# Disorders of the ankle and subtalar joints

58

CHAPTER CONTENTS



## 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

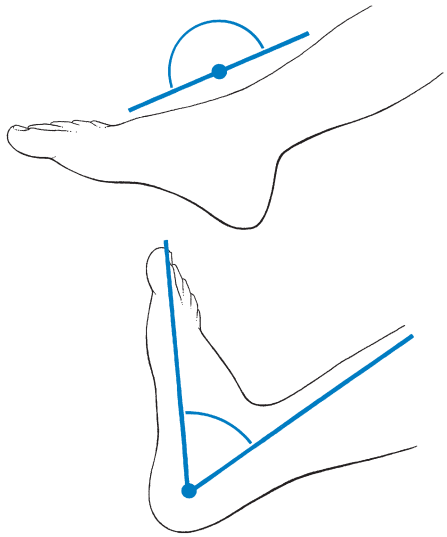


**Fig 58.1** • The normal range of plantiflexion (upper) and dorsiflexion (lower).

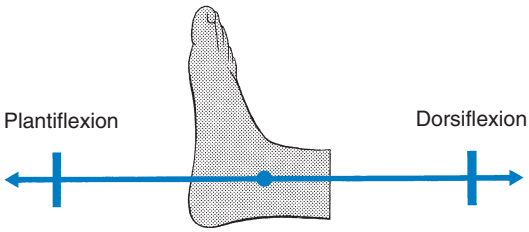



**Fig 58.2** • The capsular pattern at the ankle joint.

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

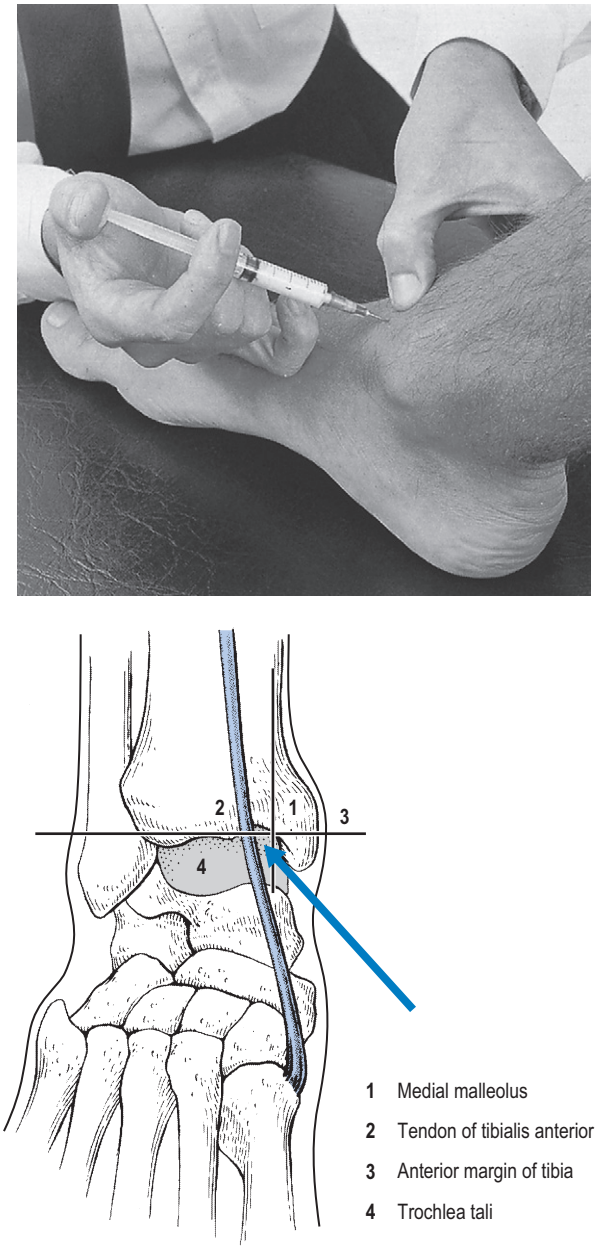



**Fig 58.3** • Landmarks for the injection of the ankle joint.

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.

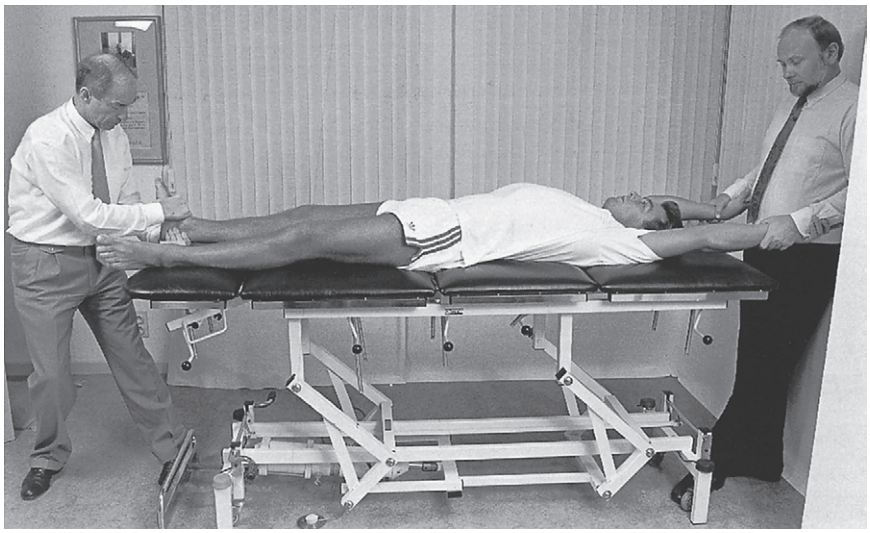
The result cannot be assessed until a week later.

## 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



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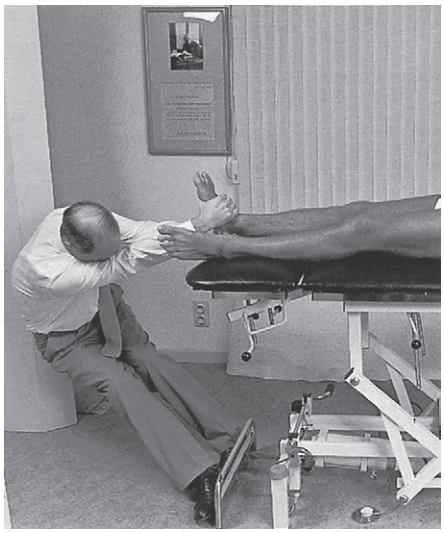

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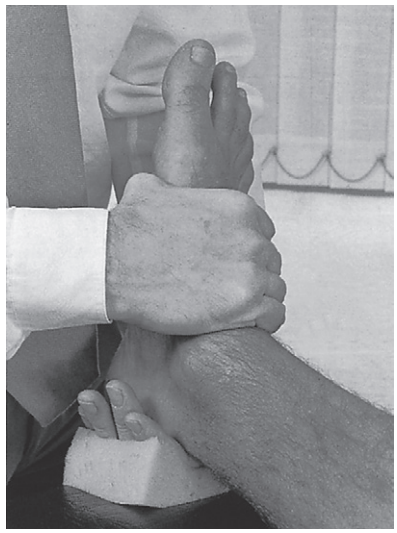


(c)

**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.

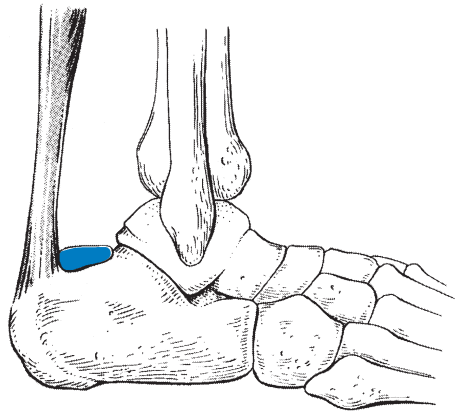


**Fig 58.5** • The Achilles bursa.

## 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.

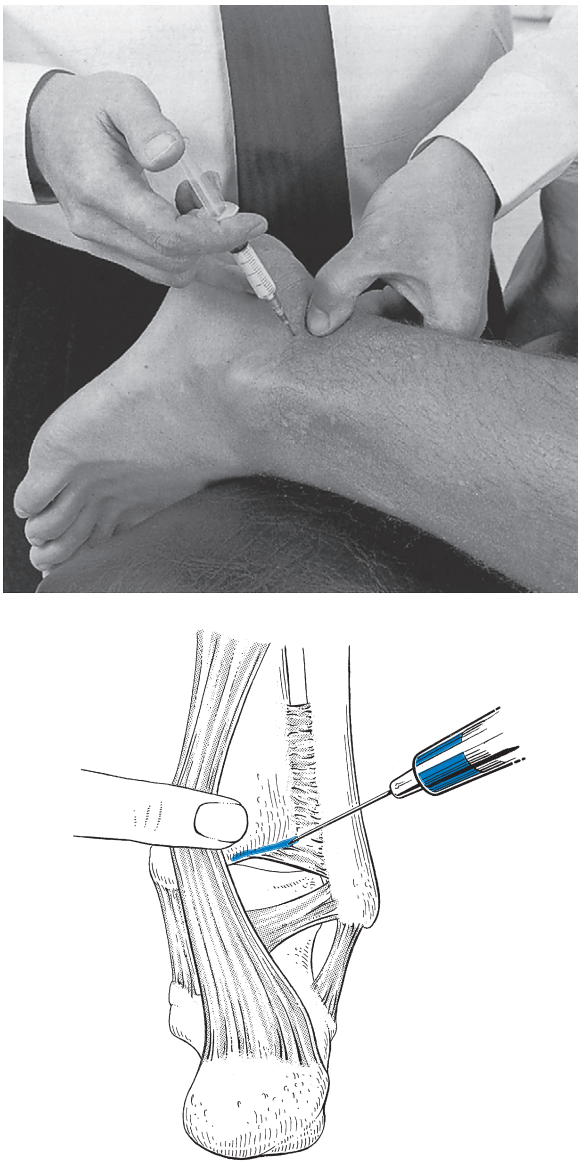


**Fig 58.6** • Infiltration of dancer's heel.

## 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.

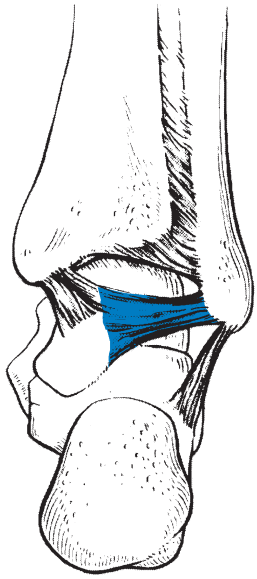


**Fig 58.7** • The posterior talofibular ligament.

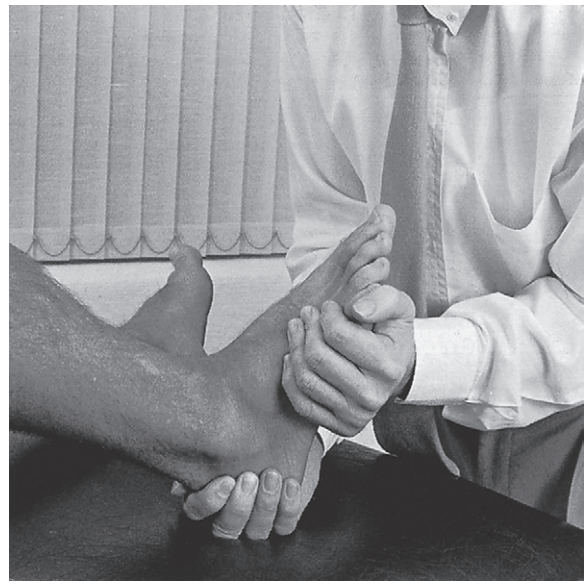


**Fig 58.8** • Accessory test in jumper's sprain.

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<br>Nocturnal pain | Capsular pattern<br>Warmth/fluid | Aspiration |
| | Rheumatic arthritis | Pain/morning stiffness | Capsular pattern | Triamcinolone |
| | Osteoarthrosis | Pain on weight bearing | Capsular pattern<br>Crepitus | Mobilization<br>Higher heel<br>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<br>Soft end-feel | Triamcinolone |
| | Dancer's heel | Posterior pain | Pain at full plantiflexion<br>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<br>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.

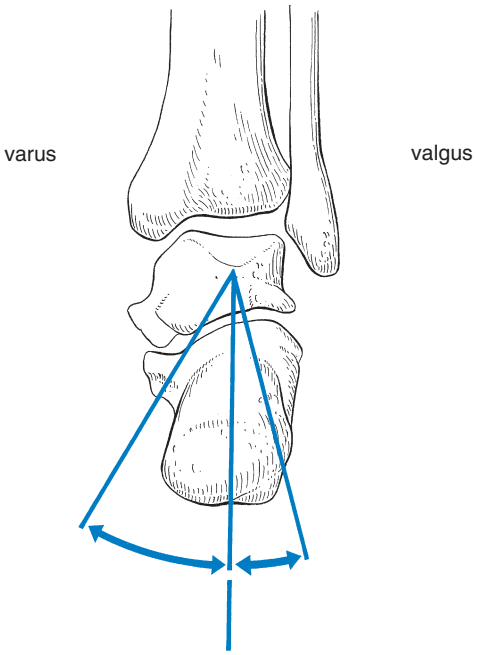



**Fig 58.9** • Normal varus and valgus movement at the subtalar joint.

### 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.

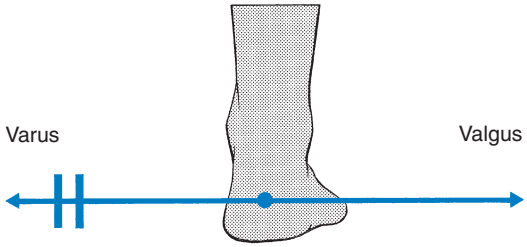
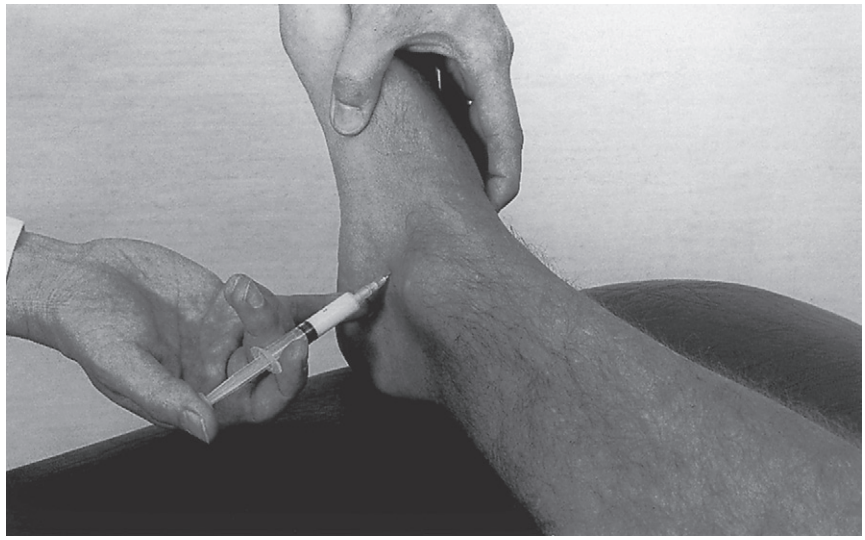


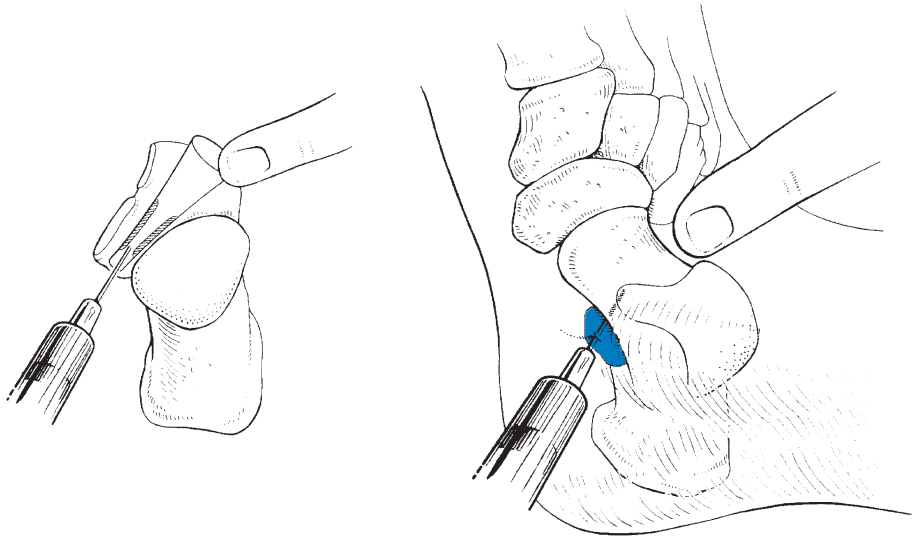

**Fig 58.10** • The capsular pattern at the subtalar joint.

***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.

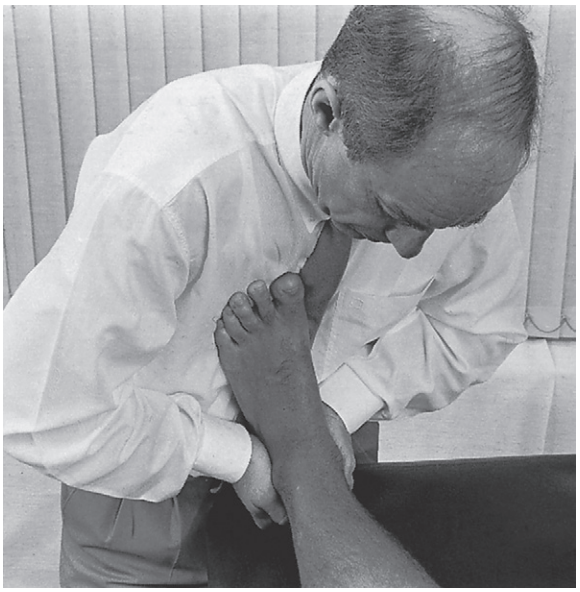


**Fig 58.12** • Mobilization for immobilizational stiffness of the subtalar joint.

## 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

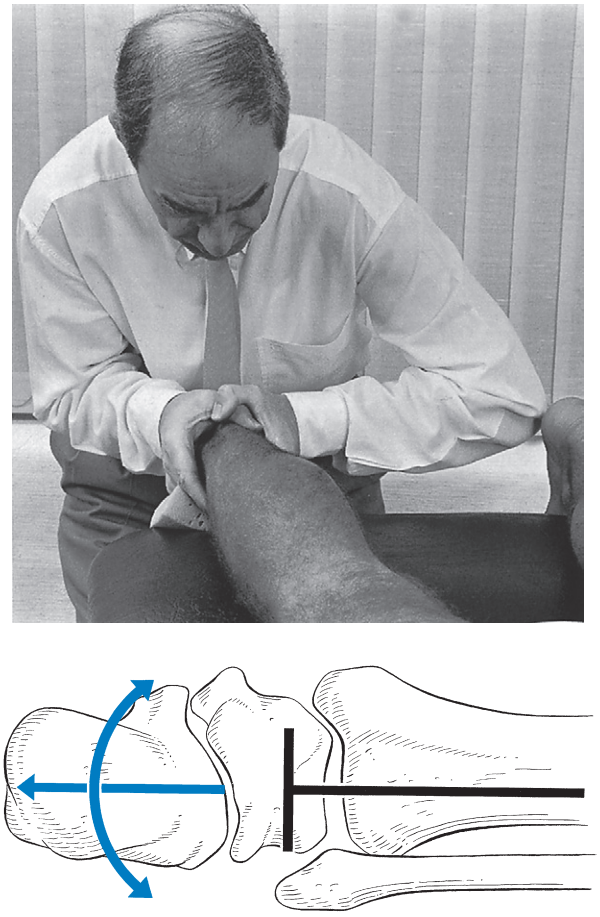


**Fig 58.13** • Manipulation for a loose body in the subtalar joint.

**Table 58.2 Summary of disorders of the subtalar joint**

| | Lesion | Symptoms | Signs | Treatment |
|---|---|---|---|---|
| **Capsular pattern** | Rheumatoid arthritis | Slow onset<br>Other localizations | Warmth<br>Capsular thickening<br>Muscle spasm | Triamcinolone |
| | Osteoarthrosis | After fracture | Hard end-feel | Arthrodesis |
| | Subacute traumatic arthritis | Previous trauma | Muscle spasm<br>Warmth | Triamcinolone<br>Immobilization |
| | Spasmodic pes planus | Young person<br>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.

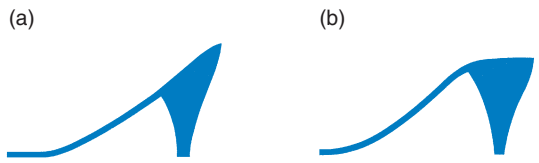



**Fig 58.14** • The upper surface of the shoe heel should not be sloping (a) but should be flat (b).

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.

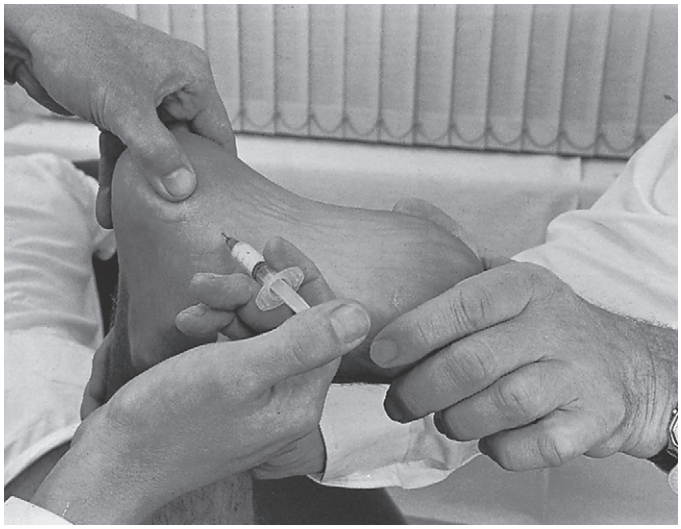
The patient's foot is held in dorsiflexion, either with the dorsum of the injecting hand or by an assistant. This position renders the plantar fascia taut and creates more room for the needle, which aims towards the palpating thumb on the tender spot. After traversing the resistant fascia, it touches 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



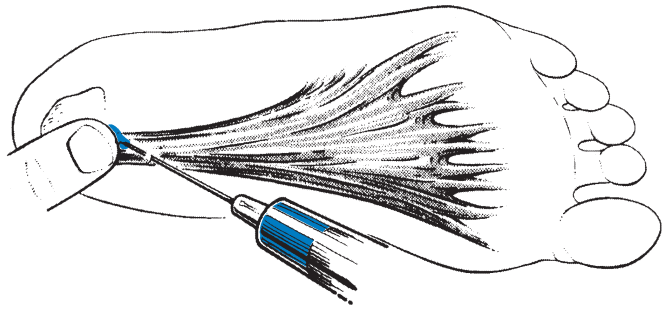


**Fig 58.15** • Injection in plantar fasciitis.

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

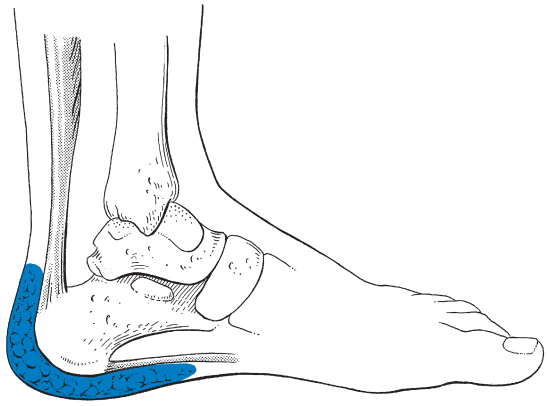


**Fig 58.16** • Localization of tenderness in heel pad syndrome.

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]

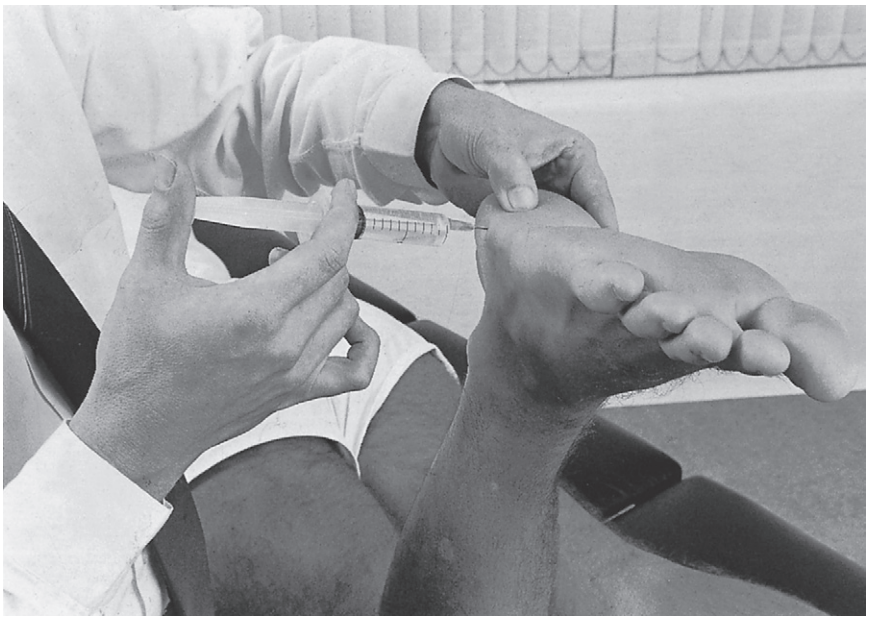


**Fig 58.17** • Injection in heel pad syndrome.

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.

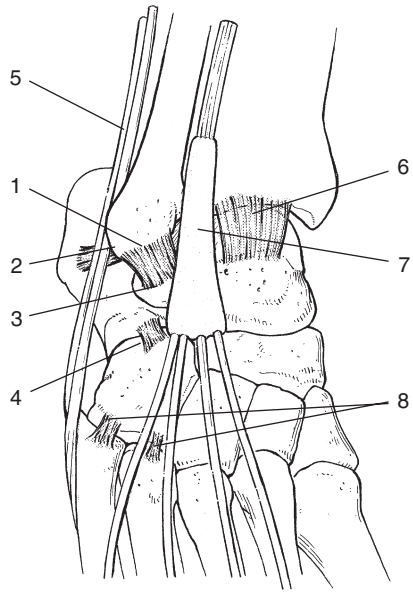



**Fig 58.18** • Sites of inversion sprain – see Box 58.1 for details.

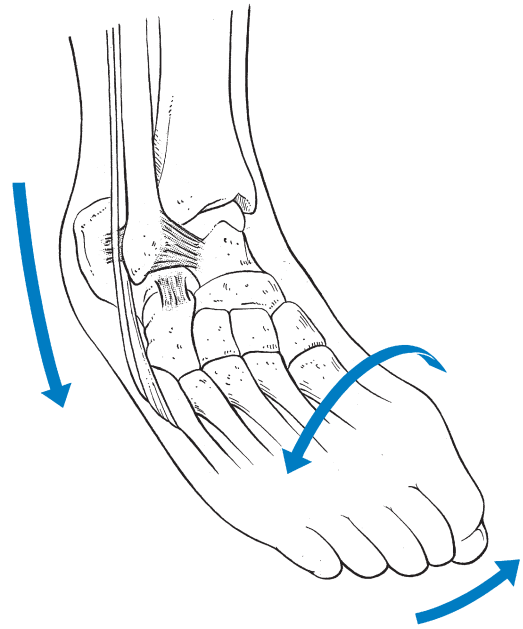


**Fig 58.19** • Mechanism of inversion sprains: varus and supination in increasing plantiflexion.

**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

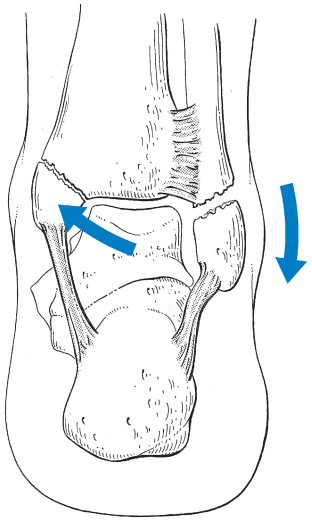


**Fig 58.20** • Malleolar fractures occurring with so-called varus sprain.

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<br>Plantiflexion |
| Calcaneofibular | Inversion<br>Varus |
| Calcaneocuboid | Inversion<br>Adduction and supination in the midtarsal joint |
| Anterior tibiotalar | Plantiflexion |
| **Tendons** | |
| Peroneal | Combined plantiflexion/inversion<br>Resisted eversion |
| Extensor digitorum longus | Passive plantiflexion<br>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<br>Dorsiflexion | Anterior talofibular/ calcaneocuboid ligament | A capsular pattern: haemarthrosis in the ankle |
| Varus<br>Valgus<br>Mortice | Calcaneofibular ligament | Excessive movement: total rupture<br>Lateral pain: lateral avulsion fracture of the fibula<br>Medial pain: medial compression fracture |
| Plantiflexion/ inversion<br>Plantiflexion/ eversion | All lateral ligaments and peroneal tendons | Excessive movement: total rupture<br>Lateral pain: lateral avulsion fracture of the fibula |
| Plantiflexion<br>Dorsiflexion<br>Pronation | Calcaneocuboid ligament | |
| Supination<br>Abduction | Calcaneocuboid ligament | |
| Adduction | Calcaneocuboid ligament | |
| Resisted plantiflexion | Peroneal tendons | |
| Resisted dorsiflexion<br>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<br>Midtarsal tests: negative<br>Resisted movements: negative |
| Calcaneocuboid ligament | Passive plantiflexion–inversion: painful, slightly limited, tight end-feel<br>Passive midtarsal supination: painful<br>Resisted movements: negative |
| Calcaneofibular ligament | Passive varus: painful |
| Peroneal tendon | Passive plantiflexion–inversion: painful<br>Midtarsal tests: negative<br>Resisted eversion: positive |
| Extensor digitorum tendon | Passive plantiflexion–(inversion): painful<br>Midtarsal tests: negative<br>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.

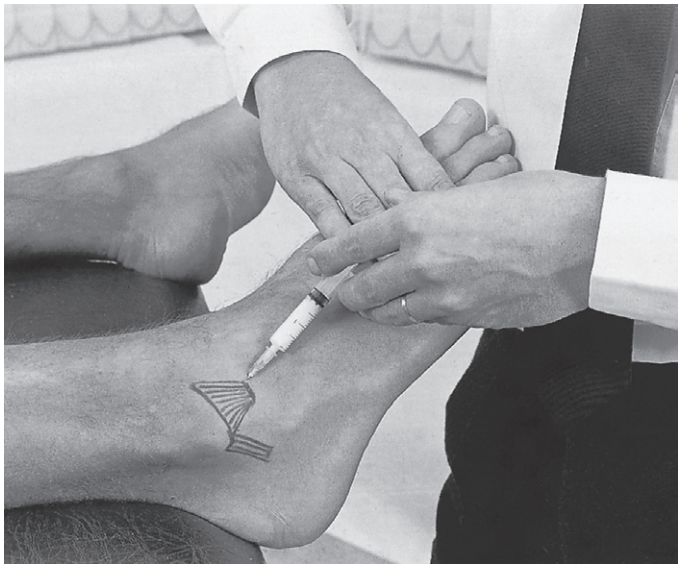


**Fig 58.21** • Infiltration of the fibular extent of the talofibular ligament.

### *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.

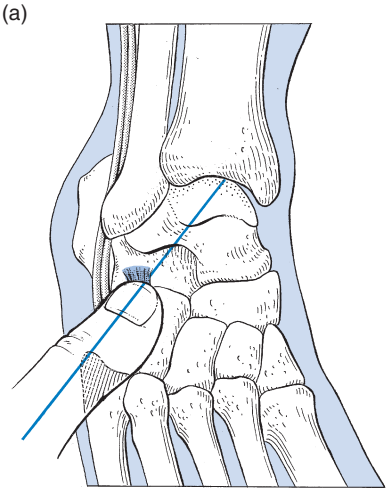
More thorough friction must be applied in the subacute stage.

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)

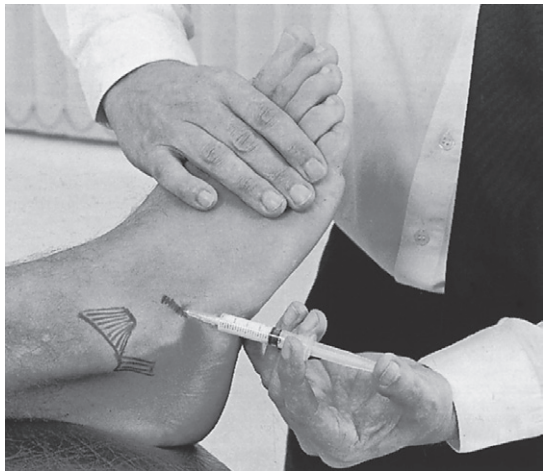


**Fig 58.22** • Calcaneocuboid ligament: (a) palpation technique; (b) infiltration.

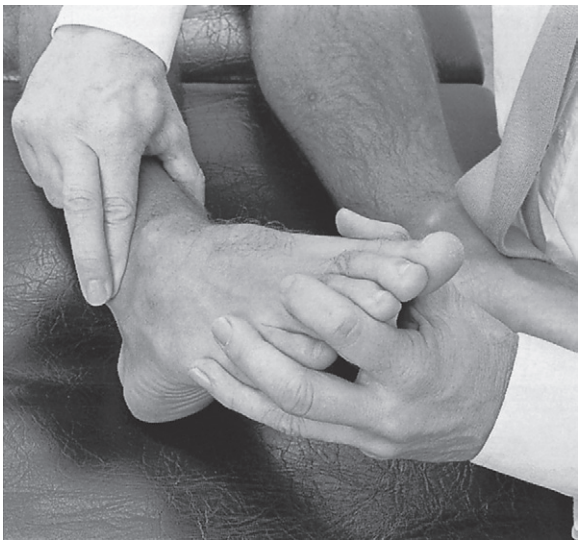


**Fig 58.23** • Deep friction of the fibular extent of the talofibular ligament.

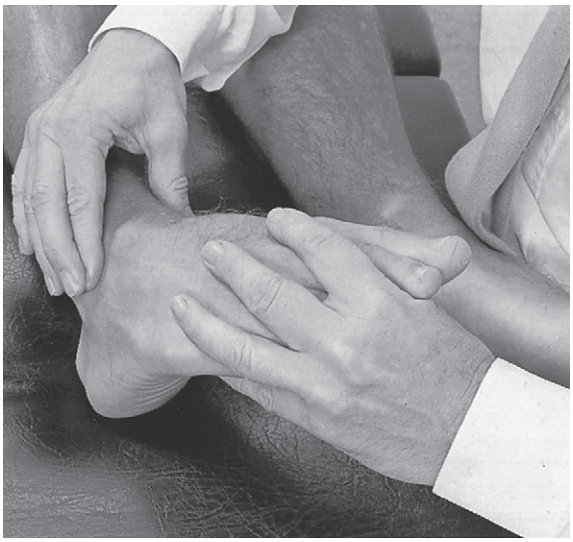


**Fig 58.25** • Friction to the fibular extent of the calcaneofibular ligament.

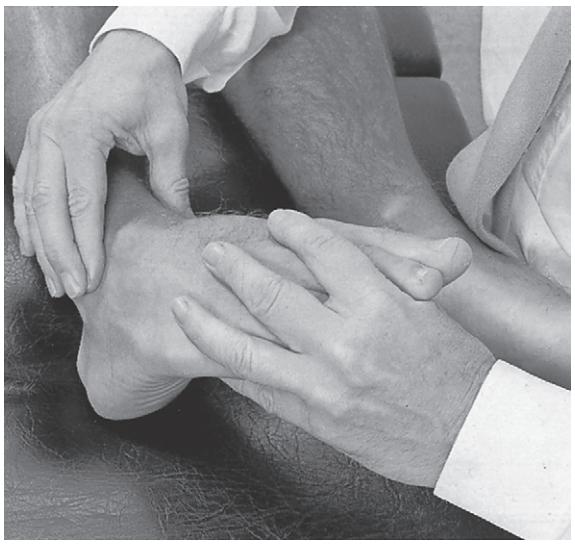


**Fig 58.24** • Friction to the talar extent of the talofibular ligament.

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,

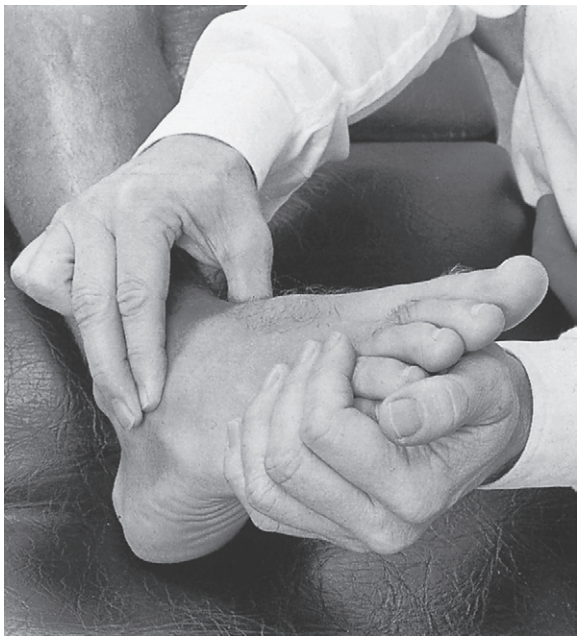


**Fig 58.26** • Friction to the calcaneocuboid ligament.

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

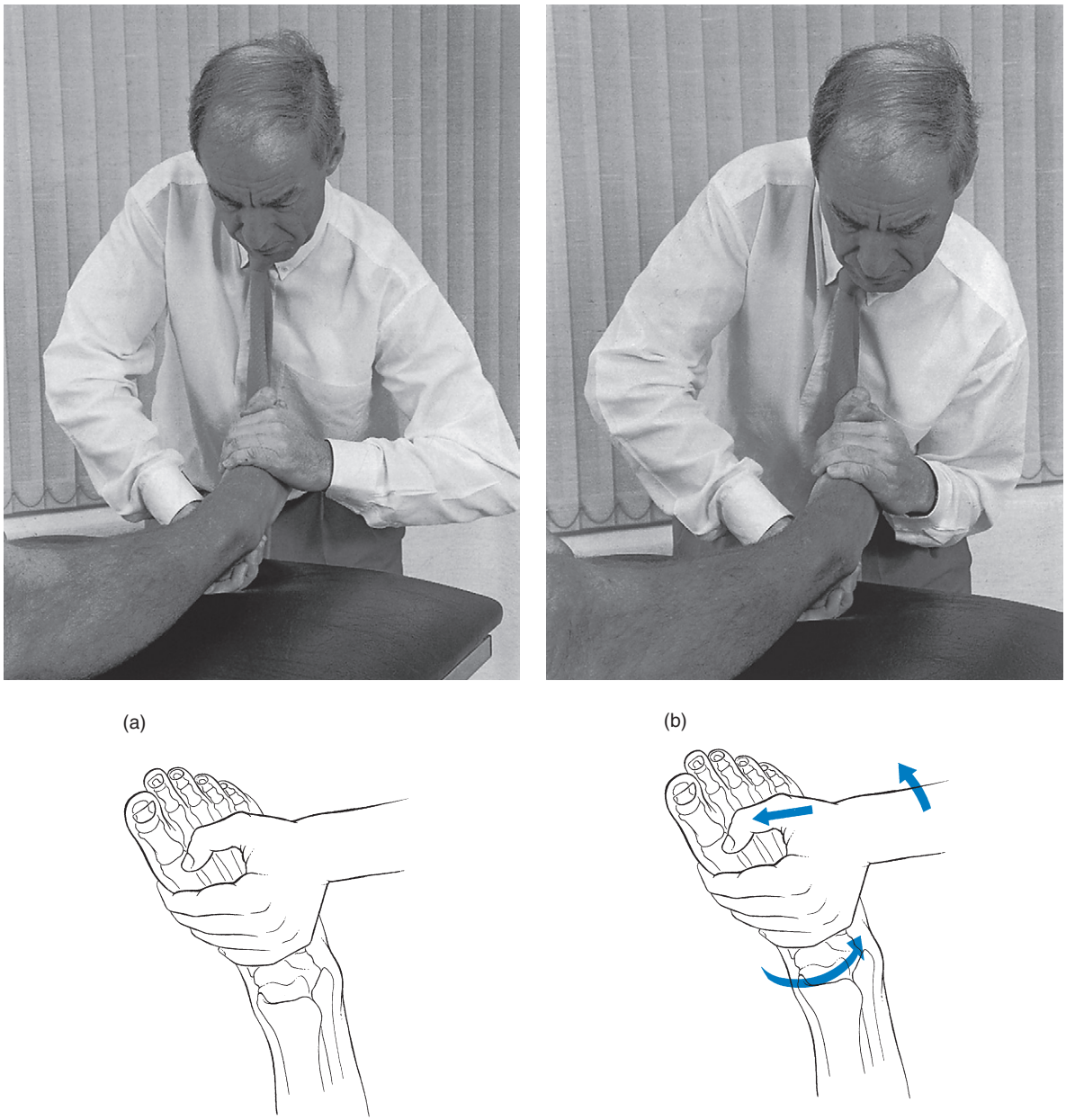



**Fig 58.27** • Manipulation of adherent lateral ligaments: start of manipulation (a) and end of manipulation (b).

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

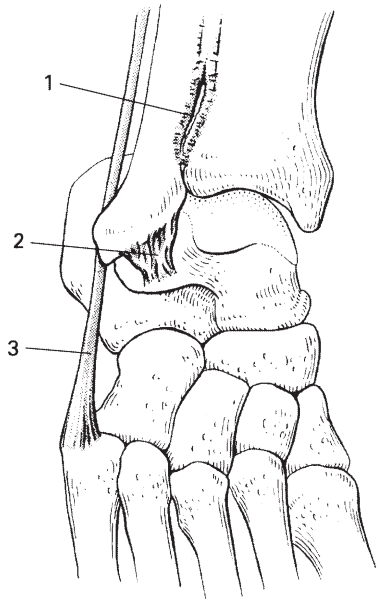



**Fig 58.28** • Causes of instability at the ankle: 1, lengthened or ruptured distal tibiofibular ligament; 2, lengthened or ruptured anterior talofibular ligament; 3, proprioceptive defects affecting the peroneal muscles.

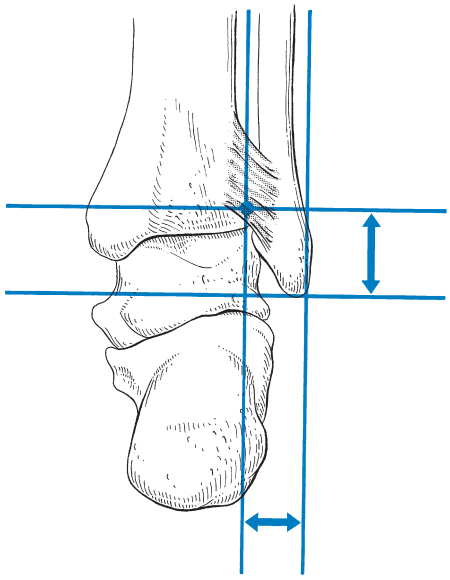


**Fig 58.29** • Landmarks and measurements which can be drawn on a plain frontal radiograph taken preparatory to sclerosing injections for an unstable mortice.

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]

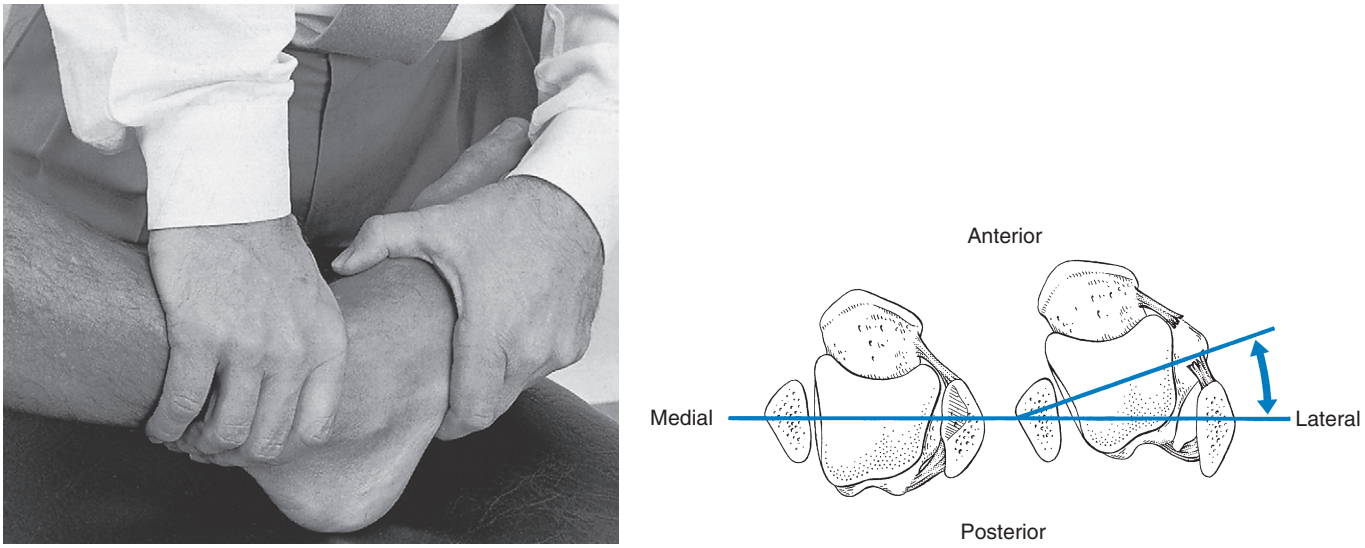



**Fig 58.30** • Anterior drawer test for ligamentous insufficiency of the anterior talofibular ligament.

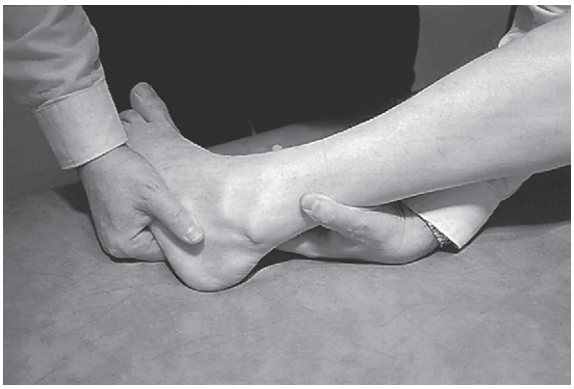


**Fig 58.31** • Positive anterior drawer test demonstrating ligamentous insufficiency of the anterior talofibular ligament.

## 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<br>Fear of the ankle 'giving way' | Foot is adequate for ordinary purposes but tends to swell and ache after vigorous or prolonged use<br>No sudden aggravation or twinges |
| **Examination (between attacks)** | **1.** Anterior talofibular ligament:<br>Normal routine examination<br>Sometimes excessive range of inversion<br>End-feel normal or 'empty'<br>Positive anterior drawer test<br>**2.** Unstable mortice:<br>Reproduction of click and pain during strong varus movement at ankle<br>Radiography shows increased distance between the two malleoli during forced varus<br>**3.** Defective propriosensory reflex:<br>Normal clinical examination<br>L5 palsy or upper motor neurone lesion<br>Weak peroneal muscles | **1.** Adhesions of talofibular and calcaneocuboid ligaments:<br>Pain at outer side of ankle and foot during full inversion and plantiflexion<br>Slight limitation<br>End-feel more tense than on the opposite side<br>Resisted movements are negative<br>**2.** Chronic tendinitis of peroneal muscles:<br>Passive inversion during plantiflexion hurts at outer side of ankle and foot<br>End-feel normal<br>Pain during resisted eversion |
| **Treatment** | **1 & 3.** Wobble board training, floated heel, taping or surgery<br>**2.** Sclerosing injections into the distal tibiofibular ligament over 3 consecutive weeks, or surgery<br>**3.** Floated heel | **1.** Adhesions rupture by manipulating the ankle and foot after vigorous deep transverse friction at the exact location<br>**2.** Deep transverse friction 3 times a week, for 2–4 weeks<br>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.

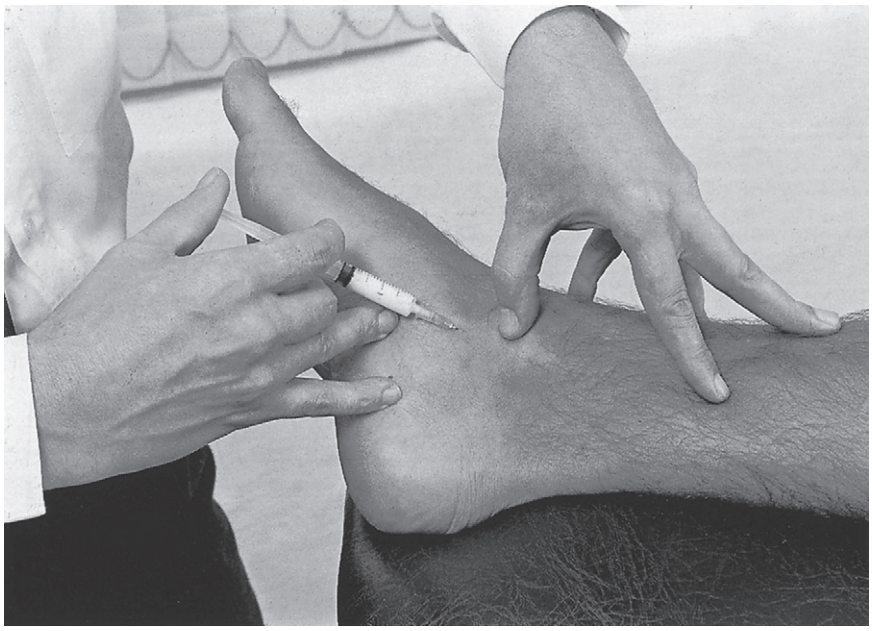


**Fig 58.32** • Infiltration of the deltoid ligament.

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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