligaments

Interspinous and supraspinous ligaments

The injection is made for both diagnostic (lidocaine 2%) and therapeutic (10 mg of triamcinolone) reasons when local inflammation in the ligament or the periosteum of the spinal process is considered to be the cause of symptoms. Sclerosing injections of the interspinous and supraspinous ligaments of the two lower segments are also given as part of the stabilizing treatment in recurrent disc displacements.

Technique

The patient lies prone, with a small pillow under the pelvis. Interspinous ligaments L5–S1 and L4–L5 are easily located: usually, the horizontal line joining the upper edges of each iliac crest falls between the supraspinous processes of L4 and L5.

A 2 mL syringe, fitted to a 5 cm 21 G needle, is filled with the solution. At the required level, the needle is inserted vertically through the ligament and moved slightly upwards so that it touches the inferior border of the spinous process of the vertebra above; 1 mL of the fluid is then injected as a series of droplets deposited at the ligamentoperiosteal junction (Fig. 40.34). Then the needle is partly withdrawn and reinserted in the direction of the upper surface of the spinous process of the vertebra below. Using the same procedure, another millilitre of the fluid is infiltrated here.

In order to steer clear of any vital structures, including those in the spinal canal, the injection should not be made unless the tip of the needle touches bone. Considerable tissue resistance may be encountered when the fluid is forced in. For this reason, the needle should not be too thin.

Should the infiltration be part of a sclerosing treatment for recurrent disc displacement, both fourth and fifth lumbar levels must be infiltrated. A mixture of 1 mL of lidocaine 2% and 3 mL of sclerosant is then distributed over both levels.

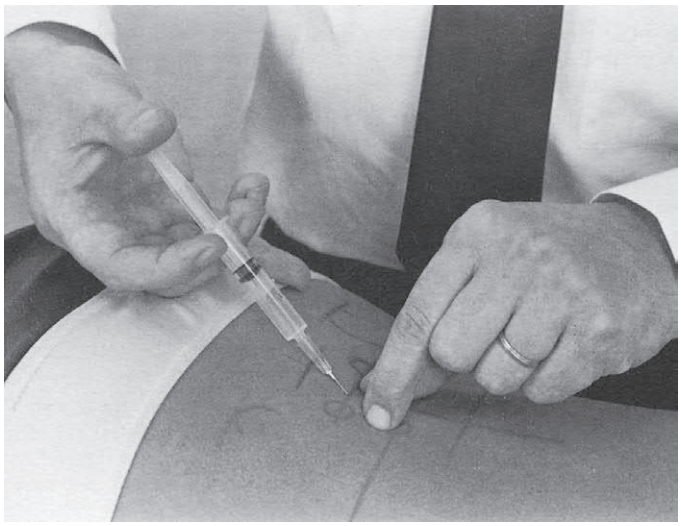


Fig 40.34 • Injection of the inter- and supraspinous ligaments (L5–S1).

Posterior capsule of the lumbar facet joint

Backache seldom stems from a facet joint lesion, which is not a very reliable clinical diagnosis.[183] However, facet joint pain may result from traumatic overstretching of the dorsal capsule.[184] Although the facet joints are richly innervated by the dorsal rami of two consecutive roots,[185] only the fibrous capsule contains nociceptive free nerve endings and can be the possible source of facet-derived pain.[186] The aim of the treatment is therefore not to inject the joint but to infiltrate the dorsal capsule.

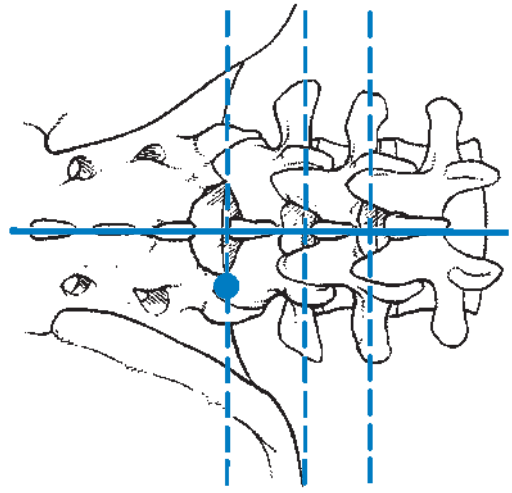
Infiltration with 20 mg triamcinolone is also indicated in ankylosing spondylitis, as well as in traumatic overstretching. However, the common reason for infiltrating the facet joints is postural backache. Here a sclerosing solution is used to cause capsular sclerosis. Capsular sclerosis is also indicated when, after reduction, the fragment of a disc proves unstable.

Some recommend that intra-articular injections should be done in the radiology department under fluoroscopic control[187,188] or under CT guidance of the needle.[189] Infiltration of the posterior capsule of the joint can, however, be performed blind. Finding the facet joint ligaments with the tip of a needle is not difficult. It has been shown that pain relief is achieved equally well with intra-articular and periarticular injections, indicating that the pain may be of capsular origin rather than synovial inflammation.[190]

Technique

The patient lies prone on a couch, with the pelvis slightly elevated in order to reduce the lordosis. This posture enables better palpation of the spinous processes and the intervals between them. The patient keeps the arms alongside the body so as to relax the sacrospinalis muscles.

A horizontal line connecting the two iliac crests is drawn. It usually falls between the spinous processes of L4 and L5. Other horizontal lines are drawn between the processes of L3 and L4, and L5 and S1. Finally, the midline is drawn. The centres of facet joints L4–L5 and L3–L4 usually lie on the cross-bars at about 1.5 cm from the midline. At the L5–S1 level, the joint is located slightly more lateral at 2.0–2.5 cm from the midline.



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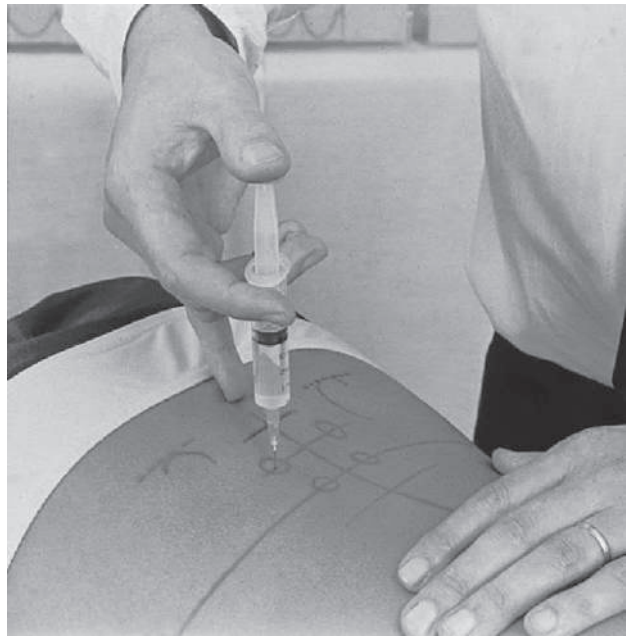


(b)

Fig 40.35 • Injection of the posterior capsule of a lumbar facet joint.

A 5 cm needle is inserted at the surface marking of the joint and thrust in vertically downwards through the sacrospinalis muscle (Fig. 40.35b). Depending on the build of the patient, the needle reaches the lamina at 3–6 cm depth. The needle should encounter tough ligament before reaching bone. As it meets bone, the point lies against the lamina. One millilitre of the fluid is injected at different places over the surface of the joint. Considerable tissue resistance may be encountered. In order to remain clear of any vital structures, no injection should ever be made unless the tip of the needle is felt to impinge on bone.

For diagnostic purposes, 1 mL of lidocaine 2% can be used. In traumatic overstretching of the joint or in ankylosing spondylitis, 20 mg of triamcinolone is injected. A sclerosant is injected in the two lower pairs of facet joints when capsular sclerosis is required (see Ch. 34).

Deep lumbar fascia

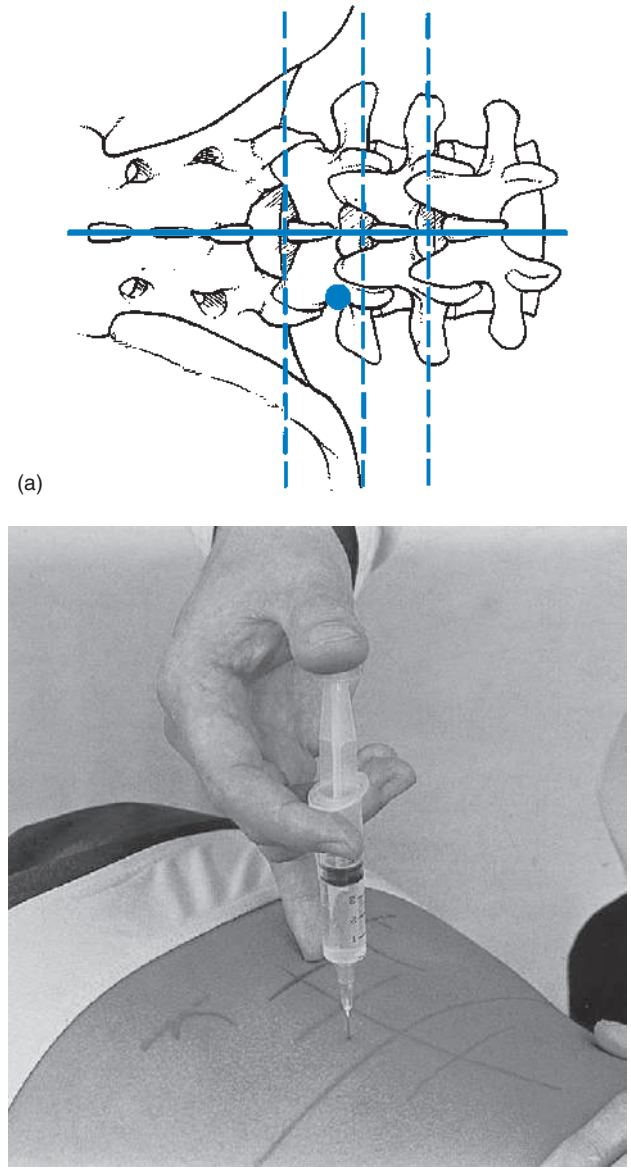
The middle layer of the thoracolumbar fascia is attached to the lumbar transverse processes and is continuous with the intertransverse ligaments. It is also attached to the edge of the lamina, where its fibres blend with those of the posterior layer of the thoracolumbar fascia.[191]

The transition between deep lumbar fascia and lamina at both L5 and L4 levels is infiltrated with sclerosant solution as part of the treatment of chronic postural backache or to induce ligamentous contraction in the treatment of recurrent disc displacements. An injection at the lateral border of the lamina also reaches the posterior ramus, where the fibrils that supply the facet joint curl round the bone to continue on the dorsal aspect of the lamina. Injection of a sclerosant (phenol solution) at the edge of the lamina will therefore also induce facet denervation.[192–194]

Technique

The patient lies prone, the pelvis slightly tilted and the back muscles as relaxed as possible. Three transverse lines, crossing the L3–L4, L4–L5 and L5–S1 supraspinous ligaments, and the midline are drawn. Points chosen between the horizontal lines and 1.5 cm from the midline lie exactly above the laminar edges (Fig. 40.36).

The syringe is filled with either a local anaesthetic or a mixture of sclerosant and local anaesthetic, and fitted with a needle 5 cm long. The needle is inserted at the appropriate level. It is then thrust vertically downwards until it touches bone at a depth between 3 and 6 cm (the depth depends mainly on the size of the patient). The needle is then partly withdrawn and reinserted in a slightly more lateral direction. This is repeated until the operator feels the needle slide along the edge of the lamina; 0.5 mL of the solution is injected here. Care is taken to maintain bony contact with the edge of the lamina during the injection. The needle is then partly withdrawn and reinserted in a slightly more medial direction, where it touches the surface of the lamina. Another 0.5 mL of the solution is then injected.



(a)

(b)

Fig 40.36 • Injection of the deep lumbar fascia.

Iliolumbar ligaments

These ligaments play an important role in stabilization of the lumbosacral junction.[195] They are especially important in maintaining torsional stability of the junction (see p. 420).[196,197] An iliolumbar strain can stem from overstretching.[198]

Technique (Troisier[142]: p. 396; Naeim *et al*[199])

Infiltration of the ligament is made at the iliac insertion. A 5 mL syringe is filled with 4 mL of the sclerosant solution and 1 mL of procaine 2%, and fitted with a 7 cm needle.

The iliac crest and the posterior superior iliac spine are palpated. A horizontal line is drawn, connecting the upper edge of each iliac crest. One thumb is held at the medial edge of the iliac crest. The needle is inserted on the horizontal line, about 3 cm lateral to the supraspinous processes (Fig. 40.37). The tip of the needle is thrust in very obliquely in the direction of the palpating thumb until it is felt to traverse a resistant ligament before touching bone. The infiltration is performed along the border, deeply and superficially by multiple withdrawals and reinsertions. It is important to realize that the zone of insertion extends over 3–4 cm. Caution must be taken to inject only when the needle touches bone.

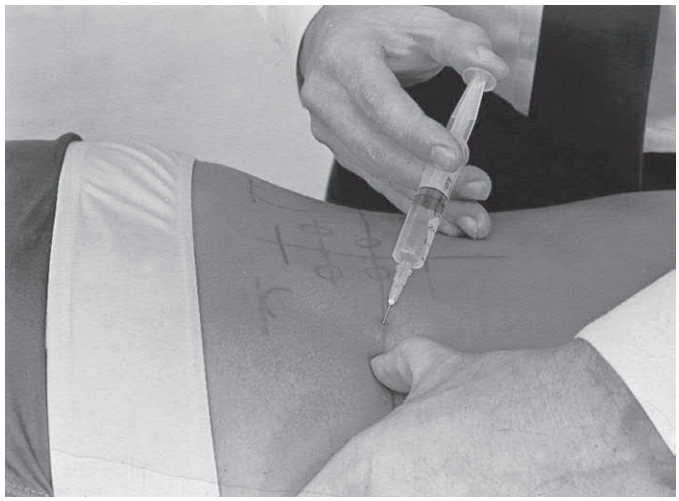


Fig 40.37 • Injection of the right iliolumbar ligaments.

Sclerosant injections at the lower lumbar spine

From 1956 onwards, Hackett used sclerosant injections at the ligamentous periosteal junctions of the posterior lumbar arch as a treatment for chronic low back pain.[200,201] The solution initially used consisted of zinc sulphate and carbolic acid. In the late 1950s, Ongley chose to use a dextrose–phenol–glycerol solution, originally developed for the treatment of varicose veins.[202] This mixture has a good safety record and causes few side effects. It provokes an effective inflammatory response, which leads to fibroblast proliferation and new collagen production (O. Troisier, cited by Cyriax[21]: p. 339).[203] The tightening and permanent shortening that result limit vertebral mobility and thus are beneficial in preventing recurrent disc displacements. Phenol is also known as a very powerful neurolytic agent.[204,205] The fact that some of the phenol solution injected at or around the medial and lateral branches of the posterior ramus causes chemical denervation[194] adds to the therapeutic effect of the injections. Chemical denervation probably explains the rapid relief (sometimes from the day after the injections) obtained by a number of patients treated with sclerosant injections.

Local sclerosant injection is the treatment of choice in a local posterior dysfunction syndrome. The purpose then is to create long-standing denervation of local nociceptors or of branches of the dorsal ramus.

In recurrent disc displacements, or in chronic backache caused by postural ligamentous pain, a series of infiltrations is made in all the dorsal ligaments of L4–L5–S1 motion segments, or the L3–L4–L5 motion segments.

Technique

Over 3 consecutive weeks, 3 mL of the solution, mixed with 1 mL of lidocaine 2%, is infiltrated in the different lumbar ligaments at the ligamentous periosteal junction, in the following sequence:

- The first injection is given at the interspinous and supraspinous ligaments L4–L5 and L5–S1 and at the iliac insertions of the iliolumbar ligaments.
- The second injection, 1 week later, reaches the posterior aspects of apophyseal joints L4 and L5 on both sides.
- The third injection, 1 week later, is given at the lateral aspects of the laminae of L4 and L5, where the ligamentum flavum and the medial aspect of the deeper layer of the fascia lumborum merge.

Remarks

Usually, there is considerable after-pain for the 24–48 hours following each treatment session but the pain seldom necessitates bed rest or absence from work.

For 6 weeks, which is the time needed to induce sufficient sclerosis of the tissues, the patient should avoid bending forwards or sitting in kyphosis, in order to avoid stretching the ligaments. If, during this period of contraction of the fibrous tissue, a new discodural interaction develops, manipulative reduction should be carried out at once.

Results

The results of these injections are reasonably good.[206] In chronic postural backache, about 70% of patients become pain-free after 6–8 weeks. If, after 6 weeks, there is improvement but no cure, the series can be repeated. If, on the other hand, no improvement is reported after 2 months, it is pointless repeating the injections. Our personal experience with sclerosant treatment shows that there is usually little or no tendency to recurrence. Sometimes the patient has to return for a 'booster' after 2 years.

Prevention of lumbar spine disorders

Introduction

Prevention is the ideal approach to low back pain. Classically, there are three types of prevention. Primary prevention aims to avoid the causes before they occur; secondary prevention endeavours to reduce the frequency and severity of low back episodes; and tertiary prevention attempts to reduce disability in patients with chronic problems.[207]

Primary prevention, although the best approach, is hard to achieve. It is extremely difficult to motivate changes in behaviour, especially if the condition at risk is not life-threatening. However, primary prevention programmes are important in industry. In a good one, efforts should not be focused on the worker alone. A good ergonomic approach to the workplace (plant, office and cars) is the first prerequisite, education of the employee the second.[208]

Secondary prevention is the responsibility of every therapist. Once resolution is complete and the patient has recovered from an episode of backache, the question arises as to how to

prevent further recurrences. The patient should be told about the mechanism of disc protrusions and the way they can be prevented, and motivated to attend a back school programme where education about proper back care and the reduction of future episodes is more thorough.[209]

In tertiary prevention, the focus is on the patient in whom treatment did not completely succeed and it is necessary to adapt lifestyle to persistent pain. The causes of backache are many and prevention strategies must be adapted to the mechanical situation that pertains.

Prevention in discodural interactions

Once a displacement is reduced, whether by manipulation, traction or the passage of time, the patient must be warned that the probability of a new attack remains very high. Because of its avascularity, repair of disc tissue is almost non-existent and what has shifted once can shift again when subject to the same provocation. Recurrent episodes of backache thus occur in up to 85% of patients. Severity and duration also tend to increase with each recurrence.[210,211] Recurrences therefore increase the risk of chronicity, disability and eventually surgery. These facts are reason enough to inform the patient in as detailed a way as possible about the mechanism of disc protrusions and how they can be prevented in the future. There are two basic principles:

- Avoid long periods of immobility.
- Keep the back in the 'physiological' position, which is lumbar lordosis.

Immobility causes drying out and quicker degeneration of disc material and is therefore deleterious to disc tissue (see pp. 438–439). The patient should be advised to change position as frequently as possible.

A slight physiological lordosis, the normal and natural posture of the lower back, is the consequence of the wedge-shape of the lumbar discs. The patient should try to keep the back hollow in all positions. First, it has been demonstrated that in this position intradiscal pressure is at its lowest.[212] Second, in a lordotic posture, the intervertebral joint is more open in front than behind. During this asymmetrical loading, the resultant of the compressive forces pushes the nucleus in a forward direction. Bending, in contrast, results in a tensile stress on the posterior part of the annulus and is a compressive backward force on the nucleus (Fig. 40.38). A posterior movement of the nucleus then threatens the dura and nerve roots. Several biomechanical studies have demonstrated that the normal nucleus moves posteriorly in kyphosis and anteriorly in lordosis.[213–216]

The patient should understand the necessity of keeping the back hollow at all times. All advice is based on the simple principle, 'keep your back hollow' (Fig. 40.39), and a real effort should be made to render this second nature.

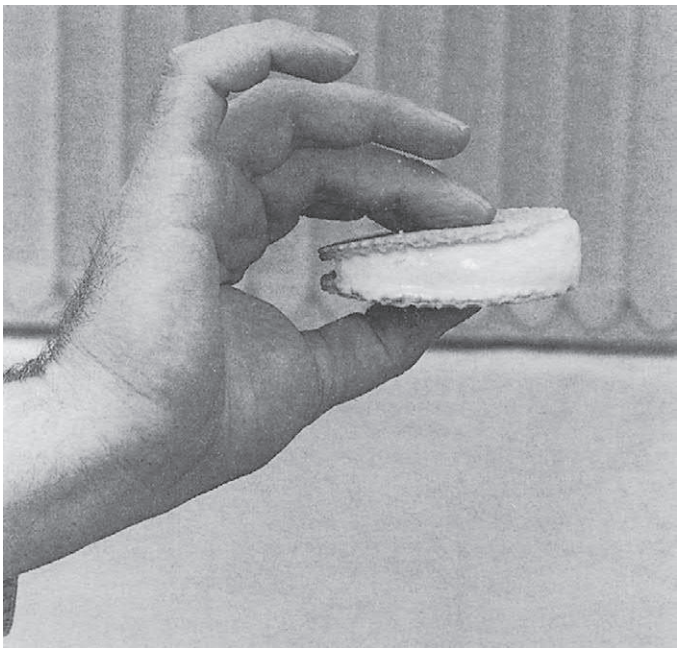


Fig 40.38 • Analogies are useful in explaining mechanical principles to patients. If the spine is loaded in kyphosis, the disc tends to move backwards.

Sitting posture

Nowadays, people sit for a great deal of their life: in cars, trains and buses, at the office and at home. It should be made clear to the patient that sitting is the worst position for the back because it increases the pressure inside the disc. Sitting positions should therefore be avoided but, if they are necessary, a proper position on a good chair must be chosen. A good chair allows the user to correct the hollow in the back. Of importance are the height and depth of the seat, and the inclination and shape of the back rest.

Height of the seat

This should adjust to the height of the individual (Fig. 40.40). Ideally, the thighs rest slightly on the seat when the knees are at right angles and the heels rest on the ground.[217]

If the seat is too low and the patient holds the legs straight, the hamstrings pull the pelvis forwards, which results in flexion of the lumbar spine from below; or the knees and hips are kept bent, which once again rotates the pelvis forwards and forces the lumbar spine into flexion.[218] If the seat is too high, the whole pelvis glides forwards and the lumbar kyphosis increases.[219]

A slight anterior inclination of the seat may be advised when the patient sits at a desk (Fig. 40.41). In this position the pelvis rotates forwards; the lumbar spine follows and is moved towards lordosis.[220] The patient adopts a straight, erect posture which is more comfortable when working at a horizontal desk and keeps the lumbar spine hollow.[221]

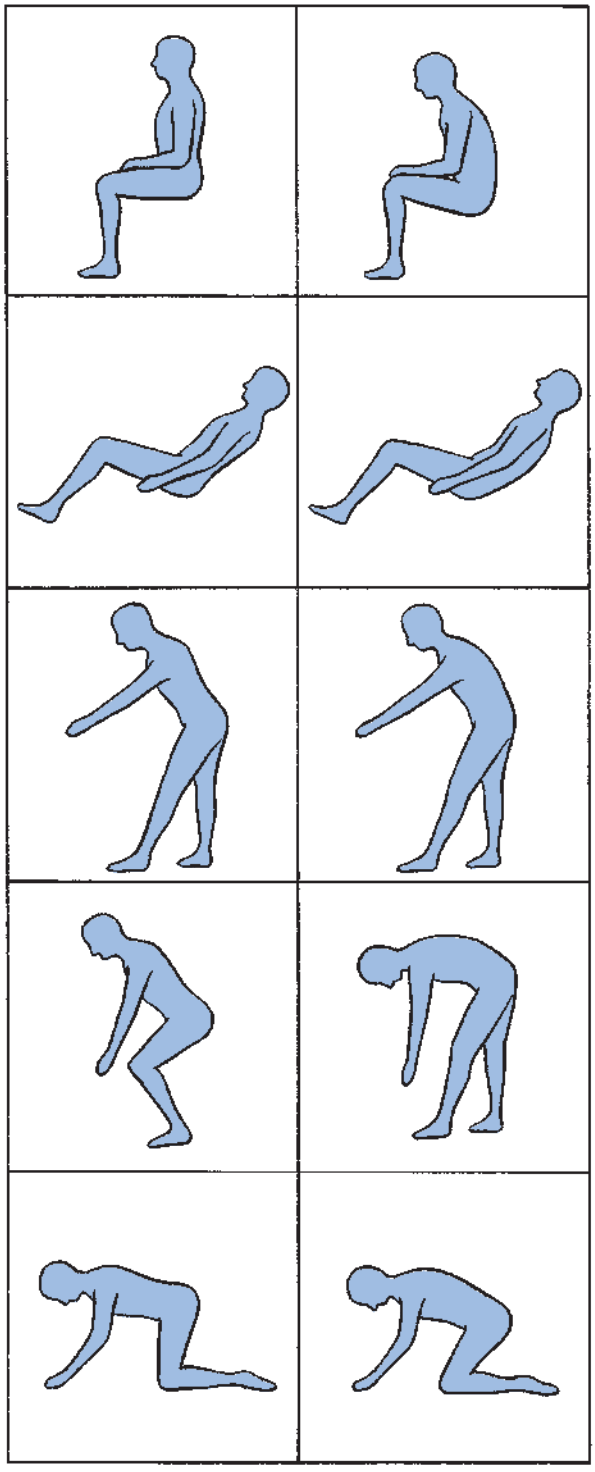


Fig 40.39 • Correct postures for keeping the back hollow (left). Postures to be avoided are also shown (right).

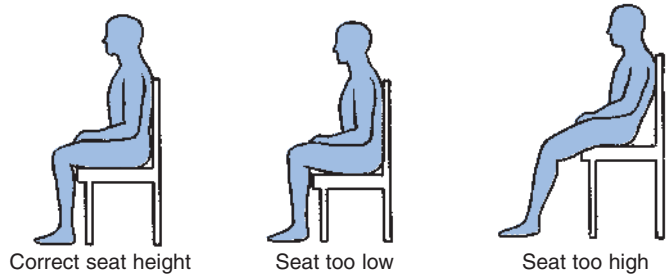


Fig 40.40 • Seat height.

Fig 40.41 • Slight anterior inclination of the seat, for sitting at a desk.

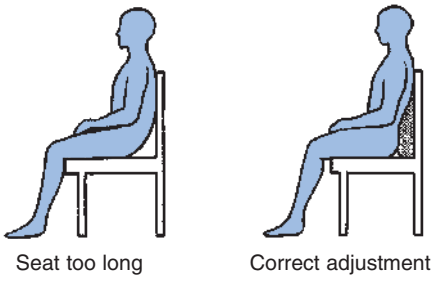


Fig 40.42 • Seat depth.

Depth of the seat

The seat should be no deeper than the distance from the back of the knees to the sacrum. If it is longer, the sitter cannot position the lumbar spine against the back rest and the spine is forced into a convex position (Fig. 40.42). Should this be

the case, the gap must be filled with a hard support such as a cushion, briefcase or rolled-up coat.[222]

Inclination of the back rest

The angle between the seat and the back rest should not exceed 100°. An increase in inclination is followed by an increased backward rotation of the pelvis and kyphosis of the lumbar spine.[223]

Lumbar support

The chair should be fitted with a well-stuffed, hard lumbar support. The level of the support is at the L3–L4 region, which is the level at which the forearm lies when put behind the back, with the elbow bent at a right angle (Fig. 40.43). Normally, this is between 19 and 26 cm above the surface of the seat.[224]

Posture in the car

Car seats often cause a great deal of trouble. Aerodynamic principles force modern manufacturers to design the coupé as low as possible. As a result, the car seat is also low, which is harmful for the driver's back: the legs are held nearly straight and pull the pelvis forwards. The principles set out above can only be followed if a backward inclination, together with a corresponding tilting of the seat, is achieved. A back rest inclination of 110–120°, with a back rest-to-seat angle of 90–100°, is optimal (Fig. 40.44). A hard lumbar support, approximately 8 cm thick and between 20 and 25 cm above the horizontal part of the seat, should be used to maintain the lordosis. Inclination of the seat has several advantages:

- The buttocks slide backwards and are pushed into the angle between the horizontal and the vertical parts of the seat.
- With the thighs resting on the seat, the knees move upwards so that the angle between thighs and lower legs decreases, which relieves tension on the hamstrings.
- The upper trunk moves dorsally, which increases the contact between the back and the back rest. A large proportion of the body weight is then transmitted to the back rest and the axial load on the spine is reduced. The distance between the back rest and the steering wheel should be such that the arms are nearly straight. A position with completely extended arms prevents adequate contact between chest and back rest, increases the load and forces the back into kyphosis.[225,226]

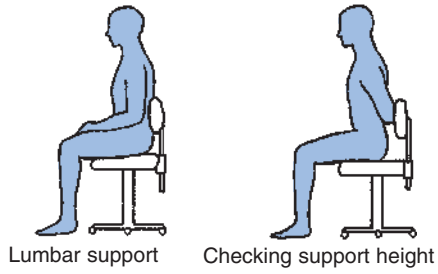


Fig 40.43 • Lumbar support.

The patient should realize that simply having a proper, well-adjusted chair is not sufficient. It must also be used in the proper way, forcing the buttocks backwards as far as they will go, until the sacrum touches the back rest; the support on the back rest then pushes the lumbar spine forwards into lordosis. A randomized study showed that sitting with a lumbar support at the L3–L4 level resulted in reduction of lumbar pain.[227] All sitting positions that are performed in kyphosis should be avoided. Sitting with the legs crossed, for instance, is harmful: the uppermost thigh draws the pelvis forwards, which flexes the lumbar spine from below. Also, in sitting with the legs out straight (i.e. in bed or in the bath), the hamstrings rotate the pelvis forwards, which results in considerable kyphosis.

Standing posture

Standing does not increase intradiscal pressure and no posterior displacements of discal tissue need be feared when a normal lordotic posture is maintained. However, working in a standing position usually implies a slight bending of the spine (e.g. working at a desk, sweeping, vacuum cleaning), which considerably increases intradiscal pressure.[228] Having the work surface at the right height and using proper equipment can prevent this (Fig. 40.45). The work surface for standing should be some 8–10 cm (the height of a fist) under the elbow. For most people, this is 30–40 cm higher than the top of the kitchen table and about 10–15 cm higher than most work surfaces. Washbasins should also be built in higher than they usually are. Cleaning materials – brushes, vacuum cleaners and floor-mops – should be long enough to allow proper use, with the body kept upright.

Lifting and carrying

'Use your legs, not your back'

In order to decrease leverage, it is extremely important to keep the distance between the load and the body as short as possible.[229] Therefore, lifting should never be done with straight legs.[230] The best way to pick up a load is by squatting (Fig. 40.46). Care should be taken to arch the back in lordosis by

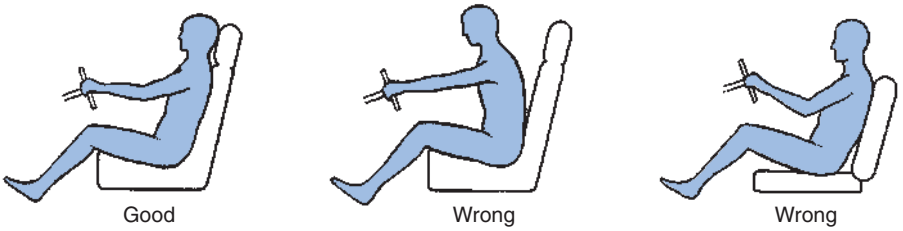


Fig 40.44 • Car seat adjustment.

Fig 40.45 • Standing posture.

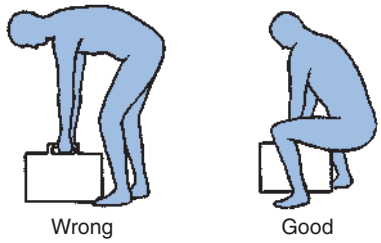


Fig 40.46 • Lifting.

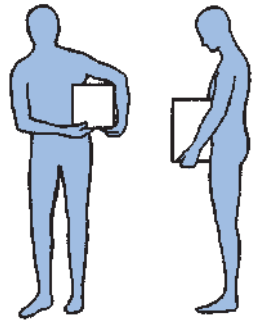


Fig 40.47 • Carrying: the load should be held close to the body.

contraction of the sacrospinalis muscles, *before* lifting starts. Straightening the knees and hips, while the lumbar spine is kept as immobile as possible, then lifts the load.

'Avoid combined flexion and twisting of the trunk'

A combination of torsion and bending maximally stretches the posterolateral annulus and is likely to cause annular failure and protrusion.[231,232] To avoid this, the patient can be taught to pivot on the feet rather than twisting the trunk.[233]

In carrying, the load is placed as close as possible to the body; the further the distance between body and load, the larger the intradiscal pressure and the more the spine is forced into kyphosis.[234] It is also a good idea to carry loads on one iliac crest (Fig. 40.47). The load is then transmitted directly to the leg, rather than via the lumbar spine.

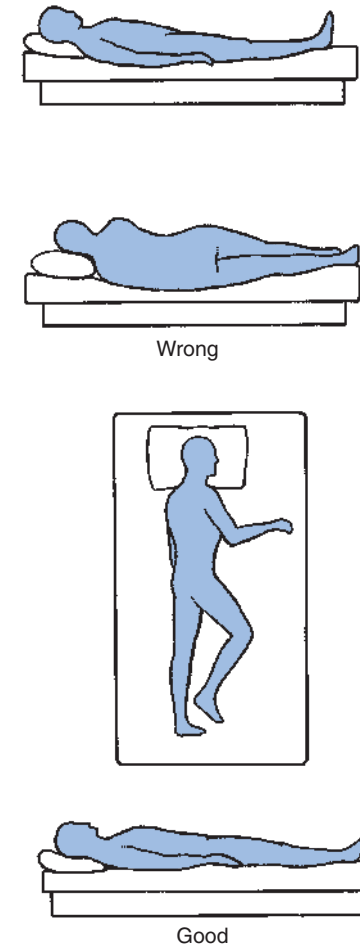


Fig 40.48 • Posture in bed.

Posture in bed

A lot of backache results from lying on sagging mattresses. Lying on the back in the hammock position forces the lumbar spine into kyphosis and may result in gradual nuclear displacements. The mattress should therefore be firm and positioned on a hard surface (boards or laths). The best way to lie on it is prone, the chest slightly turned to one side. The upper leg is slightly bent, and the lower leg out straight (Fig. 40.48). This position is excellent, as it maintains the lumbar concavity.

What about exercises?

Exercises that enhance muscle strength cannot prevent disc displacements. The patient should be told that a strong back and strong abdominal muscles do not prevent posterior migration of disc tissue. Only the maintenance of good posture during the activities of daily living can do this. It is not the strength of the muscles but the way they are used that is of importance. Furthermore, back exercises aiming at increasing muscle power can be damaging for the disc: prone-lying extension exercises increase the intradiscal pressure five-fold, and sit-up exercises six-fold.[235] Exercises that augment or maintain lumbar mobility are also contraindicated. Displacements will be prevented by keeping the lumbar spine still, not by moving it.

In contrast, exercises that increase strength and mobility of the hips and the knees should be encouraged.[236] Transferring the stress from the spine to the legs implies good function (strength and mobility) of these; therefore exercises make sense but only as a complement to instruction and postural training.

Only one prophylactic 'exercise' is logically defensible and proves to be of some value in patients with recurrent attacks of back pain: extension exercise under passive traction (Cyriax[21]: p. 226). The patient puts the hands on a table and straightens the arms. The legs are placed as far back as possible. By bringing the shoulders almost vertically above the outstretched arms, the trunk is allowed to sag. This results in passive extension during traction provided by the weight of pelvis and legs (Fig. 40.49). The position is sustained for 20–30 seconds. The traction causes a decrease in intradiscal pressure and the extension results in a forward shift of the disc. The exercise is performed 5–10 times a day.

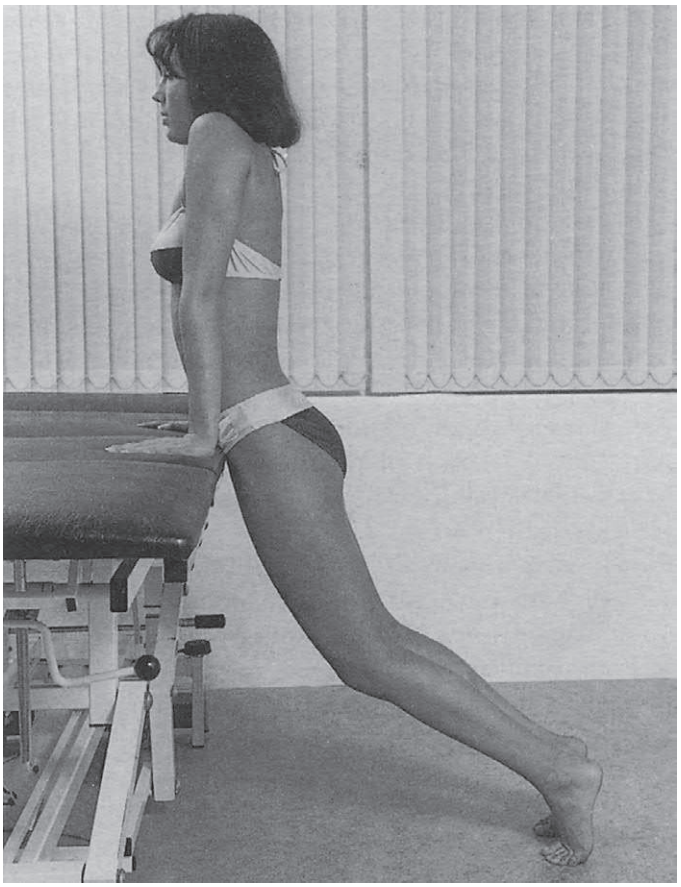


Fig 40.49 • Extension exercise under passive traction.

What about sport?

Swimming, walking and running are safe. In swimming the trunk floats, which precludes any compression strain. In running and walking, the lumbar spine remains in physiological lordosis, which prevents posterior migration of the disc. Riding also seems to be quite harmless, as long as the rider uses the muscles to keep the back hollow.

Ball games may provoke sudden annular displacements. The player does not always have full control over the movements performed or the positions the body is forced to adopt. Tennis, badminton and volleyball have an especially bad reputation because they are 'jerky' and require a lot of stooping and twisting. Basketball and soccer are safer.

Rowing involves full forward bending. Once a disc lesion has developed, it is extremely difficult to continue the sport, even if the rower trains to achieve perfect technique. Canoeing requires a sitting position with extended knees. This is a bad posture because the hamstrings pull the pelvis forwards, which results in kyphosis. Patients with back pain should therefore avoid this sport.

Prevention in ligamentous disorders

(Box 40.10)

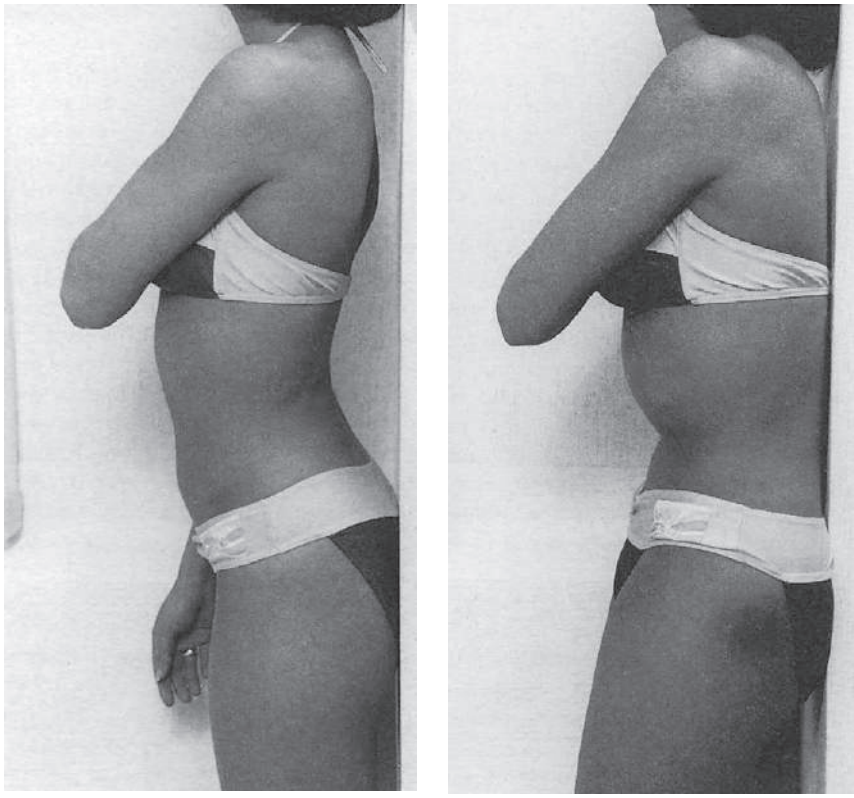
Lumbar ligamentous pain appears when the posterior ligaments are subjected to abnormal mechanical stress: because of the decrease of intervertebral height, too much load is applied on the posterior compartment of the spinal axis. The clinical picture is intermittent pain, induced by maintenance of prolonged positions and abolished by posture correction or movement. Maintenance of lordosis or hyperlordosis especially stresses the posterior facets and increases postural pain.

To prevent postural pain, the patient should avoid constant static postures. Hyperlordosis in particular should not be permitted. Proper attention should be paid to correction of the hyperlordotic position. Pelvic tilting in the standing position or while standing against the wall with the knees slightly flexed (Fig. 40.50) should be performed several times if the patient has to stand for any length of time. In addition, moving the body weight from one leg to the other changes postural tone and thus prevents postural backache. It is also advisable, when standing, to place one foot on a small footstool or bar (15–20 cm high; Fig. 40.51). This allows slight flexion of the hip and relieves the lordotic pressure.

Box 40.10

Prevention in ligamentous disorders

- Avoid the maintenance of prolonged positions
- Avoid hyperlordosis
- Keep moving



(a) (b)

Fig 40.50 • Correction of hyperlordosis (a) by pelvic tilting (b).

Fig 40.51 • Use of a footstool to relieve lordotic pressure.

Prevention in stenotic disorders (Box 40.11)

Compression of the nerve roots in a narrowed spinal canal is not constant. The size of the canal changes in relation to posture. An axial load on a degenerated spine decreases the size of the canal: the posterior longitudinal ligament folds and buckles in a posterior direction, the superior articular process moves forwards and upwards in relation to the vertebra above, and the pedicles tend to push downwards on the nerve roots. Extension further narrows the space and therefore adds to the compression on the roots. Flexion relieves the compression, as it stretches the posterior longitudinal ligament and moves the superior articular process away from the root.

Patients suffering from stenotic disorders must therefore be encouraged to flatten the lumbar spine as much as possible in all positions. The sitting position in kyphosis is most comfortable. In standing and walking, however, it is extremely difficult to maintain some kyphosis in the lumbar area. In order to achieve this, the patient can be trained in pelvic tilting (active forward movement of the pelvis, which straightens the back), first while lying on the floor, then while standing against the wall with the knees slightly flexed. Once the technique of pelvic tilting has been mastered, it should be practised for 5–10 minutes several times a day. In early cases of spinal stenosis or lateral recess stenosis, these pelvic tilting techniques have proved to be efficient.

Box 40.11

Prevention in stenotic disorders

- Avoid lordosis
- Avoid axial load

Spinal surgery

The incidence of spinal surgery is highly variable and differs from country to country. In the industrialized world the lowest incidence is in the UK: fewer than 10/100 000 Britons undergo disc excisions or lumbar fusions each year. The rate of back surgery in the US is more than five times that in England and Scotland. In the USA, it is estimated that 69.5/100 000 have a lumbar discectomy each year, and an additional 31/100 000

undergo spinal fusion.[237,238] Even within the same country there remain large differences. Volinn and colleagues have demonstrated almost 15-fold differences in low back surgery rates among counties in Washington State.[239] Given that there is no difference in the incidence of sciatica and low back pain, this large variation between regions and countries can only be explained by a lack of medical consensus on the indications for surgery. A report of the US Institute of Medicine also concluded that 'surgery for chronic low back pain is overused and often misused'.[240] Moreover, recent studies demonstrate that the higher the regional disc surgery rate, the poorer the outcome[241] and the higher the reoperation risk.[242]

Successful surgery to the lumbar spine is based on an unequivocal cause of symptoms; congruence between this and the findings on imaging; a lack of undue influence exerted by the psychosocial circumstances surrounding the patient's illness; and conviction on the part of all concerned that surgery will produce better results than allowing the condition to evolve naturally. Violation of these principles is the dominant cause of failure, rather than failure to choose the right operation from the variety of surgical procedures currently available.[243] Surgical intervention should therefore not be undertaken lightly and adequate weight must be given to non-surgical treatment or even to natural spontaneous resolution. Sometimes, however, surgical treatment is absolutely necessary and should not be postponed.

In deciding for or against surgery, the following factors are important. First, most of the conditions that are broad indications for lumbar spine surgery have a normal and natural recovery.[244,245] Second, the results of surgical procedures are not perfect. Even in the best hands and after using strict preoperative criteria for selection, the immediate success rates of discectomy for sciatica do not exceed 90%,[246] and long-term success is less than 80%.[247,248] If the main complaint has been backache, only half the patients will find relief of their symptoms.[249,250] The rate of reoperation during the subsequent 4 years is 10%,[251] and increases to 15% if the interval is 5 years.[252] Further surgical procedures on those who have not obtained relief have a very low chance of success.[4,178] Third, the risk of non-intervention should be known. Is there a possibility of continuing disability if a patient with an incipient muscle paresis is not sent for surgery? It has generally been accepted that motor weakness and/or sensory loss are major reasons for surgical intervention. There is little or no debate that a cauda equina syndrome is an absolute and urgent indication for lumbar disc excision. However, most muscle paresis of the lower limb is not severe and is a sign that is detected by the doctor rather than a phenomenon apparent to the patient – patients with weakness are usually quite unaware of it. In a single root paresis, recovery is the rule. Usually after some months, and sometimes before the patient loses the root pain, the muscles begin to strengthen and the skin regains its sensitivity (Cyriax[21]: p. 330). Hakelius and Hindmarsh[253] showed that delays of up to 3 months had only a minimal effect on the ultimate recovery of strength. In a retrospective study of 166 patients who underwent surgery and 417 patients who were treated non-surgically, Hakelius[254] could not find a statistical difference between the two groups in recovery from muscle paresis and from sensory loss. Also, the classic prospective controlled study by Weber[255] showed equally good restoration of muscle weakness in surgical and non-surgical groups. However, if a disc protrusion causes a double palsy of two adjacent roots, permanent weakness may be found. Such may be the case in an L4–L5 palsy caused by a protrusion at the fourth lumbar level and resulting in a drop foot. If permanent weakness of the dorsiflexors is feared, decompression surgery should be performed without delay because delayed laminectomy does not guarantee restoration of muscle power.

Surgery for discodural or discoradicular interactions

Lumbar disc herniations can be treated by different interventions: discectomy via the intralaminar approach, microdiscectomy, percutaneous discectomy and chemonucleolysis with chymopapain. Comparison of these treatments is not possible because no controlled prospective studies have been carried out.

Lumbar discectomy has, in the past, been referred to as lumbar laminectomy. This term is not appropriate because it suggests complete exposure of the neural elements.

The advantages of microdiscectomy are the smaller incision and the more rapid recovery. The disadvantages are technical difficulty, a higher infection rate, the increased risk of missing significant disorders, operating at the wrong level and the higher recurrence rate.[256]

In percutaneous or suction discectomy, a cannula is placed against the annulus. A cutting tool is moved into the sheath to carve a hole in the annulus. Small curved forceps are then introduced into the disc space and as much nuclear material as possible (usually 4–6 g) is evacuated.[257] The success rate of the procedure is between 50 and 75%.[258,259] In percutaneous laser disc decompression, the cannula contains a glass fibre, enabling the application of laser energy to destroy the nuclear material.[260] Although the exact mechanism of these techniques is not known, the benefit of the procedure presumably relies on the mechanism of disc decompression. The procedure is contraindicated for a sequestered disc.[261] The advantages of the technique are the low risk of complications, the short duration of treatment and the quick recovery. However, the results are still too poor to consider the treatment a useful intervention: in a recent randomized clinical trial, the treatment was only successful in 44% of the patients.[262]

Chemonucleolysis is the treatment of intervertebral disc lesions by intradiscal injection of the enzyme chymopapain.[263] The protease rapidly degrades proteoglycan core protein and generates fragments containing only a few glycosaminoglycan chains. These diffuse out of the tissue and the water-binding capacity of the disc is lost, so reducing the intradiscal pressure and hence pressure on the entrapped nerve root.[264] The results are slightly inferior to those of surgical excision.[262] A serious disadvantage of the procedure is increasing low back pain for up to 6 weeks after the injection. Also, a small number of catastrophic allergic and neurological complications have been recorded.[265–267] Chemonucleolysis can be a viable treatment option for younger patients with a lumbar disc protrusion causing radicular pain.[268,269]

Indications

There is little debate about the absolute indications for lumbar disc excision when the patient presents with a cauda equina syndrome or an incipient drop foot. The dominant complaint, however, in the majority of patients with lumbar disc herniation, is pain rather than significant neuromuscular dysfunction. Although muscle paresis is usually considered by most neurosurgeons as an indication for early surgery, it still has to be proved that recovery from neuromuscular dysfunction occurs more rapidly with surgery than with non-surgical treatment.[270,271]

Absolute indications (Box 40.12)

Third and fourth sacral root palsy

Weakness of the bladder with incontinence during an attack of acute lumbago or sciatica calls for immediate laminectomy.[67] The outcome of a cauda equina syndrome is dependent on rapid recognition and early surgery because the compression is preganglionic and therefore no recovery will take place. The important features are bilateral root pain with bilateral neurological symptoms; perineal pain, paraesthesia and/or numbness; and the presence of urinary dysfunction and/or loss of rectal sphincter tone.

Incipient drop foot

Dorsiflexion of the foot is controlled by the L4 and L5 motor roots. A large herniation at the fourth lumbar level may cause a pressure atrophy of both. The tibialis anterior and the dorsiflexors of the toes are then completely paralysed and usually remain so. Hence, if there is major and rapidly progressive muscle weakness of the dorsiflexors of the foot, immediate laminectomy should be considered as the best hope of rapid restoration of muscle power.

Severe intractable root pain

What matters is not the severity of the lesion but the severity of the pain. This indication has nothing to do with the existence of neurological signs but rather with the presence of continuous and intractable pain; the patient has received all possible conservative treatment, including epidural local anaesthesia, possibly with the addition of triamcinolone and nerve root blocks, but the symptoms remain as severe as ever. Discectomy most often proves necessary in sciatica, with or without neurological deficit, and accompanied by gross lumbar deformity: the patient presents with a gross side flexion or flexion deformity and any attempt to straighten the back results in severe root pain shooting down the limb (see Ch. 36). Alternatively, there may be considerable deviation away from the side of the pain during flexion, and side flexion towards the pain is inhibited by severe root pain.

Relative indications

Most surgeons consider root pain with neurological signs as a primary indication for discectomy.[246] However, it is our firm belief that slight or moderate muscle weakness alone does not indicate that surgical intervention will be necessary. If only one nerve root is involved, recovery from the palsy is the rule. Usually, the muscle begins to recover soon after the pain has ceased. Although it is true that root atrophy may sometimes lead to permanent weakness and loss of sensitivity or permanent loss of the ankle jerk, these slight inconveniences cannot always be prevented by an operation. Several retrospective and prospective studies comparing surgically and non-surgically treated patients did not demonstrate any difference in recovery from the palsy.[253,255,272]

Therefore weakness is not a primary indication for surgery but for epidural local anaesthesia; desensitization of the nerve root is all that is required. Pain passes away quickly and often permanently; recovery from neurological weakness usually follows after some months.

It should never be forgotten that most patients with sciatica recover within a year, no matter what sort of treatment has been instituted. It is appropriate to recall that, in the prelaminectomy era, all but a few patients with sciatica recovered from their pain and disability. Therefore, a patient who suffers from tolerable root pain, even in combination with neurological signs, and who does not respond to conservative treatment should be advised to wait until spontaneous recovery has had a chance to take place. If the signs and symptoms remain unchanged after 8 months, surgery may then be considered. Unfortunately, leg pain lasting more than 8 months also correlates with a less favourable postoperative outcome.[273]

Box 40.12

Surgery for discoradicular interactions: indications

- Fourth sacral root palsy
- Incipient drop foot
- Severe intractable root pain
- No result after 6–12 months of conservative treatment

Failure after decompressive surgery

The estimated rate of failure following decompressive surgery varies widely and ranges from 5% to over 50%, with an average of 15%.[274,275] The reason for failure is either incorrect diagnosis, surgery at the wrong level, the wrong operation, an unsuitable patient or technical problems. The history once again may be very helpful in assessing the probable cause of failure. There are three possibilities, as described below.[179]

The patient has no initial relief of symptoms or the symptoms are worse: diagnosis, indication or level was wrong

A major reason for failure is psychosocial: the patient is not cooperative or is not anxious to get well – there may be a compensation claim, job dissatisfaction or psychological disturbances.[276,277] Operations should be considered carefully in such patients: prolonged disability after the operation is to be expected. Even if there is genuine organic trouble, the operation will be followed by intermittent backache.[278] An operation should therefore always be avoided if the patient's personality

is thought to preclude the achievement of a satisfactory outcome.

Sometimes failure results from missing the actual level of involvement. This follows segmentation abnormalities or mislabelled imaging studies. The latter is not uncommon and results from the rather low specificity of diagnostic imaging.[279–283] The accuracy of CT and myelography is between 80 and 87%,[284,285] and therefore it should not be surprising that the wrong level can be operated on if imaging studies only are taken into account in the decision to operate.

Furthermore, one of the most important sources of continuing radicular pain is the overlooking of a concomitant lateral spinal stenosis at the time of surgery.[286–288]

There has been initial relief, followed by gradual onset of recurrent radiculopathy at the same segment

It is likely that gradual scarring around the nerve root has set in; it is estimated that such root scarring accounts for 12% of all failures.[289,290]

There has been complete relief of symptoms but recurrent problems develop months or years later

A new disc displacement, at either the same or another level, may have occurred. Alternatively, the disc space narrowing resulting from the excision is contributing to the development of a lateral recess stenosis.

Surgery for spinal stenosis and lateral recess stenosis

In a narrowed central canal, a wide laminectomy that extends laterally on both sides as far as the facet joints is recommended.

When lateral recess stenosis is present, laminectomy and facetectomy are performed. The operation is usually executed on both sides because the opposite side is often narrowed too. So at any one level, four nerve roots should be explored (L4–L5 at the level L4 and L5–S1 at the level L5).[175]

The outcome of surgery for spinal stenosis is good to fair in 75% of the patients, although about 33% continue to have severe backache.[291] Also, the reoperation rate seems to be rather high: 23% of the patients undergo reoperation of the spine during a follow-up period of 8 years.[292] Factors associated with a good outcome are: a clear myelographic stenosis, no prior surgical interventions, no diabetes, no hip joint arthrosis and no preoperative fractures of the lumbar spine.[293]

Lumbar arthrodesis

Spinal arthrodesis may be indicated when gross lumbar instability has set in. A number of different approaches and techniques exist to stabilize two consecutive vertebrae or the lumbosacral junction. Using a posterior approach, these techniques include: posterolateral fusion, posterior lumbar interbody fusion (PLIF), and transforaminal lumbar interbody fusion (TLIF). More complex techniques use an anterior approach (e.g. anterior lumbar interbody fusion – ALIF) or a combined approach, known as anteroposterior fusion or 360° fusion. All use bone grafts after the segment has been stabilized using different types of fixation (cages, screws, plates, wires, hooks and rods).[294,295]

Indications

Spondylolisthesis

The disorder is found in 5–7% of the population[296] but surgical stabilization is only called for if specific signs and symptoms are present:[297]

- Progressive and symptomatic slippage of more than 33% or an initial symptomatic slip of more than 50% in a child
- The persistence of ligamentous lumbar pain or bilateral sciatica despite sclerosant therapy.

Degenerative spondylolisthesis

A degenerative spondylolisthesis is a vertebral slip in spite of an intact neural arch. The slippage is caused by a combination of disc degeneration, general joint laxity, increase of mechanical stress and structural abnormalities of laminae and facets.[298–300] It occurs most often at the L4–L5 level and seldom progresses by more than 33%.[301–303] As the displacement progresses, the patient may present with backache or bilateral sciatica. Arthrodesis must be considered if sclerosant injections do not provide benefit.[303]

Lumbar instability

The patient suffers from recurrent severe low back pain caused by repetitive discodural interactions. The pain and dysfunction may be promptly relieved by manipulation but reappear after an apparently trivial twist or strain. If the condition continues despite conservative treatment such as back school and sclerosant injections, arthrodesis should be performed. The key premise, however, is that selective fusion will only relieve the symptoms if the 'unstable level' can be identified with certainty. To define the level, discography[304] or sinuvertebral blocks (Cyriax[21]: p. 223) can be useful. Also, short-term external fixation can select those patients who will benefit from fusion.[305]

Nuclear self-reducing protrusions

Pain comes on in the morning and gets slightly worse as the day goes on. The patient is comfortable lying down. Each morning the pain has vanished but reappears on getting up. Repeated sclerosant injections into the dorsal ligaments have not helped; nor did epidurals. The only effective treatment in this case is arthrodesis (Cyriax[21]: p. 338).

Total disc replacement

The purpose of this technique is to restore and maintain spinal segment motion and to relieve pain, while avoiding instability and protecting the adjacent discs and facet joints from undue degeneration. The surgical approach is similar to the one used to obtain an anterior lumbar interbody fusion and carries

Box 40.13

Indications for spinal surgery

Discectomy

Absolute indications:

- Third and fourth sacral palsy
- Incipient drop foot
- Severe intractable root pain

Relative indications:

- Failure of conservative treatment

Laminectomy

- Narrowed central canal
- Narrowed lateral recess

Arthrodesis

- Gross lumbar instability:
  - Spondylolisthesis
  - Recurrent disc protrusions
  - Nuclear self-reducing disc

similar rates of vascular complications.[306,307] Replacing a degenerated joint instead of fusing it was considered for the spine due to the success of total knee and hip arthroplasty.

Several reviews of the published literature and clinical trials have been performed to investigate the efficacy and safety of total disc replacement. The conclusion is that the long-term complications and benefits of total disc replacement are similar to those for lumbar fusion.[308–310]

Indications for spinal surgery are summarized in Box 40.13.

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Clinical examination of the sacroiliac joint

41

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Introduction

Sacroiliac arthritis, both rheumatic and septic, is a common cause of unilateral gluteal pain. The sacroiliac joint is a true synovial joint and therefore subject to the same inflammatory and infectious conditions that affect other synovial joints.[1,2] However, the sacroiliac joint as a primary source of mechanical backache remains controversial and diagnoses such as 'sacroiliac dysfunction' or 'sacroiliac subluxation' are ill defined and difficult to substantiate.[3–5] Much of the controversy is caused by lack of agreement on clinical criteria that justify the diagnosis of sacroiliac lesions. If the diagnosis is made only on the basis of pain localization or tenderness on palpation, sacroiliac lesions will be seen frequently. If the diagnosis is based on a thorough clinical examination of lumbar spine and pelvis, sacroiliac lesions will be rare. Unilateral pain and tenderness in the region of the sacroiliac joint is usually a manifestation of the confusing phenomena of referred pain and referred tenderness from a dura mater at a low lumbar level and not a reliable clinical proof of a sacroiliac disorder.[6–9] Furthermore, mechanical testing of the sacroiliac joints – exerting tension on the sacroiliac ligaments *without* affecting the lumbar spine and the hip joint – is not easy. Most commonly used tests lack this requirement, which may explain the differences of opinion between examiners.[10–12]

A discussion of the disorders affecting the joint requires definition of the following terms:

- *Sacroiliac strain/sprain* is overstretch or rupture of the capsuloligamentous structures as a result of abnormal joint movement.
- *Sacroiliac instability* is a condition of abnormal joint mobility caused by capsuloligamentous laxity.
- *Sacroiliac subluxation* is permanent displacement of the bony parts forming the joint.
- *Sacroiliac dysfunction* is a reversible decreased mobility of the joint, the result of articular causes.
- *Sacroiliac arthritis* is an inflammatory condition of the joint.

The concept of a 'sacroiliac sprain' as a common cause of backache and sciatica was introduced by Goldthwait and Osgood in 1905 and reinforced by others.[13,14] It still persists. However, MacNab[8] noticed that sacroiliac sprains only occur below the age of 45 and in circumstances creating considerable force on the joint, such as those generated by falls from a height or motor vehicle accidents. Cyriax[7] (his p. 364) advised reserving the term for cases of pain arising from the sacroiliac ligaments in the absence of arthritis. However, difficulty arises in those examples of early sacroiliitis in which pain and clinical signs precede the appearance of radiographic sclerosis.

Diagnosis can also be suggested by negative findings. For example, absence of articular signs in the lumbar spine, full range on straight leg raising, a normal end-feel to the hip joint and the absence of neurological and arterial signs suggest possible involvement of the sacroiliac joint, when there is pain in the buttock, posterior thigh and calf.

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Clinical assessment does not differ from that described for the lumbar spine in low back pain disorders (see Ch. 36). When data from the history and functional examination suggest the possibility of a sacroiliac lesion, special tests can be performed and additional imaging studies requested.[15]

Referred pain

In sacroiliac joint lesions, pain may be either localized or referred to the buttock and leg. The distinction between pain referred *to* and *from* the joint is very important and should be the first consideration.

Pain referred to the region of the sacroiliac joint

Most instances are the result of lumbar disc protrusions with segmental (L1, L2, L3 and S1, S2) or multisegmental (lumbar dural) reference of pain. Arthritis of the hip joint is another possibility: pain may be felt in the inner and upper part of the buttock because of segmental reference of this joint in the third lumbar segment (see p. 11).

Pain referred from the sacroiliac joint[16]

The innervation of the sacroiliac joint and its ligaments is wide and complicated.[17] Studies confirm that the anterior portion of the joint is innervated by the ventral rami of L5, S1 and S2, and the dorsal portions of the joint by the dorsal ramus of L5 and a plexus derived from the dorsal rami of the sacral nerves.[18–20] The superior part of the joint and the iliolumbar ligaments have an L2 and L3 origin.

In sacroiliac strain or arthritis, pain is most often felt in the buttock, with radiation to the back of the thigh and the calf but never the foot. Often there is groin pain too.[21] A typical distribution of pain is shown in Figure 41.1.[22]

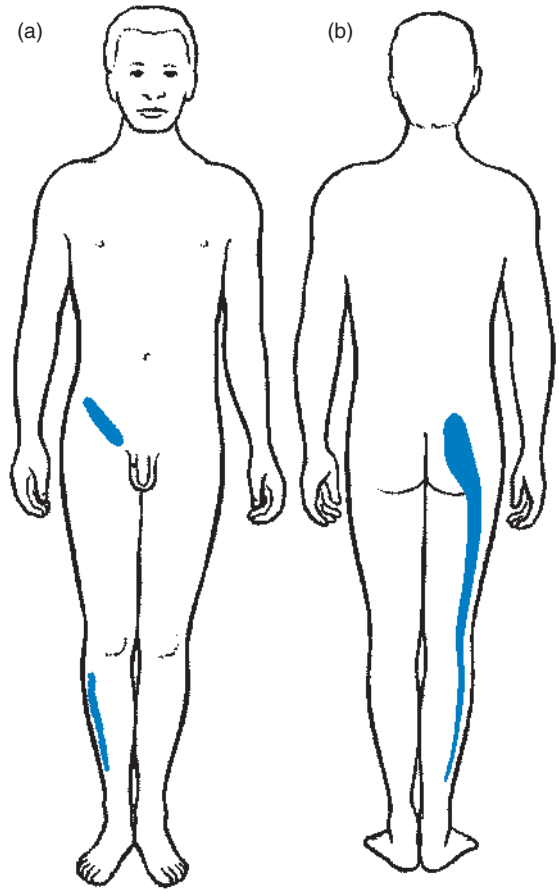

Fig 41.1 • Pain referred from the sacroiliac joint. (a) Pain in the groin may refer from the superior part of the joint. (b) Typical pain referred from the posterior capsule and ligaments.

History

History taking should cover preceding disease, trauma, pregnancy and delivery, occupation and working habits, as well as sports and recreational activities. Family and social history are of similar importance.[23] In pelvic peripartum instability, the pain often starts in the third month or within the first few weeks after delivery.

In sacroiliac lesions, a typical finding is that there is unilateral gluteal pain (often deep, dull and ill defined[24–26]), perhaps together with reference to the S1–S2 dermatomes, chiefly the posterior thigh. Pain may alternate in the right and left buttocks which strongly suggests a manifestation of early ankylosing spondylitis, particularly when it is found in males aged between 15 and 35.

Coughing usually jars the buttock or posterior thigh as the result of the sudden increase in abdominal pressure which distracts the ilium from the sacrum. This sign may be interpreted as being of dural origin, based on the assumption that a disc lesion is present at a low lumbar level. However, if such a lesion is present, pain is normally felt in the lumbar area rather than the buttock.

Some authors describe the adoption of an antalgic gait in painful disorders of the sacroiliac joint[27,28] and a tilt of the trunk, most frequently towards the painless side.[27] Some patients avoid sitting on the buttock of the affected side.[28]

Neurological symptoms, such as paraesthesia, weakness or numbness, are absent.

Functional examination

Introduction

Clinical examination of the sacroiliac joint is not routinely undertaken in low back pain and sciatica, except for one test – sacroiliac distraction (see Fig. 41.3 below) – because this is the most sensitive test to detect inflammation in the joint.

Apart from the positive sacroiliac distraction test, attention may be drawn to the sacroiliac joint by some discrepancies between gross symptoms and slight signs during standing lumbar movements. For example, in acute sacroiliac arthritis,

Box 41.1

Indications for specific sacroiliac tests

- Positive sacroiliac distraction test during the routine lumbar examination
- Discrepancy between gross lumbopelvic pain and slight signs during routine lumbar examination
- Suggestive history for sacroiliac disorder and negative routine lumbar examination

full trunk flexion and side bending to the painful side may provoke pain, but the pain becomes much more severe as soon as the sacroiliac joints are directly tested. Movements of the hip joint may also prove painful, especially both rotations, as the result of an indirect strain on the sacroiliac joint at the end of the movement.

Routine clinical examination of the lumbar spine can be negative but some symptoms may point in the direction of possible sacroiliac involvement because of low back pain and/or sciatica. In sacroiliac strain, the basic tests are usually not capable of evoking pain.[29,30] It is only when movements really stretch the ligament and when this is prolonged that an injured ligament may react painfully. A diagnosis of ligamentous strain is thus based on the history, painful reactions after prolonged load, and the outcome of special sacroiliac tests (Box 41.1).

Sacroiliac tests

These tests can be divided into those that assess movement or position by palpation (palpation tests) and those that stress the structure to reproduce the patient's symptoms (pain provocation tests).

Palpation tests

The best-known palpation tests are the standing and sitting flexion palpation tests and the Gillett test.[31] The Gillett test is performed as follows.

The patient stands with the back to the examiner (Fig. 41.2), who places one thumb just underneath the posterior superior iliac spine and the other on the second sacral spinous process or on the contralateral superior iliac spine. The patient is asked to lift one knee as high as possible, which rotates the ilium on that side posteriorly and therefore moves the posterior superior iliac spine inferiorly relative to the opposite side. Fixation of the sacroiliac joint prevents this movement or, paradoxically, even gives rise to elevation of the posterior superior iliac spine as the patient compensates by tilting the pelvis at the point of maximal hip flexion. This manœuvre is usually painful in symptomatic patients.

The test presupposes normal hip joint function; interpretation is difficult and even impossible in leg-length inequality, lumbar scoliosis and particularly in obese patients, in whom the bony landmarks are difficult to palpate.

To date, palpation tests have not demonstrated acceptable levels of reliability[32–35] and therefore we do not use them.

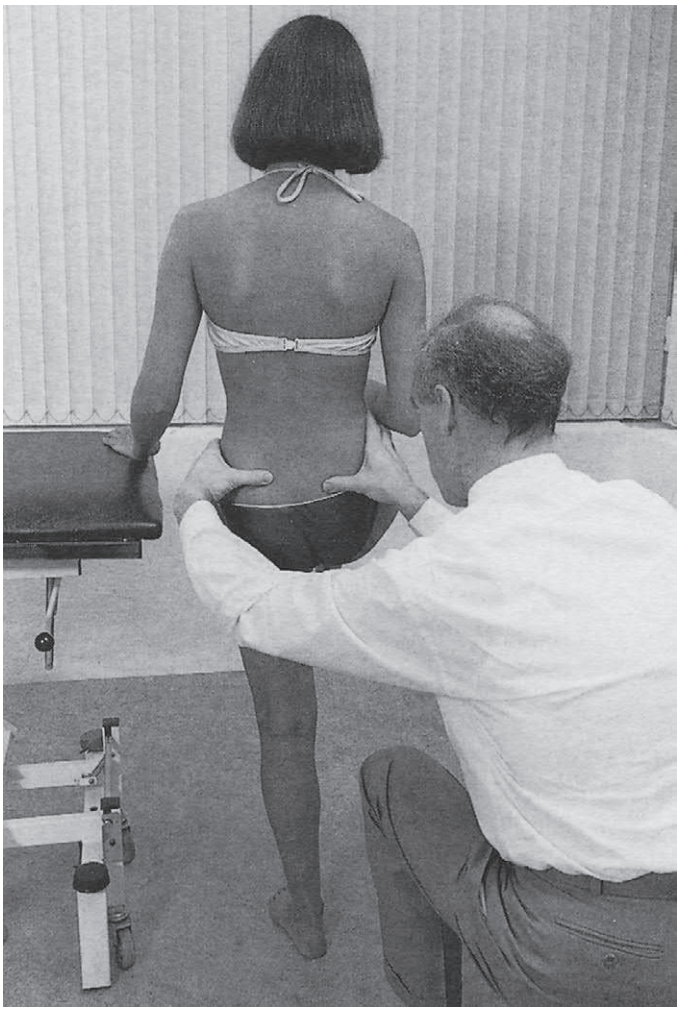

Fig 41.2 • Gillett test to estimate rotation of the sacroiliac joints. The knee on the right-hand side is raised as high as possible. The ilium on that side rotates posteriorly, which can be established by palpation of the posterior superior iliac spine.

Pain provocation tests

Pain provocation tests aim to stress the structures in an attempt to reproduce the patient's symptoms. Studies that have examined these tests show good interexaminer agreement.[36–39]

The main tests are:

- Distraction test or anterior gapping test
- Compression test or posterior gapping test
- Sacral thrust or downward pressure test
- Cranial shear test
- Posterior shear or thigh trust test
- Pelvic torsion or Gaenslen's test
- Yeoman test
- Patrick's or 'faber' test.

Anterior gapping test (Fig. 41.3)

This test, always performed routinely in the basic functional examination of the lumbar spine, has a high sensitivity and an almost 100% specificity for sacroiliac arthritis.[40,41]

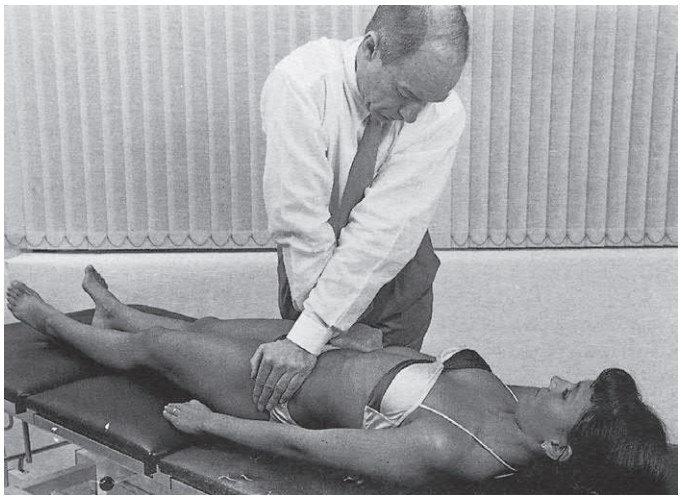

Fig 41.3 • Anterior gapping test.

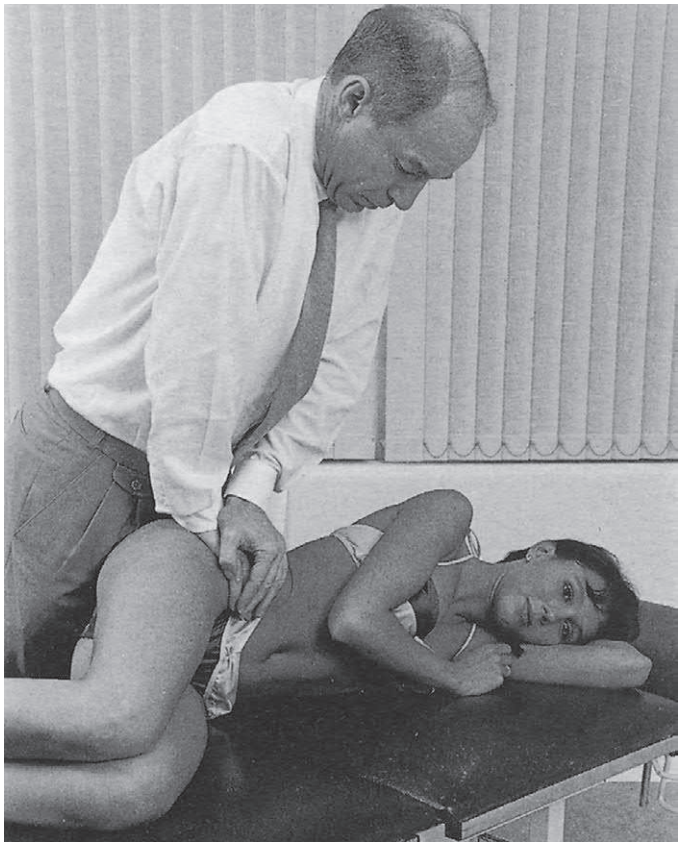

Fig 41.4 • Posterior gapping test.

The patient lies supine. The examiner applies increasing pressure to the anterior superior spines of the ilia in a downward and outward direction. The examiner crosses the arms to do this, in order to increase the strain on the ligaments. If the patient states that the test is painful, it must be ascertained whether pain is located unilaterally gluteal or posteriorly crural. All other outcomes, such as lumbar pain or tenderness at the anterior superior iliac spines, are not relevant. In gluteal or posterior crural pain, the test is repeated, with the lumbar

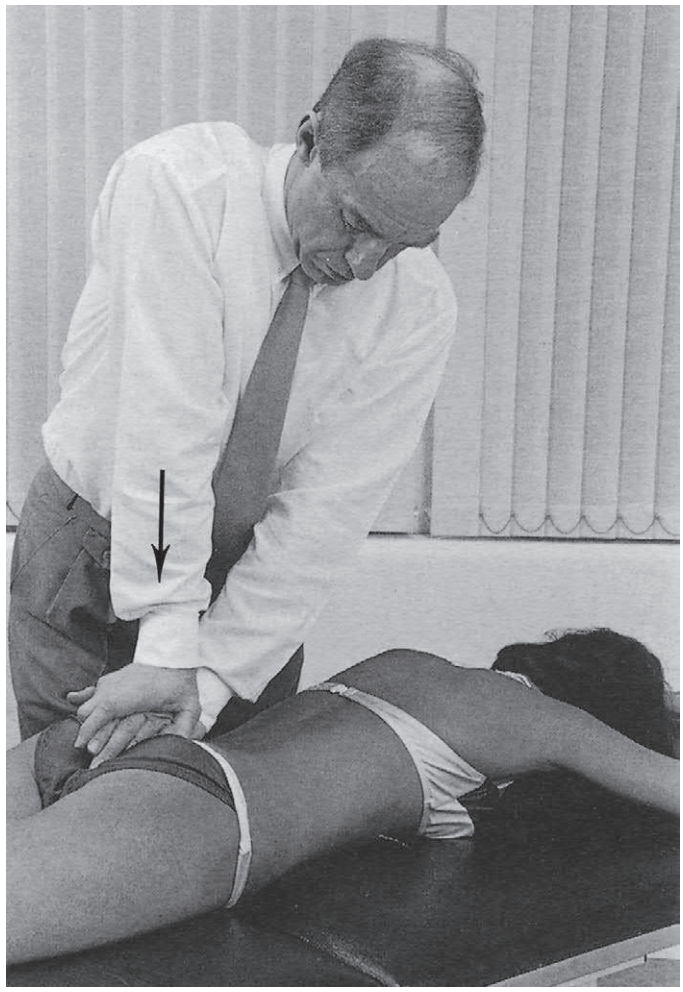

Fig 41.5 • Downward pressure on the sacrum.

region supported by the patient's forearm so as to stabilize the lumbar joints and also prevent compression of a tender part of the sacrum or ilium on the couch. If this does not alter the intensity of pain, it is almost certain that the anterior sacroiliac ligament is at fault.

Other tests to stretch the anterior sacroiliac ligament exist but are less specific because tension is also applied to other structures. Forced lateral rotation at the hip joint with the leg held in 90° flexion is one example and is a routine test in the basic functional examination of the lumbar spine.

Posterior gapping test (Fig. 41.4)

The patient lies on the painless side. The examiner stands behind the patient and exerts pressure on the uppermost iliac crest, directing it towards the opposite iliac crest. The test stretches the posterior sacroiliac ligaments and compresses the anterior part of the sacroiliac joint. This test has the advantage that the sacrum and the iliac spine are not in contact with the couch, excluding the possibility of pain being a result of pressure on a tender area.

Sacral thrust or downward pressure test (Fig. 41.5)

Pressure is applied directly to the sacrum while the patient lies prone. The heel of one hand, reinforced by the other hand, is

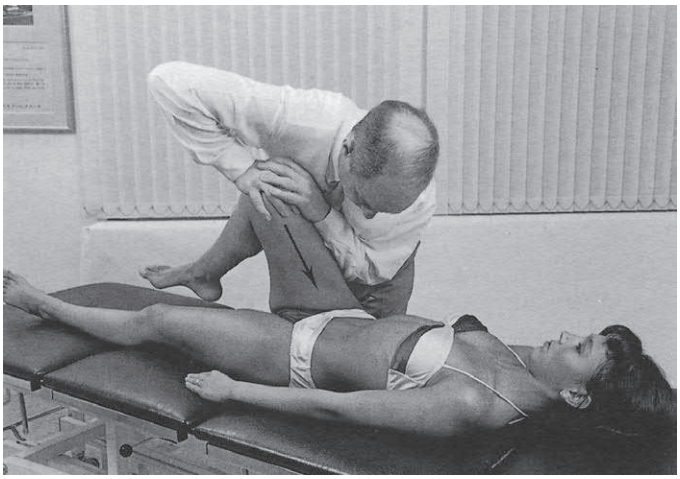

Fig 41.6 • Alternative test to stretch the posterior sacroiliac ligaments (thigh thrust test).

on the centre of the sacrum (S2), and the force is directed anteriorly against the ilia, which are fixed against the examining couch. In sacroiliac lesions, gluteal pain may be provoked on attempted extension by similar pressure on the lumbar spine but is much more severe when pressure is directed to the sacrum. In the *cranial shear test*, the hand is on the coccygeal end of the sacrum and the pressure is applied in a cranial direction.

The following set of sacroiliac pain provocation tests all use the femur as a lever. The results should therefore be interpreted cautiously.

Posterior shear or thigh thrust test (Fig. 41.6)

The patient lies supine and the examiner stands on the painful side. The hip is flexed and slightly adducted. The examiner applies a posterior shearing stress to the sacroiliac joint and ligaments through the femur. Excessive (to the end-feel) adduction of the hip is avoided and the stress should be in a longitudinal direction and not towards further adduction. Some authors believe that this test in particular puts strain on the iliolumbar ligaments and that, if the thigh is maximally flexed and adducted towards the opposite shoulder, axial pressure falls on the posterior sacroiliac ligaments; if the thigh is pushed towards the same shoulder, axial pressure is believed to tense the sacrotuberal ligament.[30]

Pelvic torsion or Gaenslen's test (Fig. 41.7)

This test is performed with the patient in a supine-lying position.[42] One hip is passively flexed and pushed to the chest. The opposing leg is extended passively, hanging over the edge of the couch. Overpressure is applied to force the sacroiliac joints to their end of range: nutation on one side and counternutation on the other. The test should be interpreted with care because it also stresses the psoas muscle, the hip joints and the femoral nerve.

Yeoman's test[43] (Fig. 41.8)

This test is performed with the patient lying prone. The examiner stands at the painful side and puts one hand on the sacrum.

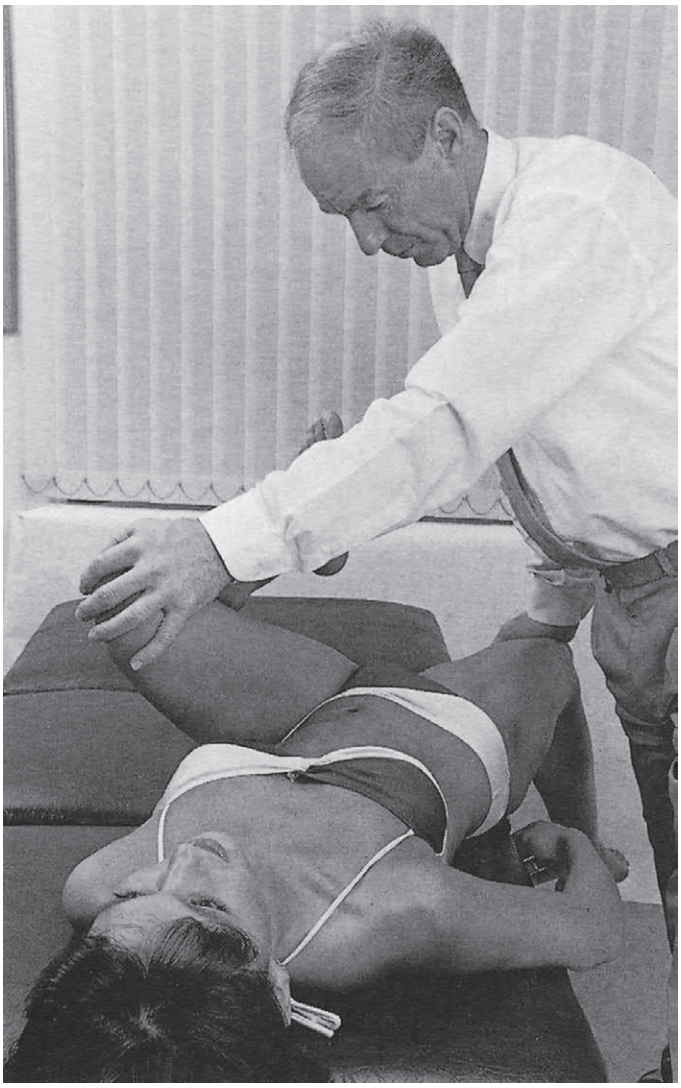

Fig 41.7 • Gaenslen's test.

The other hand extends the hip and at maximum range forces the ilium into anterior rotation. A positive test produces pain over the sacroiliac joint. This test also stretches other structures – hip joint and psoas muscle – and via the latter also puts tension on the lumbar spine. For this reason, this test is not always reliable and should, at the very least, be compared with the results of hip extension with the ilium fixed (the stabilizing hand on the ilium instead of on the sacrum).

Patrick's test (Fig. 41.9)

This test *f*lexes, *a*bducts and *e*xternally *r*otates (*faber*) the femur at the hip joint. After reaching the end of the movement, the femur is fixed in relation to the pelvis. The examiner holds down the anterior superior iliac spine on the opposite side and increases the pressure at the medial side of the knee. This stresses the anterior sacroiliac ligaments, on the side of the abducted leg in particular.[44]

It is good to recall that almost all routine functional tests of the hip indirectly put stress on the sacroiliac joints.

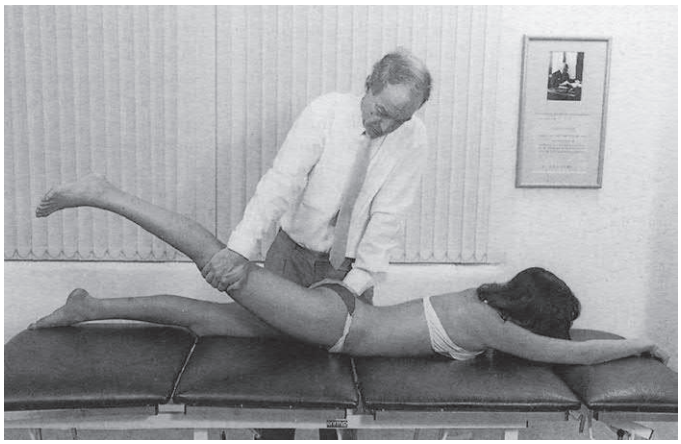

Fig 41.8 • Yeoman's test.

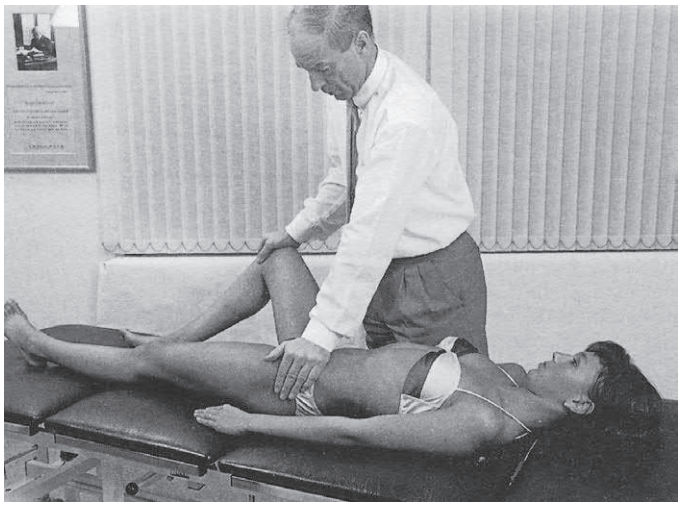

Fig 41.9 • Patrick's test

Box 41.2

Sacroiliac pain provocation tests

Anterior ligaments

- Anterior gapping test
- Patrick's test

Posterior ligaments

- Posterior gapping test

Anterior and posterior ligaments

- Sacral thrust test
- Cranial shear test
- Thigh thrust test
- Pelvic torsion test (Gaenslen's)
- Yeoman's test

- Forced lateral rotation with the leg held in 90° of flexion and resisted adduction of the thigh exert a distraction force at the sacroiliac joints and indirectly stretch the anterior sacroiliac ligaments.
- Forced medial rotation at the hip joint with the hip and knee held in 90° of flexion and resisted abduction of the thigh pull the ilium away from the sacrum. In the absence of hip joint disease, pain experienced over the sacroiliac joint is highly suggestive of a sacroiliac lesion.[8]

Epidural local anaesthesia

Positive confirmation by local anaesthesia, normally so useful in difficult cases, is impracticable here because the anterior sacroiliac ligaments cannot be infiltrated and the posterior mass of ligaments is too large. Cyriax advises the induction of epidural local anaesthesia in patients with what appear to be genuine complaints, painful lumbar and sacroiliac tests and an uninformative radiograph.

Radiology

In sacroiliac strain, nothing is revealed by the radiograph. In arthritis, radiological assessment of the sacroiliac joints is of vital importance. However, clinical signs may precede the appearance of early sclerosis by months or years (Cyriax[7]: p. 360). Normal appearance of the sacroiliac joint therefore does not exclude a diagnosis of ankylosing spondylitis. During recent decades, magnetic resonance imaging (MRI) has become the most suitable method of detecting the early inflammation and structural damage associated with sacroiliac arthritis,[45] and has an estimated sensitivity and specificity of 90%.[46]

Pelvic instability may be detected if the X-ray is taken while the patient is standing on one leg. A shift of the pubic bone on the non-weight-bearing side to a more caudal position implies an unstable symphysis pubis and sacroiliac joint.

Conclusion: the diagnosis of sacroiliac joint disorders should be established from the findings of a careful history and the outcome of physical examination.

Arthritis and strain are accepted conditions. If two or more specific tests to stress the joint prove to be positive, such a disorder is to be considered. However, these tests are only significant when the history and remaining physical findings rule out other syndromes. It is also important to remember that patients with sacroiliac joint lesions can have coexisting sources of pain: for example, lateral recess spinal stenosis or a herniated disc lesion.

Dysfunction and subluxation are difficult to prove, remain obscure and may not exist.

Access the complete reference list online at www.orthopaedicmedicineonline.com

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Interpretation of the clinical examination of the sacroiliac joint and coccyx

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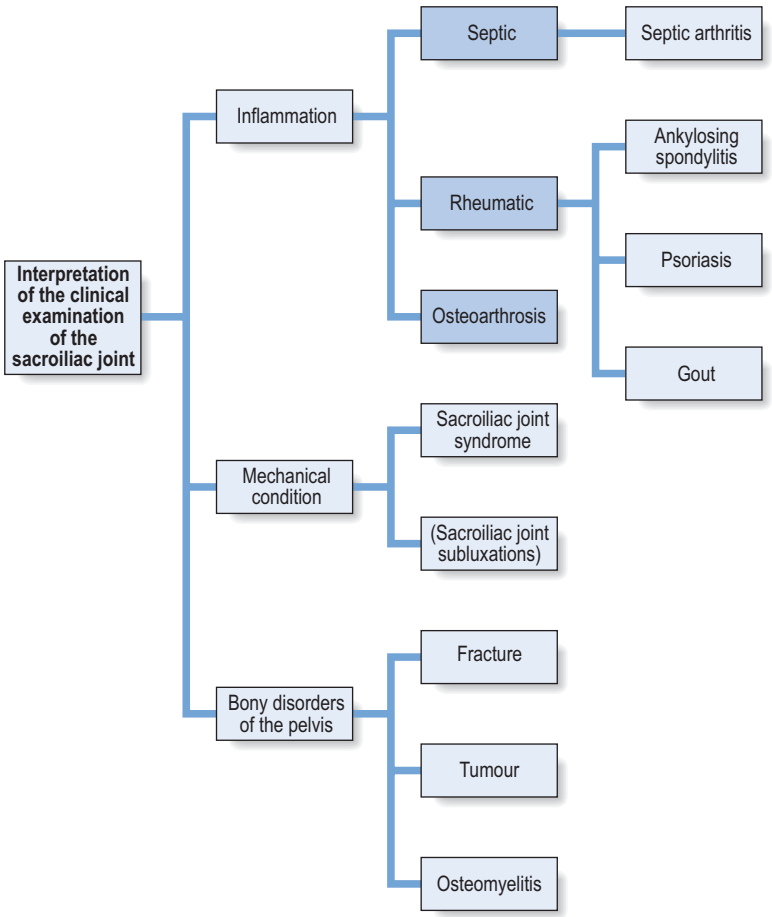

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Disorders of the sacroiliac joint

CHAPTER CONTENTS

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Introduction

Sacroiliac joints are true synovial joints and thus subject to various forms of arthritis and degenerative processes. Although they are relatively immobile – the joint can only rotate 3–5° in the younger subject – they may be susceptible to mechanical trauma.

After the fifth decade of life, fibrosis takes place between the cartilage surfaces and by the seventh decade the joint has usually undergone fibrous ankylosis. The available range of movement decreases as fibrous ankylosis increases.[1]

Most pain in the sacroiliac or gluteal region does not originate from the sacroiliac joint but is referred pain of discodural origin (see Ch. 33); every diagnosis of a 'sacroiliac lesion' should be made with caution and only after other common sources of 'sacroiliac pain' have been ruled out.

The pathological conditions affecting the sacroiliac joint are inflammatory and mechanical. The latter is usually referred to as 'sacroiliac joint syndrome'.[2] The exact nature of the syndrome is not known but it is generally accepted that mechanical pain stems from minor subluxations and/or ligamentous strain.

Sacroiliac arthritis

In the assessment of patients with pain in one buttock, perhaps radiating to the back of thigh and calf, the clinician must always bear in mind the possibility of an inflammatory lesion of the sacroiliac joint. Sacroiliitis is usually the first manifestation of ankylosing spondylitis but may also be associated with inflammatory bowel disease, psoriasis and other more uncommon rheumatic disorders. Sacroiliac gout has been observed. Pyogenic infection of the sacroiliac joints is rare.

Ankylosing spondylitis

Introduction

Once considered a rare disease, ankylosing spondylitis (AS) is now recognized as relatively common, affecting up to 0.5–1.0% of the population.[3] The ratio of occurrence in males and females is approximately 5:1, although it was previously thought to be 20:1. Several studies now suggest that it may occur almost as frequently in females as in males, although in a milder form and with more peripheral localization.[4,5] The disease is characterized by fibrosis and ossification of ligaments and capsules rather than the joint destruction so typical of rheumatoid disease.[6]

Ankylosing spondylitis almost invariably starts at the sacroiliac joints and then extends upwards to involve the spine at increasingly higher levels. However, the sacroiliitis very often remains silent. It has been estimated that no more than 1 case in 10 ever has pain in the buttock. Most spondylitis begins as a diffuse lumbar ache, and sometimes the earlier symptoms

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Box 43.1

Modified 'New York' criteria for ankylosing spondylitis (AS)

Clinical criteria

- Low back pain and stiffness for more than 3 months not relieved by rest
- Symmetrical limitation of lumbar movement
- Limitation of chest expansion to 2.5 cm

Radiological criteria

- Bilateral sacroiliitis, grade II or more
- Unilateral sacroiliitis, grades III–IV

Definite AS is diagnosed if the radiological feature is associated with at least one of the clinical criteria

Probable AS is considered if three clinical criteria are present

are thoracic or cervical (Cyriax[7]: p. 366). AS frequently involves extraspinal joints, tendons and ligaments. The disorder may affect all body systems: iritis, pulmonary diseases, chronic prostatitis and cardiovascular diseases are now recognized as possible complications of the disease.[8–12]

Diagnosis of AS is not always easy, particularly in the early stages when only the pelvis is affected. Clinical criteria have been developed during recent decades.[13,14] These criteria are usually not appropriate. Radiologically documented sacroiliitis is obligatory for making a definite diagnosis but it may take years before the radiological abnormalities of the sacroiliac joints can be demonstrated without doubt.[15,16] Recently, magnetic resonance imaging (MRI) has proven its value in the early detection of sacroiliitis, with an estimated sensitivity and specificity of about 90%.[15] Active sacroiliitis on MRI precedes the future appearance of sacroiliitis on radiographs by 8–9 years.[17,18] The clinical criteria, such as decreased chest expansion and symmetrical limitation of spinal movements, also occur relatively late in the course of AS, at a time when the disease should be obvious on other grounds (Box 43.1).

The natural history of the disease in an individual is extremely difficult to define or predict. Some patients have disease limited to the pelvis and the majority have a good outlook for a successful life pattern. Only in a small minority of patients does AS progress to the well-known total ankylosis.[19]

Sacroiliitis

It is well recognized that AS begins at the sacroiliac joints but it is not widely appreciated that the disease may be silent here. Many patients with advanced disease cannot recollect ever having pain in the sacroiliac region or the buttock. Even if sacroiliitis causes symptoms at its onset, these may be taken for an S1 disc lesion. However, routine clinical examination of the spine performed carefully can always detect the lesion.

Symptoms

The patient is usually between 15 and 40 years old and complains of unilateral gluteal pain. Because the sacroiliac joints are largely derived from the first and second sacral segments, the pain commonly radiates to the back of the leg as far as the heel. The localization of the pain is thus the same as in S1 or S2 nerve root compression. However, in sacroiliac arthritis it never spreads to the foot, and paraesthesia is absent. As in discodural problems, coughing (which increases intra-abdominal pressure) may cause pain in the buttock and down the leg. The localization and extent of pain, together with the painful cough, may lead to the assumption that an ordinary discoradicular conflict is present.[20] Some specific characteristics then help to distinguish sacroiliac arthritis from disc diseases. The main feature is that the pain comes and goes in an irregular and unpredictable way. During a flare-up, the pain is constantly present; during remission, the patient can exercise freely without an increase in symptoms. An attack is usually unprovoked: if pain is present it is usually increased by exertion, but if it is absent it cannot be stimulated. This is the reverse of the history in disc lesions, where the pain always follows certain activities and subsides after their avoidance. Another important feature is that sacroiliac pain often alternates from one side to the other, though it is seldom bilateral except when it changes sides.[21]

Signs

Given the similarity with S1 or S2 root compression, the index of suspicion usually remains low and the diagnosis is often missed (Table 43.1).

During the examination in a standing position no suspicion arises. There may be a slight increase in gluteal pain during extension and bending towards the painful side; flexion is limited because of increasing pain in the buttock and thigh; and sometimes a slight deviation towards the painful side can be noted during flexion.[22] Straight leg raising may also cause pain at the end of range.[23]

It is only when the anterior part of the sacroiliac joint is tested (see Ch. 41) that the diagnosis becomes obvious. Unilateral or gluteal or posterior crural pain during the test incriminates the sacroiliac joint. This manœuvre is an extremely sensitive method of deciding whether the sacroiliac joint is affected, and a positive distraction test often precedes radiological evidence of sacroiliac arthritis by years. Although many other tests for the sacroiliac joints have been described, the distraction technique as described earlier is the most significant test of the status of the joint; it applies immediate stress to the anterior part of the joint, without using a lever – distraction forces using the patient's femur as a lever are very non-specific and should therefore not be used as screening tests. Because of the specificity of the sacroiliac distraction test, it is an essential part of the routine clinical examination of the lumbar spine.

If the patient is examined during a flare-up, passive and resisted hip movements can also cause gluteal pain, especially passive external rotation and resisted flexion, abduction and extension.

Although some authors find tenderness over the sacroiliac joint highly indicative of the existence of sacroiliac arthritis,[24] we believe that palpating for tenderness adds no further information and only confuses the examiner. First, the joint, covered as it is by the overhang of the ilium and the sacral extent of

Table 43.1 Differential diagnosis of sacroiliac arthritis and S1–S2 disc lesions

	Arthritis	S1–S2 root compression
History		
Gluteal pain	Spreading in S1–S2 dermatomes	Spreading in S1–S2 dermatomes
Flaring/constant pain	Flares	Constant
Coughing	Painful	Painful
Morning symptoms	Pain on awakening, improved by walking Prolonged morning stiffness	Pain on getting out of bed
Association with activity	Pain irrespective of exertion	Pain follows certain activities
Pain localization	Alternating Not beyond the ankle	Unilateral Often in the foot
Paraesthesia	Never present	In foot or toes
Clinical examination		
Extension and side flexion	Full	Often limited
Flexion	May be slightly limited	Usually grossly limited
Straight leg raising	May be painful at the end	Usually limited
Sacroiliac distraction test	Painful	Painless

Table 43.2 Sacroiliac changes in ankylosing spondylitis[26]

Grade	Changes
0	None
I Suspicious	Patchy periarticular osteoporosis
II Minimal	Loss of definition at the edge of the joints Some sclerosis Minimal erosion
III Definite	Definite sclerosis on both sides Blurring and indistinct margins Loss of joint space
IV Ankylosis	Complete fusion of the joint

the sacrospinalis muscle, remains beyond the direct reach of the palpating finger. Second, the sacroiliac region is a common site for referred tenderness in lumbar discodural conflicts.

Further examination

Radiological evidence of sacroiliitis is accepted as being obligatory for the diagnosis of AS. However, the clinical symptoms may predate the radiological abnormalities by months or even years. In the early stages, when radiological signs are minimal and of questionable significance, it may help to use computed tomography (CT) for demonstrating joint narrowing and fusion.[25]

The changes are classified according to the New York criteria in five grades (grades 0–IV; Table 43.2). Initially, there is patchy periarticular osteoporosis, leading to loss of definition of the subchondral bone plate. The joint thus appears to be widened. Further evolution of the process results in superficial erosion, together with focal sclerosis of subchondral bone. Further proliferative changes result in irregular bridging across the articular cavity. This causes blurring and indistinct margins on both sides of the joint. Finally, the radiograph shows complete osseous fusion.

The best way to detect active sacroiliitis is on MRI. An MRI is considered as 'positive' if the areas of bone marrow oedema (BME) are located at typical sites, i.e. they are periarticular to the sacroiliac joints. When only one BME lesion is visible on an MRI slice, it should be clearly visible on consecutive slices. Enthesitis, capsulitis and synovitis reflect active inflammation as well and are certainly compatible with AS; however, they are not sufficient for a 'positive' MRI if present without concomitant BME.[27]

Association with HLA-B27

The association between the genetic marker HLA-B27 and AS is well known.[28,29] The frequency of HLA-B27 in healthy populations is between 1% (Japanese and African) and 14% (Caucasian), whereas the marker is present in 90% of the AS population.[17] However, the presence of HLA-B27 plays little or no role in diagnosis of the disease: a patient with repeatedly normal radiographs is unlikely to have the disease, regardless of HLA status; in contrast, a B27-negative individual with symptoms suggesting AS has the disease if the radiograph shows the typical changes.[30]

Natural history

The prognosis for an individual is difficult to predict. In some patients the disorder is limited to the pelvis, whereas others

quickly develop spinal and extraskeletal disease. The younger the patient is at the age of onset, the worse the outcome, and men usually fare worse than women.[31] When sacroiliac arthritis appears after the age of 25 years, the disease is likely to follow a mild course: bilateral sacroiliitis continues flaring up and subsiding for some years until bony ankylosis is complete and the pain disappears. If the disease spreads upwards, its spread is very slow and the thoracic spine is only affected when the patient is 40 or 50 years of age. In these patients the cervical spine usually remains unaffected and the hips retain full mobility. In contrast, when sacroiliac arthritis appears before the age of 20 years, or spondylitis has reached the lumbar spine before the age of 25 years, early and severe disablement is very probable; pain and stiffness spread upwards along the spine very quickly and there is also a strong chance of hip involvement within 20 years of onset.[32]

Treatment

It is vital for patients to have some knowledge of the natural history of the disease. They should be told that the concept of inevitable, progressive stiffening of the joints, ending in complete ankylosis and crippling disability, is not correct. The diagnosis of AS is usually not as serious as is generally believed. The patient should be made aware that the majority have a good prognosis for a normal social, family and professional life, and that the disease leads to incapacity in only a few cases.

No specific treatment of a curative nature presently exists. The aim of treatment is therefore preventive and symptomatic: avoidance of pain and deformity.

In order to prevent further deformity, the patient should adopt an appropriate routine. A strict daily routine of positioning and extension exercises is more valuable than physiotherapy. Sleeping on a hard mattress and avoiding lying bent on the side are basic. Lying face downwards on a rigid surface at least once a day for half an hour is also recommended. During the day, extension exercises should be performed as often as possible. Attention should also be paid to posture at work and all opportunities for mobility exploited. Swimming is the best routine sport.

Pain and inflammation are treated by non-steroidal anti-inflammatory drugs (NSAIDs). Indometacin is considered the drug of choice. The patient must be informed that therapy should be continuous and that the purpose of medication is to allow normal activities to be pursued and the daily posture and exercise routine to be carried out. Anti-tumour necrosis factor (TNF) agents are recommended in the case of NSAID failure.[33] Over the past few years, several placebo-controlled and open trials have shown a dramatic response in active AS to TNFα-blocking agents (infliximab and etanercept). In these trials, 50–70% of patients showed an improvement of 50% or more.[34,35]

Psoriasis

The true prevalence of sacroiliitis in psoriasis is unknown. The majority of estimates are in the range of 20–30%.[36,37] The link between the skin condition and the joint disease is unknown. The disease is frequently unilateral or asymmetrical, and can be asymptomatic. The clinical presentation is pain and a positive sacroiliac distraction test – the same as in AS. Treatment of the sacroiliitis is with NSAIDs.

Reiter's syndrome

Reiter's syndrome is the classic triad of arthritis, conjunctivitis and non-bacterial urethritis. The arthritis affects several joints, usually asymmetrically. The cause is unknown but evidence tends to point to an infectious agent. Males are predominantly affected and the onset of the disease is usually between the ages of 20 and 40 years.

Although a high percentage (more than 30%) of patients with the syndrome show severe radiological sacroiliitis,[38] only a small percentage develop clinical evidence of unilateral or bilateral sacroiliac arthritis. Clinical evidence of sacroiliac joint involvement may occur as early as 3 months from the onset of the illness.[39]

Septic arthritis

A pyogenic infection of the sacroiliac joints is rare, although in recent years more reports have been published on this topic.[40–42] The infection reaches the joint by the haematogenous route or by direct extension from a contiguous abscess. Predisposing factors are pregnancy, intravenous drug abuse and immunosuppression.

The initial diagnosis is often overlooked because of its rarity and the poorly localized symptoms and signs. The condition should be considered in cases of acute or subacute onset of pain in the gluteal region, hip or low back, accompanied by fever. An apparent acute abdomen may be present, especially in children.[43,44]

The disease may also present with symptoms and signs of femoral or sciatic nerve root irritation if the distended anterior joint capsule comes into contact with the lumbosacral plexus.

The diagnosis is strongly suspected when the 'sign of the buttock' is found during clinical examination of the back (see p. 637). Roentgenograms are often normal. CT scan and MRI may be useful tools but radionuclide scanning with ^{99m}Tc or ^{67}Ga usually affords early confirmation of the condition.[45–47] Generally, antibiotic treatment leads to complete recovery.

Gout

Gout is usually considered to be a disorder of the peripheral joints. However, since 1965, it has been recognized that the sacroiliac joint is also radiologically affected at a late stage in a significant percentage (7–17%) of patients with tophaceous gout.[48,49] The sacroiliitis usually remains clinically silent, acute attacks being rare.[50]

Osteoarthrosis

The incidence of degenerative arthritis in the sacroiliac joints increases with age.[51] It is not considered to be a cause of symptoms. Osteoarthrosis of the sacroiliac joints is a radiological finding only and has no clinical significance (Cyriax[7]: p. 372).

Sacroiliac joint syndrome

Introduction

The ability of mechanical lesions of the sacroiliac joint to cause backache and referred pain to the buttock and posterior leg was first recognized by Goldthwait and Osgood in 1905.[52]

Some schools of thought have put great emphasis on the joint and consider the sacroiliac joint syndrome as a common source of low back and pelvic pain.[53] Although it is generally accepted that most pain in the sacroiliac region is of dural origin and has nothing to do with an actual lesion of the joint,[7,54] it is logical to accept the sacroiliac joint as a prime cause of pain because it is a synovial joint and thus subject to the same dysfunctional conditions that affect other synovial joints. It is not difficult to accept ligamentous sprain and overuse phenomena in and around the sacroiliac joint as possible sources of 'dysfunctional' pain. It is, however, more difficult to identify a 'blocked sacroiliac subluxation' as the main cause of the dysfunction.

Numerous tests to detect dysfunction of the sacroiliac joint have been described in the chiropractic and manual medicine literature. Although commonly used, many of these tests are difficult to perform or to interpret, and consequently their intertester reliability is low (see p. 599). In particular, those tests that assess motion (or the lack of it) in a sacroiliac joint are not reliable. Pain provocation tests – stressing the structures in an attempt to reproduce the patient's symptoms – have a much better intertester reliability and can be used to detect sacroiliac joint syndrome.

We therefore do not discuss blocked sacroiliac joints as a possible source of sacroiliac dysfunction. The issue is too controversial and today there exists no clear evidence for the disorder. Sacroiliac strain, by contrast, is a well-delineated entity with typical signs and symptoms. Recent studies demonstrated temporary pain relief after local blocks of the sacroiliac joint, thus confirming the sacroiliac joint as a real source of low back pain.[55–57] However, it is worth repeating that the diagnosis requires typical physical findings and that tenderness over the sacral sulcus and the posterior sacroiliac joint line are not sufficient in themselves to make the diagnosis.

History

Sacroiliac joint syndrome or strain usually occurs in women between the ages of 15 and 35 years. The ligaments may have been strained by a fall on the buttocks or a motor vehicle accident. Other more trivial mechanisms of injury may also be linked to the development of sacroiliac joint syndrome, such as stepping into an unexpected hole or miscalculating a height.[58] Alternatively, the strain can be associated with pregnancy. Hormonal changes can cause relaxation of the sacroiliac ligaments; during pregnancy and parturition, the joint will therefore be more susceptible to strain.

Pain is usually unilateral (although bilateral pain may occur), is never alternating, and is localized in the sacroiliac region with reference to the buttock and the posterolateral aspect of the thigh and calf.[59]

The pain has typical postural characteristics in that it appears only after prolonged or increased loading of the ligaments. It is therefore typical that pain is brought on by the maintenance of prolonged postures.[60] As a rule, it is increased by excessive standing or by strolling, and is abolished by correction of posture or by movement. Resting will also decrease the symptoms, although lying down – for example, in a fixed position – may cause pain. Pain is often aggravated by bending and by climbing stairs. Dural signs, such as pain during coughing and sneezing, are absent. Neurological symptoms – paraesthesia and weakness – do not occur.

The condition is persistent, though rare cases of spontaneous recovery do occur.

Clinical examination

As pain at the sacroiliac area is usually of dural origin, the diagnosis of sacroiliac strain (summarized in Box 43.2) must always be made sparingly. Examination of the lumbar spine shows a full range of movement, sometimes with pain at the end of range of flexion or extension. There may be pain if weight is borne on the ipsilateral extremity. Straight leg raising is full-range, although pain at the end of range may be encountered.[61] In severe cases, some hip movements (flexion, medial rotation and extension) may be painful at the end of range. Resisted abduction and resisted extension of the leg may also provoke pain.

It is possible for the sacroiliac distraction test (included in the basic lumbar clinical examination) to remain negative. As this test pulls on the anterior ligaments only, the stress is not always adequate to provoke pain in the posterior ligaments. More vigorous movements, performed with leverage of the hip, will then be required to stress the posterior ligaments (see pp. 949–952). It is worth emphasizing again that most of these tests are non-specific in that they assume that the hip is completely normal. A positive test is therefore significant only when the clinical examination has ruled out lumbar and hip disorders.[62]

Box 43.2

Summary of the diagnosis of sacroiliac strain

- Postural pain in the sacroiliac region after:
  - Pregnancy
  - A fall or road traffic accident
- Negative lumbar examination
- Negative examination of the hip
- Positive sacroiliac tests

Treatment

Training programme

Vleeming *et al* consider an inadequate 'force closure' of the sacroiliac joints as an important cause of sacroiliac strain. Force closure is defined as the compressive stabilizing forces exerted by ligaments and coupled bilateral gluteal and back muscles. The authors hypothesize that ligaments alone are not capable of transferring lumbosacral load effectively from the spine to the iliac bones. This is particularly the case in heavy load situations and conditions of sustained load, such as sitting and standing for a long time in a relatively counternutated position of the sacrum.[63–66] Muscle weakness and inadequate coordination between muscles diminish force closure, which consequently increases the load on the pelvic ligaments. The ligaments become strained, leading to pain and laxity.

The authors advise a specific training programme as one of the treatment measures to compensate for the lack of force closure. Strength and coordination of the gluteus medius and contralateral latissimus dorsi should be trained. The erector spinae, the multifidus muscle fascicles and the oblique and transverse abdominals should also be part of the active stabilizing training programme because of their direct or indirect attachments with the sacroiliac ligaments.

Belt

The symptoms may be abolished permanently if the joint and the ligaments are protected for a month or so by the wearing of an appropriate belt. The most suitable type is a very tight, non-elastic 6 cm wide belt, placed around the pelvis between the iliac crest and the greater trochanter (Fig. 43.1).

The biomechanical effects of such a belt in human pelvis–spine preparations were studied by Vleeming *et al*,[65] who found that it led to a significant decrease in rotation at the sacroiliac joints. Both the location of the belt and the degree of loading were crucial. An optimal decrease in movement was reached with a belt worn at the level just cranial to the trochanter.

The active straight leg raising test, described by Mens *et al*,[67] seems to have some prognostic value. Lying supine, the patient is asked to lift the leg about 5 cm off the couch. In a serious pelvic dysfunction the patient is unable to do so or the strength on one side is considerably less. The test is repeated after stabilizing the pelvis with a belt or by manual pressure on the iliac spines from the lateral side. If this lateral pressure converts a painful active straight leg raising test into a painless one, wearing a belt will lead to a good result.

Sclerosing injections

If wearing a pelvic belt fails to improve the patient's condition, sclerosing injections into the posterior sacroiliac ligaments are indicated. We use Ongley's solution (2% phenol, 25% dextrose, 15% glycerol). This mixture has a good safety record and, apart from considerable pain for up to 2 days after the injection, it causes no side effects. It induces an inflammatory response, which leads to fibroblast proliferation and new collagen production (see pp. 112–114). Because of pain, the solution must be mixed with 2% lidocaine, in a proportion of 80% sclerosant and 20% lidocaine.

Fig 43.1 • A belt for sacroiliac strain.

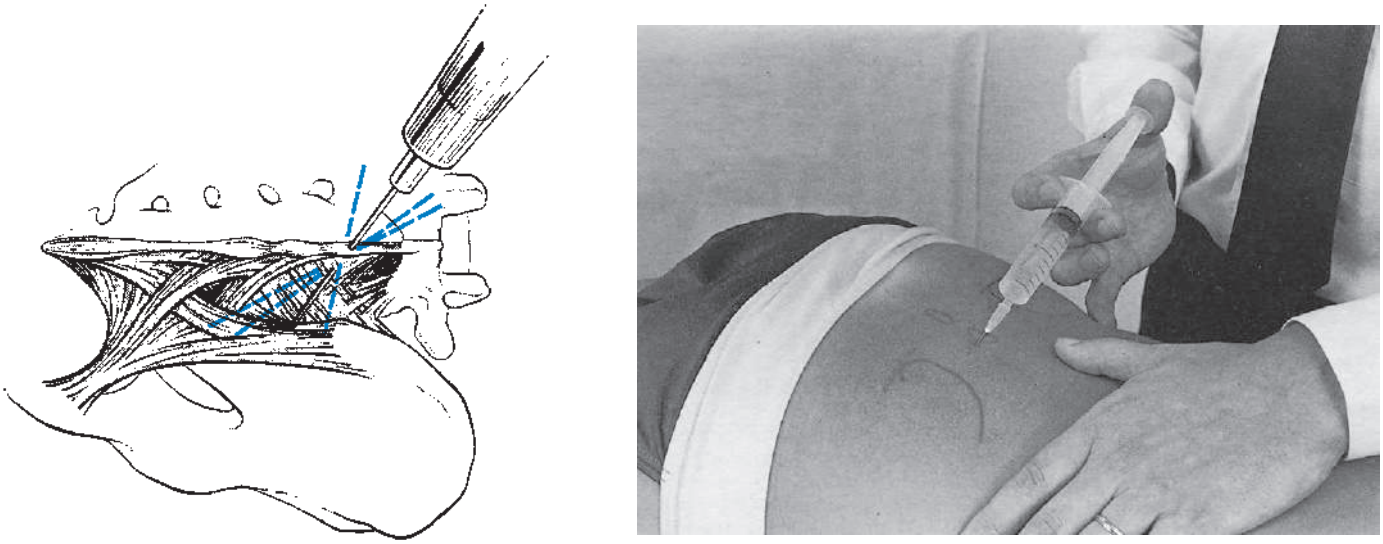
Usually all the sacroiliac ligaments are treated at their ligamentoperiosteal junction. Although it is possible to be very selective and to infiltrate only small groups of ligaments, better results will be achieved if all the ligaments on both sides are infiltrated.[68]

Technique

A 10 mL syringe, filled with 8 mL sclerosant and 2 mL lidocaine, is fitted to a 7 cm long needle.

The skin is punctured at the level of the tip of the first sacral spine. From here, the following ligaments on both sides can be infiltrated: the posterior sacroiliac, the interosseous sacroiliac, the sacrotuberous and the sacrospinous (the latter two form the sacral attachments). To reach the *posterior sacroiliac ligament*, the tip of the needle is directed at an angle of 30° to the skin and thrust laterally until it touches bone. Four to five small injections are made along the posterior aspect of the posterior superior spine. It should be stressed that no fluid is introduced unless the tip of the needle is felt to impinge on bone (Fig. 43.2).

The needle is then partly withdrawn and reinserted at an angle of about 45° to the horizontal to reach the iliac attachments of the *interosseous sacroiliac ligaments*. Bone is reached at a depth of 5–7 cm, where small infiltrations are made.



(a) (b)

Fig 43.2 • Sclerosant infiltration of the posterior sacroiliac ligament.

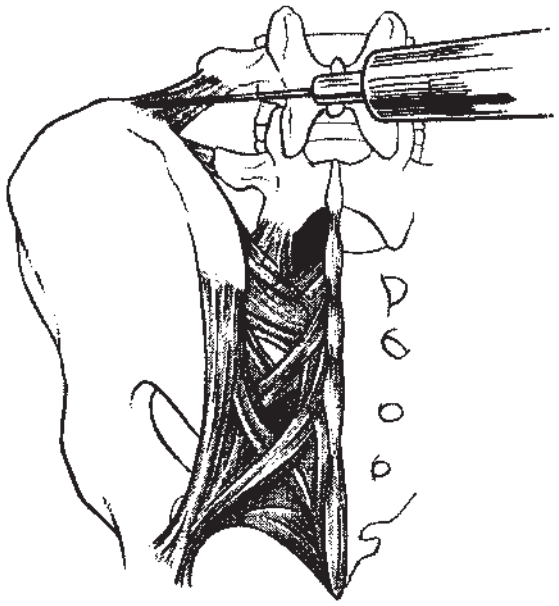


Fig 43.3 • Sclerosant infiltration of the iliac insertion of the iliolumbar ligament.

To reach the sacral attachments of the *sacrotuberous* and *sacrospinous ligaments*, the needle should be almost completely withdrawn and, together with the skin and subcutaneous tissue, moved down towards the coccyx as far as possible. The free thumb palpates the lateral side of the sacrum at the lower three levels. The needle is then pushed under the palpating thumb and small infiltrations (with bony contact) are made.

The *iliac insertion of the iliolumbar ligament* is infiltrated via a separate skin puncture. The needle is inserted about 3 cm lateral to the fifth supraspinous process. The palpating thumb is placed at the medial edge of the iliac crest. The tip of the needle is thrust in very obliquely in the direction of the thumb, until it is felt to traverse a resistant ligament before touching bone (Fig. 43.3). An infiltration of 1 mL is performed along the border, deeply and superficially, by multiple withdrawals and reinsertions. Care is taken to inject only when the needle touches bone.

Follow-up

There is considerable pain at the time of injection but the anaesthetic soon takes effect. After an hour and subsequently for up to 2 days, the back is painful, sometimes to such an extent that the patient is forced to rest in bed. This unpleasant reaction usually lasts no longer than 2 days.

The infiltration is repeated twice, at weekly intervals. For 6 weeks after the last infiltration, the patient should avoid all movements and postures that strain the sacroiliac joints, such as standing, bending and climbing stairs. The result should be judged after 6 weeks. Insufficient relief indicates the need for another infiltration.

Bony disorders of the pelvis

Tumours

Sacral tumours, both primary and secondary, are rare lesions. They often escape early diagnosis.

Most patients with sacral tumours have a non-specific complaint of low back pain. However, the history will reveal some unusual features typical of non-mechanical lesions in the lumbar spine, currently referred to as 'warning symptoms' (see Ch. 39):

- Continuous pain, not altered by changing positions or activities
- Increasing pain, slowly getting worse
- Expanding pain
- Bilateral sciatica.

Late in the course of a serious sacral lesion, disturbance of urinary and/or bowel control may occur.

Clinical examination may reveal local tenderness and swelling. Both lumbar examination and sacroiliac pain provocation tests may be positive. The most striking clinical finding is usually the appearance of a 'sign of the buttock' (see p. 637), which draws immediate attention to a serious pelvic lesion.

Apart from metastases, a sacrococcygeal chordoma is the most common type of malignant sacral tumour. The neoplasm is believed to take its origin from remnants of the notochord; it grows slowly but is locally infiltrative and destructive. Symptoms may initially be mild and may present months or years before the diagnosis is made. As the disease progresses, pain may become intractable. Death usually results from complications or extensive local tumour growth. Diagnosis can be made via a careful rectal examination, which almost always reveals the firm, presacral tumour mass, which is extrarectal and fixed to the sacrum.

Radical resection is the treatment of choice for sacral chordomas. Addition of radiation after subtotal resection improves the disease-free interval, although radiation therapy can generally be used only once.[69,70]

Fractures of the sacrum

The increased incidence of motor vehicle and industrial trauma during recent decades has led to an increase in fractures of the sacrum.

The diagnosis and treatment of these lesions are beyond the scope of this book. However, insufficiency fractures of the sacrum usually develop in the absence of obvious trauma and must therefore be included in the differential diagnosis of sacroiliac lesions. Insufficiency fractures of the sacrum usually occur in elderly women with postmenopausal osteoporosis. They are often confused with disc lesions, spinal stenosis and cauda equina syndrome. Sacroiliac tests are very painful and there is a 'sign of the buttock' (see p. 637).

A CT scan is often necessary to demonstrate the fracture line. Treatment consists of rest.[71,72]

Access the complete reference list online at
www.orthopaedicmedicineonline.com

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44

Anatomy and disorders of the coccyx

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Anatomy of the coccyx

The coccyx is formed by fusion of four rudimentary vertebrae. The first coccygeal vertebra has a base for articulation with the apex of the sacrum and two cornua (cornua coccygea) that are usually large enough to articulate with the cornua sacralia. The sacrococcygeal joint is a true joint with a joint capsule and ligaments. Other ligaments covering the posterior aspect of the coccyx are the posterior intercoccygeal ligaments. The gluteus maximus partly inserts at the dorsolateral aspect of the coccyx via its coccygeal fibres (Fig. 44.1).

The coccyx and overlying skin are innervated via the dorsal rami of S4 and S5.

Disorders of the coccyx

Usually, coccygeal pain results from a local lesion, but referred coccygodynia is possible and psychogenic pain also occurs.

Referred coccygodynia

It is well known that coccygodynia can arise from a lumbar disc lesion, usually as the result of extrasegmental reference.[1] Coccygodynia may also arise from irritation of lower pelvic structures (neoplasms of rectum or prostate).[2] The history and clinical examination can easily distinguish referred pain from a local disorder. In referred coccygodynia from a disc lesion, pain arises not only during sitting but also during lumbar movements. Coughing is also painful. Physical examination additionally shows pain during lumbar movements, and straight leg raising often increases the discomfort. However, palpation does not assist in diagnosis because local tenderness is to be expected, whichever variety of coccygodynia is present. Tumours or other causes of inflammation affecting the sacrum should be suspected if the pain is not relieved by lying down or if there is nocturnal discomfort. In case of doubt, epidural local anaesthesia can be useful.

Local coccygodynia

True coccygodynia nearly always affects women and the most common cause is traumatic. Usually, a direct contusion during a fall in the half-sitting position is responsible. Because the coccyx usually lies 2 cm above the two ischial tuberosities it can only be contused when the body is tilted backwards during a fall on the buttocks. Alternatively, in a fall onto a narrow object, the force may strike the coccyx rather than the ischial tuberosities.

In 'idiopathic' coccygodynia, no particular injury is noted. It has been suggested that certain anatomical variations of the coccyx predispose to repeated microtraumas, which then cause chronic irritation. A coccyx with a sharp forward angle seems to be more prone to painful stretching.[3]

Childbirth also causes injury sometimes and postpartum coccygodynia is a well-recognized entity.

Coccygodynia is related to sitting. Pain is felt at the coccyx only and does not spread in any direction. Sitting on a hard surface or with the buttocks over the border of the chair relieves the pain. Lumbar movements, standing and lying down do not provoke the pain. Coughing and sneezing is painless but defecation sometimes hurts. As a rule, walking is painless except when the coccygeal fibres of the gluteus maximus muscle are involved.

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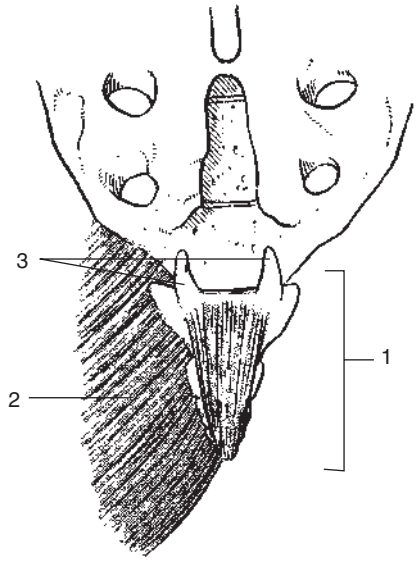

Fig 44.1 • The coccyx (1) and its articulation with the sacrum; the insertion of the gluteus maximus (2); the two cornua (3) and the intercoccygeal ligaments.

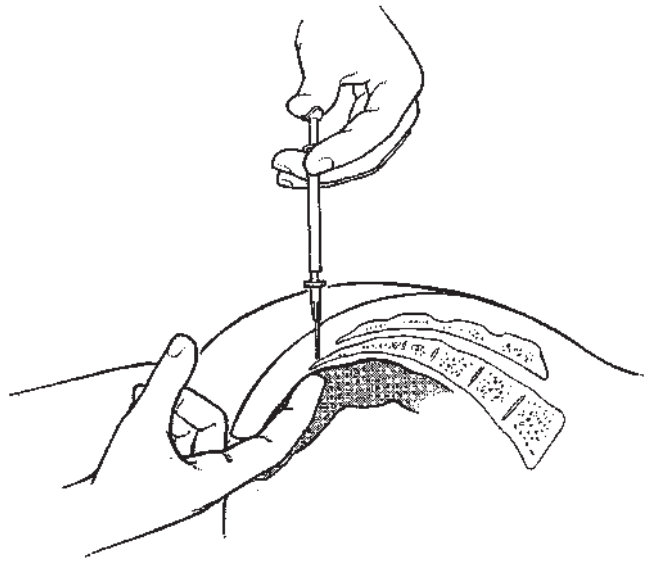

Fig 44.2 • Steroid infiltration of the coccyx.

The routine clinical examination of the lumbar spine, sacroiliac joints and hips will be found to be completely normal. Palpation will reveal a localized tender area. As already mentioned, tenderness is also present in referred and psychogenic coccygodynia, so the diagnosis should be made before palpation starts.

Palpation starts at mid-sacrum and four types of coccygodynia may be found:[1]

- Contusion of the tip of the coccyx and the immediate surrounding tissue. This is the most common type.
- Sprain of the posterior intercoccygeal ligaments.
- Sprain of the sacrococcygeal joint.
- Irritation of the coccygeal fibres of the gluteus maximus muscle. In this condition the pain is unilateral and may spread slightly to one buttock. Occasionally the patient complains that walking is uncomfortable.

Treatment

Apart from seating modifications that transfer the weight to the ischia, treatment may consist of deep transverse friction, steroid infiltrations or surgery.

Deep transverse friction

When applied to the affected ligaments, this is very often quickly effective. Six to eight sessions two or three times a week usually suffice, except in contusion of the tip of the coccyx, where infiltration of a local steroid usually affords better and quicker results.

Technique

The patient lies prone with a pillow under the pelvis. The legs are slightly abducted and internally rotated. The physiotherapist sits next to the patient and places one thumb on the affected spot. If the lesion is at the sacrococcygeal or dorsal intercoccygeal ligaments, friction is given using alternating, up-and-down movements of the thumb. If the lesion lies at the insertion of the gluteal fibres, the thumb should be pressed deeply between the muscle and the lateral border of the coccyx. Friction is then given by drawing the thumb up and down along the edge of the bone.

Steroid infiltration

This can be done if the lesion lies at the tip of the coccyx or when deep friction does not succeed. The injection is usually very effective, but if the patient does not take preventive measures in the form of seating modifications, then relapses may be encountered.[4]

Technique

The patient lies prone on the couch, the pelvis slightly tilted and the legs internally rotated. The tenderness is carefully sought. Sometimes it is necessary to perform rectal palpation (Fig. 44.2). The coccyx is then pressed between the index finger and the thumb. In this position the injection can be given precisely and without fear of penetrating the rectum. This precaution is particularly appropriate if the sacrococcygeal joint or the apex of the coccyx is infiltrated.

A 1 mL tuberculin syringe, filled with 10 mg of triamcinolone, is fitted to a thin 2 cm needle. After careful preparation of the skin, the needle is introduced at the localized spot until it hits bone. By half-withdrawing the needle and reinserting it at a slightly different angle, the entire lesion is infiltrated with a series of tiny neighbouring punctures. The palpating thumb and index finger feel exactly where each droplet is deposited.

After injection, the coccyx remains sore for a few days before the symptoms abate. The patient is re-examined after 2 weeks. A second infiltration is made if some residual tenderness remains.

If the condition recurs frequently, the steroid solution should be replaced by two infiltrations of sclerosant solution (1.5 mL of sclerosant and 0.5 mL of lidocaine 2%). Although

very painful during the days following the injections, it usually affords permanent results.

Coccygectomy

This is only considered in intractable incapacitating pain persisting in spite of adequate conservative treatment. However, the operation is very rarely indicated and the results are far from good.[5,6] Furthermore a high incidence of Gram-negative infection following coccygectomy has been reported.[7]

Psychogenic coccygodynia

Coccygeal pain may be of psychogenic origin. Because the diagnosis is usually made by eliciting tenderness, the elimination of a psychogenic case is extremely important. Usually the history helps: genuine local coccygodynia does not spread and psychogenic pain is usually vague and radiates in various (impossible) directions. In local coccygodynia, lumbar or hip movements do not elicit pain, whereas they all hurt in a psychogenic case. If suspicion of psychogenic coccygodynia arises, the patient must be given enough freedom during the history and functional examination for contradictions to emerge (see online chapter *Psychogenic pain*).

Access the complete reference list online at **www.orthopaedicmedicineonline.com**

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Clinical examination of the hip and buttock

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Introduction

Pain felt in the hip and buttock does not necessarily originate from a lesion in these areas. Cyriax stated that most pain in the buttock derived from the lumbar spine (reference from L1 and S2), whereas pain in the thigh is referred from the lumbar spine and hip region as often as it has a local origin. When dealing with pain in this area, it is often not easy at first to determine if there is a problem in the lower back, the sacroiliac joint or the hip. Therefore, a detailed and chronologically ordered history is first taken, as described for the lumbar spine (see Ch. 36). Past and present symptoms are noted and the examiner is informed about the exact site and nature of the pain. Next, a preliminary examination must be performed that includes the whole lower quadrant: from the lumbar spine, over the sacroiliac and hip joints to the upper leg. Once it is clear that the symptoms do not arise from the lumbar spine or sacroiliac joint, the structures of the hip are examined more intensively.

If the diagnosis is still obscure after taking the history and carrying out a physical examination, the focus should turn to disorders outside orthopaedics that could be responsible for the symptoms – usually intra-abdominal lesions. In such conditions the pain is usually unrelated to movements that have been undertaken during the examination. Another cause of pain in the buttock of non-orthopaedic origin is occlusion of the common iliac artery with intermittent claudication. Finally, it must be remembered that hip complaints may be claimed but can have a non-physical basis in the psyche.

Referred pain

Pain referred to the buttock and hip region

Most pain in the buttock and hip results from a lumbar lesion with a segmental (L1–S2) or multisegmental (dural) reference of pain. Knowledge of the dermatomes that meet in the buttock and hip is therefore essential.

The first lumbar dermatome is represented by an area of skin at the outer and upper buttock which is partly overlapped by the second and third lumbar dermatomes (Fig. 45.1).

The skin of the lower part of the buttock is derived from the first and second sacral segments. The fourth and fifth lumbar segments are not present in the buttock. In spite of

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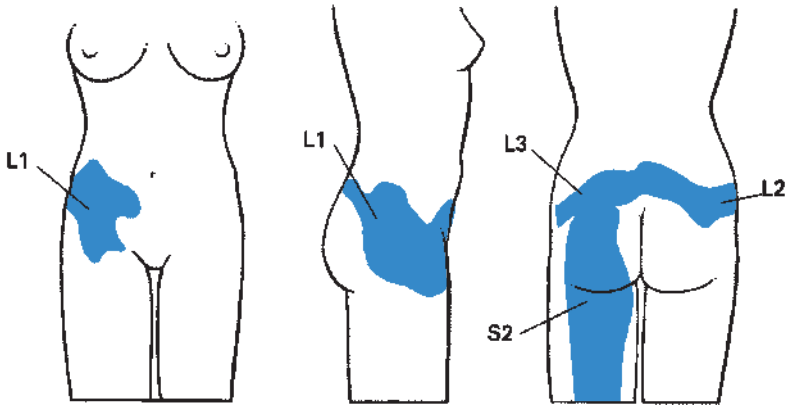


Fig 45.1 • Dermatomes at the hip and buttock.

this, fourth and fifth lumbar disc protrusions are the commonest cause of pain in the buttock,[1] and are an expression of dural pain.

It is worth remembering that the first lumbar dermatome also covers the lower abdomen and the groin. The second lumbar dermatome is from the front of the thigh to the patella. The third lumbar dermatome is positioned over the inner aspect and the front of the thigh, then down the leg to just above the ankle. The first and second sacral dermatomes cover the gluteal area, the back of the thigh, the posterior aspect of the leg and the sole.

Multisegmental dural pain in the buttock is broad and may spread diffusely to both legs, excluding the feet.

Pain in the groin may also result from intra-abdominal pathological conditions: appendicitis, gynaecological disorders or inguinal or femoral hernia.

Box 45.1

Summary of referred pain

Pain referred to the buttock and hip region

Segmental	Groin	L1–L2
	Thigh	L2–L3
	Buttock	S2
Multisegmental from the lumbar dura		

Pain referred from the buttock and hip region

Hip joint		L3
Sacroiliac joint		S1–S2 (L3–L4?)
Gluteal bursitis		L4–L5
Psoas bursitis		L2–L3
Muscles	Iliopsoas and adductor longus	L2–L3
	Rectus femoris	L3
	Quadratus femoris	L5–S1
	Hamstrings	S1–S2

Pain referred from the buttock and hip region

Muscles

The muscles of the buttock are derived from the fourth and fifth lumbar and first sacral myotomes. Lesions are a rarity and so is referred pain from these muscles. The segmental distribution of the most frequently injured muscles is:

- Iliopsoas – L2 and L3
- Adductor longus – L2 and L3
- Rectus femoris – L3
- Quadratus femoris – L5 and S1
- Semimembranosus/semitendinosus – S1 and S2
- Biceps femoris – S1 and S2.

Hip joint

The hip joint is formed largely from the third lumbar segment. Therefore pain referred from the joint may be felt in the upper and inner part of the buttock, and the inner aspect and front of the thigh and leg as far as the medial malleolus. Rarely, the joint has developed mainly from the fourth lumbar segment. In such cases, pain spreads along the outer side of the mid-thigh and leg, and may reach the big toe. Because referred pain does not always occupy the whole dermatome, it is possible for a hip lesion to refer pain to the knee only, perhaps also spreading along the front of the tibia.[1,2] But pain felt only at the upper inner quadrant of the buttock strongly points to the low back or the sacroiliac joint.

Sacroiliac joint

The sacroiliac joint normally gives rise to pain along the back of the thigh and calf. In rare instances, pain is felt in the groin as well (see p. 598).

Bursae

The bursae may also give rise to referred pain in the same way as other soft tissues. In gluteal bursitis, pain is felt at the lateral or posterior trochanteric area and may be referred to the outer thigh, i.e. a lesion in a tissue derived from the fourth or fifth lumbar segments. The psoas bursa is developed from the second and third lumbar segments. Pain is felt in the groin and may spread to the front of the thigh.

Referred pain in the buttock and hip is summarized in Box 45.1.

Box 45.2

Summary of symptoms

- Onset: congenital/acquired (traumatic/non-traumatic)
- Evolution
- Current symptoms
  - Site of pain
  - Influence of posture, movement
  - Nocturnal pain
  - Twinges
  - Coughing and pain
  - Instability
  - Functional disablement
- Symptoms in other parts of the body

History

History taking is largely the same as in lumbar spine disorders (see Ch. 36) because it is not always clear from the onset if the patient has a lumbar, sacroiliac or hip problem. However, once it has become more or less apparent that the complaints are the outcome of a hip lesion, some particular questions should be asked.

After the usual questions on the patient's age, sex, occupation and hobbies, the examiner tries to find out what the actual problem is: pain, functional disability or instability? The problem should then be worked out systematically via a chronological approach: when and how did the problem start, what was its evolution and what are the current symptoms (Box 45.2)?

Onset

- *When did it start?* Is it an acute, subacute or chronic problem?
- *What brought it on or how did it start?* Was there an injury or did the pain appear without obvious reason?

If there was an injury:

- *How did it happen?* In what position was the body and what forces were applied to the hip?
- *What were the immediate symptoms?* Where was the pain? Was there any swelling? Was there any functional disablement?

If there was no injury:

- Did the symptoms come on suddenly or gradually?

Evolution

In long-standing cases or in traumatic conditions it is important to have an idea of the evolution of the complaints in terms of:

- *Intensity:* Did it worsen, improve or remain constant, or did it fluctuate with complete or no complete relief between attacks?
- *Localization?* Did the pain expand or move? Moving pain is characteristic of a loose body.
- *Treatment?* What was the effect of previous treatments?

Current symptoms

- *What is the problem now?* The examiner makes further enquiries about pain, pins and needles, instability or functional disability.
- *Where do you feel the pain (which dermatome)?* As exact a description as possible must be obtained.
- *Do you have pain at rest or during the night?* Nocturnal pain indicates a high degree of inflammation and may point to a serious disorder such as arthritis, haemarthrosis, tumour, metastasis or fracture. However, in an ordinary gluteal bursitis, lying on the affected side at night is also often painful.
- *What brings the pain on?* Sitting, standing up, walking and running, climbing stairs, sitting or lying? If the pain starts after walking a certain distance, ask if it disappears after standing still for a while and reappears after walking the same distance: this suggests claudication in the buttock.
- *Does a particular movement provoke the pain?*
- *Does the pain appear at the beginning, during or after some sort of exertion?*
- *Do you have twinges, and when?* This symptom is defined as a sudden, sharp and unexpected pain and is clearly indicative of momentary subluxation of a loose body. On walking, a severe twinge is felt shooting down the front of the thigh and the leg gives way at this point.
- *Is any movement accompanied by a click?* Clicking may be indicative of loose bodies or acetabular labrum tears.[3,4]
- *Does coughing hurt?* This dural sign is highly suggestive of a lumbar intervertebral disc lesion but is also found in sacroiliac arthritis.
- *Do you have a feeling of instability?* Any disorder altering the anatomical relations in the hip region – for example, congenital dislocation, coxa vara or epiphysiolysis – may lead to instability. Painful conditions at the hip and neurological disorders, such as paresis of the fifth lumbar root involving the gluteus medius, or of the third lumbar root involving the quadriceps, are other possibilities.
- *Do you have any functional disability?* This may be stiffness on standing up or starting to walk, or inability to put on shoes. All these direct attention to an arthrotic joint.
- *Do you have symptoms in other parts of the body?* The possibility of systemic disease arises: for example, rheumatoid arthritis or ankylosing spondylitis.

Inspection

Gait

When the patient enters the room, observe the gait. Disorders that affect the hip joint or any other joint of the lower extremity become especially perceptible during walking. The patient's

gait can be restricted so as to avoid a painful component. Excessive movement of the pelvis may compensate for a stiff hip joint. In gross weakness of the gluteus medius or gluteus maximus, an abduction or extension lurch respectively is present.

The patient's face should be in harmony with the reported symptoms: for example, haggard due to sleepless nights. While the patient undresses, note should be taken of any manœuvre that seems painful or awkward.

Posture

Next, posture is assessed, especially in relation to the lower back, pelvis and lower extremities. Details of observing the patient's posture are dealt with in the chapter on the lumbar spine (see p. 498).

Hip joint position

The position of the hip joint can be informative about a pathological condition. In acute arthritis and gross osteoarthrosis, the hip joint is often in flexion, which is compensated for by an anterior tilt of the pelvis together with increased lordosis of the lumbar spine. The femur is also slightly abducted and laterally rotated. This in turn influences the position of the knee and foot, which are also rotated. It is important to remember, however, that excessive external rotation of the leg, with 'toeing out', also occurs in external femoral neck retroversion or a slipped upper femoral epiphysis and in pelvic torsion. Posterior rotation of the innominate bone may also be responsible for slight external rotation of the leg. In contrast, 'toeing in' may be the result of extreme femoral neck anteversion.[5]

In third lumbar root pain, patients may also adopt a flexed position to relax the nerve root.

The combination of excessive internal rotation together with adduction is typical of a non-organic cause.

Muscle contours

The contours of the glutei, hamstrings and quadriceps muscles are observed. Unilateral or bilateral muscular atrophy is noted.

Skin folds

Lastly, the skin folds, i.e. gluteal folds and groin, are assessed. These are normally situated symmetrically. Asymmetrical folds may be the result of underlying anatomical alterations, such as muscular atrophy, pelvic obliquity, leg-length discrepancy or congenital dislocation of the hip joint.

Box 45.3 summarizes the aspect of inspection.

Functional examination

Preliminary examination

Before starting the clinical examination of the buttock and hip, a preliminary examination of the lumbar spine and sacroiliac joint (summarized in Box 45.4) should be performed to confirm the absence of a lesion in these regions (see Ch. 36).

Box 45.3

Summary of inspection

- Observation of:
  - Gait
  - Face
  - Unusual movements
- Posture/position of the hip
- Contours of muscles
- Skin folds

Table 45.1 Fifteen functional tests

Supine	Prone lying
Passive tests	
Flexion	Extension
Medial rotation	Bilateral medial rotation
Lateral rotation	
Adduction	
Abduction	
Resisted tests	
Flexion	Bilateral lateral rotation
Extension	Bilateral medial rotation
Adduction	Flexion knee
Abduction	Extension knee

Basic functional examination

Routine clinical examination consists of 15 functional tests (Table 45.1). If signs warrant or the history is indicative, complementary tests can be performed.

Supine

Passive movements

The range, painfulness and end-feel of passive flexion, lateral and medial rotation, adduction and abduction are noted, carefully comparing both sides.

Passive flexion

The anterior thigh is moved upwards until it touches the abdomen (Fig. 45.2). The average range of movement is 140°, with a soft end-feel caused by tissue approximation. It is important to remember that the last 30° of this apparent hip movement is carried out by the pelvis, which flexes at the lumbar joints. This backward tilt of the pelvis also moves the other thigh towards extension, and when there is a restriction

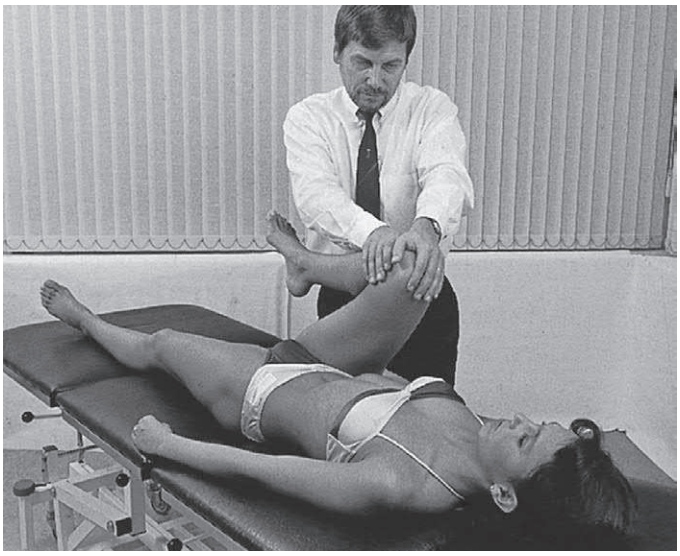
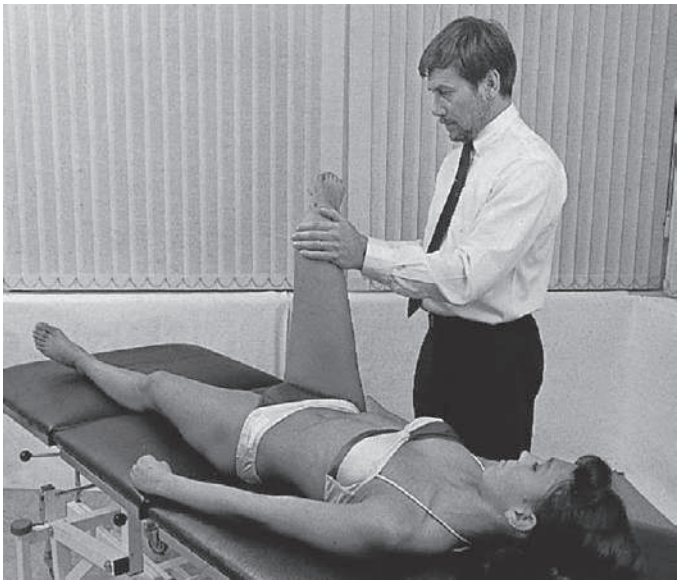


Fig 45.2 • Passive flexion.

(a)



(b)

Fig 45.3 • Passive rotation: (a) lateral; (b) medial.

of extension in the contralateral hip joint, the thigh will move upward (Thomas's sign of flexion contracture of the hip).[6]

Passive rotations

The hip and knee are bent to 90° in order to examine rotation movements (Fig. 45.3). The contralateral hand is used to stabilize the femur at the knee, while the other hand, placed at the distal end of the lower leg, performs medial and lateral rotation movements. At the end of range, a capsular elastic end-feel should be found. The average range of passive lateral rotation is 60°, and that of medial rotation is 45°. In advanced arthrosis or arthritis, the end-feel is hard. However, a soft end-feel replaces a hard one when swift erosion of the femoral head occurs in arthrosis.

In gross arthritis or arthrosis, there is 90° limitation of flexion and no range of medial rotation, although lateral rotation is full. In very early arthrosis or arthritis, medial rotation is the first movement to become measurably restricted, soon followed by slight limitation of flexion. In arthrosis or arthritis, medial rotation is the most painful passive movement. In bursitis or an impacted loose body in a joint that is not yet arthrotic, the end-feel is soft. Usually, in these last two disorders, lateral rotation hurts and is the only clinical finding.

Passive adduction

This is tested after raising the other leg, so as to get it out of the way (Fig. 45.4a). The range of movement is 30° on average. In a normal joint, the end-feel is elastic, caused by stretching of the capsule and muscles that lie on the outer side of the hip. When movement is painful at the outer side of the hip, a lesion of the iliotibial tract[1] should be considered. If some resisted movements are also painful, gluteal bursitis is probably the cause.

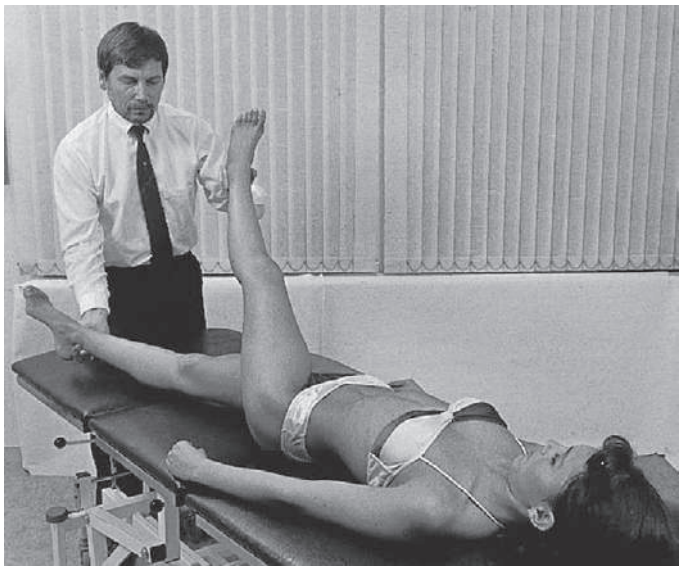
Passive abduction

The ipsilateral hand is placed at the medial and dorsal side of the thigh, as far distal as possible. The knee is put into 90° flexion, which eliminates the influence on the movement of the biarticular part of the adductors, i.e. the semitendinosus, semimembranosus and gracilis. The other hand stabilizes the pelvis (Fig. 45.4b).

On full passive abduction, the structures on the medial side of the thigh are stretched, while on the outer side the bursae may become compressed between the greater trochanter and the ilium. In arthritis and arthrosis, movement is restricted because of muscle spasm and bone-to-bone contact, respectively.

Resisted movements

Four resisted movements are then tested: resisted flexion, extension, adduction and abduction. Because muscle lesions in the buttock are very rare, pain on resisted abduction or extension usually results from compression of a nearby tender bursa. Muscle sprains in the thigh do occur and are mainly found in young adults as the result of sports injury.



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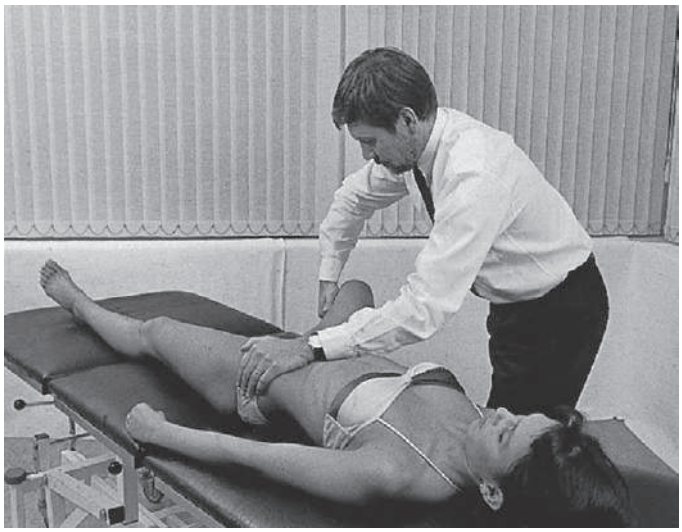
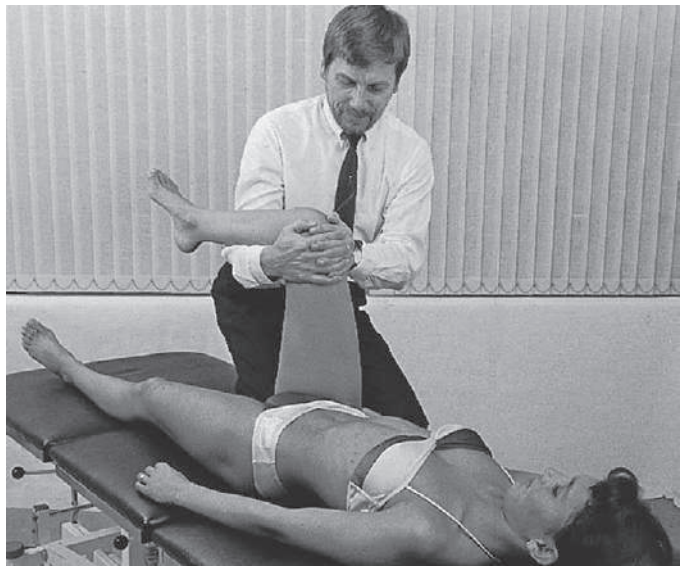


(b)

Fig 45.4 • Passive adduction (a) and abduction (b).



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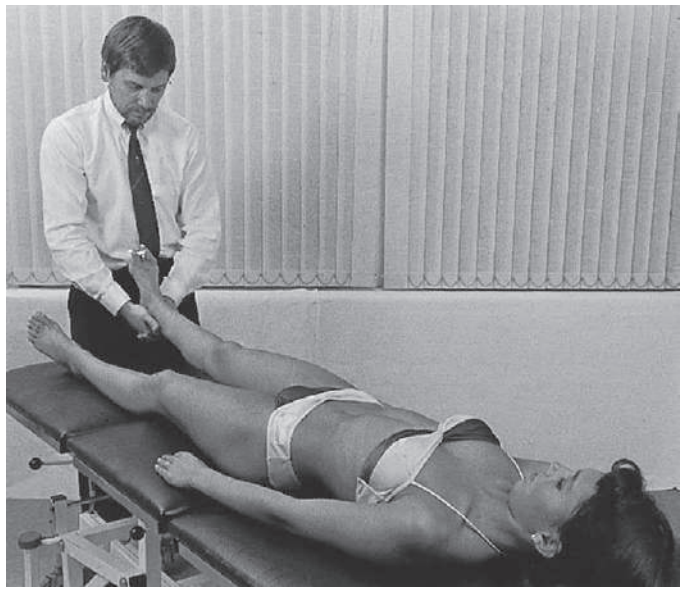


(b)

Fig 45.5 • Resisted flexion (a) and extension (b).

Resisted flexion

This is performed with the hip joint flexed to 90°. Both hands are placed at the anterior and distal end of the thigh so as to exert counterpressure, while the patient attempts to flex the hip. The lower leg is supported in 90° of flexion at the knee. To stabilize the ilium, the examiner places one knee against the tuberosity of the ischium (Fig. 45.5a). Pain and weakness are noted and again carefully compared with the other hip. This test gives a positive result in the following circumstances:

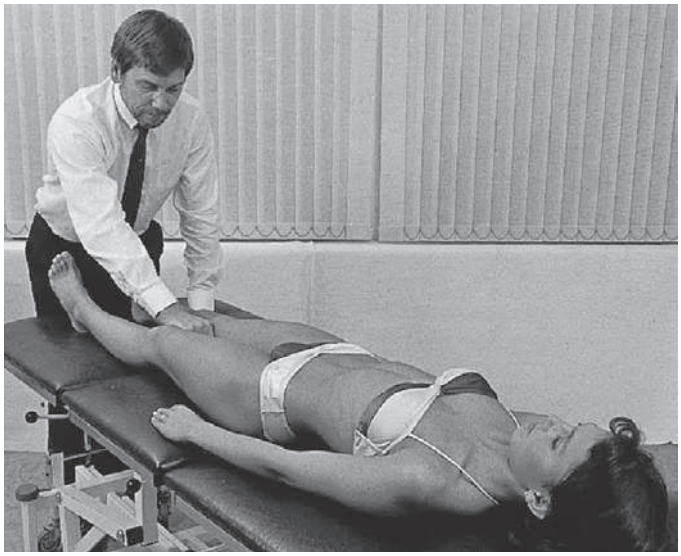
- *If pain alone is provoked*, the possible lesions are: strain in the psoas, sartorius or rectus femoris muscle; obturator hernia is another possibility.
- *Painful weakness* is found in avulsion fracture of the lesser trochanter, abdominal neoplasm infiltrating the psoas muscle or metastasis in the upper femur.
- *Painless weakness* is the result of paresis of the psoas muscle, usually the consequence of a second lumbar root palsy but rarely a third. If the palsy is bilateral, neoplasm at the second lumbar level should always be suspected.

Resisted extension

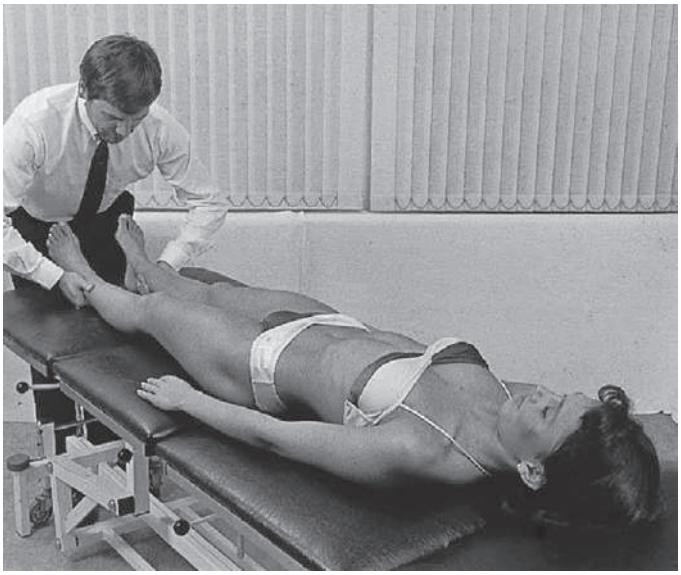
This tests the gluteus maximus and the hamstrings. The hip joint is slightly flexed and the knee joint remains extended. The examiner places both hands at the heel of the foot and resists extension (Fig. 45.5b).

Pain and weakness are noted:

- *Pain* usually results from a hamstring lesion but gluteal bursitis or sacroiliac strain is also a possibility.
- *Weakness* indicates a lesion of the first sacral root.



(a)



(b)

Fig 45.6 • Resisted adduction (a) and abduction (b).

Resisted adduction

The examiner places the clenched fist between the patient's knees and asks the patient to squeeze (Fig. 45.6a).

- *Pain* is usually the result of an adductor longus lesion.[7] Fracture, neoplastic invasion of the pubic bone and a sacroiliac lesion are other possibilities.

Resisted abduction

The hip joints are placed in a slightly abducted position. The examiner resists the abduction movement at the ankles (Fig. 45.6b). This test primarily activates the gluteus medius, gluteus minimus and tensor fasciae latae.

- *Pain* is usually the result of the compression of underlying structures, such as an inflamed gluteal bursa, or is the consequence of stress placed on strained or inflamed sacroiliac ligaments.
- *Weakness* is found in a palsy of the fifth lumbar root from a disc herniation at the same level. It may also be the result of anatomical changes, as in congenital dislocation of the hip or coxa vara; in such circumstances the muscle's origin lies closer to its insertion, which makes the contraction less efficient.

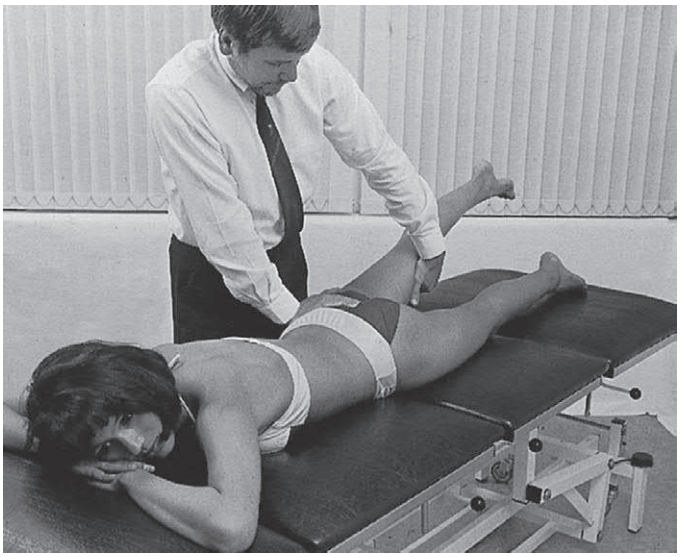


Fig 45.7 • Passive extension.

Prone

Passive movements

There are two passive movements.

Passive extension

The ipsilateral hand is placed over the mid-buttock. The other hand grasps the thigh just below the patella. The test is performed by simultaneous movement of both hands in opposite directions. The knee should stay extended, to prevent tension on the rectus femoris. By pressing the pelvis firmly on to the couch, the examiner prevents any stress from reaching the sacroiliac and the lumbar joints (Fig. 45.7). The average range of movement is 30°. The normal end-feel is capsular-elastic. Extension is one of the movements that become restricted in arthritis or arthrosis.

Passive medial rotation

Both hips are examined together, even though a separate assessment will have been made in the supine position. The knees are flexed to a right angle. The thighs are rotated by pressing the feet apart (Fig. 45.8). Care should be taken to ensure that the buttocks are level.

A small amount of limitation of medial rotation can now be ascertained.

- *Minor limitation* is often important, as this is the first movement to become restricted at the onset of arthritis.[1]

Resisted movements

There are four resisted movements.

Bilateral resisted lateral rotation

This is performed with the knees flexed to a right angle. With crossed arms, the examiner presses the hands against the inner side of both the patient's legs at the internal malleoli of the ankle (Fig. 45.9a).

- Pain may be the result of muscle strain, i.e. the quadratus femoris. As already stated, it more commonly results from compression of an inflamed bursa.

Bilateral resisted medial rotation

This is performed in the same way but both hands are pressed against the outer malleoli (Fig. 45.9b). This creates tension in the medial part of the hamstrings, tensor fasciae latae and gluteus medius and minimus. However, pain is more often the result of compression of an inflamed bursa.

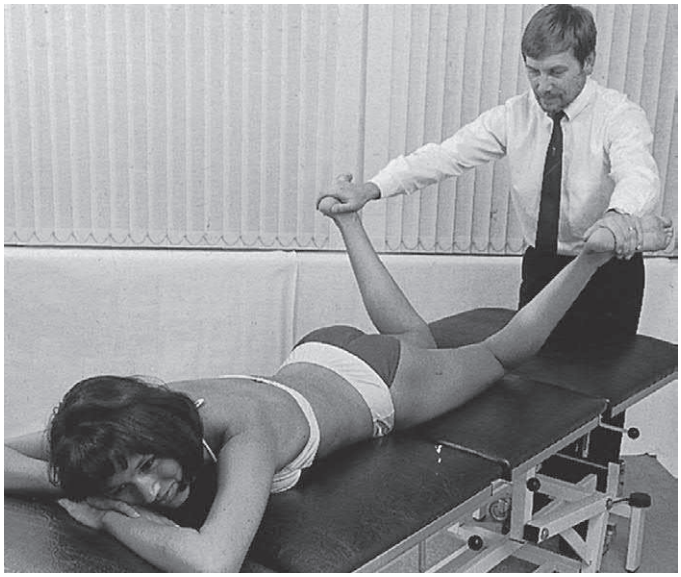


Fig 45.8 • Passive medial rotation.

Resisted flexion of the knee

This tests the hamstrings. The knee is positioned in 70° of flexion. One hand fixes the ilium, while the other hand presses against the distal end of the lower leg (Fig. 45.10a).

- *Pain* in the thigh results from a lesion in the semitendinosus, semimembranosus or biceps femoris. Pain at the ischial tuberosity indicates tendinitis of one of these muscles or a lesion of the sacrotuberous ligament.
- *Weakness* is present in first and second sacral root palsy and is usually the result of disc herniation at the fifth lumbar level.

Resisted extension of the knee

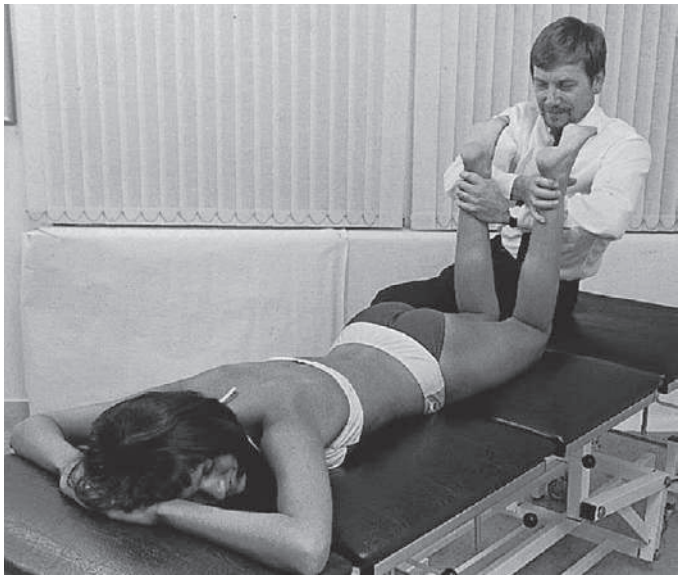
This tests the quadriceps. The knee is held in 70° of flexion. The ipsilateral hand is used to stabilize the thigh on the couch. The elbow of the other arm is placed at the distal end of the lower leg to resist extension movement (Fig. 45.10b). In order to be able to withstand even the strongest extension, the hand of this arm may grasp the stabilizing arm.

- *Pain* in the thigh is from a lesion in the quadriceps.
- *Weakness* results from a third lumbar root lesion.

Palpation

When tendinitis is suspected, points of tenderness are sought. Both sides should be compared. However, it is only when a resisted test is positive that it is worthwhile palpating for the exact site of the lesion.

In bursitis, palpation affords little assistance. It is the response to resisted movements that helps to indicate the bursal relationship with a muscle.



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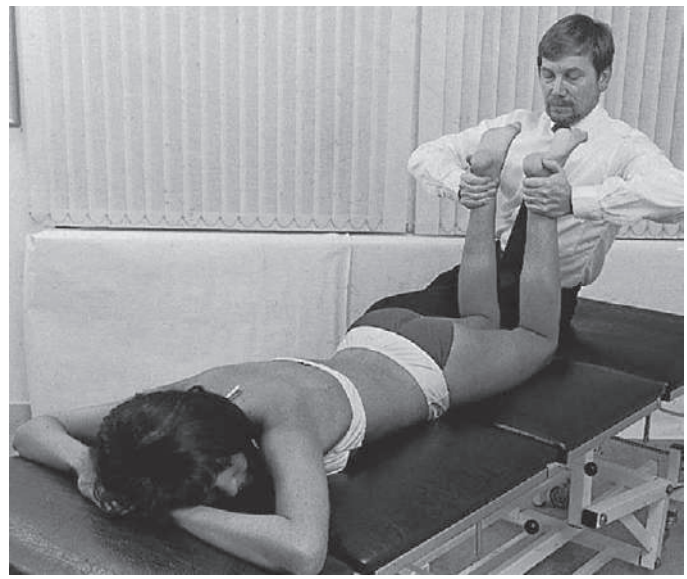
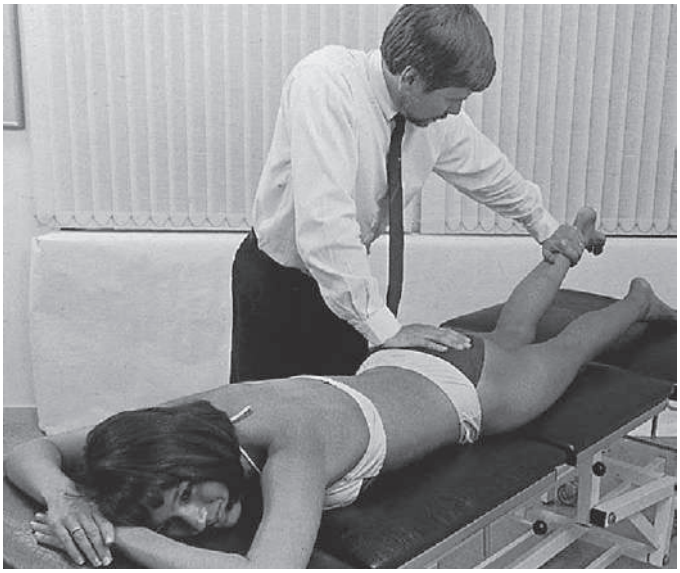


(b)

Fig 45.9 • Bilateral resisted lateral (a) and medial (b) rotation.



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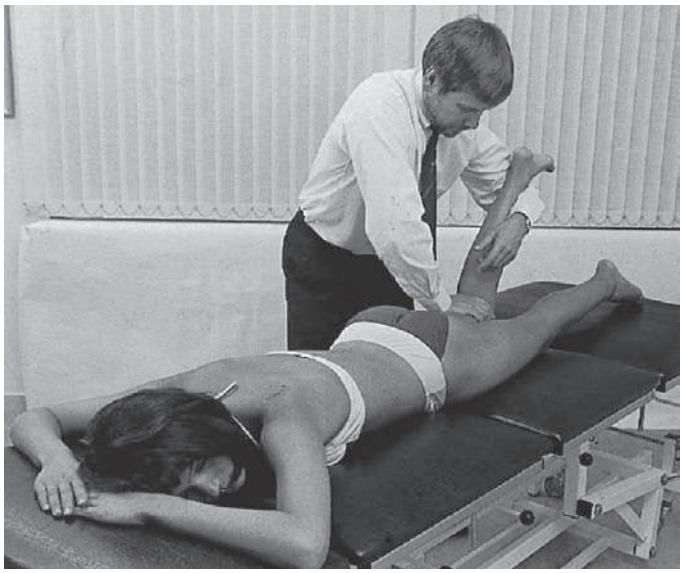
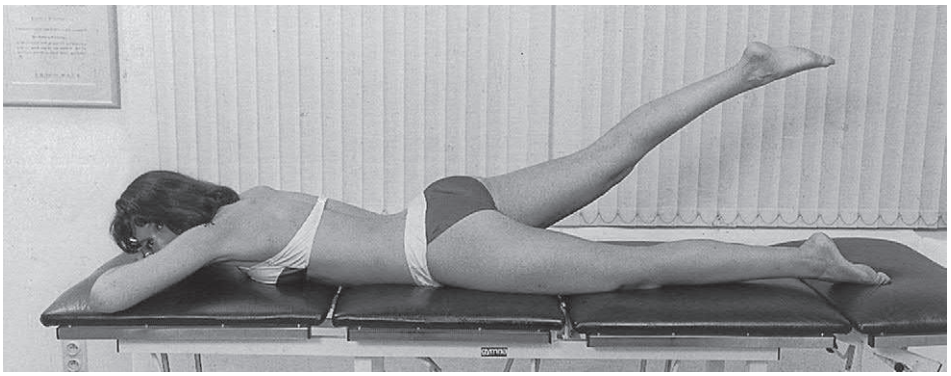


(b)

Fig 45.10 • Resisted flexion (a) and extension (b) of the knee.

Fig 45.11 • Sustained active extension of the hip in the prone position.



Accessory tests

If signs warrant or the history is indicative, accessory tests can be performed.

Palpation of the femoral arterial pulse (see p. 516)
The pulse is just inferior to the inguinal ligament, halfway between the anterior superior iliac spine and the pubic symphysis.

Combined passive flexion/adduction
This test should be interpreted with care because it includes several structures. It may be a localizing sign both in tendinitis of the rectus femoris and in psoas bursitis. The implication is that the lesion lies in a position where it can be compressed. The test is also painful in arthritis of the hip joint and in sacroiliac conditions.

Sustained active extension of the hip in the prone position
This test (Fig. 45.11), continued for several minutes, provokes gluteal pain in claudication due to a block in the common iliac or internal iliac arteries.

Technical investigations

Interpretation of the results of technical investigations without previous clinical diagnosis may often be misleading. The classic example is asymptomatic osteoarthrosis, easily visible on the X-ray but not causing any pain, or a loose body in an osteoarthrotic joint, where the radiograph shows the arthrosis but not the subluxated piece of cartilage. However, if symptoms and clinical signs warrant, technical investigations become an obligatory part of assessment.

This is especially the case in:

- Hip joint lesions in children
- Suspected aseptic necrosis
- Positive sign of the buttock (see p. 637).

In these circumstances radiography, computed tomography (CT), magnetic resonance imaging (MRI), sonography or arthroscopy will be performed to confirm or exclude a particular diagnosis.

Two diagnostic techniques have become popular during recent decades. *Ultrasonography* (ultrasound) is an excellent

Box 45.4

Summary of examination

Preliminary examination

- Lumbar spine
- Sacroiliac joint

Basic functional examination

- Supine
  - Five passive movements
  - Four resisted movements
- Prone
  - Two passive movements
  - Four resisted movements

Palpation

- Tendinitis
- Bursitis?

Accessory tests

- Palpation of femoral arterial pulse
- Passive flexion/adduction
- Sustained active extension of the hip
- Trendelenburg test

Technical investigations

method to detect intra-articular fluid.[8,9] It may also be a very useful auxiliary method in estimating the degree of tendon and muscle ruptures[10] and in localizing bursitis.[11] However, the method requires a lot of experience, and diagnostic precision depends entirely on the skill of the examiner.

There is general agreement that hip *arthroscopy* is valuable as a diagnostic investigation in patients with catching or transient locking of the hip (loose bodies, synovial tags and lesions of the labrum).[12–14]

Examination of the hip and buttock is summarized in Box 45.4.

Access the complete reference list online at
www.orthopaedicmedicineonline.com

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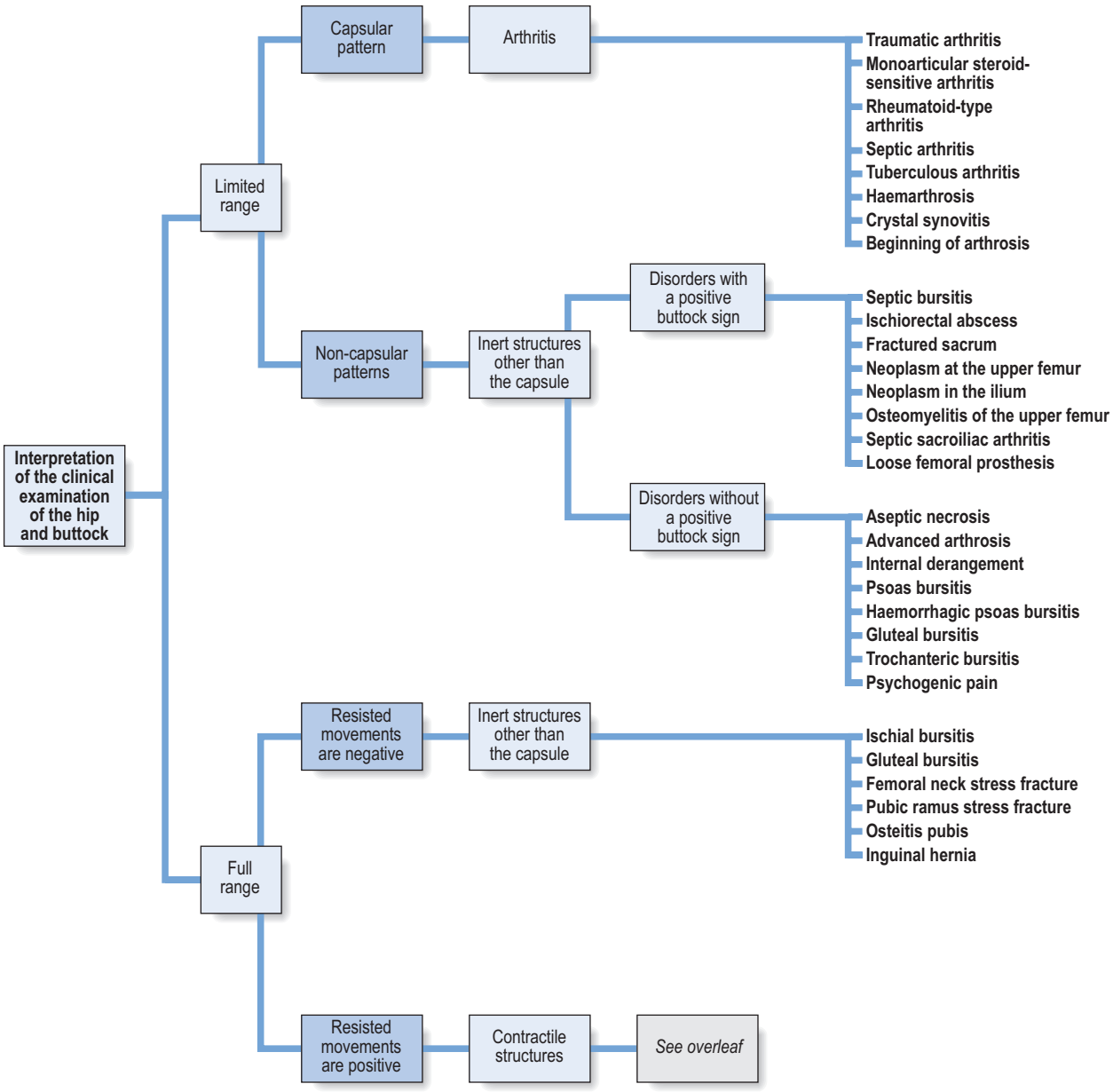
Interpretation of the clinical examination of the hip and buttock

46



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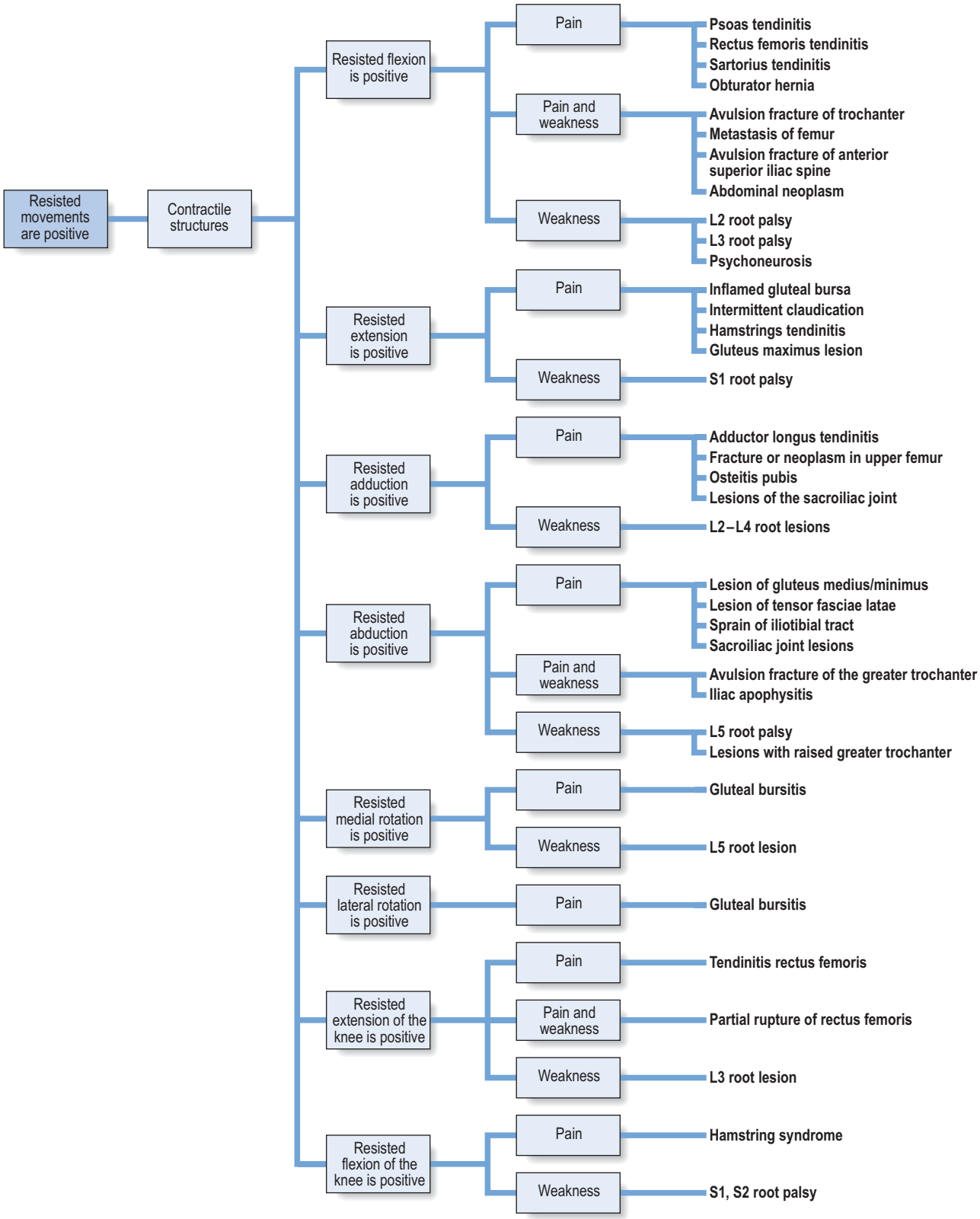
Resisted movements are positive
Contractile structures
Resisted flexion is positive
Pain
Psoas tendinitis
Rectus femoris tendinitis
Sartorius tendinitis
Obturator hernia
Pain and weakness
Avulsion fracture of trochanter
Metastasis of femur
Avulsion fracture of anterior superior iliac spine
Abdominal neoplasm
Weakness
L2 root palsy
L3 root palsy
Psychoneurosis
Resisted extension is positive
Pain
Inflamed gluteal bursa
Intermittent claudication
Hamstrings tendinitis
Gluteus maximus lesion
Weakness
S1 root palsy
Resisted adduction is positive
Pain
Adductor longus tendinitis
Fracture or neoplasm in upper femur
Osteitis pubis
Lesions of the sacroiliac joint
Weakness
L2–L4 root lesions
Resisted abduction is positive
Pain
Lesion of gluteus medius/minimus
Lesion of tensor fasciae latae
Sprain of iliotibial tract
Sacroiliac joint lesions
Pain and weakness
Avulsion fracture of the greater trochanter
Iliac apophysitis
Weakness
L5 root palsy
Lesions with raised greater trochanter
Resisted medial rotation is positive
Pain
Gluteal bursitis
Weakness
L5 root lesion
Resisted lateral rotation is positive
Pain
Gluteal bursitis
Resisted extension of the knee is positive
Pain
Tendinitis rectus femoris
Pain and weakness
Partial rupture of rectus femoris
Weakness
L3 root lesion
Resisted flexion of the knee is positive
Pain
Hamstring syndrome
Weakness
S1, S2 root palsy

47

Disorders of the inert structures

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Capsular pattern

The capsular pattern at the hip joint is gross limitation of medial rotation, abduction and flexion, less limitation of extension, and little or no limitation of adduction and lateral rotation (Fig. 47.1). In advanced arthritis, abduction and internal rotation are impossible and associated with obvious limitation of flexion and extension.

A capsular pattern in the hip joint of a child or adolescent always implies a serious problem. The slightest limitation of movement should be reason enough to put the child on bed rest and start diagnostic procedures to detect the cause. Weight bearing is prohibited until the reason for the capsulitis is discovered (see online chapter *Hip disorders in children*).

If a capsular pattern is present at the hip and resisted movements do not hurt, the conditions discussed in this section should be considered.

Traumatic arthritis

This results more often from overuse of a stiffened and arthrotic joint than from a single injury. When it occurs in a child, transitory arthritis or the beginning of Perthes' disease should always be suspected (see online chapter *Hip disorders in children*).

In traumatic arthritis in adults, a single intra-articular injection with 40 mg of triamcinolone often leads to permanent cure.

Monoarticular steroid-sensitive arthritis

The patient complains of considerable aching in the L3 dermatome, first during exertion but later at night too. In an early case, there is only slight limitation of movement with a capsular pattern, which gradually becomes more obvious. The end-feel is that of muscle spasm and resisted movements are negative.

Radiographs are negative in early cases, whereas in more advanced arthritis a generalized loss of joint space may be seen. If the disorder is left untreated, there is a risk of early osteoarthrosis supervening, which then complicates the condition.

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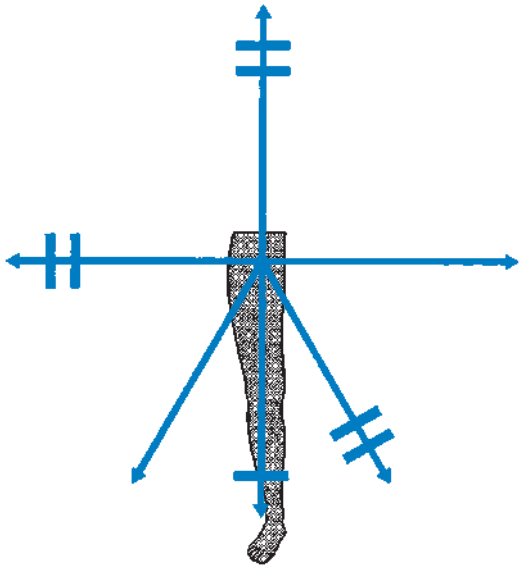


Fig 47.1 • The capsular pattern at the hip joint.

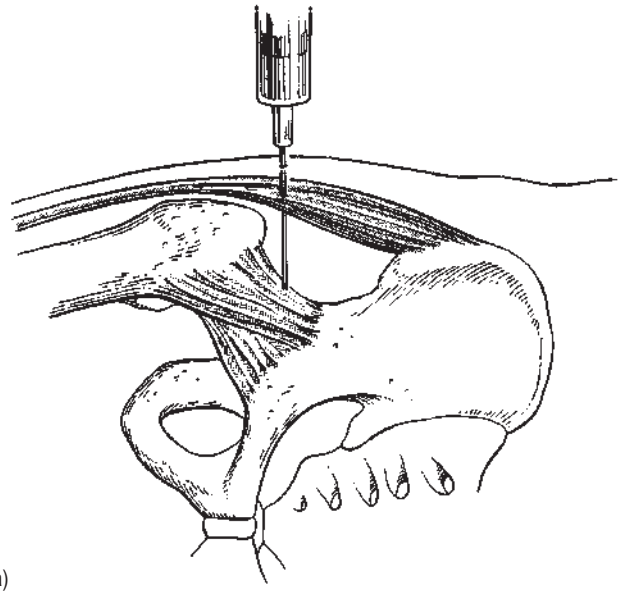
As in idiopathic monoarticular arthritis of shoulder, knee and elbow, the true aetiology remains unclear but the condition subsides immediately and lastingly with two intra-articular injections of 50 mg of triamcinolone.

Technique: injection

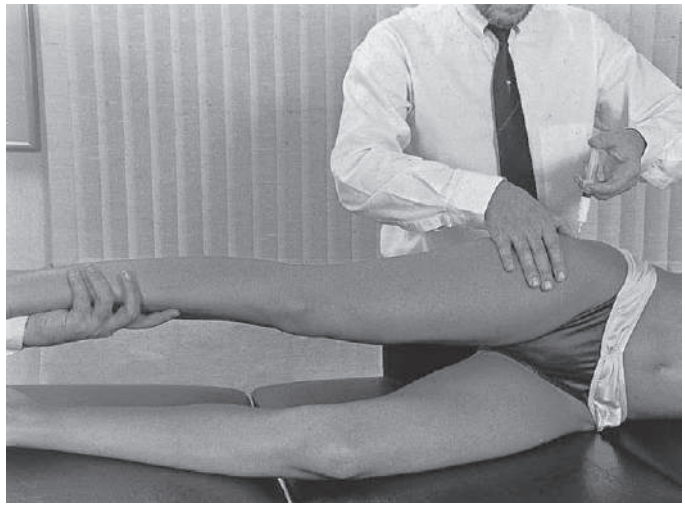
Although there are many different approaches to the hip joint, the safest is from the lateral aspect because there are no important blood vessels or nerves on this aspect (Fig. 47.2).[1]

The patient lies on the painless side, with outstretched legs and a small pillow between the knees, or with the upper leg supported. This relaxes the iliotibial tract and makes the upper border of the trochanter more easily palpable. The femur is kept in the anatomical position and the leg is not allowed to rotate internally. In this position, the trochanteric border lies vertically above the acetabulum. A point is chosen in the middle of the border and a 7 cm needle is thrust in vertically downwards. At 4–7 cm depth, the tip of the needle is felt to pierce the thick ligamentous structure of the capsule, before striking the bone of the femoral neck; 50 mg of triamcinolone is injected. No particular resistance is experienced when the drug is forced in but the patient may feel some aching down the leg.

Although some after-pain may be present for the next 12 hours, there is considerable subjective improvement from the second day on, and the following night the patient will have what is probably the first undisturbed sleep for months. The injection is repeated after 2 weeks. The patient should be told to avoid hard work and exercises for a further 2 weeks. Usually, there are no recurrences.



(a)



(b)

Fig 47.2 • Intra-articular injection of the hip joint.

In ankylosing spondylitis, the arthritis progresses with fluctuations until the hip becomes almost completely fixed in flexion. Apart from systemic treatment, an intra-articular injection can be given during flares but caution should be taken with repeated injections of triamcinolone, for fear of steroid arthropathy; no more than three injections per year should be given. During periods when symptoms subside, gentle stretching can be undertaken to counteract the progressive stiffening.

In polymyalgia rheumatica, both shoulders and hips are involved early in the course of the disease. Systemic therapy quickly relieves signs and symptoms.[2]

Rheumatoid conditions

In rheumatoid conditions, the hip is affected only late in the evolution of the disease.

Septic arthritis

As a rule, this is caused by haematogenous dissemination, although it can also be the result of an intra-articular injection.

Septic arthritis at the hip is not only a disaster for the joint but can also be life-threatening.[3]

Acute pain in the L3 dermatome, together with fever and a gross capsular pattern, draws attention to this disease.

Local aspiration should be carried out and systemic antibiotic therapy should be administered at once.[4]

Tuberculous arthritis

Insidious onset and slow progression of a monoarticular arthritis, together with muscle wasting, are the first features to draw attention to this disorder. The capsular pattern is gross, the hip sometimes fixed in flexion. At the onset, systemic manifestations may be minimal or absent.

The diagnosis is made on radiography and examination of the synovial fluid.

Chondrocalcinosis and gout

The lesions very rarely affect the hip joint. The diagnosis is suspected when acute hip pain and a gross capsular pattern are encountered, especially if there is a previous history of attacks of acute pain in other joints. The diagnosis is confirmed by the demonstration of crystals in the synovial fluid.

Monoarticular arthritis in middle-aged people

This condition was described by Cyriax[5] (his p. 386). For no special reason, a middle-aged patient experiences aching at the anterior aspect of the thigh during exertion. Clinical examination of the hip shows only a slight capsular pattern, with some limitation of internal rotation and flexion. There is pain at the end of range and the end-feel is elastic. The radiograph reveals nothing but a normal hip joint. The condition continues unchanged for months. Intra-articular injection with triamcinolone seems to be ineffective, but the lesion responds very well to stretching of the capsule, which relieves the pain quickly and permanently (see p. 633–634 for technique).

Osteoarthrosis

Aetiology

It is widely accepted that the most likely causative factor in the development of arthrosis of the hip is the incapacity of (parts of) the hip to withstand mechanical stresses. In the literature, a distinction is made between primary and secondary arthrosis.[6]

Secondary arthrosis originates from a pre-existing anomaly at the hip, such as Perthes' disease, acetabular dysplasia and epiphysial dysplasia. Rapidly developing osteoarthrosis most often results from aseptic necrosis.

When the osteoarthrosis results from an undetermined abnormality of the cartilage or the subchondral bone, the condition is called idiopathic or primary. Primary osteoarthrosis is extremely rare – in more than 90% of cases, previous abnormalities in the hip joint can be demonstrated.[7–9]

Initiation and progression of osteoarthrosis at the hip seem to be caused by continuous interaction between such factors as deterioration of articular cartilage, changes in the subchondral bone, stiffening of the capsule, dysfunction in the neuromuscular system and chemical changes in the composition of the synovial fluid.

Cartilage fibrillation

Articular cartilage is a viscoelastic substance. It deforms slightly under load. The main purposes of the tissue are (a) to transmit joint forces to underlying bone structures, (b) to minimize the frictional drag of joint motion and (c) to maximize the contact area of the joint under load.

Strain may induce loss of proteoglycan molecules in the collagen network.[10] This results in an increase in hydration and loss of tensile strength, and initiates the fibrillation of the cartilage.[11,12]

Subchondral bone

The health of articular cartilage depends largely on the mechanical qualities of its bony subchondral bed: the greater the discontinuity in elasticity between cartilage and subchondral bone, the more shear stress will occur.[13] The presence of stiffened subchondral bone thus increases the likelihood of the progression of cartilage lesions.[14]

Capsule of the joint

Inflammation of the synovial membrane can also be responsible for the degeneration of cartilage: connective tissue activating peptides and catabolites are set free during inflammatory reactions and have a negative influence on cartilage and subchondral bone.[15,16]

In addition, rigidity of the capsule, so often seen in the early stages of arthrosis,[17] plays a role in its progression: because of the loss of laxity, the normal gliding movement of the cartilage surfaces is altered, which imposes a change in load distribution and thus increases stresses on particular areas of the joint.

Synovial fluid

The composition of the synovial fluid may enhance the development of osteoarthrosis: a decrease in viscoelastic quality provokes more friction between the joint surface. It is well to remember that immobilization also decreases synovial fluid hyaluronan levels and may thus contribute to the progression of osteoarthrosis.[18]

Muscular dysfunction

Muscular dysfunction and disturbed neuromuscular balance are quite common in osteoarthrosis of the hip. They cause the joint to work under abnormal conditions and may play a role in the development or continuation of hip osteoarthrosis.[19] A pattern of tightness and overactivity of the psoas, adductors,

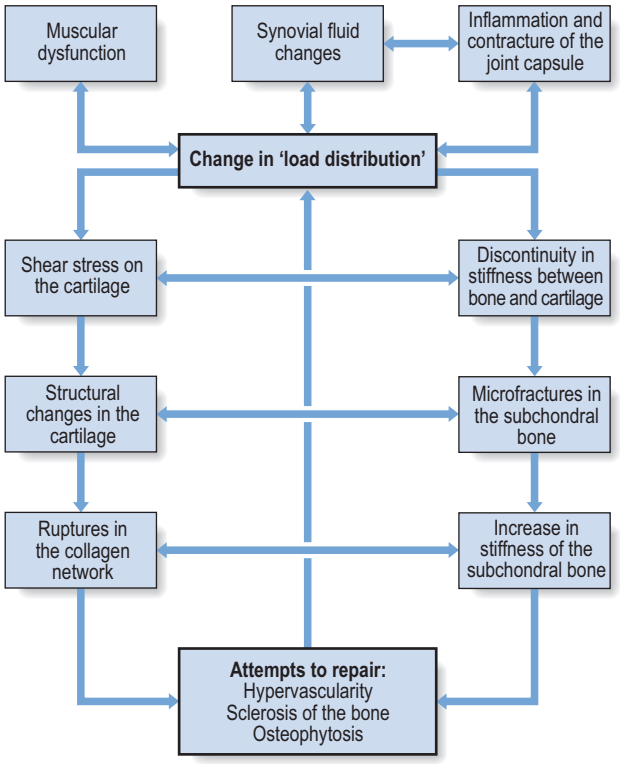


Fig 47.3 • The aetiology of osteoarthrosis.

tensor fasciae latae and rectus femoris is typical in arthrosis of the hip joint, whereas the gluteals show a tendency towards weakness and inhibition.[20,21]

The continuous interaction between the changing structures of the joint imposes a physiological imbalance, which starts the process of degeneration. Vigorous and persistent attempts to repair the degenerative changes aggravate the already disordered joint function and set up a vicious circle. Hypervascularity, weakening of the subchondral bone, fatigue fractures, localized zones of collapse, flattening of the femoral head and formation of osteophytes then become inevitable (Fig. 47.3). This whole process can lead to rapid destruction of the joint; however, this is not always the case and spontaneous clinical and radiological improvements can occur.[22]

Conclusion: osteoarthrosis is a 'joint failure', similar to 'heart failure'. The aetiology is multiple but each of the components of the joint (cartilage, subchondral bone, synovial fluid and joint capsule) plays a role in the pathogenesis.

Symptoms and signs

Although the diagnosis of advanced arthrosis of the hip is easy, it may be difficult to diagnose the early stages. Also, there is sometimes a striking lack of correspondence between the clinical picture and the radiographic appearance.

Symptoms

The pain is located in part of the L3 dermatome: the groin, the anterior aspect of the thigh, the knee and the leg as far as the ankle joint. It is important not to forget that a separate part of the dermatome is located in the region of the sacroiliac joint, so patients with arthrosis of the hip may also present with unilateral pain in the lower back.

In the early stage, pain is only present during and after exercise but later it becomes continuous and can even disturb sleep. It is suggested that pain at night in coxarthrosis is associated with an increase in intracapsular pressure and subsequent joint contracture.[23] If the patient mentions twinges during normal walking, the disorder is considered to be complicated by impacted loose bodies (see later).

Signs

The examination often reveals a capsular pattern, with internal rotation the most limited and some limitation of flexion, extension and abduction, but this is certainly not always so. Many cases of hip osteoarthrosis present with other movement restriction patterns: for example, gross limitation of both internal and external rotation.[24] As a rule, there is considerable difference between the clinical signs of an early osteoarthrosis and those found in advanced cases.

In the early stage, there is merely a capsular stiffening, without much erosion of cartilage or osteophyte formation. The clinical findings are therefore a capsular pattern with a less elastic end-feel. Most commonly, internal rotation is found to be the most painful and limited, followed by limitation of flexion, abduction and extension.

In advanced instances, gross limitation is found, with loss of all rotational movement. In extreme cases, a 'hinge joint' develops, allowing only flexion and extension in an oblique plane: the femur moves laterally when flexion is forced. The end-feel is hard and marked coarse crepitus can be palpated. Muscle tightness can sometimes be detected by performing muscle length test procedures such as those described by Janda and Lewit.[25–27]

Radiography

'Osteoarthrosis of the hip' must be a clinical diagnosis and it is unwise to rely entirely on the radiograph for estimation of functional incapacity and for deciding on optimal treatment. First, there is a considerable lack of correspondence between the degree of pain, the mobility of the joint and the radiograph appearances.[28] Second, the patient may suffer from other lesions at or around the arthrotic hip. These lesions – loose body, psoas or gluteal bursitis (see pp. 642–647) – are not radiographically visible. If a radiological examination is performed without a full history and proper clinical examination of the hip, such conditions will be missed and the painless arthrosis will be blamed for the pain.

The radiological changes in hip osteoarthrosis are: presence of subchondral sclerosis in femoral head and acetabulum, joint space narrowing, femoral head deformity, marginal

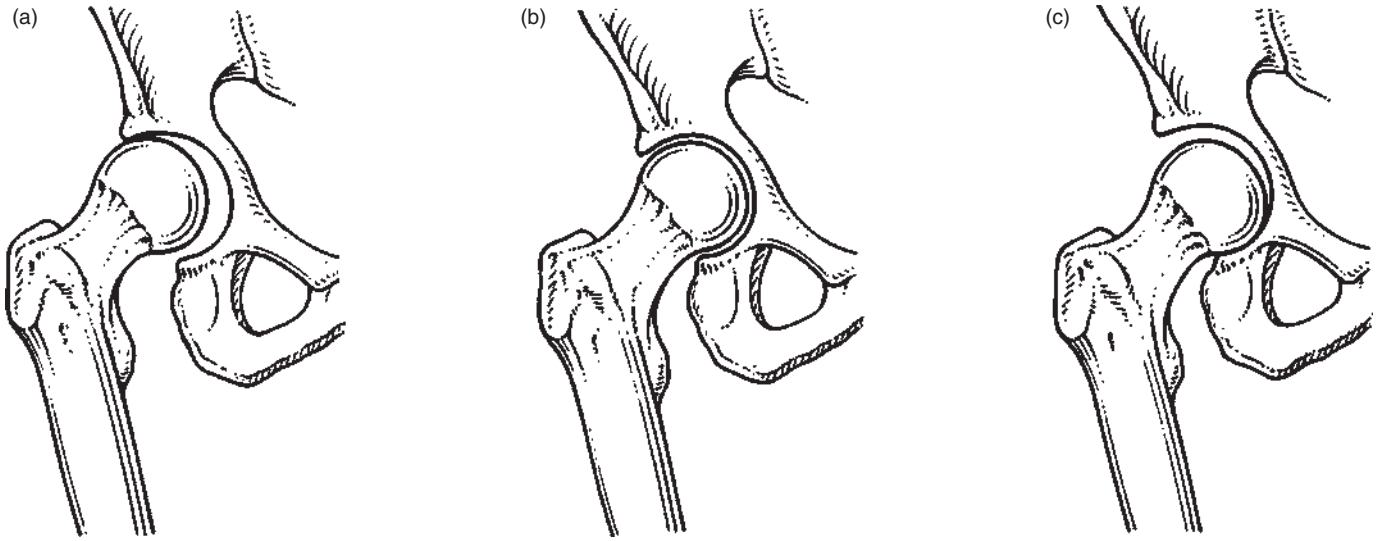


Fig 47.4 • Migration of the femoral head: (a) superolateral migration; (b) non-migratory hip osteoarthrosis; (c) medial–axial migration.

osteophytes, cystic changes in the femoral head and the acetabulum, and migration of the femoral head. The severity of the radiological changes is classified into four grades (Kellgren 1–4). Classically, there are three radiographic types of arthrosis of the hip, according to the direction of migration of the femoral head.

- Most osteoarthrotic hips show superolateral migration of the femoral head with localized erosion of cartilage at the lateral border of the labrum and a widening of the inferomedial part of the joint.[29] Cameron and MacNab suggest that this is the form of osteoarthrosis that is primarily related to capsular restrictions and responds well to capsular stretching.[30]
- A medial–axial migration occurs in about 10–15% of cases. This presentation is usually associated with gross osteophytosis at the lower border of the femur and labrum.
- Another 10–15% of cases are non-migratory hip osteoarthrosis, associated with superior or concentric loss of cartilage space and concentric formation of osteophytes (Fig. 47.4).

Treatment

Early treatment of osteoarthrosis is vital. There is evidence that reduced motion of the hip (from capsular tightening and muscular imbalance) further increases the degenerative process in cartilage and subchondral bone. Several studies have demonstrated the beneficial effect of exercise on pain and disability.[31] The treatment of choice is therefore early stretching of the joint (grade B mobilizations). Treatment with injections has only a limited indication. In later stages or in quickly developing osteoarthrosis, conservative treatment is useless and surgery is indicated (Box 47.1).

Box 47.1

Treatment of osteoarthrosis of the hip

Early and/or slowly developing osteoarthrosis

Capsular pattern, ligamentous end-feel and absence of coarse crepitus

- Capsular stretching – angular mobilizations
  - In flexion
  - In extension
  - In medial rotation
- Traction (manual or mechanical)
  - In maximally loose-packed positions
  - In other positions
- Muscular re-education
  - Stretching of shortened muscles
  - Activation of inhibited muscles
  - Gait control
- (Intra-articular injections)

Advanced and/or quickly developing osteoarthrosis

Non-capsular pattern, hard end-feel and coarse crepitus

- Surgery
- (Intra-articular injections may give temporary relief)

Capsular stretching

It is generally believed that early stretching of a tight capsule may prevent joint damage or at least slow further progression.[32] Therefore, stretching is the treatment of choice in the early stage of the disease. The decision to use it depends largely on the clinical findings: early arthrosis with a slight capsular end-feel usually responds quite well to such treatment. Stretching is of no use in advanced arthrosis with gross limitation of movement, a hard end-feel and coarse crepitus because these are

the clinical indications of gross cartilaginous destruction and formation of large osteophytes.

It is vital to start stretching treatment as early as possible. A stiff and inflamed capsule is one of the reasons for disturbed load distribution, responsible for further progression of degeneration. Furthermore, overuse at a stiffened capsule provokes consecutive strains, resulting in traumatic inflammation and pain.

Treatment is given 2–3 times a week, for 10–20 sessions. The joint is mobilized in three directions – flexion, extension and internal rotation – for 5–10 minutes each. The patient may experience slight aching for 1 or 2 hours after the forcing. This is an important criterion to the therapist, who must adjust the vigour of the treatment according to the length of time that the increased pain lasts. Slight after-pain for 1–2 hours is acceptable. If the patient suffers from increasing pain over 1 or 2 days, it is clear that the joint has been forced too much. If neither after-pain nor improvement follows, greater stress, more persistently applied, is used during the next visit.

The result that can be expected is not a marked increase in range but merely a decrease in pain. It is remarkable that pain at night, even of many months' standing, can often be abolished by a few sessions of capsular stretching. Used at an early stage, years of relief can often be obtained, although it may be necessary to repeat the mobilizations once or twice a year to keep the capsule as mobile as possible.

Technique: forced flexion

The patient adopts a half-lying position on the couch. The therapist stands on the affected side, level with the patient's knee. The hip is flexed as far as is comfortably possible. One hand rests on the anterior aspect of the knee, while the other presses the opposite thigh down towards the couch. With steadily increasing pressure applied to the knee, the thigh is forced towards flexion (Fig. 47.5). This position is maintained for as long as the patient can bear it: e.g. 1 minute. The pressure is then slowly released to give the patient a break and the same procedure is repeated.

Technique: forced extension – 1

The patient lies prone on a high couch. The therapist stands level with the thighs. To prevent stress on the lumbar spine, it is vital to keep the patient's pelvis on the couch when extension of the hip is forced. Therefore, with one hand, the therapist presses on the lower part of the buttock, which will obstruct movements at the lower back and the sacroiliac joint. With the other hand, the therapist grasps the thigh from the medial side, just above the patella. Both arms are kept rigid. Bending the body sideways, the therapist then pulls the patient's thigh upwards, while the patient's pelvis is pushed forcefully downwards (Fig. 47.6).

Technique: forced extension – 2

Some elderly people have difficulty lying on their stomach for any length of time and an alternative method can be used. The patient lies flat on the back, with the head supported with a cushion. The painless hip is now forced as far into flexion as possible, which tilts the pelvis and makes the other thigh lift off the couch. Sufficient extension can now be forced when sustained downward pressure is applied just above the patella (Fig. 47.7).

Technique: forced medial rotation

The patient adopts a prone-lying position on a high couch. The knee at the affected side is bent at a right angle. The therapist stands on the affected side and level with the pelvis. The ipsilateral hand is pressed on to the ilium. The other hand is placed at the medial malleolus and forces the hip into medial rotation. The pelvis rotates and moves the iliac crest backwards on the opposite side. Pressure applied on the pelvic region now forces

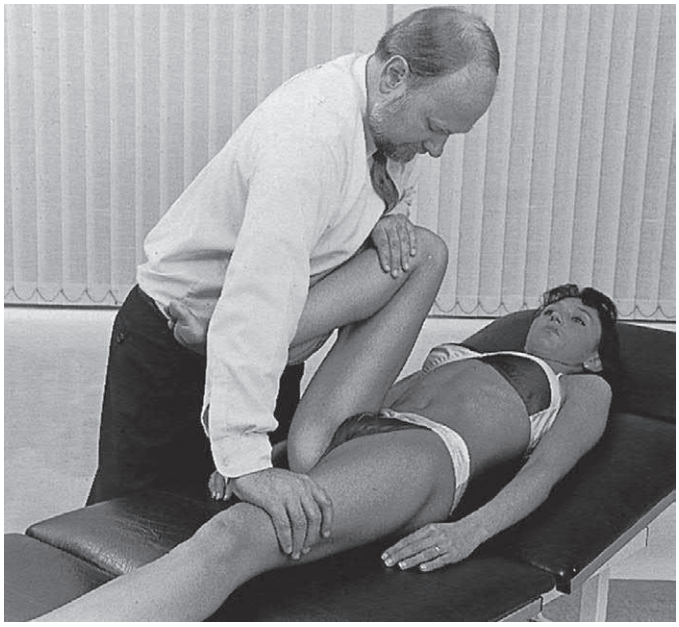


Fig 47.5 • Forced flexion of the right hip.

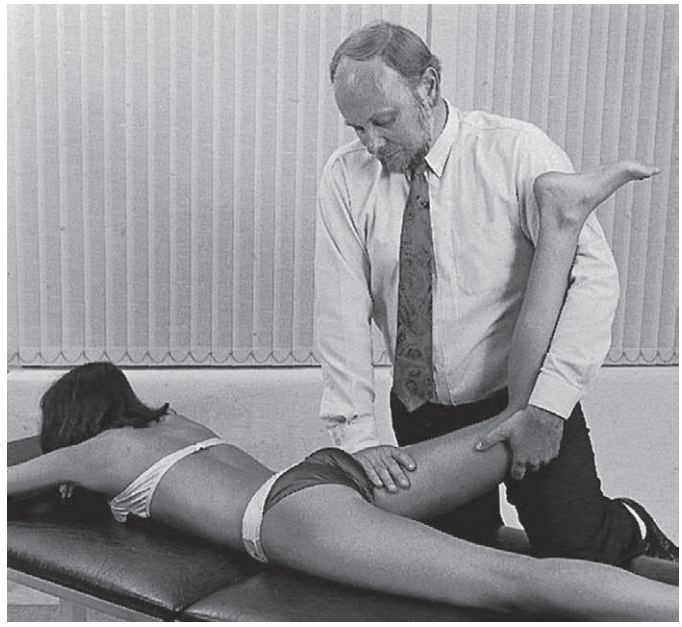


Fig 47.6 • Forced extension of the right hip – 1.

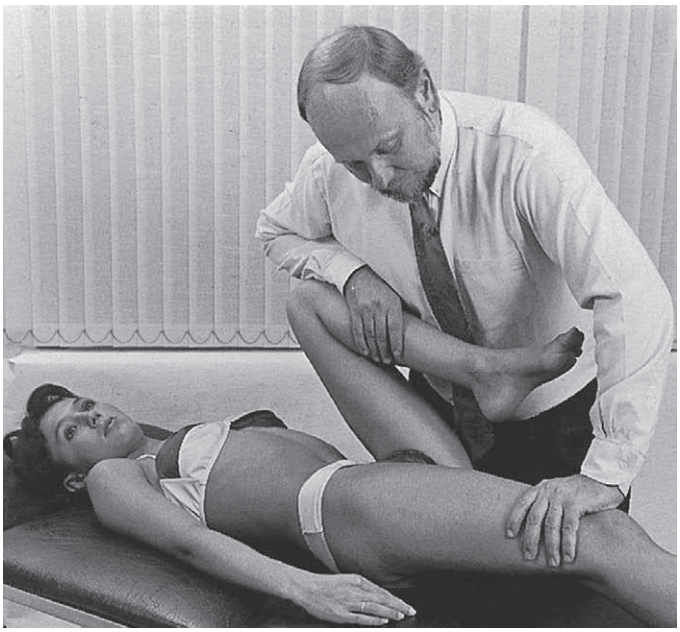


Fig 47.7 • Forced extension of the right hip – 2.

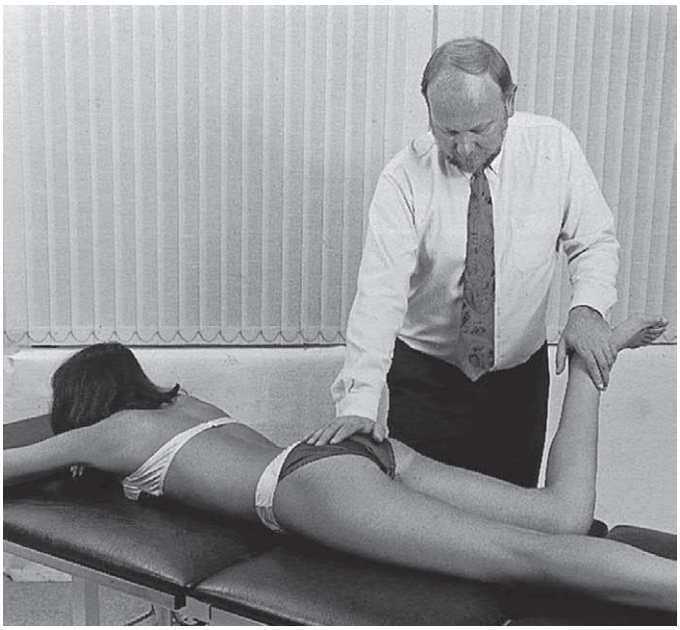


Fig 47.8 • Forced medial rotation of the right hip.

the iliac crest downwards again. When the thigh on the affected side is held firmly in the rotated position, this downward pressure will considerably increase the outward stress on the affected hip (Fig. 47.8).

Traction

Traction (either manual or mechanical) is an alternative technique to stretch the joint capsule.[33–35]

Practitioner's checklist

- The ratio of pain to end-feel will indicate the degree of applied force (see general principles of treatment, p. 93). Movements should be performed to the point of discomfort but not of pain. In suitable cases, the force can be gradually increased during the session. A whole session takes about 20–30 minutes, with pauses after every 30 seconds–1 minute.
- Slight passive oscillatory movements can be added: type I and II mechanoreceptors come into play and suppress a painful reaction from the nociceptors (see p. 92). To prevent adaptation of the mechanoreceptors, oscillations should be constantly changed in rhythm, amplitude and direction.
- Although osteoarthrosis and osteoporosis do not seem to appear simultaneously, the former protecting from the latter and vice versa,[36,37] forcing medial rotation should always be carried out with caution in elderly people, for fear of fracturing the neck of the femur.

Manual traction (distraction) can be given in two different ways: with an extended knee, in which case the traction is performed via the ankle; or with the leg flexed to more than 90°. Traction is then carried out via the proximal part of the upper leg. Because of the direction of the hemispherical acetabulum, in either case the femoral head moves inferiorly, anteriorly and laterally. Depending on the position of the joint, some parts of the capsule are stretched more intensively.

Traction I

The patient lies supine near the edge of the couch. To prevent the pelvis being pulled down or sideways, or lifted off the couch, two bands are used for fixation: one at the groin, and the other transverse over the pelvis just beneath the anterior superior iliac spines.

The therapist grasps the patient's ankle. According to the direction of the acetabulum – inferiorly, laterally and anteriorly and in agreement with the maximally loose-packed position of the joint capsule – the leg should be brought into a position of about 30° of flexion, 30° of abduction and slight lateral rotation.

Traction is performed by leaning backwards with straight arms (Fig. 47.9). Once the therapist feels the patient relaxing the muscles, a jerk can be tried by pulling the arms towards the body. At this point, slight separation of the femoral head from the acetabulum can be felt.

Traction II

The therapist sits or stands at the level of the pelvis. The leg should be flexed to at least 90° and slightly laterally rotated. Both hands take hold of the upper part of leg (Fig. 47.10a). Traction is performed in the direction of the acetabulum: inferiorly, laterally and anteriorly.

Use of a band makes it possible to lessen the effort of the therapist a great deal. Both hands then hold the leg in position at the knee (Fig. 47.10b). Traction is achieved by leaning backwards.

Traction II is not suitable for manipulating the joint.

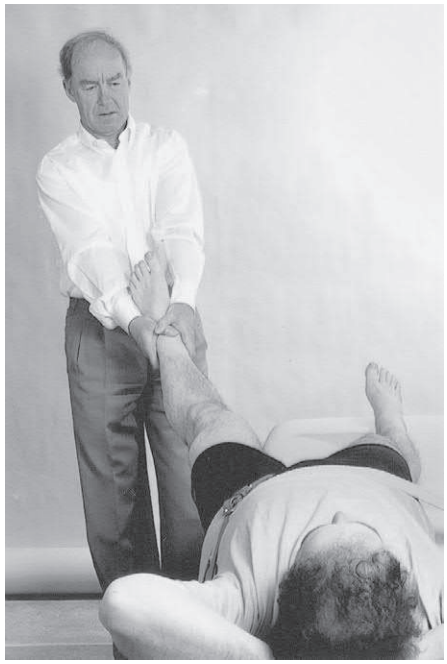


Fig 47.9 • Manual traction.

It must be noted that distraction of the hip joint can only be effective if the pelvis is completely immobilized; otherwise, the lumbar spine will compensate for the movement. To this end, two fixation belts are used. One should resist the movement of the pelvis caudally, while the other resists anterior and lateral displacement.

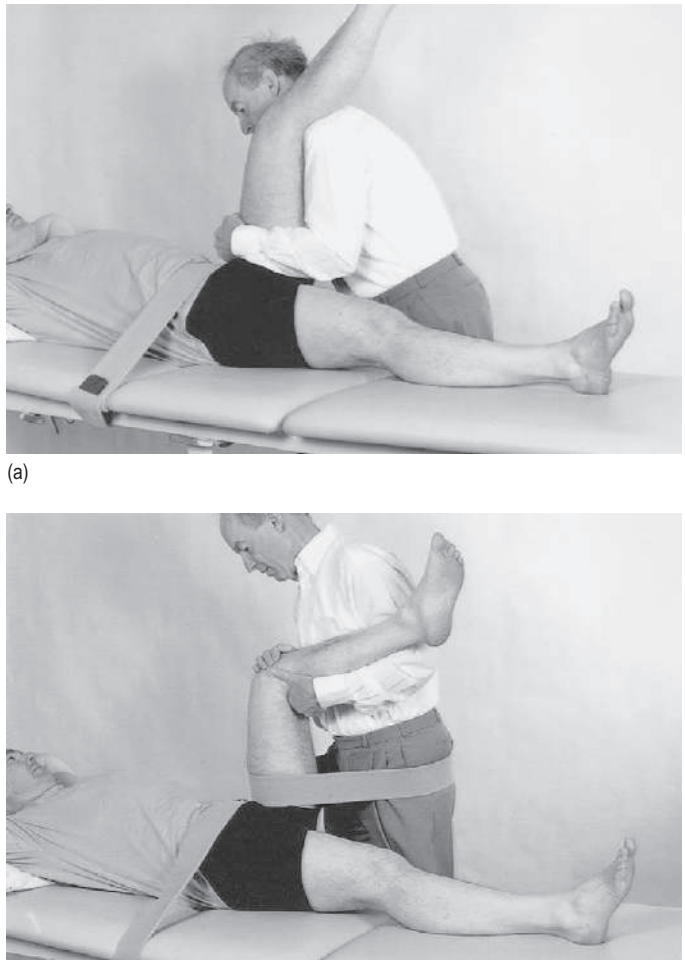
Muscular re-education

In order to correct the pattern of muscular dysfunction and a disturbed neuromuscular balance, selective activation of inhibited, weak muscles and stretching of tight, shortened muscles is advocated by several authors.[38–41] The second measure is of even greater importance because tight, hyperactive muscles interfere with the activation of inhibited muscles. These muscles (usually psoas, tensor fasciae latae and rectus femoris) are stretched slowly without straining the joint.

Activation of inhibited muscles is achieved by exercises with low loads to prevent overflow into other muscles. It is also advisable to perform exercises as closely as possible to their functional manner. For this purpose, closed kinetic chain exercises are advocated because the weight-bearing component effectively stimulates mechanoreceptors around the joint, so improving muscular contractions.[42]

Hip extensors can be activated lying supine, the lower leg over the edge of the couch, the hip extended and the knee in 90° of flexion. Pushing the foot on the floor facilitates both the gluteus maximus and the posterior part of the gluteus medius.

Activation of the *hip abductors* is performed first sitting near to the edge and at the corner of the couch, one leg extended and in contact with the floor. Supporting him- or herself on this leg, the patient activates the hip abductors. Standing with the feet about 25 cm apart and resisting pressure against the pelvis from the contralateral side is another effective exercise



(a)

(b)

Fig 47.10 • Mechanical traction.

to activate the ipsilateral hip abductors. This can also be achieved if the patient is asked to lower and raise the contralateral pelvis. Placing the hands on a chair at each side of the body for support reduces the load.

In order to reduce the harmful effect of a limp, a walking stick in the contralateral hand may be necessary at first. It decreases the contraction of the ipsilateral hip abductors and thereby reduces compressive forces on the joint.

Finally, it is necessary to be aware of any harmful effect produced by the trunk or lower limb that might influence the development and continuation of the hip joint problems, i.e. stiffness of the lumbar spine, leg-length differences, or dysfunction at the knee or the talocrural or subtalar joint.

Intra-articular injections

If the patient suffers from a subacute exacerbation because of traumatic arthritis superimposed on a stiffened arthrotic capsule, one intra-articular injection with 50 mg of triamcinolone will relieve the traumatic inflammation and the pain but increased mobility will not follow. To avoid arthropathy, this injection should not be repeated.[43]

Also, intra-articular injections with hyaluronic acid seem to be a safe and effective method of treatment for patients with advanced hip arthrosis and may lead to a significant reduction in pain scores, disability scores and analgesic use.[44–46]

Surgery

A McMurray intertrochanteric osteotomy is indicated in a painful hip that has good mobility. It has the advantage of diminishing pain while maintaining a useful range of motion in the 'natural hip'.

Total hip replacement is indicated in advanced osteoarthrosis. It is one of the most commonly performed operations in the United States, with over 280 000 procedures reported annually.[47] The benefits of total hip replacement in terms of reduced pain and improved function and quality of life for patients with advanced coxarthrosis have been well documented in the literature.[48] The prosthesis is made up of two parts: an acetabular component made of a metal shell with a plastic inner socket (the socket portion) that replaces the acetabulum, and a femoral component made of metal (the stem portion) that replaces the femoral head.

There are two major types of artificial hip replacement: cemented and uncemented. Cemented total hip replacement uses cement to secure an implant to the bone, while with cementless technology the bone heals directly to the prosthesis. The choice is usually made based on age, body weight and lifestyle. Patients with poor bone quality or with less active lifestyles are candidates for cemented total hip replacement.

The life expectancy of the prosthesis is between 15 and 20 years.[49] Aseptic loosening with or without osteolysis is the major problem and accounts for 71% of the revisions, but the incidence had decreased three times during the past 15 years to less than 3% at 10 years in Sweden.[50] However, long-term durability of the acetabular components remains a major concern.[51]

Concerns regarding high rates of failure among young, active patients and a desire to preserve bone for future revision operations led to the development of hip resurfacing arthroplasty. This differs from total hip replacement in that the femoral head is resurfaced rather than resected, thereby preserving femoral bone stock, which could theoretically decrease morbidity and improve patient outcomes associated with future revision operations.[52,53]

Non-capsular pattern

A non-capsular pattern on clinical examination of the hip indicates the possibility of lesions of the joint itself or disorders of nearby tissues such as the buttock and the groin. The straight leg raising test and resisted movements help to differentiate the two possibilities.

Serious lesions in the buttock are characterized by an interesting pattern of physical signs, called the 'buttock sign', summarized in Box 47.2. Non-capsular lesions of the hip itself comprise loose bodies, bursitis and aseptic necrosis of the femoral head.

Box 47.2

'Sign of the buttock'

- More limitation of passive hip flexion (with flexed knee) than of straight leg raising
- Other hip movements are limited in a non-capsular way

Lesions

- Septic bursitis
- Ischiorectal abscess
- Fractured sacrum
- Neoplasm at the upper femur
- Neoplasm in the ilium
- Osteomyelitis of the upper femur
- Septic sacroiliac arthritis
- Loose femoral prosthesis

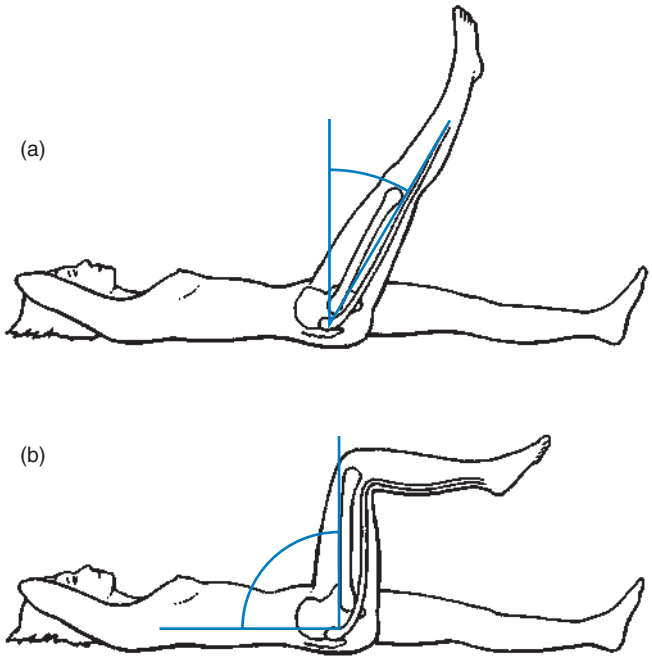


Fig 47.11 • The buttock sign: passive hip flexion is more limited and/or painful than straight leg raising.

Disorders with a positive 'buttock sign'

This clinical syndrome, described by Cyriax[5] (his p. 375), always indicates a major lesion in the buttock.

The *buttock sign* is characterized by more limitation and/or pain on passive hip flexion with a flexed knee than with an extended knee (i.e. straight leg raising; Fig. 47.11). The other passive movements at the hip joint are limited in a non-capsular way. This strange pattern immediately draws attention to the gluteal region. If the hip joint itself were affected, straight leg

raising would not be limited, except in those gross articular patterns in which flexion cannot reach 90°. If the nerve roots, the sciatic nerve or the hamstrings were affected, hip flexion with a flexed knee would not be painful or limited because it does not stretch these structures. The fact that both movements are limited and painful implicates other structures in the gluteal region.

Checking for the buttock sign is very important in pain syndromes at the gluteal region. Because there will probably be nothing characteristic about the pain, only a comparison between the results of the straight leg raising test and passive hip flexion can detect serious disorders in the buttock.[54–56] When this typical combination of signs emerges, a very careful examination of passive and resisted movements must follow. Passive movements disclose a non-capsular pattern, almost always with a full range of medial rotation. The end-feel of the limited movements is 'empty': as a consequence of the increasing pain, the examiner has to stop the movement, even though it is felt that the end of range has not been reached. Some resisted movements are painful and weak as well because they increase the tension on the affected tissues. As a rule, resisted extension and internal rotation are the most painful. Palpation may disclose a painful swelling.

To refine the diagnosis, the general temperature must be noted, a rectal examination performed and radiographs and/or bone scans done.

Septic bursitis

This is by far the commonest cause of a positive 'buttock sign'. The condition usually occurs after direct inoculation during an intramuscular injection, although haematogenous dissemination is also possible, especially in the elderly.

History

The history is often rather trivial, with increasing gluteal pain spreading down the back of the thigh to the knee and calf. During the first few hours or days, the pain eases at rest and increases during movement. Coughing does not hurt. The features do not draw immediate attention to the buttock but rather suggest a nuclear disc lesion.

Clinical examination

The patient's gait is hobbling, as if the weight can hardly be borne on the affected leg.[57] This major disability contrasts markedly with the minor degree of discomfort and is the first warning for the examiner.

Examination of the lumbar spine shows limitation of flexion, sometimes with a list towards the pain. This is logical since the pain increases when tension on the affected tissues increases during forward flexion of the spine. The other lumbar movements are of full range and painless, while straight leg raising is painful and somewhat limited.

These clinical examination findings correspond perfectly with the classic findings of a disc protrusion, so the diagnosis will be missed if the examination is not properly performed. When passive flexion at the hip joint is tested, the buttock sign will be apparent at once.

The local temperature is raised and palpation reveals a tender area just above and behind the greater trochanter. In advanced cases, an abscess may have formed, revealed by a swollen, tense and fluctuating area in the buttock.

Treatment

Treatment consists of ice packs, bed rest and antibiotic therapy. If there is an abscess, the lesion must be drained.

Sometimes the buttock sign and the localized and fluctuating swelling result from an acute haemorrhage after trauma: for example, a fall on the buttocks. Here the history of trauma is informative and the temperature is normal. Aspiration usually leads to complete cure.

Ischiorectal abscess

Occasionally, an anorectal abscess points towards the ischiorectal fossa instead of to the rectal region – an ischiorectal abscess.[58]

Usually, the patient complains of gluteal pain, rather than of local rectal pain, and nothing in the history suggests an infection of rectal origin; the pain is related to movement and posture and not to function of the bowel.

Sitting is impossible. The patient limps badly and even putting the foot to the ground causes considerable pain.[59] The hip is held constantly in slight flexion but further flexion is prevented by increasing pain, as is straight leg raising, indicating the buttock sign. Apart from fever, other toxic symptoms may be present. The abscess may be felt during bidigital rectal examination with the index finger in the rectum and the thumb external.

The treatment is surgical and consists of prompt incision and adequate drainage.[60]

Fractured sacrum

Sacral fractures are associated with pain, swelling, ecchymosis and tenderness on palpation. In the presence of neurological symptoms, the diagnosis is usually not difficult. Neurological damage is not present, however, if the fracture line lies through the ala. Because of the position of the sacroiliac ligaments, the fracture remains stable and the diagnosis is then frequently missed.[61] The patient may ascribe discomfort to local bruising and sometimes continues to be mobile.

In a spontaneous sacral insufficiency fracture in an elderly woman the diagnosis is more difficult.[62,63]

There is pain during walking and standing but also local tenderness during sitting or lying. Coughing and sneezing also hurt. Careful clinical examination discloses the buttock sign. Sacroiliac tests are very painful. During digital rectal palpation of the sacrum, the anterior part is found to be as painful as the posterior, a strong sign of sacral fracture.

Because the sacrum is curved, diagnosis on a radiograph is not always easy. It is therefore advisable to obtain anteroposterior and lateral tomograms.[64]

If the fracture is markedly displaced or associated with neurological deficit, surgical reduction and stabilization must be performed. In uncomplicated fractures, treatment consists of aspiration of the haematoma and rest for 4–6 weeks, although bony union without deformity seems to take place whether the patient rests or not.[65]

Neoplasm at the upper femur

Metastases or primary tumours in the upper femur give rise to increasing pain in buttock and thigh. After a short development of the disease process, the functional disability may be so gross that it prevents the patient bearing weight on the affected side. There is also pain at rest.[66]

Examination reveals the buttock sign. If the lesion lies in the neighbourhood of the lesser trochanter, resisted hip flexion is painful and weak. Marked wasting of the thigh muscles is present in advanced cases. The diagnosis is confirmed by radiography.

Neoplasm in the ilium

Again, the patient complains of increasing gluteal pain, not only during movement (the patient can only hobble with assistance) but also at rest. The buttock sign is present but the other passive movements are of full range and almost painless. Some resisted movements are painful and weak, and marked muscular atrophy is seen. The contrast between the extreme pain and the almost full range of passive movement, in combination with the buttock sign, makes the diagnosis obvious. The radiograph confirms it.

Osteomyelitis of the upper femur

Osteomyelitis is suspected when local symptoms and signs are accompanied by high fever and toxic manifestations. Sometimes, however, the patient is afebrile, and then only complains of local symptoms – increasing and continuous pain in the buttock and thigh. This contrasts with the signs: hobbling with a bad limp, suggesting gross arthritis, but further clinical examination reveals a non-capsular pattern at the hip joint together with a clear sign of the buttock. Early in the evolution it is possible for the radiograph to be negative. If there is any doubt, a bone scan should be obtained.

Septic sacroiliac arthritis

This disease affects young patients. Predisposing factors are immunosuppression, drug addiction and childbirth.[67,68] In addition to demonstrating the positive buttock sign, the sacroiliac distraction test is extremely painful. Fever and general illness are present,[69] together with local tenderness over the joint.

Radiological examination remains negative[70] in the early stage but computed tomography (CT) or bone scan usually confirms the diagnosis.[71]

Loose femoral prosthesis

A sign of the buttock in a patient who underwent a hip arthroplasty in the past may indicate a loose femoral prosthesis. Radiological diagnosis may be difficult and obscured by artefacts.[72]

Aseptic necrosis of the hip

Aseptic necrosis (osteonecrosis, ischaemic necrosis and avascular necrosis are synonymous) of the hip is an osteoarticular disorder characterized by bone marrow ischaemia and death of trabecular bone of the femoral head. As bone repair occurs, weight-bearing bone becomes mechanically weakened and flattened and may eventually collapse. Secondarily, this leads to quickly developing osteoarthritis of the hip. In children it is called Perthes' disease (see online chapter *Hip disorders in children*).

Aseptic necrosis of the hip in adults was first described in the German literature.[73] Two types – post-traumatic (complicating fractures or dislocations) and idiopathic – exist. The latter has been the subject of many experimental and clinical studies over recent decades. All observations show an increasing incidence,[74] although this is probably due to improved diagnostic techniques. The fact that the disease is not uncommon was shown by Streda in 1971,[75] who found that 68% of patients with current osteoarthritis had pre-existent osteonecrosis.

Pathogenesis

It has been accepted for years that non-traumatic osteonecrosis of the hip results from progressive ischaemia, caused by interruption of the arterial supply of the femoral head.[76] Experimental and clinical studies, however, indicate that other mechanisms can induce bone necrosis. The fact that aseptic necrosis occurs most frequently in patients treated with high-dose corticosteroids[77] and in those with a history of alcohol abuse[78] suggests a direct toxic effect on the osteocytes.[79] This results in local inflammatory exudate and increased intraosseous bone marrow pressure. Elevation of intraosseous pressure is transmitted to small venules and capillaries within the bone, causing a decrease in blood flow to the bone. This uncompensated increase in intraosseous pressure is thought to result in irreversible circulatory disturbances and subsequent tissue damage, which further magnifies the initial result.[80] The problem with osteonecrosis is that the radiological evidence only appears months after the process has started: necrosis of bone is only the irreversible end result of severe and prolonged ischaemia. Furthermore, it is not the necrosis itself but the reaction of the tissue to the ischaemia that shows up on a radiograph. For these reasons, a standard radiograph cannot assist in early diagnosis. However, diagnosis and treatment at

Table 47.1 Stages of bone necrosis of the femoral head, adapted from RP Ficat[88]

Stage		Clinical features	Radiographic signs	Bone scan
Early				
0	Preclinical	0	0	Reduced uptake?
I	Preradiographic	+	0	Increased uptake
II	Before flattening of head or sequestrum formation	+	Diffuse porosis, sclerosis or cysts	+
Late				
III	Collapse	++	Flattening Crescent sign Broken contour of head Sequestrum	+
IV	Osteoarthrosis	+++	Flattened contour Decreased joint space Collapse of head	+

an early stage are extremely important if gross damage to the femoral head is to be avoided.

Diagnosis

Early diagnosis of aseptic necrosis is important because the disease occurs in relatively young individuals (average age 20–50 for idiopathic forms) and treatment options for more advanced disease are frequently unsuccessful.

Any sudden and progressive groin pain – eventually spreading down the L3 dermatome – in a patient under the age of 50 should immediately arouse suspicion of the onset of aseptic necrosis, especially if there is a clear discrepancy between marked symptoms (pain) and nearly absent signs (full range of movement in the hip).

The classic radiological techniques (plain radiographs and a CT scan) are inefficient in making an early diagnosis and other methods of investigation should be used.

Bone scan

A bone scan usually shows increased uptake of the radionuclide from the very beginning of the disease – the sensitivity is about 70%.[81] Both sides should be compared.

Magnetic resonance imaging (MRI)

Nowadays, MRI is the most sensitive non-invasive method for the diagnosis of aseptic necrosis, which is diagnosed when a peripheral band of low signal intensity is present on all imaging sequences, typically in the superior portion of the femoral head, outlining a central area of marrow. This peripheral band is most apparent on T1-weighted sequences.[82–85] Because the two hips are frequently involved, it is necessary to image both, not just the symptomatic one. The sensitivity of MRI is between 75 and 100%.

Table 47.2 MRI staging of aseptic necrosis[87]

Class	T1	T2	Definition
A	Bright	Intermediate	'Fat' signal
B	Bright	Bright	'Blood' signal
C	Intermediate	Bright	'Fluid' or 'oedema' signal
D	Dark	Dark	'Fibrosis' signal

Staging

There are two staging classifications of aseptic necrosis, one based on radiographs (Table 47.1) (Ficat and Arlet[86]) and the other on MRI signal intensities[87] (Table 47.2).

The staging of Ficat and Arlet is based on clinical features, radiographic signs and scintigraphy. The accuracy of radiographic staging may be improved by using CT to detect subchondral lucency. However, CT does not depict the earliest marrow abnormalities.

Stage 0

This is a theoretical, preclinical and preradiological stage.

Stage I

The earliest clinical manifestations are sudden groin pain (sometimes referred to the L3 dermatome), often pain at night, and increasing pain on weight bearing.[89] Coughing may hurt.

Clinical examination normally shows a full range of movement. Sometimes there is pain in a non-capsular pattern: external rotation and flexion hurt at the end of range.[90] This could suggest the presence of a psoas bursa or an impacted loose

body. Sometimes a forceful axial and upward blow on the heel also provokes groin pain.

Standard anteroposterior and lateral radiographs are entirely normal but a bone scan shows increased uptake of the radionuclide.[91] The MRI is positive.

Stage II

This early radiographic stage can continue for several months. The clinical symptoms and signs persist or increase. Radiographic changes, although very subtle, appear: slight diffuse sclerosis and slight decalcification, either generalized or in the form of small cysts. Again, the bone scan shows increased uptake and the MRI is positive.

Stage III

There is increasing L3 pain. The patient limps or enters the room leaning on a stick. The clinical examination shows a slight capsular pattern, and forceful axial thrust on the heel provokes the pain.

This stage is characterized by pathognomonic radiographic appearances:

- A 'crescent line', due to a subchondral fracture and a segmental flattening of the femoral head.
- Later, a sequestrum becomes manifest, followed by collapse of the femoral head.

Stage IV

This is the terminal phase of the necrotic process. There is clinical evidence of gross arthrosis. The radiographic picture is that of an arthrosis, superimposed on a deformed femoral head.

MRI staging of aseptic necrosis is based on the signal intensity of the centre of the marrow inside the dark line of necrosis.[87] Radiographically occult aseptic necrosis will generally be depicted on MRI as any of classes A–C. Unlike radiographic staging, MRI classes have little predictive value regarding the prognosis for collapse of the femoral head. However, MRI size and position of the necrotic lesion are related to prognosis.

Treatment

The most important factor in effective treatment of non-traumatic osteonecrosis of the hip remains early diagnosis.[92]

While patients with advanced aseptic necrosis usually end up having hip arthroplasty, some of those whose lesion has been diagnosed early (at pre-collapse stage) have been managed with hip salvage surgery.[93] A variety of drugs have also been used, such as lipid-lowering drugs, anticoagulants, vasodilators and bisphosphonates.[94–96] Their use has been considered on the basis of specific physiological risk factors for osteonecrosis, such as lipid emboli, adipocyte hypertrophy, venous thrombosis, increased intraosseous pressure and resorption of bone.

Early decompression of bone (core decompression) is the treatment of choice in the early stages, before irreversible damage has taken place. The rationale for the use of core decompression is based on the concept that increased intramedullary pressure is involved in the pathogenesis of avascular necrosis. The aim of the proponents of core decompression is to decrease the intramedullary pressure and thus arrest or reverse the process of avascular necrosis before it is evident.[97] Cortical bone grafting can be added to the core decompression in an effort to provide structural support for the subchondral bone and articular cartilage, and to prevent collapse during the repair process. A meta-analysis of 24 reports analysing 1206 hips treated by core decompression with or without cancellous bone grafting revealed an overall clinical success rate of 63.5% (range 33–95%). Less than 33% of the hips required a replacement or salvage procedure during the follow-up period.[98]

In stage III, an osteotomy can be performed to rotate the necrotic or collapsing segment of the hip out of the weight-bearing zone, replacing it with a segment of articular cartilage of the femoral head supported by healthy viable bone. In addition to the biomechanical effect, osteotomy may also reduce venous hypertension and decrease intramedullary pressure.[99]

Symptoms, signs, investigations and treatment of aseptic necrosis of the hip are summarized in Box 47.3.

Stress fracture of the femoral neck

Stress fractures of the femoral neck are not uncommon. The disorder presents in athletes,[100] military recruits[101,102] and the elderly[103] with specific symptoms and findings. However, the diagnosis can be easily missed.

Patients present with unremitting, localized hip and groin pain without a history of significant trauma or unusual increase in daily activity. As in the initial stages of aseptic necrosis of the hip, there is a discrepancy between the obvious symptoms (localized pain and limping) and the minimal clinical signs.

Box 47.3

Summary of aseptic necrosis of the hip

Symptoms

- Age between 30 and 50 years
- Frequently a history of steroid treatment or excessive alcohol intake
- Sudden groin pain or pain in the L3 dermatome during weight bearing
- Sometimes nocturnal pain

Signs

- Walking with a limp contrasts with the normal movements or the slight non-capsular pattern found at the hip
- Upward thrust to the heel sometimes evokes hip pain

Technical investigations

- Early stage: MRI (radiography is not informative)
- Later stages: radiography

Treatment

- Early stage: core decompression and modified rest
- Advanced stage: osteotomy or arthroplasty

There is a non-capsular pattern with full and painless range of flexion and extension but considerable pain at the end of internal and external rotation of the hip. Resisted movements are painless.[104] The initial radiographic features may prove negative.

An early diagnosis of spontaneous stress fracture of the femoral neck may be made when the scintigraphic examination shows increased focal radionuclide uptake.[105] MRI seems to be the most accurate diagnostic tool in the early detection of a femoral neck stress fracture.[106]

The sequelae of undetected spontaneous fractures are subcapital fracture with displacement, angular deformity and avascular necrosis of the femoral head.

Treatment depends on radiological differentiation of the lesion. A transverse stress fracture is potentially unstable and the recommended treatment is prompt internal fixation. The compression stress fracture has a benign prognosis. Restriction of weight bearing for a few weeks is usually sufficient to relieve symptoms. Prophylactic internal fixation should be considered only in cases of significant weakening of the trabecular bone mass.

Internal derangement of the hip

Internal derangement of the hip is not uncommon but is frequently overlooked. It is caused by a loose body, a torn portion of the labrum or impingement of a small focus of synovitis.

Cyriax[5] suggested that a small piece of exfoliated articular cartilage, secondary to trauma or osteoarthrosis, becomes loose in the joint. When the fragment lies inside the capsular fold, level with the femoral neck, it is harmless and painless, but when it is pinched at the acetabular edge, sudden twinges result.

During arthroscopy, Dorfmann and Boyer sometimes found a small focus of synovitis on the anterior and anteroinferior aspects of the femoral neck in patients with symptoms of internal derangement. They hypothesized that this was probably due to impingement of the capsule by the psoas muscle tendon.[107]

Recent arthroscopic studies suggest that most internal derangement may be the result of impingement of acetabular labral tears.[108] The latter are caused by repetitive contact between a widened femoral neck and the acetabular rim.[109] Most tears are radial flaps located in the anterior section of the acetabulum and are more tag-like than massive.[110] Anatomical studies have shown that the labrum is richly innervated with free nerve endings, capable of nociception. This may cause a direct painful response from the nipped flap.[111]

Symptoms

The pathognomonic sensation in internal derangement of the hip is the presence of a 'twinge' – a sharp, severe pain in the groin or the trochanteric area. It appears suddenly and unexpectedly during ordinary walking or descending stairs. The patient feels a pain shooting down the thigh to the knee and the leg feels as if it 'might let the patient down'. Sometimes the pain disappears immediately or the patient has to stand on the good leg for a while.[112] This painful twinge may be repeated on each step, some 100 metres later or not at all for the first few weeks. If patients have many twinges a day, they will be severely disabled, because they happen at any moment without warning and nothing can be done to prevent them.

Signs

There is a difference in signs between an impacted loose body in a normal joint and the presence of a loose fragment in an osteoarthrotic joint.

Examination of a joint not complicated by osteoarthrosis shows a full range of movement, with some discomfort at the end of one or two movements – as a rule, external rotation and flexion. As these two movements are also painful in psoas bursitis (see later), the differential diagnosis will be supplied by the existence of twinges and the slightly different end-feel. Some authors report successful diagnosis of impingement lesions with the so-called Thomas test.[113,114] This involves flexion and external rotation of the hip, then allowing the extremity to abduct. The hip is then moved into extension, internal rotation and adduction. A positive test result is indicated by a palpable or audible click and the production of pain.

The commonest occurrence of a loose body, however, is as a complication of osteoarthrosis. Unfortunately, these cases are often missed because the twinges mentioned by the patient are usually ignored, all attention being given to the radiograph. Examination not only shows the capsular pattern with the usual hard end-feel but also one or two movements may have a softer end-feel and cause pain. Sometimes only the capsular pattern of osteoarthrosis is present, and then the diagnosis will rely entirely on the characteristic history of twinges.

Current modalities for imaging the hip joint, including arthrography and MRI, are poor at directly identifying a labral tear (sensitivity is between 13 and 24%).[115] Indirect MRI findings of labral tears include a large α angle, an osseous bump formation at the femoral neck, a deep acetabulum and posteroinferior or anterosuperior cartilage lesions.[116]

Treatment

The ultimate proof of the existence of a loose body is the immediate and usually lasting improvement after a manipulative reduction, which means the fragment has been moved to a position within the joint where it no longer becomes 'nipped' on movement.

Technique: reduction of a loose body – 1

The manipulation is a combination of three movements conducted simultaneously: a movement towards extension during strong traction, in combination with medial or lateral rotation.

The patient lies supine on a low couch. An assistant presses on both anterior superior iliac spines in order to fix the pelvis firmly to the couch during the whole procedure (Fig. 47.12). To prevent local pain, a thick layer of foam may be placed between the hands and iliac crests. The pressure on the iliac

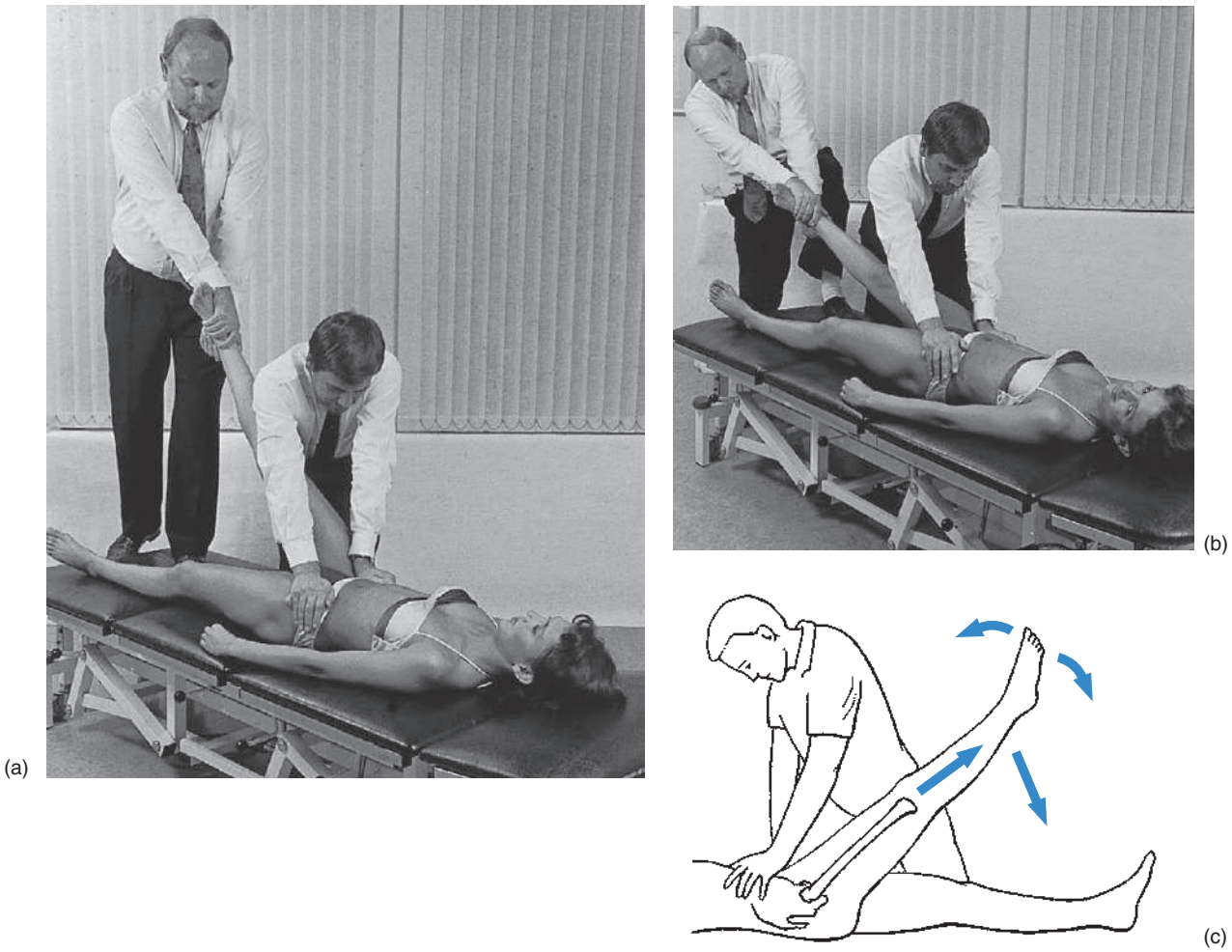


Fig 47.12 • Reduction of a loose body in the hip joint – technique 1: start (a) and end (b) of the manipulation.

spines should be directed downwards and cranially to withstand the force of the traction.

The manipulator stands on the end of the couch, holding the patient's ankle. From this point, the procedure depends on the direction of rotation required.

Medial rotation

The contralateral hand encircles the heel. The other hand is placed at the distal side of the leg, level with the malleoli (Fig. 47.13). This position of the hands is vital in protecting the ligaments of the ankle when rotation is performed. The manipulator raises the outstretched leg to 70 or 80° and leans backwards, inducing as much traction as possible. As soon as the muscles are felt to relax, the manipulator steps gradually off the couch, meanwhile rotating the leg with a jerk to full medial rotation and back to the neutral position as the leg is extended. This manœuvre is repeated three or four times during extension.

Lateral rotation

The ipsilateral hand is used to grasp the heel. The foot is held at a right angle and the other hand encircles the medial border of the foot (Fig. 47.14). The manipulator leans backwards after

Fig 47.13 • Position of the hands during medial rotation.

Fig 47.14 • Position of the hands during lateral rotation.

having lifted the patient's leg to about 70°. Again, when the muscles relax, a movement backwards is made and then downwards, meanwhile rotating the leg laterally. At the end of the rotational movement, a sharp jerk is added and the leg is turned back into the neutral position. This rotational movement is performed three or four times as the leg is extended.

As a rule, manipulation in the most comfortable direction is done first. After each manœuvre, the joint is re-examined to find out whether there is an improvement. The most effective rotation is then carried out several times during the same session.

Attention should be paid to two important technical points:

- The rotation must have the largest possible amplitude. Thus, starting at least from the neutral position, the leg has to be rotated until the end-feel is reached. It is then rotated back to the initial position, to restart the same procedure.
- Traction should not be lost during extension. Care should be taken to maintain strong traction throughout the whole manipulation until the joint is extended completely. Therefore, the manipulator keeps one foot on the couch, in order to maintain sufficient traction.

Technique: reduction of a loose body – 2

If the previous manipulation did not reduce the subluxated loose body, an alternative technique should then be tried, applying stronger traction and rotation, which can be achieved by using the lower leg as a lever.

The patient adopts a supine-lying position on a low couch. As in the previous manipulation, an assistant holds the pelvis down on the couch, putting all the body weight on the anterior superior iliac spines. The manipulator places the contralateral foot on the couch, just beyond the patient's buttock, and puts

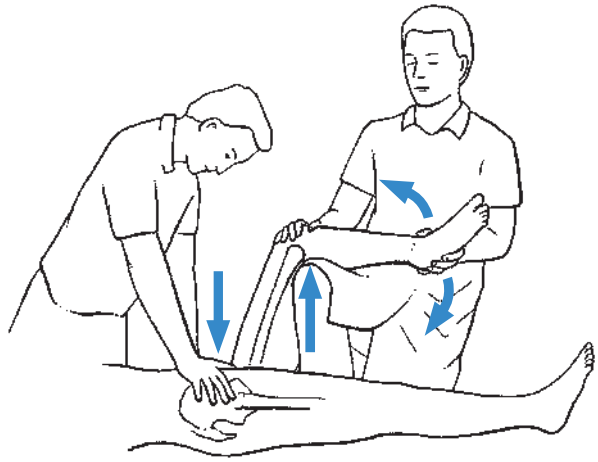


Fig 47.15 • Reduction of a loose body in the hip joint – technique 2.

the back of the patient's bent knee over the thigh. The contralateral hand holds the knee and the other hand grasps the ankle (Fig. 47.15).

Considerable traction on the hip can now be achieved when the lower leg of the patient, used as a lever, is pushed down. This levers the buttock off the couch. In the meantime, the assistant applies as much counterpressure as possible. At the moment the muscles are felt to relax, the femur is rotated by means of the lever provided by the patient's leg. At the end of range, a quick rotational thrust is added. The direction of rotation can be chosen at random but usually the rotation found to be the most beneficial during the previous manipulation is done first.

Results

This manipulation can have spectacular results. The patient no longer has the repeated twinges that make walking so difficult. This happy outcome can be achieved in two-thirds of patients complaining of twinges at the hip. The osteoarthrosis itself, if present, will not be changed but it seldom causes problems other than a little discomfort after a long walk. As a rule, two or three manipulation sessions will be sufficient. If no

improvement is achieved by then, defeat must be conceded and the patient referred for arthroscopy.

Warning

Because the leverage of both traction and rotation is considerable, the manipulation should *never* be performed in elderly people, for fear of fracture of the femoral neck.

Psoas bursitis

The psoas bursa is one of the largest bursae in the human body.[117] Located between the lesser trochanter, the musculotendinous portion of the iliopsoas muscle and the anterior capsule of the hip joint, it can be up to 7 cm long and 4 cm wide.[118]

As the bursa is derived from the second and third lumbar segments, pain is usually felt in the groin, anterior thigh, knee and leg. It appears during walking and during specific movements: for example, crossing the legs.[119] Untreated, the disorder can persist for years.

The lesion is generally overlooked and the symptoms are usually blamed on the slight arthrosis visible on radiograph rather than the (invisible) psoas bursa.[120] Clinical examination, however, shows a non-capsular pattern: lateral rotation is painful and the end-feel soft, and flexion is slightly painful at the end of range. Sometimes there is also pain at the end of extension or adduction.[121] An accessory test is provided by passive adduction in flexion, which is the most painful movement because it squeezes the bursa. This test also provokes considerable stretching of the sacroiliac ligaments and tissues in the buttock, so it is essential to ascertain that the reproduced pain is in the groin and is not some vague sensation of pulling in the buttock. Resisted movements are strong and painless. The bursa may be palpably enlarged, as is sometimes the case in rheumatoid conditions.[119,122,123]

Differential diagnosis should be made from a loose body in the hip and early aseptic necrosis. In the former the patient complains of sudden twinges and in the latter there will often be a gross discrepancy between the functional incapacity and the moderate signs. Ultrasonography is the best confirmatory diagnostic test.[124]

The diagnosis is also confirmed when a diagnostic infiltration with local anaesthetic is found to abolish the clinical features. This injection, perhaps repeated once or twice, is very often therapeutic too.[125]

Technique: infiltration

The patient adopts a half-lying position on the couch, keeping the legs outstretched, which relaxes the tissues at the groin. Three landmarks are identified: the anterior superior spine of the ilium, the femoral artery and the greater tuberosity. A perpendicular line is drawn from the anterior superior spine, and a horizontal line 5 cm under the tip of the greater tuberosity. The needle is inserted at the crosspoint, which is well lateral to the inguinal vessels.

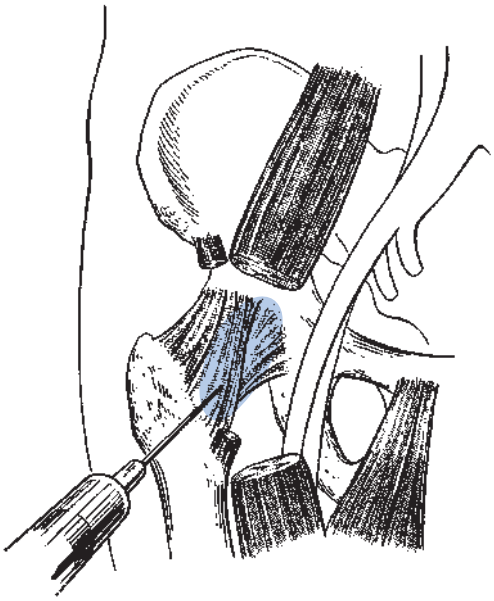


Fig 47.16 • Infiltration of the psoas bursa.

A needle 7 cm long is fitted to a 50 mL syringe, filled with procaine 0.5%. The needle is inserted at the identified spot, and thrust in at a 45° medial and upward direction (Fig. 47.16).

At the same time, the free hand is used to palpate the femoral artery continuously as a safeguard to avoid the inguinal vessels. If the tip of the needle hits bone, it lies near the junction of the femoral head and neck; this is where the infiltration is given, using the classic technique of withdrawals and reinsertions. If the needle pierces the capsule, it must be withdrawn a little until the drug can be placed without resistance.

Results

Diagnostic infiltration often has a lasting therapeutic result, regardless of the duration of symptoms. If necessary, the infiltration can be repeated once or twice, at weekly intervals. If no lasting relief is afforded and the injection only acts

temporarily, 50 mg of triamcinolone should be injected at the same site at the next attendance.[126]

Haemorrhagic psoas bursitis

As in haemorrhagic bursitis at the shoulder, this condition sometimes occurs in the elderly. The patient states that the hip was hurt during a sideways slip of the leg, which was immediately followed by extreme pain at the front of the thigh and knee. Spontaneous haemorrhage complicating a pigmented villonodular synovitis of the bursa has also been reported.[127]

Examination shows a gross non-capsular pattern, with up to 90° limitation of flexion and gross limitation of lateral rotation and extension, but almost a full range of medial rotation.[128] Resisted movements are full-range and painless. Sometimes a tense, tender swelling can be palpated in the groin. Aspiration confirms the diagnosis and is also the required treatment.

Gluteal bursitis

This syndrome, also referred to as 'greater trochanteric pain syndrome', is one of the most frequent causes of pseudoradicular pain in the limb.[129] The condition most frequently occurs in late-middle-aged females and shows little tendency to spontaneous cure.[130] Patients are usually in their forties or fifties and complain of pain at the gluteal or tronchanteric area, spreading to the outer or posterior thigh and down to the calf muscles and outer malleolus.[131] Unlike the pain caused by a disc lesion, the symptoms are not related to sitting but to walking, prolonged standing or transitioning to a standing position.[132] They are also exacerbated by sitting with the affected leg crossed and by climbing stairs.[133] The patient will not mention the twinges that are so typical of an impacted loose body but may state that a particular movement causes a sharp pain down to the leg. Sometimes the patient has nocturnal pain when lying on the affected side. Sitting with the painful leg crossed over the other also hurts.[134] Coughing is painless. The combination of long-standing leg pain, related to hip movements and without symptoms typical of disc lesions, suggests the existence of gluteal bursitis.[135]

There is a full range of movement but some movements hurt in a non-capsular way at the end of range. The end-feel is normal. Some resisted movements may be painful because they squeeze the tender bursa. A typical pattern is pain on passive external rotation and passive abduction and resisted external rotation or resisted abduction.[136,137] Pain on resisted hip movements may be explained by compression of the bursa. However, recent histopathologic findings show that tendinopathy and bursa pathology may coexist in greater trochanteric pain syndrome.[138]

Most of the affected bursae are located near the greater trochanter and between the gluteus maximus and gluteus medius (Fig. 47.17).

Localization depends on the findings on palpation, although sometimes the approximate area can be deduced from the general examination: when full passive internal rotation together with resisted external rotation causes pain, the bursa at the insertion of the piriformis is affected; pain on passive abduction and passive flexion, together with resisted abduction, indicates that the bursa between the gluteus medius and gluteus maximus is at fault.[139] Caution is required in drawing conclusions from palpation. Because the gluteal region is an awkward area and the affected tissues lie deeply, tenderness can be elicited in a normal buttock if the pressure applied is considerable. It is therefore always wise to compare the unaffected side with the affected one.

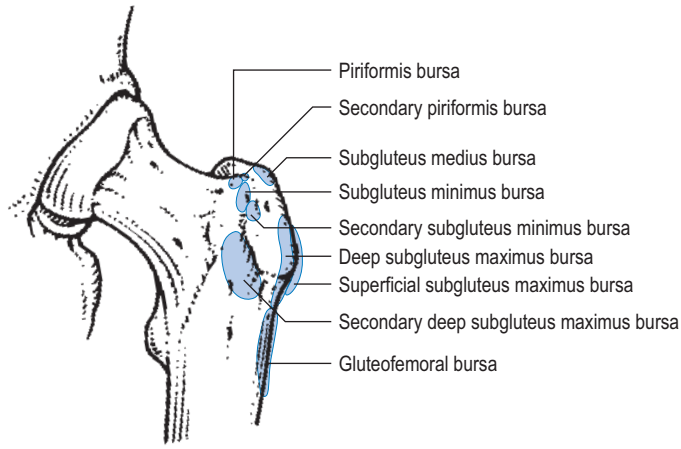


Fig 47.17 • The bursae at the greater trochanter.

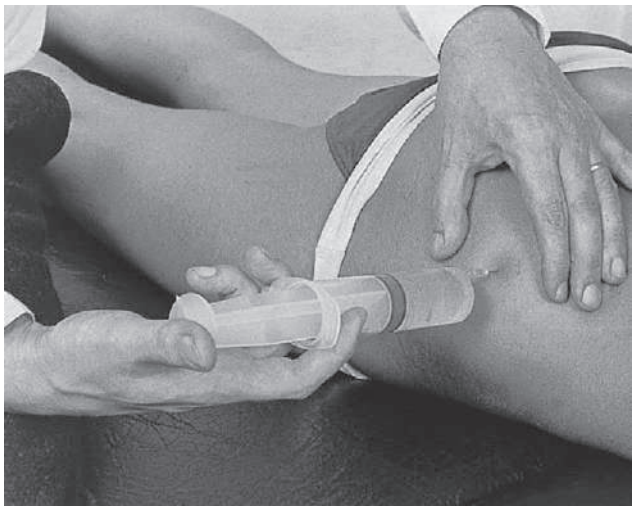
During the last few decades there has been increasing use of ultrasonography and MRI for visualizing lesions of the greater trochanteric area. It should be mentioned, however, that several studies identified gluteal abnormalities in asymptomatic hips,[140–142] sometimes in as many as 60%.[143] It is therefore unwise to rely entirely on the result of these tests to establish the diagnosis.[144]

The diagnosis is settled by local anaesthesia. If, after infiltration, the movements known to hurt are tested again and are no longer painful, the right point has been found.[145] This diagnostic injection often has a lasting result; therefore the patient should re-attend after a week for re-examination. If there is considerable and lasting relief, the injection is repeated two or three times at weekly intervals. If there is no therapeutic result, 50 mg of triamcinolone is injected in the same site. This injection may also have to be repeated once or twice but this time at an interval of 2 weeks.[146] Sometimes an intractable case is encountered; though injections relieve the symptoms for 2 or 3 weeks, the latter subsequently recur; as operative exploration of the buttock is a very difficult procedure and rarely effects a cure, it is best to admit defeat and hope a spontaneous remission will take place.[147]

Technique: injection – 1

When the patient has evidence of a highly localized bursitis, the lateral approach is used (Fig. 47.18). The patient lies prone and the physician sits or stands on the affected side. A 7 cm needle is fitted to a syringe containing 50 mL of procaine 0.5%. The tender area is localized and the needle inserted horizontally until it reaches the ilium. It is then withdrawn by 1 or 2 cm and a large area is infiltrated using the classic technique, employing withdrawals and reinsertions at different angles.

(a)



(b)

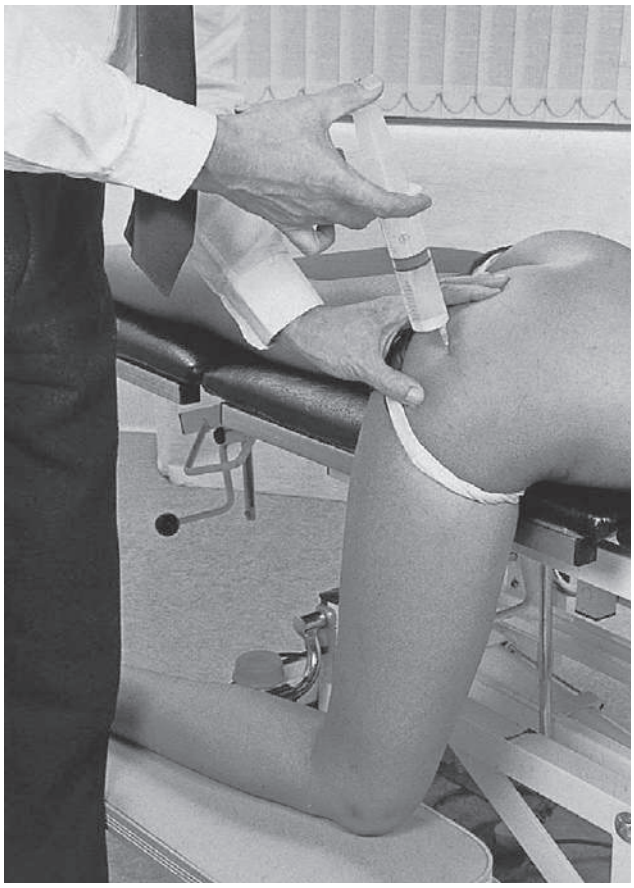


Fig 47.18 • Infiltration of a gluteal bursa by technique 1 (a) and technique 2 (b).

Technique: injection – 2

When the bursitis is located at the upper or inner edge of the trochanter, this alternative technique must be used. The patient lies prone on a high couch but with the affected thigh hanging vertically downwards over the edge. In order to support the leg, a low support is placed under the bent knee. Slight adduction of the hip is preferable in order to make the inner portion of the trochanter more accessible. In this position, it is easy to palpate the upper part of the trochanter from above.

The tender spot is identified. The needle is inserted from above and medially, and thrust downwards until the tip touches bone. It is then slightly withdrawn and the affected area is infiltrated all over with 50 mL of procaine 0.5%.

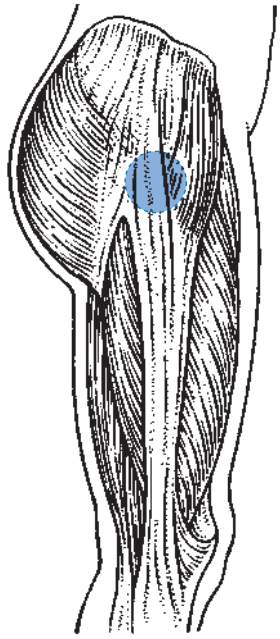


Fig 47.19 • Location of the trochanteric bursa.

Trochanteric bursitis

The trochanteric bursa (Fig. 47.19) lies at the lateral aspect of the trochanter tip, between the iliotibial tract and the bone, level with the insertion of the gluteus medius and the tensor fasciae latae. It can become inflamed after a local blow,[148] although trochanteric bursitis may also result from overuse in long-distance runners.[149] The incidence of trochanteric bursitis peaks between the fourth and sixth decades of life and is more common in women (male : female ratio 1 : 10).[150]

The symptoms are increasing pain at the trochanteric area, spreading down to the lateral aspect of the knee, during walking or running. Walking upstairs is most painful. Occasionally, lying on the affected side is also painful and can disturb sleep.

Clinical examination shows a non-capsular pattern: external rotation is very painful and sometimes limited, with a soft end-feel; however, when external rotation is retested with the hip and the knee extended, there is no pain at all. Passive abduction may also be painful. The resisted movements are painless, except sometimes abduction.[151] Palpation reveals a tender area at the lateral aspect of the trochanter.

Treatment consists of infiltration with procaine.

Technique: infiltration

The patient lies prone; the tender spot is identified and the needle (7 cm long) inserted locally. It pierces the thick structure of the iliotibial tract before hitting bone. The needle is

Table 47.3 Differential diagnosis of non-capsular patterns of the hip

Disorder	Diagnosis	Treatment
Major disorders (all have a positive buttock sign)		
Septic bursitis	Intramuscular injection? Raised temperature Localized swelling	Antibiotics
Ischiorectal abscess	Sitting is not possible Raised temperature Rectal examination	Surgery
Fractured sacrum	Bilateral buttock sign Positive sacroiliac tests Radiograph	Rest
Neoplasm at upper femur	Increasing/gross disability Painful and weak resisted flexion Radiograph/bone scan	
Osteomyelitis of ilium	Increasing pain Normal rotations Radiograph/bone scan	Antibiotics
Osteomyelitis of femur	Discrepancy between symptoms and signs Radiograph/bone scan	Antibiotics
Septic sacroiliac arthritis	Temperature Positive sacroiliac tests Bone scan	Antibiotics
Minor disorders		
Loose body	Twinges Often pain at the end of flexion and lateral rotation	Manipulation
Loose body complicating osteoarthritis	Twinges Painless capsular pattern Some movements painful in a non-capsular pattern	Manipulation
Aseptic necrosis of the hip	Corticosteroid treatment or excessive alcohol intake? Sudden pain in the groin/nocturnal pain/pain during coughing, sneezing Discrepancy between symptoms and signs Early stages: Slight noncapsular pattern/pain during axial compression Diagnosis by bone scan/MRI or functional examination of bone Later: gross osteoarthrosis/radiological evidence	Early diagnosis essential Early stage: core decompression Later: osteotomy or hip arthroplasty
Psoas bursitis	Pain at the anterior aspect of thigh Full range in a non-capsular pattern Painless resisted movements	Infiltration with procaine or triamcinolone
Haemorrhagic psoas bursitis	Elderly patient/blow or fall Sudden pain in groin and thigh Limitation of flexion to 90° Echography	Aspiration
Gluteal bursitis	Pain at posterolateral aspect of buttock and thigh Full range with some movements painful at the end of range Some resisted movements hurt Local tenderness	Infiltration with procaine or triamcinolone

Continued

Table 47.3 Differential diagnosis of non-capsular patterns of the hip—cont'd

Disorder	Diagnosis	Treatment
Trochanteric bursitis	Trochanteric and lateral thigh pain Passive external rotation painful and limited Local tenderness	Infiltration with procaine or triamcinolone
Ischial bursitis	Localized pain on sitting, immediately relieved when standing up Completely negative examination of lumbar spine and sacroiliac and hip joint Local tenderness	Infiltration with triamcinolone
Psychogenic pain	Limping in internal rotation Multiple inconsistencies	

partly withdrawn and the large area between bone and tendinous tissue is infiltrated in the classic way, first with 20–30 mL of procaine 0.5% to prove the diagnosis. If no lasting benefit results, 50 mg of triamcinolone is substituted.

A condition similar to acute subdeltoid bursitis can occur in the trochanteric bursa. The patient complains of serious and rapidly increasing pain at the trochanteric area. Clinical examination shows a non-capsular pattern, with limited external rotation and an extremely tender trochanteric area. The radiograph often reveals calcification.[152] Treatment consists of local infiltration of the whole bursal wall with triamcinolone, which, as in acute subdeltoid bursitis, affords immediate and lasting relief.

Ischial bursitis

The ischial bursa is painfully squeezed between the ischial tuberosity and the hard surface of a chair during sitting. Because the condition is not very common, it will not be the first thing to occur to the clinician in the presence of ischial pain. When a patient complains of pain in the buttock, coming on during sitting and easing as soon as he or she is standing, the first condition to come to mind is a disc lesion at a low lumbar level. However, in ischial bursitis, the patient experiences pain as soon as the buttocks touch the chair, whereas in a discodural lesion the pain gradually increases during sitting or the moment the patient stands again. If the routine lumbar, sacroiliac and hip examinations are negative, this condition should be considered.[153] The only clinical finding is local tenderness at the ischial tuberosity. The diagnosis can be confirmed by introducing some local anaesthetic but the injection seldom gives lasting relief. Treatment consists of one or two infiltrations with 20 mg of triamcinolone and avoidance of further compression.

Psychogenic pain

Sometimes pain in the hip or buttock does not have an organic basis. It is possible for psychogenic pain to make the hip almost functionless. The label 'hysteria' is given to the condition of a patient who has a firm belief in the reality of the symptoms. If the patient is faking pain and disability, the term 'malingerer' is used.

Psychogenic pain affecting the hip, just like the other, more organic lesions at the hip joint, has a specific and typical pattern. The patient walks in with a remarkable gait: the hip is fixed in internal rotation, which is completely the reverse of the fixation in external rotation of a capsular pattern. Suspicion arises immediately this type of gait is seen and the patient must be subjected to a thorough examination from the lumbar spine to the toes. A good routine is to start with the examination of movements not relevant to the hip – ankle and foot. Few patients with psychogenic pain will be able to resist the clear invitation to allege hip pain when several tests of, for example, the foot are performed. When, finally, the hip is examined, other multiple inconsistencies will be revealed: passive hip flexion is limited to 90° but rotations are of full range, or there is complete weakness of the psoas muscle in the supine-lying position, although the patient can step off the couch without elevating the limb manually. If a movement is found to be grossly limited, sustained pressure in the direction of limitation will, in the end, afford a full range of movement with a normal end-feel, proving that the limitation was voluntary.

The differential diagnosis of non-capsular patterns of the hip is summarized in Table 47.3.

Access the complete reference list online at **www.orthopaedicmedicineonline.com**

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48

Disorders of the contractile structures

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Pain in the buttock usually results from a lesion of the lumbar spine, the hip joint or the sacroiliac joint. Muscular lesions are very uncommon. Even if resisted movements elicit pain in the buttock, gluteal bursitis is more likely to be the cause, especially if one or more passive movements also elicit the pain.

Pain in the groin more often results from a tendinous or muscular lesion. It is good to remember, however, that groin pain may also result from a lesion of the lumbar spine, the hip joint or, uncommonly, the sacroiliac joint. Also, a number of intra-abdominal pathological conditions, such as appendicitis, gynaecological disorders and inguinal or femoral hernia, may cause groin pain (see online chapter *Groin pain*).[1]

Resisted flexion

This test primarily examines the psoas muscle. However, a strain or weakness may be obscured because of active contraction of other synergistic muscles such as the rectus femoris, sartorius, tensor fasciae latae and some of the adductors.

Pain

If resisted flexion is strongly opposed and is painful, the following conditions should be considered:

- Tendinitis of the:
  - Psoas
  - Rectus femoris
  - Sartorius
- Obturator hernia.

Tendinitis of the psoas

Tendinitis of the psoas is rare. It may result from an acute hyperextension of the hip or from an overuse injury.[2–6] Iliopsoas impingement is also an uncommon cause of pain after total hip replacement.[7] The lesion is always located in the femoral triangle and thus accessible to the palpating finger. It can be identified just below the inguinal ligament between the pulsating femoral artery medially and the sartorius muscle laterally.

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Treatment

Deep transverse friction is very effective.[8] Sonography-guided iliopsoas peritendinous injections with steroid have also been proposed as a safe and effective method of treatment.[9]

Deep friction

The patient sits upright on the couch with the hip joint in 90° of flexion and the knees extended – with the hip extended in the supine position, it is not possible for the finger to penetrate deeply enough because of the taut overlying tissues. The therapist sits at the patient's side facing the thigh, with the index and middle fingers placed at the painful tendon in the femoral triangle, just lateral to the femoral artery and medial to the sartorius muscle. The thumb is placed at the outer part of the hip and used as a fulcrum (Fig. 48.1). The transverse movement of friction is imparted by alternating flexion and extension of the wrist and elbow, together with some adduction–abduction movement at the shoulder. In acute cases, treatment can be started the day after onset. However, really deep friction should not last more than 1 minute and is repeated daily, with a gradual increase in the treatment time. In the second week, treatment is performed on alternate days. It is then carried out deeply throughout and for about 15 minutes. A good result is to be expected in 2 weeks. Chronic strain requires 15 minutes of friction, two or three times weekly, depending on the result of each session. A lesion that has persisted for years will respond to 6–8 sessions of deep transverse friction. Treatment is very painful but, in our opinion, there is no alternative. Finding the exact point is not easy and requires a good understanding of local topographical anatomy.

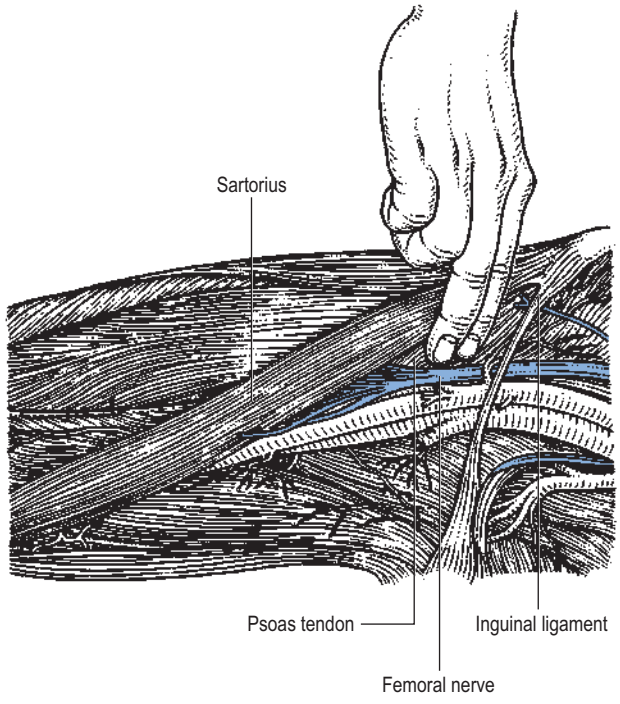

Fig 48.1 • Deep transverse friction to the psoas tendon.

Tendinitis of the rectus femoris

Resisted flexion is painful, especially if the test is executed with an extended knee (active straight leg raising). Resisted extension of the knee in prone lying position is also painful. The lesion is discussed in 'Pain on resisted extension of the knee' (see Ch. 54).

Lesions of the sartorius

Lesions of the sartorius muscle are rare and typically occur in soccer players. Resisted flexion of the hip is painful, especially if some external rotation with a flexed knee is added to the movement. Palpation reveals the localization of the lesion, which is either at the tenoperiosteal insertion or 1 or 2 cm more distal in the tendon. Treatment consists either of deep transverse friction or, if the lesion is tenoperiosteal, of one or two infiltrations with triamcinolone.

In young athletes (aged 15–18) who are skeletally immature, an avulsion fracture of the anterior superior spine of the ilium is more common than a muscle strain or tendinitis (see 'Pain and weakness').[10]

Obturator hernia

This lesion is principally found in thin, elderly women, often with a history of recent weight loss, obstipation or chronic respiratory disease. The patient complains of numbness or pins and needles, which may eventually culminate in intense pain at the anterior and medial side of the thigh down to the knee. These symptoms result from compression of the obturator nerve at the obturator foramen by a prolapsed fold of peritoneum. Absence of the adductor reflex test is a sign of involvement of motor conduction of the same nerve.[11] Pain on resisted hip flexion is explained by pressure exerted by the psoas on the hernia. The differential diagnosis can be made when resisted flexion becomes negative after the patient has been in the Trendelenburg position for upwards of 2 minutes; the effect of gravity is to reduce the prolapse, which is no longer painfully squeezed during active contraction of the psoas.

Pain and weakness

Pain and weakness on resisted flexion can be present in the following conditions.

Avulsion fracture of the anterior superior iliac spine

Avulsion fractures occur more commonly in skeletally immature athletes than in adults because young patients' tendons are stronger than their cartilaginous growth centres. The same stress that causes a sartorius tendinitis in an adult can cause an avulsion fracture of the anterior superior iliac spine in an adolescent. The fracture does not become widely displaced because of the surrounding thick periosteum.

The lesion is well known in young sprinters, soccer players and jumpers.[12] While running, the subject feels a sudden painful click in the groin and upper part of the thigh. From that moment further activity is impossible and the athlete leaves the track with a limp; even walking is painful.[13,14] On examination, resisted flexion and resisted lateral rotation of the hip and resisted flexion of the knee are all painful. Palpation reveals ecchymosis and palpable tenderness at the anterior superior iliac spine where the sartorius muscle is attached. Radiography shows slight separation of the iliac spine. Spontaneous recovery is the rule and takes 2–3 weeks. During this period, however, total bed rest is not necessary. Movement should be permitted to the limits of pain but return to sports activity should be allowed only from the time that clinical examination becomes fully negative.

Avulsion fracture at the apophysis of the lesser trochanter

This may be seen in schoolboys and young athletes. There is no history of sudden onset because the lesion appears to be caused by overuse.[15] The complaint is of groin pain during walking. Clinical examination shows a normal range of passive movement but resisted flexion is weak and provokes pain. This should be reason enough to obtain a radiograph, which shows separation at the lesser trochanter. In most instances 2 or 3 weeks' bed rest in a half-sitting position will be enough for recovery to take place. Once the patient can walk without pain, standing is allowed.

It is important to remember that avulsion fractures of the lesser trochanter in adults are almost always the result of metastatic bone disease.[16]

Abdominal neoplasm

Infiltration of an abdominal neoplasm into the psoas muscle, although rare, is another cause of considerable weakness of hip flexion and pain in the iliac fossa.

Metastasis in the upper femur

Weakness and pain in the thigh will be accompanied by marked articular signs in the hip joint and probably a 'buttock sign' (see p. 637).

Painless weakness

Lumbar root palsy

Painless weakness is a major sign in the rare condition of a second lumbar root palsy in a second lumbar disc protrusion. In a third lumbar root palsy, it is less obvious and is also accompanied by weakness of the quadriceps.

Psychoneurosis

Weakness of hip flexion is also a common finding in psychoneurotic patients complaining of pain in the lower back or thigh. The diagnosis can only be made if this sign is accompanied by other inconsistencies in the history and clinical examination (see online chapter *Psychogenic pain*).

Serious non-specific disorder

Because discoradicular conflicts at the second lumbar level almost never occur, painless weakness of hip flexion should always arouse suspicion of a serious non-specific disorder (see p. 534), such as inflammatory disease or neoplasm.

Resisted extension

Pain

Pain on resisted extension hardly ever has anything to do with the gluteus maximus, in which a lesion occurs only after a direct blow and recovers spontaneously within a few days. However, a contracting gluteus maximus may compress an inflamed gluteal bursa and thus indirectly provoke pain in an inert structure (transmitted stress). Pain after prolonged contraction of the gluteal muscles indicates a particular form of intermittent claudication.

If there is hamstring tendinitis or sprain of the sacrotuberous ligament, resisted flexion of the knee is then also painful (see 'Resisted flexion of the knee', p. 658).

Inflamed gluteal bursa

An inflamed gluteal bursa is a much more frequent cause of pain on resisted extension, especially if one or more passive movements also elicit the pain (see 'Gluteal bursitis', p. 646).

Buttock claudication

Claudication is the symptomatic expression of peripheral artery disease in the leg. It is confined as pain (aching, heaviness, cramping or burning) that is produced with a similar level of walking, disappears after several minutes of standing and occurs at the same distance once walking has resumed.[17] Classically, it appears in the calf and/or thigh, but sometimes is confined to the buttock region only and is then caused by a proximal arterial stenosis (bifurcation of the aorta, common iliac, internal iliac and gluteal arteries).[18] The essential history is that of a buttock pain that forces the patient to stop walking, improves in a minute or two and reappears when the patient starts walking again. The routine clinical examination is completely negative. An additional test – active extension of the hip in prone-lying position – may reproduce the buttock pain (see p. 625). The usual reduction in femoral pulse may be absent if the stenosis is located on the hypogastric or gluteal artery and there is no substantial damage to the aorta–iliac

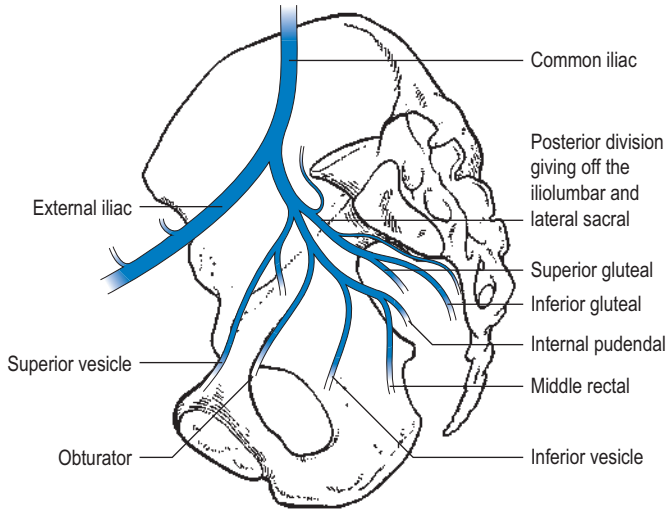

Fig 48.2 • Locations of a proximal arterial stenosis.

axis.[19] Arterial stenosis is confirmed by Doppler ultrasound or arteriography (Fig. 48.2).[20]

Painless weakness

Painless weakness on resisted extension is indicative of a first sacral root palsy. In the exceptional instance of an extensive paralysis of the gluteus maximus, an 'extensor' or 'gluteus maximus' lurch may be found; the patient has to thrust the trunk posteriorly to maintain hip extension during walking.

Resisted adduction

Pain

Adductor longus

Pain on resisted adduction that is localized to the groin usually points to the adductors. While the adductor longus, adductor magnus, adductor brevis and pectineal muscles are all adductors of the hip, of these the adductor longus is most often injured in sports.[2,21–24]

The mechanism of an acute injury is usually that of a sharp cutting movement, which causes a forceful eccentric contraction of the muscle. Acute lesions are common in soccer players and often result from a sudden slip on a muddy field, which stretches or tears the muscle or tendon fibres.[25] Alternatively, the lesion starts as an overuse phenomenon, such as repetitive abduction and adduction movements of the leg in the skating stride or in defence movements in basketball. It also occurs in high-jumping athletes and ballet dancers[26] and has been described in middle-aged cricket bowlers and ice-hockey players.[27,28] Lastly, it is a well-known lesion ('rider's sprain') in horse sports.

Pain is provoked mainly on resisted adduction but full passive abduction may also hurt because it stretches the injured muscle fibres.[29] Imaging procedures are usually unnecessary. However, ultrasound, although operator-dependent, can confirm the diagnosis.[30]

There are three possible locations of the lesion:

- tenoperiosteal
- musculotendinous
- proximal extent of the muscle belly.

The exact site of the lesion is found on palpation and is more often musculotendinous, a few centimetres below the pubic bone, than tenoperiosteal. Exceptionally, the uppermost part of the muscle belly itself is at fault.

Differential diagnosis

Although pain in the groin during resisted adduction usually indicates a lesion of the adductor longus, this is not always so. Strong adduction indirectly pulls on bones and ligaments of the pelvic ring and, if a pathological condition is present in this region, the transmitted stress causes pain. A detailed discussion of pubic lesions can be found in the online chapter, *Groin pain*. The following three lesions are the most important.

Fracture or neoplasm in the pubic bone

Palpation of the adductor muscles reveals nothing, whereas a radiograph establishes the presence of a lesion.[31]

Osteitis pubis (periostitis at the pubic symphysis)

This occurs in soccer players and race walkers as the result of repeated shearing of the pubic bones at the symphysis.[32] Palpation of the adductor muscles reveals nothing but the pubic symphysis is very tender (see online chapter *Groin pain*).[33]

Lesions of the sacroiliac joint

Sacroiliitis or sacroiliac strain may also be responsible for painful resisted adduction at the hip, caused by distraction of the ilium from the sacrum. Usually, the pain is felt in the buttock and attention is immediately directed to the sacroiliac joint. However, in some instances, pain is felt in the groin or inguinal region and diagnosis can then be more troublesome.[34]

Treatment

Deep transverse friction is usually effective in tendinous or musculotendinous lesions, whereas tenoperiosteal lesions can be treated by friction or infiltration with triamcinolone. Muscular lesions react well to infiltration with local anaesthetics or to deep friction.

Treatment should always be associated with relative rest to avoid further strain on the involved structure. In persistent cases, surgery is indicated.[35,36] The procedure used includes division of the adductor longus tendon close to its origin from the pubis. The results seem to be good and most athletes can return to sports after 8 weeks.

Technique: deep friction

The patient adopts a half-lying position on the couch, with the affected thigh in slight abduction and lateral rotation. The therapist sits level with the patient's knees and facing the body. In musculotendinous lesions, the affected area is grasped between the thumb and index and middle fingers (Fig. 48.3). Friction is imparted by a drawing movement of the hand

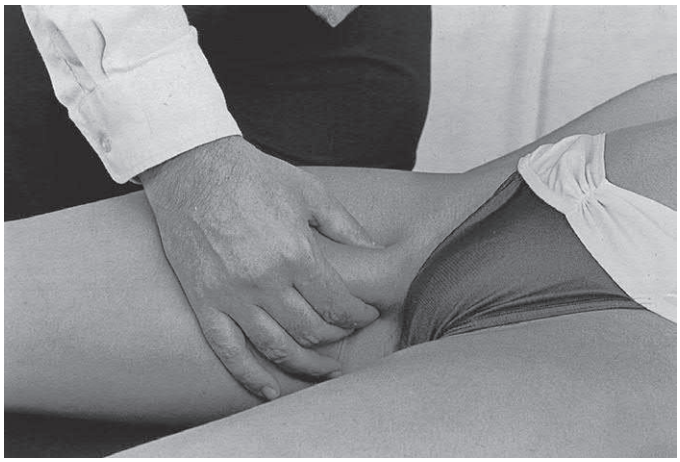

Fig 48.3 • Deep transverse friction to the adductor longus muscle (musculotendinous junction).

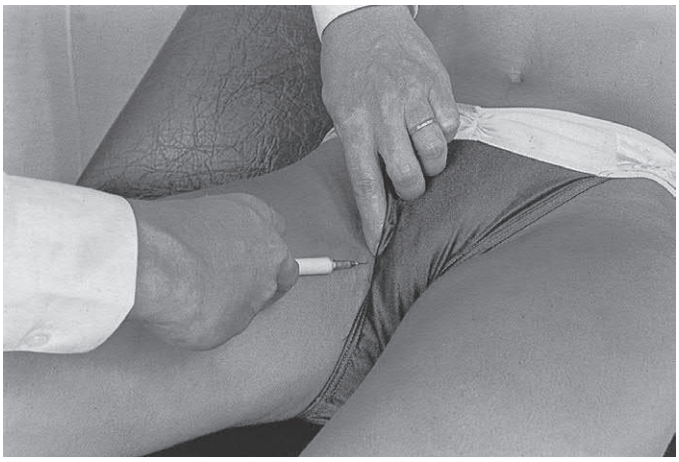

Fig 48.5 • Infiltration of the adductor longus (tenoperiosteal junction).

Fig 48.4 • Deep transverse friction to the adductor longus muscle (tenoperiosteal junction).

medially. At the end of range, the affected structures have passed beyond the fingers and the movement is then repeated.

In tenoperiosteal lesions, the index finger, reinforced by the middle finger, is placed on the pubic bone (Fig. 48.4) and friction is carried out by alternately flexing and extending the elbow, together with a slight adduction and abduction movement at the shoulder.

In the acute stage, treatment should be brief and not vigorous, but can be repeated daily for the first few days and on alternate days thereafter.

Long-standing scars that may have formed in the absence of treatment at an early stage are given truly deep friction for as long as possible – say, 20 minutes – twice a week.

The results are good in all recent and most chronic cases. Exertion is best avoided until the patient is symptom-free.

Technique: injection

The patient adopts a half-lying position on a high couch and puts the thigh into slight abduction. The affected spot is identified at the tenoperiosteal junction on the pubic bone. A 2 mL syringe is filled with triamcinolone and fitted with a 4 cm needle. The needle is inserted just distal from the palpating finger on the periosteum and directed upwards until it hits bone (Fig. 48.5). Using a series of small withdrawals and reinsertions at slightly different points, the whole area is infiltrated. Meanwhile, the palpating finger is kept on the spot, to detect where the tiny bulges of drug appear.

Another infiltration may be required when the patient is seen 2 weeks after wards, if the resisted adduction test remains positive.

Resisted abduction

Pain

Lesions of the hip abductors are rare. The diagnosis is often difficult because pain on resisted abduction of the hip is often caused by transmitted stress on inflamed bursae (trochanter and gluteal) or strained sacroiliac ligaments.

Glutei medius and minimus muscles

Muscular lesions seem not to occur. Local pain during resisted abduction is far more likely caused by compression of an underlying inflamed bursa, which takes place during contraction of the gluteal muscles.[37] If passive abduction is also painful, bursitis is almost a certainty and results from compression of the bursa between the greater trochanter and the ilium (p. 646).

Lesion of the tensor fasciae latae

This muscular overuse lesion may occur in dancers and athletes. It is often caused by repetitive increased tension on the iliotibial band, which results from pelvic tilt or certain activities such as habitual running on the sides of the road ('downside leg'). Localized pain is felt between pelvic crest and trochanter. The signs are characteristic: lumbar examination reveals pain

on side flexion of the trunk towards the painless side, although the other lumbar movements are free. Passive and resisted movements of the hip are normal. Resisted abduction may also be painless, especially in the prone-lying position with the hip extended. When a sprain of the tensor is suspected, resisted abduction should be performed in the side-lying position, with the uppermost hip (test limb) flexed to 45°. Alternatively, an accessory test is performed: in a standing position, the patient crosses the painful leg behind the other and bends sideways towards the painless side, taking all the body weight on the affected limb. Pain – sometimes severe – indicates a lesion of the tensor fasciae latae. Palpation then reveals a painful spot between the iliac crest and the greater trochanter.

Treatment

Treatment of a local lesion of the tensor fasciae latae is by deep transverse friction.

Technique

The patient lies on the painless side. The upper leg rests on the couch in an extended and slightly adducted position to stretch the iliotibial tract. The lower leg is flexed to about 45° to stabilize the pelvis. The therapist stands level with the hip and dorsal to the patient. The thumbs are placed on top of each other at the affected area, with the fingers opposite the thumbs for counterpressure. Friction is imparted by moving the thumbs forwards over the iliotibial tract in a transverse direction (Fig. 48.6).

At the same time, the elbows are slightly extended together with a flexion movement at the shoulders. Friction is continued for 20 minutes and is repeated twice a week. Even in long-standing and neglected cases, it will take no more than 6–8 sessions to achieve a satisfactory result.

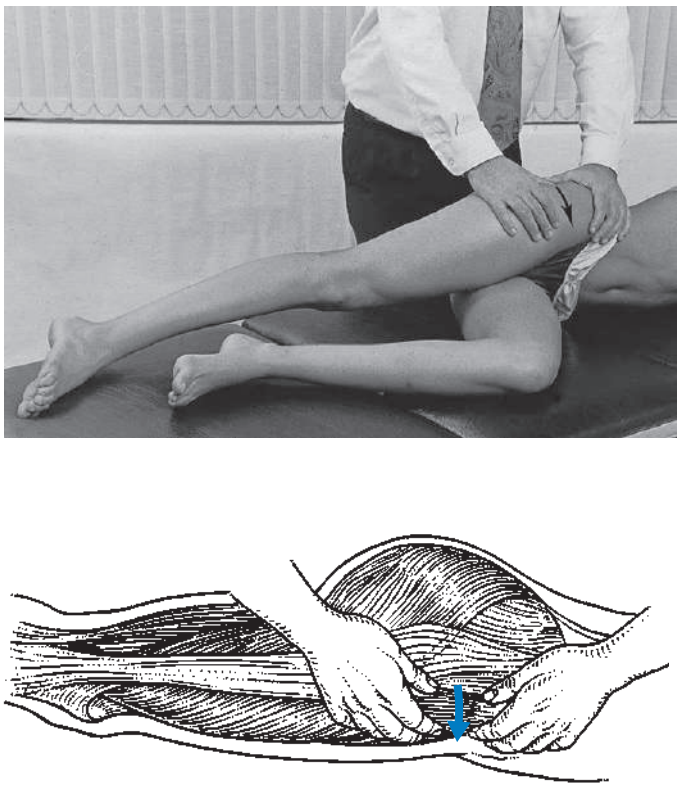

Fig 48.6 • Deep friction to the tensor fasciae latae.

Sprain of the iliotibial tract

This lesion is often described as the 'lateral snapping hip syndrome'. It is caused by slipping of the iliotibial band over the posterior part of the greater trochanter and is frequent in runners, dancers and basketball players.[38,39]

In addition to the audible snapping phenomenon, the syndrome is characterized by pain in the region of the greater trochanter which radiates to the buttocks or thighs. The painful snapping can be observed by placing the palm of the hand on the trochanteric area during walking. Signs on examination of the hip may be identical to those of an inflamed bursa underlying the iliotibial tract (see p. 647).

The non-operative treatment of choice is infiltration with triamcinolone. In refractory circumstances, surgical treatment is indicated. The procedure usually consists of excising an ellipsoid-shaped portion of the band overlying the greater trochanter.[40]

Sacroiliac joint

MacNab[41] also points to the sacroiliac joint as a possible source of pain. When the gluteus medius contracts to abduct the hip, it pulls the ilium away from the sacrum. In the absence of hip joint disease, pain experienced over the sacroiliac joint on resisted abduction of the leg is then highly suggestive of a lesion of the sacroiliac joint (see p. 600).

Pain and weakness

In adolescents, painful weakness of abduction is indicative of an avulsion fracture of the greater trochanter but iliac apophysitis can be another possibility. It develops in young long-distance runners as the result of repeated contractions of the gluteus medius and tensor fasciae latae on an immature iliac apophysis (see online chapter *Hip disorders in children*).[42]

Painless weakness

Painless weakness usually has a neurological cause – weakness of the gluteus medius in a fifth lumbar root palsy. It is also a common finding in disorders of the hip joint and femoral neck, and leads to a raised greater trochanter – protrusion of the acetabulum, epiphysiolysis, congenital dysplasia and malunion after a fracture of the femoral neck. In these instances, resisted abduction is weak and the pelvic–trochanteric muscles are ineffective in stabilizing the pelvis on the leg during walking. In placing the centre of gravity over the hip joint, the patient forces a lurch towards the involved side. Such a movement is called an 'abductor' or a 'gluteus medius' lurch. In mild cases, only a Trendelenburg gait is found – characteristic dipping of the pelvis towards the leg that is off the ground.

Resisted medial rotation

Pain

Pain produced during this resisted movement is rare and usually attributable to an inflamed gluteal bursa. The muscles (the gluteus medius, the anterior fibres of minimus and the tensor fasciae latae) are involved only following a direct blow that causes bruising.

Resisted lateral rotation

Pain

When pain is produced, the quadratus femoris, gemelli and piriformis are initially suspected of being the site of the lesion[43] but, except in a direct injury, they are seldom affected. Pain during resisted lateral rotation of the hip is most often the result of transmitted stress of the contracting muscles acting on an inflamed gluteal or trochanteric bursa (see 'Gluteal bursitis', p. 646).

Resisted extension of the knee

Pain

Tendinitis of the rectus femoris presents as pain felt in the groin. It has been found in 12% of all groin injuries.[2] It is usually an overuse phenomenon that occurs as a result of prolonged, repetitive strengthening exercise or intensive goal-shooting training in soccer.[44] Pain is provoked more on resisted extension of the knee than on resisted flexion of the hip, particularly when the patient lies prone with the hip extended. This finding has already been mentioned and is the result of the constant-length phenomenon. Pain is also felt on full passive flexion of the hip and even more so in a combined movement of flexion and adduction, when the tender part is pinched between the upper femur and the anterior spine of the ilium.[45] Additionally, full passive rotation of the hip, done with the hip and knee held in 90° of flexion, can sometimes cause enough localized stretch to be painful.

The lesion is located just below the inferior iliac spine (in the body of the tendon). Alternatively, the tender point lies at the proximal part of the muscle belly.[46] Treatment is deep transverse friction or infiltration with triamcinolone.

The same stress that causes a muscle strain in an adult can cause an avulsion fracture in an adolescent. The fracture occurs at the apophysis, which becomes separated from the underlying bone. The fracture is never widely displaced because of the surrounding thick periosteum.[47] Most patients can be treated non-operatively with 6–10 weeks of relative rest.

Lesions of the muscle bellies of the quadriceps are discussed in the chapter on the knee (Ch. 54).

Differential diagnosis

Tendinitis of the rectus femoris should be differentiated from lesions of the second and third lumbar roots, usually the result of a disc lesion. Pain is reported over the anterior aspect of the thigh. Knee flexion with the patient in a prone-lying position may provoke the pain. However, testing the muscle for strength is painless, although there may be some weakness. When there is any doubt, an epidural local anaesthetic can be given and confirms the diagnosis.

Treatment

Deep transverse friction of the tendon or infiltration with triamcinolone is effective.

Technique: deep friction

The patient sits on the couch, with the hip joint in flexion, in order to make it possible for the therapist's palpating fingers to reach the overlying tissues. The therapist sits at the patient's side facing the thigh. The painful tendon is found about 8 cm below the anterior superior iliac spine and in line with it, bordered by the sartorius muscle medially and the tensor fasciae latae laterally. Two or three fingers are placed just medial to the tendon, using the thumb at the outer side of the thigh for counterpressure (Fig. 48.7).

Friction is imparted by repetitively pulling the fingers over the tendon in a transverse direction. During this movement, the wrist is extended and the shoulder adducted. In acute cases, treatment is started the day after onset. At this point, deep friction should not last more than 1 minute and is repeated daily, with a slow increase in duration. During the second week, treatment is undertaken on alternate days. In chronic cases, treatment takes about 20 minutes, twice a week. Recovery is achieved in 6–8 sessions. During this period, all activities that are likely to worsen the condition should be avoided.

Technique: infiltration

The patient adopts a half-lying position on a high couch. The affected spot is identified at the tenoperiosteal junction in the triangle between the muscle bellies of sartorius and tensor fasciae latae. A 2 mL syringe is filled with triamcinolone and

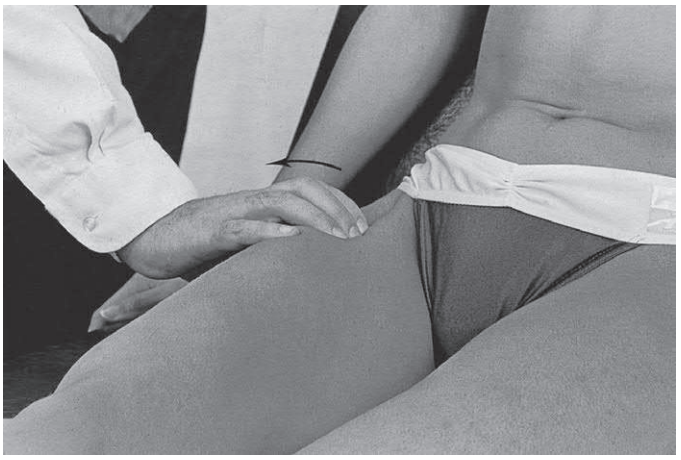

Fig 48.7 • Deep friction to the rectus femoris.

fitted with a 4 cm needle. The needle is inserted just distal to the palpating finger on the periosteum and directed upwards until it hits bone. Using a series of small withdrawals and reinsertions at slightly different points, the whole area is infiltrated. Meanwhile, the palpating finger is kept on the spot, to ascertain where the tiny bulges of drug appear. Another injection may be required if the resisted adduction test remains positive when the patient is seen 2 weeks afterwards.

Pain and weakness

This characterizes a partial muscle rupture.

Painless weakness

Painless weakness of the quadriceps is present in third lumbar nerve root compression (see p. 517). If weakness is bilateral, myopathy or myositis should be suspected.

Resisted flexion of the knee

Pain

Painful resisted flexion of the knee implicates the hamstrings (biceps femoris, semitendinosus and semimembranosus). Lesions of the muscle bellies of the hamstrings are discussed in the chapter on the knee (Ch. 54). Tendinous and tenoperiosteal lesions also occur, cause pain in the gluteal area and posterior aspect of the thigh, and are generally referred to as the 'hamstrings syndrome'.[48,49]

Hamstrings syndrome

This lesion is common in hurdlers and ballet dancers and is also found in all athletes who engage in rapid acceleration and short intense sprinting, such as baseball, tennis and soccer players.[50] The cause of a hamstring strain is not fully understood but most authors today agree that the basic causative reason for its development is the muscle strength imbalance between the hamstrings muscle group and the quadriceps femoris muscle.[51] Clinical features of the hamstrings syndrome include pain in the lower gluteal area which radiates down the posterior thigh to the popliteal area. The pain typically increases during performances that stress the hamstring muscles (sprinting or hurdling). Another characteristic complaint is pain felt while sitting. Driving a car typically provokes the pain because of local pressure on the lesion and the increased tension of the hamstring muscles in that position.

Clinical examination shows painful resisted flexion of the knee and resisted extension of the hip. Straight leg raising is of full range but quite painful at the extreme of range. One common clinical test that may induce the pain is the 'shoe wiping test' of Pecina[52]: it consists of having the patient imitate movements typically used when wiping shoes on a doormat; the hamstrings are stretched and at the same time are contracting, which causes the typical pain.

Palpation reveals tenderness either in the tendon or at the tenoperiosteal insertion. The tendons are usually affected at the upper 5 cm of their extent. In tenoperiosteal lesions, the sprain lies at the tuberosity of the ischium.

Differential diagnosis

First and second sacral root lesions

Tendinitis should be differentiated from these lesions, which are usually the result of disc protrusion at the fifth lumbar level. Pain may be reported in the thigh and there is probably also pain at the end of lumbar flexion or straight leg raising, but testing the muscle for strength is painless. Local tenderness is also absent. In case of doubt, epidural local anaesthetic is given.

Ischial bursitis

Resisted movements are also painless in ischial bursitis or 'weaver's bottom'. Local tenderness is then the only clinical finding and points to involvement of the bursa (see p. 648).

Treatment

In tendinous lesions, the patient is treated with deep transverse friction or infiltration with triamcinolone. Persistent lesions can be treated surgically.[48]

Technique: deep friction

The patient adopts a side-lying position or a supine one with hip and knee flexed to 90° and a chair supporting the lower leg. This position is necessary to move the gluteus maximus upwards, bringing the tuberosity of the ischium directly within reach. It also keeps the tendon fairly stretched; otherwise it lies too lax and deep to be properly subject to friction. The therapist stands on the ipsilateral side, level with the patent's hip. Friction is imparted by applying two or three fingers at the medial aspect of the affected area and moving across the tendon by alternating adduction–abduction at the shoulder and a slight flexion–extension movement at the wrist. The thumb supplies counterpressure and acts as a fulcrum at the outer aspect of the thigh (Fig. 48.8). Treatment is given for 20 minutes, two or three times a week. Recent-onset cases respond sufficiently in about 2 weeks. In chronic cases, 2 months may

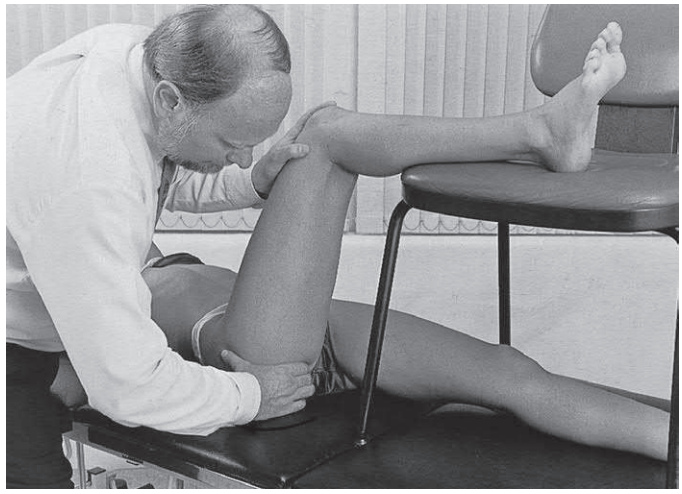

Fig 48.8 • Deep transverse friction to the hamstring tendons.

Table 48.1 Summary of disorders of the contractile structures

Test	Pain	Painful weakness	Painless weakness
Resisted flexion	Psoas tendinitis Rectus femoris tendinitis Sartorius tendinitis Obturator hernia	Avulsion fracture anterior superior iliac spine Avulsion fracture lesser trochanter Abdominal neoplasm Metastasis upper femur	2nd (3rd) lumbar root Psychoneurosis Serious non-specific disorder
Resisted extension	Inflamed gluteal bursa Intermittent claudication Hamstrings tendinitis Sprained sacrotuberous ligament		1st sacral nerve root palsy
Resisted adduction	Adductor longus lesion Fracture/neoplasm pubic bone Osteitis pubis Sacroiliac joint lesion Hip joint lesion		
Resisted abduction	Inflamed gluteal bursa Lesion of the tensor fasciae latae Sacroiliac joint lesion Sprain of iliotibial tract	Avulsion fracture greater trochanter Iliac apophysitis	5th lumbar nerve root palsy Raised greater trochanter
Resisted medial rotation	Inflamed gluteal bursa (tensor fasciae latae)		
Resisted lateral rotation	Quadratus femoris lesion Inflamed bursa Gluteus maximus or medius lesion		
Resisted extension knee	Tendinitis rectus femoris	Partial rupture rectus femoris	3rd lumbar nerve root palsy Myopathy Myositis
Resisted flexion knee	Hamstrings tendinitis	Partial rupture hamstrings	1st/2nd sacral nerve root lesion

be required for a response; therefore infiltration with steroid suspension is the preferred treatment.

Technique: infiltration

Unless the hip is slightly flexed, the origin of the hamstrings at the ischium remains covered by the gluteus maximus. In order to render the insertion palpable, the patient must adopt a prone position, the pelvis well supported on a high cushion. The origin of the hamstrings is then palpable just distal to the lower gluteal border.

The physician stands next to the patient's hip and palpates for the tender spot. A 2 mL syringe is filled with triamcinolone and fitted with a 5 cm needle. The needle is inserted well distally and the tip is moved upwards until it strikes bone. Using a series of small withdrawals and reinsertions at slightly different points, the whole area is infiltrated. Another injection may be required if the resisted flexion test remains positive when the patient is seen 2 weeks afterwards.

Painless weakness

Painless weakness of knee flexion is typical of a full root syndrome at S1 and/or S2.

Disorders of the contractile structures of the hip and buttock are summarized in Table 48.1.

Access the complete reference list online at **www.orthopaedicmedicineonline.com**

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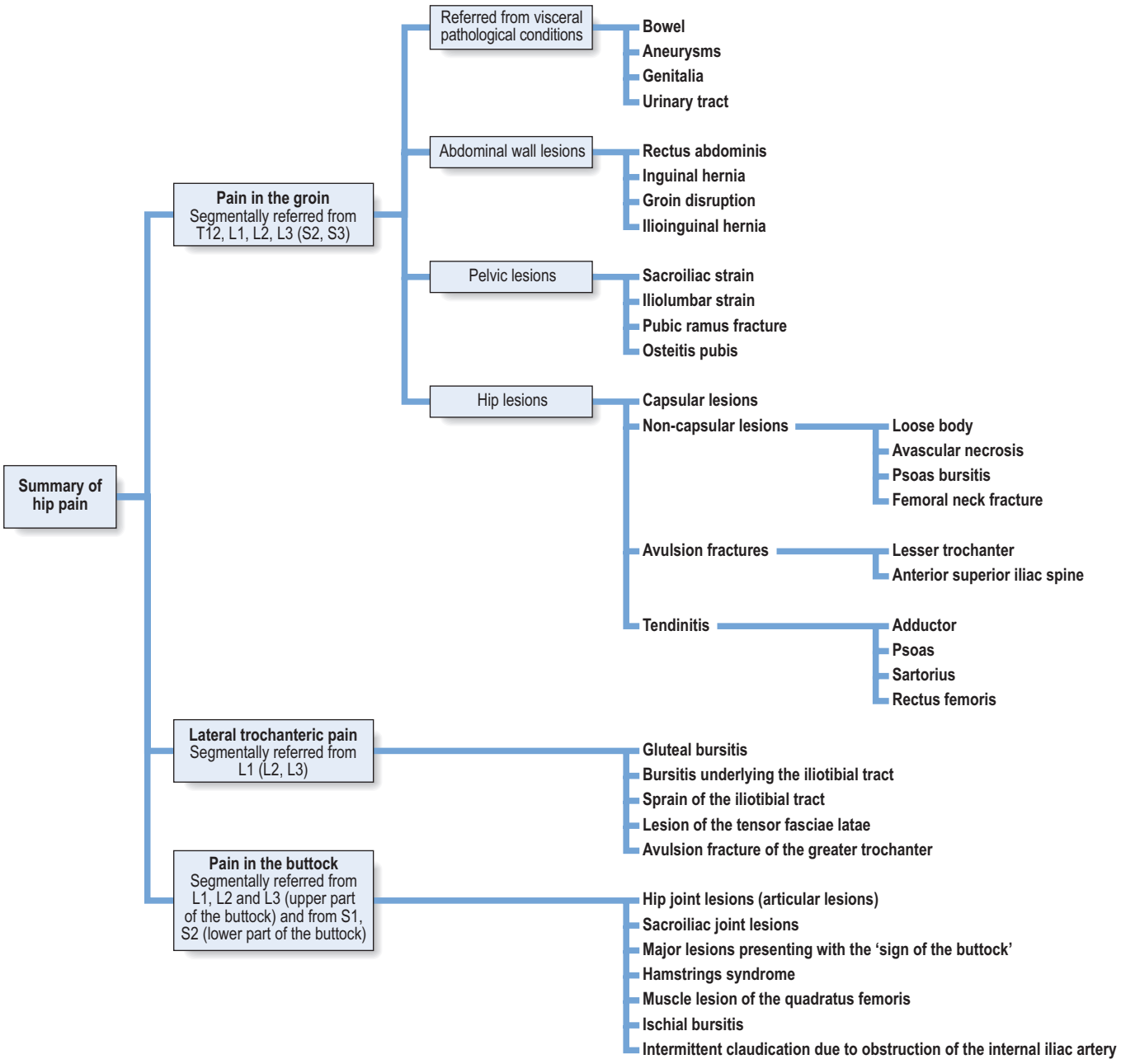
Summary of hip pain



Multisegmental (dural) pain about the hip can be classified into pain in the groin, lateral trochanteric pain and pain in the buttock.

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Clinical examination of the knee

50

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Referred pain

Pain referred to the knee

The front of the knee represents the second and third lumbar dermatomes. Two structures, the hip and third lumbar nerve root, are apt to give rise to referred pain in this area. When referred pain to the knee is suspected, the diagnostic points in the history are the indefinite area of complaint and radiation 'upwards' along the anterior aspect of the thigh. When asked for the exact site of pain, the patient points to the whole suprapatellar area and the front of the thigh. In vague anterior knee pain, the lumbar spine and the hip joint must be examined immediately. This is especially so in children who complain of knee pain but who are in fact developing hip disease, such as aseptic necrosis or epiphysiolysis. Another common error is to take a radiograph of an elderly person's knee because the pain is felt here, and then almost certainly to find some osteoarthrosis at the knee but to miss the osteoarthrotic hip.

The back of the knee is innervated by the first and second sacral segments. Disorders of the first and second sacral roots, and also the sacroiliac joints, can refer pain to this area. Again, the patient cannot point exactly to the site of pain. The pain distribution remains vague and spreads upwards along the thigh or downwards in the calf. There is also no history of relevant trauma. Compression of S1, caused either by a primary posterolateral protrusion in a young adult or by a narrowed lateral recess in an elderly person, may also provoke pain at the back of the knee only. As pain is not felt in the back or buttock at the onset, the symptoms do not draw immediate attention to the possibility of a lumbar disorder. Once again, a careful history suggests the diagnosis. With a primary posterolateral protrusion, the patient will have noticed that sitting and coughing hurt the knee, whereas walking does not. In compression of the nerve root in a narrow lateral canal, complaints are

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related to walking and standing, whereas bending usually relieves the pain.

Pain referred from the knee

Lesions of the knee are usually located accurately by the patient at or in the knee. Sometimes an impacted loose body complicating osteoarthrosis can cause pain referred down the leg and slightly up the thigh but this is rather exceptional.

History

Knee problems are always difficult to evaluate and every possible assistance is needed to make a proper diagnosis. A chronological history, as summarized in Box 50.1, is therefore the first, and sometimes even the most important, element. Cyriax used to say that one who 'doesn't have a diagnosis after the history, will hardly get one after the clinical examination'.

Age is a very important factor because some disorders at the knee appear at a certain time of life only. Anterior knee pain in an adolescent can be the outcome of Osgood–Schlatter disease, whereas the same pain in a 30-year-old sportsman is the result of infrapatellar tendinitis and in a 50-year-old lady patellofemoral arthrosis. Symptoms of internal derangement in a 17-year-old boy almost certainly indicate osteochondritis dissecans, while in a 25-year-old sportsman they may suggest a meniscus lesion and in a 60-year-old lady are probably the result of impaction of a small loose body.

The patient should be questioned about occupation and sporting activities.

Box 50.1

Summary of history taking

- Age, sex, occupation and sport
- Site of pain
- Onset
- Trauma
  - Mechanism
  - Immediate symptoms
  - Symptoms after 24 hours
- Non-traumatic
  - Sudden or gradual onset
  - What affects it?
- Evolution
  - Better/worse
  - Changing localization
  - Treatments and results
- Current symptoms
  - Localization
  - Swelling
  - Influence of movements
  - Instability
  - Twinges
  - Clicks

In knee problems there are three important symptoms that provide a great deal of information: 'locking', 'twinge' and a 'feeling of giving way'. Because the meaning of these words is not always totally understood and patients frequently confuse them, it is vital to describe what exactly is meant when asking about these symptoms.

- *Locking*: sudden (painful) limitation occurs during a movement, whereas other movements are free and painless. The knee can be locked in flexion (extension being limited) or extension (flexion being impossible).
- *Twinge*: a sudden, sharp and unexpected pain is felt. For example, the patient feels abrupt, unforeseen and sharp pain at the inner side of the knee during walking. The pain disappears immediately and normal walking again becomes possible.
- *Feeling of giving way*: this is the typical sensation in instability – a sudden feeling of weakness. It feels as if the knee cannot bear the body weight during a particular movement. The knee tends to 'collapse'.

In order to work out the diagnosis systematically and chronologically, it is as well to start with the onset of the symptoms before concentrating on symptoms at the time of examination.

Onset

- *When did it start?* Is this an acute, subacute or chronic problem?
- *How did it start?* Did the pain come on for no apparent reason or was there an injury?

If there was trauma

Describe the exact mechanism:

- In what position were the body and the leg?
- What forces were acting on the knee?

Describe the immediate symptoms:

- *Where was the initial pain?* At one side, all over or inside the joint?
- *Was there any swelling?* Immediately or after some time? An immediate effusion is always haemorrhagic and therefore indicates a serious lesion. If a swelling appears after some time, it is the consequence of a synovial reaction.
- *Did the knee give way?* Immediately or after some time?
- *Was there any locking?* If so, was the knee locked in flexion (which is typical for meniscal lesions) or was it in extension (as in impacted loose bodies from osteochondritis dissecans)? How did the knee become unlocked? By manipulation (meniscus) or spontaneously (loose body)?
- *Were you still able to walk home after the accident?*

If there was no trauma

- Did the pain come on suddenly or gradually?
- What were you doing when the pain first appeared?

- Describe the first symptoms. These may include localization, swelling, locking or loss of function (see above).

Evolution

In long-standing cases or in traumatic conditions, it is very important to have an exact idea of the evolution of the complaints.

- *Did the pain change from one side of the joint to the other or did the pain spread?* Pain moving from one side of the joint to another is characteristic of a loose body: the localization of the pain travels with the impacted loose fragment.
- *What was the evolution of the swelling?*
- *For how long were you disabled?*
- *What treatment did you have and to what effect?*
- *Have there been any recurrences?* If so, what brought them on and how did they progress?

Current symptoms

Finally, the current complaint is discussed.

- *Describe the exact localization.*
- *What brings the pain on?*
- *Do you have nocturnal pain or morning stiffness?* Pain at night usually indicates a high degree of inflammation. It occurs in acute ligamentous lesions, haemarthrosis and arthritis. Long-standing morning stiffness is usually an indication of rheumatic inflammation.
- *What is the effect of going upstairs and downstairs, and which is the more troublesome?* Going downstairs loads not only the extensor mechanism but also the posterior cruciate ligament and the popliteus tendon. Going downstairs is also very painful in impacted loose bodies.
- *Do you have twinges?* Very often, a twinge means an impacted loose body or a meniscus.
- *Does the knee give way?* Does it actually give way or just feel as though it might?
- *Does the knee click or grate?*
- *Have any other joints been affected?*

At the end of history taking, patients must be asked about their general state of health.

Inspection

In the standing position

The lower extremities are first viewed with the patient standing. Alignment of the femur, varus or valgus positions of the lower leg, pronation of the feet and alignment of the patella are observed from the front. Some genu valgum deformity in children is normal and usually disappears with growth. When the cause is a valgus position of the heel and inversion of the forefoot, appropriate measures can be taken. Excessive genu valgum deformity in elderly patients may suggest osteitis deformans. The view from the side detects any recurvatum or lack of complete extension. Observation for tibial torsion is done standing above the patient's knees and looking downwards along the tibial tuberosity and anterior tibial crest. The coronal plane of the knee is then compared with an imaginary line connecting the medial and lateral malleoli of the ankle. Tibial torsion between 0 and 40° is normal.

In the sitting position

The most important observations of the patellar position are made with the patient seated on the examination table, the legs hanging free and the knees flexed to 90°. The examiner first assesses the patellar position and the position of the tibial tuberosity and patellar ligament by viewing the knee from the lateral aspect. Thereafter the examiner views the knees from the anterior aspect while the patient holds both knees together. Normally positioned patellae face straight ahead. Malalignment of the kneecap is seen as a patella 'looking' up and over the shoulders of the examiner (see p. 722).

In the supine position

The supine-lying position is the best from which to observe swelling at the knee joint.

Diffuse swelling indicates fluid in the joint and/or synovial swelling. In advanced arthritis or in large effusions of blood, the knee may adopt a slightly flexed position.

Diffuse swelling with muscular wasting may indicate severe and long-standing arthritis. Swelling with reddening of the skin suggests sepsis or gout.

Localized swellings are caused by bony outcrops, cysts or inflamed bursae, such as a prepatellar or infrapatellar bursa, a semitendinosus bursa, a bursa under the medial collateral ligament, a lateral or medial meniscus cyst or a Baker's cyst.

Functional examination

The routine clinical examination of the knee consists of 10 passive movements, two for the joint and eight for the ligaments, and two resisted movements (Table 50.1). If signs warrant, or if suspicion of meniscal lesions or instability arises from the history, complementary tests can be performed.

Palpation for tenderness is only carried out along the structure identified by the functional examination and therefore is only performed after the functional examination. However, palpation for heat, fluid and synovial thickening is performed before the functional examination.

Two primary movements for the joint

As in the elbow, the range of rotation becomes restricted only in advanced arthritis. Therefore extension and flexion

(Fig. 50.1) are the two movements used to test the mobility of the joint.

Extension

Normally, the knee can be extended until the tibia comes into line with the femur, but in young people some hyperextension can occur and is normal. Extension is limited by the posterior cruciate ligament and the posterior capsule. The end-feel is hard.

The evaluation of the end-feel during extension is extremely important and can only be tested if a correct technique is used. One hand takes the heel of the patient; the fingers of the other hand support the knee, while the thumb presses on the tibia, just below the patella. A rapid, short extension movement is now performed by simultaneous upward movement of the heel and downward pressure on the tibia.

Flexion

A normal knee can be flexed until the heel reaches the buttock. Flexion is normally limited by contact between the thigh and calf muscles; the end-feel is therefore of the extra-articular type – softish.

A painful arc during flexion–extension is rather exceptional. When present, it indicates an impacted loose body, a torn meniscus or localized erosion of the articular edge of the femur. A painful arc can also be present in lesions of the iliotibial tract (friction syndrome or bursitis). Here pain is elicited as the tract rides over the lateral femoral condyle.

Table 50.1 Functional examination

Testing	Movements	
1. Test for heat, swelling, synovial thickening		
2. Testing movements		
Joints	Flexion	Extension
Ligaments	Varus Medial rotation Anterior drawer test Medial shearing	Valgus Lateral rotation Posterior drawer test Lateral shearing
Resisted tests	Resisted flexion (Resisted medial rotation)	Resisted extension (Resisted lateral rotation)
3. Retest for heat, swelling, tenderness		

Eight secondary movements for the ligaments

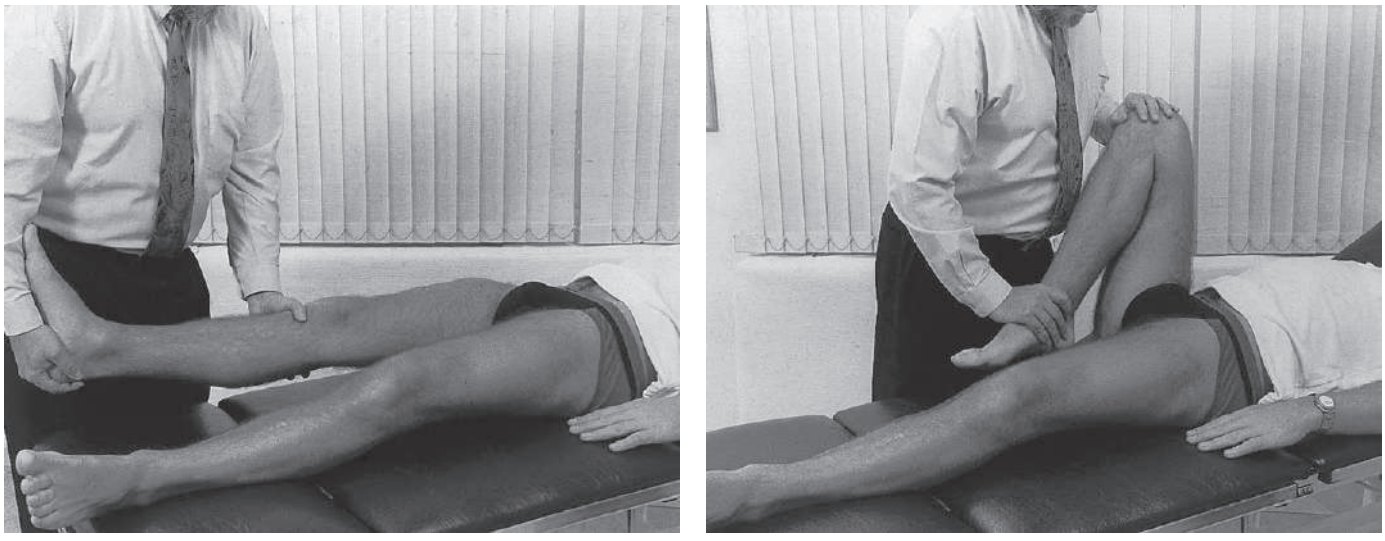
Stretching the ligaments tests them for pain and laxity.

Valgus strain

Strong valgus movement applied with counterpressure at the lateral femoral condyle tests the medial collateral ligament (Fig. 50.2a). Normally, this is done in full extension. In a minor sprain or in a minor degree of instability resulting from previous overstretching, pain and laxity are probably better disclosed if the test is repeated in slight flexion (30°).

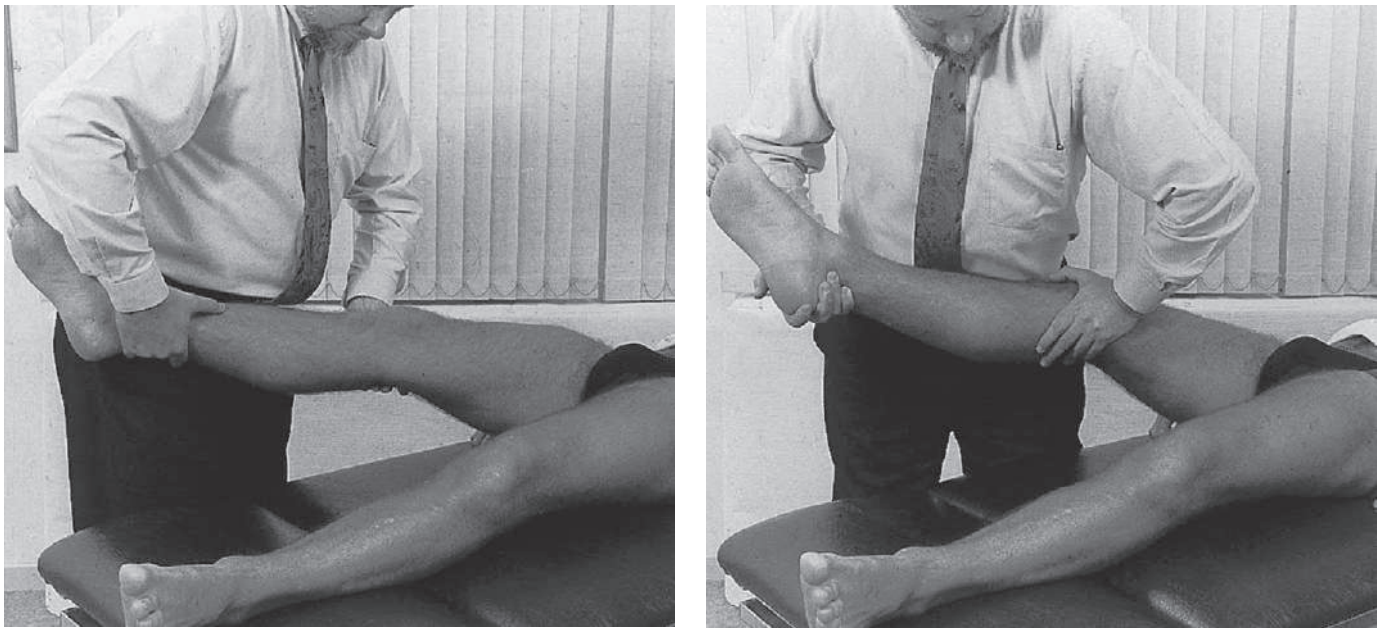
Varus strain

Strong varus movement is applied during counterpressure at the medial femoral condyle and tests the lateral collateral ligament (Fig. 50.2b). Again, the test can be repeated in slight flexion (30°).



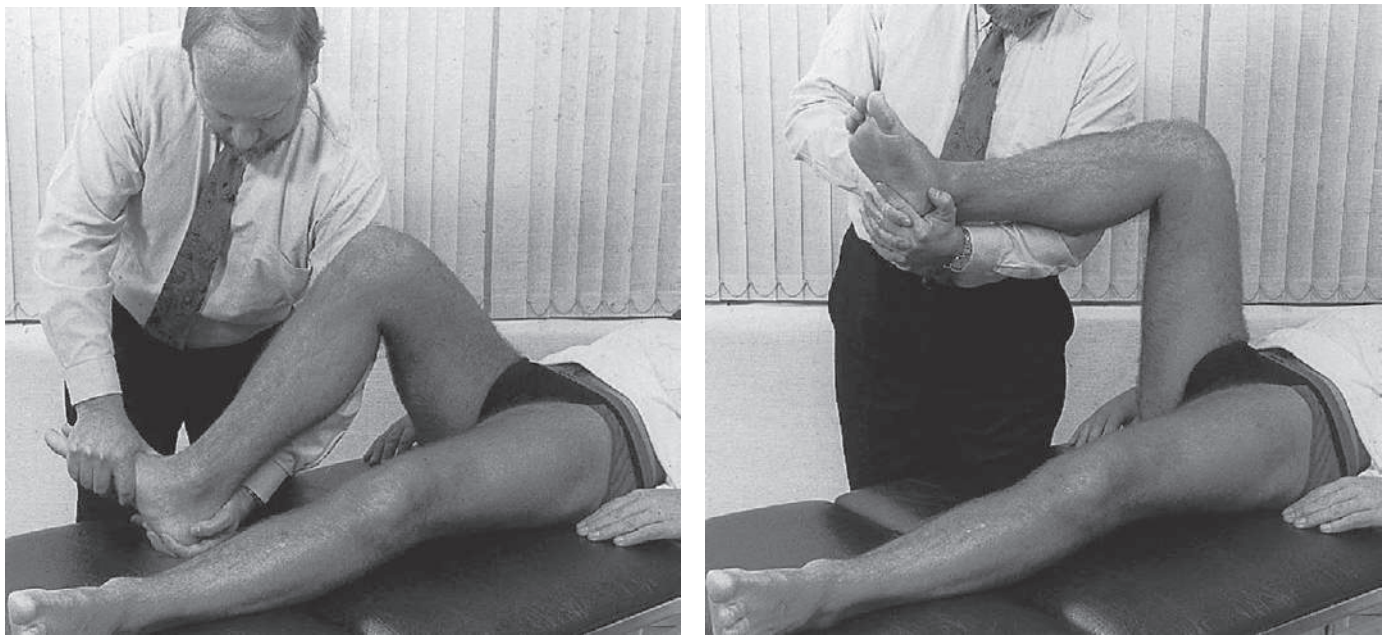
(a) (b)

Fig 50.1 • Extension (a) and flexion (b) of the knee.



(a) (b)

Fig 50.2 • Valgus (a) and varus (b) movement.



(a) (b)

Fig 50.3 • Lateral (a) and medial (b) rotation.

Lateral rotation

Lateral rotation of the knee puts stress on the medial coronary ligament and the posterior fibres of the medial collateral ligament. The knee is flexed to a right angle and the heel rests on the couch. To prevent rotation in the hip, the examiner places the contralateral shoulder against the knee, the arm under the lower leg and a hand under the heel. The other hand is placed at the inner side of the foot, which is pressed upwards in dorsiflexion. Lateral rotation is now easily performed by using the foot as a lever (Fig. 50.3a). The normal end-feel is elastic.

Medial rotation

Medial rotation puts stress on the lateral coronary ligament and the anterior cruciate ligament. The hip and knee are flexed to

right angles. The lower leg is supported by the contralateral forearm of the examiner. The hands are clasped tightly about the patient's heel, which is forced into dorsiflexion. With a combined movement of both wrists, the leg is turned into medial rotation (Fig. 50.3b). In order to protect the lateral ligaments of the ankle, it is important to exert pressure at the ankle and not beyond the calcaneocuboid joint line. The normal end-feel is elastic.

Anterior drawer test

The knee is flexed to a right angle, and the examiner sits on the patient's foot and places one hand on the patella. The other hand is placed at the back of the upper tibia, which is drawn forwards with a strong jerk to test for pain if the anterior cruciate ligament is damaged (Fig. 50.4a). The anterior drawer test in 30° of flexion ('Lachman's test') seems to be more precise in disclosing elongation or rupture in the anterior cruciate ligament (see Ch. 53).

Posterior drawer test

The examiner once again sits on the patient's foot. One hand is placed on the tibial tuberosity, while the other rests at the back of the knee. A strong backward jerk is exerted on the tibia to test the integrity of the posterior cruciate ligament (Fig. 50.4b). In a normal joint, no movement takes place and the test is completely painless.

Medial shearing strain

The knee is held at a right angle. The examiner sits opposite the patient, interlocks the fingers and places the heel of one hand at the lateral tibial condyle, with the heel of the other hand at the medial femoral condyle. By applying a strong shearing strain, an attempt is made to move the tibia medially on the femur (Fig. 50.5a). Pain may be elicited when a loose body is present. In a tear of the lateral meniscus, this manœuvre can displace part of the meniscus to the other side of the femoral condyle. A loud click is then heard and the full range of passive extension is immediately lost.

Lateral shearing strain

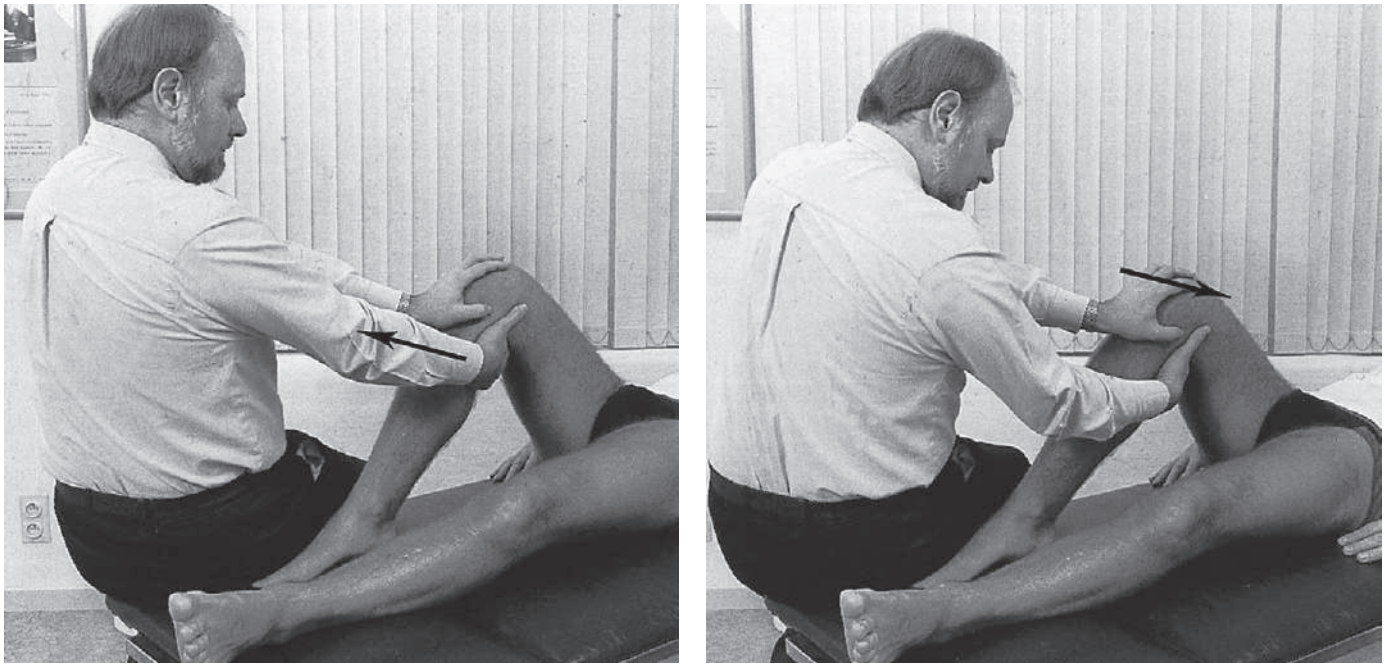
This action is the reverse of medial shearing strain. The heel of one hand is placed on the lateral femoral condyle and the heel of the other on the medial tibial condyle (Fig. 50.5b). A strong shearing force moves the tibia laterally on the femur and may provoke a click when a loose body or a longitudinal tear of the medial meniscus is present. It also elicits pain when a strain of the posterior cruciate ligament is present.

Two resisted movements for the contractile structures

For convenience, resisted flexion and extension are tested with the patient supine, but stronger force can be exerted if he or she is prone.

Resisted extension

The knee is kept slightly bent. The examiner places one arm under the patient's knee. The other hand is placed on the distal end of the tibia, where it resists extension by the



(a) (b)

Fig 50.4 • The anterior (a) and posterior (b) drawer tests.

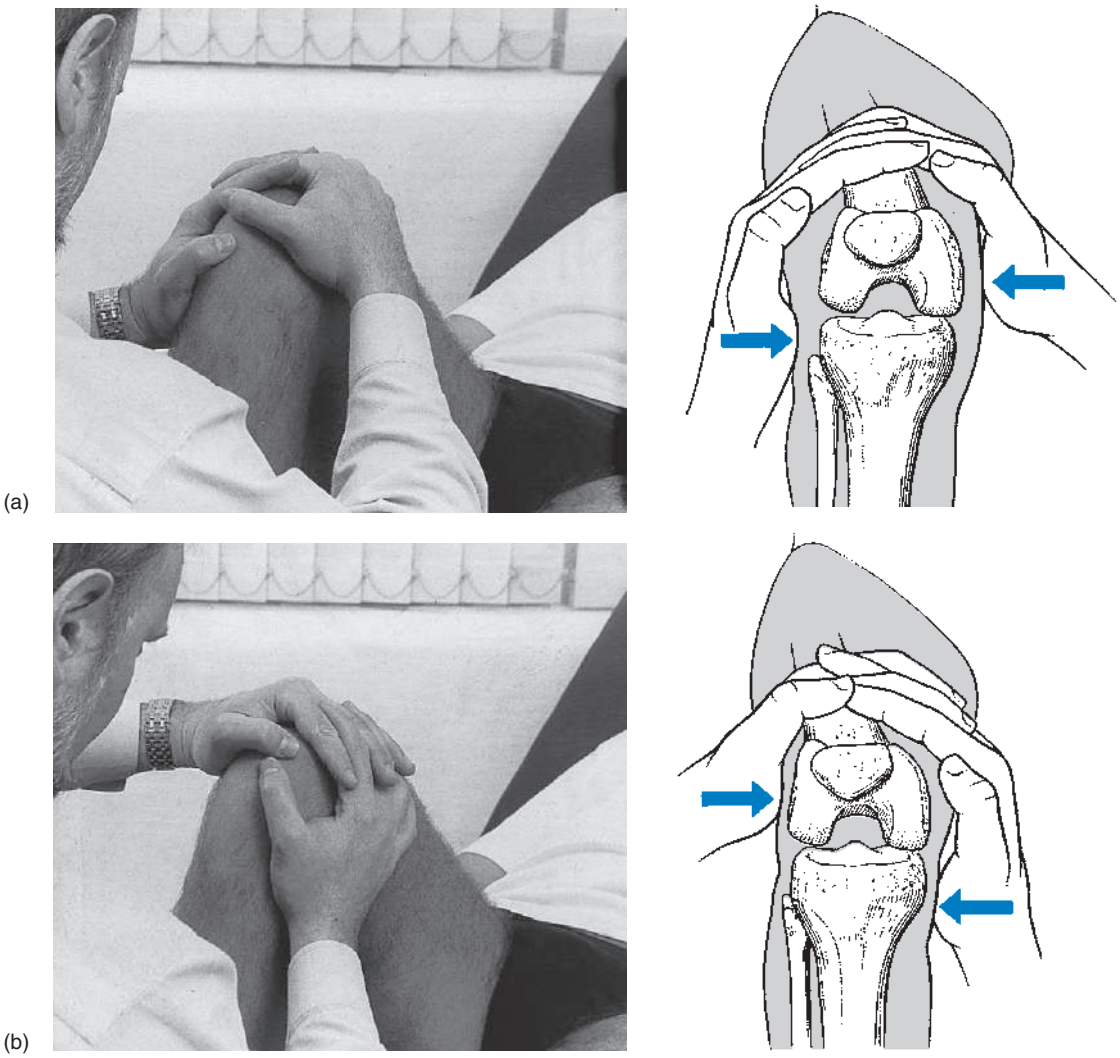


Fig 50.5 • Medial (a) and lateral (b) shearing strain.

patient (Fig. 50.6a). Pain and weakness are noted. If there is any pain, a lesion of the quadriceps mechanism is likely. If there is any weakness, a lesion of the nerve supply, usually the third lumbar nerve root, is present. Pain *and* weakness occur in a fractured patella or after a major rupture of the muscle belly.

Resisted flexion

The hip and knee are bent at right angles. The patient's heel is supported by the hands of the examiner. The patient tries to move the heel downwards while the examiner applies strong counterpressure (Fig. 50.6b).

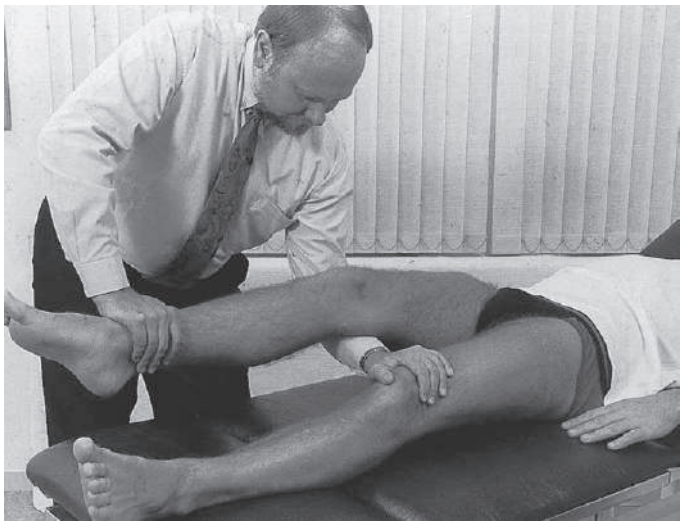
If flexion is painful, the test is repeated, this time in almost full extension, to disclose lesions of the tibiofibular ligament and the posterior cruciate ligament. Then, with the patient in a sitting position, medial and lateral rotation is tested against resistance while the knee is held passively flexed. This test distinguishes between lateral (biceps) and medial (semimembranosus, semitendinosus and popliteus) rotators of the knee.

Palpation

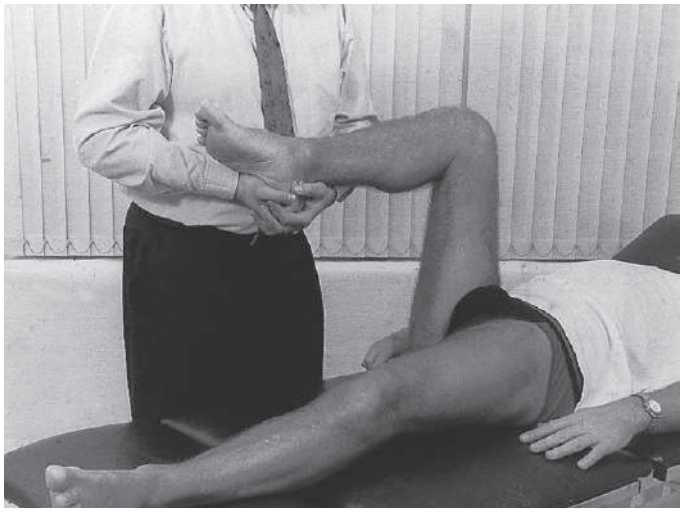
Palpation for warmth and fluid in the stationary joint is done before the clinical examination, and palpation for synovial thickening, tenderness, warmth and irregularities is done after the clinical examination. Finally, crepitus is sought during movement (Box 50.2).

Fluid

Fluid in the knee joint is a sign that is common to many disorders (traumatic, inflammatory or crystalline). Therefore, 'water on the knee' is only a statement of a sign, never a diagnosis. Testing for fluid in the joint can be done in three ways.



(a)



(b)

Fig 50.6 • Resisted extension (a) and flexion (b).

Box 50.2

Palpation for:

- Fluid (three tests)
- Heat
- Synovial thickening
- Irregularities
- Crepitus
- (Tenderness)

Patellar tap

This is the classic test. Manual pressure empties the suprapatellar pouch and moves the fluid under the patella. In the meantime, the thumb and middle finger of the other hand are used to press on the medial and lateral recesses until they empty. Any fluid now lies between the patella and femur. Next the index finger of the lower hand pushes the patella downwards

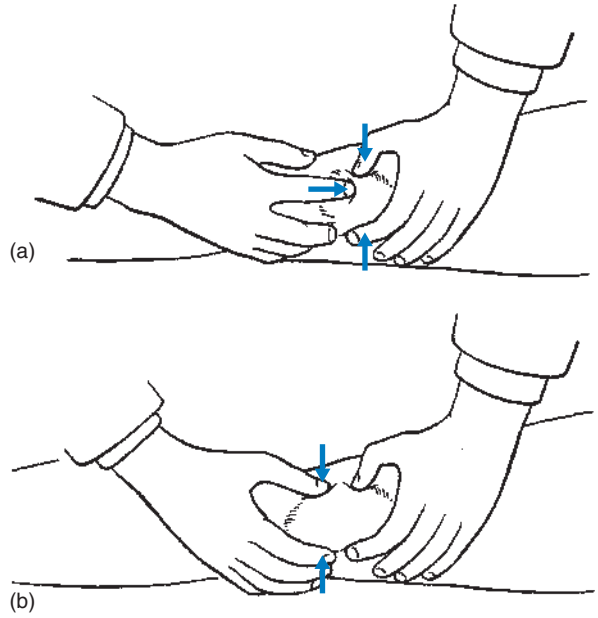


Fig 50.7 • Testing for fluid in the joint by patellar tap (a) and by eliciting fluctuation (b).

(Fig. 50.7a). If fluid is present, the patella is felt to move. When it strikes the femur, a palpable tap is felt, followed by an immediate upward movement. This is the sensation of an ice-cube pushed downwards in a glass of water: although the patella moves downwards, the pressure of the fluid immediately shifts the bone upwards against the palpating finger. In a normal knee, the patellar tap is not elicited.

Eliciting fluctuation

The examiner's thumb and index finger are placed at each side of the patient's knee, just beyond the patella. With the interdigital web I–II of the other hand, the examiner squeezes the suprapatellar pouch, pushing all the fluid downwards under the patella, which forces the two fingers of the palpating hand apart (Fig. 50.7b). This sensitive test will detect even very small volumes of fluid and enables an experienced examiner to differentiate between blood and clear fluid. Blood fluctuates *en bloc*, like a mass of jelly, whereas a clear effusion flows like water.

Visual testing by eliciting fluctuation

This test is not strictly palpation but relies on vision. Stroking in a sweeping motion with the back of the hand over the lateral recess and the suprapatellar pouch moves the fluid upwards and medially (Fig. 50.8a). In a minor effusion, all the fluid is moved to the medial part of the suprapatellar pouch. The lateral recess is then empty and can be seen as a groove between patella and lateral femoral condyle. Sweeping with the back of the hand over the suprapatellar pouch and downwards over the medial recess will now transfer the fluid laterally (Fig. 50.8b), where a small prominence appears. This is the most delicate test for effusion in the knee joint and demonstrates as little as 2 or 3 mL of fluid.

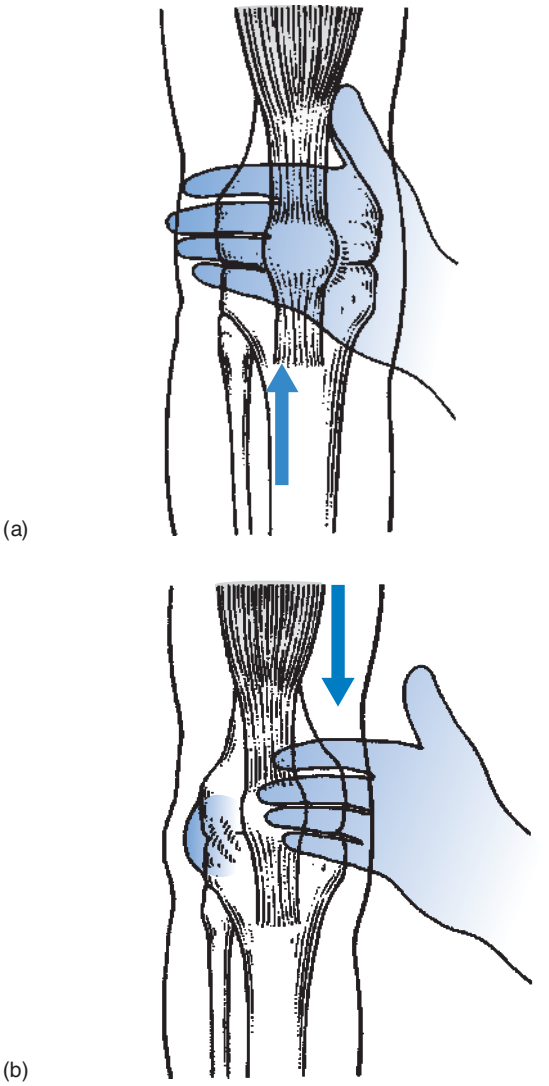


Fig 50.8 • Visual testing for fluid by eliciting fluctuation. (a) A sweeping motion over the lateral recess moves the fluid upwards and medially. (b) Sweeping downwards over the medial recess transfers the fluid laterally where a small prominence appears.

Heat

Palpation for heat is performed before and after the functional examination.

Heat, whether localized or generalized, means that the lesion is in an active stage. Generalized heat indicates capsulitis. Localized heat, sometimes only appearing after the clinical examination, indicates the presence of a minor lesion.

Heat appears in the following conditions:

- Blood in the joint
- Recent trauma or operation
- Arthritis of rheumatoid, crystalline, traumatic or bacterial origin
- Loose body impacted in an osteoarthrotic joint or an impacted meniscus
- Fracture

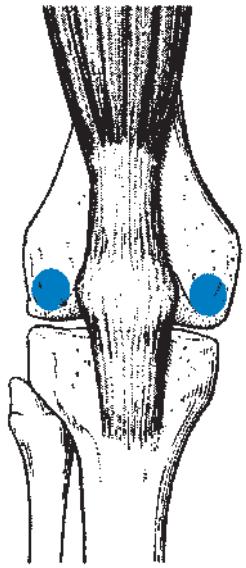


Fig 50.9 • Synovial swelling is most easily detected at the medial and lateral condyles of the femur.

- Metastases
- Osteitis deformans
- Ligamentous lesions.

Synovial thickening

Synovial thickening is a vital clinical finding. It indicates primary inflammation of the synovia and differentiates this from a secondary synovitis.

Synovial swelling is best detected at the medial and lateral condyles of the femur, about 2 cm posterior to the medial and lateral edges of the patella (Fig. 50.9). Here the synovium lies almost superficially, covered only by skin and subcutaneous fat. It is palpated by rolling the structures between fingertip and bone. Normally nothing except skin can be felt. In synovial thickening, a dense structure can be felt.

Deformities

After a fracture, or when osteophytes are present, bony deformities may be felt. Previous Osgood–Schlatter disease results in a prominence of the tibial tuberosity. Long-standing infrapatellar tendinitis may cause a bony outcrop at the patellar tip. Calcified areas in the suprapatellar pouch may form a palpable thickening but are not of clinical significance. In osteitis deformans at the tibia, the sharp anterior edge of the tibial plateau may be lost and is eventually accompanied by localized warmth.

Localized swellings may be felt all over the knee.

Some swellings are more obvious with the knee straight, whereas others are thrown into relief by flexing the knee. Ganglia and cysts related to tendons or menisci feel tense or even hard. Inflamed bursae feel softer and bimanual palpation can usually disclose some fluctuation.

Tenderness

Tenderness can be sought in the structure identified by the history and functional examination, provided the lesion lies within reach of the fingers. The two knees must always be compared carefully. Because most of the structures affected can be palpated, great diagnostic accuracy is possible.

Palpation of the moving joint

This can give an idea of the state of the opposed surfaces of the articular cartilage. However, it is important to remember that crepitus, cracking and clicking may very well appear without any pain or disablement. Some fine crepitus is also normal in middle-aged individuals. In contrast, rough crepitus indicates marked fragmentation of the surface of articular cartilage and hard creaking of bone against bone indicates that cartilage has been completely eroded. In patellofemoral arthrosis, the marked crepitus characteristic of this condition is felt only during squatting.

Accessory tests

These tests, summarized in Box 50.3, are performed only if the history or clinical examination warrants them. Meniscus tests are thus performed when the history includes periods of locking.

Stability tests are used when the patient mentions a feeling of giving way, or when some laxity is detected during the routine functional examination. Patellofemoral tests are used if the history is that of anterior knee pain or patellofemoral dysfunction.

Bilateral passive rotations

The range of knee rotation can best be compared with that of the contralateral side by performing the movement simultaneously at both knees. The patient lies prone and flexes both knees to a right angle. The examiner stands at the end of the couch, grasps the patient's feet, pushes them in dorsiflexion and then simultaneously rotates both feet laterally to test external rotations and medially to test internal rotations. Range of movement and end-feel are compared.

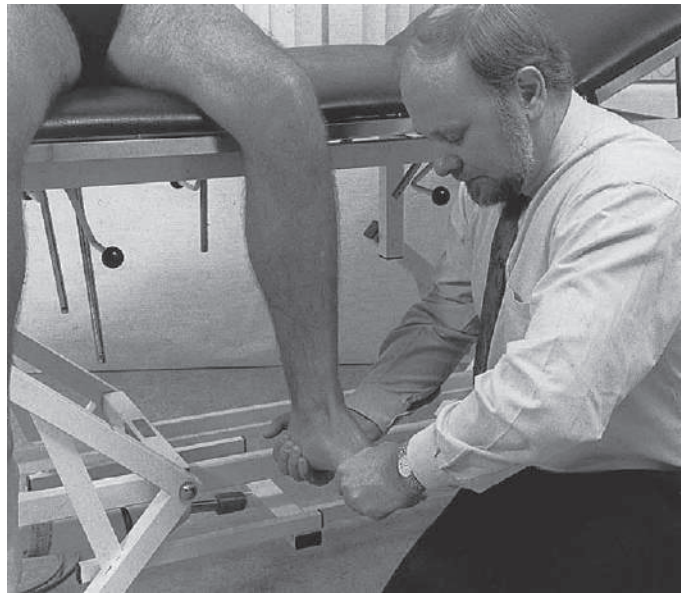
Box 50.3

Accessory tests

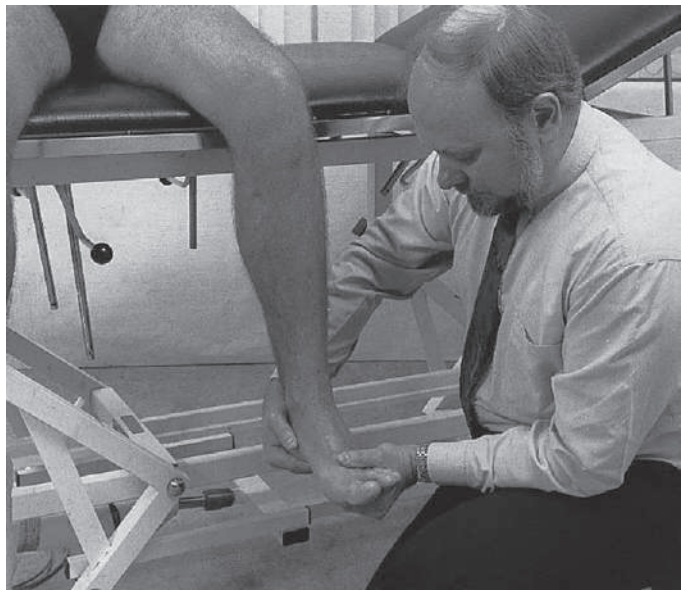
- Bilateral passive internal and external rotation
- Resisted internal and external rotation
- Stability tests
- Meniscus tests
- Patellofemoral tests
- Squatting

Resisted internal and external rotation

The patient sits on the table, the knee flexed to a right angle and the legs hanging over the edge. The examiner holds the foot in dorsiflexion and asks the patient to press outwards and inwards, meanwhile maintaining the neutral position with both hands. Internal (medial) rotation tests the medial hamstrings and popliteus (Fig. 50.10a). External (lateral) rotation tests the biceps femoris and the upper tibiofibular joint (Fig. 50.10b).



(a)



(b)

Fig 50.10 • Resisted internal (a) and external (b) rotation.

Box 50.4

Summary of the clinical examination of the knee

Inspection

- Standing
- Sitting
- Supine-lying

Basic functional examination

- Two primary movements for the joint
  - Passive flexion
  - Passive extension
- Eight secondary movements for the ligaments
  - Valgus strain
  - Varus strain
  - Lateral rotation
  - Medial rotation
  - Anterior drawer test
  - Posterior drawer test
  - Medial shearing strain
  - Lateral shearing strain
- Two resisted movements
  - Extension
  - Flexion

Palpation

- Fluid
- Synovial thickening
- Deformities
- Tenderness
- Palpation of the moving joint

Accessory tests

- Bilateral passive internal and external rotation
- Resisted internal and external rotation
- Stability tests
- Meniscus tests
- Patellofemoral tests
- Squatting

Squatting

Palpation during squatting is performed when a lesion at the patellofemoral joint is suspected. The palpating hand ascertains whether there is crepitus, which is typical in patellofemoral disorders.

Other tests

Tests for capsuloligamentous instability, meniscal lesions and patellofemoral disorders are described in Chapters 52, 53 and 54 respectively.

Clinical examination of the knee is summarized in Box 50.4.

Interpretation of the clinical examination of the knee

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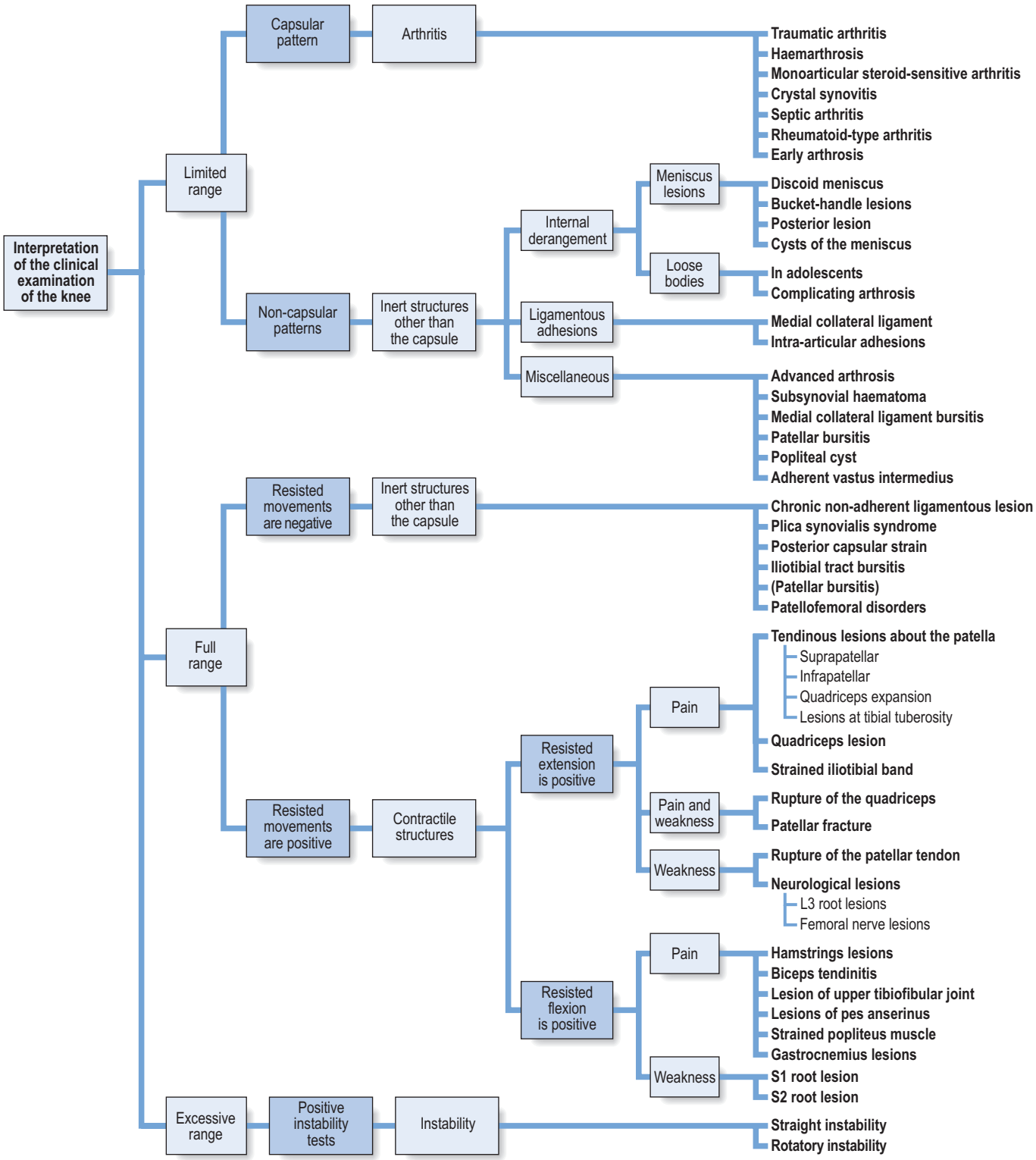

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Disorders of the inert structures: capsular and non-capsular patterns

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Capsular pattern

The normal capsular pattern at the knee joint is gross limitation of flexion and slight limitation of extension; the ratio of flexion : extension is roughly 1 : 10 (Fig. 52.1). Thus 5° of limited extension corresponds to 45–60° limitation of flexion and 10° to 90–100°. The range of rotation becomes restricted only in advanced arthritis. Recently, the concept of a capsular pattern of motion restriction at the knee was supported by a study of patients with inflamed knees.[1]

A number of conditions of the knee show a limited range in a capsular way but all can be differentiated by findings from the history and clinical examination. Therefore, the important features of capsular disorders are a traumatic or non-traumatic, slow or sudden onset, synovial thickening, redness, nocturnal pain and so on.

Traumatic arthritis

Trauma to the capsule invariably leads to traumatic arthritis. Depending on the site and stage of the lesion, the capsular pattern may be gross (acute sprain of the medial collateral ligament) or more subtle (coronary sprain).

The diagnosis is relatively simple: after an injury, a capsular pattern appears, with effusion but no capsular thickening. Care must be taken not to overlook a traumatic haemarthrosis; when the question of blood in the knee joint does arise, diagnostic aspiration must be carried out at once. If blood is present in the aspirate, not only does it indicate a serious lesion but also the joint must be fully aspirated. On the other hand, if the aspirated fluid is clear, there is no point in emptying the joint because it is not the fluid that matters but the primary lesion. Hence treatment of traumatic arthritis showing a clear effusion only during aspiration involves dealing with the underlying primary disorder.

Rheumatoid and reactive arthritis

Rheumatoid arthritis is a common cause of knee inflammation. The symmetrical distribution of the affected joints is typical of this disease (Fig. 52.2).

Reactive arthritis is an inflammation of the joint secondary to a generalized infection but in which the articular fluid remains aseptic (Crohn's disease, Reiter's syndrome, aseptic gonococcal arthritis).[2,3] The arthritis can remain monoarticular but may involve other joints in an asymmetrical manner.

In rheumatoid and reactive arthritis, intra-articular injections with triamcinolone give excellent but often only temporary results. Steroids injected into weight-bearing joints can

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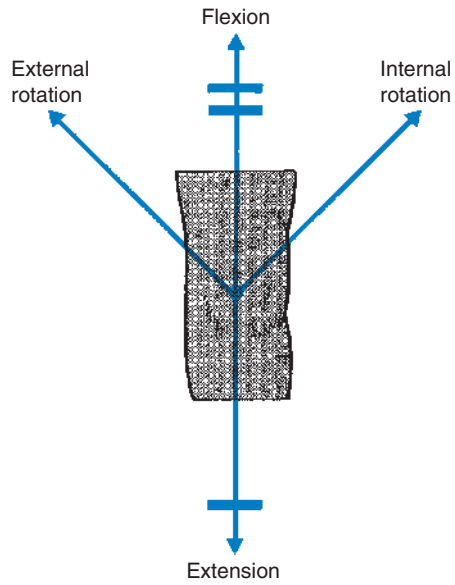


Fig 52.1 • Capsular pattern at the knee joint.

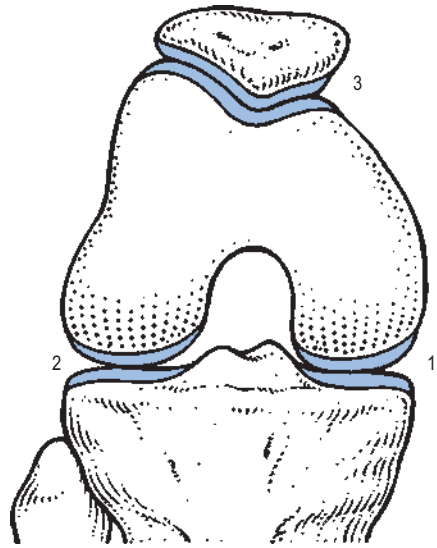


Fig 52.3 • Localized arthrosis at the knee: 1, medial compartment; 2, lateral compartment; 3, patellofemoral compartment.

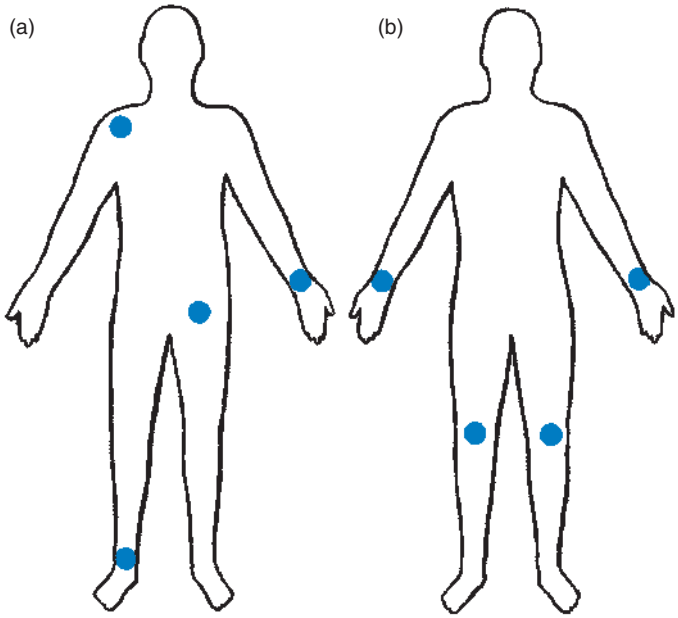


Fig 52.2 • The reactive (a) and rheumatoid (b) patterns of joint involvement.

cause arthropathy if injected too often.[4,5] Therefore, injections must be restricted to two or three a year, which is enough to keep the patient comfortable. Nevertheless, considerable doubts about the dangers of repeated intra-articular steroid injections have arisen since a study report by Balch *et al*.[6] They studied 65 patients with rheumatoid arthritis, who each received a minimum of 15 and a maximum of 167 injections at monthly intervals. Only two showed gross osteoarthrosis. These findings were confirmed later.[7]

Osteoarthrosis

The prime cause of gross degeneration in the knee joint is a disturbed distribution of load, which happens, for instance, when the patient suffers repeatedly from attacks of internal derangement because of a torn meniscus.[8,9] Early osteoarthrosis will also occur if a valgus deformity is present from youth or there is an old malunited fracture that produces uneven load and repeated shearing strains. This also occurs if the meniscus is completely removed and its load-bearing function is therefore lost; the femoral condyles then exert pressure on the tibial platform over a much smaller area and the joint is apt to wear out rapidly.[10,11] The knee is often affected in osteitis deformans and marked osteoarthrosis can then supervene. Aseptic necrosis of the medial condyle has been blamed as the cause of rapidly progressive, symptomatic osteoarthrosis in the elderly.[12–14]

The loss of articular cartilage, the structural changes of subchondral bone and the reactive osteophyte formation are the typical and well-known radiological appearances of progressive degenerative arthritis at the knee.[15] It may be localized at the lateral, medial or patellofemoral compartment or may be generalized throughout the joint (Fig. 52.3).

Although osteoarthrosis at the knee is considered very common, its prevalence is overestimated. The reason is quite simple: radiographs taken in a middle-aged or elderly patients complaining of knee pain will always show some evidence of cartilaginous degeneration. As age advances, some narrowing of the joint space and osteophyte formation always appear; they are normal and cause no harm.[16] Therefore the diagnosis of symptomatic osteoarthrosis should be made not by radiography but on the typical findings on clinical examination. A minor degree of osteoarthrosis does not give rise to symptoms and it is only when gross osteoarthrosis with

disintegration of the cartilage supervenes that the complaints can be attributed to it. What commonly, but incorrectly, is labelled 'osteoarthrosis' is a monoarticular steroid-sensitive arthritis or an impacted loose body. Both occur in middle-aged or elderly patients with osteophytes showing radiologically, and the diagnosis is missed if full clinical examination is not carried out properly.

The symptom of osteoarthrosis is pain during weight bearing, which disappears at rest. In gross arthrosis with complete erosion of cartilage, nocturnal pain from an increase of venous pressure in the tibial bone may sometimes supervene.

The characteristic clinical features in uncomplicated osteoarthrosis of the knee joint are a cold joint, devoid of synovial thickening, with limitation of movement in the capsular pattern.

The end-feel is bony and hard. In severe osteoarthrosis, the marked limitation of movement in a capsular pattern is accompanied by loud creaking of bone against bone.

However, the typical capsular proportion of limitation may be absent if the osteoarthrosis, although severe, remains localized. This is often the case in localized arthrosis of the patellofemoral compartment: passive flexion may be markedly limited and painful but passive extension remains normal. In this case, the flexion–extension movement is accompanied by rough crepitus.

The treatment of osteoarthrosis is mainly with non-steroidal anti-inflammatory drugs (NSAIDs) and intra-articular injections with steroids. NSAIDs provide some relief of the symptoms but have clinically important side effects – they are estimated to cause 3300 deaths annually among the elderly in the US.[17] Intra-articular injections with steroids also improve symptoms but the benefits are usually short-lived[18,19] and there is always the potential for long-term joint deteriorations if injections are repeated too often. During the last decade, a number of promising reports on the effect of intra-articular injections with hyaluronic acid have been published. Hyaluronic acid is an important component of healthy synovial fluid and cartilage tissue; it is thought to protect the articular cartilage and soft tissue surfaces of the knee by acting as a lubricant and, because of its high viscosity, by imparting viscoelastic properties to the joint. Because intra-articular hyaluronic acid concentrations are lower in the synovial fluid of arthrotic knees,[20] intra-articular injections with hyaluronic acid have been proposed as a means of restoring the viscoelastic properties of the knee joint, thereby providing symptom relief and improving joint function. Efficacy and safety of hyaluronic acid have been compared with that of saline,[21–23] corticosteroids[24] and NSAIDs. All the double-blind and randomized studies on the effect of hyaluronic acid on osteoarthrosis of the knee confirm that five intra-articular injections of sodium hyaluronate at weekly intervals are superior to placebo and are well tolerated in patients with osteoarthrosis of the knee, conferring a symptomatic benefit which persisted for 6 months.[25–27] As hyaluronic acid seems to provide beneficial effects with minimal adverse reactions in a significant number of patients, it may be considered as a substantive addition to the therapeutic armamentarium in osteoarthrosis.

When the condition is too advanced, joint replacement is the only solution.

Monoarticular steroid-sensitive arthritis

Cyriax described a monoarticular arthritis in the knee having some similarities with monoarticular arthritis in the shoulder and elbow. The cause and pathogenesis of this arthritis remain unclear but it heals immediately and lastingly after two injections of triamcinolone, thus earning it the title of 'steroid-sensitive' arthritis (Cyriax[28]: p. 404).

The patient describes a gradual onset of unprovoked swelling in one or both knees. At first, the swelling is completely painless. After some weeks or months, the knee starts to hurt all over. The pain is not provoked by movement and is often nocturnal.

In the initial stage, clinical examination shows only some diffuse warmth, fluid and synovial thickening. These marked symptoms and signs contrast strongly with the functional examination, which is almost completely normal. Only in advanced cases does a clear capsular pattern supervene.

Differential diagnosis

The differential diagnosis of a monoarticular steroid-sensitive arthritis, summarized in Table 52.1, must be made from the following conditions[29]:

- *Polyarthritis*: in monoarticular steroid-sensitive arthritis, disease does not spread to other joints and the laboratory findings remain normal, distinguishing it from rheumatoid and reactive arthritis.[30,31]

Table 52.1 Differential diagnosis of a capsular pattern at the knee

Type	Disorder	Signs/symptoms
Monoarticular arthritis		
Acute onset	Haemarthrosis	Trauma Haemophilia Spontaneous
	Septic arthritis	Intra-articular injections? Predisposing factors? Fever–illness
	Gout/pseudogout	Typical X-ray Typical aspirate
Slow onset	Steroid-sensitive	Typical history Synovial thickening
	Arthrosis	Hard end-feel No fluid No synovial thickening
Twinges	Loose body complicating arthrosis	
Polyarticular arthritis		
Symmetrical distribution	Rheumatoid arthritis	
Asymmetrical distribution	Reactive arthritis	

- *Villonodular arthritis of Jaffé and Lichtenstein*[32] is an advanced arthritis, with marked synovial hyperplasia and thickening of the capsule.[33] This type of arthritis is frequently complicated by spontaneous and recurrent haemarthrosis.[34,35] In doubtful cases, arthroscopic biopsy is indicated.[36] Treatment with steroid injections usually fails. Synovectomy is then advised.[37] Good results have also been reported with intra-articular injections of thiotepa.[38]
- *In middle age, a loose body complicating osteoarthrosis* may be very difficult to distinguish. The distinct features of a loose body are sudden twinges, localized warmth and absence of synovial thickening. In long-standing arthritis, secondary wasting of the quadriceps muscle may make the knee feel weak and climbing stairs difficult; consequently, a complaint of twinges is not always a good sign. The only certain criterion may then be synovial thickening, which is not always ascertained easily, especially in middle-aged women, whose subcutaneous tissues are already somewhat thickened. In such circumstances, manipulation for a suspected loose body can be tried; if several attempts do not succeed, the joint should be injected.
- *Gout and pseudogout* have an acute onset with an immediate and gross articular pattern.

Treatment

The treatment of monoarticular steroid-sensitive arthritis at the knee is two injections of 40 mg of triamcinolone at an interval of 2 weeks (Fig. 52.4).

Technique: injection for monoarticular steroid-sensitive arthritis

The patient lies on the couch and relaxes the quadriceps muscle (Fig. 52.4a). If gross arthritis is present, it may be necessary to place a small cushion under the knee in order to keep it in slight flexion. The operator stands level with the other knee. With one hand the patella is pushed up and towards the operator, which makes the medial edge more prominent. A 5 mL syringe is filled with steroid suspension and fitted with a thin needle, 4 cm long, which is placed near the upper border of the patella and thrust in horizontally just between the patellar edge and the medial femoral condyle (Fig. 52.4b). It lies intra-articularly at about 2 cm depth.[39]

Crystal synovitis

Gout

An acute arthritis at the knee occurs in 25% of all attacks of gout.[40] There is considerable pain, swelling and redness, which makes the condition difficult to differentiate from septic arthritis, haemarthrosis or pseudogout. Sometimes analysis of the joint fluid is necessary to distinguish these conditions.

Pseudogout

Calcium pyrophosphate crystals are believed to induce the synovitis of pseudogout. The term chondrocalcinosis refers to the presence of these calcium-containing salts in the articular cartilage,[41] which are seen on radiographs.

The disease may be familial and is also commonly associated with a wide variety of metabolic disorders – for example, haemochromatosis, hyperparathyroidism, ochronosis and diabetes mellitus – and is also found in patients undergoing dialysis.[42,43]

As a rule, patients are over 60 years of age. Ninety percent of attacks of pseudogout are localized to the knees and there

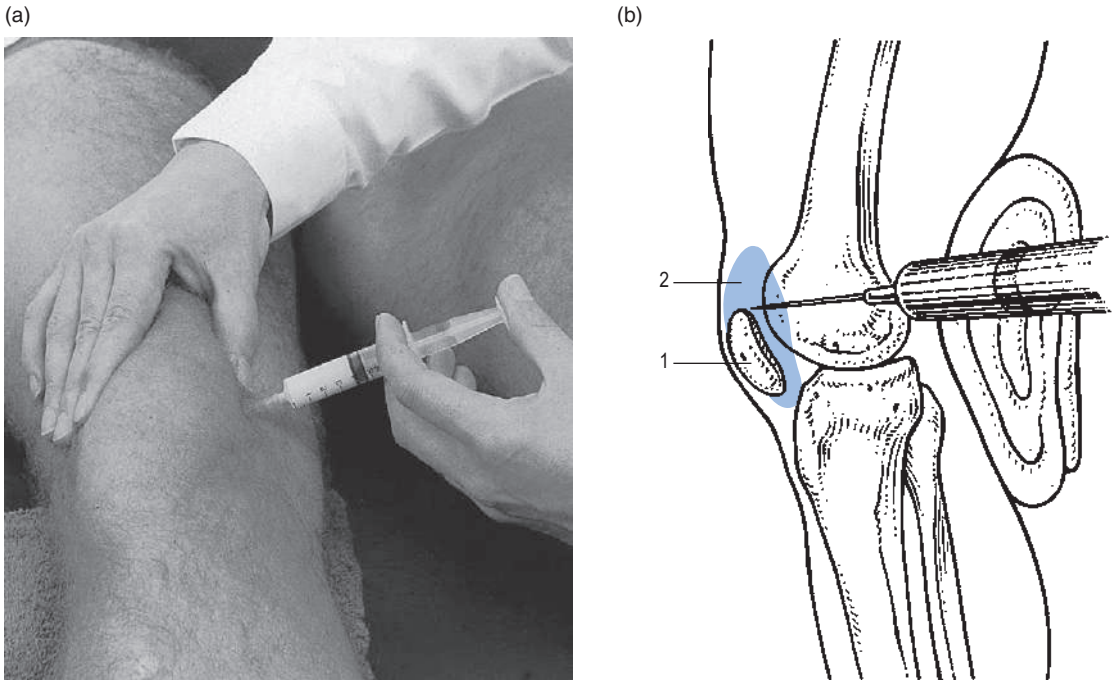


Fig 52.4 • Intra-articular injection of the knee. Structures in (b): 1, patella; 2, suprapatellar pouch.

is an acute and recurrent arthritis which, untreated, lasts 1–4 weeks. Plain radiography may show calcification of the articular cartilage or menisci. Diagnosis is by aspiration, which demonstrates calcium pyrophosphate crystals in the fluid.

Pseudogout is best treated with an immediate intra-articular injection of triamcinolone. Alternatively, some of the oral anti-inflammatory agents can be prescribed.

Haemarthrosis

Post-traumatic haemarthrosis

This usually indicates a serious lesion, most commonly rupture of a cruciate ligament or an intra-articular fracture (see p. 599).[44,45] Sometimes it is the result of a direct capsular contusion.[46]

The patient states that, after the injury, the knee became painfully swollen within 2–3 minutes. The speed of swelling, together with the severe pain, indicates an intra-articular haemorrhage; blood fills the joint at once and is a strong irritant. Blood left within the joint for weeks can cause severe arthritis and serious erosion of the articular cartilage. It should be removed at once.[47–49] After a few days, it may be necessary to aspirate the remaining blood-tinged synovial effusion.

Haemophilia

About half of all haemophiliac articular effusions occur at the knee joint. The patient is typically an adolescent who states that suddenly, and without apparent reason, the knee became swollen and very painful. Clinical examination shows a hot, swollen knee, with a severe capsular pattern (90° limitation of flexion and up to 70° limitation of extension). Even if the patient is seen some weeks after the onset, limitation of movement remains unaltered. When the tendency to bleeding is only minor, the patient is probably not known to be a haemophiliac, but the acute onset in the absence of a causative injury, the hot, swollen joint and the extreme capsular pattern should arouse suspicion. Aspiration will reveal a haemarthrosis but the diagnosis must be confirmed by haematological investigations.

Spontaneous haemarthrosis in the elderly

Sudden pain and swelling without previous injury in an elderly patient will often be caused by an intra-articular haemorrhage, presumably as the result of the rupture of an intra-articular vein. Haemarthrosis should be suspected particularly when the patient is on anticoagulant therapy.[50,51]

Treatment is aspiration, repeated after a few days.

Villonodular synovitis

Haemarthrosis can occur in villonodular synovitis.[52,53] Localized pigmented villonodular synovitis is a rare lesion that may affect any joint but is most often found in the knee. Treatment usually involves arthroscopic resection of the lesion.[54]

Septic arthritis

Bacterial infection of the knee is an extremely serious disorder. If it is not properly treated, it will not only lead to total destruction of the joint but can also be life-threatening.

Septic arthritis can follow haematogenous dissemination from focal infections, such as urethritis, cystitis, skin infections[55] or dental abscesses. This is particularly so when resistance is decreased: diabetes, rheumatoid arthritis, renal failure or a deficient immune system are all possible causes of decreased resistance.[56] Intravenous drugs and gonococcal infections, particularly with antibiotic-resistant gonococci,[57] have also been blamed for the increased incidence of septic arthritis of the knee. Another cause of septic arthritis is direct inoculation of organisms during intra-articular injection.

The symptoms of septic arthritis are hyperacute pain, swelling, redness and a gross articular pattern. The local symptoms are accompanied by high fever, chills and nausea.

Treatment is with high doses of antibiotics intravenously and daily aspiration of the knee.[58]

Non-capsular pattern

Internal derangement

Introduction

Impaction of loose tissue within the joint cavity, causing mechanical interference with the normal movement between femur and tibia, is usually referred to as 'internal derangement of the knee'. The loose tissue consists of meniscal parts, cartilaginous tissue peeled off from meniscus, joint cartilage or bony fragments, as is the case in osteochondritis dissecans.

Symptoms and signs in internal derangement are quite typical. The pain is localized, of the mechanical type and has a sudden onset. 'Locking of the joint' and 'twinges' are typical sensations. The former describes sudden and painful limitation of one movement; the latter is abrupt, unforeseen and sharp pain that appears during weight bearing.

The clinical signs are a non-capsular pattern and a typical bouncing or springy end-feel. The latter is typically found during passive extension.

A thorough clinical assessment can usually provide sufficient information to make a definitive diagnosis of internal derangement.[59] Plain radiographs are of no use in the diagnosis of internal derangement. Arthroscopy may be useful but should never be performed without first completing a full preoperative examination.

Lesions of the meniscus

These can be grouped into congenital anomalies, traumatic conditions, cysts and metabolic disorders.

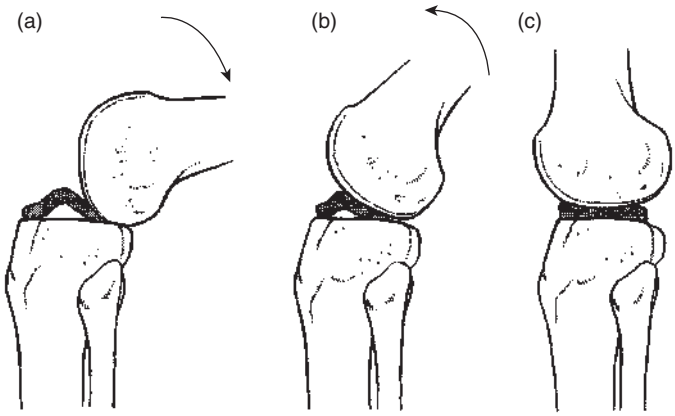


Fig 52.5 • Discoid lateral meniscus: the meniscus which rolls up in flexion (a) is trapped during extension (b) until it reduces with a 'snap', allowing full extension (c).

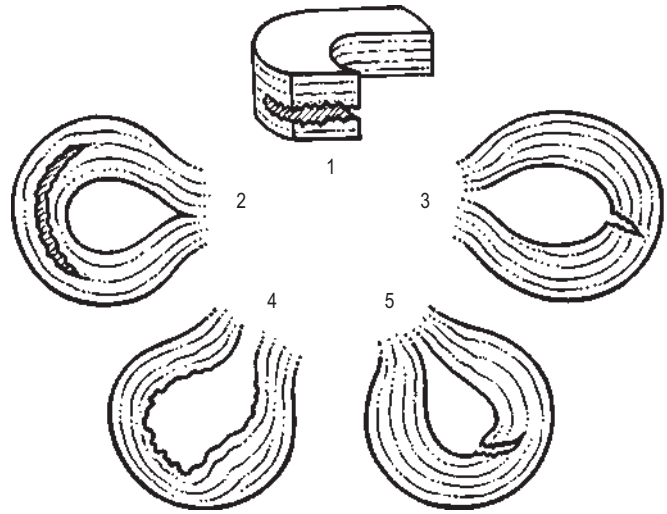


Fig 52.6 • Main types of meniscal tear: 1, horizontal; 2, longitudinal; 3, radial; 4, degenerative; 5, flap.

Congenital anomalies

The most common congenital anomaly with pathological sequelae is a discoid lateral meniscus. The incidence in autopsy specimens is between 0% and 7%,[60] but most do not cause problems.[61] Symptomatic disorder seems to occur only in the Wrisberg ligament type of discoid meniscus. This type is classically described as lacking a posterior capsular attachment and presents in childhood with a 'snapping' knee: the meniscus rolls up in front of the lateral compartment during flexion, as a consequence of increased friction and greater mobility; during extension, the rolled meniscus becomes trapped anteriorly, until it reduces suddenly with a dramatic audible snap (Fig. 52.5).[62]

The disorder affects children and adolescents. The presenting symptom is internal derangement at the lateral side of the knee. Clinical examination reveals an audible click and visible alteration of the knee at approximately 10–20° of extension.[63] Diagnosis is confirmed with sonography or magnetic resonance imaging (MRI).[64]

Treatment of a congenital discoid meniscus is complete or partial meniscectomy.[65]

Traumatic meniscal lesions

Disruption of collagen fibres within the meniscus occurs as the result of either an acute injury or gradual age-dependent degeneration (Fig. 52.6). The split may be either horizontal or vertical.

Vertical tears

Vertical tears are the most important clinicopathological condition of the meniscus and usually result from excessive force applied to a normal meniscus. Transverse tears are less common than longitudinal ones. The latter frequently involve the thin inner edge of the meniscus, which, when it separates, creates the bucket-handle tear, so characteristic of the locking knee.[66] Peripheral longitudinal tears are defined as those occurring within 3 mm of the meniscosynovial junction and comprise about 30% of all vertical tears.[67] Left untreated, most of these peripheral lesions heal spontaneously.[68]

The less mobile medial meniscus is much more commonly involved than the lateral meniscus. The medial meniscus is closely bound to the medial collateral ligament and the medial coronary ligament, which attaches the meniscus to the tibia, is only 4–5 mm long. The lateral meniscus, however, is separated from the lateral collateral ligament by the popliteus tendon, and its coronary ligament is much longer (13–20 mm).[8] This difference in mobility is the reason for the higher proportion of torn medial menisci.

The typical vertically torn meniscus, which results in a bucket-handle lesion, occurs between the ages of 15 and 30 years. The mechanism of injury is as follows: during flexion–extension, the menisci move with the tibia, to which they are attached (see online chapter *Applied anatomy of the knee*), but during rotational movements they follow the femoral condyles. During combined movements, the normal displacement of both menisci may be prevented and the menisci become trapped between tibia and femur. This typically happens in a football player who, in an attempt to kick the ball sideways, severely twists on the slightly flexed and weight-bearing knee: the rotation force of the femur on the immobilized tibia then ruptures the cartilage. It has also been suggested that meniscal lesions may appear as the result of knee instability, in particular after anterior cruciate lesions.[69,70]

The typical history of a bucket-handle lesion is that of a patient, usually a male soccer player aged between 16 and 30 years, who felt agonizing and localized pain, usually at the inner side of the knee, during a vigorous rotation with weight bearing. The knee gave way and the patient fell to the ground. When he stood up again, the knee could not be straightened; although it could be bent, it was impossible to force it into extension. Full extension was only regained when either the patient, the trainer or the doctor forced the knee to unlock by a combined rotational and extension movement. After an audible click, full extension immediately became possible again. After reduction, the knee became swollen and sore for a couple of days. After a week or two, the patient may then find the knee is 'cured', providing it is not twisted while weight bearing. Should that

happen, something painfully 'going out' in the joint would be felt and, as before, straightening the joint would not be possible. Again, the limitation of movement disappears only if the knee is manipulated.

The biomechanical basis for the locking is that, in a normal knee, the collateral ligaments and the posterior aspect of the joint capsule all become taut during extension. When the slack in the ligaments has been taken up, the tibia and femur are strongly approximated and extension beyond 180° is prevented. At full extension, there is no room for a displaced piece of cartilage between the two bones and therefore the movement must be somewhat limited by its presence. Sudden locking of the knee in partial flexion, with immediate unlocking on manipulation, is therefore pathognomonic of a bucket-handle tear.

Horizontal and posterior cracks

It is generally accepted that these lesions result from normal forces acting on a degenerating meniscus.[71] Degenerative horizontal cleavage lesions are therefore more common in individuals of more than 40 years of age and usually occupy the posterior half of the menisci.[72,73] They are so common that most authors regard them as part of the usual degenerative processes in the knee (Noble and Turner,[74] Fahmy *et al*,[75] Smillie[9]: pp. 79, 97, 108, 145, 153).

In a posterior crack, the history is less dramatic than in that of a vertical tear.[72] For instance, the patient finds that, if there is a twist on the knee, something is occasionally felt to 'give way', usually at the back of the joint. It is immediately difficult to straighten the leg, but when it is shaken or kicked out, a click is heard and the limb returns to normal function. Sometimes the patient gives a different history: slight rotation and flexion during weight bearing – for example, on getting out of a car – produce an uncomfortable click which is relieved immediately on straightening the leg.

Diagnosis

Ruptures with displacement

In a longitudinal vertical tear, the history is most informative: the patient states that on twisting the leg during weight bearing a click is heard, localized unilateral pain is felt and the knee immediately locked in flexion. All unassisted attempts to straighten the knee fail.

The patient enters the room with a characteristic gait: hopping on one leg with the knee on the affected side flexed and the foot plantiflexed with the toes just touching the ground.

Clinical examination reveals a warm and slightly swollen joint. Flexion is normal or somewhat limited by the traumatic arthritis but when extension is attempted, limitation of 5–10°, with a characteristic springy block, is detectable. Rotation away from but not towards the affected side is painful.

Palpation usually reveals a very tender joint line at the affected side, because of a coincident sprain of the coronary ligament.

When a middle-aged patient presents with a posterior displacement, extension may be just possible but very painful, and once again the end-feel of a springy block is informative. In this type of lesion, tenderness is not found on palpation.

In a horizontal lesion, a visible and palpable 'tag' can form, which then projects at the joint line. Often digital pressure will suffice for reduction but this is seldom permanent.

Ruptures without displacement

If the patient is seen some time after reduction of the meniscus, the knee may appear normal or merely shows a sprained coronary ligament. Alternatively, the patient presents with a history of a minor posterior crack but the routine functional examination is normal. In these cases, the following tests can be used to elicit signs of a ruptured meniscus (Fig. 52.7).

Test 1. The knee is fully flexed. The examiner holds the index finger and thumb at both sides of the patient's infrapatellar

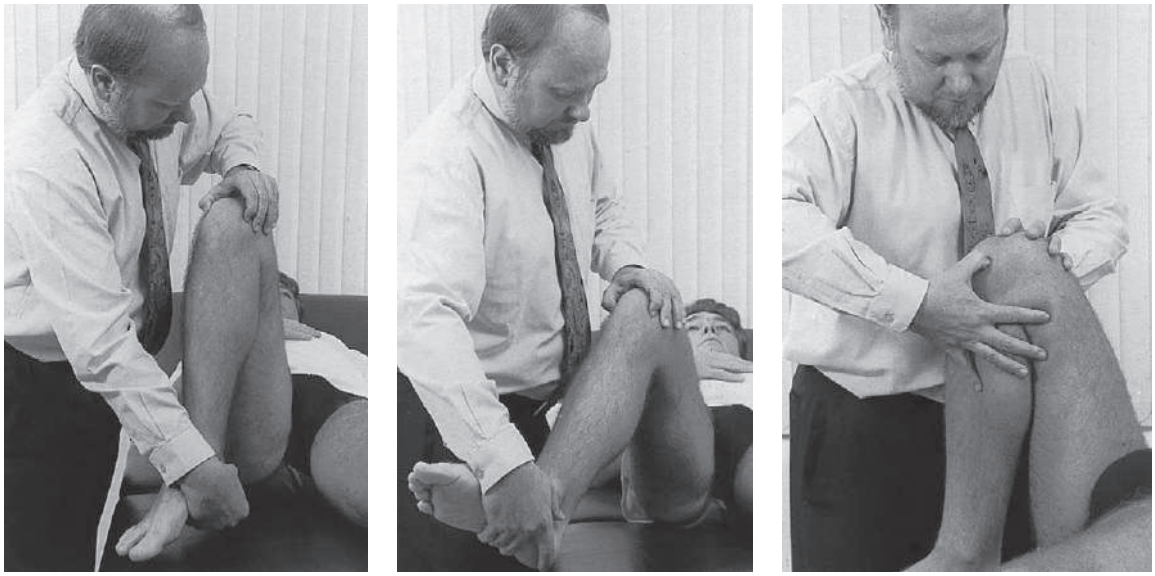


Fig 52.7 • (a) Test 1: to detect clicks during rotation in full flexion. (b) Test 2: to detect a click during extension movement under external rotation. (c) Test 3: palpation of a displaced rim.

tendon, level with the joint line. With the other hand, the heel is grasped and the leg rotated quickly to and fro. When clicks are felt at the joint line, a ruptured meniscus should be suspected.

Test 2. The knee is fully flexed and rotated. One hand is placed on the knee, the index finger at the side to be tested and level with the joint line. The examiner now slowly extends the patient's knee, while the pressure maintaining rotation is maintained. As extension proceeds, a click indicating a meniscal tear may be felt, usually as the leg approaches the neutral position at almost full extension. This test can also be performed during varus or valgus compression and is then called the McMurray test.

Test 3. The knee is held well flexed. The examiner passes the flexed thumbtip from above downwards over the joint line at the affected side. A ruptured meniscus should be suspected when it is possible to hook the rim of the meniscus and pull it downwards until it jumps back into place again. Normally this is accompanied by an audible click.

Test 4. Medial and lateral shearing strain has been discussed in the chapter on clinical examination (p. 670–671). The knee is flexed at a right angle. The examiner sits at the foot of the couch and interlocks the fingers on the patient's knee. A strong shearing strain can now be exerted to move the tibia laterally or medially on the femur. This manœuvre may elicit pain or even displace the loose part of the meniscus to the other side of the condyle. A loud click is heard, with immediate loss of full passive extension. A torn meniscus is obvious, in that the test has provoked a subluxation. Manipulative reduction must follow, of course.

Test 5. If there is a horizontal split, a 'tag' of cartilage can be felt to protrude at the joint line, and can be pushed back into place by the palpating finger. The discomfort on full extension then immediately ceases but recurrences are common. If this is the case, the patient can learn to perform the 'manipulation' themselves.

Arthroscopy

During recent decades, arthroscopy has made the diagnosis and treatment of a torn meniscus much easier.[76,77] Nevertheless, arthroscopy does not make a clinical diagnosis redundant and routine arthroscopy without a previous thorough functional examination can lead to wrong conclusions and treatment. Because meniscal lesions are so common that they are a frequent finding in middle-aged people, it is very possible that a meniscal tear, found incidentally during 'routine' arthroscopy, is not the cause of the patient's complaints. For instance, if a patient with a chronic ligamentous lesion of the medial collateral band or the coronary ligaments is subjected to arthroscopy before a clinical diagnosis has been made, the damaged meniscus will be blamed and thus unnecessarily removed.

The use of the arthroscope has encouraged the widespread assumption that the source of chronic pain in the area of the knee is to be found in the interior of the joint itself. The more likely cause of pain, however, is in the surrounding ligaments, but this can only be demonstrated by a thorough clinical assessment.[78] We, like Goodfellow ('He who hesitates is saved'),[79] believe that, although meniscal tears can be detected by arthroscopy with almost 100% accuracy, caution is needed in ascribing symptoms at the knee to meniscal lesions. Diagnosis of meniscal lesions should be made clinically and arthroscopy should be reserved for confirmation of clinical findings as an aid in therapeutic decision making.

Treatment

Manipulative reduction

Manipulation should be tried at once in a patient who presents with a displaced meniscus. The manipulation is not completely painless and anaesthesia may sometimes be required, especially if the patient is tense or anxious. Although some authors advise general anaesthesia, sufficient pain relief can be obtained after an intra-articular injection of 5 mL of lidocaine 2%.

Technique: starting position. The patient lies supine on the couch and flexes the hip and knee to right angles. Every effort must be made to encourage the patient to relax the muscles as much as possible.

In a displaced medial meniscus, the manipulator must open up the inner aspect of the knee as far as possible. Therefore, strong valgus pressure must be exerted during the whole procedure. In order to achieve this, one hand is placed at the outer side of the knee, so that the thumb lies at the back of the knee. This thumb position is important in controlling extension during the preparatory phase of the manipulation. The other hand encircles the heel to rotate the leg to and fro during the manipulation.

Technique: preparative phase. The hip is slightly rotated medially and strong valgus pressure is exerted at the knee (Fig. 52.8). In this position, repeated extensions are performed but not beyond the end of range – i.e. if the extension range is limited by 10°, the movement should stop at about 15°. In order to ensure this, the thumb placed in the popliteal fossa is used as a brake during the procedure. Once flexion and extension movements have been performed several times in this way, rapid to-and-fro rotations of the knee are added.

Technique: manipulation. The movements are now performed rapidly and repeatedly. When the patient is most relaxed, one quick thrust towards further extension is added. This is achieved by strong, swift, downward and inward pressure of the hand at the knee joint at the end of the extension. As the full range of extension is reached, reduction is signalled by a small click, at which point extension at the knee immediately becomes free.

If symptoms of a sprained coronary ligament persist after successful manipulation, deep transverse friction should also be applied. Further splinting or immobilization, in order to heal the tear, is of no use.

Natural history

The meniscal lesion itself causes no pain because the body of the meniscus is almost completely insensitive. Pain is the result of ligamentous strain caused by the repeated luxations and subluxations. However, damaged menisci frequently do not provoke any symptoms. In a postmortem study in patients above and below the age of 50, Noble and Hambden found that up to 60% of the former and 19% of the latter had meniscal tears.[80,81]

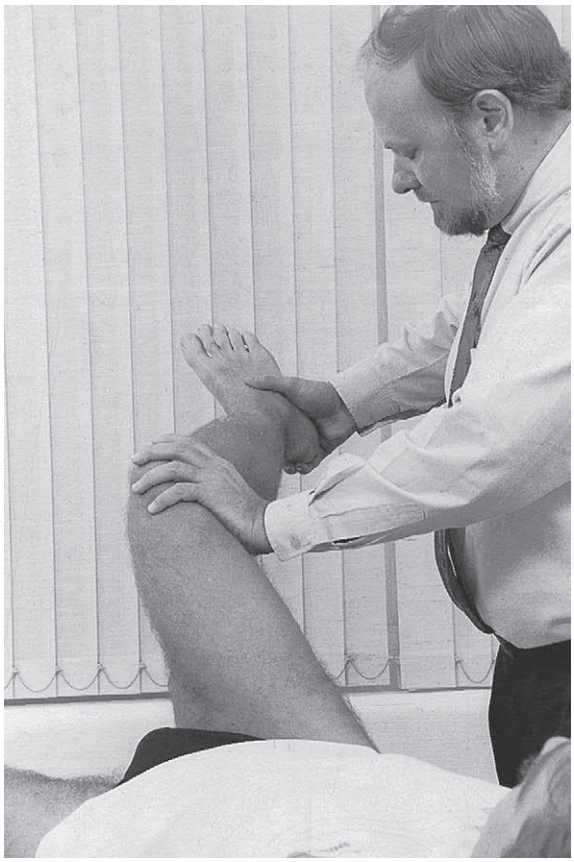


Fig 52.8 • Manipulative reduction of a displaced medial meniscus.

It is important to realize that most complaints following subluxation of a meniscus stem from a sprained coronary ligament. If a patient with a history of a former locked meniscus complains of persistent and localized pain but proves to have a full range of movement with a normal end-feel, a sprained coronary ligament should be suspected and appropriate treatment given. After successful manipulation, the reduced piece of cartilage may never subluxate again and the patient is cured for good. This was emphasized by Casscells,[82] who stated that not all meniscal tears need surgical treatment. Asymptomatic meniscal tears identified at the time of arthroscopic examination for ligamentous lesions are best left in place or unrepaired, as they will remain asymptomatic.[83] Furthermore, laboratory studies have demonstrated that torn menisci may have normal biomechanical function if the peripheral circumferential fibres remain intact.[84,85]

Surgery

Although surgery of the meniscus should not be undertaken lightly, it is unwise to permit repeated attacks of internal derangement because they initiate a premature, troublesome and intractable osteoarthrosis.[75,86]

The choice lies, then, between total or partial meniscectomy or repair. Total meniscectomy leads to loss of integrity of the articular cartilage and impairment of joint stability.[87–91] It is no longer considered the treatment of choice because of increasing dissatisfaction with the long-term results.[10,92] Instead, and if this is technically possible, only the loose fragment is removed and the outer part of the meniscus, together with the coronary ligament, is left alone.[93] The result is no increase in weight-bearing stress on the tibia.[94] The outer rim of the meniscus can still bear weight and thus preserve the articular cartilage, as well as play a useful role in stabilizing the joint. In experienced hands, this partial removal of the meniscus using arthroscopy is much less damaging to the joint and requires only 2 days' bed rest. Subsequent return to work and sport is also quicker.[95] Several long-term studies have demonstrated the technique's superiority over total meniscectomy.[96–100]

In recent years, suturing of the relatively vascular outer edge of the meniscus has been shown to result in healing by fibrous tissue.[101–107] Meniscal repair is nowadays considered as the treatment of choice in single, vertical, longitudinal tears in the outer third of the meniscal substance.[108] Good to very good long-term results may be expected if the longitudinal tear is less than 3 cm long and within 3 mm of the periphery, and the meniscal tissue is not degenerating.[109,110] Ligamentous instability is a relative contraindication to repair. In combination with insufficiency of the anterior cruciate ligament, the rate of retearing approaches 40% and therefore anterior cruciate ligament reconstruction should be performed at the same surgical intervention.[111,112]

Cysts of the meniscus

When cysts form, they are almost always at the lateral meniscus; a cyst of the medial meniscus is extremely rare.[113] There is evidently a connection between cyst formation and the existence of ruptured menisci because half the cystic menisci are also torn.[114]

The history can be indicative of internal derangement or the patient may complain of localized pain when standing for some time.

Clinical examination reveals a normal range of movement and a normal end-feel. Ligamentous tests are negative. When the joint line is palpated during full extension, the cyst is felt there as a small firm mass that varies in apparent size depending on the position of the knee, usually being most prominent in 20–30° of flexion and disappearing on full flexion (Pisani's 'disappearing' sign).[115] Some tenderness is elicited at the joint line and over the cyst. The diagnosis can be confirmed with MRI.[116]

Treatment is aspiration of the cyst. It is important to use a large-bore needle and to insert it in several different directions, as the cyst is often multilocular.[117] Arthroscopic treatment is used if the cyst recurs.[118–120]

Metabolic disorders affecting the meniscus

The most important metabolic condition affecting the menisci is chondrocalcinosis. A recent random autopsy study found calcium pyrophosphate dihydrate crystals in 7% of the menisci examined.[121] Chondrocalcinosis of the meniscus is considered in the previous section of this chapter ('Capsular pattern').

Loose body in young patients

Loose bodies, which are often multiple, can form in the knee in teenagers and young adults. They are usually the result of

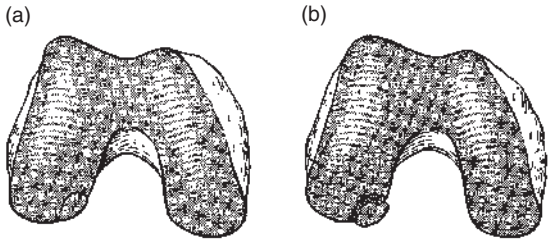


Fig 52.9 • Stable osteochondral lesion (a) and loose fragment (b) at the lateral aspect of the medial femoral condyle of the left knee (interior view).

osteochondritis dissecans but sometimes originate from chondromalacia patellae or a small osteochondral chip fracture.

Osteochondritis dissecans is well known, although the true cause is still not completely understood. In those under the age of 13 years, the condition seems to result from an abnormality of ossification. In teenagers and adults, however, trauma may have a significant part to play in the development of the lesion.[122,123] More than 75% of the osteochondral lesions are on the lateral side of the medial femoral condyle.[124] About 10–15% are situated at the lateral femoral condyle and in less than 10% of the patients the lesions are bilateral.[125]

Few symptoms result, as long as the osteochondral lesion remains stable and embedded in the condyle (Fig. 52.9). There is some vague pain during weight bearing which can also be elicited during pressure over the medial condyle. Symptoms of intermittent pain and locking of the joint occur only when the fragment has become loose, which typically happens between 16 and 20 years of age.

The history is of a sudden and temporary *locking in extension*. The patient finds that, from time to time while walking along, the knee suddenly locks. If this happens while doing sports, a fall to the ground is usual because the knee is locked straight when the patient expects to be able to bend it. When flexion is attempted again, this is successful and walking on it is immediately possible. Some aching and traumatic arthritis result for the next few days. Recurrence of this sequence is common and the location of the pain may vary, indicating that the loose body has shifted to another part of the joint.

After the attack of internal derangement, there are signs of a traumatic arthritis only: the knee is warm, contains some fluid and shows a slight capsular pattern. The ligamentous tests are normal and there is no local tenderness. Sometimes the loose body can be felt in the suprapatellar pouch. Wilson[126] describes a test that aids in the diagnosis. The knee is flexed to a right angle and the tibia rotated internally; the examiner then slowly extends the patient's knee. At about 30° of extension, pain may occur, presumably arising from the lesion at the inner condyle pressing against the tibial spine.

Loose bodies have an osseous nucleus and therefore can be seen on a radiograph. In true osteochondritis dissecans, the gap at the lateral side of the medial femoral condyle is also seen on an anteroposterior and a tunnel view.

Stable osteochondritis dissecans has a successful healing rate of 50% after 6 months of relative rest.[127] However, because the chance of spontaneous healing is low for juvenile osteochondritis dissecans lesions that present with disruption of the articular cartilage surface, and the risk of further damage is high, most surgeons recommend surgical stabilization of all unstable juvenile osteochondritis dissecans lesions at the time of presentation.[128] The method used depends on the size of the defect and the congruity of the fragment with its bed. Sometimes replacement is tried, and sometimes removal of the loose fragments by arthroscope.[129]

The differential diagnosis and treatment of loose body and meniscal tear in a young patient are summarized in Table 52.2.

Table 52.2 Differential diagnosis and treatment of loose body and meniscal tear in a young patient

	Loose body	Vertical tear of the meniscus
Age group	Teenager or young adult	Young adult
Locking	In extension	In flexion
Unlocking	Spontaneous	Manipulative
Examination findings	Traumatic arthritis Positive radiograph	After unlocking: Traumatic arthritis Sprained coronary ligament During locking: Limited extension Springy block
Treatment	Surgery	Manipulative reduction (Deep friction to coronary ligament) Surgery

Loose body complicating osteoarthrosis

Cyriax[28] (his p. 403) pointed out that:

> When a middle-aged or an elderly patient presents with signs of a ligamentous sprain, or a traumatic arthritis, and there has been no injury, it is highly probable that he suffers from a loose body.

Contrary to popular belief, minor or moderate osteoarthritis at the knee does not cause symptoms.[130] However, if there is some crepitating osteoarthritis, the articular cartilage is roughened. A small piece may then exfoliate. Alternatively, a small piece of degenerated meniscal cartilage may peel off and escape into the joint space. Such a loose fragment can occupy a harmless position at the back of the joint, or in the suprapatellar pouch, but sometimes impacts between the articular surfaces, where it occupies space, induces overstretching of the ligaments and causes localized pain.

History

The history of a loose body is typical. The patient is middle-aged or elderly and states that, for no reason, swelling

and *localized* pain appeared in one knee, usually at the medial side. Pain may be present on waking or suddenly each step hurts when walking. Sometimes pain is felt at the lateral side, or right in the joint, but *never all over the joint*. Curiously, the pain can sometimes spread up to the distal part of the thigh or down to the proximal part of the leg. If the pain moves from one side of the joint to the other, the diagnosis is obvious because moving pain suggests a moving (and free-lying) lesion.

The patient is hesitant to go downstairs for fear of a sudden twinge which makes the knee give way. Therefore descent is usually one step at a time, firmly holding on to the banister. Less often, the twinges and the feeling of instability are experienced during ordinary walking. Twinges indicate a momentary impaction in the knee joint and in middle-aged people they are almost pathognomonic of the existence of a loose body.

Sometimes pain also occurs at night; this is likely to happen when it is at the medial side of the knee. One possible explanation could be local tenderness at the medial collateral ligament when the knees are held together while the patient lies on the side.

Clinical examination

The signs vary with the position of the loose fragment within the joint but there are some that are always present and draw attention to the condition:

- *Localized warmth*: There is localized warmth on the painful side of the joint. If the joint is not warm at the beginning of the clinical examination, the examiner proceeds with the functional tests and repeats the palpation at the end. Local heat is then found which has been provoked by the minor stresses imposed on the joint during examination.
- *Fluid*: often, some fluid is present.
- *Non-capsular pattern*: there is usually a non-capsular pattern. It is obvious that when extension is limited by, e.g. 5° and flexion is not, or flexion is 30° limited but extension is of full range, a block due to internal derangement should be suspected. Such an obvious non-capsular pattern is rather uncommon and, as a rule, the signs are quite subtle. Extension is full but painful and has a smoother end-feel than on the opposite side, while flexion is full and painless. Alternatively, full extension is free from pain but flexion is slightly limited, with localized pain and a soft end-feel. In other words, the stop is not hard, as if muscle spasm due to arthritis was present, but feels as if it would go further, the limiting factor being pain.
- *A varus movement hurts at the inner side of the joint*: this indicates that a space-occupying lesion is present in the medial compartment. Alternatively, lateral or medial shearing strain may hurt at the inner or the outer side of the joint. Care should be taken not to press on the tender medial collateral ligament during these tests.
- *Localized pain and positive ligamentous tests*: there is localized pain at the end of range and some ligamentous tests are positive. As a rule, valgus strain and external rotation hurt at the medial side. The medial collateral ligament is also very tender to the touch. Usually these findings, together with localized warmth, fluid and absence of synovial thickening, are characteristic of a sprained ligament. However, the patient does not mention trauma. The conclusion is that, if the ligament is not being strained by external forces, the cause of the sprain must lie within the joint: the ligament becomes sprained when a small cartilaginous loose body is displaced between femoral condyle and tibial plateau, where it will put stress on the ligaments on every attempt to extend the joint. Cyriax called this 'a sprain without a sprain'.

If, after the clinical examination, the conclusion is that there is an impacted loose body, an attempt should be made to move the piece of cartilage to a site where it no longer interferes with joint mobility. Confirmation that the diagnosis is correct comes from the immediate disappearance of symptoms and signs after manipulation.

Special investigations

The knees of elderly patients always show radiological evidence of osteoarthrosis. Loose bodies, however, usually being composed entirely of cartilage, are not visible on radiography. When the radiograph shows osteoarthritis, the current tendency to depend on diagnosis by such means rather than by good clinical assessment can result in the clinical features being ignored and the patient being erroneously regarded as suffering from osteoarthrosis of the knee. The acute onset and the warmth are then mistakenly labelled as 'acute episodes' of progressing osteoarthrosis.

It is also apparently very difficult to see small loose bodies during diagnostic arthroscopy. It is striking, however, to hear that surgeons often see an improvement of symptoms and signs in an 'arthrotic' knee after such a procedure.[131–133] In all likelihood, the loose bodies are washed out inadvertently during the drainage that is necessary for arthroscopic investigation and it is a consequence of this that the patient notices an improvement.

Differential diagnosis (Table 52.3)

Spontaneous osteonecrosis of the knee occurs in elderly patients, usually at the medial femoral condyle.[12] The condition causes continuous pain which gets worse at night, but twinges are not mentioned. Clinical and radiological examinations are negative during the first few weeks of the condition's development,[134–136] and early diagnosis must be made by a bone scan[137,138] or by the use of MRI. The treatment is proximal osteotomy or prosthetic replacement.[139]

The most common error, however, is to regard the patient with a loose body as suffering from osteoarthrosis or a monoarticular steroid-sensitive arthritis. Because all these lesions occur in middle age or in the elderly and the signs are sometimes subtle, the distinction is not always apparent. In case of doubt, it is always wise to manipulate the knee and see whether there is any improvement.

Table 52.3 Differential diagnosis and treatment of an impacted loose body complicating arthrosis at the knee

	Osteoarthrosis	Monoarticular steroid-sensitive arthritis	Loose body
History			
Onset	General pain throughout the joint during weight bearing	Gradual, with swelling and pain throughout the joint	Sudden, localized, with pain
Pain at rest?	No	Yes	Twinges/apprehension Sometimes localized pain
Examination			
Fluid	Absent	Present	Present
Warmth	None	Generalized	Localized
Synovial thickening	Absent	Present	Absent
Pattern	Slight capsular	Capsular (±)	Non-capsular
End-feel	Hard	Muscular spasm	Soft
Other	Crepitus		Painful varus or shearing strains Painful ligamentous tests
Treatment			
	Hyaluronic acid Surgery	Two injections with triamcinolone	Manipulative reduction

Treatment

As soon as impaction of a loose body in a knee is diagnosed, manipulation must be performed. The intention behind the manipulation is to move the loose body from its position between the tibial and femoral articular surfaces into a position in the joint where it is no longer pinched, i.e. under the posterior recess. To make room between the tibia and femur, the whole procedure is performed during strong traction.

First manipulation

The principles of performing a manipulation for a loose body are:

- Traction should be as strong as possible.
- The manipulation starts with the ligaments in a relaxed position (knee in flexion).
- During movement towards extension, the knee is rotated three or four times, first in one direction and then, if no improvement results, in the other.

Technique: starting position

The patient lies prone on a low couch, the knee flexed to a right angle. An assistant holds the thigh just above the popliteal fossa. The manipulator stands level with the patient's knee. The ipsilateral hand grasps the calcaneus. The other hand is placed at the dorsum of the foot in such a way that the second metacarpal bone presses against the neck of the talus (Fig. 52.10a). The pressure against the talus and the simultaneous traction on the calcaneus hold the foot in dorsiflexion during the whole procedure (Fig. 52.10b).

The manipulator places the contralateral foot on the couch, just in front of the patient's knee. The patient's thigh is lifted off the couch and the foot is hooked on the manipulator's knee. The assistant now presses the thigh downwards as hard as feasible. This ensures maximal traction on the relaxed ligaments and creates some distraction of the joint surfaces. This position is maintained for a few seconds until the manipulator feels the quadriceps muscle relax, and the tibia is distracted from the femur.

Technique: manipulation

The manipulator now removes the leg from the couch and, leaning sideways towards the end of the couch, places the foot as distally (in relation to the patient) as possible (Fig. 52.10c). By doing so, maximum traction is ensured during the whole procedure. Extension is accompanied by a series of full lateral or medial rotations (Fig 52.10d). To this end, both shoulders and elbows must be used in order to reach the very end of rotation. The end-feel will once again be a guide in deciding whether the knee is at full range. In practice, rotation is tried first in one direction and then, if this does not succeed, in the other. The assistant who is holding the leg down feels a click, indicating that reduction has taken place, but the manipulator does not feel anything. After each manipulation, whether the knee has clicked or not, the joint is examined again. If there is improvement after a particular manœuvre, the same manipulation is done again.

Results

If the manipulation only reduces an impacted loose body incompletely, then other techniques must be tried.

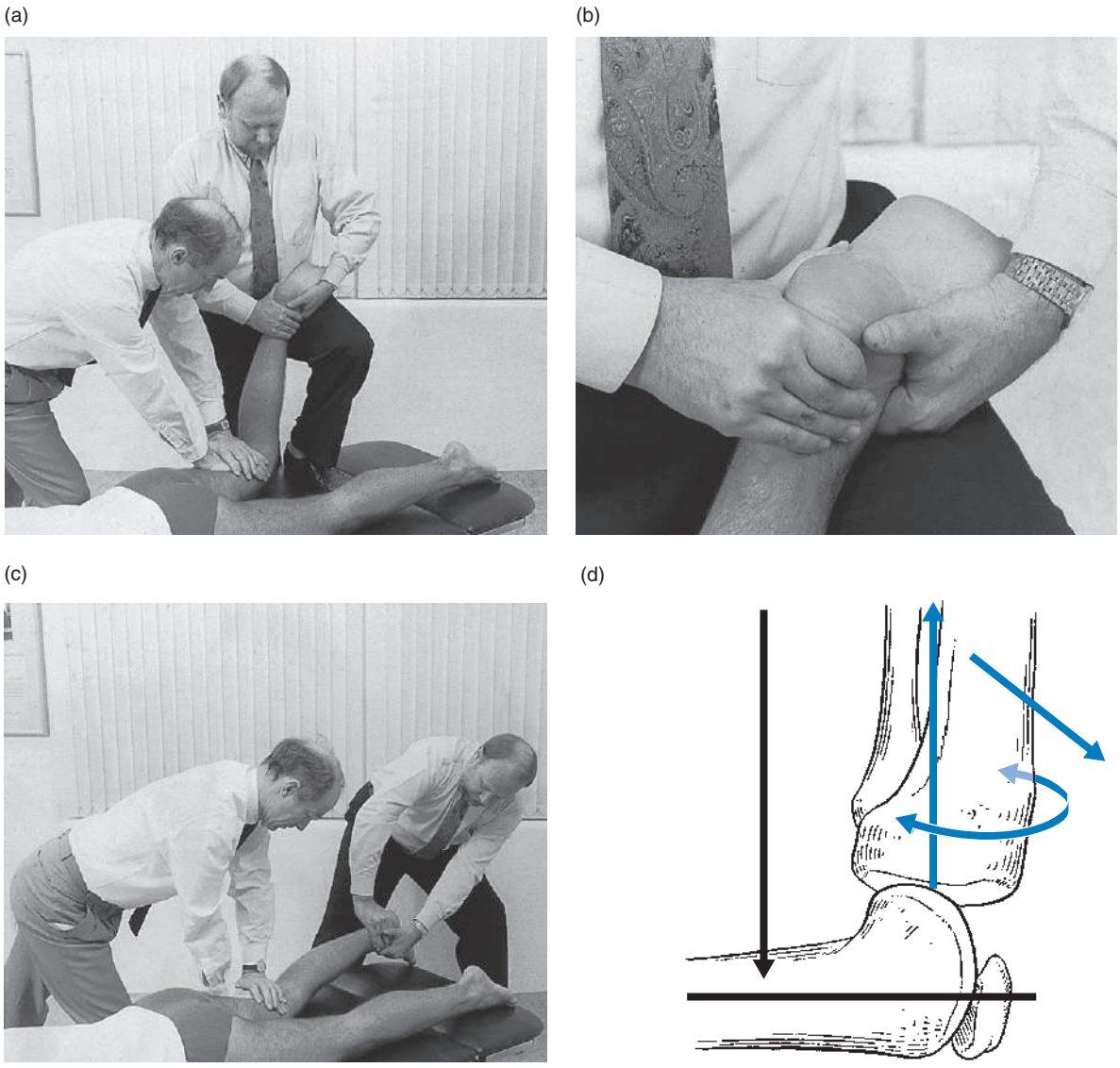


Fig 52.10 • First manipulation for a loose body impacted in the knee.

This manipulation is painless and can be repeated up to 10–20 times during one session. Usually one or two sessions suffice for full relief, even if the condition has been present for months.

Second manipulation

If the previous manœuvre has only partially improved flexion but has not completely reduced the loose body, techniques that use leverage can be employed next. If the wrist is placed at the back of the patient's knee, forced flexion of the tibia on the femur will strongly distract the joint surfaces and thus make room for the loose fragment to move.

Technique

The patient lies supine on the couch. The manipulator places one wrist in the popliteal fossa, between tibia and femur, while the other hand presses at the distal end of the tibia. The knee is then bent as far as it will go (Fig. 52.11a). When progressive ligamentous resistance indicates that the slack has been taken up, the tibia is quickly forced into greater flexion with a small thrust. A click may be felt. Re-examination will show whether further reduction has taken place.

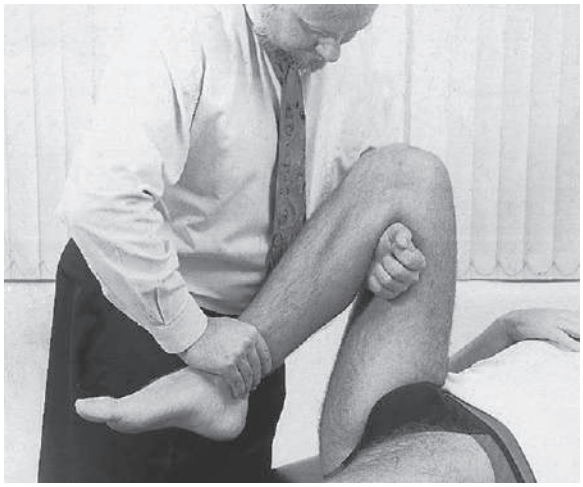
If this manipulation fails, the same movement can be repeated during rotation. The manipulator places a firm rolled bandage in the popliteal fossa (Fig. 52.11b). This permits the patient's foot and wrist to be held with both hands. Rotational movements can be added during the forced flexion. First one rotation is tried; should this fail, rotation in the opposite direction is attempted.

It is important to verify the range of extension after each attempt, because full and painless extension at the knee is more important than some improvement of flexion. Should the range of flexion improve but extension become painful or limited, this manipulation should be terminated immediately.

Third manipulation

This technique is used when the first manipulation does not achieve full and painless extension. In most cases, extension is not limited but is painful and has an altered end-feel.

(a)



(b)

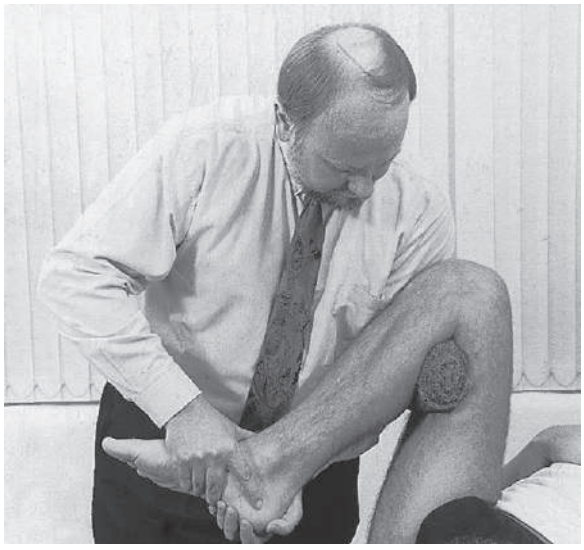


Fig 52.11 • Second manipulation for a loose body impacted in the knee: two alternative methods are shown.

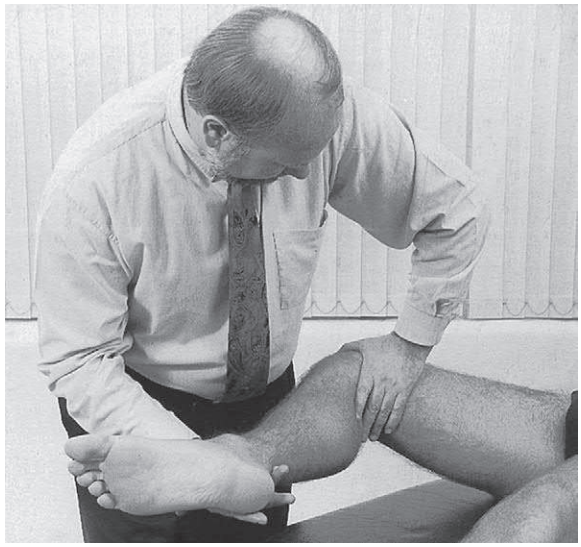


Fig 52.12 • Third manipulation for a loose body impacted in the knee.

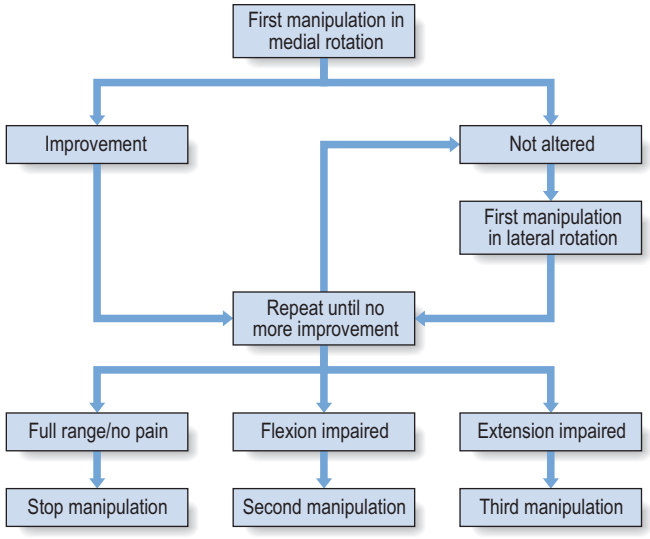


Fig 52.13 • Manipulative session for a loose body.

Technique

The patient lies supine. The manipulator stands level with the knee, and bends it to a right angle while the hip is flexed and laterally rotated. The hand is applied at the inner side of the knee and the other hand at the lateral border of the distal tibia in such a way that a strong varus movement is achieved (Fig. 52.12).

The patient's cooperation is now sought because the knee must be actively and slowly extended while the manipulator maintains the varus pressure. At almost full extension, and maintaining as much varus pressure as possible, the manipulator adds a quick jerk towards full extension. If this final jerk proves to be too painful, it should only be done a few times, after repeated active extension by the patient.

Once again, re-examination should follow each attempt and the end-feel will indicate whether reduction has taken place (Fig. 52.13).

Instructions on after-treatment

During the following days the patient must repeat the manipulation several times a day. To do this, the patient sits on a low chair or on the ground, the knee flexed and the hip laterally rotated. The lateral border of the foot now rests on the floor. Pressing strongly on the inner side of the knee, and at the same time actively extending the leg, results in a varus movement during active extension (Fig. 52.14). Fixation of the lateral border of the foot against the floor ensures the counterpressure needed for a good varus stress. At nearly full extension, a small thrust is added.

Results

Most impacted loose bodies complicating osteoarthrosis are reduced by one or two manipulative sessions. No after-treatment to the strained ligaments or the slight traumatic arthritis is necessary, because they will recover within a few

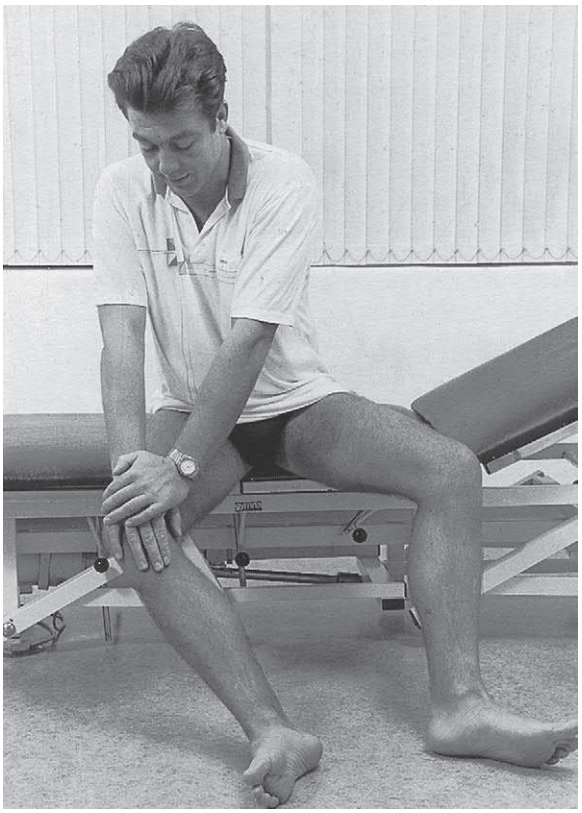


Fig 52.14 • After-treatment exercise by the patient.

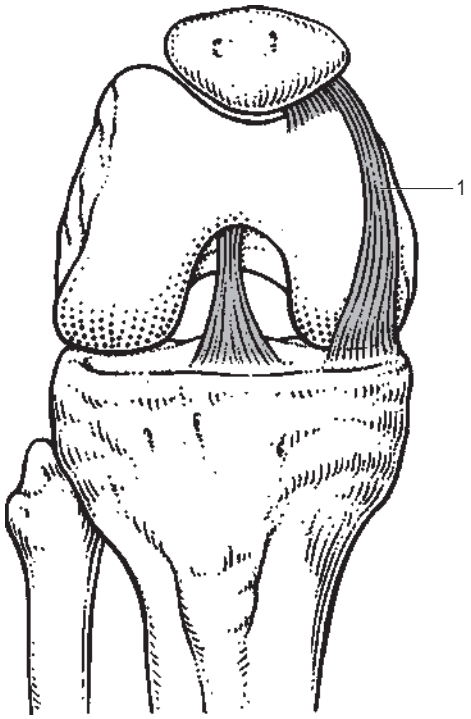


Fig 52.15 • Medial plica (1).

days without specific treatment. Recurrences of the impaction are always possible, mainly after kneeling or keeping the knee bent for some time. Patients should therefore be told not to squat or to sit sideways on a couch with the legs tucked underneath, a sitting position often adopted by women.

Plica synovialis syndrome

Plicae synoviales are remnants of the embryological dividing walls in the knee. Arthroscopy has recently brought their existence to prominence. They are present in more than 20% of all knees,[140] but it is only when pathological changes take place that pain and disability are likely to occur. Injuries and excessive strains are thought to be responsible for these pathological alterations.[141,142]

Although there are several types of synovial plicae, it is the plica mediopatellaris, or medial shelf (Fig. 52.15), which seems to cause most problems.[143]

This plica was first described by Iino in 1939.[144] It has its origin on the medial wall of the knee joint and runs obliquely down towards the synovium, inserting into it and covering the medial infrapatellar fat pad. During flexion it glides over the medial condyle like a wiper over a windscreen, which in normal circumstances is harmless and painless; if the plica is pathologically altered or the medial condyle has undergone arthrotic changes, however, symptoms may result.

One of these symptoms is a sudden twinge when the inflamed plica is pinched between the patella and the medial condyle of the femur. Sometimes there is pain and a slightly modified end-feel at the end of extension. A painful arc during flexion–extension can occur when a damaged and thickened plica rides over a chondral defect at the medial condyle, and it can be aggravated when this movement is performed during lateral rotation.[145,146]

The clinical diagnosis of a symptomatic medial plica is made from a history of localized pain during flexion–extension or pain at the end of extension.[147] The plica can sometimes be felt if the examiner rolls the medial capsule of a slightly (45°) flexed knee under the thumb.[148] When this rolling is painful, 20 mg of triamcinolone is locally infiltrated.[149,150] Some authors have reported good to fair results with flexibility training of the flexor and extensor muscles of the knee.[150,151] If the symptoms persist, arthroscopic resection is performed.[152]

Because the presence of a medial shelf is normal,[153] one should be cautious in ascribing symptoms to it. The diagnosis should be made clinically and not by relying on accidental findings during arthroscopy.

Intra-articular adhesions

There is an unusual disorder of the knee, characterized by an increasing limitation of flexion while extension remains full and painless. As this pattern occurs after operation or an unexceptional sprain, Cyriax ascribed it to intra-articular adhesions.

The history is as follows. There has been surgery to the medial side of the knee or an atypical sprain (hyperextension or hyperflexion) that has given rise to the lesion. In spite of vigorous physiotherapy, the knee stiffens progressively and almost painlessly, the main symptom being inability to flex the

joint. During the initial weeks, 15–30° of flexion are lost; after a month there is loss of about 90° of flexion. While the range of flexion decreases day by day, extension remains full and painless. Finally, the knee may stiffen with a flexion range of 45°, extension still being full-range.

Clinical examination immediately reveals the gross non-capsular pattern. Furthermore, the joint is cold but not swollen; nor is there any capsular thickening. The combination of the non-capsular pattern, gross limitation of flexion and absence of local signs is so striking that this remarkable condition will be recognized easily, especially if there is a history of trauma or surgery.

The only lesion that can be confused with these intra-articular adhesions is Stieda–Pellegrini disease, in which the calcified medial collateral ligament can cause a similar selective limitation of flexion. A radiograph establishes the diagnosis.

Treatment

Gentle forcing does not prevent the progressive decrease of flexion and any conservative treatment, except forceful manipulation towards flexion, is futile. It is easiest to force the knee when the patient adopts a prone position.

Technique

The manipulator stands at the level of the knee, which is bent as far as possible. The elbow is curved around the distal aspect of the tibia. The other forearm is placed at the back of the knee, just above the popliteal fossa. The hands are locked. Flexion is now performed by a side bending of the body towards the patient's head. A great deal of force can be obtained if the manipulator keeps the arms still. The arm on the thigh keeps the knee on the couch, while the other arm uses the tibia as a lever. The manipulation is executed during a quick side bending of the whole body, with as much force as possible. Often a loud sound of ripping, tense tissue can be heard; flexion can be achieved immediately, thus proving that there were adhesions.

Follow-up

Within 24 hours, there is considerably more pain than before the manipulation and the patient has symptoms of significant arthritis: a warm joint, full of fluid and demonstrating an articular pattern. During the following days, active and passive exercises are prescribed in order to maintain the range of flexion. When arthritis is gross and pain severe, an intra-articular injection with 50 mg of triamcinolone can be given in order to diminish the traumatic capsulitis.

Subsynovial haematoma

A severe blow on the front of the thigh may cause a localized haematoma between the suprapatellar pouch and the front of the femur (Fig. 52.16). Flexion is limited but extension remains free.

The history is typical: a severe knock just above the patella has caused immediate pain and localized swelling.

During clinical examination, the knee is found to be warm and swollen. Extension is full but probably painful at the end of its range. Passive flexion is grossly limited and there is localized pain just above the patella. Resisted extension is painless, which indicates that neither the quadriceps tendon nor the patella is to blame. Palpation reveals some fluid in the joint and a solid, tender swelling at the suprapatellar pouch.

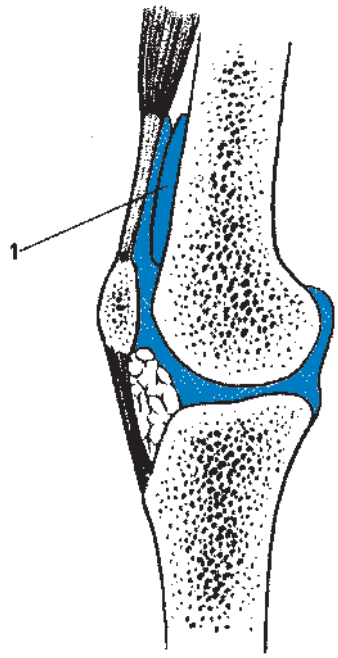


Fig 52.16 • Subsynovial haematoma (1).

Fluid aspirated from the joint is clear or slightly blood-stained. There is no improvement in range after aspiration, which establishes that the effusion is a secondary one. If the needle is passed just above the patella, in the direction of the femur, blood from the haematoma can be obtained and confirms the diagnosis. It is important to note that aspiration is possible only during the initial few days because the haematoma organizes rapidly.

After aspiration, the knee should be mobilized during the following days in order to prevent persistent adhesions.

Adherent vastus intermedius

The vastus intermedius may become adherent to the femur after a fracture of the latter. Gross limitation of flexion then results. Only surgical treatment gives a satisfactory outcome.

Posterior capsular strain

There is a history of a severe hyperextension strain. When traumatic arthritis has subsided and flexion becomes full and painless, extension continues to hurt at the back of the knee. The rest of the clinical examination shows the knee to be normal and therefore strains of anterior or posterior cruciate ligaments are excluded. In long-standing cases, an X-ray may show the typical appearance of slight calcification in the posterior capsule.

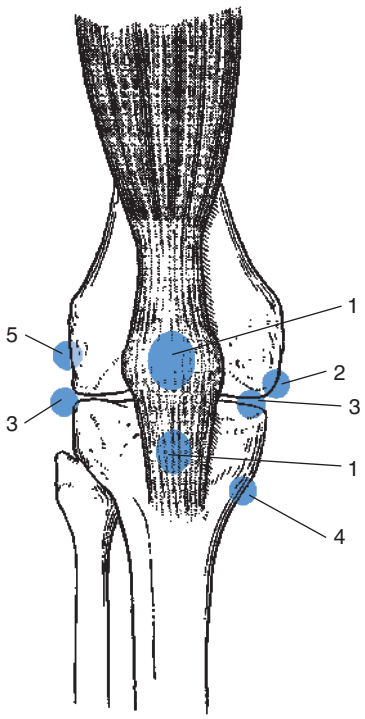


Fig 52.17 • Cysts and bursitis around the knee: 1, patellar bursitis; 2, medial collateral ligament bursitis; 3, meniscal cysts; 4, pes anserinus bursitis; 5, bursa between the iliotibial tract and lateral epicondyle.

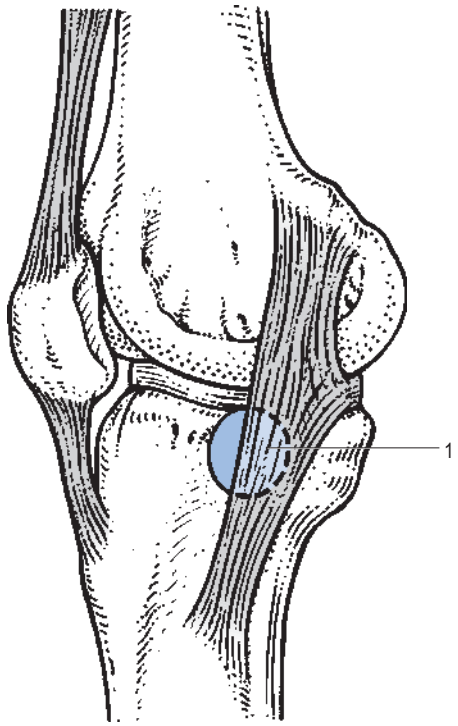


Fig 52.18 • Medial collateral ligament bursitis (1).

Treatment consists of infiltration with triamcinolone at the appropriate side, using the same technique as for posterior strains of the posterior cruciate ligament (see p. 709).

Cysts and bursitis

It is important to note that localized swellings may limit movements in a non-capsular way (Fig. 52.17). For example, a bursa underlying the medial collateral ligament, a prepatellar bursa or a cyst under the iliotibial tract can each cause a specific pattern.

Medial collateral ligament bursitis

The patient is usually middle-aged and complains of localized pain at the medial side of the knee, which has appeared without a specific cause (Fig. 52.18). The pain is worse during activity and eases at rest, but nocturnal pain may occur. There is no symptomatic evidence of internal derangement, such as twinges, locking and fear of giving way.[154] Spontaneous cure is uncommon[155]: one of our patients had had unchanged symptoms for more than 6 years.

Clinical examination reveals a cold joint, with no fluid or synovial thickening. Extension is normal, while flexion is limited by 15–45°. The end-feel is soft, the limiting factor being pain, which is strictly confined to the inner side of the knee. Valgus strain and lateral rotation are painful. Palpation reveals a solid swelling under the medial collateral ligament, level with the joint, which can be so hard that it is mistaken for a large osteophyte. Unlike a cyst of the meniscus, the bulge does not disappear during flexion, but enlarges and becomes firmer, especially when it lies at the dorsal aspect of the ligament.[156]

It is important to differentiate between chronic ligamentous sprain and bursitis under the medial collateral ligament, the signs of which are very similar. In sprain, the distinctive factors are a history of previous trauma and absence of palpable swelling. The treatment required – deep friction and manipulation – would undoubtedly worsen bursitis, and therefore the differential diagnosis should be made carefully. In case of doubt, MRI can demonstrate the fluid collection under the medial collateral band very well.[157]

Treatment is aspiration. A thick needle (19G × 4 cm) should be used because the fluid is so viscous that it is very difficult to remove. Injecting 10–20 mg of triamcinolone into the cavity will prevent early recurrence of symptoms. After aspiration, immediate full and painless flexion is possible. Permanent cure is often achieved.[158]

Patellar bursitis

Prepatellar bursitis is by far the most common type of bursitis, especially in patients who have to kneel repeatedly in their work (e.g. gardeners, bricklayers). The infrapatellar bursa can also become inflamed (‘clergyman’s knee’).

The patient complains of pain and swelling in front of the knee. The diagnosis is made on simple inspection. If the

swelling is gross, it can result in limitation of flexion because of the painful stretching of bursa and skin. Palpation shows that the effusion lies between skin and patella. The presence of heat suggests haemorrhage into the bursa, whereas heat and redness indicate the possibility of sepsis.[159]

Aspiration should be performed to disclose the nature of the fluid. If the bursa refills repeatedly, surgical removal must be considered.[160]

Bursa between the iliotibial tract and the lateral epicondyle

A bursa can form under the iliotibial tract where it rides over the lateral condyle. This is a common condition in long-distance runners, cyclists and skiers. The patient complains of localized pain while walking or running. Clinical examination reveals a painful arc at 30° of flexion. Resisted flexion and extension of the knee are negative. A swelling can be palpated between the condyle and the iliotibial tract.

Bursitis at this site must be differentiated from a strained iliotibial band, which also occurs in athletes and gives rise to localized pain at the outer side of the knee. In this condition, pain is the result of a lesion of the tract itself, confirmed by discomfort on resisted extension and lateral rotation (see p. 1143).

Aspiration, followed by local infiltration with corticosteroid suspension, is the remedy. If the condition fails to respond to this treatment, the posterior 2 cm of the band should be transversely sectioned.[161]

Pes anserinus bursitis

The pes anserinus bursa is situated between the medial collateral ligament and the overlying pes anserinus (tendons of sartorius, gracilis and semitendinosus).[162] Inflammation of this bursa commonly occurs in long-distance runners.[163]

Clinical examination reveals a full range of movement. Sometimes resisted flexion and medial rotation cause pain because these movements squeeze the bursa painfully. A tender mass can be palpated at the tibial insertion of the medial collateral ligament.[164]

Treatment is local aspiration and infiltration with triamcinolone.[165]

Meniscal cysts

Asymptomatic cysts need not be treated. If symptoms arise, treatment is by aspiration (see above).

Popliteal cyst

If a cyst forms at the back of the knee, the result may be some limitation of flexion. The end-feel will be of soft tissue approximation. There is only slight pain.[166]

Treatment consists of aspiration; surgical removal is seldom necessary.

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Disorders of the inert structures: ligaments

53

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Introduction

Ligamentous lesions at the knee are quite common. The joint is relatively uncovered by muscles, which makes it vulnerable to direct injury. Furthermore, the indirect forces acting on the knee have a large leverage, whereas the active and passive stabilizers of the joint have only a small leverage (Fig. 53.1) and thus give very inadequate protection.

Few other sports injuries cause as much concern as ligamentous lesions. Each contusion, even slight damage to the medial collateral or the coronary ligament, can cause serious trouble. Knee traumas should always be taken seriously because a neglected knee injury may lead not only to instability but also to the formation of adhesions.

There are great differences in attitudes towards the treatment regimes for the different injuries. The wide variety of traumas, the different degrees of damage and the combination of various lesions make it very difficult to compile a list of clear recommendations for treatment. In the classic textbooks, it is advised that minor ligamentous sprains are immobilized, whereas for serious or combined lesions, surgical intervention is generally recommended, especially when the patient is a young athlete or when development of later instability is feared.

We firmly believe that immobilization is never a good method. If there is a serious grade III lesion (see below) in a young athlete and instability is feared, the patient should be sent for surgery. If, for one reason or another, the patient is not treated surgically, early mobilization, deep transverse friction and functional treatment should be used. This treatment regime gives the needed physiological stimulus for quick and proper healing of the lesion and prevents the formation of adhesions, so often the cause of persistent trouble.[1]

Classification

Most accounts divide ligamentous injuries into three grades: grade I is slight overstretching with some microtears within the structure of the ligament; grade II is a severe sprain with a partial tear of the ligamentous fibres; and grade III implies a ligament which is completely torn across. In our opinion, this classification is rather arbitrary and, although it might be possible to distinguish a small lesion from a total rupture, the difference between grade I and grade II will always remain subjective.

Ligamentous injuries can also be classified according to which structure has been damaged. The commonest injuries are at the medial collateral and the anterior cruciate ligaments. Sometimes these occur together and in combination with a torn medial meniscus – the 'unhappy triad', or the triad of O'Donoghue[2,3] – or in combination with a tear of the lateral meniscus.[4] Lesions at the lateral collateral ligament and the posterior cruciate ligament are rare. In our experience, tears at the medial coronary ligament are very common; however, these are often misdiagnosed as medial collateral tears or meniscus lesions.

Sprained knees can also be classified according to the time that has elapsed since the causative accident. Here the terms acute, subacute and chronic are used:

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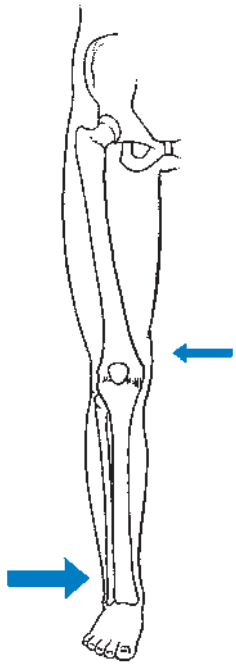


Fig 53.1 • Forces on the knee: direct (small arrow) and indirect (large arrow).

Table 53.1 Classification of ligamentous injuries according to severity, structure and time

Severity	Structure	Time
Grade I: slight overstretching	Isolated sprain	Acute: less than 2 weeks
Grade II: partial tear	Combined sprains	Subacute: between 2 and 6 weeks
Grade III: complete tear	In combination with meniscal tears	Chronic: more than 6 weeks

- *Acute*: less than 2 weeks since injury
- *Subacute*: 2–6 weeks
- *Chronic*: more than 6 weeks.

This temporal division is important in choice of treatment (Table 53.1).

Diagnosis

Even in the era of arthroscopy, a clinical approach to ligamentous lesions continues to be vital.

In the acute stage, information obtained from the history and clinical examination enables the examiner to make the distinction between serious and mild lesions. If symptoms and signs warrant, the patient is then referred for further assessment by arthroscopy.[5]

In the chronic stage, only a thorough functional examination can lead to the diagnosis of ligamentous adhesions or estimate the degree of functional instability.

Box 53.1

History of knee injury

The injury	Position of the knee Direction and severity of the forces
Initial symptoms	Localization of pain Swelling Functional disability
Evolution	Pain Swelling Functional disability
Problem now	Pain? Where? What provokes it? Swelling Instability? What provokes it?

History

It is vital to obtain a very detailed history of the mechanism that has led to the injury, as summarized in Box 53.1, especially in acute sprains of the knee.

- *The injury*: at the moment of injury, what was the position of the knee, what forces acted where on the knee and in which directions were they applied?
- *The initial symptoms*: what was the immediate result of the trauma? Where was the initial pain? Was there immediate swelling? Was there immediate functional incapacity caused by locking or instability or could the patient continue activities? Did pain, swelling and functional disability appear only after a certain lapse of time?
- *The evolution*: what was the evolution of pain, swelling and disability after the first few days?
- *Treatment received*: what sort of treatment did the patient receive and what was the result?

In long-standing cases, the current symptoms should be ascertained:

- *Pain*: is it still present? What is its localization and when does it appear?
- *Swelling*: does the knee swell?
- *Instability*: is there any feeling of instability and giving way?

Examination

The functional examination described in Chapter 50 is carried out.

In acute cases, the ligamentous tests will sometimes be overshadowed by the capsular pattern of the traumatic arthritis, which makes it very difficult to estimate the degree of damage. In subacute and chronic cases, the capsular signs have largely subsided and the tests of ligamentous integrity become more informative. Sometimes, laxity is found during the routine examination, in which case instability tests are then carried out (see online chapter *Disorders of the inert structures: ligamentous instability*). Sometimes, it is not so much instability but rather pain and limitation of a non-capsular type that are detected; these indicate the formation of adhesions around the healed tissue.

Table 53.2 Acute ligamentous lesions: contrasting histories of serious and less serious lesions

	Serious lesions	Less serious lesions
Impairment of function	Immediate inability to continue the sport or activity	Most initial pain disappears and sports and activities can be continued after a short time
Swelling	Immediate or developing during the first hour, indicating haemarthrosis[5]	Appears a few hours after the accident, with functional incapacity and generalized pain
Feeling of instability?	Yes	No
Aspiration	Blood may be present	Clear fluid is obtained
Possible diagnoses	Triad of O'Donoghue? Isolated rupture of a cruciate ligament?	Isolated sprain of a collateral, coronary or cruciate ligament

Table 53.3 Differential diagnosis of chronic ligamentous sprain and instability

	Chronic ligamentous sprain	Instability
History		
Stiffness/'giving way'	Stiffness after the knee has been kept still for some time	Feeling of 'giving way' during unexpected movements under load
Pain localization	The knee hurts at one small spot during and after exercises	Slight discomfort over the whole joint after the initial unstable feeling
Swelling	Some recurrent swelling	
Clinical examination		
Local pain	During ligamentous tests	None
Range	Sometimes limited in a non-capsular way	Sometimes increased range of movement
Instability	None	Detection of instability during the additional tests

Acute ligamentous lesions

In acute ligamentous lesions at the knee, the history will be what first indicates severity. A few hours after a serious sprain, the knee will start to hurt considerably and develop a distinct capsular pattern, protected by muscle spasm that makes it almost impossible to perform ligamentous tests. In order to distinguish between a serious injury and a less important lesion, a number of elements gained from the history may be of value (Table 53.2).

When the history indicates a serious lesion, especially if the patient is an athlete, it is wise to arrange arthroscopic evaluation. In contrast, in a more modest lesion, a conservative approach is indicated.

Chronic ligamentous lesions

Because the treatment is quite different, it is vital to differentiate between instability as the result of a total ligamentous rupture and chronic ligamentous adhesions leading to a 'self-perpetuating inflammation'. Once again, history and clinical examination are the first approach to differentiate between these conditions and, in instability, to estimate its degree (Table 53.3).

Post-traumatic adhesions at the knee have the following signs and symptoms. The knee hurts locally after (vigorous) exertion or during the first few steps after it has been kept still for a while (e.g. in the morning or after sitting for a few hours). Sometimes slight swelling is also induced. Ordinary walking does not hurt. Clinical examination reveals slight limitation of movement, positive ligamentous tests and local tenderness.

Instability is characterized by a sensation of 'giving way' during unexpected movements under load. There may be accompanying pain and discomfort, sometimes lasting for a day or two. Additional instability tests will usually detect the type and degree of ligamentous insufficiency (see online chapter *Disorders of the inert structures: ligamentous instability*).

Treatment for a 'chronic ligamentous sprain', whether it is adhesion to nearby structures or self-perpetuating inflammation in the ligament itself, is relatively simple and as a rule gives quick and permanent results. Treatment of an unstable knee is more difficult and often requires surgical intervention.

Treatment: the principle of early mobilization

To most orthopaedic surgeons, ligamentous lesions of the knee, especially if they are grade III, require immobilization or surgical repair. The reasoning is purely anatomical: there is a rupture and the main medical task is to repair both ends, either by immobilization or by surgery. Our opinion tends more in the direction of mobilization and functional management.

Experimental studies over the past few decades have demonstrated that regeneration of injured connective tissue is significantly better with the application of continuous passive motion. Under functional load, the collagen fibres are oriented in a longitudinal direction and the mechanical properties are optimized.[6] Therefore, functional conservative treatment is advised for all coronary ligament sprains and all isolated grade I, II or III sprains of the medial collateral ligament[7] and posterior cruciate ligament and isolated grade I or II lesions of the anterior cruciate ligament.[8] However, in combined lesions and in anterior cruciate ligament tears with a positive pivot shift phenomenon, surgery is the treatment of choice.[9]

Mobilization not only is the best promoter of ligamentous repair but also prevents ligamentous adhesions within or around the healing structure. Another advantage of early mobilization is the positive effect on muscle strength[10] and proprioceptive reflexes,[11] which ensures the active stability of the joint.

This conservative and functional approach to recent and isolated tears was first advocated by Cyriax[12] and has recently received much support. Several studies have demonstrated that the non-operative management of an isolated medial collateral ligament injury, especially of grade I and II, is as good as, if not better than, a primary surgical approach.[13–18] The

conservative treatment of grade III sprains of the medial collateral ligaments also gives results that are equally as good as the surgical approach but with significantly quicker rehabilitation.[19–21] Jones *et al*[22] treated 24 high-school football players with an isolated grade III injury of the medial collateral ligament. They administered mobilization, using a regime of muscle strengthening and agility exercises. Knee stability was achieved in 22 cases, with an average recovery time of 29 days. The players returned to competitive sport after a mean of 34 days. Similar results were obtained in a long-term study of 21 grade III medial collateral ligament tears.[23] The overall conclusion was that non-surgical treatment of a complete tear of the medial collateral ligament was extremely successful, provided there was no associated structural damage to the anterior cruciate ligament.

However, in advocating early mobilization, one common difficulty arises: the serious traumatic arthritis and the intense pain during the slightest movement are very strong impediments to early activity. As for ligamentous lesions at the ankle, this problem can be solved in two ways:

- Relieve the inflammation and pain as soon as possible so the patient can mobilize the knee. This can be achieved by local infiltration of a small amount of triamcinolone.
- As advised by Cyriax, mobilize the ligament over the bone by deep transverse friction instead of moving the bone under the ligament as in ordinary mobilizations. The relative movement will be the same, as is the mechanical stimulus to the regenerating fibrils.

In long-standing and chronic ligamentous lesions, where scars have been allowed to form abnormal attachments in or around the healing tissues, the approach is to break the adhesions and remodel the fibrils in the functional longitudinal direction. This can be achieved by deep friction, sometimes with manipulation in addition.

Isolated sprains

Medial collateral ligament

The medial collateral ligament prevents valgus deviation of the knee and, through its posterior fibres, checks external rotation of the tibia.[24] An understanding of this is of importance in the interpretation of clinical tests in a torn medial collateral ligament.[25]

Diagnosis

The medial collateral ligament is the most commonly injured ligamentous structure of the knee. The classic mechanism of injury is a forced valgus movement on a partly flexed and externally rotated knee,[26] which typically occurs when a soccer or American football player receives a kick or blow at the outer side of the weight-bearing knee.[27] The patient experiences a crack and feels sudden pain at the inner aspect of the knee. Most of the pain disappears fairly quickly and the sportsman or woman can probably return to the game or walk off the pitch. At first, the knee is not swollen and there is only slight disability. The real incapacity, with increasing swelling and pain, starts after a few hours. By the next day, the patient can hardly stand and can only hobble with assistance.

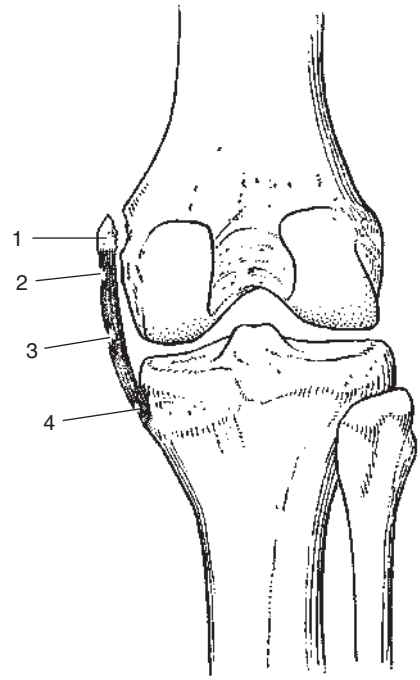


Fig 53.2 • Medial collateral ligament lesions: 1, avulsion of a bony fragment; 2, proximal tear; 3, mid-portion tear; 4, distal tear.

Clinical examination shows a hot and tender knee full of fluid. There is a gross articular pattern, with muscle spasm at the end of the range of movement: extension is probably 5–10° limited and flexion can be limited up to 90°. In this acute stage, it is impossible to perform proper ligamentous tests but the patient knows that the inner side of the knee was initially sprained, and localized tenderness is easily found at some point along the ligament.

The tear can be proximal, be related to the mid-portion of the ligament or be situated at the distal, tibial portion (Fig. 53.2). Mid-portion tears are the most common, but also the most disruptive, because they also involve the deeply situated meniscotibial and meniscofemoral portions of the ligament.[28] In a proximal tear, it is wise to perform radiography to exclude avulsion of a bony fragment, which is an indication for surgical repair.[29,30]

The natural history of an injured medial collateral ligament is as follows. In the acute stage there is a traumatic arthritis, lasting about 2 weeks. Thereafter, during the subacute stage which lasts 4–6 weeks, the limitation of movement slowly diminishes. Although the joint remains warm at the inner side, the swelling goes down. Testing the ligament by applying strong valgus pressure (in 0° and in 30° of flexion) will elicit pain and, if the ligament has been totally ruptured (grade III), an excessive range will be detected. This is an indication for further stability testing.

After 2–3 months, the traumatic arthritis has totally subsided. At this stage, three situations are possible:

- There has been good healing, with a strong non-adherent ligament.
- The ligament is permanently lengthened, resulting in an unstable knee.
- The ligament is adherent, leaving the patient with recurrent and localized disability.

If an abnormal and adherent scar forms between the ligament and the surrounding tissues, any vigorous effort will sprain the impaired ligament afresh. In this case, the history is typical: the knee is quite adequate for ordinary walking and even running, but one small area at the inner side hurts when the patient takes vigorous exercise. The knee also feels stiff after it has been kept still for some time. Clinical examination shows a non-capsular pattern of limitation with, as a rule, painful full extension and limitation of flexion by 5–10°. External rotation and valgus strain hurt at the medial side of the knee. The other ligamentous tests are painless and instability cannot be detected. Resisted movements are strong and painless. There is no fluid or warmth, unless the patient is seen the day after some additional exertion. Tenderness should be sought along the medial collateral ligament. The usual localization is at the joint line. However, if a full range of movement is found after a former sprain but there is a clear history suggesting a chronic sprain and examination shows the medial collateral ligament to be at fault, tenderness is usually found at the femoral origin of the medial collateral ligament.

Stieda–Pellegrini disease

Sometimes calcification of the medial collateral ligament develops after an apparently ordinary sprain of the inner side of the knee. Suspicion arises when increasing limitation of flexion, together with a hard end-feel, is found 4–6 weeks after the accident. Radiography shows ossification along the inner side of the medial femoral condyle.[31]

Conservative treatment is ineffective but good results have been reported with surgical treatment.[32] Spontaneous recovery takes 6 months to 1 year.[33]

Treatment

All grade I and grade II sprains (partial tears), as well as isolated grade III sprains (complete tears) of the medial collateral ligament should be treated conservatively. Surgery is indicated only in combined lesions of medial collateral ligament and meniscus and/or anterior cruciate ligament.[34]

The technique chosen depends on the stage of the lesion, as summarized in Box 53.2. If the patient is seen during the first 24 hours after the accident, local infiltration with triamcinolone can be considered. Deep transverse friction in combination with early mobilization can be applied during the first 6 weeks following the accident. If the patient is seen after more than 6 weeks, the lesion must be considered as chronic and manipulation is given.

Acute stage

Infiltration and cold compression

Immediately after the accident, an ice compress is applied in order to prevent an excessive inflammatory reaction. As soon as the patient is seen, a small dose of triamcinolone (20 mg) can be injected at the site of the tear. Because this injection is extremely painful, a local anaesthetic must always be added.

During the next 24 hours, the patient remains in bed with an ice bag on the affected area. From the second day on, flexion is strongly encouraged, as is extension without weight bearing. During the first week, however, no attempt should be made to straighten the knee completely on walking; the medial collateral ligament is taut in extension and too much stretching at the point of injury would cause harm. On account of the anti-inflammatory effect of the triamcinolone, pain and swelling abate very quickly and normal gait is restored after 1–2 weeks. Walking and modest movement of the knee should be encouraged because they provide the best stimulus to normal healing and prevent the formation of adhesions. The patient can usually return to sports after 6 weeks.[35] By that time, movements are of full range and ligamentous tests negative. It is wise to recommend a protective knee brace during the first few weeks of sporting activity, especially if the patient returns to contact sports.

Technique: infiltration

The patient lies supine, with a pillow under the knee. The physician, standing at the inner side of the knee, outlines the correct area. A fine needle is fitted to the syringe, filled with a mixture of 1 mL of lidocaine 2% and 20 mg of triamcinolone. The needle is thrust in almost horizontally (Fig. 53.3) and the whole tender area is infiltrated by a series of small droplets.

Friction

Because a steroid injection, even a small amount, undoubtedly not only abates the excessive inflammation but also interferes in the union of the ruptured ligament, it is wise to use an alternative treatment modality if the patient is a young or professional athlete. Deep friction, applied from the first day, is an excellent alternative and gives identical results, providing the massage is given correctly.

Friction serves a double purpose: it prevents any fibrils from binding the ligament to bone and applies a positive stimulus to the healing connective tissue. As, in the acute stage, adhesions will not yet have formed, just 2 minutes of deep friction are required in both flexion and extension, though the preparatory phase of gentle massage, followed by slight superficial friction (which renders the ligament anaesthetized), may take up to 20 minutes. The patient is given this deep friction every day

Box 53.2

Treatment of medial collateral ligament sprains

Acute stage	• Infiltration with triamcinolone (if performed during the first 24 hours following the accident) + active mobilization • Deep transverse friction + mobilization within pain-free range
Subacute stage	Deep transverse friction + gentle passive movements in flexion/extension/external rotation
Chronic stage	Manipulation in the limited direction, preceded by vigorous deep friction

Fig 53.3 • Infiltration of the medial collateral ligament.

during the first week, after which the subacute stage is entered. If the massage is given adequately, the range of flexion increases dramatically during the first few days. The patient should be encouraged to walk and to move actively, although straightening the last 20° or flexing the last 60° must be avoided during the first week because these movements put too much longitudinal strain on the ligament.[36] The joint can be protected with a partial mobile brace, the coronal straps of which prevent excessive valgus movement.[37]

Technique: deep friction

The patient lies supine on the couch and the exact point is identified. Friction is first given in as much extension ( ) as possible, then in as much flexion ( ) as possible. The therapist places the index finger at the exact point, which is most often the central part of the ligament at the joint line. The index finger is reinforced by the middle finger, and the thumb is placed at the outer side of the joint so that it can be used as a fulcrum (Fig. 53.4). Friction is imparted by an extension movement at the wrist, which draws the index finger anteriorly over the ligament. Releasing the grip and flexion of the wrist moves the index finger back to the starting position.

Subacute stage

In the subacute stage, massage is given more thoroughly and for longer, applying the same technique as described for the acute stage (see Fig. 53.4). By moving the ligament across the bone, the normal movement of bone over ligament is mimicked. Immediately after the massage, gentle movements in flexion, extension and external rotation are carried out by the therapist (passive movement). In order to prevent atrophy of the thigh muscles, a programme of resisted exercises should also be followed. Treatment three times a week for two weeks usually suffices for full recovery. Return to competitive sports is allowed only when running ability has returned to normal. This may take up to 6 weeks.[38]

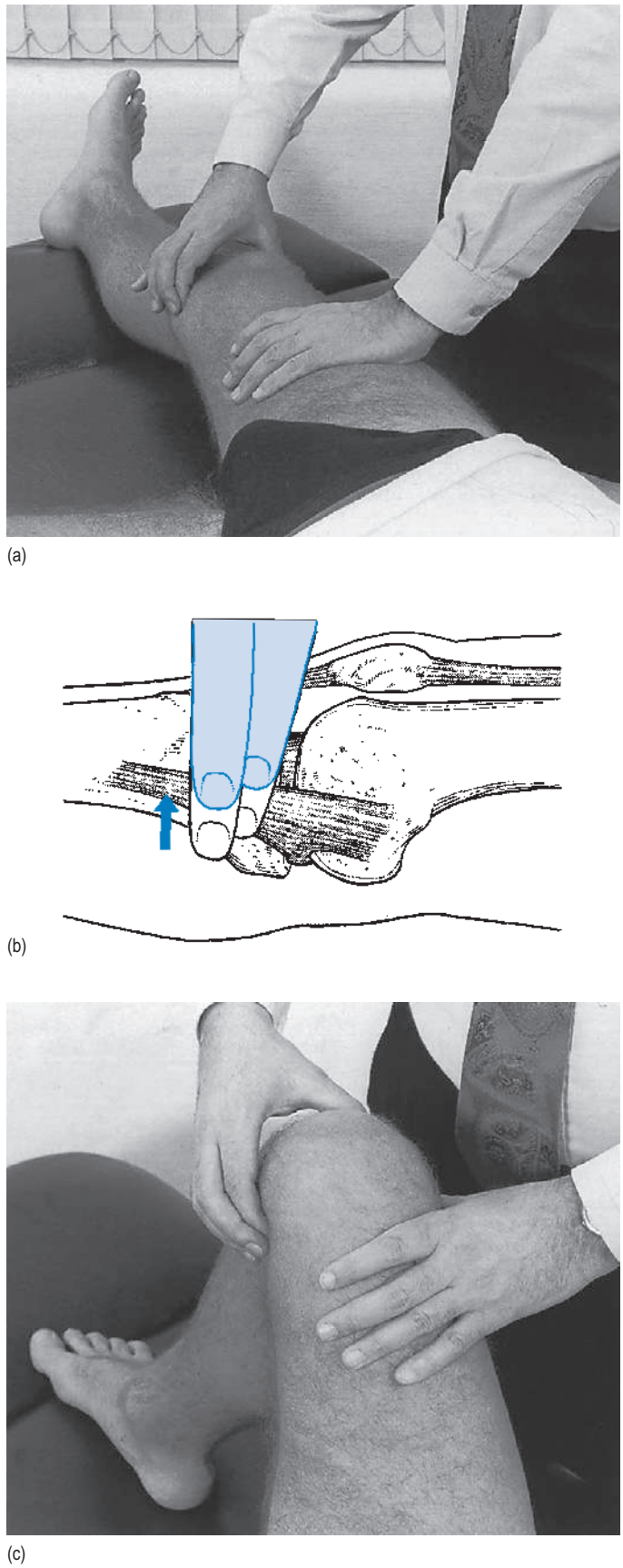


Fig 53.4 • Friction to the medial collateral ligament: (a), (b) in extension; (c) in flexion.

Chronic stage

When the patient is first seen in the chronic stage, adhesions have already formed between the ligament and bone – a situation that would never have been reached if the patient had received proper treatment from the start, but which is often encountered when the knee has been immobilized. The principle of treatment is to restore a full range of movement by rupturing the abnormal adhesions. In order to do so, vigorous deep friction to the affected part of the ligament is applied for 20 minutes: 10 minutes in as much flexion as possible and 10 minutes in as much extension as possible (see Fig. 53.4). This massage anaesthetizes the affected tissue. Manipulation in all the impaired directions is now performed. It is important for the manipulations to be within normal physiological limits; attempting to overstretch the medial collateral ligament has no part in restoration of range at the knee joint, as it would create instability.

Manipulation techniques

There are four techniques.

Manipulation in flexion

The patient lies supine or adopts a half-lying position on the couch. The hip is bent and the knee flexed as far as it will go. The therapist presses the ipsilateral hand just above the ankle. The other hand is placed on the knee (Fig. 53.5a), with the fingers on the ligament to feel the 'tearing' during the manipulation. The slack is taken up at the end of flexion and the tibia is forced vigorously backwards to the end of range. A snap is heard, which demonstrates the tearing of the adhesions.

Manipulation in extension

The patient lies supine on the couch, the knee extended as far as possible. The therapist stands to the side, level with the knee. The ipsilateral hand supports the heel and the other hand encircles the knee, just below the patella (Fig. 53.5b). The knee is slightly flexed and suddenly moved into full extension with a strong, quick jerk. A tiny snap is heard, which indicates that the adhesion has broken.

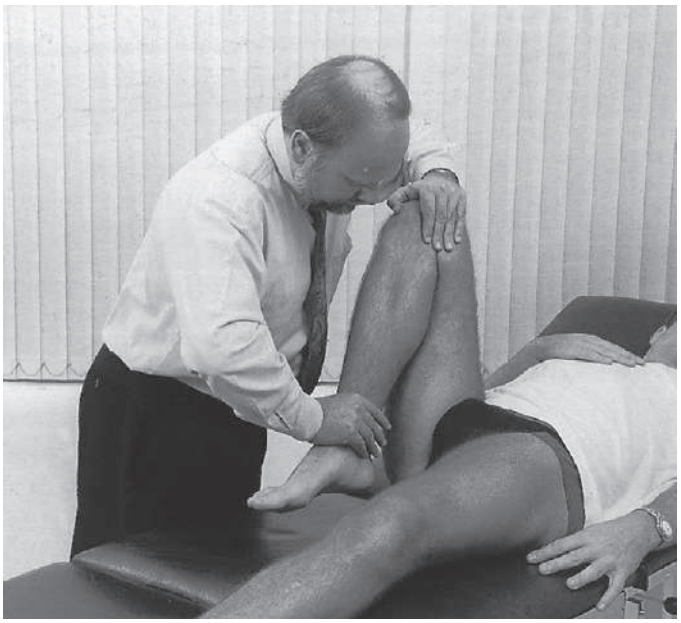
Manipulation in lateral (external) rotation

The patient adopts the half-lying position, with the hip flexed and the knee at 90°. The therapist stands level with the patient. The ipsilateral hand is clasped behind the heel, which rests on the couch. The foot is dorsiflexed, the inner side against the therapist's forearm. The contralateral hand is used to stabilize the knee and the femur (Fig. 53.6a). Lateral rotation is now easily performed by using the foot as a lever. The slack is taken up at the end of range and the manipulation performed by a quick adduction movement of the shoulder.

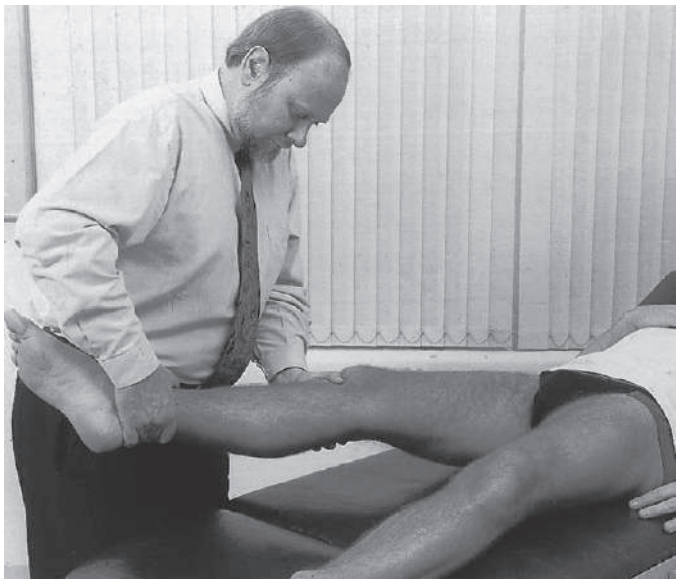
Manipulation in medial (internal) rotation

It is unusual for an adhesion of the medial collateral ligament to cause pain on medial rotation. If it does, this technique is used. The patient adopts a half-lying position, with the hip and knee well flexed. The therapist stands at the affected side, level with the thigh, and encircles the heel with both hands. The contralateral hand clasps the inner side of the foot, while the arm passes behind the leg and supports it (Fig. 53.6b).

In order to protect the lateral ligaments of the ankle, fixation is applied to the distal end of the leg and not to the foot.



(a)



(b)

Fig 53.5 • Manipulation to rupture medial collateral ligament adhesions: (a) in flexion; (b) in extension.

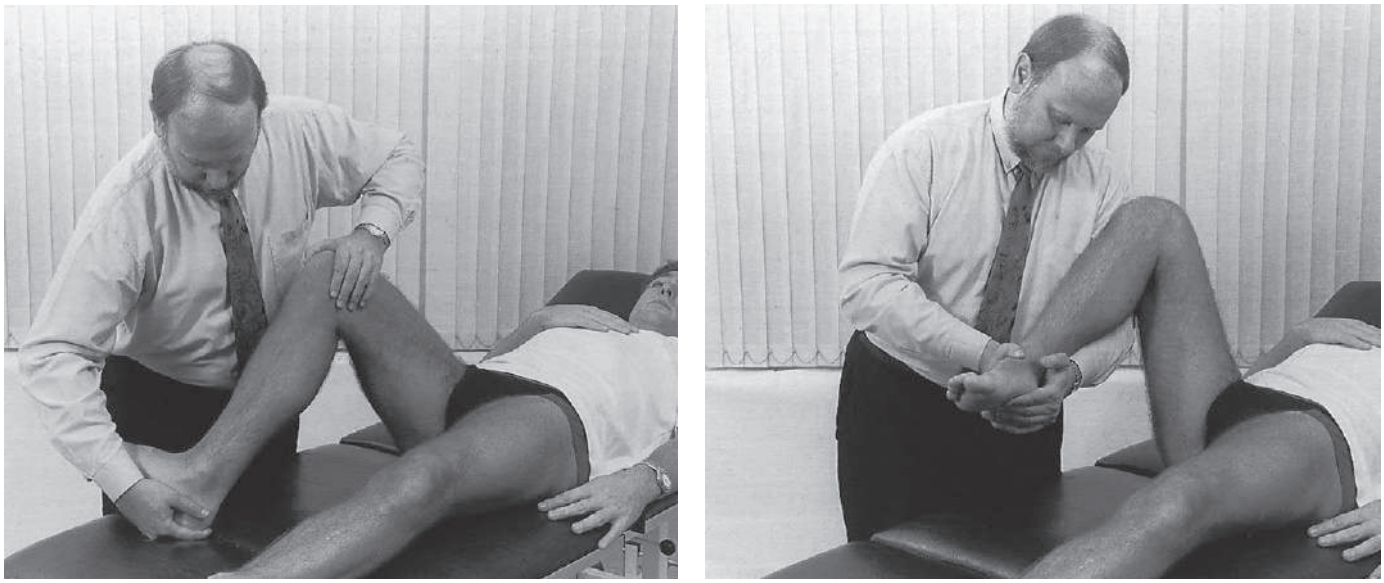
If the outer hand is kept on the calcaneus and fibula, the lateral ligaments of the foot are protected.

The foot is brought as far into medial rotation as possible, and the manipulation is performed with a quick movement of both hands.

After-treatment and results

Immediately after manipulation, there is a full and painless range of movement. It is remarkable that there is no pain or reaction in the joint.

For the first few days after the manipulation, the patient must repeatedly perform flexion, extension and rotation movements in order to maintain the regained mobility at the



(a) (b)

Fig 53.6 • Manipulation to rupture medial collateral adhesions: (a) in lateral rotation; (b) in medial rotation.

Table 53.4 Summary of isolated medial collateral ligament injuries

	Symptoms/signs	Treatment
Acute stage	Typical history Swelling Capsular pattern Three localizations	Infiltration with triamcinolone or daily deep friction followed by progressive active movements
Subacute stage	Decreasing traumatic arthritis Positive valgus test	Deep friction (20 minutes) 3 times a week, followed by active movements
Chronic stage	Localized pain after exercises Slight limitation of movements Positive valgus (and external rotation)	Manipulation in all the impaired movements after vigorous deep friction, followed by active exercises
Differential diagnosis	Medial collateral ligament bursitis Loose body	

knee. Flexion is performed by squatting and extension by a hyperextension movement during weight bearing. Rotational movements in a lateral or a medial direction are achieved during weight bearing with a slightly bent knee, whereby the femur rotates inwards or outwards on the stationary tibia. These exercises are performed every few hours during the following days.

Usually, a single manipulation followed by the appropriate after-treatment suffices to cure chronic ligamentous adhesions of the medial collateral band, no matter how long the problem has existed.

The symptoms/signs and treatment of isolated medial collateral ligament injuries are summarized in Table 53.4.

Lateral collateral ligament

Diagnosis

The lateral collateral ligament is very rarely sprained. The mechanism of sprain is a forced varus movement on an outstretched knee. Since the ligament lies relatively distant from the joint, the immediate articular signs are less dramatic than in sprains of the medial collateral ligament. Although the knee is warm and contains some fluid, the capsular pattern is minor or even absent. Varus strain hurts and palpation reveals the exact localization.

Practitioner's checklist

- When the uppermost, immobile part of the medial collateral ligament is affected, limitation does not occur in the chronic stage; manipulation is of no use and localized deep friction without forcing the joint suffices.
- When ligamentous strain is secondary to a loose body at the inner side of the joint (see p. 689), deep friction is ineffective and manipulation makes matters worse.
- Friction and manipulation also worsen an inflamed bursa under a ligament; treatment is aspiration (see p. 695)

The natural history of a lateral collateral ligament injury is as follows. The acute stage, with light traumatic arthritis, lasts 2 weeks. Thereafter, the lesion enters the subacute stage: pain and discomfort during movement only, a further decline of the capsulitis and a painful varus test. A chronic sprain of the lateral collateral ligament causes no adhesions; thus all movements will be free but a strong varus movement remains painful.

Fig 53.7 • Friction to the lateral collateral ligament.

Treatment

Grade III lateral collateral ligament tears, especially if they are combined with ruptures of the posterolateral ligament complex, should be repaired immediately after the injury. Isolated grade I or grade II lesions of the lateral collateral ligament are treated conservatively.

Treatment in the acute stage consists of infiltration with triamcinolone (only during the first 48 hours) or daily deep friction.

During the subacute or chronic stage, only deep friction is effective. Because adhesions never form in this condition, manipulation is not performed.

Technique: deep friction

The patient lies supine, the knee in extension. The therapist sits on the opposite side. The index finger, reinforced by the middle finger, covers the affected part of the ligament. The thumb is placed at the inner side of the knee, to be used as a fulcrum (Fig. 53.7). Via alternate flexion and extension of the wrist, the forefinger will be moved to and fro over the ligament, while the thumb is kept still. In a recent injury, when there is a lot of local tenderness, a gradual start to the friction is necessary – preparing the area for the 2 minutes of thorough friction which is required. In chronic cases, 20 minutes' friction may be necessary, three times a week, for about 2 weeks.

Coronary ligaments

Cyriax[39] was the first to draw attention to sprains of the coronary ligaments, which are very common but mostly go undiagnosed because the localization of the pain and nature of the onset resemble a meniscus lesion or a sprain of the medial collateral ligament. Recently, magnetic resonance imaging (MRI) examination[40,41] and arthroscopy[42] have confirmed the existence of coronary ligament lesions. The treatment is simple but effective and the injury is usually cured after 1 or 2 weeks' deep friction or after one infiltration. Without treatment, the lesion recovers spontaneously in about 3 months, although in some cases it can persist indefinitely.

Diagnosis

The classic mechanism of a coronary ligament sprain is almost identical to that of a torn meniscus. The patient describes a rotational strain, usually during slight flexion of the knee. As in meniscal lesions, the medial side is more often damaged than the lateral. This is the typical lesion incurred by a tennis player, twisting the body, and thus the femur, on the stationary tibia during a forced and unintentional forehand drive. If the foot is anchored to the ground, the medial coronary ligament may be overstretched. The important fact which differentiates a coronary ligament sprain from a meniscal lesion is that in the former the knee is not locked after the accident. The patient can straighten the knee and walk normally, and is often able to continue playing. It is only after some hours that serious pain, swelling and limitation of movements appear.

The next day, the patient is unable to walk without a limp. Clinical examination reveals traumatic arthritis: warmth, fluid and a capsular pattern. Medial rotation is painful when the lateral coronary ligament is overstretched and painful lateral rotation implicates the medial coronary ligament. Applying valgus and varus strain does not hurt, so eliminating a lesion of the collateral ligaments. The appropriate ligament is found to be tender. Palpation must be performed with the knee well bent and in rotation away from the affected side.

During the subacute stage, the capsular pattern subsides slowly, but if adequate treatment is not given, warmth and fluid can persist for several months. During this time, not only lateral or medial rotation but also passive extension remains painful. These features can be explained by the fact that the menisci are forced forwards during full extension of the knee, which pinches the inflamed ligaments and elicits pain.

In the absence of adequate treatment, the lesion can become chronic. Although no adhesions will form here, a self-perpetuating inflammation will persist in the healed tissue, giving rise to recurrent, though slight, disability.

Differential diagnosis, history and examination

The differential diagnosis from a meniscal lesion is extremely important and once again relies almost entirely on history and clinical examination. In our opinion, it is unwise to refer a patient for arthroscopy before a lesion of the coronary ligament has been excluded. Meniscal lesions are so common that certainly not all cause symptoms.[43,44] Although arthroscopy detects meniscus tears with almost 100% accuracy, it does not necessarily follow that the detected tears are the cause of the patient's symptoms.[45] The differential diagnosis between a meniscus lesion and a coronary ligament sprain is thus made on information gained from the history and clinical examination.

Although the same mechanism is responsible for both a torn meniscus and a sprained coronary ligament, there is an essential difference between their histories. In a subluxated meniscus, passive extension is lost at once, whereas in a coronary ligament sprain, extension becomes impaired only when the traumatic arthritis is established.

In the acute stage of a coronary ligament sprain, the clinical examination shows some extension to be lost because of the post-traumatic arthritis, which implies more limitation of

flexion (probably 45–60°). The end-feel is muscle spasm. In an impacted meniscus, however, limitation of extension contrasts with a full range of flexion, the end-feel of extension being a springy block.

In the subacute stage of a coronary ligament sprain, when extension is painful at the end of range or only slightly limited, the end-feel is never springy but rather is ligamentous.

Treatment

The treatment of a coronary ligament sprain, whether acute, subacute or chronic, is the same: deep friction. Although it is reasonable to infiltrate the ligament with steroid, this hardly ever succeeds because adequate infiltration of the thin line of tissue between the meniscus and the tibia is technically almost impossible. However, infiltration of the coronary ligament can be considered in two instances: if the lesion is located on the tibial border, at the ligamentoperiosteal insertion of the ligament, and in persistent problems after meniscectomy; here the hypertrophied scar tissue in the line of intersection often causes permanent trouble but infiltration with triamcinolone usually gives very good results.

Technique: friction to the medial coronary ligament

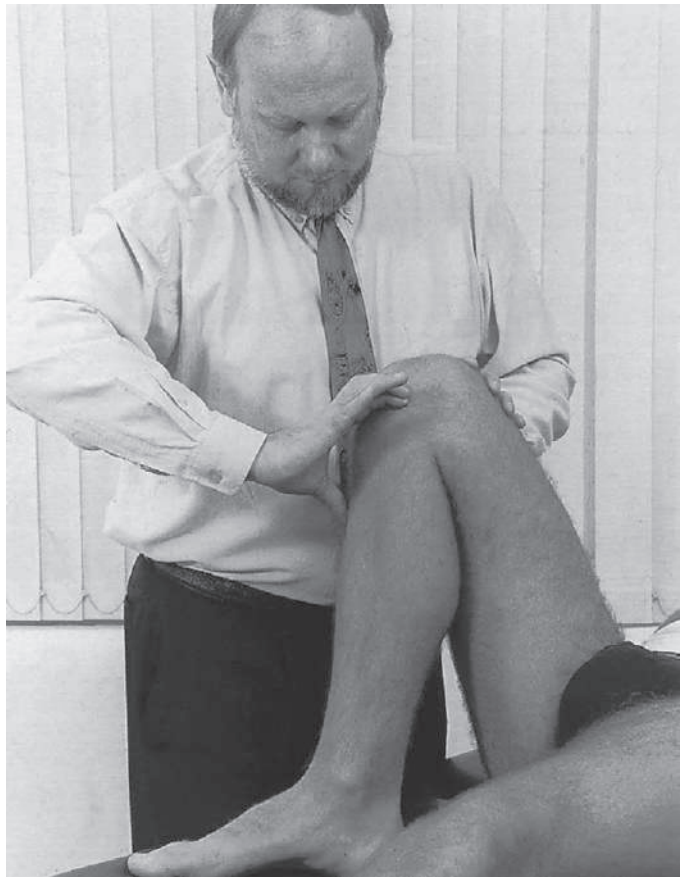
The patient lies on the couch with the knee flexed to 120°. Lateral rotation moves the medial tibial condyle further away from the meniscus, rendering the medial coronary ligament more accessible to the palpating finger. The physiotherapist sits or stands level with the injured knee. The tibial condyle is identified and the site of the lesion ascertained.

The index finger of the ipsilateral hand is placed on the lesion, and reinforced by the middle finger. Because deep friction only works if it is applied to exactly the right point, it is important to make sure that the fingertip lies on the coronary ligament and not elsewhere. Therefore the fingertip must press downwards on the tibial condyle, moving the coronary ligament between finger and tibial condyle. *Consequently, the index fingernail lies horizontally and the finger is slightly flexed.*

The thumb is placed at the lateral side of the knee, where it is used as a fulcrum. The therapist ensures a vertical downward pressure of the index finger by placing the thumb as distally as possible on the leg. Massage is now given by drawing the fingertip horizontally along the condyle, around the fixed thumb (Fig. 53.8). As usual, there is an active phase of sweeping the index finger anteriorly and a relaxing phase when the finger is moved backwards to the previous position. In order to give sufficient excursion to the fingertip, the therapist should not sit or stand too close to the patient, so that the entire arm, elbow and wrist can be moved during the massage.

Technique: friction to the lateral coronary ligament

The technique is the same as described for the medial coronary ligament but the therapist stands on the opposite side of the damaged knee. The well-bent knee is now brought into inward rotation. Friction is performed by the contralateral hand. Once again, the fingertip of the index finger is placed *horizontally* on the affected ligament. The thumb now lies downwards on the inner side of the knee and acts as a fulcrum (Fig. 53.9). Friction is given by a movement of the whole arm, drawing the index finger along the tibial condyle.



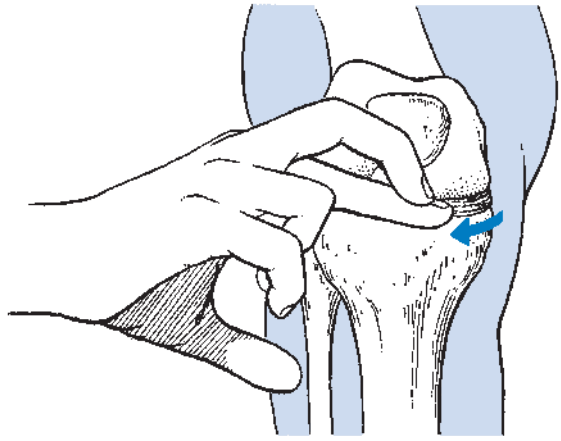


Fig 53.8 • Friction to the medial coronary ligament.

Comments

In acute sprains, friction is given daily for 10 minutes, in subacute or chronic cases on alternate days for 10–15 minutes. As no alternative treatment exists for coronary ligament sprains, the technique must be absolutely correct. To reach the ligament, it is vital to place the index finger flat on the ligament and the thumb on the opposite side of the leg as caudally as possible. If these principles are observed, all patients recover after 2–3 weeks' treatment, regardless of whether they are first seen the day after the sprain or many months later. If appropriate friction is unsuccessful, considerable doubt must arise as to the diagnosis and the patient must be re-examined at once.

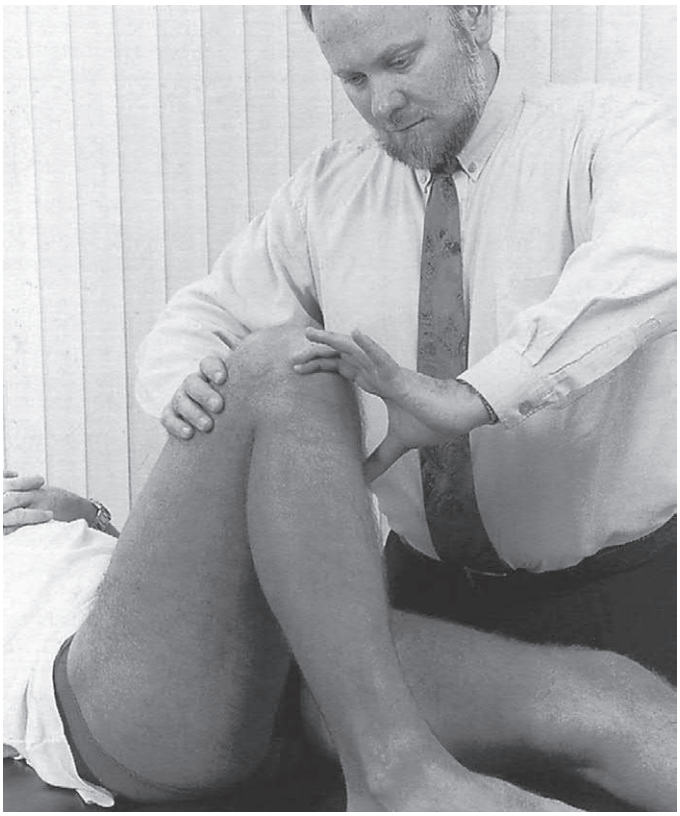


Fig 53.9 • Friction to the lateral coronary ligament.

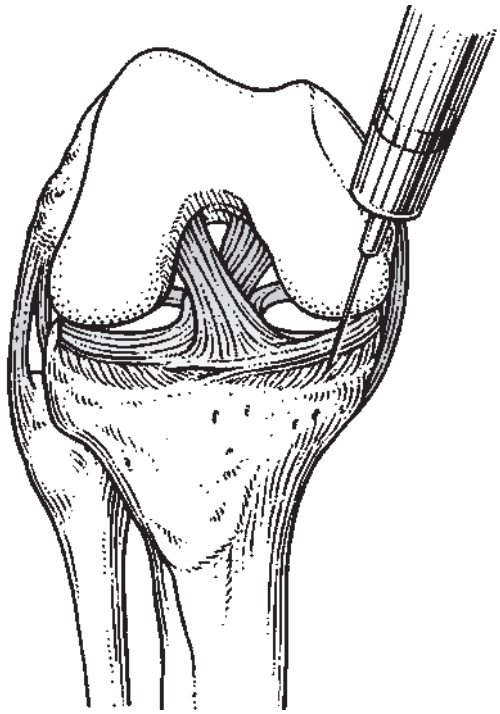


Fig 53.10 • Infiltration of the medial coronary ligament.

Technique: infiltration of the medial (lateral) coronary ligament

The knee is well bent and brought into lateral (medial) rotation. The tibial condyle is identified and the site of the lesion ascertained. A small, thin needle is fitted to a 1 mL syringe, filled with 10 mg of triamcinolone and inserted from above until it hits bone. A series of small droplets are injected all over the tender area (Fig. 53.10).

Anterior cruciate ligament

An isolated tear of the anterior cruciate ligament may be caused by a hyperextension strain combined with medial rotation.[46] Alternatively, the ligament may tear when the knee is loaded in valgus position, with the femur internally rotated and the pivot shifted to the lateral femorotibial compartment.[47] Both situations occur frequently in soccer, basketball and volleyball, when an athlete decelerates, pivots/turns quickly or lands heavily from a jump.[48] Combined lesions of the anterior cruciate and medial collateral ligaments, together with a torn meniscus, occur in contact sports (e.g. rugby, American football and judo).[49,50] It has also been demonstrated that chronic anterior cruciate deficiencies can result from longitudinal meniscal tears.[51,52]

Diagnosis

Immediate swelling after the injury indicates haemarthrosis, which suggests a complete rupture of the ligament.[53–55] In moderate sprain, only mild traumatic arthritis may result. Blood is a strong irritant to capsule and cartilage,[56] and should be removed at once. If the patient is an athlete competing in a high-performance sport, arthroscopic assessment of the integrity of the anterior cruciate ligament must always follow the detection of a traumatic haemarthrosis. Alternatively, the patient is referred for MRI examination because this technique has become the imaging method of choice to evaluate the integrity of the anterior cruciate ligament.[57]

Further clinical examination discloses whether the unmodified anterior drawer test and its modification at 30° are painful or have an excessive range. Excessive anteroposterior movement shows the ligament to be ruptured or elongated. If the patient complains of pain only and no instability is detected during this test, inflammation at the ligamentoperiosteal border is present. It is very important to distinguish an inflamed but strong ligament from a painless and elongated one. In painful inflammation, treatment is infiltration with 20 mg of triamcinolone at the ligamentoperiosteal junction, no matter whether the lesion is new or of long standing. Because the ligament does not lie within the reach of the palpating finger, massage is not an alternative treatment. Spontaneous cure of a sprained anterior cruciate ligament is very slow; it can take months or years, or the lesion may fail to recover at all.

Sometimes a sprain leads to permanent lengthening, resulting in serious instability. The problem of instability is discussed later but it is as well to remember that lengthening of the anterior cruciate ligament, or even complete rupture, does not always lead to permanent functional instability.[58–60] For its anterior stability, the knee depends not only on the anterior cruciate ligament but also on the capsular ligaments, the integrity of the menisci and good proprioceptive reflexes from the

quadriceps and hamstring muscles.[61] Messner and Maletius re-examined 22 patients with minor knee instability caused by partial rupture of the anterior cruciate ligament after a mean of 12 years and found excellent knee function that showed no differences from that of a control group.[62]

Treatment

Treatment of a painful but strong anterior cruciate ligament consists of one or two injections at the site of the lesion. These injections should be given as soon as possible, regardless of whether the lesion is in an acute or chronic stage. The lesion always lies at the ligamentoperiosteal junction but sometimes it is hard to decide at which end. If there is any doubt about localization, the anterior end is infiltrated first; if no benefit has been gained within a week, the other end is then treated similarly.

Technique: infiltration of the anterior end of the anterior cruciate ligament

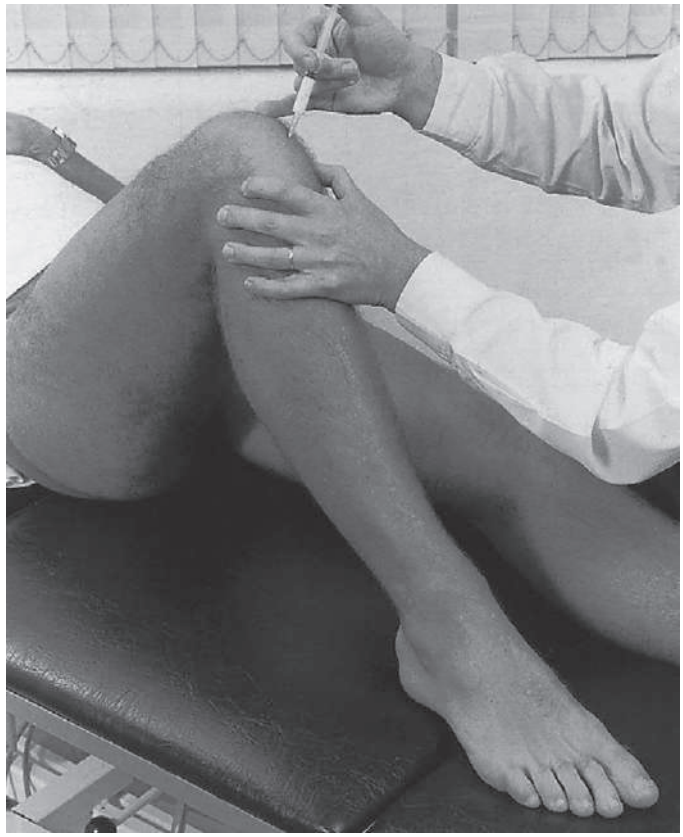
The patient lies on the couch, with the knee bent at a right angle. The border of the medial tibial condyle is identified, together with the medial side of the patella and the infrapatellar tendon. A point is chosen just medial to the inferior edge of the patella, about 3 cm above the tibial rim. A 5 cm long needle is introduced here, in a backward and medial direction, aiming at the spine of the tibia (Fig. 53.11). The needle must cross dense tissue before it touches bone. If bone is hit immediately, this indicates that the needle does not lie within the ligament. If the correct position is reached, an area 1 cm^2 is infiltrated with 20 mg of triamcinolone, by means of a series of small withdrawals and reinsertions. It is important to note that the infiltration must be made only at the ligamentoperiosteal junction, which is when the tip of the needle hits bone.

If the entire area has been infiltrated correctly, recovery will be complete within 2 weeks. If the injection has only afforded partial relief, a second injection can be given. More than two injections are seldom required.

Technique: infiltration of the posterior end of the anterior cruciate ligament

The patient lies prone, with the knee extended. The medial and lateral femoral condyles form the landmarks. The free thumb is placed on the lateral condyle. The femoral attachment of the anterior cruciate ligament lies at the medial surface of the lateral condyle. It cannot be reached by a downward thrust and the needle must be brought in from a medial aspect, using an almost horizontal insertion. A 6 cm long needle is inserted at the apex of the medial condyle and moved at an angle of 30° to the horizontal in the direction of the lateral condyle. This approach ensures that the needle passes well posterior to the popliteal vessels (Fig. 53.12). The needle has to penetrate a tough ligamentous structure before it touches bone. The infiltration is given via a series of small droplets at different spots of the tenoperiosteal junction.

More than two injections are hardly ever required. The results are uniformly good, provided the right point has been reached. Obviously, these injections only have an effect on inflammation and pain and do not alter instability.



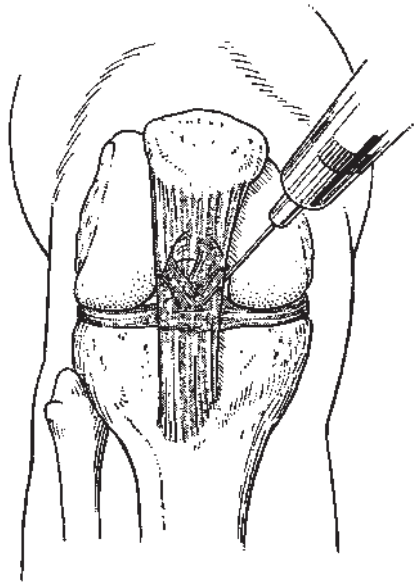


Fig 53.11 • Infiltration of the anterior end of the anterior cruciate ligament.

Posterior cruciate ligament

A severe lesion of the posterior cruciate ligament is always serious because the ligament creates the axis about which the knee rotates.[63]

Furthermore, most patients with posterior cruciate ligament injuries have combined ligamentous and/or chondral knee

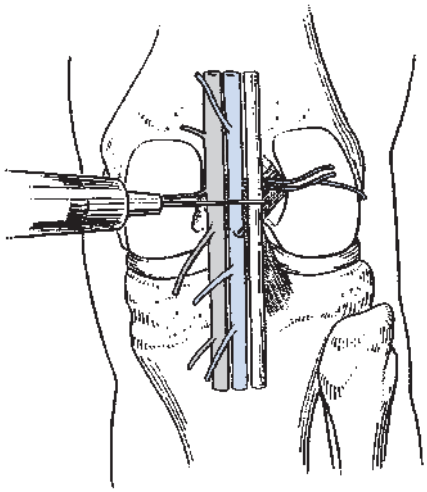


Fig 53.12 • Infiltration of the posterior end of the anterior cruciate ligament.

damage.[64] Therefore, complete rupture of the ligament will often lead to considerable instability and limited function of the knee.[65]

A mild, isolated sprain at the tibial or femoral insertion, however, does not lead to gross instability.[66] Nevertheless, it can induce persistent problems due to the formation of painful, self-perpetuating inflammation.

In this section we deal with mild sprains only. Complete and/or combined ruptures of the posterior cruciate ligament are discussed in the online chapter *Disorders of the inert structures: ligamentous instability*. A severe lesion should always be suspected if haemarthrosis is present. Escape of blood through a rupture of the posterior capsule may mask this sign.

Diagnosis

Motor vehicle accidents are the most common cause of injury, but sports-related trauma (football, skiing) has increased in recent years. The usual mechanism of injury in motor vehicle accidents is a 'dashboard' injury: during a car crash the femur is forced forwards on the immobilized tibia.[67] Sports-related injuries result from severe hyperextension of the knee or from hyperflexion with the foot typically plantarflexed. The latter mechanism is the most common cause of isolated posterior cruciate ligament injuries.[68]

During the first few days after the accident, examination shows traumatic arthritis, with a warm and swollen knee containing clear fluid. Tests for the collateral ligaments are negative but internal rotation can be painful at the end of range. The posterior drawer test demonstrates pain rather than laxity. Shearing the tibia laterally on the femur is also painful, as is resisted flexion with the knee in a 90° flexed position. As a rule, the lesion lies at the tibial border. Sometimes a small bony chip may be avulsed and is visible on a plain radiograph.[69]

During the last few decades, MRI has proven to be very accurate in the diagnosis of posterior cruciate ligament lesions and in demonstrating associated injury.[70–72]

After the traumatic arthritis has subsided, the posterior drawer test remains painful, sometimes for years; without proper treatment the patient will be permanently unable to run or play sports.

Treatment

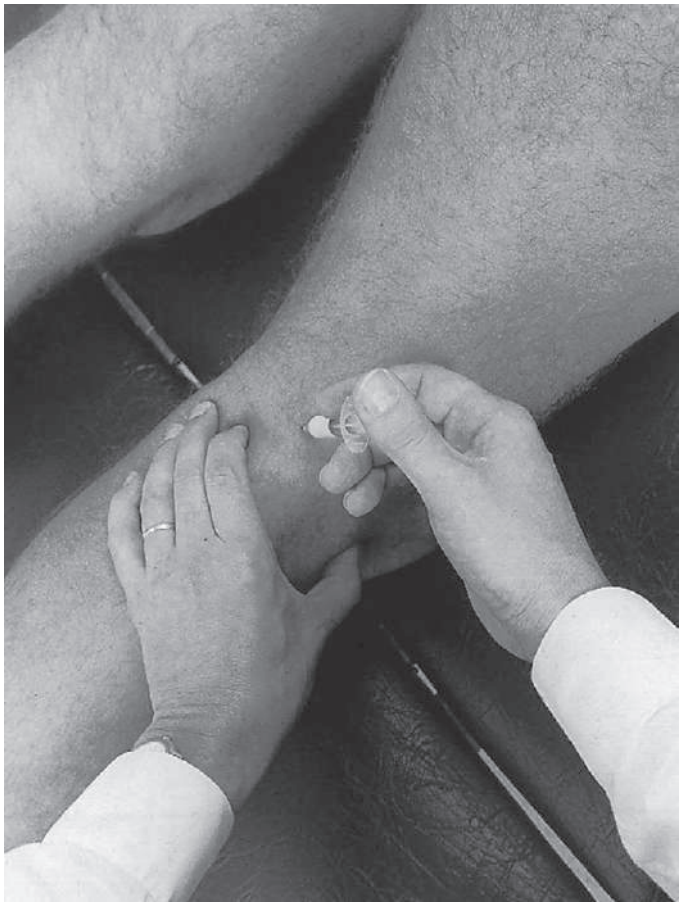
Treatment of posterior cruciate ligament lesions depends on their severity, the existence of concomitant lesions and the presence and degree of laxity.[73,74] Isolated, partial posterior cruciate ligament injuries (grades I and II) are best treated non-operatively, while complete injuries (grade III) may require operative treatment based on clinical features. All combined ligamentous injuries usually respond best to surgical management. In recent years, several studies have demonstrated that knees with an isolated injury to the posterior cruciate ligament, without concomitant articular damage, may be successfully managed conservatively and that the majority of athletes with isolated posterior cruciate ligament injuries who maintain muscle strength return to sports without any functional disability.[75–78]

When there is no gross instability but merely pain, treatment consists of one or two injections at the site of the lesions; if given during the acute stage, this may avoid many months of traumatic arthritis.

The results are remarkable, and after just a few weeks the patient is able to return to activity: for example, football. In chronic sprain, the infiltration produces equally good results. The lesion is inaccessible to friction, and therefore infiltration is the only successful form of treatment. The posterior end of the ligament is much more frequently affected than the anterior. Therefore, this end should be infiltrated first. If the condition has not improved after 2 weeks, the anterior side should be treated.

Technique: infiltration of the posterior end of the posterior cruciate ligament

The patient lies prone, with the knee extended. The landmarks here are the lateral femoral condyle – which is easily identified – the popliteal artery and the posterior margin of the tibia. The latter cannot be palpated directly and must be marked by



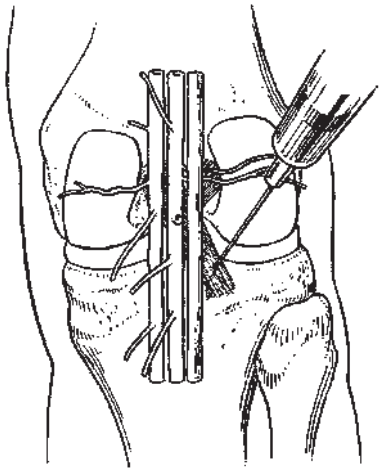
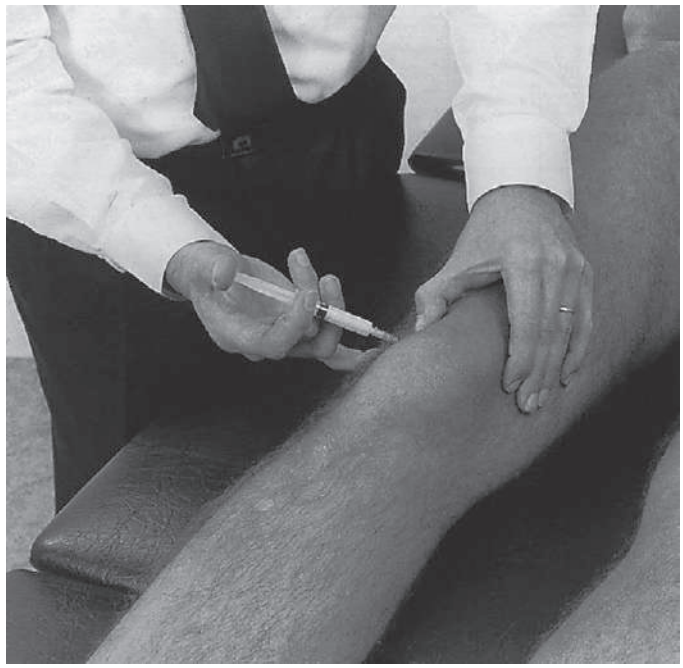


Fig 53.13 • Infiltration of the posterior end of the posterior cruciate ligament.

palpation of the joint line at the anterolateral side. The level of the posterior edge of the tibia can then be estimated. The posterior end of the posterior cruciate ligament is at the midpoint of the tibia, extending somewhat to the lateral side. A 5 cm long needle is inserted at the apex of the lateral femoral condyle, about 2 cm above the estimated level of the tibial surface and directed inferiorly and medially, at about 60° to the horizontal. The palpating index finger is placed on the popliteal artery and the joint line. The lateral approach,



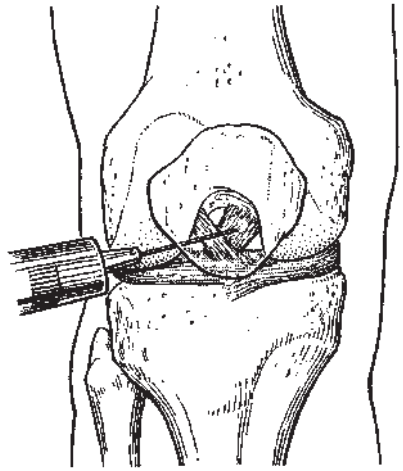


Fig 53.14 • Infiltration of the anterior end of the posterior cruciate ligament.

together with palpation of the artery, avoids the popliteal vessels. The tip of the needle is advanced until the characteristic feel of a ligament is encountered before bone is felt (Fig. 53.13). If the needle passes without obstruction and hits cartilage, this indicates that it has passed into the knee joint. This is too far proximal and the tip should be moved in a more oblique direction until it penetrates ligament and then touches bone. This is the exact area sought, just below the articular edge of the tibia. Infiltration of 20 mg of triamcinolone should be given here by means of a series of small insertions and withdrawals along the mediolateral borders.

Technique: infiltration of the anterior end of the posterior cruciate ligament

The patient lies supine, with the knee extended. The medial and lateral femoral condyles are identified. The anterior end

Table 53.5 Summary of ligamentous lesions

Acute major lesions (gross or combined sprains)				
Immediate swelling Immediate functional incapacity Haemarthrosis				
Lesions:				
Rupture of a cruciate 'Unhappy triad'				
Isolated sprains				
History: Information about the injury should be as detailed as possible Swelling and functional incapacity appear some time after the accident Examination findings and treatment depend on the stage of the lesion:				
Stage	**Medial collateral ligament**	**Lateral collateral ligament**	**Coronary ligaments**	**Cruciate ligaments**
Acute				
Warmth and fluid	Present	Present	Present	Present
Capsular pattern	Gross	Slight	Moderate	Slight
Local tenderness	Present	Present	Present	Absent
Treatment	Infiltration or friction	Infiltration or friction	Friction	Infiltration
Subacute				
Warmth and fluid	Reduces	Reduces	Reduces	Reduces
Capsular pattern	Reduces	Reduces	Reduces	Reduces
Positive ligamentous tests	Valgus External rotation	Varus	Rotation	Drawer
Treatment	Friction and mobilization	Friction	Friction	Infiltration
Chronic				
Formation of adhesions?	Yes	No	No	No
Limited range of motion?	Yes	No	No	No
Treatment	Manipulation	Friction	Friction	Infiltration

of the ligament lies at the lateral surface of the medial condyle and is covered by the patella, so that direct access from above is not possible. The physician has to move the patella as far as possible medially and upwards by tilting the outer edge with the thumb. A 5 cm long needle is now inserted just under the inferolateral edge of the patella, and pushed up in the direction of the medial condyle, until it hits bone (Fig. 53.14). It is then moved little by little, until ligamentous resistance is felt. A series of small droplets are injected when the needle is felt to touch bone after piercing the ligament.

Ligamentous lesions of the knee are summarized in Table 53.5.

Access *Disorders of the inert structures: ligamentous instability* and the complete reference list online at **www.orthopaedicmedicineonline.com**

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Disorders of the contractile structures

54

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Extensor mechanism

Quadriceps strains and contusions

A muscular tear of one of the quadriceps bellies is a common disorder in sprinters and soccer players. An abrupt, vigorous contraction during a sprint breaks some of the muscle fibres and is felt as a sudden, painful 'giving way' at the front of the thigh. Alternatively, the muscular lesion may result from a direct contusion during contact sports (judo or American football), known as 'Charley Horse'.

Patients who suffer an acute quadriceps strain will usually know right away. They are typically involved in sports requiring kicking, jumping, or initiating a sudden change in direction while running. Frequently, a sharp pain is felt, associated with a loss in function of the quadriceps. Sometimes pain will not fully develop during the athlete's activity while the thigh is warm; consequently, the extent of the injury is underestimated. Stiffness, disability and pain then set in some time afterwards, e.g. late at night, and the following morning the patient can walk only with a limp.[1]

Clinical examination shows a normal hip and knee, although passive knee flexion is painful or both painful and limited, depending on the size of the rupture. Resisted extension of the knee is painful and slightly weak. As a rule, the lesion is in the rectus femoris, usually at mid-thigh level.[2] The affected muscle belly is hard and tender over a large area. Sometimes a haematoma can be palpated. In serious lesions, a space can be detected by palpation. This is particularly the case in major ruptures just above the suprapatellar tendon, which occur mainly in patients over 40 years of age.[3] In such cases, there is not only pain but also weakness during resisted extension, and the patient is unable to straighten the knee actively over the last 30°.

Ultrasonography is an excellent imaging modality for visualizing the quadriceps muscles; it has the ability to image the muscles dynamically and assess for bleeding and haematoma.[4]

Development of myositis ossificans as a complication of a thigh contusion is not uncommon in adolescents and young adults.[5] It occurs in 9% of quadriceps contusions and seems to be associated with five risk factors (knee motion less than 120°, injury occurring during American football, previous quadriceps injury, delay in treatment of more than 3 days and ipsilateral knee effusion).[6] Early diagnosis is important and is usually made with the help of sonography.[7,8] There is no proper

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treatment but the lesion undergoes spontaneous cure within 2 years.[9] Therapy with deep transverse massage is contraindicated.

Treatment

Initial haemorrhage may be reduced by elastic strapping and an ice bag over the lesion. In the case of a contusion, holding the knee in 120° of flexion for 24 hours following a quadriceps contusion also appears to shorten the time needed for the patient to return to unrestricted full athletic activities.[10]

Minor ruptures in the quadriceps muscle require the same treatment as muscular lesions elsewhere. As soon as the patient is seen, the lesion is infiltrated thoroughly with 50 mL of procaine 0.5%. From the next day on, deep transverse friction is given, followed by active isometric contractions with the muscle in a shortened position.

When the rupture is major, operative suture may be advised, especially if the patient is an athlete with high functional demand on the knee extensors. It is then important that surgical treatment is undertaken as soon as possible.

Technique: deep friction to the quadriceps muscle

The patient sits with the knees outstretched. Flexion at the hips, together with maximal extension of the knees, induces complete relaxation of the quadriceps. The physiotherapist stands at the patient's side. The correct level is sought. The fingers of one hand are placed deep to the affected fibres. The other hand can reinforce the palpating one. The thumbs are placed laterally to be used as a fulcrum. By flexion of the fingers, during an upward-drawing movement, the physiotherapist drives all the fibres between fingers and femur (Fig. 54.1).

At the end of the movement, the fingers are slightly extended and brought back to the previous spot, under the lesion. The skin must move with the fingers. Since this is an extremely tiring technique, periods of rest must be built into the treatment session, which lasts about 20 minutes.

Fig 54.1 • Friction in minor rupture of the quadriceps.

After the friction, active isometric contractions are done over 5–10 minutes. Again, this is carried out in a fully relaxed position, so that the contraction cannot exert tension on the healing tissue (see Ch. 5).

Treatment with deep friction and contractions is given daily for the first week and on alternate days from the second week onwards. In athletes, there is a significant tendency for the lesion to recur and the patient should not restart a training programme until a week after complete clinical cure. In the meantime, treatment should continue. The training programme must be built up gradually over the following 3–4 weeks.

Adherent vastus intermedius

This condition occurs typically after a fracture of the femur. There is gross limitation of flexion, whereas the other knee movements are all full-range. Only surgical treatment can achieve an acceptable outcome.

Tendinous lesions about the patella

The quadriceps tendon has a superficial and a deep layer. The superficial layer runs without interruption from the quadriceps muscle, over the patella to the tibial tuberosity. The deep layer inserts all around the patellar border, effectively making the patella a sesamoid bone in the tendon. Tendinous lesions therefore occur not only at the inferior aspect of the bone but also at the superior, medial and lateral borders. Tendinosis about the patella is a typical overuse phenomenon in sports characterized by high demands on leg extensor speed and power, such as volleyball, basketball, soccer and athletics. It was first described by Sinding-Larsen in 1921[11] and Johansson in 1924.[12] Kulund[13] and Ferretti[14] also reported cases of tendinosis at the upper border of the patella. Cyriax[15] describes three possible sites: the upper border (suprapatellar tendinosis), the apex – the classic 'jumper's knee' (infrapatellar tendinosis) and at either side of the patella (tendinosis of the quadriceps expansion) (Fig. 54.2). The lesion was uncommon until the mid-1960s, when the syndrome of jumper's knee became more and more frequent because of increased training and higher performance goals for athletes.[16,17] During the last few decades, these lesions have reached epidemic proportions[18]; in sports medicine centres, patellar tendinopathy is one of the leading reasons for consultation and often contributes to the decision to give up an athletic career.[19]

The history is obvious: during or after exertion, there is localized pain at the front of the knee. In mild examples, there is probably only a little pain after activity, whereas in a severe case pain forces the athlete to stop, and pain at rest can also supervene. The patient also states that walking upstairs or standing up from sitting is painful. The lesion can be classified by its symptoms into four stages (Table 54.1).[13,20,21]

Clinical examination reveals a normal knee with a full and painless range of movement and normal ligamentous tests. Only resisted extension is painful or uncomfortable. When the history suggests a slight quadriceps tendinitis but resisted extension is negative, the patient should take part in a training

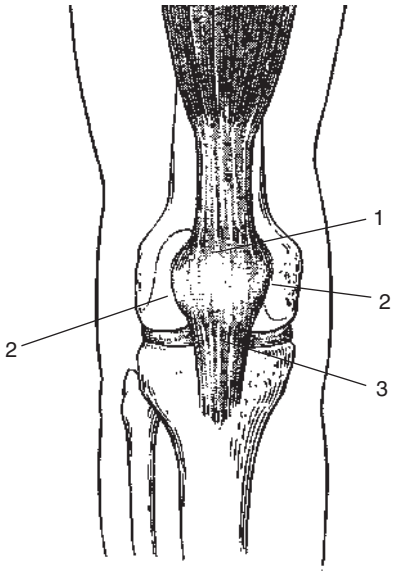


Fig 54.2 • Localizations of quadriceps tendinitis (with permission from Cyriax[15]: p. 408): 1, suprapatellar; 2, expansion; 3, infrapatellar.

Table 54.1 Staging of quadriceps tendinitis[21]

Stage	Symptoms
I	Pain after activity
II	Pain at onset, disappearing during activity and reappearing after it
III	Continuous pain, inability to perform
IV	Weakness due to rupture

session and be re-examined immediately thereafter; this will probably reveal a minor lesion.

Palpation identifies the site. To make the deeper layers of the tendon accessible for the palpating finger, the patella must be tilted as much as possible (see pp. 716 and 717).

Conventional radiographic techniques are of no use in the diagnosis of quadriceps tendinitis. Ultrasonography, however, seems to have some value in staging the disorder.[22–24]

On magnetic resonance imaging (MRI), swelling of the tendon and changes in the intrasubstance signal may be present.[25] However, the sensitivity and specificity of MRI is 75% and 29%, respectively. In younger patients with relatively mild symptoms, MRI does not show significant changes and in older, active patients changes may be present in asymptomatic knees.[26,27]

Treatment

Classically, conservative treatment for patellar tendinopathy consists of rest, ice, taping and anti-inflammatory drugs. Other modalities, such as electrotherapy, ultrasound, laser therapy and extracorporeal shock wave therapy, have also been used.[28] If these measures are insufficient or fail to relieve the symptoms, surgery is usually advised.[29] The patellar tendon is incised and the hyaline inflammatory tissue (usually the middle portion of the patellar ligament) is removed.[30,31] However, the benefits of open tenotomy can be questioned on the basis of the results, which show a success rate that varies between 58%[14] and 78%.[32] Sporting success is seen in about 50% of tenotomy patients, with a median time to return to pre-injury level of activity of 10 months.[33]

Eccentric training as a treatment option for tendinopathy has gained credit in recent decades. The technique was first presented by Curwin and Stanish in 1984, with encouraging results.[34] The programme is based on eccentric drop squats, which are performed with some level of discomfort. During the programme, the athlete should be removed from sports activity. When training becomes pain-free, load is increased by first increasing the speed of the eccentric phase and then adding weight.[35,36]

Our advice is to treat all peripatellar lesions with either a local infiltration of triamcinolone or a series of deep transverse friction.[37] At the upper border and the apex, infiltration with triamcinolone can be used safely and with very good results, providing the injection is done exactly at the right spot, using a weak solution (10 mg/mL) and according to the general principles of infiltration. Deep transverse friction is preferred if the lesion is too extended for infiltration. If a small spot remains resistant to treatment, it can subsequently be infiltrated.

Lesions of the quadriceps expansion at either side of the patella respond extremely well to deep transverse friction but infiltration is of no use at this site.

Because relative rest is always the first measure in tendinous lesions, the patient should not return to athletics until complete remission of the lesion has been achieved. After successful treatment, and in order to prevent recurrence, it is important to detect the possible causes of the tendinitis (inappropriate or faulty training programme or structural factors) and to correct these.[38]

In recalcitrant cases, one or two infiltrations with sclerosing solution to the tenoperiosteal border can be tried.[39]

Technique: deep friction to the suprapatellar tendon

The patient sits on the couch with the knee completely extended and the hip flexed, so that the quadriceps muscle is fully relaxed.

The patella must be tilted in such a way that the affected spot can be reached. This can be achieved by pressing the lower pole of the patella posteriorly with the web of the thumb or with thumb and index finger, which steadies the bone and forces the upper border upwards and forwards. With the middle finger of the other hand, the tender area can now be sought more easily. It is important to move the finger as far as possible under the inclined patella in order to catch the tendinous fibres of insertion deep under the bone. Deep friction is performed by pressing the tendon between the middle finger and the superior aspect of the patella (Fig. 54.3). The index finger is used to reinforce the middle finger. The thumb is placed as far distally as possible on the leg and used as a fulcrum. The friction consists of a to-and-fro movement of the whole forearm and hand, in a transverse direction over the tendinous fibres.

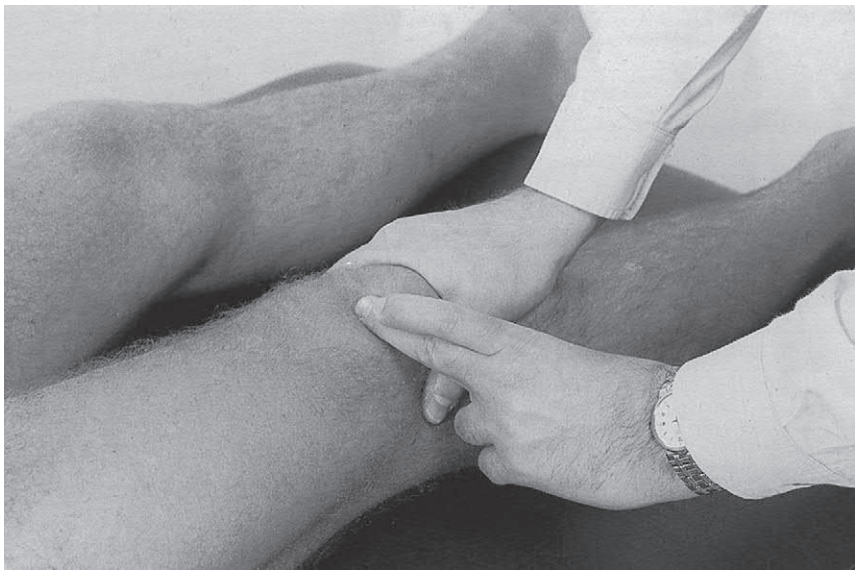


Fig 54.3 • Deep friction to the suprapatellar tendon.

Twenty minutes of really hard friction are needed to produce a good result, which may be expected after 10–20 sessions.

Technique: infiltration to the suprapatellar tendon

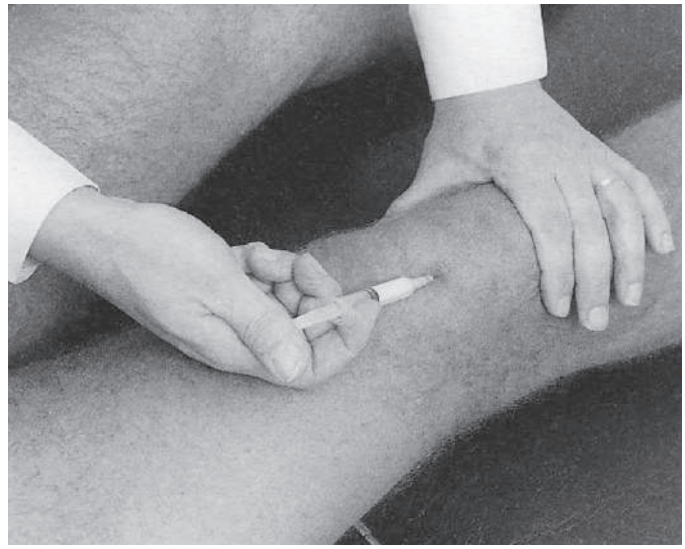
The patient sits with the knee outstretched and the hip flexed, so that the quadriceps is fully relaxed. The patella is fixed as described earlier. The upper edge of the patella is marked and the tender area identified. Usually it lies at the medial edge of the upper border, less frequently at the lateral edge of the upper border, and as a rule it is no larger than 1 or 2 cm. A spot is chosen about 1.5–2.0 cm above the centre of the affected area. A 3 cm needle fitted to a 2 mL syringe is introduced at this spot and moved in the direction of the patella (Fig. 54.4). The more the patella is tilted, the better the needle can reach the affected area. Small injections are given only when the tip of the needle is felt to pierce tendinous structure and touch bone. Considerable resistance is felt during the injection. Caution is taken to infiltrate the whole tender area by the usual technique of fanwise insertions and withdrawals.

Significant discomfort may be expected for a day or two. If, after 2 weeks, clinical examination still evokes pain, a second injection must be given. Two or three injections cure most patients.

Technique: deep friction to the quadriceps expansion

Infiltration of the quadriceps expansion seems to have little therapeutic effect. Deep transverse friction is therefore the only appropriate approach. Full and lasting relief is always attained, provided the massage technique is correct.

Once again, the patient sits with the knees outstretched and the quadriceps muscle fully relaxed. The therapist sits level with the patient's knees. With the thumb of one hand, the patella is pushed distally and over towards the affected side.



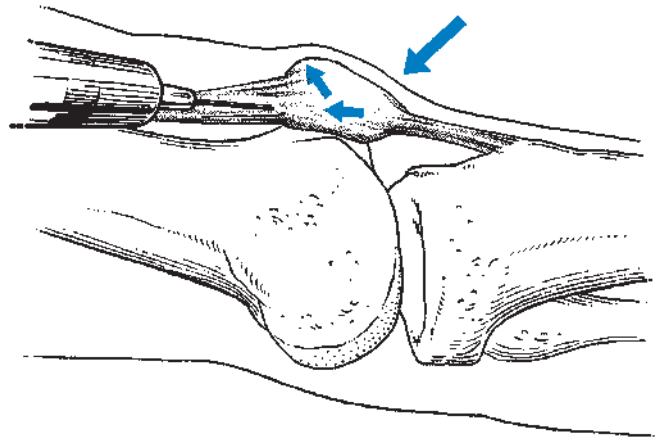


Fig 54.4 • Infiltration of the suprapatellar tendon. The patella is tilted (small arrows) by pressing on the inferior pole (large arrow).

This allows the edge of the patella to project outwards. The little finger of the other hand is extended as far as possible. The ring finger, reinforced by the middle finger, is placed under the patellar edge in such a way that it presses upwards, squeezing the tendinous fibres against the posterior aspect of the patella. Caution is observed so as to bring the hand into full supination, which means that the operating finger is horizontal, with pressure being applied against the back of the projecting edge of the patella. A vertical finger just presses against the femur and no friction on the tenoperiosteal junction results. Hand and forearm are held in line with the tibia, and friction is produced by a horizontal movement along the patellar edge (Fig. 54.5).

Twenty minutes' friction, three times a week for 2–3 weeks, nearly always results in cure.

Technique: deep friction to the infrapatellar tendon

Infrapatellar tendinitis is by far the most common tendinitis at the patella. The lesion lies over an area of 1–2 cm at the inferior pole of the patella. In order to make this area more accessible, pressure is exerted at the upper part of the patella, in the following manner. The physiotherapist stands level with the extended knee. One hand is placed just above the patella, so that the web of the thumb can exert downward pressure. This stabilizes the patella and tilts the apex upwards, so that the tender area can be sought with great accuracy. Friction is given with the middle finger of the other hand, reinforced by the index finger. The thumb is placed as far proximally as possible on the thigh. Pressure is exerted upwards, so that the lesion becomes squeezed between finger and bone (Fig. 54.6). Sometimes a large osteophyte can be palpated at the apex; if so, it is important to investigate to establish which side of the osteophyte the tenderness is located. Friction is given by alternate adduction and abduction movements at the shoulder, which draw the finger strongly along the inferior border of the patella.

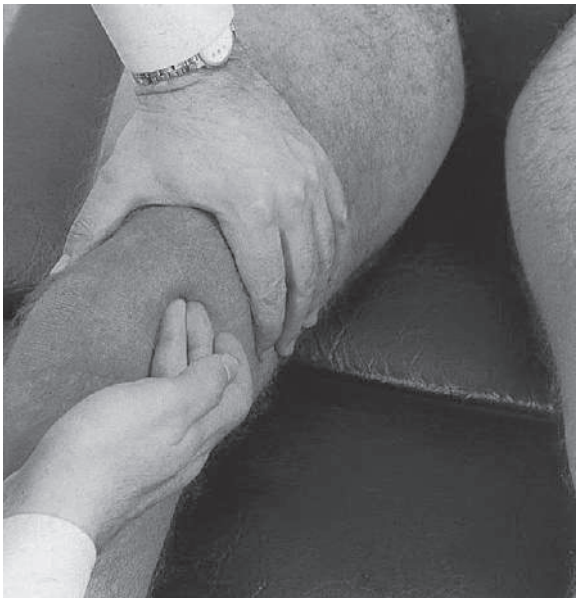
Twenty minutes of friction on alternate days, for 10–20 sessions, is sufficient for cure. If no improvement is seen after 10 sessions, the patient is referred for triamcinolone infiltration.

Technique: infiltration of the infrapatellar tendon

The patient sits supine with the quadriceps relaxed. The therapist presses with one hand on the superior border of the patella, in such a way that the inferior pole moves distally and upwards. This makes the infrapatellar tendon accessible to the palpating finger. The exact location of the lesion is now identified, in the way described earlier. The lower pole must be palpated very carefully, to determine the exact borders of the lesion. As already mentioned, caution should be observed in locating the lesion when an osteophyte is present.

Once the exact location is identified and the borders marked on the skin, a point is chosen 1.5–2 cm inferior to the midpoint of the affected area. A thin 3 cm needle is fitted with a syringe, containing 2 mL of triamcinolone. With the patella remaining tilted, the needle is inserted at the identified point and moved upwards, through the infrapatellar tendon, until it hits bone (Fig. 54.7). It is important to reach the posterior aspect of the patellar apex because most lesions lie deeply at the tenoperiosteal junction. The infiltration is now given using a series of small droplets along the inferior border. Considerable pressure has to be exerted on the syringe to push the drug in. Care is taken to inject only when the tip of the needle is felt to hit bone.

After the injection, the patient should take relative rest for about 10 days. Usually, considerable discomfort is experienced for 24–48 hours. The patient is seen again after 2 weeks. If



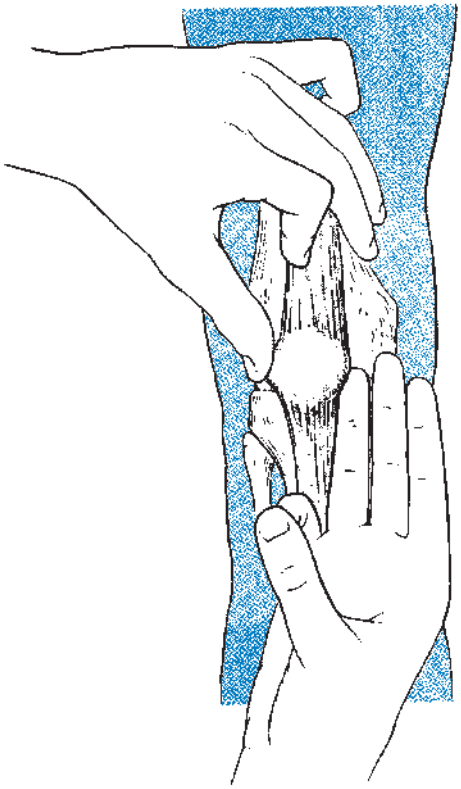
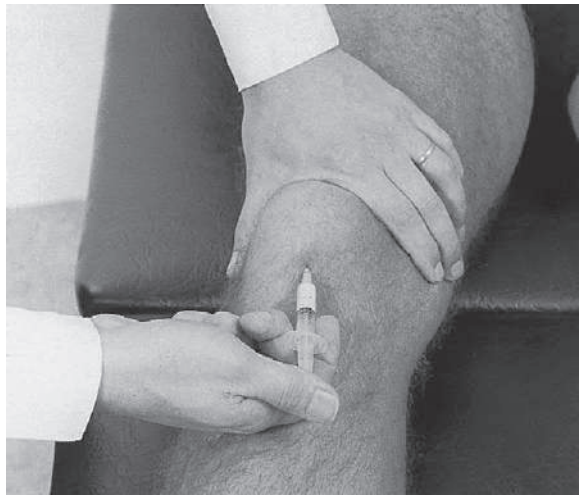


Fig 54.5 • Deep friction to the quadriceps expansion.

Fig 54.6 • Deep friction to the infrapatellar tendon.



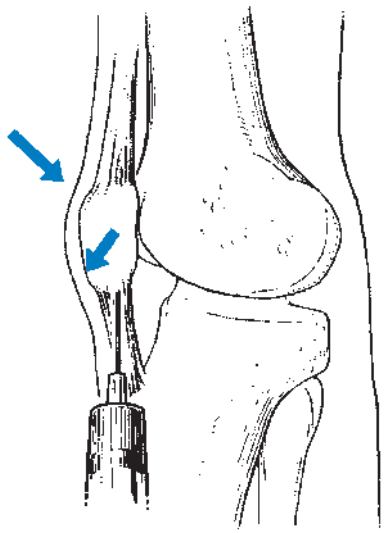


Fig 54.7 • Infiltration of the infrapatellar tendon. The patella is pressed distally, which moves the inferior pole upwards (arrows).

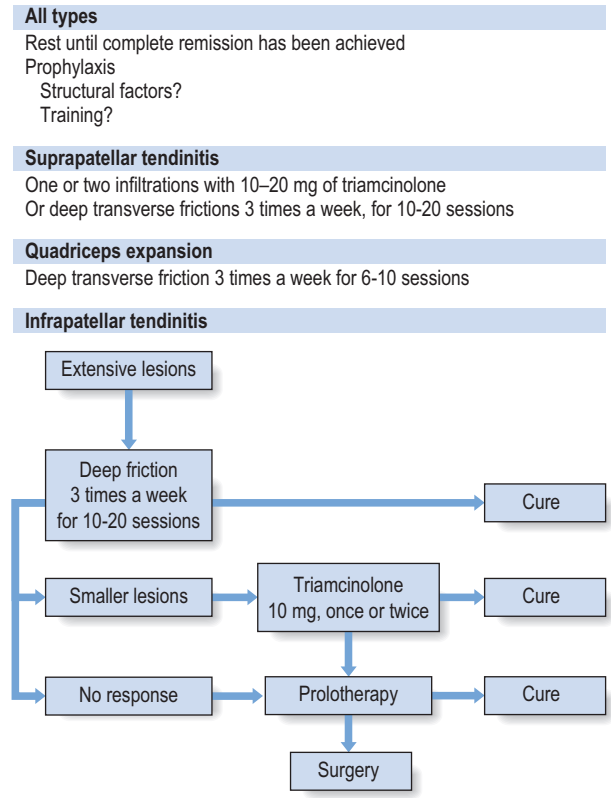


Fig 54.8 • Treatment of quadriceps tendinitis.

pain is still elicited by resisted extension, infiltration is repeated. Usually 1–3 injections are required to obtain total and lasting relief.

Return to sport must be gradual, and a training programme that does not impose too much strain on the quadriceps during the first few weeks is advised. An underlying biomechanical factor as the basis for a possible recurrence should be sought.[40,41,38]

Treatment of quadriceps tendinitis is summarized in Figure 54.8.

Rupture of the quadriceps tendon

Rupture of the suprapatellar quadriceps tendon is an uncommon injury. Most of the patients are elderly, and sometimes there is diagnostic confusion with other causes of inability to use the legs, such as a mild stroke.[41] The mechanism of injury usually involves forced hyperflexion or rapid extension from a weight-bearing position.[42] Pain, swelling, a palpable gap and inability to straighten the leg against resistance are the cardinal features. Treatment is operative repair.

Lesions of the infrapatellar tendon

Tendinitis of the infrapatellar tendon (ligamentum patellae) is unusual and occurs only in rheumatic disorders. Inflammation of the peritendineum, with pain, thickening and coarse crepitus, can be found. Treatment consists of triamcinolone infiltration along the surface of the tendon.

A patellar tendon rupture may be caused by acute or chronic excessive traction force.[43] The tendon is then pulled from the lower pole of the patella.[44] Degenerative rheumatic disease may also give rise to rupture of the infrapatellar tendon.[45,46]

Treatment is surgical.[47]

Lesions of the insertion at the tibial tuberosity

Tenoperiostitis

This exceptional lesion sometimes occurs in athletes. Resisted extension of the knee is painful at the insertion of the infrapatellar tendon. Palpation reveals the exact spot at the tibial tuberosity. Friction can be tried but is not always very helpful, and treatment mostly consists of one or two infiltrations with a small amount of triamcinolone.

Apophysitis

Osteochondrosis of the tibial tuberosity, or Osgood–Schlatter disease, occurs in boys between 10 and 15 years of age.[48]

The typical symptoms are local pain during sports or when walking upstairs. Local tenderness makes kneeling impossible.[49] Clinical examination reveals local ache during resisted extension of the knee. Palpation shows an enlarged, tender and warm tuberosity.

Radiography confirms the diagnosis and is important in differentiating the condition from more serious disorders such as osteomyelitis, arteriovenous malformations and osteosarcoma.[50]

In Osgood–Schlatter disease, nothing other than reduction of athletic activity should be advised. Spontaneous recovery takes place in 2 years, often with a permanently enlarged tuberosity (knee knob).

Patellar fracture

Most patellar fractures are stellate fractures, occurring after a fall or a direct blow (dashboard injuries). In the last decade, an increasing number of patellar fractures in association with total knee replacement have been described.[51,52]

Clinical examination shows warmth, a capsular pattern and a joint that contains blood. Resisted extension is markedly weak and painful. The combination of a gross articular pattern and marked quadriceps signs shows that the part of the quadriceps mechanism that overlies the knee joint must be at fault. Radiography confirms the diagnosis.

In young patients, an uncomplicated fracture heals quickly but early osteoarthrosis has to be expected.[53] When accurate reduction and stable fixation cannot be achieved, partial or total patellectomy must be considered and seems to produce satisfactory results.[54,55]

Transverse fractures are uncommon, except in an elderly person with an osteoporotic patella. After the trauma, the patient can walk with pain; because the capsule enclosing the patella is not ruptured, displacement is avoided and therefore the disability is minor.[56]

Patellofemoral disorders

Introduction

Patellofemoral pain is a common condition. About 25% of the population will, at some stage in their lives, suffer from patellofemoral symptoms.[57] There is no clear consensus in the literature concerning the terminology, aetiology and treatment for pain in the anterior part of the knee. The term 'anterior knee pain' is suggested to encompass all pain-related problems. By excluding anterior knee pain caused by intra-articular disorders, peripatellar tendinitis or bursitis, plica syndromes, Osgood–Schlatter disease and other rarely occurring conditions, it is suggested that those remaining patients with a clinical presentation of anterior knee pain could be diagnosed as having patellofemoral pain syndrome (PFPS).[58] The term chondromalacia patellae, although once used as an all-inclusive term for anterior knee pain, is now only accepted to describe pathological lesions of the patellar articular cartilage found at arthroscopy or arthrotomy. A diagnosis of 'chondromalacia patellae' for anterior knee pain is made far too often.[59,60] Stougard detected cartilaginous lesions at the posterior aspect of the patella in almost every knee joint at autopsy[61] and Paar and Riehl observed damage to the patellar cartilage in almost every operated knee.[62]

The cause of patellofemoral pain is unknown.[63] Currently, there are two theories to explain its origin: the mechanical and the neural theory.[64]

Mechanical theory

Most authors stress the importance of the congruency between the articular surfaces of the femur and the patella.[65–67] During the last decade, a great deal of emphasis was given to the idea of imbalance of the soft tissues surrounding the patella as the prime cause of patellar 'maltracking'. The soft tissue balance is particularly critical in the first 20° of knee flexion, where the position of the patella in relation to the femur is determined by the interaction of the medial and the lateral soft tissue structures; after this 20° of motion, the patella engages the femur and the bony architecture becomes more important for the position of the patella relative to the femur.[68] Any imbalance in the soft tissue structures in the first 20° of flexion will mean that the patella does not encounter the trochlea optimally, which may lead to undue stress on various tissues around the joint and thus to pain. The imbalance may be the consequence of multiple biomechanical defaults. Weakness of the vastus medialis obliquus is thought to be the single most important factor causing momentary patellar subluxation.[69] Also, the passive stabilizing structures are usually more extensive and stronger on the lateral than on the medial side. Active stabilization is provided by the quadriceps muscle – on the lateral side the vastus lateralis and on the medial side the vastus medialis. The fibres of the vastus medialis are divided functionally into two compartments: the vastus medialis longus and the vastus medialis obliquus (VMO). The latter does not play a role in active extension of the knee but keeps

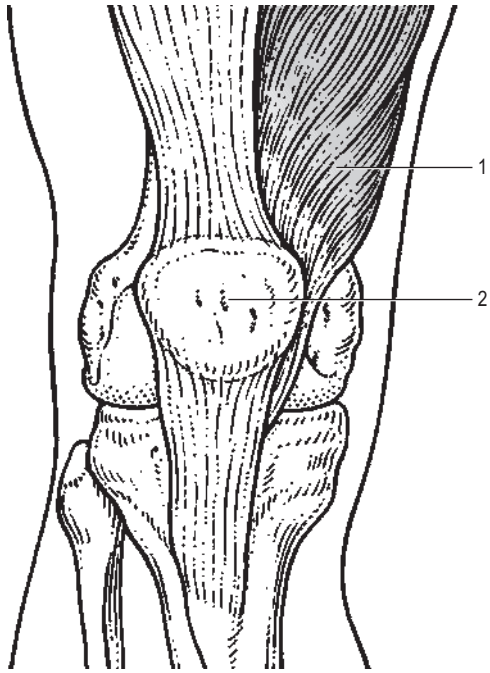


Fig 54.9 • Active medial stabilization is provided by the vastus medialis obliquus (VMO): 1, VMO; 2, patella.

the patella centred in the trochlea of the femur throughout knee extension.[70]

The VMO is the only active stabilizer of the patella (Fig. 54.9); therefore the timing and the amount of activity in the VMO is critical to patellofemoral function and it is hypothesized that small changes in its activity may have considerable effects on the patellar position.[71] Electromyography (EMG) studies have revealed that, in patients with patellofemoral pain, the VMO contracts at a significantly slower rate than the vastus lateralis, whereas in normal subjects the reflex response time of both muscles is equal.[72–74] A recent MRI study examined VMO size in patients with patellofemoral problems and found atrophy in all of them. Although it is not clear whether this atrophy is a result or a cause of PFPS, there is obviously a strong relationship between the two.[75]

Other biomechanical factors that may contribute to the development of patellofemoral pain are tightness in the lateral structures, increased Q angle (see p. 722) and faulty foot mechanisms.[76–78] In a retrospective study on 282 male and female students enrolled in physical education classes, Witvrouw *et al* found a significant correlation between four parameters and the development of patellofemoral pain: a shortened quadriceps muscle, an altered VMO muscle reflex response time, a decreased explosive strength and a hypermobile patella.[79]

Neural theory

The normal articular cartilage is aneural, so defects in the surface are not thought to produce pain. It has therefore been suggested that irritation of the subchondral bone produces the pain.[80] Schneider *et al* could reproduce the typical pain sensation by raising intrapatellar pressure in about 40% of patients suffering from persistent patellofemoral pain. This subgroup was treated with a new method of intraosseous drilling and decompression. Of the patients so treated, 90% experienced pain relief more than 3 years postoperatively, which may indicate osseous hypertension as a cause of the pain.[81]

Some authors have sought the origin of the pain in soft tissue structures around the knee. Nociceptive nerve fibres and neural markers (S-100 protein, neurofilament protein, substance P and neural growth factor) have been demonstrated in the medial and lateral retinacula and in the infrapatellar fat pad.[82,83]

From the existing studies it can be concluded that both theories are complementary rather than exclusive: the neural factor precipitates the symptoms in patients with certain biomechanical anomalies who subject their knee to overuse.

Clinical examination

It is important to stress the fact that most anterior knee pain is not caused by patellofemoral disorders but stems from infra-, supra- or peripatellar tendinitis, or a coronary ligament lesion. A basic functional examination should therefore always be performed first (see Box 54.1). However, if specific symptoms warrant, and the basic functional examination is totally negative, attention must be directed towards any peculiarities found during inspection of the patella and surrounding tissues.

Inspection on standing

Alignment of the lower extremity is evaluated first by observing the patient standing. Excessive varus or valgus of the extremities and internal femoral rotation are often associated with an extensor mechanism malalignment. Hyperextendable knees may also contribute to the development of anterior knee pain. McConnell points out that shock absorption throughout the lower leg then comes from internal rotation of the femur, rather than from knee flexion.[84]

Inspection on sitting

Some of the most important observations are made with the patient in the sitting position, with the legs bent to 90°. The position of the patella is assessed by viewing the knee from the lateral and the frontal aspects. In a lateral view (Fig. 54.10), the patella should be situated on the distal end of the femur, with the proximal patellar pole level with the anterior femur and the anterior surface of the patella in line with the tibial shaft. In 'patella alta', the upper border of the patella sits more on the anterior surface of the femur and 'faces towards the ceiling'. Viewed from anterior, a normally positioned patella faces straight ahead (Fig. 54.11); 'outfacing' and 'infacing' patellae are easily recognized in this position.

Attention is also directed to the alignment of the patella and the patellar tendon in relation to the tibial tuberosity and the tibial crest.

The bulk and the level of insertion of the VMO muscle is then assessed. The patient is asked to hold both knees actively at 45° of flexion. Normally, the VMO is seen as a substantial muscle, arising from the adductor tubercle and medial

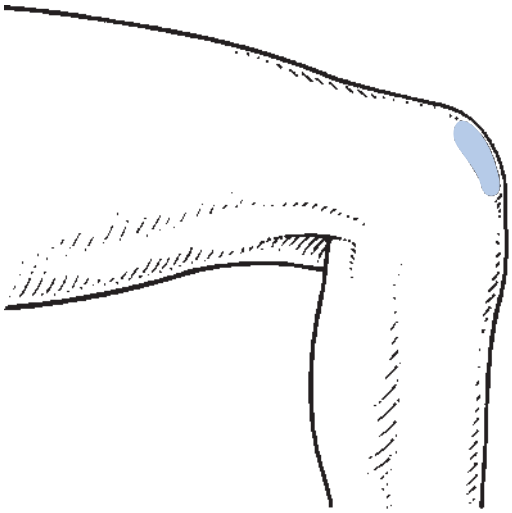


Fig 54.10 • In a lateral view the proximal pole of the patella should be level with the upper thigh.

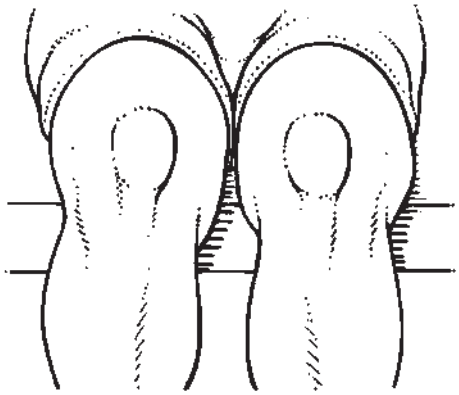


Fig 54.11 • Viewed from anterior, normally positioned patellae face straight ahead.

intermuscular septum and inserting into the upper third to half of the medial patellar border. In the obese knee, it is advisable to palpate along the inner border of the patella during active contraction in 45° of flexion.

Inspection in the supine position

The knee is in almost full extension (a small cushion is placed in the popliteal fossa) and the quadriceps is fully relaxed. The position of the patella in relation to the femur is observed. In the obese knee it may be necessary to palpate the patellar borders with one hand and the femoral condyles with the other. Four components affect the patella statically and dynamically: lateral glide, mediolateral tilt, anteroposterior tilt and rotation. An optimal patellar position is one where the patella is parallel to the femur in the frontal and the sagittal planes and midway between the two condyles of the femur when the knee is flexed to 20°.[84]

The glide component is determined by measuring the distance from the mid-pole of the patella to the medial and lateral femoral epicondyles. The patella should be precisely in the middle. The amount of mediolateral tilt is detected by

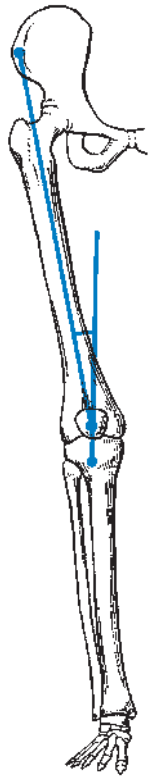


Fig 54.12 • The normal Q angle is about 10° in males and 15° in females.

comparing the height of the medial patellar border with that of the lateral patellar border. The two borders should be level compared to the horizontal.

The anteroposterior component evaluates the position of the inferior pole of the patella relative to the superior pole.

A rotation of the patella is determined by examining the position of the long axis of the patella, relative to the long axis of the femur.

Q angle

Next the Q angle is measured. This is the angle at the centre of the patella between two imaginary lines, from the anterior superior iliac spine and from the tibial tuberosity to the geometric centre of the patella. A Q angle of up to 10° is normal in the male and up to 15° in the female (Fig. 54.12).[85]

Passive movements of the patella

Patellar mobility is best assessed on a knee flexed to ±20° (the knee resting on a cushion or on the thigh of the examiner), with the quadriceps completely relaxed. The examiner's thumbs are placed on the medial border of the patella and the index fingers on the lateral border as attempts are first made to push the patella laterally and then medially. Quantitating the degree of mobility can be done by dividing the width of the patella into four longitudinal quadrants (Fig. 54.13). In normal subjects, the patella cannot be displaced more than three quadrants laterally and should allow a medial shift over at least one quadrant. When the patella is moved in a medial

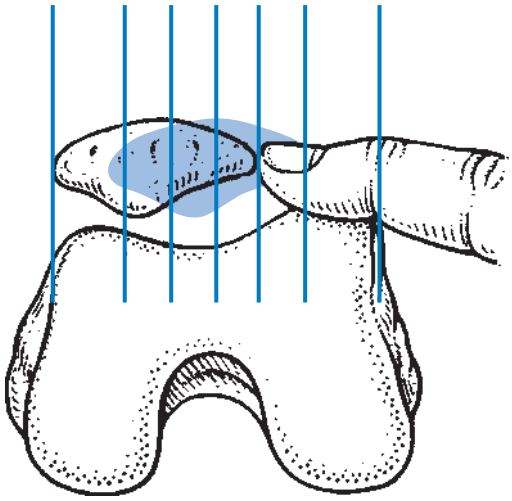


Fig 54.13 • Quantitating the degree of mobility can be done by dividing the width of the patella into four longitudinal quadrants.

direction, it should initially remain parallel to the femur and not tilt during the first quadrant of the movement.

During lateral movement of the patella, the examiner is looking not only for patellar mobility but also for any associated apprehension in the subject: as the patella tends to slip over the lateral condyle of the femur, the patient suddenly contracts the quadriceps muscle and draws the patella back. This sudden reflex would not be present if the patella was not apt to subluxate, and is typical of cases of subluxation.

Inspection during active movement

Close observation of the patella in relation to the femur is then continued during active contraction of the quadriceps. The patella should be pulled directly superiorly, or superiorly and slightly laterally. Excessive lateral displacement represents lateral overpull by the quadriceps and has been referred to as the 'lateral pull sign' (Fig. 54.14).[86]

Further observation is made during active flexion of the knee. The patella should remain centred on the trochlea. A patella which was sitting slightly lateral on the trochlea at full extension should move medially to be centred on the trochlea as soon as the knee flexes.

Clinical examination of the patellofemoral articulation is summarized in Box 54.1.

Clinical manifestations

From a clinical perspective, patellofemoral problems in the skeletally mature patient fall into three broad categories: patellofemoral instability (subluxation or dislocation), patellofemoral pain with malalignment but no episodes of instability, and patellofemoral pain without malalignment.[87]

Recurrent dislocation and subluxation of the patella

Dislocation mostly affects girls and begins at puberty. The history is that of a knee suddenly and painfully giving way. The youngster falls to the ground and feels something is out of place at the lateral aspect of the knee. When the leg is straightened, there is a loud click and extension becomes possible at once. For some days afterwards the knee swells and hurts, consequent upon traumatic arthritis. Sooner or later the incident recurs.

More often, there is a subluxation of the kneecap. The patient does not report the dramatic features described above but merely a 'catching' of the knee, followed by medial pain. The latter is caused by tearing of the medial capsular structures and should be differentiated from lesions of the medial meniscus.[88]

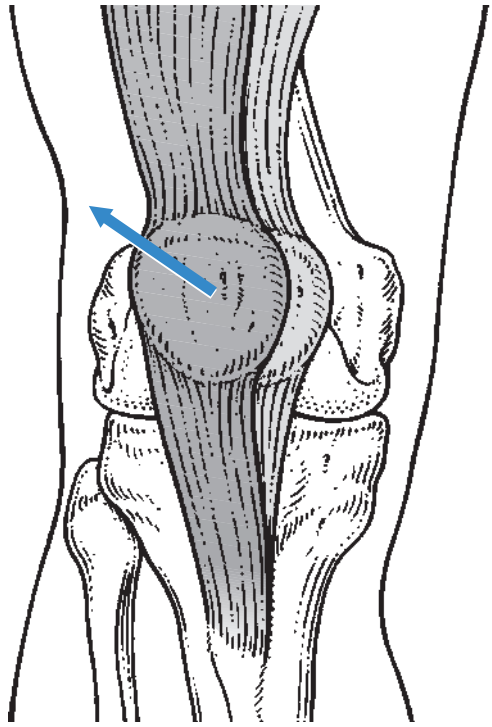


Fig 54.14 • Lateral pull sign: the patella moves laterally during contraction of the quadriceps.

Box 54.1

Clinical examination of the patellofemoral articulation

- Inspection standing
- Inspection sitting with the knees hanging over the edge of the couch
  - From anterior
  - From lateral
- Contraction of the quadriceps at 45° of flexion
- Inspection in the supine position
  - Position of the patella: four components
  - Q angle
- Passive movements of the patella
  - Mediolateral glide
  - Apprehension
  - Mediolateral tilt
- Active contraction of the quadriceps
- Active flexion of the knee

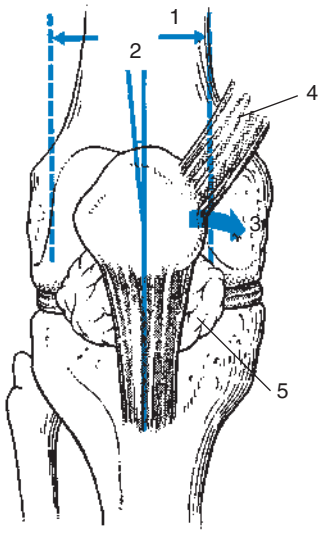


Fig 54.15 • Anatomical changes in recurrent subluxation of the patella: 1, increased patellar mobility; 2, decreased Q angle; 3, outfacing patella; 4, vastus medialis deficiency; 5, fat pad enlargement.

Clinical examination often reveals an enlarged fat pad and patella alta. There is deficiency or atrophy of the VMO. Some authors find a significantly decreased Q angle.[89] Lateral patellar mobility is increased (more than three-quarters of the width of the patella) and apprehension can often be provoked during glide mobilization of the patella (Fig. 54.15).

Treatment

In acute or recurrent dislocation, surgical intervention is advised but only after puberty, when the epiphysis has closed. Good subjective results are to be expected in about 80% of patients.[90]

In recurrent subluxation, the decision to operate should not be taken lightly because it has been demonstrated that long-term results continue to show an incidence of persistent instability and late patellofemoral arthrosis.[91–93] Arthroscopic release only gives a satisfactory result in 40% of cases.[94] Treatment of patellofemoral instability should therefore be conservative, surgical interventions being reserved for patients who do not respond adequately. Conservative treatment consists of intensive functional vastus medialis rehabilitation, together with the use of a brace (see treatment of PFPS, p. 1143).

Patellofemoral pain syndrome without subluxation

The source of pain in 'patellofemoral pain syndrome' cannot be adequately explained. The hypothesis is that patients with PFPS have lateral displacement of the patella within the femoral trochlea.[95] This lateral 'malalignment' may result from a number of factors, including malalignment of the lower extremity and/or the patella, tight lateral soft tissue structures and muscular imbalance of the lower extremity. All may lead to areas of increased stress on the patellofemoral joint and pain.[96]

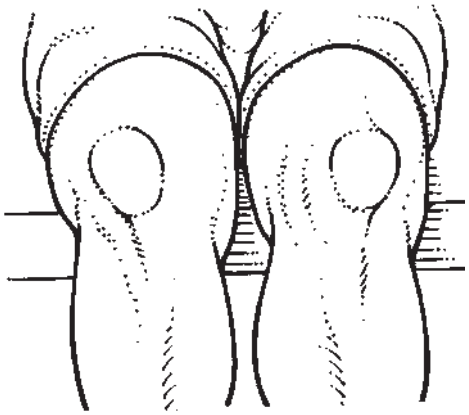


Fig 54.16 • Laterally tilted patellae.

Most patients with knee pain attributed to PFPS are young women. The common symptom is of a dull and vague aching behind the kneecap during and after physical activity, especially during body-weight loading of the lower extremities (walking up- or downstairs and squatting). The knee is usually also sore in a sitting position and a few patients have nocturnal pain too.[97]

Routine clinical examination reveals nothing. There is a full and painless range of movement, with normal end-feels. Resisted extension is strong and painless. Inspection may reveal some typical abnormalities. Excessive varus or valgus position of the lower extremities is often associated with a malalignment of the patella. The position of the patellae is first assessed in the sitting position. Laterally tilted patellae (Fig. 54.16) appear to be 'looking' up and over the examiner's shoulders towards the ceiling, the so-called 'grasshopper eyes' kneecap.

The VMO is then assessed by asking the patient to hold the knees actively at 45° of flexion. In patients with atrophy of the VMO muscle, there is a visible and palpable lack of muscular tissue at the upper medial border of the patella.

The Q angle is often increased in patients with PFPS. Another common finding is tightness of the inert lateral structures which make the patella tilt laterally: the medial border of the patella is higher than the lateral border, the undersurface of which is hard to palpate. Any attempt to push the patella medially results in more anterior tilting of the medial border. If the lateral structures are tighter more distally on the patella, it externally rotates, and if the tightness is greater in the superior pole, the patella will internally rotate during lateral pressure. Slight tightness of the lateral structures is revealed by a decreased medial glide: a medial displacement of only one quadrant indicates a tight lateral retinaculum. Again, the patella tilts laterally during the manœuvre.

Excessive lateral movement of the patella during active contraction of the quadriceps (the 'lateral pull sign') or during active extension of the knee in a sitting position indicates predominance of lateral forces. They are common findings in PFPS (Fig. 54.17).

The classic radiological examination techniques to visualize the patellofemoral articulation are the Merchant[98] and Laurin[99] views, whereby the joint surfaces are visualized with the knee flexed to 45° and 30° respectively. MRI has also been found to

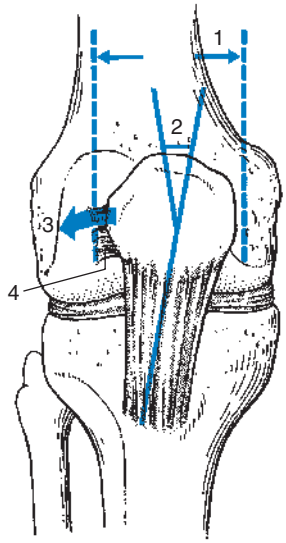


Fig 54.17 • Anatomical changes in PFPS: 1, decreased patellar mobility; 2, increased Q angle; 3, lateral tilted patella; 4, hypertrophied lateral bands.

be an accurate and reproducible method of measurement of the patellar tilt angle.[100,101]

Arthroscopy is of little value in the diagnosis of PFPS. The incidence of damaged cartilage in asymptomatic patients is so high that arthroscopy on its own cannot determine whether the symptoms should be attributed to chondromalacia. Hertel detected lesions of the articular surface of the patella in about 50% of routine arthroscopies.[102] Paar and Schneider found clear damage to the cartilage in 67% of the joints in which they undertook arthroscopies.[103] These two series establish that arthroscopic lesions occur far more frequently than do clinical manifestations, and that overestimation of disease is a hazard if diagnosis is based totally on the result of arthroscopy.

Treatment

In a number of patients, the condition seems to be self-limiting, as demonstrated by Sandow and Goodfellow, who reported spontaneous cure in more than 20% of anterior knee syndromes. Of 54 girls suffering from PFPS, only 9 were permanently unable to participate in sports after a follow-up period of 4 years.[104]

Many different treatment protocols for PFPS have been described in the literature. It is clear that, if abnormal tracking of the patella is believed to be the reason for the pain, the initial treatment must be to try to restore normal alignment. An optimal patellar position is achieved by stretching the tight lateral structures and by changing the activation pattern of the VMO.[68]

Stretching is performed passively by the therapist. However, the most effective stretch to the shortened retinacular tissue is obtained by sustained low load, using tape, which facilitates a permanent elongation of the tissue.[105–107]

Specific training of the vastus medialis includes timing and intensity of the VMO relative to the vastus lateralis (VL). Ideally, the VMO should come in slightly earlier than the VL.[73] This is mainly achieved during weight-bearing activities. It has been proposed that training causes changes within the nervous system that allow an individual to coordinate the activities of muscle groups better.[108] Biofeedback machines are extremely useful to facilitate this process.[109] Adding some hip adduction in training will also cause an increase in VMO activity relative to VL.[110]

If symptoms persist despite a conservative treatment programme, surgery may be indicated. An arthroscopic approach, with shaving of the affected area[111] and/or lateral release of the lateral retinaculum, is recommended.[112,113] Before deciding to operate, however, it is important to remember that the long-term results are not as good as might be hoped.[114,115]

Patellofemoral arthrosis

This can occur after a flake fracture of the articular surface of the patella.[116] It is not clear whether chondromalacia patellae precedes osteoarthritic changes because the anatomical location of the two conditions differs. Chondromalacia is most often found near the median ridge, whereas osteoarthrosis first appears on the very medial side of the patella.[13] Erosion of the cartilage at the medial edge may start before the age of 20 and has been shown to be present in three-quarters of individuals by the age of 30.

As in other localizations of osteoarthrosis, radiographic signs do not always equate with symptoms. Sometimes the patient is aware only of grating or cracking when walking upstairs. Alternatively, the symptoms may be very similar to those in chondromalacia: anterior pain when walking upstairs, squatting or sitting with bent knees.[117] Palpation during squatting reveals pain, together with 'crackling' and 'popping' sensations.

Treatment depends on the severity of the symptoms. Intra-articular injections with hyaluronan has proven to be an effective treatment modality for patellofemoral arthrosis, with beneficial effects on pain, function and patient global assessment.[118,119] In intractable cases, patellofemoral arthroplasty can be advised.[120]

Strained iliotibial band

The iliotibial band, a thick extension of the fascia lata, inserts at the tibial tubercle of Gerdy. The band lies anterior to the flexion–extension axis of the knee in extension and posterior in flexion. The tensor fasciae latae, acting through the band, is thus a weak external rotator and extensor of the knee. Iliotibial band friction syndrome is an overuse injury usually seen in military personnel, cyclists and long-distance runners.[121] With an incidence of between 1.6 and 12%, it is the most common running injury of the lateral knee in long-distance runners.[122,123]

The presumed pathogenesis is that during activities involving repetitive knee flexion (such as running), the iliotibial band repetitively shifts forwards and backwards over the lateral femoral epicondyle, causing friction and thus inflammation of the iliotibial band (Fig. 54.18). Friction thus occurs at, or slightly below, 30° of flexion.[124] This can produce either irritation and subsequent inflammatory reactions within the iliotibial tract itself, or formation of a painful bursa between the band and the epicondyle.[125,126]

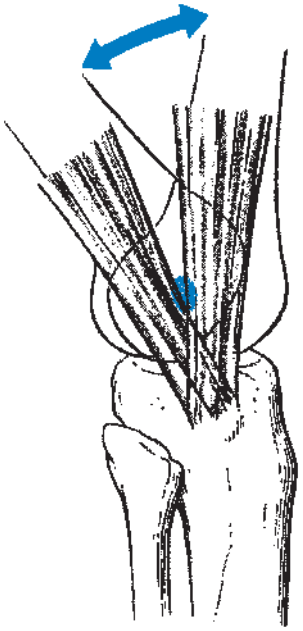


Fig 54.18 • A strained iliotibial band results from the band rubbing on the lateral epicondyle during repeated flexion–extension movements.

While running, athletes notice increasing pain at the outer side of the knee, which forces them to stop. The pain is more intense when the foot comes in contact with the ground when running downhill or during deceleration.[127] In severe instances, the pain may be so sharp that walking with a normal gait becomes impossible.

Examination reveals no abnormality of the joint but some discomfort is experienced when extension and lateral rotation are carried out against resistance. Sometimes a painful arc is found when the band slips over the lateral epicondyle at 30° of flexion. When the routine examination is negative, a simple confirmatory test may be done. In 90° of flexion, pressure is applied at the iliotibial band over the lateral epicondyle; as the knee is gradually extended, the epicondylar area becomes painful at about 30° of flexion.[128] MRI may be useful in confirming the diagnosis.[129–131]

Treatment

Preventive measures include avoidance of excessive distance and hill running and a reduction in length of stride.

In most cases, 2–3 infiltrations with triamcinolone, deeply between band and epicondyle, are successful.[132]

Partial resection is advised in persistent lesions.

Flexor mechanism

When resisted flexion of the knee causes pain in the posterior aspect of the thigh, the fault is in the hamstrings. Resisted internal and external rotation then differentiates between lesions of the medial hamstrings and of the biceps femoris. Should resisted flexion hurt at the knee, other possibilities arise. These are a strained popliteus muscle, tendinitis at either origin of the gastrocnemius, a sprained posterior cruciate ligament or a lesion of the upper tibiofibular joint.

Hamstring strains

The hamstrings have a double function. They not only extend the thigh and flex the knee (concentric function), but also antagonize the quadriceps during knee extension in running (eccentric function). It is probable that a sudden change from stabilizing activity to an active function is the main reason for a hamstring strain.[133–135]

Lesions of the hamstrings are frequently seen in athletes, and rugby, American football and soccer players.[136–138] Classically, a strain occurs as a sprinter leaves the starting blocks or during a slide in soccer. The athlete feels a snap, immediately followed by pain and functional loss. In lesions of the muscle belly, straight leg raising is limited by pain because of muscle spasm. Prone-lying resisted knee flexion is painful. Resisted external or internal rotation can then distinguish between a biceps lesion or a lesion in the medial hamstrings. The short head of the biceps seems to be the most frequently strained.[139,140] Sometimes there is a haematoma and the tender area is swollen.[141]

Treatment

An elastic wrap and an ice bag should be applied immediately after the incident.

Minor lesions require the same treatment as other muscular lesions. As soon as the patient is seen, whether this is on the first day or after a week, the lesion is infiltrated with 50 mL of procaine 0.5%. The patient lies prone, with the hip extended and the knee well flexed and supported by a pillow, so that the muscle belly is completely relaxed. The tender area is now identified, which may not be easy because the lesion may be deeply situated, especially when the short head of the biceps is affected.

From the next day on, daily deep transverse friction is given, together with isometric contractions, until the lesion is completely cured.

Technique: deep friction to the hamstrings

The technique is very similar to the one used in quadriceps lesions but here the patient lies prone, with the hip extended and the knee passively flexed to 90°. This position produces complete relaxation of the muscle belly. The therapist sits or stands level with the patient's limb. The fingers of one hand, perhaps reinforced by the other hand, are placed deep to the affected part of the belly. The thumbs are positioned laterally on the femur and used as a fulcrum. An extension movement at the wrists moves the flexed fingers upwards and pulls on the fibres, which are felt to slip under the fingers (Fig. 54.19). The effect of deep friction can be increased somewhat by a rotational movement around the thumbs at the end of this movement. As this treatment is extremely tiring, three periods of 5 minutes' friction over a total of 20 minutes is as much as is practicable.

After friction, electric or active isometric contractions should be given for about 10 minutes. The purpose is to expand

the muscle transversely as much as possible, without strain on the healing fibres. This prevents intramuscular adhesions, often responsible for recurrent or chronic strains of the hamstrings.[142] The possibility of recurrence is considerable if the patient goes back to athletics too soon.[143] Therefore treatment should continue with massage and electric contractions for a week after clinical cure is established. Return to sport should be progressive, and special care should be taken to 'warm up' adequately.

Prophylaxis should also include progressive strengthening exercises and stretching before and after activity.[144]

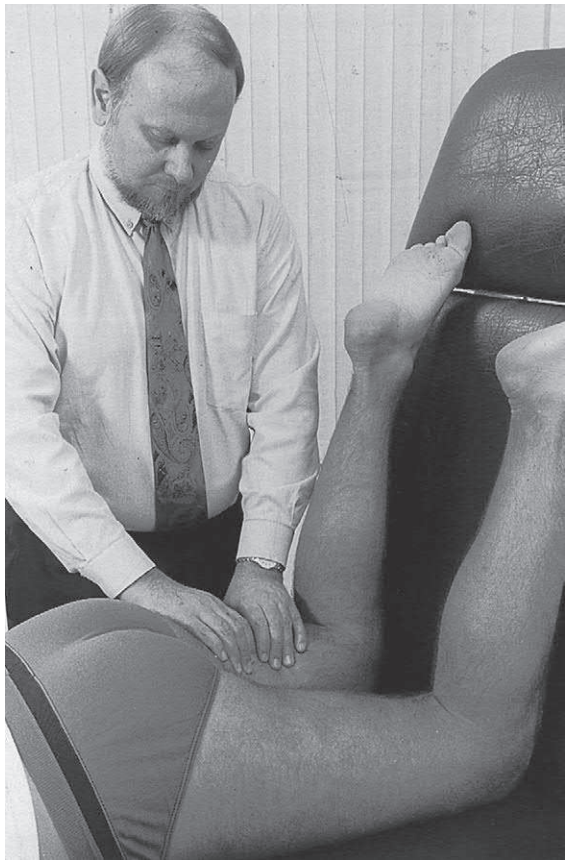


Fig 54.19 • Deep friction to the hamstrings.

Biceps tendinitis

This usually occurs as an overuse lesion in athletes. Pain develops at the outer side of the knee. Functional examination is entirely negative, except that resisted flexion hurts at the outer side. Resisted external rotation is also painful. Tenderness is found at the biceps tendon, usually at the tenoperiosteal insertion at the head of the fibula.[145,146]

Treatment consists of deep transverse friction. Alternatively, an infiltration with 20 mg of triamcinolone can be used but this is only effective if the lesion occurs at the insertion.

Technique: friction to the biceps tendon

The patient lies prone with the knee fully outstretched. The physiotherapist stands at the opposite side, level with the knee. The head of the fibula is easily identified. The index finger is placed on the tendon at the level of the lesion. Counterpressure is exerted with the thumb placed at the medial side of the knee (Fig. 54.20). Massage is now given using small, alternating extension and flexion movements at the wrist, while the thumb is kept still.

Twenty minutes, three times a week for 3–6 weeks, may be required.

Lesions of the upper tibiofibular joint

Dislocations or fractures may occur after violent twists or direct blows, as when a rider's knee is caught between a tree and the horse, for example.[147] It is clear that the examiner must be especially alert for these injuries because they can lead to peroneal nerve palsy.

Fig 54.20 • Massage of the biceps tendon.

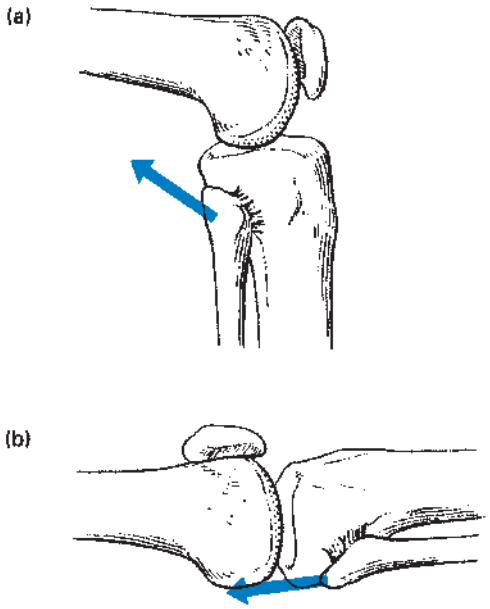


Fig 54.21 • Resisted flexion at a right angle (a) and in full extension (b) differentiates between a lesion of the upper tibiofibular joint and the biceps tendon.

A sprain of the upper tibiofibular ligament can develop after trauma to the ankle joint or as the result of repetitive strains.[148]

The findings on clinical examination are the same as in biceps tendinitis. Because contraction of the biceps pulls the fibula backwards on the tibia, resisted flexion and resisted external rotation hurt at the outer side of the knee (Fig. 54.21a). However, when the biceps tendon is examined for tenderness, none is found. Resisted flexion in almost full extension is also painless, because the fibular head is now pulling upwards instead of backwards and stress is no longer imposed on the tibiofibular ligaments (Fig. 54.21b). Also, passive backwards movement of the fibular head elicits pain and the anterior tibiofibular ligament is tender to the touch.

Treatment is infiltration of the tender structure. One or two infiltrations with 20 mg of triamcinolone solution usually obtain permanent cure.

Lesions of the pes anserinus

These lesions are rare but are brought to mind if resisted flexion and internal rotation hurt at the inner side of the knee. Palpation usually reveals a tender spot at the insertion of the semimembranosus. Deep transverse friction clears the symptoms.

It is important to remember that part of the semimembranosus inserts through the posterior oblique ligament at the posterior border of the medial meniscus. In lesions of the posterior horn, pain on resisted flexion can therefore complicate the clinical picture.

Strained popliteus muscle

Should resisted flexion and internal rotation hurt at the lateral or posterolateral aspect of the knee, the popliteus tendon or muscle is suspected (Fig. 54.22).

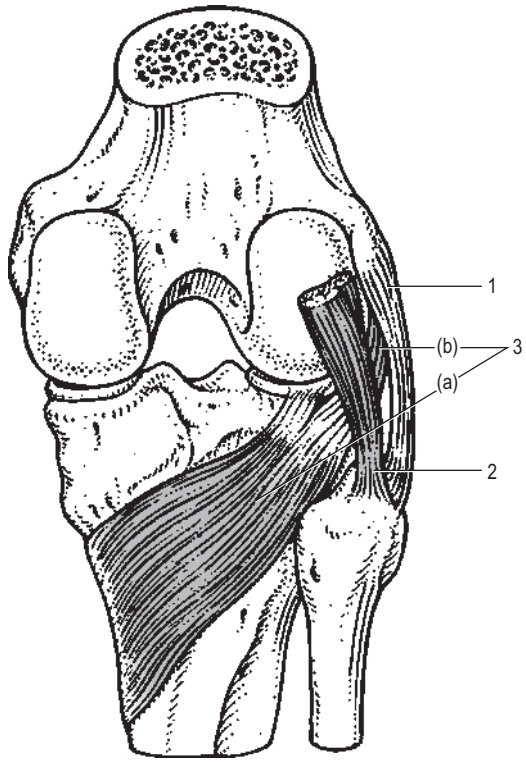


Fig 54.22 • Anatomy of the lateral aspect of the knee: 1, lateral collateral ligament; 2, biceps tendon; 3a, popliteus muscle; 3b, popliteus tendon.

The popliteus tendon originates from the lateral femoral condyle. It passes deeply to the lateral collateral ligament, which it separates from the lateral meniscus.[149] The tendon also attaches partly to the lateral meniscus.[150,151] The muscle belly inserts with a broad and short tendon at the posterior aspect of the tibia, in the popliteal fossa. Although the muscle is a flexor and internal rotator of the leg, its main function is to prevent a sliding forwards of the lateral femoral condyle on the fixed tibia during the stance phase in walking or running, especially in running downhill or descending stairs.[152–154] This makes the popliteus an important active stabilizer of the lateral and posterolateral aspect of the knee joint and explains the frequency of popliteal lesions in long-distance runners and downhill skiers. The prevention of forward sliding is also a function of the posterior cruciate ligament (passive stabilizer), and a strained popliteus muscle can closely simulate a ligamentous strain of the posterior cruciate. Only a careful functional examination differentiates the two lesions.[155]

Localization of the pain and palpation for tenderness further reveal that the lesion lies at the tendon or in the muscle belly.[156,157]

Treatment

Tendinous lesions respond very well to one or two infiltrations with 10 mg of triamcinolone.

Alternatively, deep friction can be tried, to both the origin and the body of the tendon, although the position of the affected structure makes access for the palpating finger very

Fig 54.24 • Deep friction to the popliteus muscle.

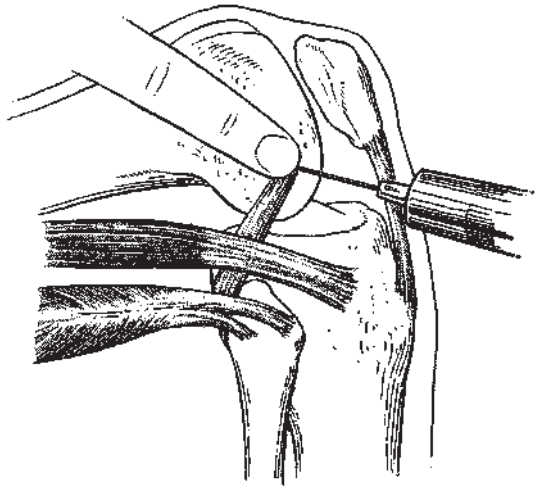


Fig 54.23 • Infiltration of the popliteus tendon.

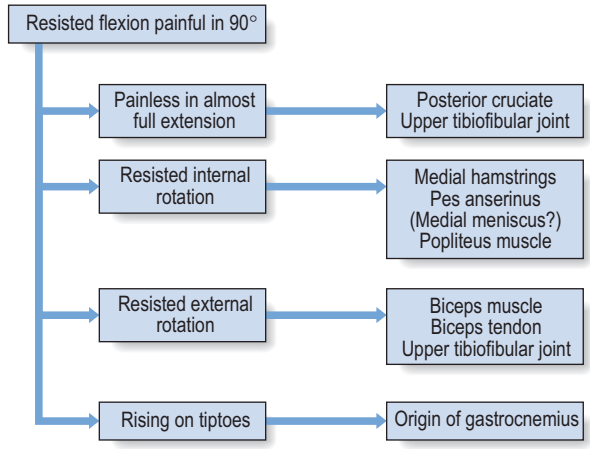


Fig 54.25 • Differential diagnosis of resisted knee flexion.

difficult. Lesions of the muscle belly respond only to deep friction.

Technique: infiltration of the popliteus tendon

The patient lies supine, with the knee flexed to 90°. The tendon is identified by careful palpation of the lateral condyle. At the most prominent aspect, two small bony projections can be palpated, with a groove running vertically downwards between them. In this groove, the origin of the popliteus tendon can easily be palpated and followed until it disappears under the lateral collateral ligament. If the origin is tender, 1 mL of triamcinolone should be infiltrated with the usual technique: the needle is brought in through capsule and ligament until it reaches bone (Fig. 54.23). A series of droplets is then injected at different areas, but only when the tip of the needle is felt to touch bone.

If the body of the tendon is tender, the injection should be made around the tendon and not in the tendon itself.

Technique: deep friction to the popliteus muscle

The patient adopts a prone position, with the knee slightly bent. The physiotherapist stands lateral to the patient's leg. One thumb, reinforced by the other, is placed on the affected spot, parallel to the muscular fibres (Fig. 54.24). Deep transverse friction is now given, by a supination movement of both arms, which makes the thumbs roll transversely over the

muscle fibres. The direction of the movement is cranial and medial.

Lesions of the gastrocnemius

Tendinous lesions at the origin of the gastrocnemii are very uncommon. We have only seen this lesion in cyclists. Diagnosis becomes obvious when not only resisted flexion but also rising on tiptoes hurts at the back of the knee. After careful palpation, the tender area is infiltrated with triamcinolone; this usually gives a good result.

The differential diagnosis of resisted knee flexion is summarized in Figure 54.25.

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55

Clinical examination of the lower leg, ankle and foot

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Referred pain

Pain referred to the foot

Pain referred from other structures (hip, sacroiliac joint, lumbar spine) is hardly ever experienced in the foot *alone*. Most commonly, it involves the foot and the ankle, with or without other parts of the lower limb. The question of referred pain only arises when the clinical examination of the foot is completely negative and the patient states that the foot pain is unaltered by walking.

Pain referred from the foot

Because the foot is a distally located part of the body, there is very little referred pain from lesions of the foot (following the principles of referred pain). Therefore pain in foot or ankle usually points fairly accurately to the site of the lesion.

History

Although the main question is the current problem – where the pain is now – it is best to try to obtain a chronological account, as summarized in Box 55.1. In order to work out the problem systematically, the questions asked should follow the order given below.

- *When did the pain start?* Is the problem acute, subacute or chronic?
- *How did it start?* Was there an injury or not?

If there was an injury:

- What was the mechanism?
- What were the symptoms at that time? Pain? Swelling? Disability?
- What was their evolution over the following days?

If there was no injury:

- Did the pain come on suddenly or gradually?
- What brought the pain on? Nothing special? Overuse?

Sometimes there will be some evolution in degree and localization of pain, swelling and functional disability during the weeks or months after the onset.

The examiner should also find out what sort of treatment the patient has already had, and what the results were.

- *What has been the evolution since the onset?* No change? Gradually worse or gradually better? Ups and downs, with complete or no recovery between attacks? Do changes depend on exertion?
- *What sort of treatments did you have?*
- *What were the results?*
- *What are the problems now?*

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Box 55.1

Summary of history taking

What is the present complaint?

- Onset
- Trauma
- No trauma

Evolution

- Better
- Worse
- Ups and downs

Present complaints

- Site of pain
- Influence of movements
- Nocturnal pain
- Twinges
- Instability
- Swelling

The present complaints should be investigated further:

- *Where do you feel the pain now?* Posterior, middle or anterior segment? Medial, lateral or all over the joint(s)?
- *Is there pain at rest or during the night?* Pain at night indicates a high degree of inflammation.
- *Is there long-standing morning stiffness?* This also suggests a serious inflammatory disorder.
- *What brings the pain on?* Is there any pain during normal walking or normal running? Can you participate in your usual sports?

Because foot disorders cause pain while the foot is performing its function, walking or running will always be the provoking activities:

- *Is there equal pain on walking and standing?*
- *Can you walk on uneven surfaces?* Because walking upstairs and downstairs demands normal mobility of the ankle joint, it is worth asking if the patient can do so without problems.
- *Is the pain provoked by particular movements?*
- *Is the pain felt at the beginning of, during or after exertion?*
- *Does the pain depend on the sort of shoes you wear?* Sometimes an in-built wedge relieves or aggravates the pain. In Achilles tendinitis or plantar fasciitis, a raised heel with a horizontal surface relieves the stress on these affected tissues and therefore relieves the pain.

Further questioning should establish whether there are 'twinges' or instability:

- *Do you have twinges, and when?* A twinge in the foot is a very important symptom. It is a sudden, sharp pain, mostly occurring during walking. It should always be differentiated from 'giving way'. In a twinge, there is only momentary pain and not a feeling of instability. By the time the patient realizes it, the pain has disappeared. A twinge is very often an indication of a momentary impaction of a loose body in the ankle or subtalar joint. If localized in the forefoot, it can be a symptom of Morton's metatarsalgia or sesamoiditis.
- *Do you have a feeling of instability? If so, describe it.* Real instability of the ankle or foot is only important in sports. Normal walking or even running on a flat surface hardly ever causes a feeling of giving way. In the occasional case where this does happen, it is the result of neurological weakness of the peronei muscles, rather than of a ligamentous lesion.

Inspection

Inspection is first made in a standing position. The shape of the legs is evaluated: valgus or varus deformity is checked. The normal intermalleolar distance should not exceed 5 cm. A slight outward rotation of the tibiae is normal, with an outward pointing of the toes of about 15°. Exaggeration of this outward rotation can be caused by short calf muscles and results in a restricted step during walking and running.

The shape of the feet in a standing position is studied next. At the calcaneus there can be a valgus or varus deformity.

The longitudinal arch of the foot is then estimated: a cavus deformity or a flat foot may be identified. At the mid-tarsal region the shape and regularity of the bones is inspected and, at the forefoot, special attention is paid to the existence of an insufficient anterior arch, hallux valgus, claw toes, hammer toes or metatarsus inversus.

After inspection in the standing position, it is sometimes advisable to check the patient's gait and how the shoes have worn down.

Inspection in the supine-lying position gives information about contours, shape, atrophy, colour of the skin, swelling, oedema, haematoma, the condition of the skin and nails, and the existence of callosities.

Sometimes a second inspection in a standing position may follow the routine functional examination, when special attention should be paid to:

- Change of the shape of the foot during weight bearing, in relation to the shape when lying down
- Which part of the foot comes under abnormal strain
- Changes in the colour of the skin (redness on suspension and pallor on elevation point to arterial insufficiency).

Functional examination

The foot is the most difficult moving part of the body to examine because a great number of strong structures, with little individual mobility, are condensed into a small volume. Testing each structure in turn without the help of a lever is a very difficult task and demands great technical and manual ability.

In the foot, the clinical tests consist of 18 movements (Box 55.2). Other than rising on tiptoe, the clinical examination is

Box 55.2

Summary of movements of the foot

- Rising on tiptoe
- Two tests for the ankle joint
  - Plantiflexion
  - Dorsiflexion
- Three ligamentous tests
  - Mortice test
  - Inversion
  - Eversion
- Two tests for the subtalar joint
  - Varus
  - Valgus
- Six tests for the midtarsal joints
  - Plantiflexion
  - Dorsiflexion
  - Adduction
  - Abduction
  - Pronation
  - Supination
- Four resisted movements
  - Plantiflexion
  - Dorsiflexion
  - Inversion
  - Eversion

performed in the supine-lying position. Consecutively, the ankle joint, the subtalar joint, the midtarsal joints, the ligaments and the muscles are tested.

Rising on tiptoe

This movement (Fig. 55.1) is used to test the plantiflexor mechanism of the foot. If rising on tiptoe hurts and resisted eversion and inversion remain painless, the triceps surae muscles must be at fault. Because plantiflexion is almost entirely performed by the contraction of the triceps, rising on tiptoe will, in particular, test the integrity of triceps, the Achilles tendon and its insertion on the calcaneus. Note is taken of whether the movement is strong or weak, painless or painful.

If rising on tiptoe is painful, the following test distinguishes between the soleus and the gastrocnemius muscles. The patient lies prone and plantiflexion of the foot is resisted, first with the knee fully extended, then with the knee bent at a right angle. Flexing the knee relaxes both the gastrocnemii muscles but does not alter the pull at the soleus muscle. Hence abolition of pain when the muscle is tested during knee flexion incriminates the gastrocnemius muscle.

If rising on tiptoe is weak, a neurological lesion can be the cause. Apart from upper motor neurone lesions, peroneal atrophy and direct injury to the sciatic nerve, the common cause of painless weakness is a first and second sacral root palsy.

Rising on tiptoe is also very important in the diagnosis of Achilles tendon rupture. If the plantiflexor muscles are examined in the supine-lying position only, active plantiflexion is not found to be lost because the flexor hallucis longus, the flexor digitorum longus, the tibialis posterior and the peronei remain intact.

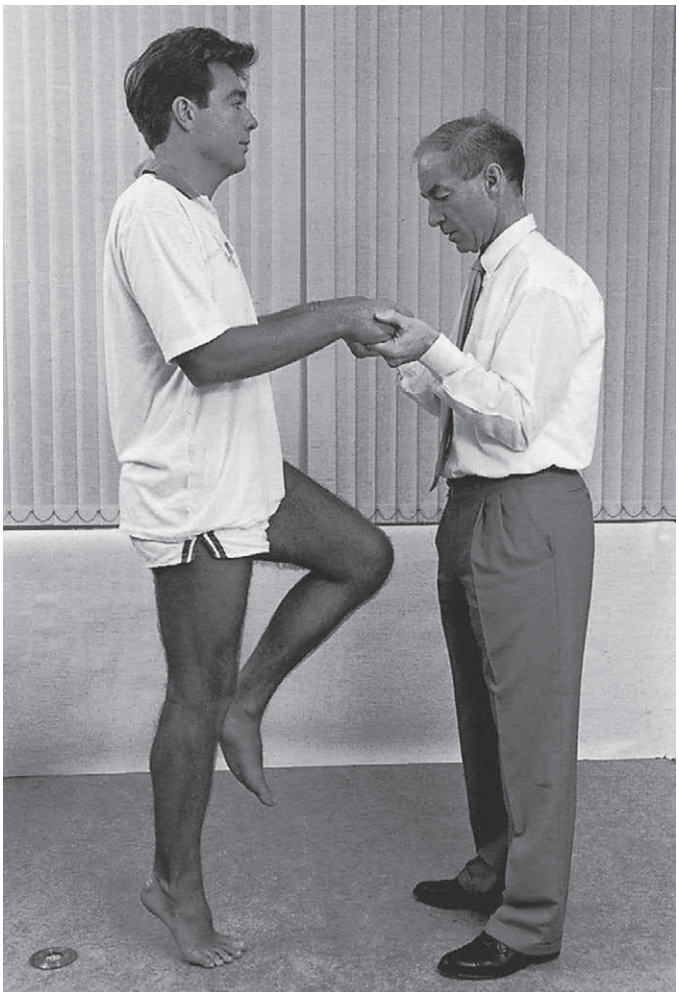


Fig 55.1 • Rising on tiptoe.

Tests for the ankle joint

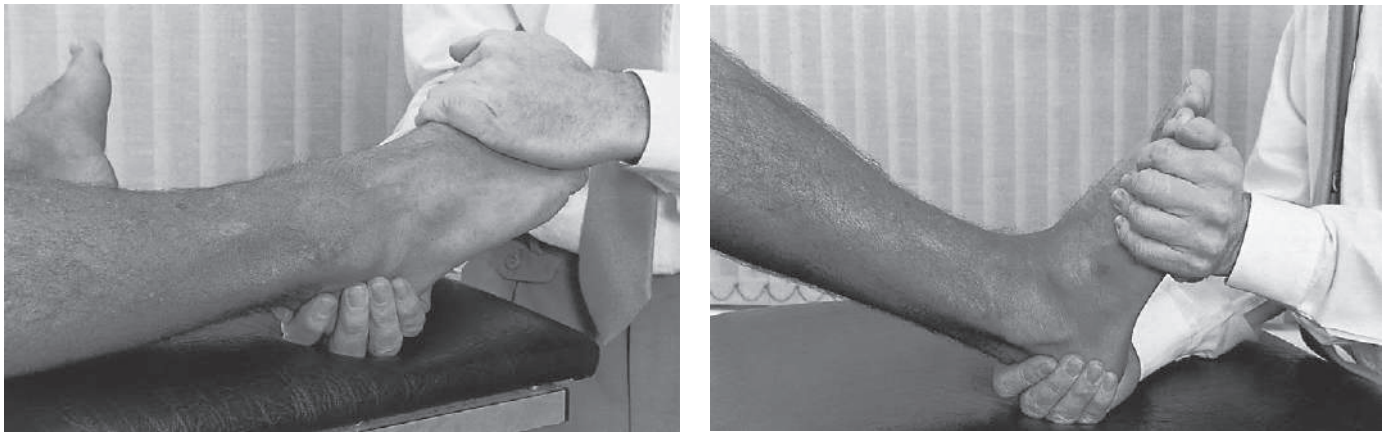
The ankle joint is a simple joint, allowing, in normal circumstances, plantiflexion and dorsiflexion only. The range of movement is measured by estimating the angle joined by the longitudinal axis of the tibia and the dorsum of the foot.

Plantiflexion

Normally, an ankle can be plantiflexed until the dorsal aspect of the foot falls into line with the leg (Fig. 55.2). Plantiflexion is limited by the engagement of the heel via the Achilles tendon against the back of the tibia. Therefore the normal end-feel is soft.

Dorsiflexion

Normally, the ankle can be moved to reduce the angle between the dorsum of the foot and the tibia to less than 90°.



(a) (b)

Fig 55.2 • Passive plantiflexion (a) and dorsiflexion (b).

Occasionally, the range of dorsiflexion is limited by the length of the calf muscles. Therefore dorsiflexion is performed with the knee slightly bent so as to relax the gastrocnemii. In this position, the movement is brought to a stop by stretching of the posterior capsule of the joint and by bony engagement between the neck of the talus and the anterior margin of the tibial surface. The end-feel of normal dorsiflexion at the ankle is soft.

Ligamentous tests at ankle and foot

Lateral ligaments

To test the outer structures of the ankle and foot, the examiner performs a strong inversion movement during full plantiflexion. Inversion produces a combination of varus at the subtalar joint and adduction/supination at the forefoot. This test stretches all the structures at the outer and anterior side: the lateral ligaments of the ankle, the subtalar joint and the midtarsal joints, together with the tendons of the peroneal and the extensor digitorum longus muscles.

Technique

The ipsilateral hand of the examiner fixes the leg at the distal and medial side (i.e. the left hand of the examiner on the right leg of the patient). The contralateral hand is placed on the midfoot, so that the heel of the hand rests at the fifth metacarpal bone and the fingers encircle the medial border. The heel of the hand now presses the foot downwards and inwards. Meanwhile, supination is performed by an upward pulling of the fingers at the medial border (Fig. 55.3).

As there are considerable differences within individuals in the range of this movement, both sides should be compared. A note is made of the range of movement, the pain and the end-feel. The normal end-feel is soft.

Medial ligaments

A combined test is used: full plantiflexion at the ankle joint, together with valgus at the subtalar joint and abduction–pronation at the midtarsal joint. These movements stretch the anterior and middle portion of the deltoid ligament, the anterior tibiotalar ligament and the calcaneonavicular ligament. At the outer side, however, the posterior talofibular ligament can become painfully squeezed.

Fig 55.3 • Testing the lateral ligaments.

Technique

With the contralateral hand, the examiner fixes the patient's lower leg at the distal and lateral side. The ipsilateral hand encircles the midfoot. The hand lies on the first metatarsal and the fingers encircle the lateral border. The hand forces the foot into plantiflexion and valgus. Meanwhile, the fingers provide a pronation movement (Fig. 55.4).

The thick deltoid ligament and the architecture of the midfoot make the foot very firm in this direction. Therefore, not much movement is achieved during this combined test. The normal end-feel is soft.

Tibiofibular ligaments

A strong varus movement applied to the talus held in a neutral position forces this bone as a wedge between the two malleoli,

so testing the integrity of the distal tibiofibular ligaments. In a normal 'mortice', the strong tibiofibular and lateral collateral ligaments prevent separation of the tibia and fibula. When there is ligamentous rupture or laxity, the fibula can be pressed outwards, a circumstance that is detected by a palpable click when the tibia and the fibula engage after their momentary separation. In total rupture of the anterior talofibular or the calcaneofibular ligaments, this test will also be positive. Differentiation should then be made using the anterior drawer test (see p. 784).

Technique

The ipsilateral hand fixes the patient's leg at its inner side, just above the ankle. This position of the hand is important, both to give counterpressure and to detect the click when pressure is released. The contralateral hand grasps the foot at the heel and forces it into varus with a strong, quick thrust (Fig. 55.5).

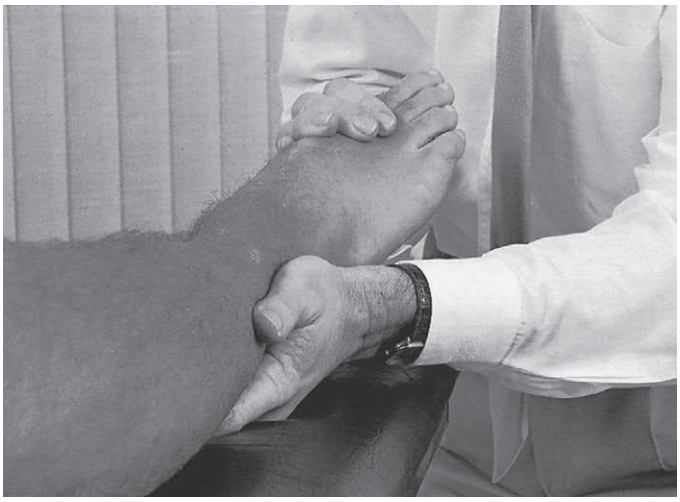


Fig 55.4 • Testing the medial ligaments.

Mobility of the subtalar joint

Varus and valgus

In order to move the calcaneus on the talus, the examiner must try to avoid any movement at the ankle joint. Because the width of the trochlear surface of the talus is smaller posteriorly than anteriorly, the medial and lateral surfaces of the body of the talus are gripped tightly during dorsiflexion of the ankle. Therefore the ankle must be forced into and kept in full dorsiflexion during varus–valgus movements.

Technique

The heel is firmly grasped between the two hands, the fingers clasped behind the heel. Dorsiflexion is performed by traction on the heel (Fig. 55.6). Because the test involves a very strong joint, with little mobility, and because it is hardly possible to obtain any leverage, the examiner must keep the heel as steady as possible. By swinging the upper half of the body, it is possible to gain a good idea of the range of motion.

A normal varus–valgus range is between 20° and 45°. Mobility should always be compared with the other side and the normal end-feel is soft. The varus movement also tests the integrity of the calcaneofibular ligament.

Passive tests for the midtarsal joints

Flexion–extension, pronation–supination, abduction–adduction

As the middle segment of the foot consists of several bones and joints, it is very difficult to assess isolated movement at the various joints. Therefore Cyriax considered the whole middle segment as one integrated structure – the midtarsal joint.

Because of anatomical characteristics, plantiflexion is often accompanied by some adduction and dorsiflexion by some abduction (see online chapter *Applied anatomy of the lower leg, ankle and foot*).

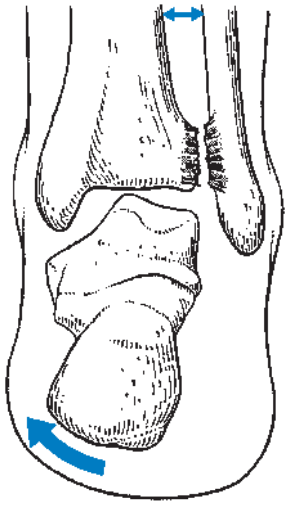
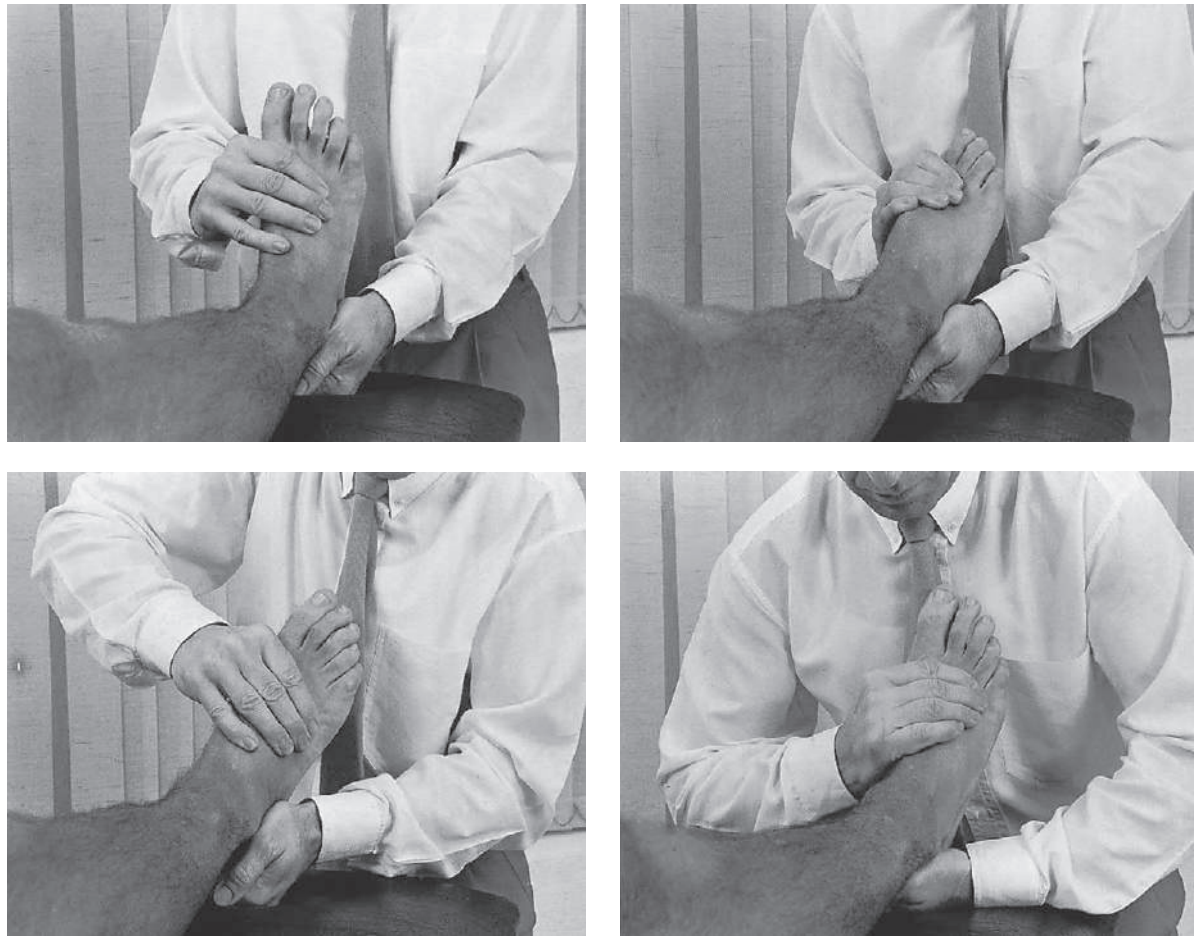


Fig 55.5 • Testing the tibiofibular ligaments (the 'mortice' test).

(a) (b)

Fig 55.6 • Varus (a) and valgus (b).

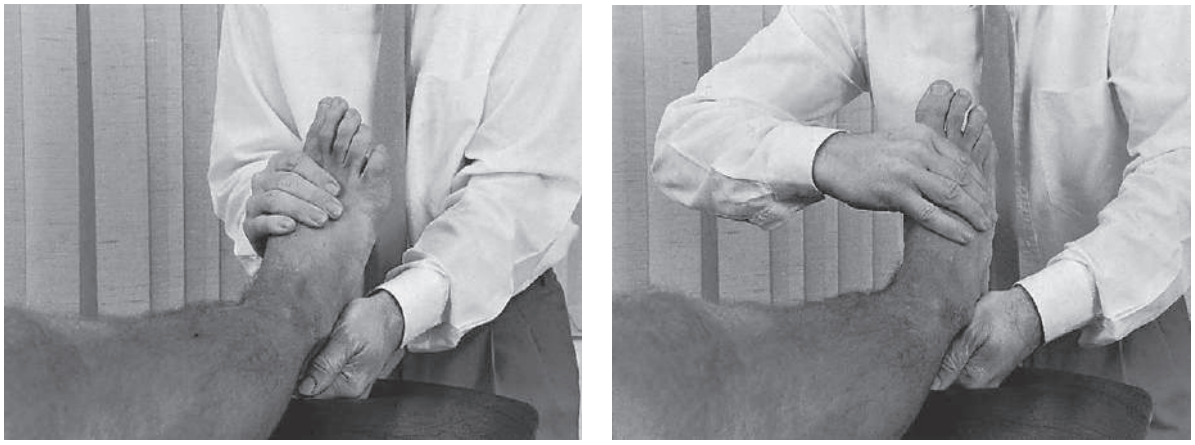


(a)

(b)

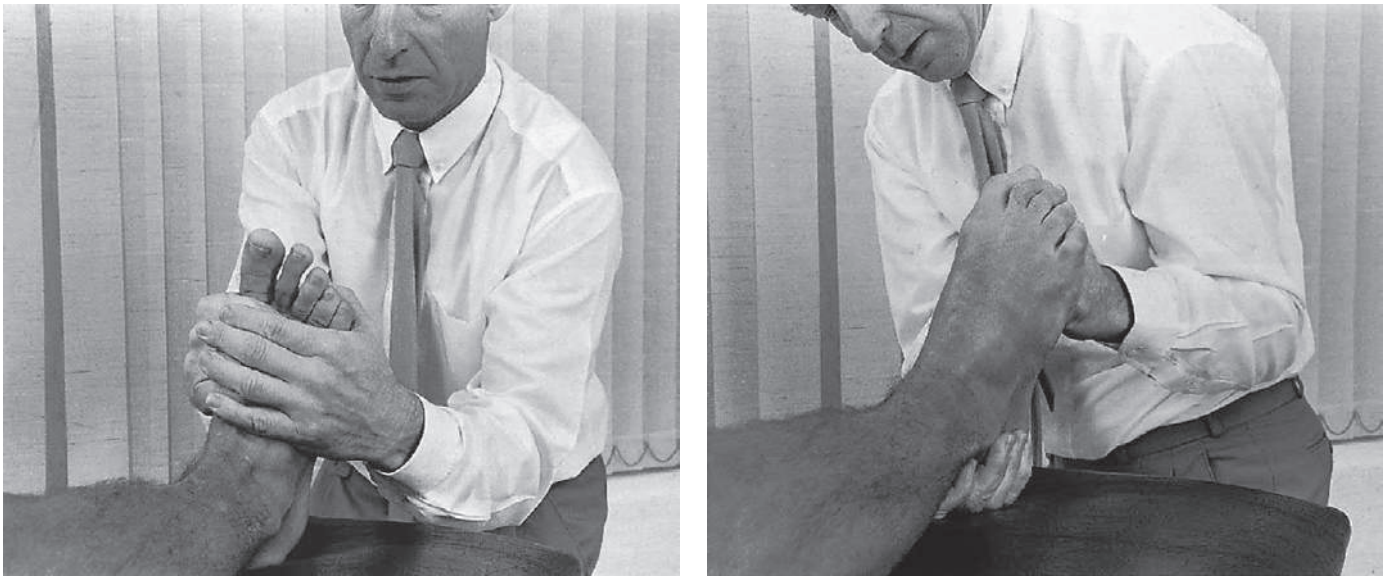
Fig 55.7 • Extension and flexion (a), abduction and adduction (b).

Continued

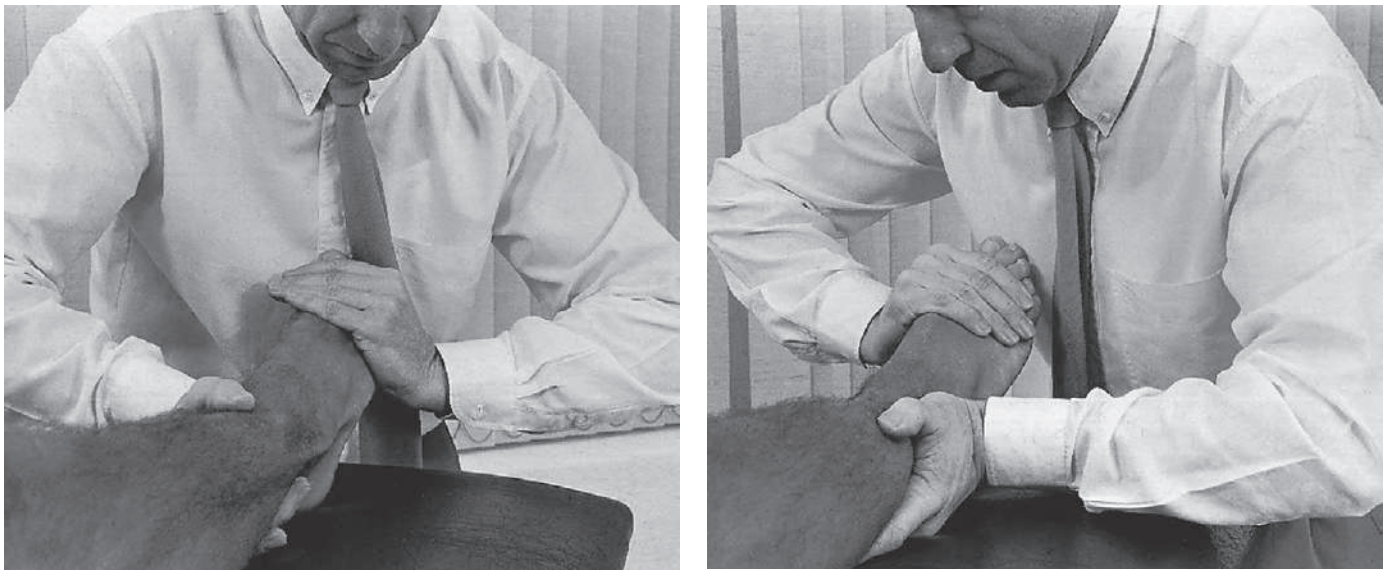


(c)

Fig 55.7 • Cont'd. Pronation and supination (c).



(a)



(b)

Fig 55.8 • Resisted dorsiflexion and plantiflexion (a), and eversion and inversion (b).

Box 55.3

Summary of the clinical examination

History

Inspection

Functional examination

- Rising on tiptoe
- Ankle joint
  - Plantiflexion
  - Dorsiflexion
- Ligamentous tests
  - Mortice
  - Lateral ligaments
  - Medial ligaments
- Subtalar joint
  - Varus
  - Valgus
- Midtarsal joints
  - Plantiflexion
  - Dorsiflexion
  - Abduction
  - Adduction
  - Supination
  - Pronation
- Resisted movements
  - Plantiflexion
  - Dorsiflexion
  - Inversion
  - Eversion

Accessory tests

- Combined movements
- Examination of the toes
- Instability tests

Pronation and supination also examine the inner and outer ligaments of the midfoot. Pronation tests the plantar calcaneonavicular ligament, whereas supination and adduction bring the calcaneocuboid ligament and the ligaments between cuboid and fifth metatarsal under stress.

Technique

First, the posterior segment (ankle and subtalar joints) must be stabilized. Therefore, the examiner uses the contralateral hand to pull strongly on the heel and force it into valgus. The traction forces the talus into the dorsiflexed position between both the malleoli, thus immobilizing the ankle joint, and the valgus position fixes the subtalar joint.

The ipsilateral hand encircles the forefoot, so that the thumb comes to lie under the metatarsal heads and the fingers at the dorsum of the metatarsal shafts. In this position, the examiner can easily perform plantiflexion–dorsiflexion in the foot by a simple pronation–supination movement of the arm. An adduction–abduction movement is achieved by an adduction–abduction movement of the wrist, and pronation–supination by a flexion–extension movement (Fig. 55.7). Although the range of movement varies considerably between individuals, it is surprising how much movement the normal midtarsal joints allow.

The end-feel in flexion–extension and in abduction–adduction is rather hard, whereas the end-feel in both rotations is soft.

Resisted movements

Resisted tests are always performed in a neutral position. No active movement of the joint is allowed. The examiner tries to evoke pure isometric contractions of the tested muscles. In order to avoid a false result, it is absolutely essential to keep the joint immobile during the contraction. Therefore a good immobilization technique is of great importance, especially when inversion and eversion are to be tested against resistance.

Technique

To stabilize the foot during resisted eversion, the patient's leg is immobilized by the examiner's ipsilateral hand, placed at the medial and distal end of the leg. The examiner uses the contralateral hand to apply counterpressure at the lateral border of the foot (Fig. 55.8).

During resisted inversion, the reverse is done: the contralateral hand fixes the leg at the distal lateral side and the ipsilateral hand, placed at the inner border, applies counterpressure.

Resisted dorsiflexion tests the tibialis anterior, extensor hallucis longus, extensor digitorum longus and the peroneus tertius.

The plantiflexor muscles tested are the triceps surae, tibialis posterior, flexor hallucis longus, flexor digitorum longus and peronei brevis and longus.

Resisted eversion tests the integrity of the peronei muscles but also the peroneus tertius and extensor digitorum longus.

Inversion against resistance tests the tibialis posterior and anterior, together with the flexor and extensor hallucis longus, and slightly tests the triceps surae.

Accessory tests

Valgus under dorsiflexion

Forcing the heel into valgus and in dorsiflexion is the only way to reproduce pain in cases of a traumatic periostitis at the anteroinferior surface of the fibula.

Examination of the toes

Passive, resisted examination of the big toe and the outer four metatarsophalangeal joints is performed when a lesion at the forefoot or at the toes is suspected.

Anterior drawer test

This tests the integrity of the anterior talofibular ligament (see p. 785).

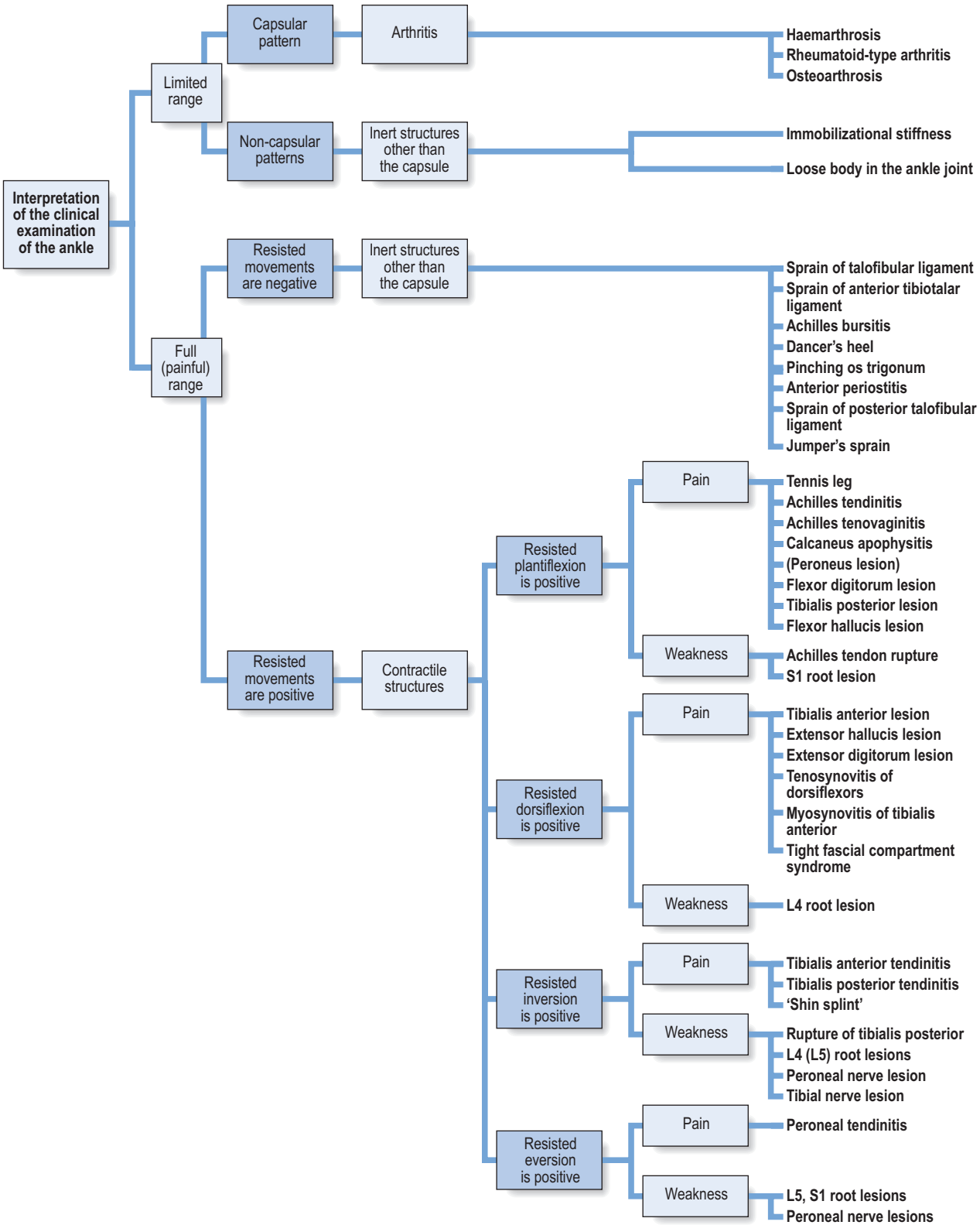
Cases with pain but a normal examination

It is possible for a foot to hurt and yet appear normal on clinical examination, probably because the momentary stress of the manual testing is insufficient to evoke pain. This happens particularly in athletes and ballet dancers who feel pain after exertion, i.e. after a strain far greater than any examiner can impose on the foot by mere clinical testing. In such cases, patients should stand up to demonstrate which particular movements hurt. If the pain only appears during or after training or exertion, the patient is asked to return when the pain has been provoked.

Clinical examination of the lower leg, ankle and foot is summarized in Box 55.3.

Interpretation of the clinical examination of the lower leg, ankle and foot

56



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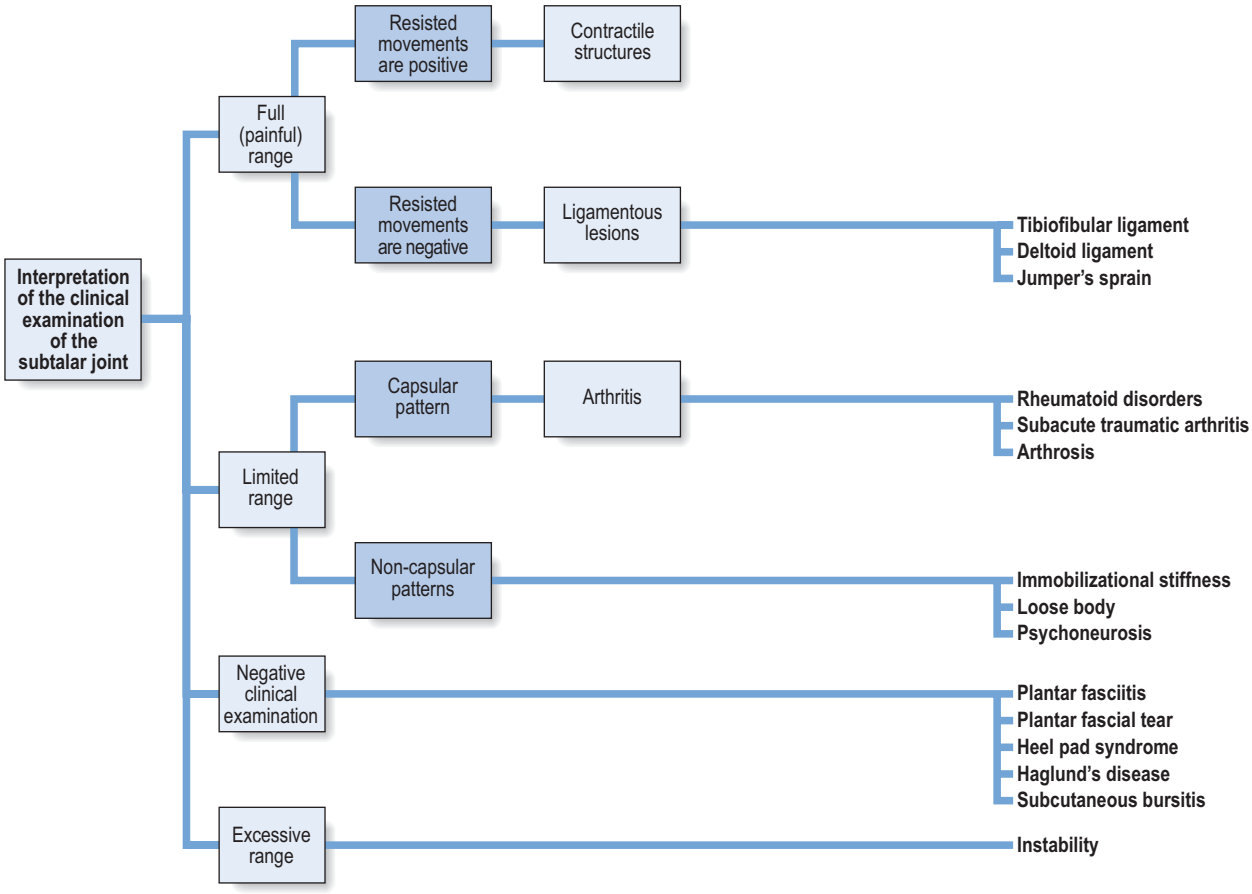
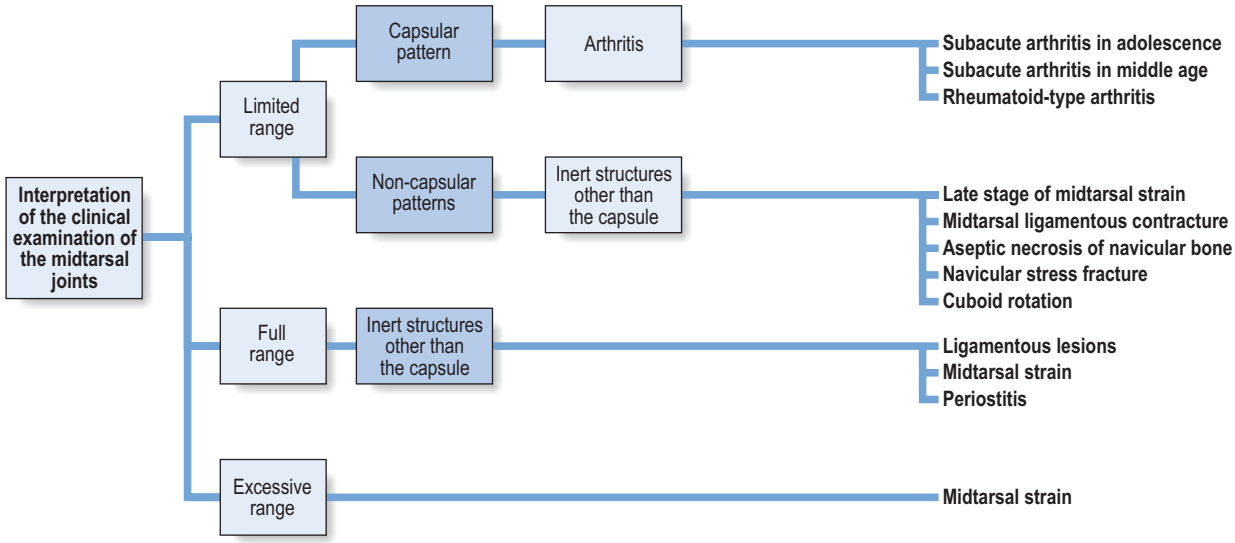

Interpretation of the clinical examination of the subtalar joint
Full (painful) range
Resisted movements are positive
Contractile structures
Resisted movements are negative
Ligamentous lesions
Tibiofibular ligament
Deltoid ligament
Jumper's sprain
Limited range
Capsular pattern
Arthritis
Rheumatoid disorders
Subacute traumatic arthritis
Arthrosis
Non-capsular patterns
Immobilizational stiffness
Loose body
Psychoneurosis
Negative clinical examination
Plantar fasciitis
Plantar fascial tear
Heel pad syndrome
Haglund's disease
Subcutaneous bursitis
Excessive range
Instability


Interpretation of the clinical examination of the midtarsal joints
Limited range
Capsular pattern
Arthritis
Subacute arthritis in adolescence
Subacute arthritis in middle age
Rheumatoid-type arthritis
Non-capsular patterns
Inert structures other than the capsule
Late stage of midtarsal strain
Midtarsal ligamentous contracture
Aseptic necrosis of navicular bone
Navicular stress fracture
Cuboid rotation
Full range
Inert structures other than the capsule
Ligamentous lesions
Midtarsal strain
Periostitis
Excessive range
Midtarsal strain

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Disorders of the lower leg

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Bone disorders

As this book covers 'non-osseous' lesions of the moving parts of the body, little attention will be paid to bone disorders. For differential diagnostic reasons, however, it is as well to bear in mind that, if the patient complains of continuous localized pain, without special clinical findings during functional examination, the possibility of a bone disorder should be considered and a radiograph or a bone scan obtained. Paget's disease, metastases, primary bony tumours and osteomyelitis are possibilities.

Children between the ages of 6 and 11 years who complain of diffuse pain in the legs but have a normal clinical examination are very often regarded as suffering from 'growing pains'. Paediatricians' estimates of the frequency of this diagnosis vary from 4%[1] to 20%[2] of all children. During recent decades, several hypotheses have been put forward but none gives a good explanation for the symptoms. As it occurs between the ages of 6 and 12, which is not the period of maximum rate of growth, 'growing' cannot be the real reason for the pain. Whatever the origin of the pain, it disappears spontaneously and completely after the age of 12 years.[3,4]

Lesions of the plantiflexors

While the patient rises on tiptoe, still the best test of integrity of the plantiflexor mechanism, the examiner notes if there is any pain or weakness.

Pain

Tennis leg

This is the common term to describe a tear in the triceps. It occurs most often in the medial belly of the gastrocnemius muscle, usually some 5 cm above the musculotendinous junction.[5]

The disorder was first described by Hood in 1884.[6] For decades it has been regarded as a ruptured plantaris tendon,[7,8] but careful clinical examination of patients suffering from this disorder shows this to be false.[9]

The history is quite simple. During a vigorous contraction – for example, when starting to sprint, pushing a car or lifting a heavy weight – the patient experienced sudden severe pain in the calf. From then on, the patient was unable to dorsiflex the foot during walking and needed to tiptoe on the affected side.

Examination reveals pain during resisted plantiflexion but no weakness. In the supine-lying position, dorsiflexion at the ankle is found to be markedly limited when the knee is in the

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extended position but becomes normal when the knee is flexed. This implicates the gastrocnemius muscle, part of which is in spasm around the tear. The difference in range between a flexed and an extended knee in a disorder of the gastrocnemius muscle is another example of the constant-length phenomenon. Palpation of the calf reveals the tender area in the medial gastrocnemius, with either *swelling and haematoma* if the lesion is recent, or *induration* around the tear if it is old. After a severe rupture, it is possible to palpate the gap in the medial gastrocnemius. Ultrasonography is the imaging technique of choice to demonstrate size and grade of the lesion.[10,11]

Differential diagnosis

Although the diagnosis is obvious, it is as well to bear in mind some other possibilities.

High rupture of the Achilles tendon

Although the similar onset and localization of the pain can cause diagnostic confusion, this condition should hardly be a problem if a proper clinical examination is done. Weak resisted plantiflexion and no spasm during passive dorsiflexion in a ruptured Achilles tendon contrast with the findings in tennis leg.[12,13] If there is any doubt, a simple test can be done as follows.[14] The patient lies prone, the foot hanging over the edge of the couch. The examiner squeezes the calf muscles. If the Achilles tendon is intact, the foot moves into plantar flexion. When there is total rupture, no movement results.

Deep venous thrombosis in the calf muscles

Differential diagnosis from this condition is very important. Instances have been described of patients with tennis leg receiving anticoagulants, which aggravated their condition.[15] The main historical difference is in the onset. In deep venous thrombosis, pain appears after immobilization or after sitting for a couple of hours,[16] and not during vigorous contraction of the calf muscles. Clinical examination does not show limited passive dorsiflexion, although passive dorsiflexion and resisted plantiflexion can be painful. The calf muscle and leg are swollen and the foot becomes oedematous because of obstruction to venous return. Palpation of the tender spot shows other differences: in thrombosis the whole calf muscle is painful to the touch and sometimes a painful 'string' can be palpated deep within it.[17,18]

Rupture of a Baker's cyst

In long-standing rheumatoid arthritis of the knee, chronic distension with fluid can weaken the posterior ligaments and during exertion these can rupture. Sudden pain in the knee, followed by swelling of the leg and oedema of the ankle, strongly suggests venous thrombosis.[19] Here, the long-standing rheumatoid arthritis and the absence of injury suggest the diagnosis, which can be confirmed by arthrography.

Intermittent claudication

The history is characteristic: pain in one calf is brought on by walking and relieved by rest. Routine clinical examination reveals nothing. Diminished or absent pulsations at the dorsalis artery of the foot and posterior tibial artery may be found. Angiography confirms the diagnosis.

Posterior compartment syndrome

The patient, usually a young man, reports an ache and swelling in the calf some hours after unaccustomed exercise. Walking is uncomfortable and increases the swelling. Examination shows that rising on tiptoe is *not* difficult and not really uncomfortable. The calf is diffusely swollen and the skin is red and warm to the touch. Passive dorsiflexion of the foot is severely limited by loss of elasticity of the calf muscles. Palpation reveals uniform tenderness of the whole calf muscle without any localized tender area.[20] The symptoms result from muscle ischaemia, produced by increased tissue fluid pressure in the closed fascial compartment. The difference from tennis leg lies in the discrepancy between marked restriction of passive dorsiflexion and almost painless tiptoe rising. Treatment is surgical division of the deep fascia.

Referred pain to the calf

This is most frequently the consequence of compression of the S1 nerve root, whether by a primary posterolateral protrusion of nuclear substance at the fifth lumbar level in a younger patient, or by a compression in a narrowed lateral recess in an older person. When these conditions are suspected, a careful history must be taken and a clinical examination of the lumbar spine must be performed. Positive findings during lumbar examination, together with painless tiptoe rising, confirms the diagnosis.

Treatment

Different types of treatment have been advocated for tennis leg. Most authorities advise partial or total immobilization for small tears[21] and surgical suture for serious ruptures.[22,23]

In our opinion, surgery for this condition is scarcely ever necessary and partial or total immobilization by plaster cast or strapping is obsolete. When such treatment is instituted, the formation of a chronic adherent scar is promoted, which results in disability lasting several months. The aim of treatment in muscle tears is to allow the torn fibres to heal in such a way that mobile and functional scar tissue is formed. If normal enlargement of the muscle is impaired by adhesions, self-perpetuating inflammation will be the result. Muscle belly lesions (tennis leg is an excellent example) therefore need a different and more functional approach. The aim must be to restore normal movement in the damaged muscle as quickly as possible. This can be achieved with combined treatment: early compression to decrease the amount of haemorrhage; aspiration and infiltration with local anaesthetic as soon as the patient is seen; and deep transverse friction and electrically stimulated or active contractions of the muscle during the next few days.[24] During the recovery period, the muscle must be protected by a raised heel pad, which enables the patient to use the unaffected parts of the gastrocnemius without strain on the line of healing.

Technique: infiltration

As soon as the patient is seen – whether on the day of the accident or some weeks or months later – local anaesthesia is induced at the site of the partial rupture. The patient lies prone, the knee slightly bent and the foot plantiflexed, to allow maximal relaxation of the muscle. The tender spot is located and gripped between thumb and index finger (Fig. 57.1). If

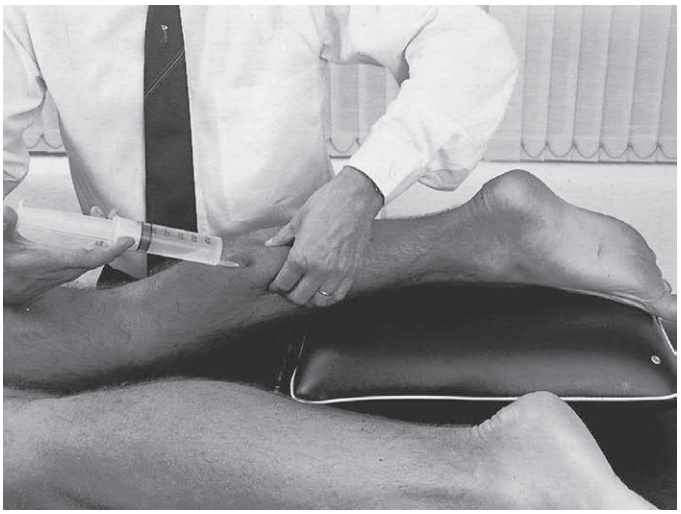


Fig 57.1 • Infiltration with local anaesthesia in tennis leg.

Fig 57.2 • Deep friction to the gastrocnemius muscle.

there is any fluctuation, an attempt is made to aspirate the haematoma. Once that has been done, 30–50 mL of procaine 0.5% is injected using the usual infiltration techniques. As the spot is never precisely localized (gentle pressure does not reveal tenderness in the deeper part of the muscle, whereas strong pressure is apt to hurt throughout the belly) and the gastrocnemius muscle is a large structure, up to 50 mL of a 0.5% solution may be required.

After the injection, pain ceases and a much greater range of passive dorsiflexion at the foot becomes possible within a few minutes. This suggests that the previous limitation was the result of spasm of the muscle. The patient is encouraged to move the foot further up and down while lying on the couch. *Never allow the patient to stand up and to put weight on the affected leg after the injection* because this might lead to a further tear. A raised heel pad must be given before the patient stands and walks.

Raised heel

While the patient is practising non-weight-bearing movements, a cork pad should be made and fitted into the shoe. By putting the weight on the raised heel, the patient will be able to use the unaffected parts of the temporarily shortened muscle, without straining the healing breach.

For the first few days, a heel pad of 3–4 cm may be needed. Thereafter, as treatment and cure proceed, the height is reduced until the pad becomes unnecessary.

Technique: friction and electrical contractions

The day after the injection, the patient is given the first session of deep friction and electrical stimulation to the muscle.

The patient lies prone with the foot fully plantiflexed. The therapist sits at the level of the patient's leg. The fingers are placed at one side of the affected area and the thumb is positioned at the opposite side of the leg so as to apply counter-pressure. The other hand may reinforce the palpating one (Fig. 57.2). Transverse friction starts deeply. By drawing the fingers upwards over the affected area, the therapist feels the muscle fibres escape from the grip until only skin and subcutaneous tissue remain. In the second stage, the fingers are slightly relaxed and moved backwards into the previous deep position, where the same movement starts again. The procedure is continued for about 15 minutes. During friction, the fingers do not move in relation to the skin – fingers and skin move as a unit over the muscle fibres.

At first, massage is given gently. After 5–10 minutes, friction can be deeper and firmer but must always remain comfortable.

After each session of massage, the muscle is given electrical stimulation and active contractions for about 10 minutes, with the knee flexed and the foot in the plantiflexed position. Deep friction prevents transverse scar tissue formation. Electrically stimulated contractions ensure expansion in the muscle belly without tension on the line of healing.

Massage and electrical stimulation are given daily for the first few days and then three times a week, and for a week after symptoms cease, so as to prevent recurrences.

The diagnosis and treatment of tennis leg is summarized in Box 57.1.

Results

When the patient is seen within the first few days of trauma and this scheme of treatment is followed, one can expect normal sports activities to be resumed after 3 weeks.

A patient who presents with a palpable gap in the muscle belly is sometimes referred for surgery. However, in our opinion, surgery is seldom required when a muscle belly is torn. This contrasts with a tendinous rupture – say, of the Achilles tendon – which often calls for surgical intervention.

Achilles tendinopathy

The terminology used to describe this painful condition of the Achilles tendon is very confusing and terms such as Achilles tendinitis, tendonitis and tendinopathy have been widely used. In this book we use the term *tendinopathy* as the general clinical label for the combination of tendon pain, swelling and impaired performance. *Peritendinitis* and *tendinosis* are the histopathological entities of the disease.[25]

Achilles tendon problems are very common among athletes as well as the general population.[1,2] Tendinitis of the Achilles tendon is chiefly a runner's problem.[26] Non-sporting people

Box 57.1

Summary of tennis leg

Diagnosis

- Acute onset
- Limitation of dorsiflexion
- Painful tiptoe rising
- Typical localization of pain and tenderness

Differential diagnosis

- High rupture of the Achilles tendon
- Deep venous thrombosis
- Rupture of Baker's cyst
- Intermittent claudication
- Ischaemic contracture
- Referred pain (S1)

Treatment

- Infiltration with local anaesthetic
  + Heel pad
- Deep friction daily during the first week
  + Electrically stimulated and active contractions
- Deep friction three times a week
  + Electrically stimulated and active contractions

can also suffer from this condition and it then starts after unaccustomed exertion or a long walk in inappropriate shoes. Nevertheless, it most often occurs in long-distance runners, and it comes as no surprise that the increase in the number of people running during the past few decades has been paralleled by an increase in the number of cases of inflamed Achilles tendon.[27,28] In a survey of 2002 running injuries seen over a 10-year period at a sports medicine referral clinic, overuse injury to the Achilles tendon was the sixth most common injury.[29] The lifetime cumulative incidence of Achilles tendinosis is 5.9% among sedentary people and 50% among elite endurance athletes.[30]

The site of the lesion lies usually at mid-tendon. Occasionally, the strain occurs level with the upper border of the calcaneus or, very rarely, at the musculotendinous junction. Achilles tendinitis is multifactorial and usually the outcome of a combination of anatomical and biomechanical characteristics, along with poor training technique.[31] The most important aetiological factors are short triceps (decreased touchdown angle), poor stretching habits and increased plantar flexion peak torque.[32,33] Some authors suggest that there might also be a vascular factor furthering the condition.[34,35] This would explain the usual location of the lesion at mid-tendon, which is the junction of its two vascular systems.[36,37]

During the last decade, fluoroquinolone antibiotics have been implicated in the aetiology of Achilles tendinitis and subsequent tendon rupture.[38–41]

The history in Achilles tendinitis is quite simple.[42] The patient states that the heel aches during or after walking or running (stage I). Sometimes there is pain at the beginning of exertion, improving after a while and reappearing on fatigue (stage II). Alternatively, there is continuous and worsening pain during exercise (stage III). Sometimes the pain does not cease after exertion but continues at rest (stage IV). As a rule, however, pain eases at rest and increases on running or walking. Nocturnal pain is absent.

Clinical examination shows rising on tiptoe to be painful, although in minor cases pain is only evoked by repetitive tiptoe rising. The rest of the functional examination is completely negative except in lesions at the tenoperiosteal junction, where full plantiflexion hurts slightly as this squeezes the affected part of the tendon between the posterior aspect of the tibia and the upper surface of the calcaneum.

Because we advocate deep transverse friction as the most effective conservative treatment for this disorder and massage only acts where it is applied, the tendon should be palpated very carefully to discover *all* the tender areas because there may be more than one. The lesion nearly always lies at the inner or outer aspect, or both, of the tendon. Very often, the anterior surface is affected but almost never the posterior.[43] Most of the lesions lie 2–6 cm above the tenoperiosteal junction.[44] The morphological and macroscopic changes in the condition are not always the same. Observations during surgery have led to the description of two separate histopathological types:

- *Peritendinitis*: the inflammatory changes are confined to the peritenon, which is thickened and shows inflammatory changes, with considerable connective tissue proliferation and adhesions between peritendineum and tendon.[45,46]
- *Tendinosis*: this is characterized by inflammatory and degenerative changes in the tendon tissue itself.[47,48] Sometimes partial ruptures of deeply situated fibres can be seen, with the superficial parts of the tendon remaining intact. Here there is a visible and palpable enlargement at the tendon from the structural changes of chronic fibrosis.[49]

Imaging methods

Ultrasonography has been used increasingly to examine Achilles tendon injuries. It is a quick, safe and inexpensive method to verify the existence and location of intratendinous lesions but is operator-dependent.[50,51] Magnetic resonance imaging (MRI) is useful in visualizing pathological conditions of the tendon but is expensive and time-consuming. It is unwise, however, to rely solely on the results of these imaging techniques. First of all, the normal anatomy of an asymptomatic Achilles tendon may vary, which causes diagnostic misinterpretation.[52] Secondly, several studies have reported intratendinous changes in up to 34% of cadaver specimens, in ultrasound and MRI images of patients without symptoms.[53–55]

Treatment

There are a number of approaches. Initial conservative treatment should be directed towards preventing adhesion formation between tendon and paratenon. Cold packs and stretching comprise most of the treatments proposed in the recent literature and may produce some benefit. Cold therapy is able to control pain and oedema, as well as to reduce regional blood flow and the metabolic demands of the tissue, and thereby helps to prevent further tissue damage at the site of injury.

Passive stretching of the Achilles tendon has been advocated as restoring normal ankle joint mobility and decreasing the strain of the Achilles tendon with normal motion.[56]

None of these therapies, however, addresses the problem of breaking the adhesion formation and restoring normal gliding between tendon and paratenon, which is the main reason for the chronic pain.

Steroid injection

The role of corticosteroid injections in the treatment of Achilles tendinopathy is controversial.[57–61] Several reports demonstrate disappointing results and injections have also been blamed for causing spontaneous rupture of the Achilles tendon.[62] However, localized paratendinous infiltrations with small doses of triamcinolone (10–20 mg) still have a place in the treatment of Achilles tendinopathy, provided the correct technique is used. The injection is never made into the body of the tendon but along the surface, between tendon and peritendineum. After the injection, the patient should avoid all exercise during the first 10 days, after which a training programme can gradually be resumed. The injection can be repeated once if there is no cure after 2 weeks. If this procedure is followed and the correct technique is used, as set out below, postinfiltration ruptures are not to be feared.[63]

Technique: injection

The patient lies prone on the couch. The foot is forced into dorsiflexion over the edge of the couch. This stretches the tendon, which facilitates palpation of the affected area. A 2 mL syringe is filled with 20 mg triamcinolone. A 5 cm needle is fitted and introduced some 3 cm away from the lesion. It is pushed through the skin and moved either upwards or downwards, parallel to the tendon, until the tip reaches the distal edge of the lesion. A small injection is given while the needle is drawn back along the surface of the tendon (Fig. 57.3); the manœuvre is repeated four or five times, each time a little to one side of the previous infiltration line. If the tip of the needle stays superficial to the tendon, no particular resistance to the plunger will be felt during the injection.

Prolotherapy

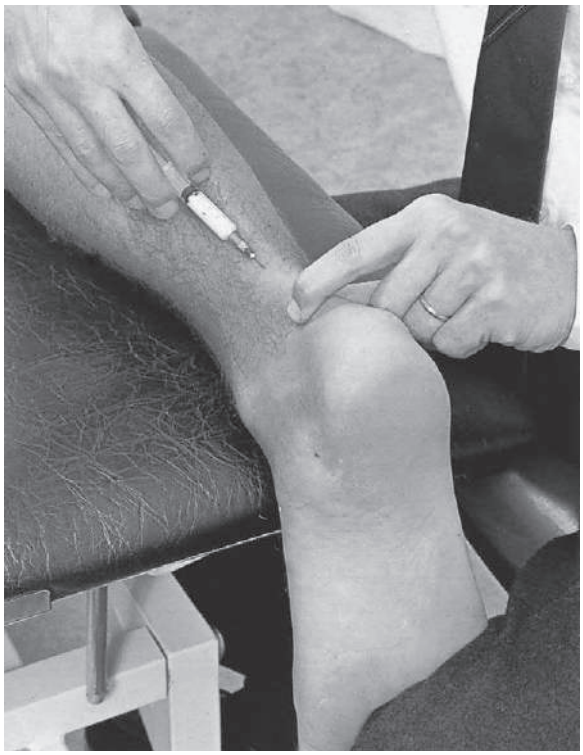
Recently, promising results have been described in studies of patients with Achilles tendinopathy who were treated with paratendinous injections of a sclerosing agent. On ultrasonography, neovascularization and tendon inhomogeneity decreased significantly as the clinical parameters improved.[64–66]

Surgery

Operative treatment should only be considered in long-standing cases when rest and conservative treatment (deep transverse friction) have failed. The surgical procedures performed depend entirely on the underlying pathological features.

In simple peritendinitis, the crural fascia and the paratenon are split longitudinally from the musculotendinous junction to the insertion on the calcaneus, and adhesions between the tendon and the sheath are released.[67]

In cases with focal tendinosis and partial rupture of the tendon, the diseased area of the tendon is excised and the tendon repaired by side-to-side suture of the remaining tendinous fibres.[68] Where excision of the degenerated tissue disrupts the continuity of the tendon, the tendon is reconstructed and reinforced with the fascia of the gastrocnemius.[69]



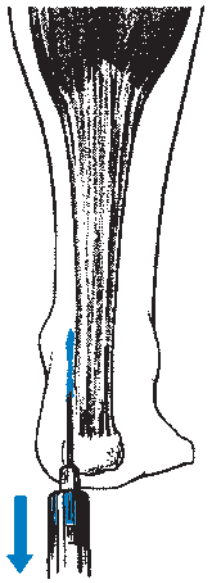


Fig 57.3 • Steroid infiltration parallel to the surface of the tendon in Achilles tendinitis.

If there is extensive tendinitis, excessive scar tissue is excised.[70] Some authors claim better results if carbon fibres are implanted between the tendinous straps.[71]

Postoperative care involves between 1 and 4 weeks of non-weight bearing, followed by 1–4 weeks of partial to full weight bearing, depending on the severity of the lesion and the type of surgical intervention.

Results are good to excellent in about 85% of cases.[72,73]

Deep friction to the Achilles tendon

This was introduced more than 40 years ago by Cyriax[74] and is, in our opinion, the best conservative treatment for the condition, provided it is given correctly: (a) at the right point, (b) with a good friction technique, (c) 20 minutes per session and (d) at regular intervals (three times a week). During the period of treatment, the patient must take relative rest.

Technique: friction to the medial and lateral edge of the tendon

The patient lies face downwards on a low couch, with the foot projecting just beyond the edge of the couch. The therapist sits at the foot and pushes the sole into dorsiflexion through a slight pressure of the contralateral knee. At the correct point the tendon is grasped between finger and thumb, or two fingers and thumb, depending on the extent of the lesion ('a' in Fig. 57.4). Slight pressure under the tendon is exerted and the hand is drawn upwards until the fingers slip towards the posterior aspect of the tendon ('b' in Fig. 57.4). In the final stage of this manœuvre, only skin remains between the two grasping fingers. The pressure is now released slightly and the fingers are pushed downwards until they lie at the starting position ('a') and the whole procedure can be repeated. The cycle is repeated for about 15 minutes.

During friction, fingers and skin are moved as a whole over the tendon. Should there be any movement between the patient's skin and the therapist's fingers, the friction will be imparted to the patient's skin and, if vigorous, will quickly cause a blister.

Friction must be given with sufficient transverse sweep, which means that the amplitude of the to-and-fro movements must be large enough to ensure that the frictional element is paramount. This is the only way to ensure effective separation of each fibre from its neighbour.

Although deep friction to the Achilles tendon is uncomfortable for the patient, especially at first, it should never be intolerably painful. The true art is to give enough friction without too much pressure. The fact that massage is painful is no guarantee that it is correctly given.

Technique: friction to the anterior part of the tendon

This localization should always be sought in addition to other tender spots.

The patient lies face downwards on the couch but further up the couch than in the previous technique, in order to allow the foot to adopt a full plantiflexion position. The relaxed tendon then has good mediolateral mobility. With one thumb at the opposite side, the examiner pushes the tendon over sideways, as far as it will go. The anterior aspect of the opposite side of the tendon can now be reached with the tip of the ring finger of the other hand. The ring finger is reinforced by the long finger, while the little finger is kept out of the way in order not to disturb the pronation–supination movement. This movement is carried out with the elbow flexed and with the finger, hand and forearm in a straight line with the patient's leg.

Once again, friction is given in two phases. First, the fingertip is placed deeply under the tendon with the forearm in a slightly pronated position ('a' in Fig. 57.5). Pressure is exerted in a dorsal and medial direction; meanwhile, the arm moves in full supination, until the lateral fibres escape under the

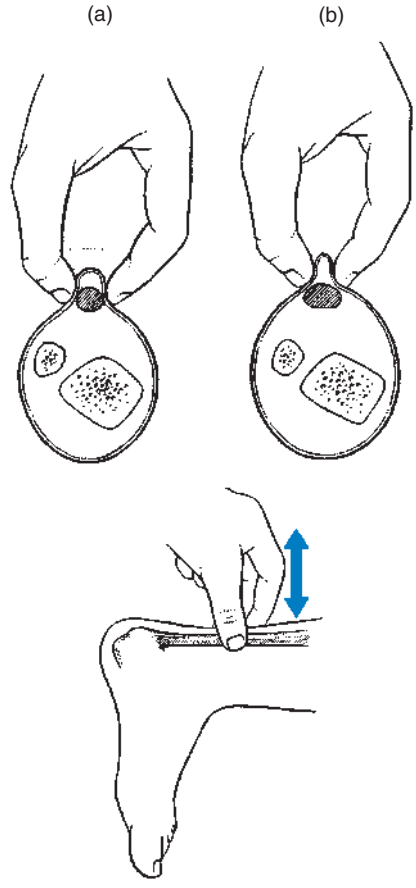


Fig 57.4 • Deep friction to the medial and lateral edge of the Achilles tendon.

fingertip ('b' in Fig. 57.5). Caution is taken not to move the finger on the skin but rather to move the skin and the fingertip as a whole. Now pressure is slightly released and the fingertips replaced in the former position ('a'), so that the whole procedure can be repeated. The to-and-fro movements are performed for 15 minutes. The fingers must not be allowed to glide on the skin, to avoid the formation of a blister.

During the whole procedure, there must be sufficient sweep, which can be achieved by a good pronation–supination movement at the elbow.

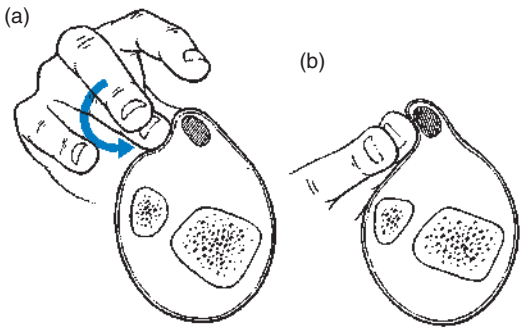


Fig 57.5 • Deep friction to the anterior aspect of the Achilles tendon.

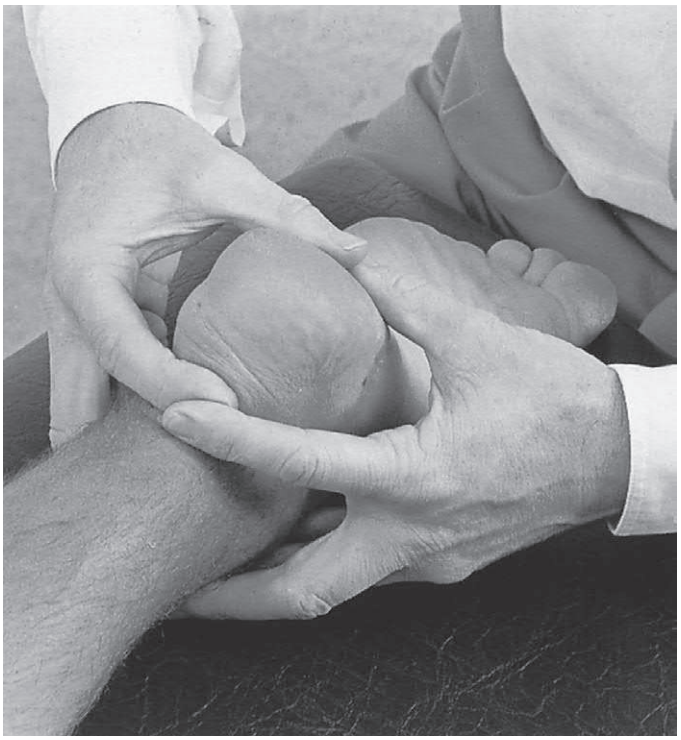


Fig 57.6 • Deep friction to the insertion of the Achilles tendon.

Technique: friction to the tenoperiosteal insertion of the tendon

A lesion at the tenoperiosteal insertion lies level with the upper surface of the calcaneus. As in the earlier technique, the tendon must be relaxed; therefore the patient moves upwards on the couch to bring the foot into full plantiflexion.

The tendon is compressed between the therapist's fingers and the upper surface of the calcaneus. Using both hands, a circle is made round the heel with one index finger, reinforced by the other, at the lesion. Both thumbs cross at the plantar surface. Pressing downwards squeezes the tendon between the fingers and the calcaneus. Deep friction is performed by moving the forearms in opposite directions. The amplitude of the movement must be large enough to reach the whole width of the tendon (Fig. 57.6).

Results of deep friction. Provided massage is given at the right point and the technique is adequate, the results of deep transverse friction are good. Usually, a schedule of 15 minutes, three times a week over 2–4 weeks, is required. In the meantime, the patient should not run or walk any further than necessary. The only failures are in nodular scarring around an old partial rupture. Our experience is that Achilles tendinitis with macroscopic fibrous changes in the deep layers of the tendon often remains refractory to conservative treatment, whereas superficial lesions without palpable changes respond quite well to friction.

Eccentric training

Recently, there has been increasing interest in the use of eccentric loading in treatment of chronic Achilles tendinopathy. (In an eccentric contraction, the muscle–tendon unit lengthens as a load is applied to it.) In 1986 Stanish *et al*[75] reported that a once-daily, 6-week eccentric loading programme led to complete relief of pain in 44% and a marked improvement in symptoms in a further 43% of patients. More recently, others have confirmed the efficacy of eccentric loading for mid-substance lesions of the Achilles tendon,[76,77] and high success rates (at least 60% either good or excellent) have also been reported in prospective randomized trials.[78–80] However, the mechanism for the efficacy of eccentric loading remains unknown.[81]

Prevention

Raised heels, shock-absorbing heels and correction of a valgus deformity are usually prescribed.[82] As the triceps is also a slight invertor, a valgus deformity must always be corrected to relieve excessive strain on the tendon.[83] Although these measures are certainly beneficial, it is important to remember that they are prescribed for prevention only because they do not lead to substantial and histological changes at the site of adhesions.

Many authors pay a great deal of attention to the preventive value of stretching exercises before running.[84,85] Although they may have such value, the worth of stretching techniques alone should not be overestimated when they are used as therapy in actual lesions. Stretching is a good method of prevention but, in our experience, has no therapeutic value.

Achilles tenovaginitis

Although the Achilles tendon does not have a sheath, inflammation can sometimes occur between the tendon and the peritenon and create a so-called *tenovaginitis* of the Achilles tendon. This can take two forms: rheumatoid and xanthomatous.

Rheumatoid arthritis and ankylosing spondylitis

In these conditions, the contrast between the mild symptoms and the marked signs is striking. It is also important to remember that gout can sometimes affect the Achilles tendon. The main complaint is not, as in tendinitis, pain during contraction of the triceps but pain during local pressure, e.g. catching the heel against the edge of the shoe. Examination shows that rising on tiptoe is indeed just uncomfortable rather than painful but the tendon is warm, swollen and very tender to the touch.[86,87] Local treatment consists of infiltration with 20 mg of triamcinolone between the sheath and the tendon. The technique is the same as described for Achilles tendinitis.

Xanthomatous tenovaginitis

The Achilles tendon is a common site of xanthomatous lesions. Similar swellings can be seen and palpated at the extensor tendons on the dorsum of both hands or occasionally on the tendons crossing the dorsum of the foot and on a line at the upper part of the ulna and tibia. Small nodules can also be seen in the upper eyelids. There is only slight discomfort during walking and tiptoe rising is painless. Both tendons can be seen to be thickened, and palpation reveals enlargement and diffuse nodularity. The nodules are painless to the touch.

Treatment consists of restoring normal lipid metabolism with clofibrate 1.5 g daily, or fenofibrate 300 mg daily.

Calcaneus apophysitis

Apophysitis of the calcaneus (Sever's disease, Osgood's disease) occurs in boys between 6 and 12 years of age and is often bilateral. The disorder has been classified among the general osteochondrosis syndromes, such as Legg–Calvé–Perthes disease at the hip or Osgood–Schlatter disease at the tibial tuberosity. The child complains of sharp pain in the heel, occurring during contraction of the triceps. The pain starts suddenly, can last a couple of hours or days, and may disappear and reappear. Rising on tiptoe can be negative but the bone is usually tender to the touch. The radiological appearances are characteristic – 'slight fragmentation of the apophysis' – and confirm the diagnosis. Spontaneous recovery occurs in a year or two, often with the development of a slight permanent prominence on the posterior aspect of the calcaneus.[88]

There is no treatment except relative rest, slightly raising the heel and inserting shock-absorbing heel pads in the shoe.[89]

Weakness

Weakness of the plantiflexor mechanism is detected by an inability to rise on tiptoe.

Apart from neurological lesions, which are discussed at the end of this chapter, the commonest cause of weakness is a rupture of the Achilles tendon, most frequent in athletes over 30 years of age.[90] The tear lies in the 'critical zone of poor vascularization', which is 2–6 cm above the calcaneal insertion.[91]

It is surprising that this simple and easy-to-detect condition very often remains undiagnosed for a time. Between 30 and 41% of Achilles tendon ruptures remain undiagnosed for more than 2 weeks.[35,92,93]

The history is typical. During a sudden contraction – for example, at the start of or during a sprint, lifting a heavy object or pushing a car – the patient hurts the heel. There is a snap, followed by a dull pain in the heel. This eases fairly quickly but, from then on, the patient finds that only hobbling on a flat surface is possible. Alternatively, there may not have been a sudden contraction of the triceps but rather a severe elongation caused by excessive and sudden dorsiflexion at the ankle.

When the patient lies on the couch, the clinical examination reveals little. There is usually no haematoma or visible thickening. Passive movements are painless. An alert examiner will probably detect an excessive range of dorsiflexion. In the supine, non-weight-bearing position, resisted plantiflexion remains possible because the plantaris, flexor hallucis longus and flexor digitorum muscles still contract. Rising on tiptoe, however, immediately reveals the diagnosis. The squeeze test is also positive: the patient lies prone, with both feet suspended over the edge of the couch; compressing the triceps surae causes plantiflexion of the foot when the tendon is intact but not when it is ruptured.[14]

Differential diagnosis

The differential diagnosis from tennis leg is quite simple. In a patient with a minor tear in the medial gastrocnemius muscle, there is pain in the calf and the foot is fixed in plantiflexion. This contrasts very strongly with the absence of pain, the inability to plantiflex against resistance and the excessive dorsiflexion range at the ankle that are characteristic of a rupture of the Achilles tendon.

Although the history of sudden pain and disability may suggest a sprained ankle, a rupture of the Achilles tendon will not be missed if the clinical examination is executed properly.

Treatment

There is still controversy concerning the best treatment for Achilles tendon ruptures. It appears that a satisfactory outcome may be achieved with either non-operative or operative treatment but surgical repair appears to provide better functional capacity[94] with better scores of strength, power and endurance[95–97] and lower re-rupture rates. The advantages of the conservative approach are that there are no risks of

anaesthesia, infection, skin adhesions and sural nerve injury.[98,99] Reports in the literature indicate that in active, young, very demanding individuals, surgical repair should be considered, with non-surgical treatment reserved for elderly or sedentary patients.[100–102] However, conservative treatment can also be considered in athletically active patients, as treatment should always be individualized to the concerns and health of the patient.[103,104]

There is no single, uniformly accepted surgical technique for Achilles tendon repair. Most acute ruptures have been treated successfully with excision of the devitalized tendon tissue and simple end-to-end suture. However, various augmentation procedures, such as reinforcement of the suture with a triceps surae tendon tip-over graft, have been combined with simple suture and have had satisfactory outcomes.[105–107] To minimize the complications typically associated with open surgery, percutaneous techniques to repair the ruptured Achilles tendon have been advocated, and the results are reported to be promising, although not without failures and complications.[108,109]

Several recent studies have reported functional benefits of early postoperative tendon mobilization in well-motivated patients, as treatment results are determined not only by the method of repair but also, and perhaps more importantly, by the early postoperative functional rehabilitation.[110,111]

Conservative treatment consists of 8 weeks in a plaster cast with the foot in equinus position, followed by the use of a 2.5 cm heel for an additional 1 or 2 months.[112,113] The thickened and enlarged scar tissue resulting from conservative treatment can be massaged twice a week for 2–3 months. The fibrous swelling at the point of the rupture never seems to disappear, however, and some residual disability can be permanent.

It can be concluded that the treatment of choice in the athletically active patient is primary surgical repair.[114] Conservative treatment is chosen in middle-aged or less active patients.[115]

Short plantiflexor muscles

Short calf muscles, resulting in equinus deformity, are among the most common complications in children with cerebral palsy.[116] Sometimes, shortening of calf muscles is an isolated finding and may easily be overlooked.

History

Usually, the parents bring the child's gait to attention. They notice that the youngster turns the feet outwards during walking or running, adopting a 'Chaplin' style of moving. Because the child cannot dorsiflex the ankle joint during walking, the feet must be turned outwards to take off. As this is not the best technique for running, the child will run conspicuously more slowly than healthy peers.

Sometimes the parents also notice that the child tends to tilt backwards on standing. The poise around the centre of gravity is incorrect, which leads to muscular fatigue. Therefore the child will complain that prolonged standing is tiring.

Box 57.2

Short calf muscles

Symptoms

- Pain on standing/muscular fatigue
- Outward position of the feet

Complications

- Achilles tendinopathy
- Metatarsalgia
- Plantar fasciitis
- Mid-tarsal strain and arthrosis

Clinical examination

This shows the dorsiflexion range at the ankle joint to be painlessly limited. A gastrocnemius equinus is typically characterized by less than 10° of ankle dorsiflexion with the knee extended.[117]

Complications

Because the patient cannot bear weight adequately on the heels, mid-tarsal hypermobility and later painful strain will result. Limited dorsiflexion at the ankle can also lead to various compensations in the lower extremity that ostensibly cause secondary problems, including Achilles tendinopathy,[118] flat foot, chronic metatarsalgia[119] and strain of the plantar fascia.[120]

Symptoms and complications associated with short plantiflexor muscles are summarized in Box 57.2.

Treatment

Younger patients

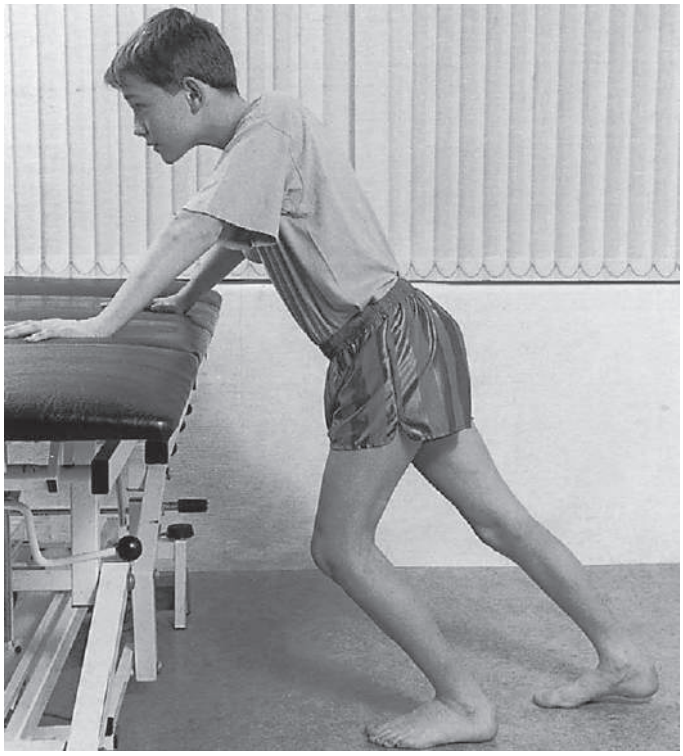
The mid-tarsal joints must be protected, together with the plantar fascia and the forefoot. Therefore the heel must be raised temporarily by a horizontal surface. The child is taught to do stretching exercises for soleus and the gastrocnemii, several times a day, for 2–6 months. These lengthen the calf muscles so that the raised heel on the shoe can be discarded.

The soleus muscle is stretched in the following way. The child performs knee flexion exercises and tries to keep the heels on the ground (Fig. 57.7a). Once this can be done easily, the patient is encouraged to lean further and further forwards so as to stretch the soleus more.

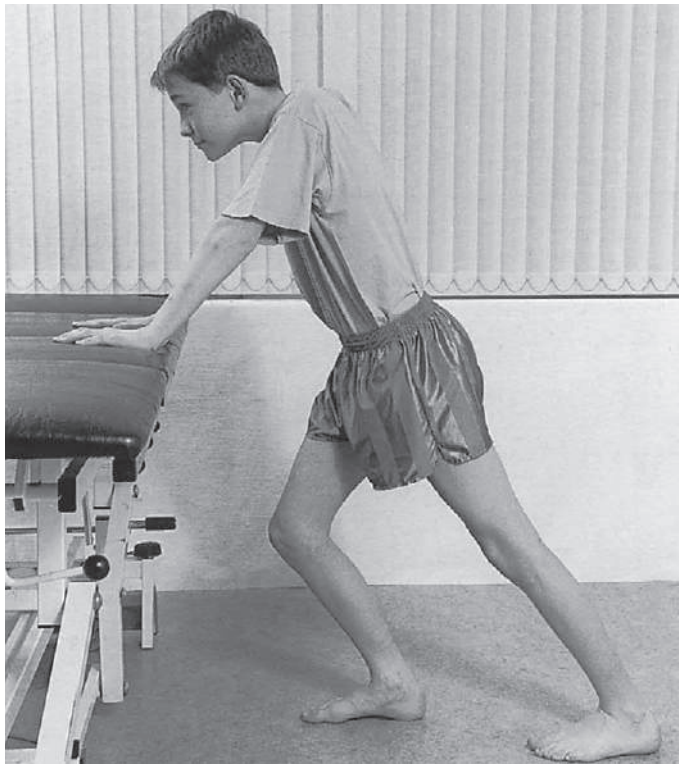
To stretch the gastrocnemii muscles, the child stands in front of a wall, with feet fully dorsiflexed and knees extended, the heels remaining on the ground. The patient then leans forwards, keeping the knees firmly in full extension (Fig. 57.7b).

Adults

The same treatment can be tried but the process of stretching the calf muscles is tedious and tends to fail. Here, the shortening can only be compensated by raising the heel of the shoe.



(a)



(b)

Fig 57.7 • Stretching of the left soleus (a) and left gastrocnemii (b).

Alternatively, surgery can be advised. Recently, good results have been reported with endoscopic release of the gastrocnemii.[121,122]

Lesions of the dorsiflexors

Pain

If resisted dorsiflexion of the foot elicits pain, a resisted movement of the hallux, and then of the toes, identifies the offending muscle: tibialis anterior, extensor hallucis longus or extensor digitorum longus. Further palpation then discloses the precise localization, which can be at the muscle, the tendon or the tenoperiosteal insertion.

Lesions of the muscle bellies respond equally well to local anaesthesia and deep friction (Box 57.3). Three injections, each of 10–30 mL of procaine 0.5%, at weekly intervals usually give good results. Friction is given according to the general principles that it should be deep and transverse, with sufficient sweep on a relaxed muscle, three times a week for 6–10 sessions.

Lesions of the tendons respond to deep friction, on a stretched tendon, three sessions a week, for 6–10 sessions.

Lesions at the tenoperiosteal junction respond to deep transverse friction and to infiltration with 10 mg of triamcinolone.

Special conditions

Ischaemic contracture or adherence of the extensor hallucis longus

The muscle may develop ischaemic contracture or become adherent after a fracture at mid-tibia. This results in a constant-length phenomenon. Each time the foot is plantiflexed, the shortened extensor hallucis muscle extends the big toe, which is forced against the tip of the shoe. This may result in extreme soreness of the big toe. The treatment is tenotomy level with the first metatarsophalangeal joint.

Tenosynovitis of the extensor digitorum longus or the extensor hallucis longus

This rare condition occurs level with the ankle and is usually a sequel of a sprained ankle. Sometimes it is seen as a rheumatoid condition. Apart from pain, a localized swelling can be seen and occasionally crepitus can be felt. In traumatic tenosynovitis, infiltration with 1 mL of triamcinolone between the tendon and sheath and deep friction have equally good results. In rheumatic conditions, infiltration is the only method of symptom relief.

Myosynovitis of the tibialis anterior muscle

The only parallel condition in the body is myosynovitis of the bellies of the abductor pollicis longus and the extensor pollicis muscles in the forearm. The lesion occurs as an overuse phenomenon in skaters, skiers and long-distance runners. It has also been described in army recruits after a long march, who are unaccustomed to wearing heavy boots.[123] Resisted dorsiflexion hurts above the front of the ankle. Painless resisted dorsiflexion of the big toe and outer toes points to the tibialis

Box 57.3

Summary of treatment of lesions of the dorsiflexors

Muscle

- *Either* 10–30 mL procaine 0.5%
- *Or* deep friction 3 times a week, 10 sessions

Tendon

- Deep friction 3 times a week, 10 sessions

Tenoperiosteal junction

- *Either* 1 mL triamcinolone (10 mg/mL)
- *Or* deep friction 3 times a week, 10 sessions

anterior muscle but palpation fails to reveal any tenderness in the anterior tibial tendon itself. If the palpating finger moves upwards to the musculotendinous junction, a painful area is detected close to the bone. If the foot is moved gently up and down, crepitus may be felt.

Treatment consists of deep transverse friction, which solves this problem very quickly: 2–4 sessions at intervals of 2 days usually give lasting relief.

Technique: deep transverse friction to the tibialis anterior muscle

The therapist sits opposite the patient. With one hand, the foot is brought into full plantiflexion and eversion in order to stretch the muscle. The thumb of the other hand is placed at the medial aspect of the leg and the fingers on the lesion (Fig. 57.8). The thumb is used as a fixed point and the fingers move in a transverse direction over the anterior aspect of the anterior tibial muscle. To allow sufficient sweep, the movement is conducted by an adduction movement of the shoulder.

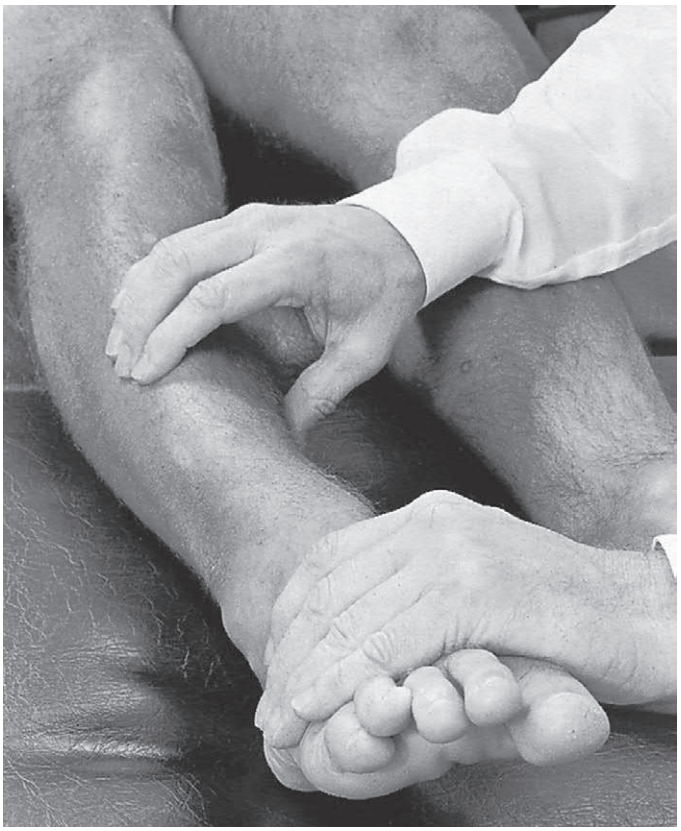


Fig 57.8 • Deep friction to the tibialis anterior muscle.

Tight fascia syndrome of the anterior compartment

This disorder may resemble intermittent claudication but the patient is much younger and physically active. After exertion – for example, kicking a football for about 10 minutes – voluntary dorsiflexion becomes impossible and what seems like a drop foot develops. This is accompanied by vague pain at the front of the leg and sometimes by pins and needles over the dorsum of the foot and the inner four toes. After a short rest, the muscle recovers and pain and weakness disappear.[124] When the patient is examined at rest, nothing special is revealed: dorsiflexion is strong and painless, and the arterial pulsations are normal. In long-standing cases, it is sometimes possible to palpate fascial hernias over the front of the leg.[125]

The symptoms result from increased tissue fluid pressure in the closed fascial compartment which is formed by the tibia, the fibula, the interosseous membrane and the anterior fascia (Fig. 57.9). During exercise, muscle volume may expand by 20%, from both increased blood flow and capillary filtration.[126,127] If the superficial fascia is not lax enough to accommodate the greater volume, the intramuscular pressure rises to levels that compress the anterior tibial artery. This

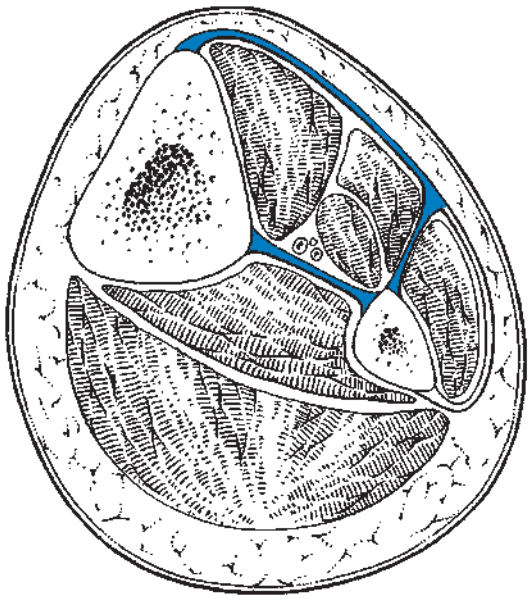


Fig 57.9 • The anterior compartment (colour indicates the fascia).

produces relative muscle ischaemia and therefore temporary paralysis and pain. The swelling may also trap the superficial peroneal nerve at its exit through the fascial foramen at mid-leg and cause paraesthesia in the outer foot.

After some minutes' rest, blood flow recovers and symptoms resolve. Because the patient is asymptomatic between

occurrences and not chronically disabled, some authors prefer the suffix 'recurrent' rather than 'chronic' to identify anterior compartment syndromes.[128] However, if the minute vessels in the belly of the muscle themselves gradually become silted up with cells, the disorder may become irreversible.[129]

The diagnosis is made on the typical history in combination with a negative clinical examination. However, if the athlete is examined after completing the exercise that reproduces the symptoms, clinical examination will show pain on passive plantiflexion and weak dorsiflexion. The anterior compartment is also tender on palpation.[130]

The differential diagnoses of an anterior compartment syndrome include shin splint, stress fracture, nerve entrapment syndromes and claudication.[131] The gold standard of diagnosing anterior compartment syndrome is the measurement of intracompartmental pressures before and after exertion.[132,133] A resting pressure >15 mm Hg, and 5 minutes post-exercise pressures >20 mm Hg are diagnostic of compartment syndrome.[134–136]

Acute and severe anterior compartment syndromes may develop after a tibial fracture[137] or a direct blow.[138] Within a few hours, pain at the mid-leg becomes intense and is not relieved by immobilization. The overlying skin sometimes becomes shiny and warm. Together with the palpable tenderness, this may give the false impression of cellulitis. Making this error is a disaster because, if the diagnosis is not made at once and the fascia divided immediately, ischaemic necrosis will ensue. Complete paralysis with a permanent drop foot will then result. There are also documented cases of bilateral compartment syndrome with permanent drop foot complicating surgery of long duration in the lithotomy position.[139]

Treatment

Apart from altering the patient's activities or training programmes, surgery seems to be the only effective treatment for recurrent compartmental syndromes. The surgical procedure consists of a subcutaneous fasciotomy.[140–142]

In acute cases, surgical decompression must be performed as soon as possible. As in a strangulated inguinal hernia, the motto is: 'operate before sunset or sunrise'.[143] Sheridan and Matsen[144] found that fasciotomy performed within 12 hours of onset of a compartment syndrome resulted in normal function in 68% of cases. However, in those decompressed after 12 hours, only 8% recovered normal function.

Weakness

Painless weakness of dorsiflexion results from neurological lesions that are discussed at the end of this chapter.

Lesions of the invertors

Pain

The main invertor muscles are the anterior and posterior tibialis muscles (Fig. 57.10). The flexor hallucis longus and the triceps are weak invertors as well.

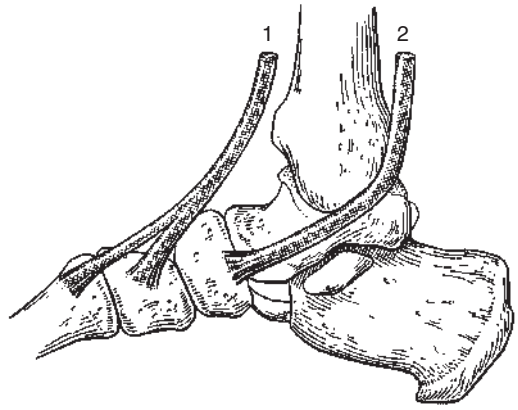


Fig 57.10 • Tendons and insertions of the main invertor muscles: 1, tibialis anterior; 2, tibialis posterior.

Posterior tibial tendinitis is usually caused by overuse, which occurs frequently in a valgus deformity at the subtalar joint.[145] Resisted inversion of the foot hurts and dorsiflexion does not. Because the tibialis posterior has an additional function in stabilizing the hindfoot during rising on tiptoe, this test may also be painful. Palpation then discloses whether the lesion lies at the insertion on the navicular bone, at the tendon (distal or proximal to or under the medial malleolus), at the muscle belly or at the proximal musculotendinous junction.

Sometimes the tendon sheath rather than the tendon is inflamed, causing tenosynovitis. This occasionally occurs in rheumatoid arthritis.

Tendinitis usually recovers with a few sessions of massage but the valgus deformity at the heel must be corrected in order to prevent recurrences. Pain resulting from overuse secondary to a valgus deformity will never be relieved unless a combined approach is used consisting of deep friction and correction of the valgus deformity with a support at the heel.

In rheumatoid tenosynovitis, 10 mg of triamcinolone is injected between the tendon and its sheath.

Deep friction techniques

The technique differs according to the site of the lesion: above or below the medial malleolus. In both instances, the patient lies on the couch with the hip in external rotation so that the inner surface faces upwards.

Technique: friction to the upper part of tibialis posterior

The therapist sits lateral to the patient's foot. With the contralateral hand, the foot is fixed in dorsiflexion. As the tendon lies deeply between the posterior aspect of the tibia and the tendon of the flexor digitorum longus, the proper spot can only be reached if the pronation–supination technique is used. The middle finger of the ipsilateral hand, reinforced by the index finger, is thus laid flat on the affected part of the tendon, just behind the edge of the tibia (Fig. 57.11). The ring and little fingers are kept extended. The fingers, the wrist and the forearm are brought into line with the tibia. Friction is imparted with a supination movement, which is continued until the tendon escapes from the finger. Pressure is then

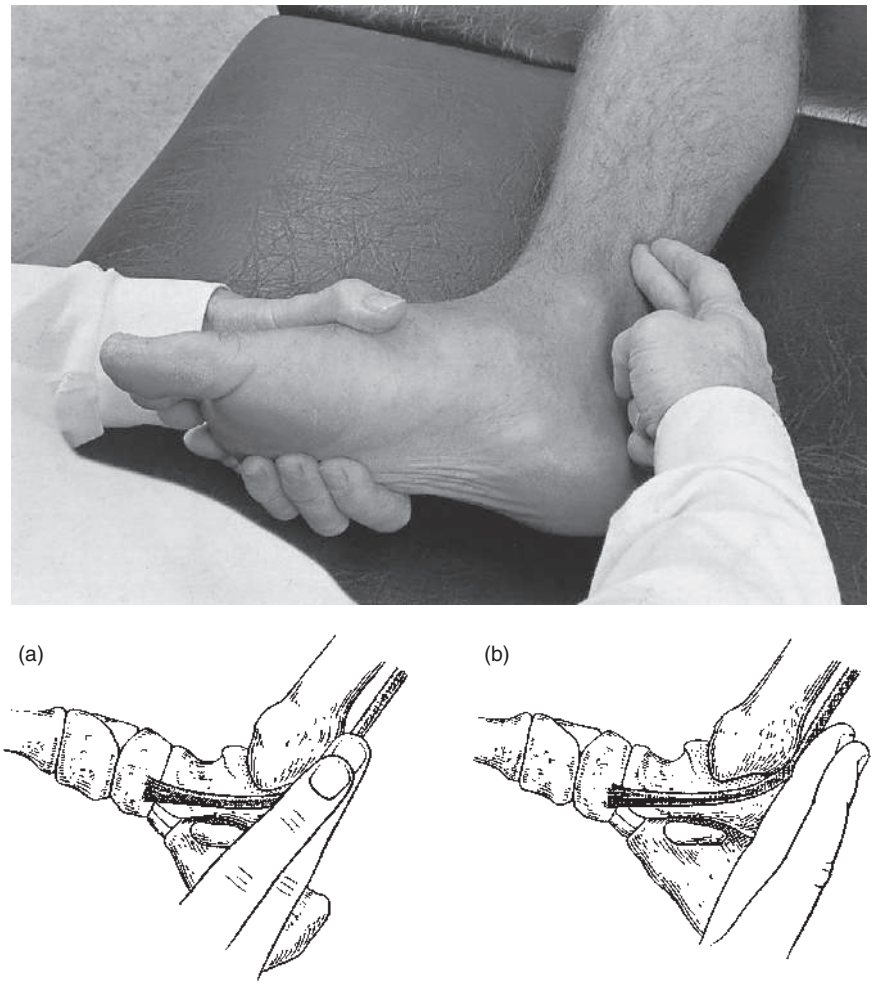


Fig 57.11 • Deep friction to the upper part of the tibialis muscle: (a) starting position; (b) end of the movement.

released a little and the finger returned to the original position by pronation.

This alternating pronation–supination movement is continued for about 20 minutes. Usually 10 sessions, three times a week, suffice to resolve the condition.

Technique: friction to the lower part of tibialis posterior

The patient's foot is dorsiflexed and the therapist sits level with the affected foot. The thumb of the contralateral hand is placed at the lateral side of the foot. The tips of one or two fingers are placed on the affected length of the tendon, just beyond it. Friction is now given with an extension movement at the wrist (Fig. 57.12), using the thumb as a fulcrum. The fingers ride over the whole width of the tendon until the latter escapes from the pressure. Using flexion of the wrist, without losing contact with the skin, the fingers are then moved to the original position behind the tendon, and the whole procedure is repeated.

Friction is continued for 20 minutes, twice a week, for 1 or 2 weeks.

Shin splints

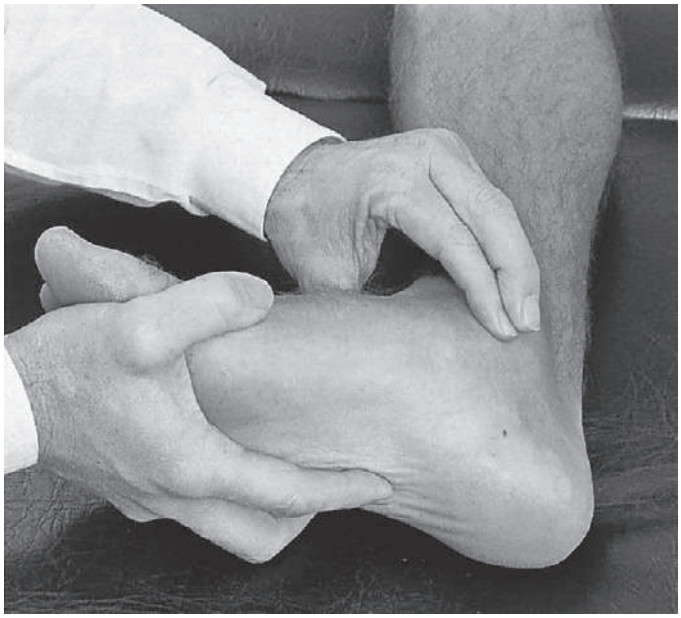
Pain at the posterior medial edge of the tibia in athletes and long-distance runners is called 'shin splints' or 'medial tibial stress syndrome'. Few conditions have caused as much controversy. The term has been used to cover a variety of conditions, such as stress fractures,[146–148] periostitis and soft tissue inflammation.[149] The American Medical Association subcommittee for classification of sports injuries restricts the name shin splints to musculotendinous lesions of the tibialis posterior muscle.[150]

The disorder appears as an overuse phenomenon in runners and is one of the most common causes of exertional leg pain, accounting for 6–16% of all running injuries.[151]

The most common complaint is diffuse pain of the lower extremity, along the middle-distal tibia, associated with exertion. In the early course, pain is worse at the beginning of exercise but gradually subsides during training and within minutes of cessation of exercise. As the injury progresses, however, pain presents with less activity and may occur at rest.[152]

Clinical examination shows little pain during resisted inversion of the foot. Palpation reveals tenderness and sometimes induration at the musculotendinous junction of the tibialis posterior muscle, usually between its upper and medial thirds.

Differential diagnosis must include exertional compartment syndrome and stress fracture. Here, pain is elicited by sharply striking the heel or tapping the anterior tibia, or the *bone* is tender to direct pressure. During the initial weeks, a stress



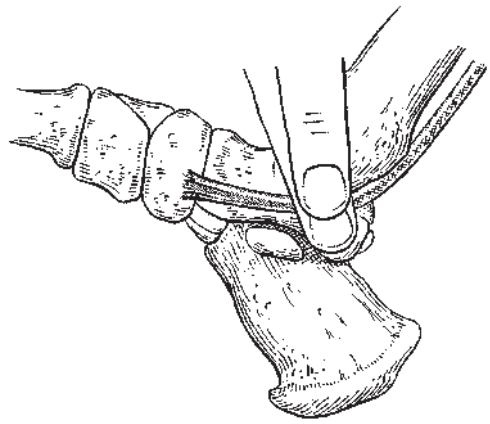


Fig 57.12 • Deep friction to the lower part of the tibialis posterior.

fracture is not visible on a plain radiograph[153] but can be detected with ultrasound.[154] In case of doubt, a bone scintigram can be obtained that easily distinguishes an inflammatory shin splint from a stress fracture.[155,156]

The condition responds effectively to a series of sessions of deep transverse friction. Good results have also been obtained with weekly infiltrations of procaine (10–20 mL) over 3 consecutive weeks.

For a stress fracture, only rest is effective. A 6- to 8-week pause in training is usually necessary.

Differential diagnosis between posterior and anterior leg pain is summarized in Box 57.4.

Weakness

Neurological lesions are discussed at the end of this chapter.

Weak resisted inversion is often caused by a rupture of the tibialis posterior tendon (TPT), secondary to a combination of degenerative changes and trauma.[157] It is usually located in a critical zone with a low degree of vascularity, posterior to the medial malleolus.[158]

Box 57.4

Leg pain: differential diagnosis

Posterior leg pain	Anterior leg pain
• Tennis leg	• Myosynovitis of the tibialis anterior
• Achilles tendinitis at the musculotendinous junction	• Shin splints
• Deep venous thrombosis	• Stress fracture
• Rupture of Baker's cyst	• Tight fascial compartment
• Intermittent claudication	• Lesion of a dorsiflexor muscle belly
• Ischaemic contracture of the calf muscles	• L3, L4 referred pain
• L5–S2 referred pain	

Rupture of the TPT does not initially cause pain and usually the patient cannot recall an acute trauma. The main symptom that draws attention to the possibility is a gradual deformity of the foot. The triad of deformity appearing during weight bearing is:

- Valgus deformity of the heel
- Loss in height of the medial longitudinal arch
- Abduction of the forefoot, which is detected by the 'too many toes sign'[159]: more of the lateral toes are seen on the affected side when the patient is inspected from behind.

Rupture of the tibialis posterior tendon thus leads to a progressive, unilateral, acquired flat foot, with increasing valgus of the heel, plantar flexion of the talus and subluxation of the talonavicular joint.[157,160]

It is difficult to detect weakness of the tibialis posterior during manual testing because the synergistic action of the other invertors of the foot substitutes for the power of the ruptured muscle. Weakness of the tendon is best detected during rising on tiptoe. In a normal foot, this test shows a typical sequence. First the TPT is activated, which inverts the foot and locks the heel and the mid-tarsal joints. Then the triceps muscle pulls on the now-rigid calcaneus, the heel rises and the body weight is imparted to the ball of the foot. With a rupture of the TPT, however, the initial varus movement at the heel is impossible and the patient rises up with the heel in valgus or does not get up on to the ball of the foot at all.[161,162]

Conservative treatment includes a medial heel wedge and a longitudinal arch support.[163]

Surgical treatment must be considered when the patient is young and active. A plantaris or extensor tendon graft may be used to bridge the defect. Recently, the transfer of the flexor digitorum longus tendon into the sheath of the tibialis posterior has been used for larger defects.[164,165]

Lesions of the evertors

Pain

Tendinopathy

If there is pain on resisted eversion of the foot, the peroneal muscles are at fault. Peroneal tendinosis is usually a simple

Fig 57.13 • Deep friction to the peroneal tendons under the malleolus.

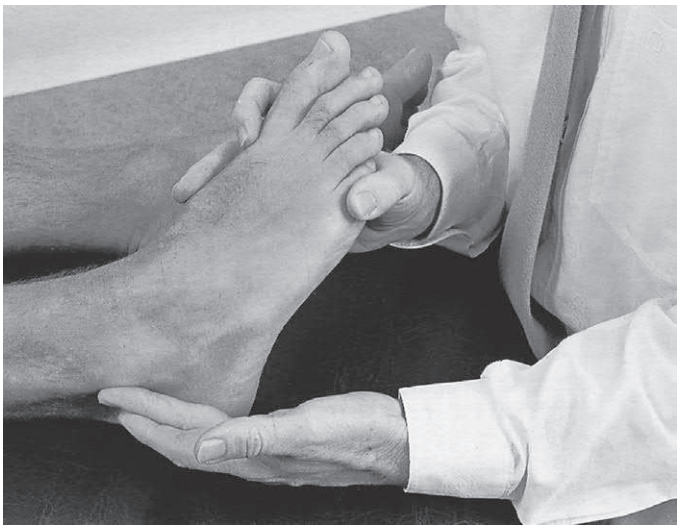


Fig 57.14 • Deep friction to the peroneal tendons level with the malleolus.

overuse phenomenon in runners and skaters,[166,167] although it may also appear as a complication of ankle sprain (see p. 774).[168,169]

As tendinosis occurs anywhere, from the lower fibula to the cuboid and the fifth metatarsal base, the exact position of the lesion can only be disclosed by palpation for tenderness along the whole tendon.

Treatment

Peroneal tendinosis is seldom sufficiently localized to be treated by steroid injection. However, it responds very well to deep transverse massage.

Technique: deep friction above and below the malleolus[170]

The patient lies supine, with the leg medially rotated. The therapist sits at the opposite side. The foot is brought into inversion; this stretches the tendon. Two or three fingertips of the therapist's ipsilateral hand are now placed on the tendon. The fingers are slightly flexed, thereby pressing the tendon against the fibular shaft. The thumb is placed at the medial side of the lower leg or ankle to provide counterpressure (Fig. 57.13). Friction is given with a to-and-fro movement of the forearm, during which the fingers ride over the tendon. The friction is in two phases: an active phase, during which the tendon is pressed and pulled, and a relaxed phase, during which the fingers are placed in the previous position to restart the movement.

Three sessions a week over 2–4 weeks may be required. During the course of treatment, the patient should avoid exercise but complete rest is not required. The results are uniformly good.

Technique: deep friction level with the malleolus

The therapist sits by the patient's foot, facing it. The foot is held in inversion and plantar flexion is produced by slight pressure of the ipsilateral hand. The therapist holds the contralateral hand and forearm in line with the leg. The middle finger, reinforced by the index finger, is placed in the sulcus, behind the peroneus tendon (Fig. 57.14). Friction is started with the finger behind the tendon. Pressure is exerted and, with a supination movement of the forearm, the tendon is pressed anteriorly until it escapes from the finger and slips backwards. The now-supinated hand is manœuvred back to the pronated position behind the tendon, where the movement restarts.

Friction is imparted by rotating the forearm in full supination and full pronation for about 15 minutes. Three sessions a week over 3–4 weeks are usually sufficient to cure the lesion.

Other conditions

'Snapping ankle'

Sometimes patients complain of a slight pain, together with a snapping feeling at the outer side of the ankle, during certain movements. The reason is a loosening of the peroneal tendons in the groove of the posterior surface of the fibula. They slip forwards over the malleolus when the ankle is dorsiflexed and 'jump back' during plantiflexion.[171] This 'snapping ankle' is usually not painful, just uncomfortable. Sometimes, however, it is the origin of a superimposed tendinitis. Non-surgical treatment should always be instituted first and consists of holding the tendons reduced in their anatomical positions while the superior peroneal retinaculum heals by scarring.[172] If considerable discomfort remains, surgery can be advised. Different methods of repair have been described.[173,174] Usually, a new retinaculum is constructed behind the lateral malleolus or the calcaneofibular ligament is transposed to the lateral side of the peroneal tendons.[175]

Mucocele

A mucocele can form in the sheath of the tendon. It results in localized swelling and can be associated with a considerable ache. The diagnosis is obvious because the swelling is located in the sheath and can be made to fluctuate upwards and downwards along the malleolus by digital pressure. Treatment is aspiration. Because the viscous fluid has a high density, the

aspiration is difficult to perform unless a wide-bore needle and a large syringe are used. Sometimes up to 50 mL of fluid can be removed. If there is any pain, 1 or 2 mL of triamcinolone are injected into the now-empty synovial space. Sometimes this procedure has to be repeated after a year or so.

Peroneal spasm

This is not an intrinsic disorder of the peroneal muscles but results from arthritis of the talocalcaneal and mid-tarsal joints. If the arthritis is cured, the spasm disappears immediately.

Weakness

Weakness of the peronei usually results from neurological lesions.

Disorders causing neurological weakness of the foot

Weakness of the calf muscles is best detected by asking the patient to stand on one foot and to rise on tiptoe. Slight weakness can only be identified by repetitive rising on tiptoe, comparing one leg with the other.

Dorsiflexion, eversion and inversion are tested in the supine-lying position. As described in Chapter 55, it is very important to give strong counterpressure, with the leg in a neutral position. Normally, the extrinsic muscles of the foot are very strong and their pull cannot be overcome by the examiner's force. Both sides should be compared.

In central or upper motor neurone lesions, the weakness is gross and distributed over different muscles and muscle groups (Table 57.1).

Table 57.1 Innervation of neurological weakness of the foot

Muscle	Segmental innervation	Peripheral nerve
Tibialis anterior	L4 (L5)	Deep peroneal
Extensor digitorum longus	L4, L5	Deep peroneal
Extensor hallucis longus	L4, L5	Deep peroneal
Peronei	L5, S1	Superficial peroneal
Tibialis posterior	L4, L5	Tibial
Flexor digitorum longus	L5–S1	Tibial
Flexor hallucis longus	L5–S1	Tibial
Triceps surae	S1, S2	Tibial

In lesions of the nerve root (sciatica caused by disc protrusion), the distribution is segmental. There is or there has been considerable pain in the respective dermatomes before the appearance of the weakness. Although weakness can be significant, especially if two consecutive nerve roots are involved, it is almost never complete.

In lesions of a peripheral nerve, weakness follows the normal afferent distribution of the nerve. Very often, the pain appears silently, without previous pain in the innervated area. As the peripheral nerves in the lower limb behave in the same way as a nerve trunk, paraesthesia will only appear when pressure on the nerve is released and not during the actual period of compression (see release phenomenon in Ch. 2).[176,177]

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Disorders of the ankle and subtalar joints

58

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The ankle joint

The ankle is a very simple joint, allowing only plantiflexion–dorsiflexion movement. Normally the foot comes into a straight line with the lower leg during plantiflexion and can be moved to less than a right angle during dorsiflexion (Fig. 58.1).

Capsular pattern

The capsular pattern of the ankle joint is slightly more limitation of plantiflexion than of dorsiflexion (Fig. 58.2). In patients with short calf muscles, however, dorsiflexion ceases before the extreme of the possible articular range is reached, which raises the question of whether limitation is capsular or non-capsular. In such a case, a clinical diagnosis of arthritis at the ankle rests entirely on the end-feel. Limitation of plantiflexion with a hard end-feel indicates arthritis. If full dorsiflexion cannot be reached because of short calf muscles, a softer end-feel is detected.

Rheumatoid conditions

Rheumatoid conditions, so often affecting the other tarsal joints, are not found in the ankle joint. If it does become inflamed, this occurs only after a long evolution of rheumatoid disease. Exceptions are psoriatic arthritis and gout, which are not uncommon at this joint. In acute arthritis without an apparent precipitating cause, a gout attack must always be suspected, especially if the patient is a middle-aged man. Gout attacks the ankle joint in almost 50% of all gout patients.[1] It responds very well to one or two injections with 20 mg of triamcinolone.

Haemarthrosis

Haemarthrosis of the ankle is not uncommon in ankle sprains. It occurs after direct trauma – for example, in soccer players. A capsular pattern at the ankle joint after an inversion sprain or direct trauma always suggests haemarthrosis. Because blood is a strong irritant to cartilage and provokes early arthrosis, it

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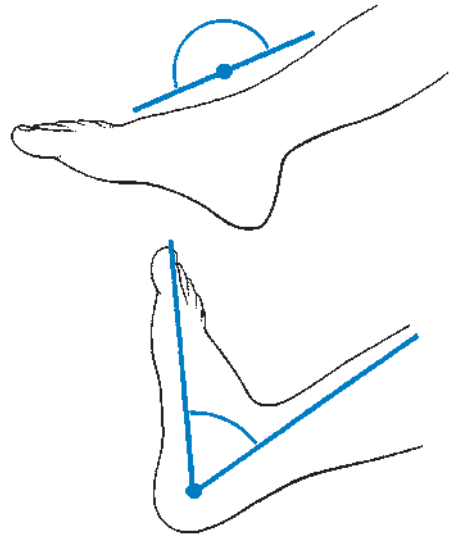


Fig 58.1 • The normal range of plantiflexion (upper) and dorsiflexion (lower).

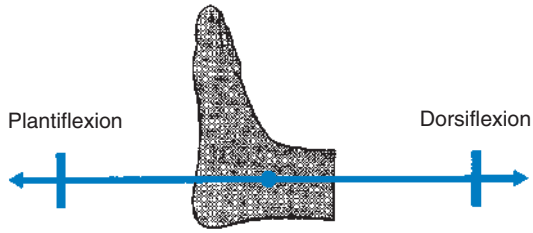


Fig 58.2 • The capsular pattern at the ankle joint.

should be evacuated at once. A radiograph of the talus and/or a magnetic resonance image (MRI) must be taken to exclude osteochondral fracture.[2]

Avascular necrosis

Avascular necrosis is bone death due to ischaemia. Loss of blood supply to the bone can be caused by an injury (trauma-related avascular necrosis), such as ankle sprain, joint dislocation or fracture of the dome of the talus, or can stem from certain risk factors (non-traumatic avascular necrosis), such as some medications (steroids), blood coagulation disorders or alcohol abuse. The earliest clinical manifestation is the finding of a capsular pattern with a spastic end-feel. MRI is the most sensitive technique for detecting talar avascular necrosis and can be used when the condition is strongly suspected clinically despite normal radiographic findings.[3]

Osteoarthrosis

Osteoarthrosis is often the result of shearing strains – for instance, after malunion of a tibiofibular fracture. Early arthrosis has also been reported after aseptic necrosis of the talus.[4] In sports in which repeated and severe sprains of the ankle occur, such as rugby, American football and judo, osteoarthrosis is common and often occurs early. Clinical examination shows a capsular pattern with a hard end-feel. Radiography may show cartilage loss, a flattened talar dome, subchondral sclerosis, intraosseous cysts and peripheral osteophytes.

The best conservative treatment is to fit the patient's shoe with a higher heel, which enables walking without much dorsiflexion at the ankle joint. However, conservative treatment of painful osteoarthrosis is seldom satisfactory. Sometimes one or two injections of 20 mg of triamcinolone may help but should not be repeated too often for fear of further destruction of the joint from steroid arthropathy. During the last decade, the use of visco-supplementation (intra-articular injections of high-molecular-weight solutions of hyaluronan to restore the rheologic properties of the synovial fluid) has been shown to be safe and efficacious in the treatment of osteoarthrosis of the ankle.[5] If the symptoms warrant and the condition worsens, arthrodesis is the only satisfactory treatment and is usually acceptable, provided the patient is fitted with adequate shoes that permit walking without difficulty.

Injection or aspiration technique of the ankle joint

Technique

This is a simple procedure. The patient lies in the supine position, the knee bent and the foot flat on the couch, which forces the ankle into a degree of plantar flexion. The medial malleolus and the tendon of the tibialis anterior are easily identified. The trochlea tali is found by flexing and extending the talus under the tibia. A 4 cm needle is introduced between the medial malleolus and the tibialis anterior tendon, just under the edge of the tibia (Fig. 58.3). The tip lies intra-articularly when it strikes cartilage.

Non-capsular pattern

Immobilizational stiffness

Limitation of both plantiflexion and dorsiflexion often occurs after long-standing immobilization of the ankle. Only strong and daily mobilization of the joint will afford any benefit. Traction and translation techniques can be of great value in the treatment of this post-immobilization stiffness.[6] Some authors report increased motion and pain relief after arthroscopy.[7]

Loose body in the ankle joint

A loose body with an osseous nucleus is well known as a result of transchondral fracture (osteochondritis dissecans) of the dome of the talus. In most cases, the aetiology is inversion sprain.[8,9] The diagnosis is made by radiography or computed tomography (CT), and symptoms may warrant surgery. However, when there is only a loose cartilaginous fragment without an osseous nucleus, radiographs are negative, and the diagnosis must be made almost entirely on the history.

The patient complains mainly of twinges in the ankle during walking. This seems to happen especially when the foot is

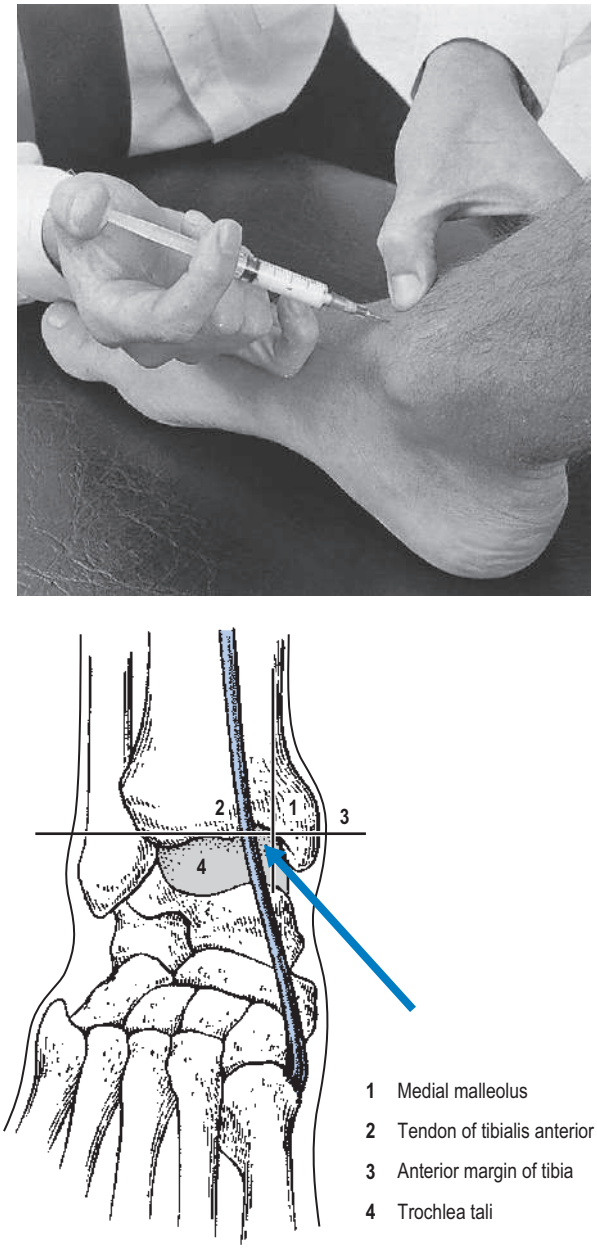


Fig 58.3 • Landmarks for the injection of the ankle joint.

plantiflexed – for instance, on walking downstairs. The twinge is sudden and unexpected, and prevents walking any further. When the foot is shaken, the disability disappears and walking can continue. There may be several twinges on one day or none for weeks. Between the bouts of twinges, no pain or disability is reported. Sometimes the patient states that the symptoms appeared after a severe sprain of the ankle; sometimes no previous trauma is remembered.

Examination reveals nothing because the subluxation is only momentary; nor does the radiograph because the fragment is cartilaginous.

Diagnosis is impossible if the typical history is overlooked. Twinges during plantiflexion of the foot, together with the negative clinical and radiographic findings, should always remind the examiner of the possibility of a loose body in the ankle joint.

Differential diagnosis

The differential diagnosis is between a loose body in the subtalar joint (p. 1214), distal tibiofibular ligament deficiency (p. 1230), a snapping peroneal tendon (p. 759) or an unstable ankle (p. 1229).

Treatment

Treatment is manipulation. The aim is to shift the piece of cartilage into a position within the joint where it can no longer subluxate. This manipulation is performed several times at one session. It is impossible to evaluate the immediate result because the usual clinical criteria, so useful in the assessment of loose bodies in other joints, are completely absent here. The patient is reassessed a week after manipulation, to determine whether the frequency of twinges has changed. If no improvement results and if the diagnosis is maintained, a Root's shoe can be tried; the anterior wedge to the heel enables the patient to walk without the foot reaching full plantiflexion.

Technique: manipulation

The patient lies supine on the couch, the heel *exactly* level with the edge. The patient stretches the arms above the head and an assistant grasps the hands, in order to apply countertraction during the manipulation (Fig. 58.4).

The contralateral hand is placed under the heel. Because this is the fulcrum, it must be protected from the hard edge of the couch by a thick foam-rubber pad. The ipsilateral hand encircles the foot from the medial side in such a way that the fifth metacarpal bone of the manipulator comes into contact with the neck of the patient's talus. The thumb is placed at the plantar side in order to press the foot upwards in slight dorsiflexion. The manipulator now leans back, pulling as hard as possible with the uppermost hand. Levering around the fixed heel, a strong circumduction movement is carried out, clockwise for the right foot and anticlockwise for the left foot. During this manœuvre, the assistant provides countertraction. The circumductory movements are repeated several times during the same session.

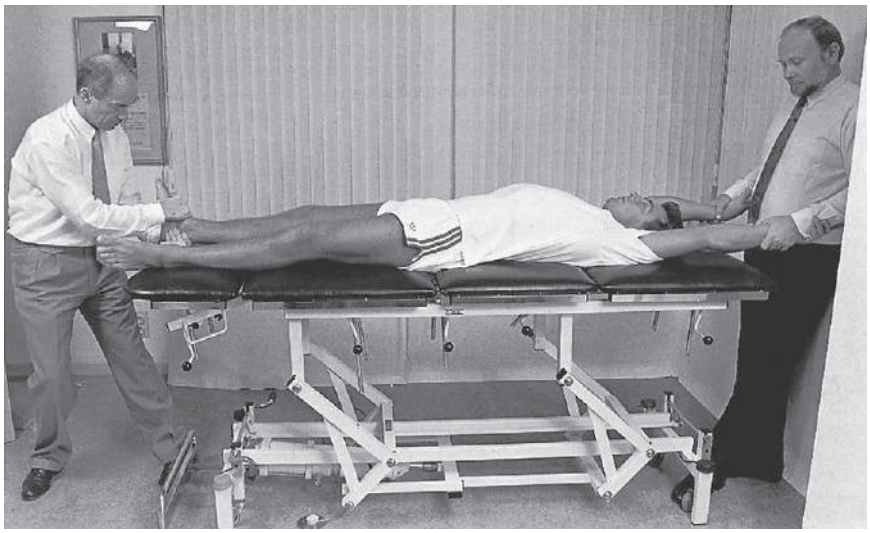
The result cannot be assessed until a week later.

Other lesions with a non-capsular pattern

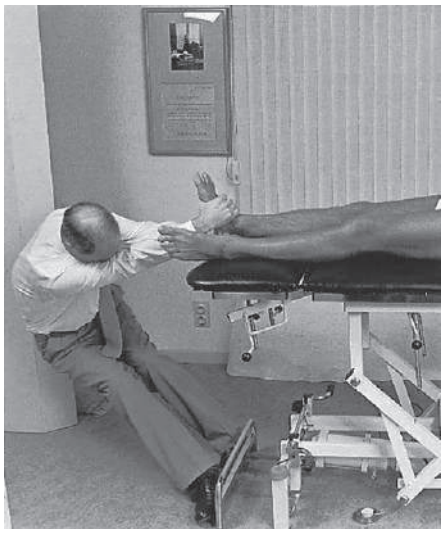
Although plantiflexion and dorsiflexion at the ankle joint show a full range of movement, they can be painful at the end of range, showing that a structure is pinched or stretched.

Sprain of the anterior tibiotalar ligament

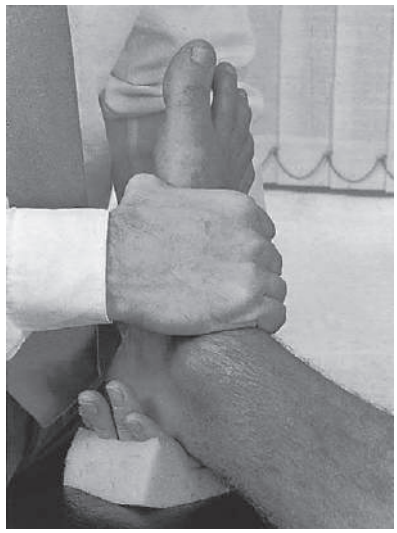
This is an uncommon injury, caused by a pure plantiflexion stress. The chronic aching that results from this type of trauma may last for many years but is never severe, unless the patient is, for example, a rugby or soccer player and needs full and painless plantiflexion mobility at the ankle joint. Kicking a heavy ball from underneath is especially painful. Examination shows that full passive plantiflexion hurts at the front of the



(a)



(b)



(c)

Fig 58.4 • Manipulation for a loose body in the ankle joint: starting position (a), manipulation (b) and detail (c).

ankle, but all the other movements, including resisted dorsiflexion of the toes, are painless. The tender spot at the front of the ankle can easily be defined if the tendons of the dorsiflexors of the toes are pushed away.

The anterior tibiotalar ligament is a very thin structure and therefore is difficult to inject. However, friction is extremely effective in this condition. The main difficulty is pushing the tendons away to reach the thin sheath of tissue joining the talus and the tibia. Three to six sessions of deep transverse friction normally suffice for cure.

In soccer players, new bone may form on the upper surface of the talar neck, as a result of traction at the insertion of the ligament. This has been called 'soccer ankle'.[10] The diagnosis is made from the radiograph. If pain persists, the bone spurs may be removed surgically.

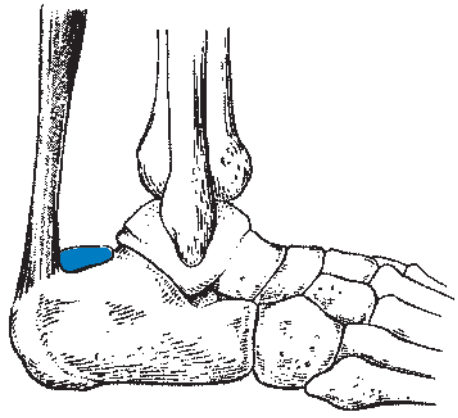


Fig 58.5 • The Achilles bursa.

Achilles bursitis

If the bursa, normally found between the Achilles tendon, the upper surface of the calcaneus and the tibia (Fig. 58.5), becomes inflamed, pain will be elicited when it is squeezed between the posterior side of the tibia and the upper surface of the calcaneus at the extreme of passive plantiflexion.[11,12]

Full plantiflexion evokes pain, this time at the back of the heel. Rising on tiptoe remains negative, thus excluding the Achilles tendon as a cause. Palpation reveals a tender spot anterior to the tendon, close to the superior border of the calcaneus.[13]

Achilles bursitis responds extremely well to one or two injections of triamcinolone. The tender spot is identified, at the lateral or medial side, just in front of the Achilles tendon. Two mL of a 10 mg/mL triamcinolone solution are introduced into the area, following the normal rule of infiltration: over a three-dimensional space and with several insertions and withdrawals. The whole procedure is repeated after 2 weeks if the condition is not completely cured by then.

Dancer's heel (posterior periostitis)

This is a bruising of the periosteum at the back of the lower tibia. The lesion lies at the junction of the cartilage and periosteum, and is caused by pressure from the upper edge of the posterior surface of the talus. It occurs in ballet dancers who, during training, develop a hypermobility in plantiflexion at the ankle joint, usually as a result of *pointe* work. The repetitive engagement of talus against the posterior tibial edge induces periosteal bruising.[14–16] Sometimes the condition results from a single vigorous plantiflexion strain, such as when a soccer player kicks the ball from underneath.

The patient complains of pain at the back of the heel during plantiflexion. Clinical examination reveals an excessive range of movement and pain is reproduced by forced plantiflexion of the ankle. Dancer's heel must be differentiated from Achilles bursitis.[17] In the latter the end-feel is soft, giving the impression of pinching some tissue, whereas in a dancer's heel the end-feel is normal.

The only effective treatment is one or two infiltrations with triamcinolone. This stops the tenderness immediately but the mechanism of the disorder must be explained to the patient so that he or she can take care to avoid the causative trauma. The soccer player has to adopt another technique in kicking the ball from underneath and the dancer must take care not to 'overpoint' the foot.

Technique: infiltration

The patient adopts a prone-lying position, the foot over the edge of the couch. The posterior articular margin of the tibia lies approximately 2 cm above the line joining the tips of the malleoli. A 2 mL syringe is filled with a steroid suspension and fitted with a fine needle, 4 cm long. The Achilles tendon is pushed medially (Fig. 58.6). The needle is inserted vertically downwards, lateral to the Achilles tendon, 2 cm above the line connecting the malleoli. The most difficult part of the whole procedure is now to palpate with the tip of the needle and feel for the line at which bone (tibial periosteum) gives way to articular cartilage. The infiltration is now made by placing a line of little droplets all *along* and just *above* this cartilaginoperiosteal border.

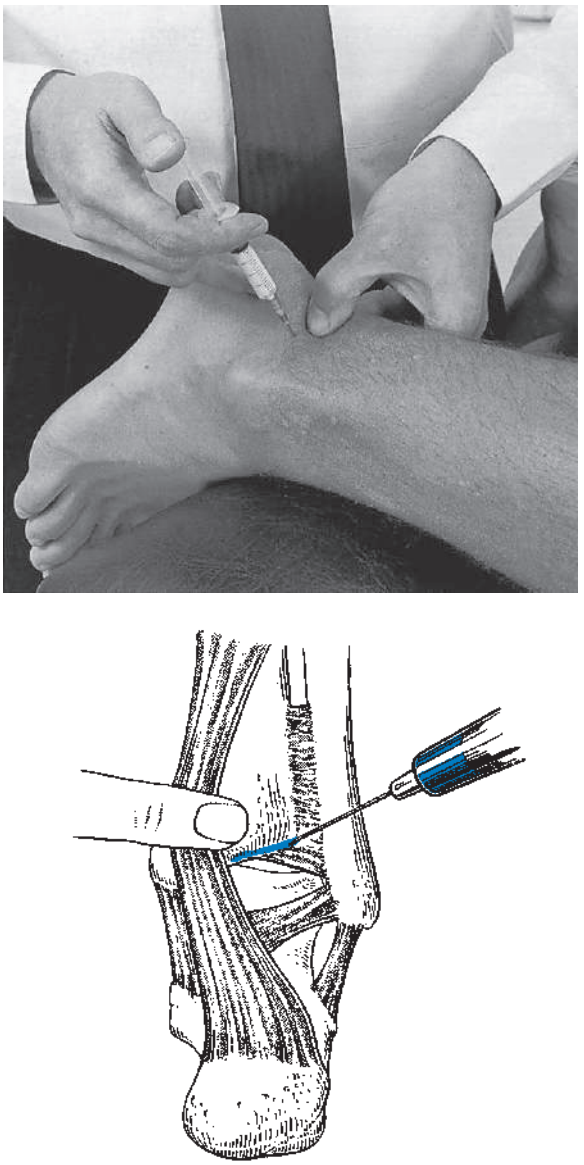


Fig 58.6 • Infiltration of dancer's heel.

Pinching of the os trigonum

Posterior ankle pain during extreme plantiflexion can also be caused by periostitis of the os trigonum. This accessory bone, located just behind the talus, is found in about 10% of the population.[18] Sometimes the ossicle is fused to the talus and is then called Stieda's process. With extreme plantiflexion, such as in ballet or soccer, the os trigonum may be pinched between talus and tibia and produce periostitis and pain.[19] The clinical diagnosis is made when posterior pain during passive plantiflexion is seen in combination with slight limitation of plantiflexion movement and a hard end-feel. Diagnosis can be confirmed by an MRI examination.[20] Sometimes a painful outcrop can be palpated in the posterior triangle.[21] Martin[22] noted not only that this reduced dorsiflexion mobility but that painfully resisted plantiflexion of the big toe was also present. This is caused by fibrosis of the flexor hallucis longus tendon in the fibro-osseous canal behind the talus.

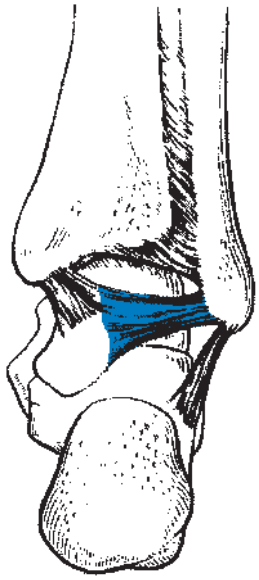


Fig 58.7 • The posterior talofibular ligament.

Fig 58.8 • Accessory test in jumper's sprain.

Treatment is infiltration with triamcinolone. If pain persists, surgical removal can be considered.

Anterior periostitis

The converse of a dancer's heel is periostitis at the anterior margin of the tibia. This is caused by pressure of the anterior lip of the tibia on the talar neck during an extreme dorsiflexion movement at the ankle.[23] The typical situation inducing this injury is when a gymnast lands flat on the feet but with the knees bent so that the ankle is forced into extreme dorsiflexion. The result is immediate pain at the front of the ankle. The sharp component of the pain disappears but the lesion does not heal completely, leaving the patient with pain during extreme dorsiflexion movements. In ballet dancers, repeated and extreme dorsiflexion necessitated by the *demi-plié* position can lead to periostitis of the anterior tibial lip.[24,25]

Examination shows a full range of movement with pain at the front of the ankle during extreme dorsiflexion. In mild cases, pain will be evoked only when the foot is dorsiflexed during weight bearing (e.g. squat with the feet flat on the ground).

Treatment is one infiltration with triamcinolone, along the anterior tibial margin. This is within the reach of a palpating finger and therefore the infiltration is easy to perform. The results are good. In recurrent cases, the patient is referred for arthroscopic removal of the bony impingement.[26]

Sprain of the posterior talofibular ligament

Sprain of the posterior talofibular ligament (Fig. 58.7) is rare. The diagnosis is difficult to make if the examiner is not aware of the possibility of this lesion being present. The only painful movement during the routine functional examination is passive eversion of the foot during full plantiflexion – a movement performed to test the anterior fasciculus of the deltoid ligament. If the pain is posterolateral instead of anteromedial, it is obvious that a tissue is being pinched rather than stretched and the condition can be considered.

The treatment of choice is an injection of triamcinolone into the tender ligament; one injection gives lasting relief. Although deep transverse friction can be used, it is very difficult to reach the lesion with the tip of a finger.

Jumper's sprain (lateral periostitis)

This is one of the classic lesions sustained by high jumpers. Before the athlete takes off to jump, the foot is forcefully twisted in valgus and dorsiflexion. Apart from lesions at the inner side of the ankle (strain of the deltoid ligament and elongation of the tibialis posterior tendon), compression at the outer side can result. During this extreme movement, the superolateral aspect of the anterior margin of the calcaneus can impinge against the inferior and anterior edge of the fibula and produce bruising, which results in traumatic periostitis.[27] Sometimes the impingement leads to chronic inflammation of the talofibular ligament, resulting in hypertrophic scar tissue.[28]

Examination reveals nothing if only the standard functional tests are performed. When the possibility of this lesion is suspected, combined dorsiflexion–valgus movement is performed to reproduce the pain (Fig. 58.8). If this manual stress is not sufficient to elicit the usual pain, the patient is asked to stand, squat with the foot flat on the ground and twist the heel into valgus. Palpation reveals localized tenderness at the anterioinferior surface of the fibula.

One or two injections of triamcinolone bring total relief, provided the athlete avoids sustaining the same trauma. Normally, a slight inner wedge (0.5 cm) within the shoe is needed, which prevents further bruising of the fibula during 'take-off'. Those patients refractory to conservative treatment require arthroscopic debridement.[29,30]

Disorders of the ankle joint are summarized in Table 58.1.

Table 58.1 Summary of disorders of the ankle joint

	Lesion	Symptoms	Signs	Treatment
Capsular pattern	Haemarthrosis	Trauma Nocturnal pain	Capsular pattern Warmth/fluid	Aspiration
	Rheumatic arthritis	Pain/morning stiffness	Capsular pattern	Triamcinolone
	Osteoarthrosis	Pain on weight bearing	Capsular pattern Crepitus	Mobilization Higher heel Arthrodesis
Non-capsular pattern	Immobilizational stiffness	After plaster immobilization	Limitation of plantiflexion–dorsiflexion	Mobilization
	Loose body	Twinges	Full range/no pain	Manipulation
	Sprain of the anterior tibiotalar ligament	Anterior pain	Pain at full plantiflexion	Deep friction
	Achilles bursitis	Posterior pain	Pain at full plantiflexion Soft end-feel	Triamcinolone
	Dancer's heel	Posterior pain	Pain at full plantiflexion Hypermobility	Triamcinolone
	Periostitis os trigonum	Posterior pain	Pain/limitation of plantiflexion	Triamcinolone
	Anterior periostitis	Anterior pain	Pain at full dorsiflexion	Triamcinolone
	Sprain of posterior talofibular ligament	Posterolateral pain	Pain at full plantiflexion and eversion	Triamcinolone Surgery
	Jumper's sprain	Anterolateral pain	Pain at full dorsiflexion and eversion	Triamcinolone

The subtalar (talocalcaneal) joint

The subtalar joint allows movement in two directions only: varus and valgus. Motion takes place around an axis through the talus (Fig. 58.9), the axis being at a 15° medial angle to a line drawn through the calcaneus and the second metatarsal.

Capsular pattern

The capsular pattern (Fig. 58.10) is progressive limitation of varus with, eventually, fixation in valgus. The valgus position is maintained by spasm of the peronei muscles.

Rheumatoid disorders

In addition to the limitation of movement towards varus by muscle spasm, local heat is present and synovial thickening can be palpated. Very often, the midtarsal joint is affected as well. In rheumatoid arthritis, the arthritis is often accompanied by characteristic changes in other joints. The possibility of early ankylosing spondylitis should be kept in mind when a young patient presents with arthritis of the subtalar joint. An early manifestation of arthritis in the subtalar and midtarsal joints is also a common finding in juvenile idiopathic arthritis.[31,32] In the case of an acute joint inflammation, gout should not be forgotten.

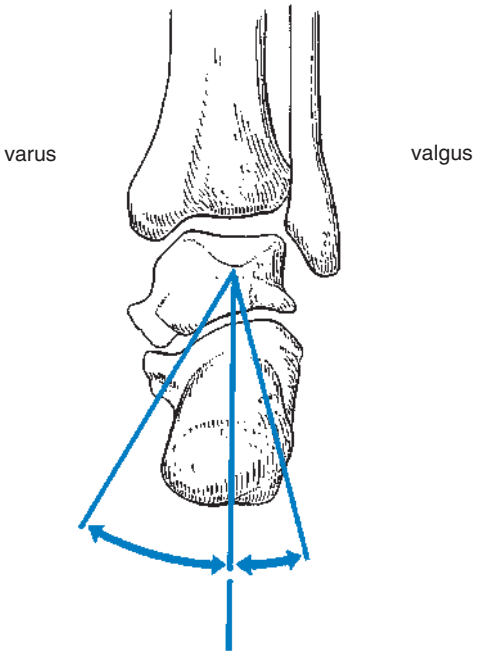


Fig 58.9 • Normal varus and valgus movement at the subtalar joint.

Treatment

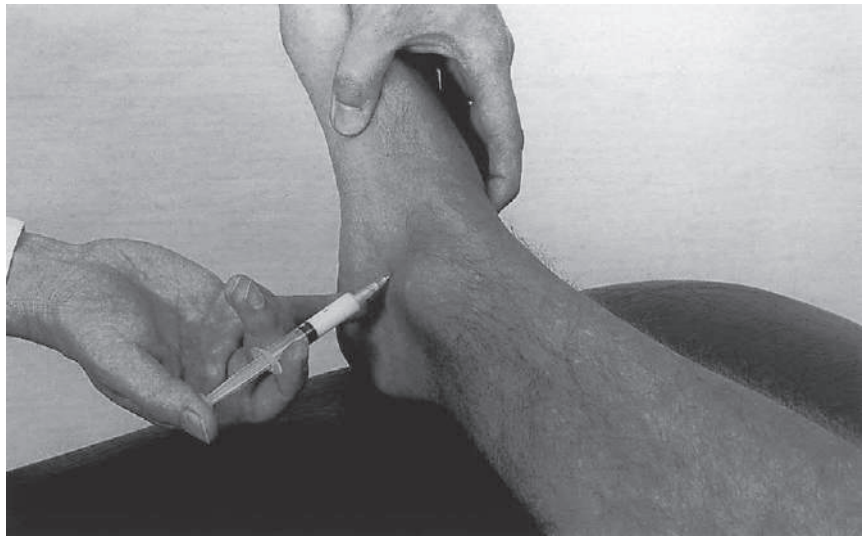
The cause of rheumatoid disorders should be treated. In addition, triamcinolone injected into the joint relieves the pain very

quickly and, even if the range of movement does not increase, the patient can enjoy some comfort for months or even years. If the pain reappears after a short interval, it is not wise to continue the injections.

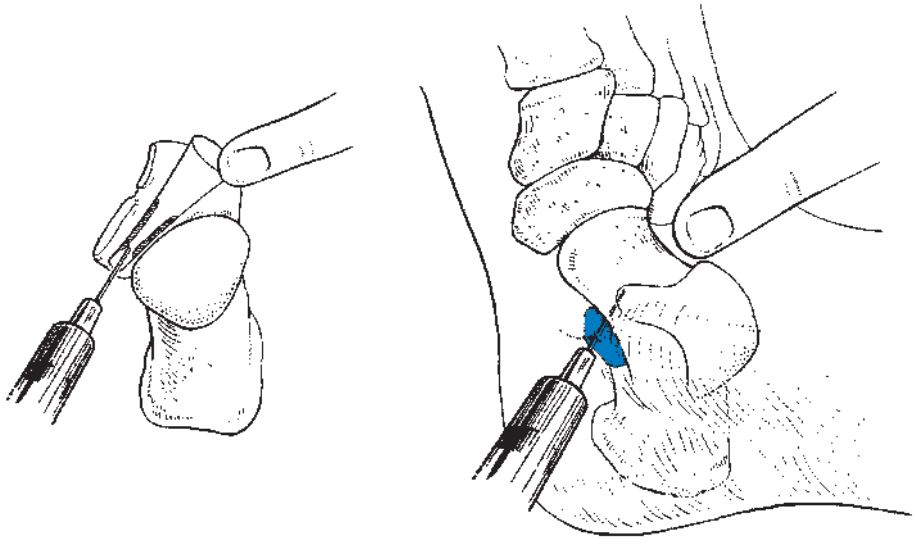
Fig 58.10 • The capsular pattern at the subtalar joint.

Technique: injection

A 2 mL syringe is filled with steroid suspension and fitted with a thin 2 cm needle. As there may be muscle spasm, the joint is fixed in valgus to create room to insert the needle from the medial side, which must be done just above the sustentaculum tali and parallel to the joint surface. The index finger of the palpating hand is placed at the lateral end of the sinus tarsi (Fig. 58.11). The needle is moved in the direction of and slightly anterior to the palpating finger. Usually it meets bone after 1 cm. The needle must then be manœuvred until it is felt to slip in further without resistance. The tip then lies within the anterior chamber of the joint, and 1 mL of the suspension is injected. The needle is then partly withdrawn and reinserted in a 45° posterior direction, where it enters the posterior chamber, and the remaining 1 mL is injected.



(a)



(b)

Fig 58.11 • Injection of the subtalar joint (a). Direction of the needle in relation to the palpating finger in the lateral opening of the sinus tarsi (b).

Subacute traumatic arthritis

Sometimes recovery after a serious varus sprain at the ankle is unduly delayed by pain and limitation of movement in the ankle and midfoot. Examination reveals that the limitation of varus movement is caused by spasm of the peroneal muscles. Usually, the midtarsal joint is also affected. Palpation sometimes reveals warmth over the subtalar and midtarsal joints. Untreated, this condition can last for months or even years. Very often, a wrong diagnosis of post-traumatic adhesions is made. If the limitation of varus movement at the talocalcaneal joint, together with the muscle spasm and the warmth, are missed, such patients will probably be treated by mobilization, or even manipulation, which aggravates the condition.

Patients suffering from this condition are often regarded as mentally unstable but it should be remembered that, in psychological disorders with projection to the foot, the heel is *always* fixed in varus, not in valgus.

Treatment consists of two injections of 20 mg triamcinolone into the joint, 2 weeks apart. If this treatment fails, the joint is immobilized for several months in plaster, which is done in as much varus position as possible, sometimes after blocking the peroneal nerve.

Osteoarthrosis

This is a common disorder after an intra-articular fracture of the calcaneus. Diagnosis is made when a typical hard end-feel is detected, in the context of a previous fracture.

The pain is incurable by conservative measures, including intra-articular injections. Persistent pain after an intra-articular fracture can only be relieved by arthrodesis.

Spasmodic pes planus

This is discussed on page 1292.

Non-capsular pattern

Immobilizational stiffness

Marked limitation of movement at the talocalcaneal joint can result from plaster immobilization for tibiofibular fractures. There is a stiff joint, with an equal limitation of varus and valgus but no muscle spasm. The joint is in mid-position, whereas in arthritis it fixes in valgus. Capsular thickening and warmth are absent.

Treatment consists of manual mobilization, which is technically difficult, for there is no lever, and the small size of the calcaneus affords very little purchase. Sometimes many months of repeated forcing will be necessary. Restoration of a full range is not always essential because slight limitation of range of movement is still compatible with good function.

Technique: mobilization

The patient lies face upwards on the couch. The therapist stands at the patient's foot. The fingers are clasped behind the heel and the calcaneus is grasped as strongly as possible between the palms of the hands. The elbows are brought forwards in order to dorsiflex the foot. This position (Fig. 58.12) immobilizes the talus in the mortice. Mobilization is performed by swinging the body from one side to the other. This forcing must be repeated for 10–20 minutes at each session, with the greatest possible vigour.

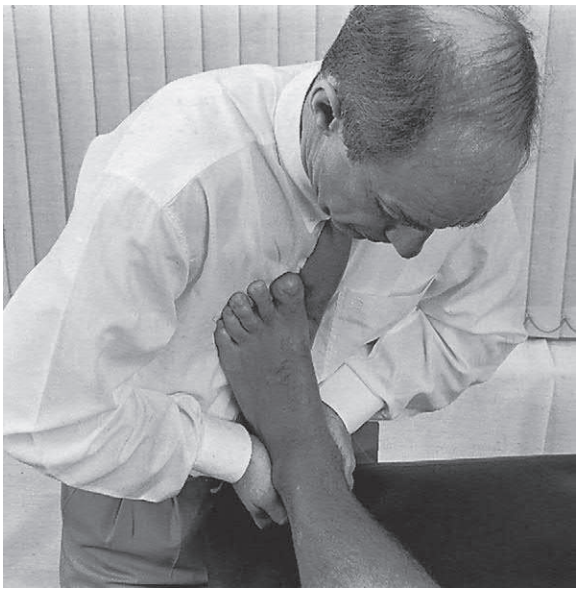


Fig 58.12 • Mobilization for immobilizational stiffness of the subtalar joint.

Loose body

The patient experiences painful twinges, usually provoked by walking on uneven surfaces, which may suggest instability of the ankle or an unstable mortice. Alternatively, the patient may have sudden attacks of painful fixation in valgus, provoked by a spasm of the peronei muscles.

Clinical findings depend on when the examination is undertaken. If the patient has twinges only during certain movements, clinical examination is negative. If the patient presents during an attack of fixation, the typical valgus deformity and muscle spasm are found. Differential diagnosis from an articular disorder then depends entirely on the history because, in the case of a loose body, the patient states that pain and fixation are intermittent rather than permanent.

If impaction of a loose body in the subtalar joint is suggested by a history of twinges or if repeated attacks of sudden fixation in valgus are mentioned, reduction must be attempted at once. Very often, it succeeds immediately but the cartilage fragment is seldom moved permanently into a position from which it no longer subluxates. It is therefore wise to repeat the manipulation several times at intervals of a few days. If no permanent relief is obtained and the signs warrant it, arthrodesis is advised.

Technique: manipulation

The patient lies prone, pulling himself or herself upwards at the upper edge of the couch until the dorsum of the foot engages the lower edge. This forces the foot into slight

plantiflexion. The manipulator stands behind the patient and locks both hands around the heel, so that the crossed fingers are placed between the dorsum of the foot and the edge of the couch. The fingers are protected by a thick layer of foam. The thumbs are crossed at the dorsum of the calcaneus. In order to exert the utmost possible traction, the feet are placed against the legs of the couch and the body leans backwards. The elbows stay in line with the calcaneus, the abdomen close to the patient's foot (Fig. 58.13). The traction produced by the body weight is reinforced by a pronation movement of both forearms. Varus–valgus movements are forced at the joint by repeatedly swinging the shoulders from one side to the other. During the whole procedure, the patient is told to maintain the pulling position and not to allow any downward movement of the body.

Disorders of the subtalar joint are summarized in Table 58.2.

Painful conditions at the heel

Sometimes the patient has a clear history of pain at or about the heel but there are no findings on clinical examination. If the complaints consist of twinges, attention is drawn to the possibility of a loose body either in the ankle joint or in the subtalar joint. If mention is made of a feeling of giving way, instability of the ankle should be suspected. The possibility of referred pain from an S1 structure (S1 nerve root or sacroiliac joint) should also be kept in mind. However, if the patient has constant pain during standing and walking but there are no signs on clinical examination, pinching of inflamed tissue underneath the heel can be the cause.

Plantar fasciitis

Plantar fasciitis is most commonly a disorder of middle age, and men and women are affected equally. Risk factors include obesity and spending prolonged periods standing or walking, particularly on hard floors.[33] It is also more common among middle-aged athletes, in whom it accounts for about 10% of running injuries.[34] The lesion is usually an overuse phenomenon, occurring in the presence of predisposing anatomical, biomechanical or environmental factors that put too much strain on the plantar fascia.[35,36] The condition seems to be more common in people with a valgus deformity, because this flattens the foot and puts more strain on the fascia.[37] Short calf muscles can also be the cause of an overstrained fascia. In this

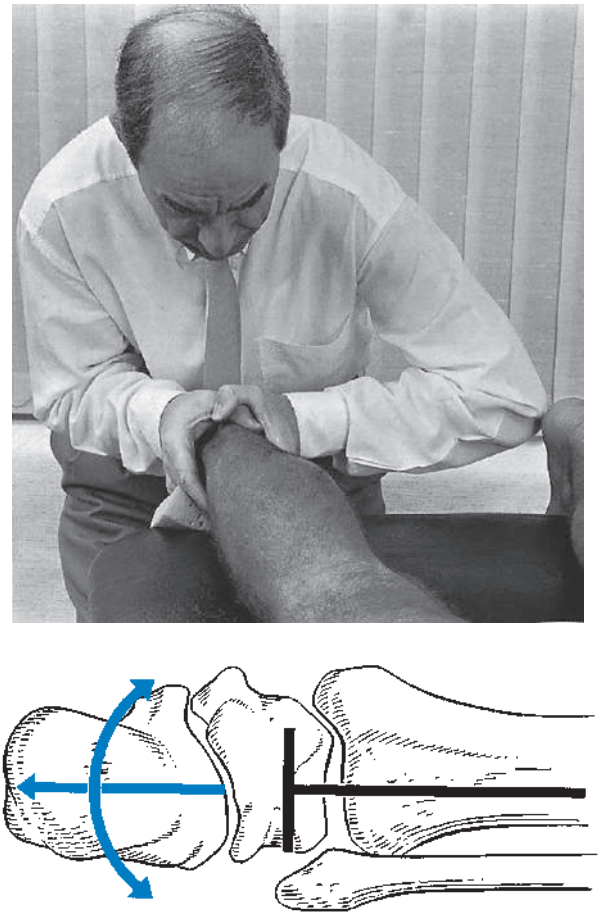


Fig 58.13 • Manipulation for a loose body in the subtalar joint.

Table 58.2 Summary of disorders of the subtalar joint

	Lesion	Symptoms	Signs	Treatment
Capsular pattern	Rheumatoid arthritis	Slow onset Other localizations	Warmth Capsular thickening Muscle spasm	Triamcinolone
	Osteoarthrosis	After fracture	Hard end-feel	Arthrodesis
	Subacute traumatic arthritis	Previous trauma	Muscle spasm Warmth	Triamcinolone Immobilization
	Spasmodic pes planus	Young person Little pain	Muscle spasm and midtarsal localization	Relief of weight bearing and support
Non-capsular pattern	Immobilizational stiffness	Plaster immobilization	Limitation of varus/valgus	Mobilization
	Loose body	Twinges or fixation	Nothing or capsular pattern	Manipulation
	Psychoneurosis		Fixation in varus	

condition, the Achilles tendon tends to pull the heel upwards during standing, which stresses the longitudinal arch and the fascia.[38]

The diagnosis is relatively easy to establish because of the typical presentation. A patient suffering from an overstrained plantar fascia complains of localized pain at the inner aspect of the sole during weight bearing. The first steps taken after sitting or lying are especially painful.

Functional examination of the foot and the ankle is negative. The only positive sign is the detection of a point of deep tenderness, usually situated at the anteromedial portion of the calcaneus – the origin of the plantar fascia. Exceptionally, the tenderness is not at the tenoperiosteal junction but in the body of the fascia, between its origin on the calcaneus and the forefoot. Ultrasound examination can objectively confirm the clinical diagnosis[39–41] but is usually not needed.

Traction spurs, projecting forwards at the anterior border of the calcaneus, are commonly seen on radiographs and traditionally have been implicated as the cause of the painful heel.[42] However, there is no relation between the spur and pain. The cause of the pain is the plantar fascial tendinitis resulting from excessive tension. The presence of a spur does not determine whether or not the patient has symptoms because a spur is very often not found in patients with obvious signs and symptoms of plantar fasciitis. Therefore a radiograph is of no particular assistance in the diagnosis of plantar fasciitis.

Treatment

The classic conservative treatment methods range from application of a heel cup, heel cushion, night splints, walking cast and steroid injection to rest, ice and anti-inflammatory drugs.[43–45] Recently, extracorporeal shock wave therapy (ESWT) has been advocated for treatment of this condition. While the first placebo-controlled trials of ESWT in chronic plantar fasciitis reported benefit of variable magnitude,[46,47] later studies concluded that shock-wave treatment was no more effective than conventional physiotherapy when evaluated 3 months after the end of treatment.[48,49] Another study showed that treatment with corticosteroid injections was more efficacious and several times more cost-effective than ESWT in the treatment of plantar fasciitis.[50]

We have found the combination of alleviating the strain on the plantar fascia and one or two localized infiltrations with triamcinolone to be effective in almost every case of plantar fasciitis. The most important measure to alleviate tension on the plantar fascia is to raise the heel horizontally by 5–10 mm, which will drop the forefoot during weight bearing. This has a double effect: first, it shortens the distance between metatarsus and calcaneus and therefore directly relieves the fascia of strain; second, it removes the tension on the Achilles tendon and therefore indirectly relaxes the tension on the fascia. A high heel can afford immediate relief, provided the upper surface is horizontal and not wedge-shaped, as is the case in women's shoes (Fig. 58.14); in the latter, a wedge that is thicker anteriorly is placed in the shoe to render the upper surface of the heel horizontal.

Sometimes this simple orthotic measure is not enough, and triamcinolone must be injected into the inflamed tissue.

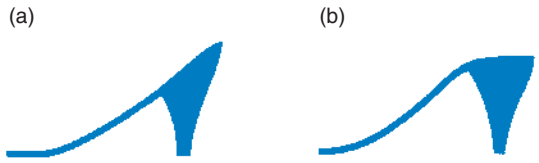


Fig 58.14 • The upper surface of the shoe heel should not be sloping (a) but should be flat (b).

However, this must always be followed by use of a raised heel. Strengthening of the short plantiflexor muscles also affords good active protection against further overstretching of the plantar fascia.

Results of the infiltration depend entirely on its accuracy. It is extremely important to localize exactly the site and the extent of the lesion before the needle is introduced. Palpation and infiltration should therefore be done with great care. Some authors even suggest placing the needle under ultrasound guidance,[51,52] although this is seldom really necessary.

In the exceptional case when conservative treatment fails, the patient is sent for operative plantar fascia release. The results in terms of symptomatic relief are generally good.[53]

If an abnormal valgus position of the heel is present, a small inner wedge should be built in as well.[54,55]

Technique: injection

The patient lies prone on the couch, the knee flexed to a right angle. The therapist stands level with the foot. One hand encircles the heel, while the thumb is placed on the painful spot. A 2 mL syringe is filled with triamcinolone and a needle (4–5 cm long) is fitted to it. A point is chosen along the medial border of the fascia, about 3 cm distal to the lesion. The reason for inserting the needle so far away from the lesion is that the skin overlying the tender spot is too thick to be sterilized. Furthermore, if an oblique approach is made, the needle is thrust in, in the same direction as the fascial fibres, and points directly at the tenoperiosteal junction.

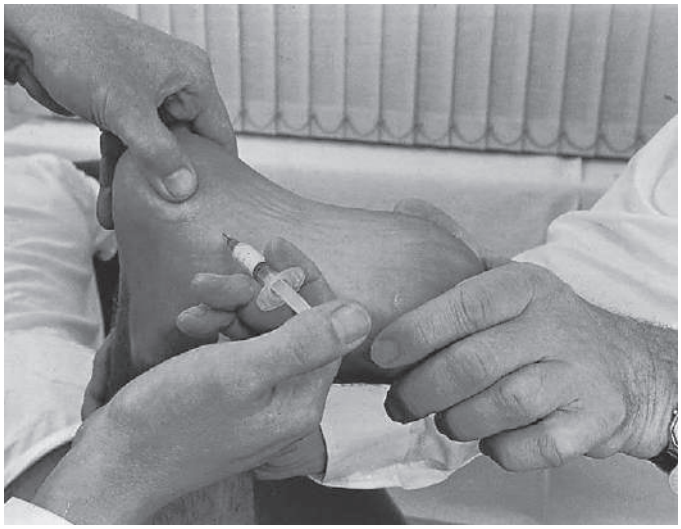
The patient's foot is held in dorsiflexion, either with the dorsum of the injecting hand or by an assistant. This position renders the plantar fascia taut and creates more room for the needle, which aims towards the palpating thumb on the tender spot. After traversing the resistant fascia, it touches bone (Fig. 58.15). The affected area at the tenoperiosteal border is now infiltrated.

This is a very painful injection. The severe pain will last 24–48 hours and the patient must be warned that it may be impossible to stand or to walk during that time. The patient is re-examined after 14 days. If the condition has not completely resolved, a second injection is given. Combined with a raised heel, the results of the injection are uniformly good.

Alternatively, tenotomy of the fascial origin at the heel under local anaesthesia may be required.[56] This minor operation is followed by a couple of days' bed rest and exercises for the short plantiflexor muscles of the foot.

Plantar fascial tear

Like a ruptured Achilles tendon, a plantar fascial tear occurs mostly in middle-aged athletes.[57] The presentation is sudden



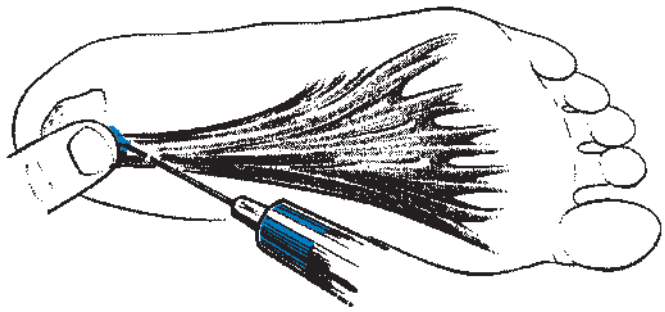


Fig 58.15 • Injection in plantar fasciitis.

pain in the midfoot during a sprint or a jump. There is an area of ecchymosis on the sole.[58] Palpation reveals a tender and swollen area at the medial plantar aspect of the foot.[59]

The immediate treatment is reduction of the haematoma and swelling by ice and elevation. Strapping, together with strengthening exercises for the foot muscles, is given to support the medial arch. Deep friction to prevent adherent and painful scarring is applied as soon as possible. Full recovery occurs in 3–4 weeks.

Heel pad syndrome

Inflammation of the heel pad between the calcaneus and the skin of the heel is also called superficial plantar fasciitis.[60] The heel pad (Fig. 58.16) consists of fatty tissue and elastic fibrous tissue, enclosed within compartments formed by fibrous septa; these connect the skin of the heel with the calcaneal periosteum. The fat pad acts as a shock absorber.[61] It can become inflamed after a direct blow or repeated minor injuries.[62] The pain is felt all over the posterior part of the sole, especially during weight bearing.

Examination shows nothing in particular except uniform tenderness over the whole inferior surface of the heel. It was recently demonstrated that the affected heel pad in plantar heel pain syndrome was stiffer under light pressure than the heel pad on the painless side, and it was hypothesized that this

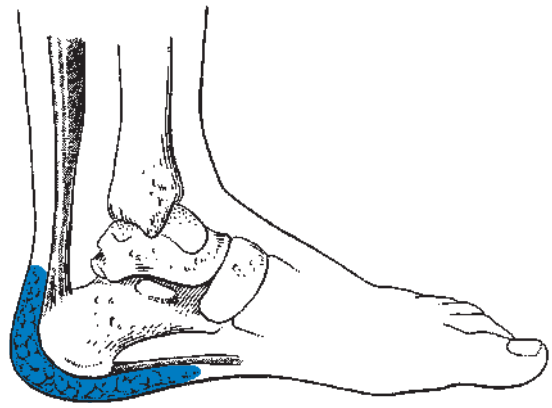


Fig 58.16 • Localization of tenderness in heel pad syndrome.

was caused by the changed nature of chambered adipose tissue.[63]

Treatment

The measures that are so effective in treating plantar fasciitis, such as raising the heel and injecting steroid, are of no value in this condition. However, injection of 10 mL of a local anaesthetic into the pad between the surface of the calcaneus and the superficial fascia is effective.

Technique: injection

The patient lies prone, with the knee flexed at a right angle. The physician stands at the foot and encircles the heel with one hand. A 10 mL syringe is filled with procaine 0.5% and fitted to a needle 5 cm long. The needle is thrust in horizontally between calcaneus and skin (Fig. 58.17). The tip of the needle is then pushed in for some centimetres until it lies at the centre of the heel. The solution is injected there and diffuses over the whole area, forming a large, tense swelling. Significant pressure is needed to force in the last millilitre.

The condition starts to improve after a few days. The patient should have another injection a week later, if necessary. It is astonishing how a couple of injections with anaesthetic cure patients who have suffered months or years of persistent and intractable heel pain.

Subcutaneous bursitis

There is no anatomical bursa between the posterior aspect of the calcaneus and the skin, but in some circumstances a bursa may form, particularly when narrow and ill-fitting shoes are worn, and especially if they are curved in at the upper posterior edge.[64] Friction of the hard border against the calcaneus results in an adventitious bursa. Chronic irritation will thicken the walls of the bursa and also the overlying skin. Palpation reveals a very tender spot at the posterior and upper surface of the calcaneus or at the lower extent of the Achilles tendon. The bursa is usually visibly inflamed and may contain some fluid. An excessive prominence of the bursal projection on the posterosuperior aspect of the calcaneus, in combination with a swollen and painful bursa, is called Haglund's disease.[65]

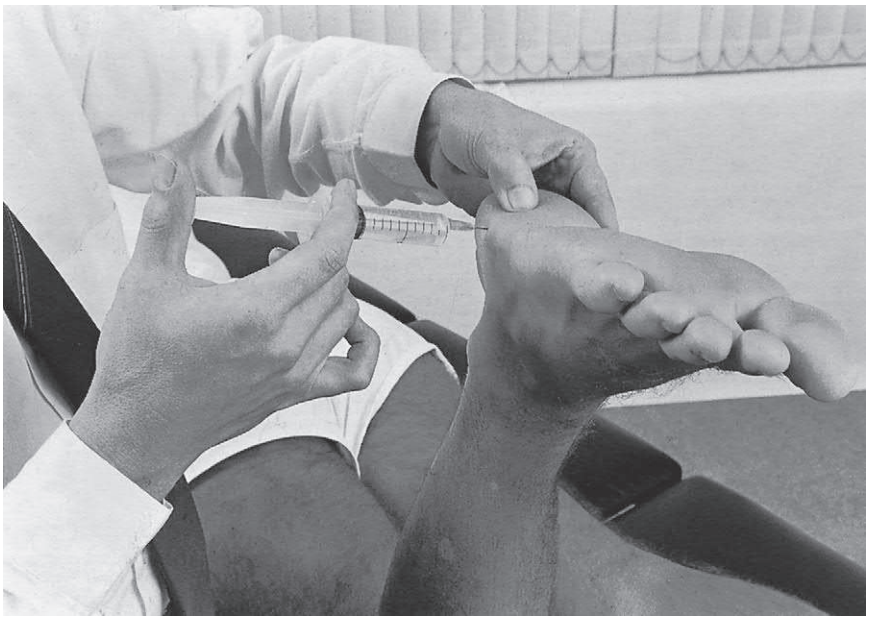


Fig 58.17 • Injection in heel pad syndrome.

The initial treatment is to alter the back of the shoe and introduce a rubber pad at the lower half of the back of the calcaneus, which keeps the upper half away from the pressing edge. If this does not succeed, the bursa can be drained by aspiration, followed by infiltration of 10 mg of triamcinolone. If such conservative treatment does not succeed, excision may be advised. The results of surgery are satisfactory, provided adequate bone has been resected.[66,67]

Subcutaneous nodules

At the posterior aspect of the calcaneus, nodules may form in the subcutaneous fascia and can cause severe pain when they are pinched between the calcaneus and the back of the shoe. Examination reveals small, tender nodules at the lower posterior border of the heel. They are the size of a rice grain and can be felt slipping to and fro under the palpating finger. Sometimes only one nodule can be palpated.

The initial approach to treatment is to provide shoes with a gap posteriorly, although this is not always acceptable to the patient. Division of the nodules by subcutaneous tenotomy under local anaesthesia gives very good results and is easy to perform.

Ligamentous disorders – ankle sprains

'Sprained ankle' is the general name for a variety of traumatic lesions to the posterior segment of the foot. It is a very common sports injury. Several conditions are so described, varying from a simple strain of the ligaments to avulsion fractures and fracture–dislocations. Sometimes only one structure is injured, and sometimes several.[68]

Box 58.1

Classification of ankle sprains according to the site of the lesion*

1. Fibular origin of anterior talofibular ligament
2. Fibular origin of anterior calcaneofibular ligament
3. Talar insertion of anterior talofibular ligament
4. Lateral fibres of calcaneocuboid ligament
5. Peroneal tendons
6. Anterior tibiotalar ligament
7. Tendons of extensor digitorum longus
8. Ligaments of cuboid–fifth metatarsal joint and cuboid–fourth metatarsal joint

*In order of descending frequency[69,70] – see Fig. 58.18.

In this book, there will be no discussion of bony lesions or of fracture–dislocations. However, it is important not to miss these during a routine clinical examination. The diagnosis should then be confirmed by radiography.

Sprained ankles have been classified according to the causative stress (varus–valgus), the tissue damaged (ligament, tendon or bone) or the degree of damage (grade I, II or III) and the time elapsed since the causative accident (acute, subacute or chronic) (Box 58.1 and Tables 58.3 and 58.4).

The severity of a sprain involving a ligament is usually expressed in grades: grade I – slight overstretching and elongation of the fibres without macroscopic disruption of their integrity; grade II – a severe sprain with a partial rupture of the ligament; and grade III – total rupture. Clinically, the degree of damage is always hard to evaluate, especially shortly after injury. Even technical investigations, such as stress radiographs, arthrograms and tenograms, do not always provide an accurate diagnosis. Therefore a classification into grades is not used in this book.

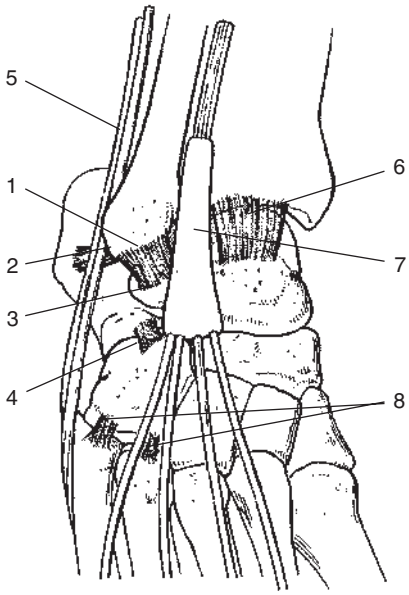


Fig 58.18 • Sites of inversion sprain – see Box 58.1 for details.

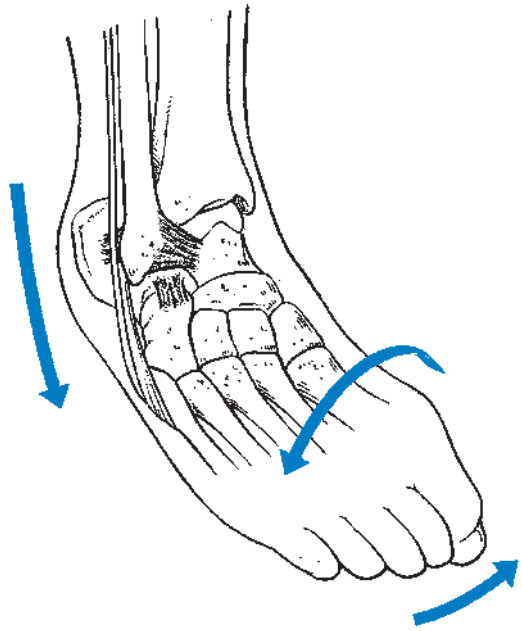


Fig 58.19 • Mechanism of inversion sprains: varus and supination in increasing plantiflexion.

Table 58.3 Classification of ankle sprain according to time since accident

Stage	Time
I Traumatic inflammation	24–48 hours
II Repair period	48 hours to 6 weeks
III Adherent scar tissue	>6 weeks

Table 58.4 Classification of ankle sprain by severity of lesion

Grade[71]	Lesion
I	Elongation of ligaments without macroscopic rupture
II	Partial and macroscopic ligamentous rupture
III	Complete ligamentous rupture

Inversion sprain

Lateral ankle sprain injury is the most common acute sport trauma, and accounts for about 14% of all sport-related injuries.[72] It is also reported to be the most common injury in college athletics in the United States.[73] Athletes involved in soccer, basketball, volleyball and long-distance running are especially plagued by these injuries.[74–76]

Mechanism

The origin of an inversion sprain is usually an indirect force produced against an inverted and plantiflexed foot, when the weight of the body forces the talus to rotate[77] and twists the forefoot into supination and adduction. Hirsch and Lewis[78] demonstrated that a rotational force of only 5–8 kg can produce a rupture of the anterior talofibular ligament.

The site of the lesion will depend largely on the degree of plantiflexion during inversion[79,80] (Fig. 58.19):

- If the ankle is in the neutral position or slightly dorsiflexed during the excessive varus movement, the calcaneofibular ligament is damaged.[81]
- If the ankle is plantiflexed during the varus stress, the talus becomes involved in the movement and undergoes a medial rotation. This imposes the greatest stress between talus and fibula, and the anterior talofibular ligament becomes stretched.
- When the ankle and subtalar joints undergo indirect violence and the midtarsal joints and the forefoot are also twisted into full plantiflexion, with supination and adduction, the stress tends to fall on more distally localized structures such as the calcaneocuboid ligament, the insertion of the short peroneal tendon at the fifth metatarsal bone or the cuboid–fifth metatarsal joint.
- During complete plantiflexion of the ankle, with slight or no varus movement, the anterior tibiotalar ligament or the tendons of the extensor digitorum longus may be damaged.

Most authors only mention the talofibular and calcaneofibular ligaments.[82,83] Very often, however, lesions of the calcaneocuboid ligament and tendinous lesions of the peronei and the long extensor of the toes result from an ankle sprain.

It is very important to note that, in most cases of sprained ankle, a combination of lesions occurs. The commonest association is a sprain of the fibular collateral ligament together with the calcaneocuboid ligament. Injury to all these structures may be correctly described by the patient as a 'sprained ankle'. It is important to realize that, after a so-called varus sprain, not

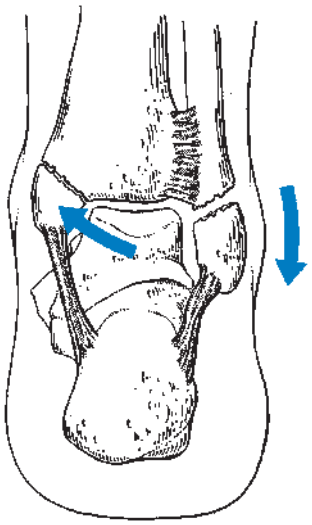


Fig 58.20 • Malleolar fractures occurring with so-called varus sprain.

only ligamentous but also osseous lesions can occur. The most frequent lesions are fractures of both malleoli (Fig. 58.20): at the lateral malleolus a traction fracture, and at the medial malleolus a compression fracture. An avulsion fracture of the base of the fifth metatarsal is also not uncommon. It is obvious that, if history or examination suggests a fracture, a plain radiograph must be taken.

Natural history

During the first few hours after the trauma, a traumatic inflammatory reaction with warmth, pain, swelling and loss of function will result, regardless of the size of the lesion, and lasts a couple of days. In this period, damaged tissue and noxious substances are eliminated and diluted. Very soon, there is ingrowth of blood and lymph vessels from the adjacent intact structures, together with migration of fibroblasts. The latter synthesize new collagen in an effort to form a scar.

It has been well established that the alignment of the collagen fibres in the scar is anarchic and disorderly if insufficient external stimulus is applied to the healing tissue. Some tension to the granulation is necessary to improve and accelerate the development of the fibrillary network into orderly layers.[84] Immobilization leads to a scar that is adherent to capsule and bone. The sprained ankle then proceeds to a chronic stage: prolonged disability for several months. Sometimes the patient never recovers, unless the adhesions are ruptured by manipulation.

The discussion of diagnosis and treatment of ankle sprains follows the natural history sequence:

- *First or acute stage*: traumatic reaction immediately following the trauma – the first 24–48 hours.
- *Second or subacute stage*: traumatic reaction disappears; period of repair – from the second day to 6 weeks.
- *Third or chronic stage*: the scar has definitely formed; if there are adhesions, permanent disability results after 6 weeks to 2 months.

Diagnosis

There is nothing easier than diagnosing a sprained ankle but locating the lesion precisely, in order to estimate the degree of damage and to predict the prognosis, is not so simple. A thorough and diligent clinical examination by an experienced practitioner is the best basis for an accurate diagnosis. The value of technical investigations should not be overestimated.

Acute stage

History

There has been an inversion injury, with sudden pain and sometimes an audible 'snap'. Afterwards there is swelling and more pain, together with some degree of dysfunction. Sometimes there is nocturnal pain; if this is the case, haemarthrosis should be strongly suspected. If the patient is immediately unable to stand on the affected leg, a fracture should be considered.

Clinical examination

This should include the normal examination for the ankle and foot. In ankle sprains of short duration, the clinical examination serves a double purpose: it detects serious lesions and localizes the exact site of the sprain.

- *To detect serious lesions* in recent ankle sprains, some 'warning signs' are built into the history and the clinical tests. These make it possible to identify cases of sprained ankle that have a high risk of complications: avulsion fractures, malleolar fractures, fractures of the fifth metatarsal base, haemarthrosis and total ruptures of the lateral ligaments.

Warning symptoms and signs

Serious complications of ankle sprain should be suspected if one or more of the following symptoms and signs is present.

Warning symptoms:

- Age over 60 years (risk of fractures)
- Immediate and continuous inability to bear weight (fracture)
- Nocturnal pain during the first 48 hours (haemarthrosis)

Warning signs:

- Capsular pattern at the ankle joint or subtalar joint (haemarthrosis/subchondral lesion)
- Gross pain during valgus (compression of a fractured fibular malleolus)
- Pain and weakness during resisted eversion (fracture of the fifth metatarsal basis)
- Localized tenderness over the tip of either malleolus (fractured malleolus)
- Little pain during passive movement of the foot (possibly in combination with a larger range of movement) is suggestive of a total ligamentous rupture.[85–87]

- *Localization of the site of the lesion* can be deduced from the pattern that emerges when passive and resisted movements are tested. Once the site of the sprain has been identified by clinical examination, tenderness of the appropriate structures can be sought. (It is important to make the diagnosis purely by inference from studying

Table 58.5 Patterns of acute ligamentous and tendinous disorders in inversion sprains

Structure	Positive tests
Ligaments	
Anterior talofibular	Combined plantiflexion/inversion Plantiflexion
Calcaneofibular	Inversion Varus
Calcaneocuboid	Inversion Adduction and supination in the midtarsal joint
Anterior tibiotalar	Plantiflexion
Tendons	
Peroneal	Combined plantiflexion/inversion Resisted eversion
Extensor digitorum longus	Passive plantiflexion Resisted dorsiflexion of foot and toes

the clinical tests and not by palpation. In recent cases, oedema and generalized tenderness are often so gross that palpation does not yield reliable information.)

The patterns of acute ligamentous and tendinous disorders are given in Table 58.5.

In combined lesions (two or more ligaments, or a ligament and a tendon), the clinical examination may be more puzzling. The information that emerges during the standard examination is summarized in Table 58.6.

Technical investigations: radiography

Radiography is widely used in the assessment of inversion ankle injuries and accounts for about 10% of all radiographic examinations performed in accident departments.[88] However, the predictive value of a plain radiograph examination in relation to fractures is rather poor when clinical warning signs are absent.[89–91] For this reason, decision rules for plain X-rays have been developed – the so-called 'Ottawa rules'. A plain X-ray of the ankle and foot should be requested if the patient is over 60 years of age or there is localized bone tenderness of the posterior edge or tip of either malleolus, or the patient is unable to bear weight immediately after the injury.[92] This rule is 100% sensitive and 40.1% specific for detecting malleolar fractures and would allow a reduction of 36% of ankle radiographic series ordered.[93,94]

A routine radiograph is of no value in the diagnosis of a total ligamentous rupture or a haemarthrosis.

In order to diagnose complete rupture, other radiological procedures have been suggested: stress radiography (evaluation of talar tilt), ultrasound and MRI.[95]

- *Stress radiography*: talar tilt (the angle between the inferior border of the tibia and the superior edge of the talus, during varus stress) depends not only on the degree of ligamentous rupture but also on the use of anaesthesia,[96] the degree of applied force and the direction of the X-ray beam. The sensitivity and selectivity of stress pictures are seriously questioned.[97,98]

Table 58.6 Summary of the diagnosis of recent inversion sprains

Positive test	Lesion	Warning sign
Tiptoe rising	Peroneal tendons	Not possible: fractures of malleolus or 5th metatarsal
Plantiflexion Dorsiflexion	Anterior talofibular/ calcaneocuboid ligament	A capsular pattern: haemarthrosis in the ankle
Varus Valgus Mortice	Calcaneofibular ligament	Excessive movement: total rupture Lateral pain: lateral avulsion fracture of the fibula Medial pain: medial compression fracture
Plantiflexion/ inversion Plantiflexion/ eversion	All lateral ligaments and peroneal tendons	Excessive movement: total rupture Lateral pain: lateral avulsion fracture of the fibula
Plantiflexion Dorsiflexion Pronation	Calcaneocuboid ligament	
Supination Abduction	Calcaneocuboid ligament	
Adduction	Calcaneocuboid ligament	
Resisted plantiflexion	Peroneal tendons	
Resisted dorsiflexion Resisted inversion	Tendons of extensor digitorum longus	
Resisted eversion	Peroneal tendons	Weakness: avulsion fracture of 5th metatarsal

- *Ultrasound*: this has been advocated for the evaluation of acute ankle ligament injuries because it allows for non-invasive and dynamic assessment of the ankle.[99] However, ultrasound is highly dependent on equipment and operator skill level.
- *MRI scans*: these are not typically indicated for acute ankle sprains, except to elucidate associated conditions (detection of talar dome injuries).[100] It is also important to note that approximately 30% of asymptomatic patients undergoing MRI have abnormal anterior talofibular ligaments.[101]

In our opinion, plain radiographs are advised only if the signs warrant them. Stress radiography, ultrasound and MRI should not be performed because their diagnostic and prognostic values are poor. Furthermore, because the majority of severe (grade III) ankle sprains may be treated non-operatively and, if residual instability occurs, late reconstruction achieves satisfactory results,[102] early detection of severe grade III lesions by means of expensive and potentially dangerous investigations is obsolete.

Table 58.7 Patterns of ligamentous and tendinous lesions in chronic inversion sprains

Lesions of the:	Findings
Talofibular ligament	Passive plantiflexion–inversion: painful, slightly limited, tight end-feel Midtarsal tests: negative Resisted movements: negative
Calcaneocuboid ligament	Passive plantiflexion–inversion: painful, slightly limited, tight end-feel Passive midtarsal supination: painful Resisted movements: negative
Calcaneofibular ligament	Passive varus: painful
Peroneal tendon	Passive plantiflexion–inversion: painful Midtarsal tests: negative Resisted eversion: positive
Extensor digitorum tendon	Passive plantiflexion–(inversion): painful Midtarsal tests: negative Resisted dorsiflexion: positive

Box 58.2

Differential diagnosis in chronic inversion sprains

Immobilizational stiffness

- Equal limitation of varus/valgus
- Rest of clinical examination: normal

Loose body

- 'Twinges'
- Clinical examination: negative

Instability

- Clinical examination: negative
- Specific tests: positive

Subacute post-traumatic arthritis

- Capsular pattern at the subtalar joint and spasm of the peronei

Box 58.3

Persistent pain after ankle sprain – differential diagnosis

- Chronic stage
  - Adhesion formation
- Tendinitis
  - Peroneal tendons
  - Extensor digitorum longus
- Instability
  - Unstable mortice
  - Ligamentous insufficiency
  - Functional instability
- Traumatic arthritis (talar dome fracture)
- Loose body
- Immobilizational stiffness

Chronic stage

History

The patient describes a previous ankle sprain from which nearly complete recovery has been made, probably after some weeks of immobilization. The foot is functionally adequate for ordinary purposes but is apt to swell and ache after vigorous or prolonged use. The patient is fit to walk and even to run, although playing sports such as football is painful, especially at the beginning of and after the game. This history might indicate that scars have formed abnormal attachments as the result of healing in the absence of enough movement. Another possibility is that the ligamentous sprain has recovered but a chronic tendinitis remains. Additionally, apart from adhesions and tendinitis, persistent trouble after a previous ankle sprain may also be caused by instability, immobilizational stiffness or a loose body in the ankle joint.

Clinical examination (Table 58.7 and Box 58.2)

Adhesions

These can form at the talofibular and the calcaneocuboid ligament. In both instances, there is pain at the outer side of the ankle on full inversion and plantiflexion. Pain during supination at the midtarsal joint implicates the calcaneocuboid ligament. Slight limitation of movement can occur and the end-feel is harder than on the unaffected side. There is no pain when resisted movements are tested.

Persistent tendinitis

In this case, passive plantiflexion–inversion movement is also painful but the end-feel is normal. Pain during resisted eversion suggests that the peroneal tendons are at fault.[103,104] Painful resisted dorsiflexion of foot and toes is caused by inflammation of the extensor digitorum longus tendons.

Other sources of persistent ankle trouble

These include instability, traumatic arthritis, immobilizational stiffness and impaction of a loose body; they are disclosed during the clinical examination.

Immobilizational stiffness, traumatic arthritis (p. 1212) and loose body (p. 1214) have been discussed earlier.

Instability at the ankle, due to a persistent rupture of the tibiofibular and talofibular ligaments or delayed contraction of peroneal muscles, is discussed later.

The differential diagnosis of persistent pain after ankle sprain is summarized in Box 58.3.

Treatment

Nowadays, it is generally accepted that 'functional treatment' with early mobilization and weight bearing and neuromuscular training exercises is the treatment of choice in grade I and

grade II sprains.[105–109] This approach achieves much better results than treatment with plaster immobilization.[110–116] Early surgery may claim equally good results in the short term but long-term studies clearly demonstrated much better results when early mobilization was used.[98,117,118] Other prospective and randomized studies also showed the best results with early functional treatment.[119–121]

For grade III ankle sprain, the treatment remains more controversial. Some surgeons recommend surgical repair[142] while others favour non-operative conservative treatment.[143] Recent research indicates that, even for total ruptures of the lateral ligaments of the ankle, the treatment of choice is still functional rehabilitation. Several prospective studies[122,123] and meta-analyses[124,125] showed that early functional treatment provided the fastest recovery of ankle mobility, and the earliest return to work and physical activity, without affecting late mechanical stability. A prospective and randomized study on 85 patients with acute grade II or grade III lateral ligament ruptures concluded that functional treatment was free from complication, resulted in shorter sick leave and facilitated an earlier return to sports than did surgery.[126,127] Furthermore, secondary surgical repair, even years after an injury, has results comparable to those of primary repair,[128,129] so even competitive athletes can receive initial conservative treatment.[130]

Early mobilization

In the management of ruptures at the ankle, most physicians and surgeons reason anatomically: a rupture is suspected or established radiologically and their approach is to repair the defect as soon as possible, by partial or total immobilization or by early suture. Their philosophy of treatment in ligamentous sprains is the same as in fractures: immobilize the two separated ends to build a strong scar. This anatomical way of thinking does not correspond to functional reality. The function of a ligament is in no way comparable with the function of a bone. Bone must be strong and have solidity. In contrast, the function of ligaments is mobility. Ligaments must allow movement and conduct movement between certain limits. To serve that purpose, ligamentous tissue must be mobile enough to change its position continuously during the imposed movement. Therefore the scar must be not only strong enough to prevent excessive instability but also mobile enough to allow sufficient movement. If this is not the case, and if abnormal attachments have formed between scar and bone, persistent functional problems may result. Furthermore, immobilization reduces proprioception and increases the peroneal reaction time, thus increasing the chance of *functional* instability.

Thus, early mobilization of the healing ligament is important for full recovery. However, in advocating this, we encounter one main difficulty: in a serious ankle sprain, the intensity of the inflammatory response leads to secondary effects that impair mobility. The slightest movement causes pain, which forces the patient to immobilize joint and ligaments. The regenerating fibrils, however, rapidly spread in all directions, leading to disorganized scar tissue structure and to the possibility of adhesion formation, which is sufficient reason to start the mobilization at the earliest possible moment.

The problem can be solved in two ways:

- *Abolish the inflammation and pain as soon as possible*, so that the patient can start mobilizing exercises (passive or active). This can be done via local infiltration of triamcinolone into the sprained structures. A small amount of steroid suffices, with no danger of causing permanent weakness of the ligament.
- *Move the ligament over the (stationary) joint*: Cyriax used to say: 'If the joint cannot be moved in relation to the ligament, since pain and inflammation force the joint in muscle spasm, it will probably be possible to move the ligament in relation to the joint.' The relative movement is the same, as is the mechanical stimulus to the regenerating fibrils. This can be achieved by gentle passive movements at the developing scar. This is the reasoning behind deep transverse friction applied to the sprained ankle.[131]

Treatment procedures

The treatment chosen depends on the stage of the lesion (Box 58.4).

Acute stage: first 2 days

Immediate infiltration with a small dose of triamcinolone at the exact point is indicated. The sooner after the injury the infiltration is made, the more spectacular the results. As it is not easy to palpate a recently sprained ankle, the exact site of the lesion must be determined by an accurate functional

Box 58.4

Treatment of acute and chronic ankle sprain

Uncomplicated ligamentous lesions

Acute lesion (<48 h)

Either:

- Infiltration with 10 mg triamcinolone
- Figure-of-eight bandage
- Followed by mobilization and 'functional treatment'

Or:

- Deep transverse massage, daily for 30 seconds, after preparative 'effleurage', until the ankle is in the subacute stage
- Figure-of-eight bandage
- Functional treatment

Subacute lesion

- Deep transverse massage, daily for a few minutes, followed by gentle passive movements
- Functional treatment

Chronic lesion (<6 weeks)

- Deep transverse massage as preparation for manipulation in the limited direction
- Functional treatment

Tendinous lesions

- Deep transverse massage
- Relative rest

examination. To facilitate the injection, most of the oedema must be removed by deep effleurage, before the needle is introduced.

Because of the anti-inflammatory reaction of the steroid, the resolution of swelling and post-traumatic inflammation is hastened. The pain abates quickly and gradual functional improvement can be expected from the second day after the injection.

Sometimes simple strapping in slight valgus is applied, which can give the patient confidence. It is also possible that the tape brings on a 'musculocutaneous reflex', whereby proprioception of the ankle is activated and so prevents an early recurrence of the sprain.[107,132] Strapping must be loose enough to enable the patient to walk and to move the ankle as much as possible.

If the patient refuses infiltration or is seen too late after the injury (after a couple of days), deep transverse massage is applied.

First some effleurage is given, to diminish the oedema and to render the lesion more accessible to the fingertips. Then very gentle massage is applied for 10–15 minutes. The intention is just to anaesthetize the injured ligament so that the patient can bear the 30 seconds of harder friction. The massage will not be vigorous, only deep enough to move the ligament on the subjacent bone and give it the necessary stimulus for correct healing.

The patient should be treated daily for 10 days to 2 weeks. Between sessions, walking for short distances must be attempted. It is important to give instructions on how to walk slowly, with a heel-and-toe gait and without limping.

Subacute stage

Infiltration with steroid suspension is of no use after 2 days have elapsed since the injury. Massage is the treatment of choice. It moves the ligament over the joint and the bone, in imitation of its normal behaviour, and gives a mechanical stimulus to the ingrowing fibrils, which prevents their adhesion to the surrounding tissues.

Once again, effleurage is applied first, in order to reduce the oedema. Gentle massage causes some local anaesthesia, after which a few minutes of firmer friction, applied deeply to the actual site of the ligamentous tear, is used.

After the friction, gentle passive movement is performed to the limit of the possible range, which is the range of discomfort but *not of pain*. Therefore it is important to perform the movement firmly but gently and prevent an excessive range by observing the patient's reactions. Unless the therapist is unnecessarily rough, there is no danger of overstretching the sprained ligaments.

Active movements follow passive ones. The patient must be persuaded to execute them repeatedly during the day. Active movements are very useful because they further the effect of deep friction in preventing scar tissue forming abnormal adhesions. The main difficulty is to get the patient to understand how much greater the painless range is than believed.

Chronic stage

Scars have been allowed to form abnormal attachments as the result of healing in the absence of sufficient movement. The foot functions well enough for ordinary purposes but it aches and swells after prolonged or strenuous use.

The only logical and effective treatment is to break the adhesions by manipulation. This is quite easy to perform and does not need anaesthesia, apart from some deep friction. One manipulation session often suffices to cure chronic adhesions that have caused continuous disability for months. After the manipulation, there is no pain and after-treatment is not required.

Before the decision to manipulate is made, the diagnosis must be certain. It is obvious that an inversion sprain which has resulted in peroneal tendinitis will not benefit from manipulation of the joint. The distinction between tendinitis and chronic adhesions is made by the end-feel during passive inversion and the negative findings during resisted eversion. Another differential diagnosis is 'subacute traumatic arthritis' at the subtalar joint. As this condition can also be the result of a previous ankle sprain, it can very easily be mistaken for post-traumatic adhesions. In subacute traumatic arthritis, there is also some limitation of movement in the calcaneocuboid joint, which is caused by spasm of the peronei. In chronic ligamentous adhesions, however, the movement at the talocalcaneal joint is normal and the end-feel is not spastic but rigid. It is clear that the arthritis will be aggravated if manipulation is undertaken.

Tendinous lesions

Early mobilization is not used if a tendon is affected. In all stages, the treatment consists of deep transverse friction. The patient should also avoid any exertion that causes pain.

Usually, the lesion is too diffuse for injection of steroid but, if a small stretch of tendon remains refractory to friction, infiltration with triamcinolone suspension can be performed.

Treatment techniques

Technique: infiltration of the fibular extent of the talofibular and calcaneofibular ligament

The injection technique is the same for both ligaments.

The patient lies supine on the couch, with the lower limb in internal rotation to bring the lateral malleolus uppermost. The foot must be held in as much plantiflexion and inversion as possible to make the lateral side accessible by the needle. After removing most of the oedema, tenderness is palpated along the inferior border of the malleolus and a line is defined from end to end. A 2 mL syringe is filled with triamcinolone, mixed with some local anaesthetic. A thin needle, 3 cm long, is fitted and inserted at a point 2 cm distal from the edge of the fibula. The needle is now moved almost parallel to the ligament, in the direction of the fibular edge (Fig. 58.21). A series of small droplets is injected from end to end at the ligamento-periosteal insertion.

It is vital to infiltrate the whole of the tender area and to inject only when the needle touches bone. The injection is only about half as painful as the previous examination and palpation but some after-pain may occur for 1 or 2 days.

From the next day onwards, the patient should use the foot and must be encouraged to walk with a proper heel-and-toe gait, without limping.

Technique: infiltration of the calcaneocuboid ligament

The patient adopts a supine-lying position with the limb in internal rotation. This brings the outer side of the foot uppermost.

With one hand, the forefoot is supinated and adducted to bring the ligament under some tension. Tenderness along the ligament is sought in the following manner. The therapist places the interphalangeal joint of the thumb on the base of the fifth metacarpal bone and aims in the direction of the midpoint between the two malleoli. The tip of the thumb now lies exactly on the lateral calcaneocuboid ligament (Fig. 58.22a). The line of tenderness along the ligament is marked from end to end. A 2 mL syringe is filled with steroid suspension and fitted to a fine needle, 2 cm long. The needle is thrust in at the lateral border of the ligament (Fig. 58.22b). It is first moved to the calcaneal extent, where a series of droplets is placed along the ligamentoperiosteal border. The infiltration is made when the needle touches bone. Once the calcaneal border has been infiltrated, the needle is withdrawn slightly and pushed on to the cuboid border, where the same procedure is repeated.

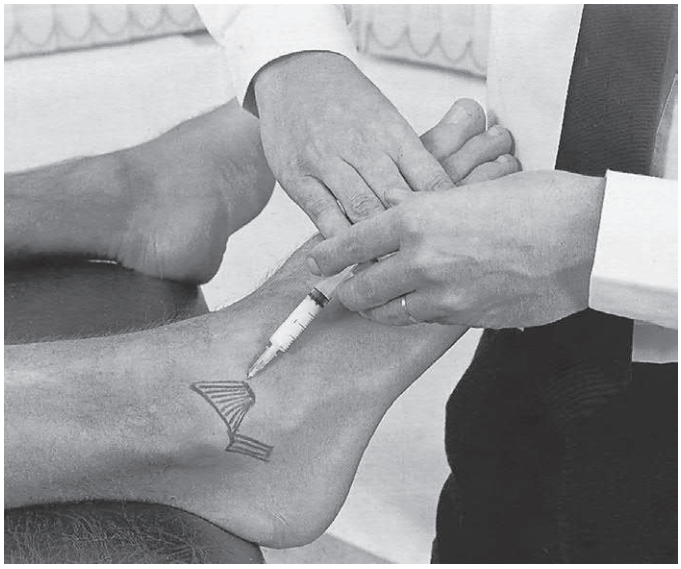


Fig 58.21 • Infiltration of the fibular extent of the talofibular ligament.

Technique: deep friction to the fibular extent of the talofibular ligament

The patient lies supine, with the leg outstretched and in medial rotation so that the outer border of the foot faces upwards.

The therapist sits at the medial side of the foot. With the contralateral hand, the patient's foot is held in plantiflexion and inversion. This position stretches the ligaments as far as is comfortably possible. In recent sprains, some effleurage is given first, so as to move most of the oedema and to define bony landmarks and tenderness more easily.

The index finger of the ipsilateral hand, reinforced by the middle finger, is now placed on the site of the lesion. To reach the exact localization under the fibula, the forearm must be pronated, so the pressure will be upwards and inwards and the ligament is pressed between finger and bone. The thumb is placed proximal to the medial malleolus to give counterpressure during the massage (Fig. 58.23).

Friction is imparted by drawing the fingers forwards during an adduction movement of the shoulder. The pressure is then slightly released and the finger turned to the previous position, where the whole procedure is repeated.

In recent sprains, the massage is given very gently, only deep enough to move the ligament on the subjacent bone. There should be only gentle and slight pressure for 15 minutes, after which some anaesthesia will occur; another 30 seconds of proper massage can then follow.

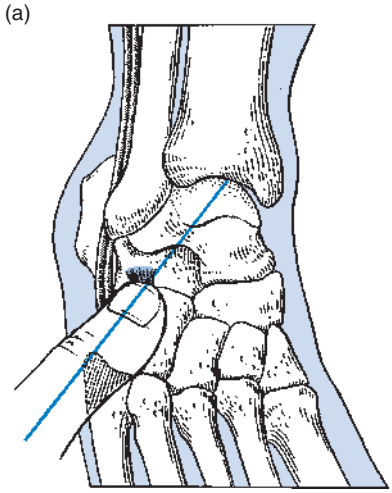
More thorough friction must be applied in the subacute stage.

In chronic stages, strong and vigorous friction is given to break some adhesions and to anaesthetize the ligament for the subsequent manipulation.

Technique: deep friction to the talar extent of the talofibular ligament

The position of the patient's foot is the same as in the previous technique. The therapist uses the contralateral hand to force the foot slightly into plantiflexion and inversion in order to stretch the ligament. The neck of the talus is sought and the

(a)



(b)

Fig 58.22 • Calcaneocuboid ligament: (a) palpation technique; (b) infiltration.

Fig 58.23 • Deep friction of the fibular extent of the talofibular ligament.

Fig 58.25 • Friction to the fibular extent of the calcaneofibular ligament.

Fig 58.24 • Friction to the talar extent of the talofibular ligament.

talar insertion of the ligament identified. The tip of the index finger of the other hand, reinforced by the middle finger, is placed on the talar extent (Fig. 58.24). The pressure is directed purely medially to the bone, so the forearm is not pronated. Friction is given by drawing the hand to and fro over the ligament. The thumb placed at the medial side of the foot acts as a fulcrum. The sweep will not be as large as in the previous technique because the talar extent of the ligament is much less.

In recent sprains, massage is gentle, whereas in chronic cases, strong friction must be given in order to anaesthetize the ligament for the subsequent manipulation.

Technique: deep friction to the fibular extent of the calcaneofibular ligament

The patient lies supine. The therapist sits at the distal end of the foot and fixes the heel in varus position with the ipsilateral hand. The index finger of the contralateral hand, reinforced by the middle finger, is placed under the fibular tip. The thumb is held at the medial malleolus so as to give counterpressure (Fig. 58.25). The friction is imparted by a flexion–extension movement at the wrist.

Technique: deep friction to the calcaneocuboid ligament

The patient lies face upwards, with the lower limb extended and in medial rotation. In this position, the outer border of the foot faces upwards. The ligament is palpated by using the technique explained above (see Fig. 58.22a). To check the correct position, the patient is asked to contract the peroneal tendons, which should lie just plantar to the fingertip.

The therapist sits at the inner side of the foot, facing its medial aspect. The foot is steadied with the contralateral hand, which forces the forefoot into adduction and supination. This brings the calcaneocuboid joint to prominence and stretches the ligament. The index finger of the other hand, reinforced by the middle finger, is placed on the joint line at the tender point (Fig. 58.26).

Friction is given by a vertical movement of the finger, imparted by an adduction movement of the whole arm. This moves the finger along the joint line and across the tender ligament in a transverse direction to the fibres. In recent sprains, the friction is not vigorous. The intention is merely to anaesthetize the tender area with 10 minutes of gentle massage, just deep enough to move the ligament slightly on the bone. This is followed by 30 seconds of more thorough friction. In subacute treatment sessions the friction may be more vigorous, and in chronic cases the intention is to rub really hard, but still within the limits of comfort, as a preparation for manipulation.

Technique: manipulation of adherent lateral ligaments

The technique is the same for both the anterior fasciculus of the fibular collateral ligament and the calcaneocuboid ligament. The intention is to rupture the adhesions at the ankle joint and the calcaneocuboid joint. Before the manipulation,

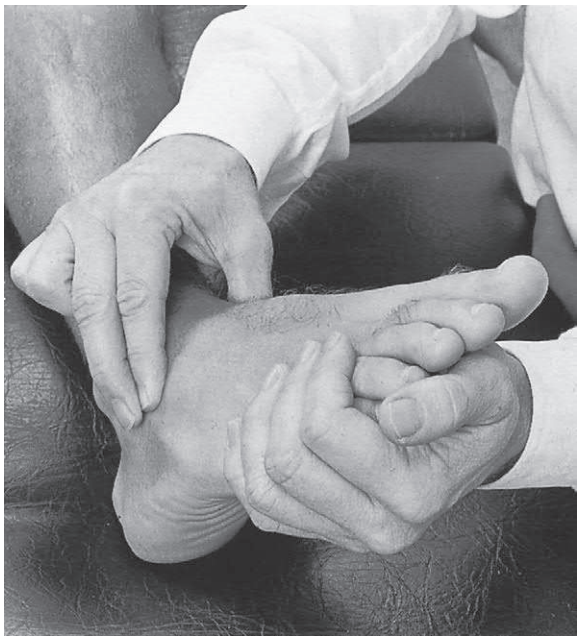


Fig 58.26 • Friction to the calcaneocuboid ligament.

vigorous friction must be given at the exact point for 15 minutes so as to decentralize the ligament.

The patient lies supine on a high couch, the leg extended. The manipulator stands facing the patient's foot, grasps the heel with the ipsilateral hand and forces it into full varus by an abduction movement of the arm. The dorsum of this hand will remain on the couch during the whole procedure. The contralateral hand is now placed on the dorsum of the foot, whereby the fingers curl round the shaft of the first metatarsal bone and the heel of the hand rests on the lateral border of the foot (Fig. 58.27).

The hand must do three things at the same time: plantiflex, medially rotate and adduct the foot. These three movements must be performed simultaneously, quickly and with a small amplitude at the end of range. Although the movement must be strong enough to break the adhesions, the physiological range must be respected. The manipulating hand must be 'motor and sensor' at the same time and the end-feel is the guide to manipulation, informing the manipulator whether it should be continued or not.

In order to carry out this triple movement perfectly, the manipulator abducts the arm and flexes the elbow to a right angle, the hand flat on the patient's foot:

- The hand presses the foot downwards to the floor, which evokes plantiflexion.
- If the manipulator exerts greater pressure at the outer border of the foot, it is forced into inversion.
- An adduction movement in the foot is now obtained, when the heel of the manipulating hand is brought inwards, pivoting around the fingers at the inner side of the forefoot.

During the entire movement, the elbow is pressed downwards and towards the manipulator. Therefore, unless the manipulator holds the elbow well away from the body before the whole procedure starts, little force can be exerted in the final movement.

The manipulation does *not have a large amplitude*. The downward, inward and rotational movement is performed slowly, until the characteristic resistance of the adherent ligament becomes perceptible. Here a final quick, strong thrust is given by an adduction movement at the shoulder, in order to restore the 1 or 2° of impaired movement.

There are a number of contraindications to manipulation (Box 58.5).

Box 58.5

Contraindications to manipulation

- No limitation of movement and a normal end-feel.
- If inversion sprain has resulted in peroneal tendinitis, no advantage accrues from manipulation; the only suitable treatment is deep transverse friction to the peroneal tendons.
- It is obvious that forced movements are not suited to recent sprains.
- If laxity of the joint rather than adhesions causes a permanent or recurrent problem at the ankle, manipulation will not, of course, be the treatment of choice.
- Post-traumatic arthritis, which occurs occasionally after an ankle sprain; the differential diagnosis is made by detection of a capsular pattern, together with peroneal spasm.

Recurrent varus sprain – instability

The patient states that, after a previous varus sprain, the ankle turns over easily and there is a fear of the ankle 'giving way'. It lacks stability and is subjected to a succession of minor sprains. This is apt to happen more frequently in athletes, who make heavy demands on the joint.

Instability at the ankle, resulting in repeated minor sprains, may have different causes (Fig. 58.28):

- A ruptured distal tibiofibular ligament, with a so-called unstable mortice.
- A permanent lengthening of the anterior talofibular ligament, with an anteroposterior instability of the ankle joint.
- Proprioceptive deficits secondary to neurological injury to ankle ligaments and capsule. As a result, the peroneal muscles are brought into play too slowly to prevent further sprains when the ankle starts turning over.[14,15]

The unstable mortice

The common complaint is an 'unstable ankle' after a previous sprain. The patient finds that the foot turns over easily, often with an audible click and momentary severe pain *in* the ankle. The pain does not last very long and, after a few moments, walking can continue. The ankle merely feels sore for a couple of days.[133]

Clinical examination reveals nothing except a positive test for the mortice, which is included in the standard clinical

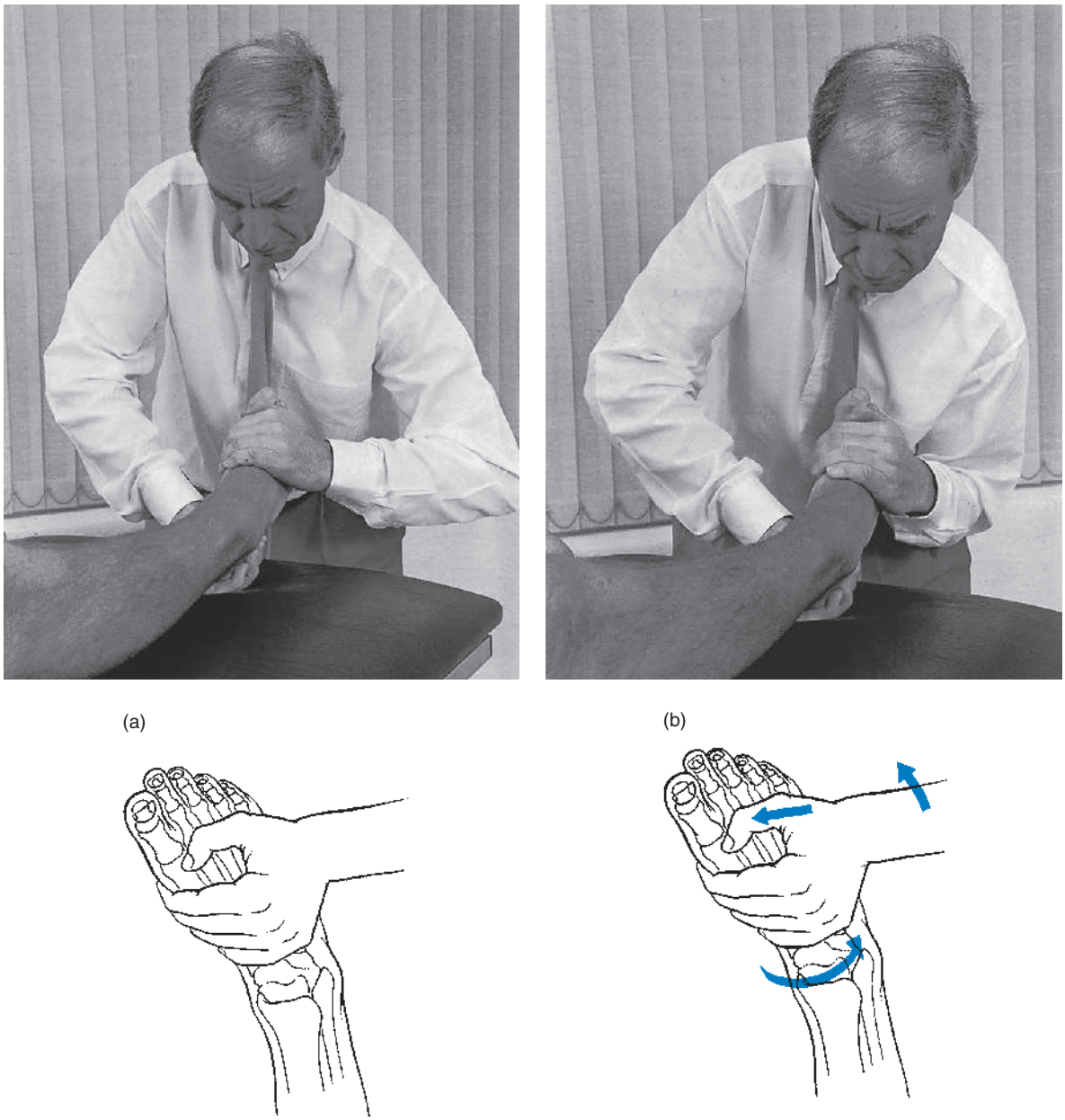


Fig 58.27 • Manipulation of adherent lateral ligaments: start of manipulation (a) and end of manipulation (b).

examination of the ankle. This is a strong, quick varus movement at the ankle, with the joint in the neutral position. If there is rupture or elongation at the distal tibiofibular ligament, an excessive range of varus movement can be demonstrated. A click can be produced at the ankle when the force is released and the two bones meet again. If this varus movement is repeated with the examiner's fingers palpating the two malleoli, they can be felt to move apart.

The diagnosis can be confirmed by radiography. If the radiograph is taken during strong varus movement, widening of the joint space between the tibia and fibula is seen.[134]

Treatment: sclerosing injections

These must always be tried first, before the patient is referred for surgery. Sometimes excellent results can be obtained.

The injections are given at weekly intervals over 3 weeks. The patient must take relative rest over another 4 weeks, to allow the injected ligaments to sclerose.

To make the injection easier, it helps to measure on an anteroposterior radiograph the distances between the tip of the lateral malleolus and the articular surface of the tibia, together with the respective widths of tibia and fibula (Fig. 58.29).

The ligament must be infiltrated from behind and from in front, each time with 1.5 mL of the sclerosant mixed with 0.5 mL of lidocaine 2%. A thin needle, 4 cm long, is used.

Technique: infiltration from behind

The patient lies prone; the lines indicating the inferior border of the tibia and the joint line between tibia and fibula are drawn

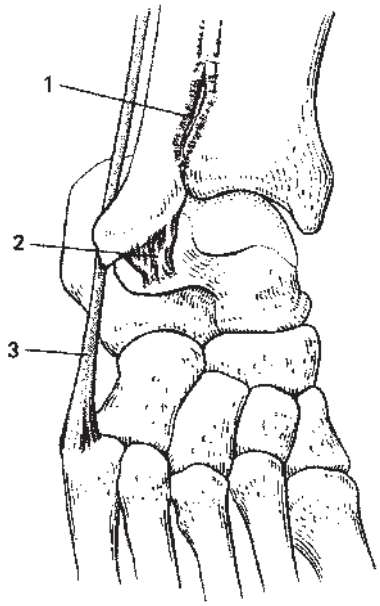


Fig 58.28 • Causes of instability at the ankle: 1, lengthened or ruptured distal tibiofibular ligament; 2, lengthened or ruptured anterior talofibular ligament; 3, proprioceptive defects affecting the peroneal muscles.

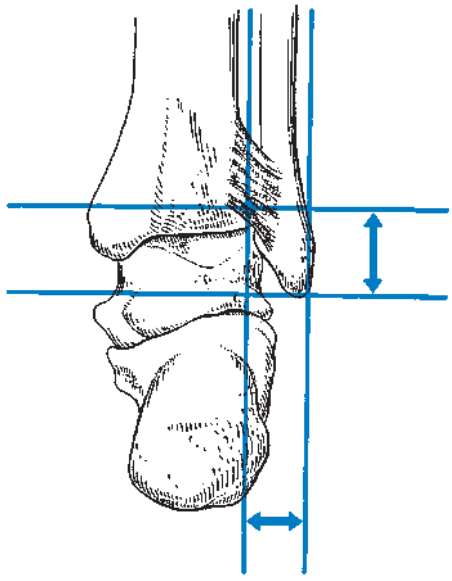


Fig 58.29 • Landmarks and measurements which can be drawn on a plain frontal radiograph taken preparatory to sclerosing injections for an unstable mortice.

at right angles. The needle is thrust in vertically downwards, a few millimetres above the intersection of these two lines. Tough ligament must be felt to be penetrated before hitting bone. This happens at about 3 cm depth. If the needle immediately hits bone, its point is too far to one or the other side of the joint: the needle is withdrawn a little and reinserted in a slightly different direction until it first traverses the ligament and then hits bone. A series of intraligamentous infiltrations along the vertical line of the ligament follows.

Technique: infiltration from in front

The patient lies supine. The two lines are drawn again, to cross each other anteriorly. The injection is now repeated from in front. Here the tendons of the extensor digitorum longus must be held apart before the needle is inserted. Again, ligament must be pierced before bone is met.

Ligamentous insufficiency of the anterior talofibular ligament

The patient has a history of previous ankle sprain, from which recovery is largely complete. There remains, however, some fear of the ankle 'giving way' and the patient is unable to rely on using the foot.

Routine clinical examination shows nothing, except probably a greater range of inversion movement.

The anterior drawer test can be used to demonstrate a rupture or elongation of the anterior talofibular ligament.[135]

Diagnostic technique: anterior drawer test[136–138]

The ankle is held in slight plantiflexion. The examiner stands at the opposite side and stabilizes the patient's lower leg with the ipsilateral hand. The contralateral hand encircles the foot and displaces the foot forwards. The lateral margin of the trochlea tali is thus shifted forwards in relation to the lateral malleolus (Fig. 58.30). It is important to notice that there is not only a forward gliding but also a medial rotation of the talus around a vertical axis at the medial malleolus.

As the anterior talofibular ligament is tense in all plantiflexion positions of the ankle, this anterior movement will only be possible if the ligament is not intact. The movement of the talus can be seen and felt. Often, a depression between talus and malleolus is noticed when the talus is moved forwards (Fig. 58.31). If a positive anterior drawer sign is present in a patient with recurrent ankle sprains or the patient fears the ankle 'giving away', a lax anterior talofibular ligament must be blamed for the symptoms.[139]

However, the reverse does not hold because only half of the patients with a positive anterior drawer sign report symptoms of ankle instability. The reason for this is probably compensation for the ligamentous laxity by muscle power and a good proprioceptive reflex.[136] Therefore an anterior drawer test is never performed during the standard examination but only when the history warrants it.

Treatment

Conservative treatment of this form of instability consists of proprioceptive training, so that a good reflex will replace the function of the insufficient ligament. Mechanical devices designed to prevent ankle sprain during high-risk sporting activities (e.g. soccer, basketball) are: wrapping the ankle with tape or cloth, orthoses, high-top shoes or some combination of these methods. Appropriately applied braces, tape or orthoses should not adversely affect performance.[140–143]

If necessary, surgical repair of the torn ligaments may be advised.[144,145] The results of late surgical repair are good.[146,147]

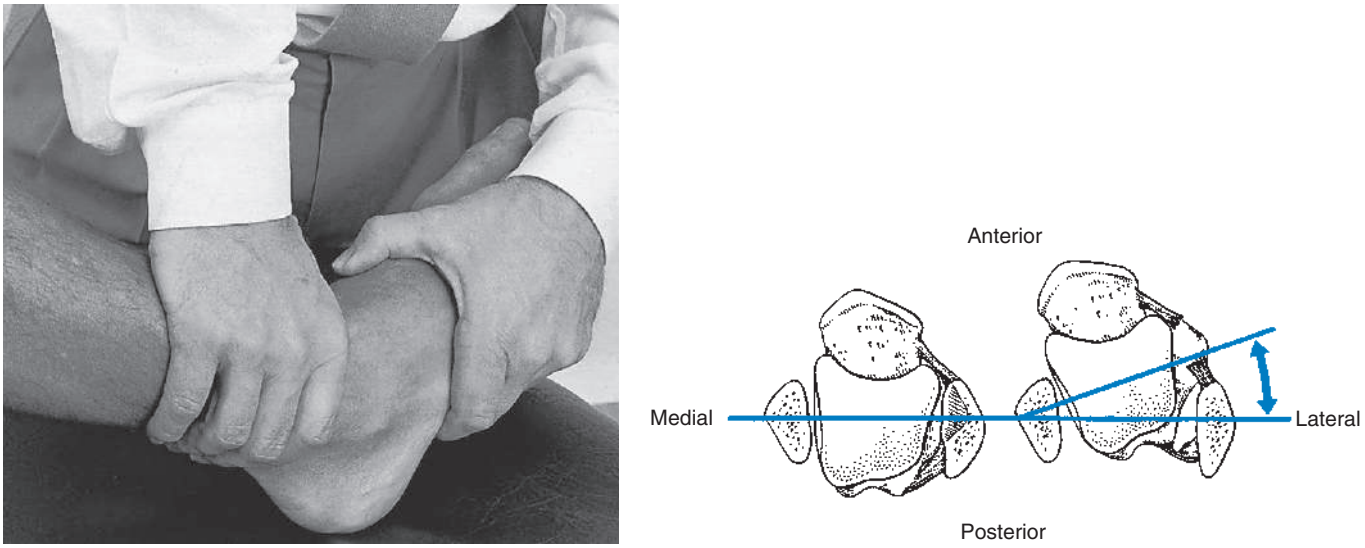


Fig 58.30 • Anterior drawer test for ligamentous insufficiency of the anterior talofibular ligament.

Fig 58.31 • Positive anterior drawer test demonstrating ligamentous insufficiency of the anterior talofibular ligament.

Functional instability

Cyriax[148] first introduced the concept of functional instability in 1954 and experimental support was provided by Freeman in 1965.[149,150]

This concept views instability after an ankle sprain as being merely the result of a failure of the muscles of leg and ankle to afford active protection, rather than the result of rupture or elongation of the lateral ligaments.[151,152] The sprain causes neurological damage at ligaments and capsule, which results in a prolonged propriosensory reflex. The increase in the peroneal reaction time is then responsible for inadequate protection against further sprain.[153,154] However, arthromuscular reflex can be trained by movement and by coordination exercises on a 'wobble board'.[155,156]

The re-education process is started on a see-saw block or a tilting board curved in one plane. While maintaining equilibrium on one leg, the patient tries to prevent either end of the block from touching the floor. The next step in regaining proprioception is training on a 'wobble board', first in a standing position, and later with tiptoeing and jumping or with some weights added to the wobble board.[157]

A recurrent varus sprain can also result from neurological weakness of the peroneal muscles, which are not strong enough to prevent inward rotation of the ankle. It is important to remember that the first complication of an upper motor neurone lesion is very often a recurrent varus sprain at the ankle.

Another condition leading to recurrent ankle sprains on a basis of weak peroneal muscles is sciatica with a fifth lumbar root palsy. Patients with a palsy of the fifth root should therefore be warned to protect the ankle until the palsy has recovered. The best way to do this is to wear a floated heel at the outer border of the shoe. Many running shoes designed for athletes have a built-in floated heel that prevents an inversion movement at the heel.

It is a remarkable fact, however, that in some conditions in which there is serious weakness of the peroneal muscle, as in peroneal atrophy, the ankle will very rarely be repeatedly sprained.

Differential diagnosis of instability

Recurrent ankle sprains and fear of the ankle 'giving way' must be distinguished from a number of other disorders at the outer side of ankle and foot which give rise to sudden pain, clicks and twinges.

- If there is a history of previous sprain, apart from chronic peroneal tendinitis, the differential diagnosis is a chronic ligamentous adhesion (Table 58.8).
- If there is no clear history of previous ankle sprains, instability must be differentiated from loose bodies in the ankle and subtalar joints, 'snapping peroneal tendon', chronic peroneal tendinitis and jumper's sprain.

Table 58.8 Differential diagnosis and treatment of recurrent disability at the outer side of the foot

	Instability	Chronic sprain
History	Ankle turns over easily and is subjected to a succession of minor sprains, with disability for some days Fear of the ankle 'giving way'	Foot is adequate for ordinary purposes but tends to swell and ache after vigorous or prolonged use No sudden aggravation or twinges
Examination (between attacks)	**1.** Anterior talofibular ligament: Normal routine examination Sometimes excessive range of inversion End-feel normal or 'empty' Positive anterior drawer test **2.** Unstable mortice: Reproduction of click and pain during strong varus movement at ankle Radiography shows increased distance between the two malleoli during forced varus **3.** Defective propriosensory reflex: Normal clinical examination L5 palsy or upper motor neurone lesion Weak peroneal muscles	**1.** Adhesions of talofibular and calcaneocuboid ligaments: Pain at outer side of ankle and foot during full inversion and plantiflexion Slight limitation End-feel more tense than on the opposite side Resisted movements are negative **2.** Chronic tendinitis of peroneal muscles: Passive inversion during plantiflexion hurts at outer side of ankle and foot End-feel normal Pain during resisted eversion
Treatment	**1 & 3.** Wobble board training, floated heel, taping or surgery **2.** Sclerosing injections into the distal tibiofibular ligament over 3 consecutive weeks, or surgery **3.** Floated heel	**1.** Adhesions rupture by manipulating the ankle and foot after vigorous deep transverse friction at the exact location **2.** Deep transverse friction 3 times a week, for 2–4 weeks The patient should not use the foot more than strictly necessary until cure is complete

Eversion sprain

An eversion sprain at the ankle is rare. When this condition is encountered, the foot should always be examined in an effort to discover why the sprain has occurred.

Mechanism

The deltoid ligament is strong. The position of the ankle and foot is such that excessive eversion movement does not take place unless the patient already stands with a valgus deformity at the heel. Alternatively, a strong valgus movement can produce bone damage rather than a ligamentous lesion. The deltoid ligament is so strong that eversion injury tends to cause avulsion of the tibial malleolus rather than tearing of the ligament.[158]

Diagnosis

The anterior fasciculi of the deltoid ligament are stretched during a combined plantiflexion–eversion movement. Palpation reveals the localization of the lesion, which invariably lies at the ligamentoperiosteal junction along the inferior border of the medial malleolus. Simultaneous posterior tibial tendinitis often occurs with a sprain of the deltoid ligament. If this is the case, resisted inversion is also painful. An avulsion fracture should be suspected when a strong varus movement also causes significant pain at the inner side of the ankle.

Treatment

Sprains of the anterior and middle fasciculi of the deltoid ligament are likely to continue causing pain for many months, or even years. The reason is that the patient, who stands with the heel in valgus deformity, overstretches the damaged tibionavicular or tibiocalcaneal ligament each time the foot is put on the ground. As each step causes a renewed strain, the fascicular tear never has a chance to heal. The 'chronic' lesion is caused by repeated traction; consequently, the worst possible treatment is manipulation, which overstretches the inflamed ligament. It is entirely wrong to compare this lesion with chronic adhesions at the lateral side of the ankle.

Treatment consists initially of relief from tension. Thus, a support (1–2 cm thick) must be fitted under the heel and the inner midtarsal area. This gives the calcaneus a neutral position and prevents further tension on the ligaments. Although it prevents the ligament from further overstretching, it does not always cure the existing inflammation. Therefore, some triamcinolone should be injected into the affected area, quickly reducing the inflammation and so leading to cure.

Massage is totally ineffective in this condition.

Technique: infiltration

The patient lies supine with the hip and leg rotated outwards. The foot is held in eversion and slight dorsiflexion, rendering the medial malleolus more prominent. The precise site and extent of the painful area are determined by careful palpation.

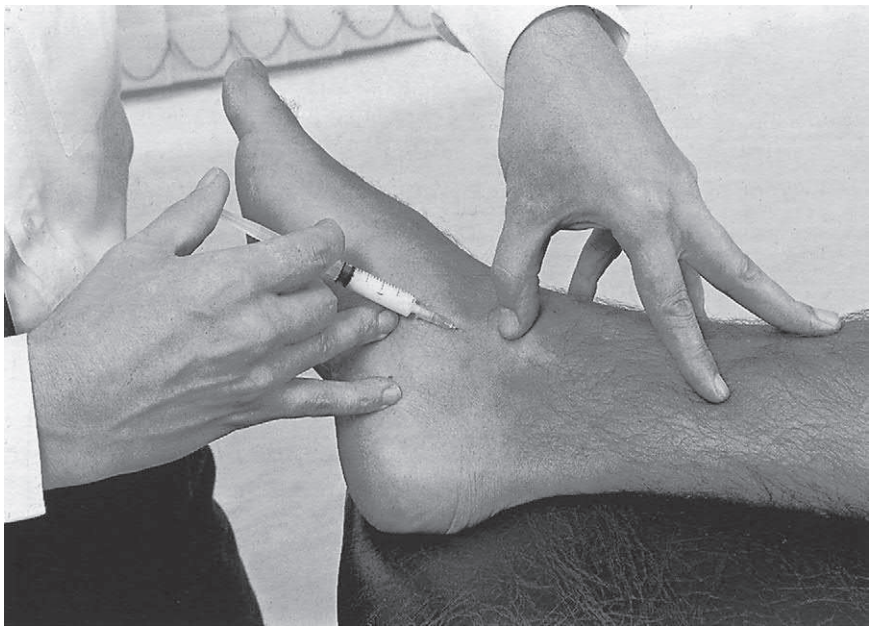


Fig 58.32 • Infiltration of the deltoid ligament.

A 2 mL syringe is filled with 20 mg of triamcinolone and fitted to a thin needle, 2.5 cm long. A point is chosen about 2 cm below the medial malleolus. The needle is inserted here and pushed upwards through the ligament until it hits bone (Fig. 58.32). By means of a series of partial withdrawals and reinsertions, droplets of the suspension are injected along the affected extent of the ligamento-osseous junction, each time with the needle making contact with bone. As the deltoid ligament is a thick structure, the infiltrations should be made superficially and deeply.

After the injection, the ankle is likely to be sore for 24–48 hours. Meanwhile, the patient should use the affected foot for weight bearing as little as possible.

For a comprehensive table summarizing the differential diagnosis of lesions at the heel and ankle, see online chapter *Differential diagnosis of lesions at the heel and ankle*.

Access the complete reference list online at **www.orthopaedicmedicineonline.com**

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Disorders of the midtarsal joints

59

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The talonavicular and calcaneocuboid joints and the tarsometatarsal joints comprise the midtarsal joints. Functionally, they act as one structure. Movement is possible in six directions: dorsiflexion–plantiflexion, adduction–abduction and pronation–supination. It is important for the ankle and subtalar joints to be stabilized when these movements are performed; this is achieved by dorsiflexion in the ankle joint and strong valgus pressure at the heel.

Due to the obliquity of the joint surfaces and the greater mobility of the talonavicular joint, dorsiflexion is usually accompanied by some abduction, whereas plantiflexion induces adduction.

At the distal (Lisfranc's) joint, considerable rotation round the second metatarsal shaft is possible (Fig. 59.1). Because of the specific structure of the joint line, plantiflexion of the metatarsals will always be accompanied by a movement towards the second metatarsal. Therefore a plantiflexion movement at the midtarsal joint increases the curvature of the anterior arch, whereas dorsiflexion is associated with flattening.

Capsular pattern

The capsular pattern at the midtarsal joints is increasing limitation of adduction, supination, plantiflexion and dorsiflexion (Fig. 59.2). The mid-foot finally fixes in an abducted and pronated position because of a spasm of the peroneal muscles.

Subacute arthritis in adolescence

The sign that first draws attention to this disorder is spasm of the peroneal muscles. Therefore it has been called 'spasmodic pes planus', which is a misnomer because it is not the spasm but the underlying capsulitis that causes the pain. Spasm is never primary but results from inflammation of the joint.

The arthritis is caused by overuse at the midtarsal joints. It is now an uncommon condition that occurs only in boys aged between 12 and 16 years. Cyriax mentioned the cause as being overuse, resulting from standing for long periods, which used to happen in the transition from school to work. Both the midtarsal joints and the talocalcaneal joints are affected. The condition is usually bilateral.

The first sign that draws attention to the disorder is a clumsy gait, which does not go unnoticed by the parents. There is also some discomfort on standing. Walking and running do not cause pain and there is also a complete absence of pain when sitting or lying down.

Clinical examination

Inspection reveals a long, thin foot, sometimes with some degree of pes cavus. An eversion deformity at the heel and the

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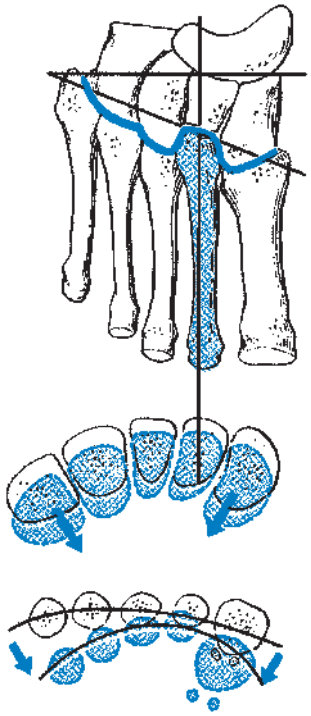

Fig 59.1 • At Lisfranc's joint, rotation round the second metatarsal shaft is possible. Because of the specific joint line, plantiflexion of the metatarsals is always accompanied by a movement towards the second metatarsal.

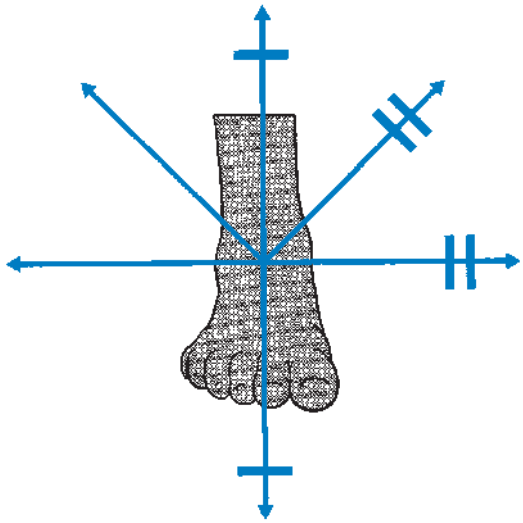

Fig 59.2 • The capsular pattern at the midtarsal joints.

mid-foot is seen to be maintained by a spasm of the peroneal and extensor digitorum longus muscles. In standing, the peroneal tendons are visible as a prominent tight band behind and below the lateral malleolus.

Functional examination shows a limitation of varus at the subtalar joint and limitation of adduction and supination at the midtarsal joints. These movements are prevented by muscle spasm. In long-standing cases, contraction of the joint capsules may increase limitation of the already restricted movements. For diagnosis, it is vital to detect the muscle spasm of capsular contracture during clinical examination in the lying position.

A radiological evaluation of ankle and foot reveals no abnormality.

Natural history

The natural history is for symptoms to subside and cease after 2 years. By that time, the foot has become permanently but painlessly fixed in the deformed position. A limp results. The disorder can also lead to later valgus sprains or lesions of the tibialis posterior and flexor hallucis longus tendons.

Treatment

In the early stage, the essence of treatment is relief of weight bearing and support for the joint. Cyriax[1] (pp. 436–438) advised that: 'The lad … requires (a) a sedentary job, (b) a bicycle, (c) … [an inner] wedge on the heels of his shoes and (d) strapping of the joint.'

The patient is first told how his condition is provoked and he must understand that avoiding weight bearing is the important part of the treatment. He should never stand if he can help it; he must seek a sedentary job, should sit when at home and should use a bicycle rather than walk.

To bring the heels towards a varus position, the shoes are fitted with inner wedges and non-elastic strapping is worn as often as possible. The patient must maintain this regime for 6–12 months until he regains a full range of movement at the subtalar and midtarsal joints. Recurrence is uncommon once this stage is reached.

In advanced cases, the subtalar joint will have become fixed in a valgus position by considerable spasm of the peroneal muscles. Only slight movement can be obtained at the midtarsal joints. If no structural contracture of the ligaments about the talocalcaneal and midtarsal joints has yet occurred and the valgus deformity is only caused by peroneal spasm, immobilization in a plaster cast, with the foot held in varus position, can be tried. The cast, from below the knee to the toes, should be worn for 6 weeks to 2 months. To obtain this position, the peroneal nerve must temporarily be blocked at the point where it curls round the neck of the fibula.

Subacute arthritis in middle age

The patients are usually overweight women in their 40s, 50s or 60s. Overuse is the common cause of the arthritis but sometimes the condition is due to an isolated sprain. The pain, limitation of movement and muscular spasm are less pronounced than in arthritis in adolescence. The condition does not seem to have a natural history: without treatment it appears to continue indefinitely but without much alteration. Cases that have persisted for 2 or 3 years are not uncommon.

Usually the arthritis is unilateral. Clinical examination reveals a capsular pattern of both the subtalar and midtarsal joints: the peroneal spasm restricts varus at the hindfoot and adduction–supination at the mid-foot. The radiological appearances are normal.

Treatment

If only one joint is affected, infiltration with triamcinolone can be successful, provided prophylaxis against recurrence is given.

If more joints are affected, which is usually the case, the following treatment is instituted:

- Modified rest and relative relief from weight bearing
- Strapping the joints in varus and supination
- Tilting the heel in varus position by a medial wedge.

If all these measures are taken, the arthritis can be expected to clear up after 6 months, but the patient should guard against renewed overuse.

Rheumatoid arthritis

Rheumatoid or reactive types of arthritis can attack the midtarsal joints. If the arthritis is severe, the patient can walk only with difficulty, and pain at rest is so intense that sleep becomes disturbed.

As well as marked fixation in valgus and abduction–pronation, clinical examination shows gross synovial thickening, warmth to the touch and tenderness. If the oedema allows, the synovial thickening can be palpated over the dorsum of the joint.

A very effective local treatment is immobilization in a plaster cast, which eases the pain within a few days; however, if the cast is removed too soon, pain quickly returns.

Spontaneous recovery takes 1 or 2 years. During this period, the joint becomes fixed in abduction–pronation.

Non-capsular pattern

Midtarsal strain

If an excessive strain is imposed on the midtarsal joints – for example, as the result of a deformity (e.g. plantaris) – the power of the musculature (tibialis posterior and flexor hallucis longus) becomes insufficient to maintain the longitudinal arches during weight bearing.[2] After some time, the midtarsal ligaments become stretched, elongate and undergo inflammatory changes, so that pain results. Elongation promotes excessive movements at the midtarsal joints, which cause the plantar arch to flatten during weight bearing. As a result, the forefoot dorsiflexes and abducts, which causes the inner side of the foot to become prominent and further overstretches the calcaneonavicular ligament. In due course, the excessive motion of the joints and their poor alignment leads to inflammation of the capsule and to structural damage of the articular surfaces, which finally results in structural deformity and arthrosis.

Mechanism

To understand the mechanism of midtarsal strain, the ankle and foot should be seen as complex structures with an intimate interaction between position and function of their different components.

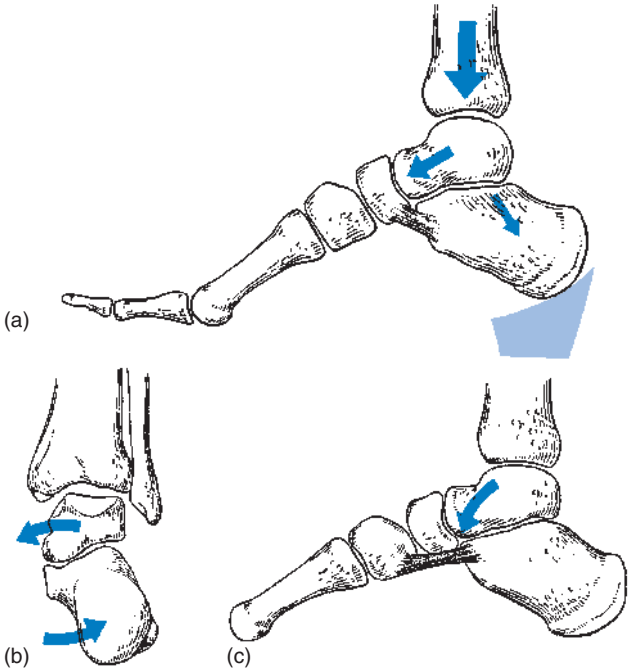

Fig 59.3 • Midtarsal strain: (a) movement of the talus forwards on the calcaneus; (b) medial pressure forcing the calcaneus towards valgus; (c) downward pressure of the talus causes increasing depression of the longitudinal arch and increasing stress on the plantar ligaments.

Initially, the body weight bears on the talus via a downward thrust through the tibia. The talus is supported by the calcaneus, on which it lies obliquely. The former therefore has a tendency to glide in a medial and forward direction, especially in patients with an equinus deformity at the ankle or in patients with too short plantiflexor muscles. In women who wear high, oblique heels, the talus also tends to be pushed forwards on the calcaneus (Fig. 59.3a).

The forward and medial gliding of the talus imposes medial and downward pressure on the anteromedial calcaneal border. The medial pressure forces the calcaneus towards valgus, which may be furthered by a shortening of the Achilles tendon (Fig. 59.3b). The downward pressure of the talus evokes dorsiflexion at the talonavicular joint.[3] This increases the depression of the longitudinal arch and can be responsible for greater stress on the plantar ligaments and the fascia plantaris (Fig. 59.3c). The downward pressure of the talus and the consequent dorsiflexion at the talonavicular joint will initiate a number of other events, which are the source of more deformity and trouble:

- Because of the obliquity of the joint surfaces, each dorsiflexion in the midtarsal joints is accompanied by some abduction. The downward movement of the talus is thus at the origin of an *abduction* deformity in the mid-foot. This dorsiflexed and abducted foot overstretches the inner ligaments (calcaneonavicular ligament and capsule of the talonavicular joint).

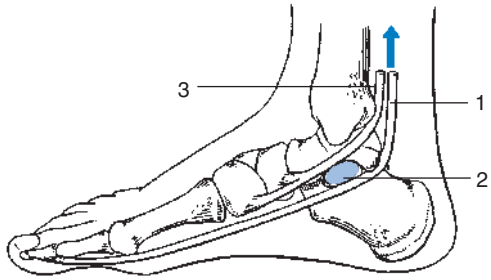

Fig 59.4 • The sustentaculum tali (2) is propped upwards by contraction of the flexor hallucis longus (1); tibialis posterior (3).

- Dorsiflexion at the midtarsal joint also causes spreading out and abduction of the metatarsals. Abduction is the result of obliquity of the joint line. Spreading out is caused by the specific cone-shaped form of the bases of the metatarsals, which move their heads away from the centre during extension (see online chapter *Applied anatomy of the lower leg, ankle and foot*). This flattens the anterior arch and results in a splay foot.

Thus, on account of an initial forward and medial gliding of the talus, a number of events take place:

- Valgus of the calcaneus with possible shortening of the Achilles tendon
- Dorsiflexion and abduction of the mid-foot, with flattening of the longitudinal arch and tension in the plantar and medial ligaments
- Flattening and abduction of the forefoot, with spreading of the metatarsal heads and loss of the anterior arch
- In due course, there will also be some effect on the tendons: that of the tibialis posterior, the first invertor of the foot, suffers strain and becomes inflamed. The tendon of the flexor hallucis longus, which is not only an invertor, but also, through its position under the sustentaculum tali, has a specific function as a stabilizer of the anterior part of the calcaneus, also becomes strained (Fig. 59.4).

The mechanism of midtarsal strain is summarized in Fig. 59.5.

Clinical examination

As midtarsal strain advances, three clinical grades can be distinguished.

Postural pain

The plantiflexion–dorsiflexion range of movement increases and therefore the abduction–pronation range of the foot too. Due to this laxity, the foot becomes 'wobbly', but neither structural changes nor clear signs of overstretched tendons and ligaments appear. There is muscular fatigue and pain after long periods of standing or after prolonged walking. Clinical examination reveals only laxity in the midtarsal joints.

Midtarsal 'strain'

This appears when the joint capsules and the ligaments become inflamed. On account of the excessive mobility and poor alignment of the joints, the protective muscular action of the tibialis anterior and flexor hallucis longus are overwhelmed and they

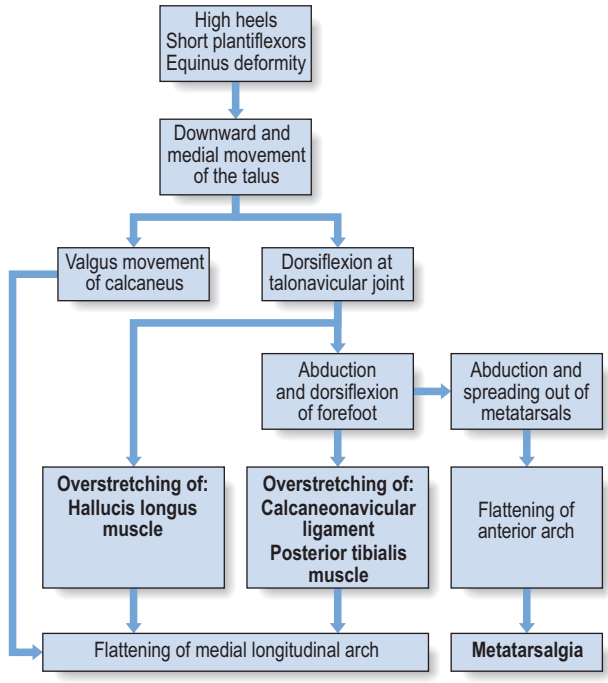

Fig 59.5 • Summary of the mechanism of midtarsal strain.

start to become inflamed. There is pain during and after weight bearing. The deformity of the foot is functional only. No structural changes appear at this stage. Clinical examination reveals excessive mobility in the joints, together with pain at the extremes of range, especially rotation. Resisted inversion of the foot and resisted plantiflexion of the big toe may be painful, because of tendinitis of the tibialis posterior or of the flexor hallucis longus.

Structural changes[4]

Due to abduction of the forefoot on the hindfoot, the medial side of the foot becomes prominent. Talonavicular arthrosis supervenes, with osteophytes at the dorsum and the inner side of the joint. As the navicular bone is now permanently depressed, painful and persistent overstretching of the talonavicular ligament results. Apart from the visible deformities at the inner side and the dorsum of the foot, the clinical findings are permanent fixation of the foot in abduction–dorsiflexion, some limitation of rotation with a hard end-feel, and tenderness at the talonavicular ligament.

Treatment

Heel raising

The heel must be raised *horizontally* (Fig. 59.6) so as to allow the forefoot to adopt a more plantigrade position in relation to the talus when weight is borne. A slight medial wedge can be added to correct the valgus position of the heel. Attempts should be made to stretch the triceps muscle if there is shortening.

Exercises for the short plantiflexor muscles

Exercises (faradic and resisted) should be given for the short plantiflexor muscles of the sole, especially the adductor hallucis,

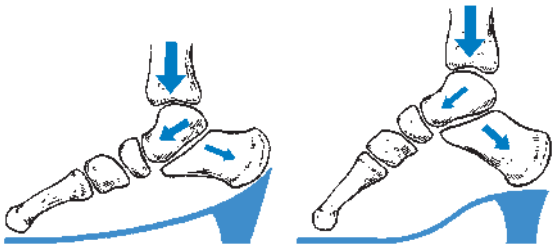

Fig 59.6 • The horizontal raised heel on the right reduces the forwards force on the talus.

flexor hallucis brevis, flexor digitorum brevis and quadratus plantae. The main purpose is to make them adequate to take the strain and thus to relieve some tension on the ligaments.

Exercises for the invertors

Exercises for the invertors should also be given; strengthening them protects the foot against further pronation–abduction. A strong and functional flexor hallucis longus muscle plays a role in 'propping' the talus upwards by lifting the sustentaculum tali (see Fig. 59.4). The posterior tibial muscle, the key dynamic support of the medial longitudinal arch of the foot, should also be strengthened.[5]

Mobilization of the joints

Mobilization of the midtarsal joints aims to allow a full range of motion to be painlessly achieved. During the initial stages, the range of movement is excessive. Nevertheless, repeated strains, followed by healing of minor ruptures, lead to the formation of painful adhesions. The self-perpetuating inflammation in these elongated ligaments is the main reason for the discomfort at the end of range. Therefore treatment also includes manipulation to break the ligamentous adhesions. This is the only area of the body in which manipulations are required at a joint that already has an excessive range of motion.

Technique: manipulation

The patient lies in a supine position on a high couch. The manipulator sits facing the patient's foot. As great strength is required and the manipulator has to work without leverage, a good starting position is vital. The heel of the ipsilateral hand is placed at the dorsum of the first metatarsal bone, and the heel of the contralateral hand is placed at the plantar aspect of the fourth and fifth metatarsal bones. Both hands are clasped about the outer and dorsal aspect of the forefoot. The foot is pressed slightly towards dorsiflexion (Fig. 59.7). Rotation is now imparted to the forefoot by a swinging movement in the shoulders and elbows, which causes the upper hand on the inner side to press towards the manipulator and the outer hand to press away from him. This movement is repeated in a rhythmic manner for a few minutes.

Steroid infiltration

Sometimes there is marked and persistent tenderness of the calcaneonavicular ligament, which calls for infiltration with triamcinolone. The injection is repeated after 2 weeks.

Technique: infiltration

The patient lies in a supine position on a high couch. The sustentaculum tali and the navicular bone are identified.

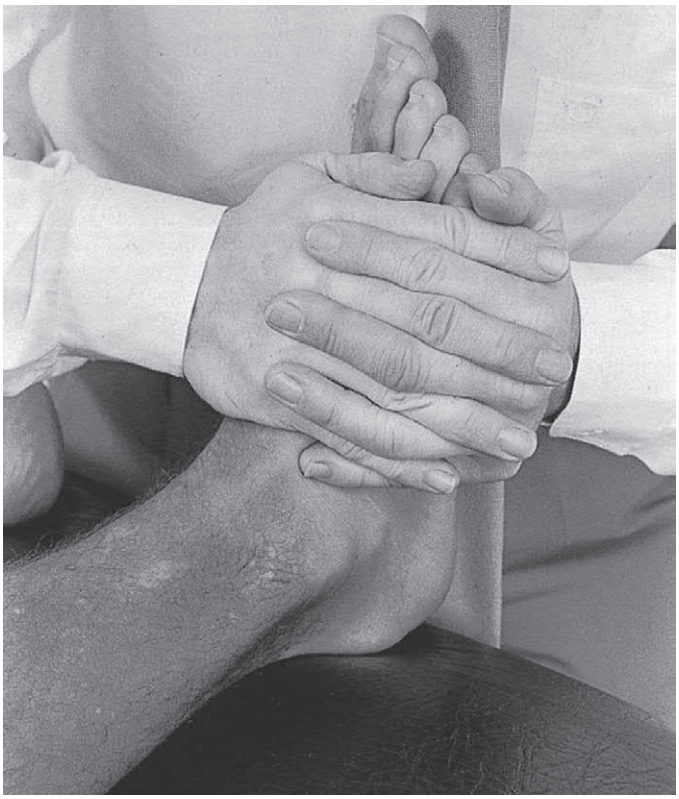

Fig 59.7 • Manipulation to break adhesions at the midtarsal joints.

Between them, the tender ligament can easily be palpated. A thin needle, 2 cm long, is fitted to a 2 mL syringe filled with triamcinolone.

The palpating thumb remains on the tender ligament and the needle is introduced between the thumb and bone (Fig. 59.8). The point of the needle is then aimed towards the calcaneal border of the ligament, where 1 mL is injected. Another 1 mL is then infiltrated at the navicular insertion of the ligament.

Deep friction

If the flexor hallucis longus or the tibialis posterior tendons are at fault, deep friction is indicated (see Ch. 57).

Surgery

If conservative management fails, the painful midtarsal joints are best stabilized by arthrodesis. In the past, triple arthrodesis was used extensively[6]; nowadays, more limited single-joint fusions are used which allow more preservation of the hindfoot motion, resulting in better function.[7–10]

Conservative treatment of midtarsal strain is summarized in Box 59.1.

Aseptic necrosis

Osteochondritis dissecans at the navicular bone appears in boys aged between 4 and 12 years and is known as Köhler's disease I. There is pain and limitation of the midtarsal movements. The diagnosis is made from a radiograph or a bone scan.[11]

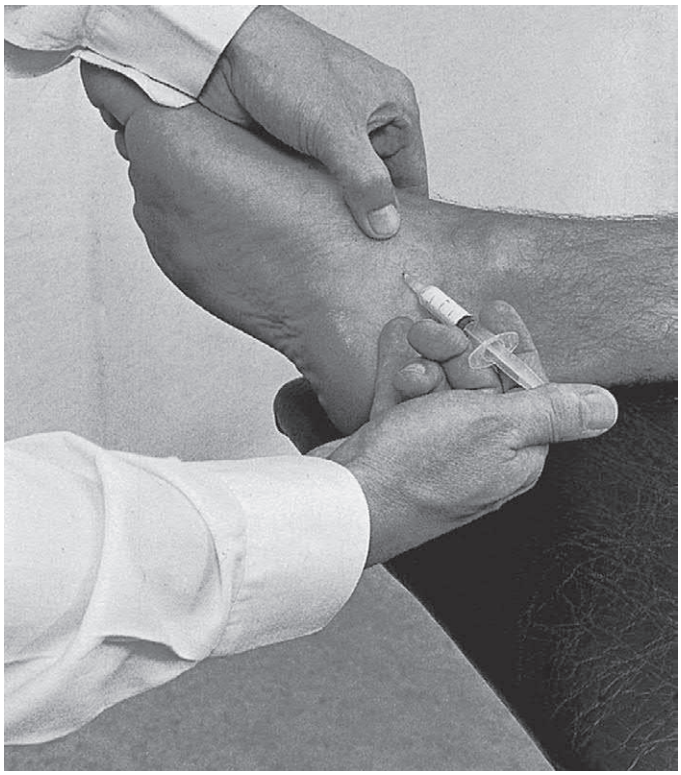

Fig 59.8 • Steroid infiltration of the calcaneonavicular ligament.

Box 59.1

Conservative treatment of midtarsal strain

- Elevated horizontal heel
- Training of short plantiflexor muscles
- Training of tibialis anterior and flexor hallucis muscles
- Mobilization of the midtarsal joints
- (Infiltrations to the strained ligaments)
- (Deep friction to the strained tendons)

The course of the disease process is benign and self-limiting.[12,13] Treatment is symptomatic and consists of weight-bearing plaster casts over 3 months.[14] With or without treatment, there is spontaneous reconstitution of the navicular bone and excellent recovery of function after an average duration of 15 months.[15,16]

Navicular stress fracture

Navicular stress fractures account for about 15% of all stress fractures.[17] They typically occur in running and jumping athletes, in whom repetitive cyclic loading results in fatigue failure of the navicular bone, the cornerstone of the medial arch. Biomechanical analysis of navicular motion during stride[18] reveals that most of the impingement force is focused at the central third of the navicular bone, which is also an area of relative avascularity.[19] This explains why the characteristic fracture is located in the central third of the bone.

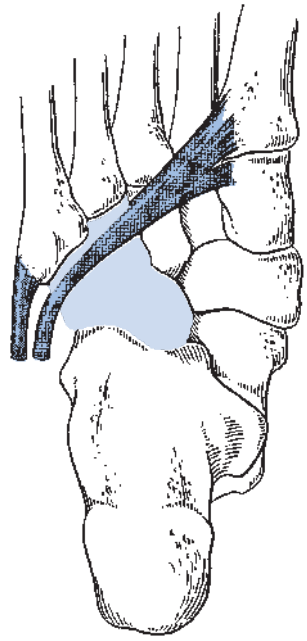

Fig 59.9 • The relationship of the cuboid bone and the tendons of the peronei.

Navicular stress fractures typically present in a running athlete who has gradually increasing pain in the dorsal midfoot, with occasional radiation of pain down the medial arch.

On examination, neither swelling nor discoloration is visible. Rising on tiptoe may be painful, as is the passive plantiflexion–dorsiflexion movement at the midtarsal joints. Clinical suspicion is raised when tenderness is elicited over the 'N spot', a nickel-sized area at the central region of the proximal dorsal navicular bone.[20] Tenderness is strongly associated with navicular stress fractures.[21]

Early diagnosis can be made by ultrasound, which is a good screening test for stress fractures.[22] Computed tomography (CT) is the best tool in the diagnosis of navicular stress fracture. It allows differentiation between stress fracture and stress reaction, and enables accurate fracture definition.[23]

Non-displaced fractures respond well to 6 weeks of non-weight-bearing cast immobilization. Displacement, comminution and delayed or non-union fractures are indications for surgical open reduction and internal fixation.[24,25]

Cuboid rotation

Subluxation of the cuboid bone occurs when a strong pull exerted by the tendon of the peroneus longus muscle causes rotation of the bone[26] (Fig. 59.9). The lateral side of the cuboid is tilted upwards and the medial side becomes depressed. The cuboid is locked in this subluxated position and pain results. Newell and Woodle[27] found this condition in some 4% of athletes complaining of pain in the midtarsal region. The condition seems to be more common in patients who have pronated feet.

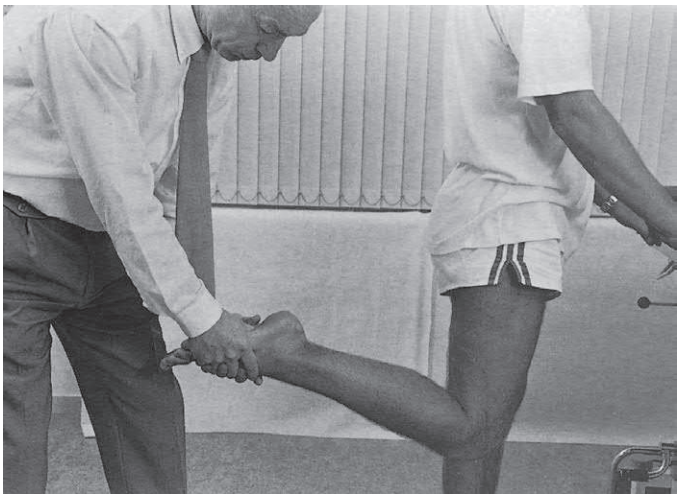

Fig 59.10 • Manipulation for cuboid rotation.

The diagnosis is primarily subjective, and must be made on the basis of the patient's history and physical findings. The patient, most often a long-distance runner or dancer, complains of lateral mid-foot pain during and after activity. Inspection may reveal a subtle forefoot valgus, a shallow depression on the dorsal surface of the foot and a palpable fullness on the plantar aspect of the cuboid. Clinical examination usually shows end-range pain during midtarsal movements and/or some limitation of dorsiflexion–plantiflexion. In addition, pressing on the plantar surface of the cuboid in a dorsal direction produces pain.[28,29]

Documentation by radiograph, CT scan or MRI is difficult. Treatment is manipulation (the 'cuboid whip').[30]

Technique: manipulation[31,32]

The patient stands with the back to the manipulator and holds on to a couch or a chair. The knee is flexed at a right angle. The physician grasps the foot with both hands so that the fingers are placed at the dorsum of the forefoot and the crossed thumbs over the plantar aspect of the cuboid (Fig. 59.10). The manipulation is now performed as a quick upward 'whiplash', while the thumbs, at the plantar aspect, apply strong downward pressure. The manipulator's elbows should be close together and the arms, wrists and hands should be fully relaxed.

In long-standing cases, or when the foot is pronated, it may be necessary to prevent recurrences with an orthotic device.

Midtarsal arthrosis

Arthrosis of the midtarsal joint can be the result of a navicular fracture, an old navicular apophysitis or an ordinary midtarsal strain.

A fracture or apophysitis can lead to gross deformities with serious disturbances to the anatomy of the foot. If this is the case and serious problems result, a steel support moulded accurately to the sole of the foot will minimize movements at the disorganized joints. If this does not lead to improvement, arthrodesis can be considered.

However, the diagnosis 'midtarsal arthrosis' is very often a misnomer because it is based only on deductions from radiological appearances. The common cause of the radiologically visible osteophytes at the dorsum of the talonavicular joint is a long-standing midtarsal strain. Once again, it is neither the osteoarthrosis nor the osteophytes that cause trouble, but rather the underlying ligamentous strain. If the ligaments are strained, treatment for midtarsal strain must be applied, whether there is radiological evidence of osteoarthrosis or not.

Lesions of the cuneiform–first metatarsal joint

The cuneiform–first metatarsal joint permits not only plantiflexion and dorsiflexion movement but also slight rotation around the base of the second metatarsal.

During routine functional examination of the foot, movements at the cuneiform–first metatarsal joint cannot be distinguished from those at the talonavicular and cuneiform–navicular joints. If a lesion of the cuneiform–first metatarsal joint is suspected, the joint must be examined separately after the routine examination.

The disorders that occur at this joint are:

- Osteoarthrosis
- Gout
- Loose body.

Osteoarthrosis

This condition usually results from previous osteochondritis (Cyriax[1]: p. 439). There is a similarity to hallux rigidus, also known to be the result of osteochondritis.[33]

The patient is an adolescent and more girls are affected than boys. The condition is usually bilateral and the onset insidious. One day the patient finds that, if wearing tight shoes, localized pain arises at the site of small projections at the dorsal and medial aspect of the foot. There is no problem unless something catches against the prominences. Rarely, the onset is sudden and the patient says that both joints became tender and swollen for about a week. After this severe phase, there is a permanent and small prominence on each foot.

There is a visible and palpable outcrop at the dorsum of each foot. If the condition is acute, there is also some swelling and local tenderness. Clinical examination is usually largely unrevealing except for slight stiffness of the range of plantiflexion–dorsiflexion. A radiograph confirms the diagnosis.

Treatment

If the arthritis is acute, a few days' rest is advised with weight bearing only in a high-heeled shoe, no part of which should touch the joint. As the condition is harmless and recurrent pain only stems from the pressure exerted when lace-up shoes squeeze the skin against the osteophytes, the treatment is to prevent this pinching. For girls, it is easy to find shoes that have no part touching the joint; alternatively, a felt ring around the

Table 59.1 Differential diagnosis and treatment of disorders at the mid-foot

Disorder	Presentation	Examination	Treatment
Subacute arthritis in adolescence	Boys Clumsy gait/slight pain	Capsular pattern with peroneal spasm	Early cases – prevention: Sedentary job Raised heel Joint strapping Long-standing cases: Plaster cast Immobilization
Subacute arthritis in middle age	Overweight women in their 50s or 60s	Capsular pattern with muscle spasm Differential diagnosis: gout and rheumatoid arthritis	Modified rest Joint strapping and tilting the heel Triamcinolone infiltration
Midtarsal strain	Pain at the mid-foot	Abduction and dorsiflexion in the mid-foot No muscle spasm First stage: 'Wobbly' foot with increased range Pain at the end of dorsiflexion/abduction Later stages: Structural deformity Strained ligaments Strained invertor muscles	Raising the heel, exercise to the short plantiflexor muscles, mobilization of the midtarsal joints
Midtarsal ligamentous contracture	Patients in their 50s and 60s Previous plaster immobilization Pain during exercises	Pain at the end of range No muscle spasm No increase in range of mobility	Infiltration of all the tender ligaments with triamcinolone
Stress fracture of the navicular bone	Long-distance runners Increasing pain during activity	Normal functional examination Local tenderness and pain during application of ultrasound	Rest
Aseptic necrosis of the navicular bone	Boys of 5–12 years	Pain and limitation Diagnosis is established by radiography	Support
Cuboid rotation	Athletes, with midtarsal pain during running	Full but painful range of rotation movements Tender ligaments at the inferior aspect of the cuboid bone	Manipulation
Midtarsal arthrosis	After apophysitis or fracture Gross deformities		Steel support Arthrodesis
Arthrosis at the cuneiform–first metatarsal joint	Teenagers with bilateral bony outcrop at the dorsum of the foot	Normal clinical examination Local tenderness	Prevention of pressure
Gout at the cuneiform–first metatarsal joint		Red, warm, swollen and exquisitely tender joint	Medication
Loose body at the cuneiform–first metatarsal joint	Athletes Sudden pain during a sprint	Normal clinical examination	Sustained traction
Chronic ligamentous adhesions at the calcaneocuboid joint	After an ankle sprain Persistent lateral pain during and after exercises	Pain during inversion/adduction and supination	Manipulation

bony outcrops can be used. For boys, this is sometimes more difficult to achieve and, if the inconvenience continues, the bony prominences have to be removed surgically.

It is important to note that gross osteoarthrosis of the cuneiform–first metatarsal joint may lead to fixation, with considerable plantaris deformation of the first metatarsal (fixation in plantiflexed position). This may, in turn, result in metatarsalgia of the first metatarsophalangeal joint or, more frequently, a lesion of the sesamoid–metatarsal joint. If this occurs, a support must be prescribed to take the weight off the head of the bone (see online chapter *Disorders of the forefoot and toes*).

Gout

It is not uncommon for gout to attack this joint, even before the metatarsophalangeal joint of the big toe.[34] If there is sudden pain, warmth, reddening of the skin, pain at night and extreme tenderness of the joint, gout should be considered.

Loose body

Athletes sometimes complain of sudden twinges at the medial border of the foot during sprinting. These are recurrent and appear in race after race but cannot be repeated voluntarily. Between races, the foot is normal and pain is not present when the foot is examined. However, localization shows that the twinges do not arise from the ankle or the subtalar joint. When the patient rises on tiptoe, subluxation of the metatarsal bone on the cuneiform does not occur.

Some internal derangement is very likely because of the typical history of sudden twinges and because the clinical examination is entirely negative.

Good results can be obtained by sustained traction on the big toe (up to 10 kg) for 30 minutes, two or three times a week.

Differential diagnosis and treatment of disorders at the midfoot are summarized in Table 59.1.

Access the complete reference list online at **www.orthopaedicmedicineonline.com**

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Applied anatomy of the cervical spine

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The anatomy of the cervical spine is complex and unique. To understand the diagnosis and treatment of the multiple disorders affecting this vital region, a thorough knowledge of the anatomy is necessary.

Bones

The cervical spine has seven vertebrae, which may be divided into two groups that are distinct both anatomically and functionally: the upper pair (C1 and C2, the atlas and axis) and the lower five (C3–C7) (Fig. 1).

Upper cervical spine

The upper cervical spine has the first and second vertebrae – the atlas and axis – and forms a unit with the occiput.

The atlas

The atlas (Fig. 2) does not have a vertebral body – this has been absorbed into the axis vertebra, to form the odontoid process (Fig. 3). A thick anterior arch remains, extending into and joining the two lateral masses, on which are the superior atlantal joint facets, which articulate with the occipital condyles; and the inferior joint facets of the axis. The posterior arch is thinner than the anterior arch and forms the posterior junction of the lateral masses. The large vertebral foramen thus formed has a larger diameter in the transverse than in the sagittal plane.

The transverse processes contain a transverse foramen through which the vertebral artery passes before it loops back above the upper surface of the posterior arch, which sometimes contains an arterial groove, although anatomical anomalies are frequently encountered.

The posterior aspect of the anterior arch has a facet for articulation with the odontoid process of the axis, which is held in place by the transverse ligament, spanned between two tubercles, which project from the inner sides of the lateral masses.

The axis

The second cervical vertebra is the axis (Fig. 3). Its vertebral body is formed by fusion with the vertebral body of the atlas to form the odontoid process (or dens), which is completely separate from the atlas. The laminae of the axis are very well developed and blend into a bifid spinous process.

Both transverse processes have a transverse foramen for the vertebral arteries. The superior articular facets of the axis articulate with the inferior articular facets of the atlas. The inferior articular facets of the axis articulate with the superior articular facets of the third vertebra.

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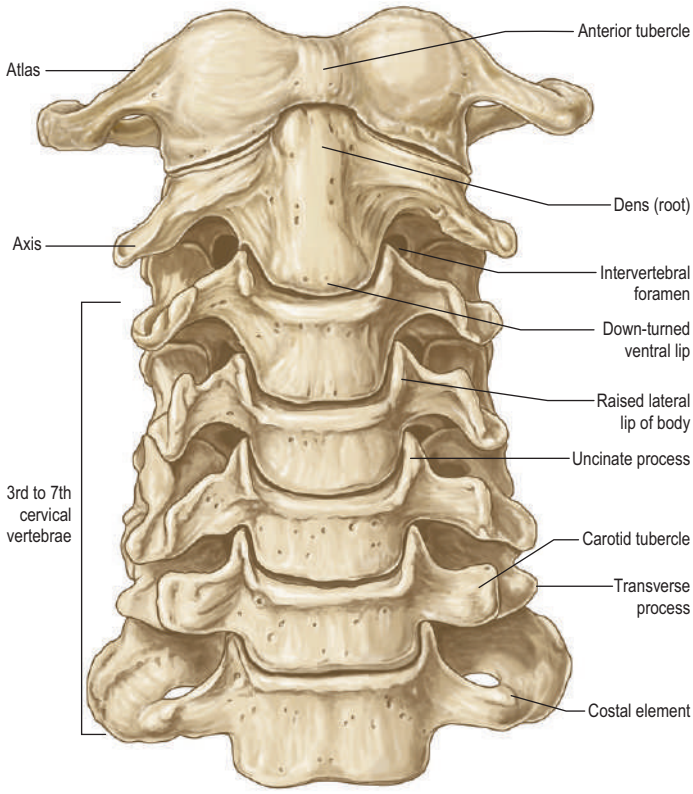

Fig 1 • Anterior view of the cervical spine. From Standring, Gray's Anatomy, 40th edn. Churchill Livingstone/Elsevier, Philadelphia, 2009 with permission.

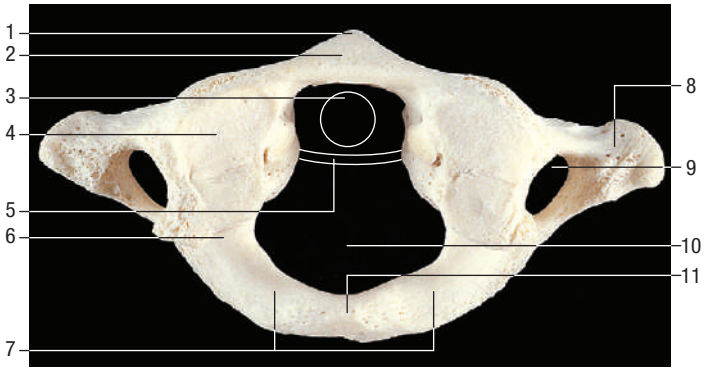

Fig 2 • The atlas, superior view. 1, Anterior tubercle; 2, anterior arch; 3, outline of the dens; 4, superior articular facet; 5, outline of transverse ligament; 6, groove for cervical artery and C1; 7, posterior arch; 8, transverse process; 9, foramen transversum; 10, vertebral foramen; 11, posterior tubercle. From Standring, Gray's Anatomy, 40th edn. Churchill Livingstone/Elsevier, Philadelphia, 2009 with permission.

Fig 3 • (A) Axis, superior view. 1, Dens – attachment of apical ligaments; 2, superior articular facet; 3, dens – attachment of alar ligaments; 4, foramen transversum; 5, pedicle; 6, spinous process; 7, body; 8, transverse process; 9, vertebral foramen; 10, inferior articular process; 11, lamina. **(B)** Axis, lateral aspect. 1, Dens – attachment of alar ligaments; 2, dens – facet for the anterior arch of the atlas; 3, groove for transverse ligament of atlas; 4, superior articular facet; 5, lateral mass; 6, divergent foramen transversum; 7, body; 8, ventral lip of body; 9, lamina; 10, spinous process; 11, inferior articular facet; 12, transverse process. From Standring, Gray's Anatomy, 40th edn. Churchill Livingstone/Elsevier, Philadelphia, 2009 with permission.

Lower cervical spine

The lower cervical spine is composed of the third to the seventh vertebrae which are all very similar. Each vertebral body is quite small (Fig. 4). Its height is greater posteriorly than anteriorly and it is concave on its upper aspect and convex on its lower. On its upper margin it is lipped by a raised edge of bone. The anteroinferior border of the vertebral body projects over the anterosuperior border of the lower vertebra.

Anterolateral (in the upper vertebrae) to posterolateral (in the lower vertebrae) on the upper surface of the body, two uncinate processes project upwards and articulate with the lower notches (or anvils) of the upper vertebra to form the joints of von Luschka or uncovertebral joints.

Laterally, the transverse processes have an anterior and a posterior tubercle, which are respectively the remnants of an embryonic rib and transverse process. The spinal nerve lies in

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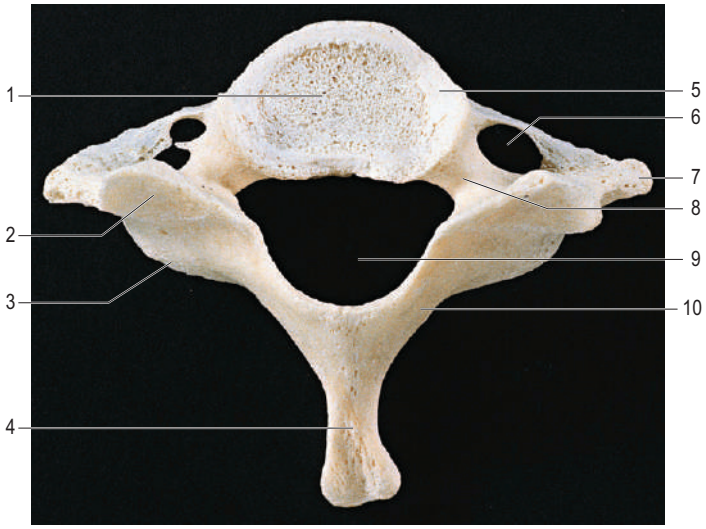

Fig 4 • Seventh cervical vertebra, superior view. 1, Body; 2, superior articular process; 3, inferior articular process; 4, spinous process; 5, uncinate process; 6, foramen transversum; 7, transverse process; 8, pedicle; 9, vertebral foramen; 10, lamina.
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the groove between the two tubercles. The transverse process also has a transverse foramen for the vertebral artery and vein. This is not so at C7, where the foramen encloses only the accessory vertebral vein.

The intervertebral foramina are between the superior and inferior pedicles. The articular processes for the articulation with the other vertebrae are more posterior. The two laminae blend together in a bifid spinous process (at C3, C4 and C5).

The spinous processes of C6 and C7 are longer and taper off towards the ends. C7 has a large spinous process and is, therefore, called the vertebra prominens.

Intervertebral discs

There are six cervical discs, because there is no disc between the upper two joints. The first disc is between the axis (C2) and C3. From this level downwards to the C7–T1 joint they link together and separate the vertebral bodies. Each is named after the vertebra that lies above: e.g. the C4 disc is the disc between the C4 and C5 vertebrae (Fig. 5).

The disc, comprised of an annulus fibrosus, a nucleus pulposus and two cartilaginous endplates, has the same functions as the lumbar disc, and so will not be discussed in detail (see Ch. 31, Applied Anatomy of the Lumbar Spine). There are, however, some differences (Table 1). At the cervical spine the discs are more effectively *within* the spine than they are at the thoracic or lumbar levels because of the superior concavity and inferior convexity of each vertebral body. They are also about one-third thicker anteriorly than posteriorly, which gives the cervical spine a lordotic curve that is not related to the shape of the vertebral bodies. The annulus fibrosus is also thicker in its posterior part than it is in the lumbar spine. The further down the spine, the more the nucleus pulposus lies anteriorly in the disc, and it disappears earlier in life than it does in the lumbar spine. For both these reasons, nuclear disc prolapses are uncommon after the age of 30.

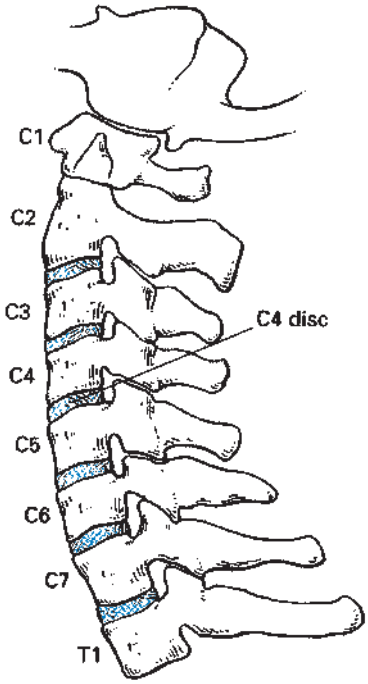

Fig 5 • The intervertebral discs.

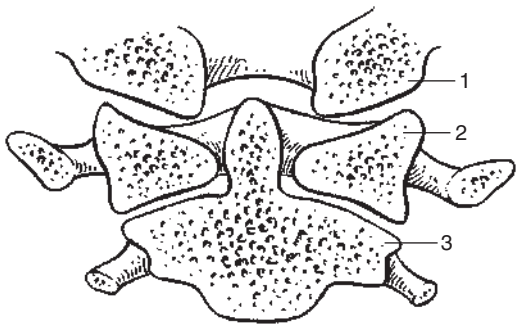

Fig 6 • The occipitoatlantoaxial joint complex: 1, occiput; 2, atlas; 3, axis.

Table 1 Differences between the cervical and lumbar discs

Cervical	Lumbar
Contained by vertebral bodies	Not contained by vertebral bodies
Thicker anteriorly than posteriorly	Equal height
Annulus thicker posteriorly	Annulus weaker posteriorly
Nucleus in anterior part of disc	Nucleus in posterior part of disc

Joints

The cervical spine is more mobile than the thoracic or lumbar. Its structure allows movements in all directions, although not every level contributes to all movements.

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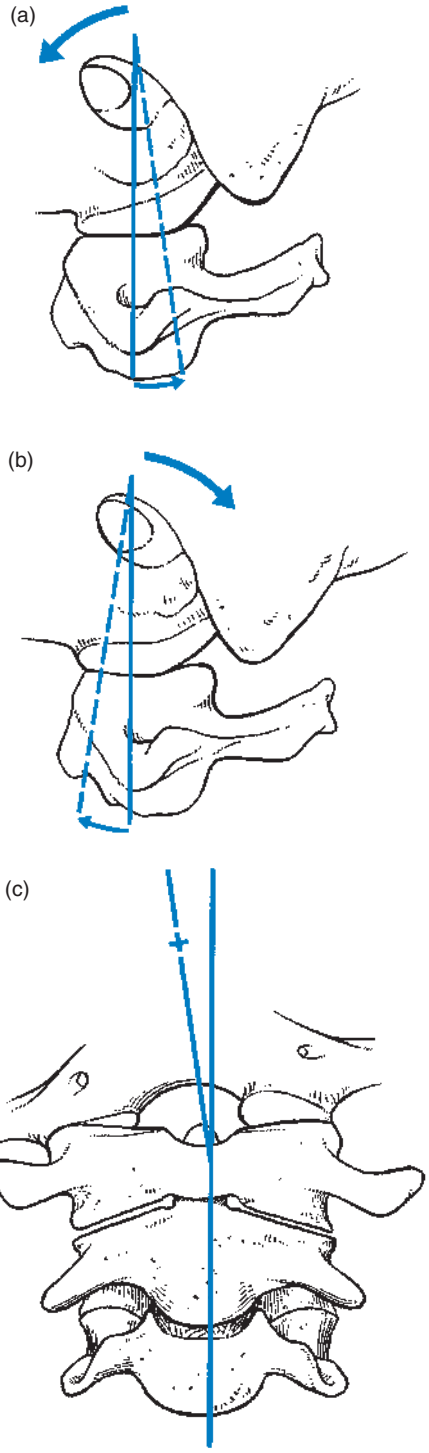

Fig 7 • The occipitoatlantoaxial joint and its movements: (a) flexion, (b) extension, (c) lateral flexion.

Occipitoatlantoaxial joints

The occipital condyles are arcuate in the sagittal plane and fit into the cup-shaped superior articular surfaces of the atlas (Fig. 6). These joints only allow moderate flexion–extension (13–15°) and lateral flexion (3–8°) movements (Fig. 7). Axial rotation is not possible at these joints.

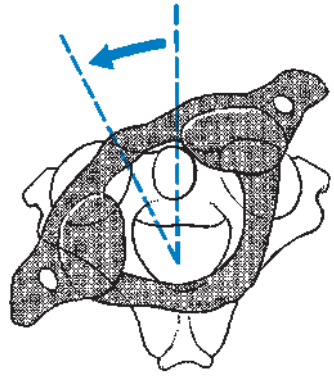

Fig 8 • The occipitoatlantoaxial joint and its rotation.

At the joint between C1 and C2 extensive rotation movement is possible (45–50°) which represents about 50% of rotation in the neck (Fig. 8). There is a moderate flexion–extension excursion (10°) but lateral flexion is impossible.

Joints between C2 and C7

Most flexion–extension takes place at the joints C3–C4, C4–C5 and especially C5–C6. Lateral flexion and axial rotation occur mainly at C2–C3, C3–C4, C4–C5.

Mobility is less in the most caudal segments and coupling of movements is present (Fig. 9a). This phenomenon is the result of the position of the articular surfaces of the facet joints (see p. 123). Lateral flexion is always combined with ipsilateral rotation. So, for example, lateral flexion to the left is accompanied by rotation to the left. This is greatest at C2–C3 and coupling rotation decreases towards the caudal aspect of the spine. The clinical importance of this becomes clear during examination: specific articular patterns may occur as its result.

Movement takes place at two sites. First, in the anterior part of the lower cervical spine, which contains the intervertebral joints (with their intervertebral discs) and the uncovertebral joints and, second, in the posterior part where the facet joints, the arches and the transverse and spinous processes are found.

Anterior aspect

Intervertebral joints

The intervertebral joint is the complex of two vertebral bodies and the intervertebral disc between them (Fig. 10). The disc has several functions: it permits greater mobility between the vertebrae; it helps distribute weight over the surface of the vertebral body during flexion movements; and it acts as a shock absorber during axial loading. The joints are mainly stabilized by the anterior and posterior longitudinal ligaments and the uncovertebral joints.

Uncovertebral joints (Fig. 11)

These develop during childhood when fissuring occurs in the lateral aspect of the intervertebral disc, leading to the formation of a cleft in the adult spine. They do not contain articular cartilage or synovial fluid and must therefore be considered as

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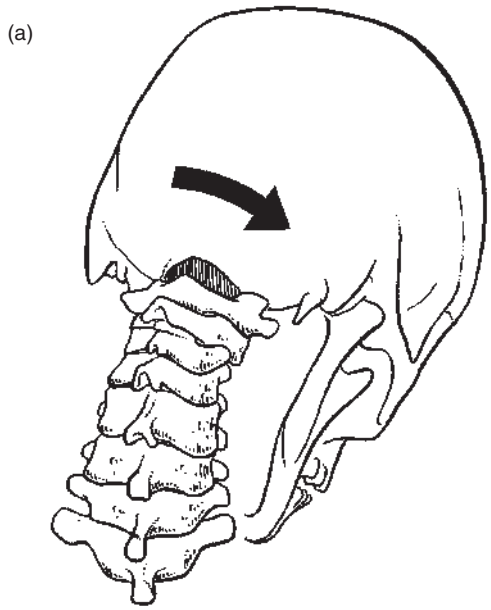

Fig 9 • Combined flexion and rotation in the lower cervical spine (a), and lateral view of disc movements (b), upper: extension; middle: neutral position; lower: flexion.

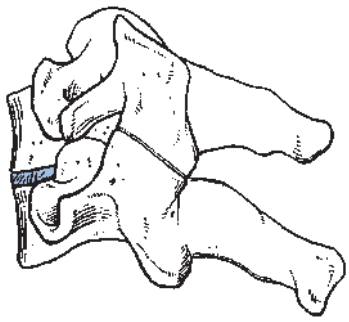

Fig 10 • The intervertebral joint (coloured).

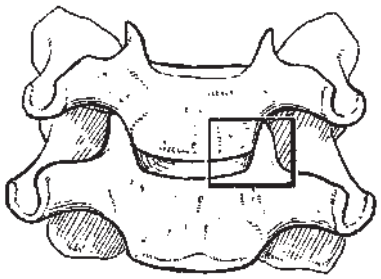

Fig 11 • The uncovertebral joint (boxed).

pseudojoints, although they do undergo degenerative changes. These secondary 'joints' add to lateral stability.

Posterior aspect and facet joints

Posteriorly the vertebrae are held together by the ligaments between the spinous processes (ligamentum nuchae and interspinous ligaments) and between the laminae (ligamentum flavum), and they articulate via the facet joints.

The facet joints are classified as diarthrodial joints: the articular surfaces are covered with cartilage; there is a synovial membrane and a fibrous joint capsule with contained synovial fluid. The joint line is oblique: it courses from anterosuperior to posteroinferior, along an angle of 45° at the level C2–C3, which decreases to 10° at C7–T1 (Fig. 12). Because the joint line is oblique and the capsule is lax, more movement is possible than at the thoracic and lumbar levels.

Rotation is always combined with ipsilateral side flexion. During rotation to the left the lower facet of the upper vertebra at the left side glides backward on the upper facet of the vertebra below. The opposite happens at the right.

During extension of the neck, the vertebral body of the upper vertebra glides backwards (Fig. 13a). The lower facets not only glide backwards and downwards but also tilt backwards, which results in opening in front and closing behind. The reverse happens during flexion: the lower facets of the upper vertebra glide forwards and upwards and tilt forwards, which opens the joint at the back and closes it in front (Fig. 13c).

Ligaments

The cervical spine has a complex ligamentous system. Ligament function is to maintain normal osseous relationships.

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Clinically, however, they are not so important because ligamentous lesions in the cervical spine are not that common and, when they occur, it is difficult to find out where exactly the problem lies.

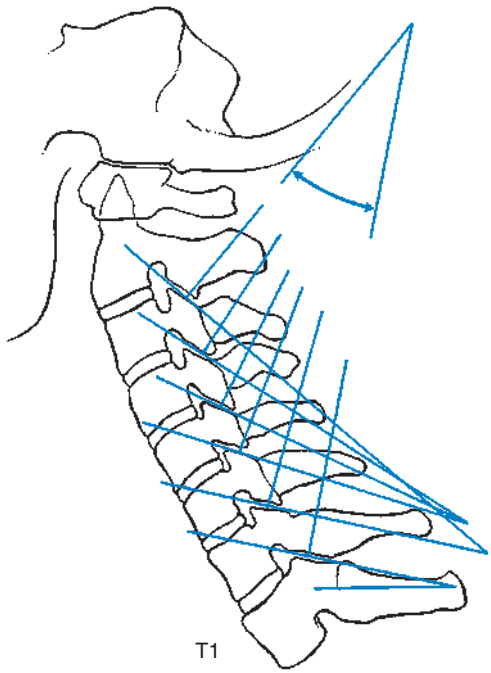

Fig 12 • The facet joints, showing the decrease of the obliquity of the joint line towards the lower part of the spine.

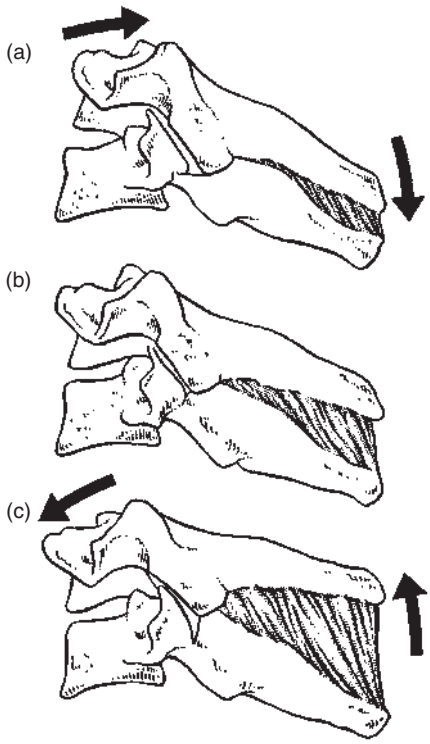

Fig 13 • Movements at the facet joints: (a) extension, (b) neutral, (c) flexion.

Two distinct sets of ligaments can be recognized: the ligaments of the occipitoatlantoaxial complex and those of the lower cervical spine.

Ligaments of the occipitoatlantoaxial complex

These are strong structures that stabilize the upper cervical spine. First, there are the ligaments connecting the occiput to atlas and axis: the *anterior atlanto-occipital membrane*, the *posterior atlanto-occipital membrane* and the *tectorial membrane* (Fig. 14). A second ligamentous complex connects the

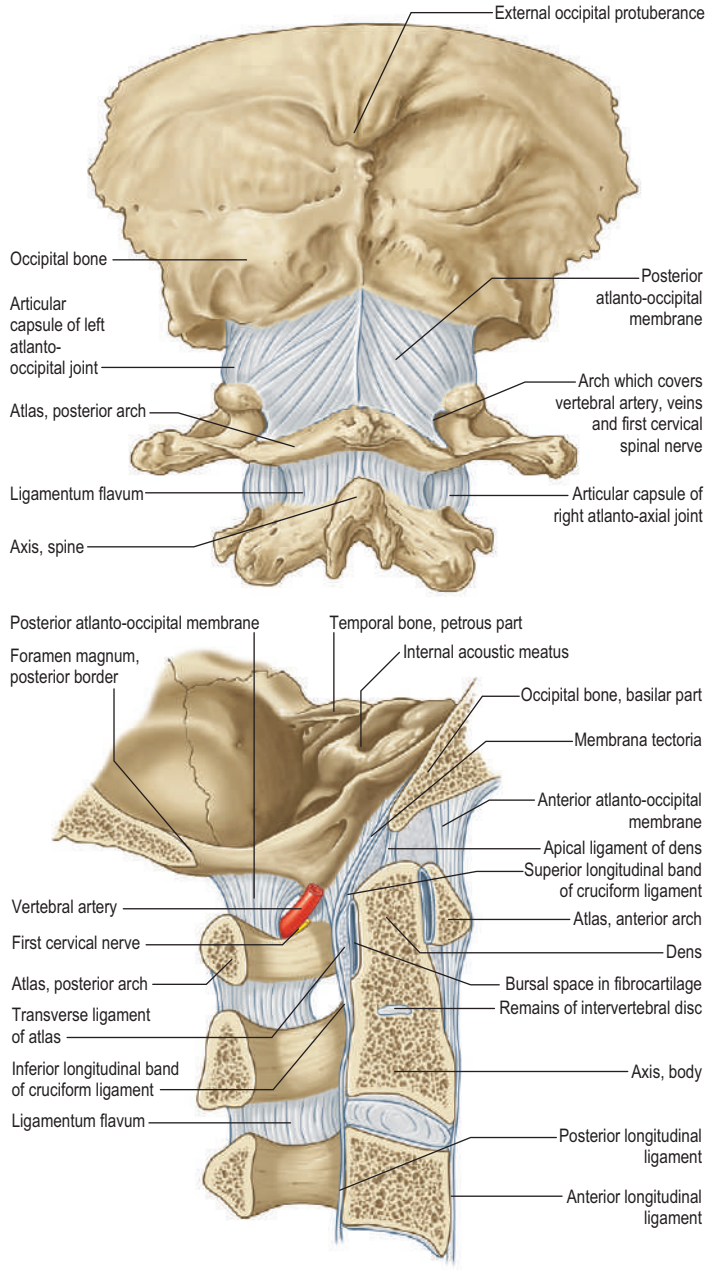

Fig 14 • Occipitoantlantoaxial complex: posterior view and sagittal section. From Standring, Gray's Anatomy, 40th edn. Churchill Livingstone/Elsevier, Philadelphia, 2009 with permission.

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axis to the occiput: the *apical ligament*, the longitudinal component of the *cruciform ligament* and the *alar ligaments* (Fig. 15). Third, there is the complex of ligaments that connect axis to atlas: the lateral component of the *cruciform ligament* (the *transverse ligament*), the two *accessory atlantoaxial ligaments* and the *ligamentum flavum* (Fig. 16). Finally, the *ligamentum nuchae* (Fig. 17) is attached above to the external occipital protuberance, lies in the sagittal plane and merges with the interspinous ligaments and supraspinous ligament, which becomes an individual ligament from the spinous process of C7 downwards. This ligament contains much elastic tissue and is stretched during neck flexion. Because of its elasticity it helps to bring the head back into the neutral position.

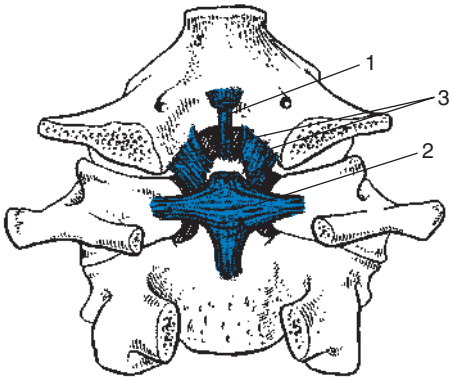

Fig 15 • The occipitoaxial ligaments: 1, apical ligament; 2, cruciform ligament; 3, alar ligament.

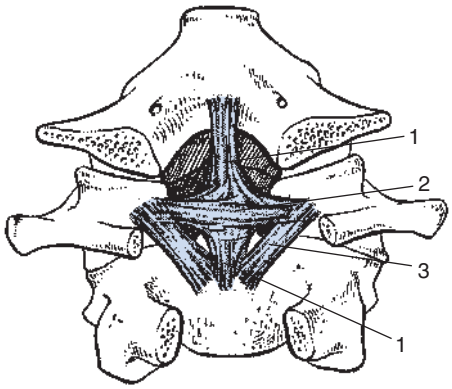

Fig 16 • The atlantoaxial ligaments: (blue) cruciform ligament: 1, longitudinal component; 2, lateral component (= transverse ligament); 3, accessory atlantoaxial ligaments; (gray) ligamentum flavum.

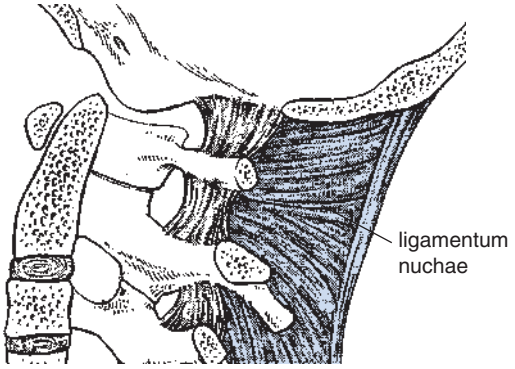

Fig 17 • The ligamentum nuchae.

Ligaments of the lower cervical spine

The *anterior longitudinal ligament* (Fig. 18) is closely attached to the vertebral bodies, but not to the discs. By contrast the *posterior longitudinal ligament* (Fig. 19) is firmly attached to the disc and is wider in the upper cervical spine than in the lower. Both ligaments are very strong stabilizers of the intervertebral joints. The lateral and posterior bony elements are connected by the *ligamentum flavum*, the *intertransverse ligaments* and *interspinous ligaments* and the *supraspinous ligament* (Fig. 20).

Muscles

Muscular action at the cervical spine is dependent on a combination of activities of a great number of muscles and whether or not they contract bilaterally or unilaterally. There are three functional groups: flexors; extensors; and rotators and lateral flexors.

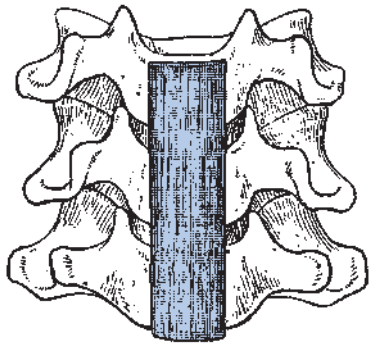

Fig 18 • The anterior longitudinal ligament.

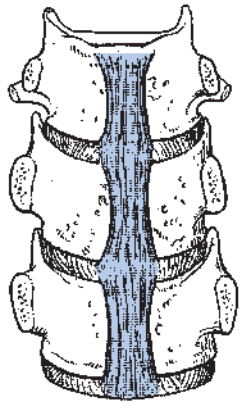

Fig 19 • The posterior longitudinal ligament (posterior view).

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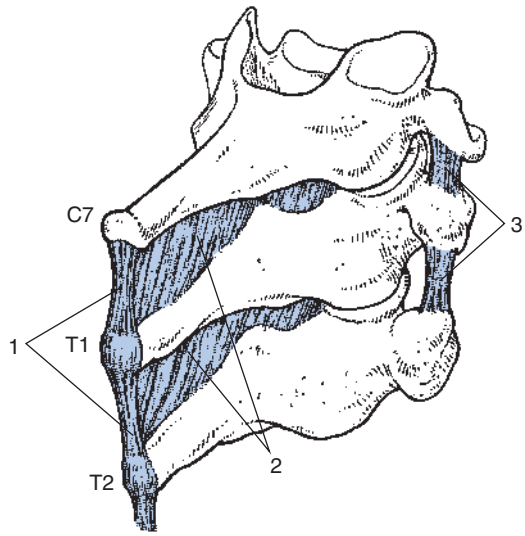

Fig 20 • The supraspinous (1), interspinous (2) and intertransverse (3) ligaments.

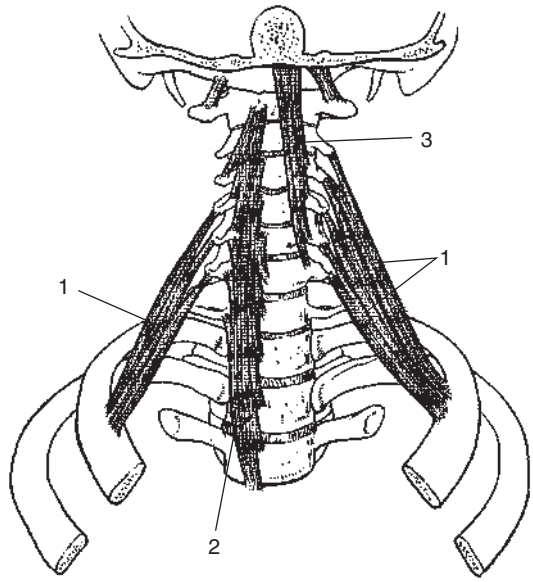

Fig 21 • Flexor muscles: 1, scalenes; 2, longus colli; 3; longus capitis.

Most muscles do not have clinical importance, in that lesions of them hardly ever occur. However, they can sometimes indirectly involve other (non-contractile) structures. On examination a resisted movement (muscular contraction) or a passive movement (whereby the muscle is stretched) may influence a lesion lying outside the muscle in, for example, an inert tissue. One contractile structure makes an exception however: an acute lesion of the longus colli causes, besides pain and weakness on flexion, serious limitations of extension and both rotations (see Standring, Fig. 28.6).

It should be noted that the function of many of the muscles is multiple and depends on the mode of action employed. For example, bilateral action of the sternocleidomastoids is involved in flexion–extension, but unilateral action results in ipsilateral lateral flexion and contralateral rotation (see Fig. 24).

Unilateral action of the flexors results in either pure rotation or lateral flexion, or a combination of both, sometimes ipsilaterally and sometimes contralaterally.

Many of the extensor muscles have other functions as well as extension, the latter occurring when they act bilaterally.

The muscles of the neck are illustrated in Figures 21–24.

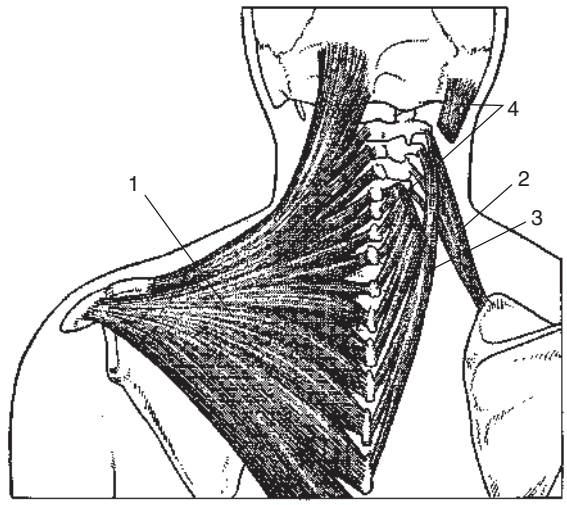

Fig 22 • Superficial extensor muscles: 1, trapezius; 2, levator scapulae; 3, splenius cervicis; 4, splenius capitis.

Nervous structures

Related structures

Spinal canal

In the cervical spine, the spinal canal is commodious compared to that of the thoracic and lumbar spine. Its outline is oval in the transverse plane and the average anteroposterior diameter is 17 mm, although this varies with movement: flexion increases it and extension decreases it. During extension there is a backward movement of the upper vertebra in relation to the lower because of the obliquity of the facet joints (Fig. 13). As the anteroposterior diameter of the spinal cord at midcervical level is about 10 mm, there is, however, a large margin of safety.

Intervertebral foramen

The intervertebral foramen lies between adjacent pedicles and through it the spinal nerve emerges from the spinal canal (Fig. 25). The foramen continues across the bifid transverse process and is orientated anterolaterally 45° and anterocaudally 15°.

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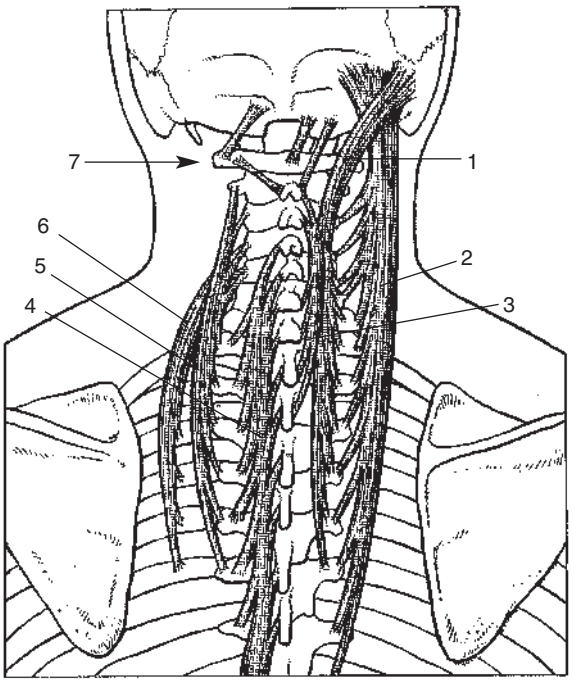

Fig 23 • Deep extensor muscles: 1, longissimus capitis; 2, semispinalis capitis; 3, spinalis cervicis; 4, multifidus; 5, longissimus cervicis; 6, iliocostalis cervicis; 7, suboccipital complex.

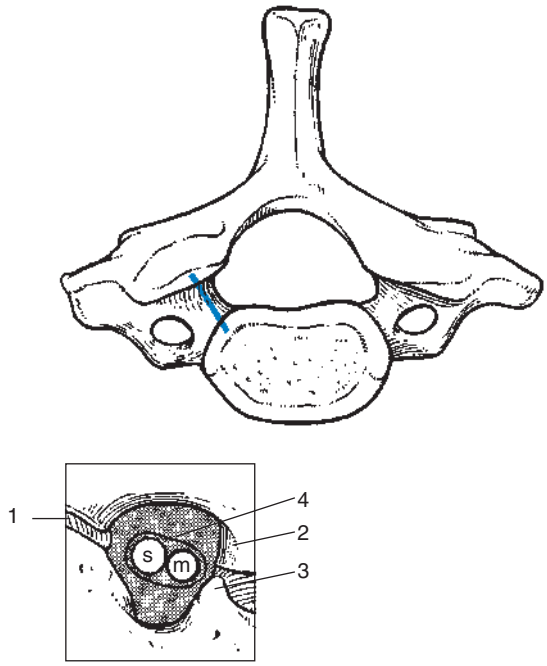

Fig 25 • The intervertebral foramen: s, sensory fibres; m, motor fibres. 1, articular facet; 2, vertebral body; 3, uncinate process; 4, dural sleeve.

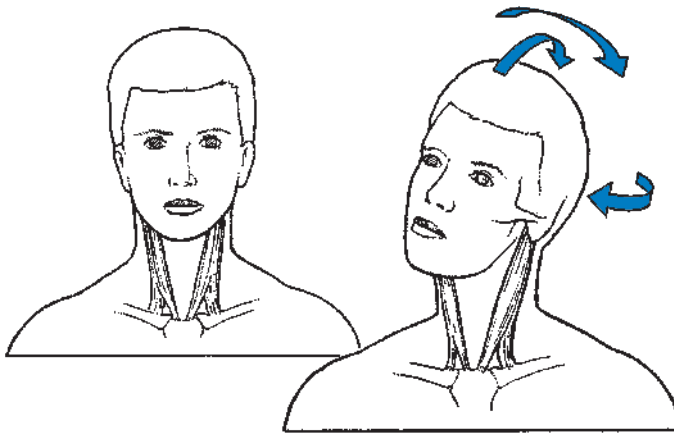

Fig 24 • Functions of the sternocleidomastoids.

The anterior border is the uncinate process and vertebral body and the articular facet is posterior. The diameter of the foramen is reduced during a combined movement of extension and ipsilateral rotation.

Spinal membranes

The dura mater is firmly attached to the rim of the foramen magnum and its fibres blend with the periosteum within the skull. In the spinal canal it is not attached to the vertebral arches, because of the presence of protective fat tissue in between. The arachnoid is a membrane which lies in close contact with the dura. The subarachnoid space is quite wide. The third layer, the pia mater, covers the spinal cord (Fig. 26). A number of dentate ligaments pass between a fold of the pia mater which extends longitudinally and the anterior and posterior nerve roots. These ligaments are attached to the dura and suspend the spinal cord in the cerebrospinal fluid.

Mobility of the dura mater

The dura mater can move considerably without influence on the cord. This is an interesting feature, in that the cervical spine lengthens approximately 3 cm during neck flexion. Consequently the dura mater, attached above and below, migrates within the spinal canal. It thus moves forwards and can be dragged against any space-occupying lesion within the canal (e.g. a disc protrusion). As a consequence the mobility of the dura can be hindered, increasing tension and resulting in pain, because the dura is also sensitive.

Sensitivity of the dura mater

The anterior aspect of the dura mater is innervated by a dense longitudinally orientated nerve plexus consisting of different fibres of the sinuvertebral nerves originating at several levels (Fig. 27). Distal to the spinal ganglion, the sinuvertebral nerve detaches and passes back into the spinal canal to innervate the posterior longitudinal ligament, the anterior aspect of the dural sac and the dural sheath around the nerve root, the anterior capsule of the facet joints and also the vascular structures (Fig. 28).

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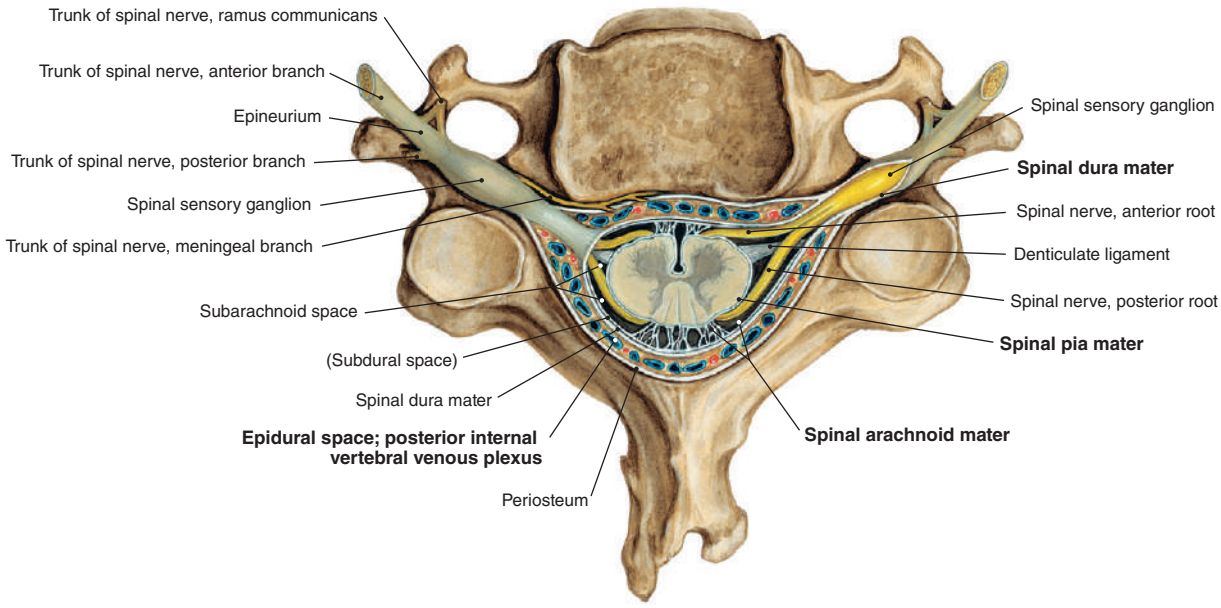

Fig 26 • The dura mater and exits from the spinal canal; cross-section at the level of the fifth cervical vertebra. From Putz, Sobotta – Atlas of Human Anatomy, 14th edn. Urban & Fischer/Elsevier, Munich, 2008 with permission.

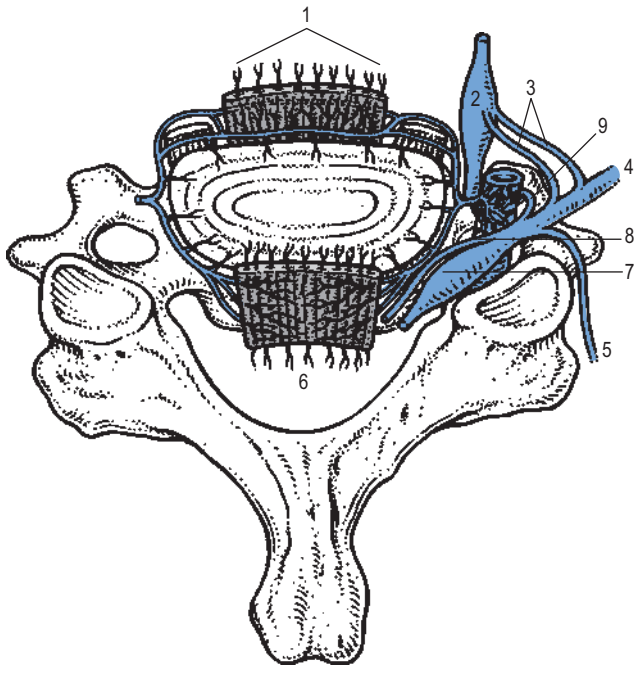

Fig 27 • The sinuvertebral nerve and its related structures: 1, anterior longitudinal ligament; 2, sympathetic trunk; 3, rami communicantes; 4, ventral ramus of spinal nerve; 5, dorsal ramus of spinal nerve; 6, posterior longitudinal ligament; 7, spinal ganglion; 8, anterior root; 9, sinuvertebral nerve.

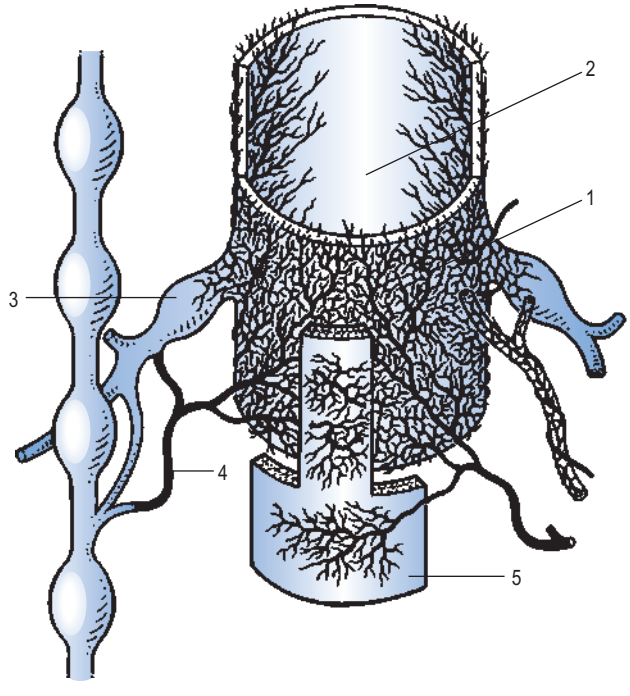

Fig 28 • The anterior part of the dura mater is innervated by a mesh of nerve fibres belonging to different and consecutive sinuvertebral nerves: 1, anterior part of the dura; 2, posterior part of the dura; 3, spinal ganglion of nerve root; 4, sinuvertebral nerve; 5, posterior longitudinal ligament.

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This could be an explanation for the 'dural pain' that occurs when the anterior part of the dura is compressed (see p. 16). The pain is multisegmental, which means it is felt in several dermatomes at a time.

The mobility and sensitivity of the dura mater are described in detail on page 425.

Nerve roots

The nerve root contains motor and sensory branches formed by the convergence of rootlets emerging from the ventral and dorsal aspect of the spinal cord. The branches remain completely separated until their fusion in the spinal ganglion – sensory above and motor below – but are surrounded by the continuation of the dura – the dural sheath. Just outside the intervertebral foramen the root divides into a dorsal and ventral branch – the dorsal ramus and the ventral ramus, the latter joined by the cervical sympathetic chain.

The dural sheath

The dural investment surrounds the nerve root from the point where it leaves the spinal cord to the lateral border of the intervertebral foramen (Fig. 26). It is sensitive and slightly mobile, which means that some migration is possible.

Mobility of the dural sheath

At the cervical spine the nerve roots are not as mobile as in the lumbar spine. The dural sleeve is tethered to the transverse process, especially at the mid-cervical level. Therefore, it is more difficult to use tension tests during clinical examination.

Sensitivity of the dural sheath

The dural sheath is sensory-innervated by its own sinuvertebral nerve. Pain that originates from the dural sheath is therefore strictly segmental and felt in the corresponding dermatome.

The parenchyma

Irritation of the parenchyma, for instance as the result of external pressure, results in *paraesthesia*. This is also segmental and felt in the same dermatome as the 'dural sheath pain'. Further irritation and destruction of the neural fibres lead to impaired conduction, which results in *motor and/or sensory deficit*.

Except for the nerve roots C6 and C7, which are slightly more oblique, the course of the root is almost directly lateral and thus horizontal, in contrast to the oblique course found in the lumbar spine. There are eight cervical nerve roots. The C1 root emerges between occiput and atlas, and the nerve root C8 between the seventh cervical and the first thoracic vertebrae. The result of this is that, for instance, a nerve root C7 can only become compressed by the disc C6 (the disc between the vertebrae C6 and C7) (Fig. 29).

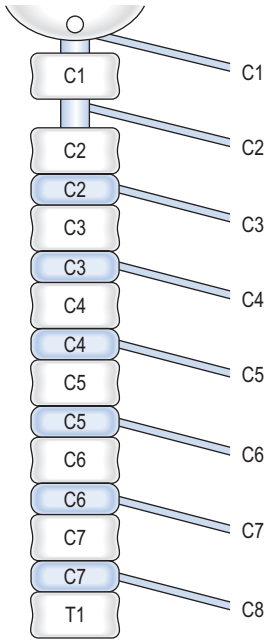

Fig 29 • The mainly horizontal course of the nerve roots and the relationship between roots and discs.

Innervation of the cervical spine

That part of the cervical spine which lies anterior to the plane of the intervertebral foramina is innervated by the anterior primary rami and their branches – the sinuvertebral nerves. The posterior aspect of the spine is innervated from the posterior primary rami.

Blood supply

The main blood supply to the cervical spine and related structures is from the vertebral arteries which originate from the subclavian arteries and finally join to form the basilar artery. During their course they give off branches to the spine and to the spinal cord (anterior and posterior spinal arteries).

The vertebrobasilar system is a 'closed' circuit (Fig. 30), starting below in the subclavian arteries and ending above in the arterial circle of Willis. The vertebral arteries run parallel on both sides of the spinal column through the canal formed by the successive transverse foramina, and are the main blood supply for the cervical spine, the spinal cord and the brainstem. Between the axis and atlas the arteries curve backwards and outwards and run over the posterior arch of the atlas. They curve upwards again and run cranially.

Inside the skull they join to form the basilar artery which then splits into a left and right posterior cerebral artery. These arteries communicate with the internal carotid artery from

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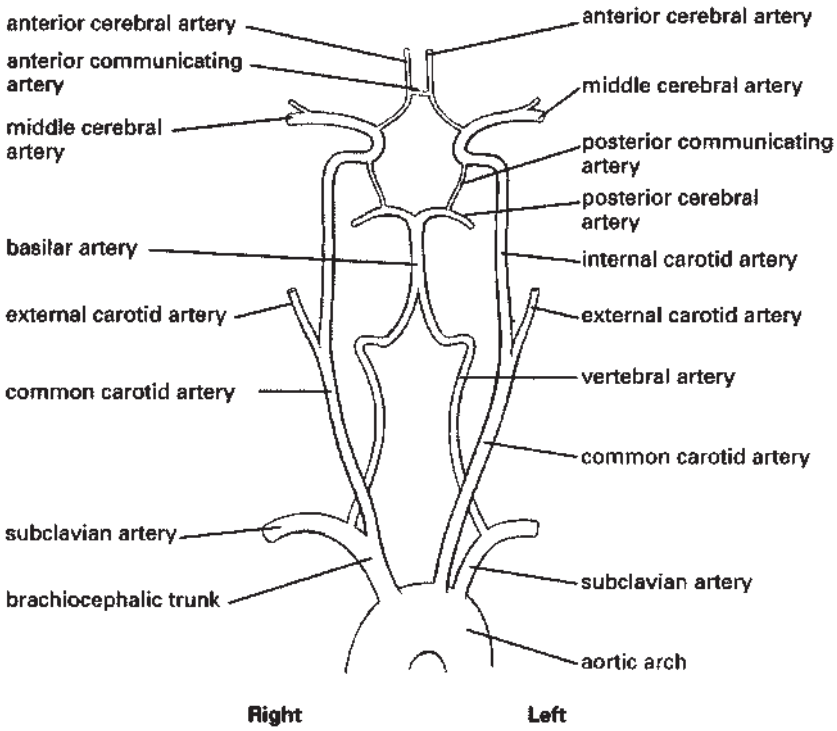

Fig 30 • The vertebrobasilar system.

which the anterior cerebral arteries branch off. For a detailed description see online chapter *Headache and vertigo of cervical origin*.

Uncoarterioradicular junction

There is a close connection between the uncovertebral joint, the vertebral artery and the nerve root. The artery is between the uncinate process and the nerve root. The latter lies behind the artery and just anterior to the facet joint. Degenerative changes leading to osseous, cartilaginous or capsular hypertrophy may result in compression of artery or nerve root. The uncovertebral joint is the main threat to the vertebral artery, the facet joint to the nerve root. Movements such as rotation, combined with either extension or flexion, also influence these structures (Fig. 31).

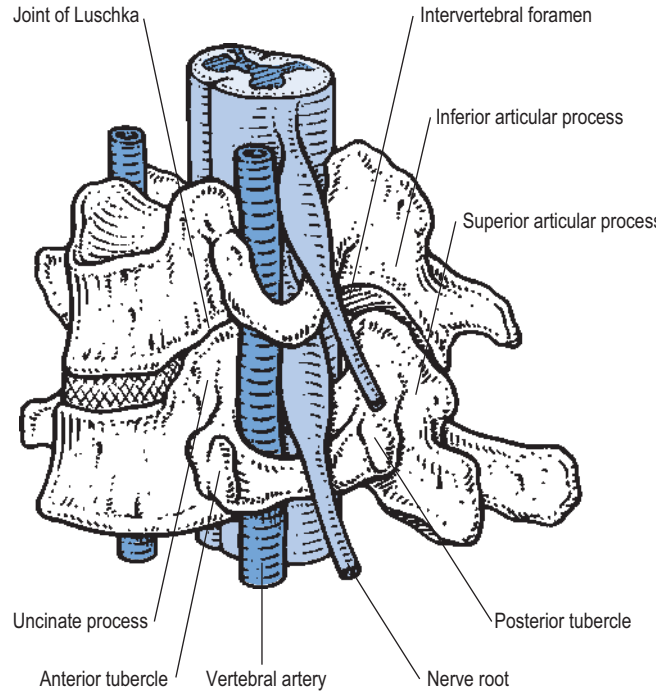

Fig 31 • The uncoarterioradicular junction.

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Non-mechanical disorders: pathology

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Classification

Non-discogenic lesions which give rise to symptoms in the neck, trapezius or scapular area or in the upper limb can be classified into the following groups:

- Osseous
- Rheumatoid arthritis and ankylosing spondylitis
- Infectious
- Intraspinal
- Neurological
- Visceral
- Psychological
- Other

Osseous disorders

Fractures and luxations

A considerable variety of fractures and dislocations follow trauma between the occiput and the first thoracic vertebra. Most are caused by car accidents, with falls and sports accidents next in frequency.[1–3] Half of all patients present with neurological problems.[4]

Early diagnosis is important. The cervical fracture is often combined with another spinal fracture and therefore the entire vertebral column must be X-rayed.[5] Plain radiographs are the first way in which diagnosis is established. Multidirectional CT is particularly advantageous in patients with facet injuries. CT seems to add most additional information in laminar or posterior column injuries, fractures of the vertebral body or in atlantoaxial subluxations.[6]

Fractures and dislocations of the atlantoaxial complex

Fractures of the axis are common. In 14–17.5% of all fractures of the cervical spinal column the lesion lies at the axis.[7] Most frequent are odontoid fractures. They are classified as type I – avulsion of the tip of the odontoid process, type II – fracture through the base and type III – fracture through the vertebral body.[8,9] These are followed in frequency by those of the vertebral body, the pedicle or the lateral mass. Less common is hangman's fracture – a bilateral fracture through the pars interarticularis of the axis. Neurological damage is not frequent in odontoid and hangman's fractures but quite common in the other miscellaneous fractures of the axis.[10]

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Dislocations of the atlas are not uncommon and may lead to serious neurological damage.[11] Fractures of the atlas are rare and seldom cause neurological problems.

In order of frequency the following fractures are found: bilateral fracture of the posterior arch, comminuted fracture of the ring of the atlas – traumatic spondylolisthesis or Jefferson's fracture, and unilateral fracture of the lateral mass.[12]

Fractures of the lower cervical spine

An accident immediately followed by the occurrence of gross limitation of movement in every direction, especially of extension (full articular pattern) strongly suggests vertebral fracture or dislocation. Apart from possible neurological problems the patient presents with severe central or bilateral neck pain and is unable to move his head as the result of muscle spasm. Properly performed radiological examination is diagnostic.

Most fractures in the cervical spine occur below C2. They range from fractures of the articular process to fractures of the vertebral body, lamina, spinous process and pedicle. Fractures are commonly classified in six groups depending (in order of frequency) on the forces that have acted on the cervical spine: compressive flexion, distractive flexion, compressive extension, vertical compression, distractive extension and lateral flexion.[13] Neurological problems most often occur when the fracture is combined with luxation.[14,15] Pathological fractures following a minor injury or a sudden effort give rise to the same clinical picture but the history is much less indicative and may even be misleading. If neurological injury is present, the diagnosis of a probable fracture or dislocation is made more simple.

Clay-shoveller's fracture

This is a fracture of one or more spinous processes in the lower cervical or upper thoracic spine (mostly C7, sometimes C6 or T1).[16]

A traction fracture may occur as the result of strong muscular force from the trapezius transmitted to the spinous processes through the musculature. It happens suddenly. A crack is felt followed by severe pain at the base of the neck and between the shoulders. The same event may occur in motor vehicle accidents where a strong flexion force is applied to the neck, and thus also in whiplash injuries.[17,18]

If the fracture is the result of a fatigue mechanism, the patient complains of a dull ache at the cervicothoracic junction coming on without clear cause.

Neck movements are not really painful but the patient is unable to bring the arms above the horizontal because of pain. The passive shoulder range is full and painless. The radiograph shows avulsion of one or more spinous processes.

Spontaneous cure takes 3–6 weeks.

Bony tumours

Primary bone tumours are uncommon. They represent only 0.4% of all tumours and cervical localization accounts for only 4.2% of the primary bone tumours of the spine. This is very much less than in the thoracic or lumbar spine.[19]

Box 1

Warning signs for cervical tumour

History

- Central neck pain becoming slowly worse
- Elderly person with rapidly increasing pain and/or stiffness of the neck
- Elderly person with neck pain for the first time
- Cord symptoms
- Dysphagia

Examination

- Full articular pattern occurring spontaneously over short period of time
- Gross limitation of rotations
- Wrong end-feel; muscle spasm, soggy, empty
- Resisted neck movements painful and weak
- Unusual weakness in the arm
  - Gross weakness in the absence of severe radicular pain,
  - bilateral weakness,
  - multiradicular weakness,
  - T1 palsy
- Horner's syndrome
- Anaemia

Benign primary tumours occur more often in the first two decades of life, while malignant primary tumours more frequently affect adults. The incidence of malignant tumours increases significantly with age. In the cervical spine, even more than in other parts of the axial skeleton, metastatic lesions are much more frequent than primary tumours.

The symptoms may vary and include local heat, tenderness, neuralgic pain, root palsy, torticollis-like limitation of neck movements[20] and myelopathy, although early in development the symptoms may mimic ordinary soft tissue lesions. Severe pain at night is often a hallmark of neoplasm.[21]

The presence of one or more inconsistencies during history and/or functional examination is a warning sign and puts the examiner on guard (see Box 1). It is again mainly the clinical approach that suggests a serious disorder. Radiography – usually the first additional examination – is not always helpful, as it appears that more than 30% of the cancellous bone of the vertebral body must be destroyed before a plain X-ray becomes positive.[22–24] More refined imaging such as CT scan, technetium scan, angiography and MRI confirms the diagnosis. A radiograph and CT scan of the chest or abdomen may be necessary in patients with unknown primary sites.

Benign tumours

The most common benign neoplasms affecting the cervical spine are, in order of frequency: osteoid osteoma, osteoblastoma, haemangioma, aneurysmal bone cyst, eosinophilic granuloma, giant cell tumour and osteochondroma. They can be found at any level, except C1, and are most common at C2, C4 and C7 levels.[25–28]

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Osteoid osteoma

This is the most frequent benign tumour in the cervical spine[20] and appears in young adults, mostly males.[29,30] It affects the cervical spine less frequently than the lumbar spine and is located in the pedicles and vertebral arches.[31]

The typical symptom is persistent localized pain, especially at night, which can usually be relieved by salicylates. The pain sometimes radiates to the upper limb.

Surgical removal is curative.

Osteoblastoma

This tumour affects the cervical spine as frequently as it does the thoracic spine. It is more common in the lumbar spine.[25] Young adults – males more frequently – are affected and the posterior elements of the vertebra are involved, which may lead to radiculopathy and myelopathy.

The neck pain is dull, especially at night and the normal lordosis may be reversed to produce a torticollis-like picture. Root and cord symptoms may mimic a disc protrusion, except that the patient is too young to have disc protrusion causing root or cord symptoms.

Excision usually leads to full recovery.[32]

Haemangioma

This occurs most often in women in their fourth decade.[33] It is common and usually has an asymptomatic development – one-quarter of all cases are in the cervical spine.

The tumour rarely gives rise to symptoms. Should it do so, localized pain progressing to cord symptoms is usual.

Treatment with low-dose radiation usually suffices.[34]

Aneurysmal bone cyst

This is seen most frequently in children and young adults, mostly females (under 30 years old).[35] Twenty-five per cent of spinal aneurysmal bone cysts are located in the cervical spine.[28] It is a destructive tumour and is mostly localized in the neural arch but may also invade the vertebral body. As it expands, it may lead to root pain and even to compression of the spinal cord.

Excision and/or curettage and stabilization with bone grafts are indicated.[36]

Eosinophilic granuloma

This is a variant of the systemic disorder, histiocytosis-X. The cervical spine can be affected in both children and adults, and the condition has a typical development. The vertebral body collapses, producing sudden pain and sometimes muscle spasm leading to torticollis. A patient with acute torticollis and a sudden onset who presents with pain, limitation and involuntary muscle spasm on careful passive examination should therefore be radiographed to exclude this disorder, which shows a 'vertebra plana'.

The fracture may heal spontaneously. However, when the flattening is significant, neurological symptoms may follow; these are reversible when treatment is started without delay. Open biopsy, followed by immobilization and irradiation may be necessary.[37,38]

Giant cell tumour

This is more frequent in the sacrum and the lumbar spine but may also affect the cervical spine. It seems to occur in younger patients (between 20 and 40 years old), especially women.[39] It leads to destruction of the vertebral body and can later involve the posterior part of the vertebra.

It may cause pain in the neck but can also give rise to radicular symptoms.

Excision with bone grafting is the treatment of choice.

Osteochondroma

This occurs mostly in young adult males. When it affects the spine it is seldom symptomatic, except in the cervical region.[40] There is slow progression but neural compression can develop.

The results of surgical excision are good.

Malignant tumours

The most common malignant neoplasms are: multiple myeloma, chordoma, solitary plasmacytoma, chondroma, chondrosarcoma, lymphoma, osteosarcoma, Ewing's sarcoma and metastases.

The primary malignant tumours represent 6.3% of all primary bone tumours of the spine and occur mainly from middle age on, much more frequently in men than in women. They are found at all levels, except C1.[26]

Multiple myeloma

This is the most frequent primary malignant tumour in the spine. It occurs more often in men, usually between 50 and 70 years of age. The patient's main symptom in early cases is pain in the neck. Anaemia and cord symptoms soon follow.

The radiograph shows round lytic defects in the bone, without a surrounding sclerotic reaction. Pathological fractures may occur.

The treatment of choice is radiation and/or systemic chemotherapy. The outlook is poor.

Chordoma

This is an uncommon, locally invasive, slow-growing malignant neoplasm that arises from the vertebral or suboccipital remnants of the embryonic notochord. In 33–38% it occurs in the upper cervical vertebrae, especially C2, and is found most often in men aged between 50 and 70 years.[41,42]

Central neck pain becoming slowly worse is a common symptom. After several months, movements become gradually limited, especially rotation, with a soggy end-feel on passive testing.

The tumour often extends anteriorly into the soft tissues and may then result in dysphagia, upper respiratory obstruction and Horner's syndrome.[43] Posterior extension may be accompanied by neurological complications, such as epidural spinal cord compression or cervical radiculopathy.[44,45]

The classic radiological finding is an expanding osteolytic lesion. This life-threatening lesion is best treated surgically by radical resection and, if this fails, repetitive local debulking procedures may be used. The prognosis is bad.

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Solitary plasmacytoma

This is a myeloma (plasma cell neoplasm) in a single vertebral body. The patient is over 60 years and complains of slowly progressing neck pain with muscle spasm. The prognosis is much more favourable than in patients with multiple myeloma.[46] Collapse of the vertebral body and cord compression may result.

The treatment of choice is radiation.

Chondrosarcoma

This tumour usually occurs in patients aged between 40 and 60 years. It forms cartilaginous tissue in the vertebral body or in the neural arch where it leads to destruction of bone. In most cases a soft tissue mass also develops.

Lymphoma, osteosarcoma, Ewing's sarcoma

Lymphoma presents either as a skeletal manifestation of a systemic disease or as an isolated tumour. It may involve the epidural space adjacent to the osseous spine. Complete recovery is only possible after total surgical excision.

Osteosarcoma and Ewing's sarcoma are extremely uncommon in the spine. These tumours evolve rapidly and osteogenic sarcoma generates early metastases to the lung. Treatment is biopsy, chemotherapy and/or radiotherapy.

Metastases

Secondary deposits are the most common malignant tumours of the cervical spine, although this part of the vertebral column is the least affected, occurring in 8–20% of patients with known metastatic disease.[47] Breast, lung, prostate, colon, kidney and thyroid are the most frequent sites of primaries.[48] Metastases in the spine may pass unnoticed for a considerable time and are sometimes discovered during routine radiography.

In symptomatic cases, pain is the earliest and most prominent feature in 90%.[49] Localized pain that starts spontaneously and becomes gradually worse, especially at night, is the most common picture of spinal metastases. It is axiomatic that a cancer patient who develops neck pain harbours a spinal metastasis until proven otherwise. If a patient presents with neck pain but has a history of a primary tumour, for example breast cancer, even a long time ago, metastases must be taken into consideration. Tumour-related pain is predominantly nocturnal or early morning pain and generally improves with activity during the day. This pain may be caused by inflammatory mediators or tumour stretching the periost of the vertebral body.[50]

Neurologic symptoms and signs often begin with radiculopathy (nerve root symptoms) and are followed by myelopathy (spinal cord compression).

The clinical features differ depending on whether the lesion is localized at the upper cervical spine (C1-C3), the lower cervical spine (C4-C7) or the upper thoracic spine (T1-T3).[51]

Upper cervical metastases

It may be difficult to detect bony tumours at this level. Some warning signs will probably be found, for example an elderly patient, who for the first time complains of neck pain or who describes rapidly increasing pain and stiffness of the neck coming on in the course of 1 or 2 months. On examination, a pronounced full articular pattern is found: active movements are very limited and, on passive testing, muscle spasm prevents every forced movement. Resisted movements are painful and weak. In the early stage the radiograph shows only arthrosis and osteophytes but the history and functional examination show the discrepancy between the actual signs and what is seen on the radiograph. Further imaging confirms the tentative diagnosis.

Lower cervical metastases

Diagnosis here is much easier. Warning signs in the history may suggest severe disease and the examination confirms this. A full articular pattern with gross limitation of movement with a spastic endfeel is present. The resisted movements may be painful and weak. The neurological examination of the upper limb may point to a manifest (or obvious) non-discal pattern: gross weakness in the absence of severe radicular pain; bilateral weakness; multiradicular involvement and palsy of different nerve roots. Also signs of cord compression may become present.

Upper thoracic metastases

Limitation of movement is difficult to detect and therefore the situation becomes clear clinically only where a bilateral root pain or root palsy occurs. It should be remembered that weakness of the intrinsic muscles of the hand due to a T1 root palsy is never caused by a disc protrusion. Especially if Homer's syndrome is also present, a malignant condition is very probable: either a pulmonary sulcus neoplasm or a neoplasm in the upper thoracic vertebrae.

If vertebral metastases are suspected, further investigations are arranged.

Plain radiographs are often ordered as the first test to evaluate a patient with cancer who has neck pain, but are relatively poor screening tests for metastases. Visualization of a radiolucent defect on plain radiographs requires a 30% destruction of the vertebral body. Additionally, metastatic tumour often infiltrates the bone marrow of the vertebral body without destroying the cortical bone. Bone scan (99mTc-MDP) is more sensitive than plain radiographs for detecting spinal metastases. The advantage of bone scan is the ability to screen the entire skeleton with a single image. However, the sensitivity is not 100%: patients with rapidly progressive, destructive tumours may not be detected and bone scan is relatively insensitive for multiple myeloma and tumours confined to the bone marrow.[52] It also has a low specificity for tumour: fractures, degenerative disease, and benign disorders of the spine (Schmorl's nodes, haemangioma) all may be positive.[53] Since MRI is widely available, it has become the most sensitive and specific modality for imaging spinal metastases. Sagittal screening images of the entire spine reveal bone, epidural and paraspinal tumour. The extent and degree of spinal cord compression can be readily appreciated.[54]

The three treatment modalities presently available for spinal metastases are chemotherapy, radiotherapy and surgery (both radical tumour resection and laminectomy).

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Rheumatoid arthritis and ankylosing spondylitis

Rheumatoid arthritis

Rheumatoid arthritis is as frequent in the cervical spine as it is uncommon in other parts of the spine and the atlantoaxial complex especially becomes affected.[55] The frequency of involvement of the cervical spine is between 25% and 80%[56] and the longer a patient has rheumatoid arthritis the more chance it has to reach the cervical level.[57] As in other joints the bony, cartilaginous and ligamentous structures are destroyed and laxity and deformation result.

Anterior atlantoaxial subluxation is the most frequent complication – it occurs in 49% of patients[58] and represents 65% of all cervical subluxations[59] – and results from laxity of the transverse ligament of the atlas and/or the ligaments of the dens (alar ligaments/apical ligament).[60] The subluxation is mostly anterior but may be lateral and occasionally posterior. When, on a flexion/extension radiograph, the distance between the anterior arch of the atlas and the odontoid process of the axis exceeds 3 mm, laxity is probable. More than 10 mm indicates gross instability[61] and requires surgical stabilization.[62] The spinal cord is seriously threatened at this stage, especially during flexion movements. This may in the end lead to spasticity, hyperreflexia (deep tendon reflexes), weakness, sensory loss and urinary problems. More recent reports show a tendency to rely more on the posterior atlantodental interval, the distance between the posterior arch of the atlas and the dens, which should be a minimum 14 mm.[63]

If the destructive process reaches both the occipitoatlantal and atlantoaxial joint complexes, the odontoid process of the axis may protrude through the foramen magnum – cranial settling or vertical odontoid subluxation[64,65] which happens in 38% of rheumatoid patients.[66] Later in the development, the atlantoaxial instability tends to decrease again,[67,68] leaving the patient with only odontoid subluxation. The lower cranial nerves, the cardiorespiratory centre and pyramidal tracts may all become compressed. Mikulowski *et al* report that 10% of rheumatoid arthritis patients may die of compression of the brainstem which is unrecognized before death.[69]

It is therefore important to do a radiographic evaluation of the cervical spine in all patients with rheumatoid arthritis, even when they are asymptomatic. Collins *et al* found that only 50% of patients with abnormalities on their radiographs had symptoms of their cervical disease.[70]

The degree of odontoid protrusion can be measured on a lateral radiograph. A line is drawn between the posterior edge of the hard palate and the inferior border of the occiput (the McGregor line). If the tip of the odontoid process lies more than 4.5 mm above this line, cranial settling is present (Fig. 1). If these landmarks are not clear, the Fischgold and Metzger measurement can be used: on an anteroposterior open-mouth tomogram the digastric line is drawn (between the points where the mastoid processes join the base of the skull), and the tip of the dens should lie 1 cm or more below this, to be classed as normal.[71]

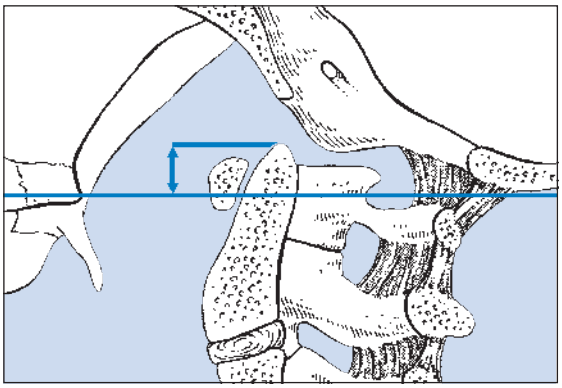

Fig 1 • The McGregor line – if the tip of the odontoid process is more than 4.5 mm above the line, cranial settling is present.

Subaxial subluxation is less frequent (10–20% of patients) but may arise at several levels,[72,73] especially C2–C3 and C3–C4. The laxity then occurs at the zygoapophyseal and uncovertebral joints[74] and may result in compression of nerve root and/or vertebral artery, the latter leading to vertebrobasilar insufficiency and its consequent clinical features.

Many patients are asymptomatic. There may be a discrepancy between the degree of destruction or instability and the symptoms. Patients may be seen with slight instability and neurological problems only, whereas others may have significant laxity without neurological symptoms.[75,76] Neurological dysfunction occurs in 7–34% of patients and may include brainstem, spinal cord and nerve roots.[77]

The disorder may cause moderate pain in the neck. Involvement of the upper cervical joints gives rise to pain often felt in the upper neck, radiating bilaterally to the occipital, temporal, auricular and retro-orbital regions. Occasionally the patient may complain of a clunking sensation with flexion movements (Sharp's and Purser's sign).[78]

On clinical examination a full articular pattern is found with a soggy or empty end-feel. This characteristic sensation immediately draws attention to the disorder. A clunking sensation on neck flexion indicates excessive movement between C1 and C2, as happens in atlantoaxial subluxation. The Sharp–Purser test may help to diagnose the condition; it appears to have a sensitivity of 69% and a specificity of 96% compared with radiological evidence.[79] The patient sits on a chair. The examiner stands to one side and places the index finger on the spinous process of C2. The palm of the other hand lies on the patient's forehead. An attempt is made to slide the head posteriorly and, if a slide between the spinous processes of C1 and C2 is felt, the test is positive.

Ankylosing spondylitis

When, late in its development, the disease reaches the cervical spine, this leads to increasing stiffness and limitation in all directions and, in the end, possibly to ankylosis in flexion. This happens in 75% of patients with a history of more than 16 years.[80] The end-feel is hard and may ultimately become bone-to-bone. As long as this bone-hard end-feel is not present the pain can be controlled with slow gradual stretching manœuvres

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at regular intervals. Once this bony end-feel has occurred, any forcing of the joints is futile.

Complications may occur, such as atlantoaxial instability[81] and fractures.[82]

Cricoarytenoid joint involvement

In long-standing cases of rheumatoid arthritis or of ankylosing spondylitis, involvement of the cricoarytenoid joints is a possibility. Dyspnoea, hoarseness and a feeling that the throat is obstructed may follow. Laryngoscopy is diagnostic.[83,84] (Cyriax:[85] p. 91.)

Infections

Infectious disease affecting the cervical spine is very uncommon compared to the thoracic and lumbar spines. Tuberculosis in the cervical spine occurs in less than 5% of spinal involvement and infection by pyogenic organisms (i.e. discitis, osteomyelitis) is extremely rare but may lead to severe neurological problems.[86,87] Infections of the spinal canal present as epidural, subdural or intramedullary abscesses. Parapharyngeal infections may lead to upper cervical ligamentous laxity.[88]

Intraspinal tumours

Cervical intraspinal neoplasms represent about 20% of all spinal tumours and may lie either outside or within the dura. The latter can be extra- or intramedullary. Clinical differentiation is often not easy because these conditions often compromise the same intraspinal structures giving rise to similar symptoms and signs.

Extradural tumours

These constitute almost one-quarter of all primary spinal tumours. The majority are metastatic[89,90] (see Fig. 2) and invade the vertebral bodies or arches: carcinoma of the bronchus is the most common primary but other sources are the breast, prostate, gastrointestinal tract, thyroid and kidney.[91]

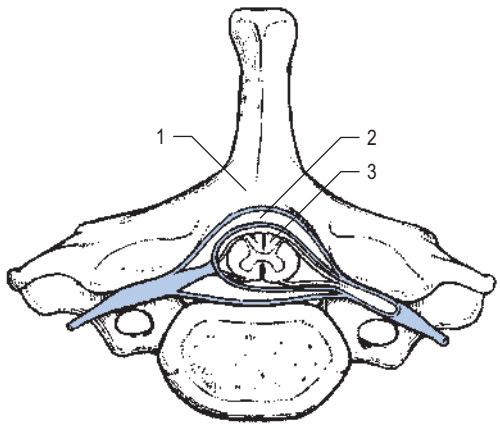

Fig 2 • Location of intraspinal tumours of the cervical spine: 1, extradural tumours (metastases, abscesses); 2, intradural extramedullary tumours (meningiomas, neurofibromas, schwannomas, sarcomas, dermoids, epidermoids, angiomas); 3, intramedullary tumours (astrocytomas, ependymomas).

These tumours must be differentiated from benign and malignant bony tumours of the spine (see pp. e14–e16), although any intradural neoplasm may also perforate the dura mater to become extradural. Pyogenic and tuberculous abscesses in the epidural space give rise to similar symptoms and signs.

These space-occupying lesions in the spinal canal very commonly lead to radicular symptoms and signs: root pain quickly follows the original local neck pain and motor and sensory deficit supervenes. Very soon in their development, the spinal cord becomes compressed and transverse myelopathy occurs with bladder dysfunction and further progressive sensory and motor deficit (see pp. 165–168).

Intradural extramedullary tumours

These form almost two-thirds of all intradural neoplasms,[92] of which meningeomas and neuromas are the most common. Neuromas (schwannomas and neurofibromas) are benign, slowly growing peripheral nerve-sheath tumours originating from Schwann cells. Schwannomas are composed entirely of Schwann cells, whilst neurofibromas contain Schwann cells, fibroblasts, perineurial cells, mast cells and axons in an extracellular matrix. Neuromas in the cervical spine are not as frequent as they are in the thoracic spine but are more frequent than in the lumbar spine.[93] Sometimes the growing tumour develops a large extradural component that expands towards the outside of the spinal canal. These so-called dumbbell neuromas present a special entity and account for 15% of all cervical neuromas.[94]

These tumours show a tendency to present with radicular symptoms very much resembling those caused by a disc protrusion. The root pain is quite constant, slightly influenced by activity and worse at night.[95] Of all patients with symptoms consistent with disc herniation, 1% have an intraspinal tumour.[96]

The ease of differential diagnosis from a cervical disc lesion may vary. In some it is quite easy, while in others it may be extremely difficult. Any of the following features should arouse suspicion (Table 1).

Warning signs and symptoms in neuroma

Warning symptoms	• Root pain, under the age of 30 • Increasing root pain over several months • Primary onset of root pain • Coughing causes pain down the arm • Bilateral root pain • Lhermitte's sign
Warning signs	• Absence of articular signs • Pronounced muscle weakness (+3 or +4) • Spinal cord signs

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Table 1 Differential diagnosis of discoradicular interactions and neurofibroma/schwannoma in the cervical region

	Discoradicular interactions	Neurofibroma/ schwannoma
Symptoms	No root pain under 35 years	At any age
	Root pain stabilizes after 2 months	Root pain continues to worsen after 6 months
	Secondary posterolateral evolution	Primary posterolateral onset
	Coughing may hurt in the scapular area	Coughing always hurts down the arm
	Unilateral root pain	Unilateral root pain may become bilateral
	Paraesthesia felt distally	Paraesthesia is felt all over the body
Signs	Articular signs	No articular signs
	Slight segmental weakness	Unusual weakness
	Symmetrical cord signs	Asymmetrical cord signs

During functional examination, the following signs may indicate the possibility of a non-mechanical lesion:

- Absence of articular signs: because the lesion is extra-articular, neck movements are negative. It should be noted, however, that this may also happen later in the evolution of a disc protrusion compressing the nerve root.
- Unusual weakness is strongly suspicious: this means weakness out of proportion to other symptoms and signs, or affecting muscles that are not usually involved or in muscles derived from more than one segment.
- Spinal cord signs may occur (spasticity, incoordination, incontinence, sensory loss) but are rarely symmetrical.

Differential diagnosis should be made with intramedullary and extradural tumours, and non-neoplastic conditions such as syrinx, spondylotic myeloradiculopathy, multiple sclerosis or spinal arteriovenous malformation.[97]

Radiographs may remain normal for a very long time. CT, myelography and particularly MRI are very useful diagnostic procedures.[98]

The treatment of choice is surgery and the prognosis is excellent.[99,100]

Intramedullary tumours

Intramedullary spinal cord tumours are rare lesions, most frequently seen in children and young adults.[101] The majority are ependyomas and astrocytomas. Most are of low histological grade and follow indolent clinical courses, their presentation pattern depending upon the tumour's anatomical location, which poses diagnostic challenges. Most lesions occur in the cervicothoracic cord.[102] The commonest symptom is pain which may be diffuse in nature. Anterior horn cell and corticospinal tract involvement lead to upper limb weakness with small hand muscle wasting and upper motor neurone signs in the legs.[103]

The traditional treatment of intramedullary tumours is biopsy and dural decompression, followed by radiotherapy. In recent times, modern neurosurgical techniques including ultrasonic aspiration, laser, evoked potential monitoring and intraoperative ultrasonography have become available facilitating safer attempts at radical excision.[104] Aggressive tumour resection reduces chances of local tumour recurrence but carries risks of major postoperative neurological impairment.[105]

Neurological conditions with positive signs on neck examination

Some specific neurological conditions can occur to give rise to symptoms that warrant a cervical examination. Positive signs will be found during the neurological examination of the upper limb. Possible conditions are:

- Mononeuritis of the long thoracic nerve: active elevation of the arm is grossly limited
- Mononeuritis of the spinal accessory nerve: active elevation of the arm is slightly limited
- Mononeuritis of the suprascapular nerve: resisted abduction and external rotation of the shoulder are weak
- Neuralgic amyotrophy: several muscles are weak at random.

These conditions are discussed extensively in the online chapter *Nerve lesions and entrapment neuropathies of the upper limb*.

Visceral conditions

Lesions of the lung may give rise to pain felt in the shoulder or the upper thoracic area. In particular, pulmonary or visceral lesions interfering with the diaphragm (a C3–C4 structure) cause pain felt at the point of one shoulder. Heart diseases may give rise to multisegmental reference of pain, especially in the C3 dermatome, or to segmental reference of pain, felt locally in the upper thorax or down the inner aspect of the upper limb (T1–T2 dermatomes). Inflammation of the gallbladder may provoke symptoms in the scapular area. Mobility of the shoulder can become impaired as the result of the dense scarring which accompanies healed apical phthisis, limiting the mobility of the costocoracoid fascia.

Shoulder girdle disorders causing cervicoscapular pain

- Posterior sternoclavicular syndrome
- Lesion of the first costotransverse joint.

Sternoclavicular arthritis (especially when the posterior fibres of the joint are affected) or a lesion of the first costotransverse joint may give rise to pain felt in the trapezius area and so are recognized during cervical examination. The former – posterior sternoclavicular syndrome – presents with pain on shrugging the shoulder and on full arm elevation. The latter gives rise to signs on three levels: cervical, shoulder girdle and shoulder.

In both cases the complete shoulder girdle examination must be done and is diagnostic. These conditions are discussed in detail in Chapter 23.

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Headache and vertigo of cervical origin

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Headache

Headache is one of the most prevalent pain disorders, affecting 66% of the global population, with a daily prevalence of 4–5%.[1] This major health problem, disturbing both quality of life and work, is comparable to back pain in that it affects mostly the active population and thus has socio-economic consequences, generating high costs for therapy as well as for absence from work. It was reported in 1999 that in the US alone, migraine headache cost American employers about $US13 billion per year because of missed workdays and impaired work function.[2] Headache is not an easy issue. It occurs in many forms and the causal mechanisms are still not well understood.[3] Furthermore, the patient's description may be so vague that distinction between the common benign forms and more serious syndromes is sometimes difficult. Sensitive structures within the skull can become stimulated by tension, stretching, compression or displacement, all of which can be the consequence of inflammation (e.g. meningitis), increase of pressure (e.g. tumour, haematoma) or decrease in pressure (e.g. lumbar puncture). The same happens when vascular structures dilate or constrict.

Very often the problem lies outside the skull, as in tension-type headache of muscular contraction, trigeminal neuralgia, temporal arteritis and conditions affecting the teeth, the temporomandibular joint, the eyes, the sinuses or other structures in this region. Headache may also be psychogenic.[4]

The differential diagnosis of headache is challenging and should proceed in an orderly fashion. Crucial elements include a thorough history, supplemented by general medical and neurological examinations, as well as laboratory testing and neuroimaging in selected patients. By using a logical and clinical classification system, the clinician will be able to identify those patients with headache suited to orthopaedic therapy.

The first step is to distinguish primary from secondary headaches. The International Headache Society (IHS) has classified headaches as primary, where there is no other causative factor, or secondary, where the headache occurs in close relationship to another disorder to which it is attributed.[5] The former tends to decline with age, while the prevalence of the latter increases.[6]

Primary headache is usually classified into three main groups. The most common form is tension-type headache, a mild to moderate dull pain, often brought on by stress which has a global prevalence of 83%.[7] Migraine has a prevalence of 10%, and cluster headache constitutes for about 3% (Box 1). Except migraine, which can sometimes be treated with techniques described in this book, these primary headache disorders fall outwith the scope of this work and will, therefore, only be discussed briefly.

Secondary headaches are related to other disorders. This can be a local lesion in the head or neck or a generalised lesion (Box 2). Orthopaedic medicine is interested in the secondary headaches in which the cause must be sought in the cervical spine.[8]

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Box 1

Primary headaches

Tension–type headache
Migraine
Cluster headache

Box 2

Secondary headaches

Headache secondary to disorders of the:

- skull (e.g. Paget disease, mastoiditis, secondary malignancy)
- ears (e.g. otitis media or externa)
- eyes (e.g. glaucoma, strabismus, ocular strain, iritis)
- nose and nasal sinuses (e.g. acute or chronic sinusitis)
- teeth (e.g. tooth abscess, malocclusion)
- cervical spine (e.g. cervical spondylosis, disc lesion, ligamentous lesions)
- cranial nerves (e.g. herpes zoster, occipital neuralgia)

Headache secondary to:

- intracranial vascular disorders (e.g. venous sinus thrombosis, ruptured aneurysm, cerebral haemorrhage)
- extracranial vascular disorders (e.g. carotid artery dissection, cranial arteritis or carotidynia)
- disorders of intracranial pressure (raised intracranial pressure or low cerebrospinal fluid pressure, e.g. post–lumbar puncture leak)
- intracranial infections (e.g. encephalitis or meningitis)

Headache attributable to:

- generalized infection (e.g. influenza, typhoid or malaria)
- medication (e.g. vasodilators)
- hypertension

Primary headache

Tension-type headache

Tension-type headaches (TTH) are the most common chronic headaches. Rasmussen and colleagues surveyed 1000 adults from the general population and reported a lifetime prevalence of TTH of 69% in men and 88% in women.[9] Jensen reported a lifetime prevalence of 78% in a general adult population.[10] Some 30% were affected more than 2 weeks a year and 3% were labelled as chronic. The term describes headaches that have previously been grouped under various ill-defined headings such as 'tension headache', 'stress headache' and 'muscle contraction headache'.

TTH is described as a dull, non-pulsatile pain, affecting the entire head, of oppressive and progressive character, moderate or severe intensity, variable duration (up to several days) and lacking the typical features of migraine. In 90% of cases the pain is bilateral, the typical location being in the occipital, parietal, temporal and frontal areas.[11,12] Though the duration and intensity of the pain is variable, this headache is not as debilitating as migraine, and sufferers are usually able to continue their daily activities.

Box 3

International Headache Society criteria for tension-type headache

A. Headache lasting from 30 min to 7 days
B. Two or more of the following pain characteristics:
   1. Pressing or tightening (non–pulsating quality)
   2. Mild or moderate intensity that does not prohibit activity
   3. Bilateral location
   4. No aggravation by climbing stairs or similar routine activity.
C. Both of the following:
   1. No nausea or vomiting (anorexia may occur)
   2. Photophobia and phonophobia are absent.

Box 3 cites the IHS operational criteria for tension-type headache.[13]

Although TTH is the most frequent type of primary headache (two-thirds of the population have suffered an episodic TTH), its physiopathology is still the cause of controversy. For many years it has been thought that TTH was directly related to muscular tension. However, more recently it has been postulated that, although muscular tension is usually present in most cases, it is possible that the origin is more central, due to the hyperexcitability of the trigeminal caudal nucleus and of other structures of the central nervous system that register, modulate and interpret head pain. Precipitating factors for tension-type headache are emotional stress, anxiety, depression and myofascial pain.[14,15] This pathology type may also be induced, intensified or made chronic by analgesic abuse.[16]

Treatment

For acute episodes, the most common treatment involves the use of simple analgesics and anti-inflammatory medications. For chronic tension-type headache and for prevention, amitriptyline[17,18] is considered the treatment of choice, but also nortriptyline, mirtazapine[19] and tizanidine can be used. Amitriptyline (AMT) is a tricyclic antidepressant, possessing an analgesic effect that is independent of its antidepressive effect. The analgesic mechanism is not precisely known. Probably, serotonin (5-HT) and noradrenaline reuptake inhibition of the CNS plays a fundamental role in the control of the pain.[20]

Migraine headache

Migraine is a neurovascular disorder characterized by neuronal aura symptoms and vascular headache. Since 1988 migraine is defined by the criteria set by the Headache Classification Committee of the International Headache Society (IHS).[13] It consists of two subgroups: migraine with aura and migraine without aura. In 20% of cases, the migraine headache is preceded by a visual hallucination/illusion known as an aura. Typically, the aura is a serrated arc of scintillating, shining, crenulated shapes, beginning adjacent to central vision and expanding peripherally over 5–20 min, within one visual field, usually followed by headache. The scintillations are followed temporarily by a blind region, after the same retinotopic

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progression from central to peripheral visual fields. Sometimes the visual hallucinations are accompanied by unilateral paraesthesia or numbness, hemiparesis and dysphasia. The typical characteristics of migraine headache are unilateral location, pulsating quality, moderate or severe intensity and aggravation by routine physical activity. The pain peaks and then subsides, and usually lasts between 4 and 72 hours in adults and 1 and 48 hours in children. The frequency of attacks is extremely variable, from a few in a lifetime to several times a week, and the average migraineur experiences from one to three headaches a month. The pain is accompanied by other features. Nausea occurs in almost 90% of patients, while vomiting occurs in about one-third of patients. Many patients experience sensory hyperexcitability manifested by photophobia, phonophobia, osmophobia and seek a dark and quiet room. Blurred vision, nasal stuffiness, diarrhoea, polyuria, pallor or sweating may be noted during the headache phase. There may be localized oedema of the scalp or face, scalp tenderness, prominence of a vein or artery in the temple, or stiffness and tenderness of the neck. Impairment of concentration and mood are common. Box 4 presents the diagnostic criteria.[21]

Many theories have been formulated in the last six decades about the pathogenesis of migraine, but the problem is still not fully clarified.

The first theory, conceived by Wolff in 1948, is build up on a vascular basis, considering three observations: (a) during the migraine attack extra-cranial vessels dilate and are throbbing in many patients; (b) the stimulation of intra-cranial vessels provokes an ipsilateral headache; (c) vasoconstrictor drugs, like ergot derivatives, show a curative effect. On these observations, he hypothesized that an intra-cranial vasoconstriction could be responsible of the migraine aura and of the following hyperaemic reaction with local vasodilatation and activation of the perivascular nociceptive endings, resulting in acute unilateral headache.[22]

Another possible interpretation of the migraine pain is proposed on the basis of a depression of the cortical electrical activity, the so-called 'cortical spreading depression theory'. Shortly before migraine headache begins, a spreading depression develops in the cortex of one hemisphere. This cortical spreading depression (CSD) is a relatively short-lasting wave of depolarization that spreads across the surface of the brain, moving from the back (occipital region) of the cerebral cortex toward the front at about 3–5 mm/minute. This phenomenon is frequently referred to in the literature as the 'spreading depression of Leao'.[23] CSD begins with a brief wave of excitation, followed by a prolonged period of neuronal depression, which is associated with disturbances in nerve cell metabolism and regional reductions in blood flow.[24] Experimental evidence supports a relationship between CSD as a cause of migraine aura.[25] Also during an aura, cerebral blood flow abnormalities are often seen. Support for the CSD theory comes from observations that, in patients who have migraine with aura, a gradual spread of reduced blood flow that mimics the rate of progression of CSD can be measured during the aura phase.[26]

Current theories however explain migraine as a neurovascular disorder[27] in which the trigeminocerebrovascular system plays an unique pivotal role. It is hypothesized that the primary dysfunction in migraine occurs within the central nervous system and that this evokes changes in blood vessels within pain-producing intracranial meningeal structures that give rise to headache pain.[28] The brain itself is not provided with pain sensitive endings, while meninges are rich of nociceptors.[29] Tracing studies have identified the trigeminal nerve as the major afferent pathway for pain from the vessels and dura mater.[30] This trigeminovascular system consists of the neurons that innervate the cerebral vessels and the dura and whose cell bodies are located in the trigeminal ganglion. This ganglion contains bipolar cells. Peripheral fibres innervate blood vessels in the meninges, the extracranial arteries, and those in the circle of Willis. These nerve fibres contain nociceptors that are capable of generating pain impulses, and the endings of these nerve fibres contain peptide neurotransmitters.[31] The centrally projecting fibre synapses in the caudal brain stem or high cervical cord. The trigeminal innervation is predominantly to the forebrain but extends posteriorly to the rostral basilar artery, whereas the more caudal vessels are innervated by the C2 and C3 dorsal roots, which also synapse with the central trigeminal neurons.

The neurovascular hypothesis proposes that either migraine triggers or CSD (cortical spreading depression)[32] can activate trigeminal nerve axons, which then release neuropeptides (such as substance P, neurokinin A, and CGRP) from axon terminals near the meningeal and other blood vessels.[33] Substance P and neurokinin A cause vasodilation and promote the extravasation of plasma proteins and fluid from nearby meningeal blood vessels, where they produce an inflammatory

Box 4

International Headache Society:

Diagnostic criteria for migraine with and without aura

Migraine without aura

A. At least five attacks fulfilling B–D
B. Headache attacks lasting 4–72 hours
C. Headache has at least two of the following characteristics:
   1. Unilateral location
   2. Pulsating quality
   3. Moderate or severe intensity (inhibits or prohibits daily activities)
   4. Aggravation by climbing stairs or similar routine physical activity.
D. During headache at least one of the following:
   1. Nausea and/or vomiting
   2. Photophobia and phonophobia.

Migraine with aura

A. At least two attacks fulfilling B
B. At least three of the following four characteristics:
   1. One or more fully reversible aura symptoms
   2. At least one aura symptom develops gradually over more than 4 minutes or, 2 or more symptoms occur in succession
   3. No aura symptom lasts more than 60 minutes
   4. Headache follows aura with a free interval of less than 60 minutes.

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response. This response is termed sterile neurogenic perivascular inflammation. The neuropeptides may also sensitize nerve endings, providing a mechanism for sustaining the headache. When activated, the trigeminal nerve also transmits pain impulses to the trigeminal nucleus caudalis, which relays pain impulses to higher centres of the brain. According to the neurovascular theory, vasodilation is not the cause of migraine headaches but is an accompanying phenomenon attributable to trigeminal nerve activation. Although the cause of this activation is not known, it may be due to ionic and metabolic disturbances in brain function, such as those associated with CSD. It has also been proposed that abnormal activity in brain stem sensory nuclei may cause antidromic activation of trigeminal sensory pathways.[34]

The integrated hypothesis of migraine pathogenesis is an attempt to consolidate various theories and explain several observations related to migraine pain. According to this theory, triggers such as stress, glare, noise, the patient's internal clock, the dilation of the internal or external carotid arteries, or other factors may activate specific centres in the brain stem. One such centre, the locus ceruleus, causes changes in epinephrine levels. Another centre, the dorsal raphe nucleus, affects serotonin levels in the brain.[35] They cause constriction of cerebral blood vessels and a localized deficiency in blood flow, provoking CSD. This, in turn, stimulates trigeminovascular fibres, eliciting neurogenic inflammation and headache pain. Nerve fibres from the locus ceruleus, the dorsal raphe nucleus, and the trigeminal nerve cause a stimulation of cranial nerves that dilate both cerebral and extracranial blood vessels. The dilation of meningeal vessels contributes to pain generation.[36] The locus ceruleus also sends fibres to higher centres of the cerebral cortex, where it influences a person's state of arousal and awareness, and descending projections interact with the body's pain control mechanisms. Likewise, the dorsal raphe nucleus sends multiple fibres to blood vessels and upward toward the cerebral cortex. These serotonin-secreting fibres help regulate sleep and neuro-endocrine functions. Other connections are made with lower brain stem areas and with the hypothalamus. A disruption in the normal function of the hypothalamus may be responsible for prodromal signs and symptoms of migraine such as mood changes, food cravings, drowsiness, thirst and yawning.[37] These signs and symptoms may occur several hours, or even as long as 1 day, before headache pain begins.

Treatment

The classic therapeutic approach has three major pillars: avoidance of migraine triggers, treatment of the acute attack by medication and regular use of preventive medications.

Acute attacks are treated with simple analgesics and NSAIDS, and more migraine-specific drugs, such as ergot-derivates[38] and triptans, which are active at 5-HT1 receptors. However, triptans (sumatriptan and its six licensed successors), because of their better tolerability, have replaced ergotamine in most cases.[39] They reduce neuronal activity via these receptors at the trigeminocortical complex and thalamic level. There are still situations where tolerability and contraindications to use are a problem. The main issue for triptans relates to their vasoconstrictor properties and related cardiovascular and cerebrovascular safety concerns. This necessitates that triptans are not used in patients with cerebrovascular or cardiovascular contraindications.[40]

Prevention

Migraine prevention is an important component of therapy aimed at reducing the attack frequency and severity. Unfortunately, the mechanisms of action of current preventives are not well understood. A potential mechanism is the inhibition of cortical spreading depression but, as noted above, the efficacy against cortical spreading depression does not necessarily predict the efficacy in treating migraine without aura. Substances that have proven beneficial in migraine, with and without aura, broadly comprise compounds from the following classes: beta-blockers (propranolol), antidepressants (amitriptyline), anticonvulsants (valproate and topiramate), calcium channel blockers (flunarizine) and serotonin antagonists (methysergide). According to the pathophysiological concepts discussed above, these drugs most probably target the activity of modulatory circuits as well as the neuronal activity in afferent sensory pathways such as the trigeminal system.[41] Although many patients can be effectively managed using the available substances, side effects and contraindications because of co-morbidities can complicate treatment. A particular problem is the prediction of which patients will respond to which substance as treatment is still largely conducted by trial and error.

Migraine and orthopaedic medicine

On empirical grounds Cyriax[42] found that manipulation of the cervical spine may have a therapeutic effect. He discovered that the onset of an acute attack could sometimes be aborted by 30 seconds of strong neck traction. Other observations have also demonstrated some relationship between mechanical stimulation of the occipital nerve and induction or abortion of migraine attacks.[43] Also nerve blocks of the greater occipital nerves proved to be curative in a high percentage of migraine sufferers.[44,45] These results reinforce previous evidence of convergence of cervical afferents on the trigeminal sensory circuit and suggest that, by modifying the central processing of pain signals in migraine in the thalamus, greater occipital nerve blocks shut down several symptom generators.[46,47] Therefore, manipulation of the cervical spine can have a preventive effect in some patients, especially the middle-aged or elderly and it is always worthwhile manipulating the cervical spine in migraineurs of over 40 years of age. When the neck movements are painless, one session usually suffices. Manipulation is performed in four directions: both rotations and both lateral flexions. Painful neck movements may require two or three sessions. The techniques are described in Chapter 11.

Cluster headache

Also called 'Horton's neuralgia', this headache is characterized by recurrent short-lasting attacks (15 to 180 minutes) of excruciating unilateral periorbital pain accompanied by ipsilateral autonomic signs (lacrimation, nasal congestion, ptosis, miosis, lid oedema, redness of the eye).[48,49] It affects young adults, predominantly males. Prevalence is estimated at 0.5–1.0/1000. Cluster headache (CH) has a circannual and circadian periodicity, attacks being clustered (hence the name) in

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bouts that can occur during specific months of the year. An attack can be triggered by alcohol, strong odours and napping. During bouts, attacks may happen at precise hours, especially during the night. Cluster headache seems to be associated with trigeminovascular activation and neuroendocrine and vegetative disturbances, though the precise causative mechanisms remain unknown. Involvement of the hypothalamus has been confirmed, explaining, at least in part, the cyclic aspects of CH.[50] The disease is familial in about 10% of cases. Genetic factors play a role in the susceptibility, and a causative role has been suggested for the hypocretin receptor gene. Diagnosis is clinical. Differential diagnoses include other primary headache diseases such as migraine. At present, there is no curative treatment. There are efficient treatments to shorten the painful attacks and to reduce the number of daily attacks. Acute treatment is based on subcutaneous or intra-nasal administration of sumatriptan.[51] Verapamil, lithium, methysergide, prednisone, greater occipital nerve blocks and topiramate may be used for prophylaxis. In refractory cases, deep-brain stimulation of the hypothalamus and greater occipital nerve stimulators have been tried in experimental settings.[52] The disease course over a lifetime is unpredictable. Some patients have only one period of attacks, while in others the disease evolves from episodic to chronic form.

Cervicogenic headache

Cervicogenic headache (CEH) is a syndrome characterized by chronic hemi-cranial pain that is referred to the head from either bony structures or soft tissues of the neck. Sjaastad and colleagues[53,54] have, in consecutive publications since 1990, established criteria for the diagnosis of headache of cervical origin (Box 5), since 1983 known as 'cervicogenic headache'.

The pain is mild, usually has an undulating course and may eventually become chronic. There is a marked female preponderance. The neck seems to be involved, either because there has been a trauma, for example whiplash, or because there is limitation of neck movement. There may be accompanying shoulder and/or arm pain. An attack can be precipitated either as the result of neck movement or of direct pressure, for example on examination.[55] In their early publications Sjaastad *et al* mention the headache as being strictly unilateral. Later they restate this and define unilaterality as follows: the headache dominates on one side; when weak, the pain may be only on that side; when severe, it may also be felt on the contralateral side, but to a lesser extent. It never dominates on the contralateral side.[56]

The fact that neck structures can give rise to headache is now generally accepted. There seems to be a consensus on the pathophysiology. The neuroanatomical basis for CEH is the 'trigemino-cervical nucleus'. This is a region of the upper cervical spinal cord where sensory nerve fibres in the descending tract of the trigeminal nerve (trigeminal nucleus caudalis) are believed to interact with sensory fibres from the upper C1–C4 cervical roots. This functional convergence of upper cervical and trigeminal sensory pathways allows the bi-directional referral of painful sensations between the neck and trigeminal sensory receptive fields of the face and head. C1 spinal nerve has some ectopic sensory ganglia and it innervates the short muscles of the suboccipital triangle.[57] The C2 spinal nerve gives sensory supply to the median and lateral atlantoaxial joints; to several neck muscles (prevertebral, sternocleidomastoid, trapezius, semispinalis and splenius muscles); to the dura of the posterior cranial fossa and the upper spinal canal. Both the C2 and C3 spinal nerves supply the zygapophyseal joints of the adjacent segments. The atlantoaxial ligaments and the dura mater of the spinal canal are innervated by the sinuvertebral nerves stemming from the C1–C3 spinal nerves. The origin is sympathetic; the nerves contain nociceptive, proprioceptive, vasomotor and vaso-sensory fibres.[58]

Box 5

Cervicogenic headache:

Diagnostic criteria[13]

Major criteria

I. Symptoms and signs of neck involvement (it is obligatory that one or more of the phenomena is present)
   a. Precipitation of comparable symptoms by:
      1. neck movement and/or sustained, awkward head positioning, and/or
      2. external pressure over the upper cervical or occipital region
   b. Restriction of range of motion in the neck
   c. Ipsilateral neck, shoulder or arm pain
II. Confirmatory evidence by diagnostic anaesthetic block
III. Unilaterality of the head pain, without sideshift

Head pain characteristics

IV. Moderate–severe, non-throbbing pain, usually starting in the neck; episodes of varying duration, or fluctuating, continuous pain

Other characteristics of some importance

V. Only marginal or lack of effect of indomethacin; only marginal or lack of effect of ergotamine and sumatriptan; female gender; and not infrequent history of head or indirect neck trauma, usually of more than medium severity

Other features of lesser importance

VI. Various attack-related phenomena, only occasionally present, and/or moderately expressed when present:
   a. nausea
   b. phono- and photophobia
   c. dizziness
   d. ipsilateral 'blurred vision'
   e. difficulties swallowing
   f. ipsilateral oedema, mostly in the periocular area

Cervicogenic headache and orthopaedic medicine

There are a number of orthopaedic medical conditions that can give rise to headache and that often can be approached very successfully by the use of local treatment (see p. 122). Lesions of the extracranial soft tissues of the locomotor system, especially the capsulo-ligamentous structures of the occipito-atlantoaxial complex, may give rise to segmental headache. The cervical dura mater is often responsible for the vague,

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multisegmental occipito-frontal, temporal or retro-orbital pain (see p. 16).

Headache, referred from the cervical dura mater

A disco-dural interaction at any cervical level or any space-occupying lesion in the spinal canal may give rise to pain felt in the head. The pain usually radiates from the mid-neck up to the temple, the forehead and behind one or both eyes but rarely to the bridge of the nose. This 'multi-segmentally referred pain' is dural in origin and will disappear when treatment to the neck is accomplished. For further details see p. 16.

Matutinal headache in the elderly

As the result of arthrosis at the upper cervical joints, ligamentous contracture may develop and can result in 'segmental' pain felt in the upper cervical dermatomes which cover the head. The patient typically complains of occipito-frontal headache felt especially in the morning. Treatment includes capsulo-ligamentous stretching. For a detailed description see p. 163.

Postconcussional headache

The upper cervical capsules and ligaments can also become sprained during trauma, for example an accident that causes concussion of the brain after which a period of immobilization follows. The subsequent build-up of ligamentous adhesions finally results in 'segmental' headache. Several authors recognize the possibility of upper cervical capsuloligamentous conditions causing pain in the head. The adhesions can be broken manipulatively. A full description can be found on p. 168.

Vertigo

Vertigo or dizziness is not an uncommon complaint of patients who suffer from disorders that affect the cervical spine. Most often these symptoms are not related to the conditions but just occur at the same time as the cervical disorder. If the dizziness is caused by the functional disturbance in the neck, it may be expected to be as 'treatable' as any other symptom originating in that area. It would therefore be very helpful to be able to differentiate 'cervical vertigo' from other possible causes.

Vertigo originating as the result of movements of the neck initially suggests a cervical origin. These movements, however, influence not only the proprioceptive system of the cervical spine but also the blood flow through the vertebrobasilar arteries. At the same time, the vestibular apparatus may also be disturbed. All these factors in control of balance are interrelated.

Anatomy

Man is aware of his position in space. The cortex therefore receives information from three different systems: optical, vestibular and proprioceptive (superficial and deep). Disturbances in one of these areas often result in vertigo.

Optical system

Visual stimuli pass via the optical nerve, the optic chiasma and the optic tracts, mainly to the occipital cortex, though some traverse the mesencephalon and interact with eye movement, vestibular activity and the muscles of the cervical spine.

Vestibular system

The vestibular system consists of a peripheral part (organ of balance and vestibulocochlear nerve) and a central part (vestibular nuclei and vestibulospinal tract).

Peripheral part

The osseous labyrinth lies in the temporal bone and has three membranous semicircular canals (membranous labyrinth) (Fig. 1). Each lies in contact with an ampulla that contains sensory receptors – the cupulae – a gelatinous substance that

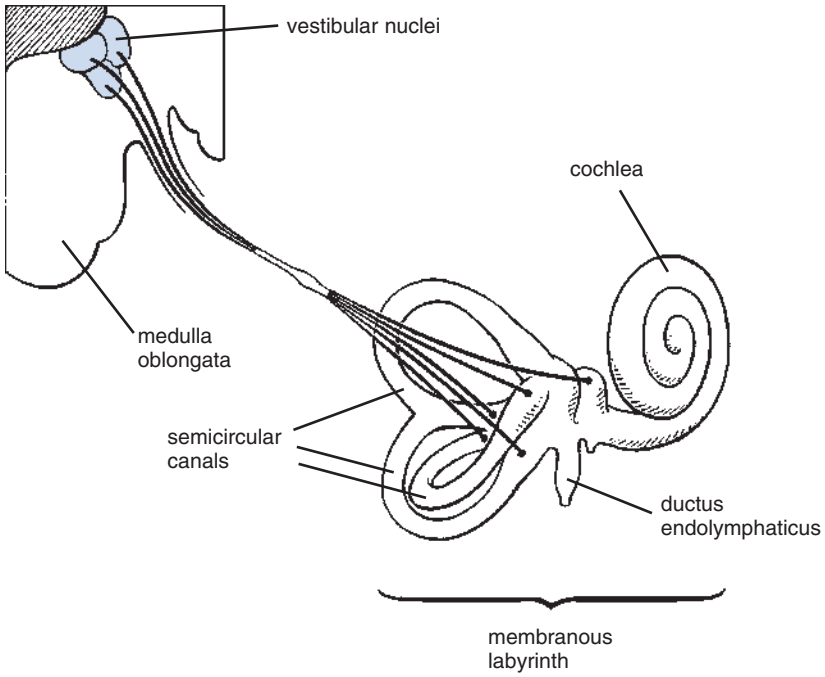

Fig 1 • The vestibular system.

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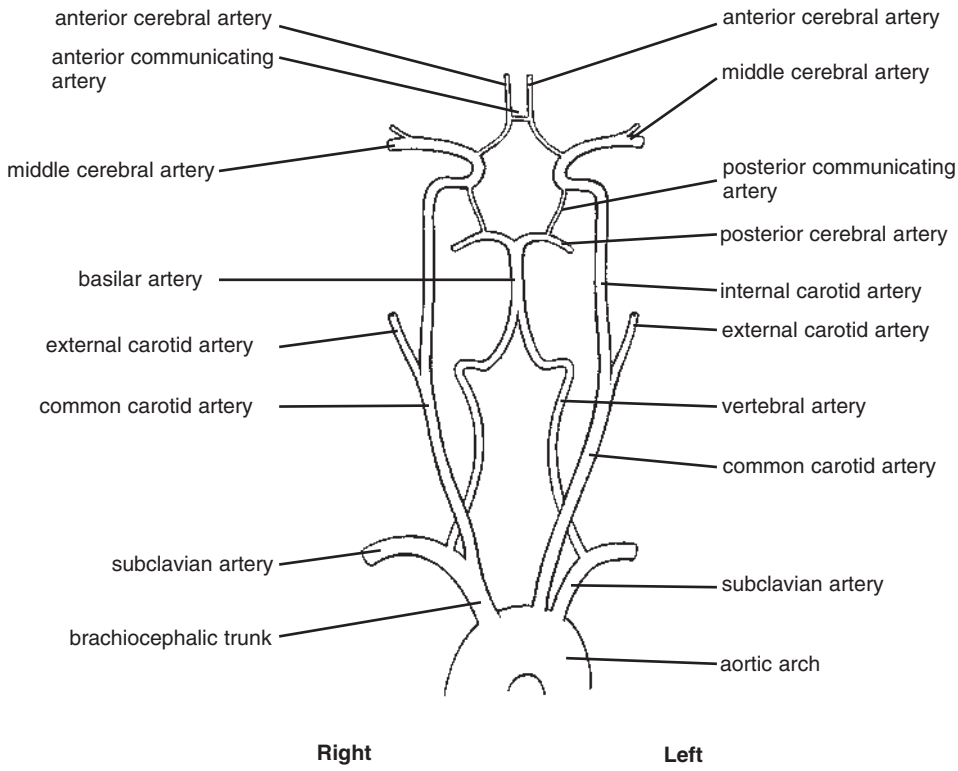

Fig 2 • The vertebrobasilar system.

moves in the endolymph and in which the otoliths float. It also contains the sensory hair cells. Impulses generated by movement of otoliths within the endolymph pass via the vestibulocochlear nerve (vestibular part) to one of the four vestibular nuclei.

Central part

This is formed by the four vestibular nuclei in the white substance of the pons cerebri from which several tracts originate (e.g. the vestibulospinal tract). These connections facilitate orientation of the position of the body.

There are also many cerebellovestibular connections, responsible for good judgement and adaptation of the direction of movements and for control of anti-gravity muscles.

Much information comes from the vestibular nuclei to modulate the activity of the oculomotor nuclei and thus the ocular muscles. These stimuli cause nystagmus, the main sign of a vestibular disorder.

Proprioceptive system

Superficial, fine tactile sensations are conveyed through the anterior spinothalamic tract. Deep tactile sensations (conscious proprioception is a combination of deep sensation, discrimination sense and vibratory sensation) are transmitted along the fasciculus gracilis and the fasciculus cuneatus. Reflex proprioception (unconscious proprioception) is conveyed centrally via the ventral and dorsal spinocerebellar tracts and the olivospinal tract.

Blood supply

The vertebrobasilar system is a 'closed' circuit, starting below in the subclavian arteries and ending above in the arterial circle of Willis (Fig. 2).

The left subclavian artery originates directly from the aortic arch as does the left common carotid artery. The right subclavian artery and the right common carotid artery are branches of the brachiocephalic trunk. The common carotid artery divides (usually at the level of the fourth cervical vertebra) into the external and internal carotid arteries.

The external carotid artery is of less importance in this context and supplies the outer parts of the head – the face, the temporal area, the occipital region, the skin of the head and the mouth.

The internal carotid artery gives off very few branches during its extracranial course but supplies a considerable part of the brain via the anterior cerebral (medial aspect of the hemispheres) and the middle cerebral artery (a continuation of the internal carotid artery, which supplies the entire outer and lateral aspects of the brain), before it anastomoses again with the vertebrobasilar system.

The vertebral arteries originate bilaterally from the subclavian arteries, of which they usually form the first and biggest branches. They run parallel on both sides of the spinal column and form the main blood supply for the brainstem, the cervical spinal cord and the cervical spine. They are closely related to this part of the spinal column and the vertebrae are adapted

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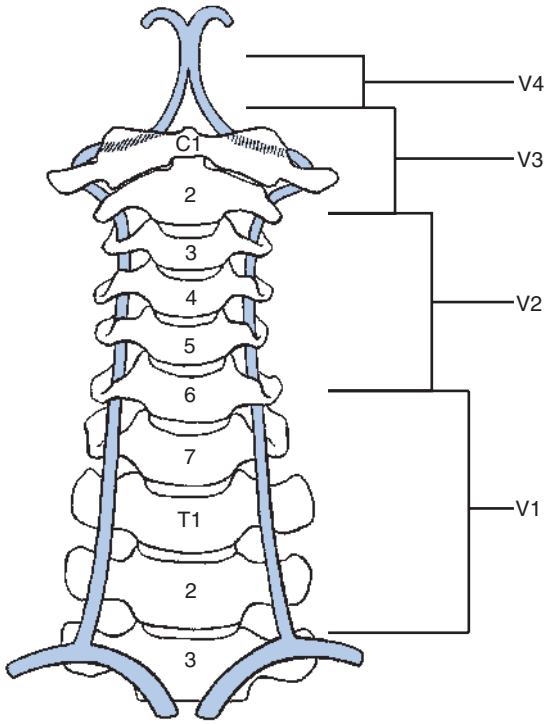

Fig 3 • The course of the vertebral artery: V1, extravertebral segment; V2, intervertebral segment; V3, atlantoaxial segment; V4, subforaminal and intracranial segment.

to the presence of the artery in that the transverse processes C1–C6 contain a transverse foramen through which the artery runs (see online chapter *Applied anatomy of the cervical spine*). A groove for the artery lies above the posterior arch of the atlas, dorsal to the lateral masses. Occasionally, this groove is closed to form an arterial canal.

The vertebral artery is divided into four segments[59,60] (Fig. 3):

Extravertebral segment

The artery runs upwards from the subclavian artery to the transverse foramen of the sixth cervical vertebra. It is surrounded anteriorly by the anterior scalenus and longus colli muscles; posteriorly and distally it is adjacent to the first rib and the transverse processes of the first thoracic and the seventh cervical vertebrae.

Intervertebral segment

From the sixth to the second vertebrae the vertebral artery passes through a canal formed by the transverse foramina and, between the vertebrae, by ligamentous and muscular structures. The anteromedial border is formed by the uncovertebral joints (von Luschka's joints).

The vertebral artery runs anterior to the nerve roots and spinal nerves, the latter lying in the sulcus for the spinal nerve. An uncoarterioradicular junction is thus formed. This is of clinical importance because arthrotic changes at these levels may have important consequences for the blood flow in these arteries.

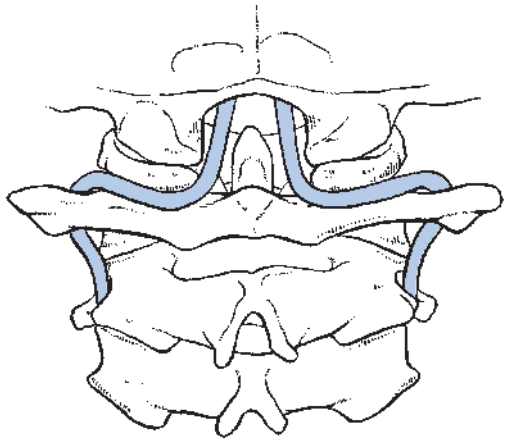

Fig 4 • The course of the vertebral artery in the atlantoaxial area.

The contents of the transverse foramina include, in addition to the vertebral artery, two vertebral veins, the periarterial venous plexus of the vertebral artery and the vertebral nerve.

Atlantoaxial segment

Between the axis and atlas the artery curves backwards and outwards (Fig. 4) because the transverse foramina of the atlas lie more laterally than those of the other vertebrae. The artery runs dorsally around the lateral mass of the atlas and loops over the posterior arch in the arterial groove. It is surrounded anteriorly by the joint capsules of the atlanto-occipital joints and posteriorly by the obliquus capitis superior and rectus capitis posterior major muscles.

Subforaminal and intracranial segment

The artery curves upwards again and runs cranially from posterolateral to anteromedial to pierce the posterior atlanto-occipital membrane, the dura mater and the arachnoid mater, and to enter the skull via the foramen magnum.

Just below the base of the brain and at the level of the pons, the left and right arteries join to form the basilar artery. The latter trunk then splits into a left and right posterior cerebral artery. From the vertebral and basilar arteries, branches originate to supply parts of the brain, especially the cerebellum.

A characteristic of the blood supply of the brain is the close connection of the different arteries by means of communicating arteries. Together they form the arterial circle of Willis. From posterior to anterior the circle of Willis contains two posterior cerebral arteries, both originating from the basilar artery. They are both connected via posterior communicating arteries with their respective internal carotid artery. From here depart both anterior cerebral arteries, connected by one anterior communicating artery. The circle is thus completed.

Blood supply by the vertebral arteries

At the cervical level the vertebral arteries give off cervical branches and cranial branches, the latter anastomosing with the spinal rami.

There are extensive anastomoses with the deep cervical artery (originating via the costocervical trunk from the subclavian artery), the thyrocervical trunk and its branch (the

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ascending cervical artery) and the occipital artery (branch of the external carotid artery). Spinal rami supply the anterior and posterior roots, the epidural blood vessels and the vertebral canal, especially the upper two vertebrae. Articular rami supply the joint capsules of the intervertebral and uncovertebral joints. Muscular rami supply the intrinsic muscles of the cervical spinal column and cutaneous branches supply the skin.

Just before both vertebral arteries join to form the basilar artery, they give off, unilaterally or bilaterally, a branch that forms (together with its fellow) the anterior spinal artery, which supplies the anterior aspect of the spinal cord.

The posterior spinal arteries also originate from the vertebral arteries and supply the posterior aspect of the spinal cord, where they anastomose extensively with the spinal rami.

Intracranially, the branches that form the posterior inferior cerebellar arteries supply the posterolateral part of the medulla oblongata, part of the posterior lobe of the cerebellum, the vermis and the cerebellar nuclei.

The basilar artery and its branches form the blood supply for the medulla oblongata, the reticular formation, the pons, the mesencephalon, parts of the cerebellum (via the anterior inferior cerebellar artery and the superior cerebellar artery), the vestibular system and its nuclear complex (via the labyrinthine artery).

The posterior cerebral arteries are part of the circle of Willis via anastomoses with the internal carotid artery. They supply parts of the thalamus and hypothalamus, the occipital lobe, large parts of the temporal lobe, the red nucleus, the substantia nigra, the nuclei of the oculomotor nerve (III) and the trochlear nerve (IV).

Blood flow

Together the vertebral arteries form a functional unit. In normal circumstances, disturbance of the function of one artery is immediately compensated for by the other, provided it is healthy. Failure adequately to compensate leads to complaints and symptoms, especially vertigo and tinnitus. Provocation tests, directed at the function of the vertebrobasilar system, are designed to test this compensation mechanism (see later).

Severely diminished flow in one vertebral artery may well lead to occlusion of the posterior inferior cerebellar artery on that side, resulting in a lateral medullary infarction – Wallenberg's syndrome (Box 6).

Blood flow in the vertebral arteries may be influenced by movement, compression, trauma or vascular factors.[61] However, the functional adaptability of structures within the spinal canal, and intervertebral and transverse foramina is so high that anatomical changes such as narrowing do not necessarily cause symptoms.

Movement

Different authors agree that maximal rotation of the cervical spine diminishes blood flow in the contralateral vertebral artery. This effect is greatly increased when rotation is combined with flexion, extension and/or side flexion.

Chrost, cited by Gutmann and Biedermann,[62] has made a survey in terms of percentage of the blood flow in the vertebral arteries in different positions of the head (Table 1). Flexion and extension movements have very little influence on the blood flow in the vertebral arteries because the axis of flexion and extension of each segment lies in the same course.

Side flexion of the local spine gives rise to moderate diminution of blood flow in the ipsilateral artery.

Rotations have greatest influence on the blood flow. 'Redundancy' of length may also help to accommodate movements. For example, the distance between the vertebral artery and the axis of rotation is greatest in the atlantoaxial segment and redundancy is therefore required at this level in the atlantoaxial loop of the artery so that, even if it is extended during full rotation of C1 on C2, neither the lumen nor flow is reduced. During 30° of rotation there is an influence on the contralateral vertebral artery and during 45° of rotation the artery is almost completely occluded. On the other hand, there is an atlanto-occipital loop between C1 and the occiput. This curve does not change during movement and may be considered as a buffer mechanism against strong arterial pulsations.

Box 6

Characteristics of Wallenberg's syndrome

- Dysphagia and ipsilateral palatal weakness (involvement of nucleus ambiguous)
- Impairment of sensation to pain and temperature on the same side of the face (involvement of descending root of the fifth cranial nerve)
- Horner's syndrome in the ipsilateral eye (involvement of the descending sympathetic fibres)
- Nystagmus (involvement of the vestibular nuclei)
- Cerebellar dysfunction in the ipsilateral arm and leg (involvement of the restiform body and cerebellum)
- Impairment of sensation to pain and temperature over the opposite half of the body (involvement of the spinothalamic tract)

Table 1 Relationship of head position and blood flow

Head position	Impact on blood flow
Neutral position	Normal flow
Rotation	Slight decrease in the ipsilateral artery Significant decrease in the contralateral artery
Side flexion	Slight decrease in the ipsilateral artery No decrease in the contralateral artery
Flexion and extension	No decrease
Extension combined with rotation	Significant bilateral decrease, most in the contralateral artery
Flexion combined with rotation	Significant bilateral decrease
Flexion combined with side flexion and contralateral rotation	Slight decrease in the contralateral artery Cessation in the ipsilateral artery

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Compressing factors

There are a number of compressing factors.

In the extravertebral segment

Exceptionally, compression may be related to anatomical anomalies (an unusual origin of the artery) or to shortening in the prevertebral musculature.

In the intervertebral segment

Osseous growth at the edges of the vertebral bodies (spondylosis) and degenerative changes in one or more intervertebral joints (spondylarthrosis) may cause compression.[63] The intervertebral disc is of lesser importance here, except in large disc protrusions where the vertebral artery may rarely become threatened.

In the atlantoaxial segment

Circulation in the vertebral artery is impaired, on the one hand, by congenital arterial anomalies (hypoplasia of one or both vertebral arteries, aplasia of one artery, anomalies in the circle of Willis) and, on the other hand, anomalies and positional changes in the upper cervical vertebrae, such as basilar impression, an extraoccipital condyle, assimilation of the atlas (fusion of the atlas with a part of the base of the skull), osseous canal for the vertebral artery at the upper part of the posterior arch of the atlas, odontoid anomalies (aplasia, asymmetry), os odontoideum (articulation between odontoid process and the body of axis) and fusion of C1–C2.

These anomalies are rare and are not detected on routine examination.

In the subforaminal and intracranial segment

Compression here is very exceptional.

Traumatic factors

Fractures, luxations and contusions (direct or indirect) may also influence blood flow in the vertebrobasilar system.

Vascular factors

Disorders of the vascular wall, haemorrhage and embolism, the latter being quite exceptional, have direct influence on blood flow. Disturbances, especially hyperactivation, in the sympathetic system may lead to vasoconstriction.

Nystagmus

The main sign that characterizes vertigo is the presence of *nystagmus*, an involuntary repetitive movement of the eyes – the cardinal sign of a vestibular disorder.[59,64]

Classification

Nystagmus can be classified according to its nature (rhythmic or not), its direction (horizontal, vertical, rotary or mixed), and its frequency or its amplitude (broad or fine) (Fig. 5).

Types[65]

Nystagmus can be physiological or pathological.

Physiological nystagmus

Focusing nystagmus occurs when a fixed point is looked at.

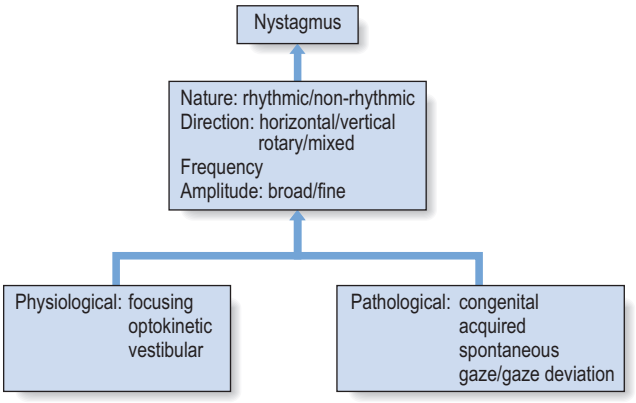

Fig 5 • Classification of nystagmus.

Optokinetic nystagmus occurs when objects pass by the onlooker with a certain regularity or when a moving onlooker passes by a number of stationary objects (e.g. looking out of a window when travelling in a train).

Vestibular nystagmus is seen when the head is quickly turned in one direction. Movement of endolymph within the canals lies in relation to that of the head and continues momentarily after the head has stopped. The cupula organs in the ampullae of the semicircular canals are moved in the direction of lymph flow and are thus stimulated. Muscles, especially the extraocular muscles, are influenced by this and the position of the eyes changes as the head is turned, in a direction opposite to the rotation. At first the eyes remain behind but are then brought back to their normal position. This eye movement, slow first and opposite to the turning direction, followed by a quick movement in the direction of the rotation is called 'nodding eye movements' or 'nystagmus'. The slow phase is caused by impulses from the labyrinth; the quick phase is the result of corrective mechanisms in the ocular muscles.

Pathological nystagmus

Nystagmus can be either congenital or acquired. In the latter, it occurs spontaneously or as the result of movement (positioning or provoking nystagmus).

Spontaneous nystagmus can have a peripheral cause. In this case, a one-sided viewing-direction nystagmus is found: a nystagmus that is latently present becomes worse when the patient is asked to look in one direction. When the problem is central, nystagmus may present in different situations:

- *Fixation nystagmus* occurs when the patient tries to fix a certain point.
- *Two-sided viewing-direction nystagmus* (symmetrical nystagmus) is seen when a person looks in one direction and then in the opposite direction.
- *Exclusive one-sided viewing-direction nystagmus* is not latently present but occurs only when looking in one direction.
- *Flagging movements* or dysconjugation of movements of the eyes is seen when the examiner makes a pendulum movement with the arm.
- *Gaze* nystagmus or *gaze deviation* nystagmus always has a central cause. It may be present for as long as the patient gazes and the eye movement may not be horizontal.

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Clinical importance

One or other type of nystagmus on clinical examination is an absolute contraindication to manipulative treatment and should be investigated by a neurologist.

Aetiology and classification of vertigo

It is usual to classify vertigo into two groups, depending on the cause: vestibular and non-vestibular vertigo. Vestibular vertigo is divided into peripheral and central types.

Vestibular vertigo

Peripheral

Vertigo is peripheral when the cause of the symptoms lies in the peripheral vestibular system, containing the labyrinth and the vestibular part of the vestibulocochlear nerve.

Peripheral vestibular vertigo is characterized by short-lived heavy turning sensations with a sudden onset. It may be accompanied by hearing disturbances[66] (tinnitus, deafness) and sometimes important autonomic nervous system symptoms, such as palpitations, anxiety, nausea, vomiting, sweating and fluctuations in blood pressure. There are some disorders that selectively involve the peripheral vestibular system to cause dizziness without hearing loss, for example benign paroxysmal positional vertigo, vestibular neuritis and bilateral idiopathic vestibulopathy.[67] Transitory spontaneous nystagmus, rotating or horizontal, and in the same direction may also be present. It is regular and with a latency period.

Most disorders in the peripheral vestibular complex lead to vertigo: injuries, infections, neoplasms, labyrinthine vascular accident, Ménière's syndrome and vestibular neuronitis.

Central

The lesion lies in the vestibular nuclei or their tracts. Central vertigo may result from injury, multiple sclerosis, tumours, cerebral arteriosclerosis and vertebrobasilar insufficiency leading to brain stem ischaemia or cerebellar stroke.[68]

Disturbances of equilibrium accompany the vertigo. Symptoms are persistent, sometimes with acute exacerbations. Auditory disturbances are rare and neurovegetative symptoms are also less pronounced.

There is persistent spontaneous nystagmus, which sometimes varies and changes direction. It is irregular and has no latency period.

Non-vestibular vertigo

This type of vertigo may have different causes: ophthalmological, psychiatric, orthostatic hypotension, visual and auditory disorders, and possibly alteration in the proprioception from the cervical joints. Hyperventilation, circulatory disorders, arterio-sclerosis, brain sclerosis, skull injury and anaemia are other possibilities.

A special form of non-vestibular vertigo occurs in disorders which affect the cervical spine. In this form, a distinction can be made between either a vascular or a proprioceptive cause. The former may follow circulatory disorders in the area supplied by the vertebral arteries; the latter results from disturbances in the proprioception of the joints of the upper cervical segments, mainly as the result of arthrosis or a disc protrusion.

Important features in the functional examination of a patient complaining of vertigo

In view of the treatment of the neck patient within orthopaedic medicine it is of extreme importance to differentiate between the following types of patients:

- The patient who shows a contraindication for any type of cervical treatment, when the problem lies within the vertebrobasilar system. The examination is then meant to recognize the possible risk factors.
- The patient whose vertigo has nothing to do with the neck, when the vestibular system is affected and in whom a normal treatment can be given to the cervical spine.
- The patient in whom the vertigo is cervicogenic and can be treated as such. This is so when the proprioceptive system is disturbed.

Requests came from several sources for the recognition of risk factors in patients who may need to receive manual and/or manipulative treatment to the neck. This has led to the development of 'Clinical Standards for Pre-manipulative Testing of the Cervical Spine'. The first and so far most important and internationally recognized standard is the Australian one,[69] from which other standards have been derived.[70–74] There are mutual differences but from these standards emerges a consensus on some important features: a patient with complaints of vertigo should be put through meticulous history-taking, an orientating otoneurological examination and a number of dizziness provocation tests.

History

The history is extremely important. The moment the patient mentions symptoms that could relate to vertigo (dizziness, tinnitus, anxiety, palpitations, excessive perspiration), the examiner should determine whether or not vertigo is truly present. This is achieved by the following sequence of questions:

- *Are there real turning sensations or just a dizzy feeling?* Turning sensations point towards a vestibular disorder, usually in the peripheral part. This also applies to motion sickness, for example car sickness, for which the cause is usually vestibular.
- *How did it start – suddenly or gradually?* This may indicate the type of vertigo present (see below).
- *How long does it last?*
- *Does it come in attacks?*
- *How severe are the complaints?*
- *Is the vertigo influenced by certain positions?*
- *Is the vertigo influenced by the position of the head?*
- *How frequently do the attacks occur?*

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Questions should also be asked about other symptoms, such as deafness, tinnitus, headache, nausea/vomiting, neurological symptoms (disturbances of vision or speech, paraesthesia, diminution of consciousness) and infection/fever.

Clinical examination is not wholly reliable and much attention should be paid to history. The diagnosis is made on the recognition of certain patterns of symptoms. The importance for the orthopaedic physician is that a correct provisional and general diagnosis is made and the patient correctly referred for specialist attention.

Based on the history, three types of vertigo can be distinguished:

- *Sudden, severe and short*: a sudden attack, coming on rapidly, lasts only a short time (from a few seconds to some hours) and disappears quite quickly. The causes can be non-vestibular (benign paroxysmal positioning vertigo, orthostatic vertigo, hyperventilation, cervical vertigo) or vestibular as the result of insufficiency of vertebral and/or basilar arteries. Differential diagnosis should be made from Ménière's syndrome, hypoglycaemia or syncopation.
- *Sudden, severe and gradually disappearing*: a sudden and severe attack is much slower to disappear (over several days to several weeks). It is caused by a peripheral vestibular condition, such as vestibular neuritis, labyrinthine injury, labyrinthine vascular accident, (peri) labyrinthitis or herpes zoster oticus. Differential diagnosis is required from multiple sclerosis.
- *Chronic, permanent/long-standing and not severe*: not very severe, but continuously present, chronic vertigo with slight exacerbations may go on for months without change. It can be caused by non-vestibular or central vestibular lesions (cerebral atherosclerosis, hypertension, tumour between the pons and cerebellum and skull injury).

Clinical examination

The information presented here is a summary of some of the clinical tests which enable distinction of a non-vestibular vertigo from a vestibular one, the latter having either a central or peripheral cause.

Orientating examination of equilibrium

The following tests are appropriate for the vestibular system:

- Neck extension
- Romberg's test
- Unterberger's test
- Babinski–Weill test
- Bárány's test.

Neck extension

The patient is asked to look at the ceiling for about 20 seconds or less if he is not able to maintain that position.

Romberg's test

Assesses static equilibrium. The patient stands with both feet together, if possible, and performs Jendrassik's grip (reinforcement) (Fig. 6). If this seems impossible, he holds his arms outstretched in front of him. When it is possible to hold the position with the eyes open, but not with the eyes closed, the test is considered positive. The patient then usually tends to fall to one side. Further neurological examination is then required.

Unterberger's test

The patient stands with the arms forwards and must try to 'step' on the spot, lifting the knees. Rotation of 45° per 50 steps is considered normal, more than this is pathological. The examiner should note the direction in which the patient moves (Fig. 7).

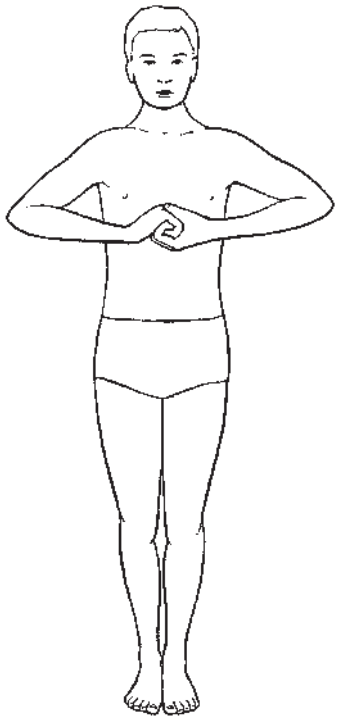

Fig 6 • Romberg's test with Jendrassik's grip.

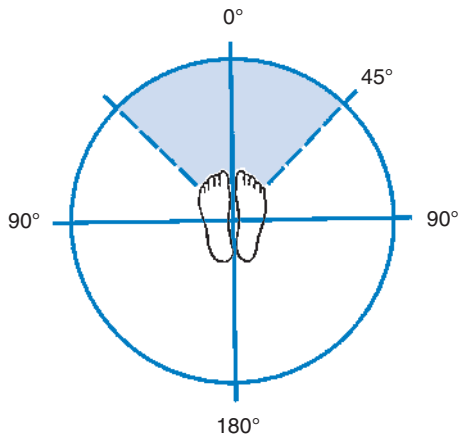

Fig 7 • Unterberger's test: the patient walks on the spot. Rotation of more than 45° per 50 steps is pathological.

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There seems to be disagreement about the value of this test. However, it was demonstrated in a prospective study of 100 patients with unilateral, subsequently histologically proven acoustic neuroma, that Unterberger's test achieved a higher sensitivity than the Romberg test.[75]

In a recent prospective, controlled study Kuipers and Oosterhuis concluded that this test does not appear to be useful for detection of abnormalities in the vestibular system or for distinguishing normal individuals from patients.[76] A few years earlier Hickey *et al* came to the same conclusion.[77]

Babinski–Weill test

The patient is asked, with his eyes closed, to walk five steps forwards, followed by five steps backwards, then forwards again and so on for 30 seconds. The test is positive when the walk is star-shaped.

Bárány's test

The patient sits. A point is fixed at a minimum distance of 2 m. The patient then closes his eyes and is asked to indicate the point 20 times consecutively. If a lesion is present, the arm deviates towards the affected side.

Examination of nystagmus

The examiner observes and analyses the involuntary eye movements. The following should be borne in mind:

- Nystagmus has a slow (pathological) phase and a quick (recovery) phase, and its direction is described by the latter. However, the side of the lesion is better indicated by the former (e.g. a quick recovery phase to the right implies a problem to the left).
- The patient cannot influence the examination, nor can compensation be achieved.

Nystagmus may occur spontaneously (Fig. 8) and a combination of opening and closing the eyes may point towards the type of nystagmus present. The examiner next undertakes tests to recognize positioning or provoking nystagmus (see types of nystagmus above). In orthopaedic medicine two tests are important: the De Kleyn–Nieuwenhuyse and the Dix–Hallpike tests.

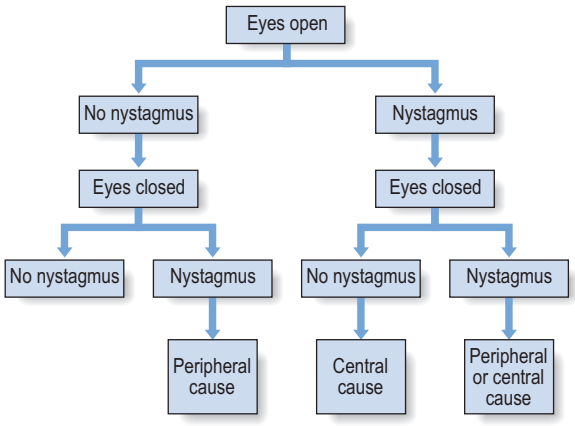

Fig 8 • Spontaneous nystagmus.

De Kleyn–Nieuwenhuyse test

The patient is in a supine lying position with his head maximally rotated in more-or-less maximum extension (Fig. 9). This provokes diminution or abolition of the blood flow in the contralateral vertebral artery, thereby testing the compensating mechanism in the ipsilateral vertebral artery and in the basilar artery and its branches. Disturbance of blood flow in the ipsilateral artery may result in the appearance of nystagmus or vertigo.

Dix–Hallpike test

The patient sits on the couch with his head turned away. The examiner then brings the patient quickly backwards under guidance. In pathological circumstances nystagmus results, but if it disappears within 30 seconds it is a benign paroxysmal positioning vertigo. Positional nystagmus together with the precipitating mechanism and the Dix–Hallpike test seem to be the best predictors of benign paroxysmal positioning vertigo.[78]

Provocation tests for dizziness

The patient, either sitting or lying, is subjected to different neck movements: they are performed rapidly and repeatedly at first and then sustained in the end range for at least 10 seconds. The neck movements are purely physiological but may be combined:

- Flexion
- Extension
- Side flexion
- Rotation
- Flexion, combined with rotation
- Extension, combined with rotation
- If manipulation is considered, the patient's head is also put in the different manipulation positions
- Movements of the trunk while the patient's head is held motionless.

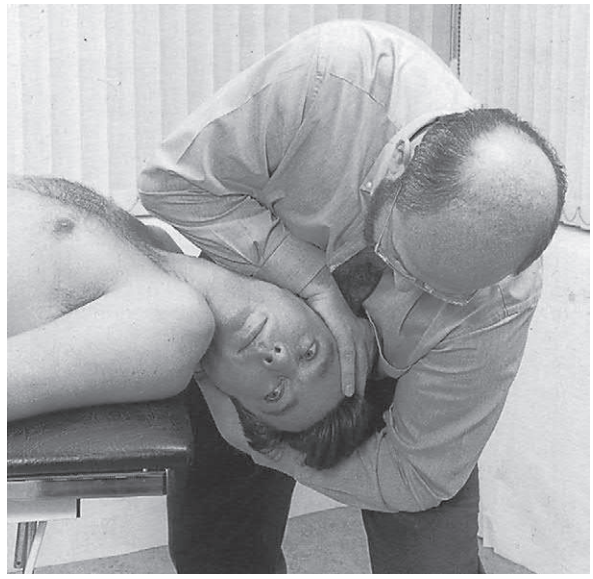

Fig 9 • The de Kleyn–Nieuwenhuyse test.

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Technical investigations

The most valuable otological examination methods are electronystagmography and audiometry. Electronystagmography seems to be useful when the cause is thought to be central or is uncertain. It is not significantly helpful in the diagnosis of peripheral lesions except as confirmation.[79]

Of the clinical neurophysiological methods, brainstem auditory evoked potentials are more useful than electroencephalography. Computed tomography and magnetic resonance imaging should be used when a central nervous system disorder is suspected.[80]

Interpretation of the examination

Careful interpretation of the data from the history and of the findings from the clinical examination (see Fig. 10) must enable the examiner to get an idea of the area where the problem lies: in the vestibular system, the vertebrobasilar system or the cervical spinal joints.

Disturbance in the vestibular system

Disturbance of the vestibular system is movement induced. The rapid execution of different neck movements may cause dizziness and probably also nausea ('motion sickness'). The examiner must keep in mind that vestibular vertigo may also be the result of acute ischaemia of the vestibular tissues, because of their blood supply via the basilar artery.

Disturbance of the vertebrobasilar system

The most important symptoms that are experienced by patients suffering from vertebrobasilar insufficiency are described by Coman[64] as the 5 Ds: dizziness, dysarthria, dysphagia, diplopia, drop attacks. Other symptoms, such as weakness, numbness, gait disturbance and visual abnormalities are also possible.[81]

The sustained neck positions provoke dizziness and/or nystagmus.

Disturbance of proprioception of the cervical spine

Elements that point towards a vestibular disorder (i.e. turning sensations, motion sickness) are absent. The patient does not mention one of the 5 D symptoms. There is no nystagmus. The neck movements are positive in that they indicate a cervical disorder: pain, stiffness, possibly diminished range of motion, articular pattern. The movements may or may not provoke dizziness. There may be balance disturbance.[82]

Benign positional paroxysmal vertigo is common in the elderly and even more common in patients with migraine.[83]

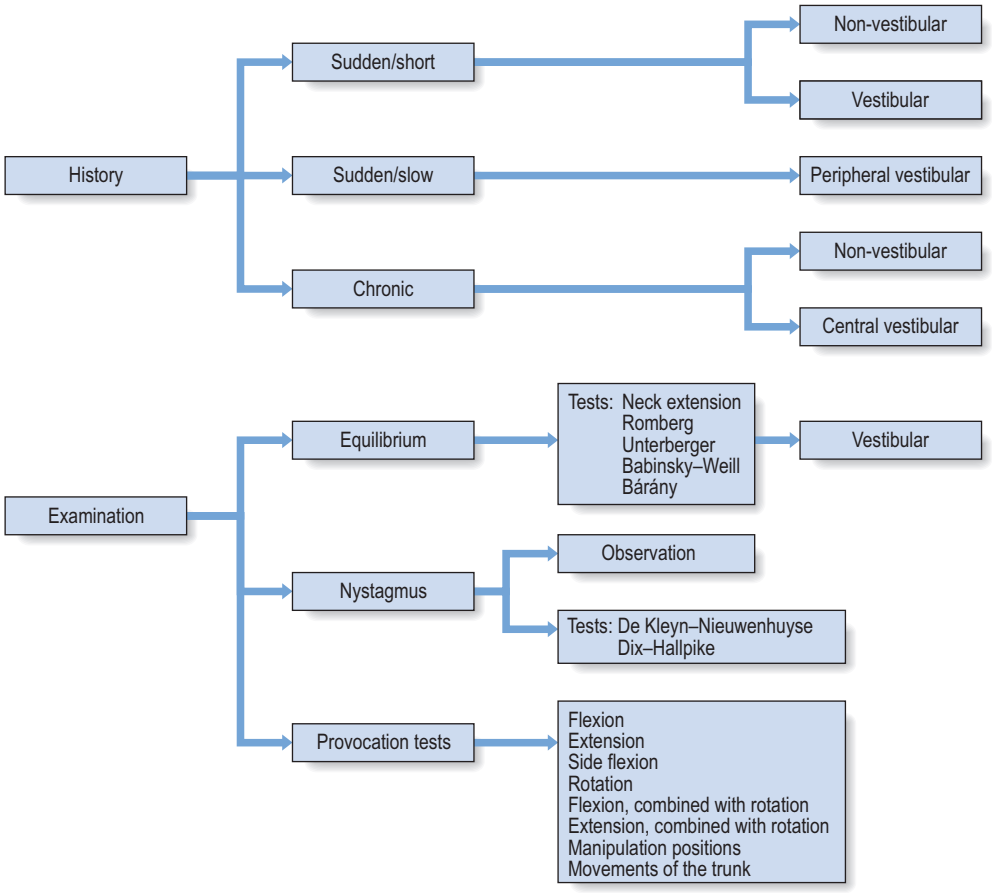

Fig 10 • History and examination of vertigo.

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Multivariate analysis demonstrated that the presence of a turning sensation and the absence of a lightheaded sensation predicted its presence.[84]

Some authors describe upper cervical joint dysfunction as a possible cause of vertigo.[85] Others doubt the existence of cervical vertigo as an independent entity and strongly emphasize the need to exclude neurological, vestibular and psychosomatic disorders first.[86,87] Posturographic assessment of human posture dynamics could be a possible future tool for use in diagnosing cervical vertigo.[88]

Oostendorp *et al* describe a turning/stop turning test (without head and neck movements) that could act as a differentiating element between vestibulogenous dizziness and cervicogenic dizziness.

Turning/stop turning test

The patient sits on a revolving stool with closed eyes. The examiner turns the patient ten times with constant speed in one direction. He then stops the movement and waits until an eventual dizziness has disappeared. The same is then performed in the other direction. The test is positive when the patient recognizes the symptoms, i.e. turning sensations.

When this test is positive the problem is rather vestibulogenous. If the cervical provocation tests are also positive there is clearly a cervicogenous component as well. When the test is negative and the provocation tests positive, the dizziness is of cervical origin.

Therapy

A patient with vestibular vertigo should be referred for specialist examination and treatment. Acute vertigo as the result of dysfunction of the labyrinth or of a serious central nervous system process demands prompt intervention to avoid long-term disability. Chronic vertigo may need surgery or rehabilitative measures, for example physiotherapeutic adaptation exercises, for lasting relief.[89]

Non-vestibular proprioceptive vertiginous complaints can have a discal origin. In these cases, manipulation with maximum traction and no articular movement (straight pull, traction with leverage) usually gives good results, especially in the elderly. This should of course only be performed by an experienced operator. For the technique, see Chapter 11). If there is any doubt, other conditions must first be excluded by specialized examination. The same techniques have the surprising result of also curing tinnitus. Kessinger mentions good results on cervical manipulation for vertigo, tinnitus and diminished hearing.[90]

Physical therapy also seems to be an effective treatment for benign positional paroxysmal vertigo.[91,92]

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Applied anatomy of the shoulder

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Introduction

The main function of the joints of the shoulder girdle (Fig. 1) is to move the arm and hand into almost any position in relation to the body. As a consequence the shoulder joint is highly mobile, where stability takes second place to mobility, as is evident from the shape of the osseous structures: a large humeral head lying on an almost flat scapular surface. Stability is provided mainly by ligaments, tendons and muscles; the bones and capsule are of secondary importance.

The function of the shoulder girdle requires an optimal and integrated motion of several joints. In fact five 'joints' of importance to 'shoulder' function can be distinguished:[1]

- The glenohumeral joint (1)
- The acromioclavicular joint (2)
- The sternoclavicular joint (3)
- The subacromial joint or subacromial gliding mechanism (4): the space between the coracoacromial roof and the humeral head, including both tubercles. This is the location of the deep portion of the subdeltoid bursa
- The scapulothoracic gliding mechanism (5): this functional joint is formed by the anterior aspect of the scapula gliding on the posterior thoracic wall.

Optimal mobility also requires an intact neurological and muscular system.

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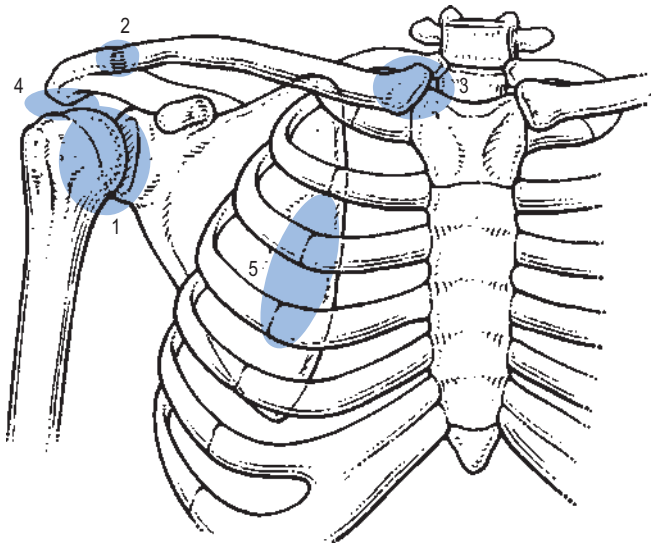

Fig 1 • A global view of all five joints of the shoulder girdle: 1, glenohumeral joint; 2, acromioclavicular joint; 3, sternoclavicular joint; 4, subacromial joint or subacromial gliding mechanism; 5, scapulothoracic gliding mechanism.

It is not our intention to give a complete anatomical review, which can be obtained from anatomy texts. Only those structures which are of specific clinical importance will be focused on.

Bones

Osseous structures of interest are the scapula, humerus and clavicle. Neither the vertebral column nor the thoracic cage is discussed here (see chapters on the anatomy of the cervical and thoracic spine).

Scapula

The scapula is a thin sheet of bone that functions mainly as a site of muscle attachment (Figs 2–3, see Putz, Figs. 286, 287, 288). Its medial border is parallel to the spine, the lateral and superior borders are oblique. It has a superior, a lateral and an inferior angle. The inferior angle corresponds to the interspinal level between the spinous processes of T7 and T8.

The scapula contains four processes: the acromion, the coracoid, the spine and the articular process (the glenoid).

The dorsum of the scapula is convex. It is divided by its spine into two fossae: the supraspinal and infraspinal fossa, containing the corresponding muscles. The scapular spine runs from the junction of the upper and middle third of the medial border, where it is rather flat, and corresponds to the level of the third thoracic spinous process. Laterally it becomes more prominent and meets the acromion at a right angle posteriorly. This angle is easily palpable and is one of the main bony landmarks at the shoulder. The acromion turns further anteriorly and covers part of the humeral head.

The coracoid process is found at the anterior aspect of the scapula. The tip points outwards and is easily palpated in the lateral part of the subclavian fossa. Further down, on the anterior aspect of the scapula, is a large concavity which contains the subscapularis muscle.

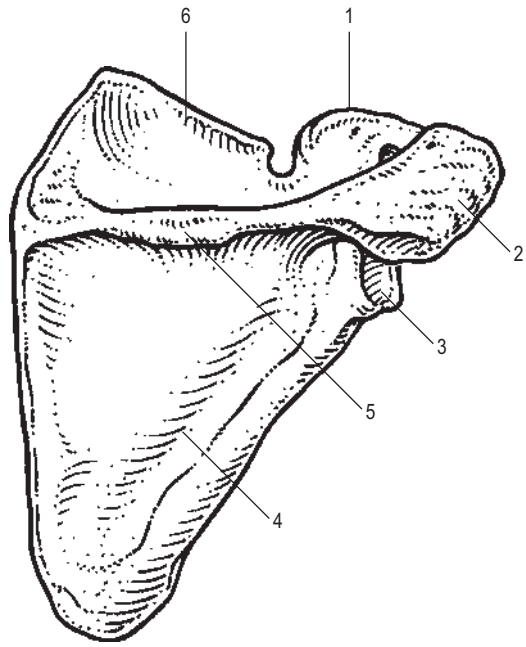

Fig 2 • Posterior view of the scapula: 1, coracoid process; 2, acromion; 3, glenoid; 4, infraspinal fossa; 5, scapular spine; 6, supraspinal fossa.

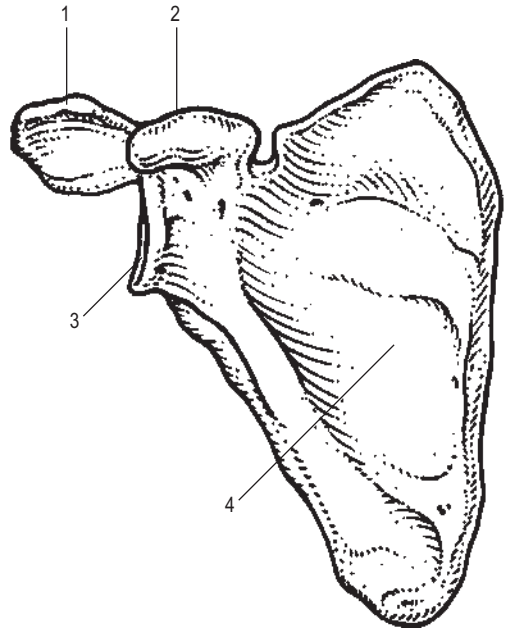

Fig 3 • Anterior view of the scapula: 1, acromion; 2, coracoid process; 3, glenoid fossa; 4, anterior surface.

At the lateral angle, just beyond the neck of the scapula, is the glenoid fossa. This has a rather shallow surface, which is directed anterolaterally and slightly cranially tilted. It is approximately one-quarter the size of the humeral head and this, plus its shallow concavity, makes the joint both very mobile and vulnerable to (sub)luxations.

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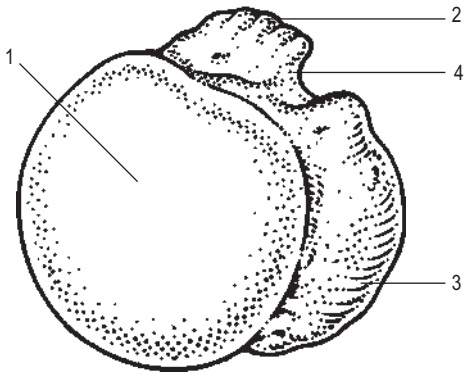

Fig 4 • Superior view of humerus: 1, humeral head; 2, minor tuberosity; 3, major tuberosity; 4, bicipital sulcus.

The ventral surface of the scapula is flat and covered with the attachment of the subscapularis muscle, except for the medial border and inferior angle where the serratus anterior muscle is inserted.

Humerus

The articular surface of the humeral head points in a medial, posterior and slightly caudal direction and is separated from the major and minor tuberosities by its anatomical neck.

When the arm is hanging down the side with its anterior aspect facing the body, the greater tuberosity lies laterally and the lesser tuberosity anteriorly. They are separated from each other by the bicipital sulcus (Fig. 4).

Clavicle

The clavicle joins the sternum to the acromion. At its medial end it has a forward convexity whereas its lateral end is rather more concave (see Putz, Fig. 284).

The joint capsules of both the sternoclavicular and the acromioclavicular joints are reinforced by several ligaments.

The clavicle has many muscular and ligamentous attachments. The insertion of the coracoclavicular ligament is of practical importance. It is found laterally on the inferior aspect of the clavicle, and just medial to it is the origin of the subclavius muscle.

The clavicle gives support to the shoulder girdle by acting as a strut between scapula and sternum. Due to its S-shape, the outer end describes a much larger rotation during arm elevation than its inner end. Therefore, lesions of the acromioclavicular joint ligaments are more frequent than are lesions of the sternoclavicular joint ligaments.

Joints and intracapsular ligaments

Glenohumeral joint

The glenohumeral joint (Fig. 5) is a ball-and-socket between humeral head and glenoid fossa. There is a remarkable

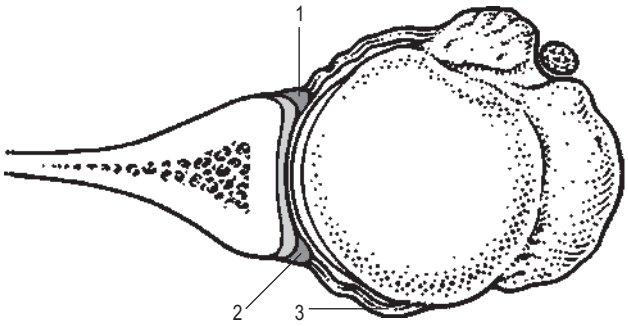

Fig 5 • Shoulder (glenohumeral) joint: 1, labrum; 2, glenoid cartilage; 3, shoulder capsule.

geometrical relationship between glenoid and head which is responsible for the considerable mobility of the joint but is also an important predisposing factor to glenohumeral instability. First, the large spherical head of the humerus articulates against the small shallow glenoid fossa of the scapula (only 25–30% of the humeral head is covered by the glenoid surface). Second, the bony surfaces of the joint are largely incongruent (flat glenoid and round humerus). However, the congruence is greatly restored by the difference in cartilage thickness: glenoid cartilage is found to be the thickest at the periphery and thinnest centrally, whereas humeral articular cartilage is thickest centrally and thinnest peripherally. This leads to a uniform contact between humeral head and glenoid surface throughout shoulder motion.

The labrum is a fibrous structure that forms a ring around the periphery of the glenoid (see Putz, Fig. 298). It acts as an anchor point for the capsuloligamentous structures and for the long head of the biceps. It further contributes to stability of the joint by increasing the depth of the glenoid socket, enlarging the surface area and acting as a load-bearing structure for the humeral head.

The synovial membrane of the joint capsule is mainly attached to the labrum, covering its inner surface, and at the anatomical neck of the humerus. It forms a sleeve around the intra-articular and inter-tubercular part of the long tendon of the biceps.

The fibrous portion of the capsule is very lax and has several recesses, depending on the position of the arm. At its caudal end it forms the axillary recess, which allows normal elevation of the arm. Very often adhesions form here.

The joint capsule is large, loose and redundant: the capacity of the glenohumeral joint capsule is larger than that of the humeral head to allow full and free range of motion of the shoulder.

At the anterior portion of the capsule three local reinforcements are present: the superior, medial and inferior glenohumeral ligaments (Fig. 6). These contribute, together with the subscapularis, supraspinatus, infraspinatus and teres minor muscles, to the stability of the joint.

The supraspinatus, infraspinatus, teres minor and subscapularis tendons reinforce the superior, posterior and anterior capsule. By virtue of the blending of their tendons with the glenohumeral capsule and ligaments, selective contraction of the cuff muscles can adjust the tension in these structures, producing 'dynamic' ligaments.

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Subacromial space

The suprahumeral gliding mechanism consists of the coracoacromial arch (see below) on one side and the proximal part of the humerus, covered by the rotator cuff and the biceps tendon on the other. Both parts are separated by the subacromial bursa that acts as a joint space (Fig. 7). Investigators point to the importance of contact and load transfer between the rotator cuff and the coracoacromial arch in the function of the normal shoulder. Normally there is no communication between the bursa and the glenohumeral joint space but it may be established by rupture of the rotator cuff.

Acromioclavicular joint

The acromioclavicular joint (Fig. 8) is the only articulation between the clavicle and the scapula. It contains a disc which usually has a large perforation in its centre. The capsule is thicker on its superior, anterior and posterior surfaces than on the inferior surface. The anteroposterior stability of the acromioclavicular joint is controlled by the acromioclavicular ligaments and the vertical stability is controlled by coracoclavicular ligaments (conoid and trapezoid).

Sternoclavicular joint

The only osseous connection between the skeleton of the trunk and the upper limb is formed by the clavicle. Its medial end lies in contact with the superolateral angle of the sternal manubrium and with the medial part of the cartilage of the first rib to form the sternoclavicular joint (Fig. 9, see Putz, Fig. 285). In both the vertical and anteroposterior dimensions, the clavicular portion is larger than the opposing manubrium and extends superiorly and posteriorly relative to the sternum. The prominence of the clavicle enables its palpation. The sternoclavicular joint is mobile along all axes and almost every

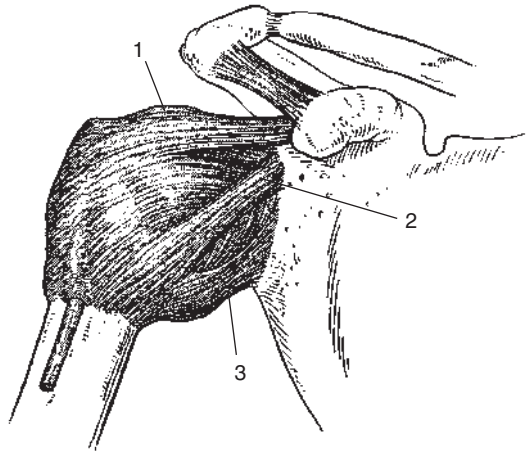

Fig 6 • The glenohumeral joint capsule and ligaments: superior (1), medial (2) and inferior (3) glenohumeral ligaments.

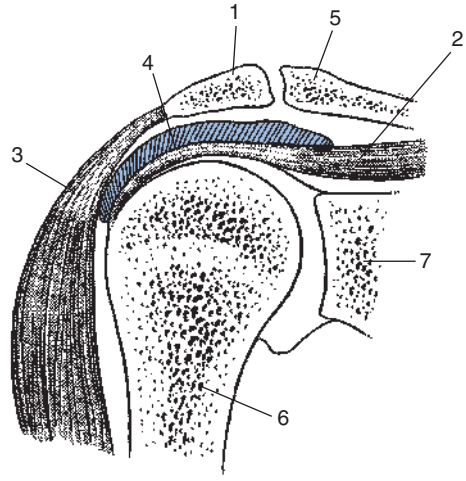

Fig 7 • The subacromial space and subdeltoid bursa: 1, acromion; 2, supraspinatus muscle; 3, deltoid muscle; 4, subdeltoid bursa; 5, clavicle; 6, humerus; 7, scapula.

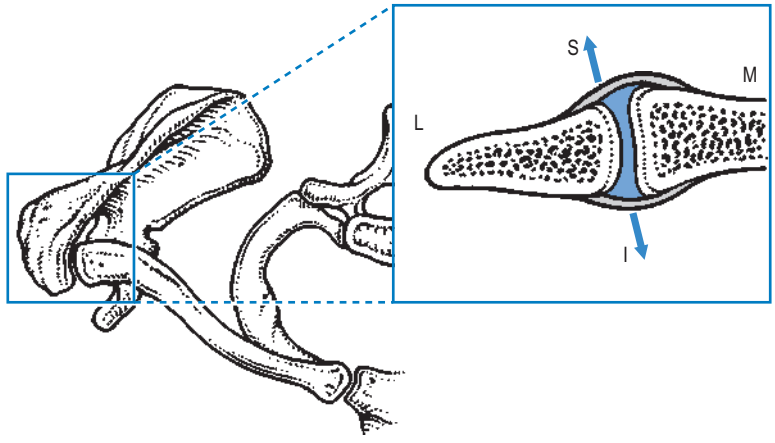

Fig 8 • Acromioclavicular joint: S, superior; I, inferior; L, lateral; M, medial.

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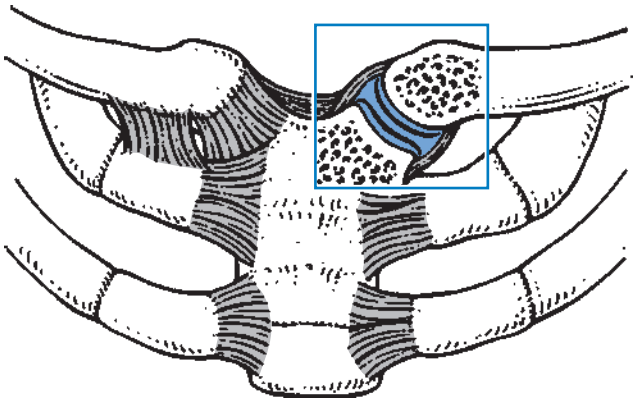

Fig 9 • Sternoclavicular joint.

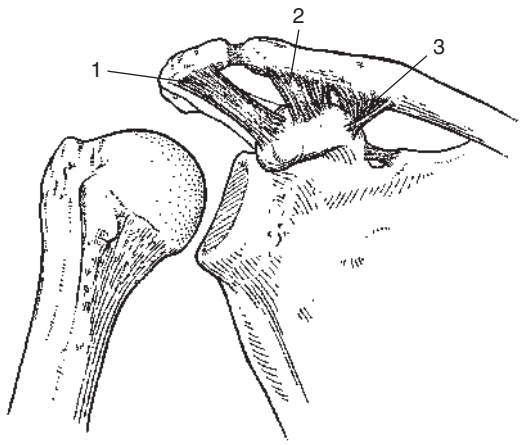

Fig 10 • The extracapsular ligaments: 1, coracoacromial ligament; 2, trapezoid ligament; 3, conoid ligament.

movement of the scapula and the arm is associated with some movement at this joint.

The capsule of the joint is thin at its inferior aspect but is reinforced anteriorly by the anterior sternoclavicular ligament, posteriorly by the posterior sternoclavicular ligament and cranially by the interclavicular ligament. The latter courses further above the sternum, to which it is firmly attached. Just lateral to the inferior part of the capsule is the costoclavicular ligament. This ligament connects the medial end of the clavicle to the cartilage of the first rib.

An intra-articular meniscus, firmly attached at its circumference to the joint capsule, to the clavicle and to the cartilage of the first rib, divides the joint into two separate cavities.

Behind the left joint is the common carotid artery, behind the right joint the innominate (brachiocephalic) vein. Both structures must be kept in mind when infiltrating on either side.

Scapulothoracic gliding mechanism

The serratus anterior muscle and the subscapularis are interposed between the scapula and the posterior thorax. The scapula is normally able to rotate, glide and tilt on the thorax. This mobility is an absolute necessity for normal function of the shoulder joint. Disorders of one of those structures, as in irregularity of the posterior thoracic wall, can result in pain on movements of the arm or scapula.

Extracapsular ligaments

(Fig. 10, see Putz, Fig. 285)

Coracoacromial ligament

The coracoacromial ligament originates from the lateral aspect of the coracoid process and runs laterally and upwards to the tip of the acromion. It forms, together with the acromion and outer end of the clavicle, the coracoacromial roof.

The coracoacromial arch provides a strong ceiling for the shoulder joint, along which the cuff tendons must glide during all shoulder movements. Passage of the proximal humerus and its cuff tendons under this roof is facilitated by the gliding surfaces of the subacromial bursa. Because there is normally no gap between the cuff and the roof, the slightest amount of superior translation may compress the cuff tendons and/or the bursa between the humeral head and the arch (Fig. 10, see Putz, Fig. 295). Changes in the coracoacromial arch have been described in association with cuff disorders along with variations of acromial shape. Classically three types of acromion morphology are described: flat, curved and hooked. There seems to be a relationship between the presence of a hooked acromion and the prevalence of cuff lesions, although a hooked acromion and a cuff defect can both be merely the consequences of age.

Coracoclavicular ligaments

The *trapezoid ligament* originates superiorly on the coracoid process and inserts at the inferior–lateral aspect of the clavicle.

The triangular *conoid ligament* lies medial to the trapezoid ligament. Its origin is at the medial aspect of the superior surface of the coracoid process and it inserts at the conoid tubercle at the inferior clavicular aspect.

Both ligaments join together and form a half of a cone. They attach the scapula to the inferior outer side of the clavicle. Their structure enables the clavicle to rotate around its long axis on elevation of the arm. In this position the clavicular insertion of both ligaments points more or less anteriorly.

Costocoracoid fascia

Deep to the pectoralis major muscle lies a group of associated muscles and fascia: the clavipectoral fascia, the subclavius and pectoralis minor muscles (Fig. 11).

The costocoracoid fascia is that part of the clavipectoral fascia which is situated superomedial to the pectoralis minor muscle. A spontaneous loss of its normal elasticity may end in a contracture of this structure, causing limitation of elevation of the arm.

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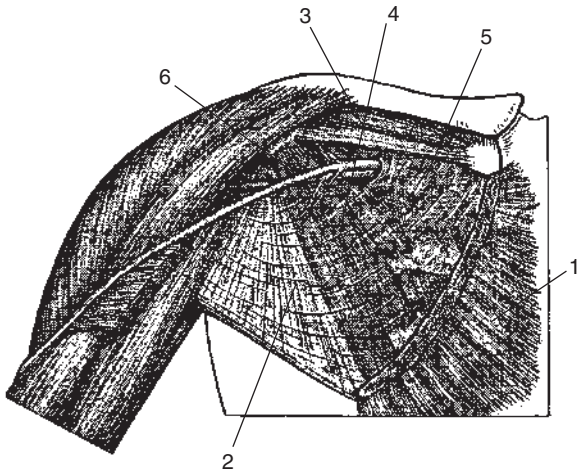

Fig 11 • The costocoracoid fascia: 1, pectoralis major muscle; 2, clavipectoral fascia; 3, subclavius muscle; 4, pectoralis minor muscle; 5, costocoracoid fascia; 6, cephalic vein.

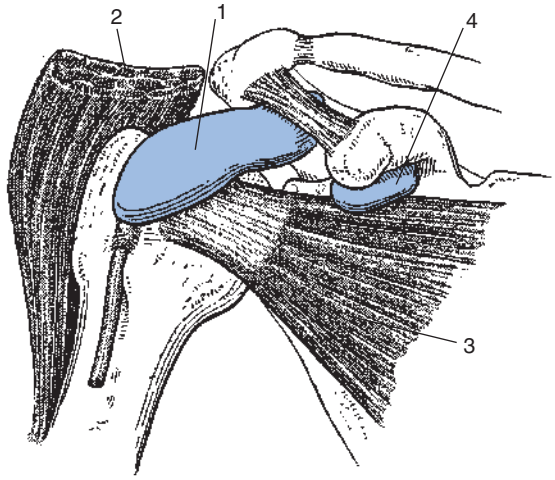

Fig 12 • Anterior view of the bursae of the shoulder: 1, subdeltoid bursa; 2, deltoid muscle; 3, subscapularis muscle; 4, subcoracoid bursa.

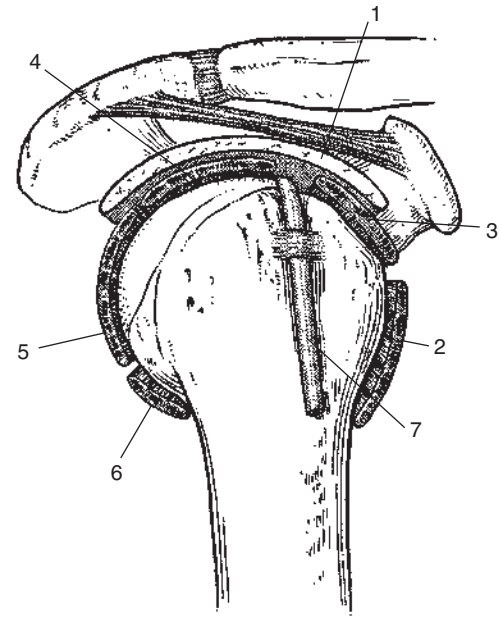

Fig 13 • Lateral view of the subdeltoid bursa of the shoulder: 1, subdeltoid bursa; 2, teres major muscle; 3, subscapularis muscle; 4, supraspinatus muscle; 5, infraspinatus muscle; 6, teres minor muscle; 7, long head of biceps.

Bursae

The bursae are shown in Figures 12–13, see Putz, Fig. 295.

Subacromial–subdeltoid bursa

It is important to realize that there is only one bursa here. However, for clinical reasons two portions may be distinguished: a deep, subacromial part and a more superficial one. The former cannot be palpated, the latter is within a finger's reach.

A good idea of the anatomical localization of this bursa is obtained if the palm of the contralateral hand is put on top of the shoulder. The metacarpophalangeal joints must lie contiguous with the lateral acromial rim. The area covered by the palm overlies the deep subacromial portion of the bursa, the area covered by the fingers, and delineates the superficial (subdeltoid) part of it.

The subacromial–subdeltoid bursa is normally only a potential 'space': two serosal surfaces in contact with each other, one on the surface of the cuff, the other on the undersurface of the coracoacromial arch (subacromial part) and the undersurface of the deltoid muscle (subdeltoid part). It normally produces a small amount of fluid which acts as a lubricant.

The bursa permits the two layers to glide in relation to each other and so can, to a certain extent, be considered as the synovial portion of the 'subacromial joint', an extension of the glenohumeral joint. Inflamed, the bursa becomes compromised by oedema and adhesions and may cause considerable pain and functional disturbance.

Subcoracoid bursa

Another clinically important bursa is the subscapularis bursa, which develops between the upper portion of the subscapularis tendon, the neck of the scapula and the base of the coracoid process (see Standring, Fig. 46.14A). The bursa is covered by the pectoralis major muscle. Bursitis here can give rise to an isolated limitation of passive lateral rotation. Since this movement stretches the muscle, it will painfully pinch the inflamed subcoracoid bursa, provoking a spasm of the pectoralis major.

Shoulder movements

The glenohumeral joint is the most mobile joint of the human body. It can move around three different axes, all transversing the head of the humerus (Fig. 14):

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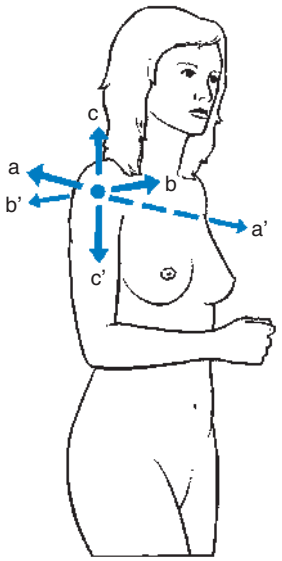

Fig 14 • The axes of the shoulder.

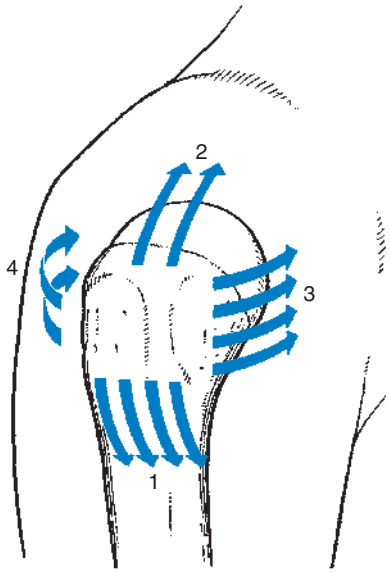

Fig 15 • Muscle functions: 1, adductors; 2, abductors; 3, medial rotators; 4, lateral rotators.

- *a–a′ – Transverse axis:* this is a horizontal axis coursing from the left to the right shoulder. Anterior and posterior directed movements around it are flexion and extension.
- *b–b′ – Sagittal axis:* this horizontal axis lies in antero-posterior sense through the head of the humerus. Movement of the arm around this axis is named adduction (the arm moves towards the body) and abduction (away from the body).
- *c–c′ – Vertical axis*: with the right arm hanging in neutral position (radial side pointing anteriorly and elbow bent to 90°), outward movement of the arm around a vertical axis, bringing the palm of the hand anteriorly, is lateral rotation. The movement in the opposite direction is medial rotation. If the arm is first abducted to 90°, movement around the vertical axis, which brings the arm anteriorly and posteriorly, is named horizontal adduction and horizontal extension.

When the patient actively elevates the arm, the initial movement takes place at the glenohumeral joint. Later the scapula rotates synchronously. Clinically this is easily checked by palpating the inferior scapular angle during elevation. Wallace[2] states that both movements take place in a ratio of about 2 : 1; that means for every 2° of movement at the glenohumeral joint there exists 1° of scapular rotation. These simultaneous movements are known as the scapulothoracic or scapulohumeral rhythm.

Mobility of the glenohumeral joint is usually tested in three directions: abduction, medial rotation and lateral rotation.

Abduction is movement in a frontal plane between a mobile humerus and a stabilized scapula. Medial and lateral rotation is assessed with the arm hanging in the neutral position. The term *elevation* indicates the movement of the shoulder complex (glenohumeral joints and shoulder girdle) in a frontal plane. Normally the arm can move through an angle of approximately 0° to 180° in elevation; the normal range for the glenohumeral joint is about 90° of abduction and the same for medial and lateral rotation.

The most important function of the shoulder – arm elevation – has been extensively studied, in particular the relationship and contribution of the glenohumeral and scapulothoracic joints and the 'scapulothoracic' rhythm has been determined. During full elevation of 180° only 90° takes place at the glenohumeral joint. The remaining half is the consequence of scapular rotation, clavicular movement and, finally, adduction of the humerus which is only possible when the scapula has been fully rotated.

Muscles and tendons

The clinically important contractile structures of the shoulder joint are those whose tendons form the rotator cuff: the supraspinatus, infraspinatus, subscapularis and, of less importance, the teres minor.

The adductors are those muscles which act below the sagittal axis, and the abductors are those acting above (Fig. 15). Medial rotators act in front of the vertical axis, lateral ones behind it.

The origins and insertions of the muscles are shown in Figure 16.

Adduction

Adduction of the arm is performed by four different muscles: teres minor and major, the pectoralis major and latissimus dorsi (see Fig. 18). The first is also a lateral rotator, while the latter three are medial rotators.

The teres minor arises at the lower outer rim of the scapula, just above the origin of the teres major muscles and inserts on the major tubercle, just distal to the insertion of the infraspinatus muscle.

The teres major runs from the lower scapular angle towards the anterior aspect of the humerus, where it inserts on the

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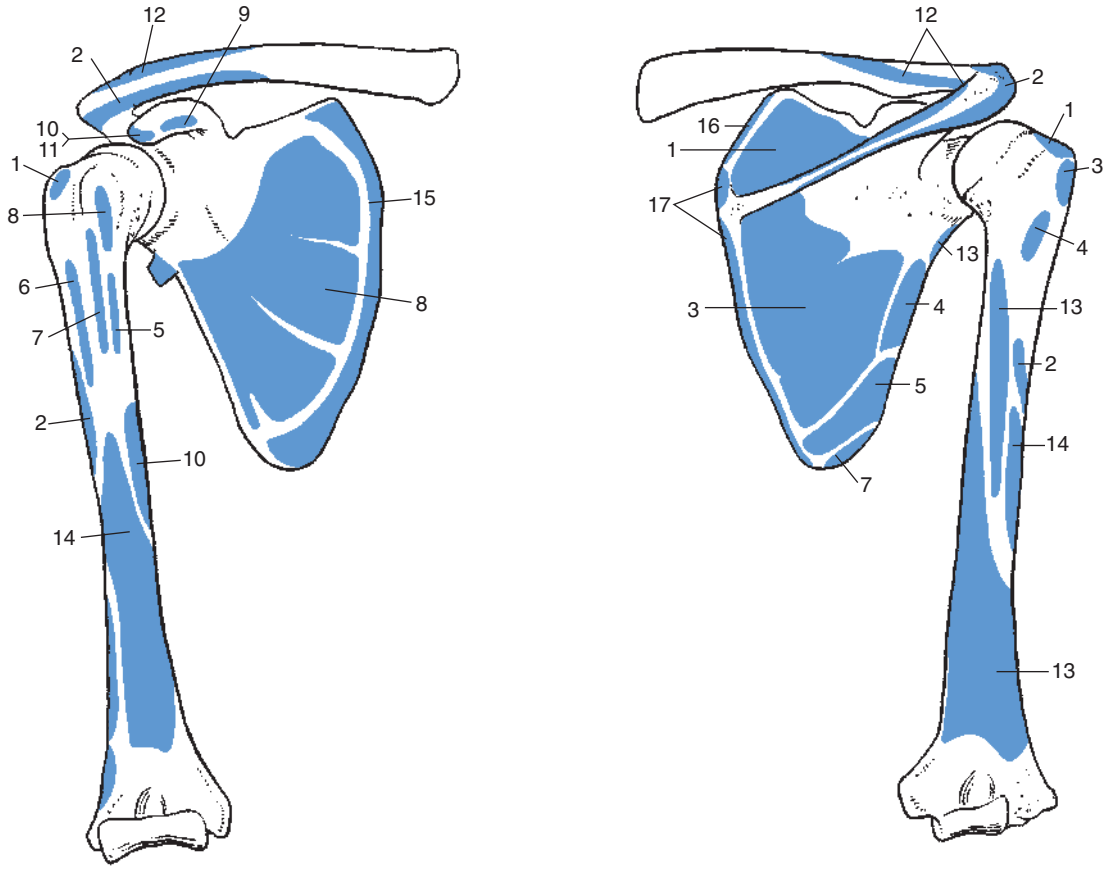

Fig 16 • Muscle origins and insertions: 1, supraspinatus; 2, deltoid; 3, infraspinatus; 4, teres minor; 5, teres major; 6, pectoralis major; 7, latissimus dorsi; 8, subscapularis; 9, pectoralis minor; 10, coracobrachialis; 11, short head of biceps; 12, trapezius; 13, triceps brachii; 14, brachialis; 15, serratus anterior; 16, levator scapulae; 17, rhomboid.

minor tubercular crest, together with the latissimus dorsi muscle.

The triangular space at the posterior aspect of the shoulder between both teres muscles is divided by the long head of the triceps muscle into a quadrilateral space laterally and a triangular space medially (see Putz, Fig. 338).

The latissimus dorsi muscle originates partly from the spinous processes of the lower dorsal vertebrae, partly from the lumbar vertebrae, partly from the iliac crest and sometimes from the lower ribs. It runs outwards and upwards in the direction of the lesser tubercular crest, where it inserts just lateral to the insertion of the teres major muscle.

The pectoralis major muscle originates from the medial end of the clavicle, the sternum, the cartilage of the second to the sixth rib and the uppermost part of the rectus abdominis muscle. The upper fibres have a slightly downward course; the lower fibres run in a craniolateral direction. It inserts on the crest of the greater tuberosity.

Table 1 summarizes the adductors.

Table 1 The adductors

Muscle	Nerve	Spinal nerve root
Pectoralis major	Pectoral	C5–C8
Latissimus dorsi	Thoracodorsal	C7 (C6, C8)
Teres major	Subscapular	C5–C8
Teres minor	Axillary	C5 (C6)

Abduction

The supraspinatus muscle together with the deltoid is mainly responsible for this movement. Clinical differentiation of the two structures is made by testing resisted movement of horizontal adduction and horizontal extension, which are secondary functions of the deltoid muscle.

The supraspinatus, which originates in the supraspinal fossa, passes laterally, underneath the coracoacromial roof and inserts at the greater tubercle of the humerus (Fig. 17). The tendon and the tenoperiosteal junction are about 1 cm in width. On palpation it feels like a rather flat cord with a typical tendinous consistency. The muscle plays a particularly important role in the initiation of abduction. When this tendon is completely ruptured the patient can no longer lift the arm actively and must make a swinging movement of the whole body in order to start the movement. Once the arm has moved through 30°

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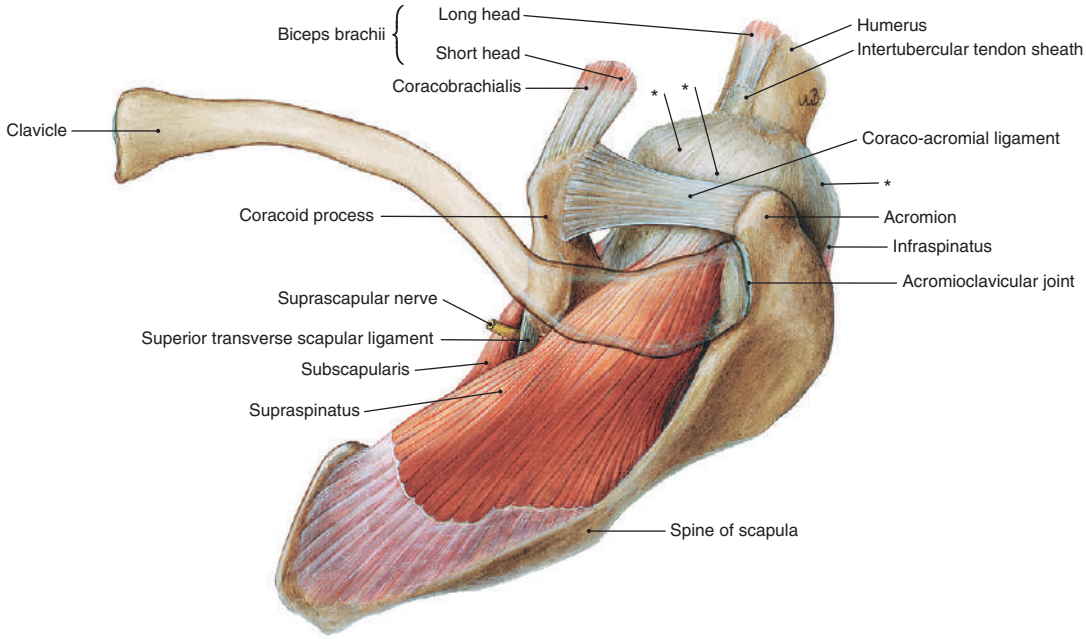

Fig 17 • Supraspinatus muscle from above and anterior. *Capsule of the joint. From Putz, Sobotta – Atlas of Human Anatomy, 14th edn. Urban & Fischer/Elsevier, Munich, 2008 with permission.

Table 2 The abductors

Muscle	Nerve	Spinal nerve root
Deltoid	Axillary	C5 (C6)
Supraspinatus	Suprascapular	C5 (C6)

the deltoid muscle takes over and carries out further abduction.

The deltoid has three different parts: the clavicular, the acromial and the scapular spinal. In addition to the muscle's overall effect in abduction, its different parts each have other functions of their own (see Putz, Fig. 345). The clavicular component helps horizontal adduction of the arm and the scapular spinal component assists horizontal extension movement. With the arm in neutral position, the former helps flexion, the latter extension.

Table 2 summarizes the abductors.

Medial rotation

Three medial rotators have been discussed already: teres major, latissimus dorsi and pectoralis major (Fig. 18, see Putz, Fig. 337). All of these are also adductors. The fourth medial rotator is the subscapularis (Figs 18, 19). Its origin lies in the subscapular fossa, and the insertion in the minor tuberosity and towards the proximal extent of the minor tubercular crest. The tenoperiosteal insertion has a width of about 3 cm, is very thin and cannot be distinguished on palpation. Location of the insertion depends entirely on bony landmarks, i.e. the minor humeral tubercle.

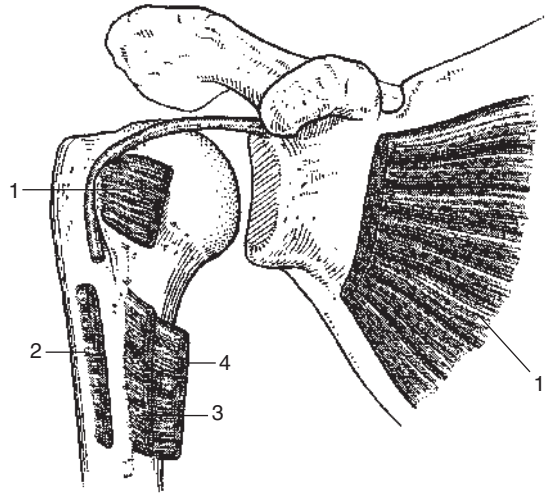

Fig 18 • Muscles used in medial rotation: 1, subscapularis; 2, pectoralis major; 3, latissimus dorsi; 4, teres major.

On abduction of the arm, the upper fibres pass underneath the acromial roof. Therefore, a momentary impingement of a lesion in this location can happen and produce a painful arc.

Horizontal adduction brings the lower half of the subscapularis insertion in contact with the coracoid process, and a lesion in this portion provokes pain on this movement.

Table 3 summarizes the medial rotators.

Lateral rotation

This movement is performed mainly by the infraspinatus, teres minor being only a weak lateral rotator. The infraspinatus originates in the infraspinal fossa and passes laterally underneath

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Table 3 The medial rotators

Muscle	Nerve	Spinal nerve root
Subscapularis	Subscapular	C5–C8
Pectoralis major	Pectoral	C5–C8
Latissimus dorsi	Thoracodorsal	C7 (C6, C8)
Teres major	Subscapular	C5–C8

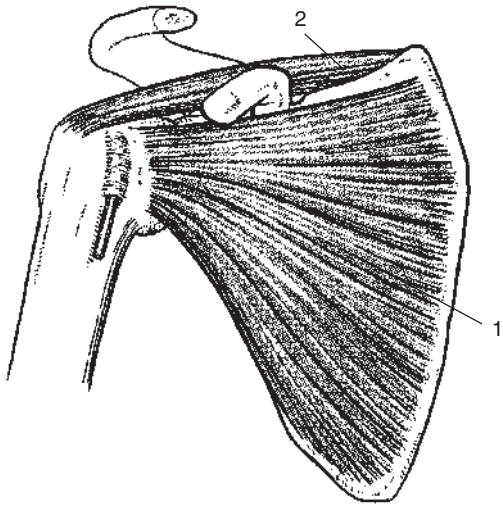

Fig 19 • Subscapularis, 1; and supraspinatus muscle, 2.

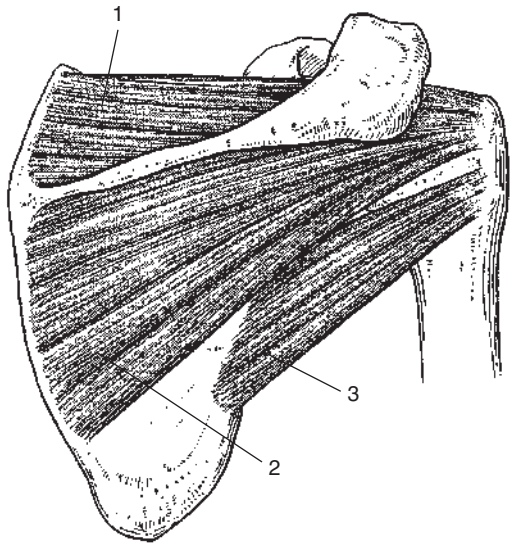

Fig 20 • Supraspinatus, 1 and muscles used in lateral rotation: 2, infraspinatus; 3 teres minor.

the acromion. It inserts on the greater tuberosity over about 2 cm. The upper fibres are in very close relationship with the insertion of the supraspinatus (Fig. 20, see Gosling, Fig. 3.64). On palpation the insertion is flatter and has a harder consistency than the insertion of the supraspinatus tendon.

The lateral rotators are summarized in Table 4.

Table 4 The lateral rotators

Muscle	Nerve	Spinal nerve root
Infraspinatus	Suprascapular	C5 (C6)
Teres minor	Axillary	C5 (C6)

Table 5 The flexors of the elbow

Muscle	Nerve	Spinal nerve root
Biceps brachii	Musculocutaneous	C5, C6

Table 6 The extensors of the elbow

Muscle	Nerve	Spinal nerve root
Triceps brachii	Radial	C7 (C6, C8)

Flexion of the elbow

Although this movement is performed by other structures as well, it is the tendon of the long head of the biceps that is the most important here (Table 5, see Putz, Fig. 346). Arriving from the supraglenoid tubercle it is directed downward towards the bicipital groove, where it lies underneath a transverse ligament. Part of it is intra-articular, where it is surrounded by a synovial membrane. On contraction of the biceps the tendon itself does not move. It plays an important role in the stabilization of the humeral head in the glenoid fossa during powerful flexion of the elbow and supination of the forearm.

The tendon of the short head of the biceps, originating from the coracoid process, seldom causes any problem.

The other flexor muscles are described in the online chapter *Applied anatomy of the elbow*.

Extension of the elbow

Extension of the elbow is performed by the triceps brachii (Table 6, see Putz, Fig. 298). The long head originates from the lower margin of the glenoid. Triceps lesions at the shoulder are extremely rare. Because the humeral head is pulled up towards the coracoacromial roof on resisted extension of the elbow, it can provoke an impingement of a lesion sited between the head of the humerus and the coraco-acromial arc. Therefore, if resisted extension of the elbow provokes pain at the shoulder, it is usually considered as analogous to a painful arc.

Flexion

The pectoralis major, biceps brachii, subscapularis and deltoid muscles, which are flexors, have already been discussed. The only other structure that can provoke pain on resisted flexion of the arm is the coracobrachialis. Its origin is at the coracoid

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Table 7 The flexors of the shoulder

Muscle	Nerve	Spinal nerve root
Coracobrachialis	Musculocutaneous	C5, C6
Pectoralis major	Pectoral	C5–C8
Deltoid (anterior portion)	Axillary	C5 (C6)
Subscapularis	Subscapular	C5–C8
Biceps brachii	Musculocutaneous	C5, C6

process, together with the short head of the biceps, and it inserts at the medial aspect of the midportion of the upper arm, below the minor tubercular crest.

The flexors are summarized in Table 7.

The anatomy of other muscles which are mainly associated with movements of the scapula – such as the subclavius, levator scapulae and others – are discussed in the online chapter *Applied anatomy of the thorax and abdomen*.

Rotator cuff

Although the rotator cuff muscles have been discussed individually, some remarks about the cuff as a whole are in order.

The rotator cuff is a complex of four muscles that arise from the scapula and attach to the tuberosities of the humerus. Although the muscles are separate superficially, in their deeper region they merge with each other, with the capsule underneath and with the tendon of the long head of the biceps.

Their intimate relationship with each other and with the capsule, together with their unique localization, provides the rotator muscles with some particular functions:

- First: the rotator cuff muscles 'rotate' the humerus with respect to the scapula – a function that has been discussed earlier. However, the insertion of these tendons as a continuous cuff around the humeral head permits the cuff muscles to provide an infinite variety of movements to rotate the humerus.
- Second: by contracting together, the rotator cuff muscles and the biceps compress the humeral head into the glenoid fossa, locking it into position and providing a secure scapulohumeral link for upper extremity function – known as 'concavity compression' (Fig. 21).
- Third: they provide muscle balance. In the shoulder, no fixed axis exists and, in a specified position, activation of one muscle will create a set of different rotational movements. In order to produce movement in one direction, the other movements initiated by the contracting muscle must be neutralized by other muscles. The rotator cuff muscles are critical elements of this shoulder muscle balance equation: by contracting selectively, they resist displacing forces resulting from contraction of the principal shoulder movers (deltoid, pectoralis major, latissimus dorsi).

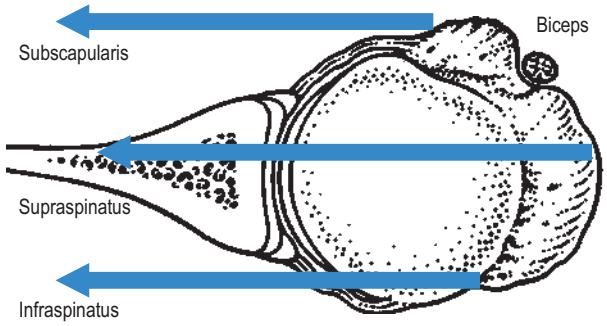

Fig 21 • Concavity compression.

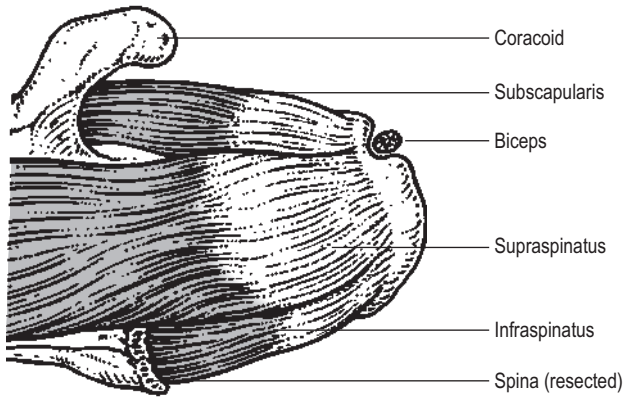

Fig 22 • The rotator cuff.

- Fourth: they have an important role in capsular stability of the joint. By virtue of the blending of their tendons with the glenohumeral capsule and ligaments, selective contraction of the cuff muscles can adjust the tension in these structures, so producing 'dynamic' ligaments (see Fig. 22).

Nerves and blood vessels

Suprascapular nerve

This nerve is derived from the brachial plexus. It contains motor fibres of the C5 (C6) root and passes in the scapular incisura, under the transverse scapular ligament (Fig. 23), whereas the suprascapular artery lies above the transverse ligament. It innervates the supraspinatus and infraspinatus muscles.

Axillary nerve

The axillary nerve (Figs 23, 24) contains both motor and sensory fibres from C5 (C6). Originating on the brachial plexus anteriorly, it passes dorsally via the lateral axillary foramen. Its close contact with the humeral neck often results in injury in the event of a dislocation.

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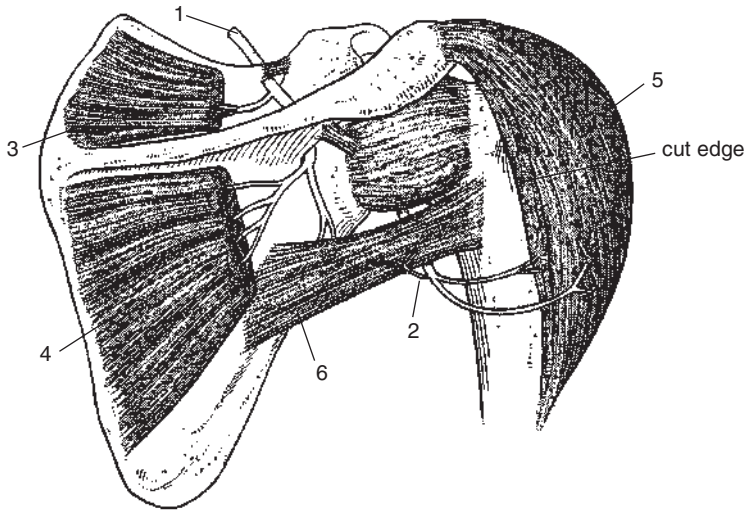

Fig 23 • The posterior scapulohumeral region: 1, suprascapular nerve; 2, axillary nerve; 3, supraspinatus muscle; 4, infraspinatus muscle; 5, deltoid muscle; 6, teres minor muscle.

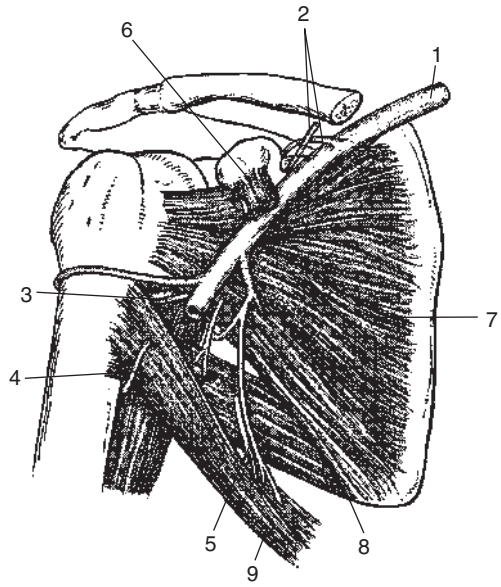

Fig 24 • The anterior scapulohumeral region; 1, subclavian artery; 2, suprascapular artery and nerve; 3, axillary nerve; 4, radial nerve; 5, subscapularis nerve; 6, pectoralis minor muscle; 7, subscapularis muscle; 8, teres major muscle; 9, latissimus dorsi muscle.

It gives a branch to the teres minor muscle and ends in the deltoid muscle. The terminal branch is the lateral superior brachial cutaneous nerve, which innervates the upper and outer aspect of the skin over the deltoid.

Subclavian artery and vein

The subclavian blood vessels are of particular importance since their compression can give rise to the subclavian steal syndrome or may play a role in the thoracic outlet syndrome. The vessels lie between the first rib and the clavicle, medially to the brachial plexus, and are separated by the scalenus anterior muscle.

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Excessive range of movement: instability of the shoulder

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Definition

Glenohumeral instability is defined as 'the inability to maintain the humeral head centred in the glenoid fossa'.[1] Recurrent instability may be characterized as dislocation, subluxation or apprehension. Dislocation is the complete separation of the articular surfaces without spontaneous relocation. Subluxation is defined as symptomatic translation of the humeral head without complete separation of the articular surfaces. Apprehension refers to the fear that the shoulder will subluxate. Instability can also be characterized according to the direction of the (sub)luxation: anterior, posterior, inferior and superior. Subluxations can also be classified according to their cause: they may be traumatic, atraumatic or voluntary. Around 95% of (sub)luxations are anterior. Most have a traumatic origin, although instability can also result from relatively small but repeated movements of the arm as may occur in swimmers, volleyball players or baseball pitchers.[2] Derangement of the labrum, intracapsular ligaments or joint capsule are regarded as the main underlying causes of chronic instability.[3] Exceptionally, osseous changes such as a fracture of the glenoid are found.

Factors in glenohumeral stability

A considerable range of mobility is essential at the glenohumeral joint. As a consequence, the shoulder has less bony and ligamentous stability than any other diarthrodial joint. There is a considerable incongruence between the two articulating surfaces with only 25–30% of the humeral head being covered by the glenoid fossa. This enables a wide range of positions without neck-rim contact.[4] Furthermore, the glenohumeral joint does not have strong isometric articular ligaments that provide stability while the joint is flexed around a defined anatomical axis as is the case in knee, elbow and ankle joints. Instead, the glenohumeral ligaments have a stabilizing function only at the extremes of motion and play no part in most functional positions of the joint.[5]

In spite of all this, the normal shoulder joint is quite stable and able to centre the humeral head in the glenoid cavity throughout most of the arc of movement.[6] This is achieved by a set of mechanisms which classically are divided into static factors (those that do not require the expenditure of energy by muscles) and dynamic factors (requiring muscle energy).

Static factors

Joint surfaces

Recent studies have demonstrated that, although the bony surfaces of the joint are largely incongruent (flat glenoid and round humerus), congruence is restored by the difference in thickness of cartilage. Glenoid cartilage was found to be the thickest at the periphery and thinnest centrally, whereas humeral articular cartilage was thickest centrally and thinnest peripherally (Fig. 1). This leads to a merely uniform contact between humeral head and glenoid surface throughout shoulder motion.[7]

The glenoid labrum contributes to stability by increasing the depth of the glenoid socket (Fig. 1) and so increasing the surface area for the humeral head.[8,9]

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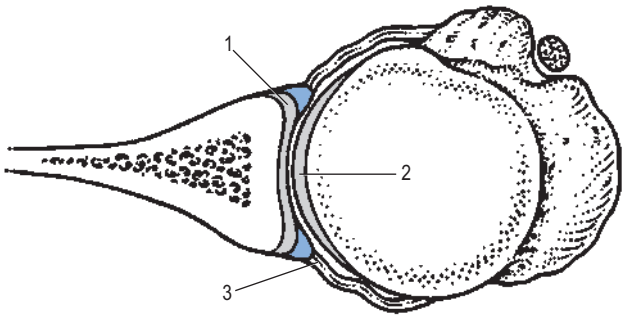

Fig 1 • Glenoid cartilage is thickest at the periphery (1); humeral cartilage is thickest centrally (2). The glenoid labrum increases the depth of the glenoid socket and the surface area for the humeral head (3).

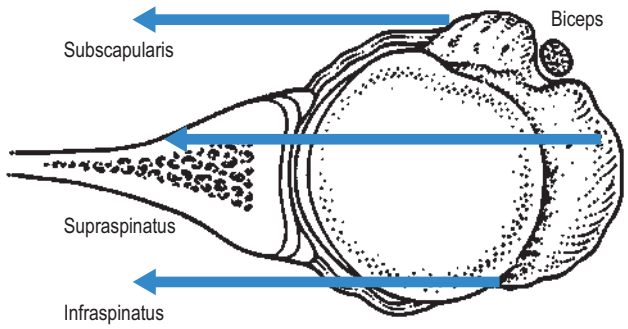

Fig 2 • By contracting together the rotator cuff muscles and the biceps press the humeral head into the glenoid fossa, locking it in position.

Cohesion–adhesion forces

Joint surfaces, wet with joint fluid, are held together by the molecular attraction of the fluid to itself and to the joint surfaces. This is called the adhesion–cohesion mechanism (compare with two wet microscope slides pressed together) and is a very strong stabilizing factor in the glenohumeral joint.

Negative pressure

There is minimal (less than 1 cc) free fluid in the normal shoulder joint. The normal shoulder is sealed by the capsule so that external fluid cannot enter it. Osmotic action by the synovium removes free fluid, keeping a slightly negative pressure within the normal joint. The negative intra-articular pressure creates a vacuum that pulls the humerus against the glenoid.[10] The limited joint volume effect is reduced if the joint is 'vented' (opened to the atmosphere) or when the capsular boundaries of the joint are very compliant.[11] This will lead to the production of a 'sulcus sign' (see below): under attempted traction on the arm, the flexible capsule is dragged into the joint.

Capsule and ligaments

The joint capsule is large, loose and redundant (the capacity of the glenohumeral joint capsule is larger than that of the humeral head). Capsule and ligaments alone cannot prevent glenohumeral translation when the joint is in most of its range of movement. Recent studies show that the main importance of the capsule for stability lies in the feedback mechanism of the proprioceptive innervation and contraction of the rotator cuff.[12,13]

Dynamic factors

Both shoulder girdle and rotator cuff muscles contribute to 'dynamic' glenohumeral stability. The muscles of the shoulder girdle (trapezius, rhomboideus, latissimus dorsi, serratus anterior and levator scapulae) stabilize the scapula as a firm platform for movement of the humeral head.[14] The 'rotator cuff' (supraspinatus, infraspinatus, subscapularis and the long head of the biceps) serves three stabilizing functions:

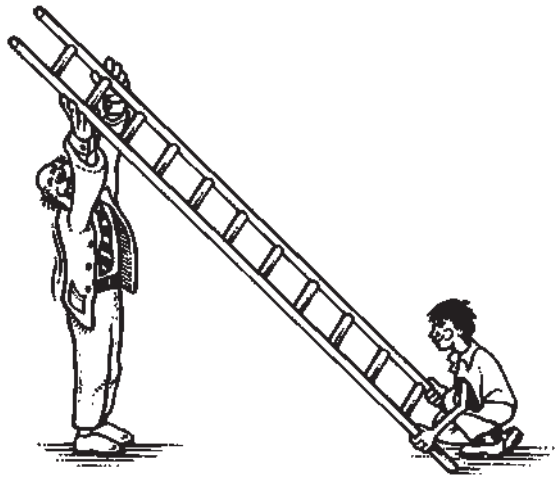

Fig 3 • By contracting selectively the rotator cuff muscles can resist the displacing forces of the principal shoulder motors.

- *Dynamic ligaments*. By virtue of the blending of their tendons with the glenohumeral capsule and ligaments, selective contraction of the cuff muscles can adjust the tension in these structures, producing dynamic ligaments.[15,16]
- *By contracting together*, the rotator cuff muscles and the biceps press the humeral head into the glenoid fossa (Fig. 2), locking it into position and thus providing a secure scapulohumeral link for upper extremity function.[17]
- *By contracting selectively*, the rotator cuff muscles can resist displacing forces resulting from contraction of the principal shoulder motors.[18] For example, when the pectoralis major and anterior deltoid muscles elevate and flex the shoulder, they tend to push the humeral head out the back of the glenoid fossa; this displacement is resisted by contraction of the subscapularis, infraspinatus and teres minor (Fig. 3). When the lateral deltoid initiates shoulder abduction, the supraspinatus and the long head of the biceps actively resist upward displacement of the humeral head relative to the glenoid fossa.

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History

The history and detailed physical examination are both very important for diagnosis. Unfortunately symptoms are often vague and clinical findings subtle.[19,20] The majority of patients mention a previous injury, usually a fall onto the outstretched arm in lateral rotation and abduction, which resulted in a luxation of the humeral head.[21] However, it should not be assumed that there is also a record of manipulative reduction at that time because in most cases the humeral head only subluxates and immediately reduces spontaneously. The earlier in life this happens, the more likely is future transient subluxation.[22] Sometimes, patients are encountered without a history of earlier displacement. This situation is called atraumatic instability and is more likely to be multidirectional. Atraumatic instability is more a syndrome that may arise from a set of pathogenic disorders: a flat glenoid, compliant capsule or weak neuromuscular control. It is often referred to as the AMBRII syndrome: **A**traumatic, **M**ultidirectional, **B**ilateral, with **R**ehabilitation directed at restoring neuromuscular control, as the most important therapeutic measure; in case surgery is necessary, it should include reconstruction of the rotator **I**nterval and tightening of the **I**nferior capsule.[23–25]

An attack of subluxation is usually described as follows: the patient experiences a sudden paralysing pain and weakness of the arm during activity. Whatever is held may be dropped. Very often the pain is brought on by unguarded movements such as raising the arm during throwing, swimming or serving at tennis. The pain is only momentary and disappears spontaneously after a few moments, after which the patient is usually able to return to activities without much pain or problem. In advanced cases, however, and after repeated attacks, the shoulder may be felt to slip out and clunk back into place with increasing ease and in an increasing number of activities.[26] Finally the shoulder may become uncomfortable even with the arm at rest and by then the acute symptoms are brought about with less forceful activities such as turning the steering wheel of a car or when the patient puts the arm in the sleeve of a coat.[27]

Functional examination

The basic functional examination may show some abnormal findings, typical for a possible minor instability: an enlarged range of movement (ROM) and a loose end-feel. Momentary subluxation can also appear as a kind of painful arc.[28] During active elevation of the arm, the humeral head subluxates to a certain extent, blocks the movement for a while and slips back into place as the movement is continued. Recently it has been shown that a real painful arc, caused by impingement, may coexist in subluxation of the shoulder as a secondary phenomenon to the instability.[29,30] An overlap seems to exist in the concepts of instability and impingement and for this reason when a painful arc is present minor shoulder instability must always be kept in mind.[31,32] On passive lateral rotation, when performed firmly, a different type of end-feel may be encountered. First resistance is felt, which then is overcome and finally gives rise to a new ligamentous end-feel. When the arm is brought back to neutral position a click is often felt. This sequence of sensation – although not necessarily painful – may indicate anterior instability. The same occurs in posterior instability for passive medial rotation.

Accessory tests

If a minor instability is suggested after the history and the basic functional examination, the classic stress tests to challenge the stability of the joint in various directions must be performed. During these tests, both apprehension and palpable subluxations are sought. To differentiate normal laxity from pathological instability, both shoulders should be compared. The uninvolved arm is always examined first.

Apprehension test for recurrent anterior dislocation (Fig. 4)

This test can be performed with the patient either standing or supine. The fingers of the contralateral hand are placed on the anterior aspect of the humeral head, the thumb posteriorly. With the other hand the examiner brings the arm into full lateral rotation. In this position, anteriorly directed pressure is applied with the thumb to the posterior part of the humeral head.[33] The test can be repeated with increasing degrees of abduction varying from 90° to full elevation. The test is regarded as positive if the patient judges that it provokes the same impression as the one felt when the shoulder moves out of place. Pain alone is not regarded as a positive apprehension sign. The examiner must be able to feel the humeral head subluxating or the patient looks and feels apprehensive and resists further motion. Thus, the apprehension is greater than the pain.[34]

Relocation test (Fig. 5)

If, with the arm still in full lateral rotation, a posteriorly directed force is applied to the anterior aspect of the humerus, it relocates the head of the humerus within the glenoid fossa. The patient loses the apprehension, the pain decreases and further lateral rotation is possible. This 'relocation test' is very valuable in differentiating a simple impingement from a real anterior subluxation.[35,36]

Apprehension test for recurrent posterior dislocation (Fig. 6)

The patient lies supine, with the arm abducted to 90° and the elbow flexed to a right angle. The examiner places the contralateral hand under the glenohumeral joint. The shoulder is then brought very gently into medial rotation – if the test is done too quickly, the humerus might subluxate. A positive test for posterior instability is indicated by a feeling of apprehension and the patient's resistance to further motion. The patient may also state that the feeling at the end of the test is what was felt when the shoulder was previously dislocated.

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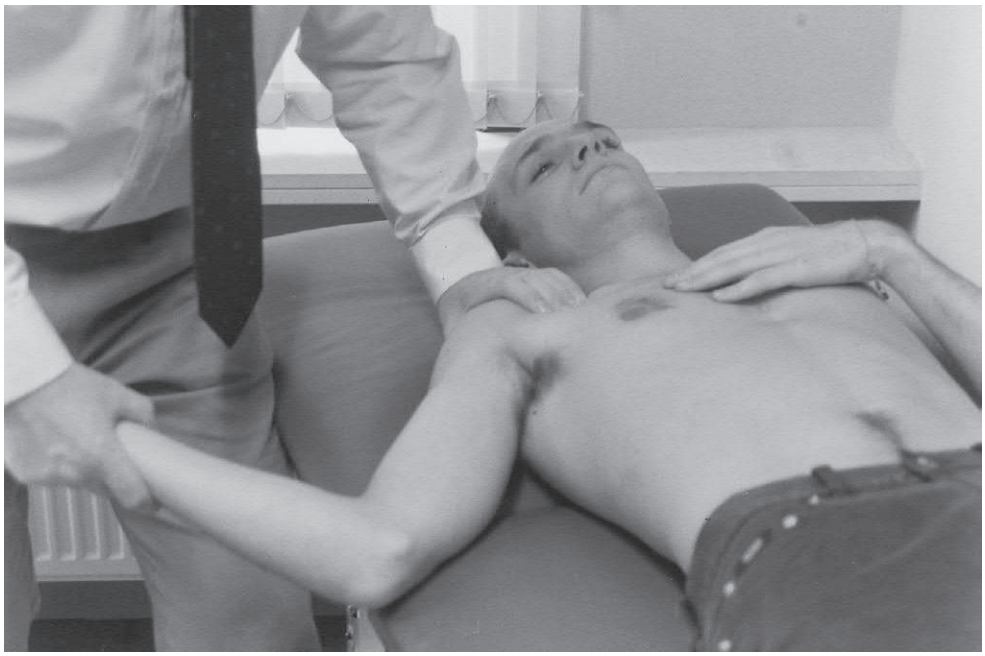

Fig 4 • Anterior apprehension test.

Fig 5 • Relocation test.

Load-and-shift manœuvres (anterior and posterior drawer) (Fig. 7)

These tests are performed with the patient in the supine position, lying with the scapula on the couch, but with the humerus beyond the edge. The contralateral hand fixates the scapula with the thumb or thenar placed on the coracoid process and the fingers on the acromion. Before assessing the amount of translation it is important that the humeral head is brought initially into its neutral position in the glenoid fossa (i.e. 'loaded'). This is achieved by placing the shoulder in a position of neutral rotation: the humerus in approximately 20° of abduction and forward flexion. The elbow is flexed and the arm directed upwards. The ipsilateral hand grasps the patient's humerus in the axilla and the shift manœuvres are performed by moving both hands in opposite directions: the anterior shift by translating the humerus anteriorly, the posterior shift by moving the humerus posteriorly in relation to the scapula. Translation of up to 25% of the humeral head diameter both anteriorly and posteriorly are considered normal,[37] although

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Fig 6 • Posterior apprehension test.

some authors state that anterior translation is less than posterior translation.[38]

Sulcus sign

With the arm hanging down and maximally relaxed, traction (Fig. 8) may provoke inferior subluxation of the head of the humerus. An increase of the space between humerus and acromion is shown in the skin being pulled towards the bone (i.e. sulcus sign).[39] When present it points towards inferior subluxation of the humeral head which is usually part of a multidirectional instability.[40]

Diagnosis

Instability tests are performed only if the patient gives a history of shoulder instability. In other conditions, for example shoulder arthritis or bursitis, these tests would be very painful. In anterior instability the apprehension and relocation tests as well as the anterior load-and-shift manœuvre are usually positive, although sometimes very subtle.[41,42] If the sulcus sign is also found, multidirectional instability is present.[43]

Diagnosis of posterior instability is based on the patient's ability to reproduce the subluxation by arm positioning in flexion and medial rotation. Posterior apprehension is usually absent and posterior translation of the humeral head on the glenoid may provide the only clue to the diagnosis of posterior instability.[44,45]

If inferior instability is also present, distal traction may reproduce the symptoms, suggesting multidirectional instability.[46] It has been reported that the best position to test for inferior instability is at 20 to 30° of abduction.[47]

Since a momentary subluxation of the humeral head on elevation can give rise to the impression of a painful arc, all other lesions causing a painful arc must also be included in the differential diagnosis. None of these lesions, however, provokes the sensation of instability. In the assessment of chronic instability of the shoulder, special X-ray views may be helpful to determine bony anatomy and pathological change.[48] CT scan, MRI and arthrography seem to have limited application in the routine roentgenographic diagnosis of the unstable shoulder.[49]

Treatment

Initial treatment for minor instability is conservative[50] and banks on three pillars:

- Strengthening of the periscapular muscles
- Training of the antagonists of the deltoid
- Training of the neuromuscular control of the rotator cuff muscles.

Provide a firm base

Fixation of the scapula must be optimal. Therefore, the scapular rotators (trapezius, levator scapulae, serratus anterior, rhomboids and pectoralis minor) must be trained to provide a stable platform for the humeral head.[51]

At first only isometric exercises should be given; later isotonic contractions are added to the training session. It is important, however, not to use long levers; only the muscles of the shoulder girdle are trained and the humerus stays alongside the trunk because long-lever exercises may put excessive strain on the rotator cuff and shoulder ligaments.

Train strength and propriocepsis of the antagonists of the deltoid: the latissimus dorsi and the pectoralis major muscles. To start with, only isometric exercises from 90° of abduction are given. The purpose is to train the muscular feeling and

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(a)

(b)

Fig 7 • Anterior (a) and posterior (b) load-and-shift manœuvres.

active relaxation of the deltoid. A caudal movement of the humeral head is encouraged. Later on isotonic exercises are added. These consist of active adduction movements, using weights and a pulley – in the beginning only with a short lever, later with the use of the stretched arm. Finally, coordination exercises complete the session: training of contractions of the antagonists during active elevation of the arm – this in an attempt to pull the head of the humerus in a caudal direction during active abduction of the arm.

Proprioceptive exercises for the rotator cuff muscles

It has been demonstrated that the muscles of the rotator cuff serve a complementary function to adjust tension in the capsuloligamentous system.[51] The simultaneous contraction of rotator cuff and biceps causes the humeral head to be compressed into the glenoid. Furthermore, selective muscular contraction of the subscapularis plays an important role in limiting

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Fig 8 • Sulcus sign.

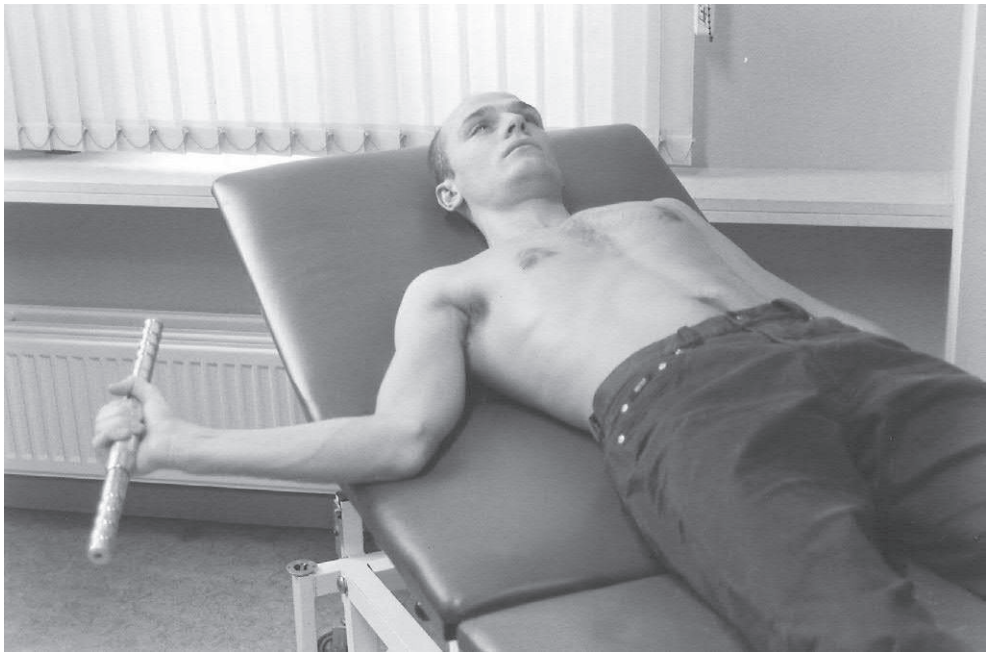

Fig 9 • Exercises for infraspinatus–subscapularis.

anterior translation,[52,53] whereas teres minor and infraspinatus have a similar function with regard to posterior instability.[54] The purpose is to train the muscular response to the capsular neuroreceptor input. This is mainly done under eccentric conditions: the muscle is activated while being stretched. Acceleration and deceleration movements are performed under high speed while using small weights.

In practice the training consists of three exercises:

- *In supine lying:* the elbow is held close to the body. A small weight (+/– 1 kg) is rotated towards and away from the trunk. The speed of rotation will change from one cycle/3–4 seconds to one cycle/second. This movement trains capsular feedback for the infraspinatus and subscapularis muscles (Fig. 9).
- *In side lying:* the shoulder is slightly flexed and in medial rotation. A small weight (0.5 kg) is lifted and brought back. The arm remains stretched and the movement is repeated, first slowly (one cycle/3 seconds) and gradually speeding to one cycle/second. This movement trains capsular feedback for the supraspinatus muscle (Fig. 10).

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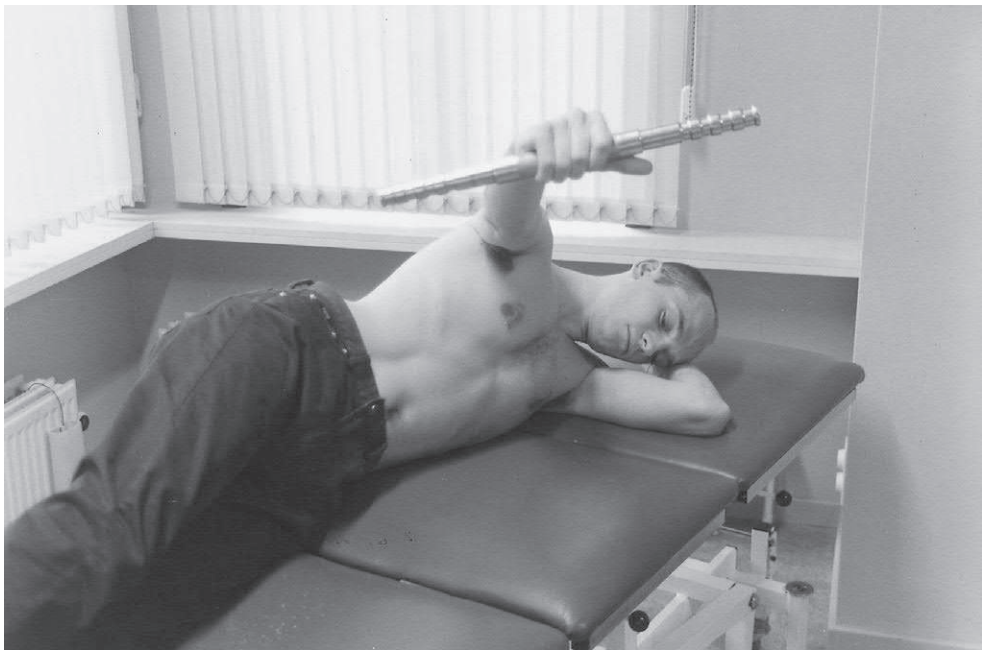

Fig 10 • Exercises for supraspinatus.

- *Standing:* the trunk is slightly flexed, the arm hanging by the side. A weight of about 2–3 kg is used. The patient is asked to let the arm hang down and to relax the deltoid muscle as much as possible. Maximal internal and external rotations are now performed. First slowly, to the very end of range, then more quickly. These acceleration–deceleration exercises, with increasing velocity, train the capsular feedback for subscapularis, infraspinatus and teres minor. The deltoid should be maximally relaxed (Fig. 11).

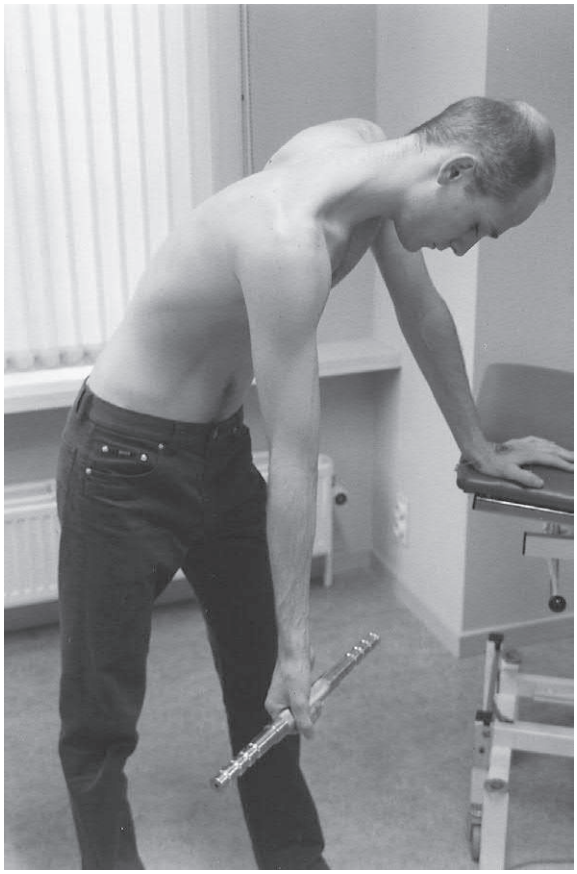

Fig 11 • Acceleration–deceleration exercises.

Practitioner's checklist

- It is important to start the exercises only when the routine basic clinical examination is painless.
- Use small weights, especially in the beginning; the exercises should be performed as relaxed as possible.
- Ask the patient to repeat the exercises as often as possible during the day in order to attain maximal neuromuscular feedback.

Surgery

When surgery is called for, it is extremely important that the differentiation between a posterior, anterior and multidirectional displacement is clear. If not, the operation will make the condition worse. The indications for operation depend on various factors such as disability during daily activities, pain and frequency of dislocations. Operative results are fairly good in anterior instability[55] depending on the type of surgical procedure[56,57] but results are less good in posterior instability in which a 40% failure rate exists.[58] Multidirectional instability is also not conducive to a good outcome.[59]

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Disorders associated with a painful arc

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A painful arc is pain felt at about half range in a movement, in the absence of pain before and after the mid-range (see p. 213). Pain may or may not recur at the end of the movement. An arc results from a momentary impingement of a lesion, lying in such a position that it is caught between the lesser or greater humeral tuberosities and the overlying anterior part of the acromion, the coracoacromial ligament or the acromioclavicular joint[1–4] (Fig. 1). A painful arc is mainly found on elevation, sometimes on medial rotation of the arm. Irrespective of the precipitating movement, the diagnostic significance remains the same: temporary impingement.

An arc on elevation is usually most pronounced during the active movement, and more on the way up than on the way down. This is due to the muscular activity, which pulls the humeral head closer to the acromion during active elevation.[5]

Sometimes the patient tries to avoid the painful moment by changing the direction of the elevation when the pain is felt. Indeed, some patients abduct until they feel the pain start at mid-range and then bring the arm in front of the body to avoid further impingement.

Occasionally the patient may experience the pain at mid-range only on the way down, or even on passive medial rotation. This has the same meaning as a painful arc on the way up. If the examiner has the impression that passive elevation or medial rotation is limited, it is important to insist that the movement is gently continued, otherwise the patient may voluntarily stop the movement too early, due to pain. The apparent limitation may be overcome by persistence, and it may be possible for the patient to then get beyond the painful moment. Thus, what might initially be regarded as a limited movement can be proved to be a painful arc with full range, which has completely different diagnostic implications.

Neer introduced the term 'impingement syndrome', now widely recognized and further divided into a 'subacromial' and a 'subcoracoid' impingement syndrome.[6–9] Cyriax always considered painful impingement as a sign rather than a syndrome: it does not implicate just one lesion but one of several.[10,11] Since the exact diagnosis is in the majority of the cases based on other tests – very often on resisted movements – a painful arc usually has value as a localizing sign, defining exactly in which particular part of the structure the lesion lies.

Movement in the subacromial space is between a convex, cuff-covered proximal humerus and a concave surface consisting of the coracoacromial roof and the inferior part of the acromioclavicular joint.[12] The gliding surface between these two moving spheres is the bursa. Pathology in one or more of these structures may partly interfere with movement and thus cause the clinical sign, 'painful arc' (Fig. 2).

Disorders of the inert structures

Acute subdeltoid bursitis

In acute subdeltoid bursitis an arc is present for the first few hours after the onset. At the end of the spontaneous course,

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say after 4–5 weeks, when the pain has become much less, the arc appears again.

In practice, the arc is seldom found because the patient presents 2–3 days after the onset, once the pain has become unbearable. At this stage, the pain is so severe that active and passive elevation beyond the horizontal is not possible (see p. 234).

Chronic subdeltoid bursitis

In almost all cases of chronic subdeltoid bursitis a painful arc is present. Here it is not regarded as a localizing sign but as a basic element in the diagnosis. It is most commonly associated with pain at the end of all passive movements, sometimes as the only sign, sometimes combined with pain on resisted abduction and lateral rotation.

When an arc is very pronounced, the diagnosis is most probably chronic subdeltoid bursitis. Occasionally, the arc is very subtle. It may be absent on lateral elevation, but when it is repeated with the arm anteriorly it may show. The same goes for lateral elevation with the arm in medial or lateral rotation. In these cases, elevation should be repeated in several different ways (see p. 246).

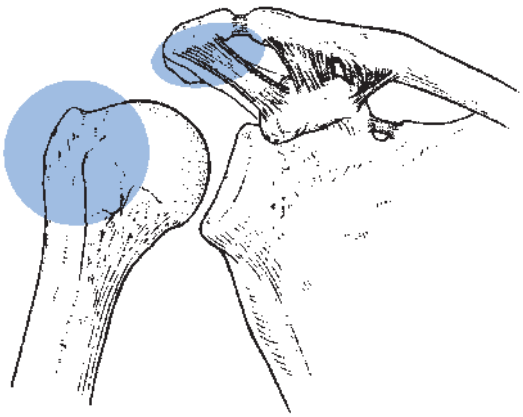

Fig 1 • Site of impingement (blue) in painful arc.

Sprain of the acromioclavicular joint

Pain at the end of all passive movements, together with more pain on passive horizontal adduction is typical of a sprain of the acromioclavicular joint. In some cases, a painful arc is also present. It indicates that the lesion lies where it can impinge, which only happens to the inferior ligament. A sprain of the superficial ligament can never cause a painful arc but palpation elicits local tenderness.

Differentiation from a chronic subdeltoid bursitis may be very difficult. A diagnostic infiltration with local anaesthetic may be necessary (see p. 241).

Acromial metastases

This is a rare disorder. Due to the metastases, the acromion becomes so tender that even the slightest pressure from below

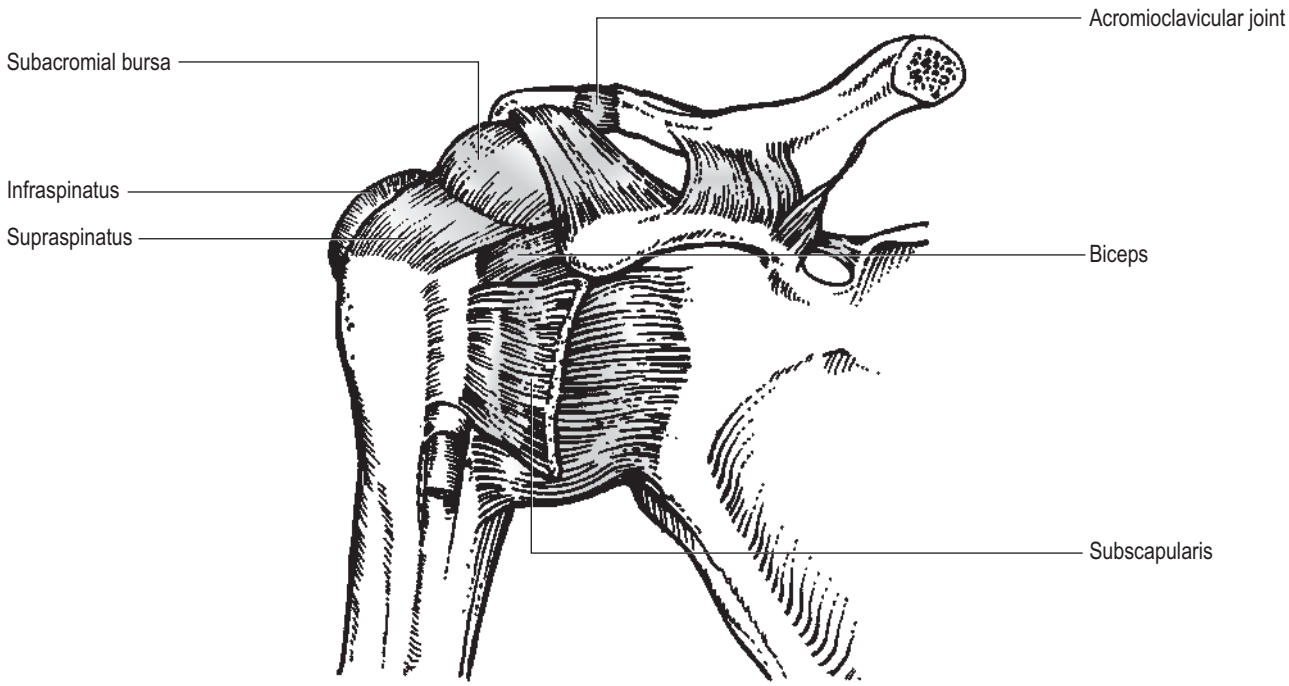

Fig 2 • Structures that may be responsible for the existence of painful arc.

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or above causes severe local pain, which does not spread in the arm. In consequence, local tenderness on palpation and a painful arc are found. Resisted abduction is usually very painful and extremely weak. These signs, together with localized warmth, call for immediate radiography.

Warning

Local tenderness on palpation and localized warmth over the acromion, together with a painful arc and painful weak resisted abduction, should prompt suspicion of metastases.

Disorders of the contractile structures

All the contractile structures mentioned below may give rise to a painful arc in that some part of them can be pinched between humerus and coracoacromial arch.[13]

Supraspinatus tendinitis

This is the most common cause of painful arc. For this reason, many authors consider that a painful arc usually means that the supraspinatus is involved.[14,15] This is not so, since the arc should be regarded only as a localizing sign; resisted abduction identifies the structure at fault. If an arc is also present it shows the lesion to lie at the superficial (bursal) aspect of the tenoperiosteal insertion (see p. 253).

Infraspinatus tendinitis

In this case, pain on resisted lateral rotation is the basic sign. The arc shows the lesion to lie at the superficial aspect of the tenoperiosteal insertion (see p. 263).

Subscapularis tendinitis

Pain on resisted medial rotation draws attention to the subscapularis muscle. As the insertion of the subscapularis is about 3 cm long, only the proximal half of it can impinge on elevation. If the lesion lies in the lower half an arc is not found but passive horizontal adduction is painful (see p. 267).

Tendinitis of the long head of the biceps

This is a rare lesion which causes pain in the shoulder on resisted flexion and supination of the elbow. The combination of these signs with a painful arc means that the lesion must lie where it can impinge. The intra-articular part of the long head of the tendon is the only site.

Disorders mimicking painful arc

Capsular laxity of the shoulder

In some instances of shoulder instability the following is encountered: when the patient lifts the arm up sideways there is a little 'stop' at about midway but once beyond this point, the normal movement continues. At the same time, the patient has the impression that something comes out of place and then slips back. In this event, the stop is caused by a partial dislocation of the humeral head, which slips back when the movement continues. Such momentary subluxation is not usually painful. It has nothing to do with an impingement and therefore is not regarded as a painful arc. Recent investigations have shown that slight instability of the shoulder may be one of the precipitating factors in the development of rotator cuff disease. It is therefore highly possible that subluxation of the humerus coexists with a lesion of one of the rotator cuff tendons and thus with a painful arc.[16–19]

Apprehension tests for shoulder instability must be performed to arrive at the diagnosis. It can also be helpful to palpate the shoulder joint during active elevation (see Chapter 14).

Snapping biceps tendon

Sometimes the long head of the biceps may slip out of the groove on active elevation, giving rise to a momentary sensation of instability. No pain is present but a sort of popping may be felt. Because no impingement occurs, this sensation cannot be regarded as a painful arc. The biceps dislocation test reveals the exact nature of the lesion (see p. 271).

Cervical disc lesion

In a cervical discodural interaction, neck and scapulo-humeral pain is sometimes provoked or increased at about mid-range during active elevation. Passive tests of the shoulder are fully negative, although some resisted movements may be painful. At the cervical spine, a partial articular pattern is found.

The presence of a painful arc in combination with signs and symptoms of a discodural interaction at the neck may confuse the examiner. Two possibilities then emerge: either the painful arc is caused by the neck problem or there is a double lesion – one at the neck and one at the shoulder.

In the former, the arc is usually felt in the scapular area, sometimes radiating down the arm. Scapular localization of the pain is very seldom due to a shoulder problem. The pain at mid-range on elevation is then explained by the muscular tension at the neck becoming greatest around the horizontal. The momentary pain cannot be considered as a true painful arc: the sign results from transmitted stress and is not provoked by any kind of impingement.

However, it is not always easy to be absolutely sure whether there is only one lesion or whether there are more. It is wise

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in these doubtful cases to start treating the most proximally located disorder (the neck) until all neck signs become normal: full range, no pain. The shoulder should then be re-examined and, if still painful, a shoulder problem must also be present and is then dealt with.

Disorders associated with painful arc are listed in Box 1.

Box 1

Disorders associated with painful arc

Inert structures

Acute subdeltoid bursitis
Chronic subdeltoid bursitis
Sprain of the acromioclavicular joint
Acromial metastases

Contractile structures

Supraspinatus tendinitis
Infraspinatus tendinitis
Subscapularis tendinitis
Tendinitis of long head of biceps

Disorders mimicking painful arc

Capsular laxity of the shoulder
Snapping biceps tendon
Cervical disc lesion

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Applied anatomy of the shoulder girdle

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The shoulder girdle forms the connection between the spine, the thorax and the upper limb. It contains three primary articulations, all directly related to the scapula: the acromioclavicular joint, the sternoclavicular joint and the scapulothoracic gliding surface (see Putz, Fig. 289). The shoulder girdle acts as a unit: it cannot be functionally separated from the secondary articulations, i.e. the lower cervical spine, the cervicothoracic junction and the upper thoracic spine, to which it is connected via the costovertebral joints.

Osteoligamentous structures

The clavicle articulates at the medial aspect with the sternum – the sternoclavicular joint – and at the lateral aspect with the acromion to form the acromioclavicular joint. In this way it connects the scapula to the trunk (Fig. 1).

Acromioclavicular joint

The rather flat articular surface at the lateral end of the clavicle articulates with the flat articular surface at the medial border of the acromion. The joint has a capsule which is reinforced by ligaments: cranially the superior *acromioclavicular ligament* and caudally the inferior one. The joint often contains an intra-articular disc, which is sometimes incomplete (meniscoid) and is subject to early degeneration. The joint line runs obliquely, from craniolateral to caudomedial (Fig. 2).

Extra-articular ligaments are important for the stability of the joint and to keep the movements of the lateral end of the clavicle within a certain range. Together they form the roof of the shoulder joint (see Standring, Fig. 46.14). They are the *coracoacromial ligament* – between the lateral border of the coracoid process and the acromion – and the *coracoclavicular ligament*. The latter consists of:

- The *trapezoid ligament* which runs from the medial border of the coracoid process to the trapezoid line at the inferior part of the lateral end of the clavicle.
- The *conoid ligament* which is spanned between the base of the coracoid process and the conoid tubercle just medial to the trapezoid line.

Movements in the acromioclavicular joint are directly related to those in the sternoclavicular joint and those of the scapula. This joint is also discussed inthe online chapter *Applied anatomy of the shoulder*.

Sternoclavicular joint

The sternoclavicular joint is more complex. It is formed by the articular surface at the medial aspect of the clavicle and the articular surface at the superolateral corner of the sternal manubrium. The two joint surfaces are discongruent and this is resolved by the presence of an intra-articular disc, which divides the joint into two cavities. The rather loose joint capsule is reinforced by anterior and posterior *sternoclavicular ligaments*. There are two extracapsular ligaments: the *interclavicular ligament*, which interconnects both clavicles and covers the jugular notch, and the *costoclavicular ligament*, between the sternal end of the first rib and the medial part of the clavicle (see Putz, Fig. 285).

Close behind the joint lie some vital structures: some important blood vessels (aorta, brachiocephalic trunk,

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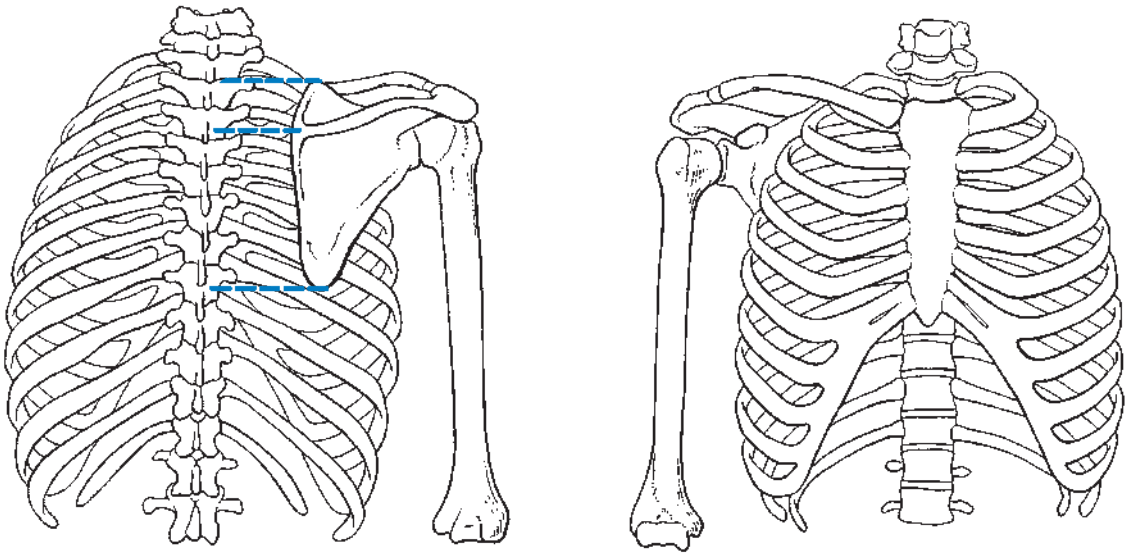
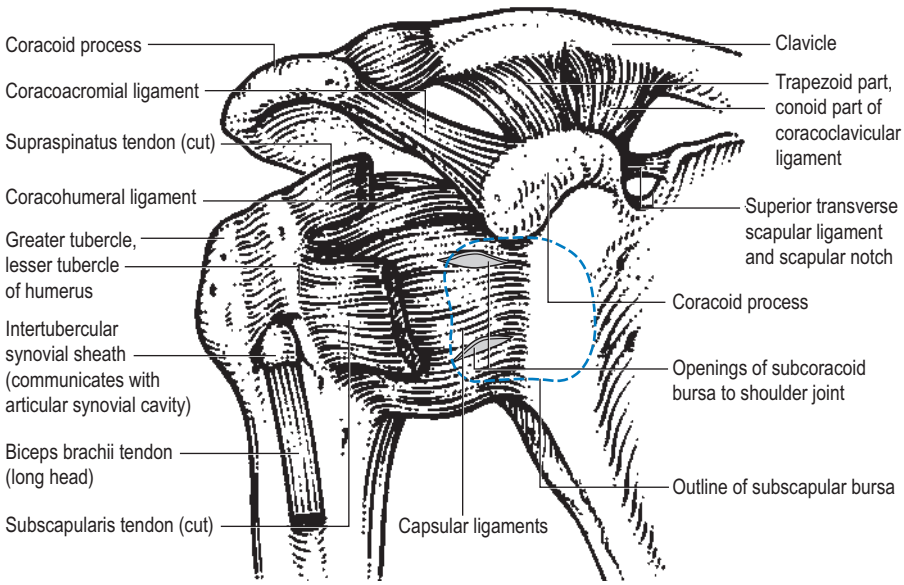


Fig 1 • Bony structures of the shoulder girdle (anterior and posterior view).

Fig 2 • The acromioclavicular joint.



brachiocephalic vein, subclavian artery, subclavian vein, jugular vein and carotid artery), the trachea, the oesophagus, the lung and pleura (Fig. 3).

Movements in the sternoclavicular joint are possible around three axes:

- Clavicular elevation and depression occur around an anteroposterior axis
- Clavicular protraction – the lateral end of the clavicle moves forwards, and retraction – the lateral end of the clavicle moves backwards, around a vertical axis
- Backwards and forwards rotation of the clavicle around its longitudinal axis.

Glenohumeral and scapular movements also mobilize the clavicle and thus influence the sternoclavicular joint. Diminution of the mobility of the clavicle will have direct consequences for the joints with which it articulates – sternoclavicular and acromioclavicular – and indirectly for the glenohumeral joint.

Scapulothoracic gliding mechanism

The scapulothoracic gliding mechanism is a 'virtual articulation' indicating that the scapula is mobile in relation to the thorax.

The gliding surfaces are formed by the anterior aspect of the scapula and the posterior aspect of the thorax. In between the scapula and the thorax lie the subscapular and the serratus anterior muscles (see below). Together with their

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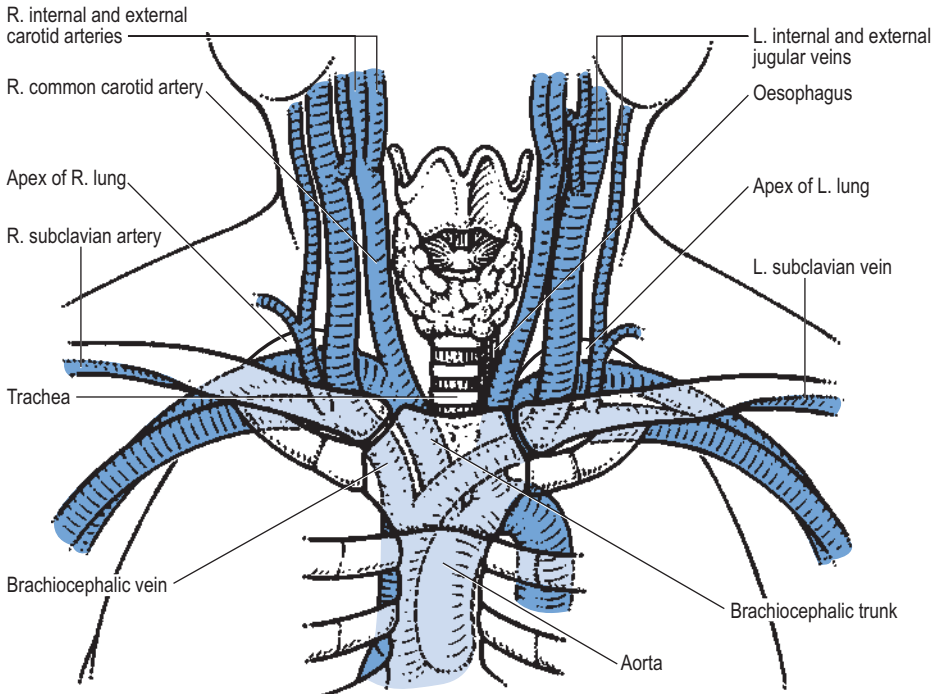

Fig 3 • Vital structures lying in the mediastinum behind the sternoclavicular joint.

fasciae they take part in the gliding mechanisms that occur during movement.

The scapula moves in three directions:

- Up and down (elevation and depression, respectively) when the lateral end of the clavicle does the same. The medial border of the scapula remains more or less parallel to the spine.
- Lateral and medial rotation – the inferior angle moves laterally and medially. These movements are mainly induced by movements of the arm towards abduction/elevation and extension.
- It glides laterally and medially – protraction and retraction or approximation – when the lateral end of the clavicle does the same. The scapula glides away from the spine but its medial border remains parallel to the spine (Fig. 4).

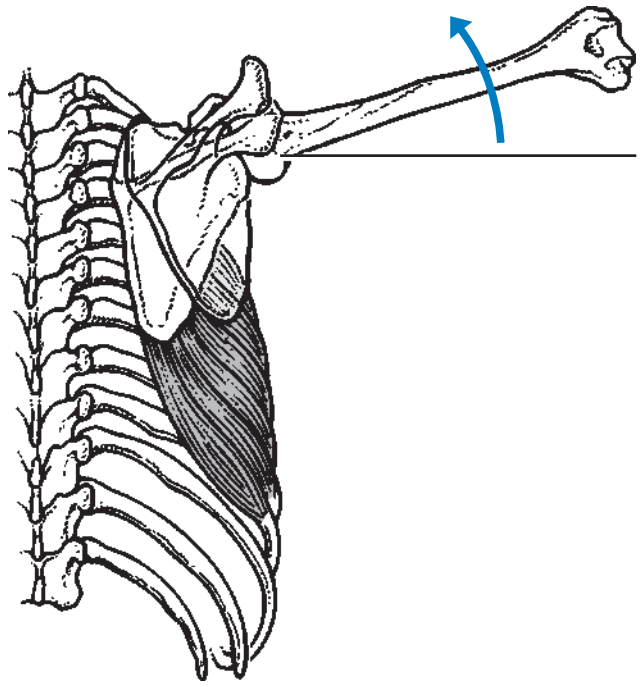

Fig 4 • The scapulothoracic gliding mechanism.

Costovertebral joints

The ribs articulate with the thoracic spine at two levels (see Standring, Fig. 54.11):

- At the vertebral body – the costovertebral joint: the head of the rib articulates with the lateral aspect of one or two vertebral bodies. The joint capsule is reinforced by the *radiate ligament of the joint at the head of the rib*. Intra-articularly the head of the rib is connected to the intervertebral disc by the *intra-articular ligament of the joint at the head of the rib*.
- At the transverse process – the costotransverse joint: the tubercle of the rib articulates with the articular facet on the transverse process of the vertebra. The joint capsule is again reinforced by several ligaments: *costotransverse ligament* – between the neck of the rib and the transverse process; *lateral costotransverse ligament* – between the angle of the rib and the tip of the transverse process; and the *superior costotransverse ligament* – between the crest of the neck of the rib and the inferior border of the transverse process of the vertebra above (Fig. 5).

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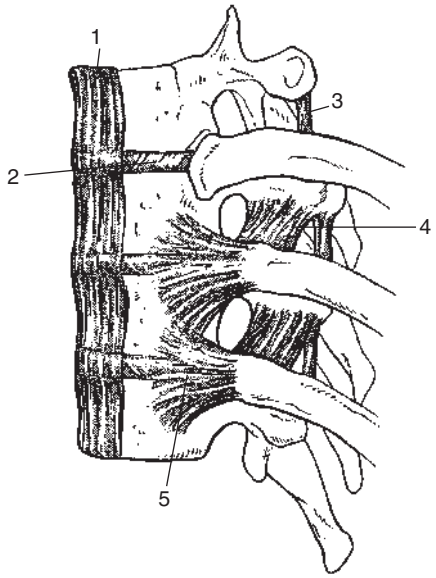

Fig 5 • The costovertebral joints. (1) anterior longitudinal ligament; (2) disc; (3) costotransverse ligament; (4) superior costotransverse ligament; (5) radiate ligament.

Muscles and their innervation

Those muscles that create movement at the acromioclavicular joint, the sternoclavicular joint or in the scapulothoracic gliding mechanism, or those muscles that connect the scapula to the trunk, can be considered as 'shoulder girdle musculature'. Many of these muscles also have influence on the secondary articulations: the cervical spine, the shoulder or the thoracic spine and are discussed in the online chapters *Applied anatomy of the cervical spine*, *Applied anatomy of the shoulder*, and *Applied anatomy of the thorax and abdomen* respectively. In this chapter only those muscles that are not discussed elsewhere are described.

Anterior aspect of the shoulder girdle

The muscles at the anterior aspect of the shoulder girdle are outlined in Table 1 and Figure 6.

Sternocleidomastoid muscle

The sternocleidomastoid muscle originates with two heads, one from the manubrium of the sternum and one from the medial end of the clavicle (see Standring, Fig. 28.5). It inserts at the mastoid process and the superior nuchal line. Contraction of the muscle puts stress onto the sternoclavicular joint.

Subclavius muscle

The subclavius takes origin at the junction between the bone and the cartilage at the sternal end of the first rib (see Standring, Fig. 46.24). It inserts at the inferior and lateral aspect of the clavicle. It pulls the clavicle against the sternum and has a stabilizing effect on the sternoclavicular joint.

Table 1 Anterior muscles of the shoulder girdle

Muscle	Nerve	Spinal nerve root
Sternocleidomastoid	Spinal accessory and cervical plexus	C1–C2
Subclavius	Nerve to subclavius	C5–C6
Pectoralis minor	Pectoral nerves	C6–C8
Pectoralis major	Pectoral nerves	C5–T1
Deltoid	Axillary nerve	C4–C6
Pectoral part	Pectoral rami	C4–C5
Biceps brachii	Musculocutaneous nerve	C5–C6
Coracobrachialis	Musculocutaneous nerve	C6–C7

Pectoralis minor muscle

The pectoralis minor muscle takes origin anteriorly at the third to fifth ribs and inserts at the inferior and medial border of the coracoid process (see Standring, Fig. 46.24). Its contraction results in a depression and protraction of the scapula.

Costocoracoid fascia

The costocoracoid fascia is a strong aponeurosis that envelops the subclavius, pectoralis minor and partly the coracobrachialis muscles. It runs deeply from the coracoid process and is attached to the anterior costal wall.

Posterior aspect of the shoulder girdle

The muscles at the posterior aspect of the shoulder girdle are outlined in Table 2 and Figure 7 and see Standring, Fig 42.52.

Levator scapulae muscle

The levator scapulae takes origin at the posterior tubercles of the transverse processes of the first to fourth cervical vertebrae and inserts at the superior angle and superior part of the medial border of the scapula (see Standring, Fig. 46.21). It elevates and medially rotates the scapula.

Serratus anterior muscle

The serratus anterior has origin with nine or ten heads at the lateral aspect of the first to eighth or ninth ribs and inserts along the entire medial border of the scapula, from the superior to the inferior angle (see Putz, Fig. 392). Three different parts are recognized: superior, medial and inferior. It fixates the scapula against the thorax and moves the scapula towards protraction and lateral rotation. The muscle is innervated by the nervus thoracicus longus.

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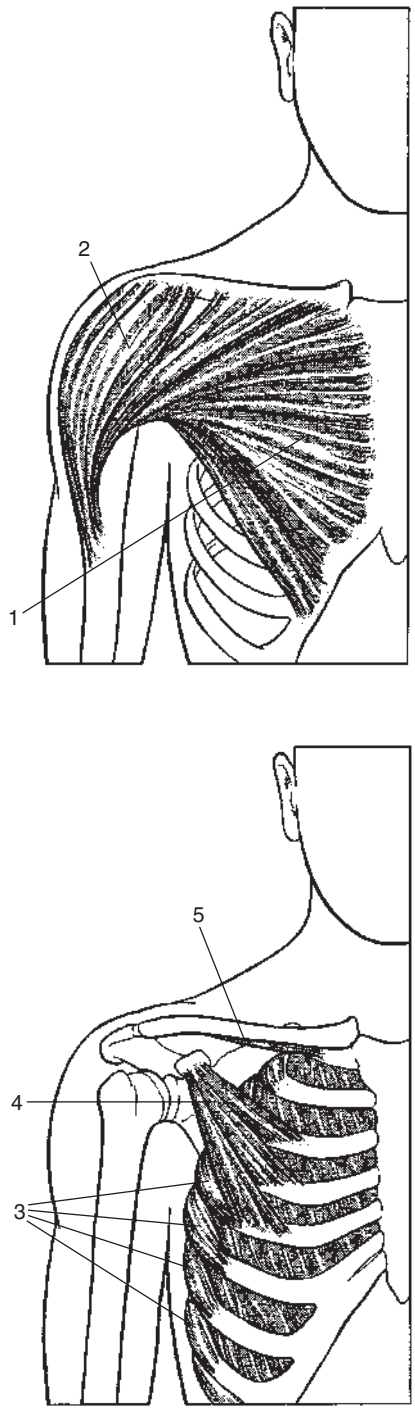

Fig 6 • Anterior muscles of the shoulder girdle. (1) pectoralis major; (2) deltoid; (3) serratus anterior; (4) pectoralis minor; (5) subclavius.

Table 2 Posterior muscles of the shoulder girdle

Muscle	Nerve	Spinal nerve root
Trapezius	Spinal accessory nerve	C2–C4
Levator scapulae	Dorsal scapular nerve	C4–C5
Rhomboids	Dorsal scapular nerve	C4–C5
Serratus anterior	Long thoracic nerve	C5–C7
Latissimus dorsi	Thoracodorsal nerve	C6–C8
Teres major	Thoracodorsal nerve	C6–C7
Subscapularis	Subscapular nerve	C5–C8
Infraspinatus	Suprascapular nerve	C4–C6
Teres minor	Axillary nerve	C5–C6
Supraspinatus	Suprascapular nerve	C4–C6
Deltoid	Axillary nerve	C4–C6
Triceps brachii	Radial nerve	C6–C8

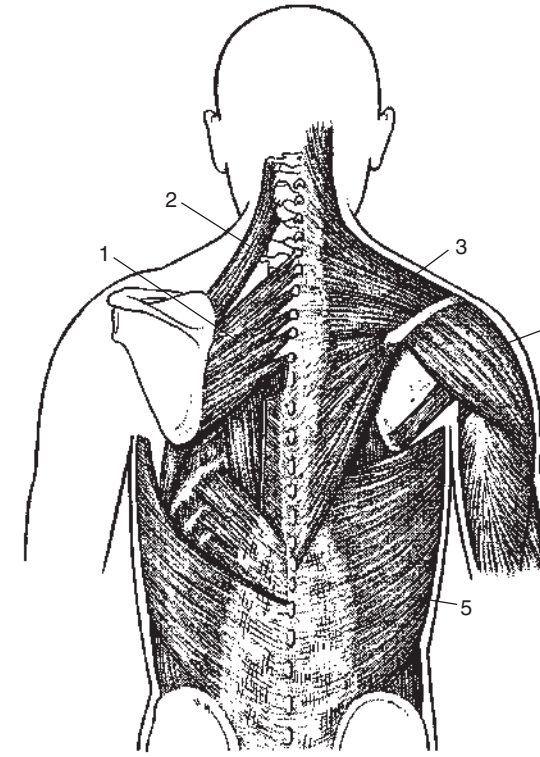

Fig 7 • Posterior muscles of the shoulder girdle. (1) Rhomboids; (2) levator scapulae; (3) trapezius; (4) deltoid; (5) latissimus dorsi.

Mobility of the shoulder girdle

Mobility in the shoulder girdle depends on the mobility of the primary joints – acromioclavicular, sternoclavicular and scapulothoracic gliding mechanism. It can only be maximal when the secondary articulations also function normally.

The movements that occur when the shoulder is moved and that can be assessed in shoulder girdle examination are:

- *Elevation*: the shoulder moves upwards in the frontal plane. The range is approximately 30–45°.

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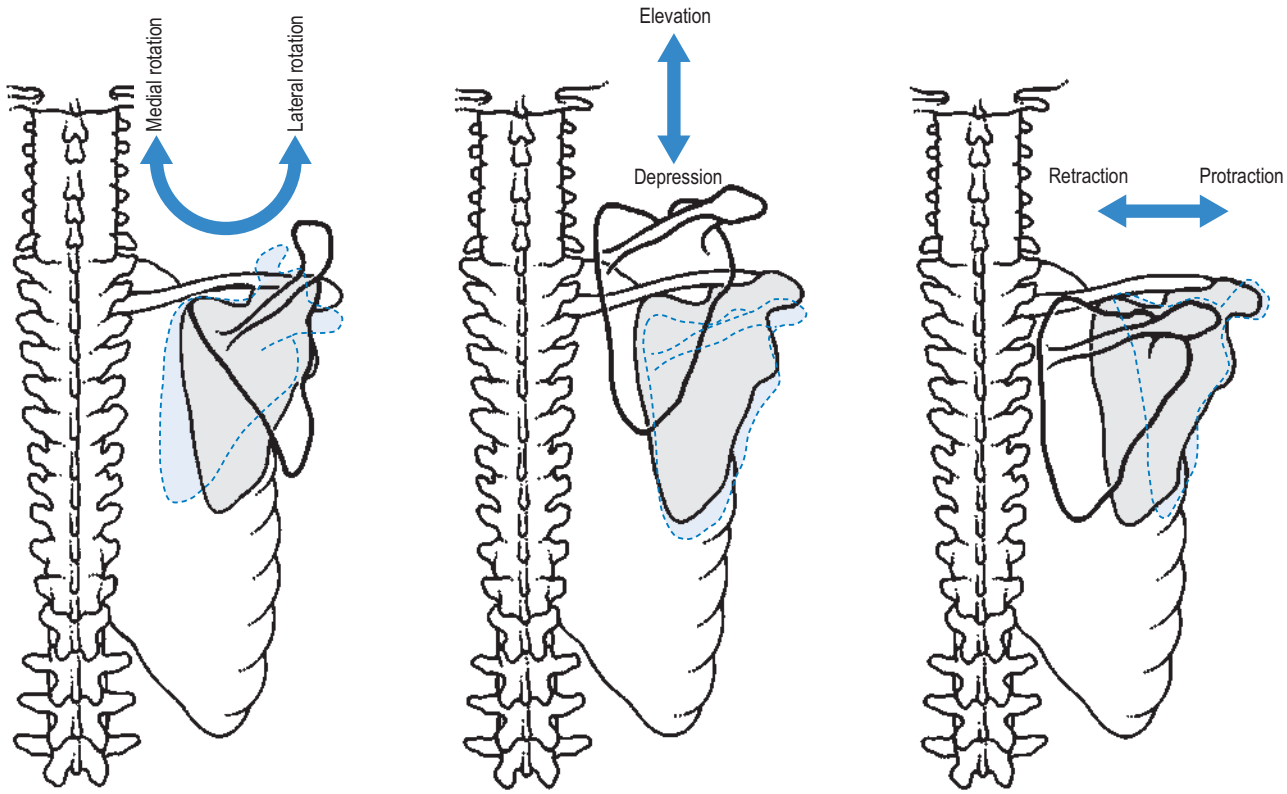

Fig 8 • Movements at the shoulder girdle.

- *Depression*: the shoulder moves downwards in the frontal plane. The range is about 5°.
- *Protraction*: the shoulder moves forwards in a transverse plane with a range of up to 30°.
- *Retraction*: the shoulder moves backwards in a transverse plane. The range is about 30°. This movement is also referred to as 'scapular approximation'.

Anterior and posterior rotation of the clavicle cannot be examined separately. It is the result of arm movements towards flexion or elevation and extension (Fig. 8).

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Clinical examination of the shoulder girdle

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The shoulder girdle acts as a zone of transition. It is localized between three other regions: the cervical spine, the upper thoracic spine and the shoulder.

Symptoms referred to the shoulder girdle

Pain originating from the cervical spine or from the upper thoracic spine is frequently felt at the shoulder girdle, for example at the base of the neck, in the trapezius muscle or in the scapular area. It may also radiate towards the subclavicular, pectoral or axillary regions or into the upper limb. This overlaps with possible symptoms from shoulder girdle disorders.

Shoulder girdle movements are therefore included in both the cervical (see Chapter 6) and thoracic (see Chapter 25) examinations. This preliminary screening may arouse suspicion about the shoulder girdle.

Symptoms referred from the shoulder girdle

Pain that originates from a disorder in the shoulder girdle is usually experienced at the base of the neck, in the pectoroclavicular area or in the trapezius muscle. It rarely spreads down the arm and thus arm pain may implicate either the spine or the shoulder.

Paraesthesia in the arm may result from a condition affecting the brachial plexus in the shoulder girdle.

History

Symptoms from a disorder in the shoulder girdle are not typical. They mimic those of neck, upper thorax or shoulder.

History-taking will therefore start in the same way as for the examination of the cervical (see Chapter 6) or thoracic spine (see Chapter 25) or of the shoulder (see Chapter 12). The examiner will notice elements that may lay blame on the spinal joints (e.g. pain shifting from the centre to one side) or features that point towards a lesion of the shoulder girdle (e.g. increase of symptoms following scapular movements).

Inspection

On inspection, special attention must be given to swelling (i.e. in the supraclavicular fossa), changes in the colour of the skin, muscular atrophy, difference in height of the shoulders and winging of the scapula.

Functional examination

The functional examination of the shoulder girdle is never done as primary testing but follows evaluation of one of the other regions, which may provide elements that implicate the shoulder girdle.

Clinical examination of the shoulder girdle is an accessory to general assessment.

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If the patient has described symptoms that could originate from the spine or from the shoulder, the cervical, thoracic or shoulder examination is performed. When, in this examination, signs are found that point towards the shoulder girdle (e.g. positive scapular tests), these will be examined thoroughly.

When the history is unspecific a preliminary examination (quick survey) of the upper quadrant is done (see p. 212). This includes tests for:

- the neck
- the shoulder girdle
- the shoulder
- the arm: as a neurological examination and to exclude local disorders in the upper limb.

Functional examination of the shoulder girdle is very simple: three active, three passive and four resisted movements are performed (see Fig. 1), summarized in Box 1.

Active movements

The active tests cause movement in the three primary articulations. During all active movements (Fig. 2), attention is paid to pain, range of motion and abnormal sensations, such as paraesthesia or crepitus. The movements may stretch inert or contractile structures and make muscles work. A decreased range of movement is usually the result of either a neurological disorder or a problem of an inert structure.

Active elevation of both shoulders ('shrugging')

The examiner stands behind the patient and asks for both shoulders to be shrugged. This movement requires full mobility of the scapulae. The normal range is between 30 and 45°.

Box 1

Summary of examination of the shoulder girdle

Active movements of the shoulders

Elevation of both shoulders
Protraction of both shoulders
Retraction of both shoulders

Passive movements of the shoulder

Elevation
Protraction
Retraction

Resisted movements of the shoulder

Elevation
Protraction
Retraction
Depression

Palpation

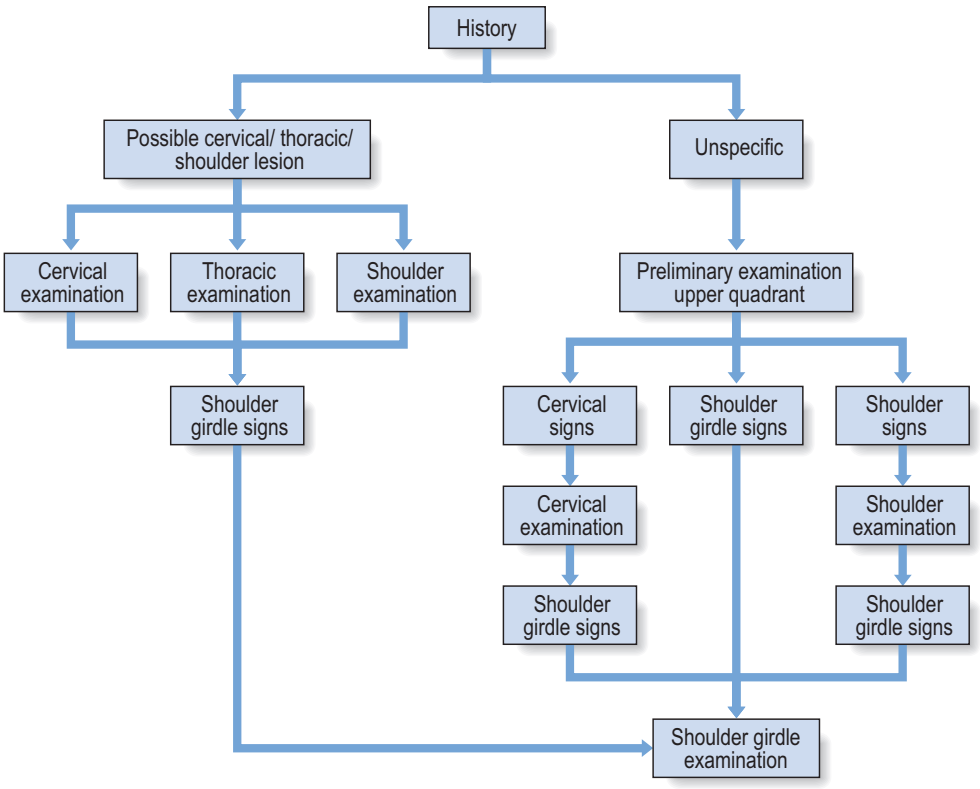

Fig 1 • Strategy for the examination of the shoulder girdle.

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(a)

(b)

(c)

Fig 2 • Active movements of the shoulders: (a) elevation; (b) protraction; (c) retraction.

Active protraction of both shoulders

The patient brings both shoulders forwards. The normal range of scapular abduction is about 30°.

Active retraction of both shoulders

The patient is asked to pull both shoulders backwards. Normally, about 30° is possible.

General considerations concerning active scapular movements

A difference in height between the shoulders on shrugging indicates impaired mobility. This may be the result of disturbance of the scapulothoracic gliding mechanism or of a neurogenic condition leading to weakness of the scapular elevators.

Pain found on active elevation may be due to a problem of a contractile or an inert structure. Passive and resisted movements reveal the true nature of the lesion. Crepitus present on active elevation means the posterior thoracic wall has roughened, which often has an unknown cause. Paraesthesia when the shoulders are kept shrugged for a while are pathognomonic of a thoracic outlet syndrome (postural variety).

Pathological findings on the other active movements may be due to muscular activity, stretching of an inert structure or a movement in one of the three primary articulations (acromioclavicular joint, sternoclavicular joint or scapulothoracic gliding surface).

Because active elevation, retraction and protraction of the shoulders pull the dura mater in a cranial direction via the first thoracic nerve root, pain on one of these movements may have a dural origin. This may occur in thoracic discodural interactions.

Passive movements

For a clear differential diagnosis between disorders of inert and contractile structures, active tests must be followed by passive and resisted movements. Passive movements (Fig. 3) put local inert structures under tension, have some influence on the joints at both ends of the clavicle and may passively stretch some contractile structures. Because the scapula glides on the thorax during these movements, the scapulothoracic gliding surface also must function properly.

The problem can be either of the scapulothoracic gliding surface, as in scapular metastases, or of one of the musculoligamentous attachments of the scapula to the trunk, or the outcome of an apical tumour of the lung. Total ankylosis of the acromioclavicular or of the sternoclavicular joint due to ankylosing spondylitis or to arthrosis is another possible cause.

For all passive tests, attention is paid to their influence on the pain, the range of movement and the end-feel. The 'normal' range of movement is the same as for the active tests; in normal subjects, the end-feel is elastic for all three passive movements, due to combined ligamentous and muscular stretching.

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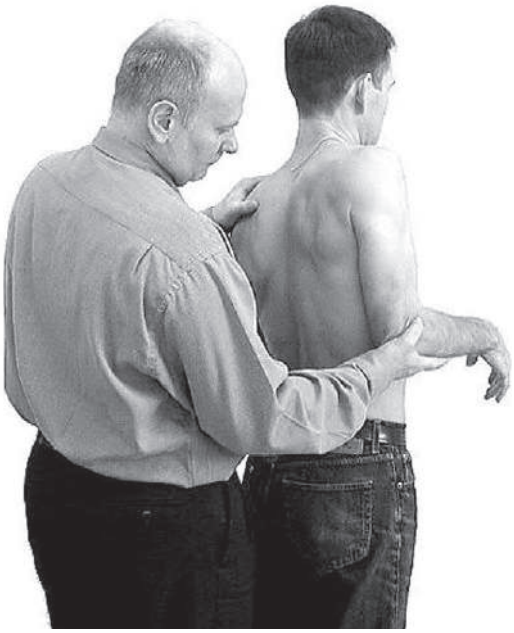
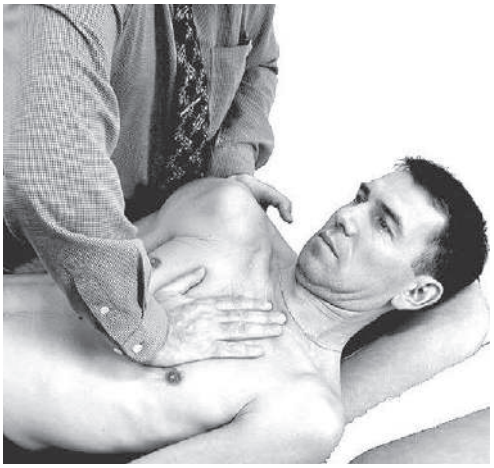


(a)



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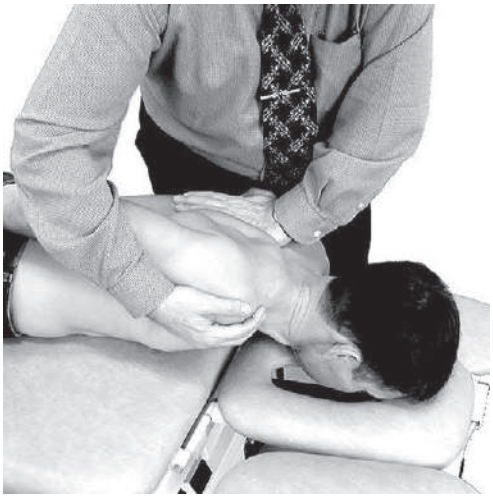

(c)

Fig 3 • Passive movements of the shoulder: (a) elevation; (b) protraction; (c) retraction.

Passive elevation of the shoulder

The examiner stands behind the patient, places one hand underneath the flexed elbow and brings the shoulder up by an upward directed pressure on the olecranon. The other hand fixates at the contralateral side of the base of the neck (Fig. 3a).

Passive protraction of the shoulder

The patient is supine. The examiner stands at the patient's painful side and asks the patient to bring the shoulder forwards actively. The ipsilateral hand on the sternum is used for fixation, with the contralateral hand on the scapula. The patient is then asked to relax and the examiner continues the movement passively to the end of range where an extra pressure is given to assess end-feel (Fig. 3b).

Passive retraction of the shoulder

The patient is asked to lie prone with the arm on the back. The examiner, at the patient's painless side, asks the patient to pull the shoulder actively backwards and then places the contralateral hand at the spine and fixes that area. The ipsilateral hand lies at the anterior aspect of the shoulder. The patient is asked to relax and the examiner continues the movement passively to the end of range where an extra pressure is given for the judgement of the end-feel (Fig. 3c).

Resisted movements

During resisted movements (Fig. 4) pain and weakness are assessed.

Resisted elevation of the shoulder

The examiner stands at the patient's painful side and puts both hands on the shoulder. The patient is asked to shrug the shoulder against the examiner's resistance (Fig. 4a). This movement tests the levator scapulae and the upper part of the trapezius, as well as the integrity of the C2–C4 nerve roots.

Resisted protraction of the shoulder

The examiner stands at the patient's side and puts one hand on the anterior aspect of the shoulder, the other one on the posterior thorax between the scapulae. The patient is now asked to press the shoulder forwards (Fig. 4b). This is a test for the pectoralis major, serratus anterior and pectoralis minor muscles.

Resisted retraction of the shoulder

The same technique is used as for resisted protraction but one hand is placed on the posterior aspect of the shoulder and one on the sternum (Fig. 4c). The following muscles are tested: rhomboids, middle and lower parts of the trapezius and latissimus dorsi.

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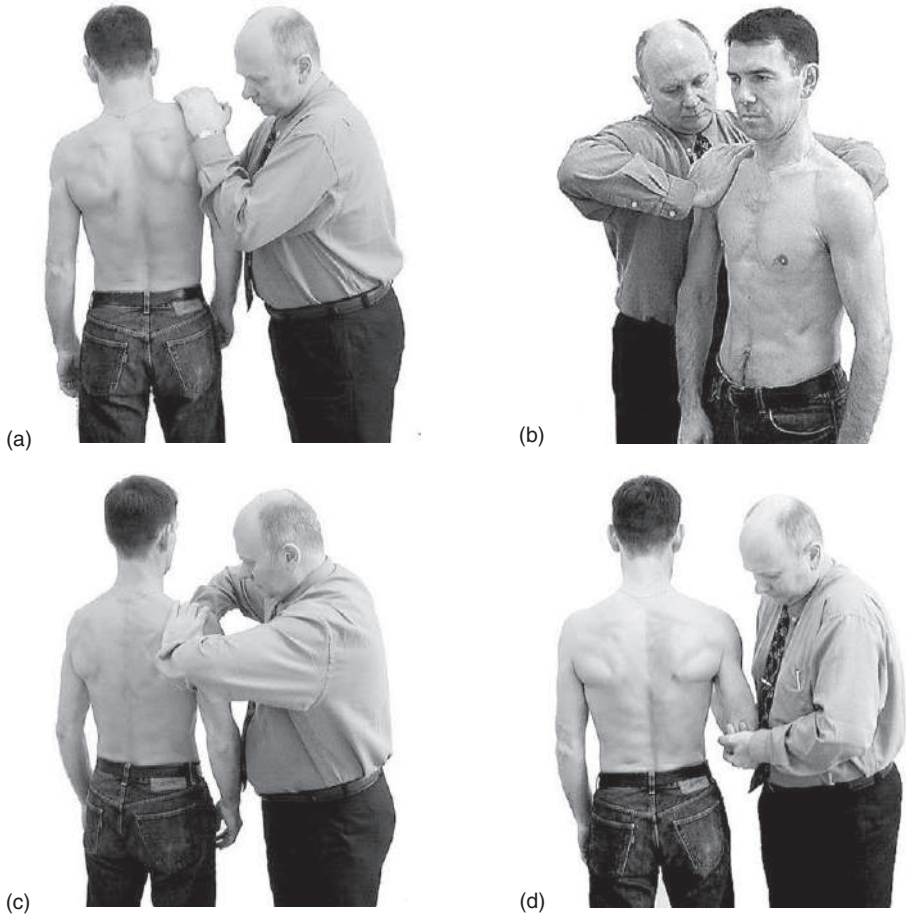

Fig 4 • Resisted movements of the shoulder: (a) elevation; (b) protraction; (c) retraction; (d) depression.

Resisted depression of the shoulder

The patient bends the elbow to a right angle. The examiner puts both hands under the elbow and asks the patient to press down (Fig. 4d). This test examines the pectoralis minor, subclavius or latissimus dorsi muscle.

Palpation

The functional tests are sometimes followed by palpation, if the structure at fault lies within the reach of the fingers.

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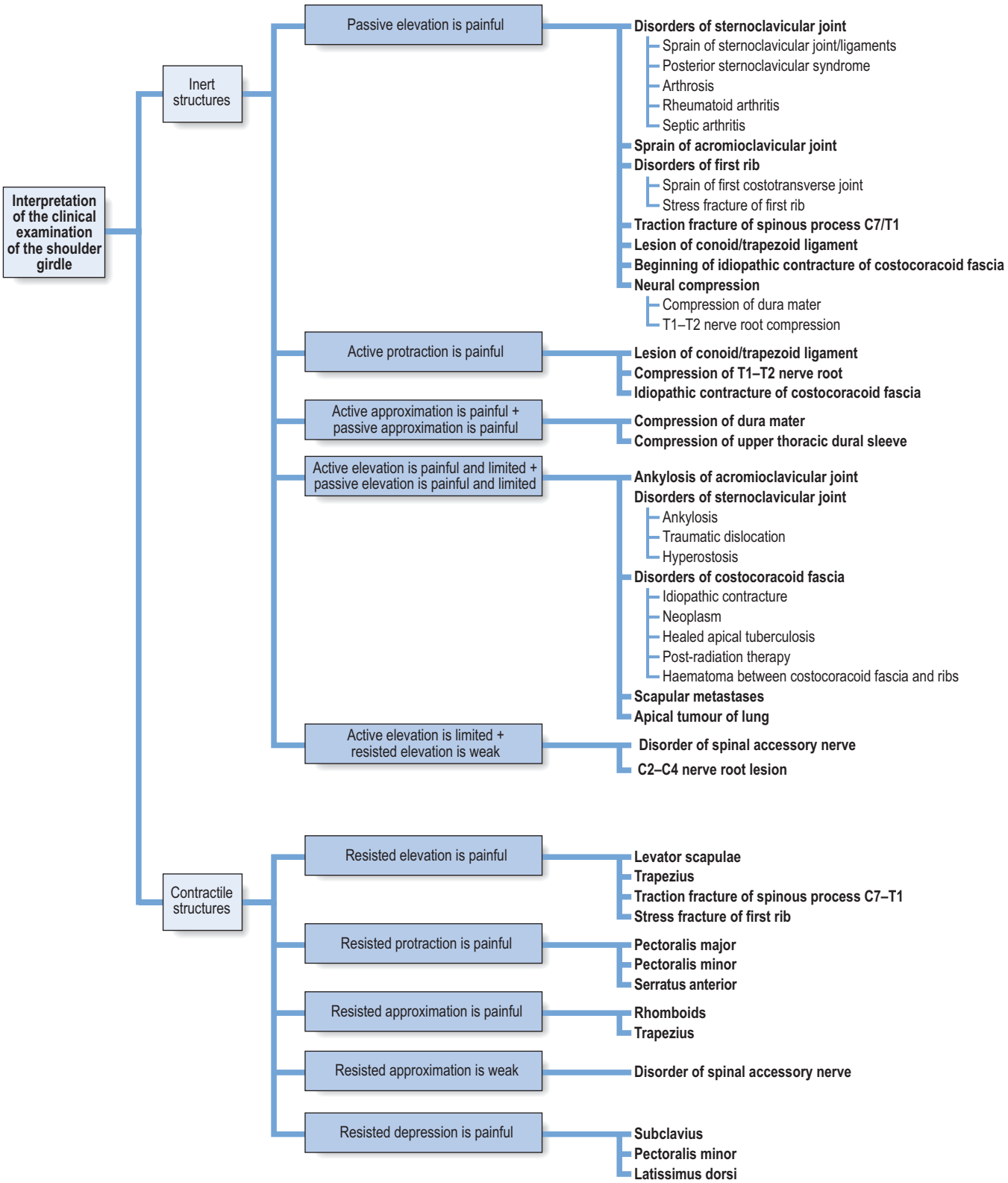
Interpretation of the clinical examination of the shoulder girdle



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Disorders of the inert structures

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Pain on active and passive elevation

(Fig. 1)

Disorders of the sternoclavicular joint

Disorders of this joint are usually the result of an injury; arthrosis, hyperostosis and rheumatoid or septic arthritis are other possibilities.[1] All conditions give rise to both scapular and shoulder signs and occasionally to signs on movements of the neck. The clinical pattern closely resembles that of an acromioclavicular joint lesion but the localization of pain at the medial end of the clavicle draws attention to the sternoclavicular joint. In posterior sternoclavicular syndrome, the pain is felt posteriorly at the base of the neck.

Positive signs are commonly found at three levels: neck or upper thorax, shoulder girdle and shoulder.

Neck signs

All active or resisted movements of the neck that involve the sternocleidomastoid muscles may also provoke some pain: active and resisted rotation towards the painless side and resisted side flexion towards the painful side.

Shoulder girdle signs

There is pain on full active and passive elevation of the shoulder. The other active and passive movements are not, or are only slightly painful.

Shoulder signs

Pain is also found on active and passive movements of the arm because almost all arm movements have some influence on the sternoclavicular joint. Pain is most marked on elevation of the arm. When a disorder of the sternoclavicular joint is suspected, passive horizontal adduction of the arm should be performed: pain is most pronounced with this test (Fig. 2).

Sprain of the sternoclavicular joint/ligaments

This is usually the result of an injury such as a fall on the outstretched hand or on the shoulder. Occasionally, a sprain is the result of overuse, which mainly occurs in arthrotic joints.

Pain is felt unilaterally over the lateral manubrial angle and may radiate into the clavicular area. It can be elicited by active and passive elevation of the scapula and by all passive movements of the arm. Resisted movements are negative. Passive horizontal adduction – an accessory test – is the most painful movement. On palpation there is tenderness over the joint line or in the ligaments.

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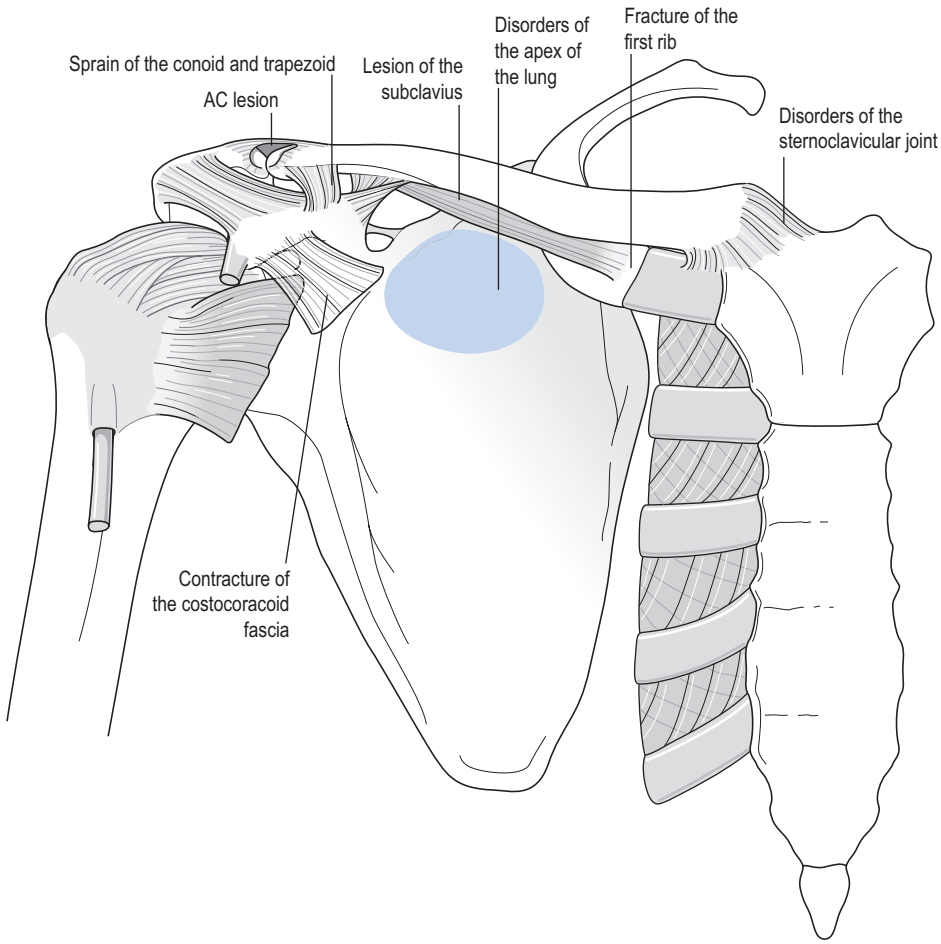

Fig 1 • Shoulder girdle lesions with clavipectoral pain.

Treatment

When the onset is recent, the arm should be placed in a sling to diminish the effect of gravity. An intra-articular steroid injection is given at once.

Technique: injection of the sternoclavicular joint

The patient lies supine. The gap between the medial end of the clavicle and the sternum is palpated. A needle of 2.5 cm is introduced, penetrating the joint to a depth of about 1 cm (Fig. 3), and 1 ml of triamcinolone acetonide is injected. If excessive resistance is encountered, the needle is in the meniscus; if this occurs, the tip of the needle should be partly withdrawn with continuous pressure on the plunger until the steroid floats in with little resistance.

Posterior sternoclavicular syndrome

This rare disorder may arise spontaneously in middle-aged people and usually causes a misleading clinical picture.

Patients complain of unilateral pain at the base of the neck. Surprisingly, the pain is felt more posteriorly and not at the sternoclavicular joint itself, which suggests a lesion of the cervical spine. Nevertheless, clinical examination of the neck does not localize the problem: there is usually full range on passive movements and no pain on active and resisted movements. Both active and passive elevation of the arm is painful. Sometimes it is even hard to achieve full range. All other movements of the arm are normal, except for passive horizontal adduction which is most painful. Clinical examination of the shoulder girdle is required. Pain is elicited by active and passive elevation of the shoulder. Resisted shoulder elevation is negative, which excludes the trapezius and levator scapulae muscles. No pain is found on palpation of the anterior portion of the sternoclavicular joint.

Treatment

One or two infiltrations of 20 mg triamcinolone into the posterior sternoclavicular ligament is helpful and can be done in one of two ways:

- By approaching the ligament from above and allowing the needle to progress behind the joint until ligamentous resistance is felt
- From the front, passing through the joint as for an intra-articular injection.

In both instances, the steroid must be infiltrated into the posterior ligament and counterpressure must be present during the whole procedure. Therefore the tip of the needle is partly withdrawn and reinserted several times, over the whole of the posterior ligament, as the product is injected. Throughout,

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Fig 2 • Passive horizontal adduction.

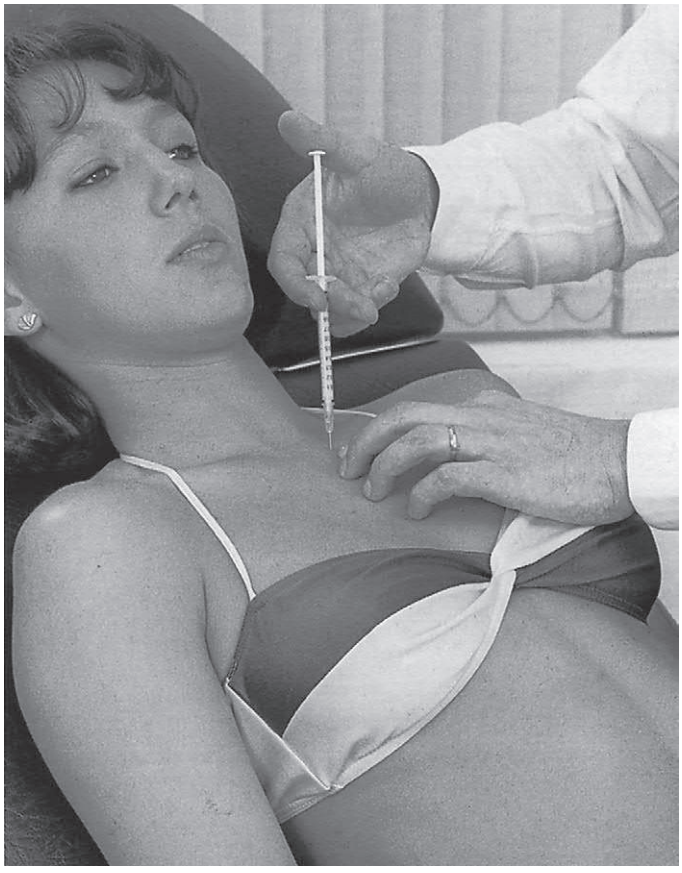

Fig 3 • Injection of the sternoclavicular joint.

special care must be taken not to penetrate neighbouring visceral structures.

The patient is reassessed after 2 weeks; if symptoms are still present a second infiltration is given. Normally two or three infiltrations suffice.

Arthrosis

Arthrosis of the sternoclavicular joint is common.[2] It occurs mainly in postmenopausal women. The chief complaint is cosmetic: there is a visible thickening of the joint. The pain, if any, is minor [3]. Elevation of the arm is limited as the result of the limitation of the shoulder girdle movement. Movements of the shoulder girdle are uncomfortable but not really painful.

Radiography shows degenerative changes (osteophytes, bone cysts, hyperostosis and diminution of the joint line), most pronounced at the inferior aspect of the clavicular head. Occasionally calcification is seen in the ligaments.[4]

For the overuse phenomenon in arthrosis an intra-articular injection of triamcinolone acetonide can be given.

Rheumatoid arthritis

Rheumatic conditions may also affect the sternoclavicular joint. This often occurs in ankylosing spondylitis.[5] It gives rise to the same clinical pattern as in a sprain, but local swelling from synovial thickening is present. A progressive ankylosis develops with pronounced limitation of movement in the shoulder girdle. Other sites of rheumatoid arthritis should bring the disorder in mind, although the sternoclavicular joint may be the first joint affected.

Treatment

Local infiltration of triamcinolone acetonide may be helpful.

Septic arthritis

Bacterial agents such as *Staphylococcus aureus*, *Streptococcus* group B and *Brucella* spp. have been isolated in septic arthritis of the sternoclavicular joint.[6,7] Septic arthritis occurs in elderly patients with a deficient immune system, with rheumatoid arthritis and diabetes mellitus, and in drug addicts.[8–10] The patient usually has fever, chills and sweating, and complains of pain and swelling in the sternoclavicular area, sometimes at the base of the neck. In about 20% a local, warm and erythematous mass is present at the joint because of abscess formation.[11–13]

A combination of neck signs, shoulder signs and shoulder girdle signs is found on examination:

- Passive and resisted neck movements are painful as the result of passive and active stretching of the sternocleidomastoid and scaleni muscles.
- Active and passive elevation of the arm are very limited.
- Active and passive shoulder girdle movements are painful and limited. There is warmth, swelling and exquisite tenderness over the sternoclavicular joint.

There is an elevated erythrocyte sedimentation rate and tomography or CT scan are mostly likely to show bony erosions and destructive changes at the medial end of the clavicle and the clavicular aspect of the sternum.

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Treatment

Inpatient treatment is required, the joint being immediately aspirated and antibiotics started. When an abscess is present, surgical drainage is necessary.[14]

Sprain of the acromioclavicular joint

Pain is usually confined to the area of the acromioclavicular joint. Both active and passive elevation of the scapula may be slightly painful but painless movement is not uncommon. The diagnosis becomes clear when the shoulder is examined. Local pain over the acromioclavicular joint is provoked at the end range of the three passive movements of the shoulder examination: passive elevation, lateral rotation and medial rotation (for a detailed description, see p. 241).

Disorders of the first rib

The first rib can become affected in that it becomes sprained at the level of the articulation with the spine or becomes the site of a stress fracture.

Sprain of the first costotransverse joint

The patient complains of unilateral pain at the base of the neck elicited by certain movements of the arm and shoulder. On examination pain is elicited by active and passive side flexion away from the painful side and resisted flexion towards the painful side. Flexion may also be positive.

Such a pattern – in fact a contractile tissue pattern – draws attention to the scaleni muscles. In this case the lesion does not lie in the scaleni but in the structure to which the muscle is attached. Pain is also present on active and passive elevation of the arm and the scapula, resulting in a mixed pattern of neck, shoulder and shoulder girdle signs.

Stress fracture of the first rib

Pain occurs spontaneously and is felt unilaterally at the base of the neck and in the scapulopectoroclavicular area. The clinical picture is typical. There are neck, shoulder girdle and shoulder signs. In the neck a 'contractile tissue pattern' is found: pain on active and passive side flexion towards the painless side, combined with pain on resisted side flexion towards the painful side. All scapular movements are more or less painful and active elevation of the arm is impossible because of the pain: the arm stops at the horizontal. Passive elevation is full but painful. Radiography is confirmative (see also p. 237).

Traction fracture of the spinous process C7 or T1

Following intense exertion the patient feels a sudden pain at the cervicothoracic junction. Neck signs are subtle but arm signs are pronounced: the patient is hardly able to bring both arms actively above the horizontal. Passive arm elevation is painful but not limited. Active and resisted shoulder movements are painful. Radiography shows avulsion of the seventh cervical or first thoracic spinous process.

Lesion of the conoid/trapezoid ligament

Pain is felt in the midclavicular area and is often due to 'smash' movements, such as those which occur in volleyball, baseball and serving in tennis. It is increased at the extremes of all passive movements of the arm but resisted movements remain negative. Forced passive lateral rotation with the arm abducted to 90° is also painful, as are the extremes of all active and passive shoulder movements (see p. 237 and online chapter *Non-mechanical disorders*).

Neural compression

Shoulder elevation is considered one of the dural signs because it stretches or moves the thoracic dura mater via traction on the intercostal nerves. The same applies to the upper thoracic nerve roots.

Compression of the dura mater

Dural mobility may be impaired as the result of compression from a space-occupying lesion in the spinal canal. In most instances it is a discodural interaction caused by a posterocentral thoracic disc protrusion. It gives rise to multisegmentally referred pain, which may increase on all active and passive scapular movements (see online chapter *Applied anatomy of the cervical spine*).

T1–T2 nerve root compression

The mobility of the upper thoracic nerve roots is reduced when a space-occupying lesion in the lateral root recess or in the intervertebral foramen causes compression. Again the disc (a posterolateral protrusion) may be responsible in rare instances but reduction of the foraminal size may also be related to degenerative conditions. The active and passive movements of the shoulder may all have an influence on the symptoms: pain and paraesthesia at the inner aspect of the upper limb. Motor deficit is uncommon and if it occurs – possibly in the intrinsic muscles of the hand – should be regarded as a warning sign. Apical tumour of the lung is most likely (see pp. 156–157).

Pain on active elevation and protraction

Pain on active forwards movement of the shoulder does not occur in isolation. It is usually combined with pain on active elevation. This clinical picture may occur in rather uncommon situations:

- *Lesion of the conoid and/or trapezoid ligament*: this gives rise to pain located in the infraclavicular fossa, provoked by both arm and shoulder movements (see p. 245).
- *Compression of an upper thoracic nerve root (T1/T2)*: a discoradicular interaction at the first or second thoracic

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level gives rise to a partial articular pattern of the thoracic spine. Dural symptoms and signs should be sought, although they are not necessarily present. Any other space-occupying lesion (e.g. a tumour) is also possible and even more probable (see p. 156).

- *Idiopathic contracture of the costocoracoid fascia*: the clinical picture strongly resembles a sprained coracoclavicular ligament. Often arm and shoulder elevation are slightly limited.

Painful limitation of active and passive elevation

Ankylosis of the acromioclavicular joint

Limitation on active and passive elevation of the scapula may be the outcome of complete ankylosis of the acromioclavicular joint as the result of ankylosing spondylitis, arthrosis or rheumatoid arthritis. The arm cannot actively or passively be raised above the horizontal. Examination of the glenohumeral joint reveals normal mobility. Examination of the shoulder girdle shows that scapular elevation is completely limited (see p. 237).

Disorders of the sternoclavicular joint

Ankylosis of the sternoclavicular joint

Ankylosis of the sternoclavicular joint as a result of ankylosing spondylitis, arthrosis or rheumatoid arthritis gives rise to an identical clinical pattern as that of ankylosis of the acromioclavicular joint. However, pain – if present – is at the medial end of the clavicle (see p. 237).

Traumatic dislocation

Traumatic dislocation of the sternoclavicular joint is not common. The medial portion of the clavicle may be displaced anteriorly or posteriorly.

Anterior luxation is by far the more common. It outnumbers posterior dislocations by up to 20 to 1.[15] This is because the posterior capsule and ligaments are stiffer than the anterior structures, so that posterior luxation is more difficult.[16] Some cranial displacement is associated because of the weight of the arm.

Symptoms and signs resemble those of a proximal fracture of the clavicle: the patient supports the painful arm, the shoulder girdle looks 'shortened' and the head is held deviated towards the affected side. The active and passive movements of the shoulder girdle are all extremely painful. The luxation deformity is clearly visible.

Subluxation is usually not inconvenient to the patient.[17] Some people can dislocate the medial portion of the clavicle voluntarily as a 'party trick'. In this event, pain is not felt but an audible click is present. Anterior displacement must be distinguished from synovial swelling, which may take place in rheumatoid arthritis. The history of an injury suggests displacement. Radiography is of little help.

A posterior dislocation may threaten or damage mediastinal structures (Fig. 4).[18–21] It may result in venous congestion of arm and head, as well as dyspnoea and swallowing disturbances.[22]

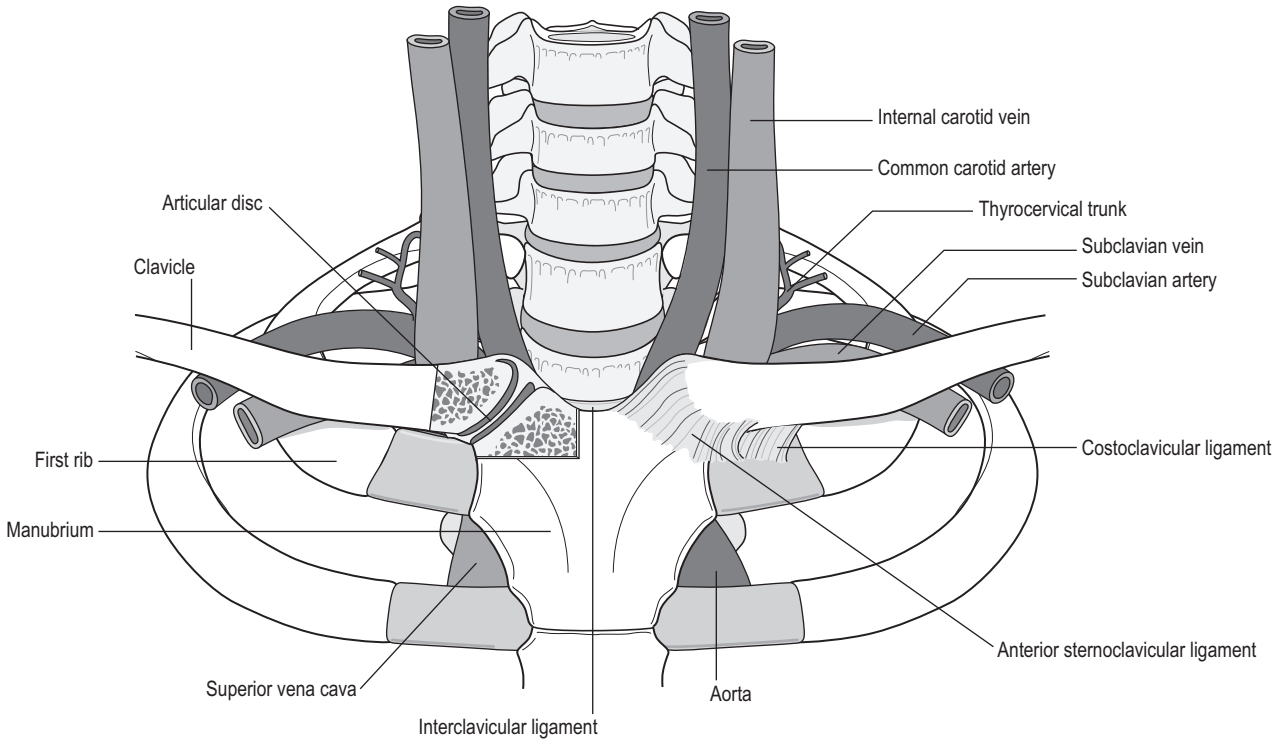

Fig 4 • A posterior dislocation may threaten mediastinal structures.

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Treatment

Luxation of the bone often needs surgical repair[23] and is necessary when neurovascular structures are involved.[24] In subluxation it usually suffices to do a passive retropulsion of the shoulder girdle by immobilization,[25] for example fixating it with a 'figure-of-eight' bandage. Intra-articular triamcinolone may occasionally be added.

Sternoclavicular hyperostosis

This is a rare condition, in which progressive ossification develops in the sternoclavicular joint, the joint between the first rib and the sternum and the sternoclavicular and costoclavicular ligaments. In advanced cases the formation of a massive osseous block between sternum, clavicle and first rib can be seen. It may be associated with seronegative spondyloarthropathy[26] and occasionally with pustulosis palmaris and plantaris. It is then known as SAPHO syndrome (synovitis–acne–pustulosis–hyperostosis–osteitis).[27–29]

Characteristically, the complaints start at a young age, with intermittent pain, local swelling and reddening over the sternum and the sternoclavicular joints. Clinical examination shows a gradual loss of mobility within the shoulder girdle, which may end in complete ankylosis.[30] Active and passive elevation of the shoulder girdle then becomes impossible. Elevation of the arm is grossly limited and shows a forward component, while the scapula remains totally immobile.

Radiology shows a progressive joint destruction in combination with an increasing hyperostosis of the sternoclavicular ligaments.[31] In most cases there is an increase in the erythrocyte sedimentation rate.[32]

Treatment

There is no causative treatment for this condition. Steroid infiltration in the ligaments may give some temporary relief. The results after surgery are poor.

Disorders of the costocoracoid fascia

Contracture of the costocoracoid fascia and/or the pectoralis minor may also limit active and passive shoulder elevation (Fig. 5), (See p. 237):

- *An idiopathic contracture* is a very uncommon cause of limited elevation of the arm.[33] It comes on spontaneously and initially causes unilateral pectoroscapular pain on full elevation of the arm and on active and passive elevation of the shoulder girdle. At a later stage, the pain becomes permanent and a limitation of about 10° on active and passive elevation of arm and shoulder may set in. The

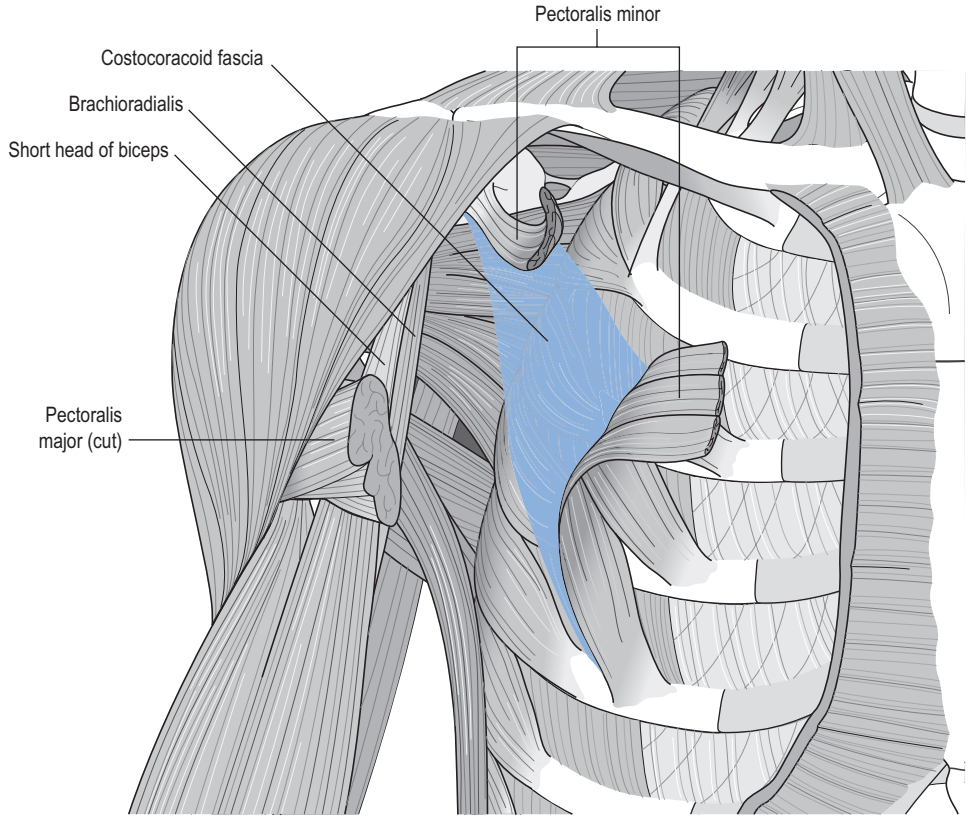

Fig 5 • Costocoracoid fascia.

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limitation of elevation of the arm suggests a psychogenic disorder, but repeated shoulder girdle examination showing an unfailing and isolated limitation in elevation of the shoulder girdle provides the key to the diagnosis. Forward movement of the shoulder (protraction) is painful but of full range, although backward movement (retraction) is normal. No resisted movements hurt. On examination of the neck, slight pain may be found on active flexion to the contralateral side and on resisted side flexion towards the painful side. Diagnosis is made on MRI, where thickening and irregularity in the upper–lateral part of the pectoralis minor can be seen. Surgical treatment – section of the thickened fibrous tissue – provides good and permanent results.

- *Neoplasm*: the previous disorder must be differentiated from an invasion of the costocoracoid fascia by a tumour. This gives rise to the same signs but is more rapid and swifter in its evolution. Other neurological signs (e.g. Horner's syndrome) should be looked for.
- *Healed apical tuberculosis*: this may also limit mobility and elasticity of the costocoracoid fascia, causing the same clinical picture as in idiopathic contracture.
- *After radiation therapy*: similar clinical features may be encountered after radiation therapy for cancer of the lung or breast and after dissection of axillary nodes in breast surgery.[34]
- *Haematoma* between the costocoracoid fascia and the ribs with subsequent traumatic fasciitis may result from injury to the anterior chest wall. This again gives rise to a similar clinical picture: pain and limitation of both active and passive elevation of the shoulder and arm. The radiograph shows no evidence of rib fracture. The disorder undergoes spontaneous cure over about 3 months.

Scapular metastases

Warning

The type and localization of the pain, together with – in its later stage – muscular atrophy and weakness, and a poor general condition, must warn the examiner, and a radiograph must be taken at once.

Scapular metastases are accompanied by unilateral scapular pain, which is increased by all active and passive movements of the shoulder. As the result of excessive pain, active and passive elevation of the shoulder may become limited.

Apical tumour of the lung

Pancoast's tumour is not uncommon. It grows from the upper part of the lung and may involve most structures in that area, such as the brachial plexus, the sympathetic ganglia at the base of the neck, the ribs and the vertebrae. It gives rise to pain felt in the pectoroscapular area which may radiate down the upper limb. It may affect the normal functioning of the thoracic spine, the shoulder girdle and the shoulder. In the shoulder girdle examination, active and passive elevation of the shoulder may be painful and limited. Accompanying symptoms and signs, such as Horner's syndrome or atrophy of the intrinsic muscles of the hand, reveal the disorder (see p. 236).

Limitation of active elevation and weakness of resisted elevation

This presentation may point towards a lesion of the nerve root C2–C4. A posterolateral herniation at these levels is extremely rare.

Warning

A physician who examines a patient complaining of pectoroscapular pain should always bear in mind the possibility of an apical lung tumour and look for unusual symptoms and signs.

Another cause of weakness of the trapezius is a disorder of the spinal accessory nerve (see online chapter *Nerve lesions and entrapment neuropathies of the upper limb*).

Paraesthesia brought on by active and/or passive elevation

If pins and needles occur in fingers or hand during active or passive elevation of the scapula, a thoracic outlet syndrome is very likely (see online chapter *Nerve lesions and entrapment neuropathies of the upper limb*).

Crepitus during scapular elevation

Painless crepitus is sometimes heard and felt on moving the scapula actively up and down.

A crepitating or snapping scapula stems from the disruption of normal mechanics in the scapulothoracic gliding surface.[35] When the patient abducts the scapula by moving the shoulders forwards the noise may stop. This can occur unilaterally or bilaterally and implies that the posterior thoracic wall, just beyond the lateral edge of the iliocostalis muscle, has become rough.[36] It is usually painless and does not require treatment. Exceptionally, scapular pain occurs after exertion or even at rest. It occurs mainly in professions where thoracic hyperkyphosis is maintained for a long period of time (e.g. computer operators and physiotherapists).

Other cases, usually associated with pain, are the result of local injury, an inflamed bursa or an inflammation of other soft tissues in this location.[37] Bursitis is frequently encountered in baseball pitchers.[38] True scapular 'snapping' is the outcome of an osseous projection on the thorax or the anterior wall of the scapula.[39]

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Treatment

Deep friction

In roughening of the posterior thoracic wall, deep friction can be tried. The affected area is outlined by asking the patient to abduct the scapula progressively, meanwhile testing scapular elevation at each degree of abduction. In the position that crepitus ceases, the scapula has been brought so far laterally that it has lost contact with the roughened thorax. The lesion now lies just medial to the medial scapular border. If local tenderness is not found on palpation, the whole upper area around the medial part of the scapula must be treated.

Technique: deep friction to the posterior thoracic wall

The patient lies prone with the shoulder well abducted and the arm in full medial rotation. The therapist stands at the opposite side and gives massage with all fingers in a mediolateral direction for about 20 minutes (Fig. 6); friction is repeated on alternate days. The crepitus will not disappear but the pain usually does, although 20 sessions may be required.

Surgery

If massage fails, the superomedial angle of the scapula can be removed.[40] In some cases the whole vertebral border has been removed.[41]

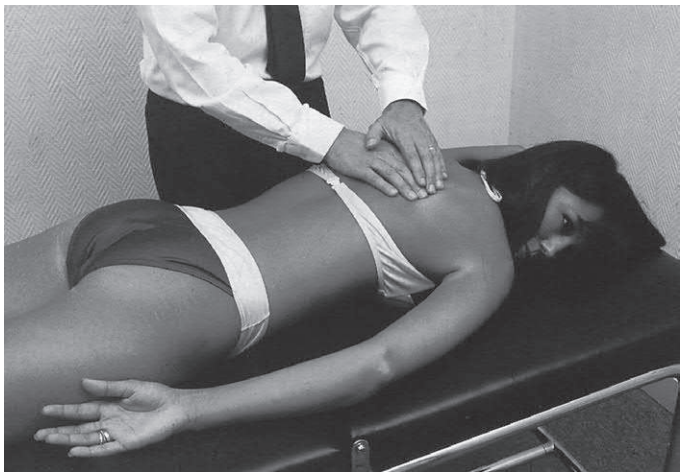

Fig 6 • Deep friction to the posterior thoracic wall.

Pain on active and passive scapular approximation

This presentation draws attention to interference with the dura mater or the dural nerve root sleeve by an upper thoracic disc protrusion or a spinal tumour.

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Disorders of the contractile structures

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Pain on resisted elevation

This, together with pain on active elevation, points to a lesion of one of the elevators of the shoulder – the *levator scapulae* or the *trapezius muscle*. Disorders of these structures are extremely rare.

If the pain is centrally located and is provoked by bilateral elevation of the arm, *traction fracture of the spinous process of C7 and/or T1* is most likely. This presents as a stress fracture of one of the lower cervical spinous processes. The lesion should be suspected when bilateral limitation of about 90° on active elevation of the arm is found. Active elevation of both shoulders is also painful, as is resisted shoulder elevation. Passive elevation is painless. Movements of the neck are only slightly painful (see p. 237).

A *stress fracture of the first rib* is another possible cause. It gives rise to unilateral pain at the base of the neck not preceded by trauma. Active elevation of the arm cannot progress beyond the horizontal and is painful but passive elevation is normal. Pain is increased by active and resisted shoulder elevation and by active and passive side flexion of the neck to the opposite side. Resisted side flexion to the painful side is also painful (see p. 236).

Lesion of the levator scapulae muscle

Pain is felt unilaterally in the upper scapular area, mostly of the dominant shoulder.[1] It is activity-related: neck movements as well as shoulder girdle or arm movements may be of influence. So are prolonged postures such as sitting with the head in slight flexion as happens during computer work.

On examination the pain is elicited during resisted elevation of the shoulder. The neck examination may be slightly positive. Palpation shows the lesion to lie at the insertion on the superior angle of the scapula.

The lesion usually lies at the tenoperiosteal insertion at the superior angle and the medial border of the scapula next to the superior angle and responds equally well to deep transverse friction or to infiltration with 20 mg of triamcinolone.

Lesion of the trapezius muscle

A muscular lesion in the trapezius is as uncommon as pain in the trapezius is common. Usually pain in that area is referred from the cervical spine: it is the most common localization of cervical dural pain (see p. 123). Because this phenomenon is often accompanied by extrasegmental tenderness, examination based on palpation will easily result in the wrong diagnosis of 'trapezius syndrome'. Proper functional testing, however, usually turns out to be negative, showing that no focal lesion is present.

Muscular lesions do occur, however, but are very uncommon. They give rise to rather localized pain which can be provoked by testing against resistance: resisted elevation of the shoulder is painful, together with resisted neck extension and/or side flexion.

The lesion responds to local infiltration of a local anaesthetic or to deep friction.

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Pain on resisted protraction

When the pain is felt anteriorly, this suggests that the lesion lies in the pectoralis minor or major muscle. Active protraction usually also elicits pain. When the *pectoralis minor* is at fault, resisted scapular depression is painful. If the lesion lies in the *pectoralis major*, resisted adduction and medial rotation of the arm is positive. Both lesions can be treated by procaine infiltrations or deep friction (see p. 260).

If the pain is located in the scapular area a lesion of the *serratus anterior* is probable.

Lesion of the serratus anterior muscle

On arm movement, the serratus anterior keeps the vertebral border of the scapula firmly attached to the thorax. In a lesion at its scapular insertion, unilateral scapular pain is felt on moving the arm. Because the pain is felt at the inner side of the scapula, the attention of the examiner is drawn to one or other structure in this area. Therefore it is discussed here, although it is likely to be encountered in the clinical examination of the shoulder.

Pain is felt on full active elevation of the arm, whereas passive elevation remains painless. Resisted abduction of the arm does not hurt. In this event, the patient is asked to bring the arm horizontal in front of the body. Resisted horizontal abduction is now performed in this position and elicits pain (Fig. 1).

Treatment is by deep transverse friction.

Technique: deep friction to the serratus anterior

The patient lies prone, the ipsilateral arm in full medial rotation, the elbow flexed to 90°, the hand lying on the back. The therapist stands at the side and brings the arm further into medial rotation by using the forearm which lifts the medial edge of the scapula off the thorax. The thumb is now placed at the anterior aspect of the medial scapular margin. The other fingers are put on the posterior aspect of the scapula and provide counterpressure (Fig. 2). Massage is given along the scapular border by adduction–abduction movements of the arm, performed in a craniocaudal direction, for about 20 minutes, three times a week. Full cure is normally achieved in 3–5 weeks.

Pain on resisted approximation

If resisted approximation is painful but pain is not elicited on passive approximation, the lesion must lie in the *rhomboid* or in the *trapezius*. The response to infiltrations with procaine is good.

Weakness of scapular approximation

This is the result of neuritis of the spinal accessory nerve. Unilateral resisted approximation of the scapula is weak, together with a loss of about 5° of active elevation of the arm (see p. 524).

Fig 1 • Resisted horizontal abduction.

Fig 2 • Deep friction to the serratus anterior.

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Pain on resisted depression

A positive result suggests that there is a problem in the subclavius, the pectoralis minor or the latissimus dorsi and is usually combined with pain on active and passive elevation, which demonstrates that one of the shoulder depressors is involved. A sprain of the *subclavius* is mostly likely. The patient complains of pain in the anterior claviculopectoral area. Alternatively, a sprain of the *pectoralis minor* may be responsible. Pain on resisted forward movement of the shoulder then establishes the diagnosis. When the *latissimus dorsi* is at fault, resisted adduction and medial rotation of the arm is also painful.

Lesion of the subclavius muscle

The patient complains of pain around the clavicle or in the upper pectoral area on certain activities or movements. Examination of the shoulder is negative which excludes the pectoralis major and the structures originating at the coracoid process (short head of biceps or coracobrachialis). Shoulder girdle examination shows the 'contractile tissue pattern': active and passive shoulder elevation is positive, as is resisted shoulder depression. On palpation a tender spot is found in the muscle belly of the subclavius.

Treatment of the subclavius is by deep transverse friction. The lesion usually does not respond to infiltration with procaine.

Technique: deep friction to the subclavius

The patient lies supine, the arm raised with the hand on the head. The therapist stands at the ipsilateral side and puts the middle finger, reinforced by the index, just below and parallel to the clavicle on the painful spot (Fig. 3). Friction is given by pronation–supination movements of the arm. This is performed for about 20 minutes on alternate days. The patient is usually cured in 10 sessions.

A summary of shoulder girdle disorders is outlined in Box 1.

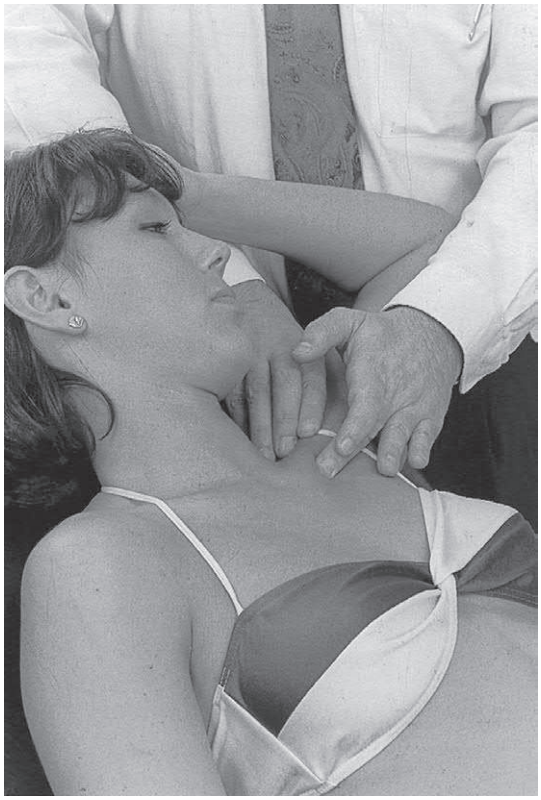

Fig 3 • Deep friction to the subclavius.

Box 1

Summary of shoulder girdle disorders

- Disorders of the inert structures
  - Pain on active elevation
    - Combined with pain on passive elevation
      - Disorders of the sternoclavicular joint:
        - Sprain of the sternoclavicular joint/ligaments
        - Posterior sternoclavicular syndrome
        - Arthrosis
        - Rheumatoid arthritis
        - Septic arthritis
      - Sprain of the acromioclavicular joint
      - Disorders of the first rib:
        - Sprain of the first costotransverse joint
        - Stress fracture of the first rib
      - Traction fracture of the spinous process C7 or T1
      - Lesion of the conoid/trapezoid ligament
      - Idiopathic contracture of the costocoracoid fascia
      - Neural compression:
        - Compression of the dura mater
        - T1–T2 nerve root compression
    - Combined with pain on active protraction
      - Lesion of the conoid/trapezoid ligament
      - Compression of T1–T2 nerve root
      - Idiopathic contracture of the costocoracoid fascia
  - Pain and limitation of active and passive elevation
    - Ankylosis of the acromioclavicular joint
    - Disorders of the sternoclavicular joint:
      - Ankylosis
      - Traumatic dislocation
      - Hyperostosis
    - Disorders of the costocoracoid fascia
      - Idiopathic contracture
      - Neoplasm
      - Healed apical tuberculosis
      - Post-radiation therapy
      - Haematoma
    - Scapular metastases
    - Apical tumour of the lung

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Box 1

Summary of shoulder girdle disorders—cont'd

Limitation of active elevation and weakness of resisted elevation
- Lesion of the C2–C4 nerve roots
- Neuritis of the spinal accessory nerve

Paraesthesia brought on by active and/or passive elevation
- Thoracic outlet syndrome

Crepitus during scapular elevation
- Disturbance in the scapulothoracic gliding mechanism

Pain on active and passive scapular approximation
- Involvement of upper thoracic dura mater or dural nerve root sleeve

- Disorders of the contractile structures

Pain on resisted elevation
- Lesion of the levator scapulae or trapezius muscle
- Traction fracture of the spinous process of C7 and/or T1
- Stress fracture of the first rib

Pain on resisted protraction
- Lesion of the pectoralis minor or pectoralis major muscle
- Lesion of the serratus anterior muscle

Pain on resisted approximation
- Lesion of the rhomboid muscles
- Lesion of the trapezius muscle

Weakness of scapular approximation
- Neuritis of the spinal accessory nerve

Pain on resisted depression
- Lesion of the subclavius muscle
- Lesion of the pectoralis minor muscle
- Lesion of the latissimus dorsi muscle

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Applied anatomy of the elbow

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Joints and ligaments

The elbow joint helps the hand to approach or move away from the body, whatever the arm position resulting from shoulder movement. It consists of three 'joints', which lie within one joint capsule and together have two separate functions.

Joints

The three 'joints' are the humeroulnar, the humeroradial and upper radioulnar joint (Figs 1–3, see Putz, Fig. 307). Their two functions are flexion/extension, which is performed at the humeroulnar and humeroradial joints, and pronation/supination, which takes place at the upper radioulnar joint in close association with the lower radioulnar joint.

The three joints work closely together and make pronation and supination movements possible whatever the extent of flexion or extension of the elbow:

- The humeroulnar joint acts as a hinge. The articular surfaces on the humerus are the spool-shaped trochlea with, proximal to it, the coronoid fossa (ventral aspect) (see Gosling et al, Fig. 3.89) and the olecranon fossa (dorsal aspect) (see Gosling et al, Fig. 3.90). The other part of the joint is formed by the olecranon, with its trochlear notch and its olecranon (proximal) and coronoid (distal) processes.
- The humeroradial joint – a ball-and-socket joint – consists of (a) the spheroidal capitulum of the humerus and (b) the proximal surface of the head of the radius. Proximal to the capitulum lies the radial fossa and beside it a capitulotrochlear sulcus (between capitulum and trochlea). The articular facet of the radius, with which the proximal part of the humeroradial joint articulates, is at the top of the head of the radius. This facet exactly follows the shape of the humeral capitulum. Because it articulates with the capitulotrochlear sulcus at the ulnar side, it allows pronation–supination movements as well.
- The upper radioulnar joint is a trochoid (cone-shaped) joint. The circumference of the head of the radius articulates with the radial notch of the ulna.

The three joints lie within the same lax joint capsule, which is spanned by muscular fibres of the brachialis, triceps and anconeus muscles.

At the distal end of the humerus lie the two epicondyles, of which the medial one is more developed than the other. They are both extracapsular.

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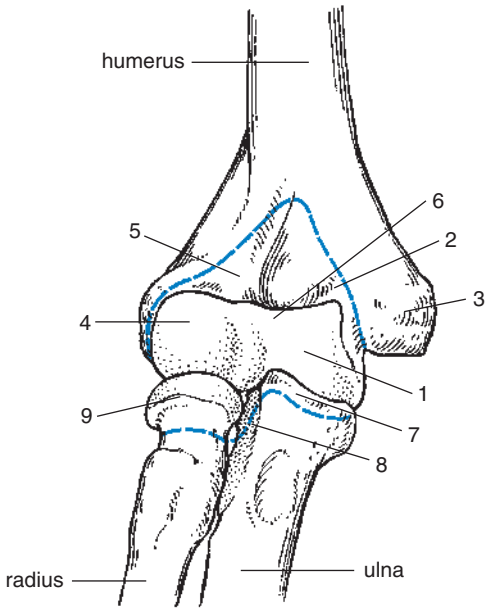

Fig 1 • Humeroulnar and humeroradial joints (anterior view). The dotted line shows the insertion of the joint capsule. On humerus: 1, trochlea; 2, coronoid fossa; 3, medial epicondyl; 4, capitulum; 5, radial fossa; 6, capitulotrochlear sulcus. On ulna: 7, coronoid process; 8, radial notch. On radius: 9, radial head.

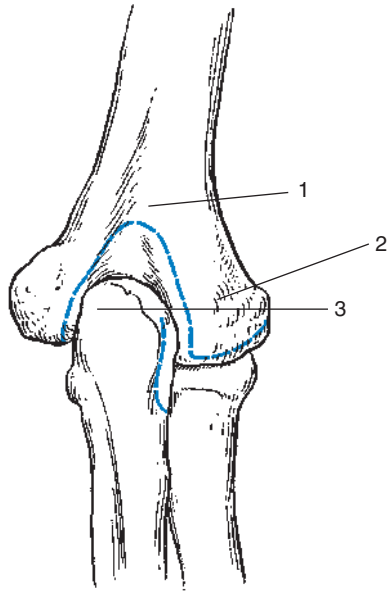

Fig 2 • Humeroulnar and humeroradial joints (posterior view). The dotted line shows the insertion of the joint capsule. On humerus: 1, olecranon fossa; 2, lateral epicondyle. On ulna: 3, olecranon process.

Ligaments

The capsule is reinforced by strong lateral ligaments (Fig. 4):

- *The medial collateral ligament* has an anterior part, which runs from the medial epicondyle of the humerus towards the annular ligament, a middle part towards the coronoid process, and a posterior part, directed towards the olecranon. All three parts are reinforced by an oblique band (ligament of Cooper) distally on the ulna.
- *The lateral collateral ligament* connects the lateral epicondyle to the radial annular ligament (anteriorly via the radial collateral ligament and posteriorly via the lateral ulnar collateral ligament which inserts at the supinator crest of the ulna) and is interwoven with the superficial extensor muscles.

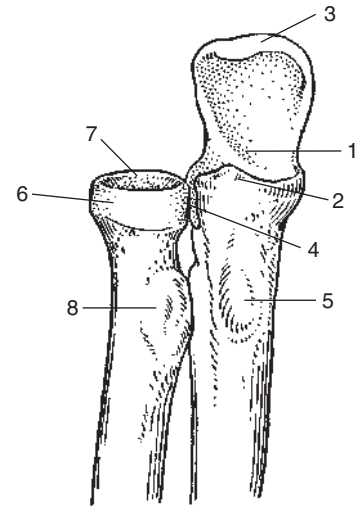

Fig 3 • Upper radioulnar joint. On ulna: 1, trochlear notch; 2, coronoid process; 3, olecranon process; 4, radial notch; 5, ulnar tuberosity. On radius: 6, circumference; 7, articular facet; 8, radial tuberosity.

These ligaments help to maintain the articular surfaces in contact with each other. They also limit lateral movements. Other ligamentous structures are:

- *The radial annular ligament*, a U-shaped fibrous collar, covered with cartilage at its inner aspect, joins the radial head to the proximal ulnar extremity so that they can articulate. It plays an important role in pronation–supination (see Gosling et al, Fig. 3.93).
- *The quadrate ligament* attaches the radial neck to the distal aspect of the radial notch of the ulna.
- *The oblique cord* extends inferolaterally from the lateral border of the tuberosity of the ulna to the radius, just below its tuberosity (see Putz, Fig. 315). It prevents downwards movement of the radius.
- *The interosseous membrane* (see Putz, Fig. 315) and the oblique cord join the two bones of the forearm to prevent any longitudinal movement and assist as a fulcrum in pronation–supination (i.e. diagonal rotation). They are both syndesmosis connections.

Biomechanical aspects

The distal extremity of the humerus is a fork, the axis of its articular surfaces running through the humeral capitulum and trochlea.

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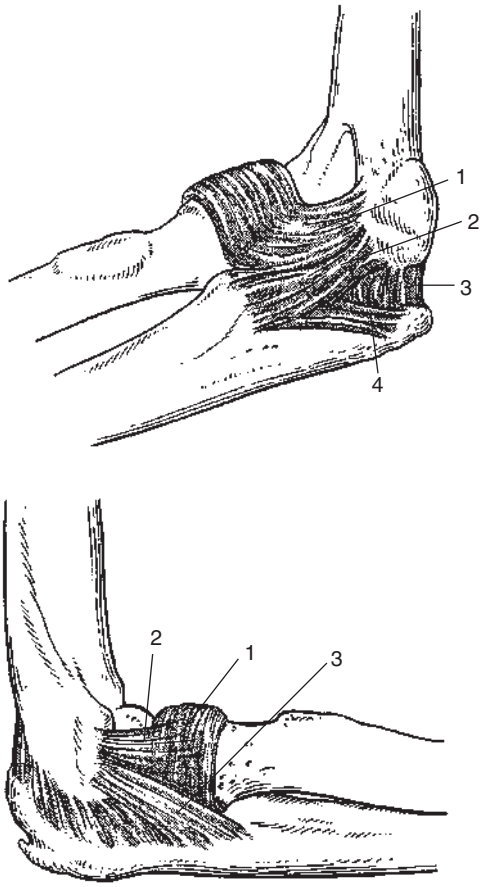

Fig 4 • The medial (upper) and radial (lower) collateral ligaments. Upper: 1, anterior part; 2, middle part; 3, posterior part; 4, ligament of Cooper. Lower: 1, annular ligament; 2, radial collateral ligament; 3, lateral ulnar collateral ligament.

The coronoid fossa, into which the coronoid process of the ulna fits, as is the case for the olecranon fossa and olecranon process, together with the forward projection of both the lower humeral and the upper ulnar extremities, allows a considerable range of flexion–extension between forearm and upper arm.

Flexion–extension

The trochlear surface of the humerus articulates with the trochlear notch of the ulna along an imaginary line A–A' which runs slightly oblique (downwards and outwards) in its posterior course. This obliquity explains the physiological valgus position of the elbow in extension (Fig. 5). In its anterior course, the axis of movement is horizontal: therefore, in flexion, the forearm lies exactly in front of the upper arm. There are, of course, individual differences.

Extension is mainly limited by the olecranon abutting against the posterior aspect of the humerus in the olecranon fossa (Fig. 6) and by stretching of the anterior part of the joint capsule. The forearm then usually lies in line with the upper arm, except in most women and in some men, in whom the elbow may sometimes overextend. This overextension is the result of the format of the olecranon fossa and process. The end-feel of this movement is hard.

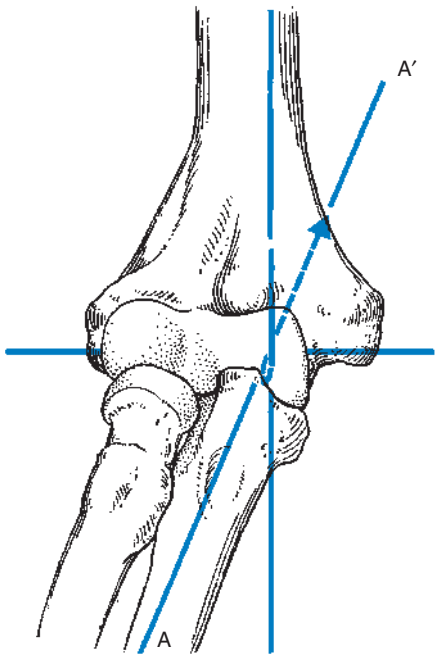

Fig 5 • The physiological valgus position. Oblique line is A–A' (see text).

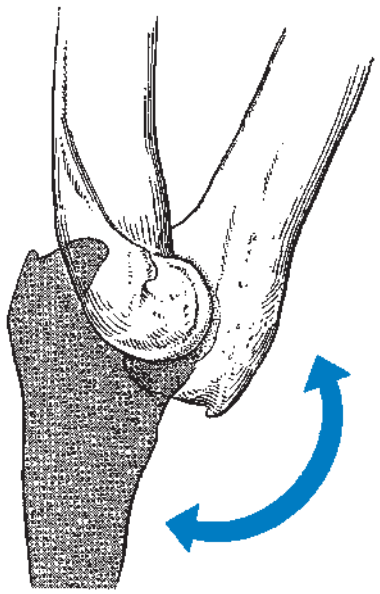

Fig 6 • The range of flexion–extension movement.

Flexion stops when, at the end of the range, the muscular masses of the upper arm and forearm come into contact with each other. The end-feel is, of course, hard in those cases in which the muscles are very poorly developed, because the movement is then stopped by the other limiting factors: (1) the contact between (a) the coronoid fossa of the humerus and the coronoid process of the ulna and (b) the head of the

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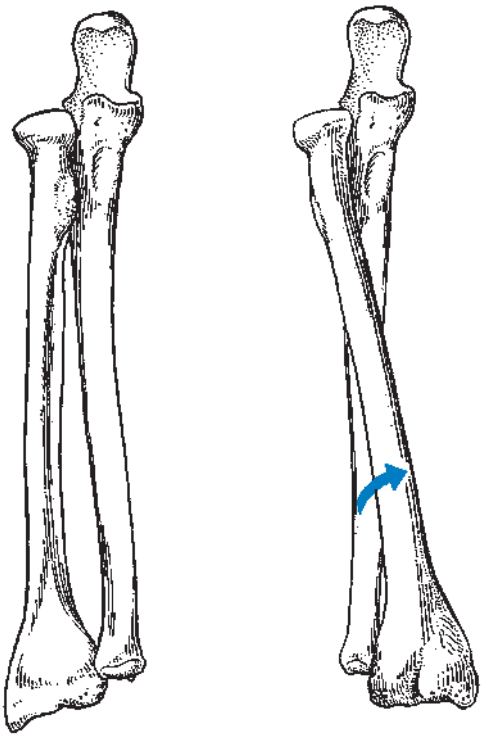

Fig 7 • Supination–pronation.

radius and the radial fossa of the humerus, as well as (2) the tension in the posterior capsule.

Pronation–supination

Pronation–supination takes place mainly in the joint formed by the radial head and the osteofibrous ring, which contains the radial notch of the ulna together with the inner aspect of the annular ligament. However, there is also movement between (a) the head of the radius and (b) the capitulum of the humerus and the capitulotrochlear sulcus.

Pronation–supination is the result of a combined action of the proximal and distal radioulnar joints, which are coaxial. Movement takes place around the longitudinal axis of the forearm, which runs from the facet on the head of the radius to the styloid process of the ulna.

Both movements can best be measured with the elbow held at a 90° angle in order to prevent compensatory rotations of the upper arm. Starting from the intermediate position with the hand vertical and the thumb pointing upwards, the supination has a range of 90°. In full supination the two bones lie parallel to each other. The limiting factor is purely ligamentous (interosseous membrane, oblique cord and anterior ligament of the distal radioulnar joint) and explains the elastic end-feel. The oblique cord prevents a downwards shift of the radius.

During pronation the radius crosses over the ulna (Fig. 7). This is possible only because of the direction of the fibres of the interosseous membrane. The radial tuberosity thereby turns towards the ulna. This movement has an 85° amplitude and is limited by stretching of the interosseous membrane and squeezing of the flexor muscles, again with an elastic end-feel.

Table 1 The flexor muscles

	Innervation	
Muscle	Peripheral	Nerve root
Brachialis	N. musculocutaneus (N. radialis)	C5–C6
Brachioradialis	N. radialis	C5–C6
Brachial biceps	N. musculocutaneus	C5–C6

In full pronation, the concave anterior side of the radius lies posteriorly.

Muscles and tendons

Flexor muscles

The flexor muscles are the brachialis, the brachioradialis and the brachial biceps (Table 1). Their action is maximal when the elbow is flexed at 90°.

Brachialis

This runs from the anterior and distal aspect of the humerus towards the ulnar tuberosity and the joint capsule (Fig. 8). It is a monoarticular muscle. Its only function is to bend the elbow, irrespective of the degree of pronation–supination of the forearm.

Brachioradialis

This muscle has its origin at the lateral supracondylar ridge of the humerus and inserts at the radial aspect of the styloid process of the radius (see Putz, Fig. 351). It brings the pronated or supinated forearm back in the neutral position between pronation and supination. In this position it acts as a flexor of the elbow, an action which diminishes when the forearm is held in supination.

Biceps

This is the dominant flexor of the elbow. It originates from the scapula, where it has two heads, the long head from the supraglenoid tubercle, the short head from the coracoid process, and it inserts with a strong thick tendon at the radial tuberosity (see Putz, Fig. 308). Part of the tendon blends into the aponeurosis of the forearm at the ulnar side (Fig. 9). Because the biceps is a biarticular muscle, it also acts on the shoulder. Its essential function is elbow flexion but its secondary function is supination of the forearm. Such supination action of the biceps increases the more the elbow is flexed and is maximal at 90°; it diminishes again when the elbow is fully flexed.

Extensor muscles

Extension of the elbow is performed by two muscles: the triceps and the anconeus (Table 2).

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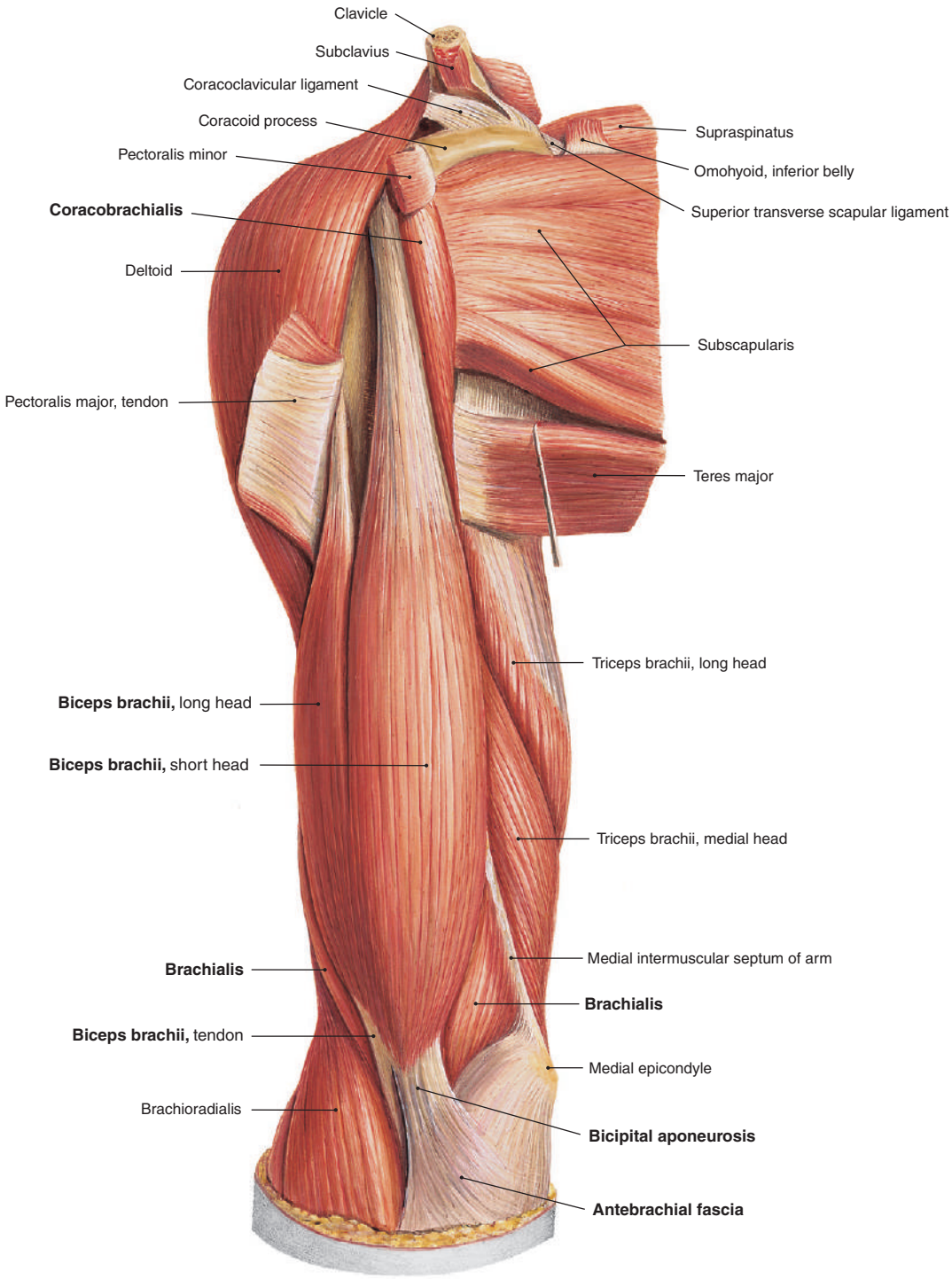

Fig 8 • Flexor muscles of the elbow: brachialis, biceps brachii and brachioradialis. From Putz, Sobotta – Atlas of Human Anatomy, 14th edn. Urban & Fischer/Elsevier, Munich, 2008 with permission.

The triceps

The triceps originates from three heads as its name implies: the long head from the infraglenoid tubercle of the scapula, the medial head from the dorsal aspect of the humerus, distally to the sulcus for the radial nerve, and the lateral head also from the dorsal aspect of the humerus, proximally to that sulcus. The three muscular bodies join in one tendon that inserts at the olecranon and at the posterior aspect of the joint capsule (Fig. 9).

Because the triceps is partly biarticular, its extension action to the elbow joint depends not only on the position of the elbow but also on the position of the shoulder. The triceps has its maximal force in a movement which combines the two: elbow extension and shoulder extension.

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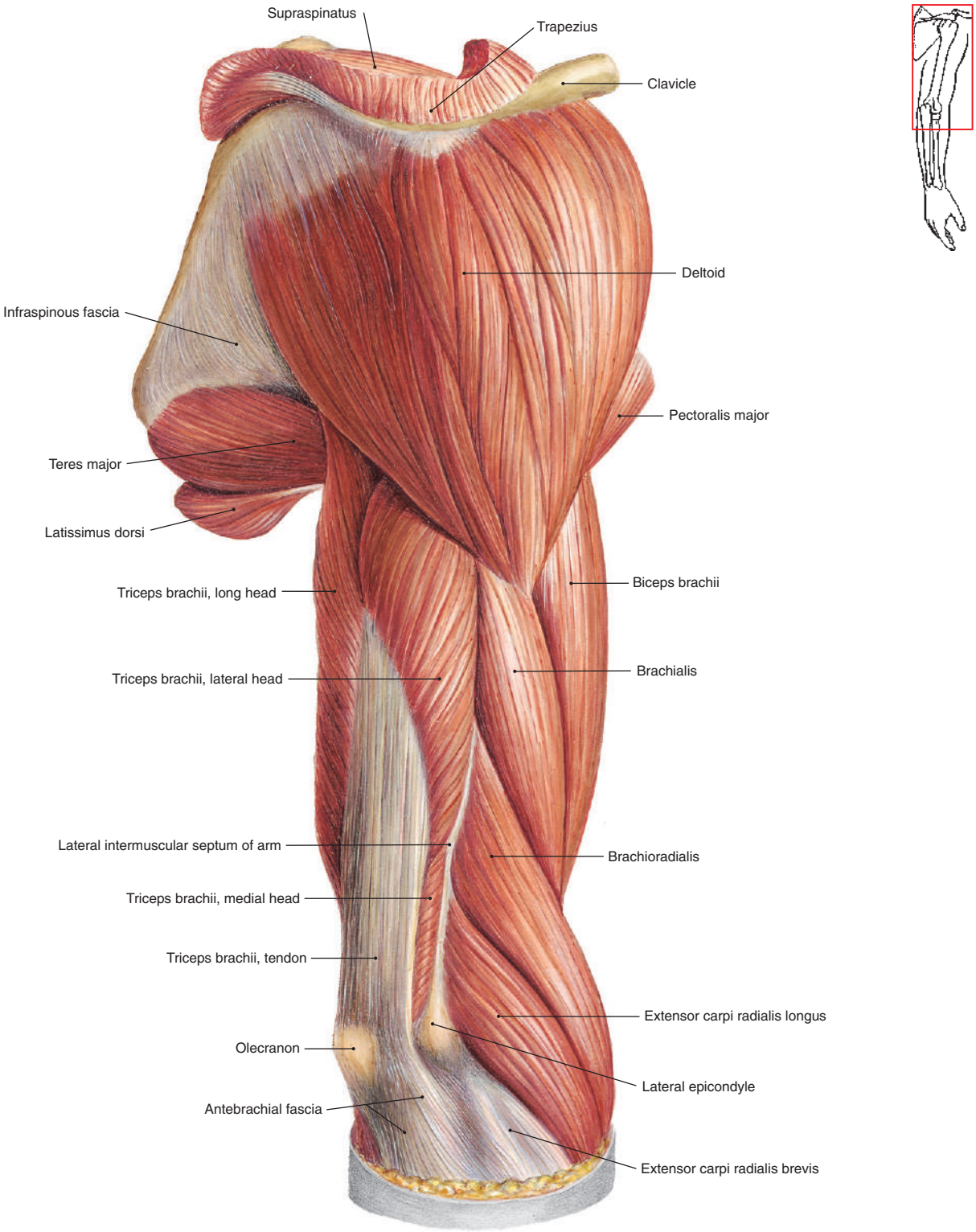

Fig 9 • The triceps: the extensor muscle of the elbow. From Putz, Sobotta – Atlas of Human Anatomy, 14th edn. Urban & Fischer/Elsevier, Munich, 2008 with permission.

Anconeus

Originating at the dorsal aspect of the lateral epicondyle of the humerus and at the lateral collateral ligament, this muscle runs towards the posterior and proximal aspect of the ulna and the joint capsule. It may be considered as a continuation of the lateral head of the triceps. It thus helps the triceps in its extensor function and also spans the joint capsule. From the clinical point of view this muscle may be ignored.

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Table 2 The extensor muscles

Muscle	Innervation	
	Peripheral	Nerve root
Triceps	N. radialis	C7–C8
Anconeus	N. radialis	C7–C8

Table 3 The supinator muscles

Muscle	Innervation	
	Peripheral	Nerve root
Supinator brevis	N. radialis	C5–C6
Brachial biceps	N. musculocutaneus	C5–C6

Table 4 The pronator muscles

Muscle	Innervation	
	Peripheral	Nerve root
Pronator teres	N. medianus	C6–C7
Pronator quadratus	N. interosseus palmaris (N. medianus)	C8–T1

Table 5 The extensor muscles of the wrist and fingers

Muscle	Innervation	
	Peripheral	Nerve root
Extensor carpi radialis longus	N. radialis	C6–C7
Extensor carpi radialis brevis	N. radialis	C7
Extensor carpi ulnaris	N. radialis	C7–C8
Extensor digitorum communis	N. radialis	C6–C8

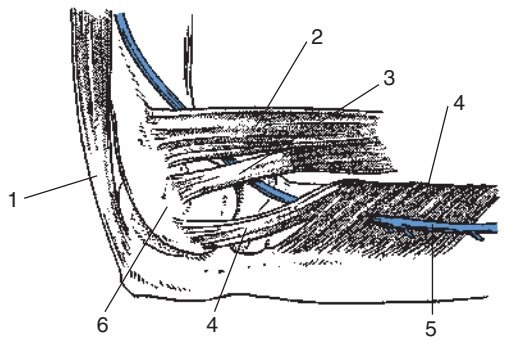

Fig 10 • Lateral view of the extensors of the elbow: 1, triceps muscle; 2, extensor carpi radialis longus muscle; 3, extensor carpi radialis brevis muscle; 4, supinator muscle; 5, radial nerve; 6, lateral epicondyle.

Supinator muscles

Supination movement is the result of the action of two muscles: the supinator brevis and brachial biceps (Table 3).

Supinator brevis

This takes origin at the supinator crest of the ulna, the lateral epicondyle, the radial collateralligament and the annular ligament. The muscular fibres encircle the radius and insert via a short tendon at the radius between the radial tuberosity and the insertion of the pronator teres muscle (see Putz, Fig. 359). The muscle supinates the forearm in whatever position there is between flexion and extension of the elbow.

Brachial biceps

This is the strongest supinator of the forearm (see above).

Pronator muscles

Pronation is performed by two muscles: the pronator quadratus and the pronator teres (Table 4).

Pronator quadratus

This is at the distal aspect of the forearm. It runs from the distal and anterior part of the ulna to the distal and anterior part of the radius, so encircling the ulna. Its role is dealt with in Section 6.

Pronator teres

The pronator teres has a multiple origin: the humeral head from the medial epicondyle of the humerus (partly from the common flexor tendon) and the ulnar head from the coronoid process of the ulna. Its insertion lies halfway down the radius at the lateral aspect (see Putz, Fig. 352). Because the pronator teres is a biarticular muscle, theoretically it also assists in the flexion of the elbow but this action is of course very secondary.

The pronator muscles are not as strong as the supinator muscles.

Muscles controlling wrist movement

Two groups of muscles originate at the elbow but control wrist movements: they are the flexors and extensors of the wrist (and fingers).

Extensors of the wrist and fingers (Table 5, see Standring, Fig. 49.14, see Gosling et al, Fig. 3.70)

Extensor carpi radialis longus

This takes its origin at the lateral supracondylar margin of the humerus, just proximal to the lateral epicondyle and at the lateral intermuscular septum (Fig. 10).

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Table 6 The flexor muscles of the wrist and fingers

Muscle	Innervation	
	Peripheral	Nerve root
Flexor carpi radialis	N. medianus	C6–C7
Flexor carpi ulnaris	N. ulnaris	C7–C8
Palmaris longus	N. medianus	C7–T1
Superficial flexor digitorum	N. medianus	C7–T1
Flexor pollicis longus	N. medianus	C7–C8

Extensor carpi radialis brevis

The origin is at the anterior aspect of the lateral epicondyle but the muscle also partly originates from the lateral collateral ligament and from the annular ligament of the radius.

Both muscles run together towards the wrist. They cause extension of the wrist (especially the extensor carpi radialis brevis muscle) as well as radial deviation.

Extensor carpi ulnaris

Running from the dorsal and lateral aspects of the lateral epicondyle, as well as from the radial collateral ligament and the fascia of the forearm towards the wrist, this assists slightly in the extension of the wrist but its main function is ulnar deviation.

Extensor digitorum communis

The origin (together with the extensor carpi ulnaris muscle) is just dorsal to the extensor carpi radialis brevis muscle from the lateral epicondyle, the radial collateral ligament, the annular ligament of the radius and the fascia of the forearm; the muscle runs towards the fingers. Its main function is finger extension but it also assists in extension (and ulnar deviation) of the wrist.

Flexors of wrist and fingers (Table 6, see Standring, Fig. 49.12, see Gosling et al, Fig. 3.72)

The flexors of the wrist and fingers take their origin at the medial epicondyle of the humerus (Fig. 11), mainly in a common flexor tendon which has a superficial and a deep layer. The superficial layer consists of the humeral head of the pronator teres muscle, the flexor carpi radialis, the palmaris longus, the humero-ulnar head of the superficial flexor digitorum and the humeral head of the flexor carpi ulnaris.

Flexor carpi radialis

Starting at the anterior aspect of the medial epicondyl, this muscle runs towards the wrist. Its main function is flexion of the wrist; its radial deviation function is secondary.

Flexor carpi ulnaris

Starting with a humeral head at the medial epicondyle, this also has an ulnar head at the olecranon and the upper part of the posterior margin of the ulna. It flexes the wrist and assists the extensor carpi ulnaris in performing ulnar deviation of the wrist.

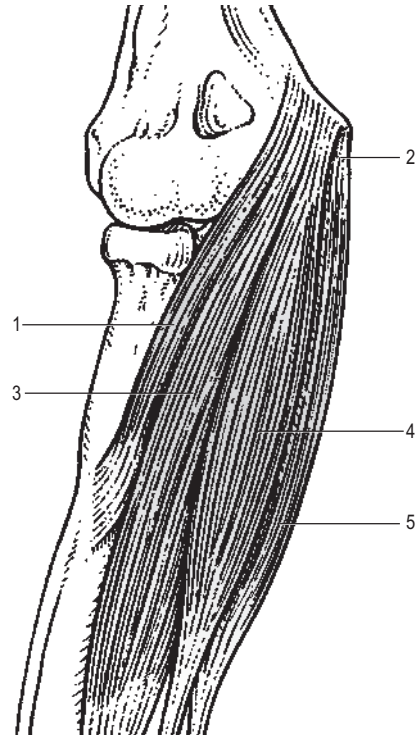

Fig 11 • Muscles originating at the medial epicondyle (anterior aspect): 1, pronator teres muscle; 2, common flexor tendon; 3, flexor carpi radialis muscle; 4, palmaris longus muscle; 5, flexor carpi ulnaris muscle.

Palmaris longus

This is not always present. When it is, it runs towards the hand into the palmar aponeurosis, of which it is a tensor. It also helps in flexion of the wrist.

Superficial flexor digitorum

The origin is threefold: a humeral head originating from the common flexor tendon at the medial epicondyle of the humerus, an ulnar head from the coronoid process and a radial head from the anterior aspect of the radius. Apart from flexion of the fingers, it also assists in ulnar deviation of the wrist.

Flexor pollicis longus

This lies in the deeper layer. It has a humeral head, originating from the medial epicondyle and, more important, a radial head originating from the anterior aspect of the radius, just distal to the supinator muscle and the interosseous membrane. It flexes the thumb and assists in flexion of the wrist.

Nerves

The nervous structures are discussed here because entrapment may occur in the elbow region. Only the appropriate part of

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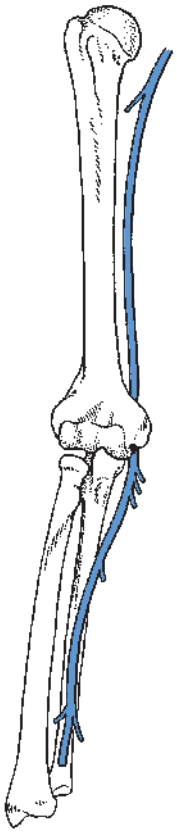

Fig 12 • The course of the ulnar nerve.

the nerve is described. For a more extensive description, see online chapter *Nerve lesions and entrapment neuropathies of the upper limb*.

Ulnar nerve

The ulnar nerve courses under the medial head of the triceps muscle towards the posterior aspect of the medial epicondyle, where it lies superficially in a shallow groove behind this bone (sulcus for the ulnar nerve; Fig. 12) and is, therefore, very vulnerable to direct contusion. It becomes stretched during flexion of the elbow. The nerve runs further distally in between the two heads of the flexor carpi ulnaris muscle, which form an aponeurotic arch. Together with the medial epicondyle, the olecranon and the medial collateral ligament, this arch forms the 'cubital tunnel' (Fig. 13, see Putz, Fig. 410).

Median nerve

At the elbow, the median nerve is of lesser clinical significance, because it is seldom affected at this level. In the upper arm, the nerve courses between the radial and the ulnar nerves. It passes through the antecubital fossa just medially to the bicipital tendon and beneath its aponeurosis. It lies between the two heads of the pronator teres muscle and runs further under a tendinous arch connecting the heads of the superficial flexor digitorum muscle (Fig. 14, see Putz, Fig. 409).

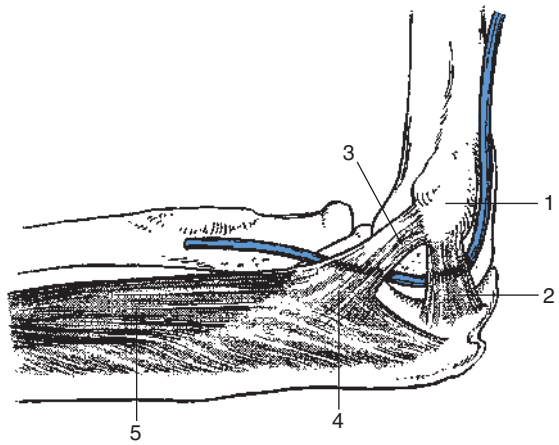

Fig 13 • The course of the ulnar nerve through the cubital tunnel: 1, medial epicondyle; 2, medial collateral ligament (posterior part); 3, common flexor tendon; 4, aponeurotic arch; 5, flexor carpi ulnaris muscle.

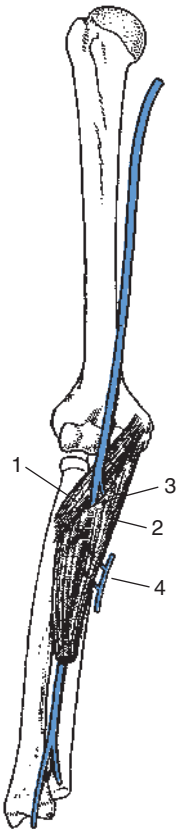

Fig 14 • The course of the median nerve: 1, pronator teres muscle; 2, superficial flexor digitorum muscle; 3, tendinous arch; 4, Martin–Grüber anastomosis with the ulnar nerve.

The median nerve is liable to anomalies consisting of either communications with other nerves or unusual branching. The commonest anomaly is the Martin–Grüber anastomosis: one or more branches from the median nerve pass to the ulnar nerve in the forearm. The practical consequence of this is that a

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median nerve palsy may involve the intrinsic hand muscles and that ulnar nerve disorders may spare the muscles usually innervated by it.

Medial cutaneous nerve of the forearm

This nerve becomes superficial at mid-arm as it runs through an opening in the brachial fascia, the basilic hiatus, and supplies the skin from the inner aspect of the elbow as far as the wrist. In the elbow region, it crosses over the median basilic vein and lies in a position where it is subject to injury by an accidental extravenous injection.

Radial nerve

In the distal part of the upper arm, the radial nerve lies lateral to the median nerve. It courses between the bicipital tendon and the proximal part of the brachioradialis muscle.

Level with the head of the radius the nerve then divides into a deep motor branch (the deep radial nerve), from which the posterior interosseous nerve branches off a bit further down, and a superficial sensory branch (the superficial radial nerve) (Fig. 15, see Putz, Fig. 411).

The posterior interosseous nerve turns around the neck of the radius and comes to lie at the dorsal aspect of the forearm where it passes through an opening in the supinator muscle: the arcade of Fröhse, formed by the edge of the upper border of the superficial part of this muscle. At this location (the

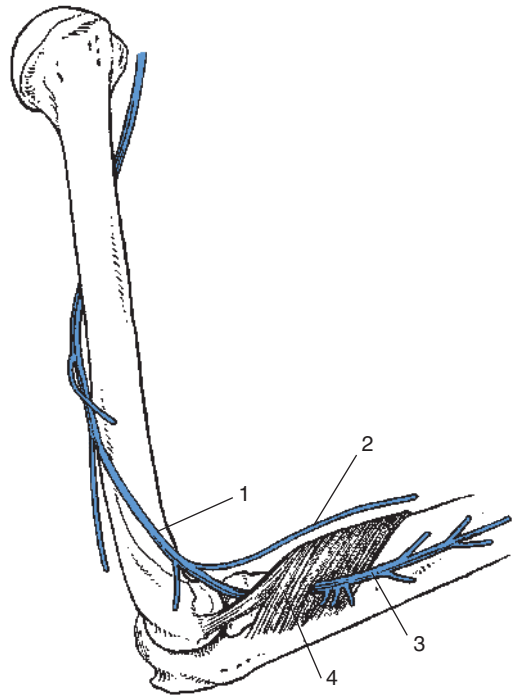

Fig 15 • The course of the radial nerve: 1, radial nerve; 2, superficial radial nerve (sensory); 3, posterior interosseous nerve (motor); 4, arcade of Fröhse in the supinator muscle (forming the 'radial tunnel').

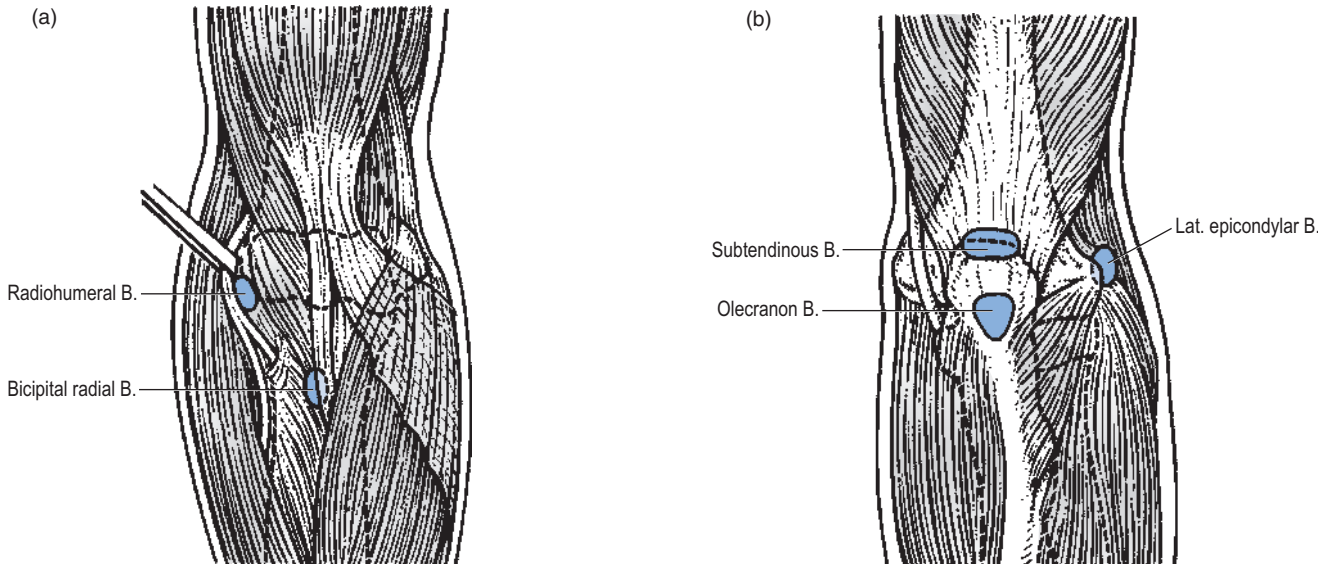

Fig 16 • (a and b) Bursae of the elbow.

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'radial tunnel') it may sometimes be compressed and cause radial tunnel syndrome (see online chapter *Nerve lesions and entrapment neuropathies of the upper limb*).

Bursae

Bursae are regularly found: in the region of the insertion of the triceps into the olecranon (subcutaneous olecranon bursa and subtendinous tricipital bursa); under the biceps tendon at its attachment to the radial tuberosity; and at the lateral aspect of the elbow, where both superficial epicondylar and radiohumeral bursae occur (Fig. 16).

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Applied anatomy of the wrist, thumb and hand

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The anatomy of wrist, thumb and hand is complex because of the presence of many different functional joints: the distal radioulnar joint, the wrist joint (containing the radiocarpal and the intercarpal joints, the carpometacarpal joints, the trapezium–first metacarpal joint), the metacarpophalangeal joints and the interphalangeal joints. The contractile structures can be divided into extrinsic and intrinsic muscles. The former – the long tendons – are quite regularly affected, while the latter are clinically of less importance.

Joints, joint capsules and ligaments

Distal radioulnar joint

The joint is a uniaxial pivot joint. The articular surfaces are between the convex head of the ulna and the concave ulnar notch of the radius. A fibrocartilaginous articular disc binds the distal end of the ulnar notch on the radius to the ulnar styloid process. This disc is part of the so-called triangular fibrocartilage complex (TFCC) and sits between the ulnar head and the ulnar carpus (lunate and triquetrum). Therefore the distal radioulnar joint can be seen as L-shaped: the short leg is between radius and ulna and the longer leg between the distal ulna and the articular disc (see Putz, Fig. 318). It has a loose capsule, reinforced with some ligaments, that allows rotation movement of the radius about the ulna. The two bones are held together mainly by the triangular fibrocartilage complex (TFCC) and to a lesser degree by the interosseous membrane and the pronator quadratus muscle (Fig. 1).

The TFCC contains different parts: (a) the triangular fibrocartilage, which has a central component – the articular disc – and two adjoining ligaments, the dorsal and palmar radioulnar ligaments. These take their origin at the dorsal and volar edges of the sigmoid notch and insert onto the ulnar styloid base (b) a meniscus homologue. This thickened ulnar part of the TFCC, inserts into the triquetral and hamate bones and the base of the fifth metacarpal; (c) the ulnar collateral ligament; (d) the ulnolunate and ulnotriquetral ligaments; and (e) the sheath of the extensor carpi ulnaris tendon, which is strongly attached to the posterolateral aspect of the TFCC. The TFCC stabilizes the radioulnar and ulnocarpal joints, transmits and distributes load from the carpus to the ulna and facilitates complex movements to the wrist (Fig. 2).

This distal radioulnar joint allows pronation–supination movements of the forearm, inevitably in combination with movement at the upper radioulnar joint, around an axis which runs through the head of the ulna. The distal end of the radius makes a circumferential gliding movement around and in front of the head of the ulna (see online chapter *Applied anatomy of the elbow*). Pronation has an amplitude of ± 85° and is stopped by the radius impacting against the ulna and by tension in the dorsal radioulnar ligament and the interosseous membrane. Consequently, there is an elastic end-feel. Supination ceases at an angle of 90° when the posterior aspect of the ulnar

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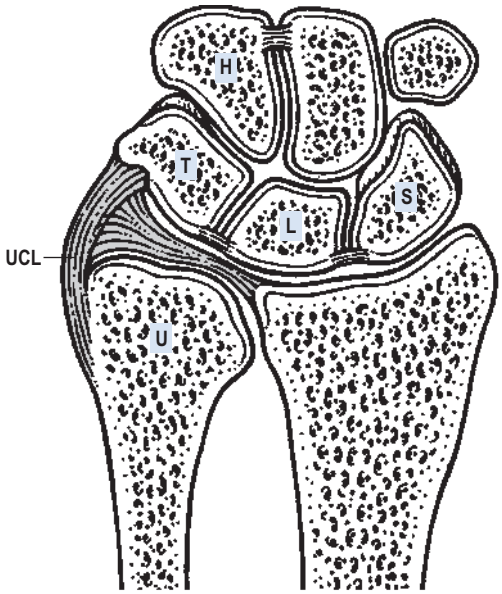

Fig 1 • Right distal radioulnar joint in supination. T, triquetrum; H, hamate; L, lunate; S, scaphoid; U, ulna; UCL, ulnocapitate ligament.

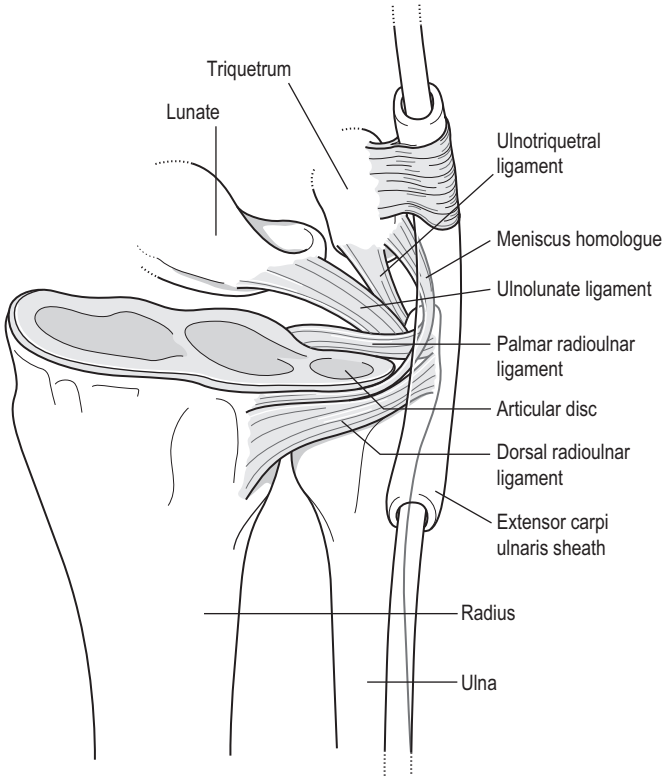

Fig 2 • Right distal radioulnar joint in pronation.

notch of the radius is brought into contact with the styloid process of the ulna through the extensor carpi ulnaris tendon. Its end-feel is also elastic (see Standring, Fig. 50.17).

Wrist joint

The wrist joint consists of two rows of carpal bones. The proximal row contains (from radius to ulna) the scaphoid, lunate, triquetral and pisiform bones, and the distal row the trapezium, trapezoid, capitate and hamate (Fig. 3, see Standring, Fig. 50.6A).

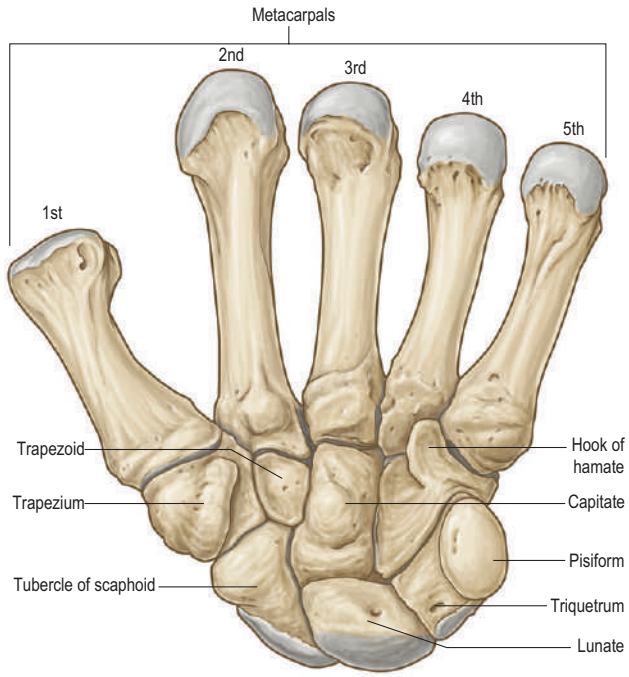

Fig 3 • Palmar carpus of the left hand. From Standring, Gray's Anatomy, 40th edn. Churchill Livingstone/Elsevier, Philadelphia, 2009 with permission.

The wrist joint is complex: there are two components, proximal and distal (see Putz, Fig. 326). Proximally the distal part of the radius and the articular disc articulate with the proximal row of carpal bones to form a condylar joint (radiocarpal joint) which has its concave surface at the radial side. The joint moves along two axes: anteroposterior for ulnar and radial deviation, and transverse for flexion and extension (Fig. 4).

Distally, the intercarpal joint is between the proximal and distal rows of bones (Fig. 5), is an open S-shape and acts as a hinge. It should not be considered as an independent joint because its function is to augment the mobility of the carpal bones and thus allow greater mobility at the wrist.

Movements are restricted by collateral (Fig. 5), palmar and dorsal ligaments (Fig. 6).

Radial and ulnar collateral ligaments

The radial collateral ligament, between the styloid process of the radius and the scaphoid bone, is taut when the hand is brought into ulnar deviation. The ulnar collateral ligament, between the styloid process of the ulna and the triquetral and pisiform bones, is under tension during radial deviation of the hand.

Dorsal ligaments

The dorsal wrist ligaments are comparatively thin. They are reinforced by the floor and septa of the fibrous tunnels for the six dorsal compartments (see below) and have a 'Z-shaped' configuration. The fibres of the dorsal radiocarpal ligaments are aligned more or less in the same axis as the forearm, those of

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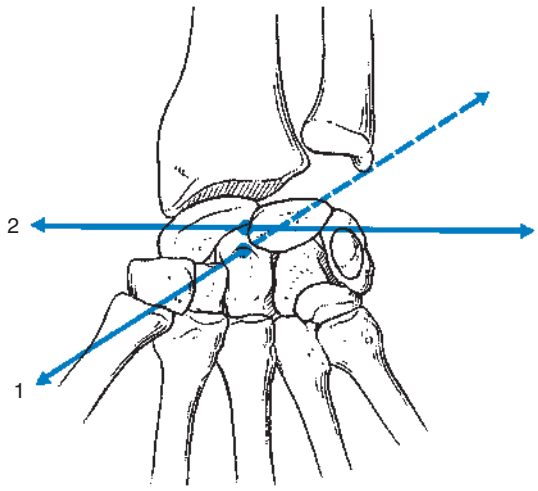

Fig 4 • The two axes of movement in the wrist joint: 1, anteroposterior axis along which ulnar/radial deviation takes place; 2, transverse axis along which flexion/extension takes place.

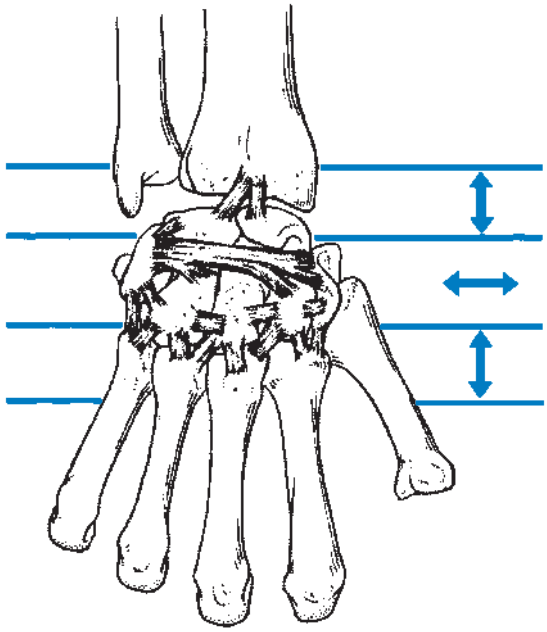

Fig 6 • The dorsal ligaments. The arrows emphasize the direction of the ligamentous fibres at the joint lines.

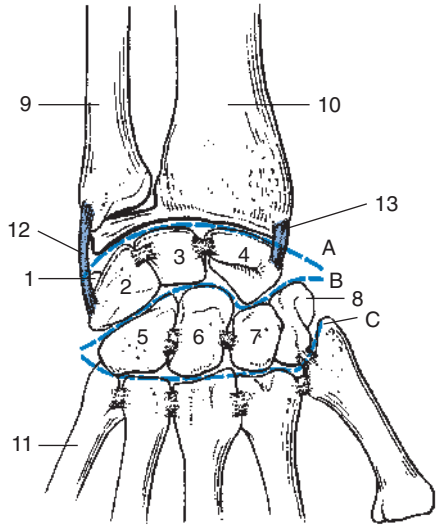

Fig 5 • The wrist and collateral ligaments (right hand dorsal view): 1, pisiform; 2, triquetrum; 3, lunate; 4, scaphoid; 5, hamate; 6, capitate; 7, trapezoid; 8, trapezium; 9, ulna; 10, radius; 11, metacarpal; 12, ulnar collateral ligament; 13, radial collateral ligament; A, radiocarpal joint line; B, intercarpal joint line; C, carpometacarpal joint line.

the intercarpal ligaments are more transverse to the carpus and the carpometacarpal ligaments are longitudinal again (see Standring, Fig. 50.15B). The pattern and shape of these ligaments is important for treatment with deep transverse friction which should be oblique to the direction of the fibres.

Palmar ligaments

On the palmar aspect of the wrist, a complex system of ligaments joins the different carpal bones to the radius and the ulna. These palmar ligaments are of little clinical importance and are taut during extension of the wrist.

Movements

The amplitude of radial deviation is only 15°, whereas ulnar deviation has an amplitude of approximately 45°, the wrist being held in the neutral position between flexion and extension. Flexion and extension both have a range of 85°, with the wrist in the neutral position between radial and ulnar deviation.

Flexion–extension has an end-feel of an elastic, capsular type. Radial and ulnar deviation has an end-feel that is quite hard but is clinically of less importance.

Carpometacarpal joints

The joints between the distal row of carpal bones and the second to fifth metacarpal bones are scarcely mobile, except for the joint with the fifth metacarpal, which permits slight movement in a palmar direction necessary to allow opposition of the fifth finger. They are provided with dorsal and palmar carpometacarpal ligaments, the fibres of which are in line with the axis of the metacarpal bones. Intercarpal ligaments 'tie' the bones together.

Trapezium–first metacarpal joint

The shape of the articular surfaces of both the trapezium and the first metacarpal bone ('saddle') allows movement in two

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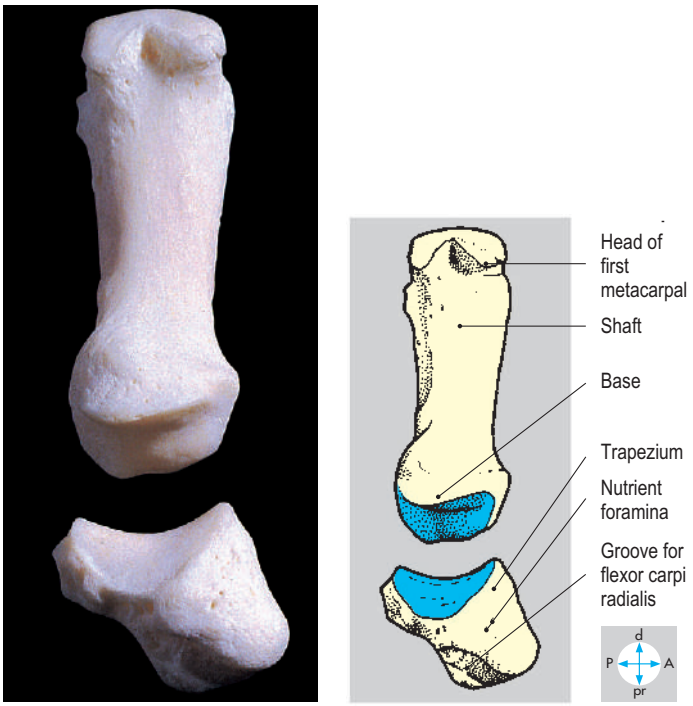

Fig 7 • Palmar view of the left trapezium–metacarpal joint. From Gosling et al, Human Anatomy, 5th edn. Mosby/Elsevier, Philadelphia, 2008 with permission.

directions: abduction–adduction and opposition–reposition (Fig. 7). The joint capsule is slack so as to allow free movement but strong ligaments control the range.

Metacarpophalangeal joints

The metacarpophalangeal joint of the thumb is different from that of the other fingers. It is a hinge and allows only a flexion movement (80–90°) and in hypermobile individuals some extension. Two sesamoid bones are often embedded in the palmar part of the capsule.

The metacarpophalangeal joint of each of the second to fifth fingers is a 'ball-and-socket' joint with a slack joint capsule, which is reinforced by strong palmar ligaments and collateral ligaments (see Gosling, Fig. 3.102). The collateral ligaments are taut when the joint is flexed.

The possible movements are flexion (90°, Fig. 8) and extension (sometimes to 90°, dependent on the laxity of the joint capsule), as well as abduction and adduction (the index finger having the greatest mobility). The rotatory movements are not clinically important.

Interphalangeal joints

The interphalangeal joints are hinges that allow flexion without extension, except in some individuals for the interphalangeal joint of the thumb and the distal interphalangeal joint of the fingers (see Gosling, Fig. 3.102). In the proximal interphalangeal joints, the range of flexion is more than 90° and increases from the index to little finger in which flexion can be 135°, which allows a 'fist' to be made.

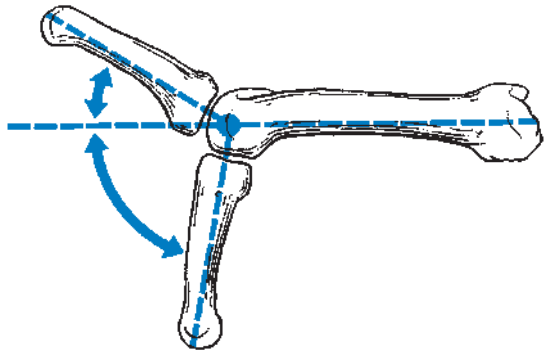

Fig 8 • Flexion and extension of the metacarpophalangeal joint.

Distally the amplitude of flexion varies between 80° and 90°, the little finger again being the most mobile. The joint capsules are reinforced by palmar and collateral ligaments.

The slight rotatory movements possible at the interphalangeal joints are not important from a clinical point of view.

Muscles and tendons

Clinically, the long tendons of the wrist, thumb and fingers – the tendons of the extrinsic muscles – are of more importance than the small muscles of hand, thenar and hypothenar – the intrinsic muscles.

Extrinsic muscles and tendons

As lesions are usually well localized, a topographical description of the tendinous structures is necessary. An understanding of exactly where they lie and where they insert is important.

Dorsal aspect

On the dorsal aspect of the wrist six osteofibrous tunnels can be distinguished (Fig. 9, see Standring, Fig. 50.14), which contain the tendon sheaths of the extensors of the wrist and fingers (Table 1) and the abductor of the thumb.

Tunnel 1

This contains the tendons of the abductor pollicis longus and the extensor pollicis brevis (see Standring, Fig. 50.38):

- *The abductor pollicis longus* inserts partly at the base of the first metacarpal bone, radial to the extensor pollicis brevis and partly into the trapezium. Its main function is abduction of the thumb, a movement that takes place between the trapezium and the first metacarpal.
- *The extensor pollicis brevis* inserts at the base of the proximal phalanx of the thumb and is responsible for extension of the thumb. Additionally, because of its close relationship with the abductor longus, it helps in abduction.

Both muscles lie in a common tendon sheath and have a further function: radial deviation and palmar flexion of the wrist.

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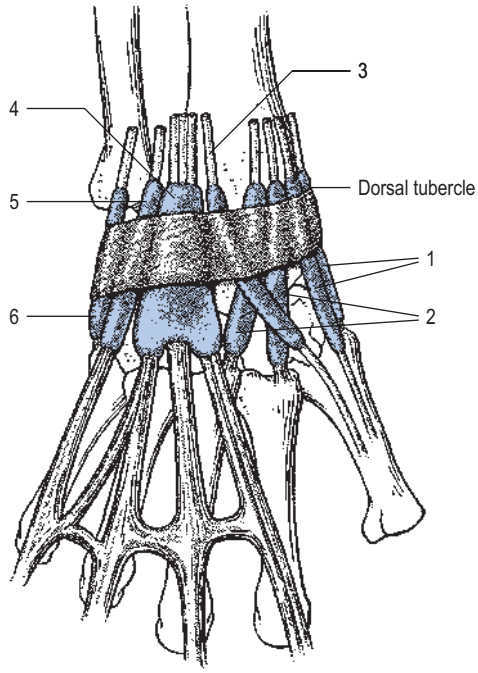

Fig 9 • The extensor tendons and their sheaths (numbers indicate the tunnels described in the text).

Table 1 The extensors

Muscle	Innervation	
	Peripheral nerve	Nerve root
Abductor pollicis longus	Radial	C7–C8
Extensor pollicis brevis	Radial	C7–T1
Extensor carpi radialis longus	Radial	C6–C7
Extensor carpi radialis brevis	Radial	C7
Extensor pollicis longus	Radial	C7–C8
Extensor indicis proprius	Radial	C6–C8
Extensor digitorum communis	Radial	C6–C8
Extensor digiti minimi	Radial	C6–C8
Extensor carpi ulnaris	Radial	C7–C8

Together they form the radial border of the 'anatomical snuffbox'.

Tunnel 2

The second tunnel contains the tendons of extensor carpi radialis longus and brevis. They diverge level with the carpus:

- *The extensor carpi radialis longus* inserts into the dorsal and radial aspect of the base of the second metacarpal bone and combines with the extensor carpi ulnaris to perform dorsiflexion of the wrist. In conjunction with the flexor carpi radialis muscle, it produces radial deviation.
- *The extensor carpi radialis brevis* attaches to the dorsal and radial aspect of the base of the third metacarpal bone. It extends the wrist and brings it back from ulnar deviation into a neutral position.

Tunnel 3

The third tunnel contains the tendon of the extensor pollicis longus, which runs along the ulnar aspect of the dorsal tubercle of the radius and then deviates 45° towards the thumb. Level with the carpus it passes over the tendons of the second tunnel and so forms the ulnar border of the anatomical snuffbox. It then continues over the dorsal aspect of the thumb towards the base of the distal phalanx. Its main function is to extend the thumb, although is also helps in extension of the wrist and abduction of the thumb.

Tunnel 4

Five different tendons are found here: the tendon of the extensor indicis proprius and the four tendons of the common extensor digitorum:

- *The extensor indicis proprius*: the fibres of the extensor indicis proprius blend with the dorsal aponeurosis of the index finger. It extends the index finger.
- *The extensor digitorum*: the tendons of the extensor digitorum merge with the dorsal aponeurosis of the second to fifth fingers and extend the fingers, especially the proximal phalanges. Their secondary functions are extension and ulnar deviation of the wrist.

Tunnel 5

This contains just one tendon, the extensor digiti minimi, which runs over the distal radioulnar joint and then parallel to the fifth tendon of the extensor digitorum into the dorsal aponeurosis of the fifth finger. Its function is identical to that of the extensor digitorum.

Tunnel 6

The extensor carpi ulnaris lies in a groove at the ulnar aspect of the head of the ulna. It then crosses the lateral aspect of the carpus to insert in the dorsal and ulnar aspect of the base of the fifth metacarpal bone. On account of its position, it functions mainly as a strong ulnar deviator of the wrist and also acts as an opponent to the abductor pollicis longus. Its extension function is very much secondary.

Palmar aspect

On the palmar aspect of the wrist lie the tendons of the flexors of wrist and fingers (Table 2). These are enclosed by the carpal tunnel and the tunnel of Guyon, which are of great importance.

The carpal tunnel

The palmar aspect of the carpus is concave and covered with the transverse carpal ligament, also called the flexor retinaculum. An osteofibrous canal is thus formed, of which the palpable boundaries are: proximally the scaphoid and pisiform bones and distally the trapezium and hamate bones (Fig. 10).

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Table 2 The flexors

Muscle	Innervation	
	Peripheral nerve	Nerve root
Flexor carpi radialis	Median	C7–T1
Palmaris longus	Median	C7–T1
Flexor digitorum superficialis	Median	C7–T1
Flexor digitorum profundus	Median Ulnar	C7–T1
Flexor carpi ulnaris	Ulnar	C7–C8
Flexor pollicis longus	Median	C7–C8

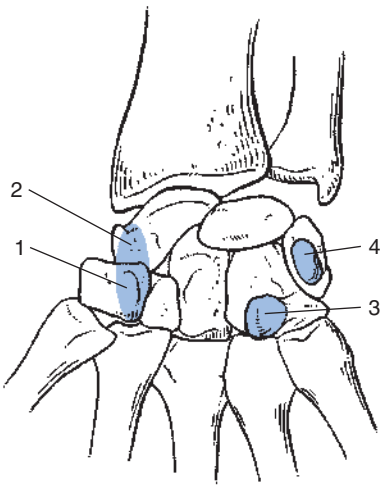

Fig 10 • Boundaries of the carpal tunnel: 1, trapezium; 2, scaphoid; 3, hamate; 4, pisiform.

The canal lies thus more distally and more towards the ulnar aspect than is often thought – at the heel of the hand.

Within the carpal tunnel are found: the median nerve; the tendons of the flexor carpi radialis and the flexor pollicis longus, in separate sheaths; and the superficial and deep flexors of the digits, within a common tendon sheath (Fig. 11).

The tendinous structures (Fig. 12, see Standring, Fig. 50.31) are the following:

- *The flexor carpi radialis* traverses the carpal tunnel along the scaphoid bone and inserts at the palmar aspect of the base of the second metacarpal. It flexes the wrist and helps in radial deviation.
- *The palmaris longus* muscle is absent in 15% of individuals. It is inserted into the palmar aponeurosis in the palm and helps in palmar flexion of the wrist as well as tensing the palmar fascia.
- *The tendons of the superficial flexors of the fingers* run, in a common sheath, through the carpal tunnel and insert into the middle phalanges of the second to fifth fingers; in doing this they separate each of the deep flexor digitorum tendons in two. Their functions are mainly flexion of the proximal phalanges of the fingers and ulnar deviation of the wrist.
- *The tendons of the deep flexors of the fingers* run through the carpal tunnel in a common tendon sheath with the superficial extensor digitorum and insert at the base of the distal phalanges of the second to fifth fingers. The muscle flexes the fingers and also the wrist.
- *The flexor carpi ulnaris* attaches to the pisiform, which some consider a sesamoid bone in the tendon of the flexor carpi ulnaris. Distal to the pisiform, the tendon divides into two ligamentous structures: the pisohamate ligament (which covers the tunnel of Guyon) and the pisiform–fifth metacarpal ligament. Together with the flexor carpi radialis, the flexor carpi ulnaris flexes the wrist and, with the extensor carpi ulnaris, performs ulnar deviation of the wrist.
- *The flexor pollicis longus* originates with a radial head at the anterior aspect of the radius, just distal to the insertion of the supinator brevis muscle and also from the interosseous membrane. There is an additional humeral head at the medial epicondyle. It runs lateral to the flexor carpi radialis muscle under which it crosses proximal to the wrist and then runs in a separate tendon sheath through the carpal tunnel to insert at the base of the distal phalanx of the thumb. It flexes the thumb and slightly flexes and radially deviates the wrist.

The tunnel of Guyon

This tunnel lies between the pisiform and the hamate bone and is covered by the pisohamate ligament, which is a continuation of the flexor carpi ulnaris tendon. The ulnar nerve and artery are also enclosed within this tunnel (Fig. 13, see Standring, Fig. 50.46).

Intrinsic muscles (Table 3)

Apart from the dorsal interossei, the intrinsic muscles of the hand (see Putz, Fig. 374) are seldom strained:

- *The thenar muscles*: these are clinically less important and include the abductor pollicis brevis, opponens pollicis, flexor pollicis brevis with its superficial and deeper head and the adductor pollicis with its transverse and oblique head.
- *The hypothenar muscles* are the abductor digiti minimi, flexor digiti minimi brevis and opponens digiti minimi.
- *The lumbrical muscles*: there are four lumbrical muscles. They originate at the radial aspect of the *tendons* of the deep flexor digitorum muscle and insert at the dorsal aponeurosis of the fingers and at the joint capsules of the metacarpophalangeal joints. They flex the metacarpophalangeal joints and extend the interphalangeal joints.
- *The palmar interossei*: the three palmar interossei adduct the fingers towards the middle finger, flex the metacarpophalangeal joints and extend the interphalangeal joints of the fingers (Fig. 14).

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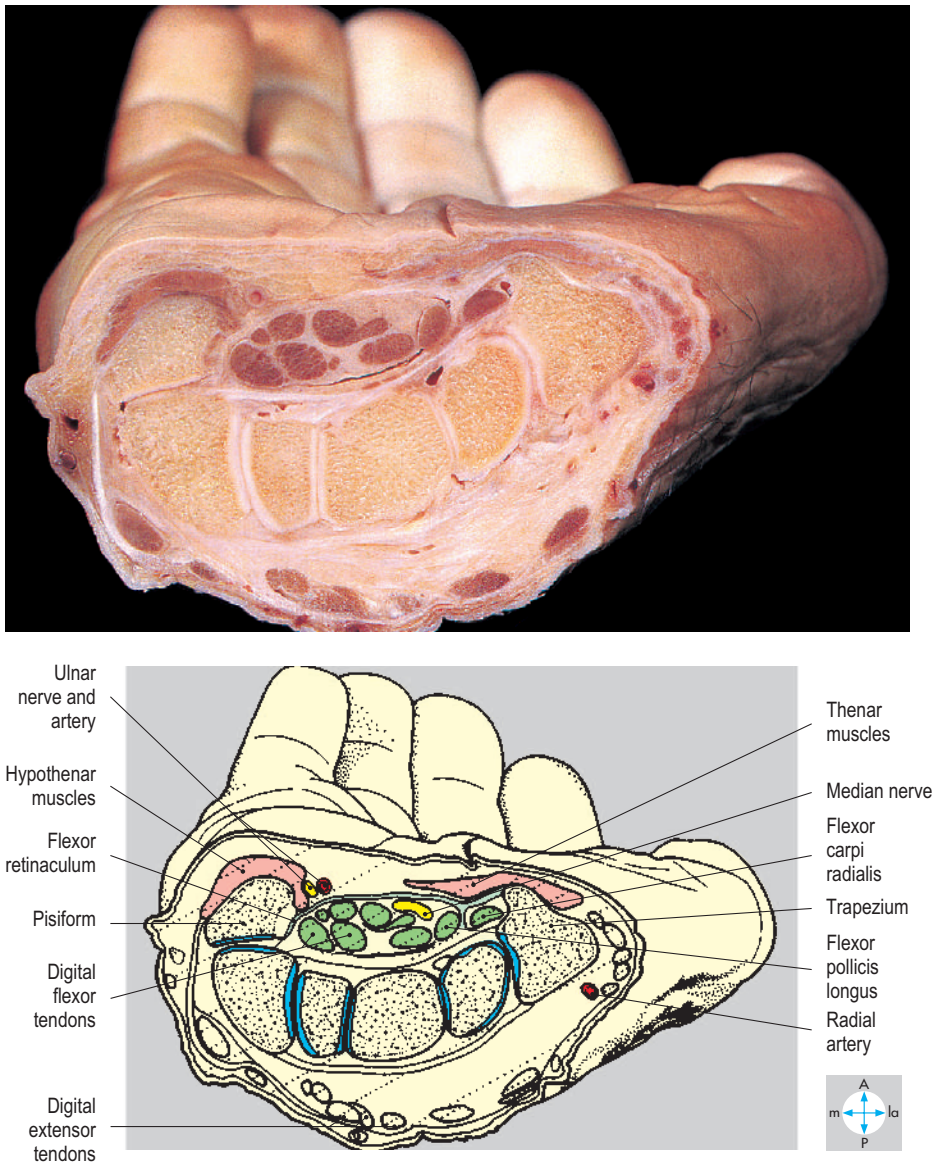

Fig 11 • Proximal view of the right carpal tunnel. From Gosling et al, Human Anatomy, 5th edn. Mosby/Elsevier, Philadelphia, 2008 with permission.

The dorsal interossei

More important are the four dorsal interossei. They originate from the sides of the five metacarpal bones, each having two heads, each in the side of two adjacent metacarpal bones (Fig. 15). They course towards the proximal phalanges, where they attach into the extensor aponeurosis of the fingers. The first dorsal interosseus goes to the radial aspect of the index finger. The second dorsal interosseus goes to the radial aspect of the middle finger, the third to the ulnar aspect of the middle finger. The fourth dorsal interosseus goes to the ulnar aspect of the fourth finger.

The dorsal interossei abduct the fingers away from the middle finger (Fig. 14). They also flex the metacarpophalangeal joints and extend the interphalangeal joints.

The palmar aponeurosis

The palmar aponeurosis of the hand is a continuation of the flexor retinaculum (the reinforcement of the distal part of the antebrachial fascia, also called transverse ligament of the carpus) and consists of transverse and longitudinal fibres (see Standring, Fig. 50.4A). The latter are part of the tendon sheaths of the flexor tendons (deep transverse metacarpal ligaments) and also connect to the capsules of the metacarpophalangeal joints. The aponeurosis is also connected to the deep fascia of the hand, which is attached to its skeleton. Together with the ligaments, septa and fasciae, the palmar aponeurosis forms a functional unit. During a strong grip it fixes the skin of the palm to the metacarpal bones and protects the soft tissues of the mid-hand.

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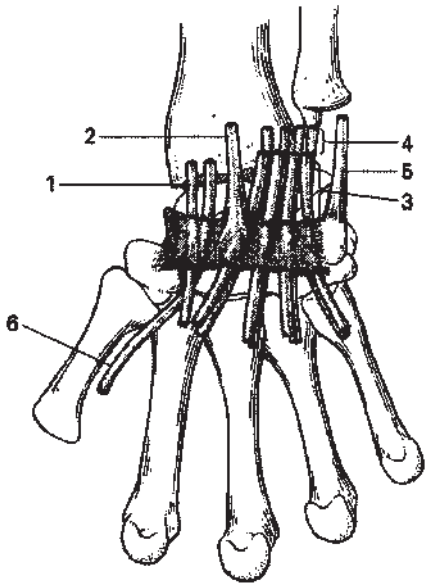

Fig 12 • The flexor tendons: 1, flexor carpi radialis; 2, palmaris longus; 3, superficial flexors of the fingers (4 tendons); 4, deep flexors of the fingers (4 tendons); 5, flexor carpi ulnaris; 6, flexor pollicis longus.

Table 3 The intrinsic muscles

Muscle	Innervation	
	Peripheral nerve	Nerve root
Abductor pollicis brevis	Median	C8–T1
Oppens pollicis	Median	C6–C7
Flexor pollicis brevis		
Superficial head	Median	C8–T1
Deep head	Ulnar	C8–T1
Adductor pollicis	Ulnar	C8–T1
Abductor digiti minimi	Ulnar	C8–T1
Flexor digiti minimi brevis	Ulnar	C8–T1
Opponens digiti minimi	Ulnar	C8–T1
Lumbricals		
Radial two lumbricals	Median	C8–T1
Ulnar two lumbricals	Ulnar	C8–T1
Dorsal interossei	Ulnar	C8–T1
Palmar interossei	Ulnar	C8–T1

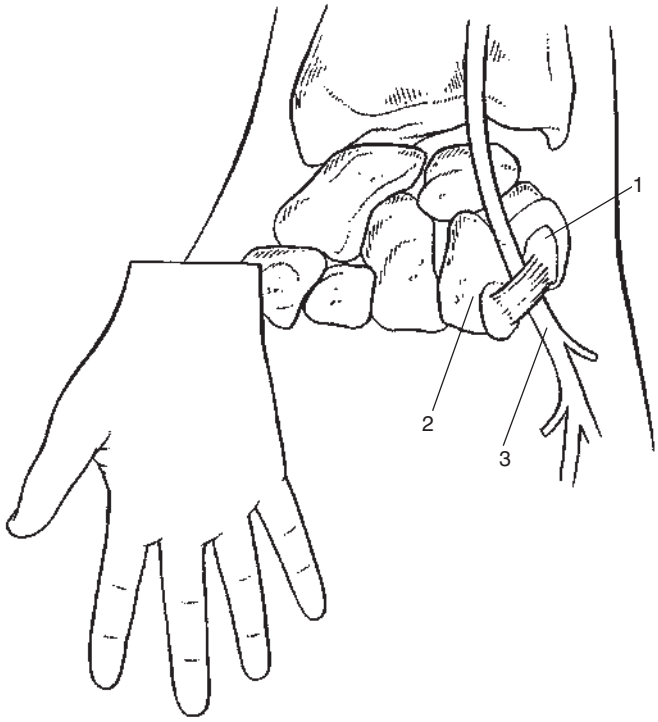

Fig 13 • The tunnel of Guyon: 1, pisiform; 2, hamate; 3, ulnar nerve.

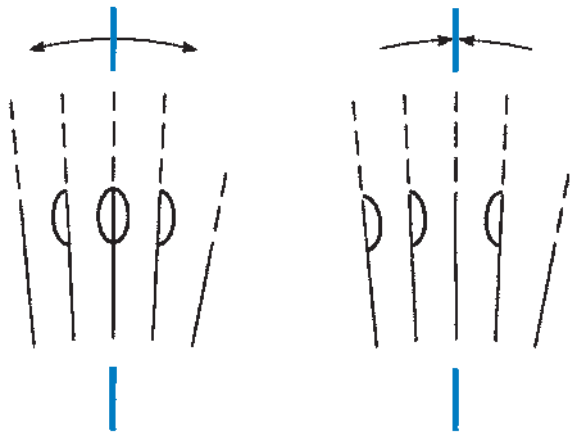

Fig 14 • (Left) Abduction, four dorsal interossei; (right) adduction, three palmar interossei.

Nerve structures

The three most important nerves in the wrist and hand are the median, ulnar and radial nerves. The median nerve is most frequently affected in the carpal tunnel, whereas the ulnar nerve becomes compressed in the region of Guyon's tunnel (see Standring, Fig. 50.45A). Lesions of the radial nerve at the wrist are less common.

Median nerve

The median nerve enters the hand through the carpal tunnel, in between the tendons of the flexor pollicis longus and flexor digitorum superficialis. Distal to the transverse ligament, the nerve divides into two branches (Fig. 16). A short motor branch goes to the thenar eminence, where it usually supplies the abductor pollicis brevis and opponens pollicis muscles, and sometimes the flexor pollicis brevis and the first and second

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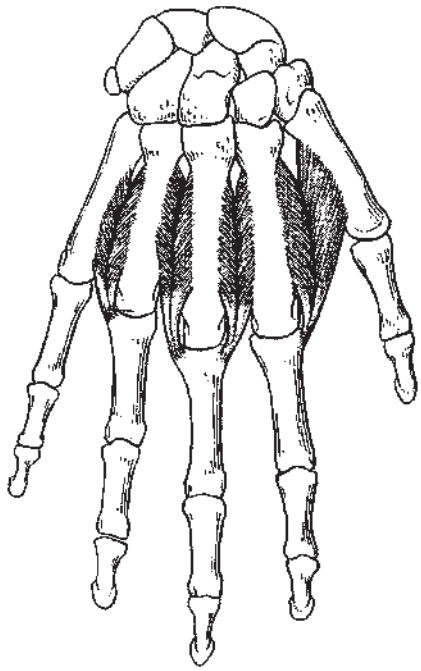

Fig 15 • The dorsal interossei.

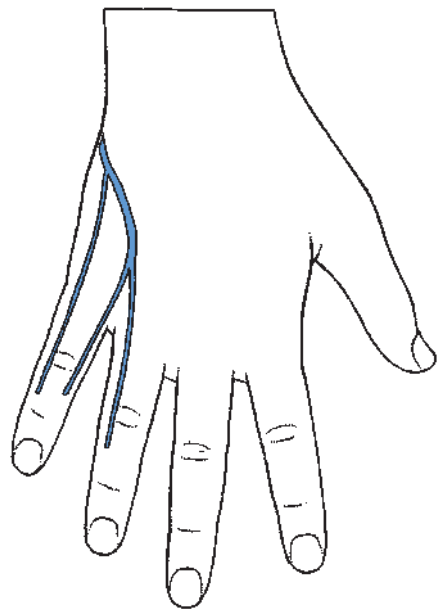

Fig 17 • The ulnar nerve: dorsal cutaneous branch.

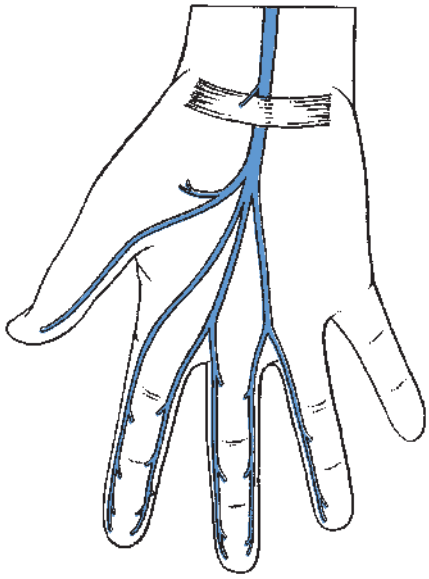

Fig 16 • The median nerve.

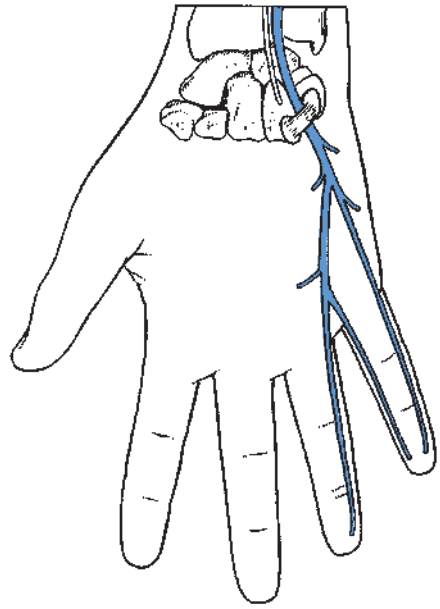

Fig 18 • The ulnar nerve: palmar branch.

lumbrical muscles. The sensory palmar digital branches innervate the palmar surface and the dorsal aspect of the distal phalanges of the thumb, the second and third fingers and the radial half of the fourth finger.

Ulnar nerve

Proximal to the wrist, the palmar cutaneous branch of the ulnar nerve arises and runs across the palmar aspect of the forearm and wrist, outside the tunnel of Guyon, to supply the proximal part of the ulnar side of the palm. A few centimetres more distally, the dorsal cutaneous branch arises and supplies the ulnar side of the dorsum of the hand (Fig. 17), the dorsal aspect of the fifth finger and the ulnar half of the fourth finger.

The ulnar nerve, together with the ulnar artery, passes between the pisiform and the hook of the hamate through the tunnel of Guyon. As it leaves the tunnel it divides into a mainly sensory superficial terminal branch, which supplies the distal ulnar border of the palm of the hand and the palmar surfaces of the fifth and ulnar half of the fourth finger (Fig. 18) and a deep terminal branch, which is entirely motor and innervates nearly all of the small muscles of the hand.

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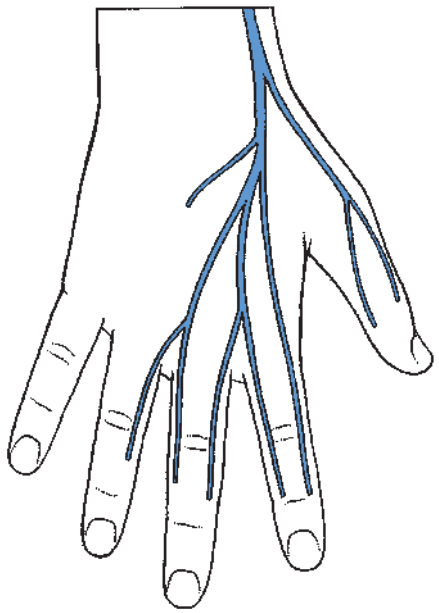

Fig 19 • Terminal digital branches of the superficial radial nerve.

Radial nerve

At the elbow, the radial nerve divides into the posterior interosseous nerve (a deep motor branch), which innervates the extensor muscles of the wrist and fingers, and the superficial radial nerve (a superficial sensory branch), which lies under the tendon of the brachioradialis muscle. In the hand, the superficial radial nerve divides into terminal digital branches that supply the dorsolateral aspect of the hand and the dorsal aspect of the first three and a half fingers, except the distal phalanges (Fig. 19).

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Disorders of the hand and fingers

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Pain and paraesthesia are two symptoms that are common in the hand. Pain is very often the result of either local trauma or overuse. Precise localization is possible in pain that is not referred from a lesion higher up in the limb. Paraesthesia may reflect a proximal lesion and the patient has difficulty in identifying the source of his symptoms.

Disorders of the inert structures

The capsular pattern

Any of the joints of the fingers may become affected by one or other form of arthritis, which results in limitation of movement with a capsular pattern.

The capsular pattern at a finger joint is an equal loss of movement at the beginning and end of the normal range in either direction. Some movement remains possible at the midpoint. Rotations are painful at the extremes of range. In very severe arthritis, rotations may also become limited.

The presence of a capsular pattern indicates that an arthritis has developed, the type of which can be defined from the history. The following questions are relevant:

- *How did it all start?* The possibilities are: no apparent cause, trauma or post-immobilization. A spontaneous onset indicates the possibility of rheumatoid arthritis or of a simple arthrosis. Trauma suggests traumatic arthritis.
- *Are other joints affected as well?* If they are, this suggests a rheumatoid condition.
- *Which joints were affected first, the distal or the proximal joints?* Arthrosis usually starts at the distal interphalangeal joints, whereas rheumatoid arthritis tends to start at the metacarpophalangeal joints.
- *Is the joint capsule swollen?* Swelling often occurs in a rheumatoid or traumatic arthritis.
- *Does the joint change colour?* The joint becomes red in gout.

Rheumatoid arthritis

Rheumatoid arthritis is undoubtedly the most deforming and most incapacitating disorder of the hand.[1]

The symptoms and signs usually develop in the course of a few weeks or months and are often symmetrical. Frequently,

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the first symptom is morning stiffness of the fingers. The basic functional examination is usually negative, but tenderness to touch can be elicited. The erythrocyte sedimentation rate is markedly raised.

Early in the course, a capsular pattern develops and one or more metacarpophalangeal joints or proximal interphalangeal joints of one or both hands show the familiar spindle-shaped swelling.

Later, when osseous destruction takes place, a palmar luxation of the fingers occurs and they deviate towards the ulnar side as the result of subluxation in the metacarpophalangeal joints. The fingers may develop the typical buttonhole and swan neck deformities: the former results in hyperextension of the metacarpophalangeal joint, flexion at the proximal interphalangeal joint and extension at the distal interphalangeal joint; the latter results in flexion at the metacarpophalangeal joint, hyperextension at the proximal and flexion at the distal interphalangeal joints. The thumb becomes Z-shaped (typical 'ninety-ninety' deformity): the metacarpophalangeal joint is fixed in 90° flexion, the interphalangeal joint in 90° extension. The joints are also warm to the touch.

Secondary rupture of tendons and ligaments may occur, with subsequent muscular contractures or muscular atrophy.

Treatment is systemic. Only in the initial stage, when few joints are affected, may intra-articular triamcinolone prove effective.

As in any other patient, symptoms may develop that have nothing to do with the patient's rheumatoid arthritis. Trigger finger, carpal tunnel syndrome and de Quervain's disease are common, and another possibility is a ganglion lying between the heads of the second and third metacarpal bones, which gives rise to vague local aching and responds well to aspiration.[2]

Traumatic arthritis

The typical history of direct contusion, indirect sprain or reduced dislocation of a finger joint indicates the presence of a traumatic arthritis – a very common condition. A chip fracture may also be responsible for the arthritis.

On inspection, a spindle-shaped swelling is often seen, which resembles the swelling of rheumatoid arthritis. Examination further reveals a capsular pattern, and on palpation warmth may be felt, especially after a severe injury. As the arthritis may be combined with a tendinous lesion, resisted movements of the fingers must also be tested.

Traumatic arthritis of the finger joints does not respond satisfactorily to any treatment. Intra-articular injection with a steroid, so useful in traumatic arthritis in the toe joints, affords no corresponding benefit in the fingers. Recovery is spontaneous over 6–18 months, depending on the severity of the original trauma and the age of the patient. Sometimes manual therapeutic techniques may favourably alter the natural course.[3] Immobilization is strongly contraindicated.

Arthrosis

Occasionally arthrosis in one joint develops as the result of severe injury but more often the condition has a spontaneous onset and affects several joints. Women between 40 and 60 years of age are often affected, and there is a strong familial predisposition.[4]

Arthrosis begins at the distal interphalangeal joints, and its knobbly appearance is quite different from rheumatoid arthritis. Both hands are usually affected more or less symmetrically. The index, middle and ring fingers are most usually affected. At the base of the distal phalanx, two small rounded bosses on the dorsum of the joint (Heberden's nodes)[5] can be seen. A varus deformity may develop at a distal joint, usually at the index. Some years later, the arthrosis may spread to the proximal interphalangeal joints (with the formation of nodes at index and middle fingers – Bouchard's nodes); it seldom reaches the metacarpophalangeal joints. From time to time, a new node forms at an affected joint and the patient will mention some aching or slight pain over 1 or 2 months, during which time the fingertip may occasionally become pink. The colour is mottled and different from the shiny red of gout. After a month or two the discolouration passes off and the node ceases to be painful.

As the joints of the hand are not weight-bearing, arthrosis is not a very painful condition. The patient merely complains of stiffness and aching especially after exertion.

The radiograph clearly shows the usual arthrotic changes – osteophytes and erosion of cartilage.

Heberden's nodes and arthrosis cause hardly any symptoms. They are unsightly and may be associated with aching and clumsiness. Since the distal finger joints finally fix in 45° flexion, arthrodesis seldom brings much improvement unless an intractable painful traumatic arthritis supervenes after injury. Some patients are pleased to have the exostoses removed surgically for cosmetic reasons.

Gout

The hands become involved only very late in the evolution of gout. According to Dieppe and Calvent,[6] the finger joints are affected in approximately 15% of cases.

The onset and the clinical appearance of chronic gout in elderly men may very closely mimic rheumatoid arthritis but diagnosis is not difficult when the patient is known to have gout and describes recurrent attacks. These typically start in the first metatarsophalangeal joint of the big toe, clear up completely and later spread to other joints. The affected joint usually has a shiny red appearance.

Tophi in the ears and a raised blood uric acid level finally appear but are of little diagnostic aid in the early, doubtful case.

Very characteristic, even diagnostic, is the rapid improvement of the arthritis within 48 hours of colchicine or phenylbutazone administration.

Non-capsular pattern

Unreduced dislocation

Dislocation is sometimes mistaken for traumatic arthritis at the interphalangeal joint of the thumb. The joint is so swollen that

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it is not obvious that it is fixed in full extension – a clear non-capsular type of limitation. In late cases, reduction is impossible and surgery is required.

Disorders of the contractile structures

Strains of muscles and tendons in the hand are not infrequent. They have no tendency to spontaneous cure. Diagnosis is not difficult and conservative treatment leads to good results. All the intrinsic muscles of the hand and their short tendons respond immediately to adequate deep transverse friction but not to infiltrations with steroids. In contrast, friction has no effect on the long flexor tendons in the palm but triamcinolone infiltration is successful.[7]

Dorsal interosseous muscles

Pain

A lesion in an interosseous muscle is usually traumatic, either the result of a direct injury or of a fracture of a metacarpal bone. Less commonly it follows overuse (e.g. musicians and keyboard operators).

There are three possible localizations for the lesion: in the muscle belly; in the tendon where it crosses the metacarpophalangeal joint; and at the insertion into the base of the phalanx (Fig. 1).

In the muscle belly, the lesion is found between the metacarpal shafts, usually distally. The patient experiences pain at the dorsal aspect of the hand, elicited by resisted abduction of the extended finger. Passive movement at the metacarpophalangeal joint may cause pain at the extreme range of movement but, as the pain is not felt at the joint itself and resisted movements are also painful, attention is drawn to the interosseous muscle. Careful palpation reveals the exact site of the lesion.

This lesion has no tendency to spontaneous recovery and it only responds – and very impressively – to deep transverse friction: two or three treatments suffice.

If the lesion is in the tendon or at the insertion, the pain is accurately felt at one side of one knuckle. The joint may be slightly swollen at the site of the lesion. Passive deviation of the finger away from the painful side is painful, as well as resisted abduction towards the painful side. Again, palpation must be performed very carefully to determine the exact painful spot.

Differentiation from traumatic arthritis is not always easy. Pain on resisted movement clarifies the diagnosis. Differential diagnosis is important because deep transverse friction will cure a tendon lesion but will have no effect on traumatic arthritis.

Untreated, the lesion may go on for years but it responds very well to deep transverse friction. The patient will be cured after four to ten treatments, however long the condition has lasted.

Technique: friction to the muscle belly

The patient sits at the couch with the hand resting on it. The therapist sits opposite the patient. The tender spot between the shafts of the metacarpal bones is palpated for, keeping the finger as parallel as possible to the metacarpals. Friction is imparted with the middle finger, reinforced by the index finger (Fig. 2a). The movement is pure pronation–supination, transverse to the muscular fibres.

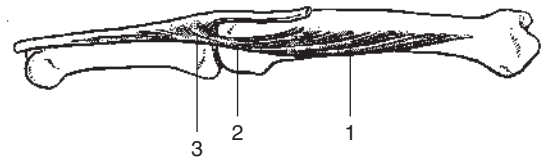
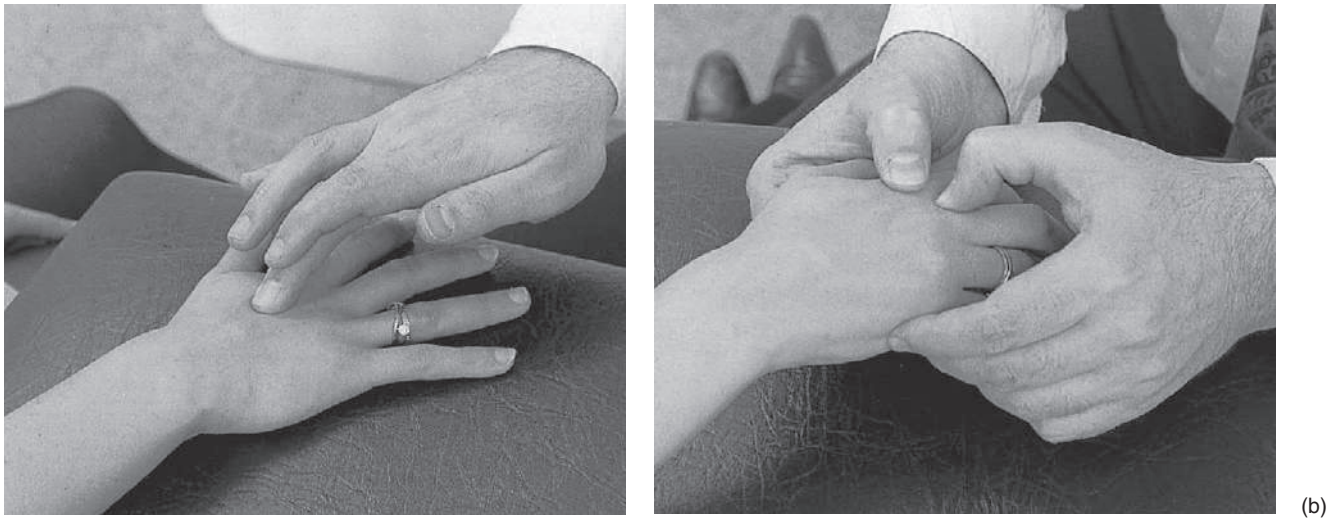


Fig 1 • Sites of lesions of a dorsal interosseous muscle: 1, in the muscle belly; 2, in the body of the tendon; 3, at the insertion into the dorsal aponeurosis.



(a) (b)

Fig 2 • Friction to (a) the belly and (b) the tendon of a dorsal interosseous muscle.

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Technique: friction to the tendon

It is sometimes difficult to get the finger to the exact spot because it lies more or less between the knuckles. Therefore the following procedure is used. With one hand the therapist takes the patient's hand, with the fingers in the palm of the patient's hand. The tips of the middle and ring fingers lie on the palmar aspect of the knuckle that contains the lesion and push the bone upwards. At the same time the thumb is laid on the dorsal aspect of the adjacent knuckle, which is pushed downwards. This manœuvre brings the affected tendon within reach of the thumb of the other hand. With the fingers of this hand counterpressure is exerted (Fig. 2b). The friction is given with the thumb, starting at the palmar aspect of the tendon and ending at the dorsal side of it. The movement is achieved by a supination movement of the forearm.

Weakness

Weakness of one or more interosseous muscles is the result of a neurological condition and is often one of the first signs of amyotrophic lateral sclerosis.

Weakness and wasting of one interosseous muscle only occurs in cases of localized pressure (usually occupational) on the deep palmar branch of the ulnar nerve in the palm of the hand (cyclist's palsy).[8]

A cervical rib can cause pressure on the lower trunk of the brachial plexus: apart from some of the interosseous muscles, the ulnar half of the flexor digitorum profundus and the flexor carpi ulnaris are also usually weak.

A first thoracic nerve root lesion may also be responsible for weakness of the small muscles of the hand.

Thenar muscles

A lesion of a thenar muscle may follow an abduction sprain of the thumb. The origin of the oblique portion of the adductor pollicis muscle at the palmar aspect of the base of the (second or) third metacarpal bone is most commonly affected. Resisted adduction of the thumb is painful, as is passive abduction.

Treatment consists of a few sessions of deep transverse friction.

Technique: friction

The patient sits at the couch, the supinated arm resting on it and the hand over the edge. The therapist sits opposite the patient. With the contralateral hand the wrist and the thumb are brought into extension in order to stretch the tendon and to bring it within reach of the fingers (Fig. 3).

Friction is imparted with the thumb of the other hand, starting at the radial aspect of the insertion and ending at the ulnar aspect.

Flexor tendons

A tenosynovitis may develop in the long flexor tendons in the palm of the hand. The rheumatoid type often presents with coarse crepitus on movements. This chronic tenosynovitis causes few or no symptoms. If necessary, the lesion responds well to infiltration with triamcinolone.

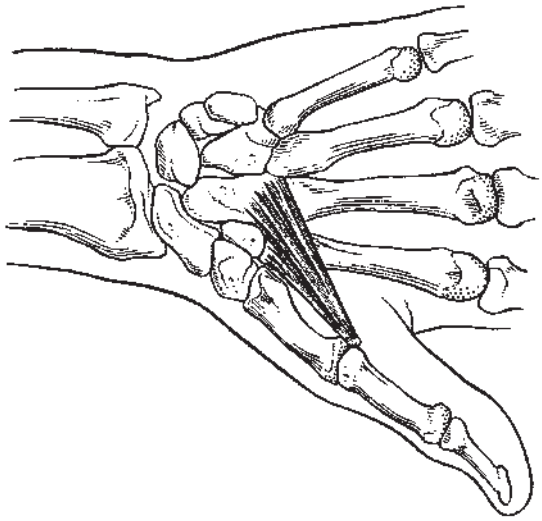

Fig 3 • Friction to the adductor pollicis.

Trigger finger

Trigger finger is a common condition causing pain and disability in the hand. It can arise spontaneously or can be the result of repetitive minor trauma or a complication of rheumatoid arthritis. Primary trigger finger occurs most commonly in the middle fifth to sixth decades of life and up to six times more frequently in women than men.[9] The third or fourth finger is most commonly involved. The disorder is caused by swelling of one of the digital flexor tendons just proximal to the metacarpophalangeal joint, in combination with narrowing of its tendon sheath.

The condition presents with discomfort in the palm during movement of the involved digits. Gradually the flexor tendon causes painful popping or snapping as the patient flexes and

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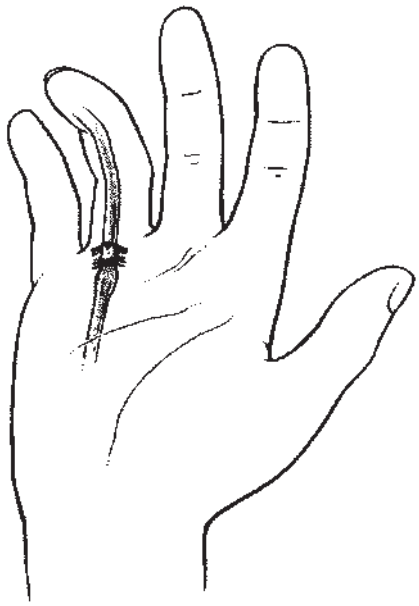

Fig 4 • Trigger finger.

Fig 5 • Infiltration for trigger finger.

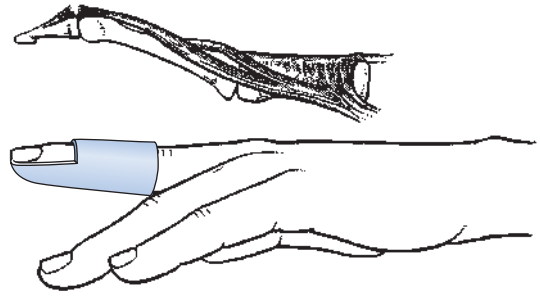

Fig 6 • Mallet finger. Splinting in extension by a stack splint.

extends the digit. As the condition progresses, the digit may begin to lock in a particular position, more often flexion, which may require gentle passive manipulation into full extension. A snap accompanies disengagement. Trigger finger arises through a discrepancy in the diameter of the flexor tendon and its sheath at the level of the metacarpal head known as the A1 pulley. This thickening of the sheath can result in a narrowed tunnel for tendon excursion and ultimately result in a block to tendon excursion.[10] However, the flexors are usually powerful enough to overcome this obstruction, whereas the weaker extensors are less able to counteract the block, resulting in the finger being locked in flexion. A painful nodule, the result of intratendinous swelling, is easily palpated in the palm, just proximal to the head of the metacarpal bone (Fig. 4).

If the condition gives rise to painful symptoms, infiltration with 10 mg of triamcinolone acetonide in and around the flexor sheath at the level of the A1 pulley should be given.[11,12] Steroid injection is an effective method of treating patients with trigger finger and should be considered as the preferred treatment.[13–16] If the result is not adequate, the tendon sheath can be slit up either at open surgery or percutaneously.[17] Success rates have been reported as over 90%; however, its use is tempered by the risk of digital nerve or artery injury and tendon bowstringing.[18,19]

Technique: infiltration of a trigger finger (Fig. 5)

The flexor tendon, the metacarpophalangeal joint and the painful nodule are identified. A 1 ml syringe, filled with 10 mg triamcinolone is fitted with a thin needle, 2 cm long. The needle, inserted at a point on the tendon, 1 cm proximal of the palpating finger, is directed at an angle of 30° to the horizontal until tough tendinous resistance is felt just underneath the palpating finger. Pressure is applied to the plunger and the needle gently manœuvred so that the tip is either deeper or more superficial to its initial position and the steroid will flow freely. This procedure is repeated a few times medially and laterally.

Tendon rupture

Mallet finger

As the result of an injury that flexes the distal interphalangeal joint while it is actively held in extension, the long extensor tendon may rupture (Fig. 6) or may become detached (avulsion fracture) from the distal phalanx. Distinction between the two can be made by radiography.

On examination, the distal joint is held in flexion and the patient is not able to extend it actively. Passive testing is normal.

Mallet injuries with and without a bony fragment may be effectively treated by splinting the distal interphalangeal joint in extension for 8 weeks, followed by 1 month of night splinting.[20] Dorsal, volar, or pre-fabricated stack type splints all can be used but care must be taken to avoid dorsal skin ischemia.[21] When a bone fragment has been retained with the extensor tendon, the opportunity to heal is enhanced because of the greater healing potential of bone compared to tendon.[22] The proximal interphalangeal joint should be left free, as

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immobilization of the proximal interphalangeal joint and its resultant stiffness may cause more morbidity than the original injury. Patients are counseled to expect a slight extensor lag (5–10°) under the best circumstances, with a mild loss of total motion.[23] Internal fixation of mallet fingers is recommended in cases of volar subluxation of the distal phalanx or in cases where the dorsal component is greater than one-third of the joint surface.[24]

Ruptured flexor tendon

Rarely, a tendon of the flexor digitorum longus may rupture at the level of the base of the distal phalanx. The whole tendon recoils into the palm of the hand. The distal interphalangeal joint cannot be actively flexed. Passive movement remains normal. Surgical repair is indicated.

Dupuytren's contracture

This disorder, named after G. Dupuytren,[25] is a painless contracture of the palmar aponeurosis. The aetiology is still unknown but it seems to occur more often in combination with alcoholism, disorders of the liver, diabetes and epilepsy.[26–28] It is common in men after the age of 30, whereas in women it does not occur under the age of 45.[29] A small node in the palm of the hand is the initial symptom. Further contraction of the palmar fascia leads to flexion contracture of the fingers, especially the ring and little fingers, which are affected in 85%.[30]

Once the contracture becomes disabling, because permanent flexion interferes with normal activities, the only treatment is surgery.

Südeck's atrophy

Reflex sympathetic dystrophy syndrome (RSDS), or Südeck's atrophy, is a curious disorder that is not uncommon. Different terminology has been used to label the same condition: Südeck's atrophy, causalgia, algodystrophy, algoneurodystrophy, reflex sympathetic dystrophy syndrome (RSDS).[31] It is a controversial condition. The controversy concerns the manner in which the sympathetic nervous system is involved in RSDS. It was redefined in 1996 by an ad hoc International Association for the Study of Pain task force that suggested changing the name to 'complex regional pain syndrome' (CRPS).[32–34] CRPS type 1 is reflex sympathetic dystrophy; type II is causalgia.[35]

It occurs most commonly as a complication of major or minor trauma (up to 5% of patients with traumatic injuries) or in patients with myocardial ischaemia (5–20%) or hemiplegia (12–20%).[36] Kozin[37] stated that RSDS was found to occur most frequently after fracture (25%) or other trauma (27%). In 27% of cases, no specific precipitating event could be identified. Central nervous system or spinal disorders, myocardial ischaemia and peripheral nerve injury are responsible for, respectively, 12, 6 and 4% of cases.

The pathogenesis is unclear. Some authors regard it as an injury to the peripheral nerve mechanism; others propose the theory of a central neurological dysfunction, causing abnormal activity in the sympathetic nervous system.

The main symptom is post-traumatic pain which is disproportionate to the injury. It spreads beyond the distribution of any single peripheral nerve, is usually felt distally in the arm and is often described as 'burning'.[38] Tenderness is always present and is occasionally associated with allodynia and enhanced sensitivity to palpation. Swelling of the affected part is often present and pitting or non-pitting oedema may be found. Dystrophic skin changes are seen: nail and hair changes (hypertrichosis), shiny and taut skin and loss of wrinkling.

The radiographic changes (patchy osteoporosis) have been well described by Südeck,[39] Kienböck[40] and Hermann *et al*.[41] A bone scan may be useful to arrive at a diagnosis.

Differential diagnosis has to be made with septic arthritis, rheumatoid arthritis, systemic lupus erythematosus, Reiter's syndrome and peripheral neuropathy.

The treatment of complex regional pain syndrome type I (CRPS-I) is subject to much debate. There is more or less concensus about the following.[42] For pain treatment, the WHO analgesic ladder is advised with the exception of strong opioids. For neuropathic pain, anticonvulsants and tricyclic antidepressants may be considered. For inflammatory symptoms, free-radical scavengers (dimethylsulphoxide or acetylcysteine) are advised. To promote peripheral blood flow, vasodilatory medication may be considered. Percutaneous sympathetic blockades may be used to increase blood flow in case vasodilatory medication has insufficient effect.[43,44] To decrease functional limitations, standardised physiotherapy and occupational therapy are advised.[45]

Disorders of the hand and fingers are summarized in Box 1.

Box 1

Summary of disorders of the hand and fingers

Disorders of the inert structures

The capsular pattern
- Rheumatoid arthritis
- Traumatic arthritis
- Arthrosis
- Gout

The non-capsular pattern
- Dislocation

Disorders of the contractile structures

Dorsal interosseous muscle

Thenar muscle

Flexor tendons

Trigger finger

Rupture of a tendon
- Mallet finger
- Ruptured flexor tendon

Dupuytren's contracture

Südeck's atrophy (reflex sympathetic dystrophy syndrome)

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Nerve lesions and entrapment neuropathies of the upper limb

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Peripheral nerve lesions in the neck, shoulder girdle and upper limb can occur anywhere along the extraspinal extent of the nerve – between the intervertebral foramen and the most distal nerve endings in the extremities. The function of the nerve becomes impaired either as the result of an entrapment phenomenon, or as the outcome of an injury causing bruising or elongation of the nerve tissue.

Entrapment phenomena occur typically at four different sites giving rise to four different mechanisms (see Chapter 2):

- Pressure on a distal nerve causes mainly analgesia as well as some paraesthesia in the territory of the nerve.
- When a nerve trunk or plexus becomes compressed, the release phenomenon – paraesthesia when the pressure ceases – is found.
- Nerve root compression is characterized by pain and paraesthesia, felt in the corresponding dermatome, and often followed by sensory and motor deficit in the same segment.
- Pressure on the cervical spinal cord is painless. An early symptom is paraesthesia with multisegmental distribution. When the compression becomes more severe, numbness, incoordination, spasticity and hyperreflexia may occur.

Disorders of the spinal cord and nerve roots are discussed in Chapters 2 and 8.

Disorders of the spinal accessory nerve

Anatomy

The accessory nerve is a cranial nerve (XI) and consists of two parts. Its main part is the spinal root; the other is the cranial root.

The spinal root takes origin in the spinal cord from a small pillar of nuclei in the anterolateral part of the anterior horn of the levels C1–C5/C6. The fibres leave the cord between the anterior and posterior rami of the nerve root. They join and form a strand that ascends parallel to the spinal cord and enters the skull through the foramen magnum.

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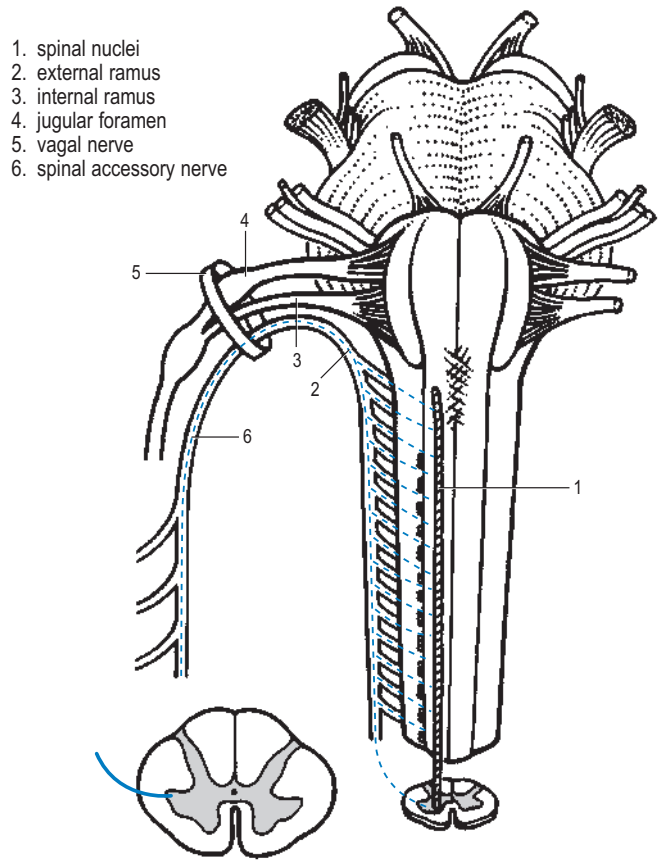

Fig 1 • Anatomy of the spinal accessory nerve.

The cranial root takes its origin in the caudal part of the nucleus ambiguus in the medulla oblongata.

Both parts accompany the glossopharyngeal (IX) and vagus (X) nerves in their exit through the jugular foramen. The fibres originating from the nucleus ambiguus then join the vagus nerve and the other fibres – the real spinal accessory nerve – descend towards the muscles they innervate (Fig. 1).

Innervation

The spinal accessory nerve is a pure motor nerve and innervates the sternocleidomastoid and the trapezius muscles (Fig. 2).

Disorders

A lesion of the spinal accessory nerve may be either idiopathic or result from a compression along its course. Idiopathic spinal accessory neuropathy may occur in isolation or in combination with a disorder of other nerves (glossopharyngeal, vagus, long thoracic or dorsal scapular).[1]

Mechanical lesions of the spinal accessory nerve can occur at different levels:

- Within the skull where the cause is usually tumourous.[2,3] This is uncommon.

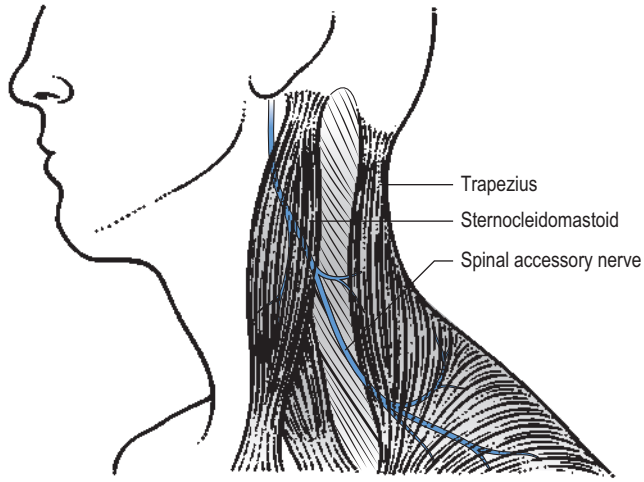

Fig 2 • Spinal accessory nerve: course and innervation.

- At the level of the exit through the jugular foramen where again, rarely, metastases or schwannomas may affect the nerve.[4,5]
- At the level of the neck where iatrogenic trauma, for example biopsy of lymph nodes in the posterior triangle, forms the commonest cause of isolated paralysis.[6–12] Injury rates from these procedures are reportedly 3–8%.[13,14] External traumas may also damage the nerve.

Mononeuropathy of the spinal accessory nerve

The patient initially complains of intermittent *pain* in the shoulder girdle area, which soon may become permanent.[15,16] At the same time, the arm starts to feel *weak* and heavy, which leads to some functional loss.[17] Exceptionally, pain is absent. Pain normally lasts for about 3 weeks, after which it disappears spontaneously.

Inspection elicits an asymmetrical neckline with drooping of the effected shoulder. This may be accompanied with lateral displacement and winging of the scapula.[18] Typically, winging is minimal and is accentuated during arm elevation, with the scapula moving upwards with the superior angle more lateral to the midline than the inferior angle. Limitation of active elevation of the arm is a consistent finding, and in one series of patients, the majority could only elevate to 80–90°.[19] Passive neck, scapular and arm movements do not influence the pain. Resisted elevation of the shoulder girdle is weak. Resisted external rotation of the arm makes the scapula more prominent medially.[20] In severe cases the trapezius muscle may be wasted. The diagnosis is confirmed by asking the patient to adduct both scapulae while the therapist applies counter-pressure at the medial border of the inferior scapular angle (Fig. 3). In neuritis of the accessory nerve, the scapula on the affected side can easily be pushed away at the side.

Spontaneous cure of motor function of the trapezius is the rule and usually takes about 4–8 months.[21,22] If inadequate functional recovery is seen after a year, additional conservative treatment is unlikely to be beneficial and surgery is indicated. In a recent review of the literature, authors have reported good

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Fig 3 • Test to confirm spinal accessory nerve palsy.

or excellent results in approximately two-thirds of patients treated with nerve surgery.[23]

Disorders of the brachial plexus

Anatomy

The ventral rami of the spinal nerves C5, C6, C7, C8 and T1 unite to form the brachial plexus. Occasionally a prefixed (C4) or postfixed (T2) ramus takes part in the formation of the plexus. Several interconnections lead to the formation of trunks, divisions, cords and branches.

Trunks

There are three trunks: superior, middle and inferior. The *superior trunk* is created by the fusion of the ventral rami of C5 and C6. The *middle trunk* is the continuation of the ventral ramus of C7. The *inferior trunk* is formed by the ventral rami of C8 and T1.

Divisions

The three trunks divide into an anterior and a posterior part. The posterior parts form the posterior cord. The anterior parts form the other cords: the superior trunk continues in the lateral cord and the inferior trunk in the medial cord. The superior and inferior trunks also give off branches for the middle trunk, thus forming interconnections.

Cords

The cords are lateral, posterior and medial according to their relation to the subclavian/axillary artery. The *lateral cord* is formed from fibres of the superior trunk, together with fibres from the middle trunk. The *posterior cord* results from the fusion of fibres originating from the three trunks. The *medial cord* is the continuation of the inferior trunk.

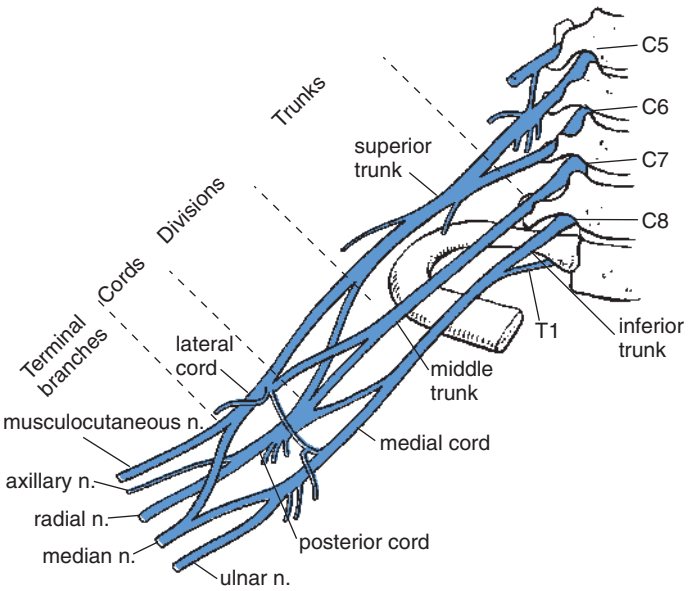

Fig 4 • The brachial plexus.

Peripheral nerve

From the three cords the major peripheral nerves of the upper limb are formed. The lateral cord continues into the *musculocutaneous nerve*. The posterior cord forms a branch that divides into two separate nerves: the *radial nerve* and the *axillary nerve*. The medial cord forms the *ulnar nerve*. From an anastomosis between the lateral cord and the medial cord the *median nerve* is formed.

Several other nerves emerge directly from the brachial plexus, either from its supraclavicular or its infraclavicular part.

From the supraclavicular part of the plexus originate: the *dorsal scapular nerve* (innervating the levator scapulae, major rhomboid and minor rhomboid muscles); the *long thoracic nerve* (innervating the serratus anterior muscle); the *thoracodorsal nerve* (innervating the latissimus dorsi muscle); the *suprascapular nerve* (innervating the supraspinatus and infraspinatus muscles); the *inferior subscapular nerve* (innervating the teres major muscle); the *subclavian nerve* (innervating the subclavian muscle); the *lateral pectoral nerve* (innervating the upper part of the pectoralis major muscle); and the *medial pectoral nerve* (innervating the lower part of the pectoralis major muscle as well as the pectoralis minor muscle).

From the infraclavicular part of the plexus the following nerves originate: the *medial cutaneous nerve of the arm* (supplying the anteromedial and posteromedial part of the arm); and the *medial cutaneous nerve of the forearm* (its anterior ramus supplying the anteromedial aspect of the forearm, and its cubital ramus supplying the posteromedial aspect of the forearm) branches of the medial cord of the brachial plexus (Fig. 4).

Innervation

The brachial plexus is responsible for the complete motor and sensory innervation of the shoulder girdle and upper limb.

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Traumatic disorders

The brachial plexus lies quite superficially within a very mobile shoulder girdle and is closely related to the different bony structures of neck, shoulder girdle, shoulder and thorax. This situation makes it very vulnerable. Traumatic disorders are therefore one of the commonest causes of brachial plexus dysfunction. As the result of traction injuries (e.g. motorcycle accidents), compression by dislocated (e.g. shoulder luxation[24,25]) or fractured bones (e.g. fracture of the clavicle[26]) or by haematomas, and intraoperative or birth injuries, larger or smaller parts of the plexus may become damaged, leading to total or partial syndromes.[27]

Upper brachial plexus palsy

This is called Erb–Duchenne's paralysis and is defined as a palsy of C5 and C6 and sometimes of C7. There is a motor deficit of the muscles innervated by the nerves originating from these fibres and possibly a sensory deficit in the C5 and C6 dermatomes (lateral and anterior aspects of arm and forearm and radial aspect of hand and fingers) (Table 1).

The patient cannot bring the arm up and has difficulty in bending the elbow; there is a visible atrophy of the deltoid, supraspinatus and infraspinatus muscles.

Middle brachial plexus palsy

If the middle part of the brachial plexus becomes damaged by trauma, the serratus anterior and rhomboid muscles remain unaffected. There is slight weakness of the deltoid and supraspinatus muscles, which results in the patient not being able to elevate the arm above the horizontal. Elbow flexion is weak because of paresis of the biceps muscle. Sensation remains normal.

Lower brachial plexus palsy

This is known as Dejerine–Klumpke paralysis and is not so common but it causes severe disability. The lesion affects the C8 and T1 segments, and quite often also C7. As a result, there is a palsy of the muscles supplied by the ulnar and median nerves as well as the finger extensors and the extensor carpi ulnaris muscle (radial nerve). A complete inability to use the hand follows. There is also sensory deficit in the C8 segment (ulnar aspect of hand and distal forearm).

Table 1 Affected nerves and muscles in a palsy of the upper part of the brachial plexus

Nerve	Muscle
Long thoracic	Serratus anterior
Dorsal scapular	Rhomboids
Suprascapular	Supraspinatus Infraspinatus
Axillary	Deltoid Teres minor
Musculocutaneous	Coracobrachialis Biceps
Radial	Brachioradialis Supinator

Space-occupying lesions

Metastatic tumours – usually originating from the breast, the lung or the lymphatic system – may invade the brachial plexus.

The superior pulmonary sulcus tumour (Pancoast) typically invades the lower trunk of the plexus as well as the sympathetic ganglia at the base of the neck.[28] A lower brachial plexus dysfunction is then accompanied by Horner's syndrome (see p. 3 of online chapter *Disorders of the thoracic cage and abdomen*).

Aneurysm of the subclavian artery and pseudoaneurysm of the axillary artery are other possible causes of compression of the brachial plexus.

Thoracic outlet syndrome

Thoracic outlet syndrome (TOS) is a vague term, only suggesting the presence of a disorder within the area of the thoracic outlet. Although it is generally accepted that the aetiology is compression of the plexus and vascular bundle in the thoracic outlet, different opinions exist about the pathogenesis. This is expressed in the various names that have been given to the syndrome (see Box 1).[29] The consequence of disagreement on the aetiology is that numerous methods of treatment are advocated.

Anatomy

The thoracic outlet is the space bounded by the upper part of the sternum, clavicle, first rib and the first thoracic vertebra.[30] Towards the centre, it is limited by the trachea and oesophagus. It forms the communicating area at the base of the neck for the passage of blood vessels and nerves from mediastinum and neck to the axilla and into which the dome of the pleura rises upward.[31] The vagus, phrenic nerves, sympathetic trunk and thoracic duct also pass through the same openings.

The first rib has a flat upper surface. In its anterior portion there are two grooves, which are separated by the tubercle at which the anterior scalene muscle inserts. The more medial groove accommodates the subclavian vein. Behind the muscle, in the posterior groove, both subclavian artery and brachial plexus are found (Fig. 5).

The clavicle overlies the first rib just cranial to the posterior groove. At this site, compression of the neurovascular structures between first rib and clavicle is possible; this mainly happens to the most medially localized trunks, which contain fibres of C8 and T1, so causing symptoms in the territory of the median and the ulnar nerve.

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Box 1

Synonyms for thoracic outlet syndrome

Shoulder–hand syndrome
First thoracic rib syndrome
Cervical rib syndrome
Brachiocephalic syndrome
Scalenus anticus syndrome
Humeral head syndrome
Costoclavicular syndrome
Nocturnal paraesthetic brachialgia
Adson's syndrome
Hyperabduction syndrome
Cervicobrachial neurovascular compression syndrome
Fractured clavicle syndrome
Pneumatic hammer syndrome
'Rucksack' paralysis
Effort vein thrombosis
Cervicothoracic outlet syndrome
Pectoralis minor syndrome
Subcoracoid syndrome
Syndrome of the scalenus medius band
Brachial plexus syndrome
Paget–Schroetter syndrome
Naffziger's syndrome
Shoulder girdle syndrome
Haven's syndrome
Sympathetic algodystrophy

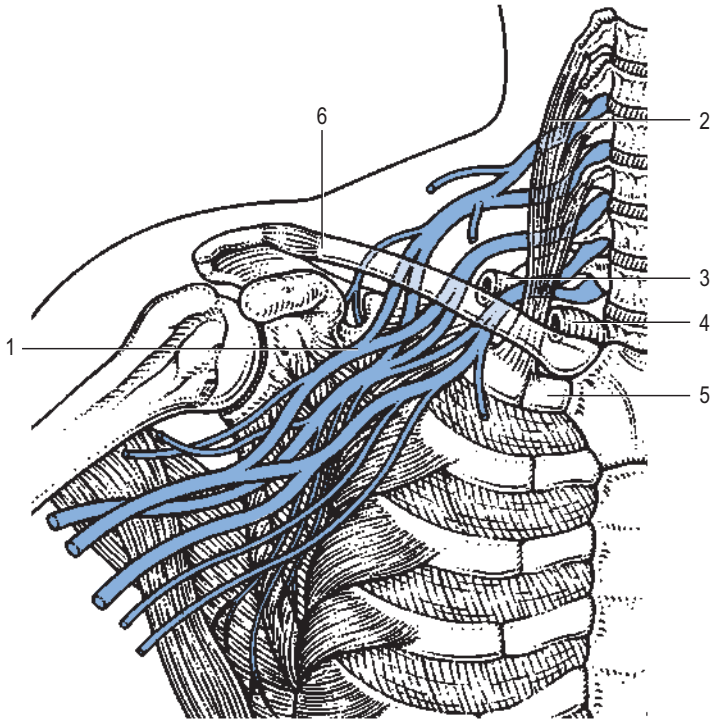
Types

According to Cyriax, the syndrome is the outcome of a compression of the most medial branches of the brachial plexus, usually occurring between the clavicle and first rib, seldom as the result of a cervical rib. The compression is usually bilateral, intermittent or continuous and may or may not involve the subclavian artery and vein.[32] It gives rise to a set of neurovascular symptoms, which are rarely present all together. Symptoms of neurological disturbances are usually found but sometimes only features of vascular compression.[33] The brachial plexus is involved in 98% of cases, the subclavian vein in 1.5%, and the artery in 0.5%.[34,35]

Cyriax recognized two main groups of thoracic outlet syndromes based on anatomical and clinical grounds: the cervical rib syndrome and the first rib syndrome. We prefer to substitute for these terms. We make a distinction between the thoracic outlet syndrome caused by anatomical changes and that from postural factors. This approaches the growing agreement about the use of four terms to indicate the presence of the thoracic outlet syndrome;[36,37] true neurologic, arterial and venous TOS – those syndromes that result from compression by a cervical rib (anatomical variety); and non-specific neurologic TOS[38,39] – the postural variety.

Anatomical variety

This is caused by structural changes – the presence of a bony cervical rib or a band of fibrous tissue which is found in 0.5% of a normal population. Only 5% of them will ever suffer from



1, brachial plexus;
2, scalenus anterior muscle;
3, subclavian artery;
4, subclavian vein;
5, first rib;
6, clavicle.

Fig 5 • A slightly schematic representation of the relationship of neurovascular structures in the thoracic outlet.

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a thoracic outlet syndrome.[40] In a fibrous band, the corresponding transverse process of C7 is larger than usual – a rudimentary cervical rib. A cervical rib sometimes gives rise to a palpable mass at the base of the neck.

Interference with the neurovascular structures usually begins at between 20 and 30 years of age and is thereafter continuous. Structural change and clinical features are frequently bilateral, although often more pronounced at one side.[41] The symptoms are the result of compression of both neurological and vascular structures.

Neurocompression

Compression of a nerve trunk can give rise to pain felt at the base of the neck, radiating unilaterally towards the shoulder and arm and even towards the chest. The pain is not very severe and is often associated with pins and needles and numbness, which may be felt in all five digits, although most frequently the fourth and fifth fingers are affected together with the ulnar side of the arm and hand.

The symptoms often appear or increase shortly after having carried a heavy object, but exceptionally they can occur during the activity itself. Some patients complain of disturbed fine motor coordination together with weakness and dysfunction of the hand.

On clinical examination, atrophy of the hypothenar (T1) or of the thenar muscles (C8), together with weakness of the interossei (T1), can be present.

Once a cervical rib has started to interfere with a nerve, the compression does not resolve. Therefore, the symptoms are continuously present and tend to increase.

Vascular compression

The compression can be venous and/or arterial.

Arterial

Compression of the subclavian artery (Fig. 6) results in coldness, pallor and easy tiredness of the hand.[42] Especially after the arm has been hanging down for a while, the hand may turn white and cold over a period of hours. A diminished pulse may be found.

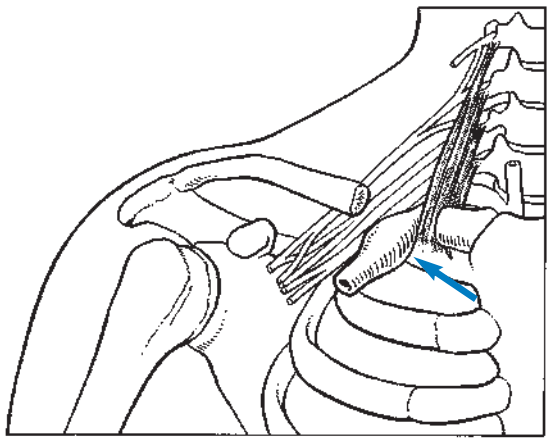

Fig 6 • Compression of the subclavian artery with poststenotic aneurysm (arrow).

In more severe cases, chronic compressive stenosis of the artery may give rise to claudication of the upper extremity. It may further lead to a poststenotic dilation, sometimes to the formation of an aneurysm. Atheromata arising from these aneurysms or from chronic compression injury of the artery can result in peripheral embolization, which may lead to irreversible damage to the hand and even to the entire arm.[34,43–45]

The presence of an acute ischaemic syndrome of the upper extremity, usually in a young female, suggests the possibility of a thrombosis in a poststenotic aneurysm.

Venous

If the subclavian vein is impaired, cyanosis, swelling and oedema of the hand and forearm are the indirect signs of this process. Thrombosis of the subclavian vein may occur.[46–48]

Postural variety

This category comprises cases that have an almost identical clinical pattern but in which there is no cervical rib or fibrous band. It is characterized by neurological symptoms typically present after lying down for, say, 2–3 hours. It is a benign disorder seldom resulting in vascular disturbance or in muscular atrophy.

There are two types, depending on the clinical features.

Acute onset

This is unusual, but the diagnosis is important although difficult to make. Frequently the patient is admitted to the hospital suspected of having had a heart attack.[49]

Young patients with acute onset of symptoms after carrying a heavy load are typical. They complain of a sudden and severe thoracic pain, radiating down the arm, feel faint and have pain on breathing. All such features draw attention to serious visceral disorders (heart attack, pneumothorax). A short time later, the arm and hand blanche. This situation resolves after a few hours, so that by the time investigations, such as electrocardiography, radiography of the thorax and possibly laboratory tests, have been done, the symptoms have disappeared and the patient is perfectly normal again.

Slow onset

This is the more common type. It is very slow in progression and has a benign evolution, seldom giving rise to neurological deficit. The diagnosis is often missed, although not difficult to establish.

It affects the middle aged or elderly, more frequently females. It sometimes occurs in pregnancy. The onset is with pins and needles in the hand and fingers, mainly at night and usually after 2–3 hours of sleep. The process is often bilateral, although worse on one side. Paraesthesia may be felt in all digits but may predominate in the median or ulnar distribution. It wakes the patient, who finds that she has to sit up or walk around for a short period of time, rubbing and moving the hands and fingers, to make the symptoms go. The symptoms disappear after a few minutes, allowing sleep to be continued, although recurrence may take place in the early morning hours. The more physical activity during the preceding day, the worse the symptoms at night.[50] In periods of rest or sickness, when

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the patient lies down for the whole day, no pins and needles are felt. Some patients also experience symptoms during the day, on activities such as knitting, holding a newspaper in front of the eyes or bicycling, all of which require some degree of shoulder elevation. Augmenting the pressure by carrying a heavy object may exceptionally provoke the symptoms as well, but normally only to a mild degree.

Cyriax explained this pattern as being the consequence of a diminishing tone in the shoulder muscles, starting in middle age. As a result, the shoulder girdle droops down during the day, resulting in compression of the most medial trunks of the brachial plexus, between the first rib and clavicle. Compression occurs during the day but the symptoms come on mainly at night after the pressure on the nerve has disappeared. He called this the 'release phenomenon' (see p. 26). The process seldom leads to damage of the nerve parenchyma with subsequent muscular atrophy because the brachial plexus can recover every night when the pressure is released.

Functional examination

The diagnosis is based mainly on the typical history, all passive movements of neck, shoulder and shoulder girdle being normal.[51] The resisted movements are of normal strength and painless, except for C8 or T1 structures in the hand, which may be weakened when the compression is the outcome of a cervical rib. This type of characteristic history should always be followed up with the following additional tests.

Sustained elevation of the shoulders

The patient sits in a comfortable position and is asked to shrug the shoulders for about 3 minutes (Fig. 7). This causes maximum release of pressure and therefore may bring on the pins and needles and abolish vascular symptoms if present. However, this test is not always positive when thoracic outlet syndrome is present; in this case, release of pressure must be tried in different positions, either fully raising the arms above the head and maintaining this position for 3 minutes (Fig. 8) or lying supine with both hands on the head for the same length of time (Fig. 9).

Auscultation, pulse and blood pressure

The subclavian area should always be auscultated for a bruit, the radial pulse must be checked and blood pressure must be measured. The diagnostic significance, however, is not certain.

Other tests

Other tests for thoracic outlet syndrome have been advocated classically. Although we regard them as less specific and reliable, they are mentioned here for completeness. Adson's test,[52] the modified Adson's test and Roos' test (elevated arm stress test)[53] are regarded by some as totally unreliable, because about 50–60% of the normal population have positive findings.[54,55]

Fig 7 • Sustained shoulder elevation test.

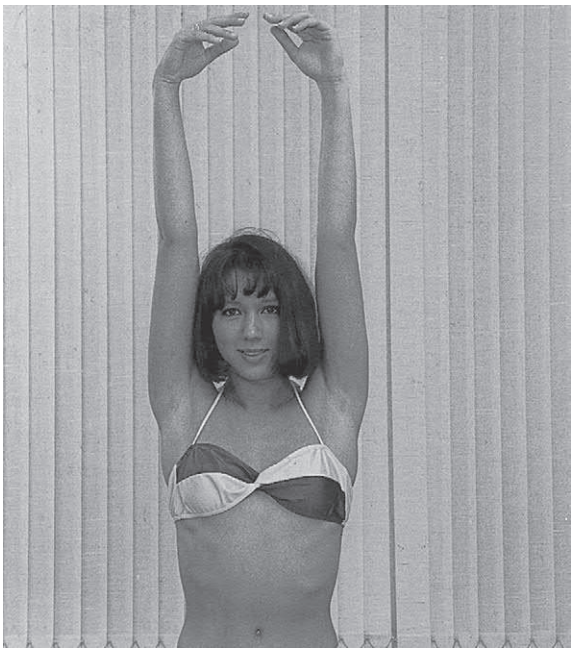

Fig 8 • Alternative sustained shoulder elevation test.

Adson's test

The patient stands with the arm resting at the side. In this position the examiner feels the patient's radial pulse. Then the patient is asked to take a deep breath and to turn the head towards the involved side. Any change in pulse degree or in blood pressure, preferably measured by a Doppler probe, is noted. If there is a change, it means that the subclavian artery

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Fig 9 • Alternative sustained shoulder elevation test.

Fig 10 • Roos' test.

is compressed and probably also the brachial plexus. Adson considered a positive test to indicate vascular compression by the scalenus anterior.

Modified Adson's test

This is similar to Adson's test but the patient turns the head away from the involved side. In this test, the scalenus medius puts pressure on the vessels. A positive test is thought to imply scalenus medius compression.

Roos' test

The patient abducts the arms to 90°, then opens and closes the hands slowly for 3 minutes. Those who are unable to keep their arms and hands elevated because of pins and needles are regarded as suffering from thoracic outlet syndrome. However, in carpal tunnel syndrome, active flexion of the fingers can bring on the pins and needles, and therefore this test does not differentiate between these two disorders (Fig. 10).

Examination of the cervical spine

If pins and needles are present in the upper extremities, a full examination of the cervical spine must always be carried out. In thoracic outlet syndrome, the passive movements of the neck are painless and of full range. When a cervical rib is present, weakness and atrophy of the thenar, hypothenar and interosseous muscles may be found.

Tests for carpal tunnel syndrome

All the specific tests for a carpal tunnel syndrome must be carried out (see below, *Lesions in the carpal tunnel*).

Technical investigations

It should be emphasized that thoracic outlet syndrome is primarily a clinical diagnosis, based on a full history and a complete clinical examination.

Electromyography (EMG) and conduction studies are of little value, for two reasons.[56,57] First, the range is quite variable in normal patients. Second, because the stimulating electrode can not be placed proximal to the level of the compression, the compound action potential which is measured does not cross the site of the nerve compression. But EMG is helpful for differential diagnosis, in excluding nerve compression at other levels, such as ulnar nerve impingement at the elbow or carpal tunnel syndrome.

A radiograph of both the cervical spine and thorax can help to detect a cervical rib, a hypertrophic transverse process of C7 (suggesting a fibrous band) or the formation of a clavicular callus.[58] It also helps to exclude a Pancoast's tumour. A CT scan may demonstrate an abnormal fibrous band.

Angiography (arteriography and/or phlebography) must be considered but is only indicated when the vascular symptoms are so severe that surgery is contemplated. Such can be the case when signs and symptoms of arterial embolism or arterial and/or venous occlusion are present.

Differential diagnosis

Cervical disc protrusions

When a posterolateral cervical disc compresses a nerve root, the cervicobrachial pain is very severe, often worse at night, coming and going without apparent reason. Some articular movements of the cervical spine increase the pain, although, surprisingly enough, their influence may be only very slight.

In posterocentral cervical protrusion with cord compression, pins and needles are felt in both hands and feet and are brought on or increased by neck flexion.

Compression of the ulnar nerve

A lesion of the ulnar nerve provokes pins and needles felt only in the fifth finger and at the ulnar half of the fourth. In compression at the cubital tunnel, some local pain around the elbow may also occur (see p. e138 of this chapter).

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Compression of the radial nerve

Pins and needles may be felt at the dorsal aspect of the lower arm, and of the 3½ radial digits. Resisted extension of the hand is weak but painless. Sometimes a drop hand is found (see p. e132 of this chapter).

Carpal tunnel syndrome

Compression of the median nerve in the carpal tunnel causes paraesthesia felt on the palmar aspect of the thumb, index and middle finger and the radial half of the ring finger. Carpal tunnel tests may be positive, although in 50% they remain negative (see p. e155 of this chapter).

Carcinoma of the superior sulcus of the lung (Pancoast's tumour)

The coexistence of pins and needles and Pancoast's tumour implies invasion of the brachial plexus, which may give rise to a palsy of the interosseous muscles at the hand. Very often a palsy of the recurrent nerve, causing hoarseness, is also present. Clinical inspection of the face may reveal Horner's syndrome (ptosis of the upper eyelid, enophthalmia and myosis; see p. 3 of online chapter *Disorders of the thoracic cage and abdomen*).

Raynaud's syndrome

This provokes only pain, and no pins and needles. Typically the hand becomes white and then blue in cold conditions.

Treatment

Anatomical variety

Thoracic outlet syndrome due to a cervical rib or a fibrous band can only be treated surgically. Paraesthesia and pain mostly disappear but wasting and weakness seldom resolve completely.

Postural variety

Because of the lack of consensus about the aetiology of this syndrome, various forms of treatment have been described (see Box 2). In the light of the mechanism that we consider responsible for the symptoms, the following approach is proposed.

Posture and exercise

Cases caused by the first rib can be helped by conservative management. However, the first step in the treatment is a clear explanation to the patient of the pressure and release mechanism of the disorder. He or she should understand that the pins and needles at night are the result of compression during daytime and that they come on when the nerve is liberated from the pressure. He or she also should realize that, to get rid of the complaints, pressure on the nerve during the day must always be avoided.

To achieve the latter, the patient is asked to keep the shoulders slightly shrugged all day. Carrying loads and wearing heavy coats must be avoided.

For some weeks, the following daily exercise must be done in the evening. Seated in an armchair, elbows resting on the arms, both shoulders are kept shrugged passively (Fig. 11). This brings the pins and needles on after a while but the position is maintained, even if symptoms become more severe. Once they diminish and disappear spontaneously, usually in half an hour, the shoulders are let down.

If the exercise is repeated daily, the patient soon finds that the paraesthesia comes on later and later at night and then appears only in the early morning hours and, after some more weeks of exercising at night, finally disappears completely. Exercises can then be stopped. But the patient must remember to continue indefinitely to keep the shoulders slightly shrugged during the day.

As mentioned previously, it is important to explain the release phenomenon in clear terms to the patient, so that he/she understands that the pins and needles are due to the nerve being fully liberated from the pressure. Lacking such understanding, the patient will mistakenly regard the exercise as harmful and discontinue it.

Box 2

Various forms of treatment for thoracic outlet syndrome

- Active strengthening exercises of the upper extremity and neck muscles
- Shoulder shrugging exercises
- Scapular adduction and abduction exercises
- Stretching of the scaleni muscles
- Mobilizations of the sternoclavicular and acromioclavicular joints
- Mobilizations of the first and second ribs
- Sustained postures and postures of prevention during activities
- Transcutaneous electrical nerve stimulation (TENS)
- Scalenectomy
- Periarterial sympathectomy
- Removal of the first rib
- Removal of the cervical rib
- Removal of a pathological fibrous band

Fig 11 • Shoulder elevation exercise for first rib syndrome.

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Table 2 Differential diagnosis and summary of anatomical and postural varieties of thoracic outlet syndrome

	Anatomical	Postural
Age	20–30 years	Middle-aged and elderly
Release phenomenon?	At first	All the time
Pins and needles?	All day	At night
Atrophy and weakness?	Thenar/hypothenar/ interossei	None
Cold hands?	Possible	None
Cynosis and swelling?	Possible	No
Treatment	Surgery	Keeping shoulders slightly shrugged at all times; daily exercises in the evening

An orthosis designed to elevate the shoulder has been described. It has good results in 77% of patients.[59]

Surgery

In cases refractory to conservative treatment, resection of the normal first rib can be performed.[60–63] However, this is major surgery and not always successful in relieving symptoms. This approach also carries potential dangers, because of the close relationship to the brachial plexus and subclavian/axillary artery.[64,65]

Table 2 provides a summary and differential diagnosis of the anatomical and postural varieties of thoracic outlet syndrome.

Plexitis

Acute or subacute neuritis of the brachial plexus has been described under different headings: amyotrophic neuritis, neuralgic amyotrophy, Parsonage–Turner syndrome, idiopathic brachial plexus neuropathy, brachial neuritis. There is a tendency to use the term 'acute brachial plexus neuropathy'.

This rather uncommon parenchymatous disorder of the peripheral nerves, described by Parsonage and Turner,[66,67] has no specific cause.[68] However, two biopsy studies, mentioned by Stewart,[36] may clarify the pathology and pathogenesis of this syndrome. They suggest that the disorder is the result of immune-mediated nerve damage following a previous viral infection or autoimmune process.[69,70]

It may develop at any age.[71] The onset is with quite sudden central neck pain or pain in one or both scapulae. After some hours to some days the pain radiates to one or both upper limbs, sometimes as far as the hands. Even in bilateral distribution the picture is asymmetrical and paraesthesia is uncommon. The pain is extreme during the first weeks, continues for 2–3 months and then gradually diminishes. Sometimes coughing or taking a deep breath may also be painful. It takes another 2 or 3 months before the patient is comfortable.

Examination in the early stage may show that the neck movements influence the cervicoscapular pain even though the lesion is not articular, but a clear pattern is not found.

Once the acute stage of an attack is over and the initial pain has disappeared, a patchy paresis and atrophy will become evident. There is visible atrophy in shoulder and shoulder girdle[72] and isometric testing reveals gross weakness in several muscles. All kinds of combinations of weakness may be possible. A typical feature of neuralgic amyotrophy is the patchiness of the motor and sensory symptoms. Histological studies have already shown that the pathologic process can cause very focal damage to one or a few of the fascicles that make up a brachial plexus trunk or cord, while simultaneously affecting several parts of the plexus as a whole.[73,74] This is clinically reflected by a wide variety in the possible distribution – and severity – of paresis. Any part of the brachial plexus, and clinically any muscle or skin area can be involved, in all sorts of combinations. It is precisely the recognition of this patchiness that is a very important clue to the diagnosis of neuralgic amyotrophy.[75] The muscles most often involved are: serratus anterior, deltoid, supraspinatus and infraspinatus, followed in frequency by biceps and triceps. The infraspinatus muscle seems to be always affected. Sensory abnormalities are much less pronounced than pain and weakness.

Analgesics may be necessary during the pain period but the other symptoms and signs recover spontaneously. In the majority of patients, the weakness abates in the next few months after the disappearance of the pain.[76]

Disorders of the long thoracic nerve

Anatomy

The long thoracic nerve takes origin in the upper trunk of the brachial plexus from the ventral rami C5, C6 and often C7. It courses behind the brachial plexus and follows the lateral wall of the thorax where it divides into several branches (Fig. 12).

Innervation

The long thoracic nerve is a pure motor nerve and innervates the serratus anterior muscle. The serratus anterior is a broad flattened sheet of muscle originating from the first nine ribs and passes posteriorly around the thoracic wall before inserting into the costal surface of the medial border of the scapula (Fig. 13).[77] The main function of the serratus anterior is to protract and rotate the scapula, keeping it closely opposed to the thoracic wall and optimizing the position of the glenoid for maximum efficiency for upper extremity motion.[78]

Disorders

The nerve can become affected:

- As the result of iatrogenic causes, such as axillary or first rib surgery[79,80]

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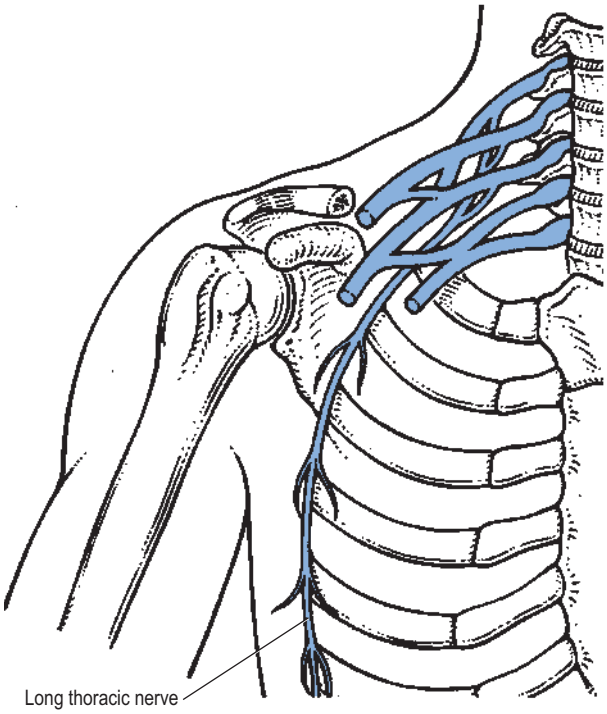

Fig 12 • Anatomy of the long thoracic nerve.

- As the result of direct compression ('backpack injuries') because of its long course along the thorax[81]
- As the result of excessive shoulder activity.[82,83] Although the association is well established, there is no consensus of exactly how trauma injures the long thoracic nerve. Work on cadavers suggests that injury may take place as the nerve exits the fascial sheath that encompasses it, either in the form of a traction injury [84] or in a 'bow-stringing' effect[85]
- As the result of idiopathic neuritis.

Mononeuropathy of the long thoracic nerve

The patient presents with pain around the affected shoulder, which either arises spontaneously or is linked to some traumatic event.[86] This pain may radiate down the arm and to the scapula. In addition, he typically complains of shoulder weakness. The pain usually resolves spontaneously over the next several weeks, but the patient is left with weakness and a winged scapula.[87]

Upon inspection, medial scapular winging is evident, with the medial and inferior borders closer to the spine and lifted superiorly when compared to the normal side.[88]

Examination of the shoulder shows a limitation on active elevation of the arm of about 45–90°. Passive movements are of full range; resisted movements are normal. The diagnostic manœuvre is to ask the patient to push against a wall with the arms stretched out horizontally in front of the body (Fig. 14).[89] In this position, the vertebral border of the scapula lifts further from the thoracic wall due to the loss of serratus anterior scapular protraction.

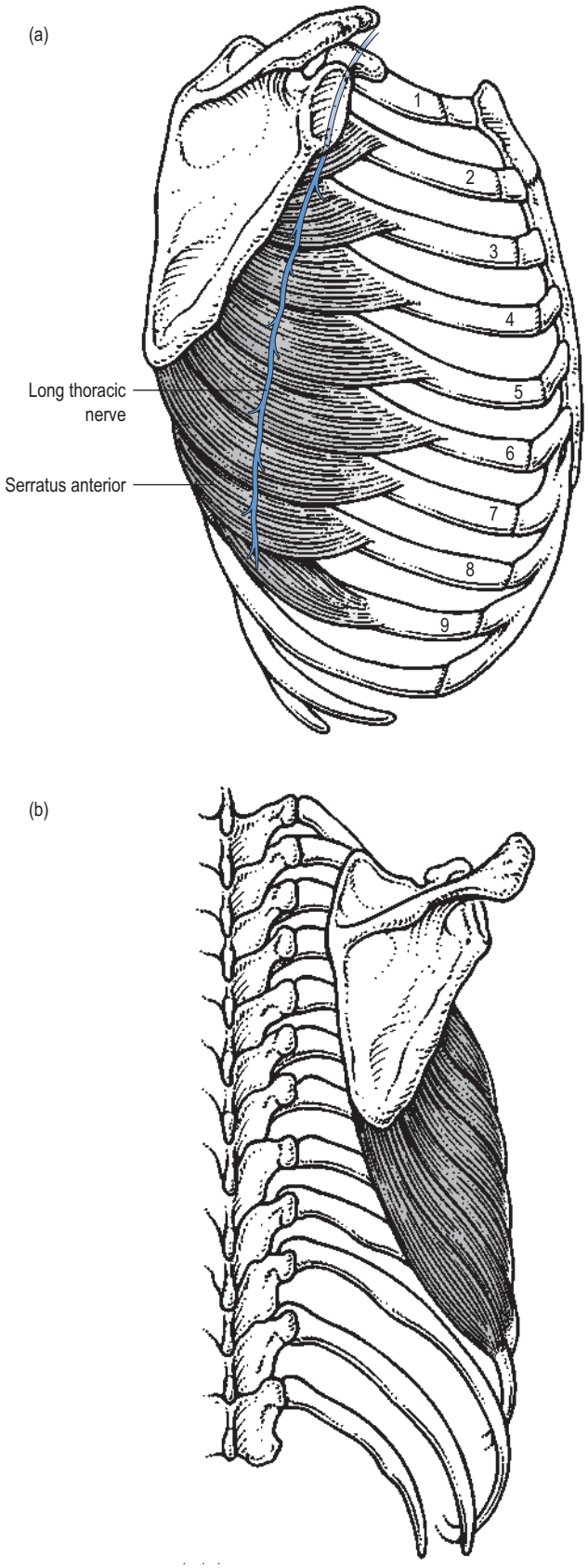

Fig 13 • Motor innervations of the serratus anterior muscle by the long thoracic nerve.

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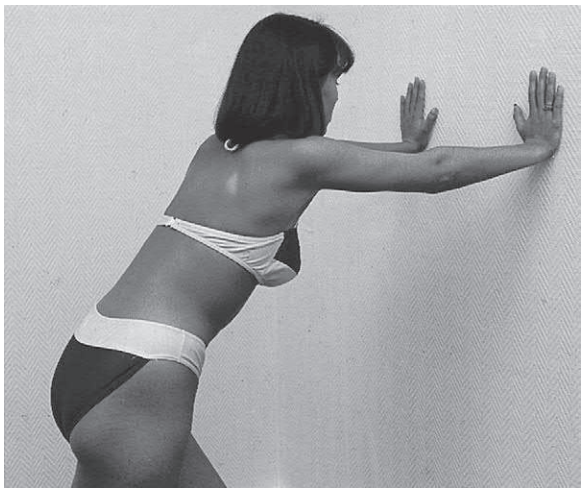

Fig 14 • Test to confirm long thoracic nerve palsy.

In most cases spontaneous recovery occurs in 4–8 months.[90] However, in approximately 25% of patients the paralysis and scapular winging will persist and such patients are then candidates for surgical reconstruction.[91,92]

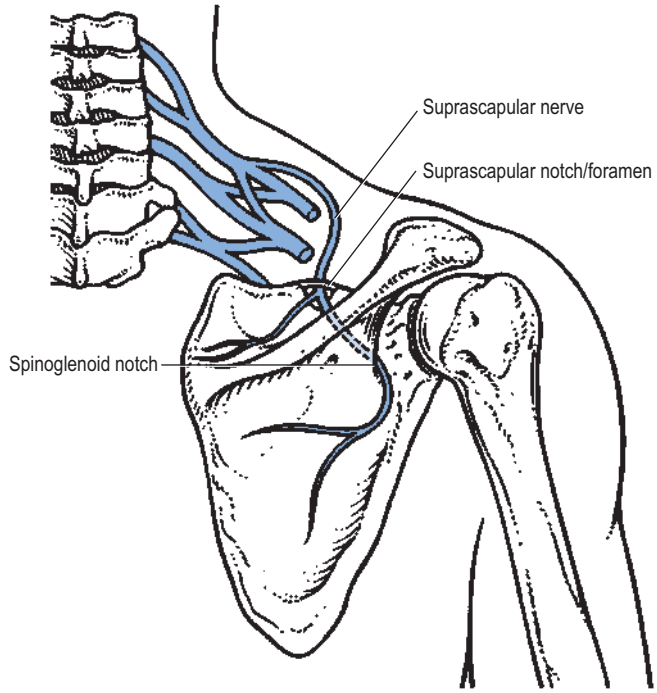

Fig 15 • Anatomy of the suprascapular nerve.

Disorders of the suprascapular nerve

Anatomy

The suprascapular nerve takes origin from the upper trunk of the brachial plexus with fibres from the C4 to C6 ventral rami. It courses through the posterior triangle of the neck, underneath the trapezius muscle. The nerve passes through the suprascapular notch of the scapula, which is bridged by the thick transverse scapular ligament[93] (Fig. 15). After entering the supraspinatus fossa the nerve gives off two motor branches to the supraspinatus muscle and then passes laterally within the fossa, providing sensory branches to the posterior capsule of the glenohumeral joint and acromioclavicular joint. It then goes around the lateral border of the base of the spinous process to the infraspinatus fossa, where the nerve terminates by supplying motor branches to the infraspinatus muscle.

Innervation

The nerve innervates the supraspinatus and infraspinatus muscles and receives sensory and proprioceptive branches from the glenohumeral and acromioclavicular joints, as well as the subacromial bursa and posterior aspect of the capsule.

Disorders

The suprascapular nerve has a short course and several sites of relative fixation, making it vulnerable to both traction and compression forces. The nerve is fixed at both its origin at the Erb point on the brachial plexus and at its terminal insertion on the infraspinatus. The nerve is relatively fixed at the suprascapular notch, and anatomical studies have shown that motion does not occur at this point.[94] Suprascapular nerve dysfunction can be caused by:

- Entrapment of the nerve by a ganglion,[95,96] or a tight ligament at the level of the suprascapular notch
- Entrapment at the spinoglenoid notch[97,98,99]
- Acute brachial plexitis (the suprascapular nerve is involved in 97% of the cases)[100]
- Trauma, e.g. shoulder luxation or scapular fracture[101]
- Idiopathic neuritis.

A lesion at *the suprascapular notch* is either the result of a ganglion cyst or a traction trauma. Downward traction of the scapula can result in opposition of the suprascapular nerve against the sharp inferior border of the transverse scapular ligament. Cross-body abduction or protraction with forward flexion, as seen in fencing, throwing sports,[102] racquet sports, and weight lifting, have also been found to maximally stretch the suprascapular nerve.

Entrapment can also occur more distally at the *spinoglenoid notch*, which is more commonly seen in athletes whose sports require rapid forceful external rotation movements, such as volleyball.[103,104] The cocking motion for the smash results in rapid external rotation of the shoulder; this rapid motion of the infraspinatus muscle is thought to pull the suprascapular nerve against the base of the scapular spine, resulting in nerve injury at this level. Injury to nerves in the spinoglenoid area has also been noted secondary to ganglion cysts.

Patients complain of a fatigue-like posterior shoulder pain which may be aggravated by activities that stretch or mobilize the nerve, such as combing one's hair or moving the scapulae.

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Patients with pathology at the spinoglenoid notch may also present with painless and isolated wasting of the infraspinatus muscle as the nerve fibres at the notch contain no sensory afferents.

Inspection shows atrophy of the supraspinatus and/or infraspinatus muscles. On clinical examination, full passive lateral rotation of the arm may provoke some pain, as may all active and passive scapular movements. Passive horizontal adduction of the arm is also painful. Depending on the level of nerve involvement, there is weakness in abduction or external rotation, or both. Proximal injury of the suprascapular nerve at the level of the suprascapular notch causes the weakness of both abduction and external rotation, while nerve injury at the level of the spinoglenoid notch affects only the infraspinatus and results in isolated weakness of external rotation.

An EMG shows a denervation of the infra- and supraspinatus which, together with the history of an injury, confirms the diagnosis. Ultrasonography can be used to show a ganglion. Magnetic resonance imaging is appropriate to diagnose entrapment.[105]

The management for suprascapular nerve entrapment includes a conservative regimen of observation, rest, analgesics, and cortisone injections into the suprascapular notch.[106,107] If this treatment is unsuccessful, surgical decompression of the nerve at the suprascapular ligament is necessary. In most patients it leads to full relief.[108,109]

Technique: injection

The patient sits on a chair. The entire spine of the scapula is marked from the medial to the lateral edge. This line is bisected and a second line drawn at a 45° angle in an anterolateral direction. A 2.5 cm mark is made on this second line and a 7 cm needle, fitted to a syringe containing 2 ml of steroid, is inserted vertically downwards at this point until it hits the bone at the base of the scapular spine (Fig. 16). The tip of the needle is then moved further anteriorly until it slips in the suprascapular notch. The steroid is injected here.

Disorders of the axillary nerve

Anatomy

The axillary nerve takes origin in the posterior cord of the brachial plexus from fibres derived from C5–C6. It curves around the neck of the humerus and passes through the quadrilateral space close to the inferior shoulder joint capsule. This quadrilateral space is bounded by the teres minor superiorly, the long head of the triceps medially, the teres major inferiorly, and the humeral neck laterally and forms a potential site of compression for the axillary nerve as it passes from the anterior to the posterior aspects of the shoulder.[110] After passing through the quadrilateral space, the axillary nerve divides into anterior and posterior branches, which supply the anterior and posterior portions of the deltoid muscle. The posterior branch of the axillary nerve also supplies the teres minor and supplies cutaneous sensation overlying the deltoid (the upper lateral cutaneous nerve of the arm). The short length of the axillary nerve renders it vulnerable to stretch injuries, especially during shoulder dislocation [111] (Fig. 17).

Innervation

The axillary nerve is mixed. The motor branches innervate the deltoid muscle and the teres minor muscle. The sensory branch innervates the skin over the deltoid region and the upper and lateral part of the arm.

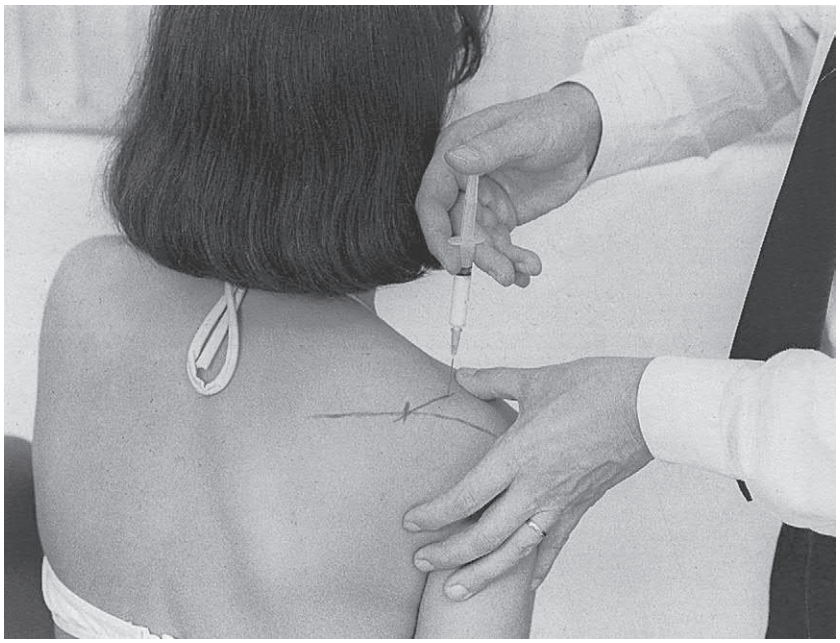

Fig 16 • Injection for suprascapular nerve entrapment.

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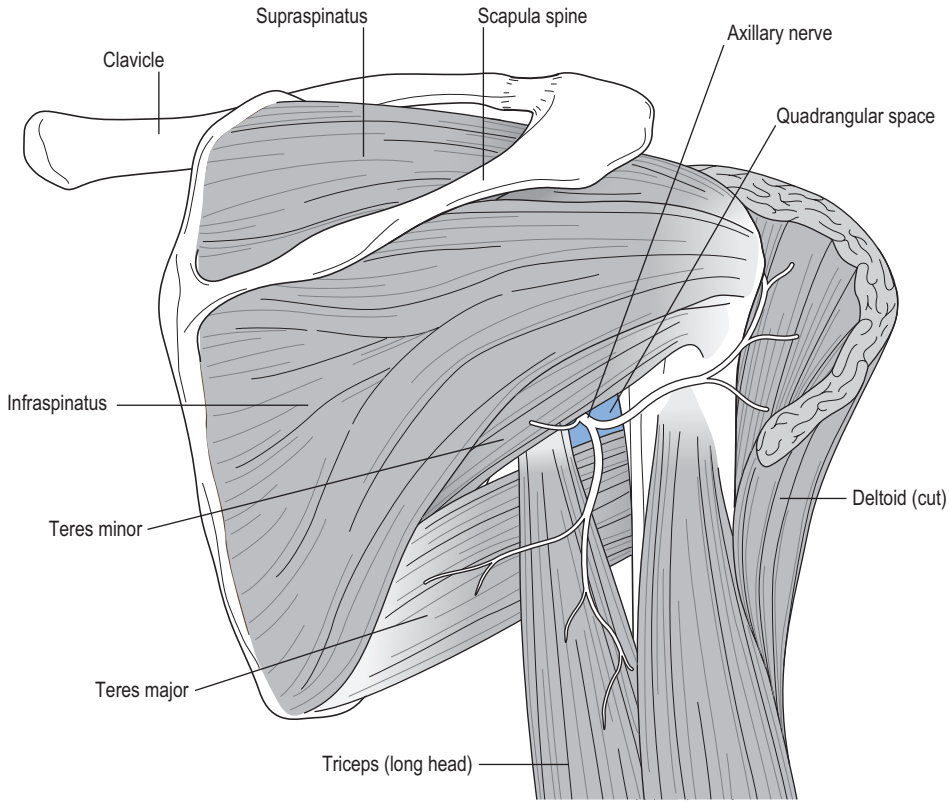

Fig 17 • The axillary nerve and its motor innervation of deltoid (1) and teres minor (2) muscles.

Disorders

- Most axillary nerve lesions occur after anterior shoulder dislocation,[73] the exact incidence ranging between 9% and 18%.[112] Blunt trauma to the anterior aspect of the shoulder without dislocation has also been implicated in axillary nerve trauma in contact sports.[113,114]
- Acute axillary neuropathy is also associated with backpacking, usually in inexperienced hikers. The cause of axillary nerve injury in 'rucksack palsy' is thought to be traction caused by depression of the shoulder from the excessively weighted backpack.
- Quadrilateral space syndrome represents a chronic compression syndrome of the axillary nerve in throwing athletes.[115] Axillary nerve entrapment then occurs insidiously in the quadrilateral space without history of trauma. Fibrous bands at the inferior edge of the teres minor have been implicated, as have randomly oriented fibrous bands found in the quadrilateral space.[116,117]
- The axillary nerve sits approximately 2 cm inferior to the usual posterior portal for arthroscopy, putting this nerve at risk during routine arthroscopic procedures.[118] An unintentional local infiltration with steroid into the axillary nerve on infiltrating too far distally for infraspinatus tendinitis may cause the same problem.

Symptoms are often vague, consisting of a dull ache and/or pins and needles over the deltoid area. Sometimes extensive visible wasting of the deltoid is present, together with an area of cutaneous analgesia in the mid-deltoid region. According to Cyriax,[119] (his p. 153) some patients tend to relax the axillary nerve as much as possible by elevating the scapula at the same side during the first few weeks after the injury. The process is a consequence of spasm of the trapezius and leads to pain on passive side flexion of the neck towards the contralateral side. Deltoid weakness is often masked by the activity of supraspinatus and pectoralis major.[120] Because the supraspinatus muscle is not involved, active elevation of the arm remains possible, and weakness is only found on resisted abduction. The diagnostic test is to ask the patient to abduct the arm to 90° and to bring it further backwards into horizontal extension (Fig. 18). This is impossible with an axillary nerve lesion.

Spontaneous cure is possible, but takes about 6 months. Care should be taken to mobilize the shoulder during the recovery so as to avoid an immobilizational arthritis. If no improvement has occurred after 6 months, surgical decompression is needed via a release of the teres minor and major tendinous insertions.[121]

Disorders of the radial nerve

Anatomy

The radial nerve takes origin in the posterior trunk of the brachial plexus and thus contains fibres from C5–T1. It reaches the lateral wall of the axilla and winds around the posterior aspect of the humerus in the groove for the radial nerve. It then pierces the lateral intermuscular septum to enter the

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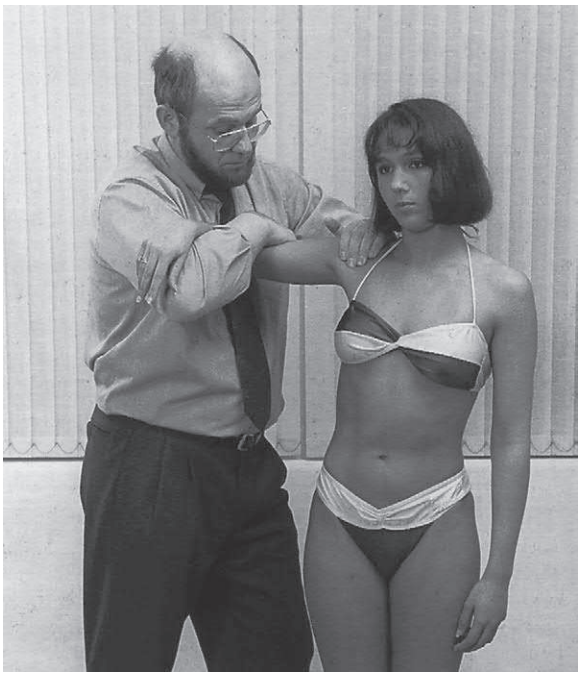

Fig 18 • Resisted horizontal extension.

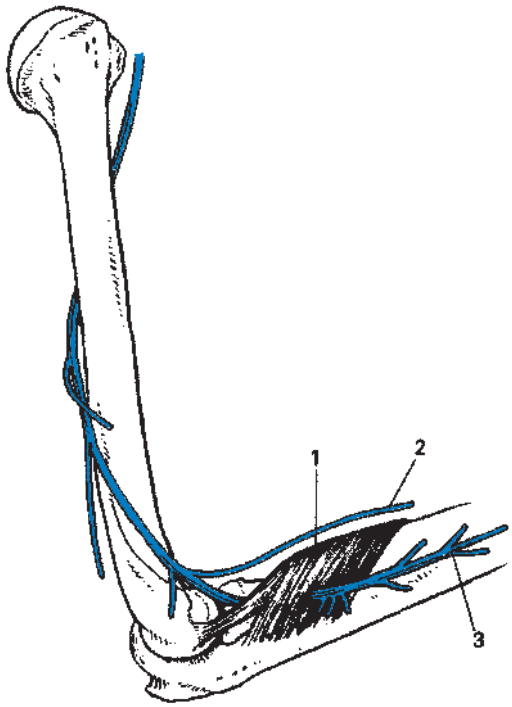

Fig 19 • The course of the radial nerve. 1, Supinator muscle; 2, superficial radial nerve; 3, posterior interosseous nerve.

anterior compartment of the arm. It lies lateral to the biceps and continues between the biceps tendon and the proximal part of the brachioradialis muscle. At the level of the elbow it divides into a superficial sensory branch (the superficial radial nerve) and a deep motor branch (the deep radial nerve). The latter continues to become the posterior interosseous nerve.

The superficial radial nerve lies under the brachioradialis muscle and follows the lateral border of the radius until at the wrist it divides into terminal digital branches to the dorsoradial part of the hand and the three radial digits.

The posterior interosseous nerve winds around the neck of the radius and goes into the dorsal compartment of the forearm. It pierces the supinator muscle through the arcade of Fröhse and runs deeply under the extensors as far as the wrist (Fig. 19).

Innervation

The motor part of the radial nerve innervates mainly the extensors of the arm and forearm: triceps and anconeus muscles, brachioradialis, extensors of the wrist, supinator and extensors of thumb and fingers.

The sensory part supplies the skin of the lateral arm, the posterior part of the forearm, the radial dorsum of the hand and the skin of the dorsal aspect of the proximal and middle phalanges of 3½ radial digits.

Disorders

The radial nerve is quite frequently affected by pathological conditions. This happens in more generalized diseases, such as poisoning by heavy metals (e.g. lead), but also in more localized lesions, either traumatic or following entrapment of the nerve tissue. The symptoms and signs depend on where, along the nerve, the lesion lies. The radial nerve may become affected at five different sites: the proximal and middle part of the upper arm, the distal part of the upper arm, the proximal part of the forearm and the distal part of the forearm (Table 3).

Lesions at the proximal and middle part of the upper arm

When the lesion results from an injury – fracture or dislocation of the humerus – it is usually combined with an axillary palsy.[101] Space-occupying lesions in the axilla or the use of old-fashioned axillary crutches are other (rare) causes.[122,123]

The full radial nerve syndrome gives rise to weakness of elbow extension (triceps, anconeus) and supination (brachioradialis, supinator), and extension of the wrist (extensor carpi radialis longus and brevis, extensor carpi ulnaris). Extension of the fingers at the metacarpophalangeal joints is also weak (extensor digitorum, extensor indicis proprius, extensor digiti minimi), as well as abduction and extension of the thumb (abductor pollicis longus, extensor pollicis longus and brevis). Sensory deficit may be found at the lateral aspect of the arm and posterior aspect of the forearm as far as the dorsum of the wrist. Paraesthesia is experienced on the dorsal aspect but not in the terminal phalanges of the thumb, index and middle fingers and the radial half of the fourth finger (Fig. 20).

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Table 3 The radial nerve and its branches

Branching off	Nerve	Supply
Upper third of upper arm	Posterior cutaneous nerve of arm (sensory)	Skin of lateral arm
Middle third of upper arm	Muscular rami (motor)	Triceps Anconeus
	Posterior cutaneous nerve of forearm (sensory)	Skin of posterior forearm
Lower third of upper arm	Muscular rami (motor)	Brachioradialis Extensor carpi radialis longus Extensor carpi radialis brevis (Brachialis)
Proximal forearm (elbow)	Superficial terminal branch (sensory) Deep terminal branch (motor)	Skin of radial dorsum of hand Supinator Extensor digitorum Extensor digiti minimi Extensor carpi ulnaris
	Posterior interosseous nerve (motor)	Abductor pollicis longus Extensor pollicis longus Extensor pollicis brevis Extensor indicis proprius
Distal forearm (wrist)	Superficial terminal branch dorsal digital nerves (sensory)	Skin of dorsal aspect of proximal and middle phalanges of 3½ radial digits

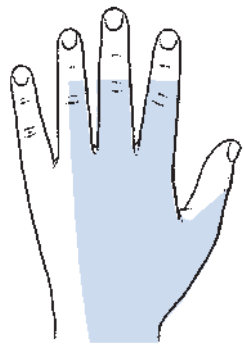

Fig 20 • Sensory supply in the hand by the radial nerve.

Examination reveals weakness of extension and supination of the elbow, together with weakness of extension of wrist, fingers and thumb. This results in a characteristic position of elbow and hand – *porter's hand* or *drop hand*. Because of the anastomoses between the posterior cutaneous nerve of the arm – first branch of the radial nerve – and other neighbouring nerves, sensory deficit is not common but when it occurs it is found at the lateral aspect of the arm and posterior aspect of the forearm as far as the dorsum of the wrist.

Differential diagnosis must be made from spinal cord, intraspinal and radicular lesions (C7), as well as conditions affecting the brachial plexus, neuritises or myopathies.

Lesions at the distal part of the upper arm

Lesions are more common at the distal than the middle and proximal part of the arm. The palsy may be traumatic – fracture of the humerus,[124,125] fracture or dislocation of the elbow – or may result from a sustained pressure just proximal to the elbow. This is typically the case in a patient who has fallen asleep with the arm over the edge of a chair or has lain all night with the arm resting against the hard edge of a bunk – 'Saturday night paralysis'. The full radial syndrome develops, except that the triceps and anconeus muscles are unaffected, as is the brachioradialis muscle. The patient awakes with a painless dropped wrist.

Sensory disturbances may occur at the dorsal aspect of the forearm when the posterior cutaneous nerve of the forearm is involved, although this area may have overlapping supply from neighbouring sensory nerves – the lateral and medial cutaneous nerves of the forearm. The superficial sensory branch of the radial nerve is responsible for cutaneous deficit at the radial and dorsal aspect of the hand, except when anastomoses with either the lateral cutaneous nerve of the forearm – a sensory branch of the musculocutaneous nerve – or the dorsal branch of the ulnar nerve at the hand provide an alternative pathway.

Lesions at the upper part of the forearm

Level with the head of the radius, the radial nerve divides in its two terminal branches – the superficial (sensory) and deep

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(motor) branches (see Fig. 19). The latter then forks off into (1) muscular rami running towards the supinator, extensor digitorum, extensor digiti minimi and extensor carpi ulnaris muscles, and (2) the posterior interrosseous nerve of the forearm, which supplies the long abductor and extensors of the thumb as well as the extensor indicis proprius. When a lesion occurs proximal to this division, symptoms of both are combined. As the branches supplying the brachioradialis, extensor carpi radialis longus and brevis, and brachialis escape the compression, there is only weakness of supination, extension of the fingers, ulnar deviation of the wrist and extension and abduction of the thumb.

Deep branch of the radial nerve

Conditions such as fracture and/or luxation of the head of the radius or local inflammatory processes, for example chronic bicipitoradial bursitis, may cause compression of the deep branch of the radial nerve where it turns around the head of the radius. The result is weakness of supination, ulnar deviation of the wrist and finger extension.

Posterior interosseous nerve of the forearm

Just below the point where the deep radial nerve comes to lie at the dorsal aspect of the forearm, the posterior interrosseous nerve of the forearm branches off and passes through the deep and superficial heads of the supinator brevis muscle[126] (Fig. 21). When the edge of the upper border of the superficial head has become fibrous, this opening forms the arcade of Fröhse,[127] also called the radial tunnel. The nerve then passes further down along the interrosseous membrane and innervates the abductor pollicis longus, extensor pollicis longus and brevis, and extensor indicis proprius muscles.

The posterior interosseous nerve can be compressed as the result of: (1) an injury, usually a fracture (or the hardware used to fix fractures),[128,129] or elbow joint dislocation;[130] (2) space-occupying lesions, such as synovial proliferations from the elbow joint in rheumatoid arthritis[131,132] or soft tissue tumours,[133,134] for example lipomas;[135] or (3) fibrous bands, which can be traumatic in origin (e.g. Volkmann's contracture) or developmental (arcade of Fröhse).[136–140]

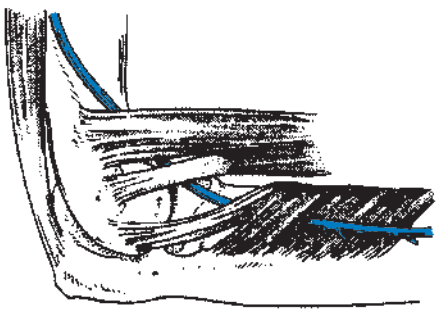

Fig 21 • Course of the posterior interosseous nerve through the supinator muscle.

Many authors believe that compression of the posterior interosseous nerve at the point where it passes through the arcade of Fröhse is a cause of lateral elbow pain and they therefore consider it as a type of tennis elbow. We do not agree that this condition should be considered to be a (resistant) type of tennis elbow, because the lesion does not lie in the extensors of the wrist. Hagert *et al*[141] also regarded epicondylitis and posterior interosseous nerve entrapment as 'two different disorders, which have nothing to do with each other, and which should therefore not be mixed up'.

Radial tunnel syndrome

In 1883, Winckworth stated that the posterior interosseous nerve could become compressed where it passes through the supinator muscle.[142] Since Roles and Maudsley described the radial tunnel syndrome in 1972,[143] this pathology has become recognized as a cause of resistant tennis elbow.[133,136,144–146] This idea is based on the reports of good results after surgical decompression of the posterior interosseous nerve at the radial tunnel.[147,148]

Nerve compression by the edge of the superficial supinator muscle seems to occur on passive pronation of the forearm. On active supination the increase in pressure is much greater, which has led to the conclusion that dynamic compression of the posterior interosseous nerve by the edge of the superficial supinator muscle is probably a cause of local nerve irritation and pain.

In compression of a predominantly motor nerve, such as the posterior interosseous nerve, the main symptom would be paralysis. Kopell and Thompson, however, state that entrapment of a motor nerve may cause diffusely localized dull aching pain.[149] The pain would originate from the nociceptive thin or non-myelinated afferent nerve fibres of muscular and extramuscular origin.[150]

However, the symptoms described by different authors are very similar to those found in tennis elbow: pain at the lateral side of the elbow, radiating distally along the posterior aspect of the forearm. The pain may be constant and can be brought on or aggravated by exertion, especially rotation movements, and the symptoms continue for some time after the causative strain has ceased. There is also diffusely localized pain on resisted supination and/or pronation as well as on resisted extension of the middle finger. Local tenderness is present over the proximal and posterior aspect of the forearm, at the suspected entrapment site. These symptoms and signs correspond with the clinical picture of what we have described as type IV (muscular) tennis elbow (see p. 313).

Treatment consists of surgical decompression. After surgery, the pain seems to disappear gradually, in the course of 6–12 months.

Werner analysed the hypothesis that posterior interosseous nerve entrapment can be a cause of lateral elbow pain.[151] The following investigations were carried out: patients with suspected posterior interosseous nerve entrapment were operated on by decompression of the nerve and then subjected to a follow-up for 2 years; the topographical anatomy was

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compared with observations at dissections; the epidemiology and symptomatology were compared with that in a series of cases of lateral epicondylitis. He concluded that lateral elbow pain may indeed be caused by dynamic compression of the posterior interosseous nerve and that it can be relieved by decompression of the nerve where it enters through the supinator muscle. The diagnosis is based solely on palpation and on positive resisted supination. Pain on resisted extension of the middle finger seems to be an unreliable test. Electrophysiological examinations (EMG) are of limited diagnostic value.

Using Cyriax's methods to examine lateral elbow pain, we have some difficulties in accepting the existence of a radial tunnel syndrome, giving rise to pain. If the main symptom is pain at the lateral aspect of the elbow and the pain is clearly aggravated by a resisted movement, one should think of a lesion of a contractile structure and try to find out what structure is at fault:

- Pain on resisted supination draws attention to the supinator brevis muscle.
- Pain on resisted extension of the middle finger does not exclude a tennis elbow, because this test also implicates the extensors of the wrist.

Differential diagnostic tests should be included (see p. 313). Pain on palpation is not reliable: the lateral aspect of the elbow is always tender to the touch and therefore the response to the palpatory manœuvres may be false-positive.

We conclude that the diagnosis of radial tunnel syndrome is probably made too often. A clear distinction should be made between the two different conditions: (1) tennis elbow, a lesion in the radial extensors of the wrist, and (2) radial tunnel syndrome (Table 4). Other authors share this view.[152,153]

We believe that what has been described in the literature as radial tunnel syndrome very often has nothing to do with the posterior interosseous nerve at all, but is simply a lesion of an extensor of the wrist, the symptoms of which have erroneously been attributed to that nerve. We speculate that the good results claimed for surgery may be the outcome of either spontaneous recovery or incidental permanent lengthening of some muscular fibres during operation. In the latter instance, the operation, ostensibly for radial tunnel syndrome, unintentionally becomes an operation for tennis elbow.

When entrapment of the posterior interosseous nerve at the elbow occurs it should give rise to a dull ache over the lateral aspect of the elbow, eventually radiating down the posterior aspect of the forearm. In due course, weakness will be found of abduction and extension of the thumb and of extension of the index finger. Paraesthesiae are absent.

Stewart confirms our view.[154] His ideas are also based on clinical findings and have been reinforced following the publication of three studies showing that, in this group of patients in whom pain is the main symptom, there is little evidence for a focal nerve lesion.[155–157]

Table 4 Differential diagnosis: type IV tennis elbow and radial tunnel syndrome

	Tennis elbow type IV	Radial tunnel syndrome
Elbow pain	Lateral pain on hand movements	Ache at elbow, especially at rest or after repetitive pronation and supination
Resisted test	Pain on wrist extension and radial deviation	Negative
Tenderness	In deep muscles overlying neck of radius	In supinator
Weakness	No muscular weakness	Possible weakness of thumb and index finger

Lesions at the distal part of the forearm

The superficial sensory branch of the radial nerve runs under the brachioradialis muscle along the radial artery and further towards the hand. At the level of the wrist it divides into five dorsal digital nerves (Fig. 22) and supplies the skin at the dorsal aspect of the thumb, index, middle and radial half of the fourth finger but not the terminal phalanges, which are supplied by the median nerve.

In the distal part of the forearm the nerve may rarely become affected as the result of external pressure (e.g. damage by handcuffs);[158,159] the result is paraesthesia and sensory deficit over the radial and dorsal aspect of the hand. If the pressure involves the medial and dorsal branch to the thumb, for example after the protracted use of small scissors, the ulnar aspect of the thumb becomes numb.

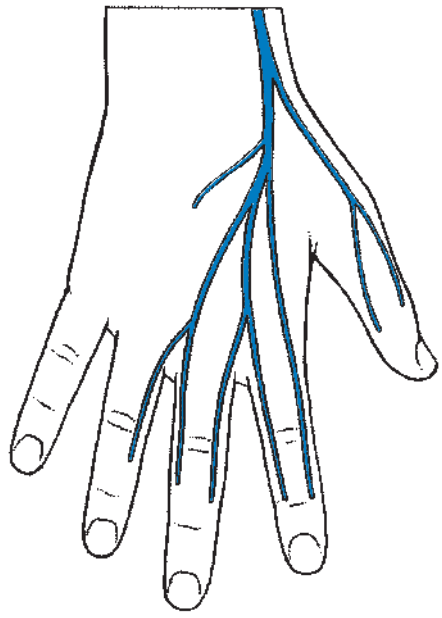

Fig 22 • Branching of the superficial sensory branch of the radial nerve.

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Disorders of the ulnar nerve

Anatomy

The ulnar nerve takes origin in the lower trunk of the brachial plexus from the spinal nerves C8–T1. It runs from the axilla down the medial aspect of the upper arm. At mid-humerus it pierces the medial intermuscular septum towards the posterior compartment. It follows the medial head of the triceps onto the retrocondylar groove at the elbow. It then courses under the aponeurotic arch joining the two heads of the flexor carpi ulnaris muscle – the cubital tunnel – and follows its course towards the wrist, under this muscle (Fig. 23). At the wrist it runs through Guyon's tunnel before it divides into its terminal branches.

Innervation

There are no branches in the upper arm. In the forearm motor branches supply the flexor carpi ulnaris muscle and the ulnar half of the deep flexor digitorum. Sensory branches supply the skin of the ulnar half of the hand. At the wrist the ulnar nerve, as it leaves Guyon's tunnel, divides into a superficial and a deep branch. The deep branch is motor and innervates the hypothenar muscles, the interossei, the two ulnar lumbricals and, on the thenar side of the hand, the adductor pollicis and the deep head of the flexor pollicis brevis. The superficial branch is sensory and supplies the skin of the little finger and the ulnar half of the ring finger.

Disorders

Ulnar nerve entrapment is one of the most frequent peripheral neuropathies, especially compression of the nerve at the level of the elbow. As it is a combined motor and sensory structure (Table 5) entrapment leads to a gamut of symptoms and signs.

The two most frequent localizations of compression are pressure in the cubital tunnel at the inner side of the elbow, and pressure in the area of Guyon's tunnel at the wrist.

The full ulnar nerve syndrome leads to weakness of the ulnar flexors of wrist and fingers (flexor carpi ulnaris, ulnar half of flexor digitorum superficialis and profundus), the hypothenar muscles (abductor digiti minimi, flexor digiti minimi brevis, opponens digiti minimi, palmaris brevis), the intrinsic muscles of the hand (dorsal and palmar interossei, ulnar two lumbricals) and part of the thenar muscles (adductor pollicis and deep head of flexor pollicis brevis). Sensory deficit is found in the ulnar half of the hand and, in the ulnar two fingers, the entire fifth finger and the ulnar half of the fourth finger.

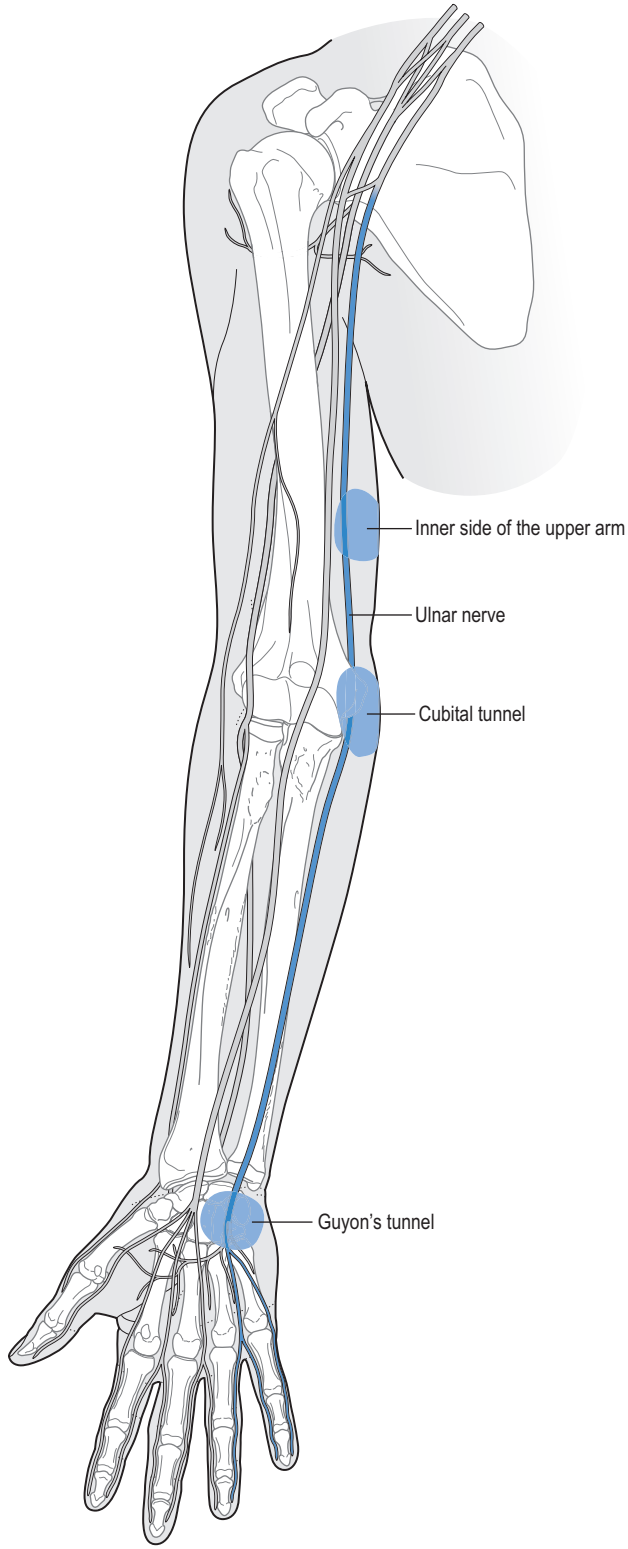

Fig 23 • Course of the ulnar nerve and the possible locations of compression:
1. Inner side of the upper arm
2. Cubital tunnel
3. Guyon's tunnel.

Lesions at the elbow

In 1877, Panas[160] was the first to describe ulnar nerve palsy, in a paper to the Académie de Médecine. Since then, several

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Table 5 The ulnar nerve and its branches

Branching off	Nerve	Supply
Upper arm	None	None
Elbow	Muscular rami (motor)	Flexor carpi ulnaris Deep flexor digitorum – ulnar half (Superficial flexor digitorum – ulnar half)
Lower forearm	Palmar cutaneous (sensory)	Skin of ulnar proximal palm of hand
Proximal to Guyon's tunnel	Dorsal digital (sensory)	Skin of ulnar dorsum of hand Skin of dorsal aspect of ulnar 1½ fingers
	Muscular ramus (motor)	Palmaris brevis
Distal to Guyon's tunnel	Muscular rami (motor)	Abductor digiti minimi Opponens digiti minimi
Hand	Superficial terminal branch (sensory)	Skin of ulnar distal palm of hand Skin of palmar (and dorsal) aspect of ulnar 1½ fingers
	Deep terminal branch (motor)	Interossei Ulnar two lumbricals Adductor pollicis Flexor pollicis brevis (deep head)

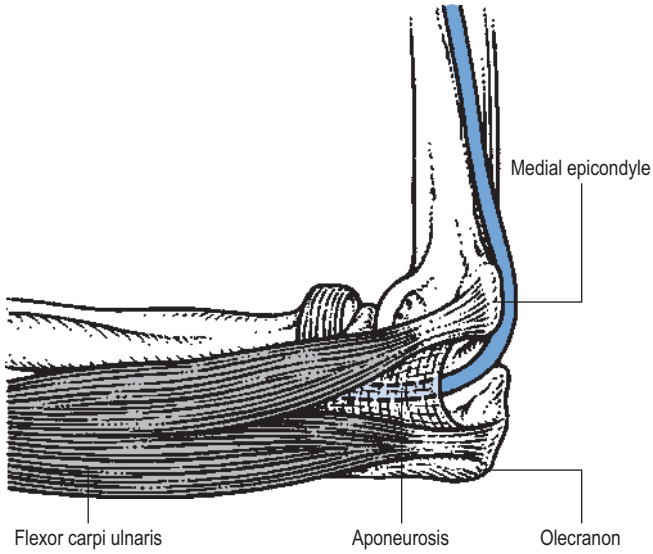

Fig 24 • The cubital tunnel.

authors have described this lesion as well as the possible surgical treatments.

The ulnar nerve courses under the medial head of the triceps muscle towards the posterior aspect of the medial epicondyle (see Fig. 23) where it lies superficially in a shallow groove behind this bone (sulcus for the ulnar nerve) and is, therefore, very vulnerable to direct contusion. It becomes stretched during flexion of the elbow. The nerve runs further distally in between the two heads of the flexor carpi ulnaris muscle, which form an aponeurotic arch. Together with the medial epicondyle, the olecranon and the medial collateral ligament, this arch forms the *cubital tunnel* (Fig. 24).

Box 3

Summary of the aetiology of ulnar nerve palsy

Friction
Traumatic
Structural
Postural
Loose body
Space-occupying lesions
Unknown

Aetiology

Entrapment of the ulnar nerve – cubital tunnel syndrome – is the second most common compressive neuropathy of the upper extremity, with only carpal tunnel syndrome presenting more frequently. Entrapment of the ulnar nerve at the elbow has several different causes, summarized in Box 3:

- *Friction* may be the result of recurrent dislocation of the nerve.[161,162] The patient notices that from time to time the nerve comes out of its groove and then replaces spontaneously. This phenomenon is sometimes accompanied by a paraesthetic twinge felt in the ulnar aspect of the hand and the ulnar one and a half fingers. Recurrent dislocation may in the end lead to ulnar palsy.
- *A fall on the elbow or a direct blow* to the medial aspect of the elbow may bruise the nerve sheath which becomes irritated. The condition is self-perpetuating, as flexion movements continue the trauma.

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- *Structural changes* may alter the stress on the nerve and thus cause friction on certain movements. These changes are excessive cubitus valgus or alterations of the position of the elbow after malunited fractures of the medial condyle.[163] Humeral trochlear hypoplasia has been described as a rare condition that may result in ulnar nerve palsy.[164]
- *Positions of prolonged flexion* (e.g. during the night) may in the end set up the known symptoms.[165] This type of ulnar nerve lesion is sometimes known as cubital tunnel syndrome.[166,167] Flexion at the elbow may have the following consequences resulting in external compression of the ulnar nerve:
  - The volume in the cubital tunnel is reduced.
  - The arcuatum ligament stretches and comes under tension as the olecranon moves away from the medial epicondyle.[168,169]
  - The ulnar collateral ligament relaxes and bulges slightly.[170]
  - The ulnar nerve elongates and is moved medially because it lies some distance from the axis of rotation of the elbow joint.
- *A loose body* causing compression of the ulnar nerve is quite uncommon. If a loose fragment in the elbow joint displaces medially at the posterior aspect of the joint, it can compress the ulnar nerve and cause paraesthesia in its territory. A non-capsular pattern will be found on examination.
- *Osteophytes, tumours or ganglia* of the nerve may compress the nerve in the cubital tunnel.
- *Idiopathic:* the aetiology very often remains unknown.[171] In as many as 50% of cases a precipitating cause is not apparent.[172]

Diagnosis

The history is obvious. The patient describes paraesthesia and/or numbness in the ulnar half of the fourth and the entire fifth finger and eventually weakness or clumsiness of the hand. A slight ache may also be felt at the elbow. A history of trauma may be elicited or merely that the symptoms came on spontaneously. In spontaneous onset, further questioning may determine whether the cause is postural.

On inspection there may be signs of joint abnormality. Furthermore, there may be wasting of the intrinsic muscles of the hand, the hypothenar muscles or some forearm muscles – flexor carpi ulnaris and deep flexors of the fingers. In severe cases, the patient may present with an ulnar palsy leading to weakness of the ulnar half of the deep flexor digitorum and of the flexor carpi ulnaris muscles, with weakness of the intrinsic hand muscles resulting in a *claw hand*.

On examination, signs are found that immediately draw attention to the ulnar nerve. A few accessory tests may give some further confirmatory information:

- The elbow is brought into maximal flexion and maintained there for a few minutes, which may bring on the pins and needles and indicates a possible postural cause.
- The elbow flexion test:[173] with the patient sitting and both shoulders and arms in the anatomical position, both elbows are fully but not forcefully flexed, with full extension of the wrist. This position is maintained for a few minutes and the patient reports to the examiner at regular intervals whether pain, numbness or tingling occur or increase.[174] When combined with pressure on the ulnar nerve the test becomes even more sensitive.[175]
- Tests for the sensitivity to light touch and two-point discrimination in the peripheral distribution of the ulnar nerve.
- Specific testing of the motor function of the abductor digiti minimi, the flexors digitorum profundi to the little and ring fingers and the flexor carpi ulnaris muscles.

On palpation of the inner aspect of the elbow, tenderness in the nerve sheath may be found and should be compared with the other elbow. A positive finding suggests frictional neuritis. Thickening of the nerve sheath indicates recurrent dislocation. Because it is not always clear whether the cause lies either at the elbow or at the wrist, tenderness should also be sought at Guyon's tunnel at the wrist.[176]

Failure to find a local cause may indicate a neuritis from metabolic problems such as diabetes mellitus, alcoholism, hypothyroidism or lead poisoning.

According to McGowan, cited by some authors,[177,178] ulnar entrapment neuropathy can be classified into three grades:

- *Grade I:* subjective symptoms combined with hypoaesthesia in ulnar fingers.
- *Grade II:* weakness and wasting of the interossei, combined with subjective symptoms.
- *Grade III:* marked weakness and wasting of the interossei, adductor pollicis and hypothenar muscles, combined with anaesthesia in the ulnar fingers.

A differential diagnosis must be made with other possible proximal causes: cervical myelopathies, anterior horn lesions, polyneuropathies, myopathies, cervical nerve root problems (C7, C8) and thoracic outlet syndrome;[179] or a more distal cause, which is compression in Guyon's tunnel.[180]

Electromyography can be useful;[181] and alternatively, an infiltration with a local anaesthetic may prove diagnostic.

Treatment

A diagnosis of cubital tunnel syndrome does not in itself necessitate surgery. In mild cases, patient education and avoidance of strains often leads to spontaneous cure.[182]

Avoidance of postural strains or pressure

The patient's daily activities should be studied to see what causes or influences symptoms. Precipitating or aggravating postures or movements which stretch or compress the ulnar nerve should be avoided. It may be necessary to explain this in some detail to the patient. Some patients can be helped by the use of a night splint, worn for several months.[183,184]

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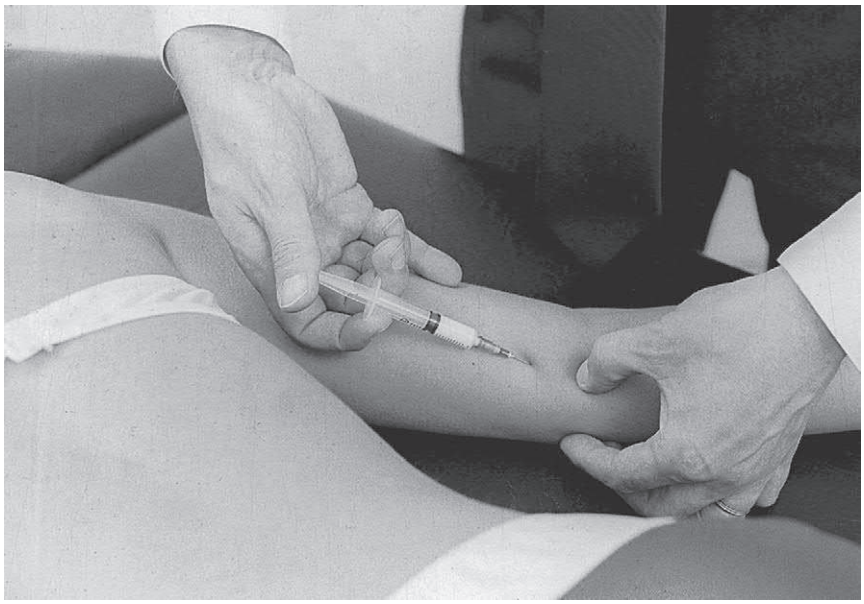

Fig 25 • Steroid infiltration for an ulnar nerve lesion at the elbow.

Infiltration

An injection of 1 ml of triamcinolone suspension about the nerve, not into it, will desensitize the nerve sheath and lead to lasting relief in those cases in which the only symptom is paraesthesia and in which conduction has not yet become impaired (Fig. 25).

Surgery

Surgical treatment is offered for more severe cases and where conservative management is deemed to have failed.[185] Simple decompression involves incising longitudinally over the cubital tunnel to release the surrounding retinacular fibres. This procedure must be performed with some care, as damage to small branches of the nerve may lead to painful neuroma.[186] Some authors believe that a release should be supplemented by medial epicondylectomy.[187,188] This eliminates the medial epicondyle as a source of compression. The remaining options involve transposition of the ulnar nerve, in which the surgeon moves the nerve anteriorly to a subcutaneous,[189,190] intramuscular[191–193] or submuscular[194] position. In the past decade, various authors have described endoscopic release of the ulnar nerve a safe and reliable treatment for the condition.[195,196]

Lesions at the wrist

Three different types of compression of the ulnar nerve at the wrist have been described: purely motor, purely sensory and a mixed form, dependent on the site of the compression.[197] Consequently, the diagnosis may not be easy. Furthermore, there is the possibility of a Martin-Grüber anastomosis (see p. e96 of online chapter *Applied anatomy of the elbow*) which again makes the diagnosis more difficult.

The ulnar nerve, together with the ulnar artery, passes through the tunnel of Guyon. This tunnel lies between two dynamic structures, the pisiform and hamate bones, and is covered by the pisohamate ligament (Fig. 26), which is a continuation of the flexor carpi ulnaris tendon.

Proximal to the wrist, the palmar cutaneous branch arises and runs over the palmar aspect of the forearm and wrist outside the tunnel of Guyon to supply the proximal part of the ulnar side of the palm. A few centimetres more distally the dorsal cutaneous branch arises and supplies the ulnar side of the dorsum of the hand, the dorsal aspect of the fifth finger and the ulnar half of the fourth finger (see Fig. 26).

As it leaves the tunnel of Guyon, the nerve divides into a mainly sensory superficial terminal branch, which supplies the distal ulnar border of the palm of the hand and the palmar surfaces of the fifth and ulnar half of the fourth finger (Fig. 27), and a deep terminal branch, which is entirely motor and innervates nearly all of the small muscles of the hand: palmaris brevis, abductor digiti minimi, opponens digiti minimi, flexor digiti minimi brevis, dorsal interossei, palmar interossei, third and fourth lumbricals, adductor pollicis and the deep head of the flexor pollicis brevis.[198]

Aetiology

The cause may be either intrinsic or extrinsic. Intrinsic causes are a ganglion, the most common cause;[199] a lipoma; an abnormal position of the abductor digiti minimi muscle;[200] or anatomical variation in the flexor carpi ulnaris tendon.[201] Extrinsic conditions are an injury with or without fractures of the pisiform or hamate bones and professional or sporting overuse (handlebar palsy).[202–204]

Symptoms

The symptoms may be compared with those resulting after compression of the nerve at the elbow. Because the ulnar nerve divides into a superficial and a deep branch at the wrist, the

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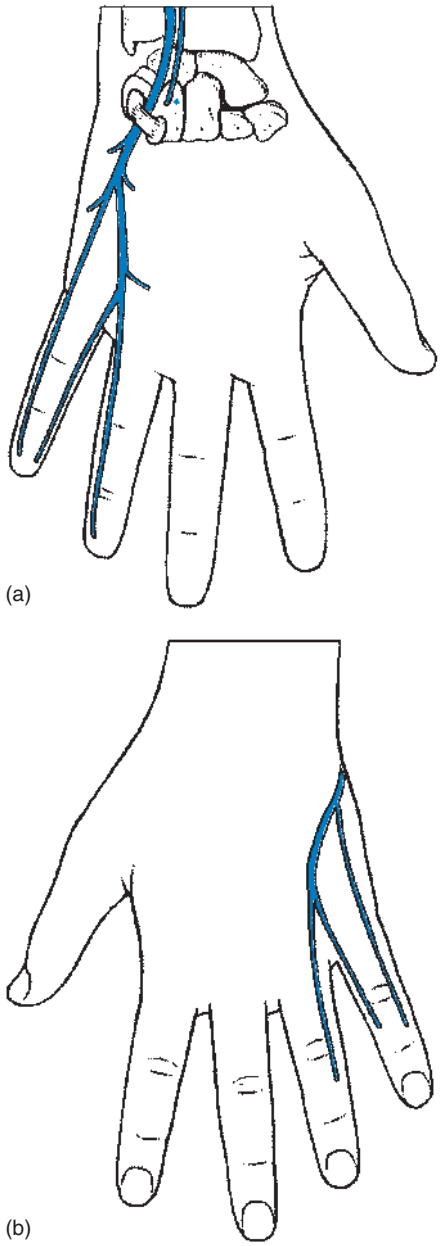

Fig 26 • The ulnar nerve passes through the tunnel of Guyon (a); but the dorsal cutaneous branch (b) does not.

symptoms may be purely motor (deep branch) or purely sensory (superficial branch). Sensation over the dorsal aspect of the fingers remains unaltered, because the dorsal sensory branch has an origin proximal to the wrist (see Fig. 26).

Four localizations are possible:[205]

- *Proximal to Guyon's tunnel*, at the pisiform bone. This causes compression of the superficial and deep branches with sensory and motor deficit (hypothenar and intrinsic hand muscles).
- *Within Guyon's tunnel*, with compression of the deep motor branch, which results in a motor deficit of the hypothenar and intrinsic hand muscles. There is no sensory deficit.

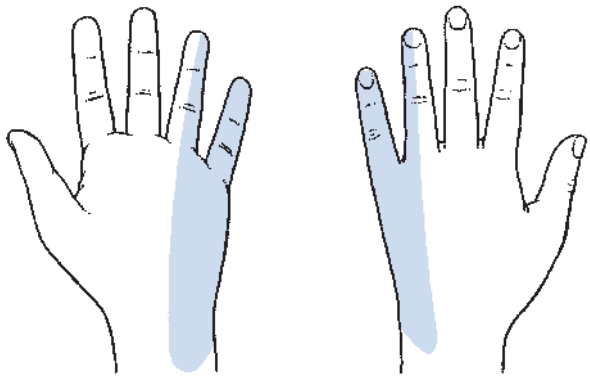

Fig 27 • Sensory supply in the hand by the ulnar nerve.

- *Distal to Guyon's tunnel*, at the hook of the hamate bone; as a result of compression of the deep palmar branch the intrinsic hand muscles are weak.
- *In the palmaris brevis muscle at the distal part of Guyon's tunnel*; the superficial branch is compressed and gives rise to a purely sensory deficit.

A fifth type of compression with motor deficit of the first dorsal interosseous and the adductor pollicis muscles has been reported by Yu-Sung *et al*.[206]

Treatment

This depends on the cause and severity of the lesion. Very often rest and avoidance of the causative strain can be sufficient. In more severe cases, local infiltration with a steroid suspension or surgical decompression may be necessary.

Disorders of the median nerve

Anatomy

The median nerve arises from the junction of the medial and lateral cords of the brachial plexus and thus from the segments C5–T1. In the upper arm the nerve lies superficial to the brachial muscle and arches around the brachial artery. In the proximal part of the forearm the nerve leaves the artery and innervates the pronator teres, flexor carpi radialis, palmaris longus, and flexor digitorum superficialis muscles. It then passes under the bicipital aponeurosis and between the two heads of the pronator teres to enter the anterior compartment of the lower arm. Just distal to that point, it gives off the anterior interosseous (ante-brachial) nerve branch that supplies the flexor digitorum profundus, flexor pollicis longus, and pronator quadratus muscles. The nerve then courses between the flexor digitorum superficialis and profundus muscles. As the muscles change to the tendons in the lower half of the arm, the median nerve runs with the tendons of index and middle fingers to the carpal tunnel. Before passing under the flexor retinaculum and into the carpal tunnel, it gives off the

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superficial palmar branch. Distal to the carpal tunnel, it divides into its terminal branches to: the thenar, the radial lumbricals and the palmar aspect and the radial side of the hand and the 3½ radial fingers (Fig. 28).

Innervation

There are no branches in the upper arm. Just distal to the elbow the first branches arise for the pronator teres, the flexor carpi radialis, the palmaris longus and the superficial flexor digitorum. The anterior interosseous nerve of the forearm innervates the pronator quadratus, the flexor pollicis longus and the radial half of the deep flexor digitorum. A sensory branch innervates the skin over the thenar and the radial half of the palm of the hand. After passing the carpal tunnel the digital nerves divide to supply the skin over the 3½ radial digits and the muscles of the thenar eminence, except those innervated by the ulnar nerve (see Fig. 28).

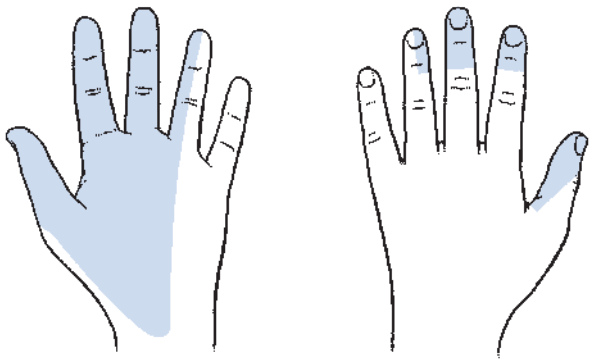

Fig 28 • Sensory supply by the median nerve.

Disorders

The median nerve (Table 6, Fig. 29) is the most important nerve of the hand, because it renders opposition of the thumb possible, combined with a circular pronation movement, as well as flexion of the radial fingers. Palsy of this nerve leads to total incapacity of the hand.

The full median nerve syndrome affects the pronator teres, the flexor carpi radialis, the radial half of the flexor digitorum superficialis and profundus, the flexor pollicis longus and the pronator quadratus, the thenar (abductor pollicis brevis, superficial head of the flexor pollicis brevis, opponens pollicis) and the radial two lumbricals. The sensory deficit is detected in the radial half of the palm of the hand, the palmar aspect of the thumb, index and middle fingers and the radial half of the ring finger, as well as the dorsal aspect of the distal phalanges of the same fingers.

Lesions at the lower part of the arm and around the elbow

The median nerve can become damaged as the result of supracondylar fractures or elbow dislocation. It can also become compressed above the elbow by a supracondylar process and the ligament of Struthers, if the latter is present. Thickening or fibrosis of the bicipital aponeurosis may also cause

Table 6 The median nerve and its branches

Branching off	Nerve	Supply
Elbow	Muscular rami	Pronator teres Flexor carpi radialis Palmaris longus Superficial flexor digitorum
Upper forearm	Anterior interosseous	Flexor pollicis longus Deep flexor digitorum (radial half) Pronator quadratus
Wrist	Palmar cutaneous	Skin of radial palmar aspect of hand
Hand	Common palmar digital Motor palmar digital branch I	Abductor pollicis brevis Flexor pollicis brevis (superficial head) Opponens pollicis Lumbrical I
	Motor palmar digital branches II–III	Lumbrical II
	Sensory palmar digital branches I–III	Skin of palmar aspect of 3½ radial digits Skin of dorsal aspects of terminal phalanges of 3½ radial digits

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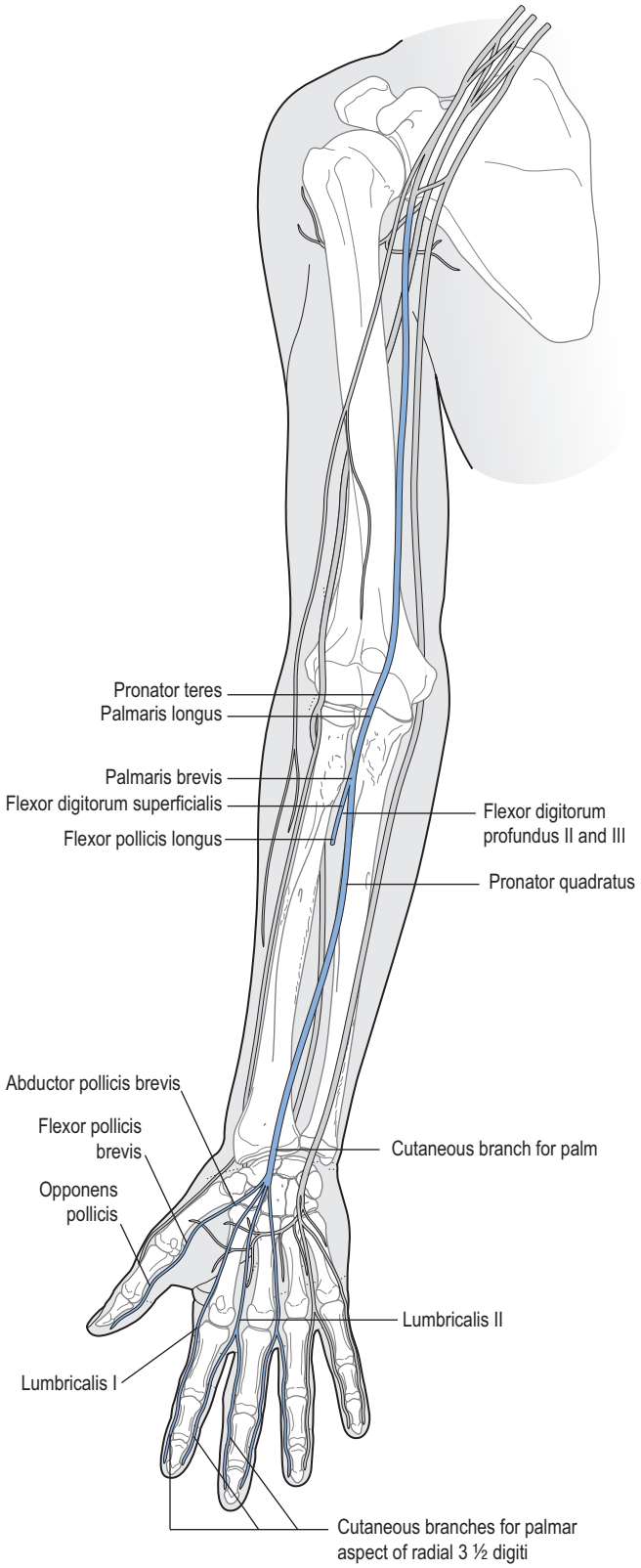

Fig 29 • The median nerve innervates the following structures:

1. Pronator teres
2. Palmaris longus
3. Palmaris brevis
4. Flexor digitorum superficialis
5. Flexor digitorum profundus II and III
6. Flexor pollicis longus
7. Pronator quadratus
8. Cutaneous branch for palm
9. Abductor pollicis brevis
10. Flexor pollicis brevis
11. Opponens pollicis
12. Lumbricalis I
13. Lumbricalis II
14. Cutaneous branches for the palmar aspect of radial 3½ digiti

compression. Below the elbow, the problem is most common at the point where the median nerve and its anterior interosseous branch dip between the two heads of the pronator teres. Thickening of the fascia that holds these two heads together can cause compression – the pronator teres syndrome.[207,208]

Pronator syndrome results from entrapment or compression of the median nerve between the humeral (superficial) and the ulnar (deep) heads of the pronator teres muscle, at the bicipital aponeurosis (lacertus fibrosus), or at the arch of the origin of the flexor digitorum superficialis (Fig. 30). Compression and entrapment may result from anatomic constraints due to congenital abnormalities in the involved tendons or muscles, such as hypertrophy of the pronator teres muscle bellies or aponeurotic prolongation of the biceps brachii muscle.[209] These conditions may be clinically silent for years and then suddenly become evident after repetitive pronation–supination stress. Less common causes of pronator syndrome include post-traumatic haematoma, soft-tissue masses and prolonged external compression.

The clinical picture includes weakness of the median innervated muscles distal to the pronator teres muscle – the flexor pollicis longus and the thenar muscles. Sensory deficit is often present. Pain is certainly not the main complaint.

The following accessory tests have been described:

- A resisted pronation during thirty seconds may provoke the symptoms.
- Sustained isometric flexion of the middle finger causes the symptoms
- Tinel's sign: percussion in the area of the pronator teres muscle.

Infiltration of steroid solution in the area of compression is usually curative. If not, surgical release is performed.

Pronator teres syndrome is probably a tunnel syndrome that is easily overestimated. The picture should be confirmed objectively by sensory and electrophysiological examination.[210] When the tentative diagnosis cannot be confirmed, possible compartment syndrome or lesion of the pronator teres muscle should be considered.

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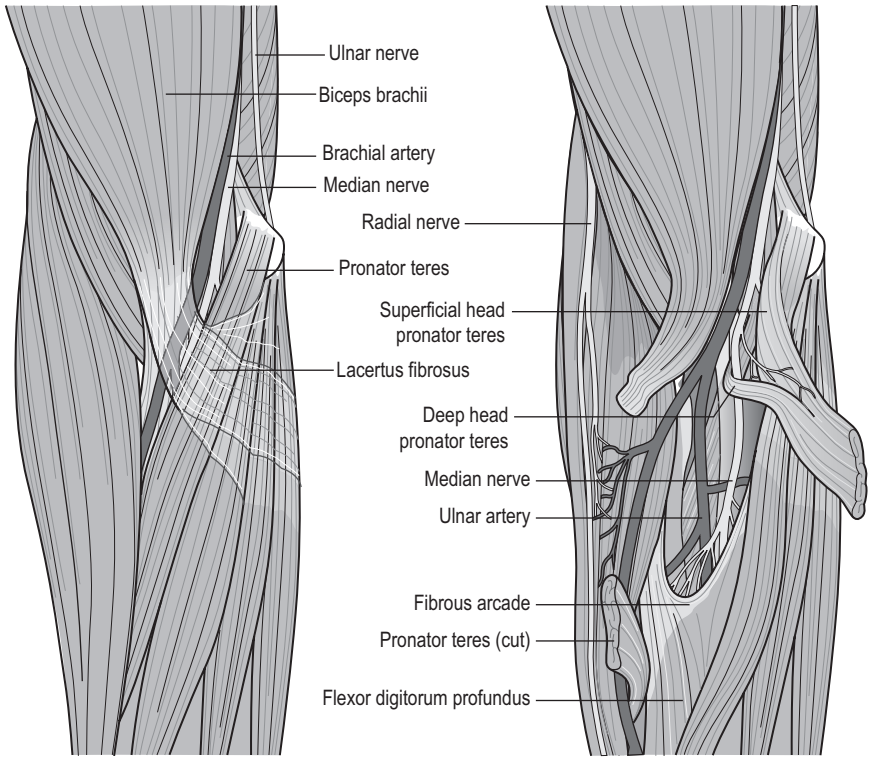

Fig 30 • Pronator teres syndrome.
Entrapment sites:
1. Lacertus fibrosus
2. The two heads (superficial and deep) of the pronator teres
3. The fibrous arcade, formed by the two heads of the flexor digitorum profundus

Lesions at the forearm: anterior interosseous nerve

Compression of the anterior interosseous nerve – a branch of the median nerve – is known as the Kiloh–Nevin syndrome.[211,212] The most frequent causes of anterior interosseous nerve syndrome are direct traumatic damage and external compression. Traumatic nerve damage may be the result of surgery, venous puncture and injection. External compression of the anterior interosseous nerve may be caused by a soft-tissue mass such as lipoma or ganglion, an accessory muscle, a vascular abnormality or cast pressure.[213]

Clinical findings

Typically, patients with anterior interosseous nerve syndrome experience a dull pain in the volar aspect of the proximal forearm, which sometimes worsens at night, combined with an acute onset of muscle weakness. The muscle weakness affects the thumb, the index finger, and occasionally the middle finger because the deep flexor muscles of these fingers are innervated by the anterior interosseous nerve. Since the anterior interosseous nerve does not innervate the skin, numbness is not associated with the syndrome.

Patients with anterior interosseous nerve syndrome have difficulties in writing and are not able to form an 'O' with the thumb and index finger. This characteristic finding, called the circle sign, is due to a lack of innervation of the flexor pollicis longus muscle and the flexor digitorum profundus muscles: the distal interphalangeal joints stay in extension during the pinch. Diagnosis must be based on clinical findings[214–217] and electrodiagnostic studies.[218]

Lesions in the carpal tunnel

The palmar aspect of the carpus is concave and covered with the transverse carpal ligament. An osteofibrous canal, the carpal tunnel, is thus formed, of which the boundaries are the scaphoid and pisiform bones proximally and the trapezium and hamate bones distally. The canal thus lies more distally and more towards the ulnar aspect than is often thought (Fig. 31).

Within the carpal tunnel are found the median nerve, the tendons of the flexor carpi radialis and the flexor pollicis longus, in separate sheets, and the superficial and deep flexors of the digits within a common tendon sheath (Fig. 32).

Carpal tunnel syndrome is the most common peripheral entrapment neuropathy[219,220] with an incidence between 1.5 and 3.5 per 1000 subjects per year.[221–223] It affects mostly women (68%) between 40 and 60 years.[224]

While first recognized by Sir James Paget in 1854,[225] it was not until 1942 that Cyriax[226] described irritation of the median nerve under the heading 'median perineuritis'. The condition remained further unrecognized until 1946, when more detailed description was given.[227,228] Full appreciation of how common the lesion is followed Phalen's article.[229]

Aetiology

Clinical carpal tunnel syndrome is caused by a raised intracarpal pressure which results from an increase in content or a

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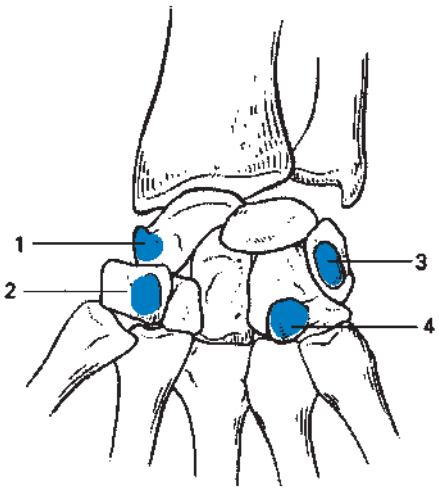

Fig 31 • The boundaries of the carpal tunnel (right hand, palmar view): 1, scaphoid; 2, trapezium; 3, pisiform; 4, hamate.

decrease in size of the carpal canal, or some combination of the two. The cause may either be local or systemic. On the basis of observation, two types are distinguished: a full syndrome (symptoms in the entire territory of the nerve) and a partial syndrome (symptoms in only a part of the nerve's territory) (Box 4).

Full syndrome

Idiopathic

Half of all cases are idiopathic.[230] Recent research suggests a multi-factorial cause. Ultrasound imaging of the carpal tunnel during dynamic stress testing manœuvres demonstrates notching, flattening, or compression of the nerve. The roof of the tunnel tightens and lowers at the same time the floor (flexor tendons) of the tunnel tightens and raises, thereby compressing the nerve.[231,232]

Traumatic

Subluxation of the lunate bone following a (possibly minor) trauma suddenly fixes the wrist in flexion, immediately

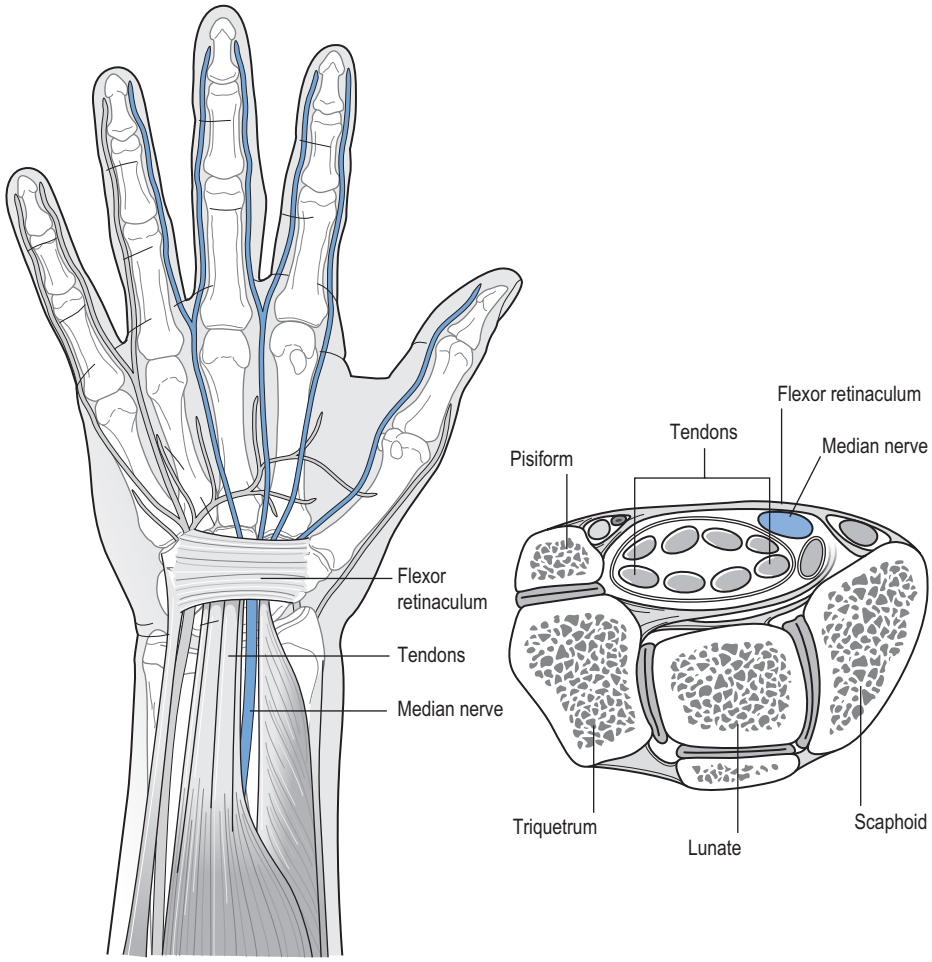

Fig 32 • Transverse section of the carpal tunnel through the proximal border (pisiform – scaphoid)

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Box 4

Aetiology of carpal tunnel syndrome

Full syndrome

- Idiopathic
- Traumatic
  - Subluxation of lunate bone
  - Occupational
  - Friction
- Inflammatory
  - Rheumatoid arthritis
  - Gout/pseudogout
  - Amyloidosis
  - Haematoma
  - Rubellar polyarthritis
- Endocrine
  - Hypothyroidism
  - Acromegaly
  - Pregnancy
  - Menopause
- Generalized peripheral neuropathy
  - Diabetes
  - Uraemia
- Tumours
  - Ganglia
  - Benign tumours: lipofibroma; neuroma
  - Malignant tumours
- Vascular
  - Haemorrhage: haemophiliacs; anticoagulants
  - Thrombosis of median artery

Partial syndromes

- Traumatic
- 'Stick palsy'
- Trigger finger

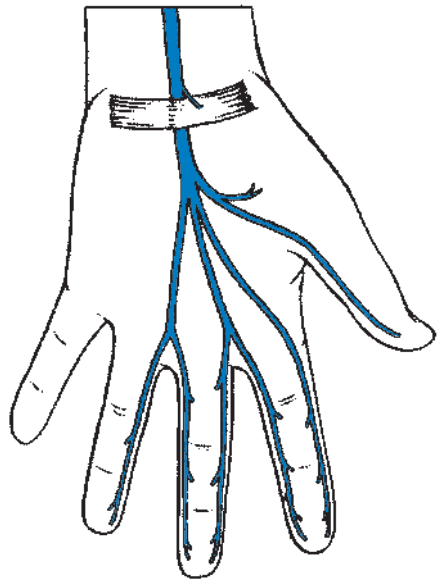

Fig 33 • The median nerve passes through the carpal tunnel to mainly supply the palm of 3½ digits.

followed by paraesthesia in the territory of the median nerve. After some days the patient also experiences numbness and some weeks later may even develop severe weakness of the thenar muscles.

It has been stated that occupations involving exposure to high pressure, high force, vibrating tools and repetitive work (in particular frequent flexion and extension and sustained force of the wrist) increase the risk for carpal tunnel syndrome (CTS).[233,234] With respect to keyboard use, however, the frequency of CTS in computer users seems to be similar to that of the general population.[235,236]

Friction of the nerve may also occur after wrist fracture, especially a Colles' fracture, as it becomes caught either by the callus or by the displaced bone.

Inflammatory

This occurs in conditions causing thickening of the transverse carpal ligament and the tendons in the carpal tunnel, such as rheumatoid arthritis.[237] Gout and pseudogout are also possible causes. The former gives rise to so much swelling that the nerve becomes severely compressed; the main symptom is then cutaneous analgesia. Other causes are amyloidosis, haematoma and rubellar polyarthritis.

Endocrine

Possible causes are hypothyroidism,[238] with accumulation of myxomatous deposits within the carpal tunnel;[239] acromegaly;[240,241] pregnancy[242] (20% of pregnant women develop a carpal tunnel syndrome after the 6th month[243]); and menopause.

Generalized peripheral neuropathy

This can occur in patients suffering from diabetes[244] or uraemia.

Space occupying lesions

These can also cause median nerve compression.[245] There are reports of tumorous conditions, hypertrophy of tenosynoviums,[246] tophaceous gout[247] and ganglia[248] that raise the intracarpal pressure and cause a carpal tunnel syndrome.

Vascular conditions

These include conditions such as spontaneous bleeding in haemophiliacs and in patients on anticoagulant therapy; thrombosis of a congenital median artery may also damage the median nerve.

Partial syndromes

These occur when pressure is exerted on one or several of the most distal terminal branches of the nerve. This can be the result of an injury, incorrect use of a walking stick or a trigger finger (see p. e149 of this chapter).

History and symptoms

The condition starts in one hand, very often the dominant one, but becomes bilateral in 30% of cases. The first symptoms are *pins and needles* felt in the territory of the median nerve (see Figs 28 and 33): the palmar aspect of the thumb, index, middle and radial half of the ring finger, as well as the dorsal aspect of the distal phalanges of these fingers. Symptoms often are worse at night and are exacerbated by repetitive flexion and extension of the wrist, strenuous gripping, or exposure to vibration. Nocturnal paraesthesiae rarely occur as the only symptom but are mentioned more often in combination with the other complaints (see below). At onset, the symptoms may be vague and diffuse and may involve only one finger – usually the middle one. After a while, the typical distribution becomes clear.

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After some months a burning *wrist pain* develops, which may radiate distally into the fingers and proximally to the forearm.[249] The pins and needles, however, do not have this proximal reference but remain distal to the point of compression.

Slight *numbness* in the radial fingers is sometimes present and, therefore, mentioned by the patient but it is often only detected when sensory deficit is sought during the examination.

In some long-standing or severe cases, the patient may complain of *weakness* and inability to use the hand: things slip from his or her fingers without noticing (loss of grip, dropping things).

In the assessment of carpal tunnel syndrome the history is most relevant. The precision of the patient's account very strongly implies the possibility of the presence of the syndrome. The area of paraesthesia described is exactly that of the median nerve and mention is made of the intermittent appearance and activity dependence of the symptoms.

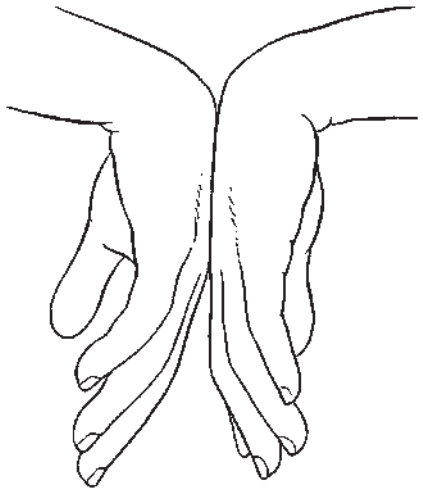

Fig 34 • Phalen's test.

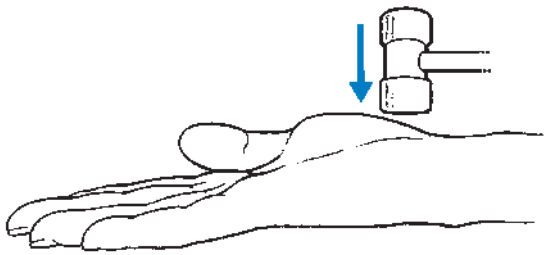

Fig 35 • Tinel's test.

Findings during inspection and examination

Inspection may reveal structural changes – post-fracture, deformities in rheumatoid arthritis, ganglia, bony subluxations – conditions that could be relevant when the overall clinical picture is considered.

In long-standing cases, and very occasionally early in the course of the condition, muscular atrophy of the thenar eminence may occur, involving the opponens pollicis and abductor pollicis brevis muscles.[250] The lateral aspect of the thenar eminence becomes flattened and gives rise to a so-called ape hand.

The standard examination of the wrist is usually negative and some accessory tests may then be performed:

- *The carpal compression test*:[251] the wrist is held in supination and extension. The examiner presses at the carpal tunnel while the patient actively flexes and extends his fingers. A positive test is indicated by paraesthesia in the 3½ fingers.
- *Phalen's test*:[229,252] the wrist is kept passively flexed for a minute. A positive test is indicated by pins and needles in thumb, index finger, middle finger and radial half of the ring finger (Fig. 34).
- *Modified Phalen's test*:[253] the wrist is held passively flexed and the thumb, index and long fingers are forcefully flexed as well.
- *Tinel's test*:[254] percussion of the carpal tunnel gives rise to pins and needles felt in the median nerve distribution (Fig. 35).

In longstanding and/or severe compression, some weakness of the thumb can be detected: abduction (abductor pollicis brevis), flexion (flexor pollicis brevis, superficial head) and opposition (opponens pollicis) may be weak.

The two main physical examination signs, Tinel sign and Phalen sign, have only a moderate sensitivity (20 to 70%) and specificity (70 to 83%).[255,256,257] However, in combination with the typical history and localization of the symptoms, they give a useful clinical indication of the probability of a carpal tunnel syndrome. The best way to confirm the tentative diagnosis is a trial injection with triamcinolone which should ease the symptoms for at least 2 weeks.

Technical investigations

During the last decades, nerve conduction studies have been considered the gold standard test for the confirmation of diagnosis of carpal tunnel syndrome with sensitivities between 49% and 84%, and specificities between 90% and 95%.[258,259] Thus, approximately 10% of patients suspected of having clinical carpal tunnel syndrome have normal electrodiagnostic studies.[260] Moreover, abnormal results in a working asymptomatic population occur between 9 and 16%.[261] Therefore, some authors advise the use of the clinical diagnosis for carpal tunnel syndrome as the criterion standard.[262,263] For the majority of patients who are considered to have carpal tunnel syndrome on the basis of their history and physical examination alone, electrodiagnostic tests do not change the probability of diagnosing this condition to an extent that is clinically relevant.[264]

Attempts have been made with cross-sectional imaging of the carpal tunnel, both with CT and MRI.[265] However, the

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sensitivity and specificity of both techniques are low (sensitivity, 23%–96%; specificity, 39%–87%), and for this reason they do not play a role in the clinical assessment of carpal tunnel syndrome.[266] Nevertheless, MR imaging does have clinical utility when the cause of carpal tunnel syndrome is a neoplasm (e.g. neurofibroma), arthritis, or a congenital anomaly and in evaluating the postoperative wrist.[267,268]

High-resolution sonography is a low-cost alternative to MRI, and has gained increasing popularity.[269] Sonography measures the cross-sectional area of the median nerve and bowing/flattening of the flexor retinaculum.[270] There seems to be a high degree of correlation between the US findings and the nerve conduction studies in diagnosing carpal tunnel syndrome.[271]

Diagnostic infiltration

A much simpler, and very reliable, diagnostic approach is the infiltration of 20 mg of triamcinolone suspension into the carpal tunnel. If diagnosis is right, all symptoms should disappear for at least a few weeks. When symptoms have not disappeared some days after the injection, the diagnosis must be reconsidered.

Differential diagnosis

Carpal tunnel syndrome must be differentiated from a cervical disc protrusion that compresses a nerve root and from thoracic outlet syndrome with compression of a part of the brachial plexus. Both are often difficult.

When distal paraesthesiae are the result of a cervical disc lesion, the symptoms usually come and go in an erratic fashion during the day as well as at night. They are transient and not particularly activity related. The distribution within the fingers is related to a dermatome, but the patient cannot localize the paraesthesia exactly. They are preceded and accompanied by severe root pain and often followed by segmental motor and/or sensory deficit. On examination of the cervical spine, a partial articular pattern is found. The symptoms disappear spontaneously in the course of a few weeks to a few months (see pp. 152–157).

In the early stage of thoracic outlet syndrome, pins and needles are strictly nocturnal and wake the patient – usually a middle-aged woman – after some hours' sleep as a result of the decompression of the nerve. The paraesthesia are felt in all the fingers and do not have any particular distribution. Decompression tests of the thoracic outlet are positive (see p. e122 of this chapter and Ch. 2).

Other differential diagnoses are cervical myelopathy, anterior horn lesions, polyneuropathies, myopathies and arthrosis of the trapezium–first metacarpal joint.

Treatment

Treatment is conservative and consists of one or more injections into the carpal tunnel. Surgery is called for in refractory or recurrent cases.

Wrist splint

Splinting with the wrist in the neutral position, especially at night, leads to good but possibly only temporary results in about 70% of patients.[272,273]

More recent studies show best results with a full-time splint.[274]

Injection

As a diagnosis of carpal tunnel syndrome can be made in spite of a negative examination, it remains provisional until therapeutic confirmation has been obtained. Relief, even if it is only temporary, after injection with triamcinolone acetonide is diagnostic. If paraesthesia is not relieved by injection, the diagnosis must be wrong. If the symptoms do not reappear for several months or years, a later recurrence can be treated with a further injection.

Technique: injection

The patient sits next to the couch. The physician stands medial to the affected hand, that lies palm upwards on the couch. The wrist is held in 45° of extension. The proximal border of the carpal tunnel (pisiform and scaphoid) and the palmar tendons of the wrist are easily detectable in this position. A 2 ml syringe is filled with 20 mg triamcinolone and fitted to a 5 cm needle. The tip of the needle is inserted between the tendons (usually ulnar of the palmaris longus) at a point 2 cm proximal to the proximal border of the carpal tunnel and inserted in the direction of the base of the fourth metacarpal bone. It slips, without resistance, between the tendons and under the transverse ligament into the carpal tunnel (Fig. 36). At 4 cm depth, the needle lies at the distal aspect of the carpal tunnel. Some fluid is injected and no resistance or painful sensation should be felt. The needle is then slowly withdrawn while the syringe is further emptied.

Results

All cases of carpal tunnel syndrome respond immediately,[275] but 60% recur within 3 to 6 months and at 2 years only 8% remain cured.[276] Several studies have demonstrated that for the first 3 months, local injections with steroids are equally good or better than surgical decompression for the symptomatic relief of mild carpal tunnel syndrome.[277,278–281] However, recurrences after injections are frequent[282,283] and, although steroid injections are safe and effective for temporary relief, most patients will eventually require surgery for long-term control of their symptoms.[284] We advise that if the symptoms recur more than 6 weeks after the injection, one or two further attempts may be made. When the result is still not sufficient or the recurrence is too rapid – within 6 weeks, or more than twice in 6 months – surgical treatment is required (Fig. 37).

Cases with severe symptoms and evidence of motor atrophy respond poorly.

Surgery

Carpal tunnel release is one of the most common surgical procedures with an annual incidence in the US of 134 per 100 000.[285] Although carpal tunnel release is an effective treatment, supported by high-quality evidence,[286,287] it has several disadvantages, including surgery-related pain and hand

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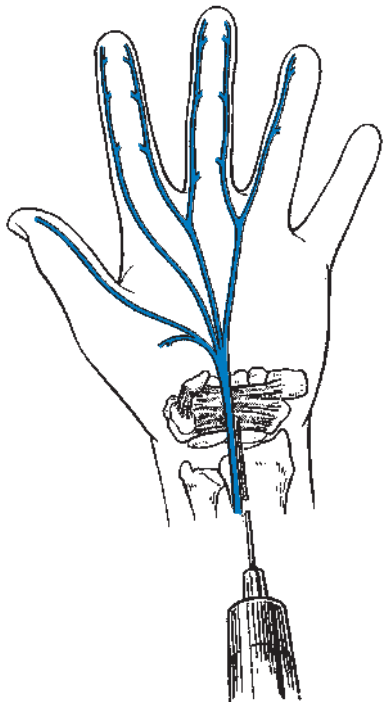

Fig 36 • Steroid injection in carpal tunnel syndrome.

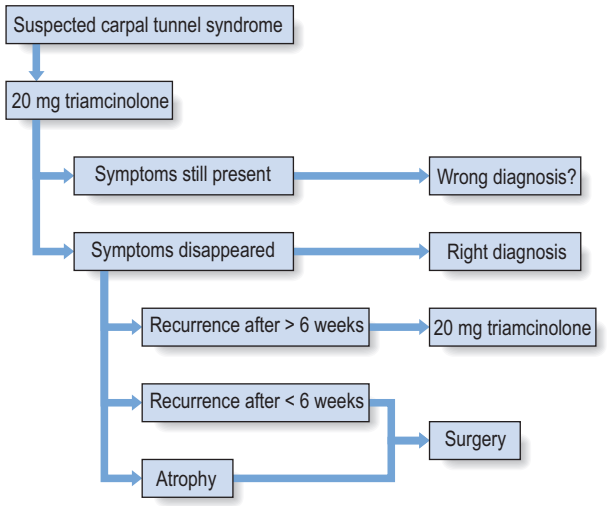

Fig 37 • Treatment of carpal tunnel syndrome.

weakness.[288,289] In addition, surgery is associated with direct and indirect costs related to work absence.[290] Therefore, patients with mild symptoms should be treated first with steroid injections. Surgery is indicated in the case of rapid or frequent recurrence[291,292] and in those patients who show objective neurologic deficit.[293–295]

The two major types of surgery are open carpal tunnel release and endoscopic carpal tunnel release (ECTR). Regardless of selection of these treatment options, the most important aspect is complete division of the flexor retinaculum. The long-term results of endoscopic release are similar to those of open release, but the former leads to quicker functional recovery.[296–298] Endoscopic release was invented to address the potential complications of the open release by using smaller incisions placed away from the middle of the palm.[299,300] It is assumed that preservation of the superficial fascia and adipose tissue over the flexor retinaculum allows faster recovery of grip strength, less scar tenderness and pillar pain, and earlier return to work.[301] However, the decision to perform ECTR is influenced by the surgeon's experience and patient factors, including occupation, socioeconomic status, and preference.[302]

Partial syndrome

Distal to the carpal tunnel the common palmar digital nerve divides into terminal motor and sensory palmar digital branches. In the hand one of these can become compressed, with the result that only part of the territory of the median nerve is involved. There are a number of possible causes.

Traumatic

As the result of a direct blow, usually a fall on the hand, the sensory palmar digital branch to the thumb at the level of its carpometacarpal joint may be bruised. The symptoms are painful pins and needles in the thumb only and there is little tendency to spontaneous cure.

'Stick palsy'

In elderly people, the sensory palmar digital branches to the index and middle fingers may become compressed in the palm of the hand, just distal to the carpal tunnel, by incorrect use of a walking stick. This results in pins and needles, felt in these two fingers. Instruction in how to use a walking stick correctly is given.

Swelling on a digital flexor tendon

A trigger lesion that has formed on the proximal part of a flexor tendon in the palm of the hand, just distal to the carpal tunnel, may rarely cause compression of the branch of the median nerve that supplies the middle and ring fingers and give rise to pins and needles at the adjacent surfaces of these two digits. Infiltration or surgery are the possible therapeutic measures.

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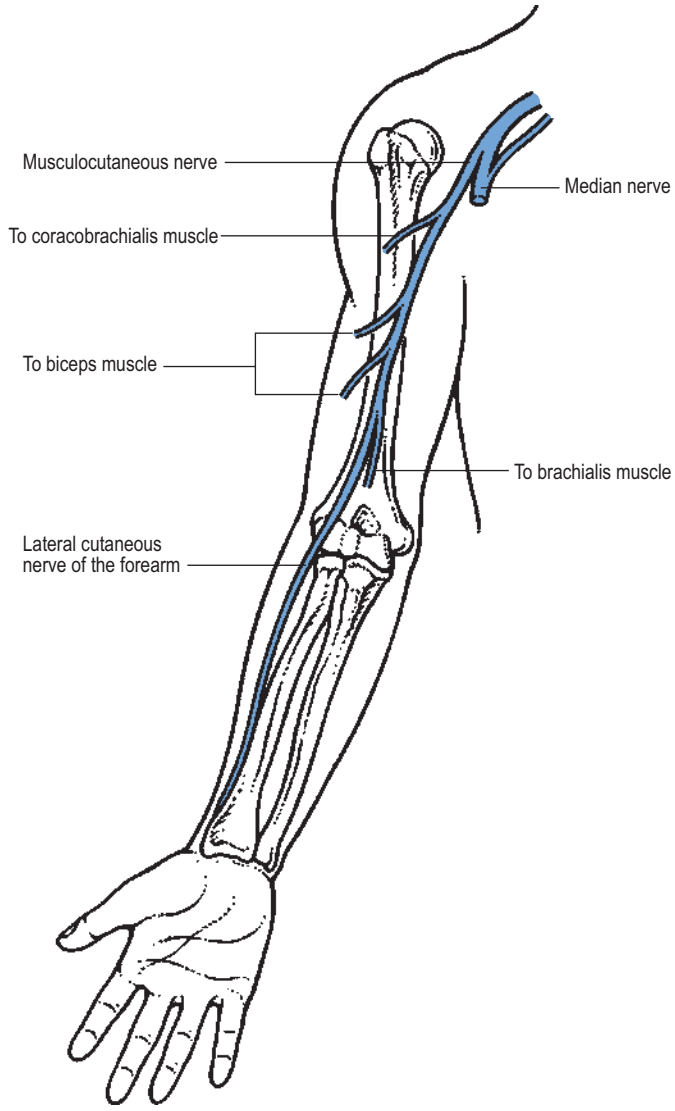

Fig 38 • The musculocutaneous nerve.

Disorders of the musculocutaneous nerve

Anatomy

The musculocutaneous nerve takes origin in the lateral cord of the brachial plexus and contains fibres from C5 to C7. It pierces the coracobrachialis muscle and descends in the anterior compartment of the arm between the biceps and brachialis muscles. It runs lateral to the distal biceps tendon and then continues as the lateral cutaneous nerve of the forearm (Fig. 38).

Innervation

The musculocutaneous nerve supplies the coracobrachialis, biceps and brachialis muscles. The lateral cutaneous nerve of the forearm innervates the skin at the anterolateral aspect of the forearm.

A lesion of the musculocutaneous nerve is rare and occurs as the result of an injury in the upper arm. This causes motor deficit with weakness of the flexors of the elbow and sensory deficit over the radial aspect of the forearm. When the lesion is at the forearm, it involves the sensory branch – the lateral cutaneous nerve of the forearm – and leads to a similar sensory deficit. This sometimes occurs after a paravenous injection.[303]

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Applied anatomy of the thorax and abdomen

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The thoracic spine

The vertebra

The thoracic spine has a primary dorsal convexity (Fig. 1) associated with intrauterine life – a phylogenetic kyphosis – whereas the cervical and lumbar spine have a compensatory lordosis.

The 12 thoracic vertebrae are intermediate in size between those in the cervical and lumbar regions. They are composed of a vertebral body – a cylindrical ventral mass of bone – continuing posteriorly into a vertebral arch (Fig. 2). The typical thoracic vertebral body is heart-shaped in cross-section and has on each of its lateral aspects a superior and inferior costal facet for articulations with the ribs (costovertebral joints). The arch is constructed out of two pedicles and two short laminae, the latter uniting posteriorly to form the spinous process. Laminae and spinous processes lie obliquely covering each other like the tiles of a roof, so protecting the posterior cord posteriorly. The pedicles carry the articular and transverse processes.

The posterior aspect of the vertebral body and the arch enclose the vertebral foramen. The spinal cord at the thoracic level is rounder and smaller than at the cervical level, and in consequence the vertebral foramina are also smaller.

Where the pedicles and laminae unite the transverse process is found, slightly posterior to the articular process, pedicle and intervertebral foramen. There is also an oval facet for the ribs on all the transverse processes, except for T11 and T12, to which ribs are not attached.

The spinous processes at mid-thorax are long and very steeply inclined: each transverse process is at a level one and a half vertebrae higher than the tip of the corresponding spinous process. In the upper and lower thorax, the spinous processes are less inclined; here, the corresponding transverse process is located approximately one level higher.

The oval intervertebral foramina are located behind the vertebral bodies and between the pedicles of the adjacent vertebrae and contain the segmental nerve roots. In the thoracic spine, these are situated mainly behind the inferoposterior aspect of the upper vertebral body and not just behind the disc. This makes a nerve root compression by a posterolateral displacement less likely at the thoracic level, whereas at the lumbar level nerve root compressions by posterolateral disc protrusions are quite common (Fig. 3, see Standring, Fig. 42.27).

The location of the intervertebral foramen depends on the level. In the upper and lower thoracic area, it is level with the tip of the spinous process of the vertebra above, i.e. level above. At mid-thorax there is a difference of about 1½ levels.

The intervertebral disc

A fibrocartilaginous disc forms the articulation between two vertebral bodies. The anatomy and behaviour of discs are discussed in Chapter 31, Applied anatomy of the lumbar spine. However, it is worth noting here that thoracic discs are narrower and flatter than those in the cervical and lumbar spine. Disc size gradually increases from superior to inferior. The

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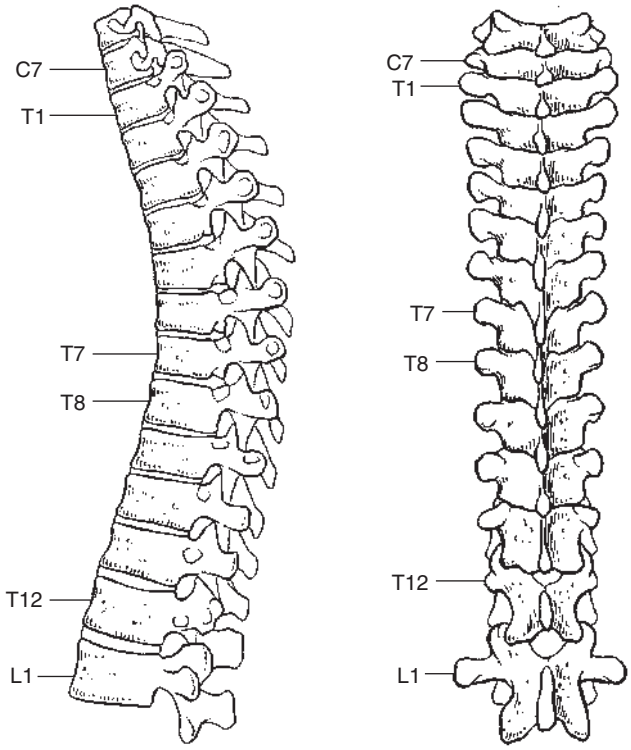

Fig 1 • Lateral (left) and posterior (right) views of the thoracic spine.

nucleus is rather small in the thorax. Therefore protrusions are usually of the annular type, and a nuclear protrusion is very rare in the thoracic spine.

The ligaments

The longitudinal ligaments run anteriorly and posteriorly on the vertebral bodies (Fig. 4, see Standring, Fig. 54.10). The anterior ligament covers the whole of the vertebral bodies' anterior aspect and some of their lateral aspect. It is firmly connected to the periosteum but only loosely to the discs. The posterior longitudinal ligament is strongly developed at the thoracic level and is wider than in the lumbar region, although it covers only a part of the posterior aspect of each vertebral body. It has some lateral expansions, which are firmly attached to the discs.

The ligamentum flavum, interposed between the laminae, extends laterally as far as the medial part of the inferior articular process. At each level the ligamentum flavum has lateral extensions to both sides to form the capsules of the facet joints (see Standring, Fig. 42.42). The transverse processes are connected to each other by the intertransverse ligaments. The supra- and interspinal ligaments bridge the gap between the spinous processes.

Facet joints

Each facet joint is composed of a superior and an inferior articular process, covered by hyaline cartilage and connected

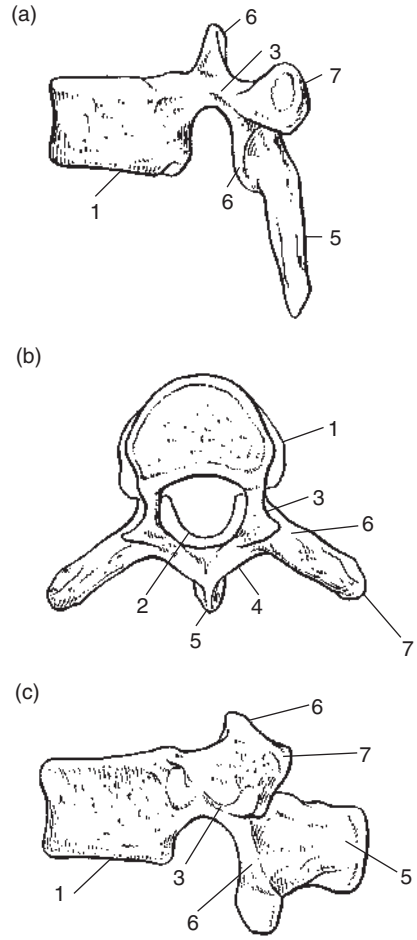

Fig 2 • Lateral and cranial views of the sixth thoracic vertebra (a, b) and lateral view of the twelfth thoracic vertebra (c). 1, vertebral body; 2, vertebral arch; 3, pedicle; 4, lamina; 5, spinous process; 6, articular processes; 7, transverse process.

to each other by a joint capsule which possesses a true synovium. The articular surface of the superior articular process points backwards, slightly upwards and outwards. The facet of the inferior articular process faces forwards, slightly downwards and inwards. These articulations lock the vertebrae together while allowing movements of flexion–extension, bilateral side flexion and rotation.

Content of the spinal canal

The spinal canal is formed by the vertebral foraminae of the successive vertebrae, the posterior aspects of the discs, the posterior longitudinal ligament, the ligamenta flava and the anterior capsules of the facet joints. In contrast to the cervical and lumbar regions, where the canal is triangular in cross-section and offers a large lateral extension to the nerve roots, the thoracic spinal canal is small and circular. It can be divided into three zones: the upper (T1–T3), and lower (T10–T12) zones are transitional, respectively, the cervical and the thoracic spine, and the thoracic and the lumbar spine. Between these is the mid-thoracic zone (T4–T9), where the spinal canal is at its narrowest (Fig. 5).

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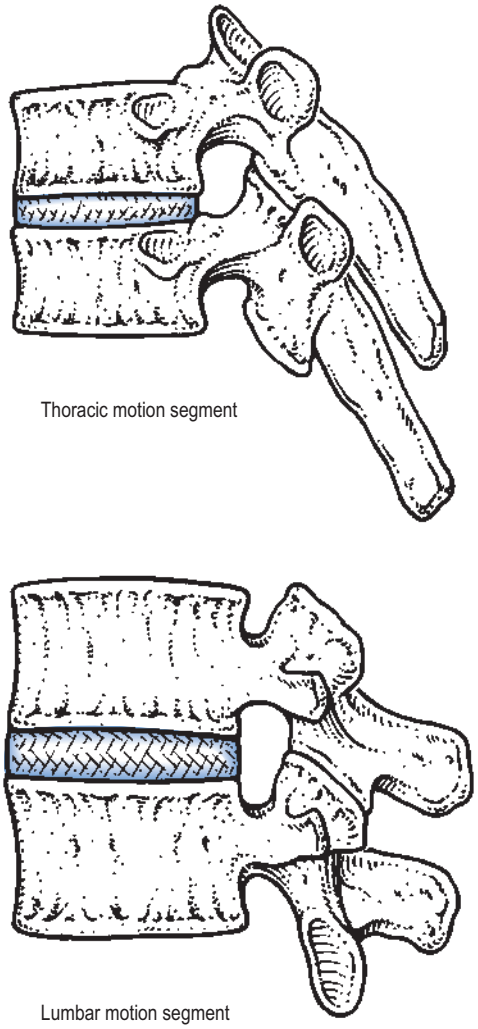

Fig 3 • Thoracic discs are smaller and flatter than lumbar discs. The intervertebral foramen is located behind the vertebral body instead of behind the disc.

The spinal canal contains the dural tube, within which are the spinal cord, the spinal nerves and the epidural tissue (see Standring, Fig. 43.3).

Dura mater

The dura mater, a blind-ended membraneous sack arising from the occiput and ending at S2 level, has similar characteristics at all levels. It is free within the spinal canal, where it is only loosely attached to the adjacent posterior longitudinal ligament allowing the dura to move and to deform on all spinal movements.

The anterior part of the dura is largely innervated by a mesh of nerve fibres belonging to different and consecutive sinuvertebral nerves. This may be the anatomical explanation for the broad and large pain reference commonly found in dural irritation at the thoracic level.

Spinal cord

The dura mater contains the spinal cord, which ends approximately at the L1 level. The spinal cord occupies the space of the spinal canal maximally at the thoracic level. This, together with its poor vascularization renders the thoracic spine very vulnerable to damage by extradural processes and vertebral trauma.

The cord depends for its blood supply on these arterial circles. The inner circle is of three longitudinal arterial channels, which run from the medulla oblongata to the conus medullaris. Their perforating arteries to the spinal cord are larger and more numerous at the cervical and lumbar level than at the thoracic level. Moreover, the inner arterial circle is characterized in the thoracic spine by a lack of anastomoses.

One of the two outer arterial circles is located in the extradural space, the other in the extravertebral tissues. These give rise to the 'medullary feeders', which arise at the cervical spine mainly from the vertebral arteries and in the thoracic and lumbar spine from the intercostal and lumbar arteries, which are segmental branches of the aorta. It is at mid and lower thorax that the spinal cord has the least profuse blood supply: the so-called critical vascular zone. When surgery is contemplated here, strict care must be taken not to impede blood flow.

Nerve roots and innervation

As at the cervical and lumbar levels, the thoracic spinal nerves emerge from the cord as a ventral and a posterior ramus, which join together to form the short spinal nerve root. The lateral part of the spinal canal that envelops the nerve root is the radicular canal. It is formed anteriorly by the posteroinferior aspect of the upper vertebra and a small part of the intervertebral disc, both covered by the posterior longitudinal ligament. The posterior boundary is formed by the lamina and the superior articular facet. The intraspinal course of the upper thoracic nerve roots is almost horizontal, as in the cervical spine. Therefore, a nerve root can only become compressed by its corresponding disc. However, the more caudal it is in the spine, the more oblique is the nerve root's course. The T12 nerve root within the spinal canal is at the height of the eleventh vertebral body, and therefore courses downwards, outwards and slightly anteriorly behind the T11 disc and the T12 vertebral body, to leave the foramen at the inferior margin of body T12. As a consequence, the lowest thoracic nerve roots can be compressed by disc lesions of two consecutive levels (T12 root by T11 or T12 disc).

The nerve leaves the spinal canal through the intervertebral foramen just behind the inferior vertebral margin and the costovertebral joints. Because the intervertebral foramina are quite large at these levels, osseous interference with the nerve roots is seldom encountered in the thoracic spine.

Beyond the intervertebral foramen, the nerve root divides into a large ventral and a smaller dorsal branch (Fig. 6). The latter gives rise to a medial and a lateral branch, which innervate mainly the skin of the posterior thorax and upper lumbar region; they also give branches to the erector spinae muscles. The ventral rami do not form plexuses at the thoracic level but retain their segmental distribution as intercostal nerves, having

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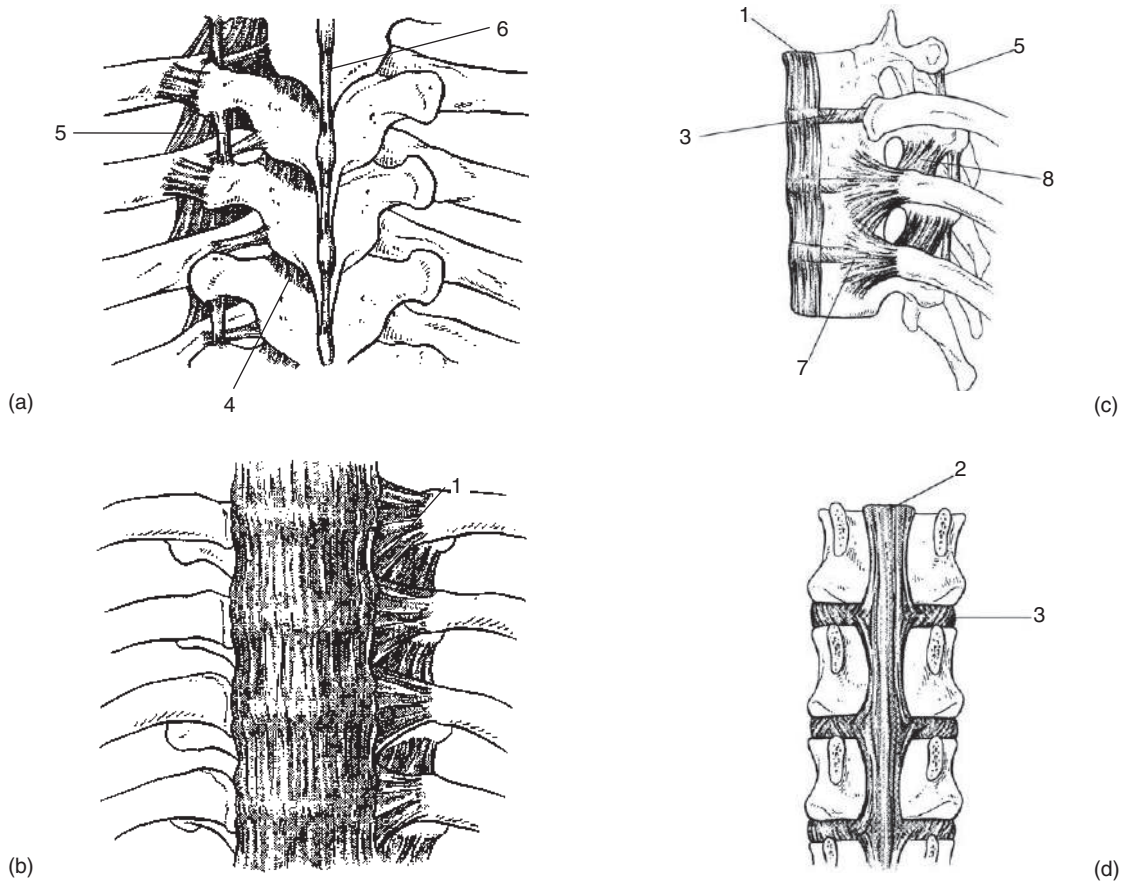

Fig 4 • Ligaments of the thoracic spine: posterior (a), anterior (b), lateral (c) and posterior with vertebral arch removed (d). 1, anterior longitudinal ligament; 2, posterior longitudinal ligament; 3, intervertebral disc; 4, ligamentum flavum; 5, intertransverse ligament; 6, supra- and interspinal ligament; 7, radiate ligament; 8, costotransverse ligament.

their course in the costal sulcus of the corresponding rib in which they lie dorsocaudal to the blood vessels. They innervate the costotransverse joints, the chest wall, the parietal pleura, the skin of the thorax and the intercostal muscles (see Standring, Fig. 54.16). As at the lumbar and cervical levels, innervation of the spinal canal is by the sinuvertebral nerve, which arises from the nerve root and re-enters the epidural space. It gives branches to the nervous network of the anterior and posterior longitudinal ligaments. Branches to the dura mater cross the midline and innervate several consecutive levels to about four segments cranial and caudal to their points of entrance. This explains why pressure to the dura mater may give rise to pain felt multisegmentally and even bilaterally (see p. 426).

The thoracic cage

The thorax is a complex system of bony, cartilaginous, ligamentous, muscular and tendinous structures. Superficial to the thoracic wall are bony and musculotendinous structures, connecting the upper limb to the trunk. These structures belong to the shoulder girdle and are discussed in the online chapter, *Applied anatomy of the shoulder girdle*.

The cranial border of the cage is the superior thoracic aperture. It is bounded by the first thoracic vertebra, the first ribs, the clavicles and the upper edge of the manubrium. The aperture does not lie in a horizontal plane but is inclined ventrally downwards. Inferiorly, the thorax is separated from the abdomen by the diaphragm.

Bony structures

Twelve pairs of ribs, together with the sternum, the clavicle and the thoracic spine, form the bony part of the thoracic cage (Fig. 7). Because the thoracic spine is kyphotic, with the apex of the kyphosis at T7, the thoracic cage is widest in sagittal diameter at this level.

The sternum (Fig. 8) is composed of an upper part (manubrium), a mid-portion (body) and a caudal part (xiphoid). Because of the overall slightly oblique alignment of the sternum the caudal end projects further anteriorly.

The upper part of the manubrium has a depression, the jugular incisura, lying between the sternal heads of both sternocleidomastoid muscles. Its superolateral sides are the sternal joint surfaces of the sternoclavicular joints.

A synchondrosis joins manubrium and body. It protrudes slightly anteriorly and is known as the sternal angle of Louis – an important landmark because the second rib is attached to the sternum at this level. At the caudal end, another

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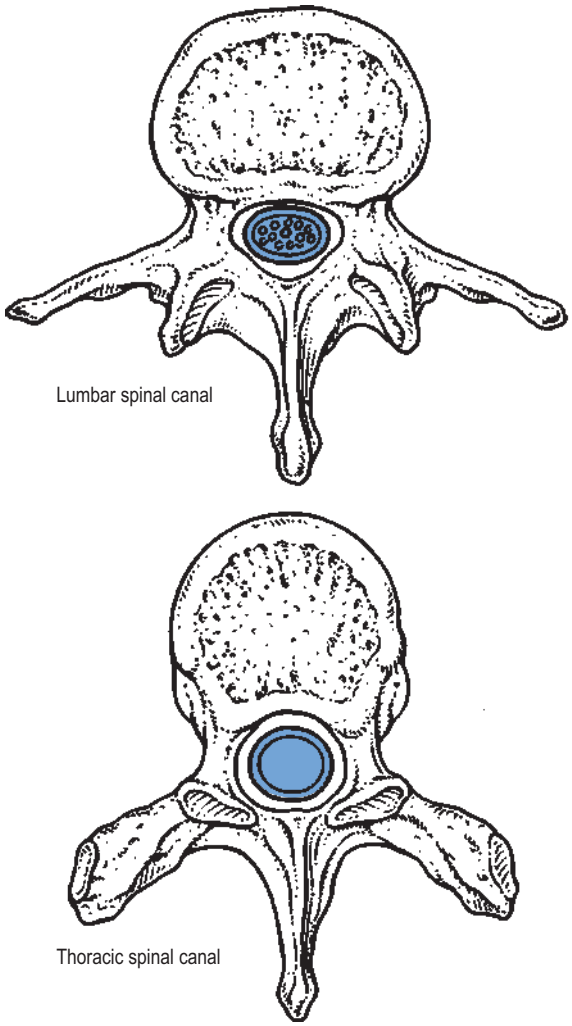

Fig 5 • The small and round thoracic spinal canal is largely occupied by spinal cord and dura.

synchondrosis connects the xiphoid process to the sternum; it is reinforced by the costoxiphoid ligaments.

The thoracic cage contains twelve pairs of ribs. Each rib consists of a head, a neck and a body (Fig. 9). The head consists of the slightly enlarged posterior end, normally carrying two hemifacets for the costovertebral joints. The first, eleventh and twelfth ribs have only one full facet here. The body is separated from the head by a thinner part: the neck. At the junction with the corpus of the rib a tubercle is present, on which another joint surface lies (Fig. 10). This is part of the costotransverse joint lying between the rib and transverse process (Fig. 11). The two lowest ribs have no articular facet on their tubercle. The costovertebral and the costotransverse joints are true synovial joints. The anterior portion of the capsule of the costovertebral joint is locally reinforced by the radiate ligament, which courses between both adjacent vertebrae and the rib. The capsules of the costotransverse joints are locally reinforced by the costotransverse ligaments.

A few centimetres beyond the vertebral column, where the curve of the rib is most pronounced, is the costal angle (see Fig. 9).

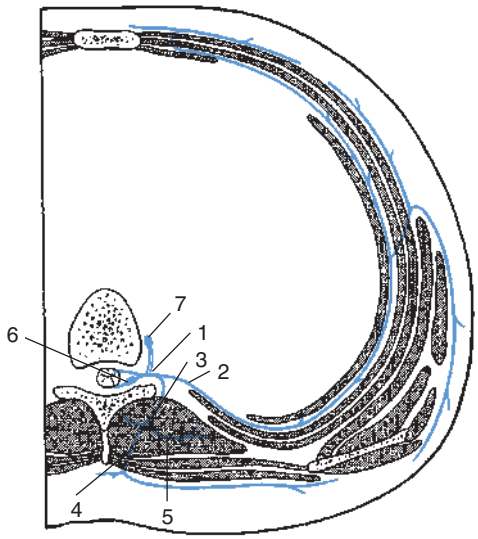

Fig 6 • Spinal and intercostal nerve supply: 1, nerve root; 2, ventral ramus and intercostal nerve; 3, dorsal ramus; 4, medial branch; 5, lateral branch; 6, spinal ganglion; 7, sympathetic ganglion.

Except for the first and second ribs, all ribs have a groove for the intercostal nerve and blood vessels at their lower margin, which is at the outer aspect confined by a sharp bony edge.

All ribs are different from each other in size, width and curvature. The first rib is the shortest. Its anteromedial part lies beneath the medial end of the clavicle. It has two sulci, separated from each other by a tubercle to which the scalenus anterior muscle inserts. Important neurovascular structures are situated medial and lateral to this tubercle. The second rib is much longer. The medial end of the cartilage lies at the same level as the manubriosternal synchondrosis. Since the first rib can hardly be palpated, the second rib is usually the first bony structure caudal to the clavicle which can be easily defined, even in circumstances where the sternal angle is not pronounced. Therefore, it is an important landmark. The rib length increases further caudally, until the seventh rib, after which they become progressively shorter.

The attachment of the ribs to the sternum is variable. The upper five, six or seven ribs have their own cartilaginous connection. The length of this varies from about 2 cm for the first rib to about 10 cm for the seventh rib. The cartilage of the eighth rib ends by blending with the seventh. The same situation pertains for the ninth and the tenth ribs, so giving rise to a common band of cartilage and connective tissue. The eleventh and twelfth ribs remain unattached anteriorly but end with a small piece of cartilage (see Standring, Fig. 54.13).

Contractile structures

The muscular and tendinous elements of the thoracic cage are the intercostals and the diaphragm. Superficial to the thoracic cage are the erector spinae muscles, levator scapulae, trapezius, rhomboid, pectoralis minor, subclavius and serratus anterior

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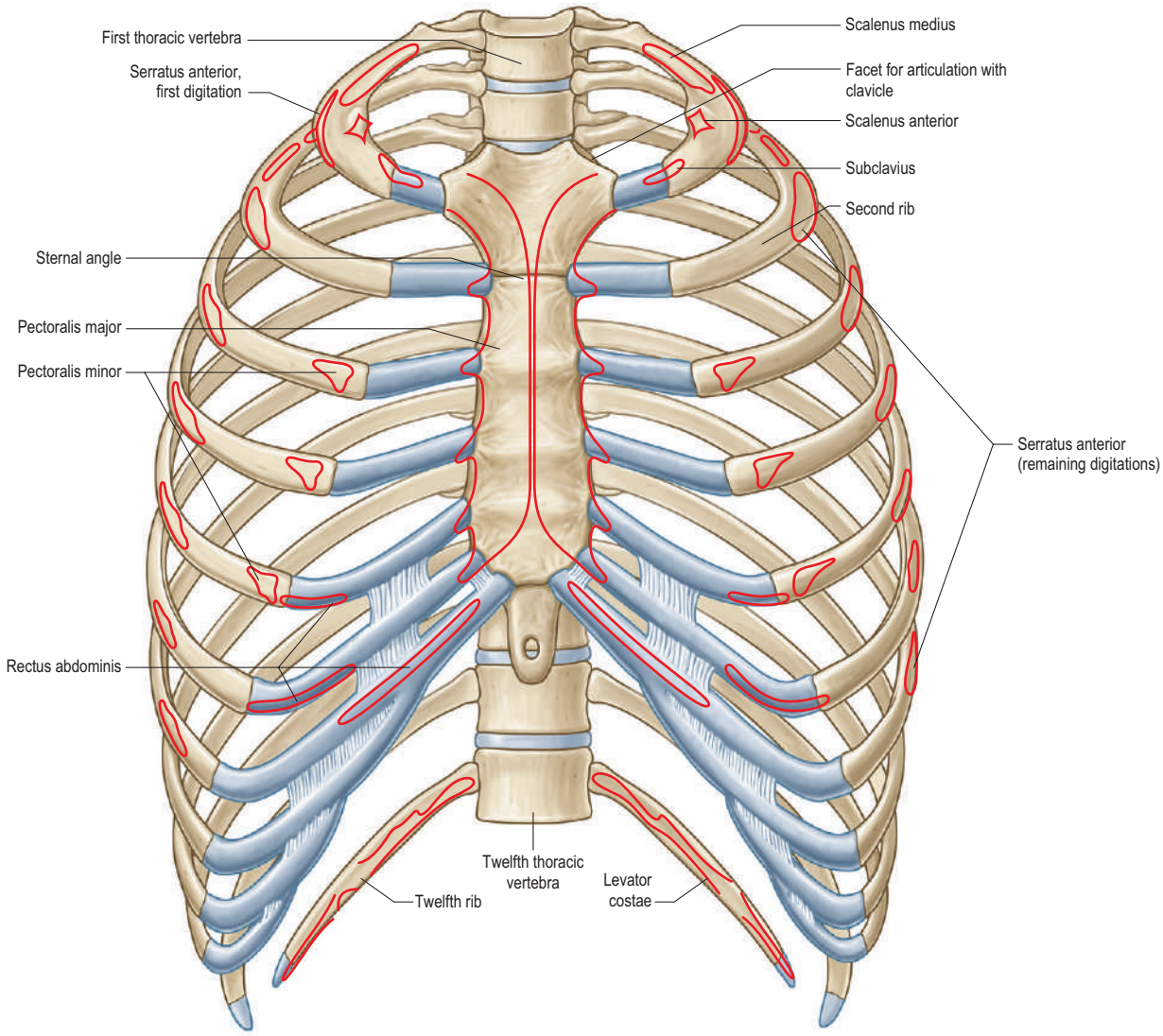

Fig 7 • The thoracic cage. From Standring, Gray's Anatomy, 40th edn. Churchill Livingstone/Elsevier, Philadelphia, 2009 with permission.

muscles. The other associated structures, such as the pectoralis major and latissimus dorsi, are discussed in Chapter 14.

The intercostal muscles

The intercostal muscles connect the ribs to each other and are the primary respiratory muscles. Three different structures can be distinguished: the external and internal intercostals and the intimi (see Standring, Fig. 54.16).

The external intercostal muscles take origin from the lower margin of the upper rib, just superficial to the costal sulcus. Their direction is from cranioposterior to caudoanterior; insertion is into the superficial aspect of the upper margin of the lower rib. Their posterior edges are level with the neck of the rib (Fig. 12). The anterior margins are at the junction of the rib and its cartilage; thus, in the most medial part of the intercostal space, an external intercostal is not present. Due to the oblique course of the fibres and the fact that leverage is greatest on the lower of the two ribs, on contraction the muscle pulls the lower rib towards the upper rib, which results in inspiration.

The internal intercostals originate from the internal aspect of the upper margin of the lower rib and insert on the inner aspect of the sulcus of the upper rib. The course of their fibres is from caudoposterior to cranioanterior. Their anterior margins are at the edge of the sternum, the posterior margin at the costal angle. They pull the upper rib downwards in forced expiration.

Between the internal and external intercostals lie the intimi. These take origin on the upper aspect of the lower rib and insert just medially to the origin of the external intercostal muscles. Their direction and function are the same as for the internal intercostal muscles.

The diaphragm (C3–C4)

The diaphragm is a dome-shaped musculotendinous structure which separates the thorax from the abdomen (Fig. 13, see

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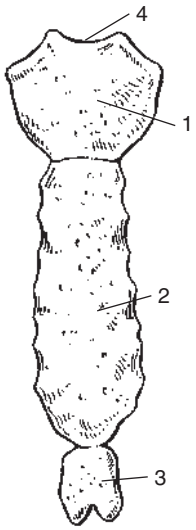

Fig 8 • The sternum: 1, manubrium; 2, body of sternum; 3, xiphoid; 4, jugular incisura.

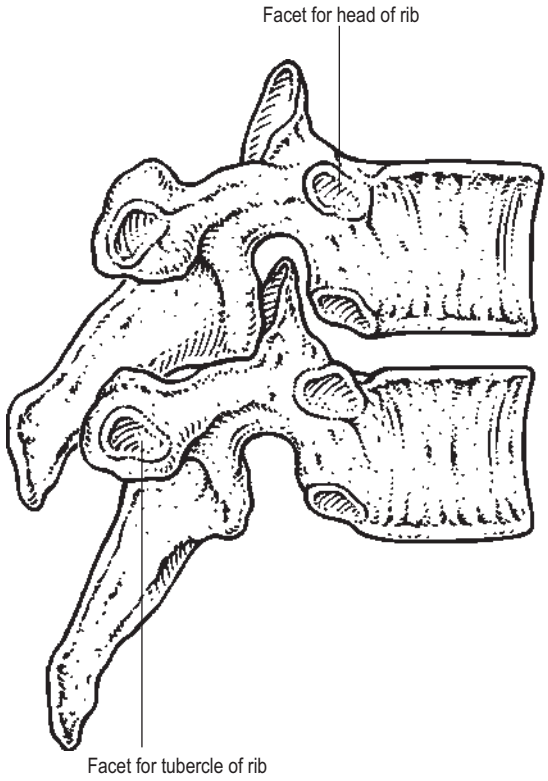

Fig 10 • Articular facets for the rib joints on the vertebral body and transverse process.

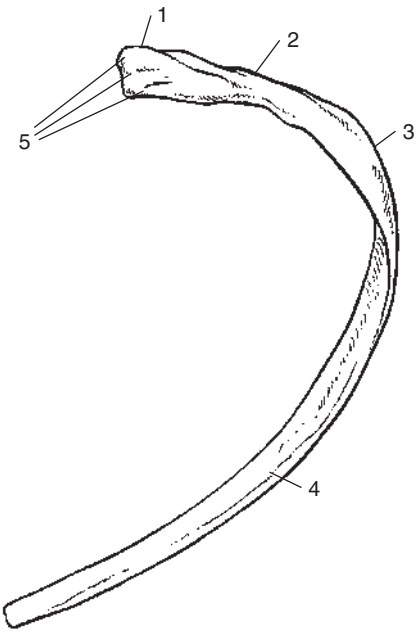

Fig 9 • Rib structure: 1, costal head; 2, costal neck; 3, costal angle; 4, costal body; 5, facet surfaces.

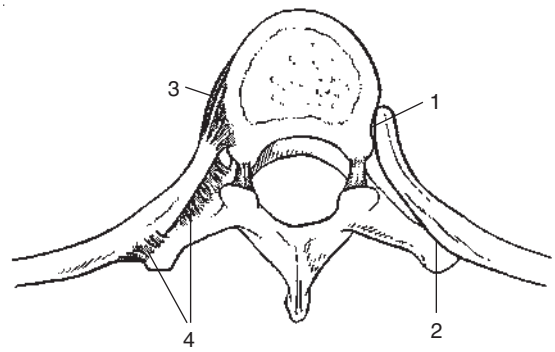

Fig 11 • The rib joints. 1, costovertebral joint; 2, costotransverse joint; 3, radiate ligament; 4, costotransverse ligaments.

Standring, Fig. 54.14). It is pierced by structures passing from thorax to the abdomen and vice versa.

Its short origin is from the inner aspect of the caudal bony thoracic aperture and can be divided into three different parts, all of which insert into a central tendinous structure that forms the roof of the dome.

The sternal part is attached to the posterior aspect of the xiphoid process and inserts anteriorly at the middle of the central tendon.

The costal part originates from the internal aspect of the six lowermost ribs and their cartilages. It inserts into the lateral part of the central tendon.

The origin of the lumbar part is slightly different on the two sides of the midline. To the left, the origin is from the anterior part of the three upper lumbar vertebral bodies; to the right, from the four upper lumbar vertebrae. These tendinous structures create an arch for the aorta and thoracic duct in front of the spine. Anterolaterally to the left, there is an opening in the muscular fibres for the oesophagus. The fibres of lumbar origin insert into the posterior aspect of the central tendon. To the right of the midline posteriorly is an aperture for the passage of the vena cava inferior.

Contraction of the diaphragm pulls the central tendon downwards, so producing diaphragmatic inspiration. The

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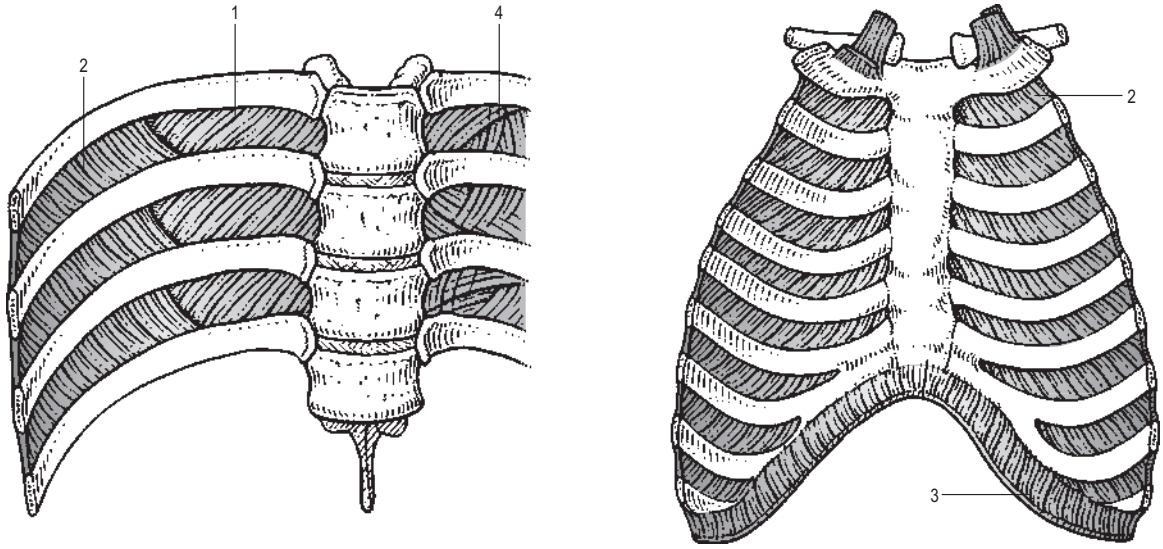

Fig 12 • The intercostal muscles: 1, external intercostal muscles; 2, internal intercostal muscles; 3, diaphragm; 4, endothoracic fascia.

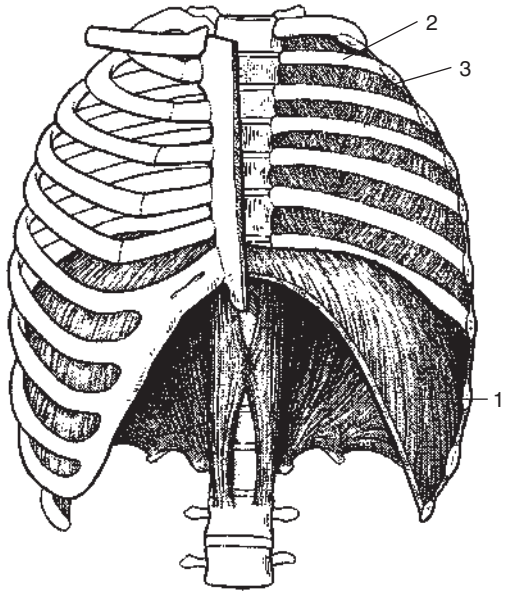

Fig 13 • 1, diaphragm; 2, external intercostal muscles; 3, internal intercostal muscles.

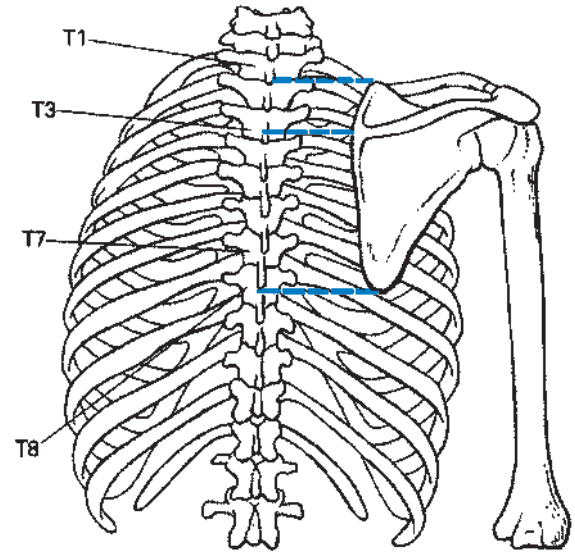

Fig 14 • The shoulder in neutral position: landmarks.

diaphragm also provides a flexible barrier between thorax and abdomen.

Other clinically important muscles

The following clinically important muscular structures, although inserting on the thoracic cage, belong to the shoulder girdle. They are discussed thoroughly in the online chapter, *Applied anatomy of the shoulder girdle*:

- Trapezius
- Rhomboid minor and major
- Levator scapulae
- Serratus posterior inferior
- Subclavius
- Pectoralis minor
- Serratus anterior
- Pectoralis major
- Latissimus dorsi.

Landmarks

With the shoulder in neutral position, the superior angle of the scapula is level with the first thoracic vertebra and the posterior end of the first rib (Fig. 14). The inferior angle corresponds to the interspinal level between T7 and T8, whereas the spine

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of the scapula lies at the level of the T3 spinous process. The medial border of the scapula is approximately 5–6 cm from the midline.

Movements of the thoracic spine and cage

The thoracic vertebral column constitutes the most rigid part of the spine, but nevertheless allows some movement in flexion–extension (in a sagittal plane), side flexion (in a frontal plane) and rotation (in a horizontal plane).

Flexion–extension movements take place mainly in the lower thoracic segments, whereas side flexion takes place at all thoracic levels but is slightly increased towards the caudal thoracic area. Rotation takes place mainly at the joints of the upper half of the thoracic spine.

Rotation is stopped by the posterior ligaments, essentially by the ligamentum flavum, together with the capsules of the facet joints. This gives rise to an elastic ligamentous end-feel on passive rotation. The degree of vertebral rotation is nevertheless limited by the close and multiple connections between the thoracic spine and the rest of the thoracic cage. Because many of the costal cartilages and ligaments lose part of their elasticity with ageing, the range of rotation is considerably decreased in elderly subjects.

On extension, the disc is compressed posteriorly and widens anteriorly, and the nucleus pulposus moves anteriorly. Extension is arrested by the spinous and articular processes, together with tension of the anterior longitudinal ligament as this is stretched. An elastic end-feel is the result. The posterior longitudinal ligament, the ligamentum flavum and the interspinous ligaments relax.

Flexion ends with tension in the interspinous ligament, the ligamentum flavum, the capsules of the facet joints and the posterior longitudinal ligament. Side flexion is limited by the impact of the ipsilateral articular processes and by the contralateral ligamentum flavum and intertransverse ligaments.

As in the cervical spine, there is a coupling characteristic to movement: on side flexion there is always some degree of axial rotation in such a way that the spinous processes move toward the concavity of the lateral curvature. Thus, on side flexion to the right, a rotation to the left occurs. This 'side coupling' is mainly confined to the upper thorax.

The abdominal wall

The abdominal wall is mainly a musculotendinous structure with some posterior and inferior bony elements (thoracolumbar spine and pelvis – see Chapter 31 and online chapter *Applied anatomy of the sacroiliac joint*) (see Standring, Fig. 61.2). The rectus abdominis muscle, situated bilateral to the midline between the thoracic cage and the pubic symphysis, takes origin from the superficial aspect of the rib cartilage of the fifth, sixth and seventh ribs and inserts into the upper surface of the pubic symphysis and pubic bone (Fig. 15). It is anchored in a fibrous sheath both anteriorly and posteriorly (see below).

The muscle assists in anterior flexion of the trunk on the pelvis and vice versa.

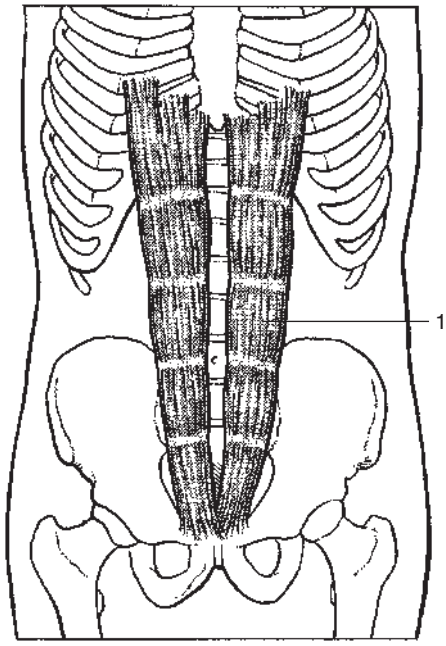

Fig 15 • Rectus abdominis, 1.

External oblique

This is the most superficial muscle of the lateral abdominal wall and takes origin from the outer surface of the seven lowermost ribs.

Its uppermost attachments alternate with those of the serratus anterior and the lower ones with the costal origin of the latissimus dorsi. The fibres course in a caudomedial direction and insert into the external edge of the anterior two-thirds of the iliac crest, into the inguinal ligament and the lateral border of the rectus sheath (Fig. 16).

Contraction causes anterior flexion of the trunk on the pelvis, rotation of the trunk to the opposite side and ipsilateral side flexion.

Internal oblique

This lies deep to the external oblique and superficial to the transversus abdominis muscle (Fig. 17). It originates from the superior aspect of the anterior two-thirds of the iliac crest, from the ventral part of the lumbar fascia and from the lateral two-thirds of the inguinal ligament. The fibres have a craniomedial course. They insert into the lower aspect of the three lower ribs and the rectus sheath.

Contraction causes an anterior flexion and an ipsilateral rotation and side flexion of the trunk.

Transversus abdominis

The origin is from the internal aspect of the six lowermost ribs, from the lumbar fascia, from the internal surface of the iliac crest and from the lateral third of the inguinal ligament. The direction of its fibres is horizontal (Fig. 18) and insertion is into the sheath of the rectus. It assists in contraction of the abdominal wall and may have some function in lumbar stabilization.

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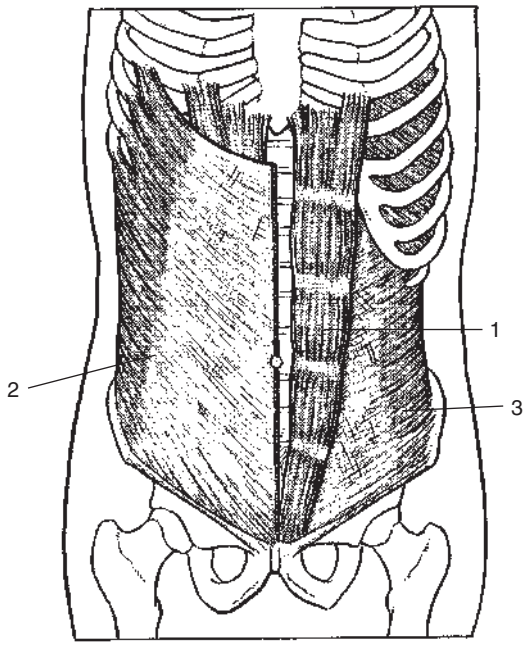

Fig 16 • Rectus abdominis, 1; external oblique, 2; transversus abdominis, 3.

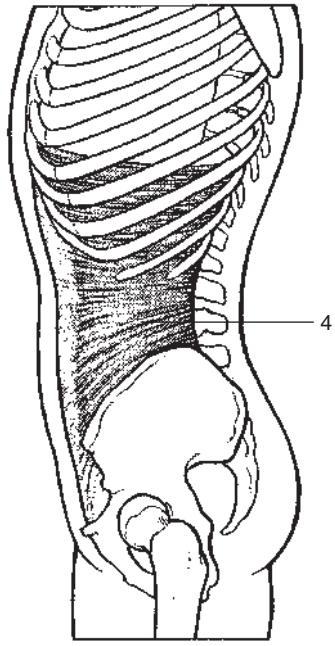

Fig 18 • Transversus abdominis, 4.

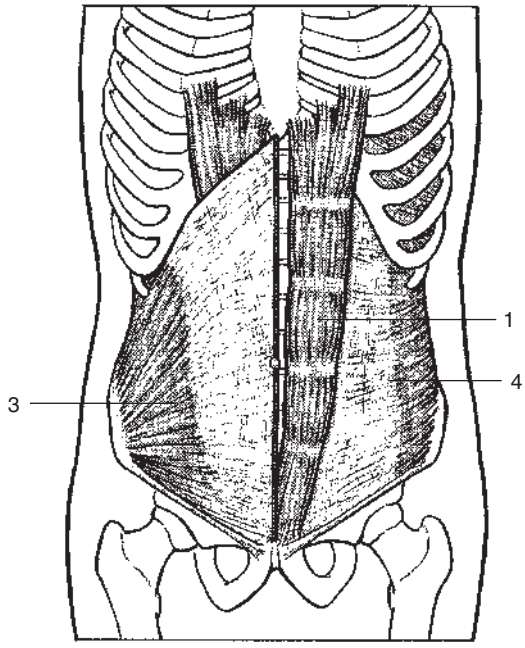

Fig 17 • Rectus abdominis, 1; internal oblique, 3; transversus abdominis, 4.

The ilioinguinal region

This is the lower ventral part of the abdominal wall. Here the internal and external oblique muscles, the transversus abdominis and the inguinal ligament merge to form the inguinal canal. The last contains the spermatic cord in the male, the round ligament of the uterus in the female and the ilioinguinal nerve in both sexes (see Standring, Fig. 61.13). The canal extends downwards and medially, parallel and a little above the inguinal ligament. The boundaries are: in front, the aponeurosis of the external oblique and muscular fibres of the internal oblique in its lateral one-third; behind, the reflected inguinal ligament and the transversalis fascia; above, the arched fibres of the internal oblique and the transversus abdominis; below, the union of the transversalis fascia with the inguinal ligament.

Protrusion of peritoneum and/or intestine into the inguinal canal is known as a direct or indirect inguinal hernia (see p. e253 of the online chapter *Groin pain*).

Rectus sheath

The fascia of the rectus abdominis muscle is reinforced by the aponeuroses of the oblique abdominis muscles, which merge in the midline to form the dense fibrous linea alba. In the upper part, the anterior portion of the sheath is formed by the insertion of the external oblique and the internal oblique muscle, which splits, to form a deep and superficial layer. The posterior part is the deep layer of the internal oblique muscle and the insertion of the transversus abdominis. In the lower part, from about halfway between the umbilicus and symphysis, layers of the flat muscles pass anterior to the rectus; behind the rectus is only diaphenous fascia (see Standring, Fig. 61.8).

A summary of the muscles of the thorax and trunk is provided in Table 1.

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Table 1 Summary of the muscles of the thorax and trunk

Structure and muscle	Function	Peripheral innervation	Spinal innervation
Thoracic cage			
External intercostal	Inspiration	Corresponding intercostal nerve	
Internal intercostal	Expiration	Corresponding intercostal nerve	
Intimi intercostal	Expiration	Corresponding intercostal nerve	
Diaphragm	Inspiration Abdominal pressure	Phrenic nerve	C4 (C3–C5)
Scapulothoracic			
Trapezius	Elevates scapula Adducts scapula	Accessory nerve and branches of cervical plexus	C2–C4
Rhomboid	Elevates scapula Adducts scapula	Dorsal scapular nerve	C5
Levator scapulae	Elevates scapula	Dorsal scapular nerve and cervical plexus	C5
Serratus posterior inferior	Pulls lower ribs downwards and dorsally	Intercostal nerves	T9–T12
Subclavius	Depresses scapula	Branch of superior trunk of brachial plexus	C5
Serratus anterior	Rotates scapula Fixates scapula	Long thoracic nerve	C5–C7
Pectoralis minor	Depresses scapula Adducts scapula	Medial pectoral nerve	C8–T1
Abdomen			
Rectus abdominis	Abdominal pressure Flexion of trunk	Middle and caudal intercostal nerves	T6–T11
External oblique	Contralateral rotation of trunk Flexion of trunk Ipsilateral side flexion of trunk	Intercostal nerves	T5–T12
Internal oblique	Ipsilateral rotation of trunk Flexion of trunk Ipsilateral side flexion of trunk	Intercostal nerves	T10–T12
Transversus abdominis	Abdominal pressure	Intercostal nerves	T7–T12

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Disorders of the thoracic spine: pathology and treatment

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Disorders and their treatment

Tumours of the thoracic spine

Spinal neoplasms, both primary and secondary, are unusual causes of thoracolumbar pain. However, because these lesions are associated with high mortality, examiners must always be aware of the possibility of neoplastic diseases and must include them in their differential diagnosis.

Malignant spinal tumours occur mainly in the middle aged and are characterized by a more or less sudden onset of the symptoms; often a minor trauma is thought to be the inciting cause and progression is swift.

According to their localization (in or outside the spinal canal) tumours can be classified as intra- or extraspinal:

- *Intraspinal tumours* are sited in the spinal canal. They are further divided into intra- and extradural tumours. The former may be intra- or extramedullary.
- *Extraspinal tumours* involve the bony parts of the vertebrae. Benign tumours are usually located in the posterior parts (spinous and transverse processes), malignant tumours in the vertebral body.

Intraspinal tumours

Thoracic neurofibroma

Pathology

This benign tumour usually originates from the dorsal root and arises from proliferating nerve fibres, fibroblasts and Schwann cells. Sensory or motor fibres may be involved.[1] Some confusion exists about the terminology: various names, such as neurofibroma, neurinoma, neurilemmoma or schwannoma have been used. Some believe that all these terms cover the same type of tumour; others distinguish some slight histological differences between them. Multiple tumours in nerve fibres and the subcutaneous tissues, often accompanied by patchy café au lait pigmentation, constitute the syndrome known as von Recklinghausen's disease.

Neuromas are the most common primary tumours of the spine accounting for approximately one-third of the cases. They are most often seen at the lower thoracic region and the thoracolumbar junction.[2] More than half of all these lesions are intradural extramedullary (Fig 1), 25% are purely extradural, 15% are both intradural and extradural, and very rarely they are seen intramedullary.[3,4] They may become symptomatic in patients at any age, but the peak incidence is around the fourth and fifth decades.[5] The tumours are benign, slow-growing and remain solitary,[5] well circumscribed and encapsulated. Often there is a cystic degeneration within the tumour. A tumour that is within the intervertebral foramen is often shaped like an hourglass or dumb-bell, one limb of which can give rise to a paravertebral extraspinal extension, which is sometimes palpable.

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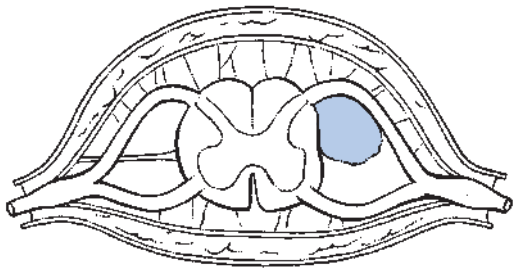

Fig 1 • Intradural extramedullary neurofibroma.

History

Symptoms are usually due to the compression of dura, spinal cord and roots. Few symptoms are present until the tumour reaches a large mass. Cystic tumours have a high risk of causing progressive symptomatic worsening as a result of cyst expansion.[6]

In the early stage, the diagnosis is often difficult because neurofibromas usually give rise to symptoms almost identical to those of a disc lesion.[7,8] Thoracic neuromas may even simulate a disc problem at the lumbar level.[8–11]

The main symptom is pain, most frequently present as root pain, exceptionally as a dural pain. It is increased by a deep breath or by a cough and may be present at night. Sometimes the patient prefers to sleep sitting up in a chair, a situation that is most unusual for a disc lesion. Activity or movement influences the pain only exceptionally. Pain increases slowly over months and years, a characteristic that indicates an unusual, slowly progressive disorder.

Involvement of the sensory fibres may result in a segmental band-shaped area of numbness. The tumour may also compress the spinal cord, affecting both motor and sensory elements (see p. 390). As a consequence, the patient may complain of stiffness of the legs, muscle spasms, extrasegmentally referred pins and needles, and disturbed sphincter function with loss of bowel or bladder control.[10] Exceptionally Brown–Séquard syndrome occurs.[5]

Clinical examination

Interference with the dura usually produces a clinical picture almost identical to that of an ordinary disc lesion.

Neck flexion and scapular approximation are often painful. These signs, together with pain on deep inspiration, are typical of a lesion that is interfering with the dura mater or with the dural nerve sleeve. They are not specific to a disc lesion and may occur in any space-occupying lesion.

Because the tumour lies outside the intervertebral joint, it does not interfere with articular movements and therefore daily activities and movements are hardly affected. Side flexion away from the painful side is sometimes the only painful and restricted movement and this pattern is unlikely to result from a disc lesion.

All signs slowly and progressively increase. Finally, neurological signs may develop but they come on much later than in malignant tumours. They may consist of a band-shaped numbness related to one dermatome. The tumour may also affect motor fibres but segmental motor deficit is very difficult to detect. Once the tumour compresses the spinal cord, any of the signs of cord compression may be encountered: depression of abdominal reflexes, hyperactive patellar and Achilles tendon reflexes and sensory loss.[4]

When a neurofibroma is suspected, the paravertebral area should always be palpated because a neurofibroma may expand extraspinally via the intervertebral foramen to give rise to a paravertebral mass.

Warning

Warning signs in neurofibroma:

- Pain: slowly increasing pain over months, mainly felt at night, uninfluenced by activities
- Patient prefers to sleep sitting up
- Side flexion away from painful side is the only painful and limited movement
- Band-shaped area of numbness related to one dermatome
- Presence of pins and needles in one or both feet or any other sign of cord compression
- Disorder mistaken as a disc lesion, which does not respond to manipulations.

Special investigations

On plain radiography there may be an enlarged intervertebral foramen and erosion of the vertebral body or the pedicles. X-ray of the thorax may show an abnormal soft tissue mass, extending outside the spinal canal (hourglass tumour).

MRI appears to be the most sensitive investigation for identifying these lesions. Nerve sheath tumours have equal or less signal intensity on T1-weighted images and mild to marked hyperintensity on T2-weighted images as compared to the spinal cord.[12] Focal areas of even greater hyperintensity on T2-weighted images often correspond to cystic portions.

Treatment and prognosis

Neurofibromas may undergo malignant change or produce cord compression. Therefore, they should be surgically removed. Small posteriorly or laterally sited tumours can usually be dealt with easily. Anterior neurofibromas present more problems and usually can be only partly excised.[13]

Other intraspinal masses

These include all other infra- and extradural tumours of the spinal canal. Despite the advantage of specialized technical investigations (radiography, myelography, CT, MRI), patients with intraspinal tumours are often misdiagnosed.

Clinical presentation

The primary and most universal symptom is pain, which is usually felt centrally in the back and may spread bilaterally as girdle pain.[14] The pain increases progressively and is relentless, despite the patient's attempts to limit activities. In anterior compression of the dural sac, L'hermitte's sign is sometimes present.[15]

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Occasionally the pain is worst at night. Although this is classically regarded as being suggestive of a tumour, it is rather rare.[8,9]

Straining and coughing may increase the pain as may active movement, but to a lesser degree than in mechanical disorders.[9,16]

The clinical pattern depends on the extent of the tumour: all tests can be completely normal or movement may be considerably limited. If the latter, anteflexion is usually involved and there is often associated muscle spasm. As a rule, in intraspinal soft tissue masses, not much is learnt from articular movements. Besides the positive articular signs, all intraspinal masses give rise sooner or later to neurological signs caused either by involvement of one or more nerve roots or by compression of the spinal cord (see p. 390).

It should be noted that a tumour is not always found where it would be expected on a clinical basis. Cases have been reported where upper thoracic tumours gave rise to pain and neurological signs in the lower limb or in the lumbar area.[8,9]

Further investigations

In all instances, further investigation is called for. Examination of the cerebrospinal fluid may show elevated proteins, a finding strongly suggesting a neoplasm.[9]

The imaging techniques used to define intraspinal tumours are MRI and CT-myelography. The advantages of MRI are: ease of obtaining sagittal sections; rapid assessment of the entire thoracic spine; patient acceptability because of the absence of radiation and low invasiveness; and lack of morbidity and complications.

Neoplasms that are frequently associated with skeletal metastases include tumours of the breast, prostate, lung, kidney, thyroid and colon.[17–20] Data from autopsy material suggest that up to 70% of patients with a primary neoplasm from one of these sources will develop pathological evidence of metastases to vertebral bodies in the thoracolumbar spine.[21] Because the majority of metastases occur in the vertebral body, they may cause anterior compression of the spinal cord, either directly by tumour growth or by a pathological fracture with retropulsion of bone and disc fragments into the spinal canal.[22] Finally compression of the cord can result from an intradural metastasis.[23]

Extraspinal tumours

Neoplasms located outside the spinal canal are called extraspinal tumours.

In general, benign tumours are located in the posterior elements of the vertebrae and are found in patients under 30 years of age, whereas malignant tumours (both primary and metastatic) are located in the anterior components of the vertebrae and are more common after the age of 50. Myelomas and metastases are the most frequent malignancies.[17]

Multiple myeloma is a malignant tumour of plasma cells. Plasma cells produce immunoglobulins and antibodies and are located throughout the bone marrow. Multiple myeloma is the most common adult primary malignancy of bone. The patients are usually in an older age group, ranging between 50 and 70. The disease leads to widespread bone destruction, abnormal immunoglobulin production and infiltration of bone marrow. Therefore a patient with multiple myeloma develops a broad range of clinical symptoms that extend beyond the orthopaedic field. Symptoms are also related to hypercalcaemia (anorexia, nausea, vomiting, kidney stones and changes in mental state), abnormal immunoglobulin concentrations (renal insufficiency and amyloidosis), haematological changes (anaemia and bleeding disorders) and generalized weakness.

Diagnosis is made on laboratory examination and radiographic evaluation.

Spinal metastases are much more common than primary tumours. The prevalence increases with increasing age. The lumbar and thoracic spines are affected in approximately 46–49% of cases, with a predilection for the thoracolumbar junction (the so-called forbidden area). Consequently signs of cord compression are often present.

In other circumstances, radicular pain occurs. This is most misleading in the thoracic area, where it often mimics visceral problems.

Clinical presentation

In 95% of cases, the first symptom is local neckache or local thoracic backache, which goes on to radiate. Suspicion should arise when this occurs in patients over 50 years of age complaining for the first time of backache not preceded by trauma. The pain tends to increase in intensity progressively and to involve a larger area: expanding pain. If radicular pain is present, it is usually worse at night (Borenstein and Wiesel:[1] p. 309).

In vertebral metastases a full articular pattern is usually present, characterized by marked painful limitation on extension and symmetrical limitation of both side flexions and rotations. Muscle spasm may also be present. Local tenderness over the spinous process is found on palpation.

Radicular signs or those of cord compression may occur abruptly, with full neurological features developing in 48 hours or more commonly subacutely over a week or more. Some patients have a much slower course – neurological symptoms and signs progressing over 4–6 months.

Tumours of T12 or L1 may compress the conus medullaris, containing the S3–S5 nerve roots. This may lead to dysfunction of the urinary or anal sphincter, which is sometimes associated with saddle anaesthesia.

Differential diagnosis (see Tables 1, 2) must be careful,[8,9,16,24–29] because the majority of the signs and symptoms also occur in ordinary thoracic disc lesions. It is based mainly on clinical examination, because up to 30% of the bone mass has to be lost before metastases may become visible on radiography.[30] When there is the slightest possibility that there is a tumour, manipulation should never be done and further investigations such as a bone scan must be carried out.

Extradural haematoma

Extradural spinal haematoma at the thoracic level can be either traumatic or nontraumatic. Spinal epidural haematoma (SEH) is an uncommon complication of severe spine injury with or without fracture of the thoracic vertebral bodies.

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Table 1 Differential diagnosis of thoracic disc protrusion and tumours

	Thoracic disc protrusion	Benign tumour	Malignant tumour	
			Intraspinal	Extraspinal
Age (years)	20–50	Any age	>50	>50
Evolution	Swift or chronic (ups and downs)	Very slow	Swift	Swift
Pain At night	Shifting Better	Expanding Worse	Expanding Worse	Expanding Worse
Dural symptoms On inspiration or cough	±	+	±	±
Articular signs	Partial articular pattern	Full range, or side flexion away from painful side is the only painful and limited test	All tests normal, or limitation of anteflexion Muscle spasm	Full articular pattern Muscle spasm
Dural signs Neck flexion Scapular approximation	 + +	 + +	 + +	 + +
Neurological signs	Unusual	+	+	±
Palpable mass	–	±	–	–

Table 2 Differential diagnosis of thoracic disc protrusion and neurofibroma

	Neurofibroma	Thoracic disc protrusion
Age	Young	20–50 years
Pain On inspiration or cough Preferred sleeping position	Slowly increasing + Sitting up	Swift onset; if chronic, no increasing pain but ups and downs + Lying down
Articular movements	Usually negative Side flexion away from the painful side: painful and limited?	Partial articular pattern
Dural signs Neck flexion Scapular approximation	 + +	 + +
Neurological signs Band-shaped area of numbness in one dermatome Segmental motor deficit Signs of cord compression	 + + +	 Unusual Unusual Unusual
Palpable mass	±	_

Compression of the cord by a haematoma is also a well-known, although rare, complication of spinal surgery.[31] The reported frequency is between 1 and 6 per 1000 operations. Traumatic bleeding in the epidural space has also been reported after epidural injections and chiropractic manipulations.[32]

Non-traumatic extradural spinal haematoma is an uncommon condition often associated with a poor outcome. There seems to be an increase of the incidence, probably from the increased use of thrombolytic and anticoagulant therapy.[33–35] Other causes of non-traumatic extradural spinal haematoma include vasculitis such as systemic lupus erythematosus (SLE), spinal arteriovenous malformations and haemophilia.[36,37]

A spinal epidural haematoma may present acutely or subacutely over a number of days or weeks and with fluctuating symptoms. The patient usually suffers from increasing and expanding thoracic back pain, followed by progressive signs and symptoms of major neurological dysfunction secondary to cord compression.[38] Conditions that may mimic an acute spinal haematoma include extradural abscess and extradural metastatic infiltration.

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It is important to make an early diagnosis because surgery may offer the best hope of restoring neurological function. MRI is the examination of choice and provides characteristic findings that allow a prompt diagnosis.[39] The technique can also provide useful information about the age of the haematoma.[40]

Spinal epidural haematoma has always been considered a neurosurgical emergency. The treatment of choice is decompressive surgery as soon as possible because permanent neurological disability or death may follow if neurosurgical intervention is delayed.[41,42] However, during the last decade several reports have been published showing that non-operative treatment may be successful in cases with minimal neurological deficits, despite cord compression revealed by MRI.[43–45]

Spinal cord herniation

Spinal cord herniation is a rare, although increasingly recognized, cause of spinal cord dysfunction. It has been ascribed to a dural defect, either congenital or acquired, in the anterior surface of the dural sac through which the spinal cord herniates.[46,47] The number of published cases in the English language literature markedly increased after 2000. Awareness of the clinical setting and the wider use of MRI in myelopathy are considered the pertinent factors in this increase.[48]

The main clinical features are thoracic pain and a Brown–Séquard syndrome. Although the dura is sensitive to pain, review of the literature shows that only 48% of the patients had thoracic pain.[49,50] About 73% present with Brown–Séquard syndrome[51,52] (spastic paralysis on the ipsilateral side together with numbness on the contralateral side). This is probably caused by tethering of the spinal cord at the side of the herniation which results in unilateral damage of the lateral funiculus.[53] Some patients have signs of only spasticity or numbness in one leg.[54]

MRI is the gold standard technique for diagnosis of spinal cord herniation. On sagittal MRI, typical features are ventral displacement, sharp ventral angulation of thoracic spinal cord, and enlargement of dorsal subarachnoid space.[55,56]

Surgery is crucial in the management of this rare entity, and duraplasty the more widely performed method.[57]

Thoracic spinal canal stenosis

This may be the outcome of either congenital deformation or hypertrophy of the posterior spinal elements. Most often it occurs in association with generalized rheumatological, metabolic or orthopaedic disorders, such as achondroplasia, osteofluorosis, Scheuermann's disease, Paget's disease or acromegaly. It is rare in the absence of a generalized disorder.[58,59] Degenerative changes in the facet joints and the intervertebral disc can diminish the volume of the spinal canal and cause cord compression.[60,61] The latter is most frequently found at T11 and T12 in middle-aged people.[62,63]

Clinical presentation

Patients usually complain of pseudoclaudication, characterized by pain in the lower back, buttock, thigh and in the legs, coming on during walking; sometimes standing may provoke symptoms.

In addition to pain, paraesthesia may be present in one or both lower limbs. The sensation is worse on walking and improves on sitting. Numbness in the lower limbs may follow and may be associated with muscular weakness, difficulty on walking and easy fatigue. Spastic paraparesis with diminished or lost reflexes in the lower limbs, together with urinary disturbance, can be present. In rare cases, hyperreflexia is noted.

Extension of the thoracic spine while upright may further compromise the cord within the narrowed canal and increase the symptoms, while improvement occurs on anteflexion.

On clinical examination there may be some degree of limitation of movement in the spine, because of osteoarthritis leading to stiffness. In such a case, a capsular pattern is found.

Arterial pulses in the lower limb are normal, which largely excludes vascular problems.

Further investigations

The radiograph is often unremarkable and myelography can be misleading. CT scan and MRI are usually required.[58,62]

Differential diagnosis

Two differential diagnoses should be considered:

- *Intermittent claudication:* when pseudoclaudication is present, differential diagnosis must be made from intermittent claudication caused by vascular abnormalities. In spinal stenosis, some patients have symptoms only on standing. In intermittent claudication the pain is brought on by walking and relieved on standing still; arterial pulses are usually diminished or absent. Doppler probe and arteriography may confirm the diagnosis.
- *Disc protrusion:* in spinal stenosis all postures or movements that bring the spine into anteflexion usually relieve the pain, whereas in a disc protrusion the opposite is more usual. Moreover, in a simple disc protrusion, activity of the lower limbs has no influence on the symptoms.

Treatment

Surgical intervention is necessary but established neurological features seldom regress.

Chest deformities

Hyperkyphosis

The normal thoracic spine is kyphotic but if the kyphosis is beyond 40°, hyperkyphosis is present.[64] The condition may occur at one or more levels and may be the result of several disorders.[65]

Juvenile kyphosis

Juvenile kyphosis has its clinical onset in adolescence between the ages of 14 and 18 years; for this reason the condition is sometimes known as adolescent osteochondritis. There is a slight preponderance in females.[66]

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It develops because of a disturbance in growth in the vertebral rim epiphysis akin to osteochondritis dissecans.[67] Although the exact aetiology is unknown, it is generally believed to result from an anterior endplate lesion, through which herniations of the intervertebral disc protrude into the adjacent bone (Schmorl's nodes).[68] The intervertebral disc itself becomes narrowed, mainly anteriorly. The protrusion interferes with the growth of the vertebral ring epiphysis, which finally leads to about 5° of anterior wedging of the vertebral body. Should this occur over several levels, thoracic hyperkyphosis (with the apex normally around T7–T9) results. In this event, the condition is named Scheuermann's disease.

Clinical presentation

In adolescents the disorder usually remains painless. It usually gives rise to progressive silent hyperkyphosis, with backache present only in a minority of cases.[69,70] At a later age, however, there is a predisposition to thoracic pain[71] (see p. e175 of this chapter).

On clinical examination, all movements are free except active extension, which may be limited. If hyperkyphosis is present, it does not disappear in prone position, and when a hyperextension thrust is given to the thoracic spine, stiffness is felt and slight discomfort provoked.

Radiography

The diagnosis is mainly based on radiographs, which are often taken for some other reason. They show narrowing of the intervertebral joint space and multiple Schmorl's nodes, giving an irregular aspect to the surface of the vertebral body. In the later stage, focal anterior wedging of the latter is seen.

Treatment

This should aim to prevent further deformation during the growth period. If the kyphosis is still reversible, active exercises for the extensor muscles of the back should suffice. In more progressive cases, in which hyperkyphosis increases or progression of anterior wedging of the vertebrae is found, a Milwaukee brace until growth has ceased is the best advice.

Hyperkyphotic posture in the elderly

Age-related hyperkyphosis is an exaggerated anterior curvature in the thoracic spine that occurs commonly with advanced age. Epidemiologic studies have demonstrated that age-related hyperkyphosis commonly affects the elderly population with estimates ranging from 20% to 40%.[72] The 'dowager hump' is well recognized, and most clinicians and patients equate this with spinal osteoporosis and vertebral compression deformity or angulation.[73] Increasing thoracic kyphosis, in particular when linked to back pain, is considered a signature of possible vertebral body compression fracture. However, approximately two-thirds of those who are most hyperkyphotic don't have vertebral fractures.[74] In the absence of vertebral compression fracture, changes in the spinal support tissues (i.e. ligaments, tendons, disk annulus and nucleus) or supporting musculature could also lead to a progressive increase in curvature.[75]

Apart from being a cosmetic deformity, most cases of hyperkyphosis do not cause much pain or suffering. However, in some instances they may lead to an increased risk of vertebral fractures[76] or may be complicated by a thoracic postural pain syndrome (see following page).

Therapy includes the treatment of the acute exacerbations (see fractures), and of a thoracic postural pain syndrome. Also pharmacological treatment of the underlying osteoporosis with calcium and vitamin D supplementation together with bisphosphonates may be indicated.[77]

Vertebral body fractures

The thoracolumbar spine is the most common site for vertebral fractures.[78] In younger patients, thoracolumbar vertebral fractures are usually caused by high-energy accidents such as falls, or motor vehicle accidents; whereas in elderly patients, osteoporosis is the dominant aetiology.[79] Vertebral body fractures may also occur spontaneously as the result of an underlying disorder, such as a vertebral tumour, infection, or ankylosing spondylitis. All are classified as pathological fractures.[80]

High-energy fractures usually arise from an axial load in combination with flexion or lateral bending. If a compression injury with a significant flexion component is the cause, a wedge-shaped deformity with considerable loss of anterior vertebral height is commonly present (Fig. 2), resulting in angular kyphosis. Lateral bending leads to lateral wedging. In some cases the whole vertebral body is shattered (burst fracture) which can lead to serious neurological complications (Fig 3). A uniformly flattened vertebral body is more indicative of a pure axial component. Vertebral compression fractures due to osteoporosis are usually wedge fractures and have a milder clinical appearance.

Osteoporotic compression fractures

A fracture occurs when the weight of the upper body exceeds the ability of the bone within the vertebral body to support the load. In cases of severe osteoporosis the trauma may be subtle, such as stepping out of a bathtub, vigorous sneezing, or

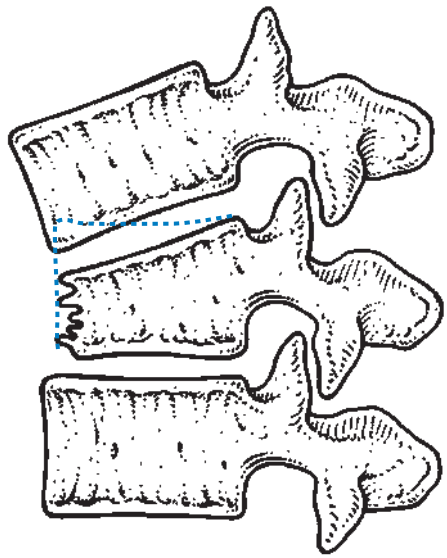

Fig 2 • Wedge compression fracture: the middle column (posterior part of the vertebral body) remains intact.

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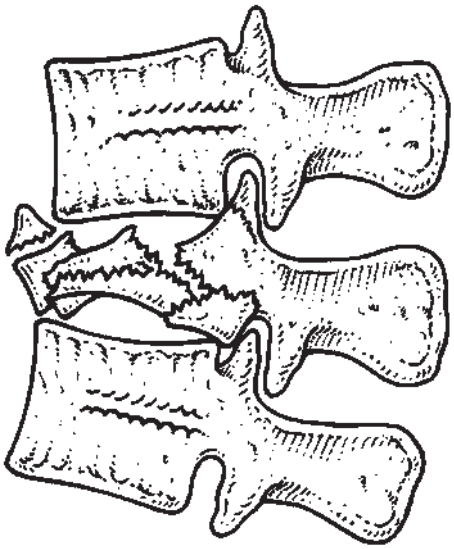

Fig 3 • Burst fracture: the posterior half of the vertebral column is disturbed and retropulsion into the neural canal may take place.

lifting a trivial object, or the trauma may result from the load caused by muscle contraction. In cases of moderate osteoporosis, more force or trauma is required to create a fracture, such as falling off a chair, tripping or attempting to lift a heavy object.

Symptoms

Central thoracic pain and bilateral girdle pain referred to the corresponding dermatomes. Standing or walking exacerbates the pain. Unsupported sitting is also uncomfortable. There may be twinges. Lying in the supine position generally relieves some of the discomfort.[81] Spinal compression fractures can also be insidious and may then produce only modest back pain early in the course of the progressive disease. This may explain the fact that only one-third of vertebral fractures are actually diagnosed, as the patient regards his back pain as a normal part of aging.[82]

Signs

Active and passive movements show a symmetrical pattern with pain at the end of both rotations and sideflexions. Extension is painful and limited and flexion is performed with support of the arms. Taking a deep breath may influence the pain. In cases of uncomplicated compression fractures, neck flexion and SLR are negative and neurologic examination is normal. Palpation shows tenderness directly over the area of the fracture, and an increased kyphosis may be noted.

Radiography

Plain frontal and lateral radiographs are the initial imaging study obtained for a suspected compression fracture. Compression of the anterior aspect of the vertebrae results in the classic wedge-shaped vertebral body with narrowing of the anterior portion. A decrease in vertebral height of 20% or more is considered positive for compression fracture.[83] Computed tomography can be used for evaluating the posterior vertebral wall integrity and for distinguishing a wedge fracture from a burst fracture. In the latter, the middle column, consisting of the posterior half of the vertebral body, the posterior longitudinal ligament and the posterior fibres of the annulus fibrosus, is disturbed, and varying degrees of retropulsion into the neutral canal takes place, provoking neurological signs (Fig. 3).[84]

Natural history

After about 2 weeks the pain diminishes progressively and it takes about 3 months for an uncomplicated wedge fracture to heal, leaving the vertebral body with a permanent wedge shape. Pain that is still present after the fracture has healed is explained by collateral damage that has been done to the disc or the surrounding ligaments. It should be noted that the kyphosis deformity may also cause later development of spinal stenosis or thoracic postural pain syndrome.

Treatment

Uncomplicated wedge fractures do not need specific treatment apart from relative rest during the period of spontaneous healing. If there is still pain after 3 months, a complete clinical re-assessment should be performed to reveal the source.

During the last decades vertebroplasty and kyphoplasty have been promoted for the management of severe recalcitrant pain. Initially reported by Galibert *et al* in 1987, vertebroplasty involved the destruction of an angioma through consolidation of the vertebral column by percutaneous injection of acrylic cement;[85] however, vertebroplasty is now commonly used in treatment of painful osteoporotic vertebral compression fractures.[86] Kyphoplasty involves the use of an inflatable bone tamp that when introduced into the vertebral body, restores vertebral height and forms a space for injection of acrylic cement.[87]

Pathological fractures

These are mainly the result of vertebral infections, ankylosing spondylitis and primary tumours or metastases.[80] If the patient complains of severe pain after trivial injury, a pathological fracture should be suspected. Sometimes the event may have been so minimal that the patient does not always mention it. The clinical presentation is the same as for other fractures of abrupt onset except that the pain may not fully disappear on recumbency.

Diagnosis is made with imaging techniques.

Warning

The following may indicate a fracture:

- Severe bilateral girdle pain after a minimal injury in the absence of pain on flexion of the neck: pathological fracture?
- Very severe pain on extension pressure.
- Capsular pattern following an injury.

Thoracic postural pain syndrome

Cyriax regarded this pain syndrome, which affects mainly young adults and the middle aged, as being the result of a posterior displacement of the whole intervertebral disc content, occurring mainly in patients with a marked thoracic kyphosis (Cyriax[7]). Although dural compression by the entire disc is very likely, ligamentous overstretching of the supra- and

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interspinal ligaments may also be a contributing factor. Prolonged and repeated anteflexion stretches the posterior longitudinal ligament, which may become too elastic. At the same time, the kyphotic posture causes a posterior directed force on the disc. After some years, the whole disc content bulges posteriorly through the longitudinal ligament. At first this occurs only during prolonged anteflexion and ceases slowly on lying down – the phenomenon is self-reducing. Later the displacement may become permanent. Once the disc comes in contact with the dura mater, or when the posterior ligaments become severely overstretched, the patient complains of pain, which is initially temporary but later becomes permanent.

The whole process depends on three elements, all of which aggravate the disorder:

- The duration of forward bending
- The degree of physiological kyphosis
- The load put on the disc.

Clinical presentations

The history is highly characteristic. Initially, the patients are well on first waking up in the morning, but pain starts after some hours of forward bending or after prolonged sitting in a kyphotic position. It is usually felt in the centre of the upper or mid-thoracic area but later may spread over the whole posterior thorax. The pain increases on carrying heavy objects or when the period of anteflexion is of longer duration. Initially, on lying down the pain disappears after some minutes, but in advanced cases it may take hours to ease off. As the condition develops, interscapular pain begins and increases slowly and steadily during the day, even if the patient does not bend forward. Finally pain becomes continuous day and night – the stage of anterior erosion has been reached.

On clinical examination, only a few articular signs are present and they are not very pronounced. In this disorder, a deep breath usually hurts at the moment the pain is maximally present. This element draws attention to a dural involvement (Fig. 4).

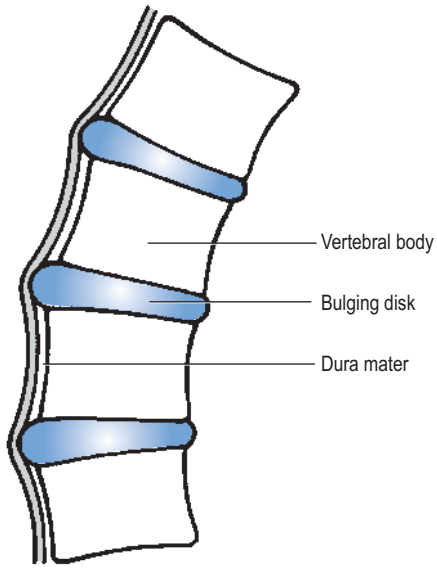

Fig 4 • Thoracic postural pain syndrome.

Treatment of the initial stage

Early treatment is in fact prevention: all kyphotic postures should be avoided.

If this is not sufficient, ligamentous sclerosant infiltrations over several adjacent levels around the maximum point of the kyphosis can be considered.

Treatment of the advanced stage

Once a patient is used to the pain, prediction of exactly after how many hours the pain will take to come on is possible. At this stage, the advice is to lie down before severe pain starts.

Besides preventive rest, daily traction can be given over 2 weeks, followed by a traction-free interval of 1 week, and with repetition for a further 2 weeks. The period without traction is progressively increased to a month, alternated with 2 weeks of traction. If this is successful, it should be continued indefinitely.

Anterior erosion

This is the final stage of the postural pain syndrome and is encountered only in the elderly. Progressive increase in hyperkyphosis finally erodes the anterior portion of the disc and the anterior bony parts of the vertebrae are in contact with each other. Bone sclerosis develops and anterior osteophytes form (Fig. 5). Pain that previously ceased at night is now constantly present and is probably due to the osseous contact between the vertebrae, with subsequent local inflammation. For this reason the pain remains local.

On clinical examination there is a marked thoracic kyphosis with very rigid joints. Therefore, the active articular movements no longer have much influence on the pain, but passive

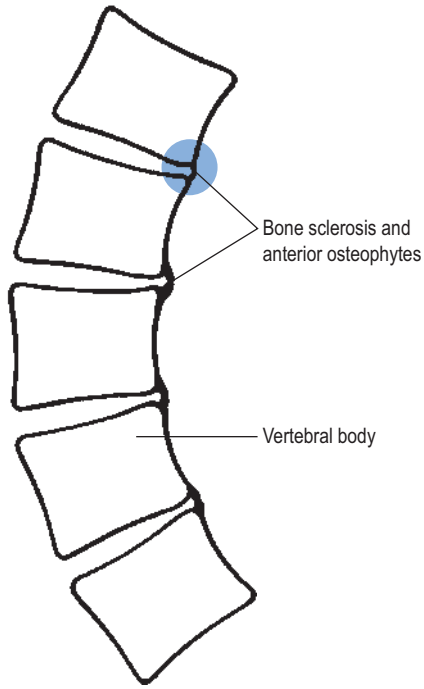

Fig 5 • Anterior erosion: bony contact between the two vertebral bodies at the anterior side may result in bone sclerosis and pain.

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articular movements may slightly increase the pain. Anterior osteophytes are visible on the radiograph.

Treatment consists of repeated traction at intervals as the complaints demand, as for the thoracic postural pain syndrome (see earlier). Recent studies demonstrated a significant improvement in pain and posture after rehabilitation of the kyphotic spine (combination of manual mobilization, taping and exercises).[88,89]

Scoliosis

Scoliosis[90] is defined as a lateral curvature of the spine of greater than 10°. The angle is determined by drawing lines across the upper surface of the vertebral bodies at which the curve changes direction – i.e. the vertebrae that tilt maximally into the concavity of the curve. Lines perpendicular to these are then drawn. The angle between the two perpendicular lines is Cobb's angle (Fig. 6). If it is greater than 10°, clinical scoliosis is present.

Sometimes scoliosis is associated with an increased thoracic kyphosis (posterior convex curvature) or a diminished lumbar lordosis (anterior convex curvature).

There are two types of scoliosis.[64] In structural scoliosis, the deformity cannot be voluntarily corrected by the patient. It is characterized by a fixed rotation on forward bending. Some cases are congenital, others acquired. Non-structural scoliosis is frequently postural and remains under voluntary control. No fixed rotation is present on anteflexion. It is not progressive.

Structural scoliosis

Congenital scoliosis

This is the result of congenital malformation such as hemivertebra. It is frequently severe and may produce grotesque cosmetic deformity, reduced pulmonary function and ultimately paraplegia.

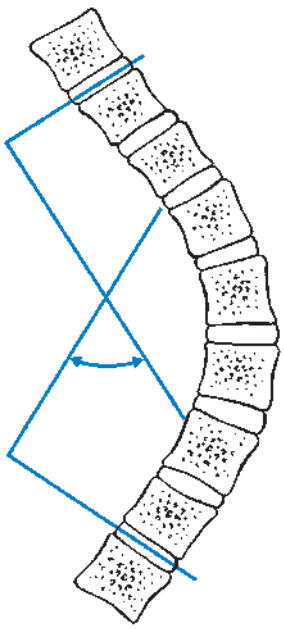

Fig 6 • Cobb's angle.

Acquired scoliosis

This is either secondary or idiopathic.

Secondary scoliosis

This has a local cause, often in association with developmental disturbances. Examples are intraspinal tumours (neurofibromatosis), neuromuscular dysfunction as in myopathies (muscular dystrophy), mesenchymal disorders (Marfan's syndrome), vertebral tumours, rheumatoid diseases, fractures and nerve root irritation. The pathophysiological mechanism of the thoracic malformation remains unknown in many of these.

Idiopathic scoliosis

In this, an underlying disorder is not present. Ninety per cent of cases are in this group, and some authors estimate the incidence as approximately 4.5% of all schoolchildren.[1,91]

Depending on the age of onset it is known as infantile scoliosis (0–4 years), juvenile scoliosis (4–10) and adolescent scoliosis (over 10 to skeletal maturity). Adolescent scoliosis sometimes has adult onset when it affects females between 20 and 40 years of age. Severe adolescent scoliosis affects girls four times more often than boys, whereas less pronounced deviations are seen equally in both sexes.[91]

One curious feature of the disorder is that in childhood, 90% of the curvatures are convex to the left, whereas in adolescence this is reversed.[92] The condition worsens as bone growth increases and rarely changes after bone growth has stopped. However, if the angle of deformity between the lumbar and thoracic spines remains less than 60°, an effect on vital functions is not to be expected.

Clinical presentation

Controversy surrounds the incidence of thoracolumbar pain in scoliosis. Some authors contend that in the main the condition is asymptomatic and that patients with scoliosis do not suffer more from back pain than those with a normal back.[93] Others maintain that the larger the scoliosis the more likely it is that pain will occur.[94] However, some patients initially experience pain at the end of the day, which stops rapidly on lying down.[94] The cause may be overstretching of ligaments, overloading of the facet joints or temporal displacement of the disc. All these possibilities are compatible with a non-articular pattern on clinical examination. The pain is usually located at the apex of the curvature. Progression of the deformity is usually associated with increased pain.

On inspection, attention must be paid to scapular asymmetry, prominence at one side of the thorax, the distance between the arms and trunk and the level of the pelvis. Because a contralateral rotation occurs on side flexion, scoliosis is associated with a unilateral thoracic prominence at the convex side of the scoliosis.[64,95] Prominence of the thorax is most easily noted when the patient bends over.

During functional examination, one side flexion is sometimes limited. Pain, if present, is of the mechanical type and gives rise to a partial articular pattern on clinical examination.

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Advanced scoliosis may lead to a decreased vital capacity and to cardiorespiratory dysfunction.

Further investigations

Radiography establishes the exact location of the curvature, its degree of severity (Cobb's angle) and the stage of skeletal maturity, all of which are important for diagnosis, follow-up and treatment. Skeletal maturation is judged from the ossification and closure of the vertebral ring apophyses.[64]

Progression is defined as an increase in Cobb's angle of at least 5° between two consecutive radiographs.

Prognosis

The higher the curvature is situated in the spine, the less favourable the outcome. Progression is possible for as long as vertebral growth continues.

Treatment

Different types of treatment may be indicated: observation, drugs, exercises, braces and surgery (see Box 1). It should be noted that there is an important distinction to be made between the treatment for children and that for adults.

Observation

This is undertaken in the following:

- All patients who have an immature skeleton, and with curve of less than 20°
- Curves of less than 40° in skeletally mature patients
- Curves that are already thought to be non-progressive.

In all such cases, observation should be made at regular intervals to detect whether the curve progresses.[96] Usually it is sufficient to see the patient every 6 months. In adolescents nearing the growth spurt, a check-up every 3 months is more appropriate. Exercises are of no use in this stage.

Active treatment

Patients with a progressive curvature of 20° or more, or with an established curve greater than 30°, and who are skeletally immature require active treatment by either orthosis or operation:

- *Brace:* a young adult having a Cobb's angle of less than 40°, in the absence of severe pain, is best treated by conservative means: non-specific anti-inflammatory agents, facet infiltrations and physical monitoring. A Milwaukee brace can also be considered. The main goal of the use of a brace is to prevent further progression, rather than to correct the curvature. A Milwaukee brace or plastic jacket can be used, which extends the spine by pushing it up cranially from the hips. However, a recent study of late-onset idiopathic scoliosis raises questions about the efficacy of spinal orthoses.[97] Braces are contraindicated in skeletally mature patients or if the curvature is over 40° – the latter usually do not respond well. A brace cannot be used in thoracic lordosis. When brace treatment is given, associated exercises become important in order to keep the muscles of the trunk and the abdomen in good condition.
- *Operation:* if the curve is over 40° in a skeletally immature patient, or when it is progressive or over 50° in a mature skeleton, spinal fusion is indicated to straighten and stabilize the spine.[96] Other indications for operative intervention are when orthotic treatment has failed, if pain becomes uncontrollable or when a thoracic lordosis is found.

Older adults are more likely to suffer from neural compression. The major aim of treatment is to maintain function. With a curve under 40° this is usually possible without surgery. If there is uncontrolled pain or a progressive curve or structural disabilities, neurological complications or cardiorespiratory problems, surgical internal fixation is essential.[5,98]

Box 1

Treatment of scoliosis

Observation	Immature skeleton, <20°
	Mature skeleton, <40°
	Non-progressive curves
Active treatment	
Brace	Indications:
	Immature skeleton, >30°
	Progressive curves of >20°
	Mature skeleton <40°, no severe pain in young adult
	Contraindications:
	Mature skeleton
	Curve >40°
	Thoracic lordosis
Spinal fusion	Indications:
	Immature skeleton, >40°
	Mature skeleton, >50°
	Thoracic lordosis
	Failure of orthotic treatment
	Uncontrollable pain
	Progressive curve in adults

Non-structural scoliosis

The deformity is mainly found in adolescents and can be corrected by a voluntary effort. It is most frequently postural, occasionally the result of mental disturbance. On forward bending, the patient aligns the head and spine over the pelvis so that the scoliosis disappears. The term 'compensatory scoliosis' is sometimes used. The condition is not of clinical importance.

Scoliosis associated with inequality of limb length is usually confined to the lumbar spine.[99]

Lateral erosion

Just as hyperkyphosis may lead to thoracic postural pain syndrome, and finally to anterior erosion, moderate and severe scoliosis may result in lateral erosion. The process is the same for both, but in scoliosis the entire disc of several consecutive levels shifts laterally to one side. After some years, this may

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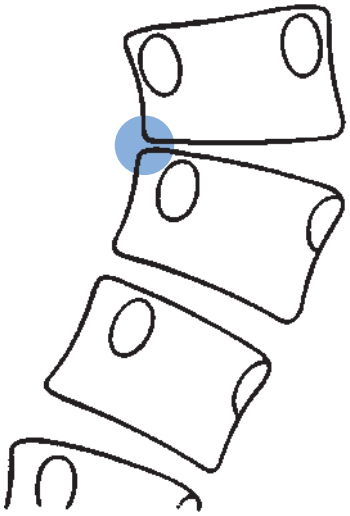

Fig 7 • Lateral erosion: bony contact between the two vertebral bodies at the concave side may result in bone sclerosis and pain.

lead to bony contact between the vertebral bodies at the concave side of the scoliosis (Fig. 7).

Because the disc is displaced directly laterally, pressure on the dura mater or nerve root is not to be expected. Consequently, pain is felt only late in the development of the condition when bone sclerosis starts. The pain remains unilateral and is not increased by a deep breath. Initially, it is felt only at the end of the day and disappears at night or after a period of recumbency. Later, the pain comes on earlier in the day and disappears only after lying down for longer and longer periods. Finally, it becomes constant.

Treatment

Traction in the same way as for anterior erosion can be given but is often not successful. Arthrodesis is the alternative.

Fracture of a transverse process

At the thoracic level this is usually the result of a direct blow or of an extension manipulation. After the injury the patient suffers immediately from unilateral segmental pain, increased on active and resisted side flexion towards the painless side. Resisted extension is also painful. Spontaneous resolution of the pain occurs in about 2 weeks.

Spinal infections

Vertebral osteomyelitis develops when an infectious organism enters the bone. A number of bacteria cause pyogenic osteomyelitis. Tuberculosis produces Pott's disease. An infection limited to the intervertebral disc is known as adult intervertebral disc space infection. In a later stage, when the infection progresses, it may also affect the vertebral body.

Both osteomyelitis and disc space infections must be differentiated from discitis or intervertebral disc inflammation, which only affects children.

Vertebral osteomyelitis

Pyogenic osteomyelitis

Since the introduction of antibiotics this disorder has changed from a disease of childhood and adolescence to one affecting mainly adults. The lumbar spine is most commonly involved.[100] The most frequent organism is a *Staphylococcus* sp.[101] In about 40% of patients, an extraspinal primary source of infection is found, very often sited in the pelvis (urinary tract, postpartum, bowel).[1] The presence of diabetes mellitus, intravenous drug abuse, underlying chronic debilitating diseases or immunosuppression, previous infections, preceding bacteraemia and recent vertebral surgery are significantly associated with pyogenic osteomyelitis.[102,103]

Clinical presentation

Although the onset may be abrupt, pyogenic osteomyelitis is most commonly insidious and progresses slowly. The pain is primarily felt locally at the site of the infection and is constantly present but often worse at night. At onset, the pain may be provoked or increased by activity and relieved in recumbency.

As the condition progresses, pain usually becomes more dramatic, leading to a full articular pattern with gross limitation mainly of extension, of both side flexions and rotations. Rigidity and muscle spasm are also present. Gross tenderness on local pressure over the spinous processes is found. A soft tissue mass may be palpable. Chills and weight loss complete the clinical presentation. Thecal sac neurocompression by the formation of an epidural abscess may cause severe neurological disturbances (paraplegia or paraparesis).[104]

Further investigations

The erythrocyte sedimentation rate (ESR) is constantly raised and is a good indicator of the activity of the disease.[105,106] An increased white cell count is found in only half of the cases, and it seldom rises above 15 000.[102,107]

Spinal radiographs become positive at the earliest after 2 or 3 weeks of infection,[107] when a narrowed disc space is the first sign found. This narrowing cannot be distinguished from narrowing that is the result of degenerative changes. Only later, after 10–12 weeks, do the adjacent endplates become more dense and eventually blurred, once destructive erosion has set in. Healing is associated with osteosclerosis and leads to bony fusion across the disc space.

Spongy bone erosion and rarefaction is apparent on CT scan before it is on plain radiography. However, gallium bone scan proves to be the most valuable technique to detect early inflammatory changes and it becomes positive earlier than does CT scan. In a recent study, gallium scanning proved to be 100% sensitive, specific and accurate.[108]

Treatment

The administration of antibiotics and bed rest until the ESR has returned to normal. Treatment of neurological deficit caused by epidural abscess is prompt surgical decompression, with or without fusion.[109]

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Tuberculosis of the spine

Vertebral tuberculosis most frequently involves the lower thoracic and upper lumbar spines. It is always the consequence of haematogenous spread from a primary visceral focus (pulmonary or urinary tract) and it is the vertebral body that is involved, because the disc is avascular. However, spread to adjacent structures – discs, muscles and ligaments – may occur and a paravertebral abscess may form.

Clinical presentation

This may be very different from a pyogenic vertebral infection. The onset is very slow and it is not unusual that many months will have elapsed before the diagnosis becomes obvious. Backache of lesser severity than in pyogenic infection is usual. Other signs and symptoms – intermittent fever, night sweating, weight loss and fatigue – may draw attention to tuberculosis.

A localized angular bony deformity from vertebral collapse may be visible. On functional examination a full articular pattern is found, together with severe local tenderness on extension pressure. Dural symptoms and signs are not present.

Further investigations

The ESR is elevated but the white cell count may vary. Radiography of the chest and bacteriological examination of urine must always be done.

On the radiograph, involvement of one vertebra is typical.[110] The most common early findings are a diminished disc space and osteolysis of the vertebral body. Later, paravertebral granulation tissue is formed, resulting in paravertebral mass.[111] This is usually followed by collapse of the vertebral body with formation of an angular kyphosis.

A positive Mantoux test is not diagnostic in itself but does arouse suspicion of tuberculosis.

Treatment

This comprises bed rest and administration of antituberculous drugs. Early surgical intervention with anterior debridement and grafting has been advocated to shorten the course of the disease, and it decreases the risk of neurological complications of deformity.[102,110]

Intervertebral disc inflammation: discitis

This disorder of unknown cause affects only children, most commonly between 2 and 6 years of age. Whether infection is involved remains uncertain. The lumbar spine is most usually affected.

The first complaint is of back or hip pain; smaller children may just refuse to walk.

On clinical examination, limited spinal movement with muscle spasm and local tenderness is found. Fever is present.

Some weeks after onset, some narrowing of the intervertebral disc space and erosion of the endplates can be seen. Although these radiographic findings are usually sufficient to establish the diagnosis of discitis, MRI is the diagnostic study of choice especially in a differential diagnosis with suspected vertebral osteomyelitis.[112] Immobilization of the spine and antibiotics are the main treatment required.

Lateral recess stenosis

In some older patients, the intervertebral disc becomes completely worn out and its fragments displace posteriorly because of physiological kyphosis. As a consequence, the intervertebral space decreases progressively and the posterior longitudinal ligament becomes relatively long. Under the influence of the body weight it bulges posteriorly and may come in contact with the dura mater, causing central backache radiating bilaterally around the trunk, or may compress the nerve root, causing unilateral root pain (Cyriax:[7] p. 211). This condition is characterized by pain that comes on immediately on standing and, unlike lumbar lateral recess stenosis, is not altered by sitting or stooping. Only when the patient lies down for 1 or 2 minutes does the pain cease, when it stops almost immediately.

Because the condition occurs mainly in an elderly patient with a very stiff vertebral column, no appreciable discomfort can be provoked by articular movements. In root compression, side flexion towards the painful side is occasionally painful.

Treatment

This depends on the site of the pain.

Central pain

Daily traction can be tried. The aim is to bring as much of the disc material back into the intervertebral space as possible, so that on load the intervertebral height diminishes to a lesser degree and compression of the dura does not occur. If successful, it should be continued indefinitely on every other day. If traction fails arthrodesis is called for.

Unilateral root pain

If instead of central pain unilateral root pain is present, an infiltration around the nerve root should be given and can make the inflammation disappear. If necessary, it can be repeated every 6–12 months (for technique, see Ch. 27).

Arthritis of the costovertebral and costotransverse joints

Disorders of these joints that give rise to clinical symptoms are rare. They may be affected by osteoarthritis, ankylosing spondylitis and by rheumatoid arthritis.[113,114] Degeneration is identified mainly in the costovertebral joints of the first, eleventh and twelfth ribs, all of which have only one articular facet. It is possible that these are more vulnerable to mechanical irritation by continuous rib motion, sometimes as the outcome of a mechanical injury.[115] Less frequently the sixth, seventh and eighth costovertebral joints are affected, and in these it is most frequently the inferior hemifacet.[116] It has been suggested that patients often have an increased thoracic kyphosis with associated scoliosis.[115]

Involvement of these joints gives rise to unilateral paravertebral pain radiating along the ribs. The pain is described as aching or burning, is usually worse in the morning and is aggravated by deep inspiration and a cough.[115–117] Exceptionally, on breathing, a sudden twinge that arrests further inspiration is felt.

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On clinical examination an asymmetrical pattern is encountered. Neck flexion and scapular movements are negative. Pain is elicited, on performing the springing test of the rib.

When the first costotransverse joint is affected, the patient complains of pain unilaterally at the base of the neck. The pain is provoked by the following movements of the neck: active and passive flexion, contralateral side flexion and resisted side flexion towards the painful side. Pain may also be present on active and passive elevation of the arm and the scapula.

Standard radiography, CT scan and technetium bone scans may show abnormalities of these joints.

Treatment

If only one intercostal joint is affected, an intra-articular injection should be given. Should this be unsuccessful, surgical excision of the joint may be considered.

Warning

There is a considerable risk of pneumothorax from these injections. Extreme care must be taken and should only be performed under radiological control.

Arthritis of the thoracic facet joints

Degeneration of thoracic facet joints most frequently affects levels C7–T1, T3–T5 and T11–L1.[118,119] The diagnosis should be considered in those patients whose complaint is a unilateral and local paravertebral pain. There are no dural symptoms or signs. The sole clinical finding is pain on one of the rotations, actively and/or passively performed. This can be either the rotation towards or away from the painful side, a situation resembling facet disorders in the cervical area. Other passive movements are far less painful or not painful at all and resisted rotations are painless. Extension pressure on the spinous processes elicits pain on two consecutive processes, indicating the level of the lesion.

These patients are often first mistakenly regarded as suffering from a disc lesion, because they show a partial articular pattern. After manipulation they may obtain some relief but it usually does not last longer than a few hours to a day. Therefore, if there is doubt about the diagnosis, the capsule of the affected facet joint should be infiltrated with 2 ml of lidocaine (lignocaine) 2%, which provides immediate relief in facet joint disorder.

Treatment

Cure is obtained with one or two infiltrations of 1 ml of triamcinolone over 1–2 weeks.

Technique: injection of a thoracic facet joint (Fig. 8)

The patient lies prone with the arms along the body. The two most tender spinous processes are marked and the facet joint between the corresponding vertebrae is identified. Depending on the level, it lies about 1 (upper and lower thorax) to 1½ (mid-thorax) interspinal levels higher than the lower tender spinous process.

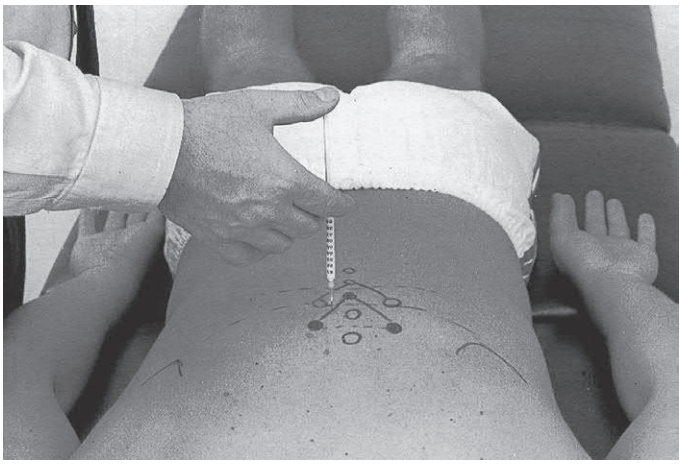

Fig 8 • Injection of a thoracic facet joint.

A syringe is filled with 1 ml of triamcinolone and a 4 cm needle is fitted. The needle is inserted vertically downwards about 1 cm from the midline. The needle is usually stopped by the lamina. It is now slightly withdrawn and moved upwards or downwards until the typical capsular resistance is felt before ending at the bone. The steroid is then injected with the needle in bony contact.

The patient must return after 2 weeks. If there is still some pain, the injection is repeated.

If the treatment fails, a blockade with phenol solution of the medial branch of the dorsal ramus of the thoracic spinal nerve can be performed. Good results have also been reported with percutaneous radiofrequency denervation of the thoracic facet.[120]

Paget's disease

This focal, non-generalized osseous disorder is characterized by thickening and deformation of the affected bone and is caused by hyperactive osteoclasts and osteoblasts, which give rise to increased bone resorption together with formation of disorganized new bone. It occurs more commonly than is generally believed and affects about 3% of the population above 40 years of age. This figure rises to about 5–10% in people aged over 80.[121] There is a slight preponderance of males and the condition is most frequently encountered in Western Europe, Australia and New Zealand.

Flat bones and the ends of the long bones are mainly affected. Most frequently involved are the spine, skull, sacrum, pelvis and femurs. About 60% of patients with Paget's disease have involvement of the lumbar spine, and 45% of the thoracic spine and sacrum. In only 15% is the cervical spine involved.[122]

Clinical presentation

Only about 12% of the patients affected by Paget's disease suffer from backache from the disease as such.[123] It should be recognized that other disorders, such as disc protrusions and facet joint problems, may be the basis of the pain in patients who are identified as having Paget's disease. In two-thirds the disease remains clinically asymptomatic.[124] These cases are

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usually detected incidentally on a radiograph taken for some other reason or by an increased serum alkaline phosphatase level.

Paget's disease usually starts in the middle aged or elderly and may give rise to local pain, local heat and deformation. The complaint is usually of a dull pain that does not increase at night. Characteristic deformities – lateral bowing of the femur, anterior bowing of the tibia, increased size of the skull and decreased length of the spine – may occur. Osteoporosis and fractures from minimal trauma are often encountered and usually present in the femur and tibia, less often in the spine. The typical Paget's stature with dorsal kyphosis may be seen. Involvement of the spine occasionally results in spinal stenosis.

Complications

Because of multiple arteriovenous fistulae in bone, there is lowered peripheral resistance with a rise in cardiac output, which may lead to cardiac decompensation. Enlargement of the skull, with secondary compression of peripheral nerves, may result in hearing loss, tinnitus and vertigo.[1]

Pagetoid bone may convert to a highly malignant sarcoma with very poor prognosis. The probability is higher when there is polyostotic involvement.

Radiology

Radiological findings depend on the stage. The initial phase is characterized by lytic bone lesions: osteoporosis circumscripta. These are rarely seen in the spine. Later, a mixed stage develops in which both demineralization and sclerosis of bone exist. The final sclerotic stage is characterized by a homogeneous increase of bone density.

In the vertebrae, coarse trabeculae and a marked edge of the vertebral body ('picture frame' appearance) are present. The central portion appears decalcified. Ultimately, the bone becomes dense. When diagnostic doubt exists, bone scans must be taken. They are more sensitive than plain radiographs.

Laboratory

Serum calcium and phosphorus concentrations are within normal limits, but the serum alkaline phosphatase is usually very high.

Treatment

Most patients are asymptomatic and do not need any treatment. More active disease may require calcitonin or bisphosphonate to reduce the symptoms but none of them is curative.

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Disorders of the thoracic cage and abdomen

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Pain in the thorax or abdomen can be the result of a local problem of either the thoracic wall or the abdominal muscles but it is more often referred from a visceral structure or from another musculoskeletal source, most frequently a disc protrusion. Therefore, it is wise to remember the only safe approach in this area is to achieve a diagnosis by both positive confirmation of the lesion and exclusion of other possible disorders.

Referred pain

Pain referred from visceral structures

All visceral structures belonging to the thorax or abdomen may give rise to pain felt in this area (see Ch. 25). In that the discussion of these disorders is principally the province of internal medicine, only major elements in the history that are helpful in differential diagnosis from musculoskeletal disorders are mentioned here. Acute chest pain is summarized in Box 1.

Heart (Fig. 1)

Ischaemic heart disease

The innervation of the heart is derived from the C8–T4 segments. Pain is therefore not only felt in the chest but can also be referred to the ulnar side of both upper limbs, though more commonly to the left.

It is traditionally accepted that pain felt in the chest radiating into the left arm is indicative of myocardial ischaemia, especially when the patient reports it as pressure, constriction, squeezing or tightness. However, none of these descriptions, which are usually regarded as characteristic of ischaemia, is of definitive aid in the differential diagnosis from other non-cardiogenic problems in the thorax. Even relief of pain after the intake of glyceryl trinitrate does not offer absolute confirmation of coronary ischaemia. For clinical diagnosis, a combination of several elements must be present, of which the most important is pain spreading to both arms and shoulders initiated by walking, especially after heavy meals or on cold days.[1]

Mitral valve prolapse

This condition usually gives rise to mild pain located in the left submammary region of the chest and sometimes also substernally. Occasionally it mimics typical angina pectoris and is sometimes accompanied by palpitations.

Pericarditis

Pain that arises from the pericardium is the consequence of irritation of the parietal surface, mainly from infectious pericarditis, seldom from a myocardial infarction or in association with uraemia. When pericarditis is the outcome of one of the latter two causes it is usually only slight. Pain is normally located at the tip of the left shoulder, in the anterior chest or in the epigastrium and the corresponding region of the back. Three different types of pain can be present. First and most

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Box 1

Summary of acute chest pain

Severe chest pain of abrupt onset should arouse suspicion of:

- Myocardial infarction
- Dissecting aneurysm
- Pneumothorax
- Pulmonary embolus
- Rupture of the oesophagus
- Acute thoracic disc protrusion

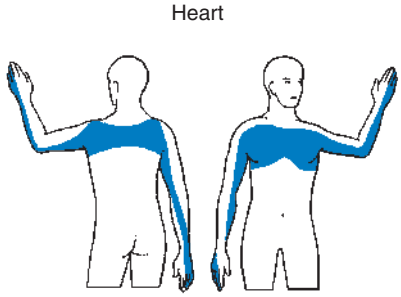

Fig 1 • Referred pain in lesions of the heart.

obvious, but rarely encountered, is pain synchronous with the heartbeat. Second is a steady, crushing substernal ache, indistinguishable from ischaemic heart disease. Third and most common is pain caused by an associated localized pleurisy, which is sharp, usually radiates to the interscapular area, is aggravated by coughing, breathing, swallowing and recumbency, and is alleviated by leaning forward.[2]

Aorta

Aneurysm of the thoracic aorta

This is most frequently the result of arteriosclerosis but is rare by comparison with the same condition below the diaphragm. The majority of small aneurysms remain asymptomatic, but if they expand a boring pain results, usually from displacement of other visceral structures or erosion of adjacent bone. Compression of the recurrent nerve may result in hoarseness and compression of the oesophagus in dysphagia. When acute pain and dyspnoea supervene, this usually indicates that the aneurysm has ruptured, with a likely fatal outcome.

Dissecting aneurysm of the thoracic aorta

This is an exceptional cause of chest pain, occurring mainly in hypertensive patients. The process usually starts suddenly in the ascending aorta, giving rise to severe substernal or upper abdominal pain. Radiation to the back is common and back pain may sometimes be the only feature, expanding along the area of dissection as it progresses distally. The patient often describes the pain as tearing. In most cases, it is not changed by posture or breathing.

When aortic dissection involves the vessels that supply the spinal cord, neurological changes and even paraplegia may result.[3]

Pleuritic pain

Pleuritic chest pain is a common symptom and has many causes, which range from life-threatening to benign, self-limited conditions. Because neither the lungs nor the visceral pleura have sensory innervation, pain is only present if the parietal pleura is involved, which may occur in inflammation or in pleural tumour. Invasion of the chest wall by a pulmonary neoplasm also provokes pain.

Clinical presentation

Pleuritic pain is localized to the area that is inflamed or along predictable referred pain pathways. Parietal pleurae of the outer rib cage and lateral aspect of each hemidiaphragm are innervated by intercostal nerves. Pain is therefore referred to their respective dermatomes. The central part of each hemidiaphragm belongs to the C4 segment and therefore the pain is referred to the ipsilateral neck or shoulder.

The classic feature is that forceful breathing movement, such as taking a deep breath, coughing, or sneezing, exacerbates the pain. Patients often relate that the pain is sharp and is made worse with movement. Typically, they will assume a posture that limits motion of the thorax. Movements of the trunk which stretch the parietal pleura may increase the pain.

During auscultation the typical 'friction rub' is heard. The normally smooth surfaces of the parietal and visceral pleurae become rough with inflammation. As these surfaces rub against one another, a rough scratching sound, or friction rub, may be heard with inspiration and expiration. This friction rub is a classic feature of pleurisy.

Aetiologies

Pneumonia

Although the clinical presentation of pneumonia may vary, classically the patient is severely ill with high fever, pleuritic pain and a dry cough.[4]

Carcinoma of the lung

In carcinoma of lung, pain is consequent upon involvement of other structures such as the parietal pleura, the mediastinum or the chest wall. Invasion of the chest wall may cause spasm of the pectoralis major muscle, which subsequently leads to a limitation of both passive and active elevation of the arm.

Pleural tumour

Malignant mesothelioma is a diffuse tumour arising in the pleura, peritoneum, or other serosal surface. The most frequent site of origin is the pleura (>90%), followed by peritoneum (6–10%), and only rarely other locations. Mesothelioma is closely associated with asbestos exposure and has a long latency (range 18–70). There is no efficient treatment and the overall survival from malignant mesothelioma is poor (8.8 months).[5,6]

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Pleuritis

This is characterized by a sharp superficial and well-localized pain in the chest, made worse by deep inspiration, coughing and sneezing. Viral infection is one of the most common causes of pleuritic pain.[7] Viruses that have been linked as causative agents include influenza, parainfluenza, coxsackieviruses, respiratory syncytial virus, mumps, cytomegalovirus, adenovirus, and Epstein-Barr virus.[8]

Pulmonary embolism

Pulmonary embolism is the most common potentially life-threatening cause, found in 5–20% of patients who present to the emergency department with pleuritic pain.[9,10]

Predisposing factors for pulmonary embolism are: phlebothrombosis in the legs, prior embolism or clot, cancer, immobilization, prolonged sitting (aeroplane), oestrogen use or recent surgery.[11]

Symptoms and signs are mainly dependent on the extent of the lesion. A small embolus may give rise to effort dyspnoea, abnormal tiredness, syncope and occasionally to cardiac arrhythmias. A medium-sized embolus may lead to pulmonary infarction, so provoking dyspnoea and pleuritic pain. In a massive pulmonary embolus, the patient complains of severe central chest pain and suddenly shows features of shock with pallor and sweating, marked tachynoea and tachycardia. Syncope with a dramatically reduced cardiac output may follow. This is a medical emergency: death may follow rapidly.

Acute pneumothorax

This is characterized by a sudden dyspnoea and unilateral pain in the chest, radiating to the shoulder and arm on the affected side and often described as a tearing sensation. Breathing and activity increase the pain. The typical features of pneumothorax are tachycardia, hyperresonance on percussion and decreased breath sounds on auscultation.

Superior sulcus tumour of the lung (Pancoast's tumour)

This warrants special attention because 90% of patients suffering from this disorder complain of musculoskeletal pain.[12,13] It is frequently mistaken for a shoulder lesion or even for thoracic outlet syndrome, an error which leads to a delay in diagnosis and treatment.[14]

The superior pulmonary sulcus is the groove in the lung formed by the subclavian artery as it crosses the apex of the lung. Because most apical tumours have some relation to the sulcus, they are often called superior sulcus tumours. They frequently involve the brachial plexus and the sympathetic ganglia at the base of the neck and may destroy ribs and vertebrae.

Pain around the shoulder, radiating down the arm and towards the upper and lateral aspect of the chest is usual and is often worse at night.

Orthopaedic clinical examination produces an unusual pattern of clinical findings. There is often a complicated mixture of cervical, shoulder and thoracic signs. Passive and resisted movements of the cervical spine may be limited and/or painful, the result of involvement of the scaleni and sternocleidomastoid muscles. On examination of the shoulder girdle, a restriction of both active and passive elevation of the scapula may be present. More positive signs are detected during examination of the shoulder.[15] Both active and passive elevations of the arm are limited because of spasm of the pectoralis major muscle. Passive shoulder movements may be considerably limited in a non-capsular way. Some resisted movements are weak.

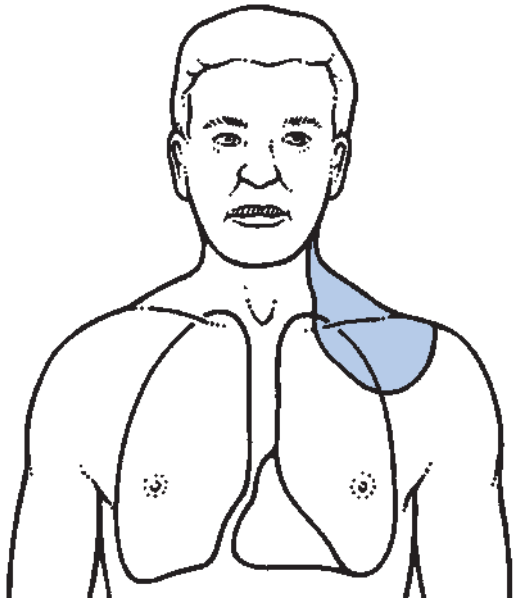

Fig 2 • The clinical symptoms of a superior sulcus tumour of the lung are produced by local extension into the chest wall, the base of the neck and the neurovascular structures at the thoracic inlet.

The neurological examination of the upper limb shows weakness and atrophy of the muscles on which consequent is extension of the tumour to the lower trunks of the brachialis plexus (Fig. 2). The only abnormal finding during thoracic examination is pain and limitation on lateral flexion towards the unaffected side explained by putting the affected thoracic wall under stretch.

The clinical picture of Pancoast's tumour may be completed by some typical findings that are caused by an ingrowth of neurological and vascular structures at the apex of the lung.[16]

These include:

- *Horner's syndrome*: this is characterized by an ipsilateral slight ptosis of the upper lid, miosis of the pupil and enophthalmos, together with decreased sweating on the same side of the face. It is the outcome of involvement of the ascending sympathetic pathway at the stellate ganglion on the side of the tumour.[17]
- *Hoarseness*: this is the result of involvement of the recurrent laryngeal nerve, which innervates the voice cords. The hoarseness is unusual and unlike that caused by local laryngeal problems.
- *Oedema and discoloration of the arm*: this occurs if the subclavian vein is obliterated by the tumour.

All the symptoms and signs mentioned (summarized in Box 2), either singly or in combination, call for careful clinical chest

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Box 2

Superior sulcus tumour of the lung

Symptoms

Aggravating shoulder–arm pain (pulmonary symptoms)

Signs

Cervical spine: impaired movements and positive resisted tests
Shoulder girdle: impaired movement
Shoulder: limited arm elevation/noncapsular pattern/weak resisted tests
Upper limb: weakness and atrophy
Thoracic spine: limited side flexion away from the affected side
Horner's triad: ptosis, miosis, enophthalmos
Hoarseness
Oedema of the arm

examination followed by further investigation by chest radiography or other imaging methods.

Mediastinal problems

Acute mediastinitis

This is a rare inflammation, usually the result of perforation of the oesophagus. The three causes are perforation of malignant tumour, ingestion of caustics leading to necrosis and Mallory–Weiss syndrome, in which vomiting without appropriate relaxation of the oesophagus causes a tear of the oesophagogastric junction, often incomplete in thickness. There is very severe substernal and central dorsal pain of abrupt onset, followed by high fever and shock. Without treatment, it is rapidly fatal.

Mediastinal emphysema

This is usually the consequence of a ruptured pleural bleb or a wound of the chest. Air spreads into the mediastinal tissues, giving rise to sudden or more gradual substernal pain, sometimes radiating into the neck, shoulders and interscapular area. Subcutaneous crepitus above the clavicle is pathognomonic for the condition.

Mediastinal tumours

These may give rise to anterior or posterior substernal pain, which is usually steady, of mild severity and often worse at night. Radiography reveals the diagnosis.

Oesophagus (Fig. 3)

Oesophageal spasm

This occurs suddenly and gives rise to substernal aching not necessarily related to the intake of food. Relief is often obtained by drinking hot water.

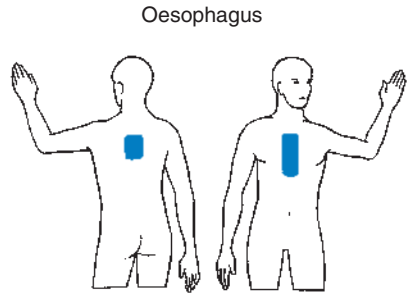

Fig 3 • Referred pain in oesophageal lesions.

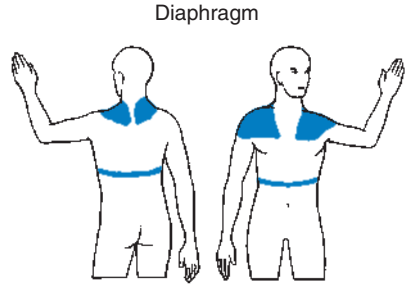

Fig 4 • Referred pain in diaphragmatic lesions.

Reflux oesophagitis

This is frequently due to a hernia of the stomach through the diaphragmatic hiatus. Pain is felt around the xiphoid process, can be very severe and may radiate to the rest of the sternum, into the back and between the scapulae.[18] Pyrosis or heartburn, which begins if the patient lies down immediately after meals, together with a burning sensation on eructation, are the most typical symptoms.

Rupture of oesophagus

The symptoms are the same as those from acute mediastinitis (see above).

Malignant tumour of the oesophagus

The initial symptoms are food lodgement at the site of the tumour. Later there may be constant anterior or posterior central chest pain, unrelated to eating and mainly the result of extraoesophageal extension of the tumour. Total dysphagia may follow, and remarkable weight loss over a short period of time is an ominous sign.

Diaphragm (Fig. 4)

Diaphragmatic irritation

This can be the result of a subphrenic abscess or of air in the abdomen after laparoscopy or laparotomy. Pain arising from the central part of the diaphragm is referred to the base of the neck and into the shoulders, mainly in the third and fourth cervical segments. Pain originating from the peripheral part is felt more at the lower thorax and in the upper abdomen.

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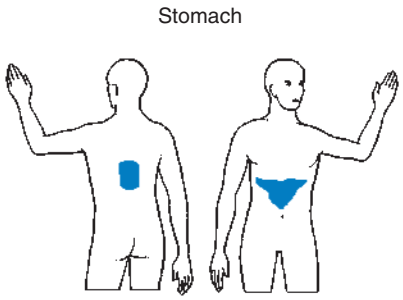

Fig 5 • Referred pain in gastric or duodenal lesions.

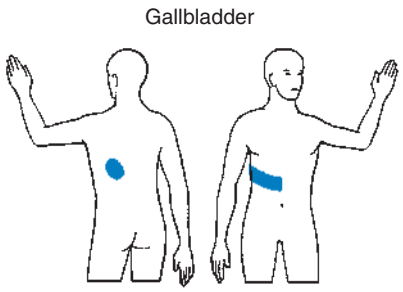

Fig 6 • Referred pain in lesions of the gallbladder.

Diaphragmatic hernia

This usually occurs due to displacement of the proximal part of the stomach as a whole when the patient is prone or head down or when intra-abdominal pressure is increased, as on straining or lifting. Pain, pyrosis and dysphagia may result. Pain usually disappears in the upright position. Hernia often causes reflux oesphagitis (see above).

Subphrenic abscess

Abscesses that are truly just below the diaphragm can occur either to the right or to the left. Many so-called subphrenic abscesses are in fact below the liver and usually follow a perforation of the gastrointestinal or biliary tract, often after surgery. Signs and symptoms are fever and upper quadrant pain, sometimes with associated shoulder pain and local tenderness along the costal margin. Dyspnoea may be associated. Persistent fever and a history of a recent intra-abdominal sepsis should arouse suspicion.

Stomach and duodenum (Fig. 5)

Gastritis

An inflammation of the superficial gastric mucosa may be the result of the intake of non-steroidal anti-inflammatory drugs, alcohol or excessive meals. There is usually epigastric pain of short duration.

Gastric or duodenal ulcer

These result in epigastric or substernal pain, often associated with inability to digest food. The pain usually ceases on intake of antacids or food. Other symptoms, such as nausea, vomiting, heartburn and flatulence, are atypical. In duodenal ulcer, the pain commonly comes on through the night and also occurs 1–1½ hours after meals. A bout of symptoms over weeks or months may be followed by a similar period of relief. Pain in the back suggests a posterior ulcer that has penetrated a structure such as the pancreas.

Gastric tumours

Poor general health with weight loss, nausea, anorexia and vomiting is the most frequent presentation. Dysphagia can occur, and epigastric pain is usually present but cannot be distinguished from that from gastric or duodenal ulcer, because it often responds to antacids or food.

Liver, gallbladder and bile ducts (Fig. 6)

Acute hepatitis

In acute hepatitis, enlargement of the liver, with subsequent stretching of the capsule, can give rise to pain felt in the right hypochondrium and upper abdomen. The development of jaundice is indicative of hepatitis and the liver is tender on palpation. It should be remembered that hepatitis B infections may be preceded in one in four cases by a polyarthritis affecting the smaller joints.

Choledocholithiasis

This provokes spasmodic pain felt mainly in the right hypochondrium. The pain may radiate posteriorly towards the inferior angle of the right scapula (T7–T9).

Cholecystitis

Though traditionally described in females of 20–40 years of age, cholecystitis can occur at any age and in either sex. Localized peritoneal irritation may occur with acute abdominal pain in the right hypochondrium. Pain may radiate into the back and to the right shoulder. Sometimes nausea and vomiting are also present. On palpation, there is local tenderness over the gallbladder.

Liver abscess

Although liver abscesses are uncommon, they may be associated with right upper quadrant abdominal pain. General illness, varying from a very slight malaise to severe illness with septic shock, may be found.

Pancreas (Fig. 7)

Acute pancreatitis

In acute pancreatitis the patient is usually acutely ill with central upper abdominal pain, which may radiate to the back. The clinical features of an 'acute abdomen' predominate.

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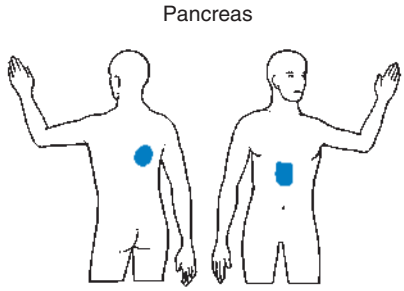

Fig 7 • Referred pain in lesions of the pancreas.

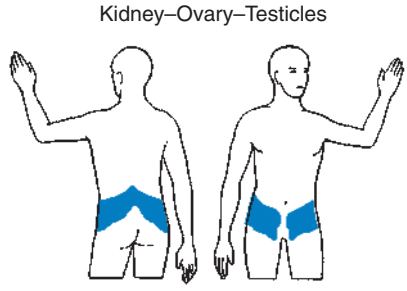

Fig 8 • Referred pain in ovarian or testicular lesions.

Subacute or chronic pancreatitis

In a high percentage of cases, pain is referred to the back at about the T8 level. The symptoms may be the outcome of obstruction of the pancreatic duct, interstitial inflammation or infiltration by a neoplasm. Patients who suffer from these disorders often get relief in a characteristic position: sitting with the trunk flexed, knees drawn up and the forearms folded around the knees to exert pressure upon it.

Spleen

Pain arising from disorders of the spleen, sometimes due to splenomegaly, is felt in the left hypochondrium and is sometimes referred to the side in the lower half of the thorax (T6–T10).

Appendix

Acute appendicitis is usually the result of an obstruction of the lumen of the appendix by a faecalith. The pain starts in the centre of the abdomen, around the umbilicus and later is sited in the right iliac fossa. Nausea and vomiting are often present. There is local tenderness and guarding on palpation of the right iliac fossa, from localized peritonitis.

Mechanical obstruction of hollow viscera

Abdominal colic is the predominant symptom. Depending on the level of obstruction, it is felt around the umbilicus (small intestine) or at the level of the obstruction (colon).

Distension of the splenic flexure of the colon by gas may give rise to pain in the left hypochondrium, in the precordial area and even in the left shoulder. The pain is often provoked by constipation and heavy meals. Patients usually suffer from gaseousness and aerophagy as well and are relieved by flatus or bowel movement.

Ureteral obstruction provokes pain felt in the patient's side and in the lateral aspect of the abdomen. It often radiates towards the genitals.

Intra-abdominal vascular disorders

Aneurysm of the abdominal aorta

This is often asymptomatic. On palpation an expansile, pulsating mass may be found. As an aneurysm expands it may give rise to pain in the abdomen or in the back. Acute rupture gives rise to very acute epigastric pain, radiating into the back. Shock supervenes and surgical intervention is urgently needed.

Mesenteric ischaemia

Mesenteric arterial occlusion sometimes comes on very suddenly, provoking catastrophic abdominal pain. In other cases, the pain is much milder and more progressive, setting in over 2 or 3 days. Vomiting and bloody diarrhoea usually occur. Careful history for other localizations of vascular abnormalities is always necessary.

Disorders of the genitals (Fig. 8)

A torsion and rupture of an ovarian cyst gives rise to symptoms almost identical to those in acute appendicitis. In subacute torsion, the patient complains of repeated episodes of sharp abdominal pain. Each attack lasts only for a short period of time.

Children and adolescents are the chief victims of testicular torsion. There is acute pain in the scrotum, sometimes radiating into the abdomen. Vomiting is often associated.

Parietal peritoneum

Peritonitis

Irritation of the parietal peritoneum can be the result of infection (as in acute appendicitis) or to chemical irritation by intestinal contents (as in a perforated ulcer) and inflammatory exudate (in pancreatitis). The pain is steady and aching and is increased by any augmentation of the intra-abdominal pressure. For this reason, the patient prefers to lie still.

Herpes zoster

Although herpes zoster is not a visceral disorder as such, it is mentioned here because it may result in unexplained pain for

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a few days before the typical rash appears. The pain is persistent and burning and varies in intensity from mild to severe. The older the patient, the more severe the pain. A rash, consisting of typical grouped vesicles with a unilateral segmental distribution, usually follows within 4 days.

Psychogenic problems

Psychogenic problems may give rise to symptoms and signs in the thoracoabdominal area as well as in the rest of the body. Specific features are absent and the vague history without direct answers to questions, together with several clinical inconsistencies, must put the examiner on guard.

Hyperventilation

Patients who suffer from hyperventilation are usually tense and anxious. They frequently complain of attacks of pins and needles in hands and feet, often associated with faintness, dizziness and precordial pain. The diagnosis is made by provoking the symptoms by asking the patient to hyperventilate.

Pain referred from musculoskeletal structures not belonging to the thoracic cage

Although some of these have already been discussed as part of the clinical examination, they are brought together here in a more schematic way to complete the account of referred pain.

Disc protrusions

Cervical disc lesion

Extrasegmental pain

A posterocentral cervical disc protrusion often gives rise to extrasegmental pain referred into the thorax, most frequently felt between the scapulae above T6. When pain is present in this area, a detailed examination of the cervical spine is immediately indicated. In a cervical disc lesion, the pain is seldom referred lower in the posterior thorax or anteriorly into the chest, but when it is, the thoracic examination remains negative except for flexion of the neck.

Segmental pain

A posterolateral cervical disc lesion gives rise to segmental referred pain, which is usually absent in the thorax. However, in a C4 root compression, pain is felt just superior to clavicle and scapular spine.

Thoracic disc lesion

Extrasegmental referred pain

A posterocentral disc protrusion in the thoracic spine is usually accompanied by posterior interscapular pain spreading extrasegmentally. Symptoms and/or signs of a disc protrusion are found on examination of the thoracic spine. Depending on the type, different pain patterns may result: acute thoracic lumbago, sternal lumbago, chronic thoracic pain and cord compression (see Ch. 27).

Band-shaped unilateral pain

This occurs in secondary posterolateral protrusions and is always preceded by posterior extrasegmental pain. A primary posterolateral protrusion provokes, from the onset, anterior pectoral pain in the absence of previous posterior pain. In the lower thorax, pain may be referred to the groin and even to the testicles.

Lumbar disc protrusion

Posterocentral lumbar disc lesions

These may occasionally give rise to abdominal pain, usually accompanied by posterior lumbar pain.

Root compression at L1–L3

This can provoke pain in the groin, because their corresponding dermatomes overlap in that area. In a secondary posterolateral protrusion the root pain is first preceded by extrasegmental lumbar pain.

Non-disc spinal lesions

Many other types of musculoskeletal disorders of the thoracic spine may refer pain into the chest or abdomen and are discussed in Chapter 28.

Disorders of the thoracic cage and abdomen

In this book, disorders are discussed in relation to the clinical examination that is most likely to reveal them. For this reason, many structures such as the sternoclavicular joint, costocoracoid fascia and the first rib, which belong anatomically to the thoracic cage, are discussed under the shoulder girdle, the neck and the shoulder joint.

Lesions of the thoracic cage commonly give rise to local pain and are often the result of a direct blow or a forceful activity during sports.

Disorders of the inert structures

Lesions of the sternum

Fracture and metastases

These give rise to well-localized sternal pain together with tenderness on palpation. A history of injury is usually obtained in the first, while a decline in general health may antedate the

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second. On clinical examination all movements hurt, as does deep inspiration. A radiograph usually confirms the diagnosis.

Manubriosternal arthritis

This can be the result of ankylosing spondylitis or rheumatoid arthritis.[19,20] Monoarticular steroid-sensitive arthritis is rare at this site.[21]

The patient complains of spontaneous pain at the angle of Louis. Local tenderness and swelling is found on palpation. In ankylosing spondylitis and rheumatoid arthritis other joints are also involved.

Laboratory tests

Tests for rheumatoid disorders should be done. In monoarticular steroid-sensitive arthritis, the manubriosternal joint is the only one affected and laboratory tests are negative.

Radiography

Arthritis may show erosion of the joint margin, presence of subchondral cysts, sclerosis of bone adjacent to the joint and widening or narrowing of the joint space. It may lead to total ankylosis, sometimes even to full destruction and subluxation of this joint.[22]

Treatment

In monoarticular steroid-sensitive arthritis, treatment consists of an intra-articular injection with 2 ml of triamcinolone, partly infiltrated into the superficial ligaments. In rheumatoid arthritis or ankylosing spondylitis, general treatment of the disorder is undertaken.

Disorders of the sternoclavicular joint

Lesions of the sternoclavicular joint are mainly characterized by local pain at the side of the manubrium or at the base of the neck. The pain is provoked by active and passive elevation of the scapula and arm. Other passive movements are only slightly painful. This combination, in the absence of any other features of a thoracic disc lesion, indicates clinical examination of the shoulder girdle (see online chapter C*linical examination of the shoulder girdle*).

Perichondritis of the costochondral joints (Tietze's syndrome)

This may have either a sudden or a gradual onset and gives rise to spontaneous unilateral pain and swelling of one of the costosternal synchondroses, most frequently that of the second rib.[23] It is found at all ages, even in children, and is equally prevalent in males and females.[24] It is a self-limiting process often associated with a respiratory infection, although this is not always the rule.

Natural history

The initial symptom is pain, which precedes the local swelling. Heat and erythema are absent. Usually the pain remains local, although radiation has been reported.[25] The intensity of the pain ranges from mild to severe, and is increased by deep inspiration and by coughing. The local fusiform or spindle-shaped mass is exquisitely tender and over-lies the affected joint.[26,27] When the xiphisternal joint is affected, the mass and pain often increase after eating as the result of a postprandial anterior displacement of the xiphoid.[28] Local tenderness is the first symptom to disappear, usually between 10 days and 2 months. Spontaneous pain remains longer, up to several months. The swelling tends to reach a maximum size and either remains stationary or regresses slowly but seldom undergoes total regression. Recurrence may occur.

Diagnosis

The diagnosis is based on the typical local swelling and tenderness, with normal radiograph. In that ultrasonographic examination of a costal cartilage is easy and quick to perform, it is the screening procedure of choice for Tietze's syndrome.[29,30]

Treatment

Perichondritis usually responds well to an infiltration with steroid into the painful junction but in costochondritis recurrence is common.[31,32]

Disorders of the rib

A fractured rib

This is usually the result of a direct blow to the chest. Nevertheless, fractures as a result of coughing or forceful muscular activity of the upper limb or trunk have been reported.[33,34] In fracture without an obvious cause, tumour must always be considered.

The patient complains of severe, well-localized pain, made worse by deep inspiration which may be arrested before reaching its peak. Every active, passive and resisted movement of the trunk which has any effect on the fractured rib is very painful. Which particular movements are positive depends on which rib is fractured and at which part. If the fracture lies in the neighbourhood of the origin of the pectoralis major (second to sixth ribs), resisted adduction and medial rotation of the arm are also painful. Slight movement of the fractured ends provokes sharp well-localized pain. Because a fractured rib is frequently accompanied by an adjacent intercostal muscle sprain, careful palpation of the muscle must follow. The pain from the fracture is felt on the rib itself, but that caused by a sprain is found in between the ribs. A radiograph confirms the diagnosis.

Differential diagnosis must be made from a primary posterolateral disc protrusion in which from the onset the pain is located in the anterior thorax. In disc protrusion, local trauma is not mentioned by the patient, pain is not felt on resisted movements and moving the rib ends on either side of the site of pain is negative. Palpation is not fully reliable because exquisite local tenderness may also be found in disc lesions.

Long floating rib

The tip of a long rib may strike the iliac crest on ipsilateral side flexion and provokes a sharp momentary pain. Severe recurrent

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symptoms of this kind may indicate removal of the tip of the rib.

The rib-tip syndrome

The slipping rib syndrome was first described by Edgar Cyriax in 1919.[33] After an injury to the anterior chest, the anterior cartilaginous tips of the eighth, ninth or tenth rib can remain detached from the bone and may subluxate inferoposteriorly.[34] Contact with the intercostal nerve can give rise to pain.

The patient complains of a sharp sudden pain felt at the anterior or inferior margin of the rib on local pressure, during vigorous activity or on deep breathing. Pain can spread posteriorly to the high lumbar level and may remain for some days. Often a popping sensation is felt.[35]

Diagnosis is based on the typical lancinating pain and the local tenderness during the 'hooking manœuvre'. The hooking manœuvre is a relatively simple clinical test, whereby the clinician places his or her fingers under the lower costal margin and pulls the hand in an anterior direction. Pain or clicking indicates a positive test.[36,37] Sometimes active abduction of the arm or ipsilateral side-flexion of the trunk provokes the pain.

Treatment

This is an infiltration of a mixture of steroid and local anaesthetic around the nerve at the tip of the rib, which brings immediate and sometimes permanent relief.[38,39] If the pain recurs, the cartilaginous tip of the rib should be surgically removed.[34]

Technique: injection

The patient lies supine, with the arms elevated. The painful tip is located by palpation. The caudal edge is identified and the lesion marked. A 2.5 cm needle is fitted to a 2 ml syringe filled with 1.5 ml of triamcinolone and 0.5 ml of lidocaine (lignocaine) 2%. The needle is inserted vertically at the site of the lesion, aiming at the lower edge at the anterior aspect of the rib, and pushed in until it touches the bone. The needle is then slightly withdrawn and moved more caudally until it passes just beyond the lower edge. The infiltration is done at this point.

Post-thoracotomy pain

A significant proportion of patients who undergo thoracotomy suffer from chronic pain. It is generally not severe but a small proportion of patients may experience persistent moderately disabling pain. The reported incidence of persistent post-thoracotomy pain lasting for more than 6 months is between 5 and 40%.[40–42] As the pain is cutaneous, it is most probably the consequence of damage to an intercostal nerve.

Patients can be treated by a nerve block around the intercostal nerve. This is tried first, with a mixture of local anaesthetic and steroid. Refractory cases require chemical rhizotomy with a 1% solution of phenol. This is usually followed by sensory loss, which is better tolerated than the pain.[43]

Tumours of the rib

These may be benign, malignant, primary and secondary.[44] Multiple myeloma involves the rib directly. Metastases usually derive from primary tumours of lung, kidney, thyroid and breast.

The initial symptom is well-circumscribed pain, spreading and increasing in severity later as the tumour grows or when it invades other neighbouring structures such as the intercostal nerves. Pathological fractures may result.

Herpes zoster

This disorder usually affects only one nerve and initially pain is present in its territory. After some days the skin turns red locally, shortly followed by the appearance of grouped vesicles. The diagnosis thus becomes clear.

Disorders of the contractile structures

Muscles of the thoracic cage

Sprained intercostal muscle

This is usually the result of a direct injury to the chest. It may exist alone or in association with a fracture of a rib.

The patient complains of a well-localized pain, which does not radiate. Deep inspiration is painful, as are all trunk movements that stretch the affected intercostal muscle. More than one muscle can be involved, so the whole of the painful area must be carefully palpated.

Differential diagnosis must be made from a fractured rib. In a sprain of the intercostal muscle, palpation is positive between the ribs and not on the rib itself. If the rib is also found to be tender, a fracture is most likely and a radiograph must be obtained.

Treatment

The lesion responds very well to a few sessions of deep transverse friction.

Technique: deep friction (Fig. 9)

The patient adopts a half-lying position and the therapist sits at the contra-lateral side. The tender point(s) in between the ribs are palpated and the middle finger, reinforced by the index finger, is placed there. The fingers are now moved to and fro in line with the ribs, movement obtained by a combination of flexion–extension at the elbow and shoulder. Every lesion should be treated each time, for about 15–20 minutes. Because this type of lesion responds very well to deep transverse friction, cure is obtained in about three to five sessions.

Lesions of the pectoralis major

The localization of the pain draws attention to the thorax, which is why this disorder is included here. However, it is the examination of the shoulder which establishes the diagnosis (see Ch. 12).

In a lesion of the pectoralis major, resisted adduction and internal rotation of the arm are painful. The patient is asked to press both hands against each other with the arms stretched

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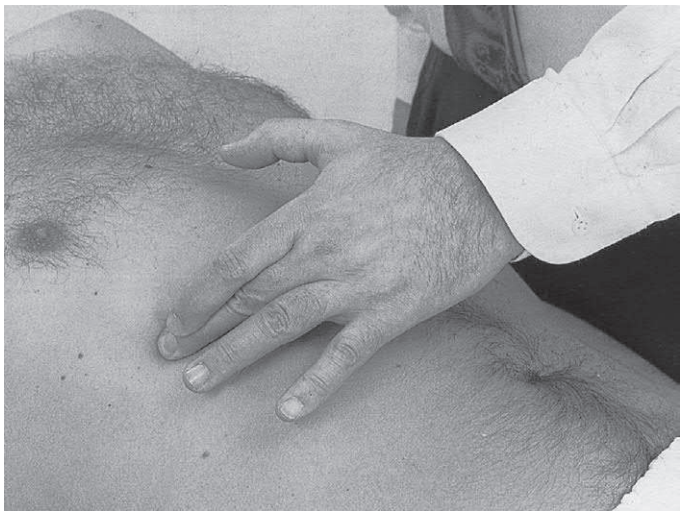

Fig 9 • Deep friction to an intercostal muscle.

Fig 10 • Deep friction to the peripheral costal insertion of the diaphragm.

out horizontally in front of the body, which is even more painful.

The lesion usually lies in the muscle belly at the infraclavicular portion or at the latero-inferior part of the muscle, almost in the axilla.

This disorder must be differentiated from a fractured rib, which may give rise to pain on the same shoulder tests. Fracture is associated with pain on deep inspiration and on coughing, as well as with some movements of the trunk. Palpation of the tender spot, with and without contraction of the pectoralis muscle, can be compared. More pain on contraction suggests a problem in the muscle, more pain with the muscles relaxed suggests that the cause is in a rib or the intercostal muscles.

Treatment is deep friction or an infiltration with procaine (see Ch. 15).

Lesions of the latissimus dorsi

This may provoke pain in the posterior thorax, sometimes even at the posterior aspect of the axilla. Pain is elicited on full passive elevation of the arm, resisted adduction and medial rotation. The basic thoracic examination is usually negative.

Treatment is either by deep transverse friction or an infiltration with procaine (see Ch. 15).

Sprain of the serratus posterior inferior muscle

This may occur in athletes (Cyriax:[20] p. 201). Local pain is felt dorsally in the lower thorax or upper lumbar area and is increased by ipsilateral resisted side flexion and rotation of the trunk. Treatment is deep transverse friction.

Lesions of the diaphragm

A severe blow on the chest can be transmitted to the diaphragm, causing a sprain.

Pain is felt on respiration, and is not provoked or altered by movements of the trunk. In this condition, all movements of the trunk should be tested while the breath is fully held, because otherwise the patient may give false-positive responses related to respiration and not to the tests as such. When the central fibrous portion of the diaphragm is affected, pain is referred to the tip of the shoulder, whereas a lesion at the insertion at the ribs provokes more local pain at the lower margin of the thoracic cage.

Treatment

The peripheral costal insertion can be treated by deep transverse friction.

Technique: friction (Fig. 10)
The patient adopts a half-lying position and fully relaxes the abdominal muscles. The therapist sits at the patient's painful side. The lesion is palpated at the inner side of the lower ribs. The fingertips of three or four fingers are hooked under them. Friction is obtained by a movement parallel to the rib, meanwhile applying anterior pressure against its posterior aspect.

Lesions of the abdominal muscles

Pain in the abdomen is usually the result of a visceral disorder. Exceptionally it arises from a musculoskeletal lesion, most frequently a thoracic or lumbar disc problem giving rise to segmental or extrasegmental referred pain.

Local muscular problems of the abdominal wall may also occur. They are usually encountered in athletes, very often soccer players. Pain is usually under the influence of movement and local pressure, sometimes of lifting and coughing. When this type of lesion is suspected, resisted flexion and rotations of the trunk must be checked. Active extension or active contralateral side flexion of the trunk can also be painful as the muscle is stretched. If, after a complete examination, the differential diagnosis between a visceral and a muscular problem

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remains unclear, palpation with the abdominal muscles relaxed is compared with palpation with the muscles in contraction. Greater pain on palpation with the muscles relaxed points towards a visceral problem, the opposite to a lesion of the abdominal muscles (Cyriax:[20] p. 219; Ramboer and Verhamme[35]).

Lesions of the rectus abdominis muscle

These are usually located in the most distal 4 cm of the muscle belly, just above the pubic symphysis. The patient complains of local suprapubic pain during activity.

Pain is found on resisted anteflexion of the trunk and on active trunk extension. Bringing the patient's ipsilateral arm upwards and backwards during active extension of the trunk increases the pain because of further stretching of the rectus muscle.

Differential diagnosis

The differential diagnosis includes lesions of the rectus femoris or the psoas muscles and osteitis pubis (see the online chapter *Groin pain*). In the former two, the pain lies more laterally and is also provoked by resisted flexion of the hip which provides the diagnostic key. In osteitis pubis the pain is at the pubic symphysis and is also provoked by resisted adduction of the hips. The fact that the pain is reproduced during the examination of both lower extremities should help in the differentiation.[45,46]

Treatment

An infiltration of 10–20 ml 0.5% procaine is normally curative.[35] Deep friction is also effective.

Technique: injection (Fig. 11)
A 4 cm needle is fitted to a 10–20 ml syringe filled with 0.5% procaine. The patient is put in the half-lying position, keeping the abdominal muscles well relaxed. The tender part of the muscle is taken between thumb and index finger and the needle thrust in obliquely in a caudal direction. Special care must be taken not to push the needle in too deeply. With several withdrawals and reinsertions the procaine is infiltrated.

The patient is reassessed 1 week later. If full cure has not been achieved, a second infiltration is given. Three infiltrations may be necessary.

Technique: deep friction (Fig. 12)
The patient is put in the half-lying position with the abdomen well relaxed. The therapist sits at the painless side and places two or three fingers on the tender spot. Counterpressure is taken with the thumb. Friction is a to-and-fro movement by flexion–extension of the elbow.

Fig 11 • Injection of a lesion of the rectus abdominus muscle.

Fig 12 • Deep friction to a lesion of the rectus abdominus muscle.

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Fig 13 • Deep friction to the muscle belly of the external oblique.

The oblique muscles

A sprain of one of the oblique muscles gives rise to pain in the anterolateral part of the abdomen. Pain is provoked or increased by resisted rotation of the trunk: pain on resisted rotation away from the painful side signifies a lesion of the external oblique muscle; pain on resisted rotation towards the painful side originates from the internal oblique muscle.

Stretching the affected fibres also hurts. Consequently active extension of the trunk or active side flexion towards the painless side is also likely to be painful.

The lesion can lie either at the costal origin or in the muscle belly. In the former, palpation should be done at the infero-posterior margin of the ribs, with the finger pressing against the internal aspect of the rib for the internal oblique muscle or against the external aspect for the external oblique muscle.

Treatment

A lesion at the origin of the external oblique muscle or in the muscle belly is treated by deep transverse friction. The origin at the ribs of the internal oblique muscle can be treated only by deep friction, using the same technique as for the costal origin of the diaphragm (see above).

Technique: deep friction to the muscle belly (Fig. 13)
The patient is put in a half-lying position with the therapist sitting at the side opposite to the lesion. The tips of two or three fingers are placed on the painful area. Friction is given by a flexion–extension movement at the elbow, transverse to the direction of the fibres – i.e. in the direction contralateral shoulder–ipsilateral hip.

Each session takes about 15 minutes. Cure is normally obtained in about 10–15 sessions.

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Applied anatomy of the temporomandibular joint

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The temporomandibular joint (TMJ) is sited at the base of the skull and formed by parts of the mandible and the temporal bone, separated by an intra-articular meniscus. It is a synovial joint capable of both hinge (rotation) and sliding (translatory) movements. Like other synovial joints, it may be affected by internal derangement, inflammatory arthritis, arthrosis, and muscular disorders.

Bones

The mandible has a horizontal part (the body) and a vertical part (the ramus). They meet at the mandibular angle. The cranial end of the ramus has two processes: anteriorly the coronoid process and posteriorly the condylar process, which has a head on top and a distinct neck below. The squamous portion of the temporal bone contains the articular surface with a concave articular fossa posteriorly and a convex articular tubercle anteriorly. The articular surface is about three times as large as that of the mandibular head and is covered with fibrocartilage which continues anteriorly into the articular tubercle, the posterior aspect of which is the most important part of the joint. From the temporal bone develops a zygomatic process which, together with the temporal process of the zygomatic bone, forms the zygomatic arch (Fig. 1).

The midline fusion of the left and right mandibular bodies provides a connection between the two temporomandibular joints, so that movement in one joint always influences the opposite one.

Joint capsule and ligaments

The joint capsule is wide and loose on the upper aspect around the mandibular fossa. Distally, it diminishes in a funnel shaped manner to become attached to the mandibular neck (Fig. 2). Its laxity prevents rupture even after dislocation.

Laterally and medially, a local reinforcement of the joint capsule is found. The lateral collateral ligament courses from the zygomatic arch obliquely downwards and backwards towards the posterior rim of the mandibular neck, lateral to the outer aspect of the capsule. At its posterior aspect, it is in close relation to the joint capsule and prevents the joint from opening widely. Medially, the joint capsule is locally reinforced by the medial collateral ligament. There are also two extracapsular ligaments: the sphenomandibular (between the spine of the sphenoid bone and the lingula of the mandible) and the stylomandibular (from the styloid process of the temporal bone to the mandibular angle).

Intra-articular meniscus

The intra-articular meniscus, or disc (see Fig. 2), is attached along its entire circumference to the capsule. It compensates for the discongruent articular bony surfaces and contributes to the stability of the joint. The meniscus divides the joint into an upper and a lower compartment, each fully separated from the other (see Hodges, 1990, in bibliography). The upper compartment is a sliding joint, the lower a hinge. Because the meniscus is malleable, it fills the whole joint space in any position of the condyle. Its anterior aspect is connected to the

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lateral pterygoid muscle. Posteriorly, the condyle is attached to the meniscus by loose connective tissue – the bilaminar zone – which is also fused to the posterior capsule. The superior surface of the meniscus relates to the middle third of the articular tubercle, the inferior surface to the anterior portion of the condyle. The meniscus always follows the movement of the condyle: when the mandibular head glides anteriorly on opening the mouth, the meniscus moves anteriorly; when it moves back on closing, so does the meniscus.

Nociceptive innervation

The articular cartilage, the synovial tissues and the central portion of the disc do not contain any pain receptors and therefore cannot give rise to pain. All other capsular and intra-articular structures do possess nociceptors; the meniscus has them mainly at the posterior edge, less on its anterior portion. They are chiefly activated by high mechanical stresses and by acute or chronic inflammation.

Muscles and tendons

The most important contractile structures are the masseter, temporal and pterygoid muscles (Fig. 3).

The masseter muscle originates at the lower edge (superficial part) and deep aspect of the zygomatic arch and at the temporal fascia (deep part). Both parts course superficially

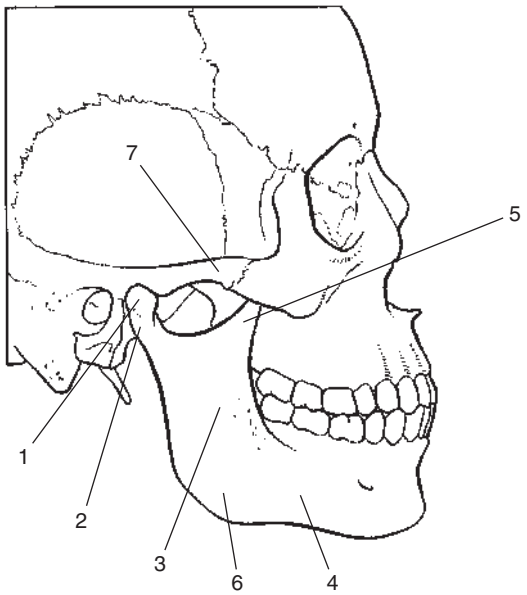

Fig 1 • Osseous structures of the temporomandibular joint: 1, mandibular head (condylar process); 2, neck of mandible; 3, ramus; 4, body; 5, coronoid process; 6, mandibular angle; 7, zygomatic arch.

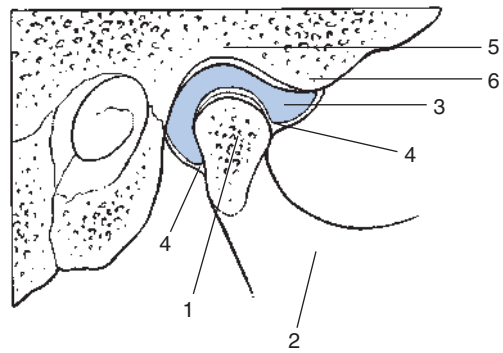

Fig 2 • The joint capsule and the intra-articular meniscus: 1, condylar process; 2, mandible; 3 meniscus; 4, joint capsule; 5, articular fossa; 6, articular tubercle.

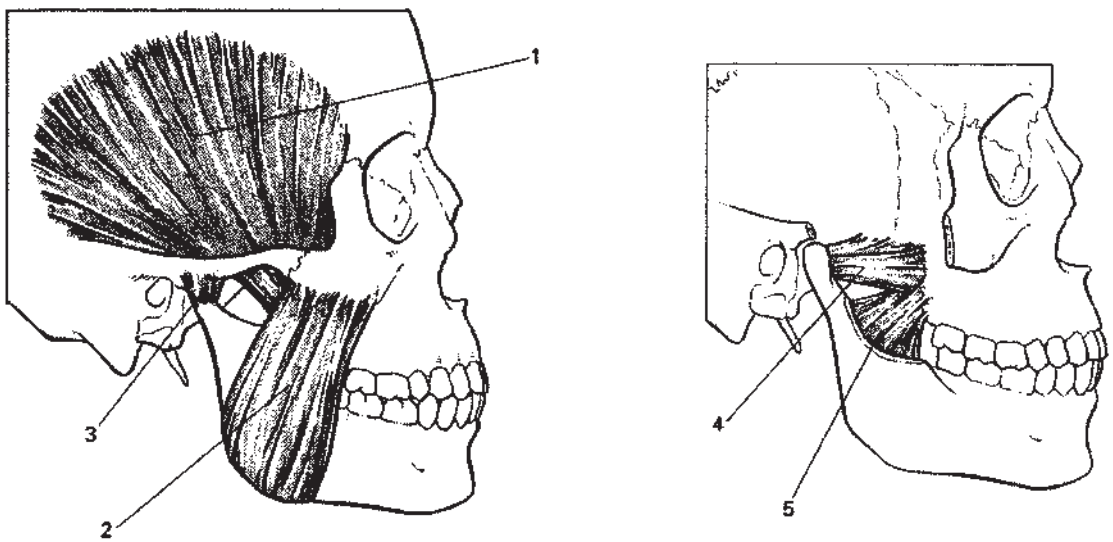

Fig 3 • Muscles of the temporomandibular joint: 1, temporalis; 2, masseter; 3, joint capsule; 4, lateral pterygoid; 5, medial pterygoid.

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from the mandibular ramus towards the angle, where they have broad insertions into the tuberosity. Contraction of the masseter closes the mouth.

The origin of the temporalis muscle is in the temporal fossa and at the temporal fascia. It stays deep to the zygomatic arch and inserts into the coronoid process of the mandible. It also closes the mouth.

The medial pterygoid originates in the pterygoid fossa at the base of skull and inserts into the deep aspect of the mandibular angle at the pterygoid tuberosity. It acts synergistically with the masseter and temporalis muscles.

The lateral pterygoid muscle lies on the deep aspect of the mandibular neck. It has two heads: one originating at the major wing of the sphenoid bone, the other at the lateral aspect of the pterygoid process. The two heads course laterally and posteriorly, join each other and insert into the pterygoid fovea of the mandibular condyle, the joint capsule and the meniscus. Because the muscle courses in an anteromedial to posterolateral direction, bilateral simultaneous action of both lateral pterygoids pulls the mandibular condyles anteriorly; which opens the mouth. Unilateral contraction provokes a contralateral deviation of the chin. The medial and lateral pterygoid muscles are too deep to be palpated.

The suprahyoid muscles – digastric and stylohyoid – and the muscles of the floor of the mouth – mylohyoid and geniohyoid – only take part in opening of the mouth when the effect of gravity is excluded, for example lying down. They are of little clinical importance.

Biomechanical aspects

The mandible allows three types of movement.

Forward movement of the mandible

The lateral pterygoid muscle pulls the mandible forwards (anterior translatory movement). During this process the mandible moves slightly downwards because the condyle is pressed down on the articular tubercle. The mandible is pulled backwards on closing by the posterior fibres of the temporalis muscle.

Opening and closing the mouth

Because the TMJ contains a meniscus, two independent movements are possible: a rotation or hinge movement and a translatory glide. From the onset of opening to the midpoint, the mandibular head *rotates* on the undersurface of the intraarticular meniscus because of the effect of gravity and the action of the muscles of the floor of the mouth. At the same time, the collateral ligaments are taut. On further movement, condyle and meniscus glide together anteriorly and slightly caudally on the articular tubercle because of contraction of the lateral pterygoid. No further rotation occurs once the anterior translatory movement has begun. If for some reason the anterior movement of the mandibular head on opening is not possible on one side, the chin deviates to the ipsilateral side.

The mouth is closed by contraction of the temporalis, masseter and medial pterygoid muscles.

Grinding movements

The mandible rotates around a vertical axis through the contralateral mandibular head by a unilateral contraction of the lateral pterygoid muscle. This is followed by a contraction of the posterior fibres of the temporal muscle, which repositions the head of the condyle. When this occurs on alternate sides a typical grinding movement results. If at the same time the other masticatory muscles contract, food can be crushed.

Nerves and blood vessels

Between the tragus and the mandibular condyle lie the superficial temporal artery and veins. The superficial temporal artery is the continuation of the external carotid artery and the veins course towards the internal jugular vein. Deep to the neck of the mandible passes the maxillary artery, a branch of the external carotid.

The auriculotemporal nerve, a branch of the mandibular nerve, which again originates from the trigeminal nerve (V), is just posterior to the blood vessels, which it follows cranially. The upper branch of the facial nerve (VII) passes superficially to the blood vessels at the level of the mandibular neck and runs horizontally forwards before breaking up into several terminal branches (Fig. 4).

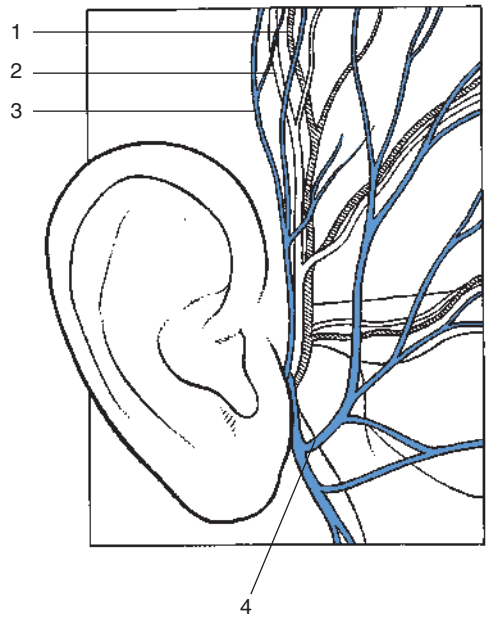

Fig 4 • Nerves and blood vessels around the temporomandibular joint: 1, superficial temporal vein; 2, superficial temporal artery; 3, auriculotemporal nerve; 4, upper branch of facial nerve.

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Clinical examination of the temporomandibular joint

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The most characteristic symptoms of disorders of the temporomandibular joint (TMJ) are orofacial pain, noises in the joint, limitation of movement – mouth opening – or a combination of these. Limitation may present suddenly as locking or may be slowly progressive.

Pain

Pain in the TMJ area usually has a local cause and is seldom referred to any distance.

The patient should also be asked about the influence of chewing, yawning, swallowing or talking. If pain is present on one of these, a disorder of the TMJ is most likely. Some disorders of the cervical spine (see Section 2) and the parotid gland may exceptionally also provoke pain on swallowing.

A clear description of the type of pain should always be sought. A sharp severe pain tends to suggest an arthrogenic problem, a diffuse ache of less intensity points more to a muscular disorder.

A painful click may be the consequence of subluxation of the intra-articular meniscus. Pain coming on spontaneously and progressively increasing over some weeks is often the result of arthritis. Continuous dull pain felt in the area of the masticatory muscles and usually worse at the end of the day may indicate myalgia.

Pain referred from the temporomandibular joint

Pain of the TMJ structures may arise from the masticatory muscles or from the joint itself. The main inert structures that can give rise to pain are the intracapsular tissues located posterior to the condyle: the posterior part of the meniscus, the meniscus attachments to the capsule, the capsule and the retromeniscal fat pad.[1] Pain is often accompanied by headache, earache or pain in the postauricular area. Pain arising from the TMJ sometimes refers to the maxilla.

Pain referred to the temporomandibular joint area

Occasionally pain is referred from the neck. When there is doubt, a preliminary examination of the neck must be performed.

Other structures may give rise to painful conditions in the TMJ area and can be divided into neurological and non-neurological disorders.

Neurological disorders

Atypical facial neuralgia

This can be uni- or bilateral and is of unknown origin. It usually causes a burning sensation, pins and needles, and continuous pain, in cycles of severity: it may occur after dental procedures.

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Trigeminal nerve neuritis

This may be encountered in patients of 45–60 years of age. It affects females more often than males and the right side more frequently than the left. The patients complain of unilateral shooting pain, from the ear towards the temporal area and the maxilla, sometimes even in the forehead and towards the pharynx. The cause of the pain may be so obscure that unnecessary dental extraction takes place. Pain is seldom accompanied by diminished sensitivity but characteristic trigger points are often found. Stimulation of these, even sometimes by light touch, results in pain felt elsewhere, which is followed by a refractory period of up to 30 seconds during which stimulation does not lead to new pain. The pain attacks seldom last longer than a few seconds. They may recur at irregular intervals, sometimes on a daily, weekly or even a monthly basis. They are isolated or come on in clusters.[2]

Herpes zoster oticus infection

This can give rise to dysaesthesia preceding the characteristic vesicles. No true trigger points are present. About 15% of all peripheral facial palsies is caused by this virus.[3]

Idiopathic peripheral facial palsy (Bell's palsy)

This is a disorder of the facial nerve, probably the result of a cranial neuritis.[4,5]

It mainly affects patients between 20 and 50 years of age. It is seldom painful although at the onset some pain around the ear may be felt. It gives rise to a palsy of the facial muscles, characterized by lowering of the ipsilateral side of the mouth. It may also cause diminished pain sensibility, changes in taste, diminished lachrymation and increased salivation.

Peripheral neuropathy

This is usually the result of diabetes, long-standing temporal arteritis or Raynaud's syndrome. It usually leads to a burning sensation and loss of sensibility on lips, cornea or conjunctivae.

Non-neurological disorders

Otitis media, otitis externa and parotitis

These all give rise to pain in the TMJ area. Pain usually remains local and increases on pressure on the tragus (otitis) or parotid gland (parotitis epidemica). These conditions mainly affect children and are usually accompanied by fever.

Paranasal sinusitis

This results in a constant, knocking pain usually felt around the orbits, sometimes radiating towards the cheek and into the teeth.

Infection of the teeth and dental abscess

Tooth infections are followed by pain in the cheek on percussion which is provoked or increased by eating sugary food. An abscess gives rise to local swelling of the gingiva.

Cluster headache (Horton's neuralgia)

This predominantly affects males, is unilaterally localized, and is associated with increased lachrymation, rhinitis and ipsilateral facial redness. Attacks of severe headache in or around the eyes, usually unilaterally, come on within 5–10 minutes and last from about 45 minutes to a few hours. Attacks occur in clusters.[6]

Temporal arteritis

This is one of the manifestations of a giant-cell arteritis, an autoimmune process.[7] It is usually seen unilaterally in males over 50 years of age and is frequently associated with polymyalgia rheumatica. It is characterized by a knocking pain around the temporal vessels. The skin overlying the artery is red, swollen and warm. The erythrocyte sedimentation rate is raised.

Leaking cerebral aneurysm

It has an explosive onset of headache, nausea and vomiting, together with photophobia and stiffness of the neck. Aneurysm at the level of the posterior communicating artery may be followed by pain in the first division of the trigeminal nerve. It is the commonest cause of so-called ophthalmoplegic migraine.[8]

History

Questions are asked about the onset of pain, its nature, localization, intensity and duration. The examiner should discover which factors increase or relieve pain.

As well as taking a history of pain, a number of other aspects should be discussed with the patient:

- *Does the joint click?* In an anterior subluxating meniscus, the normal relation between meniscus and condyle is disturbed, giving rise to a click on opening the mouth.
- *Is movement limited, either in range or by locking?* If there is a diminished range of opening of the mouth, did the limitation come on suddenly or was it more progressive? If 'sudden' locking is mentioned, can the patient still open or close the mouth? Inability to open suggests meniscus displacement, which is usually unilateral, and in which at least 1 cm of mouth opening is always retained. If closing is impossible, a luxation of the mandibular condyle is most likely. Excessive limitation coming on rapidly may be the result of hysteria or of tetanus; mouth opening is impossible in these circumstances. A limitation of slow development is usually the outcome of arthrosis of the TMJ.
- *Is there crepitus?* Crepitus is the result of movement across an irregular surface because of advanced changes in the joint. It may be present in osteoarthrosis.
- *Does the patient suffer from clenching or grinding?* This occurs mainly at night in stressed people. The patient may not be aware of it, relatives may have to be asked.
- *Is there tinnitus, vertigo or a hearing problem?* Vertigo may result from differences in vestibular impulses as a result of TMJ problems. Other symptoms, such as mild deafness, a sensation of fullness in the ear and tinnitus, may also be present.

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- *Have there been changes in sensibility?* These can indicate peripheral neuropathy. It frequently affects the lips, cornea and conjunctivae. In atypical facial neuralgia, severe diminished facial sensibility is often found. Trigeminal neuritis is seldom accompanied by disturbed sensibility.

Inspection

On inspection, attention must be paid to local swelling, deformation, deviation of the chin and teeth wear.

Swelling may be the result of a bacterial or an inflammatory arthritis (frequently rheumatoid, seldom due to psoriasis or gout), or in children[10] may be caused by an inflammation of the parotid gland.

In Bell's palsy, there is lowering of the ipsilateral side of the mouth and a smoothing out of wrinkles.

Severe inflammatory disorders of the TMJ area during childhood may result in asymmetrical development of the lower face because of disturbance of the growth centre in the mandible. Advanced arthrosis may lead to asymmetry of face and head and to narrowing of the external auditory canal. Synovitis usually causes an ipsilateral deviation when the mouth is opened and a contralateral deviation when closed.[9]

Abnormal wear and tear of the teeth may be a sign of bruxism or grinding. Malocclusion and missing teeth may result in a TMJ problem. A bilateral relationship between the teeth and TMJs exists. Changes in the dental relationship, as in malocclusion and missing teeth, may lead to adaptation in the TMJ. Problems with the joint can cause changes in dental occlusion.

Functional examination

Active movements

The influence of all five active movements on pain, range of movement, deviation, abnormal sounds and crepitus are noted.

Active opening of the mouth (Fig. 1)

Because it is difficult to measure the range of motion of the TMJ in degrees, the interincisal distance at maximum opening is used. It is about 36–38 mm in adults but may vary between 30 and 67 mm, depending on sex and age.[11,12] A practical and quick way of checking range of motion is to ask the patient to insert the knuckles in between the front teeth (Fig. 2).

Active closing of the mouth

The patient is asked to close the mouth (Fig. 3).

Active deviation of the mandible to the left and right (Fig. 4)

When the mandible deviates to the side it rotates around a vertical axis through the ipsilateral mandibular ramus. The contralateral mandibular head moves anteriorly at the same time.

Fig 1 • Active opening of the mouth.

Fig 2 • Checking the range of motion.

Active forward protrusion of the chin (Fig. 5)

This is performed by the lateral and medial pterygoid, masseter, geniohyoid and digastric muscle. When it is disturbed, this is usually the consequence of an inert problem.

Resisted movements

Resisted opening of the mouth (Fig. 6)

The examiner places one hand underneath the patient's chin, the other on the vertex. With the mouth open about 1 cm, the patient is now asked to open further while the examiner

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Fig 3 • Active closing of the mouth.

Fig 5 • Active forward protrusion of the chin.

Fig 4 • Active deviation of the mandible.

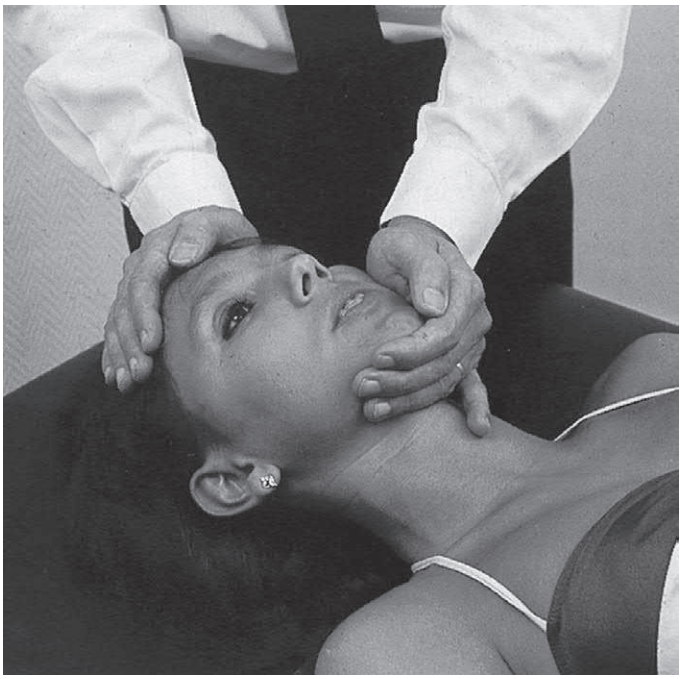

Fig 6 • Resisted opening of the mouth.

provides strong resistance, so preventing any movement. The strength of the lateral pterygoid is tested by this manœuvre.

Resisted closing of the mouth (Fig. 7)

A rubber pad about 1 cm thick is put between the teeth. The patient is asked to bite as hard as possible. This is a test for all the muscles that close the mouth: masseter, temporal and medial pterygoid.

Resisted deviation of the mandible to the left and right (Fig. 8)

The examiner puts one hand on the left side of the patient's chin and holds the head stable by placing the other hand against

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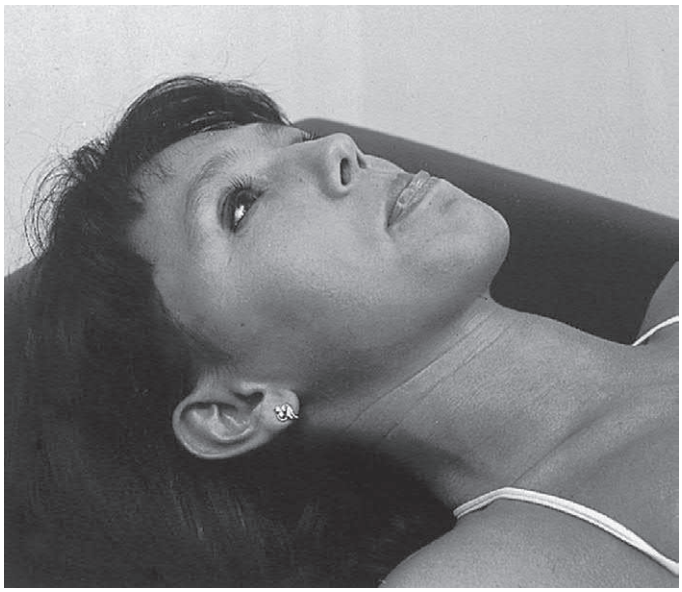
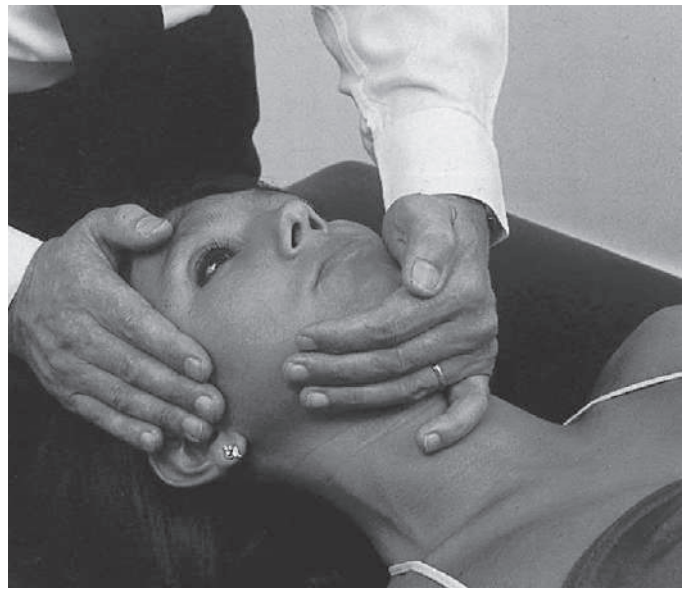
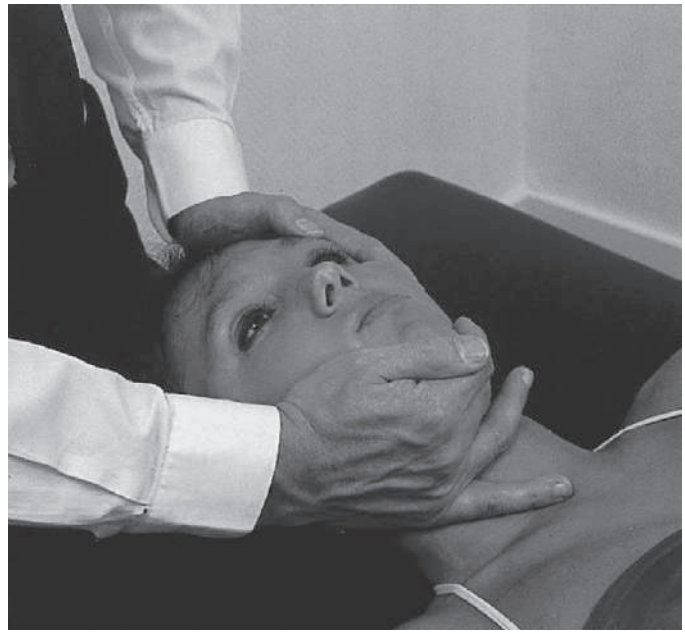


Fig 7 • Resisted closing of the mouth.



(a)



(b)

Fig 8 • Resisted deviation of the mandible (a) to the left; (b) to the right.

the right temporal area. The patient is now asked to deviate the chin to the left against the resistance offered by the examiner's hand. The test is repeated to the opposite side. This movement tests the contralateral lateral pterygoid.

Palpation

The joint is palpated during active opening and closing and during active deviation to the left and right.

On opening, the TMJ is palpated with the finger below the zygomatic bone just anterior to the condyle or, as for closing, with the tip of the finger placed either just anterior to the tragus (Fig. 9a) behind the condyle or in the external auditory meatus (Fig. 9b), exerting some anterior directed pressure against the posterior aspect of the joint. The examiner normally feels a depression on opening. If a severe effusion is present, a bulge may be palpated. Attention must be paid to abnormal sounds and crepitus and to the anteroposterior gliding movement of the condyle.

The coronoid process can be palpated on opening and closing the mouth when the fingers are placed just below the zygomatic arch. The process is felt through the masseter muscle.

Further palpation is done to elicit local tenderness of some masticatory muscles, the joint capsule and bone around the tooth sockets. The masseter muscle can be palpated on opening the mouth and on clenching the teeth. Palpation of the temporal muscle is performed on clenching the teeth.

Technical investigations

The erythrocyte sedimentation rate is frequently elevated in systemic diseases and infections.

Plain radiography does not provide much information except for evidence of arthrosis.[13] A CT scan can determine more accurately the position and condition of the meniscus and the joint.[14,15]

In recent years, magnetic resonance imaging has been increasingly used to investigate temporomandibular disorders, for example internal derangement.[16–18]

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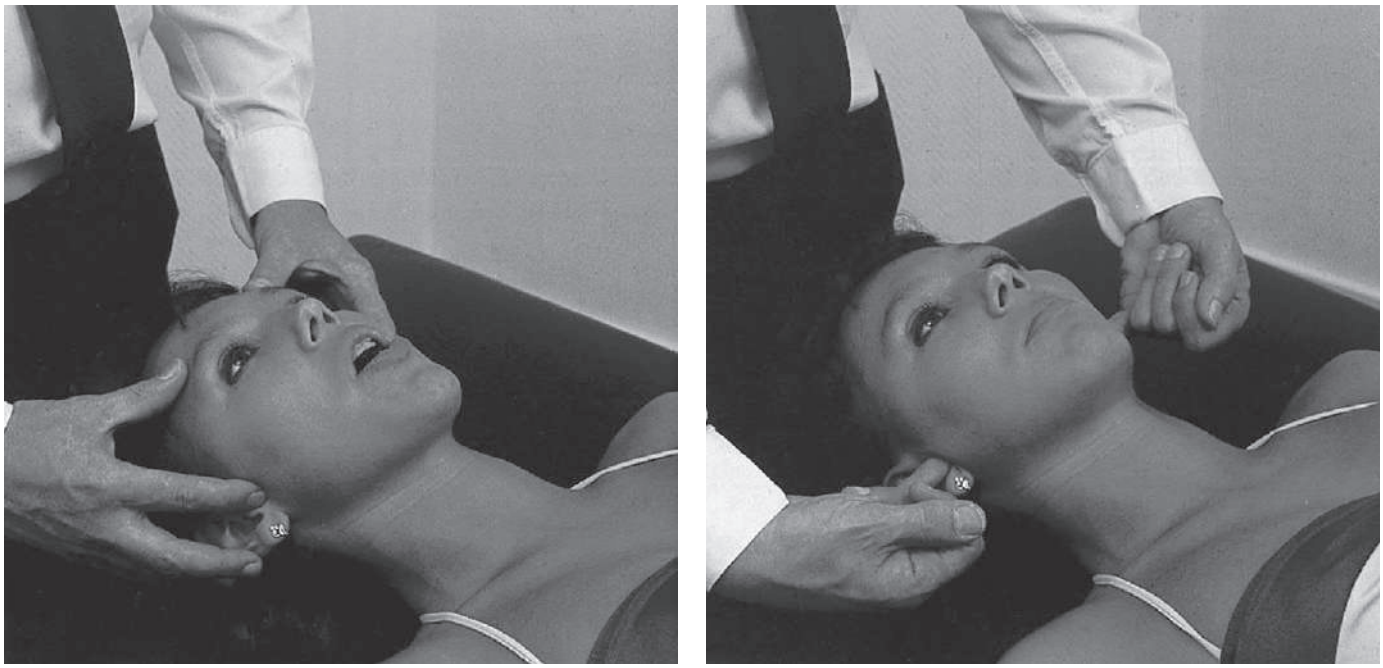

(a) (b)

Fig 9 • Palpation of the temporomandibular joint: (a) anterior to the tragus; (b) in the external auditory meatus.

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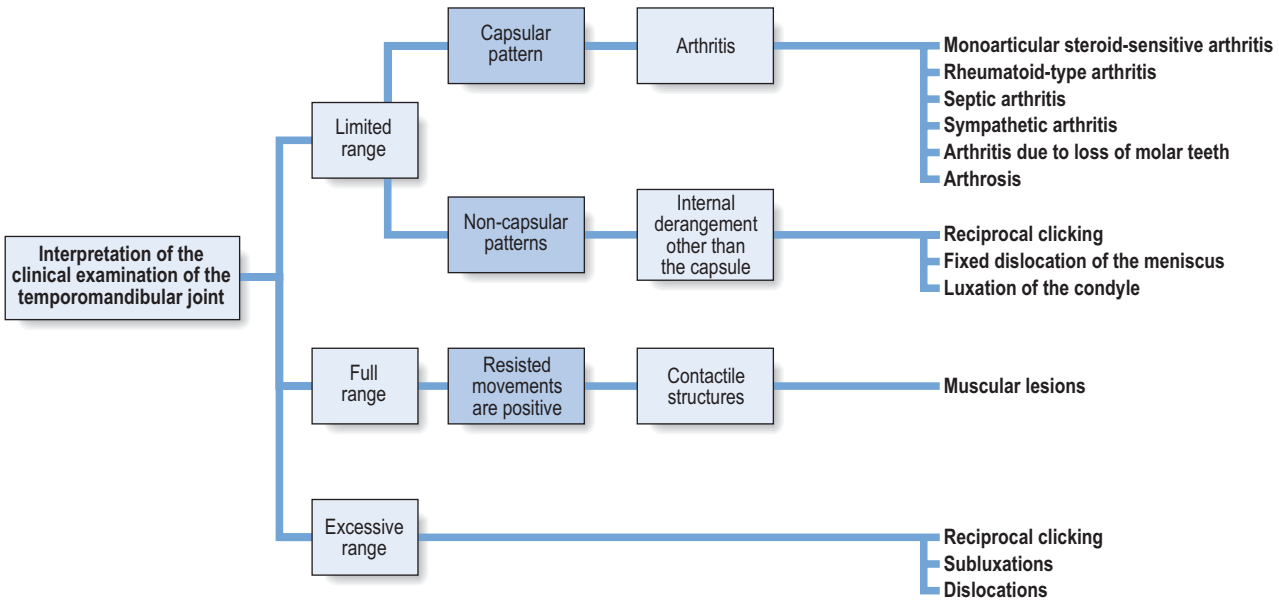
Interpretation of the clinical examination of the temporomandibular joint



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Disorders of the temporomandibular joint

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A great number of people (60–70%) have some problem with the temporomandibular joint (TMJ) but most are asymptomatic. About 25% of this group has symptoms that can be attributed to the TMJ.[1,2] Of this group only 5% will get treatment and these patients are mostly women – they outnumber male patients by at least 4 : 1.[3] Temporomandibular disorders occur at any age but most often in young adults.[4,5]

Pain the temporomandibular area is the most common complaint and is easily related to a lesion of the TMJ.[6,7] However, the exact cause of TMJ problems is often difficult to determine because several disorders may be present at the same time and combined muscular and inert tissue disorders are not uncommon. As a consequence, it may be necessary to treat both joint and muscle. Conservative treatment is still the most effective management for more than 80% of patients.[8]

There are several major causes of TMJ problems:[9]

- *A single major injury*, even if it has been only a light blow to the mandible or extreme stretching of the TMJ, can permanently injure the joint and ligaments.[10]
- *Repetitive microtraumas*, such as clenching, grinding or atypical chewing for a prolonged period of time, may damage the joint and then lead to muscular problems.
- *Monoarticular arthritis and inflammatory polyarthritis*, such as rheumatoid arthritis, but seldom psoriatic arthritis, may affect the joint.[11]
- *Abnormal biomechanical loading* as a result of structural changes of the teeth may also be harmful. However, this should not be overestimated as a cause of TMJ problems.[12]

Disorders of the temporomandibular joint are summarized in Table 1.

Disorders of the inert structures

The most common disorders of the inert structures are internal derangement and arthritis. The former may be present in hypermobile joints or may affect a normal joint, so leading to hypomobility.

Internal derangement

Internal derangement is usually the result of lack of coordination between the meniscus and the condyle, in which the meniscus displaces anteriorly and the condyle posterosuperiorly. Acute or chronic repetitive injury may be the cause. Acute injury, such as whiplash, traumatic tooth extraction or intubation during anaesthesia, may displace the condyle posteriorly, so stretching the posterior attachments of the meniscus. Repetitive microtraumas occurring over a longer period, as a result of loss of posterior teeth or interference with the incisors, may provoke a posteriorly directed force on the jaw, which again displaces the mandibular head posteriorly.

Initially, this process is characterized by reciprocal clicking, later by temporary or continuous locking. Finally, osteoarthrosis may result.

Each stage of internal derangement may give rise to painful chronic irritation of the synovium, with resulting arthritis.

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Table 1 Summary of disorders of the temporomandibular joint

Disorder	Symptoms	Signs	Treatment
Disorders of the inert structures			
Internal derangement			
Reciprocal clicking	Mainly on opening Temporary locking Stiff feeling	Opening slightly diminished Chin deviation to ipsilateral side	Stretching capsule and ligaments Repositioning splint
Fixed dislocation of meniscus	Sudden locking	Opening strongly limited Chin deviates to ipsilateral side Pain on deviation to contralateral side	Manipulation
Arthrosis	Grinding sound	Pain at all tests and during the whole of each movement	Spontaneous cure Stop clenching
	Crepitus	Crepitus Opening limited Contralateral deviation limited On opening: chin deviates to ipsilateral side Back of condyle tender on palpation	Dental correction Friction to joint capsule Intra-articular injection Forced stretching Arthroplasty
Luxation of condyle	Cannot close mouth	Closing of mouth limited Elastic recoil on passive closing	Manipulative reduction
Synovial disorders			
Monoarticular steroid-sensitive arthritis	Crepitation Pain Stiffness	Opening painful and limited Ipsilateral deviation on opening Contralateral deviation when closed Resisted opening: pain	Intra-articular injection
Inflammatory polyarthritic diseases	As in monoarticular steroid-sensitive arthritis	As in monoarticular steroid-sensitive arthritis	General treatment of underlying disorder
Infections	Pain	Severely ill Opening painfully limited and ipsilateral deviation	Hospitalization Aspiration Antibiotics
Sympathetic arthritis	Pain	Opening slightly diminished Pain on all movements Tenderness on palpation Abscess	Spontaneous cure Antibiotics
Arthritis from loss of molar teeth	Continuous pain	Opening slightly decreased Pain at extreme of all movements	Dental correction
Disorders of the contractile structures			
Myalgia	Dull aching, continuous pain worse in cold weather and at end of day	Pain on resisted closing and on maximal active opening Painful bands	Stabilization appliance Stop bruxism or tooth clenching
Muscular trismus	Vague pain	Tremor of jaw Incoordination on movement Diminished opening	Solve underlying problem
Abscess in pterygoid muscles	Pain on chewing	Opening diminished Pain on clenching Resisted contralateral deviation painful Fever	Antibiotics Drainage

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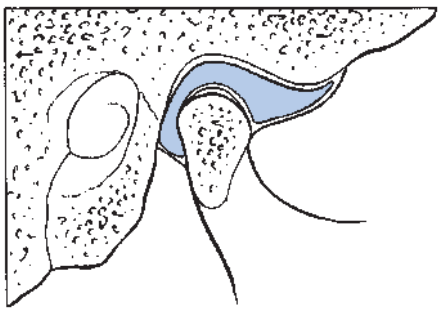

Fig 1 • Anterior displacement of the disc.

Reciprocal clicking

Clinically there is a 'click', usually most noticeable on opening the mouth and less so on closing. A click implies that the TMJ, which is normally a friction-free joint, is functioning abnormally. With the mouth closed, the meniscus lies too far anteriorly in relation to the condyle (Fig. 1). On opening, the click occurs when the condyle moves anteriorly and snaps beneath the meniscus and its posterior attachments to fall into its normal position. The opening click implies a reduction of the meniscus.

Clicks may occur early, intermediate or late in relation to the range of opening of the mouth. Early clicks indicate a small degree of anterior displacement; clicking more towards full opening indicates that the displacement is greater, as a result of progressive stretching of the posterior attachments. A closing click does not occur unless there is a preceding opening one. The click is present just before the mouth is closed and is the result of the condyle sliding posteriorly to the posterior band of the meniscus so that the meniscus is displaced anteriorly.

In addition to repetitive clicking, there is often a stiff feeling and a short period of pain after the joint has been immobile for a while.

On clinical examination, opening of the mouth is slightly decreased and provokes a small deviation of the chin to the affected side because the condyle cannot move fully anteriorly.

Treatment

Treatment aims to fully restore the normal range of motion, mainly the lost anterior translatory glide. The technique used is called 'joint liberation'[13] and consists of passive movements applied in all directions in order to realign the shortened fibres of the capsule. All the techniques can be performed under steady pull or, if pain is excessive, with small vibrations. Each manœuvre is performed over about 20–30 seconds and repeated several times. The whole procedure takes about 15 minutes.

Technique: distraction

The patient sits on a chair with the therapist facing the painless side and stabilizing the patient's head with the ipsilateral hand. The other thumb, protected by a rubber pad, is placed on the molar teeth and the fingers pass round the mandibular body. Downward pressure is applied to the molar teeth, which distracts the joint surfaces, allowing repositioning of the meniscus on the condyle.

Technique: distraction with anterior glide

In the same position, the tip of the index finger of the manipulating hand is hooked around the mandibular angle. Downward pressure and anterior pull are applied. This restores the anterior translatory glide which has been lost because of capsular retraction or because of the anteriorly displaced meniscus.

Technique: combined distraction, anterior glide and lateral stretch

The mandible is additionally pulled to the side, towards the therapist, thus realigning the fibres in all three planes of space.

Technique: lateral glide without distraction

The position and fixation of the patient is the same as for previous techniques. The hand grasps the mandible around the angle and pulls it towards the therapist, restoring lateral joint movement.

Home exercise programme

The patient is shown how to perform self-mobilizing techniques on opening, on forward movement and on lateral excursions of the mandible.

The muscles of mastication should be gently stretched to their full length. Coordination can be increased by practising hinge-type movement of the jaw and then making specific lateral movements without protrusion. Jaw movements must be limited to the click-free range and chewing on the side that provokes any noise must be avoided.

If significant painful clicking is associated with a possible meniscus displacement, a repositioning occlusal splint can be useful. It is used only if a small change in the position of the mandible stops the click on opening and closing of the mouth. It should initially be worn 24 hours a day for 8–10 weeks, except while eating. Once the clinical symptoms have diminished, use of the appliance is progressively decreased, although a part-time repositioning appliance is needed.[9,14]

Resisted muscular exercises on opening, forward movement and lateral deviation may be used in association.[15]

Fixed dislocation of the meniscus

Progression of the disorder may finally lead, via momentary self-reducing dislocations, to a permanent luxation of the meniscus. From time to time the joint becomes suddenly locked, with the meniscus lodged anterior to the condyle.

With a dislocated meniscus, clinical examination shows a reduced range of opening of the mouth. Because of loss of anterior translatory glide, only rotation is possible. In such a condition the patient can always open the mouth by at least 1 cm but often not any wider. Full closure remains possible and is painless. The chin deviates to the ipsilateral side and deviation to the contralateral side is painful.

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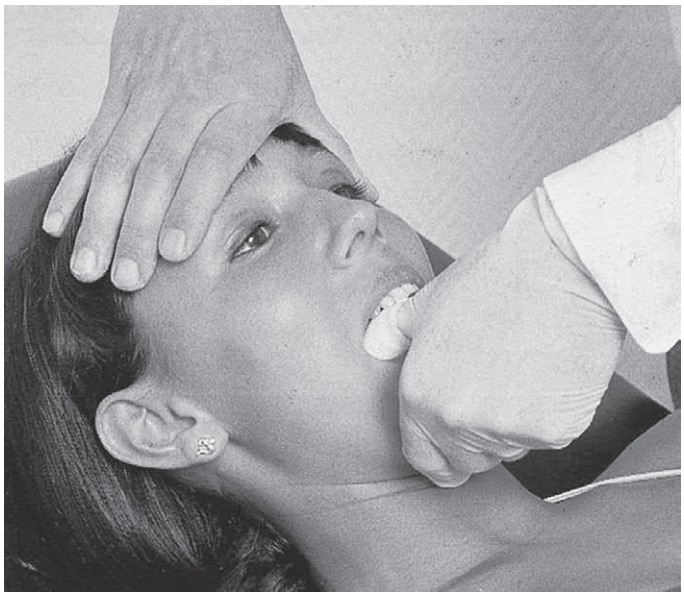

Fig 2 • Manipulative reduction of a dislocated disc.

Treatment

Manipulative reduction

Manipulative treatment aims to restore the normal relationship between the meniscus and the joint surfaces.

Technique (Fig. 2)
The patient lies on a high couch. The manipulator stands at the patient's opposite side and puts one thumb, protected by a thick pad, on the molar teeth of the affected side. The other hand is put around the patient's head and holds it steady. Caudal pressure is now applied to the molar teeth and swift translatory movements of the patient's mandible are performed, three or four times. During the manœuvre, the reduction click is felt.[16]

Manipulative reduction usually succeeds in one session.

Problems arising because of hypermobility

Hypermobile joints may also give rise to reciprocal clicking, subluxations and dislocations because of excessive translatory glide. On clinical examination, excessive movements are found together with clicking. Treatment consists mainly of stabilizing the joint and avoiding excessive anterior translatory gliding of the condyle by controlling the rotation in the joint. To do this, the patient is instructed to put and to keep the distal third of the tongue flat against the palate during opening, which limits opening to rotation movement only and reduces the tendency to anterior sliding. This method of opening should also be performed when chewing and protects the joint from further wear and tear.

Arthrosis

Arthrosis of the TMJ may be the final stage of internal derangement or is sometimes due to bruxism or missing molar teeth. In primary osteoarthrosis, no apparent cause is present.[17]

The outcome of progressive change in internal derangement is shortening and fibrosis of the meniscus–condyle attachments, and occasionally perforation or total rupture of the meniscus. Paradoxically if the latter occurs, there is only a small limitation or even complete freedom of movement, so that subjectively the patient feels better although radiography clearly shows the degenerative changes. The click changes to a more grinding noise.

Patients with arthrosis are usually over 40 years of age and it remains unilateral.[18] Usually, it does not give rise to significant inflammation or swelling, or to severe pain or limitation of movement. Only when the capsule is inflamed does it cause pain, in which case it is often present in any part of the range of movement. Crepitus is frequently found; limitation of opening of the mouth and of contralateral deviation of the chin may be present. On opening, the chin deviates to the affected side. Palpation of the back of the condyle is usually tender. Pain and stiffness increase during the day.

A radiograph shows flattening of the anterior slope of the condyle and the posterior slope of the articular tubercle, with loss of joint space and formation of osteophytes.

The complaints usually diminish progressively over 2–3 years with little painless residual disability. However, about one in five patients still has pain after 2 years.[19] Ankylosis is uncommon.

Treatment

Treatment should be directed towards the cause: dental correction is performed and clenching and grinding is stopped. Symptoms may be relieved by deep friction to the joint capsule followed by forced movements.[15]

An intra-articular injection with steroid should be given if stretching alone is insufficient. The results are better than forced movements and friction alone.

If intra-articular injection is without effect, or when ankylosis appears imminent, capsular stretching should be performed.

If either intra-articular injections or capsular stretching have failed, arthroplasty may be indicated. Because open surgery can be followed by formation of fibrotic adhesions in the joint, so limiting the normal range of movement, or cause malocclusion, surgical treatment must only exceptionally be recommended.

Technique: deep friction (Fig. 3)
For friction the patient lies on a couch, painful side up. The therapist sits behind the patient and places the index of the ipsilateral hand, reinforced by the middle finger, in front of the joint line. This is easily palpable, just cranial to the mandibular head, on opening and closing the mouth. The other hand stabilizes the head on the skull. The index finger is now pulled backwards while pressure is applied.

Friction is given for about 20 minutes, three times a week.

Technique: intra-articular injection (Fig. 4)[16]
The patient lies with the affected side up. The posterior aspect of the mandibular condyle is palpated, anterior to the tragus and below the zygomatic bone. The space to be injected lies

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Fig 3 • Deep friction to the temporomandibular joint capsule.

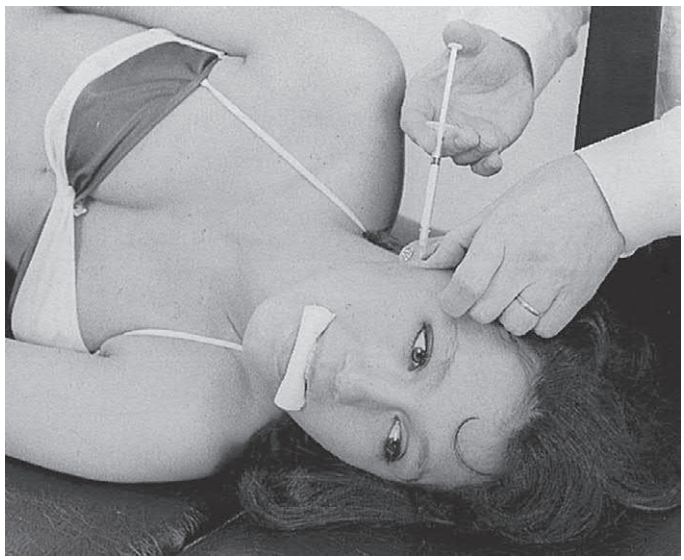

Fig 4 • Intra-articular injection of the temporomandibular joint.

just posterior to the condyle and is located with the patient's mouth wide open, moving the condyle as far anteriorly as possible. The injection should be given into the inferior compartment of the joint, beneath the meniscus.

A 2.5 cm needle is fitted to a syringe containing 1 ml triamcinolone acetonide. The needle is thrust in almost vertically and penetrates the joint space about 1 cm deep to the skin. The full amount of fluid is injected provided there is no resistance. If resistance occurs, the needle must lie either in the articular cartilage, the meniscus or under the periosteum. Injection into cartilage must be avoided because of the risk of further damage. The needle is slightly withdrawn and its tip relocated.

The patient is reviewed after a week. Usually, one to two injections suffice.

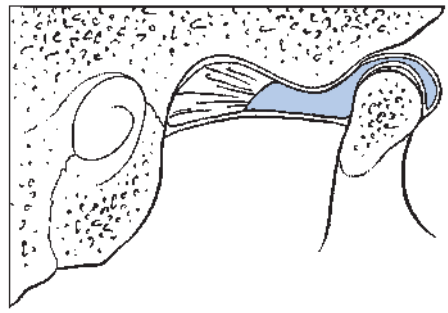

Fig 5 • Luxation of the condyle.

Fig 6 • Manipulative reduction of luxation of the condyle.

Luxation of the condyle

In luxation, both condyle and meniscus lie in front of the articular tubercle (Fig. 5). It is usually bilateral and is encountered most frequently in hypermobile joints. The onset is often a result of yawning or excessive laughing with the mouth wide open. The patient is suddenly unable to close the mouth. If closing is tried passively, there is an elastic recoil.

Sometimes the dislocation is temporary and can be reduced by the patient. In other cases, manual reduction is needed.

Treatment

Treatment is by manipulative reduction.

Technique: manipulative reduction (Fig. 6)
The patient sits in a chair. The manipulator stands at the front, puts both thumbs on the molar teeth and grasps the mandible with the fingers. Caudal pressure is now applied with the thumbs and simultaneously the chin is pulled in the cranial direction so as to close the mouth.[20]

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Synovial disorders

Arthritis of the TMJ is clinically characterized by pain on movement, crepitus and tenderness over the joint. Sometimes progressive limitation of opening of the mouth follows but the joint is only exceptionally ankylosed.

Monoarticular steroid-sensitive arthritis[15]

For no apparent reason, a patient may develop progressive pain in one of the TMJs without other joints being affected.

Opening the mouth becomes progressively painful and limited and may lead to inability to take solid food. On mouth opening the chin deviates towards the affected side but at rest in the closed position it deviates towards the other side. The disorder may persist for months. Because the lateral pterygoid muscle is attached to the anterior part of the joint capsule, in a severe case resisted opening of the mouth may also be painful.[18]

Pain and stiffness are worse in the morning and improve after movement. Technical investigations for inflammatory disorders, such as radiography and laboratory tests, are negative.

Treatment

An intra-articular injection with steroid is usually effective and may be repeated after 1 week (see above).

Inflammatory polyarthritic diseases

Juvenile rheumatoid arthritis, ankylosing spondylitis and rheumatoid arthritis sometimes involve the TMJs. They affect the synovium and may subsequently lead to destruction of bone. The clinical findings are the same as for monoarticular steroid-sensitive arthritis. The fact that other joints are affected, together with radiographic findings and laboratory tests positive for rheumatological disorders, reveal the exact nature of the condition.

Psoriatic arthritis, gout and lupus erythematosus are very rare and seldom give rise to bone destruction. There is no separate treatment for the TMJ; the joint is included in measures against the underlying disorder.

Infections

Infections of the TMJ are rare and are usually the result of an adjacent infection of the external auditory canal, the parotid gland or the middle ear. Inflammation may sometimes occur in viral diseases such as infectious mononucleosis, measles and mumps.[18] Gonococcal infection has been reported.

The patient may be severely ill and complains of temporomandibular pain and limitation of mouth opening, with deviation towards the ipsilateral side. Bacterial infection of the TMJ requires hospitalization and, after culture of aspirated pus,[21] is treated by antibiotics.

Sympathetic arthritis

An abscess in the neighbourhood of a TMJ may provoke a 'sympathetic' arthritis.[15] Causes are peritonsillar abscess, pericoronitis of a partially erupted wisdom tooth or molar extraction. There is progressively increasing difficulty in opening the mouth, usually starting, if the cause is dental, 1–2 days after an intervention. Pain is found on all movements, and the bone at the base of the tooth is tender on palpation.

Spontaneous cure is usual in 2–3 weeks after molar extraction. If active treatment is necessary, it should be directed to the underlying disorder and not to the arthritis as such.

Arthritis due to loss of molar teeth

Patients lacking all molar teeth (unilaterally or bilaterally) sometimes develop unilateral arthritis which may occur at either side in relation to the missing molar teeth. Molar teeth normally have a distance-maintaining effect in apposition between the mandible and maxilla. Should this be lost, excessive upward pressure by the mandibular condyle on the temporal fossa occurs, which may lead to arthritis. This can also occur as a result of treating patients, who have missing molars, by cervical traction.

Patients complain of a continuous deep burning pain in the temporomandibular area, not necessarily influenced by eating. On examination pain is found at the extreme of all movements and slight limitation of opening may be present.

Treatment consists of dental correction of the distance between mandible and maxilla. When cervical traction has to be given, a splint of sufficient thickness to divert the strain to the posterior part of the mandible should be used (Cyriax:[15] his p. 199).

Disorders of the contractile structures

Muscular disorders are usually the consequence of bruxism or clenching, which is commonly an unconscious way of coping with the stress of daily life and is seen more often in patients who are more subject to anxiety and depression than are members of the average population.[22]

In addition to muscular pain, tooth wear, alveolar tenderness, mobility of the anterior teeth, pain and morning jaw stiffness are usually found.

The pain is elicited on maximum resisted isometric contraction.

Myalgia

Myalgia is described as a dull aching pain felt continuously in the area of the masticatory muscles and it may affect any of these.[23] Cold weather may precipitate the symptom which, although present at all times, is usually worse at the end of the day, sometimes also disturbing the patient's sleep. Resisted closing of the mouth is painful, as is maximum active opening,

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which stretches the muscles. The muscles are usually very tender, and firm palpable bands within them are often present.

The differential diagnosis includes temporal arteritis, which can also lead to myalgic pain. Redness and swelling over the artery are characteristic of arteritis.

Treatment

Primary therapy is a full-arch occlusal stabilization appliance. This involves the construction of a flat occlusal surface that is adjusted to have multiple tooth contact in a habitual comfortable jaw closure position.[9,24] Initially it is worn continuously, except on eating, for 6–8 weeks and is adjusted several times to establish a comfortable jaw position. As the symptoms decrease, the amount of daytime wear of the appliance is progressively reduced.

In musculoskeletal pain from bruxism or tooth clenching the patient must break the habit. Advice is given to reduce all physical and mentally stressful activities during treatment. Muscle relaxants or anti-anxiety drugs can be useful. Hard or chewy foods are avoided. Application of moist heat to the temporal and masseter muscles for 20 minutes, three or more times a day, can be helpful.

Muscular trismus

Pathological muscular stimulation or inhibition may limit the opening of the mouth but only very rarely affect closing. Limitation is often the result of attempting to avoid pain. Trismus can be the outcome of a chronic pain problem or of a recent local event (injury or surgery).

When movement is attempted, there is often tremor of the jaw and incoordination. Treatment consists of solving the underlying problem.

Hysterical trismus is characterized by a total loss of ability to open the mouth. A vague pain, which extends over the whole of the head and face, is usually described. Very often these patients also hold the neck extremely still. Because it is very difficult to examine the TMJ in an unwilling patient, it is better to proceed with caution: multiple positive findings and inconsistencies should warn the examiner of a severe disorder or hysteria.

Warning

When trismus is present, *tetanus* should not be forgotten. Painless stiffness coming on abruptly at the TMJ, and causing complete inability to open the mouth, may be an early sign of the condition. In this event, both joints are usually affected.

Abscess in the pterygoid muscle

An anaesthetic injection for dental work may be followed some days later by an abscess. The cheek begins to hurt and feels stiff. The patient complains of problems on opening the mouth and has pain on chewing.

Clinical examination reveals a loss of range of mouth opening, together with pain on clenching. Resisted deviation towards the painless side also hurts.

Signs of infection including pyrexia should be sought. Treatment is with antibiotics and drainage.

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Non-mechanical disorders of the lumbar spine: pathology

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Disorders

Spondylolisthesis

In 1782, the Belgian gynaecologist Herbiniaux described a severe case of lumbosacral luxation, which he considered as a potential obstetrical problem.[1]

A precise definition of spondylolisthesis was first given by Kilian in 1854[2] – a spinal condition in which all or a part of a vertebra (*spondylo*) has slipped (*olisthy*) on another.

Wiltse *et al*[9] described five major types:

- *Dysplastic spondylolisthesis* is secondary to a congenital defect of the first sacral–fifth lumbar facet joints, with gradual slipping of the fifth lumbar vertebra.
- *Isthmic or spondylolisthetic spondylolisthesis* is the most common type of spondylolisthesis. The basic lesion is in the pars interarticularis. The vertebra above can slip as the result of a lytic process, an elongation without lysis or an acute fracture (subtypes a, b and c). If a defect in the pars interarticularis can be identified, but no slip has occurred, the condition is called a 'spondylolysis'.
- *In degenerative spondylolisthesis* an advanced degeneration of the facet joints and a progressive change in the direction of the articular processes allow the vertebra to slip forwards. The condition occurs four times as frequently in females than in males and nearly always at the fourth lumbar level (Cyriax:[3] p. 288; Rosenberg[4]). The slip is never severe. This condition has been discussed in Chapter 35 on the stenotic concept.
- *Traumatic spondylolisthesis* results from a fracture of a posterior element other than the pars interarticularis.
- *Pathological spondylolisthesis* develops as the result of weakness caused by a local or generalized bone disease.

In this chapter we discuss only the spondylolytic spondylolistheses.

Aetiology

Isthmic spondylolisthesis has been defined as 'a condition in which fibrous defects are present in the pars interarticularis, which permit forward displacement of the upper vertebrae and separation of the anterior aspects of the vertebra from its neural arch' (Fig. 1).[5] The aetiology of this bony defect (spondylolysis) has been discussed for decades but it is now widely accepted to be the result of a congenital weakness. The defect itself is not present at birth, however, but develops in childhood, probably as the result of repeated stress and trauma.[6–8] Stress fractures form in the weakened pars interarticularis; fibrous tissue fills the gap, and further tension enlarges the defect.[9] Forward slipping of the vertebral body therefore occurs most frequently between the ages of 10 and 15 years, and progression is unlikely after adolescence.[10,11] The reported incidence of spondylolisthesis is between 4 and 7%,[12–14] although a higher incidence has been reported among Eskimos (18–56%).[15,16]

Grading

Spondylolysis is visualized by an oblique view of the lumbar spine which shows the well-known 'collar on the Scottie dog's neck' (Fig. 2).

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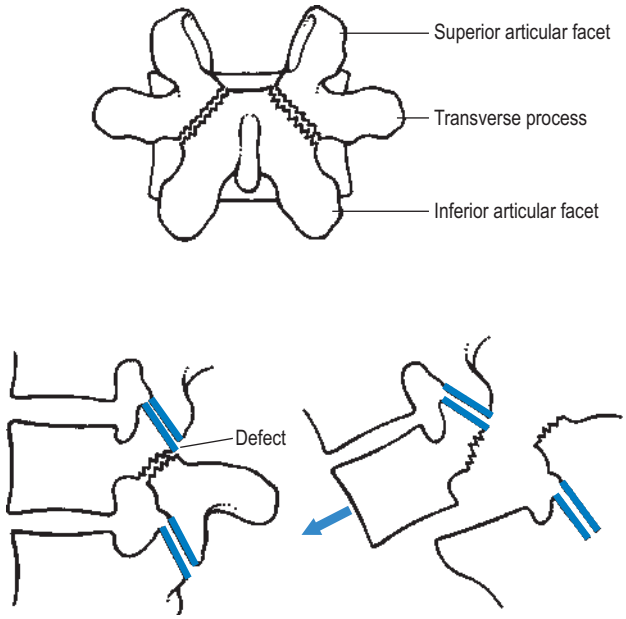

Fig 1 • Spondylolisthetic spondylolisthesis: fibrous defects in the pars interarticularis permit forward displacement of the upper vertebra and separation of the anterior part of the vertebra from its neural arch.

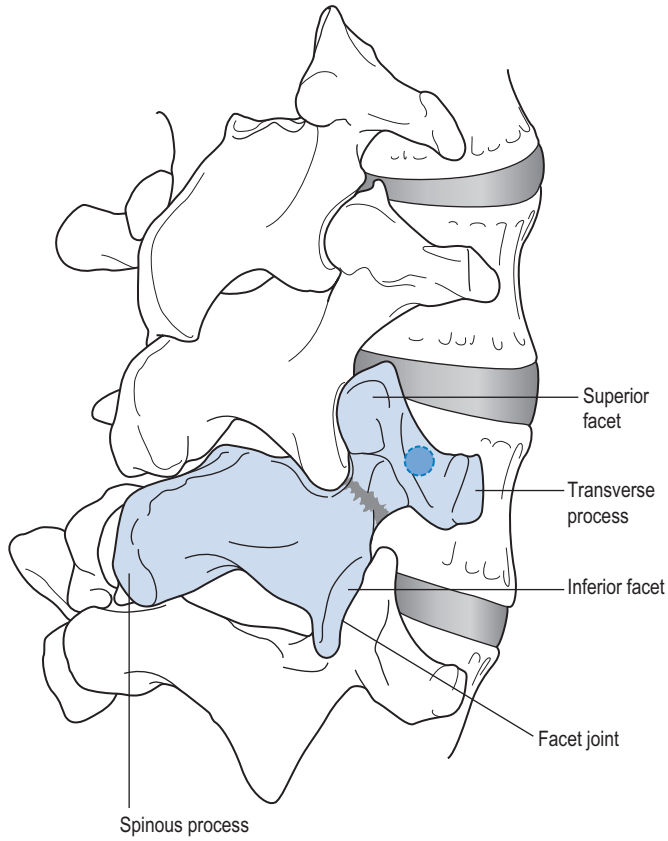

Fig 2 • The defect in the pars interarticularis is best visualized in an oblique view ('Scottie dog with collar'). From Magee 2008 Orthopedic Physical Assessment, 5th edn, Saunders, St Louis. Reproduced by kind permission.

Forward slipping is best visualized on a lateral radiograph and the amount of listhesis is graded by the Meyerding's system (Fig. 3):[17] the upper sacrum is divided into four parallel quarters and the degree of slipping is calculated from the distance that the posterior edge of the fifth lumbar vertebra has shifted on the posterior edge of the sacrum in relation to the total width of the upper sacrum. Grade I is a shift of less than 25%, grade II between 25 and 50%, grade III between 50 and 75% and grade IV more than 75%.

Some authors emphasize that there is a significant difference in measurements when the radiographs are taken with the patient in a recumbent rather than erect position.[18]

Clinical findings

It should be emphasized that most cases of spondylolisthesis are asymptomatic. Even severe displacements may be present in very active patients, without the slightest discomfort. In a radiological study of 996 adult patients with low back pain, MacNab found spondylolisthesis in only 7.6%, which is not significantly higher than in the population as a whole (4–6%).[19] Therefore caution must be taken before ascribing back pain or sciatica to spondylolisthesis and the radiological demonstration of a defect in a patient with back pain does *not always* indicate that the source of the symptoms has been discovered.[20,21]

Spondylolisthesis can produce backache or sciatica in two ways: spondylolisthesis as the basis of a secondary disc lesion and the spondylolisthesis itself causing symptoms.

Spondylolisthesis with secondary disc lesion

As early as 1945, Key stated that symptoms in spondylolisthesis were far more often caused by a disc lesion than by slippage of the vertebra.[22] The clinical features are exactly the same as in patients without spondylolisthesis, and nothing in the history or clinical examination arouses suspicion, except some irregularity of the spinous processes on examination. Radiographs carried out in the erect posture disclose the slip. It is obvious that the management of disc lesions occurring in spondylolisthetic spines is exactly the same as in those without bony defects. The only difference is probably the liability to recurrence of acute or chronic discodural conflicts. As in other forms of lumbar instability, sclerosing injections can have a good preventive outcome after reduction has taken place.

Spondylolisthesis of itself causing symptoms

Spondylolisthesis can cause both backache and sciatica. The former has postural ligamentous characteristics: the ache is central, sometimes with vague and bilateral radiation over the lower back. The discomfort is associated more with maintaining a particular position than with exertion. Dural symptoms

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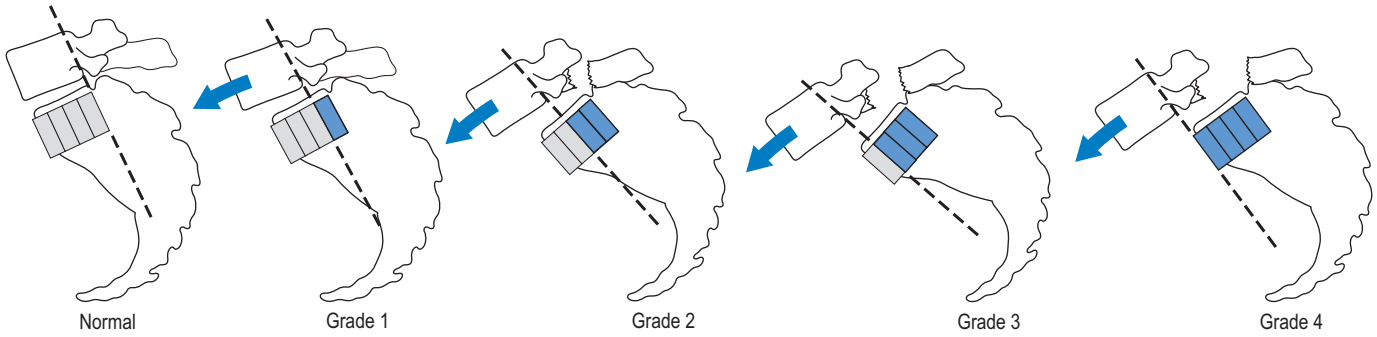

Fig 3 • Grades of spondylolisthesis (from Meyerding).[17]

are absent. There are no articular signs or symptoms; lumbar mobility is full and painless. Root signs are also absent. The only clinical finding is a bony irregularity palpated over the spinous processes. Treatment is that for ligamentous backache and consists of sclerosing injections (see p. 579).

Spondylolitic sciatica very much resembles a bilateral lateral recess stenosis but the patient is much younger. Increasing pain and paraesthesia appear in the standing position and may force the patient to sit or lie down, which causes the symptoms to disappear. Dural symptoms are absent. Clinical examination reveals little: there is a normal range of movement without pain. Root signs, such as positive straight leg raising, weakness or sensory loss are not found (J. Cyriax, personal communication, 1983; Calliauw and Van[23]).

The cause of sciatic pain in spondylolisthesis is unknown. The different hypotheses are:

- The forward movement of the listhetic vertebra drags on the nerve roots, which engage painfully against the shelf formed by the stable vertebra below (Cyriax:[3] pp. 287–290).
- A fibrocartilaginous mass, with or without small ossicles, may form at the defect in the pars interarticularis. Adhesions around the nerve root and compression result.[24]
- With the forwards and downwards drop of the vertebral body, the pedicles descend on the nerve roots and kink them as they emerge through the foramen.[19]
- A forwards slipping of the vertebral body moves the transverse processes in a forwards and downwards direction, allowing the L5 roots to be pinched between the sacrum and transverse process (the 'far-out' [25]).

Treatment

Patients with an accidentally detected spondylolisthesis do not need treatment. If the pain is caused by a secondary disc lesion, the patient is treated by the normal procedures used in discodural or discoradicular problems: manipulation, traction or epidural anaesthesia. The liability to recurrences after successful treatment, however, is much increased and in patients with both spondylolisthesis and a disc lesion the displacement will reappear. In these instances, sclerosing injections are used as a prophylactic measure.

Spondylolisthesis which of itself causes lumbar pain should always be treated non-surgically. Sclerosing injections to the ligaments often give good results.

If leg pain is a significant problem, nerve root infiltrations can often abolish it. The patient can also be advised to wear a corset during occasional strenuous activity. If root pain cannot be abolished by these conservative measures, surgery should be considered. Surgical intervention can also be considered if the listhesis is progressive or the patient presents with a Meyerding grade III or IV. The gold standard of surgical treatment is fusion in situ.[26] The different techniques for fusion have mixed and variable results[27–29], and the possibility of complications.[30] Recently, reduction of the listhesis and stabilization, whether by bilateral lateral fusion or interbody fusion, has been recommended.[31,32]

It should be remembered, however, that even in grade III and IV listhesis, good results have been described after non-surgical treatment.[33] Apel *et al*.[34] reported on the long-term results (40 years) after surgical and non-surgical treatment of grade I and grade II spondylolisthesis. Of the conservatively managed patients, all functioned well. Among those undergoing surgery, poor results were confined to those patients in whom the fusion failed, and a pseudarthrosis developed (40%). Frennered *et al*[35] stated that operative treatment for low-grade spondylolisthesis does not seem to give better results than conservative treatment. More recent prospective studies, however, conclude that surgical management of adult isthmic spondylolisthesis improves function and relieves pain more efficiently than an exercise programme.[36,37]

Osseous disorders

Osteoporosis

Osteoporosis is a metabolic disease, related to several different disorders. It is characterized by a reduction of bone mass which occurs predominantly in the axial skeleton, the femoral neck

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and the pelvis. By radiographic criteria, 18% of men and 29% of women between the ages of 45 and 79 years of age have evidence of osteoporosis and more sensitive methods for determining vertebral bone mineral density show that 50% of women past the age of 65 have asymptomatic osteoporosis.[38] The radiographic appearances are changes in bone porosity, trabecular pattern and vertebral body shape (the so-called biconcave fishmouth vertebrae).[39] It is a common mistake to believe that these changes account for patients' backache. It should be remembered that uncomplicated osteoporosis does not cause any symptom except some loss of height of the spine. Thus the major explanation for long-standing back pain in the elderly does not appear to be related to spinal osteoporosis and, if a radiograph shows uncomplicated osteoporosis in a symptomatic patient, other sources for the pain should be sought.[40] Osteoporosis may, however, lead to a pathological fracture. If this takes place, a sudden pain in a girdle distribution will result.

Paget's disease

Osteitis deformans or Paget's disease of bone[41] is a localized disorder characterized by a remarkable hyperactivity of osteoclasts and subsequent increase of osteoblastic bone deposition. As a result, the normal bone architecture is completely disturbed.[42] In a vertebral body this can result in softening, broadening and collapse of the bone. The disease is reported to occur in approximately 4% of individuals over the age of 40.[43] In the majority, the disease is restricted to a few bones.

It must be emphasized that most patients with Paget's disease are asymptomatic.[44] The main problem for the clinician therefore is not the discovery of the Paget's disease but the association of the back symptoms with the Pagetoid lesion. Back pain and the associated angular kyphosis arise as the result of collapse of the vertebral body. Sometimes new bone growth in the vertebral arch may compress nerve roots, resulting in a spinal stenosis or a lateral recess stenosis.[45,46]

Fractures

Crush fracture of the vertebral body

This causes a wedge deformity. It usually occurs at the upper lumbar or at the thoracolumbar level and usually results from axial trauma or from flexion injuries (see online chapter *Disorders of the thoracic spine: non-disc lesions – disorders and their treatment*). Wedging of a vertebral body may also result from a pathological fracture which is the consequence of senile osteoporosis, tumour, Paget's disease or tuberculous caries.

Immediate post-traumatic pain is referred bilaterally in the appropriate dermatome. The diagnosis is not missed if backache follows a gross trauma. In a pathological fracture, however, the patient probably does not recall an injury that can be related to the onset of the symptoms. Nothing in the history then warns the examiner of the possibility of a crushed vertebra. However, if the history is taken carefully, and compared with the clinical findings, some unlikelihoods will immediately become obvious. They are:

- the pain is usually located in the upper lumbar area: pathological fractures occur more often in the 'forbidden area'
- dural signs are absent: although the patient describes an intense backache, coughing does not hurt
- inspection reveals an angular kyphosis
- examination shows a capsular pattern, with symmetric limitation of lateral flexion
- there are no dural signs: straight leg raising is normal, which is always suspicious in a case of acute lumbago.

The girdle pain lasts a week or two, whereafter a localized bone pain remains. After 3 months the fracture will have united and symptoms have ceased. Although a wedged vertebra results in lasting malalignment of the related posterior joints, usually little ligametous pain results. Any continuous pain after the fracture has healed is from a coincident disc lesion. Indeed, any force sufficient to break bone will also threaten disc tissue and it is not hard to imagine that, after a crush fracture of a vertebral body, the disc above or below the lesion may also be damaged. The possibility of a coincident disc displacement should therefore be considered in a case with persistent pain. Because of the permanent kyphosis and the possible elongation of supra- and interspinous ligaments, the protrusion may be very unstable and difficult to treat. Although manipulation usually affords excellent results, the improvement is not lasting unless sclerosing injections are given to stabilize the joint.

Spondylolysis

Isthmic spondylolysis is considered to represent a fatigue fracture of the pars interarticularis of the neural arch. There is a relatively high incidence of radiographically identified spondylolysis in the general population, but the vast majority of these lesions probably occur without associated symptoms.[6,47,48] The incidence of spondylolysis in the young athletic population shows an almost fivefold increase compared to the general population,[49] with the highest rates in weight lifters,[50] gymnasts[51] and football players.[52] Given this high incidence of asymptomatic lesions, the relation between unilateral or bilateral back pain and a fatigue fracture of the pars interarticularis remains unclear.[53]

However, recent histological studies could identify a well-developed ligamentous structure covering the defect ('the spondylolysis ligament') and containing thin unmyelinated nerves.[54,55] Infiltration of bupivacaine hydrochloride (Marcain) into the pars defect produced temporary symptom relief, which proves the existence of symptomatic lesions.[56]

Symptomatic lesions appear to be particularly a clinical problem in adolescents, especially adolescent athletes. Although clinical features of active spondylolysis do not differentiate this condition from other causes of low back pain,[57] suspicion may arise when an adolescent athlete presents with unlilateral backache without dural signs nor symptoms, and pain is provoked by full extension.[58,59]

On plain radiography, the defect in isthmic spondylolysis is visualized as lucency in the region of the pars interarticularis. The lesion is commonly described as having the appearance of

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a collar on the 'Scotty dog' seen in lateral oblique radiographs (see Fig. 2). Plain radiography has limited sensitivity, however, and nowadays bone scintigraphy with single photon emission computed tomography (SPECT) is considered as the gold standard of investigation.[60,61]

Conservative treatment consists of relative rest and the avoidance of activities associated with increased pain.[62]

Fractures of the transverse processes

These may occur after direct trauma to the back. Alternatively they result from gross muscular effort, frequently a resisted rotation strain; lumbar manipulation is a rare cause of fracture.

The patient complains of post-traumatic and localized unilateral pain. Clinical examination reveals a partial articular pattern, with pain during side flexion away from the affected side. However, there is also pain during resisted movements: resisted side flexion to the painful side and resisted extension in the prone-lying position are both positive. The lesion may appear insignificant on radiograph.

Pain usually ceases after a fortnight. If pain persists, a coincident disc lesion should be suspected. Alternatively, an emotionally unstable patient may capitalize on radiological evidence of a 'fractured spine'.

Stress fractures of the lumbar pedicle

Stress fractures of the contralateral pedicle in patients with unilateral spondylolysis has recently been reported[63,64] and termed 'pediculolysis'.[65] The development of a unilateral spondylolysis probably leads to a redistribution of forces, resulting in a stress fracture of the contralateral pedicle. Alternatively, not a fracture but a compensatory sclerosis and hypertrophy of the contralateral pedicle develop.[66,67] To date, it is not clear if the lesions are responsible for particular clinical syndromes.[68]

Tumours

Classically, neoplastic lesions in the lumbar spine are classified as benign or malignant lesions. The latter are subdivided into primary malignancies and metastases. Benign and primary malignant neoplasms are rare in the lumbar spine, whereas secondary deposits are common. Although the diagnosis of tumours of the lumbar spine is largely dependent on radiological examinations, it must be remembered that 30% of the osseous mass of bone must be destroyed before a lesion is radiologically evident.[69] Therefore radiographs do not reveal early disease and too much reliance on radiographic appearances can give both the patient and the physician a false feeling of security. Therefore, in the diagnosis of neoplastic lesions, the history and clinical examination remain vital. Special attention must be paid to warning signs. When routine radiographs fail to support the clinical impression, a radioisotope scan must be obtained, in order to demonstrate the presence of a malignant lesion and the extent of the spinal involvement.

Benign tumours

Benign tumours of the vertebrae predominantly affect patients under the age of 30 years and are mostly localized in the posterior wall. Chronic localized backache that is not dependent on posture and movement and does not ease with recumbency is the main characteristic of a benign tumour. Clinical examination may show a limited range of flexion due to a muscle spasm. If the lesion compresses nerve roots, slowly progressing sciatica will supervene. Early detection of the lesion on the radiograph is not always easy.

Osteoid osteoma

This constitutes about 12% of all benign tumours (Dahlin and Unni:[70] pp. 88–101) and appears mostly in children and adults below the age of 30 years. The pain is frequently exacerbated at night and is often relieved by small doses of aspirin.[71] Treatment consists of local excision of the tumour.

Osteoblastoma

This is a rare benign neoplasma of bone but has a predilection for the spine: approximately 40% of all osteoblastomas are found in the posterior elements of the spine and sacrum.[72] The tumour is seen most frequently in males under the age of 30 years.[73] The back pain is localized, insidious in onset, with a duration of months or years and not as severe as in osteoid osteoma. Clinical examination may reveal muscle spasm and localized tenderness. Because of the expansive nature of the tumour, slowly progressive compression of nerve root(s), with radicular pain and evidence of neurological deficit, may occur.

Haemangiomas

These account for less than 1% of symptomatic primary bone tumours (Mirra:[74] pp. 492–497), although postmortem studies have demonstrated that asymptomatic lesions exist in 12% of all vertebral columns. This implies that most of these lesions remain asymptomatic throughout life. The thoracic spine is the location for 65%, the cervical spine 25% and the lumbar for only 10%. Patients with symptomatic haemangiomas are usually between 40 and 50 years of age.[75] The main complaint is localized pain. Clinical examination may show limitation of movement from muscle spasm and localized tenderness. Increased weakening may result in a pathological fracture, which in turn may cause neurological symptoms.[76] Since vertebral haemangiomas are usually asymptomatic and have a benign course, treatment is expectant. Radiation seems to afford a good outcome in patients with constant, disabling pain.[77]

Eosinophylic granuloma

This is a rare bone lesion characterized by the infiltration of bone with histiocytes, mononuclear phagocytic cells and eosinophils. It was first described by Jaffe and Lichtenstein in 1944.[78] It occurs most commonly in children and adolescents[79] and only 10% of the lesions are localized in the spine. Local and constant back pain is the first symptom. Clinical examination shows muscle spasm and local tenderness. If the lesion

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affects a vertebral body, a flattening – vertebra plana – will result. This spontaneous collapse of the vertebral body in children was first described by Calvé in 1925[80] and was thought to be a manifestation of osteochondritis juvenilis.[79] It seems that the collapse of the vertebra induces spontaneous healing of the granuloma, in that symptoms usually cease after the body has collapsed.[81,82]

Aneurysmal bone cyst

This is a benign, cystic vascular lesion of bone. The majority of aneurysmal bone cysts occur in the long bones of the extremities of young adults.[83] The lumbar spine is affected in only 10% of cases.[84] The clinical presentation is lumbar pain that usually has an acute onset and increases in severity over a short period of time. Depending on the location and size of the lesion, the other clinical manifestations vary. If the lesion is at the spinous or transverse processes, the pain remains local.[85] If the vertebral body is affected, the lesion may expand, which can result in weakening of the bone, pathological fractures and serious neurological deficits. Although aneurysmal bone cysts are benign lesions, they may cause severe damage because of their expansive characteristics. The lesion therefore must be diagnosed early and treatment instituted without delay in order to keep disability to a minimum. Treatment is by surgery, radiotherapy or cryotherapy.

Malignant tumours

Malignant tumours of the spine predominantly affect patients over the age of 50 and are mostly localized in the anterior spine elements. Metastatic lesions of the axial skeleton are much more common than primary malignant lesions (chordoma, myeloma and chondrosarcoma), the overall ratio being 25 : 1 (Francis and Hutter;[86] Mirra:[74] pp. 448–454).

Chordoma

This is a slowly developing malignant tumour that originates from the remnants of notochordal tissue and therefore occurs exclusively in the midline of the axial skeleton. It has a predilection for either end of the spinal column: 50% of cases occur in the sacrum and 38% in the skull base.[87] The lesion is rarely reported below the age of 30 years and most tumours become evident between the ages of 40 and 70.[88] Chordomas are slow-growing tumours with a locally invasive and destructive character.

The common sacral tumours may be difficult to detect. The patient initially presents with localized pain in the sacral area or with coccygodynia. The pain is dull, constant and not relieved by recumbency. Often it is of long duration and only moderate, so that it does not force the patient to seek treatment.[89] Chordomas of the sacrum extend anteriorly into the pelvis. Because the dural sleeve is not involved, presacral invasion of the nerve roots does not provoke radicular pain. Straight leg raising is also not limited. However, gross muscular weakness of one or both legs, together with considerable sensory deficit is detected. Sometimes the patient presents with urinary or bowel incontinence as well.[90] Such a gross paresis in the absence of root pain always suggests a tumour. A radiograph of the lumbar spine and sacrum discloses lytic bone destructions with calcified foci and a pre-sacral soft tissue mass.[91,92]

Patients with chordomas of the lumbar spine may present with localized central lumbar pain sometimes radiating bilaterally. Involvement of nerve roots may induce bilateral sciatica. Clinical examination then shows muscle spasm and bilateral weakness.[93]

Treatment consists of total resection of the tumour, which usually presents a major problem. Often partial resection, followed by radiation therapy, is the only option.[94] Chemotherapy is ineffective.[95]

Chondrosarcoma

This is a malignant tumour that forms in cartilaginous tissue. The tumour is frequently located in the pelvis and lumbar spine and grows extremely slowly. The usual age of onset is between 40 and 60 years (Dahlin and Unni:[70] pp. 227–259). The tumour may be symptomless over many years. Local pain is very suggestive of actively growing tumour. When neural elements are compressed by the tumour, abnormalities are found on neurological examination. The treatment of choice is total resection of the tumour.

Myeloma

This is a malignant tumour of plasma cells and is the most common primary tumour of bone; the spine is almost always involved. The disseminated form is *multiple myeloma* and accounts for 45% of all malignant bone tumours (Dahlin and Unni:[70] pp. 193–207). The patients are usually in an older age group, in that the disease is rare below the age of 50 years. Plasmacytoma is the solitary form and affects the spine in about 50% of patients.[96]

The most common complaint is of back pain, which does not vary with exertion, although initially may be relieved somewhat by bed rest. Malignant disease is suggested by steady worsening of the backache which eventually becomes continuous, irrespective of posture or movement. As the backache becomes more severe, sciatica, which is often bilateral, appears.[97] The fact that the backache does not cease after the root pain comes on and that the root pain is bilateral, immediately draws attention to the possibility of an expanding lesion. Alternatively, the backache is sudden as the result of a pathological fracture.

Findings on clinical examination depend on the extent of the disease. In the early stage, there is usually only lumbar muscle spasm and localized tenderness. In later stages, angular kyphosis and signs of nerve root compression at different levels can be seen. Finally, signs of generalized illness, such as fever, weight loss and pallor, become prominent.

Radiographically, multiple myeloma is characterized by the presence of round lytic defects in the bone without any surrounding reactive sclerosis. Occasionally, lytic defects may not be obvious, and the radiograph shows nothing more than a diffuse osteopenia.[98] In such circumstances the differential diagnosis must be made by laboratory examinations,[99] which consistently reveal an elevated erythrocyte sedimentation rate – seldom less than 100 mm/h. Characteristically, abnormal Bence-Jones proteins can be demonstrated in the urine.[100] The most important test is serum protein electrophoresis, which

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identifies a monoclonal spike in more than 75% of patients and hypogammaglobulinaemia in 9%.[101]

The usual course of multiple myeloma is one of gradual progression. Systemic therapy with melphalan and cortisone may improve clinical symptoms, but the average survival seldom exceeds 5 years.[102]

Metastatic tumours

The most common malignant tumour in the spine is metastatic cancer. The prevalence of metastases increases with increasing age, and patients who are aged 50 years or older are the population at greatest risk. Neoplasms frequently associated with spinal metastases are of prostate,[103] breast, lung, thyroid and colon.[104] Up to 70% of patients with a primary neoplasm will sooner or later develop metastases in the thoracolumbar spine.[105] The predilection of metastases for the lumbar spine may be explained by the functioning of Batson's plexus. This is a venous network, located in the epidural space between the bony spinal column and the dura mater. Because this plexus has no valves to control blood flow, metastatic cells may easily enter it and lodge in the connected sinusoidal systems of the red bone marrow of the vertebral bodies.[106]

Some suspicion may arise when, for the first time, a patient over the age of 50 presents with an attack of low back pain. Especially if the pain has a gradual onset and increases in intensity over time, the patient should be suspected of suffering from a malignant disease. The concern should be even greater if there is a prior history of malignancy. In the beginning the pain is localized but very soon it spreads down the leg in a distribution not corresponding to a single root. Sometimes there is bilateral sciatica and the lumbar pain does not ease but becomes even worse when the sciatica appears. Because the tumour often extends into the epidural space, dural symptoms may be present. However, not all skeletal metastases cause pain: symptoms may occur only when the lesion is complicated by a pathological fracture.[107]

Results of the clinical examination depend on the stage; at first, there will be muscle spasm, markedly limiting movements, especially side flexions. Localized tenderness, particularly at the 'forbidden' upper lumbar area causes more suspicion. Later, distinctive neurological signs will immediately draw attention to the possible existence of a spinal neoplasm:

- Bilateral weakness
- Weakness of the psoas muscle
- Signs of involvement of two or three consecutive roots, or non-adjacent roots
- Discrepancy between pain and weakness
- A warm foot on the affected side.

It is important to stress that radiographs may be normal and are not reliable early in the course of a metastatic lesion. Clinical symptoms and even signs of gross muscular weakness may appear before the radiograph shows erosion or collapse of bone.[108] If the clinical features of metastasis are present but the radiographic examination remains negative a bone scan may be necessary to establish the diagnosis.[109]

MRI examination is a quite sensitive complementary technique and appears to be more specific for metastasis in certain locations of the spine.[110]

Cure is seldom possible and treatment of metastatic deposits in the spine is palliative: it includes radiation therapy, corticosteroids and decompressive laminectomy.

Rheumatological disorders

Ankylosing spondylitis

This disease usually affects the sacroiliac joints initially, and then appears in the thoracolumbar area. Thereafter, the lower lumbar, the thoracic and the cervical spine also become affected[111] (see Ch. 43). Although the lesion invariably starts at the sacroiliac joints, it is possible that this does not cause any symptoms and the first complaint is then of backache.

Backache in ankylosing spondylitis is typically intermittent; it comes and goes irrespective of exertion or rest. However, the pain and the stiffness are greatest in the morning and usually improve with movement. Several segments at the upper lumbar and thoracolumbar level become involved at about the same time.[112] Because the pain is usually limited to the central part of the spine and does not refer laterally, the patient complains of vertical distribution (Fig. 4a). This contrasts with the more or less horizontal, gluteal and asymmetrical reference of dural pain in a lumbar disc disorder (Fig. 4b).

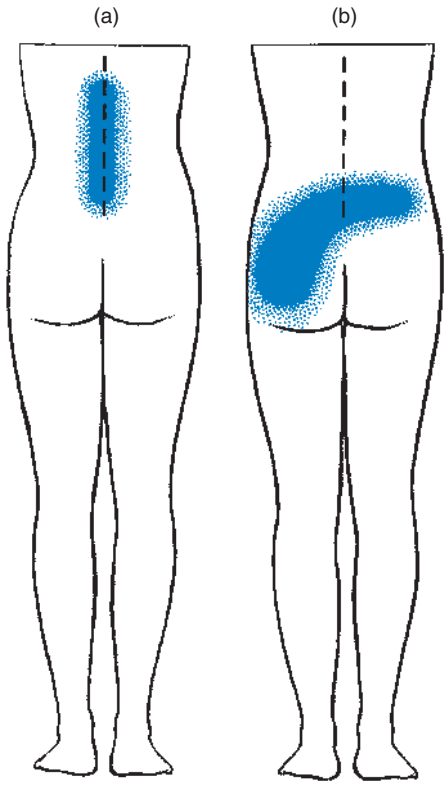

Fig 4 • Localization of pain affecting the upper lumbar level in ankylosing spondylitis (a), dural reference of low back pain in a lumbar disc disorder (b).

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Inspection usually shows a flat lumbar spine, together with the beginning of an upper thoracic kyphosis. The motion of the spine is impaired in a symmetrical way, which is best demonstrated by a gross limitation of side flexion in both directions. There is also upper lumbar tenderness and the end-feel during a downwards thrust on the prone spine is hard.

Sometimes an acute increase in pain caused by a sprain of the stiffened lumbar joints can simulate an attack of lumbago. The patient then states that lifting something heavy caused immediate and agonizing pain at the *upper lumbar* level. Although the history probably suggests a disc protrusion, there is a complete absence of dural signs, which is rather unusual in such an acute case. Furthermore, there is a symmetrical limitation of side flexion and the pain on the palpation is upper lumbar, in the 'forbidden area'.

Diagnosis is confirmed by radiography of the sacroiliac joints. Because lumbar manifestations occur some years after sacroiliac manifestations, plain radiographs of the latter will almost certainly reveal the typical narrowed joint spaces and surrounding sclerosis. In later stages, radiographic abnormalities also appear in the lumbar and thoracolumbar spines. First there are signs of osteitis of the anterior corners of the vertebral bodies. This results in the typical 'squaring' of the vertebrae. Healing of the inflammation leads to a reactive sclerosis in the anterior portions of the vertebral bodies. Later on, thin, vertically orientated calcifications of the annulus fibrosus and anterior and posterior longitudinal ligaments appear. These growing 'syndesmophytes' can enclose the whole axial skeleton, which is then called a 'bamboo spine'.[113]

Rheumatoid arthritis

Rhematoid arthritis, a systemic chronic inflammatory disease which involves synovial joints, may affect the facet joints of the lumbar spine, although it is found more frequently in the cervical articulations. The disease does not affect the sacroiliac joints.[114] Those who develop low back pain secondary to rheumatoid arthritis usually have a long-standing history of disease in the joints usually affected by the illness.[115]

Pain stems from the facet joints and therefore its reference does not spread beyond the hips.[116] The symptoms are inflammatory in nature, with pain and stiffness increasing with rest, greater in severity in the morning and improving during activity. Clinical examination reveals limitation of movement in a capsular way and localized tenderness. The diagnosis is based upon the typical history, the clinical appearances of the peripheral joints and the characteristic laboratory findings.

Reiter's syndrome

This is a triad of urethritis, arthritis and conjunctivitis.[117] It is the most common cause of arthritis in young men and primarily affects the joints of the lower extremity. The disease results from the interaction of a specific infection and a genetically predisposed host. Although back pain is a frequent symptom of patients with Reiter's syndrome, pain usually stems from the sacroiliac joint and lesions of the lumbar spine area[118] (see Ch. 43).

Ankylosing hyperostosis

This disease is also known as the vertebral hyperostosis of Forestier.[119] More recently 'diffuse idiopathic skeletal hyperostosis' (DISH) has been suggested, in recognition of the frequent combination of both spinal and extraspinal foci.[120] According to autopsy findings, it seems to be a common entity in the eldery.[121]

Despite the impressive anatomical abnormalities, most patients rarely have significant disability from the illness, the principal complaint being increasing stiffness. Some patients develop a vague and local ache in the entire trunk.[122] Clinical examination shows marked limitation of movement at every spinal joint. Dural and radicular signs are of course absent.

Laboratory parameters are normal and the diagnosis of DISH is a radiographic one. The criteria are a flowing calcification along the anterolateral aspect of four contiguous vertebral bodies, preservation of the normal intervertebral disc height and absence of apophyseal arthrosis or sclerosis.[123]

Treatment is seldom necessary, in that the complaints are rather minor.

Infections

Infections of the spine are rare. However, it is important to remember them as a potential source of backache. An early diagnosis is vital, because the prognosis of infections of the vertebral column is excellent if the disease is recognized early. Infections involving the lumbar spine include vertebral osteomyelitis, intervertebral discitis and herpes zoster, and viral infection of the dorsal root ganglia.

Vertebral osteomyelitis

Pyogenic vertebral osteomyelitis

This occurs as the result of haematogenous spread through the blood stream. Pelvic lesions, such as urinary tract or rectosigmoid infections, preferentially spread to the vertebral column through the venous plexus of Batson.[124] During recent decades the clinical features of vertebral osteomyelitis have changed.[125] Before the antibiotic era, it used to be a disease of children and adolescents, with a rapid evolution and in most cases caused by *Staphylococcus aureus*. Now, the mean age of patients reported with osteomyelitis is 50 years, the onset is insidious and the development is slow.[126] The diagnosis of vertebral osteomyelitis is frequently missed because the patient's symptoms are ascribed to mechanical problems.

Onset of back pain is insidious and it becomes more and more severe. The central ache is usually constant, although it is sometimes increased by exertion. If the lesion becomes more invasive, the patient will find it difficult to stand or sit upright. In a later stage, the infection may extend beyond the bone and can produce a psoas abscess: the patient may then present with an abdominal syndrome or with hip pain.[127] Should the infection drain into the spinal canal, an epidural abscess or meningitis will result.[128]

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In the early stage, the signs may be insignificant, with only slight limitation of movement. As a rule, side flexions are symmetrically limited. Local tenderness at the affected level can be detected during passive forcing of extension in the prone-lying position.[129] Because the first and second lumbar vertebrae are the levels in the axial skeleton most commonly affected,[130] the discovery of pain at the 'forbidden area' during selective examination will alert the examiner.

As the pain worsens, side flexions become more and more limited and muscle spasm limits flexion. Because of severe pain, the patient finds it more and more difficult to remain upright. This could suggest an attack of lumbago but when it comes to further clinical examination, straight leg raising is found to be of full range and completely painless.

Patients with psoas muscle irritation also present with decreased hip motion and a painful weakness of hip flexion. It is important to notice that, in most cases, fever and signs of general illness only appear if an abscess forms.[131]

Radiographic evidence of the disease follows the symptomatic onset by 1–2 months. Bone scintigraphy usually demonstrates abnormalities at an earlier stage of disease, although it should be remembered that false positives and negatives do occur. Computed tomography may also show bony changes before their appearance on routine radiographs.[132]

Treatment includes antibiotics and bed rest. The choice of antibiotic is dependent on the identification of the organism causing the infection.[133] Surgical interventions such as laminectomy, discectomy or vertebral fusion may be indicated in case of neurological compromise or mechanical instability.[134]

Tuberculous vertebral osteomyelitis

This has a clinical course that can be distinguished from pyogenic infections. Before the antibiotic era, it was a disease of children but nowadays patients with spinal tuberculosis have an average age between 40 and 50 years.[135] Alcoholics and drug addicts are at greatest risk of developing the disease.[136]

Tuberculous spondylitis occurs as the result of haematogenous spread from foci in either the lungs or the genitourinary tract. The lesion begins under the bony endplate and, although initially only the vertebral body is affected, in a later stage the infection can spread to disc, soft tissues and spinal cord. Abscess formation is present in about 50% of the cases.[137] The disease is very insidious, and the time elapsing before a diagnosis is made may be as long as 3 years.[138]

Initially there is a vague and localized backache. Later the pain can spread to the buttock. A careful history also reveals the existence of constitutional problems such as anorexia, weight loss, intermittent fever and night sweats.

Limitation of movement in a capsular way and muscle spasm, together with localized tenderness over the involved vertebra, are the main clinical findings in the early stage. Later there is localized bony deformity, resulting from vertebral collapse or neurological abnormalities. Because of the insidious nature of the disease and the insignificant and local symptoms, the onset of paraplegia is sometimes the first manifestation of tuberculous spondylitis.[139]

Because the disease has a slow development, patients with tuberculous spondylitis usually present with identifiable destruction of vertebral bodies. Investigation by CT seems to be the best way of searching for abscess formation.[140]

Treatment consists of antituberculous drugs and immobilization. Surgery must be considered if, despite antituberculous therapy, abscesses progress.

Intervertebral disc space infections

Infections of the intervertebral disc can develop secondary to haematogenous invasion via the blood stream. The primary lesion is usually an infectious endocarditis or a urinary tract infection.[140] The most common cause for spondylodiscitis, however, is a direct complication of disc surgery.[141–143]

The clinical picture is that of an acute lumbago which appears 1 week to 2 months after the discectomy. The pain radiates in a dural manner to the gluteal region, groin and limbs, is exacerbated by movement and is relieved by absolute rest. Dural symptoms are present.

There are dural and articular signs. Fever is rarely present but the erythrocyte sedimentation level is usually elevated.[144] Since the condition closely resembles a discodural conflict, nothing except the previous disc surgery will draw attention to the possibility of an infection. It is therefore wise to consider every case of 'acute lumbago' occurring in the first months after discectomy as a discitis until the reverse is demonstrated.

Radiographs do not show abnormality during the first few weeks. The earliest change is a decrease in the height of the affected intervertebral disc space. Later, relative sclerosis and irregularity at the vertebral endplates will be noted. If a disc infection is suspected, a bone scintillation scan is indicated and identifies an area of increased bone activity in the adjacent vertebrae.[145] During the last decade, MRI has become the radiological method of choice for establishing the diagnosis of spondylodiscitis, in particular with regard to differentiating between cases with and without abscess formation.[146,147]

Treatment includes antibiotics and immobilization.

The childhood form of discitis develops in children between 2 and 6 years old. The patient presents with antalgic posture, muscular defence, unexplained fever and increased erythrocyte sedimentation rate.[148,149]

Herpes zoster

This is a sequela of previous infection with chickenpox. After the termination of the illness, the virus remains dormant in the posterior spinal sensory ganglia. During a period of low host resistance, the virus multiplies, which results in pain and skin lesions.[150] Herpes (shingles) occurs more frequently in the elderly and in patients with impaired immune function.

The patient complains of segmental pain which is burning or shooting in character. Since the pain antedates the appearance of the vesicles by 4–7 days, the early diagnosis can be missed and the patient may be mistakenly regarded as suffering from sciatic pain, although examination of the lumbar spine and the neurological examination of the limbs are normal.[149] Sometimes, however, dysaesthesia in the area of skin supplied by the affected nerve root can be detected. Once the skin lesions develop in a segmental distribution, the diagnosis will

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be obvious. Sometimes patients with herpes zoster may also demonstrate a mild and temporary paresis in the motor nerve that corresponds to the affected level.

Treatment is directed at controlling the pain.

Intraspinal lesions

Pathological processes may affect tissues inside the spinal column (Fig. 5). In the lumbar area they can be extradural or intradural.

Extradural lesions

Extradural neoplasms

These are metastatic lesions that have invaded the intraspinal space from contiguous structures. They usually remain extradural because the dura is resistant to invasion by the neoplasm.

Epidural abscesses and epidural haematomas

These present as acute backache with severe dural signs and symptoms. There is a rapid progression to bilateral sciatica and neurological weakness.[151–154]

Intraspinal synovial cysts

Cysts arising from the synovium-lined facet joints can exert pressure on the nerve root sleeve and cause unilateral sciatica.[155] Cauda equina compression has also been reported.[156] Diagnosis is made by CT or MRI and the treatment is surgical.[157]

Intradural lesions

Arachnoiditis and arachnoid cysts

Inflammatory changes to the arachnoid space (arachnoiditis) develop after myelography with iophendylate, in spinal injuries and as a complication of haemorrhages or meningitis.[158] The patient complains of diffuse and constant back pain, sometimes radiating to both legs. Paraesthesia and dysaesthesia may be present. The prognosis is poor and there is no effective treatment.

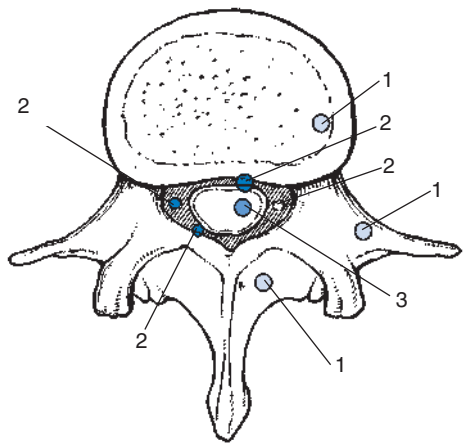

Fig 5 • Intraspinal lesions: 1, extraspinal, osseous lesions; 2, intraspinal, extradural lesions; 3, intraspinal, intradural lesions.

Neurofibromas and meningomas

Meningomas and neurofibromas are slowly growing benign tumours, arising from meninges and spinal nerves. They are rarer at the lumbar spine than at the cervical level. As intradural spinal tumours can compress the dura mater, the nerve roots and the nerve root sleeves, dural and radicular symptoms and signs will be present and the clinical picture usually mimics a herniated disc – between 0.8 and 1.0% of patients presenting with symptoms consistent with disc herniation have intraspinal tumours.[159,160] If the tumour involves the nerve roots of the first or second level, diagnosis is not very difficult because first and second lumbar disc lesions are extremely rare. At the lower lumbar levels, however, neuromas are very difficult to detect, which explains the considerable delay in diagnosis.[161, 162]

The patient presents with back pain and/or root pain. Sometimes the pain increases at night or in the supine position. Dural symptoms, such as pain during coughing and sneezing, are always present – a neuroma is unlikely if a cough does not hurt. Unlike a disc lesion, coughing usually hurts in the limb rather than in the back.

Clinical examination shows limitation of spinal movements and straight leg raising. Motor and sensory deficit together with reflex disturbances can be present. Differential diagnosis of neuromas and lumbar disc lesions is almost impossible if only the actual symptoms and signs are considered. However, when the duration and evolution of the symptoms are analysed, certain features should arouse suspicion. The evolution of radicular pain in neuroma is that of a slowly progressive lesion. This is completely different from the evolution in discoradicular interactions, in which the sciatica becomes rapidly worse and reaches a peak within 1–2 months; severe symptoms may then persist for a certain length of time, which seldom exceeds 2 months. Although primary posterolateral protrusion in young patients can have a slower and more gradual onset, the evolution usually does not take more than 6 months. Hence any case of root pain that is getting worse at the end of 18 months is suspect (Cyriax:[3] p. 293). Also, increasing backache after root pain has set in must alert the examiner. The range of straight leg raising is not much help in the differential diagnosis: if the lesion lies upwards in the canal, straight leg raising may be normal, but if the neuroma lies near the intervertebral foramen it will be markedly and often bilaterally restricted.[163] Palsies affecting two non-adjacent roots may suggest a neuroma, and bilateral sciatica should also alert the clinician to the possibility of a cauda equina tumour (Fig. 6).[165,166] Late manifestations are marked motor deficit, with drop foot or widespread weakness and muscle atrophy. Bladder dysfunction also tends to be a late manifestation, although urinary and rectal incontinence sometimes appear early in the evolution.[167,168]

Sometimes the diagnosis of a neuroma is suggested by the unusual response to a sacral, epidural anaesthesia. Although the introduction of the fluid creates considerable and sometimes

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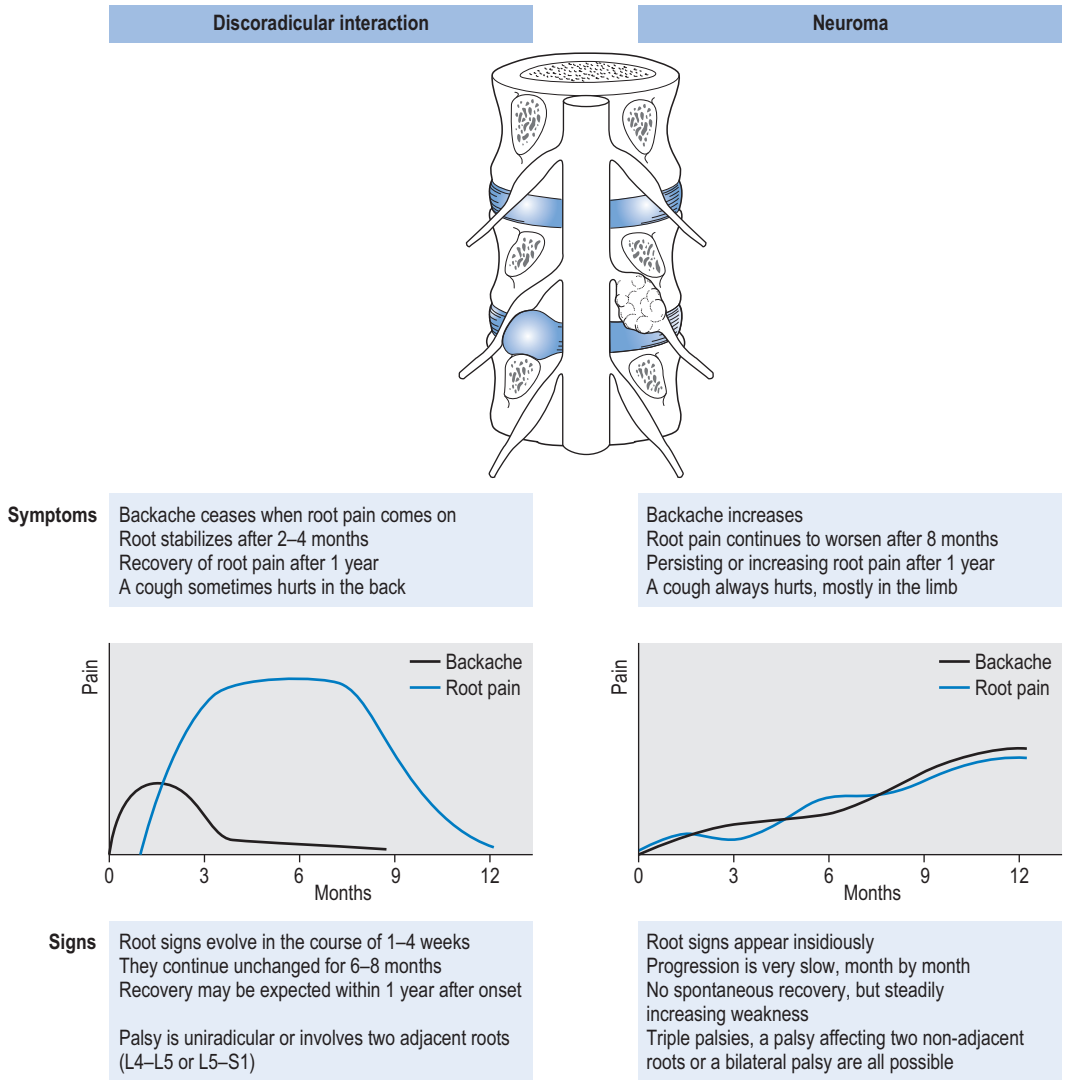

Fig 6 • Differential diagnosis of discoradicular conflict and neuroma in the lower lumbar area.[164]

unbearable root pain, it has no immediate effect on the symptoms or on the range of straight leg raising because the fluid cannot touch the neuroma, which is intradural. Had the root pain been caused by an inflammation of the dural sheath, the injection would have created some anaesthesia immediately afterwards. No improvement of straight leg raising after the epidural injection thus strongly suggests the possibility of a neuroma and the patient should be referred for further investigation.

MRI is the most useful method for the differentiation of spinal tumour from lumbar disc herniation.[167] In MRI imaging, schwannomas commonly appear isointense on T1-weighted images and markedly hyperintense on T2-weighted images.[169] Computed tomography is not the technique of choice because a lesion higher up in the spinal canal will always be missed. In addition, as disc protrusions are often asymptomatic,[170] the clinical picture may be wrongly ascribed to such a cause, surgical opinion sought and an exploration of the disc undertaken.[171] Craig *et al*[172] summarized this in 1952 when they stated: 'It is not unusual at this clinic to remove a spinal cord tumor from a patient who has had a previous operation for protruding disc without relief of symptoms'.

Treatment consists of laminectomy and total excision of the tumour, which gives excellent results, provided there is not too much neurological dysfunction.

Differential diagnosis

The following conditions may resemble a neuroma.

Adherent nerve root

There are rare cases of root adherence to the posterior aspect of the intervertebral joint, which occur after the herniation has shrivelled away as the result of the natural course of the disc lesion. The patient's sciatica slowly subsides and after 2 years the pain may have gone. However, the patient continues to find that they cannot bend forwards, and examination shows about 45° limitation of straight leg raising on the affected side.

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Attempted trunk flexion causes pain in the back and the leg. Apart from that, there are no other complaints and the sciatic pain is slight. The pain also does not increase; there is no appreciable neurological deficit and certainly not an increasing one. The condition thus presents as sciatica, usually in a young man in whom the symptoms have abated but the signs continue. As in neuroma epidural local anaesthesia does not alter the range of straight leg raising.

Increasing protrusion on an atrophied root

The patient has suffered from severe sciatica some years ago. There is sudden loss of pain, together with considerable weakness of one or two roots, which indicates that root atrophy has taken place. There is no recovery from the palsy; however, one day, without pain, the patient notices increased weakness and numbness. This can be explained by a further protrusion of disc substance at the original level. Because it impinges against the atrophied nerve root there is no pain, but there is further loss of sensory and motor conduction.

A second disc protrusion

The patient is suffering from sciatica, which is improving (as expected) after some months. After a while, a new disc lesion develops at an adjacent level, impinging against an adjacent or a contralateral root. Of course, weakness extending to another level after some months' sciatica is suspect, but the differential diagnosis will easily be made on a myelogram.

Neuralgic amyotrophy (Cyriax:[3] p. 300)

This uncommon parenchymatous disorder of the peripheral nerves usually afffects three to four consecutive roots at the same side. The origin is unknown but the disease has a spontaneous and complete recovery within a year.

The patient, usually a man aged between 50 and 70 years, experiences a sudden and severe ache in one leg. The pain is considerable and not altered by position or motion. There is no pain on coughing or sneezing. After about 3 months the symptoms slowly ease, and 6 months after the onset they have disappeared.

Examination shows a full range of movement and full and painless straight leg raising. Considerable muscle weakness is detected where the muscles are innervated by two to three different roots. An important differential diagnostic finding is that the palsy is maximal from the first onset and does not increase or spread to the other limb. There is no sensory loss.

There is no treatment for this condition. As the pain ceases, so the muscles gradually recover. The disorder completely resolves 1 year after its onset.

Pain referred to the back

It should be remembered that back pain occurs not only due to lesions of bones, discs and ligaments, but also as referred from intra-abdominal or pelvic lesions. The prominent feature will then be the complete absence of articular, dural and radicular signs during routine clinical examination. Lesions of the aorta and genitourinary or gastrointestinal diseases should be suspected in such circumstances.

Lesions of the aorta

An arterial aneurysm is a localized or diffuse enlargement of an artery. In the abdominal aorta it occurs most commonly in men over the age of 50 years.[173] The majority of patients with a stable abdominal aneurysm are asymptomatic, and pain is only associated with enlargement or rupture. Most frequently the pain is abdominal, dull and steady. Sometimes the main symptom is severe and increasing backache, accompanied by L3 pain in the left leg.[174] Increasing back pain and L3 pain in an elderly patient with a negative functional examination should therefore always arouse suspicion. The patient should be referred immediately for ultrasonography and CT of the abdomen. These methods are non-invasive and very reliable in identifying the location of the lesion.[175]

Acute occlusion of the abdominal aorta presents as acute and severe low back pain and abdominal pain together with acute claudication of the lower extremities.[176]

Genitourinary diseases

The colic and excruciating pain caused by a kidney or ureteral stone is well known. The pain is sudden, sharp and has a spasmodic character. It is upper lumbar and radiates to the lateral flank and along the course of the ureter into the ipsilateral flank and along the course of the ureter into the ispilateral testicle (men) or labium (women).

Renal infarction causes a sudden and sharp pain in the costovertebral angle. Renal cancers may present as increasing upper lumbar and flank pain, which is constant and dull.[177] Clinical examination reveals a limitation of the side flexion away from the painful side as the only finding. This warning sign should always prompt the physician to refer the patient for further internal examination.

Diseases of the bladder and the prostate rarely cause lumbosacral pain. Lesions of the testis, however, often cause backache and back pain may be a presenting symptom in 10–21% of patients with testicular carcinoma.[178] Patients may even present with back pain in the absence of any testicular symptoms.[179] In testicular carcinoma, the pain is dull and persistent, localized over the lumbosacral and paravertebral region. Functional lumbar examination is completely negative, but examination of the testicles reveals a testicular mass together with diffuse induration.

In women, referred pain from the genital organs (uterus, fallopian tubes or ovaries) is possible, although backache from gynaecological causes should not be overestimated. For instance malposition of the uterus in times past was regarded as a main source of backache in women, but this certainly does not cause any back trouble.[180] In contrast, endometriosis, a disease associated with the presence of endometrial tissue outside the uterine cavity, is often associated with intermittent back pain. The pain is lower lumbar and often irradiates to the buttocks or thighs. The symptoms characteristically increase at the time of menstruation and persist throughout the entire period of bleeding. Other symptoms associated with the disease are dyspareunia, infertility and menorrhagia.[181]

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It is a well-known fact that women complain more often of low back pain during pregnancy. The incidence reported varies between 24 and 49%.[182–185] The increase in backache during pregnancy is not very well understood. Hormonally induced laxity probably allows increased biomechanical stress on the pelvis and spine, which results in more postural ligamentous pain.[181,186] But there are several studies indicating that pregnancy is also a risk factor for lumbar disc herniations.[182,183]

Pelvic inflammatory disease, an acute or chronic infection of the fallopian tubes, is also associated with pain at the lower back, radiating into the buttocks. The chief clinical symptoms, however, will be lower abdominal and pelvic pain, with a feeling of pelvic pressure.[183]

Gastrointestinal diseases

Diseases of the pancreas, stomach, duodenum and colon may be associated with low back pain. The gallbladder has a T5–T9 origin and pain is therefore felt in the thoracic region.

Cyriax described cases of gastric ulcer adherent to the lumbar spine. The back symptoms are connected both with eating and with posture. The pain is upper lumbar, with radiation into one or other iliac fossa. It is not brought on by exertion but appears during eating. The only finding during clinical examination is central discomfort during extension. The combination of pain influenced by posture and abdominal visceral function brings the diagnosis to mind, and the diagnosis is confirmed by gastroscopy.

A peptic ulceration of the posterior duodenal wall can also cause low back pain. Again the pain is localized to the L2–L3 lumbar level, but appears 1–3 hours after the meal, and is relieved by further intake of food. The pain has no relation to physical activity.[187] Sometimes there is tenderness at the upper lumbar ('forbidden') area.

Pain from chronic pancreatitis or carcinoma of the pancreas is felt in the epigastrium and is referred to the upper lumbar area.[188] Disease processes that affect the head of the pancreas cause pain to the right of the spine, while lesions of the tail are felt at the left side.

Lesions of the colon – both diverticulitis and carcinoma – can be associated with low back and flank pain.[189] Patients with a rectal carcinoma often complain of sacral pain. Invasion of the nerve roots at the front of the sacrum gives rise to gross weakness of the muscles in both legs. As the dural sleeve is not affected here, the lesion does not provoke pain and the weakness often remains silent. Similar back pain may be the first sign of recurrence after apparently successful excision of a rectal cancer.

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Applied anatomy of the sacroiliac joint

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The joint

The sacroiliac joint possesses all the characteristics of a true joint: a joint cavity containing synovial fluid,[1] adjacent bones having ligamentous connections, cartilaginous surfaces which permit movements and an outer fibrous joint capsule with an inner synovial lining.[2–5]

The joint most commonly links the posterosuperior part of the medial aspect of the iliac bones with the first, second and the upper part of the third segment of the sacrum (Fig. 1). A transverse section shows that the joint is situated rather anteriorly (Fig. 2). The clinical consequence of this is that it is not possible to elicit tenderness by digital pressure at the joint.

The sacrum can be regarded as a wedge that fits vertically between the two iliac bones. The sacrum also fits between these bones in the transversal plane.[6]

The dorsal width of the sacrum exceeds the ventral width only in the middle portion of the joint (see Fig. 4c). At the lower and upper parts, the wedge shape is quite reversed (see Fig. 2). Several investigators, however, have demonstrated a high degree of variability in the plane of sacroiliac joints, in both the vertical and transverse directions.[1,7,8]

The articular surfaces are ear- or C-shaped and exhibit irregular ridges and depressions that fit into each other.[9,10] The anatomical configuration, together with strong ligaments, make the joint very stable. These features are more pronounced in men than in women, suggesting the likelihood of increased mobility in the latter. Some authors also associate this increased mobility with the position of the centre of gravity, which in women lies dorsal to the hip joint and not in line with the axis of support (Fig. 3). This exerts a strong rotational force in the sacroiliac joint.[11,12]

Cartilage covers the joint surfaces. It is thicker and smoother at the sacral than at the iliac surface.[5]

Joint capsule and ligaments

A tight articular capsule is attached close to the margins of the articular surfaces of the ilium and sacrum.

Powerful ligaments support the joint and sharply limit movements (Fig. 4). These ligaments can be divided into the massive interosseous sacroiliac ligament, the posterior and anterior ligaments, and three accessory ligaments – the sacrotuberous, sacrospinous and iliolumbar ligaments.

The interosseous ligament fills the irregular space between sacrum and ilium at the level of S1 and S2, immediately behind and above the joint. The shortest and strongest part of this ligament is in the frontal, horizontal axis of movement of the sacrum and it is therefore also known as the axial ligament.

The strong posterior sacroiliac ligament consists of several fascicles of different lengths which overlie the interosseus ligament and pass in an oblique direction from the lateral crests of the sacrum to the posterior superior iliac spine and the posterior end of the inner lip of the iliac crest. Those fibres from the third and fourth sacral segments are longer and constitute the long posterior sacroiliac ligaments.[13]

The thin anterior sacroiliac ligament consists of two bands which reinforce the anterior and inferior parts of the fibrous capsule and also pass across the joint obliquely from sacrum to ilium.

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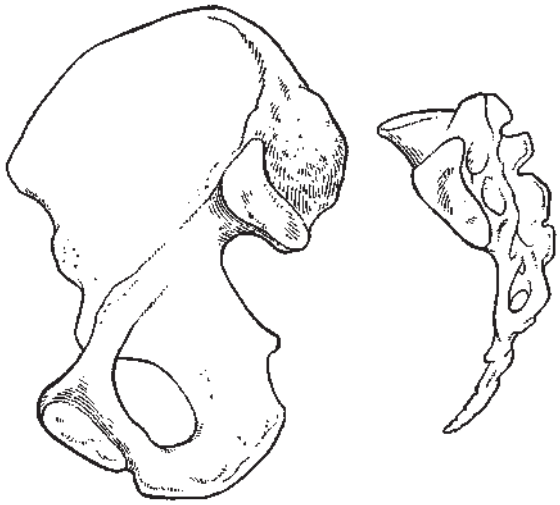

Fig 1 • Articular surfaces of the sacroiliac joint.

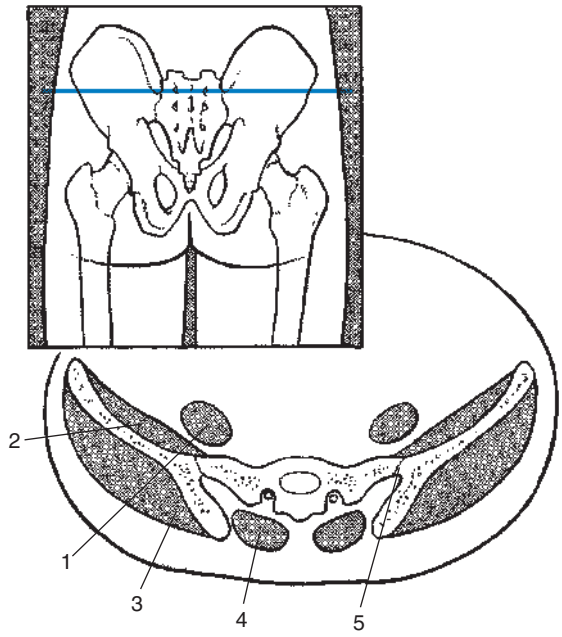

Fig 2 • Transverse section at the level of the sacroiliac joint: 1, psoas; 2, iliacus; 3, gluteus; 4, erector spinae; 5, sacroiliac joint.

The sacrotuberous ligament is attached to the posterior iliac spines, the lower part of the sacrum and the upper part of the coccyx. Its fibres run obliquely downwards and laterally to insert at the medial margin of the ischial tuberosity. The gluteus maximus, the thoracolumbar fascia, the multifidus muscle and, in some individuals (especially females), long head of biceps femoris are partly attached to this ligament.

The thin *triangular sacrospinous ligament* attaches to the lateral margins of sacrum and coccyx in front of the attachments of the sacrotuberous ligament. The fibres run in a lateral, caudal and anterior direction to insert at the spine of the ilium.

The iliolumbar ligament takes origin at the lower part of the transverse processes of the fourth and particularly the fifth lumbar vertebrae. Its fibres radiate to the anterior part of the upper surface of the sacrum as well as to the crest of the ilium.

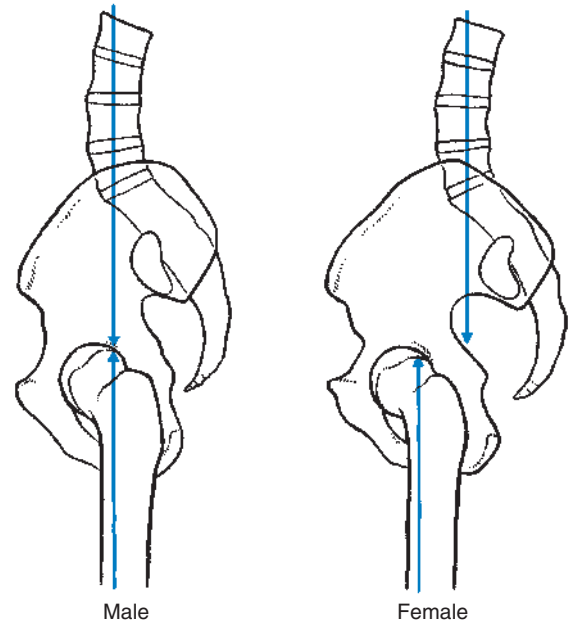

Fig 3 • Differences between the position of the centre of gravity in the male and female. The position of the centre of gravity lies in line with the axis of the support (male), or lies dorsally (female).

Muscles

The sacroiliac joint is surrounded by some of the largest and most powerful muscles of the body, i.e. the erector spinae, psoas, quadratus lumborum, piriformis, abdominal obliques, gluteal and hamstrings. However, there are no muscles designed to act on the sacroiliac joint to produce active, physiological movements. All muscles that cross the joint are designed to act on the hip or the lumbar spine.[1,2,14–16] None of the small movements of the sacroiliac joint is produced by active movements of the sacrum. The movements are indirectly imposed by gravity and muscles acting on trunk and lower limbs.

Whether these muscles could contribute to active stability of the joint (force closure) is still open to debate. Some authors suggest that the latissimus dorsi and the contralateral gluteus maximus are coupled via the thoracolumbar fascia. The forces of these two synergists cross the sacroiliac joints perpendicularly and it is hypothesised that this could stabilize the joint indirectly.[17–20]

Innervation

No authoritative anatomical studies exist today but most authors report a posterior innervation from the lateral branches of the posterior rami L4–S3 and an anterior innervation from the L2–L3 segments.[21]

The fact that capsule and ligaments contain nociceptors suggests that the sacroiliac joint is a possible source of low back pain and also plays a role in somatic referred pain.[22]

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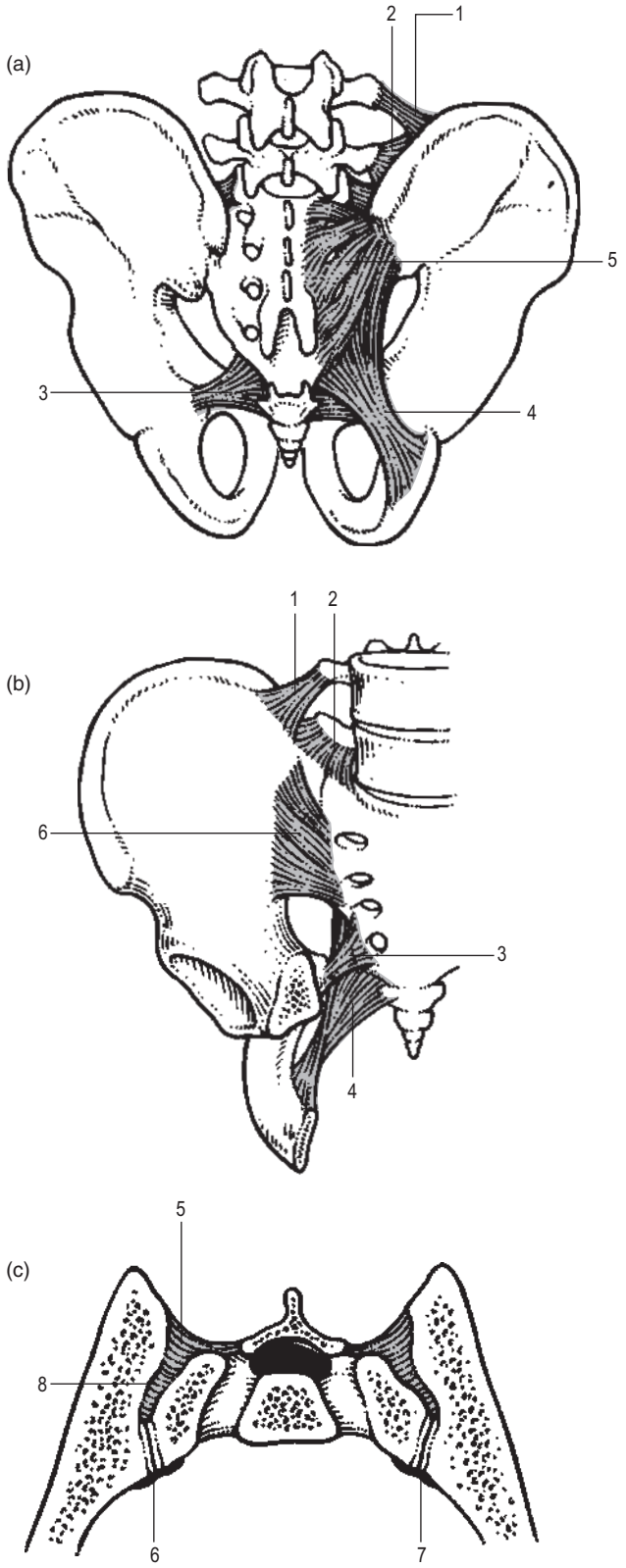

Fig 4 • (a) Posterior and (b) anterior aspect. 1, 2, superior and inferior bands of iliolumbar ligaments; 3, sacrospinous ligament; 4, sacrotuberous ligament; 5, posterior sacroiliac ligaments; 6, anterior sacroiliac ligaments. (c) Transection through the sacroiliac joint: 7, sacroiliac joint; 8, interosseous ligament.

The wide range of segmental innervation probably accounts for the variable referred pain patterns seen in sacroiliac joint lesions,[1,23] although pain is localized most commonly at the buttock and the posterior thigh.

Biomechanical aspects

In essence the sacroiliac joint is a stress-relieving joint that serves as a buffer between the lumbosacral and hip joint.[10] The sacroiliac joint transmits forces from the vertebral column sideways into the pelvis and then to the lower limbs. Conversely, forces from the lower limbs can be transmitted through pelvis and sacrum to the vertebral column. Despite its size, the sacroiliac joint cannot be considered the same as any other major joint of the body: its ranges of movement (nutation–counternutation, more complex movements and pelvic torsion) are very small and are not controlled by active muscular contractions.

The precise nature of motion in the normal joint is still unclear and joints such as the sacroiliac joint with extensive ridges and depressions can be expected to have a very limited amount of mobility. In Kapandji's words: 'There is more a tendency to movement than actual movement since the extremely powerful ligaments preclude any movement from the start'.[6]

Both *in vivo* and *in vitro* kinematic studies have demonstrated various types of minor motion in the sacroiliac joints, such as gliding, rotation, tilting, nodding and translation.[24–26] Mobility is always increased temporarily at the end of pregnancy and for up to 3 months afterwards.[15,27,28] It decreases in men at between 40 and 50 years, and after the age of 50 in women.[29]

Nutation–counternutation

Most authors[1,15,30–33] accept that there is a small rotatory movement about a frontal, horizontal axis at the level of S1–S2, constituted by the shortest and strongest part of the interosseous ('axial') ligament.

During anterior rotation, called *nutation*, the promontory of the sacrum moves inferiorly and anteriorly while the apex of the sacrum and the coccyx move posteriorly (Fig. 5a). As a result of the converging planes of the sacral joint surfaces, the iliac bones are approximated, whereas the ischial tuberosities move apart (Fig. 6). This movement also results in slight distraction of the symphysis pubis. Nutation is limited by the tension of the anterior sacroiliac ligament, the interosseus ligament, the short upper part of the posterior sacroiliac ligament and the sacrospinous and sacrotuberous ligaments.

During *counternutation* (posterior rotation), the promontory of the sacrum moves superiorly and posteriorly while the apex and the coccyx move anteriorly (Fig. 5b). As a result, the iliac bones move apart, whereas the ischial tuberosities are approximated. Counternutation is limited by the tension of the posterior sacroiliac ligaments, especially the long part of these ligaments.[6]

Weisl[30] showed that the length of the anterior–posterior diameter of the superior aperture of the pelvis changed by

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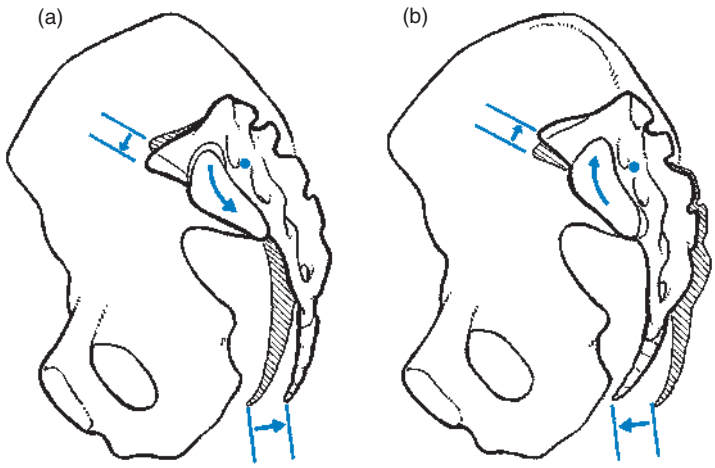

Fig 5 • Movement of nutation (a), counternutation (b).

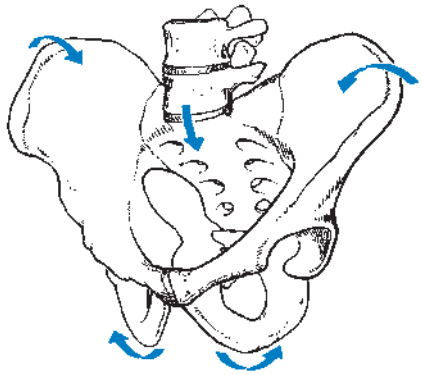

Fig 6 • Nutation of the sacrum. The iliac bones approximate, whereas the ischial bones move apart.

5–6 mm during combined nutation–counternutation. This mobility pattern has been confirmed in women.[34]

More complex movements

Radiological stereophotogrammetrical analysis[35] has identified very small movements in the sacroiliac joints. In changing position from supine to standing or sitting and between standing and hyperextension of the leg, the movements proved even to be three-dimensional, although again most constant around the frontal, horizontal axis. The mean values obtained were: 2.5°, translation 0.7 mm.

In a recent study of sacroiliac kinematics for reciprocal straddle positions in relatively young subjects, it was established that the pelvis showed a tendency to move in three directions – flexion–extension, side flexion and rotation.[36] Another study, using rigidly fixed external devices in 21 volunteers, examined the range of motion of the sacroiliac joint during maximal flexion and extension of the trunk and during standing on one leg. The conclusion was that there was a small range of movement (less than 1°) but with an irregular direction – during flexion of the trunk, the sacrum was just as likely to flex as to extend around a transverse axis. Consequently, in a sample of individuals and for one particular test, the true range of motion of the sacroiliac joint is 0°.[37]

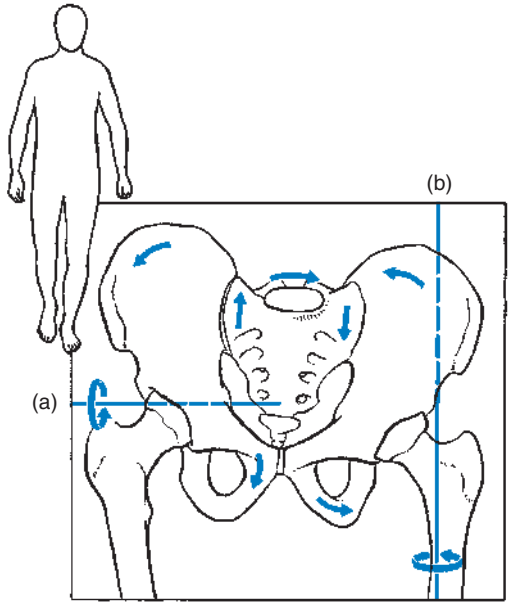

Fig 7 • Complex movements of the iliac bones and sacrum during walking (from Cramer A[38], with permission). (a) Rotation of the iliac bone at the non-weight-bearing side, around a frontal, horizontal axis. (b) Rotation of the iliac bone at the weight-bearing side around a vertical axis.

Torsion of the pelvis

Cramer discusses more complex movements.[38] During walking (Fig. 7), when a person lifts one leg and puts the entire body weight on the other leg, the load of the trunk moves the sacrum forward and caudally but also causes forward rotation. The ilium on the weight-bearing side is pushed cranially and rotates dorsally and externally. On the non-weight-bearing side, the movement of the sacrum is dorsal and cranial, with posterior rotation; on this side the ilium slides caudally and rotates forward in relation to the sacrum. Overall, the movements of the iliac are torsional. This mobility of the sacroiliac joints incontrovertibly relieves part of the strain on the lumbar spine and is therefore clearly beneficial.

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It has been conjectured that jumping from a height or stepping onto a high stool increases these complex movements and perhaps even subluxates the joint. Vleeming *et al*[39,40] have stated that:

> Abnormal loading conditions could theoretically force the sacroiliac joint in a new position where ridges and depressions are no longer complementary. Such an abnormal joint position could be regarded as a blocked joint. Being part of the complex kinematic chain between legs and spine, even a small displacement of the sacrum or ilium could be responsible for abnormal displacement or stress of the lumbar vertebrae.

In the literature, however, they have found (as yet) no definite proof of this phenomenon. In the radiological stereophotogrammetrical analysis mentioned earlier, Sturesson *et al*[35] also found that the mobility of symptomatic joints had a mean value equal to that of asymptomatic joints.

Mobility normally decreases with age, which is regularly shown on the radiograph in elderly patients with osteophytes at the lower margin of the joints or even bony ankylosis. In middle age the joint has already reduced in width and becomes obliterated by fibrous and fibrocartilagenous adhesions.[4] In ankylosing spondylitis complete fusion is found even in young adults.

Conclusion

Movements at the sacroiliac joint are small in extent. At the end of pregnancy (and for some months afterwards), mobility is increased temporarily due to hormonal influences which favour the process of childbirth. Rotatory movements of the sacrum about a frontal and horizontal axis (nutation and counternutation) seem to occur but strong sacroiliac ligaments, as well as ridges and depressions at the articular surfaces, restrict these movements. Radiological stereophotogrammetrical analysis shows movements to be three-dimensional, although rotation about a frontal and horizontal axis is most significant. Mobility normally decreases with age. Subluxation and dysfunction are as yet unproven phenomena.

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Applied anatomy of the hip and buttock

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The hip joint

The hip joint is a ball-and-socket joint, formed by the femoral head and the acetabulum (Fig. 1, see Standring, Fig. 80.15). The articular surfaces are spherical with a marked congruity; this limits the range of movement but contributes to the considerable stability of the joint. In the anatomical position, the anterior/superior part of the femoral head is not covered by the acetabulum. This is because the axes of the femoral head and of the acetabulum are not in line with each other. The axis of the femoral head points superiorly, medially and anteriorly, while the axis of the acetabulum is directed inferiorly, laterally and anteriorly.

The cup-shaped *acetabulum* is a little below the middle third of the inguinal ligament. The acetabular articular surface is an incomplete cartilaginous ring, thickest and broadest above, where the pressure of body weight falls in the erect posture, narrowest in the pubic region. The rough lower part of the cup, the *acetabular notch*, is not covered by cartilage. The centre of the cup, the *acetabular fossa*, is also devoid of cartilage but contains fibroelastic fat. The *acetabular labrum*, a fibrocartilaginous ring attached to the acetabular rim, deepens the cup and enlarges the contact area with the femoral head (see Standring, Fig. 81.5). The part of the labrum that bridges the acetabular notch does not have cartilage cells and is called the *transverse acetabular ligament*. If forms a foramen through which vessels and nerves may enter the joint. The acetabular labrum is triangular in section. The base is attached to the acetabular rim and the apex is free.

The *femoral head* is ovoid or spheroid but not completely congruent with the reciprocal acetabulum. It is covered by articular cartilage except for a rough pit for the *ligamentum teres*, a flattened fibrous band, embedded in adipose tissue and lined by the synovial membrane (Figs 2, 3). The ligament connects the central part of the femoral head with the acetabular notch and its transverse acetabular ligament. The ligament is extra-articular and contains a tiny branch of the obturator artery partly responsible for the vascular supply of the femoral head.

The femoral head and neck also receive arterial supply from the capsular vessels, arising from the medial and lateral circumflex arteries (see Fig. 3).

Capsule and ligaments

The capsule is a cylindrical sleeve, running from the acetabular rim to the base of the femoral neck. It is supported by powerful ligaments.

Anteriorly these are two ligaments: the fan-shaped iliofemoral ligament of Bertin situated craniolaterally; and the

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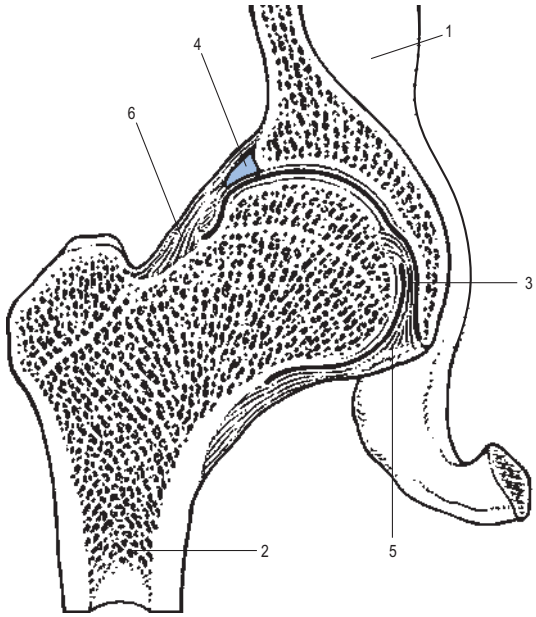

Fig 1 • The hip joint: 1, ilium; 2, femur; 3, ligamentum teres; 4, labrum acetabulare; 5, transverse acetabular ligament; 6, joint capsule.

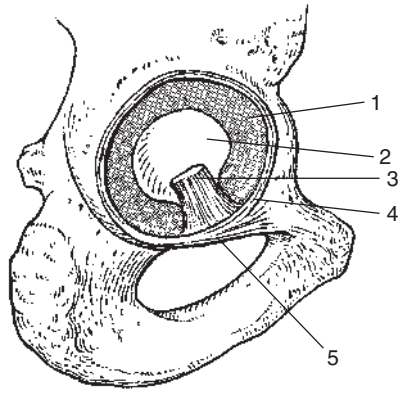

Fig 2 • The acetabulum: 1, articular cartilage; 2, acetabular fossa; 3, ligamentum teres; 4, labrum acetabulare; 5, transverse acetabular ligament.

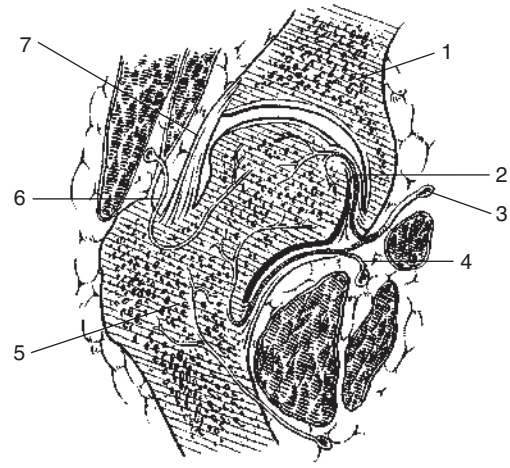

Fig 3 • Coronal section through the hip: 1, ilium; 2, ligamentum teres; 3, branch of obturator artery; 4, branch of medial circumflex artery; 5, femur; 6, branch of lateral circumflex artery; 7, joint capsule strengthened by the iliofemoral ligament (lateral part).

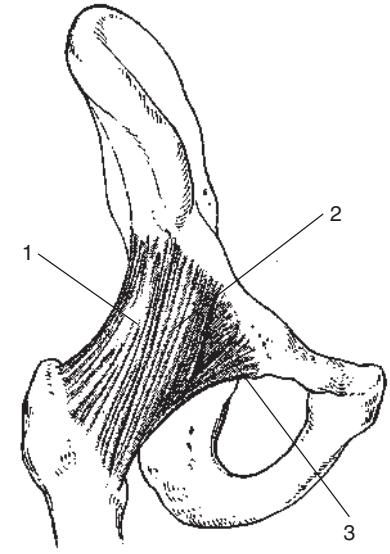

Fig 4 • Anterior ligaments: 1, iliofemoral, lateral part; 2, iliofemoral, medial part; 3, pubofemoral.

pubofemoral ligament, in a more caudomedial orientation. Together they resemble the letter Z (Fig. 4). Posteriorly the capsule is strengthened by the ischiofemoral ligament (see Standring, Fig. 81.3). These three ligaments are coiled round the femoral neck. Extension 'winds up' and tautens the ligaments, thus stabilizing the joint passively (Fig. 5a); flexion slackens them (Fig. 5b). Lateral rotation tightens the iliofemoral ligament and also the pubofemoral ligament. Medial rotation tightens the ischiofemoral ligament. Abduction tightens the pubofemoral and the ischiofemoral ligaments. Adduction tightens the lateral part of the iliofemoral ligament.

The ligamentum teres plays only a minor role in the control of hip movements. Adduction from a semi-flexed position is the only movement where this ligament is under tension.

Muscles

The hip joint is surrounded by a large number of muscles. According to their function these are divided into six groups: (1) flexors, (2) extensors, (3) abductors, (4) adductors, (5) lateral rotators, (6) medial rotators. In this chapter the anatomical and kinesiological aspects of particular importance in orthopaedic medicine are discussed.

Flexor muscles

The flexor muscles of the hip joint (Table 1) are anterior to the axis of flexion and extension.

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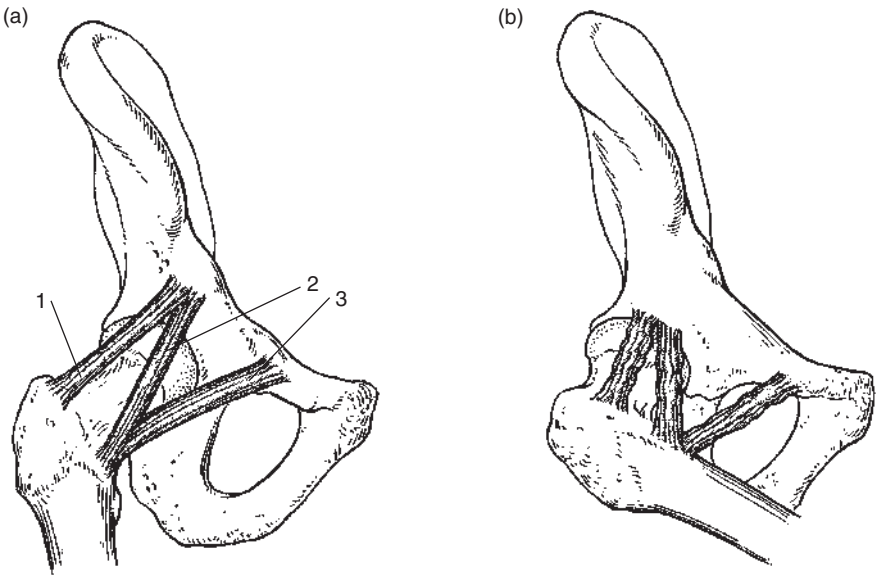

Fig 5 • Lateral view of the ligaments in extension (a) and flexion (b): 1, iliofemoral, lateral part; 2, iliofemoral, medial part; 3, pubofemoral.

Table 1 Flexor muscles

Muscle	Innervation	
	Peripheral nerve	Spinal
Iliopsoas*	Femoral and lumbar plexus	L2, L3
Sartorius*	Femoral	L2, L3
Rectus femoris*	Femoral	L3
Tensor fasciae latae	Superior gluteal	L5
Pectineus	Femoral and obturator	L2, L3
Adductor longus*	Obturator	L2, L3
Adductor brevis	Obturator	L2, L3
Adductor magnus	Obturator and sciatic	L3, L4
Gluteus medius	Superior gluteal	L5
Gluteus minimus	Superior gluteal	L5

*Clinically important muscles.

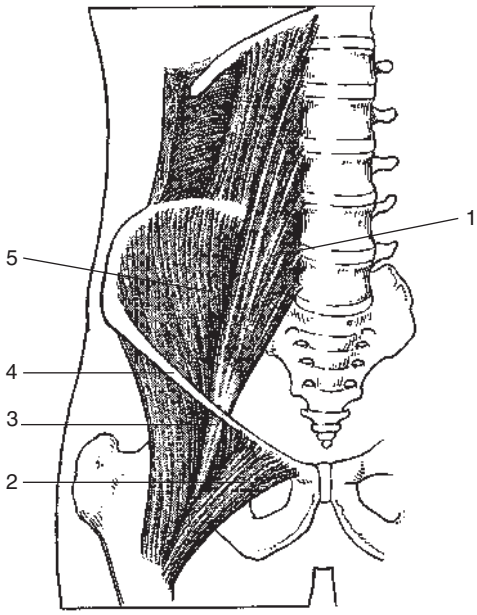

Fig 6 • The iliopsoas and associated structures: 1, psoas; 2, pectineus; 3, iliopsoas tendon; 4, inguinal ligament; 5, iliacus.

The iliopsoas is the most powerful of the flexors (Fig. 6). It originates at the lumbar vertebrae and the corresponding intervertebral discs of the last thoracic and all the lumbar vertebrae, the superior two-thirds of the bony iliac fossa and the iliolumbar and ventral sacroiliac ligaments. The insertion is to the lesser trochanter. Although its main function is flexion, it is also a weak adductor and lateral rotator. The distal part of the muscle is palpable just deep to the inguinal ligament, where it lies bordered by the sartorius muscle laterally and the femoral artery medially (Fig. 7).

The sartorius is mainly a flexor of the hip, originating at the anterior superior iliac spine and inserting at the proximal part of the medial surface of the tibia (see Fig. 7). Consequently the muscle acts on two joints, with the accessory function of lateral rotation and abduction of the hip as well as flexion and medial rotation of the knee. At the surface, the muscle divides the anterior aspect of the thigh into a medial and a lateral femoral triangle. During active flexion, abduction and lateral rotation at the hip and 90° flexion at the knee, the muscle becomes prominent and is easily palpable.

The rectus femoris combines movements of flexion at the hip and extension at the knee. Its origin is at the anterior inferior iliac spine, a groove above the acetabulum and the fibrous capsule of the hip joint and inserts into the common quadriceps tendon at the proximal border of the patella (see Fig. 7). The origin can be palpated only in a sitting position

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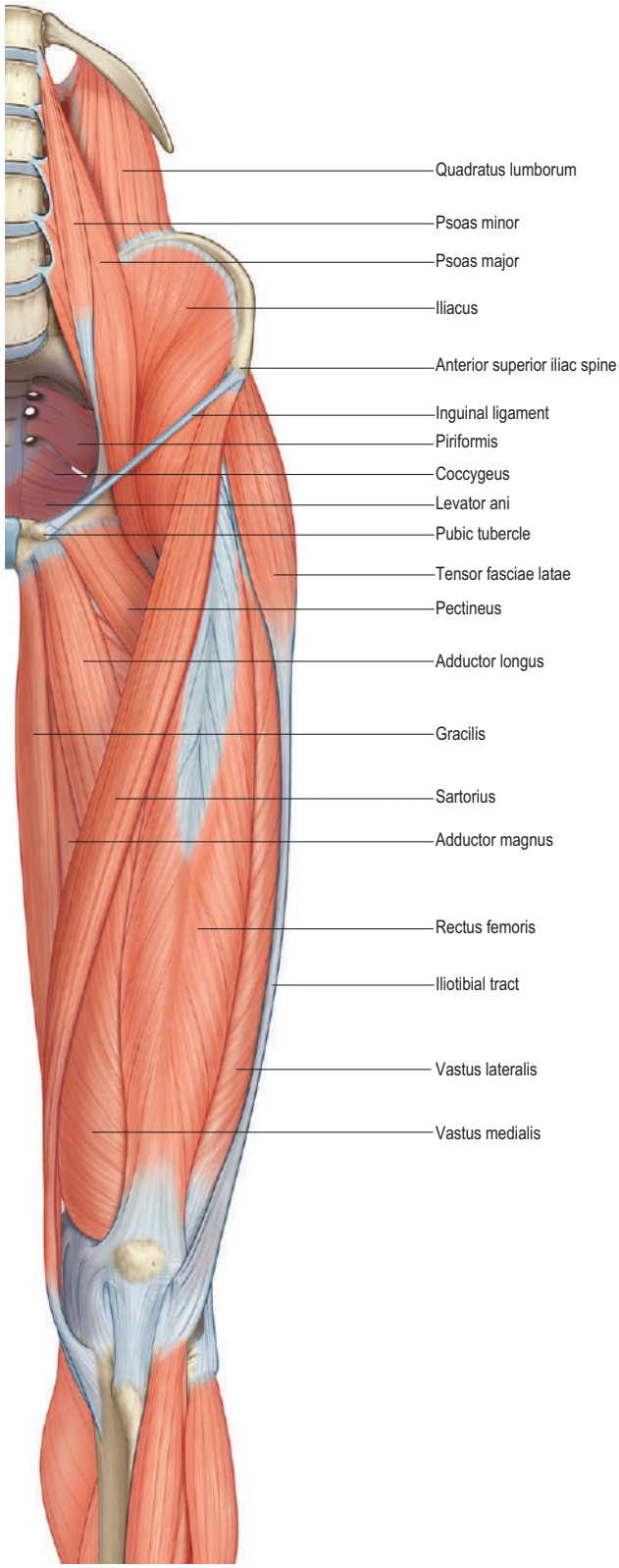

Fig 7 • Anterior view of the hip muscles. From Standring, Gray's Anatomy, 40th edn. Churchill Livingstone/Elsevier, Philadelphia, 2009 with permission.

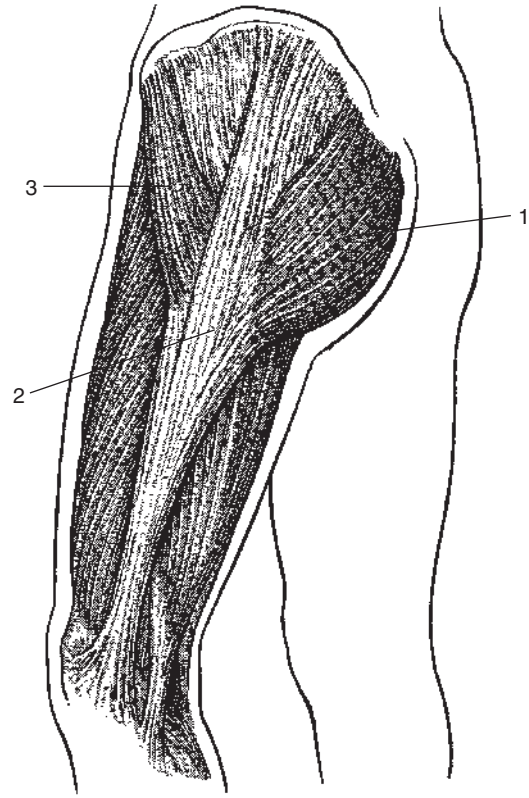

Fig 8 • Lateral view of the hip muscles: 1, gluteus maximus; 2, iliotibial tract; 3, tensor fasciae latae.

because of tension in the overlying structures. The tendon and muscle belly are bordered medially by the sartorius muscle, and laterally by the tensor fasciae latae and the vastus lateralis, the largest part of the quadriceps.

The tensor fasciae latae (see Fig. 7) originates at the outer surface of the anterior superior iliac spine, and inserts into the proximal part of the iliotibial tract – a strong band which thickens the fascia lata at its lateral aspect. Thus the course of the tensor is dorsal and distal. Acting through the iliotibial tract the muscle extends and rotates the knee laterally. It may also assist in flexion, abduction and medial rotation of the hip. In the erect posture, it helps to steady the pelvis on the head of the femur (Fig. 8). The muscle can be palpated easily during resisted flexion and abduction of the hip with the knee extended.

A number of other muscles are also active during flexion of the hip joint but only via their accessory function. These are the pectineus, adductor longus and brevis, and the most anterior fibres of the adductor magnus and the glutei (medius and minimus).

Extensor muscles

These (Table 2) are posterior to the axis of flexion and extension of the hip.

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Table 2 Extensor muscles

Muscle	Innervation	
	Peripheral nerve	Spinal
Gluteus maximus*	Inferior gluteal	Mainly S1
Semimembranosus	Sciatic	S1, S2
Semitendinosus	Sciatic	S1, S2
Biceps femoris	Sciatic	S1, S2

*Clinically important muscle.

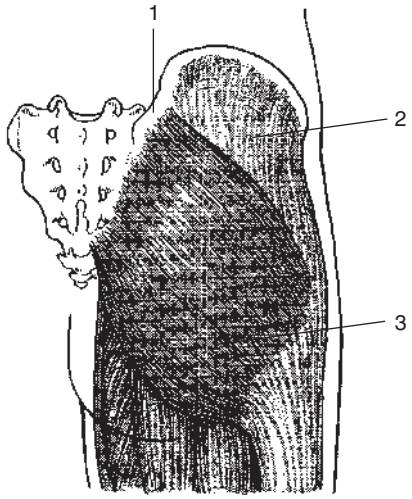

Fig 9 • 1, Posterior superior iliac spine; 2, gluteus medius; 3, gluteus maximus.

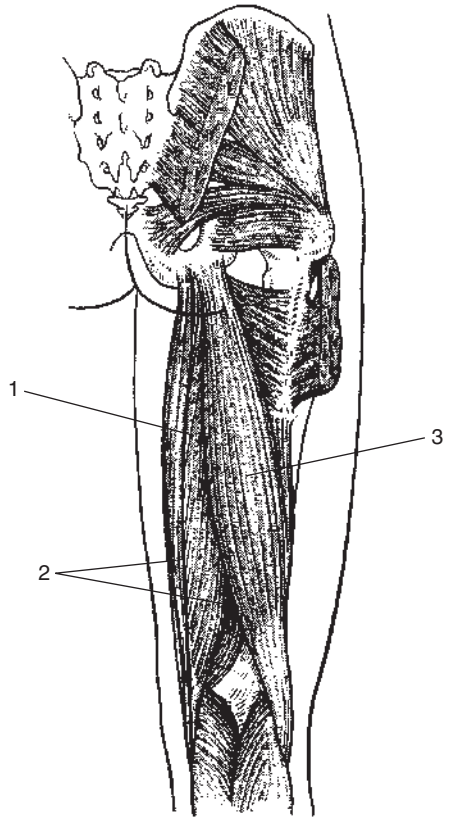

Fig 10 • Extensors of the hip (hamstrings): 1, semitendinosus; 2, semimembranosus; 3, biceps femoris.

Gluteus maximus

The most important extensor is the gluteus maximus (Fig. 9) which takes origin from the posterior gluteal line and crest of the ilium, the lower part of the sacrum, the coccyx and the sacrotuberous ligament, and runs in a lateral and caudal direction. Three-quarters of the muscle inserts at the proximal part of the iliotibial tract (see Fig. 8) and the other part into the gluteal tuberosity of the femur. It is a strong extensor. The lower fibres also have a function in lateral rotation and adduction and the upper fibres assist in powerful abduction. In standing, the muscle is inactive and remains so during the forward bending at the hip joint. However, in conjunction with the hamstrings, it is active in raising the trunk after stooping.

Hamstrings: semimembranosus, semitendinosus and biceps femoris

These muscles (Fig. 10) are also important extensors of the hip. Because the muscles cross two joints, their efficiency at the hip depends on the (extended) position of the knee. Their origins are from the upper area of the ischial tuberosity.

The semimembranosus inserts at the posterior aspect of the medial condyle of the tibia. An additional attachment is to the posterior capsule of the knee joint. Throughout its extent the muscle is partly overlapped by the semitendinosus and is therefore only palpable on each side of the latter.

The semitendinosus inserts at the proximal part of the medial surface of the tibia, behind the attachments of the sartorius and gracilis. Both muscles have accessory functions in medial rotation and adduction of the thigh, and in flexion and medial rotation of the knee joint.

The tendon of the biceps femoris splits round the fibular collateral ligament and inserts into the lateral side of the head of the fibula. The accessory function of this muscle is lateral rotation and adduction of the thigh, and flexion and lateral rotation of the knee.

The collective origin of these muscles at the tuberosity of the ischium is best palpated in the side-lying position or supine lying, with the hip flexed to 90°. This position moves the gluteus maximus upwards so permitting direct palpation of the tuberosity. Palpation of the muscle bellies is performed in prone lying, with the knee flexed to less than 90° and during slight contraction of the muscles. Resisted medial rotation of the lower leg makes semitendinosus and semimembranosus prominent. Resisted lateral rotation of the lower leg tenses the biceps and also makes the tendon visible and palpable at the lateral and distal part of the thigh.

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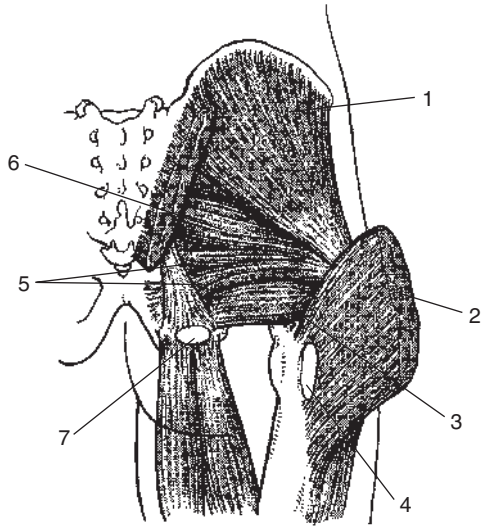

Fig 11 • The gluteus medius muscle – the main abductor of the hip joint: 1, gluteus medius; 2, gluteus maximus (reflected back); 3, quadratus femoris; 4, gluteal bursa; 5, gemelli; 6, piriformis; 7, ischial bursa.

Table 3 Abductor muscles

Muscle	Innervation	
	Peripheral nerve	Spinal
Gluteus medius	Superior gluteal	L5
Gluteus minimus	Superior gluteal	L5
Tensor fasciae latae	Superior gluteal	L5
Gluteus maximus	Inferior gluteal	Mainly S1

Abductor muscles

The main abductor muscle is the gluteus medius (Fig. 11). It originates from the external surface of the ilium, just below the iliac crest. Insertion is on the lateral surface of the greater trochanter of the femur. The muscle is partially covered by two other muscles: anteriorly the tensor fasciae latae, posteriorly the gluteus maximus.

The gluteus medius stabilizes the pelvis in the transverse direction. Standing on one limb, strong action of the gluteus medius, powerfully assisted by gluteus minimus and tensor fasciae latae, keeps the pelvis horizontal. This stabilization of the pelvis is essential for normal walking. In mild or moderate weakness of these muscles, the characteristic sign of Duchenne–Trendelenburg syndrome is demonstrated, i.e. the patient is unable to keep the pelvis horizontal, which is tilted to the opposite side. In gross weakness an excessive movement of the trunk towards the affected side compensates the paralysed hip abductors ('abduction lurch').

The other important abductors are the gluteus minimus, the tensor fasciae latae and the upper part of the gluteus maximus (Table 3).

The lateral aspect of the hip is covered by a wide muscular fan (see Fig. 8) made up of two muscle bellies: anteriorly the tensor fasciae latae, posteriorly the superficial fibres of the gluteus maximus. Both insert into the anterior and posterior borders of the iliotibial tract at its proximal aspect. Because of the triangular shape and the anatomical and functional similarity to the deltoid muscle of the shoulder joint, these muscles are sometimes known as the 'deltoid of the hip'.

The iliotibial tract is a long and strong band, which is part of the fascia lata and attached to the anterior aspect of the lateral tibial condyle. This structural arrangement permits the muscles to influence the stability of the extended knee joint and thus help to maintain the erect posture.

The iliotibial tract overrides the greater trochanter, where it is vulnerable to strain.

Adductor muscles

The adductors lie medial to the central axis of the hip joint. Although the adductor magnus is the most powerful it is clinically not important. The adductor longus (see Fig. 7) is more easily strained. Its origin is at the anterior aspect of the pubis at the junction of the pubic crest and symphysis, and it inserts at the middle third of the linea aspera of the femur. During resisted adduction the adductor longus is the most prominent muscle of the adductor group and forms the medial border of the femoral triangle. Its accessory function is flexion of the hip.

Other adductors are the pectineus, adductor magnus, quadratus femoris, external obturator and the greatest part of the gluteus maximus. Another adductor is the gracilis, the most superficial of the adductor group. It arises broadly from the inferior ramus of the pubis. The muscle belly lies just dorsal to the adductor longus (see Fig. 7). The tendon of this biarticular muscle passes across the medial condyle of the femur, posterior to the tendon of the sartorius, and is attached to the upper part of the medial surface of the tibia. Because of this course it is also a flexor and medial rotator of the knee.

Finally, the semitendinosus, semimembranosus and biceps femoris also assist in adduction of the hip (Table 4).

There is a strong functional relationship between the abdominal muscles and the adductors of the hip joint. 'Adductor tendinitis' and 'rectus abdominus tendinitis' are often seen simultaneously.

Lateral rotator muscles

When the resisted lateral rotation test is painful, the quadratus femoris should be sought first because the lesion is usually in this muscle (see Fig. 11). This flat quadrilateral muscle arises from the upper part of the lateral border of the tuberosity of the ischium and inserts just distally from the intertrochanteric crest of the femur.

Other lateral rotators of the hip (Table 5) that cross the joint posteriorly, such as the piriformis, the obturator muscles, pectineus, the posterior fibres of the adductor magnus, the gluteus maximus and the posterior fibres of gluteus medius, are clinically unimportant (see Standring, Fig. 80.21).

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Table 4 Adductor muscles

Muscle	Innervation	
	Peripheral nerve	Spinal
Adductor longus*	Obturator	L2, L3
Adductor magnus	Obturator and sciatic	L3, L4
Gracilis	Obturator	L2, L3, L4
External obturator	Obturator	L3, L4
Pectineus	Femoral and obturator	L2, L3
Quadratus femoris	Sacral plexus	L4, L5, S1
Semimembranosus	Sciatic	S1, S2
Semitendinosus	Sciatic	S1, S2
Biceps femoris	Sciatic	S1, S2

*Clinically important muscle.

Table 5 Lateral rotator muscles

Muscle	Innervation	
	Peripheral nerve	Spinal
Quadratus femoris*	Sacral plexus	L4, L5, S1
Piriformis	Sacral plexus	S1, S2
Internal obturator	Sacral plexus	L5, S1, S2
External obturator	Obturator	L3, L4
Pectineus	Femoral/ obturator	L2, L3
Adductor magnus	Obturator	L3, L4
Gluteus medius	Superior gluteal	L4, L5, S1
Gluteus maximus	Inferior gluteal	L5, S1, S2
Biceps femoris	Sciatic	S1, S2
Sartorius	Femoral	L2, L3
Iliopsoas	Femoral	L2, L3

*Clinically important muscle.

The long head of biceps also laterally rotates the thigh when the hip is extended.

Anteriorly, the sartorius and iliopsoas have only an accessory function during lateral rotation.

Medial rotator muscles

The medial rotators (Table 6) that cross the hip anteriorly are the tensor fasciae latae and the anterior fibres of gluteus medius and minimus. Posteriorly, semimembranosus and semitendinosus have an accessory function in medial rotation when the hip is extended.

Table 6 Medial rotator muscles

Muscle	Innervation	
	Peripheral nerve	Spinal
Tensor fasciae latae	Superior gluteal	L5
Gluteus medius	Superior gluteal	L5
Gluteus minimus	Superior gluteal	L5
Semimembranosus	Sciatic	S1, S2
Semitendinosus	Sciatic	S1, S2

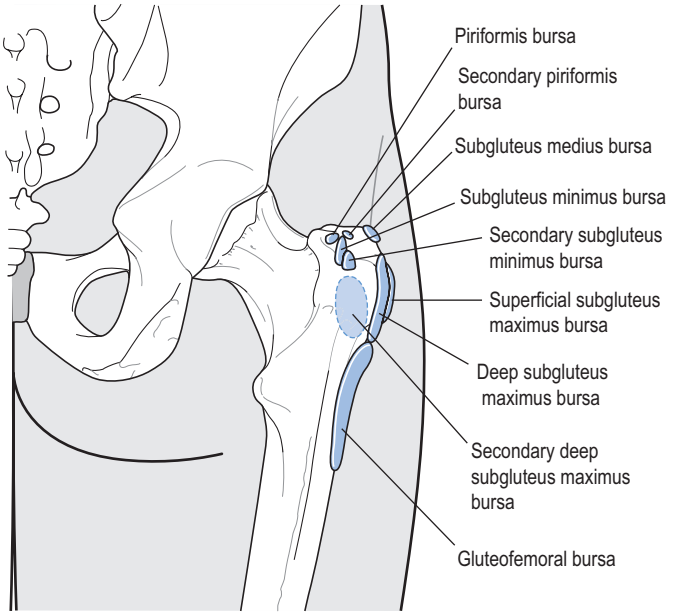

Fig 12 • The bursae at the level of the greater trochanter.

Bursae

The gluteal bursae are situated deeply, just above and behind the greater trochanter underneath the gluteus medius and maximus (see Fig. 11).

The trochanteric bursa (or subgluteus maximus bursa) is situated more superficially, laterally between the greater trochanter and the iliotibial tract (Fig. 12).

The psoas bursa, also called the bursa iliopectinea, lies deep to the iliopsoas muscle at the floor of the femoral triangle and just in front of the hip joint, with which it may communicate (Fig. 13).

The ischial bursa lies distally at the tuberosity just covered by the edge of the gluteus maximus (see Fig. 11). In a flexed, i.e. sitting, position the muscle is pulled up slightly so that in bursitis pain results because of compression of the inflamed bursa between the seat and the tuberosity.

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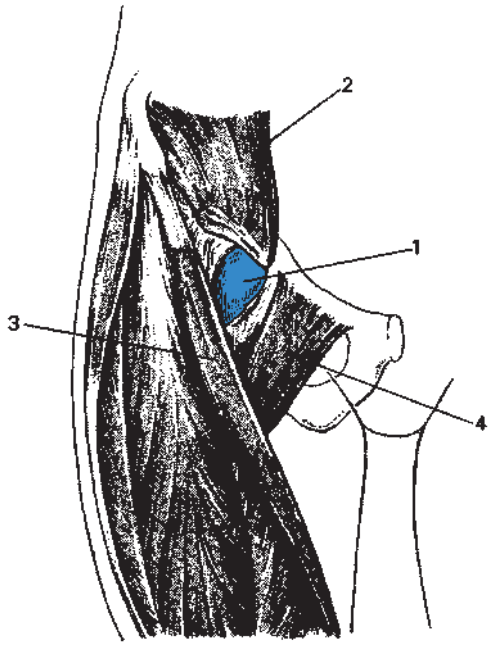

Fig 13 • The psoas bursa, 1; 2, psoas (resected); 3, sartorius; 4, pectineus.

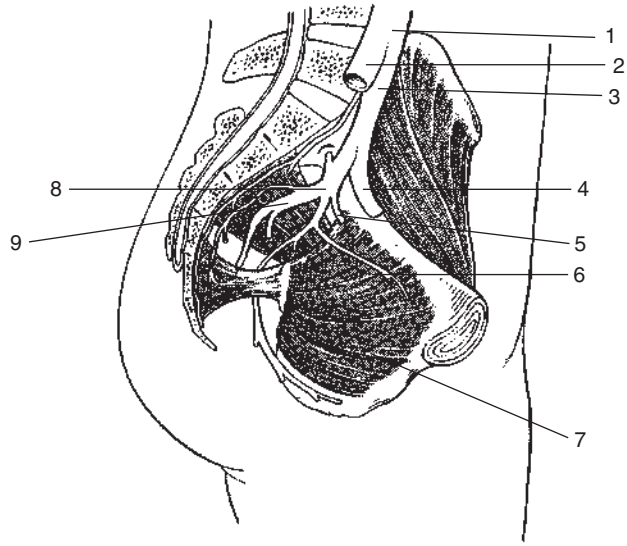

Fig 14 • Arteries: 1, aorta; 2, right common iliac artery; 3, left common iliac artery; 4, external iliac artery; 5, superior gluteal artery; 6, obturator artery; 7, internal obturator muscle; 8, internal iliac artery; 9, inferior gluteal artery.

Nerves

The femoral nerve (see Fig. 15) arises mainly from the second and third lumbar spinal nerves. It passes down between the psoas major and iliacus muscles, then behind the inguinal ligament to enter the thigh. At this proximal level, the nerve lies just lateral to the femoral artery (see Standring, Fig. 80.28). Muscular branches supply the iliacus, pectineus, sartorius and quadriceps muscles. The skin on the front of the thigh is supplied by several cutaneous branches.

The lateral cutaneous nerve of the thigh arises from the second and third lumbar spinal nerves. It passes behind or through the inguinal ligament about 1 cm medial to the anterior superior iliac spine. At the proximal part of the sartorius it divides into two branches to supply the anterolateral part of the thigh as far as the knee.

The obturator nerve arises mainly from the third and fourth lumbar spinal nerves. It enters the thigh through the obturator foramen. Some cutaneous branches are given to the skin on the medial side of the thigh, whereas another branch supplies the capsule of the hip joint. Muscular branches are distributed to the pectineus, adductor longus, gracilis, adductor brevis, external obturator and adductor magnus.

The sciatic nerve (see Fig. 17), the largest nerve in the body, arises from the fourth and fifth lumbar and first and second sacral spinal nerves. It passes out of the pelvis through the greater sciatic foramen below the piriformis muscle. On its medial side it is accompanied by the inferior gluteal artery and the posterior cutaneous nerve of the thigh. The nerve descends just medial to the midpoint of a line joining the greater trochanter of the femur and the tuberosity of the ischium. Muscular branches are distributed to the semimembranosus, semitendinosus and biceps femoris muscles. It also supplies the capsule of the hip joint through articular branches.

Blood vessels

The abdominal aorta bifurcates at the level of the fourth lumbar vertebrae into right and left common iliac arteries (Fig. 14).

At the level of the sacroiliac joint, these vessels divide into external and internal iliac arteries. Passing behind the inguinal ligament just midway between symphysis pubis and anterior superior iliac spine, the external artery becomes the femoral artery. The internal iliac arteries divide into three: the superior and inferior gluteal arteries supplying the buttock and the obturator artery which supplies the hip joint and the adductor muscles (Fig. 15).

Topographical anatomy

Anterior side

The anterior superior iliac spines (ASIS) are easily palpable except in obese patients. They form the point of origin of the sartorius muscle anteriorly and the tensor fasciae latae laterally. The spines are not level when there is leg length difference. The same occurs in pelvic torsion. However, the level of the posterior superior iliac spine (PSIS) on the same side is then just reversed, i.e. in a posterior rotation of the right innominate bone the ASIS on that side is higher whereas the PSIS is lower compared with the other side.

The anterior inferior iliac spines, lying just beneath the superior ones, are best palpated in a sitting position, with the

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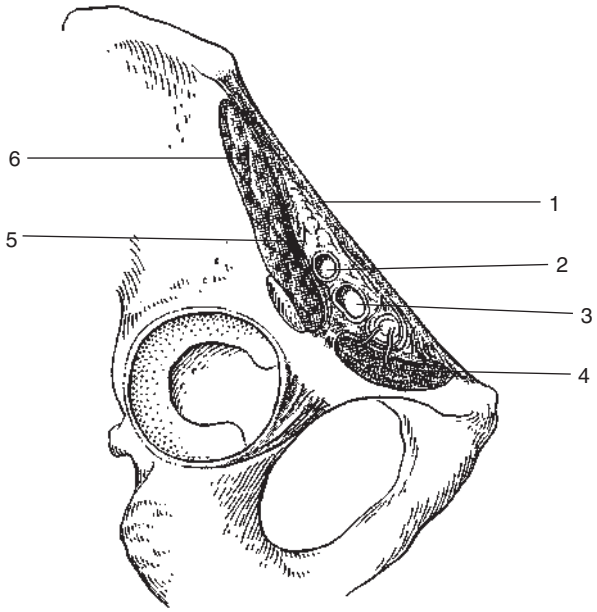

Fig 15 • Iliopsoas tendon and neurovascular structures of the groin: 1, inguinal ligament; 2, femoral artery; 3, femoral vein; 4, pectineus; 5, femoral nerve; 6, iliopsoas.

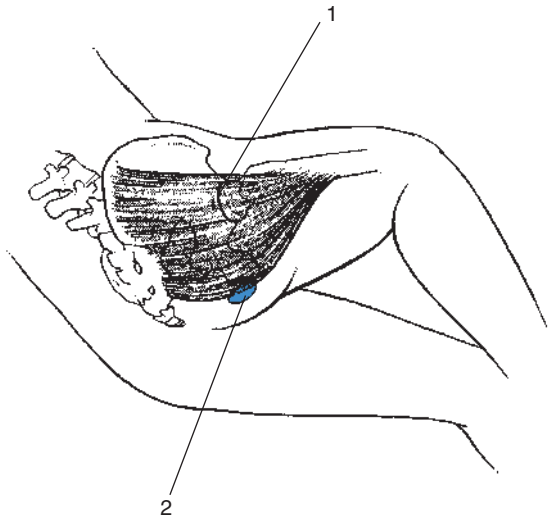

Fig 16 • The bursa at the ischial tuberosity: 1, gluteus maximus; 2, ischial bursa.

hip flexed to 90°. These are the points of origin of the rectus femoris muscles.

Towards the midline, the pubic tubercles provide attachment for the medial end of the inguinal ligament. They are palpable as bony prominences lying at the same level as the superior aspect of the greater trochanters. The rectus abdominus inserts with a medial and lateral head just cranial to these tubercles at the crest of the pubic bone.

For topographical reasons, it is also important to have an understanding of the anatomy of the femoral triangle (see Fig. 7), which is defined superiorly by the inguinal ligament, medially by the adductor longus and laterally by the sartorius. The floor of the triangle is formed by portions of the iliopsoas on the lateral side and the pectineus on the medial side. The femoral artery lies superficial and medial to the iliopsoas muscle and is easily found by palpation of the pulse as it emerges from behind the inguinal ligament (see Fig. 15). Deep to the iliopsoas lies the psoas bursa and still deeper the hip joint.

The femoral head, however, is not palpable because of the presence of the overlying muscles. The femoral nerve and vein (lateral and medial to the artery, respectively) are not palpable.

Several lymph nodes are situated medially in the femoral triangle. They can be palpated only when they are enlarged.

Lateral side

Palpation of the greater trochanter is relatively easy at its posterior edge, where the bone is not covered by muscles. The greater trochanter is an important landmark and, in the standing position, both greater trochanters should be level and at the same distance from the iliac crest and the anterior superior iliac spines. Their upper aspects should also be on the same level as the pubic tubercles.

Posterior side

The ischial tuberosity is covered by the gluteus maximus and adipose tissue (Fig. 16).

If the hip is flexed, the gluteus maximus moves upwards and the ischial tuberosity becomes easily palpable. It provides attachment for the hamstring muscles posteriorly and the quadratus femoris laterally. At its distal aspect, the bone is covered by a bursa (see Fig. 11).

Midway between the greater trochanter and the ischial tuberosity the sciatic nerve passes to the leg (Fig. 17). With the hip in a flexed position this nerve may be palpable underneath the adipose tissue.

The posterior superior iliac spines (see Fig. 9) are easily palpable, where they lie directly underneath the visible dimples just above and medial to the buttocks.

Joint movements

The hip has three degrees of freedom: flexion–extension, adduction–abduction, and medial and lateral rotation.

Flexion is forward movement in the sagittal plane (Fig. 18a). The range depends on the position of the knee. With the knee flexed and the movement performed passively, the anterior aspect of the thigh comes in close contact with the abdomen, so that the range exceeds 140°. With the knee extended, flexion is limited by the tension of the hamstrings. These muscles are biarticular and therefore restrict hip flexion when the knee is extended – the constant-length phenomenon.

Extension backward movement in the sagittal plane (Fig. 18b) is considerably restricted by the tension of the iliofemoral ligament. Passively an average range of 30° can be reached.

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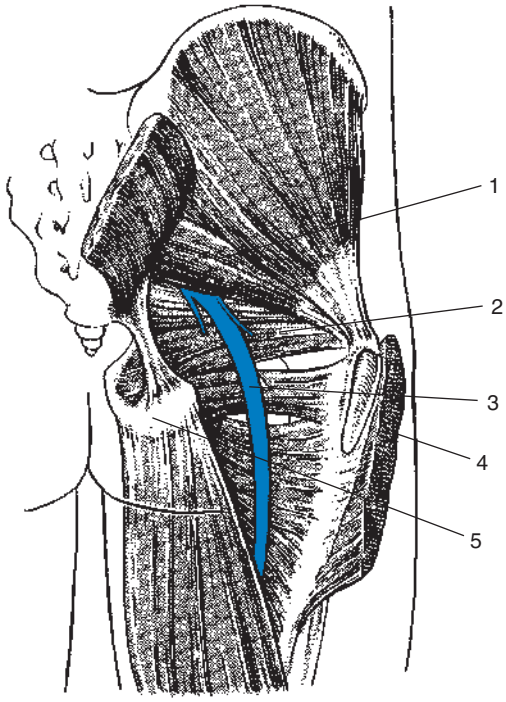

Fig 17 • The sciatic nerve: 1, piriformis; 2, gemellus superior; 3, sciatic nerve; 4, gluteus maximus (resected); 5, ischial tuberosity.

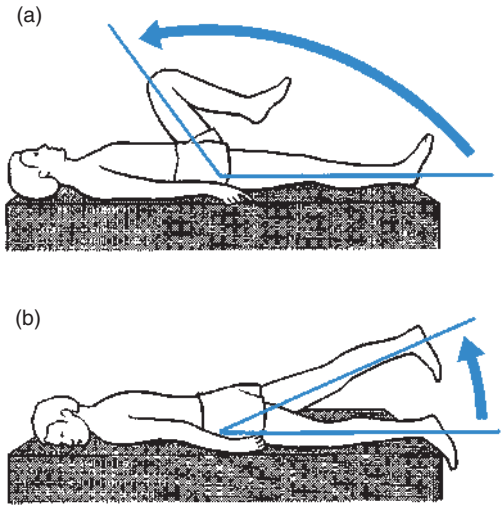

Fig 18 • Flexion (a) and extension (b).

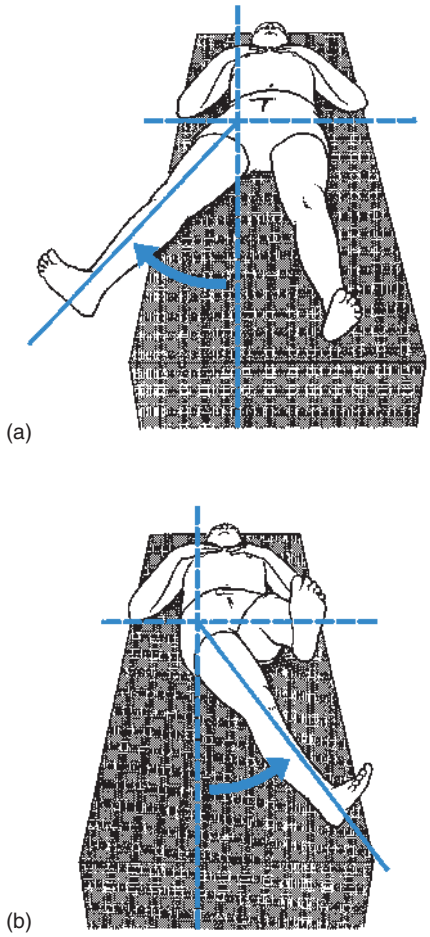

Fig 19 • Abduction (a) and adduction (b).

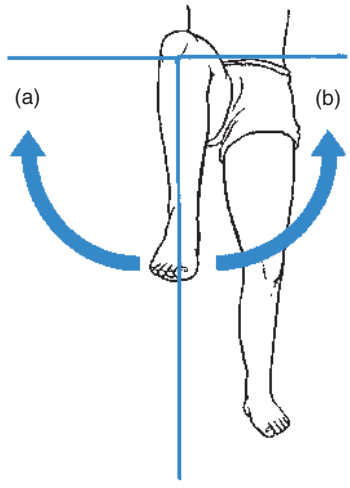

Fig 20 • Medial (a) and lateral (b) rotation.

Adduction and abduction are movements in the frontal plane (Fig. 19). The average range of adduction is 30°, and abduction 45°. In the latter, the constant-length phenomenon again plays a vital part. Here the biarticular gracilis restricts the range of abduction when the knee joint is fully extended.

Rotational movements of the hip are measured in the supine position, with the thigh in 90° of flexion (Fig. 20), or lying face down, the hip at 0° and the knee flexed to 90°. The normal range of medial rotation is about 45°, whereas lateral rotation reaches about 60°. With the thigh flexed, lateral rotation can be increased, because of relaxation of the iliofemoral and pubofemoral ligaments.

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Groin pain

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Introduction

Groin injuries tax the diagnostic and therapeutic abilities of the clinician.[1,2] Although they are considered to be common and the most frequent overuse syndromes in some athletic activities such as soccer,[3] they are difficult to manage correctly.

First, groin lesions involve a complex regional anatomy and often produce unusual presentations. Second, the lesions are often described using inaccurate terminology. For example, *groin* is a confusing term and *groin injury* usually describes multiple clinical conditions without clearly defining location or cause. It can, as is usually the case, mean a thigh muscle strain; it may refer to the genitalia; it can indicate hip disorders; or it may mean a problem in the lower abdominal wall. Finally, lesions in the back, in the sacroiliac region and within the abdomen may cause pain referred to the region of the groin.

History

The clinician confronted with the prospect of evaluating a patient who complains of groin pain should first of all ask the patient to point to the pain and also establish whether it is localized or diffuse. The groin area is not only the crossing site of trunk and lower extremity muscles but is also a region of considerable dermatomical overlap which includes T11, T12, L1, L2, L3 and S4 (Fig. 1). Pain in the groin can also be of extrasegmental origin.

Once the localization and any radiation of the pain have been clarified, the examiner continues with the routine history which relates to problems in the back, sacroiliac region and the hip (see Ch. 36, 45, 61). Finally questions related to possible intra-abdominal disorders may be indicated. For example:

- Are fever, sweats or chills present (infection or neoplasm)?
- Has there been weight loss (neoplasm)?
- Are urinary symptoms such as dysuria, urgency, frequency or haematuria present?
- Are there bowel symptoms – diarrhoea, mucus or blood in the stool?

Clinical examination

The physical examination of the groin region should proceed through several steps to search for various disorders. A start is always made with a routine examination of the lumbar spine, always followed with a similar procedure on the thoracic spine. A thorough examination of the hip follows. If the signs and symptoms warrant, a complete accessory examination of the sacroiliac joints must also be performed.

Finally, and if appropriate, an abdominal examination may be added: palpation for pain, rebound and guarding, pulses, nodes, hernia and masses such as an abdominal aortic aneurysm.

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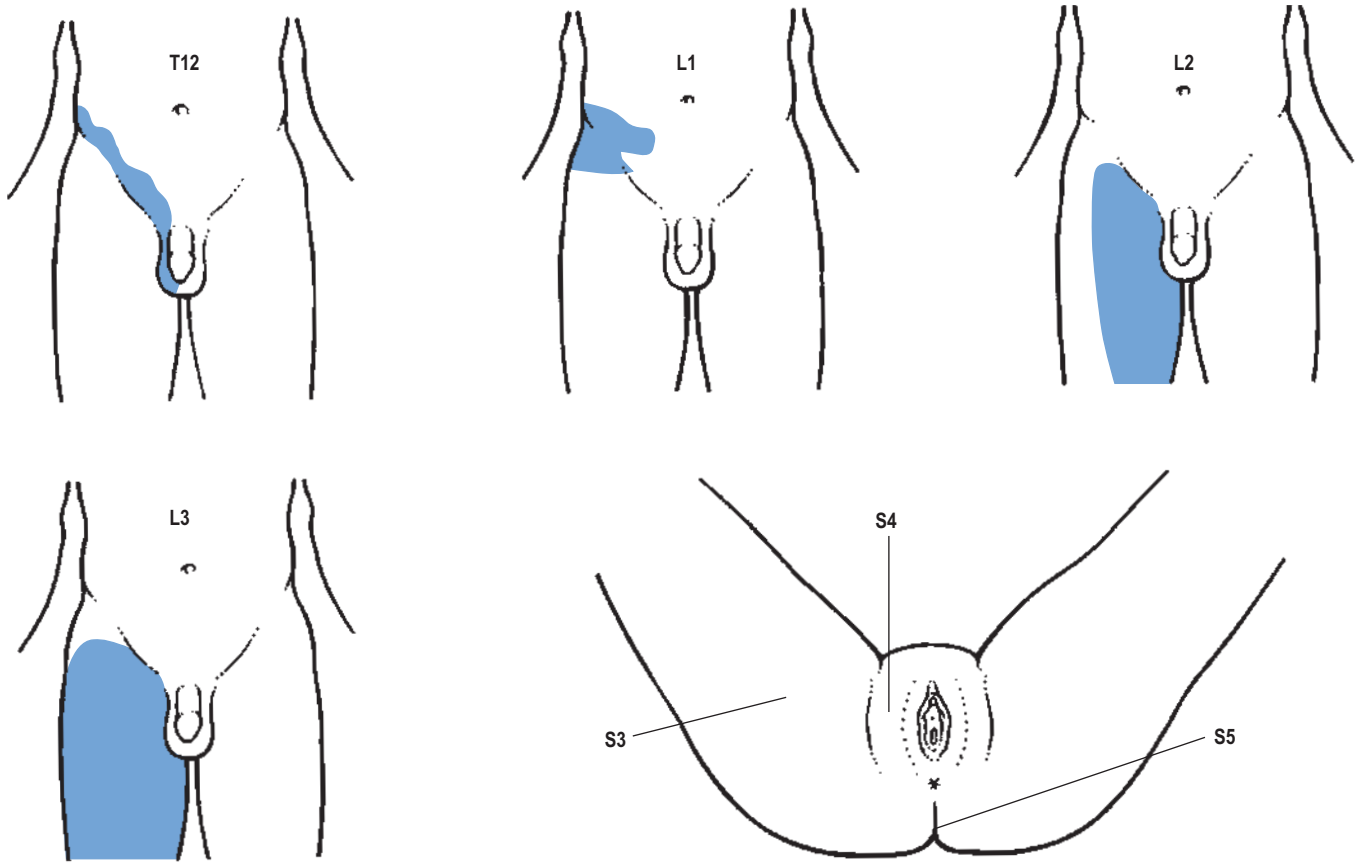

Fig 1 • The groin is a region of intense dermatomic overlap.

Interpretation

The causes that can result in groin pain are numerous and are summarized in Table 1. Differential diagnosis is not difficult if the guidelines and principles for a good functional examination of the lumbar spine and hip are followed. However, occasionally the results may be confusing. This is usually caused by the fact that resisted movements in and around the groin not only stress the activated contractile structures, but often also induce transmitted stress on inert structures (bones, ligaments). For example, resisted ad- and abduction movements of the hip may indirectly put stress on the sacroiliac or iliolumbar ligaments or on the pubic symphysis.

Most lesions have been discussed in the chapters on the hip and the thoracic spine. However, there remain some common sport lesions that cause groin pain and deserve particular attention.

Femoral neck stress fracture

This overuse injury occurs primarily in endurance athletes (often in thin women who are frequently amenorrhoeic). Risk factors include training errors, inadequate footwear, running on poor surfaces and coxa vara (angular deformity of the hip).

The fracture causes groin pain, sometimes radiating down the anterior part of the thigh. The pain (often merely an ache) increases with walking or running and is relieved on cessation of activity. Pain at night may be present in long-standing instances.

Examination reveals an antalgic gait. There is usually a discrepancy between the obvious gait and the rather subtle signs on clinical examination: a full range of motion with pain produced at the extremes of hip rotation and on axial compression.[4,5]

Early radiographs may be negative and absence of a fracture line does not rule out a stress fracture. A bone scan should be positive 2–8 days after symptoms appear. Further imaging studies such as computed tomography (CT) or magnetic resonance imaging (MRI) should be undertaken early if clinical suspicion warrants it.[6]

Treatment is based on the type of fracture. If the bone scan is positive but there is no visible fracture on plain film, initial treatment will consist of modified bed rest. This leads on to non-weight bearing with crutches and then pain-free weight bearing.[3] If there is visible fracture on the plain film, open reduction and internal fixation is the treatment of choice because of the high risk of displacement. An already displaced fracture is considered an orthopaedic emergency and requires open reduction and internal fixation.

Athletes must be told that stress fractures of the femoral neck are serious injuries that can compromise an athletic career. Even in a successful recovery, return to participation in the chosen sport may take as long as 4–5 months.

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Table 1 Lesions that can cause groin pain

Referred pain	Abdominal organs	Abdominal wall	Pelvis	Hip
Extrasegmental (dural) pain	Bowel • appendicitis • diverticulitis	Rectus abdominis tendinitis	Sacroiliac strain Iliolumbar strain	Capsular lesions Loose body
T12		Inguinal hernia	Pubic ramus fracture	Avascular necrosis
L1	Aortic aneurism	'Groin disruption'	Osteitis pubis	Psoas bursitis
L2	Genitalia • ovarian cyst	Ilioinguinal neuralgia		Femoral neck stress fracture
L3	• ectopic pregnancy and other pelvic inflammation • prostatitis			
S2	• testicular torsion			Avulsion fracture of anterior superior iliac spine
S3	Urinary tract • infection • lithiasis			Avulsion fracture of lesser trochanter Adductor tendinitis Tendinitis of • psoas • sartorius • rectus femoris

Acetabular labral tears

Recent arthroscopic studies suggest that most internal derangement in the hip may be the result of impingement of acetabular labral tears.[7] The history is that of internal derangement: a feeling of giving way or a sharp 'twinge' in the groin that radiates into the anterior thigh, especially with a rotation of the hip while rising from a seated position. On examination, a non-capsular pattern of limitation is found, with limitation of lateral rotation (see p. 642).

Some authors report successful diagnosis of impingement lesions with the so-called Thomas test.[3] This involves flexion and external rotation of the hip and then allowing the extremity to abduct. The hip is then moved into extension, internal rotation and adduction. A positive test result is indicated by a palpable or audible click and the production of typical pain. Arthrography, MRI and arthroscopy can be used to confirm the diagnosis.

Treatment consists of manipulation (p. 642), although arthroscopic or open operative excision may be necessary in recalcitrant cases.

Pubic ramus stress fracture

Stress fractures of the pubic ramus occur mostly in distance runners and joggers, with a higher incidence in females. Traction forces produced by the muscles attached to the pelvis have been implicated as possible aetiological factors. Pain in the inguinal, perineal or adductor region is the usual presenting symptom.

On examination there is a considerable discrepancy between the marked signs on standing and walking (an antalgic gait with an inability to stand unsupported on the affected leg) and the full and painless passive range of hip motion in the supine-lying position. However, resisted movements of the hip (especially resisted adduction) do provoke the pain. Exquisite tenderness over the affected pubic ramus is also common.

Plain radiographs may not show a fracture until several weeks after the injury. Bone scan is necessary for early diagnosis.

Treatment consists of cessation of running activities. Most athletes will show complete union of bone at 3–5 months.[3]

Osteitis pubis

Although osteitis pubis is a well-known entity following surgery of the bladder or the prostate;[8] it has also been reported after athletic endeavours.

Osteitis pubis (an inflammatory lesion of the bone adjacent to the symphysis pubis) in athletes is thought to be the result of mechanical strain from trauma, excessive twisting and turning in sports such as soccer or repetitive shear stress from excessive side-to-side motion.[9] It is quite common in ice-hockey players, soccer players and in long-distance runners. It is also frequent in women who exercise in the postpartum period because of the particular instability of the symphysis after birth.[10]

Complaints develop gradually and the patient cannot link the onset to any known injury. Pain is described as originating from the pubic region, with radiation into the lower abdomen,

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the groin and the adductor regions. The pain is linked to the athletic activity and gradually disappears upon resting. Coughing or sneezing may be painful.[11] In severe examples the athlete may develop an antalgic or waddling gait.

On examination there is a full range of passive movement of the hips with pain elicited by passive abduction and resisted adduction. The fact that the pain is reproduced during the examination of *both* lower extremities should help in the differentiation from a tendinitis of the adductor longus (p. 654). Palpation also causes pain along the pubic bones and the symphysis itself and not at the tendinous junction of the adductor muscles.

Bone scintigraphy, which typically shows increased uptake unilaterally or bilaterally at the pubic bones, is effective in making an early diagnosis. Radiographic changes may not be visible for 2–3 weeks but then show a symmetric resorption of the medial ends of the pubic bone, widening of the symphysis and rarefaction or sclerosis along the pubic rami. CT scan demonstrates early degenerative changes in the cortical bone, including erosions, cysts and osteophytic spurs at the symphysis.[12]

Treatment initially includes relative rest because the condition is usually considered as self-limiting within 8–12 weeks. One or two infiltrations with 20 mg triamcinolone into the symphysis may hasten healing.[13]

However, the condition may be chronic or recurrent. If symptoms persist, three infiltrations with sclerosant solution at weekly intervals usually give good results.

Groin hernia

Inguinal and femoral hernias are sufficiently common that every patient who suffers from groin pain should be examined specifically to eliminate this cause.

An inguinal hernia is located above and at the medial end of the inguinal ligament. A femoral hernia, more common in female patients, is below and lateral to the inguinal ligament (Fig. 2).

The more common type of inguinal hernia is *direct* which pushes out through the posterior wall of the inguinal canal lateral to the lateral border of the rectus abdominis muscle. It is usually the consequence of a weakness which has developed in the posterior wall of the inguinal canal (fascia of the transverse abdominal muscle). Most direct inguinal hernias are symptomless apart from the presence of a bulge.

An *indirect* inguinal hernia is congenital in origin and is caused by a failure of the processus vaginalis of the peritoneum soundly to close. It therefore originates at the internal inguinal ring, appears at the external ring and may extend into the scrotum. The relationship to injury is uncertain and, in contrast to the 'sports hernia' described below, many authorities consider that it develops from a weakness or tear of the posterior wall of the inguinal canal more lateral than that in a direct inguinal hernia in the presence of a potentially patent processus vaginalis.[14]

In most instances, activities that significantly increase intra-abdominal pressure or may involve repeated Valsalva manœuvres, for example weight lifting, cause or exacerbate inguinal hernia. While the pain might initially occur only after activity, it typically will increase in frequency to the point of occurring during activity and even with simple trunk and hip movements. In males the pain may radiate into the proximal thigh or the scrotum.

Examination for both types of inguinal hernia involves invaginating the scrotal skin along the spermatic cord using the index finger in males (Fig. 3) or direct palpation in females. A palpable mass may or may not be detected.

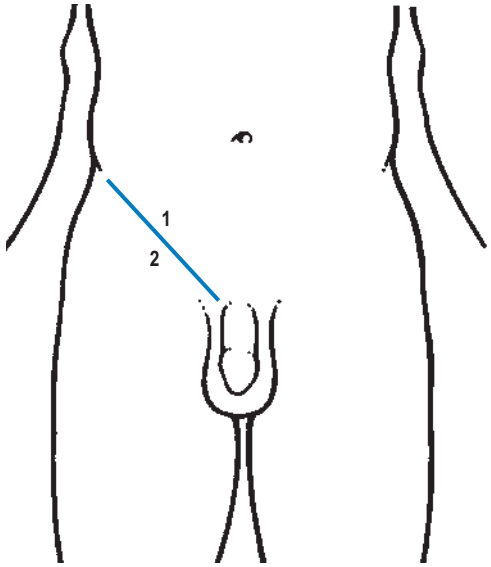

Fig 2 • Hernia localization: 1, inguinal hernia; 2, femoral hernia.

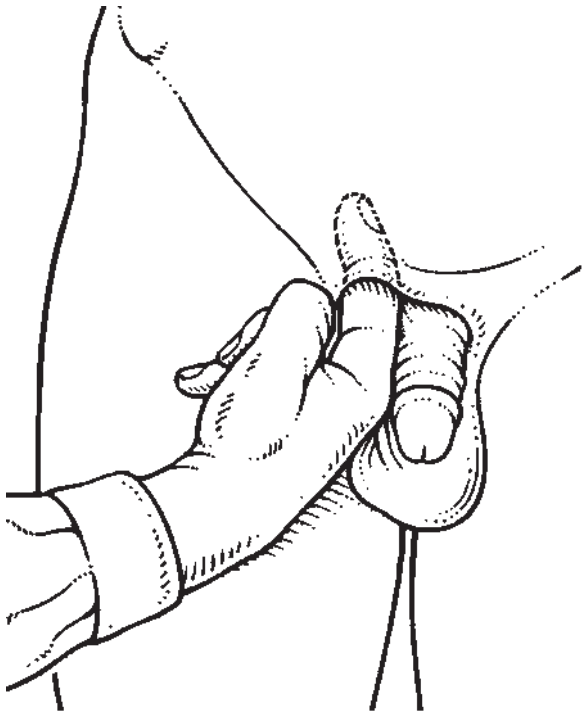

Fig 3 • Palpation of the inguinal canal via invagination of the scrotal skin.

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Manœuvres to increase intra-abdominal pressure, such as coughing or tensing the abdominal musculature, may produce a cough impulse (a sign of hernia), or may make a mass more prominent.

Treatment of inguinal hernias should be by surgical repair, not only to relieve pain and discomfort but also to prevent complications – incarceration, obstruction and strangulation with infarction of the bowel.

'Sports hernia' or 'groin disruption'

Athletes in fast-moving sports that involve twisting and turning – such as soccer, rugby and ice hockey – may be at particular risk of a tendinous disruption in the area of the inguinal canal.[15] This injury, often called a 'sports hernia', usually involves the posterior wall of the inguinal canal and can appear as a tear of the transversus abdominis muscle or as a disruption to the conjoined tendon – the tendon of insertion of both the transversus abdominis and internal oblique muscles (Fig. 4).[16] A sports hernia may, however, also involve a lesion of the external oblique aponeurosis and cause a dilatation of the external inguinal ring.[17]

A sports hernia should be differentiated from the more common inguinal hernia in that it does not involve a clinically detectable hernia (there is not a protrusion of any abdominal tissue through the walls of the abdominal cavity).[18] The term 'disruption' is therefore more appropriate to describe this type of lesion.

A sports hernia typically produces unilateral groin pain during exercises. In chronic cases, however, the patient may have symptoms during activities of daily living. Onset of pain is usually insidious but may occur suddenly. It is typically localized to the conjoined tendon but can involve the inguinal canal more laterally. Sudden movements often exacerbate the pain.[19]

Examination for a sports hernia in men is generally done by inverting the scrotal skin with a finger and palpating the inguinal ring and canal. The uninvolved side should be examined first so that the examiner may put the patient at ease and also assess the normal structures. The examining finger is inserted into the scrotal sac just below its junction with the abdominal wall and carried superiorly over the pubis up to the external inguinal ring. The ring, conjoined tendon, pubic tubercle and mid-inguinal region are checked for size and the presence of abnormal tenderness.

Conventional imaging modalities such as bone scan, ultrasound, CT and MRI all fail to reveal the defect. Surgical exploration is currently the only method to confirm the diagnosis.

Treatment is generally surgical and involves restoration of the normal anatomy by repairing the conjoined tendon and/or the external oblique aponeurosis.

Ilioinguinal neuralgia[20]

The ilioinguinal nerve originates from the Ll–L2 nerve roots. It passes between the ilium and psoas major to perforate the

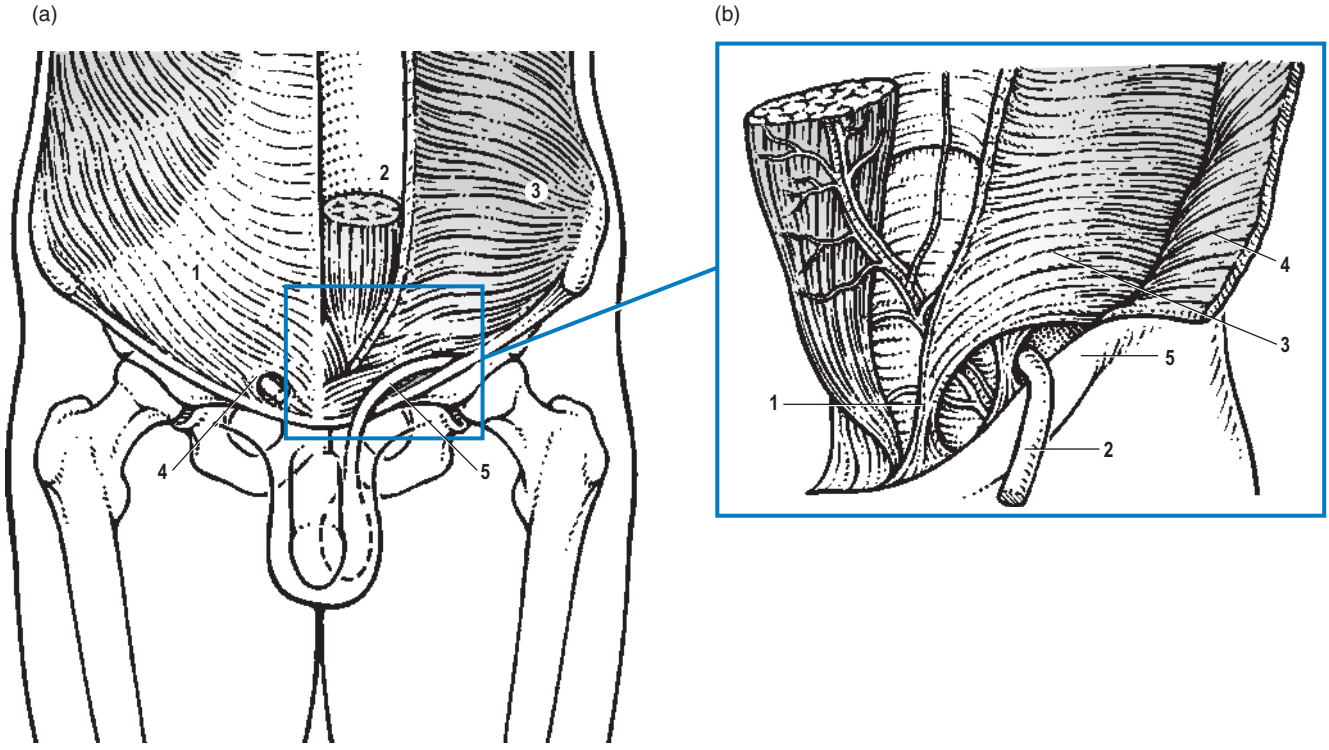

Fig 4 • Anatomy of the inguinal canal. A (overview): 1, external oblique muscle and aponeurosis; 2, rectus abdominis muscle; 3, transversus abdominis muscle; 4, external inguinal ring; 5, spermatic cord. B (insert): 1, falx inguinalis (conjoined tendon); 2, spermatic cord; 3, transversus abdominis muscle; 4, external oblique muscle; 5, inguinal ligament.

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transversus abdominis near the anterior superior iliac spine (Fig. 5). The nerve pierces the internal oblique, transverses the inguinal canal below the spermatic cord, emerging with it from the superficial inguinal ring to supply the proximomedial skin over the penile root and the scrotum in men or that covering the mons pubis and the adjoining labium majus in women. During its course it innervates the lowest portions of the transversus abdominis and internal oblique muscles as well as the skin overlying the inguinal ligament.

Ilioinguinal nerve entrapment is a well-established cause of chronic inguinal pain in patients who have had lower abdominal and inguinal hernia surgery (e.g. appendectomy or inguinal herniorrhaphy).[21] Direct trauma, intense abdominal muscle training or inflammatory conditions can also lead to entrapment of this nerve as it passes through or close to the abdominal muscle layers.[22,23]

Patients describe a burning or shooting pain in the distribution of the nerve. Light-touch sensation in the inguinal area may be altered and pain may be exacerbated by hyperextension of the hip. There is usually a well-circumscribed trigger point medial and below the anterior superior iliac spine where the ilioinguinal nerve pierces the fascia. Relief of pain by infiltration of a local anaesthetic confirms the diagnosis.

Treatment consists of three infiltrations at weekly intervals at the confirmed site with 5 ml procaine 0.5 to 1%.[24] In severe cases 20 mg triamcinolone may be added to the solution. Nerve ablation may be indicated if the lesion does not respond to infiltrations.

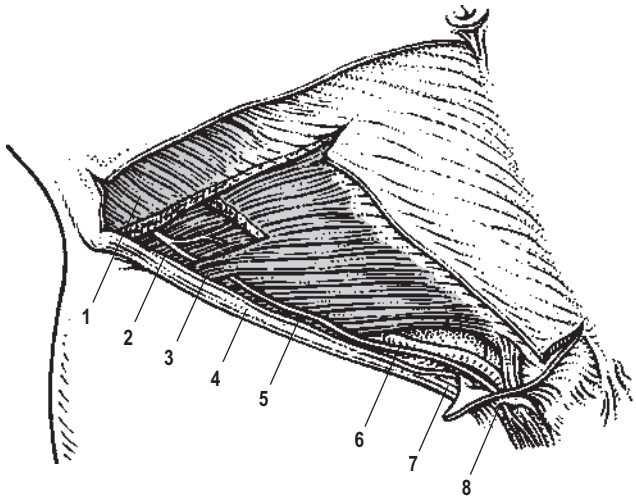

Fig 5 • Course of the ilioinguinal nerve: 1, anterior superior iliac spine; 2, transversus abdominis muscle; 3, external oblique muscle; 4, inguinal ligament; 5, ilioinguinal nerve; 6, spermatic cord; 7, pubic tubercle; 8, superficial inguinal ring.

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Hip disorders in children

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Every complaint of groin, thigh or knee pain in a child should be taken seriously and examined very carefully without delay. Furthermore, any child who limps or shows limitation of movement at the hip joint should undergo a thorough examination. Most hip lesions in children can be diagnosed adequately with the help of a radiograph. For almost all these lesions, there are good and effective treatments, provided the diagnosis is made early enough.

Congenital dislocation of the hip

The incidence of congenital dislocation is between 1 and 2 cases per 1000, but it is four times as frequent in girls as in boys.[1] There seems to be a hereditary factor, because there is about a 5% chance of a second child being affected. This rises to 36% if one of the parents also had congenital dislocation of the hip.[2]

Although there are still questions about its aetiology, the condition seems to occur in the presence of some weakness of the joint capsule and elongation of the ligamentum teres.[3] A deficiency of the rim of the acetabulum may also contribute in the development of the disease.[4] However, the most important aetiological factor seems to be an imbalance between the action of the psoas muscle and the hip extensors.[5] A relative shortening of the psoas presses the head of the femur upwards and laterally when the hip is extended by the contraction of the stronger hip extensors. In the presence of a weakness of the capsule, dislocation then occurs.

It is vital to make the diagnosis of a congenital dislocation as soon after birth as possible. Conservative treatment with an abduction brace[6] before the child begins to walk is completely adequate, but after the age of 4 even surgical repositioning is difficult and after the age of 7 it is almost impossible. Without treatment the dislocation may lead to a shorter leg, the formation of a neoacetabulum at the blade of the ilium and the supervention of early arthrosis.

Radiographs are not reliable in making the diagnosis of congenital hip dislocation in newborn babies. Early diagnosis, therefore, relies entirely on clinical tests. Those most used are known as Ortolani's and Barlow's tests.[7,8] It should be remembered, however, that positive tests only indicate a capsular laxity and therefore the *possibility* of dislocation. For instance, the incidence of positive Ortolani's and Barlow's signs in newborns is about 10–20 per 1000, which is 10 times more than at the age of 2 months, which suggests that there is a tendency towards resolution of the capsular laxity.[9]

Clinical tests

Ortolani's test

During this test the luxated hip is replaced manually.

Technique

The baby lies on the back, hips flexed to 90° and the knees completely flexed (Fig. 1a). The examiner grasps the leg with

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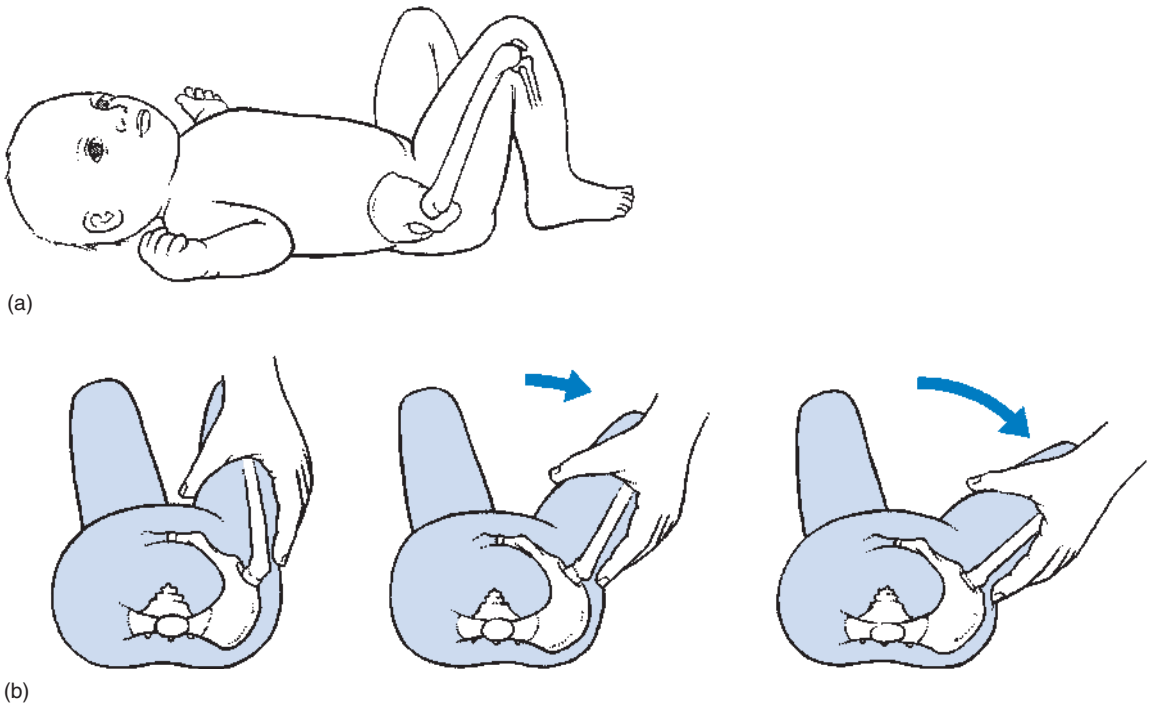

Fig 1 • Ortolani's test for congenital dislocation of the hip: (a) position of the baby, (b) reposition of the dislocated hip by abduction.

the contralateral hand in such a way that the thumb presses on the inner side of the thigh. The ring and middle fingers are placed along the outer thigh, so that the fingertips touch the trochanter. The other hand fixes the opposite hip. Abduction of the hip is now performed (Fig. 1b). In a subluxated hip, resistance will be felt at 45–60°. The moment the resistance is overcome, the femoral head rides over the acetabular edge and reduces. This is *felt* by the examiner. This sensation has wrongly been called a click, whereas the feeling is rather that of moving two knuckles of the wrist over each other. Ortoloni described this as a 'segno dello scatto' (ridge sign). Under this definition, the sensation has to be felt rather than heard.

Barlow's test

The purpose here is to dislocate the hip first and then replace it. The hips of the baby are grasped in the same way as Ortolani's test.

Technique

In this test, the thumb is placed further proximally on the femur. If the capsule is elongated or weak, the examiner can easily push the femoral head over the posterior rim of the acetabulum. This is indicated by a click.

Pressure with the middle finger behind the trochanter then moves the head anteriorly, where it comes to lie in the acetabulum again (Fig. 2). This to-and-fro movement of the femoral head over the acetabular rim indicates that the joint is unstable.

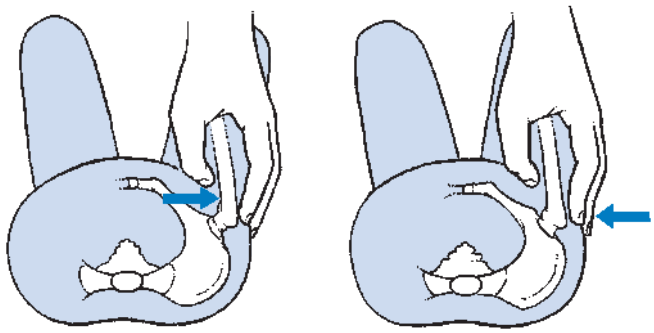

Fig 2 • Barlow's test for congenital dislocation of the hip.

Technical investigations

The diagnosis can usually be made with plain radiographs. However, during recent decades sonographic screening examinations have been shown to be superior in the early detection of congenital dislocations of the hip.[10,11] In a study of sonographic screening examinations on 4177 newborns, all cases of congenital dislocation of the hip joint were found and classified.[12] Consequently, ultrasound screening is currently offered to all newborns in Austria and Germany and to newborns with selected risk factors in the United Kingdom, Scandinavia, Italy and France.[12,13,14]

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Treatment

The majority of unstable hips at birth stabilize in a very short time,[15] but since the outcome of an 'unstable hip' is impossible to predict, all cases of hip instability in newborns must be taken seriously and treated. Hence, as soon as a positive Ortolani's or Barlow's test is found, an abduction brace is given until these clinical signs disappear.[16–18]

If the diagnosis is made when the child starts to walk, surgical repositioning is required. Surgical reduction is almost impossible after the age of 7 years. If a 'false acetabulum' develops at the iliac bone, the functional disability may be small, especially if the disorder is bilateral.[19] Pain and serious disability then start only in the fourth or fifth decade, when pseudarthrosis has occurred. If pain is not too severe, treatment can be conservative;[20] often some relief is obtained by one or two infiltrations with 50 ml of procaine 0.5% at the posterior aspect of the capsule.[21] If pain and disability are too severe, surgery is required.[22]

Congenital limitation of extension

Some children have an excessive forward tilt of the pelvis, as the result of congenital limitation of extension at the hip joints. They walk with a hyperlordosis, extending at the back instead of at their hips. This limitation is probably the result of a shortening of the psoas muscles.

Treatment consists of stretching the psoas and capsule in the same way as for early osteoarthrosis (see p. 633).

Arthritis of the hip in children

Transient synovitis

This is the commonest cause of hip pain in children and usually occurs under the age of 10 years. The child complains of pain in the groin, thigh and knee after some previous exertion. Sometimes only knee pain is present. The child limps and clinical examination of the hip shows a capsular pattern.

Although the pathogenesis of the disorder remains obscure, the conventional view is that it results from ordinary overuse. However, a correlation between transient arthritis and the development of Perthes' disease has been shown repeatedly.[23,24]

Sonographic examination, together with X-ray examination, represent the first choices in the evaluation of a capsular pattern in a child and in the follow-up of transient synovitis.[25] In transient synovitis there is effusion but the effusion persists for less than 2 weeks. In the early stage of Legg–Calvé–Perthes' disease the capsular distension persists for more than 6 weeks. A differential diagnosis between transient synovitis and Legg–Calvé–Perthes' disease is therefore only possible by observing the ultrasonographic course of the disease.[26]

Treatment is bed rest and non-weight bearing. It is advisable, if excessive intra-articular effusion is demonstrated by ultrasonography, to aspirate the fluid.[27] Most cases recover completely and without sequelae after a few weeks of bed rest.[28]

Septic arthritis

The differential diagnosis with a transient synovitis is difficult.[29] A septic arthritis of the hip may have similar early symptoms as a transient synovitis, with the spontaneous onset of progressive hip, groin, or thigh pain; limp or inability to bear weight, fever and irritability.[30] Clinical examination shows a capsular pattern. A variety of clinical, laboratory, and radiographic criteria are used to help differentiate septic arthritis from transient synovitis. The four clinical predictors to differentiate between septic arthritis and transient synovitis are: history of fever, pain at rest, erythrocyte sedimentation rate of at least 40 mm per hour, and serum white blood-cell count of more than 12 000 cells per mm^3.[31] In cases of doubt, hip aspiration should be performed.

Tuberculosis of the hip

The child complains of pain in the groin, thigh and knee, and limps. Clinical examination shows a gross capsular pattern, sometimes to such an extent that the hip is fixed in flexion, abduction and lateral rotation. There is considerable wasting of the thigh muscles. Symptoms of general illness, with slight fever and fatigue complete the picture. Radiography and examination of articular fluid give a decisive answer.

Haemarthrosis

The hip is very rarely affected in haemophilia. Acute hip pain and gross articular pattern in a haemophiliac reveal the diagnosis. Prompt aspiration and specific anti-haemophiliac measures must be taken in order to protect the cartilage against the erosive effects of the presence of the blood.

Perthes' disease (pseudocoxalgia)

Perthes' disease, an idiopathic avascular necrosis/osteonecrosis of the femoral epiphysis, was described in 1910 by Legg as 'an obscure affection of the hip joint',[32] by Calvé as 'pseudocoxalgia'[33] and by Perthes' as 'arthritis deformans juvenilis'.[34] The disease affects 4 to 10 year olds, peaking between 5 and 7 years. It affects about four boys for each girl and is bilateral in 10%. Although it is generally accepted that the necrosis is the result of a change in the blood supply to the femoral head,[35] there still remains much controversy about the way this occurs. There have been reports suggesting retarded skeletal ageing in relation to the chronological age.[36] Some studies have also demonstrated an association with pituitary growth hormone deficiency.[37] However the most likely aetiology for Legg–Calvé–Perthes' disease seems to be the hypothesis of clotting abnormalities with vascular thrombosis.[38,39]

In the early stage, the only symptom is a slight limp or pain in the hip, thigh or knee. Examination of the knee is normal

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but there is a capsular pattern of the hip. The Trendelenburg sign may also be positive: when the patient stands on one leg, pain in the weight-bearing hip or weakness of the ipsilateral gluteus medius results in a downwards pelvic tilt on the contralateral side. In a later stage, limitation of adduction during flexion is noticed – as passive flexion is performed, the knee gradually moves into abduction. When the flexed hip is now drawn toward the opposite side, limitation, pain and a hard end-feel are noted.

Radiographs vary with the stage of the disease but may show evidence of bone necrosis, fragmentation, reossification or remodelling and healing. The earliest signs include decreased size or increased density of the proximal femoral epiphyses on an antero-posterior view and crescent sign (a subchondral fracture that correlates with the extent of necrosis) on the lateral view.

Treatment and prognosis depend on the age of onset (the younger the age of onset the better the prognosis), the physical signs and the radiological changes.[40]

Late sequelae of the disease are leg-length shortening and degenerative joint disease that develops in the majority of patients by the sixth or seventh decade of life.[41]

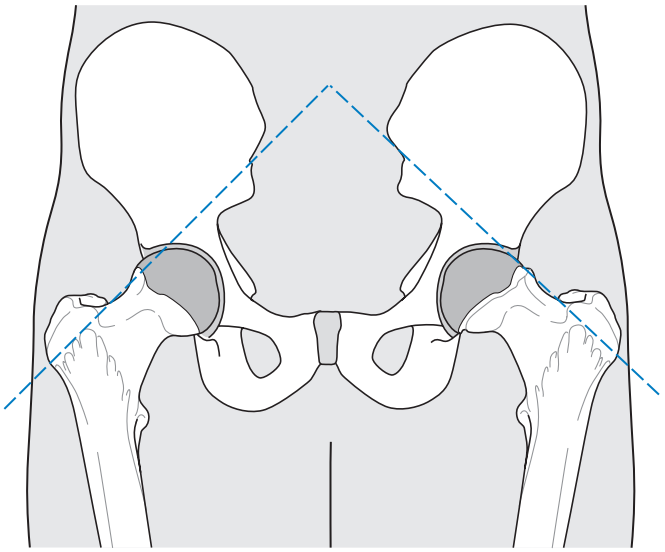

Fig 3 • Normal hip (left) and mild slip (right). In a normal hip, a line drawn along the upper border of the femoral neck bisects a segment of the epiphysis but not in a slip.

Slipped epiphysis

Due to gravity, a lysis of the proximal epiphysial junction results in downwards and backwards slipping of the epiphysis in relation to the neck of the femur. This causes a varus position and slight outwards rotation of the leg – coxa vara in adolescence. It is a disorder of teenagers (aged between 12 and 17 years), with an incidence of 5–10 per 100 000.[42] The epiphysial junction softens for an unknown reason, although there is strong suspicion that some hormone imbalance could cause the weakening.[43,44]

Early symptoms can be mild, variable and insidious in onset. Children presenting with a slipped femoral epiphysis may complain of pain in the affected hip or groin, thigh or knee. The pain may be mild or intermittent, or so severe that the child is unable to bear weight.[45,46]

Gait abnormality is frequently observed: the child may walk with a limp and the affected leg in external rotation. Trendelenburg's sign is usually present. Clinical examination reveals a clear non-capsular pattern with limitation of medial rotation and increase of lateral rotation.[47] This can be observed best during bilateral medial rotation in the prone lying position.[48] Resisted movements are strong and painless. Diagnosis is confirmed by antero-posterior and lateral hip radiographs which demonstrate the posterior and inferior displacement of the epiphysis relative to the metaphysis[49] (Fig. 3).

Treatment and prognosis depend largely on the degree of the slip.[50,51] It is therefore vital to make the diagnosis as soon as possible, and a child or teenager in whom epiphysiolysis is suspected should not be allowed to walk around until the result of radiography is known. *Epiphysiolysis is an orthopaedic medical emergency and requires immediate relief of weight bearing.*[52,53] Treatment is surgical and consists of in situ cannulated-screw fixation[54] of the slipped capital femoral epiphysis or an open replacement of the femoral head.[55,56]

Avulsion fractures about the hip

Avulsion fractures occur more commonly in skeletally immature athletes than in adults because young patients' tendons are stronger than their cartilaginous growth centres. The same stress that causes a muscle strain in an adult can cause an avulsion fracture in an adolescent. These fractures occur at the secondary growth centres, or apophyses, which become separated from the underlying bone. The fractures do not become widely displaced because of the surrounding thick periosteum.[57]

The mechanism of injury is a sudden, violent muscle contraction or excessive repetitive action across the apophysis. Hip avulsion fractures are common in young sprinters, soccer players and jumpers.[58] Patients typically describe local pain and swelling after an extreme effort and report no external trauma.

Physical examination reveals pain during the resisted movements that activate the contractile structures inserting at or nearby the fracture line:

- The anterior superior iliac spine – sartorius
- The anterior inferior iliac spine – rectus femoris
- The ischial tuberosity – hamstring
- The lesser tubercle – psoas.

There is also palpable tenderness (and often ecchymosis) at the specific bony sites (Fig. 4).

Plain radiographs reveal the avulsion fracture. It is useful to compare the injured side with the contralateral side. During the healing phase of an avulsion fracture the abundant reactive ossification in the soft tissues may clinically and radiographically be mistaken for neoplasia.[59]

Treatment is rest. Most patients can be treated non-operatively over 6–12 weeks, although some authors recommend surgery for severely displaced fragments in rare cases.

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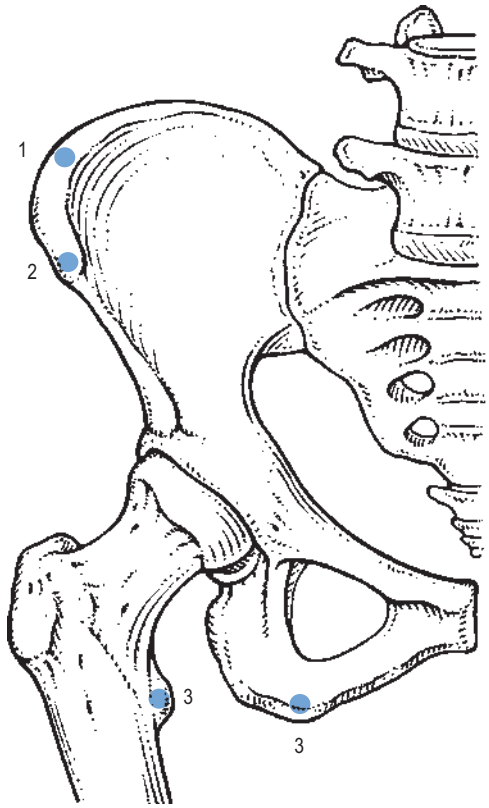

Fig 4 • Bony sites of the pelvis: 1, anterior superior iliac spine;[59,60] 2, anterior inferior iliac spine;[61,62] 3, ischial tuberosity; 4, lesser tubercle.[63]

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Applied anatomy of the knee

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In nearly all circumstances, the knee works in axial compression under the action of gravity. It must therefore reconcile two opposed requirements, namely mobility and stability. This problem is resolved by an ingenious arrangement of ligaments, menisci and tendons: the ligaments and menisci provide static stability and the muscles and tendons dynamic stability. Nevertheless, the exposure of the knee to external forces makes it very vulnerable in many occupations and sports.

The main movement of the knee is flexion–extension; secondary movement – internal and external rotations of the tibia in relation to the femur – is possible only when the knee is flexed. To measure the extent of internal and external rotation, the knee must therefore be flexed to a right angle.

Articular surfaces

During flexion–extension, the knee acts as a hinge joint, whereby the articular surfaces of the femur roll (and glide) over the tibial surface.

The distal femur can be compared with a double wheel, in which the medial and lateral condyles are the components and the intercondylar notch the junction between them (Fig. 1). The condyles are convex in both planes. The medial condyle extends a little more distally than the lateral. The greater prominence of the lateral femoral condyle prevents the patella from sliding laterally.

The tibial aspect of the joint is two curved 'gutters', separated by an anteroposterior eminence. These gutters are not congruent with the corresponding condyles but this lack of compatibility is corrected by the menisci.

The anteroposterior elevation between the tibial condyles corresponds to the femoral intercondylar notch. If the surfaces of the tibial condyles are projected anteriorly, they coincide with the articular surface of the patella which corresponds to, and is almost congruent with, the anterior surfaces of the femoral condyles. If the intercondylar eminence of the tibia is projected anteriorly, its plane is continuous with the vertical ridge on the patella just as the intercondylar notch of the femur continues in the central groove of the patellar surface of the femur. This arrangement resembles a twin-wheel rolling on a central rail (Fig. 1a). During flexion and extension, tibia and patella act as one structure in relation to the femur.[1]

The rounded surfaces of the femoral condyles in relation to the flatter tibial ones might suggest that the former roll during flexion–extension. In fact this is not so. As long ago as 1836 the Weber brothers demonstrated that the femoral condyles roll and slide almost simultaneously, and that these movements are in opposite directions. During flexion, the femoral condyles roll backwards and slide forwards on the tibia, whereas during extension they roll forwards and slide backwards (see Standring, Fig. 82.20). The ratio of rolling to sliding differs with the degree of flexion or extension, which means that during the

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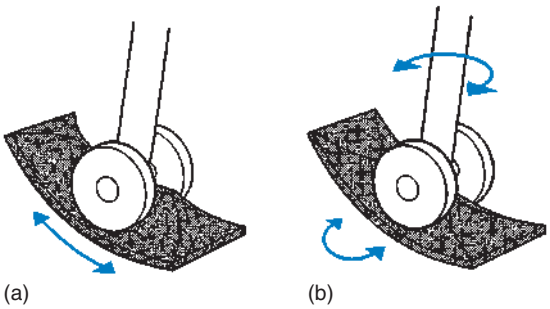

Fig 1 • (a) The knee as a hinge joint: the femoral condyles (twin wheel) in relation to the tibial and patellar surfaces ('rails'). (b) By flattening the anterior and posterior end of the 'rail', rotational movements become possible; the intercondylar spines act as the central pivot.

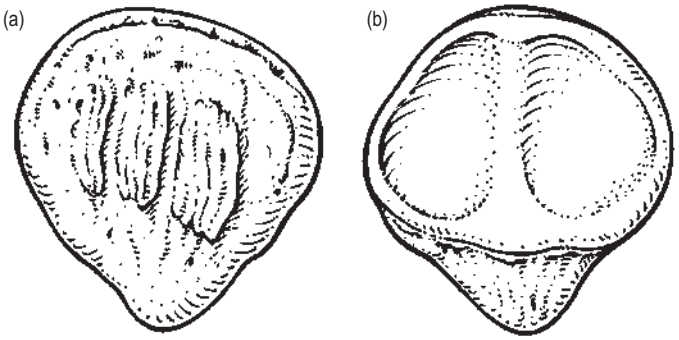

Fig 2 • Surfaces of the patella: (a) anterior; (b) posterior.

first 30° of flexion the movement is almost entirely rolling, whereas at nearly full flexion the condyles slip over the tibial plateau without rolling.[2]

The knee joint is thus primarily a hinge, with the wheel-shaped surfaces of the femoral condyles gliding and rolling in a twin set of concave curved gutters: the tibial and patellar surfaces. However, while this is a satisfactory concept in terms of flexion and extension, in reality the situation is more complex because the knee allows not only gliding and rotation around a horizontal axis but also rotation through a vertical axis, i.e. internal and external rotation of the tibia in relation to the femur. Were the knee to be only as so far described – a hinge joint with a long tibial intercondylar eminence gliding between the two femoral condyles – rotation would be precluded. However, if the anterior and posterior ends are flattened, rotation becomes possible (Fig. 1b). The remaining middle part of the eminence, forming the 'intercondylar spines', is then the central pivot about which the movements of axial rotation occur.

The knee joint is a hinge joint during flexion–extension but in a flexed position modifications enable axial rotation around a central pivot.

The patella

The patella is a flat, triangular bone, situated on the front of the knee joint (Fig. 2). It is usually regarded as a sesamoid bone, developed in the quadriceps femoris tendon. Its convex anterior surface is covered by an expansion from the tendon of the quadriceps femoris which is continuous below with the superficial fibres of the ligamentum patellae. It is separated from the skin by a bursa (prepatellar bursa).

The posterior surface presents above a smooth, oval, cartilaginous area, which is divided by a rounded vertical ridge into a larger, lateral portion, for articulation with the lateral condyle of the femur, and a smaller, medial portion, for articulation with the medial condyle of the femur. Below the articular surface is a rough, convex, non-articular area, the lower half of which gives attachment to the ligamentum patellae; the upper half is separated from the head of the tibia by adipose tissue.

The superior border of the patella is thick, and sloped from behind, downwards and forwards; it gives attachment to that portion of the quadriceps which is derived from the rectus femoris and the vastus intermedius muscles.

The medial and lateral borders are thinner and give attachment to those portions of the quadriceps femoris, which are derived from the vasti lateralis and medialis.

The apex is pointed and gives attachment to the ligamentum patellae.

The patella serves to protect the front of the joint and increases the leverage of the quadriceps femoris by making it act at a greater angle.

The menisci

There are two menisci in the space between the femoral and tibial condyles. They are crescent-shaped lamellae, each with an anterior and a posterior horn, and are triangular in cross section. The superior and inferior surfaces are in contact with the femoral and tibial condyles, respectively, and the peripheral surfaces are adherent to the synovial membrane of the capsule. The anterior and posterior horns are anchored to the tibial condyle in the anterior and posterior intercondylar fossae, respectively. The horns of the medial meniscus are further apart than those of the lateral, which makes the former nearly semilunar and the latter almost circular. The menisci correct the lack of congruence between the articular surfaces of tibia and femur, increase the area of contact and improve weight distribution and shock absorption.[3–6] They also help to guide and coordinate knee motion, making them very important stabilizers of the knee.

Movement between the tibial surface and the menisci is limited by the coronary ligaments connecting the outer

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meniscal borders with the tibial edge (Fig. 3, see Standring, Fig. 82.9). The coronary ligaments of the medial meniscus are shorter (4–55 mm) and stronger than those of the lateral meniscus (13–20 mm).[7] The medial collateral ligament of the knee is attached by its deep fibres to the outer border of the medial meniscus. In contrast, there is no connection between the lateral meniscus and the corresponding collateral ligament (Fig. 4). These anatomical differences between the medial and the lateral meniscus may explain the lesser mobility and the greater vulnerability of the former (see Fig. 3).[8]

There are only a few muscle fibres attached to the menisci. The popliteus sends a fibrous expansion to the posterior border of the lateral meniscus and a few fibres of the semimembranosus tendon run to the posterior edge of the medial meniscus.

Menisci do not contain pain-sensitive structures and are consequently insensitive to trauma. Their outer third has some blood supply and therefore a slight ability to heal. The inner non-vascularized part receives nutrition through diffusion of synovial fluid.[9,10]

Movements of the menisci

The inner sides of the menisci, attached by their horns to the tibial plateau, move with the tibia. The body of each meniscus is fixed around the femoral condyle and moves with the femur. Therefore, during movement between tibia and femur, distortion of the menisci is inevitable. Because the horns of the lateral meniscus are attached closer together and its body is more mobile, distortion is more marked in it.

During flexion of the knee, the body of the meniscus moves posteriorly and during extension it moves anteriorly. In lateral (axial) rotation, the menisci will follow exactly the displacement of the femoral condyles, which means that the lateral meniscus will be pushed forwards on the tibia and the medial meniscus will be pulled backwards (Fig. 5a). On the medial aspect the anterior part of the medial coronary ligament then comes under tension. The same effect occurs in the medial rotation: the medial meniscus is pressed forwards and the

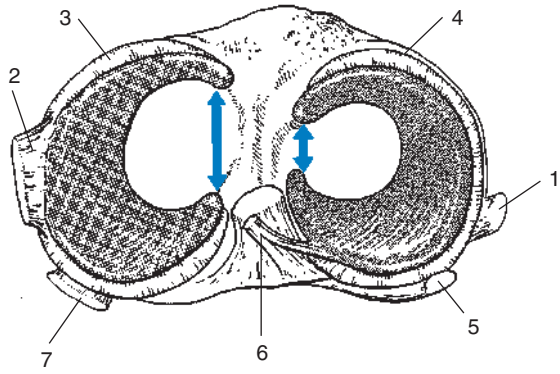

Fig 3 • Superior view of the menisci and their attachments (right knee). 1, lateral collateral ligament; 2, medial collateral ligament; 3, medial coronary ligament; 4, lateral coronary ligament; 5, popliteus tendon; 6, posterior cruciate ligament; 7, semimembranosus tendon.

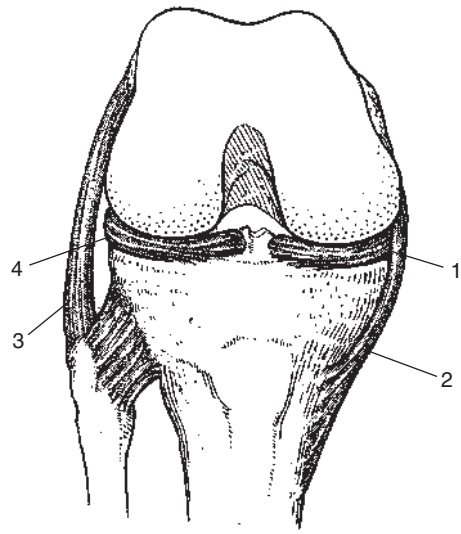

Fig 4 • Anterior view of the menisci and their relations with the collateral ligaments: 1, medial meniscus; 2, medial collateral ligament; 3, lateral collateral ligament; 4, lateral meniscus.

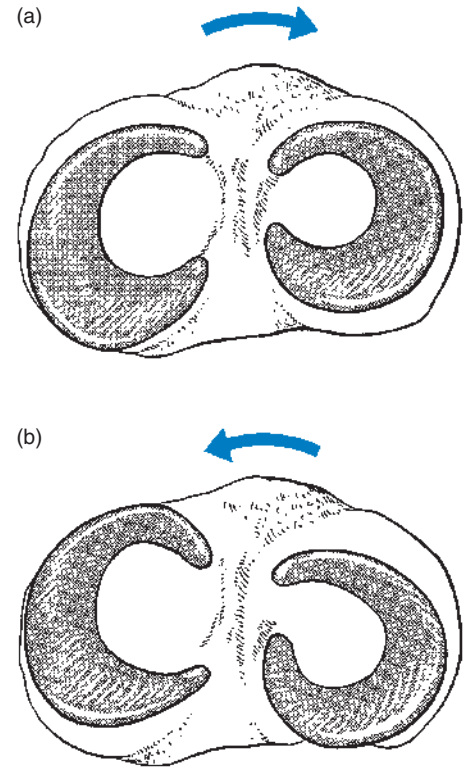

Fig 5 • Relative movement between tibia and menisci during rotation under 90° of flexion: (a) lateral rotation of the tibia; (b) medial rotation of the tibia.

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lateral meniscus drawn backwards (Fig. 5b), which causes tension in the anterior part of the lateral coronary ligament.

Joint capsule and ligaments

Capsule

The capsule connects the distal end of the femur with the proximal border of the tibia. It consists of a fibrous sleeve and a synovial membrane, the attachments of which do not always coincide: only the synovial membrane is adherent to the peripheral surface of the menisci. Reinforcements in, or along the fibrous capsule keep the bones in contact, giving the joint passive stability.

The tibial attachment of the capsule (Fig. 6a) is attached to the borders of the articular surfaces of the tibia and therefore follows its anterior, medial and lateral edges. At the posterior border the synovial membrane follows the edges of the medial and lateral condyles, to form a loop around the insertion of the anterior cruciate ligament. At the anterior border the synovial membrane is projected backwards by a sizeable pad of adipose tissue – the infrapatellar pad.

The femoral attachment of the capsule also follows the articular surfaces of the femur, although there are some points to be noted:

- Anteriorly the capsule is attached proximal to the edge of the articular surface, thus forming the suprapatellar pouch (Fig. 6b).
- On the lateral condyle the attachment of the fibrous capsule lies above the insertion of the popliteus tendon, which makes the latter lie intra-articularly, covered only by the synovial membrane (Fig. 6c).
- Posteriorly and following the articular surfaces of the femoral condyles, the attachment of the synovial membrane dives into the intercondylar notch to form a loop around the femoral insertions of the cruciate ligaments. The cruciate ligaments are thus kept outside the capsule by an invagination of the synovial membrane, which forms a partition in the sagittal plane of the joint space.

At the patella, the capsule follows the margins of the bone.

Ligaments

The medial ligamentous complex

The medial ligamentous complex has two layers: the deeply situated capsular reinforcements with an anterior, medial and posterior part, and the strong and more superficially localized medial collateral ligament (MCL). The MCL is the primary stabilizer of the medial side of the knee. It is a broad, flat and almost triangular band with a large femoral attachment on the posterosuperior aspect of the epicondyle. Its fibres run obliquely anteriorly and inferiorly to insert at the medial aspect of the tibia, just behind and slightly under the insertions of the semitendinosus muscle (see Gosling et al, Fig. 6.79). The anterior fibres of the ligament are separated from those of the deep capsular reinforcements and therefore the anterior border of the ligament can be palpated easily. The posterior fibres, however, blend intimately with the capsule and with the medial border of the medial meniscus (Fig. 7).[11]

The strong MCL stabilizes the knee against excessive valgus forces and external rotation. Although it slackens during flexion, the posterior fibres attached to the meniscus remain taut, an observation that is important in the consideration of chronic ligamentous adhesions of the MCL (see p. 700).

The lateral collateral ligament

The lateral collateral ligament (LCL) is part of the so-called lateral quadruple complex (biceps tendon, iliotibial tract, popliteus and LCL), responsible for the lateral stability of the knee. It is round in cross-section and runs from the lateral epicondyle of the femur to the head of the fibula, deep to the

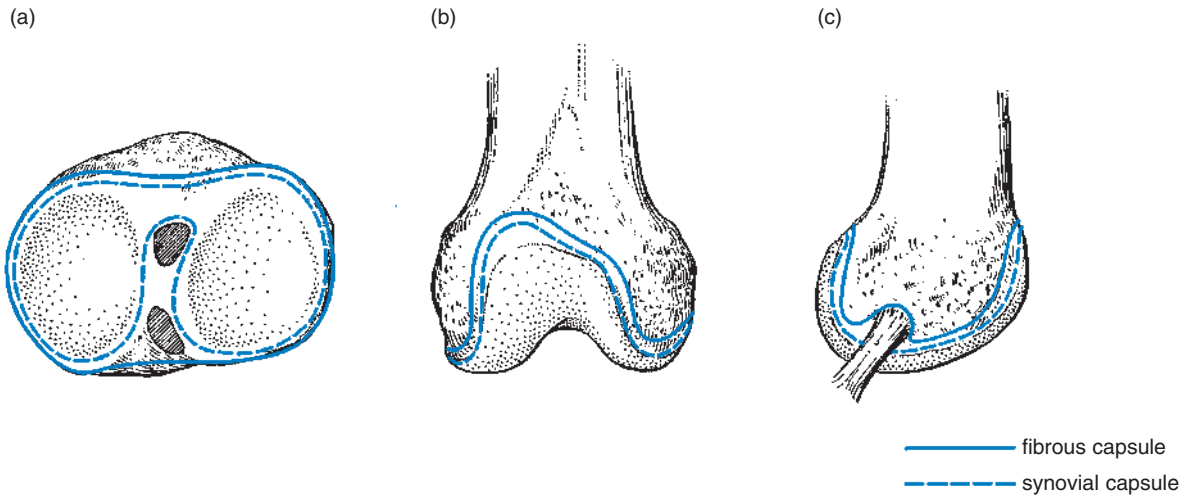

Fig 6 • (a) Insertions of the capsule into the tibia, and its relations with the cruciate ligaments (shaded). (b) Anterior and medial insertions of the capsule into the femur. (c) Lateral insertion and relationship of the capsule and the popliteus tendon.

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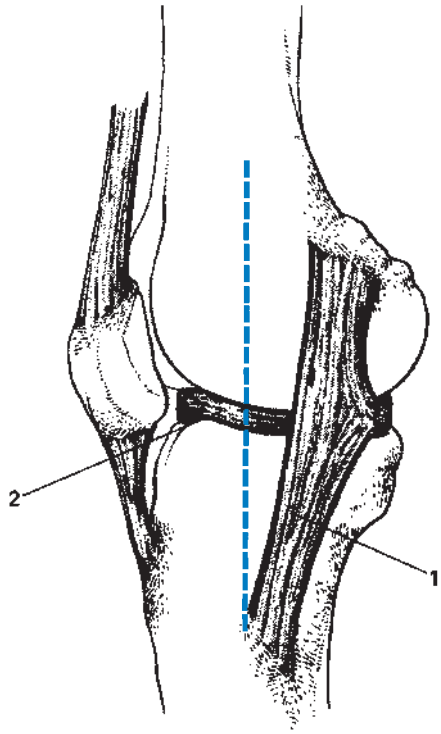

Fig 7 • The medial collateral ligament (1) and medial meniscus (2).

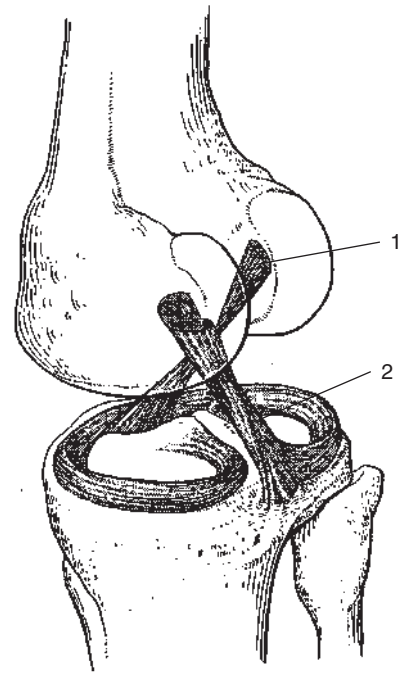

Fig 8 • Posteromedial view of the cruciate ligaments: 1, anterior; 2, posterior.

insertion of the biceps. The ligament lies completely free, separated from the capsule and the lateral meniscus by the popliteus tendon (see Figs 3, 10, see Gosling et al, Fig. 6.78). It stabilizes the knee against excessive varus movement. A small bursa lies between the popliteus tendon and the LCL.

The posterior capsular reinforcements

The posterior capsule is strengthened by two irregular ligamentous structures: the oblique popliteal and the arcuate popliteal ligaments. The former is an expansion of the semimembranosus tendon and reinforces the central posterior part of the joint capsule. The fibres of the latter run obliquely medially and upwards from the rear side of the fibular head to the posterior aspect of the femur.

The cruciate ligaments

Although the cruciate ligaments lie in the centre of the joint (Fig. 8), they remain extrasynovial because of the posterior invagination of the synovial membrane. They ensure the anteroposterior stability of the knee and, together with the collateral ligaments, prevent rotational movements during extension.

The anterior cruciate ligament

This is attached to the anterior intercondylar area of the tibia, between the anterior horns of the medial and lateral menisci. Its fibres run laterally and posteriorly to the internal aspect of the lateral condyle of the femur.

It has three coiled bundles (anteromedial, intermediate and anterolateral), which spiral from one insertion to the other. In extension, the anterior bundle is tight but in flexion the posterior fibres are stretched, which causes the anterior cruciate to be tightest at the extremes of range. During flexion, the femoral condyles tend to roll backwards over the tibia. This is checked by the anterior cruciate ligament, which induces a simultaneous anterior gliding of the femur. The anterior cruciate ligament also prevents excessive external rotation and varus movement of the tibia.

The posterior cruciate ligament

The tibial attachment of the posterior cruciate ligament is at the posterior intercondylar area, but fans out at the posterior border of the tibial plateau. The ligament runs in a medial and anterior direction and crosses the anterior cruciate ligament from medial and from behind to insert at the lateral surface of the medial condyle, deeply in the intercondylar fossa. Like the anterior cruciate, the posterior cruciate ligament has a complex architecture but is twice as strong. It is a basic knee stabilizer, tightest in mid-position. During extension, the posterior cruciate ligament pulls on the femur, causing backwards sliding during its anterior rolling. The posterior cruciate ligament also prevents anterior gliding of the femur during squatting, resists hyperextension and has an important role in the medial stability of the knee.[12]

The coronary ligaments

The meniscotibial or coronary ligaments connect the periphery of the menisci with the edge of the tibial condyle (Fig. 9).[13,14] Some anatomists do not consider them to be true ligaments, but merely reinforcements in the synovial membrane.[15]

The fibres of the medial coronary ligament are 4–5 mm long, while those of the lateral coronary ligament are 2 cm long

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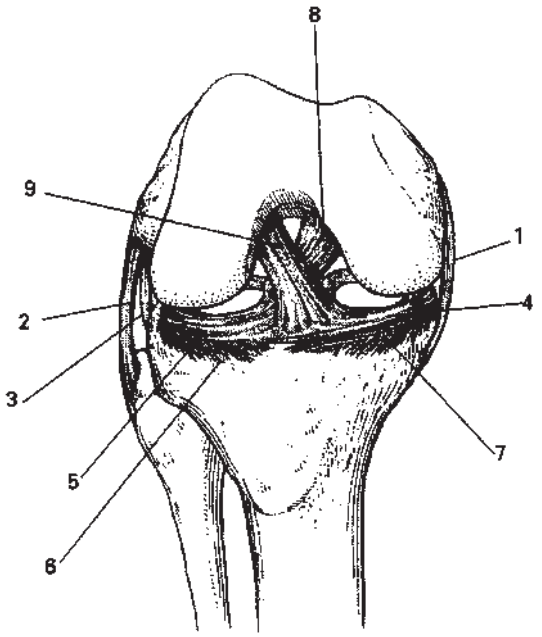

Fig 9 • Anterior view of the coronary ligaments, with the knee in flexion: 1, medial collateral ligament: 2, lateral collateral ligament; 3, popliteus tendon; 4, medial meniscus; 5, lateral meniscus; 6, lateral coronary ligament; 7, medial coronary ligament; 8, posterior cruciate ligament; 9, anterior cruciate ligament.

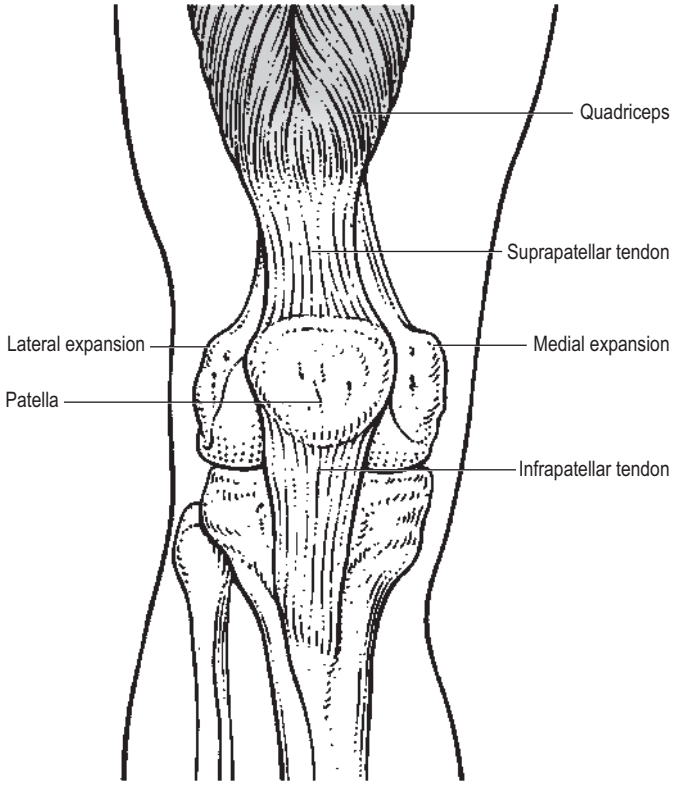

Fig 10 • The extensor mechanism of the knee.

at the front and 1.3 cm at the back. The fibres of the lateral ligament are also looser than the medial ones.

The medial coronary ligament is stretched during external rotation, while the lateral is stretched during internal rotation.

Muscles and tendons

The extensor mechanism (Fig. 10)

The extensor of the knee is the quadriceps; it consists of four muscle bellies. Three are monoarticular muscles – the vastus medialis, the vastus intermedius and the vastus lateralis. One is biarticular and spans knee and hip joints – the rectus femoris. These separate bellies have a common tendon, which inserts into the anterior tibial tuberosity. Imbedded in the tendon is the patella, a triangular sesamoid bone. The function of the patella is to increase the efficiency of the quadriceps contractions.

It is important to note that the patella lies fully within the tendon, which means there are tendon fibres all around the patellar edge. This has significant clinical consequences, since a tenoperiosteal tendinitis at the patella will occur not only at the distal but also at the proximal and sometimes even at the medial and lateral sides of the patella. For convenience, we call the part of the tendon above the patella the suprapatellar tendon (quadriceps tendon), the inferior part the infrapatellar tendon (or patellar ligament), and the medial and lateral fibres the medial and lateral quadriceps expansions; they are, nevertheless, only parts of a single tendon.

The biarticular nature of the rectus femoris and the fact that it runs anterior to the flexion–extension axis of the hip gives it the specific function of a hip flexor (see online chapter *Applied anatomy of the hip and buttock*).

Some deeply situated fibres of the vastus intermedius run to the superior capsule of the knee joint, where they pull the loose capsule away from the joint and prevent the suprapatellar pouch from being pinched during knee motion.

Another, but weak, extensor of the knee is the tensor fasciae latae, which acts through the iliotibial tract, but only when the knee is extended. In flexion greater than 30°, the iliotibial tract becomes a weak knee flexor and an external rotator. The main function of the tract is to create static lateral stabilization of the knee.

Flexors of the knee

The knee flexors are the hamstrings (semitendinosus, semimembranosus and biceps femoris), the sartorius and the gracilis, the popliteus and the gastrocnemii:

- *The gastrocnemii*, although strong plantiflexors and invertors of the heel, are only weak flexors of the knee (see Standring, Fig. 82.3). They also help in active stabilization of the joint. The medial head is a weak internal and the lateral head a weak external rotator.
- *The semitendinosus, gracilis and sartorius* insert under and in front of the medial condyle of the tibia in the so-called 'pes anserinus', just medial to the tibial tuberosity and

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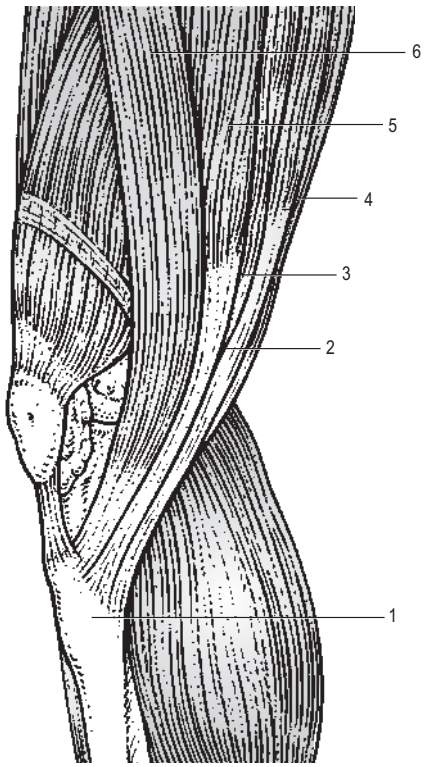

Fig 11 • Medial view of the knee showing the relations between the tendons: 1, insertion of the pes anserinus; 2, semitendinosus; 3 and 4, semimembranosus; 5, gracilis; 6, sartorius.

anterior to the tibial insertion of the MCL. They are flexors and internal rotators of the knee (Fig. 11).

- *The semimembranosus* inserts at the medial condyle of the tibia but has some fascicles attaching to the oblique popliteal ligament and a few fibres attaching to the posterior edge of the medial meniscus. It is a flexor and an internal rotator.
- *The popliteus* muscle originates within the joint at the lateral epicondyle of the femur (Fig. 12, see Standring, Fig. 83.11, see Gosling et al, Fig. 6.77). Other origins are situated at the dorsal aspect of the capsule and the lateral meniscus. The muscle belly lies deeply in the popliteal fossa under the lateral gastrocnemius and the plantaris. The broad insertion is at the upper and posterior aspect of the tibia. The popliteus is a flexor and an internal rotator of the knee. It also spans the posterior capsule of the knee and pulls the lateral meniscus in a posterior direction during flexion. Another important function of the muscle is to prevent the femur from slipping forwards on the tibia during squatting. This active function of the popliteus is very similar to the static role of the posterior cruciate ligament.
- *The biceps femoris or lateral hamstring* inserts at the top and posterior aspect of the fibular head in front of and behind the insertion of the lateral collateral ligament (see Fig. 12). Some fibres also attach to the posterolateral aspect of the tibia and the lateral part of the joint capsule. It is a strong flexor and external rotator of the knee.

Muscular function of the knee is summarized in Box 1.

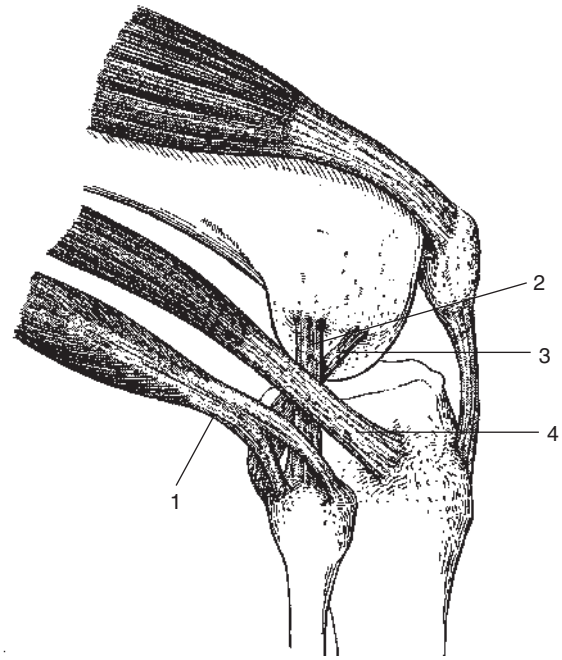

Fig 12 • Lateral view of the knee, showing the relations between the lateral muscles and ligaments: 1, biceps femoris; 2, lateral collateral ligament; 3, popliteus; 4, iliotibial tract.

Box 1

Summary of muscular function

Extension

Quadriceps femoris
(Iliotibial tract)

Flexion

Internal rotators
- Gracilis
- Sartorius
- Semitendinosus
- Semimembranosus
- Popliteus

External rotators
- Biceps femoris
- Iliotibial tract

Stretching the capsule

Medial
- Semimembranosus

Lateral
- Popliteus

Anterior
- Vastus intermedius

Innervation of the muscles

This is detailed in Table 1.

Nerve structures and blood vessels: the popliteal fossa

The lozenge-shaped popliteal fossa is an anatomical space of particular importance because it contains the vessels and nerves of the lower limb (Fig. 13, see Standring, Fig. 82.2). Its lower borders are formed by the heads of the gastrocnemius. The upper lateral border is the biceps femoris and the upper medial border the semitendinosus and semimembranosus muscles. Its

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Table 1 Innervation of the muscles

	Peripheral nerve	Spinal innervation
Extension		
Quadriceps	Femoral	L2, L3, L4
Flexion		
Semitendinosus	Tibial	L5, S1, S2
Semimembranosus	Tibial	L5, S1, S2
Gracilis	Obturator	L2, L3, L4
Sartorius	Femoral	L2. L3
Popliteus	Tibial	L4, L5, S1
Biceps femoris	Sciatic	L5, S1
Tensor fasciae latae	Superior gluteal	L4, L5
Gastrocnemius	Tibial	S1, S2

floor is the posterior capsule and the popliteus muscle. The popliteal fossa is bounded posteriorly by the deep popliteal fascia.

The lozenge is traversed vertically by (from lateral to medial) the tibial nerve, the popliteal vein and the popliteal artery. The common peroneal nerve descends along the inner border of the biceps. At the head of the fibula it splits into a deep peroneal branch, which curls around the fibular head to enter the anterior compartment of the leg, and a superficial peroneal branch, which supplies the peroneal muscles in the lateral compartment.

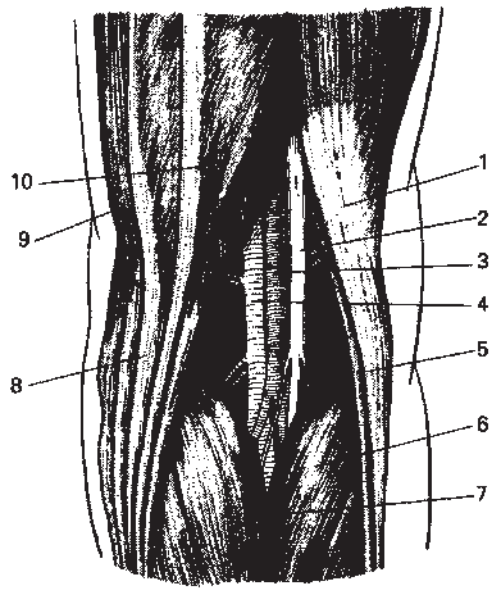

Fig 13 • The popliteal fossa: 1, biceps femoris; 2, tibial nerve; 3, popliteal vein; 4, popliteal artery; 5, common peroneal nerve; 6, lateral gastrocnemius; 7, medial gastrocnemius; 8, semimembranosus; 9, gracilis; 10, semitendinosus.

The upper tibiofibular joint

This joint is separate from the knee. The capsule is reinforced in front and behind by two tibiofibular ligaments. Little movement takes place at the tibiofibular joint. The biceps femoris can pull the fibular head slightly backwards during active knee flexion, and slight passive upwards and downwards movements of the fibula may follow valgus and varus movements of the talus.

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Disorders of the inert structures: ligamentous instability

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Ligamentous instability

If the knee becomes unstable after a ligamentous injury, permanent problems may result, especially if the patient is an athlete with high functional demand.

If the history indicates that there is possibly instability, an examination to detect the exact location and degree of this is performed. It is important to make this supplementary examination only when symptoms and signs found in the routine clinical assessment indicate ligamentous instability to be the cause of disability. Indeed, it is important to realize that not every lengthened ligament leads to problems.[1] If the athlete's muscles are strong enough to provide dynamic stability, a slightly unstable knee will not cause trouble. Conversely, it is quite possible that an athlete with slight laxity of one or more ligaments has a chronic painful lesion as well. If the diagnosis of the latter is missed because the routine functional examination is not performed, it may well be that the patient is sent for surgery, which will solve the slight instability but does not cure the main problem.

Types of instability

Knee instability is the result of loss of static and dynamic function. Stability depends on the tautness of the ligaments, congruency of joint surfaces, effectiveness of the menisci and the well-balanced action of all musculotendinous units acting across the knee joint. Of these structures, muscles and tendons play a central role in joint stabilization. They are said to be the 'first defenders' in distortion and loading situations.[2]

Degree of instability

The degree of instability is demonstrated during stress tests and can be graded on the following scale (Fig. 1):

- *mild/1+* = 5 mm or less separation of joint surfaces
- *moderate/2+* = between 5 and 10 mm separation
- *severe/3+* = greater than 10 mm separation.

This scale is not very precise but is effective for clinical and therapeutic purposes. It should be remembered, however, that under anaesthesia the results of the stress tests are often exaggerated so that patients with 2+ instability often show 3+ instability in the test.

Classification of instability

Instability may be classified as straight or rotatory.

Straight instability

This is defined as an increased range of angular movement in the frontal plane, i.e. valgus/abduction or varus/adduction movement (Fig. 2), or an increased range of gliding movement in the sagittal plane, i.e. the simultaneous forward or backward gliding movement of the tibial condyles in relation to the femoral condyles (Fig. 3).

Rotatory instability

This implies increased rotation movement of the tibia on the femur. The posterior cruciate ligament, located in the centre of the joint, is the fundamental stabilizer and is the axis of the joint, both in flexion–extension and in rotation. Consequently, this ligament is always intact in rotatory instabilities; from the moment the ligament is completely torn, there is no longer a centre of rotation and any kind of straight instability can result.[3,4]

There are three types of rotatory instability:

- *Anteromedial rotatory instability*: there is an abnormal forward gliding movement of the medial tibial plateau

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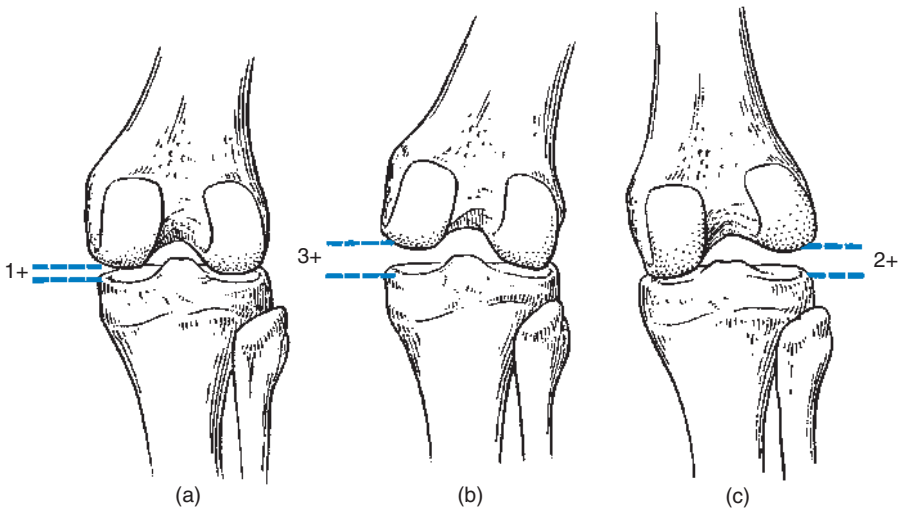

Fig 1 • Degrees of straight instability in the frontal plane: A, mild; B, severe; C, moderate.

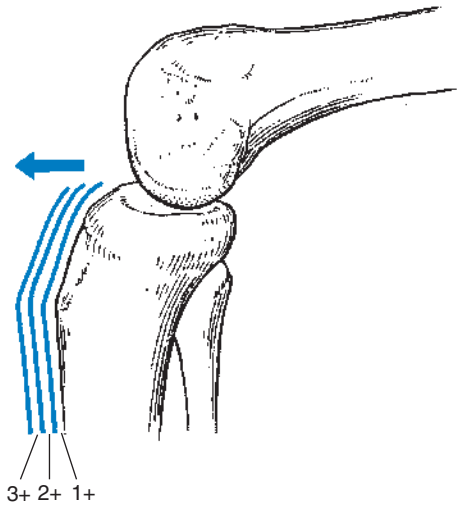

Fig 2 • Degrees of straight instability in the sagittal plane.

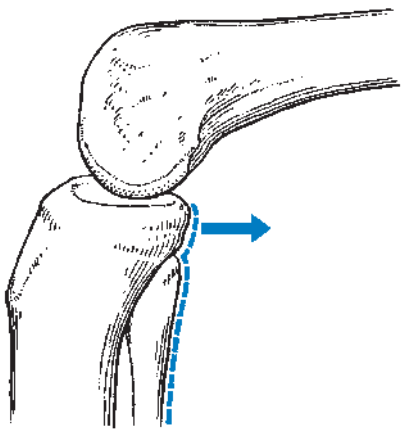

Fig 3 • Straight instability: increased range of gliding movement in the sagittal plane.

with respect to the medial femoral condyle, while the lateral tibial plateau retains a relatively normal relationship with the lateral femoral condyle (Fig. 4b).

- *Anterolateral rotatory instability*: there is an abnormal forward gliding movement of the lateral tibial plateau with respect to the lateral femoral condyle, while the medial tibial plateau retains a relatively normal relationship with the medial femoral condyle (Fig. 4c).
- *Posterolateral rotatory instability*: there is an abnormal backward gliding movement of the lateral tibial plateau with respect to the lateral femoral condyle. Again, as in anterolateral rotatory instability, the medial tibial plateau remains in normal contact with the corresponding femoral condyle (Fig. 4d).

Various combinations of these rotatory instabilities can occur. The most commonly encountered are combined anterolateral and anteromedial, and combined anterolateral and posterolateral.

Posteromedial rotatory instability does not exist because an intact posterior cruciate ligament prevents any backward gliding movement of the medial tibial condyle in relation to the medial femoral condyle.

Functional examination

If the history or routine clinical examination indicates the possibility of instability, a supplementary examination is performed to detect the exact localization and degree of ligamentous insufficiency.

Acute stage

In the acute stage, only the first few hours after the accident are suitable for clinical detection of ligamentous laxity. After this, effusion and muscle spasm resulting from the capsulitis will prevent a detailed examination. This is particularly the case in lesions of the medial collateral ligament because of its close relationship with the capsule. So assessment should be made as soon as possible, preferably at the scene of the

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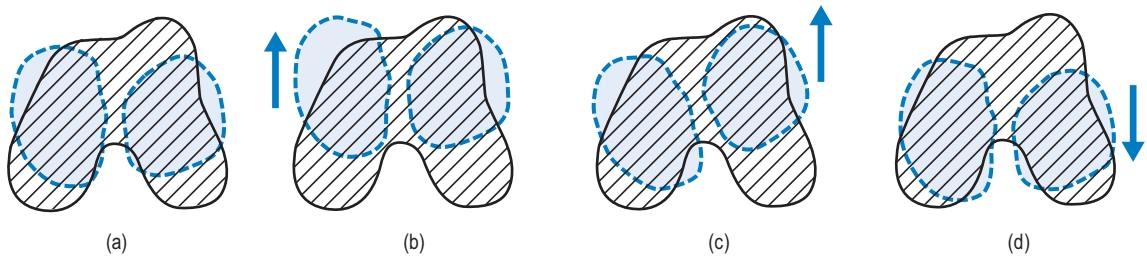

Fig 4 • Rotatory instability: (a) starting position; (b) anteromedial; (c) anterolateral; (d) posterolateral.

accident. If intra-articular bleeding is present, which especially accompanies tears of the anterior and posterior cruciate ligaments as well as of the medial collateral ligament, the first few minutes are often the only time available to make a proper clinical assessment of ligamentous laxity. When time has elapsed, examination under general anaesthesia, eventually followed by arthroscopy, is the only course of action. However, if the lesion seems to be less serious, supplementary examination can wait until a proper clinical evaluation can be made.

Chronic stage

In the chronic stage thorough clinical examination suffices to estimate, in almost all instances, the degree of functional instability and to determine the type of treatment required.

Tests

Passive abduction or valgus stress test in 30° of flexion

The patient is placed supine on the couch, the head resting on a pillow to enable observation of the examiner without active raising of the head (which often causes tightening of the hamstrings). The thigh rests on the couch and the lower leg hangs over the side, with the knee in 30° of flexion. One hand is placed about the lateral aspect of the knee, the other grasps the lower leg at the ankle (Fig. 5). Then abduction stress is applied gently and repeatedly, gradually increasing up to the point of pain. The degree of movement and the end-feel are estimated.

The normal limb is assessed first, especially in acutely injured patients, because this shows the patient that the examination will not be rough and is unlikely to be painful. The examiner also gets a measure of the stability of the unaffected knee.

Instability indicates a tear limited to one or more of the medial compartment ligaments, i.e. the medial collateral ligament, meniscotibial or meniscofemoral capsular ligaments and the posterior oblique ligament.

If the abduction stress test in full extension also shows instability, the posterior cruciate ligament is probably also ruptured. It should be borne in mind that, in the fully extended position, an intact posterior cruciate ligament still holds the joint surfaces in firm apposition, even in complete tears of the medial compartment ligaments.

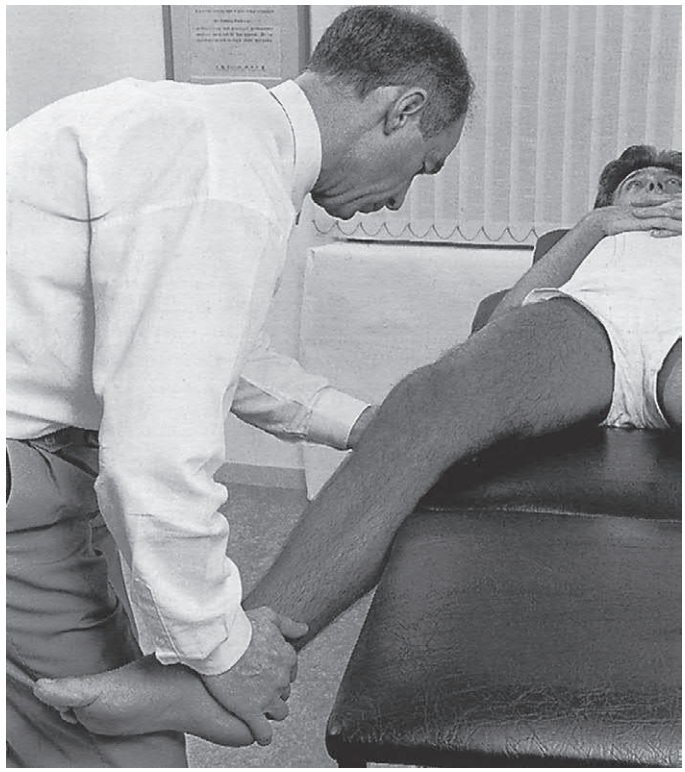

Fig 5 • Passive abduction or valgus stress test in 30° of flexion.

Passive adduction or varus stress test in 30° of flexion

This test is performed in the same position as the previous one, but the hands are changed; so one hand is placed about the medial aspect of the knee while the other grasps the lower leg at the outer side of the ankle (Fig. 6). The degree of movement and end-feel are estimated.

Adduction stress is applied to test the stability of the lateral compartment ligaments: the lateral collateral ligament, meniscotibial or meniscofemoral capsular ligaments and the arcuate ligament.

Instability of the lateral compartment is less common but causes significantly more disability than a comparable amount of instability medially.

Lateral instability cannot be detected when the test is performed with the knee in full extension because the intact tight posterior cruciate ligament precludes any movement in this

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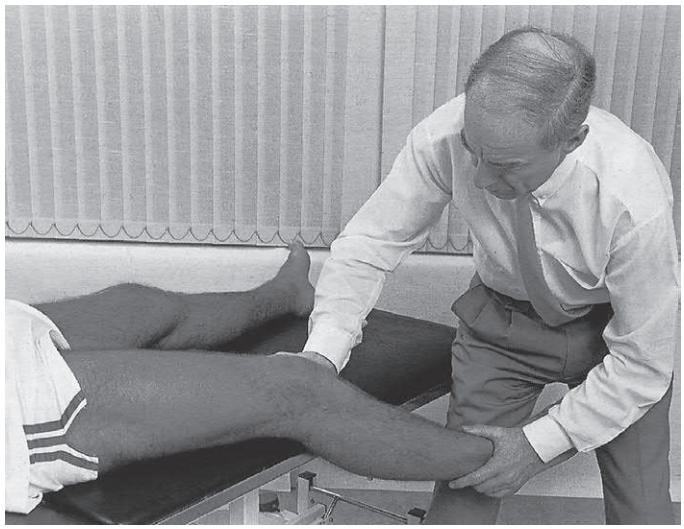

Fig 6 • Passive adduction or varus stress test in 30° of flexion.

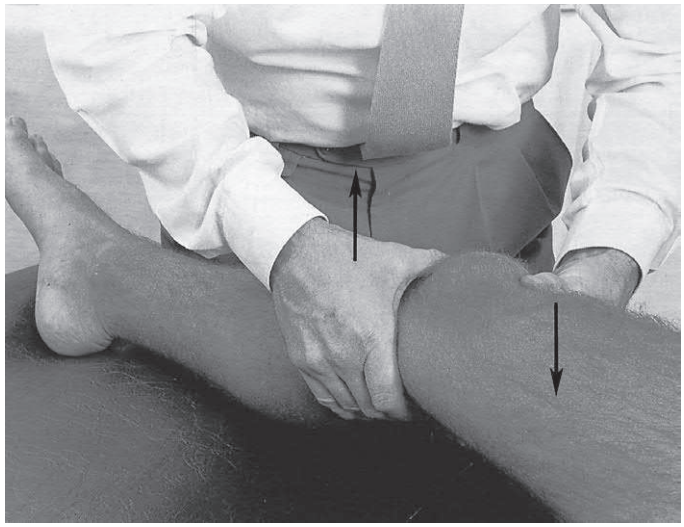

Fig 7 • Lachman test.

position. If an adduction stress test in full extension shows lateral instability, rupture of both the lateral compartment ligaments and the posterior cruciate ligament should be feared.

Lachman test

The test is performed with the patient supine and the knee in 20–30° of flexion. The distal femur is grasped from the lateral side to obtain control just above the lateral femoral condyle. The examiner uses the opposite hand to displace the proximal tibia directly anteriorly (Fig. 7).

In acute rupture of the anterior cruciate ligament this test is preferred to the anterior drawer test in the 90° flexed position because muscle defence by the hamstrings may prevent the forward movement of the tibia and give rise to a false-negative result.[5] Liljedahl *et al*[6] found only 3 of 35 patients to have a positive anterior drawer test in 90° of flexion, whereas with anaesthesia 31 and on arthrography 29 were positive.

Anterior drawer test in external and internal rotation

The patient is positioned as for the abduction–adduction stress tests. The hip is flexed to 45°, the knee to 80–90°. The foot is placed on the couch, well fixed by a portion of the examiner's buttock resting on the dorsum of the forefoot.

The examiner places both hands around the upper part of the tibia with the index fingers palpating the hamstring tendons to make sure they are relaxed. Both thumbs are placed at the anterior border of the joint so that they can estimate the range of movement. Then the proximal part of the lower leg is pulled forward repeatedly, first gently then with a somewhat stronger pull.

The test is first performed with the lower leg and foot externally rotated beyond the neutral position and as far as is comfortably possible, then internally rotated (Fig. 8).

The findings are compared with those of the basic examination, for example in 0° rotation, as well as with the other knee.

With the tibia in external rotation

Anteromedial rotatory instability is indicative of a tear of one or more of the medial compartment ligaments: the medial collateral ligament, meniscotibial or meniscofemoral capsular ligaments and the posterior oblique ligament. An associated anterior cruciate ligament tear increases the amount of subluxation of the medial tibial condyle. This ligament is the second line of defence; in an acute isolated tear of the anterior cruciate the anterior drawer test will be negative.

With the tibia internally rotated

In patients with a medial compartment laxity, the test becomes negative because of the increased tension of the posterior cruciate ligament. Simultaneous forward subluxation of the tibial condyles indicates that the posterior cruciate ligament is torn. In this case, the rotational element is eliminated and straightforward instability exists.

Isolated forward subluxation of the lateral tibial plateau, for example anterolateral rotatory instability, can only be demonstrated if the knee is held in the neutral position, because internal rotation of the tibia will tighten an intact posterior cruciate ligament and prevent any subluxation. This type of instability is indicative of a tear of the middle third of the lateral capsular ligaments.

Prone rotation test[7]

Sometimes doubt remains as to whether the range of rotation has been increased. A decisive assessment can be made with the patient prone and the knees flexed to a right angle (Fig. 9). The movement of external or internal rotation is performed bilaterally and the range of movement is assessed by the twisted position of the feet.

An increased range of lateral rotation results from a lack of stability of inert structures at the medial compartment (medial collateral ligament, meniscotibial and meniscofemoral capsular ligaments and the posterior oblique ligament) and/or the arcuate complex (arcuate ligament, lateral collateral ligament). An associated tear of the anterior cruciate ligament still increases the amount of rotation.

An increased range of medial rotation is indicative of laxity of the middle third of the lateral capsular ligaments, the anterior and the posterior cruciate ligaments.

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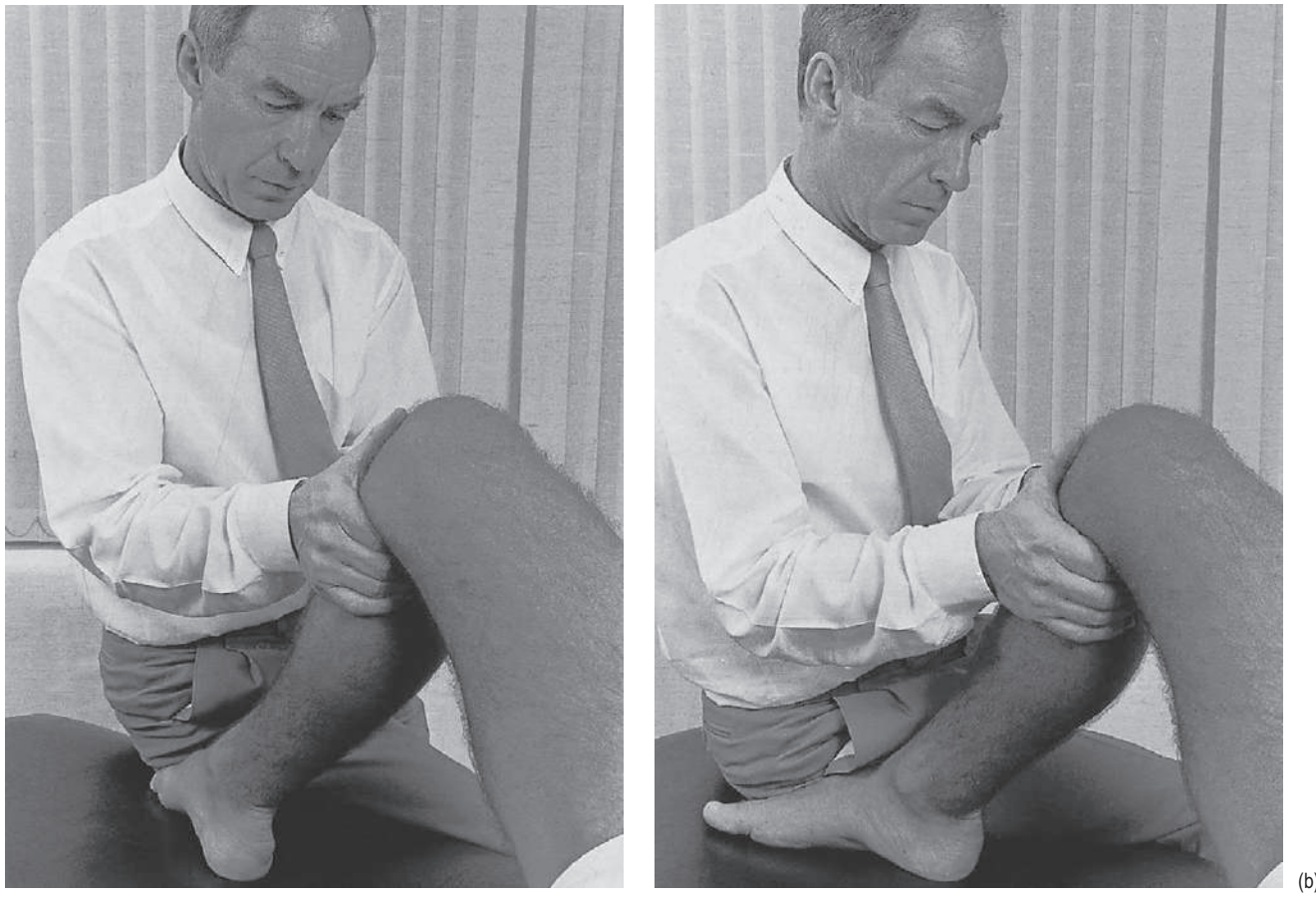

(a) (b)

Fig 8 • Anterior drawer test in external (a) and internal rotation (b).

Posterior drawer test

The patient is in supine position with the hip flexed to 45° and the knee to 90°. The foot is flat on the couch, in neutral position. The heels of both hands are placed anteriorly against the proximal part of the tibia (Fig. 10). Both thumbs at the anterior border of the joint can estimate the range of movement. The proximal part of the tibia is then pushed backwards repeatedly, first gently then with a strong jerk.

The posterior drawer test, which includes palpation of the tibia–femur step-off, was recently demonstrated to be the most sensitive and specific clinical test for posterior cruciate ligament deficiency (accuracy 96% with a 90% sensitivity and a 99% specificity).[8]

'Jerk' test and pivot-shift test

The patient lies supine, the hip flexed to about 45°, the knee to 90°. The examiner supports the patient's leg, with one hand at the foot, the other at the proximal end of tibia and fibula. The hand at the foot rotates the tibia slightly internally while the other hand exerts a mild valgus stress at the knee (Fig. 11). Extreme internal rotation may dampen a shift significantly. Maintaining slight internal rotation and mild valgus stress, the examiner gradually extends the knee.

A positive result is indicated if, on attaining about 30° of flexion, relocation occurs with a sudden movement, which is called a 'jerk'. The forwards shift can be seen and felt by the examiner. At the same moment, the patient will recognize the feeling of instability.

The test demonstrates an anterolateral rotatory instability because of a tear in the middle third of the lateral capsular ligaments. Frequently, the anterior cruciate ligament and/or menisci are also ruptured and increase the instability.[9]

Anterolateral instability can also be demonstrated by carrying out the manœuvre in the reverse direction, starting from a position of a few degrees of flexion: the pivot-shift test.

Bach *et al*[10] found that the degree of pivot shift, probably because of the role of the iliotibial band, strongly correlates with the position of the hip and knee joint. They advised 30° of hip abduction and 20° of tibial *external* rotation as the position in which instability is most clearly demonstrated.

The examiner stands at the foot of the couch in order to monitor hip position carefully. The ipsilateral hand cradles and holds the foot in external rotation. The other hand, slightly supinated, holds the proximal and lateral leg. An axial load and valgus movement are applied simultaneously as the knee is slowly flexed passively from the extended position.

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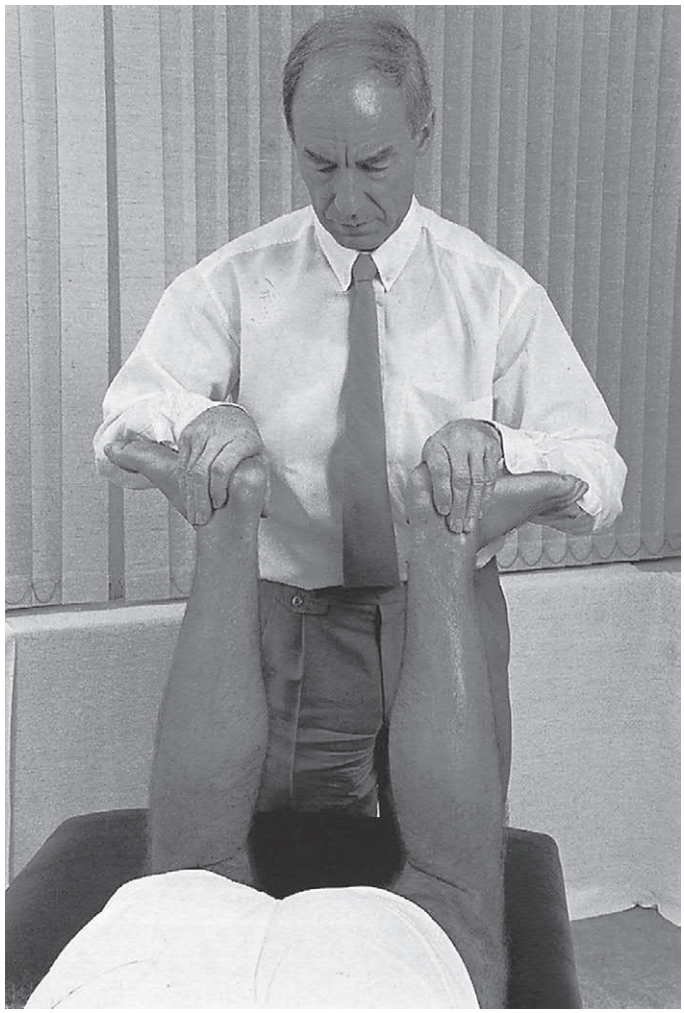

Fig 9 • Prone rotation test.

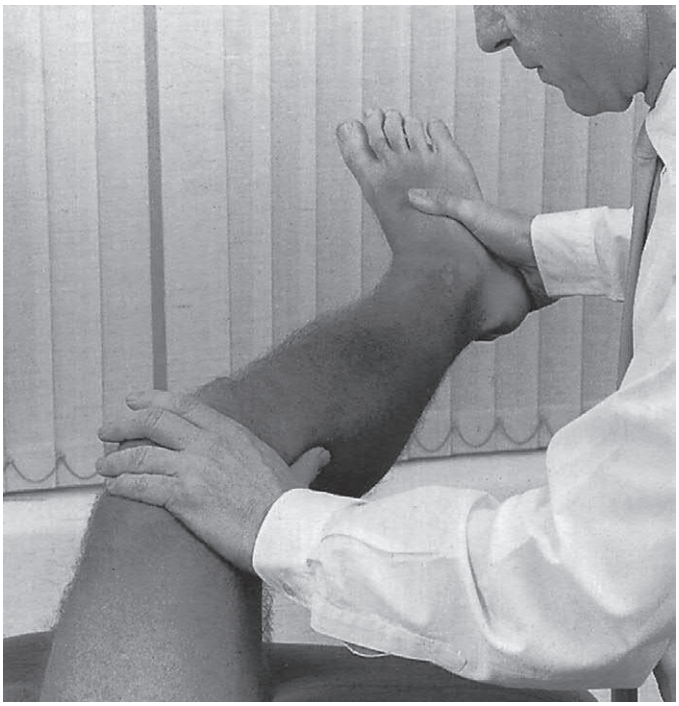

Fig 11 • The 'jerk' test.

Practitioner's checklist

- An associated acute partial tear of the medial compartment ligamentous structures makes the test too painful to undertake
- A false-positive test may occur in the presence of a torn meniscus
- In children, and in patients who have mild to moderate congenital genu recurvatum without symptoms, it is normal to find the 'jerk' and pivot-shift tests, and the anterior drawer test with the tibia in 0° rotation to be mildly positive on both sides

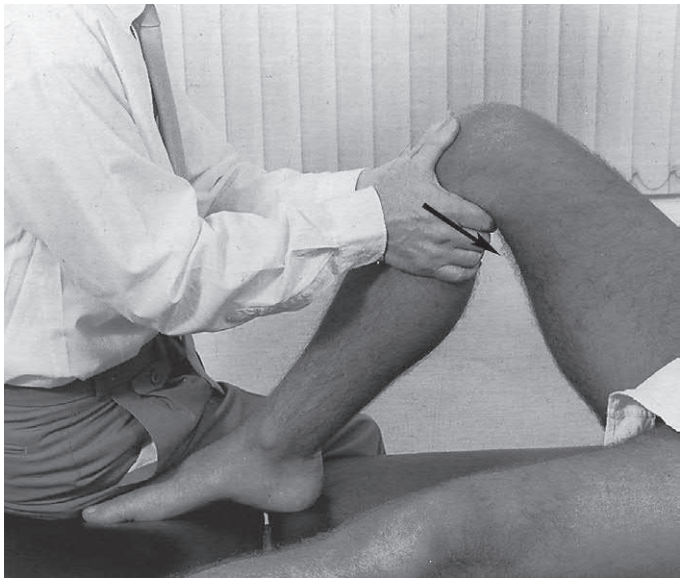

Fig 10 • Posterior drawer test.

External rotation–recurvatum test

The patient lies supine on the couch. Both legs are lifted simultaneously by grasping the big toes (Fig. 12). The amount of external rotation of the proximal end of the tibia and the degree of recurvatum are observed. In a positive test, unilateral excess of external rotation and recurvatum is obvious, which is seen as increased tibia vara.

The test demonstrates posterolateral rotatory instability, which is located in the structures of the arcuate complex: the arcuate ligament, lateral collateral ligament and possibly the popliteus tendon. It is also advisable to observe the patient standing and actively extending the knees maximally or to observe a barefoot walk.

Sometimes external rotation–hyperextension is absent, while these patients often walk with a slightly bent knee to avoid terminal extension.

Clinical tests for knee instability are summarized in Table 1.

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Table 1 Summary of clinical tests for instability

Type of instability	Structures probably ruptured	Diagnostic test
Straight valgus	Capsuloligamentous structures of medial and posteromedial compartment, and posterior cruciate ligament	Abduction stress test in full extension
Straight varus	Lateral compartment ligaments and posterior cruciate ligament	Adduction stress test in full extension
Straight anterior	Middle third of medial and lateral compartment ligaments, and anterior cruciate ligament	Anterior drawer test Lachman test
Straight posterior	Posterior cruciate ligament and arcuate complex	Posterior drawer test
Rotatory anteromedial	Medial and posteromedial compartment ligaments, and anterior cruciate ligament	Anterior drawer test from mid-position and in internal rotation Abduction in 30° flexion Lachman test
Rotatory anterolateral	Middle third of lateral compartment ligaments, and anterior cruciate ligament	Anterior drawer test from mid-position Adduction in 30° flexion 'Jerk' test Pivot-shift test
Rotatory posterolateral	Arcuate complex	External rotation–recurvatum test Posterior drawer test from mid-position and in external rotation Adduction in 30° flexion

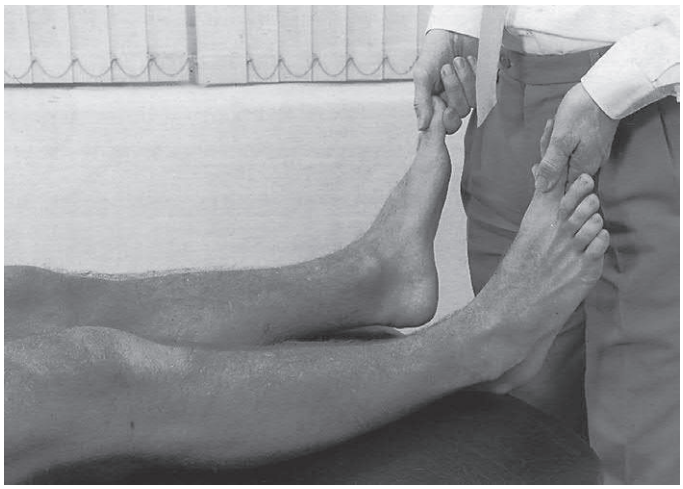

Fig 12 • External rotation–recurvatum test.

Treatment

The best chance of success in treating a knee affected by ligamentous instability is within days of the original injury.[11] Management can be non-surgical or by surgical repair. Which is chosen depends on the structure(s) at fault, the degree of rupture and the extent of the lesion.

Conservative treatment

In general, conservative treatment is indicated in all grade I and II sprains and in isolated grade III sprains of the medial collateral ligament. Acute complete tears of the anterior cruciate ligament, with an absent (0) or mild graded (1+) pivot-shift test, show excellent or good results in half of the cases.[12]

The results of conservative treatment of isolated grade III sprains of the lateral ligament compartment are generally poor and not acceptable.[13]

Surgery

Surgical treatment of knee ligament injuries, particularly of the anterior cruciate ligament, is still controversial because the complex functional role of these ligaments makes them difficult to reconstruct effectively.[14] Given that reconstruction is performed using high-strength bone–ligament–bone autogenous graft, placed isometrically and securely fixed internally, failure rates after 2 years' follow-up have decreased to about 30%.[15]

Conservative treatment in acute instability

Medial collateral ligament

Isolated acute grade III lesions of the medial collateral ligament seldom result in lasting instability. Successful conservative treatment of these lesions has been reported, with only a very small number of patients requiring subsequent reconstruction.[16–20] However, this applies only to isolated injuries in which, for example, valgus stability in full extension is normal. The key to success in non-surgical management of complete tears of the medial collateral ligament is to exclude those with concurrent damage to the anterior cruciate ligament or menisci.[18,20,21,22]

Therapy must also include a structural rehabilitation programme with early protected motion, followed by progressive strengthening. A small residual amount of medial laxity does not appear to have any influence on the ability to return to competitive athletics.[22]

When surgical and non-surgical treatment is compared in isolated acute grade III lesions of the medial collateral

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ligament, similar results are obtained. However, non-surgically treated patients are rehabilitated in significantly less time.[18] In *combined* injuries, it has been shown that the non-surgical management is successful only in 15%.

Rehabilitation in non-operated, acute complete tears[22]

In the first 2 weeks the knee is partially immobilized in a rehabilitative brace that prevents the joint from moving beyond the painful range and protects against hyperextension, excessive lateral rotation and valgus strain. In order to maintain mobility while healing, Cyriax advocates moving the ligament by deep transverse friction to and fro over the adjacent bone in imitation of its normal behaviour.[23] The first day, 1 minute's friction thus suffices, in that no unwanted adhesions have yet formed. Friction is followed by passive flexion and extension movements, within the limits of pain. This treatment is repeated daily. Within a week, tenderness is declining and friction is performed for an increasing time, for up to 15 minutes, on alternate days during the second week (see p. 701).

From the moment the patient can undertake straight leg raising, weight bearing with crutches is started, provided the patient can tolerate it. At this time, isometric exercises are commenced (three times daily, 15 contractions) in order to avoid muscle wasting.

This period is followed by 4 weeks of controlled mobilization in a rehabilitative knee brace that allows a range of motion to be set within the limits of pain; hyperextension should still be prevented. This provides the opportunity to perform stationary biking and to start isotonic exercises for the quadriceps and hamstrings muscles. Thereafter, isokinetic exercises are added.

After 6 weeks the orthosis is replaced by a functional knee brace. Isotonic and isokinetic muscle training are continued. Running exercises can be started from the moment the quadriceps has reached at least 60% of the strength of the unaffected muscle. At this stage, hamstrings strength is equal to that of the opposite leg.

If quadriceps strength has returned to 80% of normal, agility drills can be undertaken. At this stage training is, if necessary, intensified to reach the level required for competitive sports. Strength, power and endurance should be compared with the unaffected leg. Contact drills are allowed, although still with a functional brace on the knee to protect it as much as possible against valgus stress.

The average time from injury to return to full activity and competitive sports is about 9 weeks.

Anterior cruciate ligament

In acute complete rupture of the anterior cruciate ligament, surgical repair is the treatment of choice,[24–26] although secondary repair operations can still be undertaken.

Non-surgically treated complete ruptures of the anterior cruciate ligament commonly lead to recurring effusion into the joint after slight trauma and, because of the integrated nature of the whole stabilizing complex, intact restraining structures may gradually be stretched as well so that a number of structures are eventually involved and chronic instability results. After some years an advanced arthrosis is often the rule.

Buckley *et al*,[27] in their study of conservatively treated *partial* anterior cruciate ligament tears, found that most patients developed a symptomatic knee but the majority did not have a sufficiently significant functional disability to warrant reconstructive surgery. Furthermore, they found that knee function following a partial tear did not appear to deteriorate with time (49 months after injury).

Rehabilitation programme in conservatively treated grade II ruptures[14]

In the first 2 weeks the knee is immobilized in 30° of flexion. Isometric hamstrings and quadriceps exercises are instituted within the first 3 days. Hamstring muscle function is essential, because the hamstrings act as dynamic agonists to the anterior cruciate ligament in preventing forward gliding of the tibial plateau during walking, running and twisting.

After 2 weeks, limited motion in flexion between 30° and 60° degrees is allowed and can be increased to 30–70° after 4 weeks. Flexing the hip should be avoided until week 3 or 4, because quadriceps activity during this movement may stretch the injured ligament.

Partial weight bearing, using crutches, is permitted approximately 2–4 weeks after the injury. Full weight bearing is allowed after 4–6 weeks.

Approximately 6–10 weeks after the injury the patient may start resisted exercises, such as swimming and stationary biking, while wearing a functional brace. Terminal extensions and bodyweight load should still be avoided.

Jogging is allowed about 3–6 months after the injury.

Muscle exercises must be continued until there are no differences between the legs in hamstring and quadriceps strength. Athletes are not allowed to return to competitive sports earlier than 6–12 months after the injury.

Any swelling during the entire course of rehabilitation indicates that some overload has taken place and the programme should be modified accordingly.

Posterior cruciate ligament

Recent studies have shown that conservative management is also acceptable in *isolated complete ruptures* of the posterior cruciate ligament. Good results are obtainable even in athletes who wish to return to competitive sport.[28–32]

In contrast, as found by Fowler and Messieh,[31] surgical techniques of posterior cruciate ligament repair and reconstructions are generally difficult and the long-term results are unpredictable.

Rehabilitation programme

This resembles the programme for the anterior cruciate ligament. However, in posterior cruciate ligament instability, it seems to be essential that quadriceps atrophy is avoided.

Lateral ligament compartment

The long-term results of non-surgically treated grade II sprains of the lateral ligament compartment of the knee are reported as good and acceptable; however, in grade III sprains the results are mostly poor and unacceptable, resulting in an unstable knee with severe symptoms and post-traumatic osteoarthritis.[13] On examination in 30° of flexion, these grade III sprains showed a severe (3+) adduction instability. In full extension there is still some adduction instability, graded as mild (1+) or moderate (2+). Kannus[13] emphasizes that in grade III sprains the anterior cruciate ligament is often injured as well, and the

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damage to it may be missed by clinical methods at the initial examination. Such ligamentous laxity seems too severe to be fully compensated for by muscle strength, i.e. dynamic stability.

Conservative treatment in chronic instability

In chronic instability the action of the mechanical receptors in the lengthened ligaments is normally decreased. This loss of reflex activity influences the 'protective reflex arc' formed by the ligaments and their companion muscles and leads to lack of active stability.

In chronic grade III instability it is impossible to improve upon the loss of proprioception within the joint without surgical repair. Such repair is thereby limited to young and well-motivated patients with no radiological evidence of degenerative disease.[33]

Late examples of posterior cruciate and isolated lateral ligament instability cannot be repaired with confidence.[11] Thus early detection and repair are most important.

In all cases of chronic instability the patient should still be encouraged to undertake an intensive programme of muscular training to improve the strength and endurance of all related muscles – knee flexors, extensors, internal and external rotators. Meanwhile the surgeon and therapist are able to judge the patient's motivation and to select patients appropriate for surgical treatment. Exercises include isometric, isotonic and isokinetic contractions performed daily. Care should be taken again not to overload the joint, because this can lead to capsulitis, patellofemoral pain or increasing crepitus. Such symptoms should be seen as a sign that progress with exercises has been too quick. During more functional actions, such as jogging on even or uneven ground, running in figures of eight and jumping, it is advisable that the patient initially wears a knee brace to control the instability. Which appliance is appropriate is determined by the type of instability that is to be controlled. The level of muscular condition determines the grade of sport activity and the types of sport that are appropriate for each individual.

Rehabilitation management after surgical repair

Rehabilitation takes at least 6 months. It should be instituted carefully, because revascularization and structural conversion of any transplanted tissue is a very slow process. The final result is obtained about 1 year after the operation.[34–36]

The rehabilitation programme can be divided into four phases:[37]

- Assisted ambulatory phase
- Early strength training phase
- Intensive strength training phase
- Return to sports.

Assisted ambulatory phase

During the last decade a trend has developed to replace the period of immobilization after surgical repair by early mobilization in order to decrease the risk of contracture building up and to prevent detrimental effects on cartilage. For example, after complete rupture of the anterior cruciate ligament, the use of a high-strength bone–ligament–bone autogenous graft, placed isometrically and securely internally fixed, made it possible to commence joint motion, muscle re-education and, with the protection of a knee orthosis, even weight bearing up to 50% from the first week.[38–42]

A study of the results of accelerated programmes of rehabilitation after anterior cruciate ligament reconstruction indicated not only the need for individualized, evaluation-based rehabilitation, but also that caution should be used when introducing high loads or repetitive exercises within the first few months after surgery.[43]

For this reason postoperative clinical evaluation of graft failure, excessive pain and effusion must be strictly and regularly performed until termination of the rehabilitation. This includes joint arthrometer tests and stress diagnosis after the first week of rehabilitation and at every second week during the first 6 weeks, so as to have an accurate and objective evaluation of anterior displacement before progression to a more strenuous phase of rehabilitation.[37]

Any *increase in joint displacement* of 2 mm or more than the last result is an indication for adjustment of the rehabilitation programme. Weight bearing must be delayed, joint motion must be limited to between 20 and 90°, and resisted exercises for hamstrings and quadriceps must be limited at an angle of 70°. These measures decrease the tension on the graft. After 1 week the arthrometer test is repeated.

Excessive synovial fluid may be another sign that therapy has been too aggressive. It inhibits not only quadriceps contraction[44–46] but also may degrade cartilage matrix.[47]

Pain must also be controlled to block harmful effects on circulation and muscular action. This can be achieved by medication and physical agents such as cool packs (10°C) and transcutaneous electrical nerve stimulation (TENS). Signs of sympathetic reflex dystrophy contraindicate the use of low temperature methods.

Programmes that are performed too aggressively, with running and agility drills 5 to 6 weeks after surgery and return to full activity and competition 16 weeks' postoperatively, show 11–52% abnormal displacements at follow-up.[48–50] In a less aggressive rehabilitation programme, with delay of high loads and strenuous activities for at least 4 months after surgery, abnormal displacements decreased to 15%, from which only 5% showed failure (6 mm or more of increase).[43]

Early strength training phase

On the second day after surgery *active assisted exercises* are started. This prevents formation of adhesions which usually occurs within 2 weeks of immobilization. Excessive scar production is estimated in 10–20% of cases. Because structures of the supra- and infrapatellar region and the posterior joint capsule are particularly prone to the development of contractures, motion exercises must include passive mobilizations of the patella in all directions.

Several studies recommend *continuous passive motion* via an externally applied force, especially if postoperative joint motion causes difficulties.[39,51,52] It has been shown not to increase effusion or haemarthrosis.[42] However, if pain and

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muscle spasm cause patients to resist these passive motions, strong forces could damage the healing graft and contraindicate the measure.

Isometric contractions and *electrical stimulation* of the quadriceps also start from the second day and are performed daily. Electrical stimulation not only stimulates quadriceps contraction, but also increases pain threshold. From the moment contraction of the quadriceps muscle is normal again, isotonic contractions, which cause higher muscular forces, are necessary to maintain strength. Because the effects on the maturing graft are unknown, and a direct relationship exists between the inert and contractile structures, it is advisable to start with a low load, which is applied close to the knee joint. Movements should also not pass 30° of extension because studies on strain in the normal and reconstructed anterior cruciate ligament during isotonic contraction have shown the final 30° of extension to be seven times that of full flexion to 30°.[53]

A training programme for the hamstrings, gracilis, tensor fasciae latae and gluteal muscles is also instituted.

After the first week, *stretching exercises* of the gastrocnemius, hamstrings and hip flexor muscles are included.

When the patient has achieved 50% weight bearing, normally about 10 days after surgery, *balance training* can be added. The patient tries for 5–10 minutes to stay with both feet on a balance board. This stimulates proprioceptive activity and arthrokinetic reflexes that increase the action of the knee muscles, which in turn protects the graft.

Attention should be paid to a normal, symmetrical *gait*, which maintains normal articular cartilage and muscle re-education.[54,55] Any abnormalities during gait must therefore be identified and treated before weight bearing is increased.

Intensive strength training phase

To regain muscular condition *resisted exercises* may start the fourth week after surgery. These are performed submaximally (about 60% of maximum effort), with a short lever arm and progressive repetition. Three sets of 10 repetitions are prescribed. Increase of strength is to be expected if the limits of fatigue are reached after each session.

Isotonic contractions of the hamstrings may be performed prone, with the patella lying free from the edge of the table. As for the quadriceps, three sets of 10 repetitions are performed two times a day. Because there is no fear of stretching the graft, this exercise may be performed throughout the full range of motion. Isotonic contractions of the quadriceps beyond 30° of extension should still be avoided. Because of its posterior drawing force, co-contraction of the hamstrings during quadriceps action is an effective measure to decrease strain on the anterior cruciate ligament.

In a study on this matter during standing, knee flexion of 30° or more, together with trunk flexion of 30° or more, increased the protective posterior drawer force of the hamstrings.[56]

Mini-squats and wall sits (patient leans backwards against a wall with the knees flexed, ranging between 30° of extension and 90° or more of flexion) are also recommended exercises, not only to increase strength but also endurance, without stretching the graft. Bending the trunk forward still increases the protective action of the hamstrings on the graft. These so-called close-kinetic exercises (standing with continued ground reaction force) also seem to reduce forces on the patellofemoral joint.

From the moment the patient has achieved full weight bearing, which is normally 6 weeks after surgery, it is advisable to start progressive muscle training with an overload at each exercise but only on condition that there is no pain, effusion or difference of tibial anterior translation between the involved and uninvolved limb and there is a full and painless range of motion. Patients with palpable patellofemoral crepitus are at risk of deterioration and are better to avoid strong resisted exercises.

The improvement of quadriceps and hamstrings must be evaluated weekly by isometric manual tests on strength and by the measurement of thigh circumference.

Proprioceptive neuromuscular facilitation (PNF) training is another recommended exercise to increase motor activity. It improves strength and endurance of the whole limb, from proximal to distal. With allowance for pain and weakness, resistance can be performed in an isometric or a concentric manner (either eccentric or isotonic).

After 2 months, training is broadened with more *functional elements* such as walking, pool walking and stepping. The use of a knee orthosis to control rotatory movements and the flexion–extension range within 60° and 20°, respectively, must be continued.

Isokinetic contractions have their own value. Because muscular defects have been shown to be more likely diagnosed at special velocities, it is of great advantage to investigate that aspect of muscular deficiency. With the use of a device, resistance can be adjusted accurately at the indicated velocity.[57] It is advisable to perform three sets of 10 repetitions twice a day on every other day and the load should not exceed 70% of maximum effort. However, because of the process of incorporation of the graft, it seems better not to start such exercises before the sixth postoperative week and also in the absence of pain and swelling. The effect of isokinetic exercises on force and endurance has been shown to be greater than that of isotonic exercises. The high loading contraindicates these exercises when there is patellofemoral crepitus, unless this is limited to the range of motion outside the crepitus.

Return to sports

After 3 months, *bicycling* can be started, which is stimulating for the quadriceps and hamstrings with little effect on the graft. At the beginning duration is 15–20 minutes. At that time resisted exercises are still increased, although muscular effort must not exceed 75% of maximum.

Running is allowed from the sixth postoperative month and, according to the patient's goals, turning, twisting, cutting and jumping may gradually be added.

The use of a knee orthosis is still continued, especially in new training situations that impose high loads on the graft.

'Joint reactions', such as recurrence of pain and effusion, patellofemoral crepitus and any increase in anterior translation of the tibia, should always modify the programme.

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To be successful in rehabilitation, exercises must also be similar to the specific demands of the individual sport or activity.[58] Strenuous and difficult aspects are trained in isolation until proficiency has again been obtained. From that moment, and on condition that pain, effusion and patellofemoral crepitus are absent and forward translation of the tibia stays normal, training in the group can restart.

Return to competition is thereafter a matter of time and methodical building of strength. The level required for competitive sport can be judged from the following schedule: six 60 metre runs at full speed with 30 seconds' rest after each run; six 60 metre runs with small side jumps; 100 metre running with 10 left and right turns without losing speed. The whole question of successful rehabilitation is primarily one of motivation. Older and athletically inactive patients tend to do less well.

Tables 2 and 3 give a survey of the whole rehabilitation programme after anterior cruciate ligament (ACL) reconstruction.[37]

Table 2 Evaluation-based rehabilitation advanced weight bearing and minimal protection programme: range of motion after ACL reconstruction. Weeks 1–5[a]

	Week 1	Weeks 2–3	Weeks 4–5
When	Weight bearing with two crutches when: — postoperative pain is controlled — haemarthrosis is controlled — patellar mobility is normal — voluntary quadriceps contraction	Weight bearing with one crutch when: — pain is controlled without narcotics — effusion is controlled — ROM 10–100° — muscle control throughout ROM	Independent ambulation when: — pain is controlled — effusion is controlled — ROM 5–115° — muscle control throughout ROM — joint arthrometer results are within 2 mm
Evaluation	Pain Haemarthrosis Patellar mobility Joint arthrometer ROM Quadriceps contraction and muscle spasm Soft tissue contracture	Pain Effusion Patellar mobility Joint arthrometer ROM Muscle control Active extension Inflammatory response	Pain Effusion Patellar mobility Joint arthrometer ROM Muscle control Complete extension Inflammatory response Gait
Treatment	Pain management Control haemarthrosis Mobilize patella Motion exercises Flexibility programme Muscle re-education Knee orthosis CPM — perform ankle pumps 5 minutes every hour to maintain blood flow	Pain management Control effusion Mobilize patella Motion exercise 0–125° Flexibility Increase quadriceps exercises and add mini-squats Muscle re-education Knee orthosis Balance training Increase in joint arthrometer result Continue ankle pumps	Pain management Control effusion Motion exercises Flexibility Continue quadriceps exercises and mini-squats Knee orthosis Endurance and advanced muscle exercises Balance training Increase joint arthrometer Address gait abnormalities
Goals	ROM 20–70° Adequate quadriceps contraction control, inflammation and effusion 25–50% full weight bearing	ROM 0–125° Muscle control Control inflammation and effusion to prevent scarring Joint arthrometer remains within 2 mm of last test 50–75% full weight bearing	ROM 0–135° Maintain ROM Muscle endurance Control inflammation and effusion to prevent scarring Joint arthrometer remains within 2 mm of last test 100% full weight bearing with a normal gait Early recognition of complications (motion loss, RSD, laxity)

[a]Rationale: Achieve full weight bearing and full range of motion without compromising graft fixation or graft maturation.
CPM, continuous passive motion; ROM, range of motion; RSD, reflex sympathetic dystrophy.

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Table 3 Evaluation-based rehabilitation advanced muscle training after ACL reconstruction. Weeks 6–52[a]

	Weeks 6–12	Weeks 13–24	Weeks 25–52
Evaluation	Hamstrings and quadriceps strength testing Swelling Joint arthrometer Patellar mobility and crepitus	Hamstrings and quadriceps strength testing Swelling Joint arthrometer Patellar mobility and crepitus	Hamstrings and quadriceps strength and endurance testing Swelling Joint arthrometer Patellar mobility and crepitus Function tests
Treatment	Discontinue EMS Increase isotonic exercise Start isokinetic exercises Start proprioceptive neuromuscular facilitation Start aerobic conditioning Proprioceptive training Therapeutic modalities Knee orthosis	Increase speed and resistance for isotonic and closed chain kinetic exercises Advance isokinetic exercises Continue aerobic conditioning Advance proprioceptive training Start plyometrics Knee orthosis	Increase isotonic exercise Advance isokinetic exercises Continue aerobic conditioning Advance running and start functional running programme Continue proprioceptive training Knee orthosis Sport-specific training
Goals	Increase strength endurance so there is no fatigue with ADL	Increase strength & endurance of lower extremity for limited activity	Increase function Maintain strength & endurance Return to previous activity level

[a]General criteria: No effusion, painless range of motion, joint stability; performs activities of daily living without pain; range of knee motion 0–125°; full weight bearing.
ADL, activities of daily living; EMS, electrical muscle stimulation.

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Differential diagnosis of lesions at the knee

	Disorder	Diagnosis	Treatment
Anterior knee pain	Patellar bursitis	Inspection and palpation	Aspiration/infiltration or surgery
	Subsynovial haematoma	History of local blow Marked limitation of flexion Normal extension	Aspiration
	Suprapatellar tendinitis	Painful resisted extension Tenderness at the upper border	Deep friction or triamcinolone
	Infrapatellar tendinitis	Painful resisted extension Tenderness at the lower pole	Deep friction or triamcinolone
	Quadriceps expansion tendinitis	Painful resisted extension Lateral or medial tenderness	Deep friction
	Infrapatellar tenosynovitis	Painful resisted extension Crepitus Local tenderness	Triamcinolone
	Apophysitis	Boys between 10 and 15 years Local pain and tenderness	Rest
	Recurrent dislocation	Attacks of collapsing, swelling and anteromedial pain Positive apprehension test Decreased Q angle	Brace Tape and training VMO (Surgery)
	Patellofemoral pain syndromes	Dull pain, also during sitting Increased Q angle Hypertrophied lateral bands Crepitus during squatting	Tape and training VMO
	Patellofemoral arthrosis	Anterior pain during walking upstairs Crepitus Sometimes pain during sitting	Infrapatellar strap
	Sprain of the anterior cruciate, anterior insertion	Trauma Acute stages: capsular pattern Chronic stage: only a positive anterior drawer test	Triamcinolone

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	Disorder	Diagnosis	Treatment
	Sprain of the posterior cruciate, anterior insertion	Trauma Acute stages: capsular pattern Chronic stage: only a positive posterior drawer test	Triamcinolone
Medial knee pain	Medial coronary ligament	History of rotation trauma Painful lateral rotation Local tenderness	Deep friction or infiltration
	Medial collateral ligament	History of trauma Acute cases: capsular pattern and local tenderness Chronic cases: limitation in a non-capsular pattern Painful valgus and lateral rotation	Acute: deep friction and mobilization or triamcinolone Chronic: manipulation after deep friction
	Plica mediopatellaris	Vague medial pain Sometimes twinges Painful arc Local thickening and tenderness	Triamcinolone or arthroscopic section
	Loose body	Middle-aged Twinges, usually on walking downstairs Non-capsular pattern Typical end-feel 'Sprain without a sprain'	Manipulation
	Medial collateral bursitis	Middle-aged Nocturnal pain Non-capsular pattern Painful valgus and external rotation Hardish swelling	Aspiration and triamcinolone
	Medial meniscus	Locking/manipulative unlocking Symptoms of sprained coronary ligament	Manipulation or surgery
	Medial quadriceps expansion tendinitis	Pain during exertion Resisted extension painful Local tenderness	Deep friction
	Pes anserinus tendinitis	Resisted flexion and internal rotation painful	Deep friction or triamcinolone
Lateral knee pain	Lateral coronary ligament	Rotation trauma Painful internal rotation	Deep friction or infiltration
	Lateral collateral ligament	Trauma Painful varus	Deep friction or triamcinolone (acute)
	Lateral meniscus tear	Locking and manipulative unlocking Symptoms of sprained lateral coronary ligament	Manipulation or surgery
	Lateral meniscus cyst	Pain on standing Palpable bulge on extension	Puncture (or surgery)
	Discoid meniscus	Children or adolescents Symptoms of internal derangement Arthroscopic diagnosis	Surgery
	Biceps tendinitis	Positive resisted flexion, also with an extended knee Positive resisted external rotation	Deep friction or triamcinolone
	Upper tibiofibular sprain	Painful resisted flexion, disappearing with the knee in extension	Triamcinolone at the anterior ligament

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	Disorder	Diagnosis	Treatment
	Popliteus tendinitis	Painful squatting and walking downstairs Painful resisted flexion and internal rotation	Triamcinolone (origin) Deep friction (body)
	Iliotibial friction syndrome	Runners/skiers/cyclists Sometimes painful arc at 30° Painful resisted extension and flexion Local tenderness	Infiltration or operative section
	Lateral quadriceps expansion tendinitis	Painful resisted extension	Deep friction
Posterior knee pain	Posterior cruciate ligament, tibial insertion	Trauma Painful passive extension Painful posterior drawer test Painful lateral shearing	Triamcinolone
	Anterior cruciate ligament, femoral insertion	Trauma Painful anterior drawer test	Triamcinolone
	Popliteus muscle	Trauma? Painful squatting and walking downstairs	Deep friction
	Medial collateral ligament	Trauma Chronic stage: slight non-capsular pattern, passive external rotation and valgus	Chronic stage: manipulation
	Posterior capsular strain	Trauma: hyperextension strain Chronic stage: only passive extension is painful Sometimes slight calcification on the radiograph	Triamcinolone
	Popliteal cyst	Slight limitation of flexion Soft end-feel Palpation	Aspiration
	Lesions of the posterior horn of meniscus	Slight internal derangement at the back of the knee	Prophylaxis or surgery
	Coronary strain at the posterior part	Trauma: rotational injury After the capsular pattern has subsided: pain during internal or external rotation Palpation at the posterior part of the joint line	Deep friction
	Tendinitis of the origin of the gastrocnemii	Painful tiptoe Painful resisted flexion, and internal or external rotation	Deep friction or triamcinolone
Pain in the knee			
Capsular pattern	Traumatic arthritis	Trauma No synovial thickening	Deal with the cause
	Monoarticular arthritis	Slow onset First swelling, then pain and finally slight capsular pattern Synovial thickening	Two intra-articular injections with triamcinolone
	Haemarthrosis	Post-traumatic/haemophilia/spontaneous Severe and immediate pain, swelling and tenderness	Aspiration Causative treatment

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	Disorder	Diagnosis	Treatment
	Rheumatoid and reactive arthritis	Polyarthritis Biomechanical changes Synovial thickening	Intra-articular triamcinolone Causative treatment
	Septic arthritis	Hyperacute arthritis Swelling, redness, tenderness and gross articular pattern	Aspiration and antibiotics
	Gout	Hyperacute arthritis Analysis of joint fluid	Aspiration (Intra-articular triamcinolone) Causative treatment
	Pseudogout	Hyperacute arthritis Calcification of cartilaginous structures Analysis of joint fluid	(Intra-articular triamcinolone) Aspiration
	Osteoarthrosis	Pain during weight bearing Cold joint No synovial thickening Crepitus	No conservative treatment Surgery
Non-capsular pattern	Loose body complicating osteoarthrosis	Twinges Moving pain 'Sprain without a sprain'	Manipulation
	Osteochondritis dissecans	Teenager Sudden locking in extension Post-traumatic arthritis (Positive Wilson's sign?)	Surgery
	Impaction of ruptured anterior cruciate	Trauma Positive anterior drawer test Limitation of extension	Surgery
	Intra-articular adhesions	After surgery or an atypical sprain Progressive and painless limitation of flexion Normal extension	Manipulation
	Posterior horn lesions of the menisci	Slight twinges Symptoms of internal derangement Slight limitation of flexion	Manipulation or surgery

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Applied anatomy of the lower leg, ankle and foot

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Definitions

The functional terminology in the foot is very misleading, because the same terms are often used for very different things. The following definitions and explanations are used here:

- *Plantiflexion–dorsiflexion* indicate movement at the ankle. Plantiflexion is movement downwards towards the ground, and dorsiflexion upwards towards the tibia. There is also a plantiflexion–dorsiflexion movement at the midtarsal joint.
- *Varus–valgus:* these movements occur at the subtalar joint. In valgus, the calcaneus is rotated outwards on the talus; in varus, the calcaneus is rotated inwards on the talus.
- *Abduction–adduction:* these take place at the midtarsal joints. Abduction moves the forefoot laterally and adduction moves the forefoot medially on the midfoot.
- *Pronation–supination* are also movements at the midtarsal joints. In pronation, the forefoot is rotated big toe downwards and little toe upwards. In supination, the reverse happens: the big toe rotates upwards and the little toe downwards. Sometimes the terms internal rotation and external rotation are used to indicate supination or pronation.
- *Inversion–eversion:* these are combinations of three movements. In inversion, the varus movement at the subtalar joint is combined with an adduction and supination movement at the midtarsal joint. During eversion, a valgus movement at the subtalar joint is combined with an abduction and pronation movement at the midtarsal joint.

The leg

The bones (tibia and fibula) together with the interosseous membrane and fasciae divide the leg into three separate compartments: anterolateral, lateral and posterior. They contain the so-called extrinsic foot muscles. Each compartment has its own blood supply and innervation.

Anterolateral compartment

The muscles of the anterolateral compartment are the tibialis anterior, the extensor hallucis and the extensor digitorum, which are all dorsiflexors. The tibialis anterior, however, is also an invertor. The muscles lie in a strong osteofibrous envelope,

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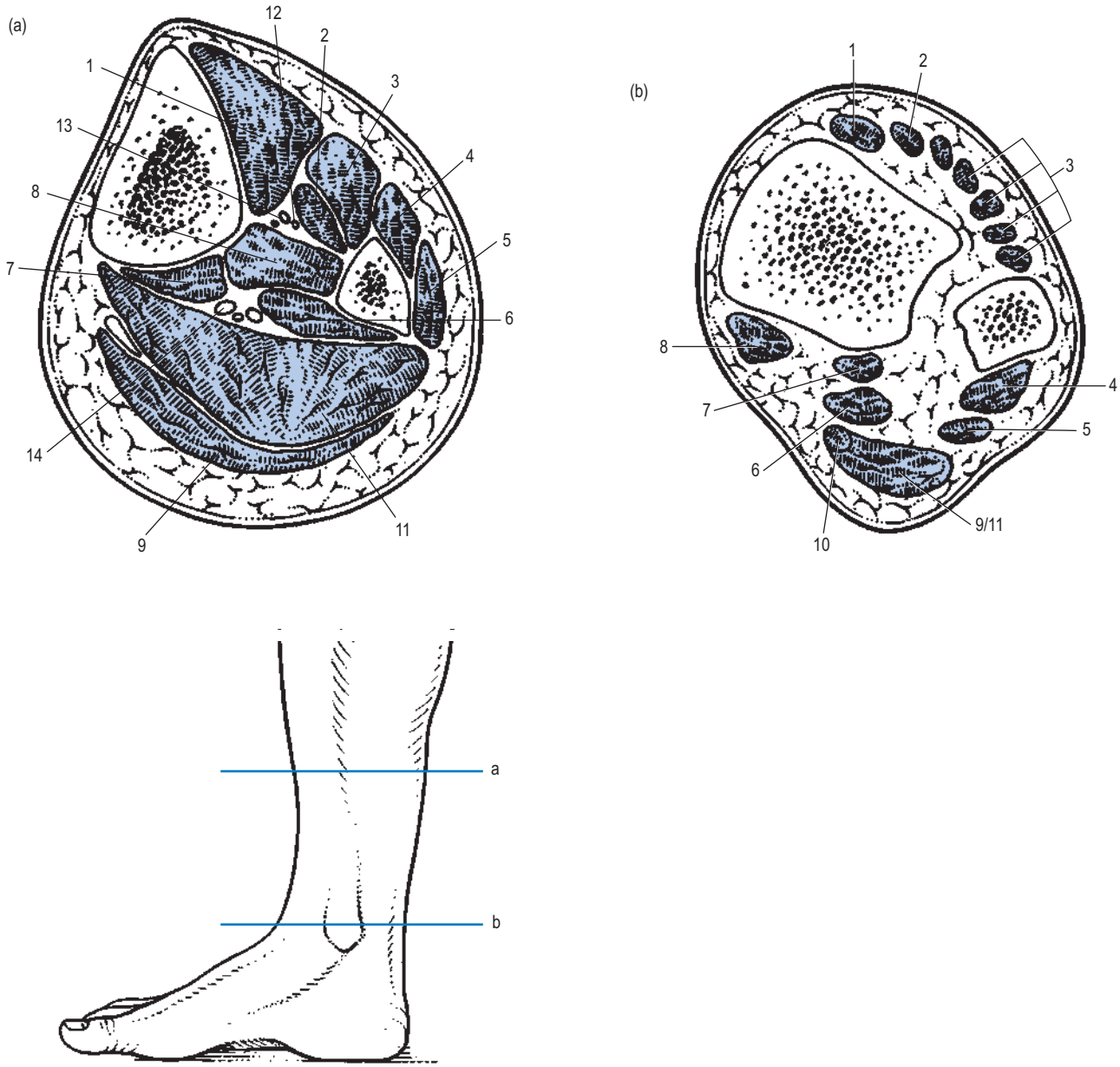

Fig 1 • Transverse sections halfway down the leg (a) and at the malleoli (b). 1, tibialis anterior; 2, extensor hallucis longus; 3, extensor digitorum longus; 4, peroneus brevis; 5, peroneus longus; 6, flexor hallucis longus; 7, flexor digitorum longus; 8, tibialis posterior; 9, soleus; 10, plantaris; 11, gastrocnemii; 12, anterior tibial nerve; 13, anterior tibial vessels; 14, posterior tibial neuromuscular bundle.

consisting of tibia, fibula, interosseous membrane and superficial fascia. The anterior tibial artery (Fig. 1) is located at the anterior surface of the interosseous membrane. It supplies the muscles of the anterior compartment and continues at the dorsum of the foot as the dorsalis pedis artery. The nerve supply of the anterior compartment is from the deep peroneal nerve, a branch of the common peroneal nerve.

Lateral compartment

The lateral compartment contains the peronei longus and brevis muscles. These are strong evertors and weak plantiflexors. Blood supply is from the peroneal artery, a branch of the posterior tibial artery and nerve supply from the superficial peroneal nerve.

Posterior compartment

The posterior compartment has a deep and a superficial part. The deep part consists of the tibialis posterior, flexor hallucis longus and flexor digitorum longus muscles. The tendons run behind the medial malleolus to the inner aspect of the sole and to the toes. Hence they are invertors of the foot and flexors of the toes. They also act as weak plantiflexors of the foot. The muscles of the superficial part are both heads of the gastrocnemius, the soleus and the plantaris. The soleus and both

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gastrocnemii form the triceps surae which inserts – via the Achilles tendon – at the upper posterior aspect of the calcaneus. The plantaris muscle usually has a separate insertion, just medial to the Achilles tendon. This group of muscles acts as a very strong plantiflexor. The artery of the posterior compartment is the posterior tibial. The tibial nerve provides the nerve supply.

The ankle and foot

The 26 bones of the foot create an architectural vault, supported by three arches and resting on the ground at three points, which lie at the corners of an equilateral triangle (Fig. 2). Ligaments bind the bones to provide the static stability of the foot. The dynamic stability of the vault is achieved by the intrinsic and extrinsic muscles.

Three functional segments can be distinguished: forefoot, midfoot and ankle (Fig. 3). Each has its particular functions and specific disorders (see Standring, Fig. 84.8A). The posterior segment (ankle) is the connection between foot and leg. It lies directly under the tibia and fibula and is connected to them by strong ligaments. This segment contains the talus (in the mortice between the fibula and the tibia) and the calcaneus.

The middle segment (midfoot) contains the five tarsal bones and is the keystone in the plantar vault. Excessive stresses falling on the vault therefore influence the midfoot in particular.

The anterior segment (five metatarsals and 14 phalangeal bones) takes the whole body weight during the 'heel-off' phase in walking or running. This specific function of the forefoot underlies pressure disorders at the plantar surface of the metatarsals and toes.

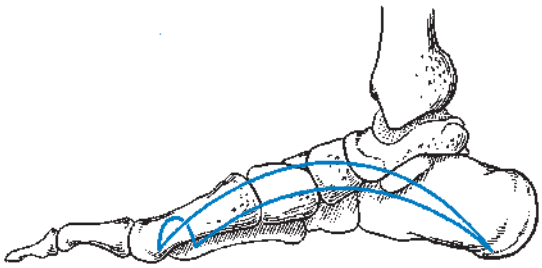

Fig 2 • The three arches of the foot.

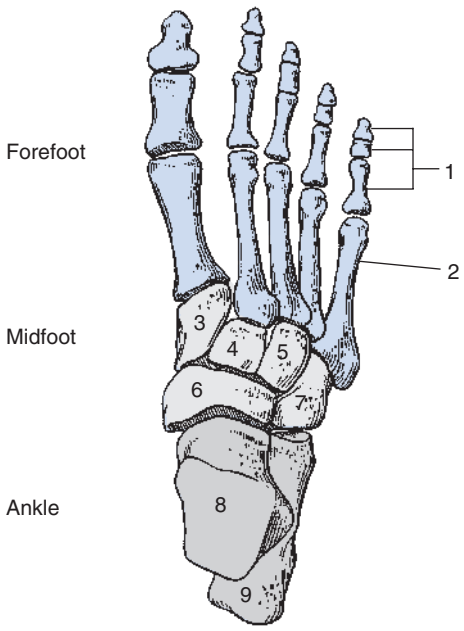

Fig 3 • The three segments of the foot: 1, phalanges; 2, metatarsals; 3–5, medial, intermediate and lateral cuneiforms; 6, navicular; 7, cuboid; 8, talus; 9, calcaneus.

The posterior segment

The talus is the mechanical keystone at the ankle (Fig. 4). It distributes the body weight backwards towards the heel and forwards towards the midfoot (medial arch of the plantar vault). The talus has no muscular insertions but is entirely covered by articular surfaces and ligamentous reinforcements. It is also crossed by the tendons of the extrinsic muscles of the foot.

Ankle joint

The superior surface and the two sides of the body of the talus are gripped between fibular and tibial malleoli, forming the ankle mortice (see Standring, Fig. 84.16). Stability of this tibiofibular mortice is provided by the anterior and posterior tibiofibular ligaments and the interosseous ligament. Because the body of the talus is wedge shaped, with the wider portion anterior, no varus–valgus movement will be possible with an ankle held in dorsiflexion, unless the malleoli or the tibiofibular ligaments are damaged (Fig. 5).

The normal movement of the ankle joint is plantiflexion–dorsiflexion, through an axis that passes transversely through the body of the talus. The lateral end of this axis goes through the lateral malleolus (Fig. 6). Hence, a normal plantiflexion–dorsiflexion movement does not influence the tightness of the calcaneofibular ligament. At the medial end, however, the axis is placed under the tip of the medial malleolus and thus under

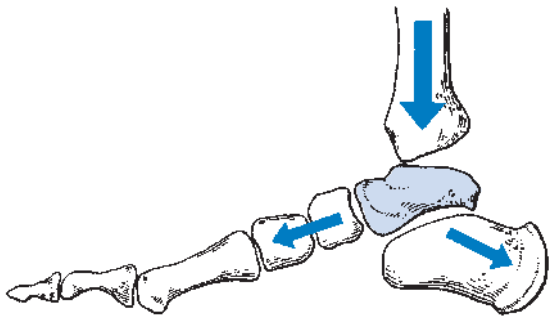

Fig 4 • The talus distributes the body weight to the heel and midfoot.

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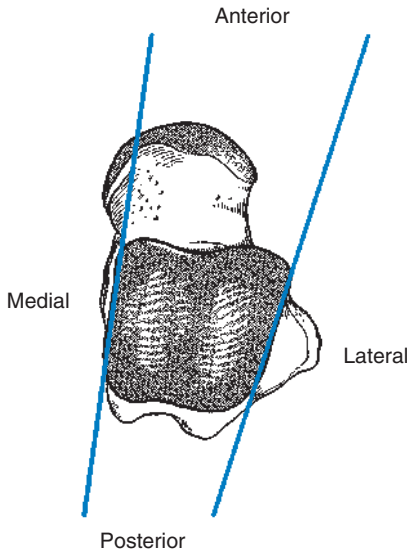

Fig 5 • The body of the talus is wedge shaped with a wider anterior portion.

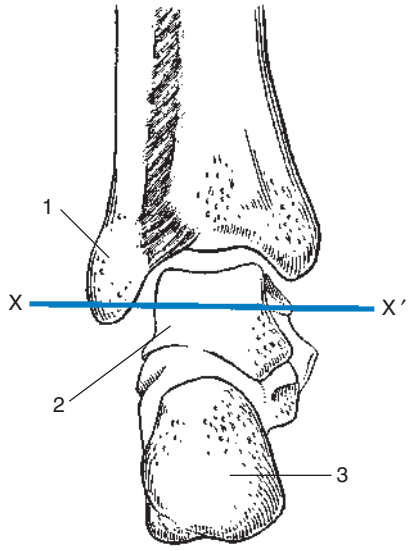

Fig 6 • Axis of plantiflexion–dorsiflexion (X–X′): 1, lateral malleolus; 2, talus; 3, calcaneus.

the malleolar point of attachment of the medial ligaments. Here, the posterior fibres of the deltoid ligament will become taut on dorsiflexion and the anterior fibres on plantiflexion (Fig. 7).

Subtalar joint

The subtalar or talocalcaneal joint consists of two separate parts, divided by the tarsal canal (Fig. 8), which is funnel shaped with the wide portion at its lateral end. The lateral end of the canal is easily palpated in front of the fibular malleolus between talus and calcaneus, especially when the foot is inverted. The canal runs posteromedially, to have its medial opening just behind and above the sustentaculum tali. In the canal a strong ligament, the interosseous talocalcaneal ligament, binds the two bones (see Standring, Fig. 84.15).

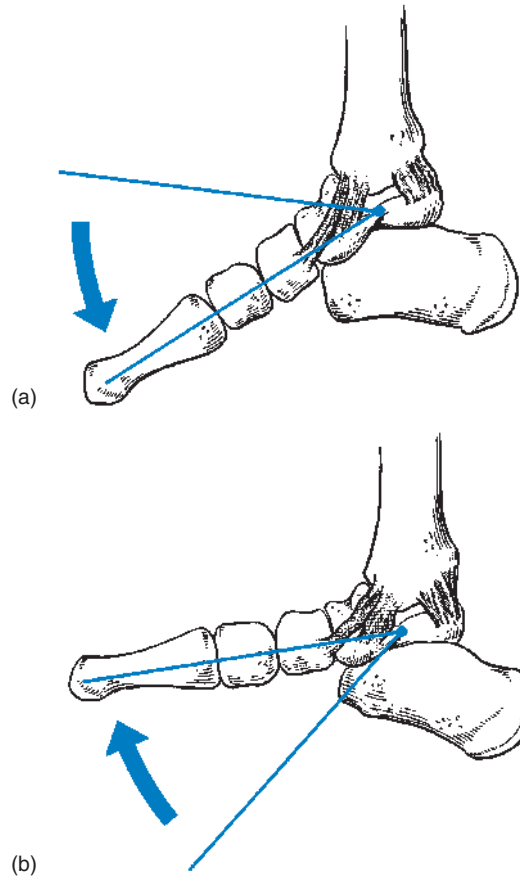

Fig 7 • The anterior fibres of the deltoid ligament become taut during plantiflexion (a) and the posterior fibres during dorsiflexion (b).

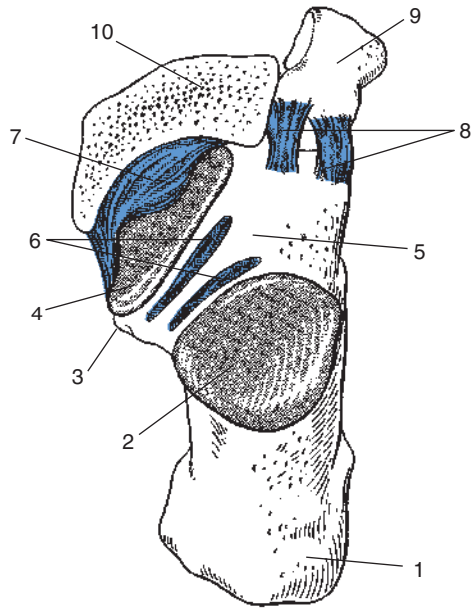

Fig 8 • View of the plantar calcaneonavicular ligament from above, with the talus removed: 1, calcaneus; 2, posterior part of subtalar joint; 3, sustentaculum tali; 4, anterior part of subtalar joint; 5, tarsal canal; 6, interosseous talocalcaneal ligament; 7, plantar calcaneonavicular ligament; 8, dorsal calcaneocuboid ligament; 9, cuboid; 10, navicular (transsection).

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The main movement of the subtalar joint is a varus–valgus movement around an axis through the talus. This axis has a 45° angle to the horizontal and a 15° angle medial to a line drawn through the second metatarsal.

Ligaments of ankle and subtalar joints

(see Standring, Fig. 84.13)

Lateral ligaments

The anterior talofibular ligament is a triangular structure, its base attached to the anterior margin of the fibular malleolus and the smaller insertion at the neck of the talus (Fig. 9), just behind the mouth of the sinus tarsi.

The calcaneofibular ligament is a strong round band running obliquely downwards and backwards from the apex of the fibular malleolus to the lateral surface of the calcaneus.

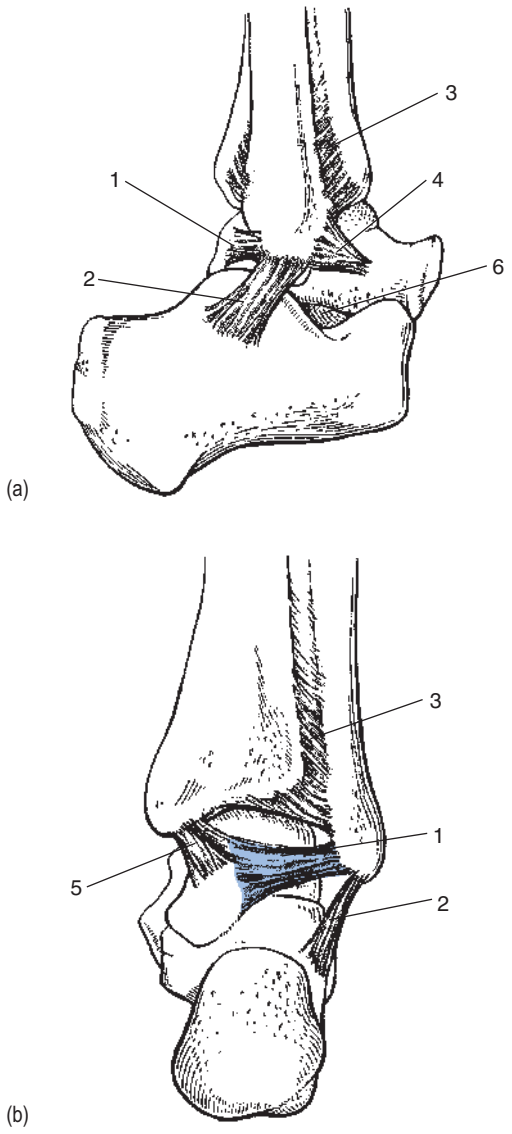

Fig 9 • Lateral and posterior ligaments of the ankle: (a) lateral view; (b) posterior view: 1, posterior talofibular ligament; 2, calcaneofibular ligament; 3, distal tibiofibular ligament; 4, anterior talofibular ligament; 5, posterior tibiotalar ligament; 6, tarsal canal.

The posterior talofibular ligament arises from the medial surface of the lateral malleolus and runs horizontally and medially to insert at the posterolateral tubercle of the talus.

Because the axis of plantiflexion–dorsiflexion runs through the origin of these ligaments (the tip of the fibula), they are not taut during these movements. The exception is the anterior talofibular ligament, the medial fibres of which originate higher up on the fibula. These become stretched at the end of the plantiflexion movement.

The main function of the lateral ligaments is to prevent excessive varus movement, especially during plantiflexion. In extreme plantiflexion, the mortice no longer stabilizes the broader anterior part of the talus; varus movement of the ankle is then possible.

Medial ligaments

The deltoid ligament is a strong and thick ligament and has deep and superficial layers. The first has fibres which run from the medial malleolus to separate parts of the talus (anterior tibiotalar and posterior tibiotalar ligaments; Fig. 10). The second runs from the medial malleolus to the sustentaculum tali (tibiocalcaneal ligament) and to the navicular bone (tibionavicular ligament).

The inferior part of the ligamentous structures at the inner side of the ankle is the plantar calcaneonavicular ligament, which runs from the sustentaculum tali to the inferior border of the navicular bone. This ligament also helps to form the talocalcaneonavicular joint (Fig. 8).

The middle segment

There are five tarsal bones: navicular, cuboid and the three cuneiforms (see Standring, Fig. 84.11). They form a semi-rigid transverse arch, held together with the interosseous ligaments and the functional stirrup of the tibialis anterior and the peroneus longus (see Standring, Fig. 84.4).

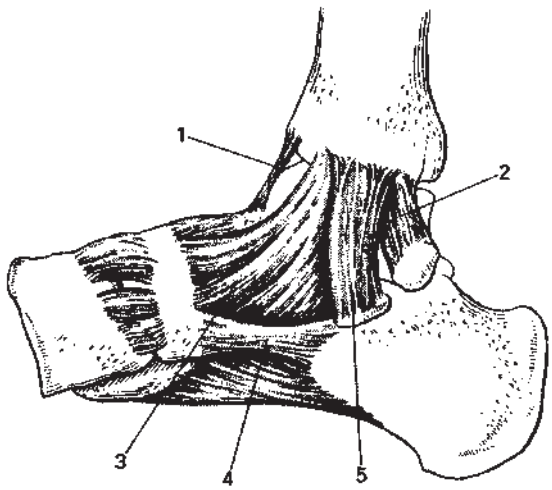

Fig 10 • Medial ligaments of the ankle: 1, anterior tibiotalar ligament; 2, posterior tibiotalar ligament; 3, tibionavicular ligament; 4, plantar calcaneonavicular ligament; 5, tibiocalcaneal ligament.

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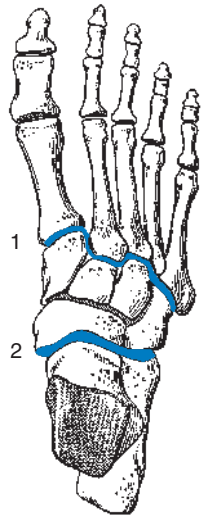

Fig 11 • Midtarsal joint: Lisfranc's (1) and Chopart's (2).

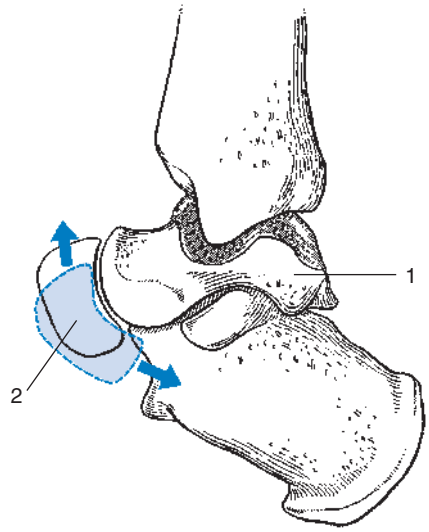

Fig 12 • Mobility of the talonavicular joint: 1, talus; 2, navicular.

The middle segment is the keystone in the plantar vault. Its elasticity permits accommodation of uneven surfaces during walking.

The joint between the posterior segment (talus and calcaneus) and the middle segment (navicular and cuboid) is the transverse tarsal or *Chopart's* joint; that between middle and anterior segment is the tarsometatarsal or *Lisfranc's* joint (Fig. 11). Functionally, all the articulations act as one structure. Therefore, Cyriax called them the midtarsal joint. Passive clinical testing evokes considerable movement in three possible directions: flexion–extension, pronation–supination and abduction–adduction.

Chopart's joint

The calcaneocuboid joint, being semi-rigid, only permits some gliding movement. At the talonavicular joint, larger movements are possible in the three directions (flexion–extension, pronation–supination and abduction–adduction). This greater mobility is because the articular surface of the talar head is larger than the surface of the adjoining navicular bone, so permitting a gliding of the navicular bone along the surface of the talus (Fig. 12).

Lisfranc's joint

The joint line is not a continuous one between the bases of the metatarsals and the anterior border of the cuneiforms and navicular bones: the intermediate cuneiform is set back and forms a fork into which fits the base of the second metatarsal (Fig. 13). Hence, the second metatarsal moves only in a plantiflexion–dorsiflexion plane and forms the axis of rotation in the tarsometatarsal joint. The base of the third metatarsal rotates around the second, the fourth around the third, and the base of the fifth moves around the fourth. The base of the first metatarsal also rotates around that of the second.

Apart from this rotational movement, the Lisfranc joint allows some abduction–adduction and considerable plantiflexion–dorsiflexion movements. The specific nature of the

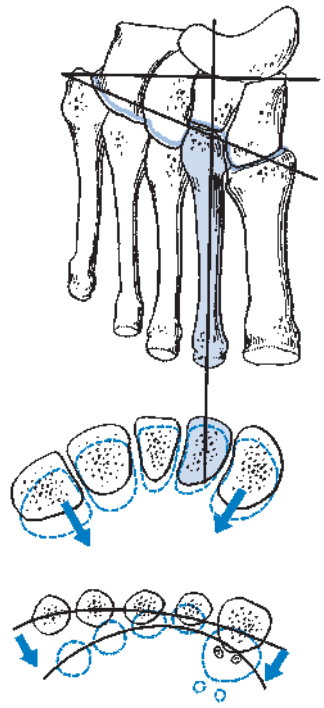

Fig 13 • Movements at Lisfranc's joint.

Lisfranc line has a particular consequence for plantiflexion–dorsiflexion: because the line of the tarsometatarsal joints runs obliquely (its medial end lies 2 cm anterior to its lateral end), dorsiflexion will include some adduction. Furthermore, the bases of the metatarsals have a cone-shaped form. During plantiflexion the heads of the metatarsals therefore do not move straight downwards but incline towards the axis of the foot and so increase the curvature of the anterior arch (Fig. 13). In contrast, extension of the metatarsals is followed by flattening of the arch. These relationships are of great importance in understanding the mechanism of splay foot.

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Ligaments of the middle segment

The architecture of the plantar vault is protected by strong plantar and interosseous ligaments. Some of these have particular clinical importance and play a role in a number of painful conditions at the midfoot. These ligaments are (see Standring, Fig. 84.13):

- *At the lateral aspect:* the lateral calcaneocuboid ligament (Fig. 14), which can be easily palpated at the lateral joint line between calcaneus and cuboid, just above the peroneal tendons. This ligament is often damaged after an inversion sprain.
- *At the medial aspect:* the plantar calcaneonavicular ligament, which is of great importance in preventing excessive dorsiflexion–abduction movements at the midfoot and thus prevents a flattening of the medial anteroposterior arch (Fig. 10).

The anterior segment

The metatarsal shafts and the tarsometatarsal joints form the transverse anterior arch of the plantar vault (see Standring, Fig. 84.4).

Changes in the curvature of the anterior arch result from movements occurring in the tarsometatarsal joints. In a plantiflexed position the anterior part of the foot is hollowed, whereas in a dorsiflexed position it is flattened. In that the arch is spanned only by relatively weak intermetatarsal ligaments and by only one muscle, the transverse head of the adductor hallucis, the plantiflexed position at the tarsometatarsal joints is of great importance in maintaining the curvature of the arch and in preventing flattening of the foot.

The two pillars of the arch are in contact with the ground through the soft tissues: the head of the first metatarsal rests on the sesamoid bones, about 6 mm from the ground; the head of the fifth metatarsal bone lies 5 mm above the ground; the head of the second metatarsal bone is, at about 9 mm, the highest of the row; and those of the third and fourth take up an intermediate position (Fig. 15).

The sesamoid bones are incorporated into the tendons of the flexor hallucis brevis and act as a fulcrum in its function. They also play a role in bearing the body weight. The base of the proximal phalanx articulates in a gliding manner upon the convex articular surface of the metatarsal head, thus allowing a considerable range of plantiflexion–dorsiflexion movement. The first metatarsophalangeal joint is normally capable of 30° of flexion and 90° of extension.

The particular insertions of the flexor hallucis longus and the flexor hallucis brevis muscles to the proximal and distal phalanges of the big toe cause the toe to be pressed to the ground during the 'foot-off' phase of walking (Fig. 16b), which distributes the body load. In the outer four toes, the proximal phalanx is free from muscle insertions, because the insertions of the flexor digitorum longus and flexor digitorum brevis fall at the base of the distal and middle phalanges. Contraction of these muscles therefore produces a prehensile movement in the forefoot (Fig. 16a).

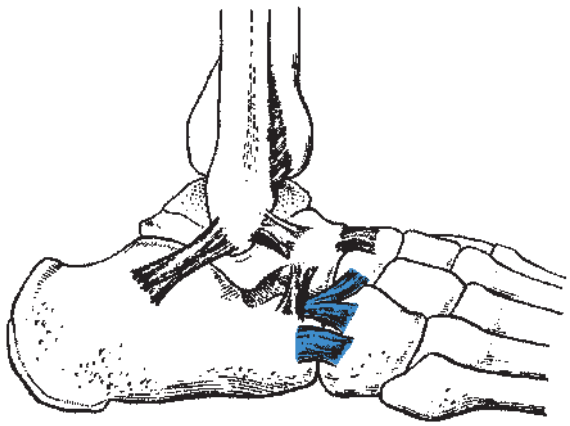

Fig 14 • Calcaneocuboid ligaments.

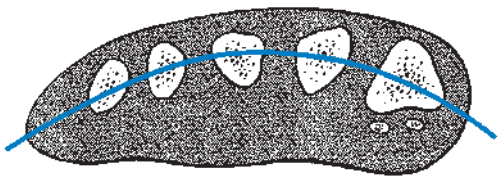

Fig 15 • The anterior arch at the level of the metatarsal heads.

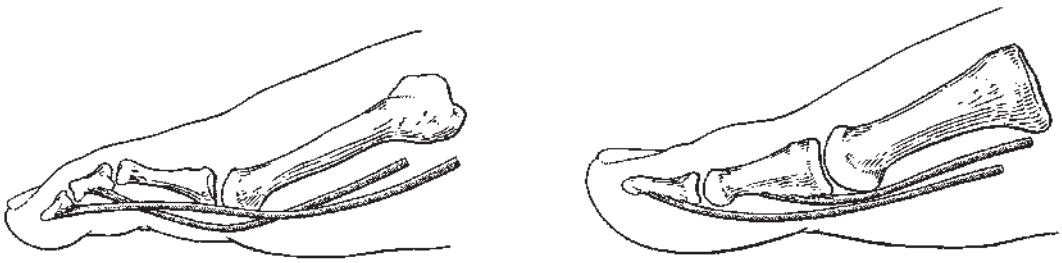

Fig 16 • Gripping movement at the outer toes during contraction of the flexor digitorum (a) and pressing movement at the big toe during contraction of the flexor hallucis (b).

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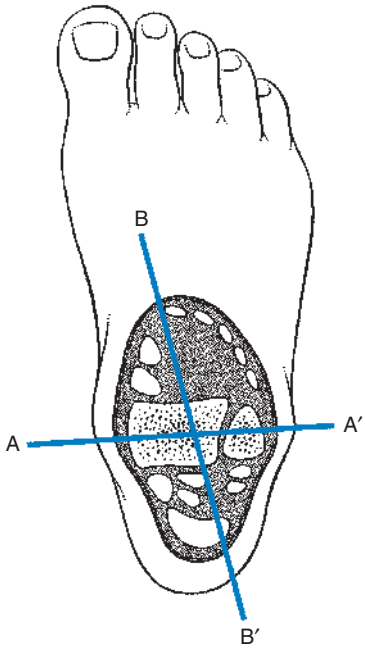

Fig 17 • Functional position of the tendons in relation to the axes of the foot.

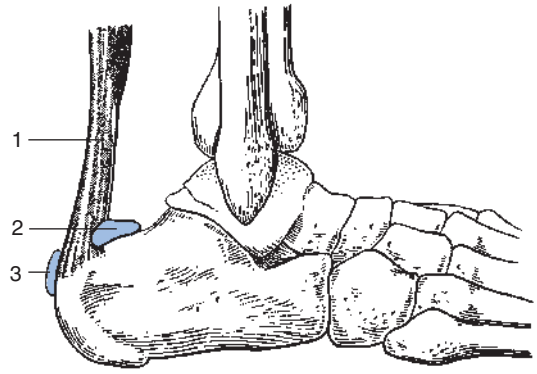

Fig 18 • Insertion of the Achilles tendon, and the relationship of the bursae to it: 1, Achilles tendon; 2, sub-Achilleal bursa; 3, subcutaneous bursa.

Table 1 The plantiflexors

	Peripheral nerve	Spinal innervation
Triceps	Tibial	S1–S2
Tibialis posterior	Tibial	L4/L5, S1
Peronei	Superficial peroneal	L5, S1
Flexor hallucis longus	Tibial	L5, S1
Flexor digitorum longus	Tibial	L5, S1

Muscles and tendons

Plantiflexors

All the tendons lying behind the axis AA′ at the ankle (Fig. 17) are plantiflexor muscles (Table 1). In practice, however, the triceps (soleus and both gastrocnemii) generates almost all the strength of plantiflexion (see Standring, Fig. 83.10).

The gastrocnemius originates by two heads, one at each femoral condyle. It spans two joints and is therefore not only a plantiflexor at the ankle but also a weak flexor of the knee.

The soleus originates from the upper tibia and fibula, below the knee joint. Halfway down the leg, the soleus and gastrocnemius muscles meet in the Achilles tendon. The anterior fibres of the soleus run further downwards along the tendon and the medial head of the gastrocnemius ends a little higher than the lateral. The strong Achilles tendon inserts at the posterior surface of the calcaneus (Fig. 18). The Achilles tendon is surrounded throughout its length by thin gliding membranes called the paratenon. The paratenon functions as an elastic sleeve (although probably not so effectively as a true tendon sheath) and permits free movement of the tendon within the surrounding tissues. A bursa lies between the upper part of the calcaneus and the anterior surface of the Achilles tendon. Sometimes a pathological subcutaneous bursa forms between the posterior aspect of the calcaneus and the skin.

Because the insertion at the calcaneus is medial to the axis of rotation (BB′, Fig. 17) of the subtalar joint, the triceps surae is also a weak invertor of the foot.

The other plantiflexor muscles – the tibialis posterior, flexor hallucis longus, flexor digitorum longus, and peronei – are rather weak (see Standring, Fig. 83.11). Although they can achieve active plantiflexion of the foot after a rupture of the Achilles tendon, they can only do this when the foot is free and unsupported. An intact triceps muscle and Achilles tendon are necessary to stand on tiptoe. The main function of the tibialis posterior is inversion and the function of both the peronei is eversion: these muscles are discussed later.

The flexor hallucis longus originates at the distal two-thirds of the posterior surface of the fibula and at the interosseous membrane. It runs behind and under the medial malleolus, in a sulcus of the talus, under the sustentaculum tali and along the inner border of the foot to insert at the base of the distal phalanx of the big toe. The flexor digitorum longus arises from the posterior aspect of the tibia and passes under the flexor retinaculum and behind the medial malleolus to attach at the distal phalanges of the four outer toes. Because its tendons cross the tendon of the flexor hallucis longus from below at the midfoot, the function of the latter will be reinforced by the first.

The three tendons at the medial malleolus run in a fibrous tunnel, formed by the tibia and the flexor retinaculum. At this level they are covered by a tendon sheath. From anterior to posterior they are the tendons of the tibialis posterior, the flexor digitorum longus and the flexor hallucis longus. The flexor hallucis longus is the only tendon of the three running behind the sustentaculum tali. This position seems to play a role in maintaining the medial arch. The long flexor tendon of the big toe acts on the sustentaculum tali as a string on a pulley, and pushes the medial arch upwards during contraction (Fig. 19).

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Table 2 The dorsiflexors

	Peripheral nerve	Spinal innervation
Tibialis anterior	Deep peroneal	L4 (L5)
Extensor hallucis longus	Deep peroneal	L4, L5
Extensor digitorum longus	Deep peroneal	L4, L5

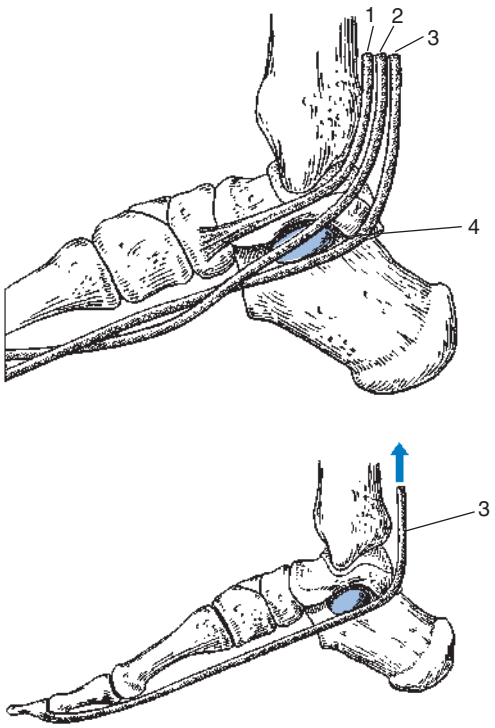

Fig 19 • The tendons at the medial malleolus: 1, tibialis posterior; 2, flexor digitorum longus; 3, flexor hallucis longus; 4, sustentaculum tali.

Dorsiflexors

The tendons lying in front of the axis AA′ at the ankle (Fig. 17) are those of the dorsiflexor muscles: the tibialis anterior, extensor hallucis longus and extensor digitorum longus (Table 2, see Standring, Fig. 83.7).

Because the extensor hallucis longus and the tibialis anterior are situated at the medial side of the axis of rotation (BB′), they act as supinators. The extensor digitorum longus, passing lateral to this axis, acts as a pronator.

The extensor hallucis longus arises from the anterior surface of the fibula and the interosseus membrane, and passes across the anterior surface of the ankle to insert at the distal phalanx of the big toe. Its main function is extension of the big toe. It also assists in dorsiflexion of the foot.

The extensor digitorum longus arises from the lateral condyle of the tibia and the anterior surface of the fibula. It runs anterior to the ankle, under the lateral half of the retinaculum extensorum digitorum, and divides into four tendons which run to the four outer toes. At its end, each tendon divides into a central slip that inserts at the base of the middle phalanx and two lateral slips inserting at both sides of the distal phalanx. The strongest function of the extensor digitorum is dorsiflexion at the ankle. It is of course also the main extensor of the toes but this function is only apparent when dorsiflexion at the ankle is counteracted by an antagonistic plantiflexion movement of the triceps surae.

The tibialis anterior is a strong dorsiflexor but first of all an adductor and supinator of the foot. Its dorsiflexion function is only possible when adduction–supination is counteracted by contraction of the peronei muscles.

Table 3 The invertors

	Peripheral nerve	Spinal innervation
Tibialis anterior	Deep peroneal	L4, L5
Tibialis posterior	Tibial nerve	L4, L5, S1
Extensor hallucis longus	Deep peroneal	L4, L5
Flexor digitorum longus	Tibial nerve	L5, S1
Flexor hallucis longus	Tibial nerve	L5, S1, S2

Invertors (adduction–supination)

The tendons of the ankle lying medial to the axis BB′ (Fig. 17) are those of invertor muscles (adduction–supination; Table 3). The two most important invertors are the tibialis posterior and tibialis anterior (see Standring, Fig. 84.2B).

The tibialis posterior originates from the posterior aspect of the tibia and fibula, runs behind the medial malleolus and inserts at the medial and plantar aspect of the tarsal and metatarsal bones. The most important insertion, however, is at the tubercle of the navicular bone (Fig. 20).

Because the tibialis posterior crosses the ankle, the subtalar and the transverse joints, it can act simultaneously on all three. The attachments to the other tarsal and metatarsal bones produce supination and are, therefore, very important in maintenance of the plantar vault.

The tibialis posterior is also a weak plantiflexor. Through its fibrous plantar attachments at the bases of the metatarsals, it also provides a plantiflexion movement at the midtarsal joints and therefore is responsible for supporting the anterior arch (see plantar movement at Lisfranc's joint). Congenital absence of these plantar attachments is therefore one of the causes of pes planus valgus.

The tibialis anterior originates from the upper lateral half of the tibia and crosses the dorsum of the foot to insert at the medial aspect of the medial cuneiform. Because it lifts all the structures of the medial arch – first metatarsal, first cuneiform, navicular and talus – it is an adductor, dorsiflexor and supinator.

Evertors (abduction–pronation)

The muscles with tendons that lie lateral to the axis BB′ act as evertors (abduction and pronation; Table 4). They are the

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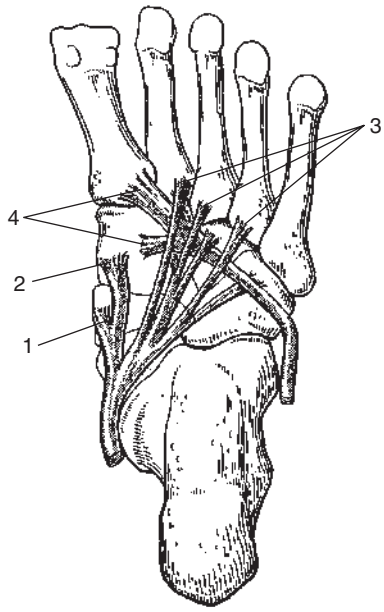

Fig 20 • Plantar view of the insertions of tibialis posterior, and its relationship with the tendon of the peroneus longus. 1, attachment to the tubercle of the navicular bone; 2, attachments to the tarsal bones; 3, attachments to the metatarsals; 4, insertions of the peroneus longus tendon.

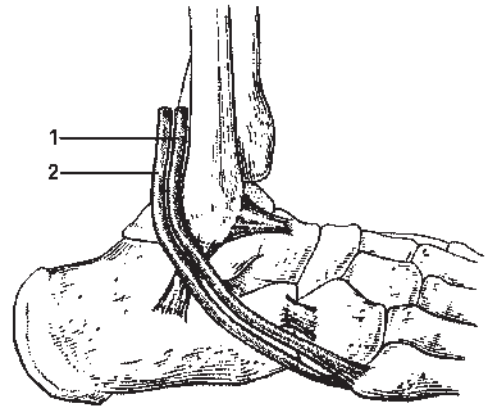

Fig 21 • The peronea.

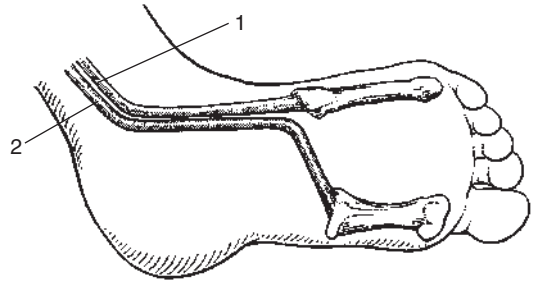

Fig 22 • Plantar view of the peronei in abduction-pronation: 1, peroneus brevis; 2, peroneus longus.

Table 4 The evertors

	Peripheral nerve	Spinal innervation
Peronei	Deep peroneal	L5, S1
Extensor digitorum longus	Deep peroneal	L4, L5, S1

extensor digitorum longus, peroneus tertius and peronei longus and brevis. The small peroneus tertius is absent in more than 10% of subjects and does not have any role in disorders of the foot.

The most powerful evertors are the peronei longus and brevis (see Standring, Fig. 84.2A). They arise from the lateral aspect of the fibula, the longus more proximally than the brevis. Their tendons run together in a common synovial sheath behind the lateral malleolus and are held in place by the retinaculum peroneorum. The tendon of the peroneus brevis, running more anteriorly and superiorly, inserts at the lateral tubercle of the base of the fifth metatarsal (Fig. 21). It acts as a strong abductor but also produces pronation of the anterior half of the foot.

The peroneus longus runs under the tendon of the brevis, along the lateral border of the foot. At the cuboid tubercle it turns obliquely medially and anteriorly to insert at the base of the first metatarsal and the first cuneiform bone (Fig. 20). The peroneus longus not only is an abductor–pronator and a weak plantiflexor of the foot, but it also plays an important role in the dynamics of the plantar arches. The abduction–pronation function is obvious from the view shown in Fig 22.

Plantiflexion is produced in two ways; directly and indirectly. Direct plantiflexion needs no further explanation, because the tendon runs behind the axis AA′ (Fig. 17). To understand indirect plantiflexion, the action of the triceps must be considered. Contraction of the triceps plantiflexes the calcaneus and the lateral metatarsals, connected through the cuboid with the calcaneus. By coupling the medial with the lateral metatarsals, the peroneus longus allows the triceps to pull on all the metatarsals at one time.

Another function of the peroneus longus is to accentuate the curvature at the three arches of the plantar vault. Together with the plantar expansions of the tibialis posterior, it provides an important muscular support of the transverse arch. In that it runs obliquely to the anterior and medial side, it also acts as a tightening device for the medial and lateral arches.

Intrinsic muscles

The sole of the foot contains a number of muscles that take origin and insert on the plantar aspect of the foot. Most arise from the calcaneus and the inferior part of the posterior segment and insert at or close to the phalanges.

The intrinsic muscles are the main contributors to the muscular support of the arches of the foot. They also assist the function of the long (extrinsic) muscles. Only the intrinsic muscles of the big toe and the dorsal interossei are clinically important.

The muscles of the big toe (flexor hallucis brevis, abductor hallucis and adductor hallucis) are inserted on the lateral aspect

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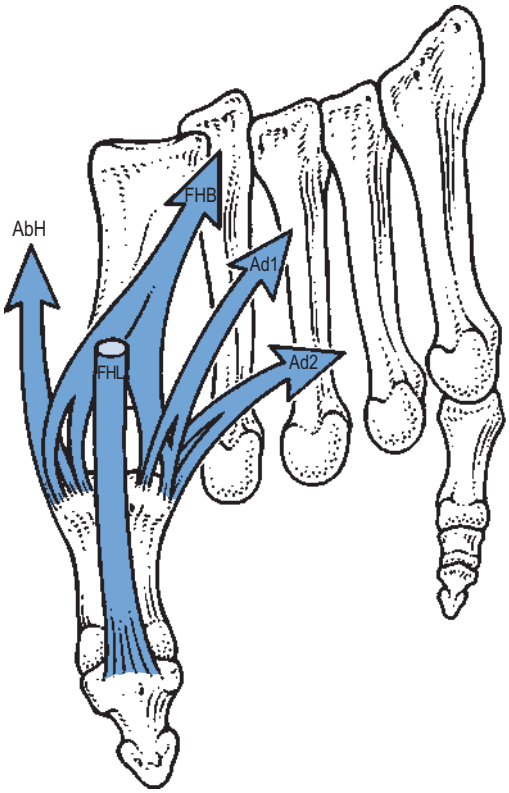

Fig 23 • Intrinsic muscles of the big toe: AbH, abductor hallucis; FHB, flexor hallucis brevis; FHL, flexor hallucis longus; Ad1 and Ad2, adductor hallucis.

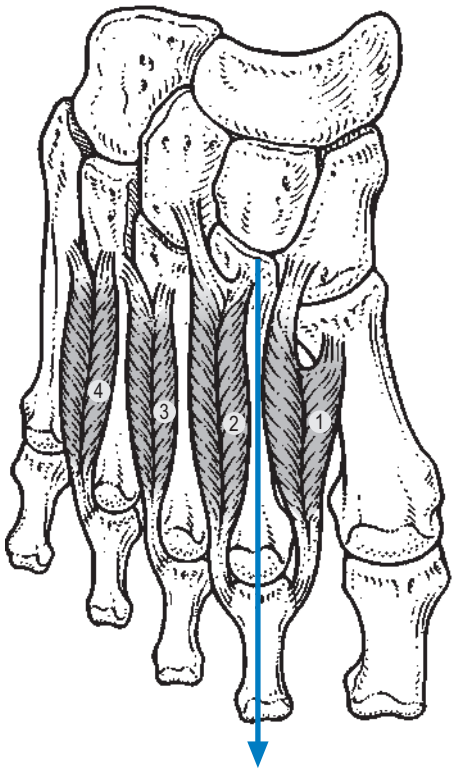

Fig 24 • Dorsal interossei of the foot.

of the basal phalanx and into the two sesamoid bones articulating with the head of the first metatarsal. The medial sesamoid gives insertion to the abductor hallucis and to the medial portion of the flexor hallucis brevis. The lateral portion of the flexor hallucis brevis and the adductor hallucis insert on the lateral sesamoid (Fig. 23).

Dorsal interossei

The four bipenniform dorsal interossei originate from the sides of the adjacent metatarsal bones. The muscle bellies fill the intermetatarsal spaces and their tendons attach to the bases of the proximal phalanges and to the dorsal digital expansions. The first interosseus inserts into the medial side of the second toe; the other three pass to the lateral sides of the second, third and fourth toes (Fig. 24). They abduct the toes away from the axis of the foot (second row) and assist in flexion and extension movement of the toes.

The plantar fascia

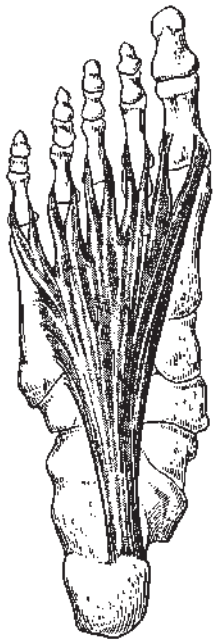

Fig 25 • The plantar fascia and its insertions.

Another structure that plays an important role in maintaining the longitudinal arches in the foot is the plantar fascia (Fig. 25). This multilayered fibrous aponeurosis originates at the medial tuberosity of the calcaneus and passes anteriorly to split into five fibrous bands, which attach to the digits in the following manner: each splits at the level of the corresponding metatarsophalangeal joint to allow passage of the short and long plantiflexor tendons and attaches at both sides of these joints and their ligaments. When the metatarsophalangeal joints are dorsiflexed during walking, the plantar fascia tightens and raises the longitudinal arch.

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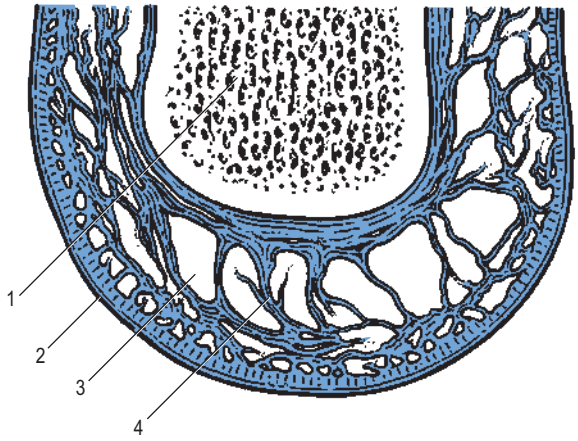

Fig 26 • Coronal section of the heel demonstrating the heel pad: 1, calcaneus; 2, skin and subcutis; 3, chambers; 4, vertical septa.

The heel pad

The heel pad is specifically constructed to function as an efficient shock absorber to attenuate the peaks of the dynamic forces during walking and running. The thick skin is connected with the periosteum of the calcaneus by large vertical fibrous septa, dividing the subcutis into separate compartments, filled with fat (Fig. 26). During compression these heel chambers are deformed and turn around the tuberosity of the os calcis. This deformation of the chambers together with the flow of the enclosed adipose tissue contributes to the shock absorption.

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Differential diagnosis of lesions at the heel and ankle

	Disorder	Diagnosis	Treatment
Posterior pain	Achilles tendinitis	Pain during rising on tiptoe Differentiate 4 types by palpating insertion, localizing sign and passive plantiflexion	Deep transverse friction Alternative: triamcinolone
	Achilles tenovaginitis	Discrepancy between symptoms and signs Warm, swollen and tender to the touch	Medication Triamcinolone
	Subachilles bursitis	Painful passive plantiflexion	Triamcinolone
	Dancer's heel	Painful passive plantiflexion Some laxity of plantiflexion movement	Triamcinolone
	Os trigonum periostitis	Painful passive plantiflexion Limitation of plantiflexion (Painful resisted flexion of big toe)	Triamcinolone Surgery
	Subcutaneous bursa	Normal examination Painful palpation	Triamcinolone Better shoes Surgery
	Subcutaneous nodules	Normal examination Palpation of small, hard, tender nodules	Subcutaneous tenotomy
	Plantar fasciitis	Typical history and localization Normal functional examination Painful palpation	Horizontal, raised heel Triamcinolone
	Painful heel pad	Typical history Normal functional examination Painful palpation	Procaine
	Sprain of posterior talofibular ligament	Passive plantiflexion and eversion hurts at the outer posterior side	Triamcinolone
	Tendinitis of tibialis posterior	Painful resisted inversion and resisted plantiflexion Localization behind the medial malleolus	Deep transverse friction
	Calcaneal apophysitis	Boys aged 12–16 years Intermittent pain Normal functional examination	Rest

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	Disorder	Diagnosis	Treatment
Lateral pain	Peroneal tendinitis	Painful passive inversion Painful resisted eversion	Deep friction Alternative: triamcinolone
	Mucocele of peroneal tendons	Painful passive inversion Painful resisted eversion Palpable, fluctuating swelling	Aspiration Triamcinolone
	Snapping ankle	Same as peroneal tendinitis Palpation of moving tendon during plantiflexion–dorsiflexion	Surgery?
	Jumper's sprain	Typical history Normal functional examination Additional test: valgus during dorsiflexion	Triamcinolone Prevention
	Chronic adhesions of lateral ligament	History of varus sprain Painful inversion Changed end-feel	Manipulation
	Sprain of posterior talofibular ligament	Passive eversion and plantiflexion cause pain at the posterolateral side	Triamcinolone
	Ankle instability (lateral ligament)	History of varus sprain and fear of the ankle 'giving way' Laxity during passive inversion Positive anterior drawer sign	Wobble board training Surgery
Anterior pain	Sprain of the anterior tibiotalar ligament	Previous plantar sprain? Painful passive plantiflexion	Deep friction
	Anterior periostitis	Painful passive dorsiflexion	Triamcinolone
	Tenosynovitis of the extensors of the toes	Painful passive plantiflexion Painful resisted dorsiflexion of the toes Crepitus/fluid	Deep friction Triamcinolone
Medial pain	Tendinitis of tibialis posterior	Painful resisted plantiflexion and resisted inversion	Triamcinolone Deep friction
	Tendinitis of flexor hallucis longus	Painful resisted plantiflexion and resisted inversion Painful resisted flexion of the big toe	Triamcinolone Deep friction
	Sprain of deltoid ligament	Painful passive eversion and plantiflexion	Triamcinolone and support
Pain in the ankle	Unstable mortice	Pain and click during strong varus	Sclerosing injection Surgery
	Psoriatic arthritis of the ankle joint	Capsular pattern Warmth Fluid	Triamcinolone
	Haemarthrosis of the ankle	Trauma Nocturnal pain Capsular pattern Tender	Aspiration
	Osteoarthrosis of the ankle	Capsular pattern Hard end-feel Crepitus	Arthrodesis

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	Disorder	Diagnosis	Treatment
Pain in the ankle	Immobilizational stiffness of the ankle	After immobilization Limitation of flexion and extension	Mobilization
	Loose body in the ankle joint	Twinges (during walking downstairs) Normal clinical examination	Manipulation Root's shoe
	Immobilizational stiffness of subtalar joint	Immobilization Marked limitation of varus and valgus No muscle spasm	Mobilization
	Osteoarthrosis at the subtalar joint	After a fracture Limitation of varus Hard end-feel	Arthrodesis
	Loose body in the subtalar joint	Twinges Fixation in valgus or full range of movement	Manipulation
	Subacute traumatic subtalar arthritis	Previous sprain or foot injury Limitation of varus by muscle spasm	Triamcinolone Immobilization
	Monoarticular subtalar arthritis	No injury Capsular pattern and muscle spasm Joint is warm, swollen and tender	Triamcinolone
	Psychogenic pain	Fixation of the subtalar joint in varus	

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Disorders of the forefoot and toes

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The forefoot

Short first metatarsal bone

This is known as Morton's syndrome. Sometimes a short first metatarsal is related to pain, sometimes it is not. If the first metatarsal is too short and hypermobile, most of the body weight will be borne by the second metatarsal. Because of the hypermobility and shortening, the transverse arch becomes depressed and metatarsalgia results (p. e308 of this chapter).

March fractures

This condition was first described in 1855 by Breithaupt,[1] a Prussian military doctor, who stated that many soldiers developed painful swellings at the dorsum of their feet after long marches. He called this *Schwellfuss* but the cause remained unclear until Stechow[2] proved that the condition is caused by stress fracture.

Stress fractures of the metatarsal bones comprise 3.7% of all sport-related injuries, with the second and third metatarsal accounting for 80–90% of the fractures.[3–5] Stress fractures of the metatarsals are also common in military recruits and in long-distance runners. It has been reported that up to 20% of stress fractures in athletes[6] and 23% of stress fractures in military recruits[7] are located in the metatarsals.

The lesion should be suspected when a patient presents with unilateral and localized warmth and oedema at the dorsum of the foot. Often the symptoms appear after a long walk but sometimes there is no specific precipitating activity. It is surprising that children are almost as liable to marching fractures as adults. Cyriax's[8] (his p. 440) youngest patient was a 6-year-old boy.

During the first weeks after the onset of pain, there is local warmth and oedema over the dorsum of the forefoot. The

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normal findings on functional examination of the ankle and midfoot contrast with tenderness at the forefoot. Because not only the bone but also the interosseous muscles at both sides are involved, the tenderness is more extensive than might be expected from the fracture itself.

During the first few days, the fracture (usually a hair-line crack) may not be revealed by routine radiography. The diagnosis is confirmed after a few weeks, when the callus formation around the fracture becomes radiologically apparent. Earlier confirmation of the diagnosis is obtainable by either radionuclide bone imaging,[9,10] or ultrasound examination.[11–13]

Differential diagnosis

Localized swelling, warmth and tenderness in the forefoot can also occur in:

- *Gout in one of the tarsometatarsal joints*. A history of recurrent acute attacks of arthritis at the same joint or in other joints is helpful.
- *Rheumatic forms of arthritis, such as rheumatoid arthritis, psoriasis and reactive arthritis*. These disorders affect multiple joints. The history is also longer than the usual 6 weeks of that of a march fracture.
- *Freiberg's arthritis at the second metatarsophalangeal joint*. Although ischaemic epiphyseal necrosis of the base of the second metatarsal bone occurs in adolescence, the disease may be asymptomatic until adult life, when the deformity of the joint and the degenerative arthritis produce pain.
- *Ringworm and erysipelas*. Local cellulitis, caused by an infection of β-haemolytic *Streptococci* spp. or fungi, can cause considerable redness, thickening and tenderness. In erisypelas, there is malaise, chills and fever, together with an increased erythrocyte sedimentation rate. In ringworm, there is considerable itching and inspection of the interdigital clefts very often reveals a small localized infection between two toes.

Treatment

March fractures, whether partial or complete, always heal spontaneously in about 6 weeks. By that time, the bone is usually firm enough to be painless and mis-union need not be feared.

Treatment consists of relative rest. Normal walking need not be forbidden during the period of union. Immobilization is achieved when the forefoot is firmly bound, so that the other metatarsals splint the broken bone. Because, even with a splinted fracture, weight bearing still hurts a little, it is up to the patient to do as much as can be tolerated. If the pain is too severe, a plaster cast can be applied for 4 weeks.

If there is persistent pain after 6 weeks have elapsed, the interosseous muscles must be at fault. It is obvious that when there is a broken bone, the interosseous muscles will suffer abnormal stresses and become strained. Such a strain, whether the cause is a direct injury, a march fracture or an overstrain, hardly ever undergoes spontaneous cure and chronic pain can last for years. The only effective treatment for this condition

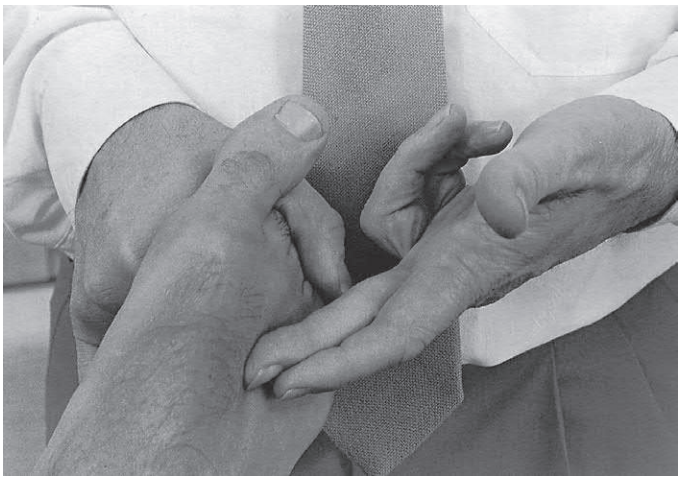

Fig 1 • Friction to the interosseus muscles.

is deep transverse friction for 15 minutes, three times a week, for about 2–4 weeks. After the massage, faradic and resisted flexor exercises are given to the toes. The results of this combined treatment are excellent.

Technique: friction

The patient lies supine on the couch. The therapist sits facing the foot and with the ipsilateral hand encircles the toes in such a way that the thumb lies at the dorsal aspect and the proximal interphalangeal joint of the flexed index finger presses under the metatarsophalangeal joints. The big toe is left free. The thumb keeps the toes flexed and the index finger presses upwards, so as to render the metatarsus convex. The shafts move apart and the muscle bellies lie closer to the surface. The middle finger of the other hand, reinforced by the index finger, is now placed in the groove between the two metacarpal bones (Fig. 1). Fingers, wrist and forearm are kept in one line and parallel to the metatarsal bones. Deep friction is imparted by rotating the fingers through an alternate pronation and supination movement of the forearm, so that the muscle is moved between the fingertip and the metatarsal shaft.

Fractures of the fifth metatarsal

Three distinct fracture patterns occur in the fifth metatarsal: tuberosity avulsion fractures, Jones fractures, and diaphyseal stress fractures.

Tuberosity avulsion fractures are the most common and usually complicate an inversion sprain (see p. 776). The majority heal with symptomatic care in a hard-soled shoe.

The true Jones fracture is defined as an acute fracture at the junction between the proximal diaphysis and metaphysis of the fifth metatarsal without distal extension beyond the fourth to fifth intermetatarsal articulation.[14] The mechanism of injury is believed to be an abduction force applied to the forefoot with simultaneous ankle plantar flexion.[15] A Jones fracture can be treated with 6 to 8 weeks of non-weight-bearing immobilization resulting in fair to good outcomes.[16,17] Operative treatment is selected if the patient is a high-performance athlete or nonoperative treatment fails.[18]

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A proximal diaphyseal fifth metatarsal stress fracture is defined as a stress fracture in the zone of the proximal fifth metatarsal immediately distal to the Jones fracture's anatomic area.[19] The mechanism of this injury is believed to be a repetitive load applied under the metatarsal head over a relatively short time, resulting in an overuse phenomenon. A proximal diaphyseal fifth metatarsal fracture may have a very long healing time and non-union can develop in as much as 25% of cases. For this reason, operative treatment is usually recommended.[20]

Splay foot

A splay foot is a broadened forefoot, with weakness of the intermetatarsal ligaments, associated with weakness of the intrinsic muscles.

Very often splay foot starts with excessive dorsiflexion movement of Lisfranc's joint, which occurs when too much weight falls on the midfoot. This is particularly likely to occur in women who wear high heels. When excessive weight falls on the midfoot, the talus is pressed downwards, and the metatarsals undergo an upwards pressure, so that they are given excessive horizontal play and the transverse arch flattens. The result is a painful overstretching of the transverse interosseus ligament and an increase in weight on the middle metatarsal heads. Calluses will form on the plantar surfaces of the heads and bruising of the plantar aspect of the capsule of the metatarsophalangeal joints (so-called chronic metatarsalgia) results. In practice, splay foot is not a specific entity but usually accompanies a midtarsal strain.

Treatment includes a high heel with a *horizontal* upper surface, which prevents the forwards gliding of the talus and thus releases the forefoot (see Ch. 59), and energetic training of the short plantiflexor muscles of the toes.

Localized splaying indicates a ganglion lying between two metatarsal heads. When the patient stands, an excessive interval is seen between two toes and palpation reveals a semisolid tumour keeping the heads apart (see p. e309 of this chapter).

The first metatarsophalangeal joint

The capsule of the first metatarsophalangeal joint is reinforced on the plantar surface by a fibrocartilaginous plate that is attached distally to the proximal phalanx and proximally to the plantar aspect of the neck of the first metatarsal. This volar plate contains the two sesamoid bones, inserted in the tendons of the flexor hallucis brevis (Fig. 2). The flexion–extension movement is a rolling and sliding of the metatarsal head within the stable support made up of the base of the proximal phalanx and the volar plate.[21]

The joint is extremely important for normal gait. There must be 60–70% of extension with the first metatarsal to allow the hallux to function normally during the stance phase of gait.[22] In the final stage of forefoot contact, 40% of body weight is imposed on the joint and the big toe[23] (Fig. 3).

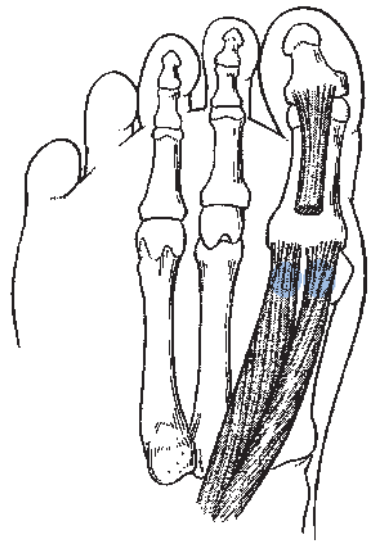

Fig 2 • The first metatarsophalangeal joint; colour indicates the location of the sesamoid bones within the tendons of the flexor hallucis brevis.

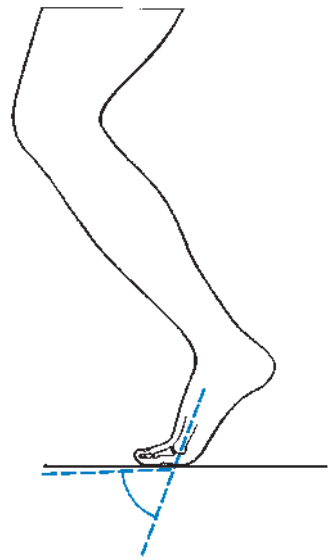

Fig 3 • The final stage of forefoot contact during walking.

During extension, the sesamoids are drawn from the head–neck junction of the first metatarsal to the distal head area. The average range of motion is 50°.[24] Apart from their function in weight bearing, the sesamoids also serve as a fulcrum that increases the mechanical advantage of the flexor hallucis brevis tendon.

The clinical examination of the big toe is summarized in Box 1.

The capsular pattern

The capsular pattern at the first metatarsophalangeal joint is slight limitation of flexion, together with marked limitation of extension (Fig. 4).

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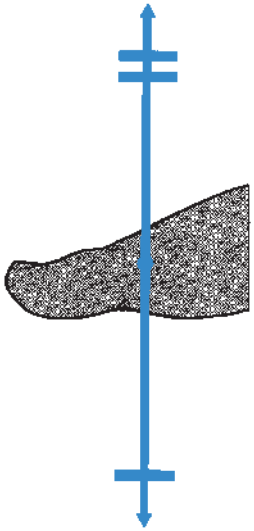

Fig 4 • The capsular pattern at the first metatarsophalangeal joint.

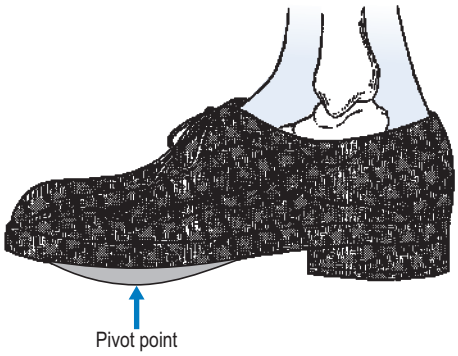

Fig 5 • A 'rocker' fitted to the shoe.

Box 1

Clinical examination of the big toe

Inspection

Redness and swelling: gout

Gross osteophytes: arthrosis

Valgus deformity and enlarged medial portion of the metatarsal head: hallux valgus

Ingrowing toe nails

Dermatological conditions

Functional examination

Passive extension and flexion

Normally the big toe can be extended to a right angle with the forefoot, whereas flexion is only 30–45°. Both end-feels are elastic

Passive abduction, adduction and rotations

Resisted extension: extensor hallucis longus and brevis

Resisted flexion: flexor hallucis longus and brevis

Palpation

For tenderness, fluid and osteophytes

Gout

The first metatarsophalangeal joint is usually the first joint to be affected in gout. The attack is characterized by a sudden onset of intense pain, frequently at night. The joint is swollen and exquisitely tender. The overlying skin is tense, warm and dusky red. The attack lasts 1–2 weeks, and asymptomatic periods of months or years commonly follow the initial acute attack.[25]

An acute attack can frequently be aborted by colchicine or by one of the non-steroidal anti-inflammatory drugs.[26–28] When attacks are frequent or other gouty complications such as tophi occur, lowering of blood uric acid is indicated.[29]

Arthritis in adolescence

Early osteoarthrosis at the first metatarsophalangeal joint, mostly bilateral, occurs in adolescence and is the result of osteochondritis dissecans.[30] It leads to the formation of a hallux rigidus in young adults.

The teenage patient, nearly always male, develops large osteophytes at the dorsum of both first metatarsophalangeal joints. The onset is slow and there is no history of overuse or of injury. Initially the pain appears only during hyperextension of the big toe. Later, the joint gradually fixes in the neutral position, leading to a hallux rigidus. Pain at every step is inevitable from stress on the rigid joint.

Clinical examination shows the big toe to be fixed in a neutral position. There is only a small range of extension and flexion. The end-feel is hard and large osteophytes can be palpated on the dorsum of the joint.

Because the patient is unable to extend the big toe during the foot-off phase, treatment must aim to introduce a forefoot movement that does not interfere with the mobility of the joint but nevertheless enables the heel to move upwards while the forefoot is on the ground. To do this, a 'rocker' is placed in or under a thick and solid sole of a shoe, at the joint line (Fig. 5).

Instead of extending at the first metatarsophalangeal joint, the forefoot will now rock during a normal gait. If this is not adequate, a steel plate in the sole prevents further stress on the rigid joint, but the resulting gait is less natural.[31]

Surgery for hallux rigidus consists of resection of osteophytes and metatarsal head (cheilectomy)[32] or resection arthroplasty.[33–35]

Traumatic arthritis

This results from a direct trauma or forceful hyperextension of the joint. This is the case in osteoarthrosis, in which a superimposed post-traumatic arthritis easily occurs. It is also encountered in certain sports, such as soccer and American football.[36–39] Treatment consists of an intra-articular injection of 10 mg of triamcinolone and prevention of further overstretching.

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Arthrosis in middle age

Osteoarthrosis at the first metatarsophalangeal joint (MPJ) is the second most common disorder affecting the foot after hallux valgus. The prevalence of the condition increases with age, and it has been reported that radiographic changes in the first MPJ are evident in approximately 46% of women and 32% of men at 60 years of age.[40] The degeneration and stiffness is not the result of a previous aseptic necrosis but has probably been induced by trauma or repetitive stresses. Sometimes a heavy weight falling on the joint or a fracture is the precipitating cause. Repetitive stresses during extension (as in women wearing high heels) can influence the degeneration.

Deterioration is slow and insidious, and therefore symptoms may not arise for years. They appear only when the joint becomes overstrained.[41] In other words, the source of the pain is not the osteoarthrosis as such but the superimposed post-traumatic arthritis. Symptoms therefore start when some dorsiflexion is lost: in men when 45° of extension range has been lost, in women who wear high heels when only 20 or 30° has been lost. The typical case is consequently a middle-aged man or woman who has pain at the big toe during and after walking.

Clinical examination shows a painful and markedly limited extension of the big toe, together with slight limitation of flexion. The end-feel is hard. Osteophyte formation and some local tenderness can be palpated at the dorsum of the joint.[42]

Because the pain is the result of a post-traumatic arthritis, it can be abolished by one intra-articular injection of triamcinolone. If the patient is careful in the future, wears appropriate shoes with thick, solid soles and avoids high heels, relapses are not to be expected. During the last decade an alternative treatment termed 'viscosupplementation' – the intra-articular injection of hyaluronan with the aim of restoring the viscoelasticity of the synovial fluid – has been proposed and studies show promising results.[43,44] Good results can also be achieved by traction. Alternatively, mobilization, using traction–translation techniques can be used.[45] In advanced cases, in which there is hallux rigidus, the use of a 'rocker' and a steel sole must be advised (Fig. 5). If all conservative treatment fails, surgery can be undertaken.[46,47]

Technique: injection

The patient sits with the foot flat on the couch. The physician grasps the distal phalanx and pulls it in an axial direction, with a slight flexion component. This distracts the joint surfaces and enables the joint line to be identified, which is not always easy to do in patients with large osteophytes: the depression between the osteophyte and the bone should not be mistaken for the joint line. A 1 ml syringe is filled with steroid suspension and fitted with a thin 2 cm needle. After identifying the joint line and the tendon of the long extensor, the needle is passed into the space between the two bones, medially to the tendon (Fig. 6) and the steroid is injected.

The patient should be warned of severe after-pain for 12–24 hours but will be pain free afterwards. If care is taken in the future, a second injection will not be necessary.

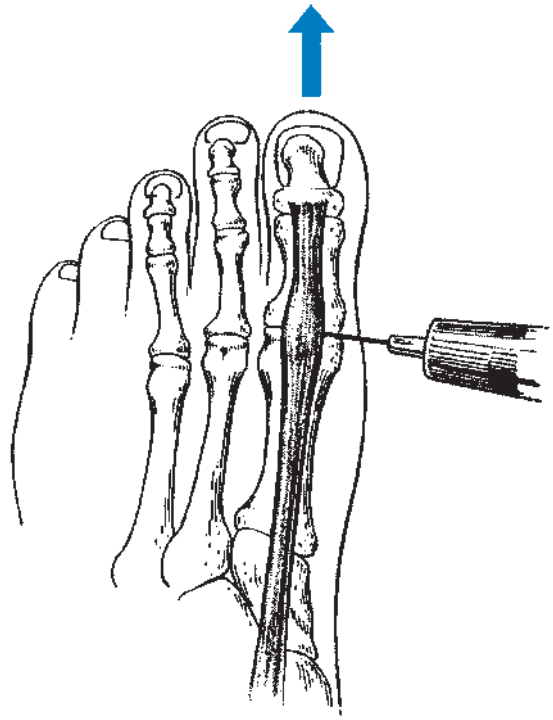

Fig 6 • Intra-articular injection of the first metatarsophalangeal joint.

Rheumatoid arthritis

This disease often affects the metatarsophalangeal joints.[48] The usual signs and symptoms of an inflamed joint are seen. The patient has nocturnal pain and morning stiffness. Clinical examination reveals a capsular pattern and a capsular thickening at a warm and swollen joint. Treatment is causative.

Non-capsular patterns

Metatarsalgia

Metatarsalgia at the first metatarsophalangeal joint is not as frequent as at the outer four toes. When it occurs, it is usually the result of an increase in the angle between the forefoot and the hindfoot. This is the case in a pes cavus deformity and also in osteoarthrosis at the cuneiform–first metatarsal joint (after a previous osteochondritis), which fixes this joint in plantiflexion and therefore increases the pressure on the plantar aspect of the first metatarsophalangeal joint during walking.

The clinical signs are pes cavus deformity of the foot, with normal mobility and painless resisted flexion of the big toe. However, there is significant tenderness at the plantar aspect of the capsule of the first metatarsophalangeal joint.

Treatment consists of an intra-articular injection of 10 mg of triamcinolone (see earlier). In a pes cavus deformity, the patient must also wear a raised heel with a horizontal upper surface (see Ch. 59). This measure relieves the excessive stress on the joint and prevents recurrences.

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Sesamometatarsal lesions

Traumatic periostitis of the sesamoid bone of the flexor hallucis longus usually results from local trauma, such as stepping barefoot on a sharp pebblestone or landing on the medial side of the extended joint.[49,50] After the accident the patient feels pain at the inner aspect of the forefoot with each step.

Clinical examination shows a full range of movement, sometimes with pain at the end of extension. Resisted flexion hurts at the plantar aspect and the sesamoid bone is tender to the touch, the exact point shifting with the position of the hallux.[24] Clinical diagnosis can be confirmed with conventional radiography and MRI imaging.[51,52]

Treatment consists of infiltration of the correct area with 10 mg of triamcinolone. Usually one infiltration is sufficient. Some authors advise orthotics[53] as a prophylactic measure but experience shows that this is never necessary. If conservative treatment is not followed by quick recovery, fracture[54,55] or post-traumatic osteochondritis[56] should be suspected. Immobilization in a plaster cast or surgical intervention is then required. Surgical treatment may include partial or complete resection of the sesamoid, shaving of a prominent tibial sesamoid or autogenous bone grafting for non-union.[49]

Alternatively, the pain can be caused by overuse, which sets up a traumatic arthritis at the sesamoid–first metatarsal joint.[57,58] This condition is similar to metatarsalgia of the big toe. Treatment is an infiltration with 10 mg of triamcinolone between the sesamoid and the first metatarsal, together with prevention by use of a raised horizontal heel.

Technique: infiltration and injection

The patient lies supine on a high couch. The tender point is identified. Because the skin is usually thick and difficult to sterilize, the medial side of the metatarsophalangeal region must be scraped until the epidermis is clean. A 1 ml syringe is fitted to a thin needle and filled with 10 mg of triamcinolone. The insertion is made from the medial aspect. The thumb of the free hand is kept on the tender spot and the needle is advanced in the direction of the palpating thumb (Fig. 7), whether at the sesamoid bone or at the joint between sesamoid and metatarsal. Depending on the lesion, an infiltration or an injection is then made.

Fig 7 • Infiltration of sesamoid lesions.

Halux valgus

This is the most common deformity of the big toe. It has been estimated that about 25% of the population have it to some degree.[59] There is no parallel between the degree of valgus and the severity of the symptoms, and many patients with severe deformity are free of symptoms.

Much has been written about the aetiology of hallux valgus.[60–62] Because hallux valgus also occurs in barefooted people who never wear shoes, there must be congenital factors predisposing to the deformity. The causative components seem to be multiple: the first metatarsal shaft is shorter than the second, there is a hypermobility of the first ray and the first intermetatarsal angle is enlarged.[63,64] All these factors contribute to the origin of a widely splayed forefoot (A in Fig. 8).[65,66] Because of the shortness of the first metatarsal, the second toe takes most of the weight during the final phase of the step. If there is a muscular imbalance between the adductor hallucis and abductor hallucis, the big toe will deviate laterally (B) and undergoes a pronation movement (C).[67] This is accentuated by the contractions of the flexors and long extensor of the toe, which act like a bowstring and shift the tip of the big toe further into adduction (D). When this predisposed splayed forefoot is now forced into a high-heeled pointed shoe, excessive weight is added and the first toe deviates increasingly and rapidly in a lateral direction. The pointed shoe increases the pressure at the medial aspect of the metatarsophalangeal joint, which results in an inflamed and painful bursa (tailor's bunion).[68, 69]

The diagnosis is made on the typical appearance:

- The forefoot is broadened and the transverse arch flattened.

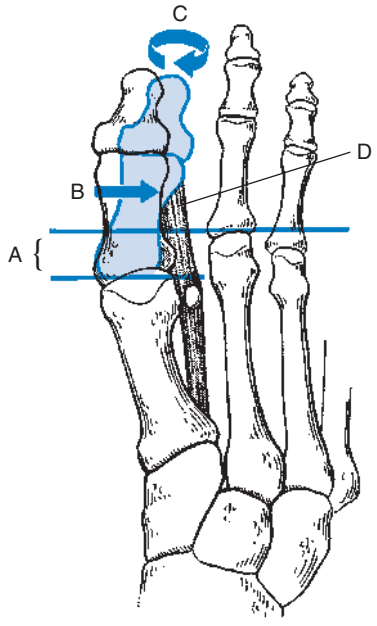

Fig 8 • Aetiologies of hallux valgus (see text for details).

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- The big toe is angled towards the second toe and frequently overlies it. The medial portion of the first metatarsal head is enlarged.
- The skin and the bursa over the medial aspect of the first metatarsophalangeal joint are inflamed and thickened.

Treatment depends on the degree of disability. It is as well to remember that many patients with extreme deformities have no pain at all. Because most of the pain stems from the compression of the enlarged forefoot in too narrow shoes, the primary conservative treatment is appropriate shoes: a wide shoe with a horizontal heel should be advised; sometimes a 'pouch' can be pressed out at the side of the metatarsal head. Local tenderness at the skin or at the inflamed bursa can often be treated successfully with ichthammol ointment. Surgery is indicated when there is continuous pain or for cosmetic reasons.[70–73] There is a choice of several procedures depending on the severity of the lesion and the age and mobility of the patient.[74,75]

The outer four metatarsophalangeal joints

The capsular pattern

The capsular pattern at the outer metatarsophalangeal joints is more limitation of flexion than extension (Fig. 9).

Rheumatoid arthritis and gout

Rheumatoid arthritis affects the metatarsophalangeal joints symmetrically. In advanced cases, the toes become fixed in the clawed position of extension at the metatarsophalangeal joint and flexion at the proximal interphalangeal joint.

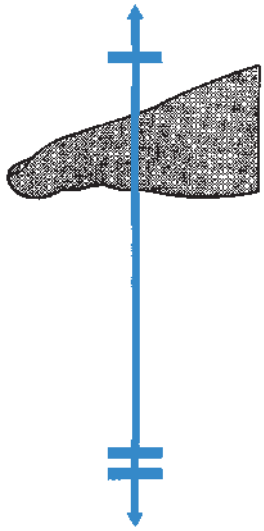

Fig 9 • The capsular pattern at the outer four metatarsophalangeal joints.

Gout is less frequent at the outer toe joints than at the big toe. The acute redness and tenderness should always be differentiated from a march fracture.

Freiberg's osteochondritis

This is an ischaemic epiphyseal necrosis of the head of the second metatarsal bone, first described by Freiburg in 1914,[76] and later by Kohler,[77,78] who showed that the third instead of the second metatarsal is involved in about 20% of cases. The disease occurs in adolescence, before the epiphyseal closure of the metatarsal head has been completed.[79]

The precise aetiology is unknown but it is assumed that the development is precipitated by an abnormally long second metatarsal bone, indirect trauma and changes in bone marrow pressure.[80,81] Sometimes the disease is asymptomatic until adulthood, when deformity of the involved metatarsal head and osteoarthrosis supervene and cause symptoms.

If the condition itself causes symptoms, examination will show localized arthritis of the second (or third) metatarsophalangeal joint, with local swelling and warmth and limitation of flexion and extension. It takes a month from the onset of pain before the characteristic radiographic changes become visible. Therefore, in its early stages the lesion is difficult to diagnose and a scintigraph or MRI image may be needed to distinguish it from a march fracture of the metatarsal shaft.[82,83]

Spontaneous recovery from this subacute stage takes up to a year. By that time the metatarsal head is permanently enlarged and palpation reveals a prominent ridge at the dorsal aspect of the metatarsal shaft. There is also some painless limitation of flexion and extension. Sometimes, and if no proper prophylactic measures are taken, metatarsalgia caused by the bony enlargement may supervene.

Later on, when the patient is 40–50 years old, osteoarthrosis may complicate the picture and the joint becomes fixed in a manner analogous to hallux rigidus.

Treatment consists of using orthotic metatarsal platforms to release pressure on the second metatarsal head. If there is limitation of extension, a 'rocker', as in hallux rigidus, can be prescribed. In advanced cases, surgery may be necessary.[84]

Traumatic arthritis

Traumatic arthritis at a metatarsophalangeal joint is rare. It can result from a direct blow or from indirect trauma, for instance during a hyperextension movement.[85] Recently, hyperplantiflexion injuries to the great toe sustained in beach volleyball players have been described. This injury is referred to as 'sand toe' and may result in significant functional disability.[86] Untreated, the capsulitis can continue for months, whereas one injection with 5 mg of triamcinolone can completely relieve pain within 2 days.

Osteoarthrosis

Local injury or a former Freiberg's osteochondritis can cause osteoarthrosis at the metatarsophalangeal joints. Limitation of flexion and extension with a hard end-feel results.

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Treatment consists of prescribing shoes with hard and thick soles, eventually fitted with a 'rocker' to render extension asymptomatic.

Non-capsular patterns

Chronic metatarsalgia

Pain in the plantar aspect of the forefoot is called metatarsalgia. Chronic metatarsalgia affects the middle three toes and arises when the metatarsal heads have to bear a disproportionate amount of the body's weight.

Aetiology

Scranton[87] differentiates secondary and primary metatarsalgia. In the former, the lesion is the result of structural changes in the joint (e.g. Freiberg's disease, rheumatoid arthritis or gout). The latter occurs if there is any incongruence between load and load-bearing capacity.

The aetiology of primary metatarsalgia is as follows. In a normal foot, only one-third of the body weight is borne by the forefoot. The transverse arch and the contraction of the long flexor muscles of the toes distribute the weight between the pads of the toes and the metatarsophalangeal joints. Because of the tautness of the anterior arch, the big toe and the outer toe take most of the weight. When too much weight falls on the forefoot, flattening of the anterior arch and insufficiency of the flexor muscles of the toes results in abnormal pressure on the plantar aspects of the second, third and fourth metatarsophalangeal joints.[88,89] In due course, a local capsulitis develops. This happens in the following conditions:

- *Splay foot:* the broadened forefoot, associated with weakness of the intrinsic flexor muscles leads to flattening of the anterior arch and some clawing of the toes.
- *Plantar deformity:* the dropped forefoot leads to too much pressure at the plantar aspects of the metatarsophalangeal capsules.
- *High heels:* if the shoe has a high heel with an oblique upper surface, the patient will stand on an inclined plane, sliding constantly downwards on the forefoot. Too much weight is imposed on the forefoot, with chronic metatarsalgia as a result. It is not the high heel itself but the oblique upper surface which is the source of the trouble. High heels with a horizontal upper surface do not cause problems and are even beneficial to women with a plantaris deformity of the forefoot. Unfortunately, no such ready-made shoes exist.
- *Pes cavus deformity:* a pes cavus deformity is very often accompanied by extreme hyperextension at the metatarsophalangeal joints, caused by shortening of the extensor digitorum muscles together with ineffective intrinsic flexors, holding the toes clawed.[90] In such cases, the toes do not bear body weight at all and serious metatarsalgia results, with large callosities under the metatarsal heads, together with corns at the dorsal aspects of the interphalangeal joints.
- *Weak flexor muscles:* sometimes the short muscles in the sole of the foot are weakened after a long rest in bed during an illness. If too much weight is borne too soon for too long a time, metatarsalgia can result.
- *Dancer's metatarsalgia:* a dancer working *en pointe* may bruise the plantar aspect of the second, third and fourth metatarsophalangeal joints.[91] To prevent this, most ballet shoes are fitted with a small semilunar pad, which ensures that the joints are protected and the body weight distributed as widely as possible.

Symptoms and signs

There is pain at the plantar aspect of the forefoot on standing and walking, relieved by rest. Sometimes a callus will form, which increases the pressure on the metatarsal head and aggravates the irritation.

Clinical examination shows pain at the end of dorsiflexion, sometimes at the end of both dorsiflexion and plantiflexion. Tenderness is noted under the metatarsal heads. Although the tenderness is described as being of the metatarsal heads, it is not the articular cartilage but the plantar aspect of the metatarsophalangeal joint that is at fault. The difference is important, because irreversible changes never take place and relief of too much weight bearing will always lead to full recovery.

Treatment

The most important measure is to avoid excessive weight bearing at the forefoot: the high heel must have a horizontal surface. This brings more weight onto the hindfoot than the forefoot and prevents the foot from sliding forwards. A support with its thickest part stopping just behind the heads of the metatarsals (metatarsal pad) must be fitted into the shoe. This ensures that the shafts of the metatarsals bear more weight than the joints themselves.

In a splay foot or after a long stay in bed, vigorous strengthening of the short flexor muscles of the toes by exercises is necessary, so that the toes flex properly at each step to take most of the body weight during walking.

In long-standing cases, good results can be obtained by a single intra-articular injection of triamcinolone into the joint.

Technique: injection

The patient lies supine. The joint is identified at the plantar and dorsal aspect; usually the joint line is more proximal than might be expected. A thin needle is fitted to a syringe filled with 0.5 ml of triamcinolone. By pulling at the toe and by moving the proximal phalanx in slight plantar flexion, the joint line becomes accessible to the needle. The needle is thrust downwards, lateral to the long extensor tendon. After 1 cm the tip of the needle is in the joint space and the steroid is injected.

Some soreness is to be expected for about 24 hours. Good results follow after 2 days. If preventive measures are also taken, the relief is permanent.

Interdigital ganglion

A ganglion arising from a flexor tendon sheath may arise between the toes at the dorsum of the foot. It can cause painful

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pressure, especially if small shoes are worn.[92] The diagnosis is obvious when a firm mass appears during standing. An excessive interval is then seen between the two toes and palpation reveals a semisolid tumour. Lying down permits the ganglion to recede. Because the mass is thick and composed of fibrolipomatous material, aspiration often fails. Excision is indicated if problems persist.

Pressure on the nerves in the forefoot

Symptoms caused by pressure on nerves at the forefoot are very often regarded as metatarsalgia. The differential diagnosis relies on the fact that the patient describes sharp, unexpected and sudden twinges instead of continuous pain during walking or running. During clinical examination, no signs of bruising of the plantar capsule of the metatarsophalangeal joints are discernible. Sometimes the symptoms can be reproduced by local pressure on a nerve.

Bruising of the second digital nerve

This is a rare condition, which is rather surprising, because the nerve passes forwards on the lateral side of the plantar aspect of the first metatarsophalangeal joint, close to the surface and unprotected from external trauma. It can easily be palpated as a thick band, before it bifurcates just distal to the joint, to supply sensation at the adjacent borders of the first and the second toes. If there is repetitive injury, bruising will persist. Consequently, the patient gets sharp twinges during walking. The twinges will be followed by pins and needles and by a constant ache.

During clinical examination the symptoms can be reproduced by local pressure on the bruised nerve.

Treatment consists of wearing a thick rubber pad under the forefoot for 3–6 months. An alternative is the injection of triamcinolone suspension around the inflamed nerve: a needle is introduced at the dorsum of the foot between the first and second metatarsophalangeal joints and thrust in the direction of the affected area, until it is felt to impinge against the plantar skin, where 1 ml of the suspension is infiltrated.

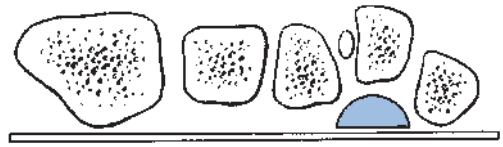

Fig 10 • Orthotic device for Morton's metatarsalgia.

Morton's metatarsalgia

This condition, first described in 1876,[93] results from a neuroma at the interdigital nerve between the fourth and fifth toes (exceptionally between the third and fourth toes).

The history is characteristic. The patient, usually a middle-aged woman, states that during walking she is suddenly seized by a sharp and unexpected pain at the outer side of the forefoot. She has to stop walking and characteristically take the weight off the foot and remove her shoe to rub and massage the painful area. After some minutes the pain ceases and she can continue to walk. The frequency of attacks is very difficult to predict, because sometimes there may be several attacks a day or none during a year. Usually recurrences tend to become more frequent. Between the attacks the patient is unaware of the condition and there are no symptoms at all.

Clinical examination is negative in most cases. Sometimes the symptoms can be reproduced by moving the heads of the adjacent metatarsal bones against each other during compression. Recently, ultrasound examination has been shown to be very sensitive in the detection of web-space abnormalities.[94,95]

The cause was described by Betts in 1940,[96] who stated that the painful condition results from a fusiform neuroma, about 1 cm long, of the fourth digital nerve proximal to its point of division.[97] When the fibrous swelling is nipped between the adjacent metatarsal heads, or pulled against a swollen transverse ligament,[98] sudden 'neuritic' pain results.

Treatment consists of relieving the pressure on the neuroma by alternating the alignment of the metatarsal heads. For instance, the head of the fourth bone can be elevated by a small support (Fig. 10). This will keep the bones slightly out of line and prevent pressure on the nerve. Steroid infiltration seldom succeeds. When conservative treatment fails, surgical excision of the neuroma may be required.[99,100]

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Nerve lesions and entrapment neuropathies of the lower limb

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Introduction

The main symptom of pressure on nerves is paraesthesia. Depending on the level of the compression, paraesthesia is accompanied by pain and numbness. The combination of these three symptoms and their interrelation are of importance in localizing the site of compression.[1]

Pressure on the distal spinal cord induces painless pins and needles in both feet, soon followed by numbness, incoordination and abnormal reflexes. This mechanism has been discussed in the chapters on the cervical and thoracic spine.

Pressure on a nerve root causes segmental pain, paraesthesia at the distal aspect of the respective dermatome and, if the pressure increases, motor and sensory deficit. For detailed descriptions of these pathologies, see the chapters on the lumbar spine.

Pressure on the lumbosacral plexus causes little pain but increasing numbness and weakness. For instance, compression of the plexus by a neoplasm does not cause pain in the limbs but only sacral or coccygeal pain. However, it does give rise to gross weakness of the muscles of one or both legs and feet.

The effects of pressure on the peripheral nerves of leg and foot are discussed in this chapter.

Sciatic nerve

Neurocompression syndromes of the sciatic nerve are not common. Some believe that the nerve can become compressed between the fibres of the piriformis muscle (the piriformis syndrome[2]).

If the nerve becomes damaged or is chronically irritated, serious deafferentation pain can result and occurs in circumstances in which the sciatic nerve is locally bruised or has undergone damage after local injection of irritating substances. Continuous and burning pain, independent of posture, is then felt in the sensory distribution of the nerve. Local pressure on the nerve can increase the pain considerably.[3–4]

Lateral cutaneous nerve

Entrapment of the lateral cutaneous femoral nerve is not uncommon and results in meralgia paraesthetica.

The lateral cutaneous nerve is sensory only. It originates at L2 and runs retroperitoneally to emerge at the outer edge of the psoas and then crosses the iliacus muscle at the lateral border of the pelvis, which it follows to the anterior superior spine of the ilium. It then passes under the lateral aspect of the inguinal ligament to follow the fibres of the sartorius muscle. Its course and the location as it exits the pelvis are very variable. Aszmann *et al*[5] investigated its relation to soft-tissue and bony landmarks in the inguinal region through dissection of 52 human anatomic specimens and identified five different types: type A, posterior to the anterior superior iliac spine, across the iliac crest (4%); type B, anterior to the anterior superior iliac spine and superficial to the origin of the sartorius muscle but within the substance of the inguinal ligament (27%); type C, medial to the anterior superior iliac spine, ensheathed in the tendinous origin of the sartorius muscle

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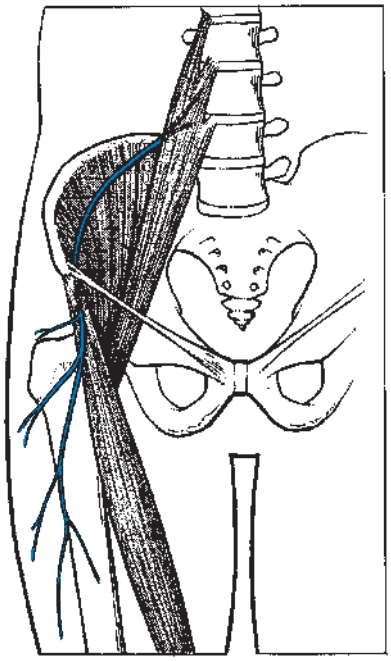

Fig 1 • Course of the lateral cutaneous nerve.

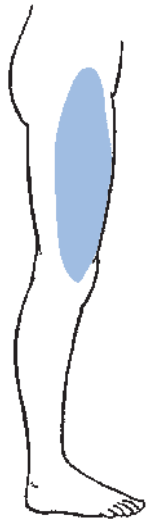

Fig 2 • Area innervated by the lateral cutaneous nerve.

(23%); type D, medial to the origin of the sartorius muscle located in an interval between the tendon of the sartorius muscle and thick fascia of the iliopsoas muscle deep to the inguinal ligament (26%); and type E, most medial and embedded in loose connective tissue, deep to the inguinal ligament, overlying the thin fascia of the iliopsoas muscle and contributing the femoral branch of the genitofemoral nerve (20%). Other studies located the nerve most commonly at 10–15 mm medial to the anterior superior iliac spine but in some cases it was located as far medially as 46 mm.[6,7] A few centimetres below the anterior superior iliac spine it emerges through the deep fascia and continues its course subcutaneously (Fig. 1).

The nerve supplies the anterolateral aspect of the thigh from the upper border of the trochanter to the level of the superior margin of the patella (Fig. 2).

The nerve can become trapped at any point along its course, although most cases result from nipping at the inguinal ligament or beyond the fascial tunnel, usually at the point where the nerve becomes superficial. Occasionally the symptoms stem from an abnormality in the pelvis, the typical example of which is meralgia during pregnancy (Cyriax:[1] pp. 297–298), encountered between the fourth and seventh months. In this condition, the symptoms are always unilateral and disappear spontaneously during the pregnancy. They have been ascribed to the pressure of a small fibromyoma against the nerve, close to where it emerges at the lateral border of the psoas. Meralgia paraesthetica has also been reported after pelvic osteotomies for Perthes' disease,[8,9] shelf operations for acetabular insufficiency[10] and after the removal of bone from the iliac crest for a graft.[11] However, most cases of meralgia paraesthetica are idiopathic, although some external causes such as tight trousers,[12] obesity,[13] the use of belts, corsets and trusses, or an overtight bandage round the pelvis[14] after an operation or during pelvic traction can compress the nerve just medial to the anterior superior iliac spine.

The symptoms are typical of any lesion of a small peripheral sensory nerve: pain, paraesthesia and numbness, confined to its distribution. The patient typically describes a burning or tingling sensation over the anterolateral aspect of the thigh. The edge is well defined and the centre is often completely anaesthetic. A common complaint is hypersensitivity: the patient even dislikes the touch of a cloth in the affected area.[15] Clinical examination reveals the extent of anaesthesia, which has a clear edge. Tenderness to pressure can sometimes be evoked distal to the anterior superior iliac spine. In some cases, the paraesthesia can be aggravated by tapping the nerve.[16,17]

Differentiation of meralgia paraesthetica from a second lumbar root lesion remains the greatest problem in diagnosis[18] and relies on the careful delineation of the paraesthetic area, the degree of numbness and a negative MR scan of the lumbar spine.[19,20] Although the L2 area and the area supplied by the lateral cutaneous nerve correspond well laterally, the second root also contributes to the innervation of the groin and the inner aspect of the thigh. Furthermore, in L2 root lesions the analgesia is very slight because of the overlap between L2 and L3, whereas in lesions of the lateral cutaneous nerve, there is almost full anaesthesia, with a clear-cut border (Box 1).

Box 1

Differentiation of meralgia paraesthetica from a second lumbar root lesion

The zone of paraesthesia is very well delineated
There are no symptoms towards the medial side of the thigh and the groin
Hypersensitivity of the paraesthetic zone
The numbness is well outlined and almost total in the centre
Tapping the nerve provokes paraesthesia (Tinel's sign)
MRI of the lumbar spine is negative

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Treatment depends on the underlying cause. In external pressure at the fascial tunnel, it is sometimes sufficient to remove the cause. Should this simple action fail, infiltration with anaesthetic at the point of contact is indicated.

Technique: infiltration

A 10 ml syringe is filled with procaine 0.5% and fitted to a thin needle 5 cm long. A point is chosen 5 cm below and medial to the anterior superior iliac spine. The needle is inserted and moved upwards along the anterior and medial side of the sartorius muscle. A fan-wise infiltration is made here.[21] Two to four weekly infiltrations may be necessary.

In intractable cases surgery can be considered.[22] It should be remembered, however, that in about two-thirds of cases the symptoms subside spontaneously over 2 years.[23,24]

Femoral nerve

Although the femoral nerve can be compressed by different processes in the psoas region, the pelvis and the groin, neither pain nor paraesthesia ever result.[25] The symptoms are a vague numbness in the anterior crural area and increasing weakness of the psoas and quadriceps femoris muscles. A new cause of femoral compression neuropathy has been reported during recent decades – retroperitoneal bleeding resulting from anticoagulant therapy.[26–28]

The *anterior cutaneous nerve* innervates the skin of the front of the thigh as far as the upper border of the patella (Fig. 3). It can be compressed at the point where it emerges through the fascia of the thigh, some 10 cm below the inguinal ligament. Local pressure or friction may cause pins and needles and cutaneous analgesia confined to the anterior aspect of the thigh, with a clear-cut border and almost complete numbness towards the centre of the area.

Treatment is removal of the external pressure and procaine infiltrations.

Saphenous nerve

The saphenous nerve is the largest cutaneous branch of the femoral nerve. It leaves the subartorial canal about 8–10 cm above the medial condyle of the knee. Some of the branches there provide innervation of the medial aspect of the knee. Another branch follows the sartorius muscle and becomes superficial just below the medial condyle of the tibia.[29] It then runs down on the leg to follow the great sapheneous vein over the anterior aspect of the medial malleolus (see Fig. 6). Its territory of distribution is the medial side of the leg, the medial malleolus and the medial border of the foot[30] (Fig. 4).

Traction on the saphenous nerve as it leaves the subsartorial canal may cause oedema, inflammation and thus compression[31–33] (Fig. 5). Direct compression of the nerve may also occur in front of the inner tibial condyle.[34,35] However, the usual site of compression is at the ankle, at the anterior aspect of the medial malleolus.

Saphenous neuralgia is also a well-known and common complication after harvesting of great saphenous vein for coronary artery bypass grafting. The main symptom is anaesthesia which may persist for a considerable time postoperatively.[36–38]

Paraesthesia over the inner aspect of the ankle and along the medial border of the foot, together with aching and numbness along the subcutaneous border of the tibia, results from either a direct contusion or sustained compression. Combined plantiflexion and eversion of the foot or flexion of the hallux may stretch the nerve and cause sharp neuralgic twinges.[39]

Procaine infiltrations of the nerve, level with the ankle joint, are often curative. Ten ml of procaine 1% is infiltrated each week, over 3 consecutive weeks.

Common peroneal nerve

The common peroneal nerve emerges at the upper and lateral aspect of the popliteal fossa, through the fascia between the

Fig 3 • Area innervated by the anterior cutaneous nerve.

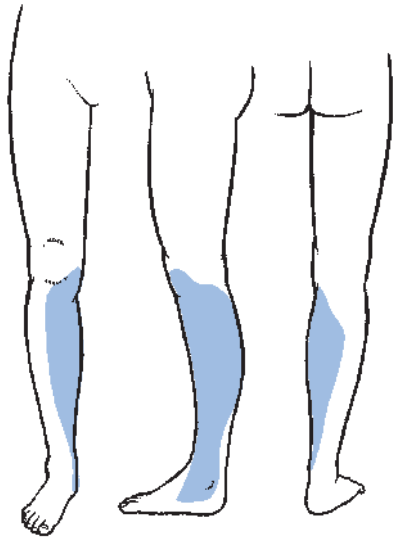

Fig 4 • Area innervated by the saphenous nerve.

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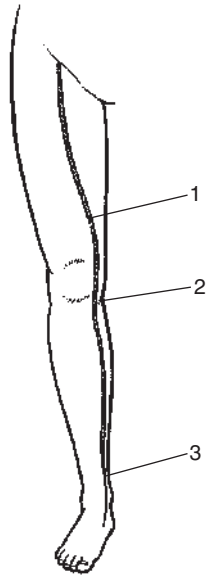

Fig 5 • Localization of entrapment along the saphenous nerve: 1, leaving the subsartorial canal; 2, in front of the inner tibial condyle; 3, at the anterior aspect of the medial malleolus.

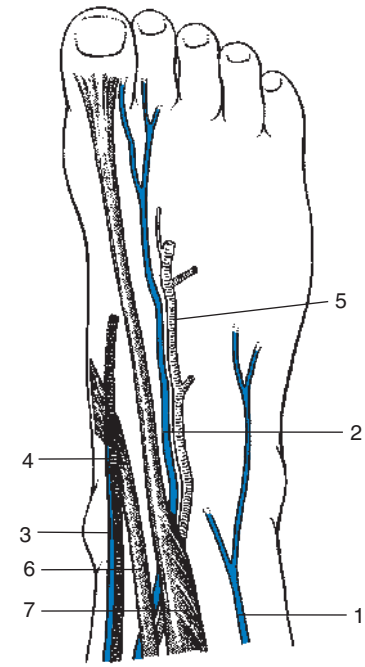

Fig 6 • Sites of compression and localization of infiltration of the superficial peroneal nerve (1), deep peroneal nerve (2), and saphenous nerve (3), the saphenous vein (4) and dorsal artery of the root (5) are also shown, (6) is the tendon of the tibialis anterior and (7) extensor hallucis longus.

biceps femoris tendon and the lateral head of the gastrocnemius.[40] It follows the biceps to the neck of the fibula where, under a fibrous edge beneath the origin of the peroneus longus, it divides into two branches. The superficial peroneal nerve continues under the peroneus longus, first along the fibula and then between peroneus longus and brevis.[41] The deep peroneal nerve winds around the fibular neck and runs through the anterior compartment between the extensor hallucis and tibialis anterior muscles until it traverses the ankle deep to the inferior extensor retinaculum.

Compression or elongation of the common peroneal nerve classically occurs where it winds round the lateral aspect of the neck of the fibula.[42] Direct compression follows immobilization in an over-tight plaster cast, fracture of the neck of the fibula or externally from pressure of the fibular head against a hard surface such as the side of a desk. Iatrogenic injury of the peroneal nerve can also occur from direct manipulation during orthopaedic surgery or with prolonged compression during lateral hip and leg rotation with knee flexion as occurs in operative positioning.[43] Elongation is frequent from sitting with the knees bent and the foot in full passive plantiflexion and inversion[44] or from prolonged squatting.[45] Long-standing compression causes atrophy and a drop foot. Slight and temporary compression or elongation of the nerve leads to 'neuropraxis' – a neurological deficit that recovers spontaneously within the course of 1–2 weeks. Chronic recurrent entrapment of the common peroneal nerve has recently been described in long-distance runners.[46,47]

There is only slight pain and there is no paraesthesia during the period of compression. Clinical examination reveals numbness of the dorsum of the foot and four inner toes, together with weakness of the tibialis anterior, extensor hallucis longus and peroneal muscles, which combine to produce a drop foot.[48]

There is no specific treatment but, in stretch or after moderate compression, spontaneous recovery is the rule. The patient must then be told how to prevent recurrence. In recurrent and transient entrapment, neurolysis of the peroneal nerve as it travels under the sharp fibrous edge of the origin of the peroneus longus can be performed and seems to give good results.[49] When the condition is caused by the pressure of an over-tight plaster or a direct blow, drop foot is usually permanent.

Deep peroneal nerve

Contusion of the terminal branch of the deep peroneal nerve can occur at the anterior aspect of the ankle, where it is relatively unprotected between the tendons of the tibialis anterior and the extensor hallucis longus, or at the dorsum of the foot[50,51] (Fig. 6). Compression neuropathy from tight boots or shoe straps has been called the anterior tarsal tunnel syndrome.[52,53] A direct blow produces the same condition.

The patient complains of aching deep in the medial and dorsal aspect of the foot and pins and needles at the adjacent borders of the big and second toes (Fig. 7). The symptoms are typically worse on activity and relieved by rest.[54]

Examination demonstrates diminished touch perception in the web space between the first and second toes. A positive percussion sign (Tinel's sign) is usually found.[55]

Treatment consists of wearing better-fitting footwear and three or four weekly procaine infiltrations around the nerve. Surgical intervention is seldom necessary.

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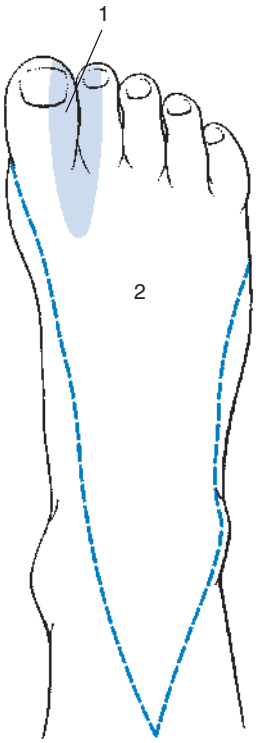

Fig 7 • Area within which sensory changes may be found in lesions of the deep (1) and superficial (2) peroneal nerves.

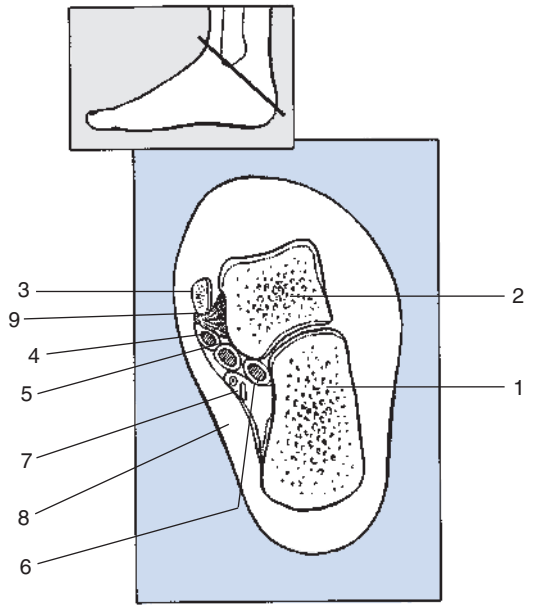

Fig 8 • The tarsal tunnel: 1, calcaneus; 2, talus; 3, tibia; 4, tibialis posterior tendon; 5, flexor digitorum longus tendon; 6, flexor hallucis longus tendon; 7, tibial nerve and artery; 8, flexor retinaculum; 9, deltoid ligament.

Superficial peroneal nerve

The superficial peroneal nerve emerges from the deep fascia at the junction between the middle and lower thirds of the leg. From this point it runs subcutaneously and is sensory only. It supplies the skin of the distal third of the front of the leg and the dorsum of the foot, except the adjacent borders of the big and second toes.

Entrapment can result from fibrosis after a direct blow[56] or surgery for chronic lateral compartment syndrome.[57] Transient tethering of the nerve during forced inversion and plantiflexion of the foot (ankle sprain) can also result in a momentarily painful stretch.[58,59]

The symptoms are pain, tingling and numbness over the dorsum of the whole foot and all the toes. Pressure or percussion (Tinel's sign) at the point of exit causes neuralgic pain and paraesthesia in the same area.[60]

Treatment consists of repeated injections with procaine at the site of compression. Should these fail, triamcinolone should be substituted. Fasciotomy and neurolysis relieve symptoms in only half of the cases.[61,62]

Tibial nerve

Entrapment of the posterior tibial nerve is most commonly seen at the medial aspect of the ankle and the midfoot in the so-called 'tarsal tunnel'.[63] The tarsal tunnel is an osteofibrous space bordered by the medial malleolus, the medial aspect of the talus and calcaneus and the flexor retinaculum. It contains the tibialis posterior, the flexor hallucis longus, the flexor digitorum longus and the posterior tibial nerve and artery[64] (Fig. 8). Consequently, compression of the posterior tibial nerve behind the medial melleolus and under the flexor retinaculum has been called the tarsal tunnel syndrome.[65,66]

The causes of compression are a space-occupying lesion (such as a bony exostosis), callus formation[67] or an inflamed and enlarged tendon sheath.[68] Excessive valgus deformity of the calcaneus with tension across the flexor retinaculum has been blamed.[69] Diseases such as rheumatoid arthritis, diabetes or a varicose vein have also been implicated.[70–72]

Patients with tarsal tunnel syndrome present with paraesthesia, burning pain and numbness in the plantar aspect of the foot and the toes[73,74] (Fig. 9).

These symptoms are frequently exacerbated during weight bearing, especially when valgus deformity causes the compression.[75] Many patients complain of nocturnal symptoms, which has been attributed to venous engorgement because the symptoms disappear when the foot is elevated. Symptoms can be reproduced by forced eversion and dorsiflexion of the foot.[76] Sometimes Tinel's sign is positive.[77] Pain and paraesthesia may also be reproduced by inflating a pneumatic tourniquet around the affected ankle.[78]

Treatment is first by correction of the underlying disorder. Infiltration with procaine or triamcinolone can be tried. If these measures fail, surgical release of the flexor retinaculum is considered.[79] However, the neurophysiological and clinical outcome of surgical decompression is only successful in half of the operations[80,81]. Recently, endoscopic techniques for the decompression of the tibial nerve and its branches have been used with acceptable results.[82]

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Fig 9 • Area within which sensory changes may be found in lesions of the tibial nerve.

Plantar nerves

These are branches of the posterior tibial nerve, running behind the medial aspect of the tuber calcanei, under the abductor hallucis and quadratus plantae. In valgus deformity of the ankle they can be compressed between the sharp calcaneal border and the shoe, to cause pain and burning sensation in the heel and foot.[83] Compression of the lateral nerve causes pain and paraesthesia at the outer side of the foot, medial nerve compression at the medial side.[84]

Treatment is correction of the valgus deformity and infiltrations with triamcinolone.[85] Should these conservative measures fail, surgery can be considered.[86]

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Psychogenic pain

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Introduction

Unless the patient has reasons for 'malingering' – the symptoms are simulated for compensation or litigation motives – pain is always indicative of some degree of dysfunction.[1] In most instances, the dysfunction is physical. Sometimes, however, the pain is devoid of any organic basis. If the patient has an unconscious belief in the reality of the symptoms, a psychogenic disorder is likely. The pain is then labelled 'inorganic' or 'functional'. There is an emotional illness and, although no peripheral tissue damage exists, the pain is as distressing as is somatic pain.

Pain is often the outcome of a combination of physical and psychological causes. It is a highly complex phenomenon: psychological factors affect the way people experience and express pain;[2] conversely, chronic pain often results in secondary personal difficulties.[3,4] Because pain is an abnormal affective state (though called into being by physical changes in the body), a heightened awareness may increase the severity of the symptoms. The patient may then present with irrational complaints that obscure genuine factors.[5]

The detection of disorders which have their origin in the patient's mind or from the desire to elicit sympathy or compensation is simple if the patient is examined by the methods set out in this book. However, it is more difficult to assess those who present with an organic lesion with psychogenic overlap. Here, much patience and clinical experience will be required to unravel the complicated clinical picture.

Orthopaedic medicine and psychogenic pain

Importance of immediate diagnosis

After the first attendance it is important to decide whether the patient does or does not have an organic lesion; this may be more difficult in an organic lesion with strong psychogenic overlap but, if the physician is alert, strong suspicion is usually aroused. For the sake of both patient and physician, psychogenic pain ought to be detected at once. If active treatment is applied to a patient who is dissembling or who is enmeshed in a compensation claim, nothing but an allegation of making symptoms worse can be expected. Also, in pain of 'inorganic' origin, the treatment will be completely inefficient and endless – the malady does not worsen but it never improves either. This is not only extremely discouraging to the physician and therapist but will also worsen the patient's mental state.

Conversely, it is unethical to regard the patient's symptoms as being devoid of organic basis simply because a treatment is unsuccessful. The label 'psychogenic pain' can never become an excuse for the physician's failure; it can only be given at the first interview and before any treatment is instituted.

Importance of a positive diagnosis

The diagnosis 'psychogenic pain' should be made on the discovery of positive inconsistencies during orthopaedic assessment and not as the result of supposition about possible psychological factors, such as anxiety, depression, stressful life situation or family dissatisfaction. A patient regarded as psychoneurotic can develop a genuine orthopaedic condition in parallel, and concentration on the psychological problem may distract the examiner from the organic cause of the pain. When there is an organic cause, thorough orthopaedic examination will reveal a simple and consistent pattern that markedly

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contrasts with the excessive nervous behaviour of the patient. The mechanofunctional aspects of the body are well defined and easy to interpret. In pain not of organic origin, the findings are self-contradictory and, given enough opportunity, the patient will sooner or later demonstrate inconsistencies.

To establish that there is not an organic basis for symptoms is extremely important in order to protect the patient from endless therapy or repeated surgery. However, to make an objective distinction between the different types of inorganic pain – whatever names they are given – is extremely difficult and requires further (psychological) assessment. An orthopaedic physician should beware of using diagnostic labels such as 'hysterical', 'hypochondriac', and 'regressed'. Because such classification depends merely on the doctor's sympathy or lack of it, it results in poor communication and engenders negative perceptions of the patient and a sense of pessimism regarding the prognosis.

Pitfalls for the examiner

The examiner must try to keep a balance between excessive scepticism and naïve trust. Not every statement should be accepted unconditionally but, conversely, not every patient with a symptom that differs from the usual should be regarded as suffering from an imaginary disorder. However bizarre the behaviour of the patient, the examiner should always face the clinical situation with an open mind that takes note of, and registers, the statements and findings. The diagnosis is then substantiated by the discovery of positive inconsistencies. The most important pitfalls are:

- the manner of the patient
- the obscurity of the history
- the potential bias of the examiner.

A patient with chronic pain who has seen several practitioners without benefit feels under suspicion or begins to have self-suspicion and develops the behaviour of a suspect. Strong emphasis on symptoms may occur with attention-seeking activity so as to convince the physician by exaggeration. However, it is not so much the current performance of the patient that may convince the physician that there is not an organic lesion but the remarkable sequence of events and the unlikely sensations that are described.

The examiner should also eschew the belief that a history or pattern not encountered previously does not exist: unknown is not the same as inconsistent. If the patient's symptoms remain within segmental boundaries, the complaints are consistent and the diagnostic movements are the same at each examination, an organic lesion is very likely. Although the examiner may not be able to make a diagnosis it must be obvious that here is a genuine, even though unfamiliar, disorder.

A serious pitfall in the detection of psychogenic disorders is the bias of the examiner towards the patient.[6] The doctor usually has initial sympathy for the patient who comes for help. However, dispassionate history and examination are essential. Only then is it possible to decide whether or not the patient's symptoms are psychogenic. A great effort is sometimes required to be completely objective but for the patient's sake it is essential. It is even more difficult for a doctor employed by an insurance company to remain unbiased; a detailed clinical approach is vital to establish facts and draw conclusions but is not partisan in a lawsuit (Cyriax:[7] p. 454).

Sometimes nothing can be found during clinical examination. Although the patient has a clear and well-defined complaint, the functional examination remains completely normal. If nothing can be found to account for pain, the decision that the symptom is psychogenic should not be taken lightly. It is possible that the examination has not been sufficiently precise to detect the organic disorder or the lesion is organic but not of orthopaedic origin. After serious internal disorders have been excluded, it is then fair to admit to the patient that one has failed to arrive at a diagnosis.

Clinical examination in psychogenic pain

Diagnosis follows the demonstration of positive inconsistencies during both the history and clinical examination. Sometimes the history contributes to the diagnosis, sometimes the examination will be the criterion but usually it is the overall picture – the combination of history and clinical examination – that is most informative. Inconsistencies may be found between symptoms, between signs and between symptoms and signs.

History

The examiner first listens to the history as related by the patient. Some accounts immediately draw attention to a possible existence of psychogenic pain or at least to a good deal of over-reaction. The patient describes symptoms in a melodramatic way: 'a spear going through my back'; the pain is 'dreadful, torturing, agonizing'. The patient's story is not a clear description of the symptoms but one of intense suffering, increasing disability and of ineffective treatments received.[8]

The examiner then asks precise questions, such as when did the pain start; how did it start; where was the pain first felt; to where did it spread? This technique of questioning is extremely helpful in defining the psychogenic causes. A patient with a genuine lesion may have difficulties in explaining himself, may be garrulous or apathetic, sullen or rambling, and fail to give a coherent account. Or, the invitation to give precise answers to precise questions will produce pleasure that at last a doctor will listen with interest and patience. A consistent report will then almost always be obtained, which quickly suggests the organic nature of the lesion. The response of patients with psychogenic symptoms is in strong contrast: because they do not know exactly what to say and refuse to commit themselves, precise answers are avoided. Pressed to describe the exact position of the symptoms they take refuge in very vague but exaggerated statements such as 'whole leg pain', 'whole leg numbness' and 'the whole leg giving way'. No position makes the pain better, or it comes and goes in the most improbable way. There is a tendency to embark quickly on the degree of suffering and the way the pain has influenced social, family or

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sexual life. When brought back to the point, there is reluctance to supply answers, which may even turn to irritation when the examiner continues with precise questions on the exact position of the pain, its variation and its spread.

Sometimes suspicion arises when none of the current and recognizable patterns emerges – the 'inherent likelihoods' (see Ch. 4). The lack of inherent likelihoods should put the examiner on guard. The patient is then allowed to go on talking so that contradiction of earlier statements may emerge.

Inspection

The patient is observed on entering the room and sitting down. Walking, sitting and undressing may demonstrate that certain muscles are not paralysed and establish that a degree of movement exists at the joints of the lower limb. The facial expression should be compared with the degree of alleged suffering. The face of a well-nourished and healthy patient does not correspond with the contention: 'I haven't had a wink of sleep in two months'.

If a limp is present, it should also be studied. The movement must be analysed and compared with the degree of dysfunction found during functional examination.[9] Joints behave in a typical way when diseased and create a characteristic gait. Psychogenic stiffness results in fixation in completely different positions which are, strangely enough, completely opposite: an arthrotic knee causes loss of extension, whereas in psychogenic disorders full extension is present; gross arthritis fixes the hip in full lateral rotation, psychogenic disorders in full medial rotation. In serious psychogenic disorders leading to fixation of joints, the 'wrong' joint is often held fixed: 'acute shoulder pain' with the shoulder girdle elevated and the neck flexed towards the pain; 'lumbar pain' with a gross deviation in the thoracic and cervical area.

Practitioner's checklist

Positive inconsistencies during the history:

- The patient's appearance does not fit with the alleged degree of suffering.
- Symptoms occupy an inconsistent area of skin: pain in the whole leg, spreading from the skull, over the scapula to the buttock and the limb, or affecting one half of the body.
- The patient cannot describe the localization and radiation of pain, or the pain always has different localizations.
- The sequence of symptoms, the variation and the spread of pain or the development of paraesthesia do not fit 'inherent likelihoods'. (Remember that what is inconsistent to the examiner is by no means so to the patient: ridiculous statements can be maintained by the complainer, despite being functionally or anatomically completely impossible).

Functional examination

In practice, functional examination is a better procedure to confirm or reject the diagnosis of psychogenic pain. Sometimes a nervous patient suggests a psychogenic component but the examination reveals a clear and consistent pattern. Conversely a patient who recounts a perfectly plausible history may show a clinical pattern highly suspicious of psychogenic pain.

There may be inappropriate, 'acting' behaviour:[10] the patient twitches or rolls about or rubs the painful area during the examination, in the meantime trying to confirm the degree of suffering by grimaces, sharp intakes of breath, groans and sighs.[11] It should be stressed, however, that acting behaviour is not proof of psychogenic pain. The patient may be so desperate in seeking attention that there is over-reaction. Although the performance may irritate the examiner, it should not alone be sufficient reason to regard the patient as not having an organic lesion.[12] It is not the behaviour of the patient but the existence of inconsistencies that leads to the diagnosis of inorganic pain.

The functional examination is started at a joint as far away as possible from the allegedly painful area: if the shoulder is said to hurt, the wrist and elbow are examined first; in supposed cervical pain, the thoracic spine and lumbar spine are tested first. The patient makes the assumption that, after the time-consuming history, the examiner will immediately start with those movements that usually cause pain and only few will resist the invitation to fake.

During an examination performed by the methods described in this book, a patient who is dissembling makes contradictions. A request is made for simple answers (it hurts; it does not hurt) regarding a large series of movements, which makes it difficult for the patient to work out quickly which movements might be expected to be painful and which not. Random answers are therefore given, forming a pattern that is not consistent with any one lesion. If the whole examination is repeated, the answers then form a completely different pattern. Alternatively the patient may state that every movement is painful. Suspicion also arises when the patient defers an answer or asks for the movement to be done again, so gaining time to formulate a reply. Patients without psychogenic pain say straight away that a particular movement hurts whereas another is painless.

When the physician suspects a psychogenic cause, attempts should be made to uncover more and more inconsistencies, further confirming an inorganic basis. Movements are tested again or in a different way, and movements not relevant to the alleged site of the pain are added to the basic set.

Positive inconsistencies in the functional examination

Fixation of the joint in a position opposite to the capsular pattern

In psychological disorders the hip is fixed in medial rotation, the knee in extension and the sublatar joint in varus.

Movements hurt at an impossible site

Flexion and extension of the knee provoke pain in the hip; pain in the back limits movements of the arm.

Repeat examination

A completely different pattern emerges during repeat examination.

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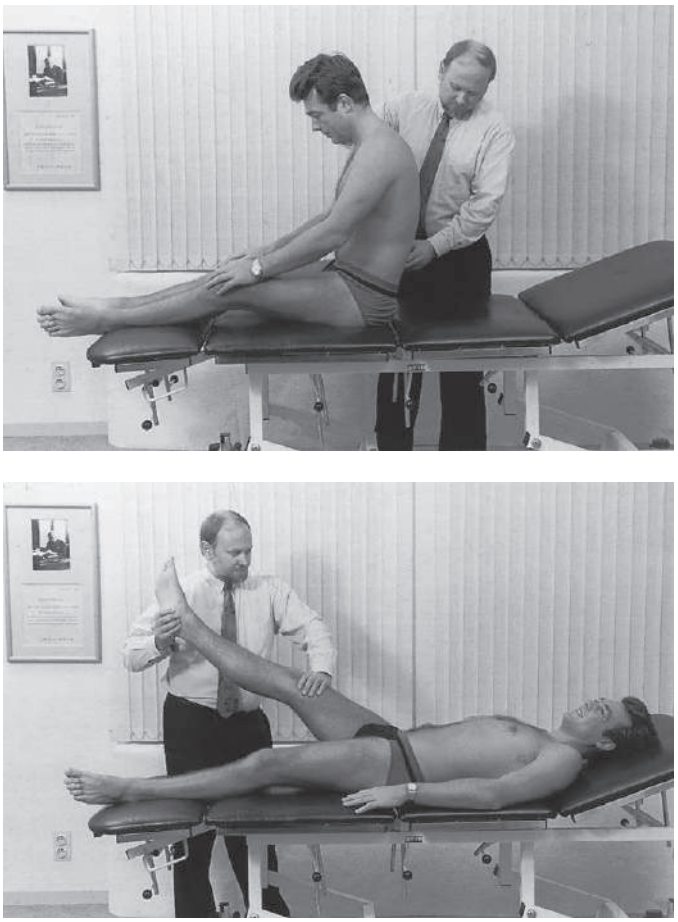

Fig 1 • Limitation of straight leg raising by pain can be demonstrated to be false if the patient can sit up with the legs straight.

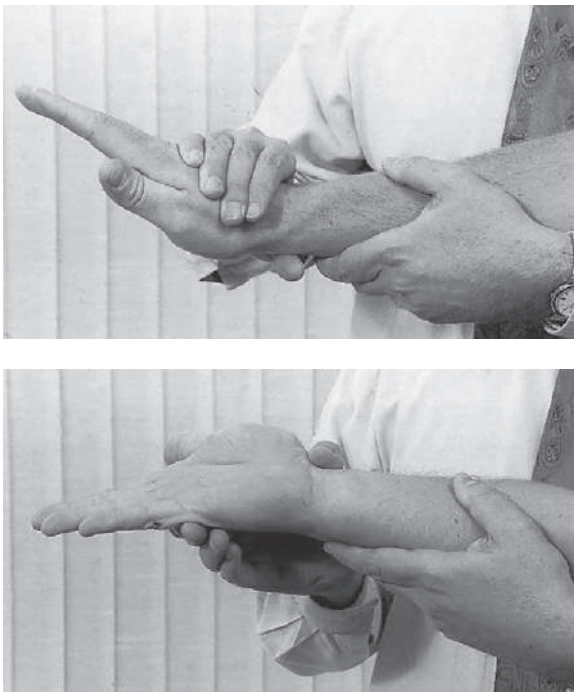

Fig 2 • Pain on resisted extension of the wrist is a false sign if the test repeated with the wrist in supination is negative.

The discrepancy between limitation in one direction and the completely normal movements of its components

Elevation of the arm is impossible but elevation of the shoulder girdle and scapulohumeral abduction are of full range.

Discrepancy between the results of the same movement carried out in different ways

The patient presents with straight leg raising grossly limited by pain but can sit up with the legs out straight (Fig. 1). A painful resisted extension of the wrist may prove to be painless if the test is carried out with the wrist in supination (Fig. 2).

Discrepancy between what the patient can do and the physical signs

A patient who sits normally must be able to flex the hip to 90°. Normal heel-to-toe walking may be observed, but resisted dorsiflexion of the foot is apparently weak.

Detection of alleged weakness

There are several ways to detect alleged weakness:

- *Discrepancy between the bulk of a muscle and weakness during manual testing:* as weakness leads to atrophy, a long-standing dysfunction always results in a decrease in muscle bulk.[13]
- *Weakness in manual testing which is not seen in other activities:* if the patient can move on and off the couch without help, there must be some strength in the flexors of the hip (Fig. 3). Alternatively a weakness may be found to be absent when the same movement is performed in another position: resisted knee flexion is weak with the patient prone but strong in the supine position.
- *'Cogwheel weakness' is found:* the patient, when asked to perform strong isometric contraction, does not fully cooperate and the examiner can feel that the contraction is actively countered by antagonist muscle activity. This results in a sequence of contractions and relaxations, which gives a typical cogwheel sensation.[14]
- *Unwillingness to cooperate can sometimes by demonstrated by simultaneous active antagonist contractions:*[15] the examiner can feel the contraction of an antagonist muscle, for example the triceps, when the agonist muscle, the biceps, is being tested. Dorsiflexion of the foot is weak, but accompanied by visible contractions of the toe flexors (Fig. 4).
- *Hoover's sign to detect an alleged weakness of the leg:*[16] the patient lies supine and the examiner lifts the heel of the affected leg. First the patient is asked to push the weak leg downwards – no pressure is felt (Fig. 5A). Then the patient is asked to lift the good leg, which this time provokes a downwards pressure in the 'affected leg' (Fig. 5B).

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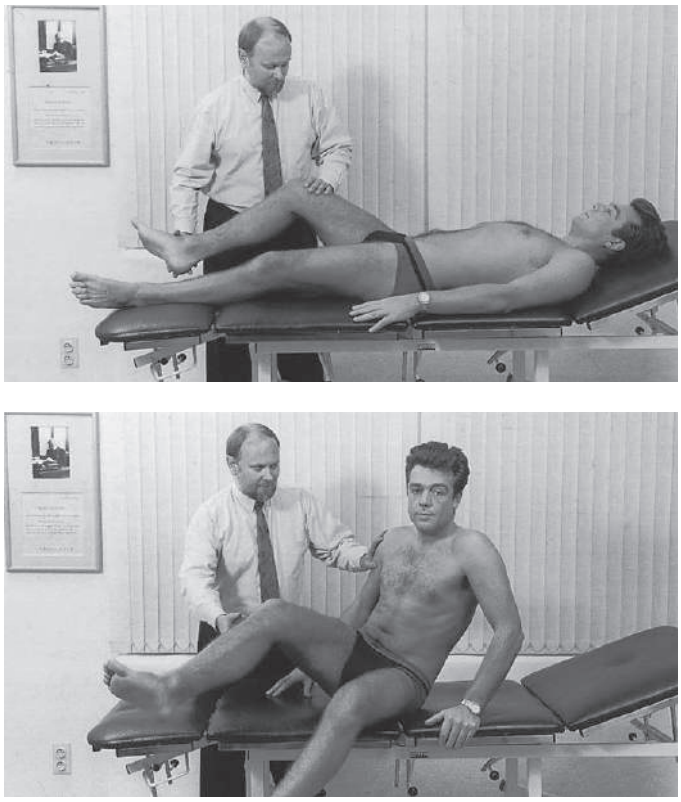

Fig 3 • Weakness on testing the flexors of the hip is not compatible with ability to get off the couch without help.

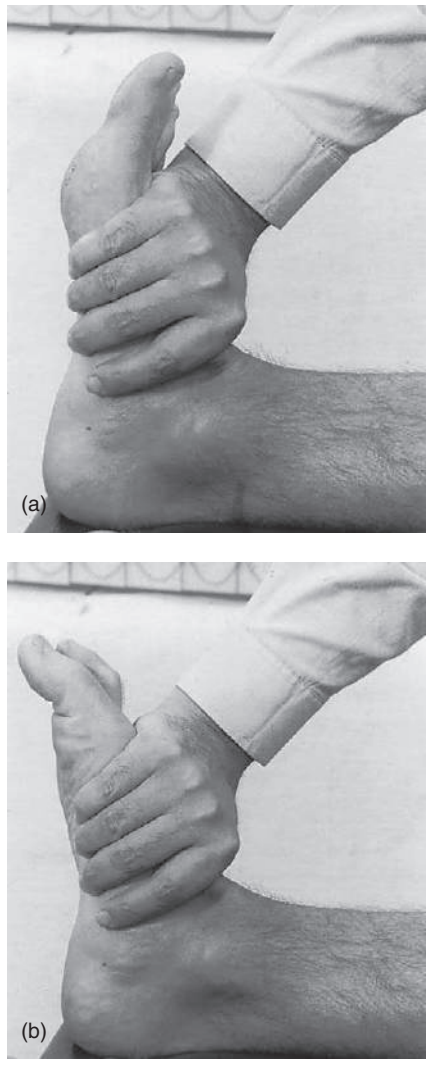

Fig 4 • In a genuine attempt to dorsiflex the foot against resistance the toes are straight (a) but contraction of the flexors is seen when the patient is uncooperative (b).

Practitioner's checklist

Positive inconsistencies in the functional examination:

- Fixation of the joint in a position opposite to the capsular pattern
- Movements hurt at an impossible site
- Repeat examination gives a different pattern
- Discrepancy between limitation in one direction and normal movements of its components
- Discrepancy between results of the same movement carried out in different ways
- Discrepancy between what the patient can do and the physical signs
- Weakness is demonstrated not to be genuine

Diagnosis of psychogenic disorders

The orthopaedic approach to suspected non-physical disorders is pragmatic: two questions need to be answered once clinical assessment is complete:

- Is there a genuine physical basis for the disability or not?
- If the pain is devoid of any organic basis, is it an unconscious (psychogenic) or a conscious process?

The answer to the first question is easy to ascertain, but the second is difficult because there are no clinical orthopaedic methods to make the distinction. If the patient is regarded as 'sincere' and possessing an unconscious belief in the condition, psychogenic pain is diagnosed – the patient is mentally ill. If it is believed that the patient deliberately assumes the symptoms – to deceive, to evade responsibility or to derive gain – the label of 'malingerer' is applied. However, no objective method of differentiation exists and sometimes one label, sometimes another, is used, depending on the examiner's sympathy or the lack of it (Box 1).

The patient who tries to reproduce symptoms or signs of an orthopaedic problem may do so in a number of ways: there may be pretence or perseveration. Pretence is used when the patient fabricates symptoms and signs; perseveration when signs and symptoms, once present, have ceased to exist but are continued by the patient.

Patients who exaggerate their condition are a particular problem: the symptoms and signs are magnified to represent more than they really are. Again the process may be conscious or unconscious. It is, for instance, quite possible that the patient tries so desperately to convince the examiner of a problem that over-emphasis occurs.[17] The symptoms and

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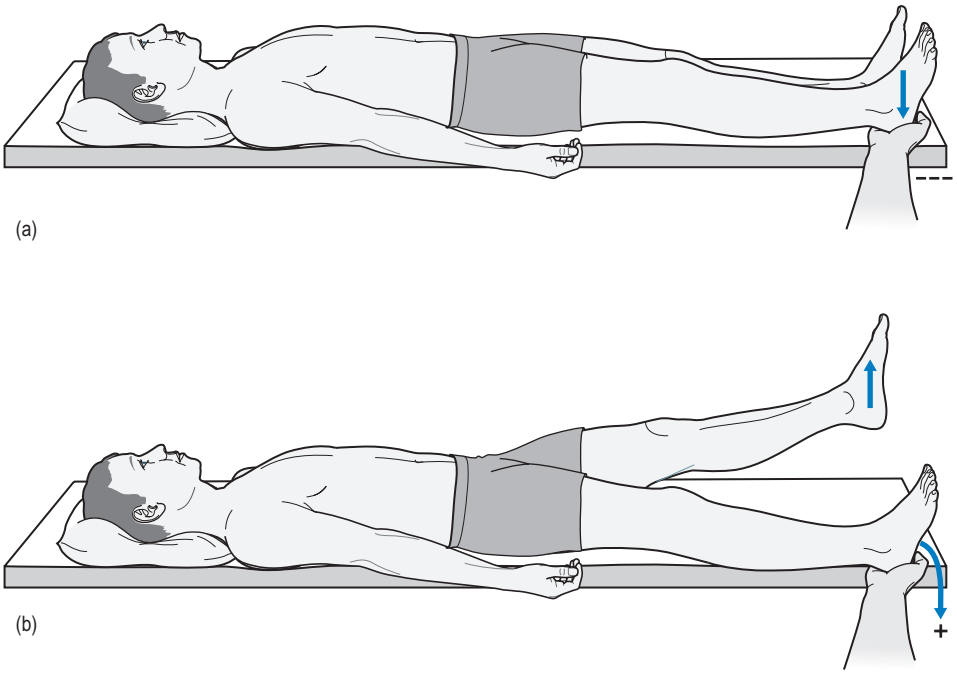

Fig 5 • Hoover's sign: No pressure is felt during active extension (A), but pressure is felt during active elevation of the contralateral side (B).

Box 1

Diagnostic possibilities in apparent psychogenic pain

Organic lesion

Organic lesion with psychogenic overlay

Organic pain in neurotic patients
Exaggeration:
- Conscious (gain)
- Unconscious

Absence of organic lesion

Pure psychogenic pain:
- Neurosis
- Hysteria
- Depression

Malingering:
- Pretence
- Perseveration

physical signs, though largely correct in quality, are excessive in quantity. Alternatively, the patient may have psychological problems but also develops a painful physical condition. In such a case, the pain generates such emotional distress that the consistent clinical pattern is overshadowed by emotional behaviour and excessive excitability. Much time, clinical experience and several repetitions of the examination may then be required to make a proper diagnosis, free from psychogenic overtones.

Treatment of psychogenic disorders

Organic pain with psychogenic overlay

Because a real – though minor – pain is present, the organic lesion should be treated first. Often the relief of the physical lesion, by depriving the patient of the basis of the malady, is curative alone. However, treatment should be adapted to the psychological condition of the patient. It is best to carry out the treatment techniques gently and carefully, in order not to provoke too much in the way of reactions. Only little should be done at once: for example one or two manipulative manœuvres, or 10 instead of 20 minutes of deep friction. The patient should also be warned, preferably in the presence of relatives, of a possible temporary increase in pain after each treatment session.

Absence of organic pain

Active physical treatment should never be given to patients whose pain does not have an organic basis. Not only is it useless but, if the patient's emotional pain gets worse, the treatment will be blamed for this. In contrast, it is very difficult to explain to a patient with psychogenic pain that the complaints are devoid of a physical basis.

The most unfortunate form of explanation is a straightforward declaration that there is nothing wrong and that the sufferer is fit to return to normal activities including work. Such a statement only induces an added determination to

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prove by more exaggeration that the disability is physical. Some method must be found to induce a change in attitude without loss of face, in that there is never willingness on the patient's side to admit to him- or herself, or to others that the disease is not organic. Communication of the absence of physical causes must be done with tact, diplomacy and in an understanding and sympathetic manner.[18]

One possibility is to say that an initial minor disorder, for unknown reasons, has triggered such a change in emotional tone that the latter has started to live its own life, although the original source of pain has now long vanished. In spite of this tactic, most patients find the diagnosis difficult to accept. Some are upset, reject the diagnosis and probably degrade the competence of the physician. It is therefore better to state the diagnosis *and the reasons for arriving at it* in the presence of the patient's nearest relatives. It is remarkable how many relatives immediately agree that the point of view is correct. Usually, they have suspected a psychogenic background but their assumption has never been supported by frank and objective medical statements. Once the relatives know the situation, they can start to help the patient in a positive way or at least provide protection from further useless and endless physical treatment or even surgery.

Another tactic for the physician is to declare, after the patient's confidence has been won by a sympathetic and benevolent attitude demonstrated during history and clinical examination, that an attempt must be made to put matters on the road to recovery. The patient is sent to the physiotherapist who applies daily electrical stimulation to 'all the affected muscles' over 2 weeks. The demonstration of normal contraction is then accompanied by praise from the therapist who reassures the patient that the muscles and nerves are normal but that there is a block to transmission of the message. The treatment, it is then suggested, will be addressed to releasing this block. Thereafter, the patient practises the hitherto impossible or painful movements. The role of the physiotherapist is essential: to be encouraging and sympathetic and try to establish a good relationship. The expression 'psychogenic pain' should never be used; the lesion from which the patient is suffering is a 'functional block'. Patients usually accept the diagnosis and such supportive treatment, with positive enforcement, is often highly successful. In a series of 107 patients regarded as suffering from purely psychogenic symptoms (therefore compensation cases were excluded) 76 declared themselves well and returned to work at the end of 3 weeks' supportive electrical treatment (Cyriax:[7] p. 460).

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Index

Notes
vs. indicates a comparison or differential diagnosis
To save space in the index, the following abbreviations have been used:
CT – computed tomography
MRI – magnetic resonance imaging

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