

## Manual of diagnostic ultrasound

vol. 2

During the last decades, use of ultrasonography became increasingly common in medical practice and hospitals around the world, and a large number of scientific publications reported the benefit and even the superiority of ultrasonography over commonly used X-ray techniques, resulting in significant changes in diagnostic imaging procedures.

With increasing use of ultrasonography in medical settings, the need for education and training became essential. WHO took up this challenge and in 1995 published its first training manual in ultrasonography. Soon, however, rapid developments and improvements in equipment and indications for the extension of medical ultrasonography into therapy indicated the need for a totally new ultrasonography manual.

The manual (consisting of two volumes) has been written by an international group of experts of the World Federation for Ultrasound in Medicine and Biology (WFUMB), well-known for their publications regarding the clinical use of ultrasound and with substantial experience in the teaching of ultrasonography in both developed and developing countries. The contributors (more than fifty for the two volumes) belong to five different continents, to guarantee that manual content represents all clinical, cultural and epidemiological contexts

This new publication, which covers modern diagnostic and therapeutic ultrasonography extensively, will certainly benefit and inspire medical professionals in improving 'health for all' in both developed and emerging countries.

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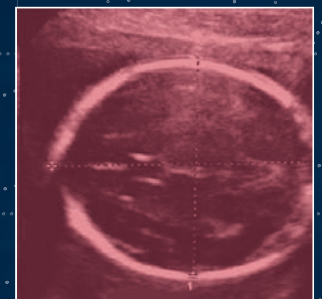
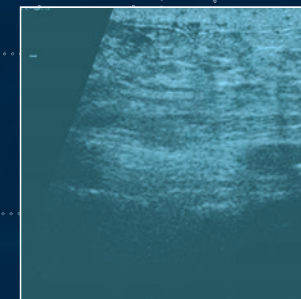
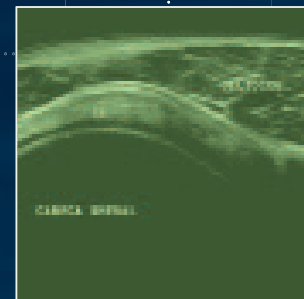
Vol. 2

Manual of diagnostic ultrasound

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volume 2

Second edition



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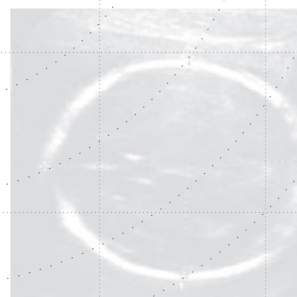
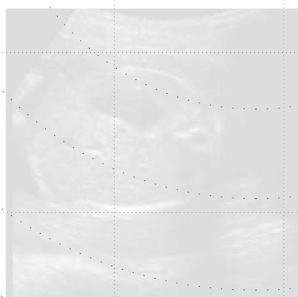
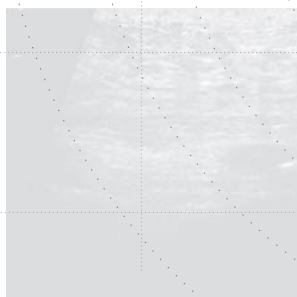


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## Chapter 6

# Musculoskeletal system

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# Musculoskeletal system

## Tendons

Use of ultrasound for studying diseases of the musculoskeletal system is increasing because of improvements in the equipment, which permit visualization of small structures that were previously inaccessible. This chapter focuses on the main diseases involving myotendinous and ligamental structures of the upper and lower members.

Tendons are composed of collagen (30%), proteoglycans (68%) and elastin (2%). Collagen fibres, 85% of which consist of type I collagen, form the primary fascicles. These give rise to secondary fascicles, which are separated by a fine, loose net of connective tissue known as the endotendon, which brings together the small nerve endings, lymphatic vessels, venules and arterioles. The endotendon is connected to the tissue surrounding the tendon, known as the epitenon. Vascularization occurs through the musculo-tendinous junction, the periphery of the tendon and the enthesis (junction with the bone). The tendon is hypervascularized during its formation but is less vascularized when mature. This basic architecture is common to all tendons.

The external covering of tendons can be vascular or avascular. Vascular tendons are covered by a single layer of synovia and loose areolar tissue, known as the paratenon, which contains the vessels that perfuse the tendons. The paratenon, together with the epitenon, gives rise to the peritendon. Avascular tendons are surrounded by a synovial sheath composed of visceral and parietal leaflets connected by a mesotendon, through which vascular structures penetrate via the vincula. These tendons receive nutrients by diffusion of synovial fluid and through the vincula. Most of the tendons of the musculoskeletal system are vascular. Only the long head tendon of the brachial biceps and the flexor and extensor tendons located in the wrists, ankles, hands and feet are avascular.

Tendons are highly resistant, and healthy tendons do not rupture. In normal tendons, lesions occur either at sites of biomechanical differences between tissues (the myotendinous junction or adjacent to bone) or in hypovascularized regions, which are considered critical, such as the third distal of the calcaneus tendon and close to the insertions of the supraspinal and brachial biceps tendons. Mechanical and vascular factors are implicated in tendinopathies, which are expressed histopathologically by the presence of tendinosis, corresponding to mucoid degeneration of the tendon, often accompanied by neovascularization, necrosis and dystrophic calcifications.

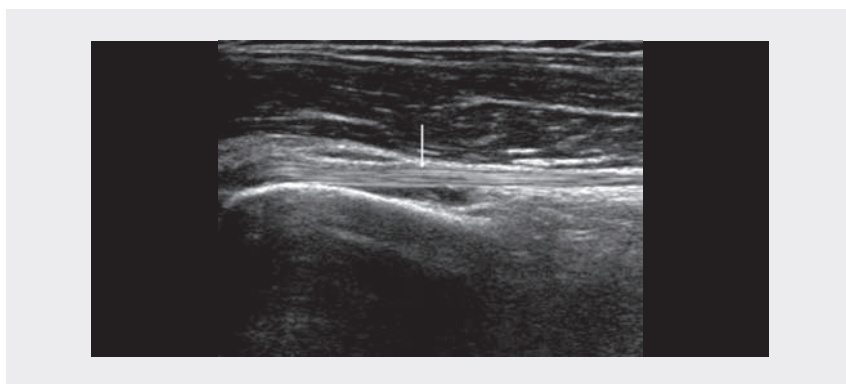
Repetitive stress on a tendon causes two types of degenerative alteration. In eccentric contraction, tendinous fibres are stretched to 5–8% more than their length, and small ruptures start to appear inside the tendon. With increased temperature, relaxation transforms 5–10% of the generated energy into heat, raising the temperature inside the tendon up to 45 °C.

## Ultrasound findings

### Normal tendon and tendinopathy

The normal tendon tends to present a fibrillar, echo-rich aspect on ultrasound (Fig. 6.1). The factors that determine the echotexture include insertion of muscle fibres inside the tendon, the tendinous architecture, entheses, the type of equipment and the examiner's experience.

Fig. 6.1. Fibrillar, homogeneous, echo-rich aspect of the tendon of the long head of the brachial biceps (arrow): longitudinal scan



The insertion of muscle fibres inside the tendon can be illustrated by the rotator cuff of the shoulder (Fig. 6.2).

In certain musculotendinous units, more than one muscle venter contributes to the structure of the tendon. The supraspinal tendon (supraspinatus) is composed of five layers, one represented by entwinement of its fibres with those of the infraspinatus tendon (Fig. 6.3).

At the entheses, the tendon changes its histology at the point of insertion into the bone and presents fibrocartilage, which is echo-poor on ultrasound (Fig. 6.4).

Fig. 6.2. (a), (b) Supraspinal muscle fascicles represented by echo-poor bands (arrows) attached inside the tendon, simulating a fracture

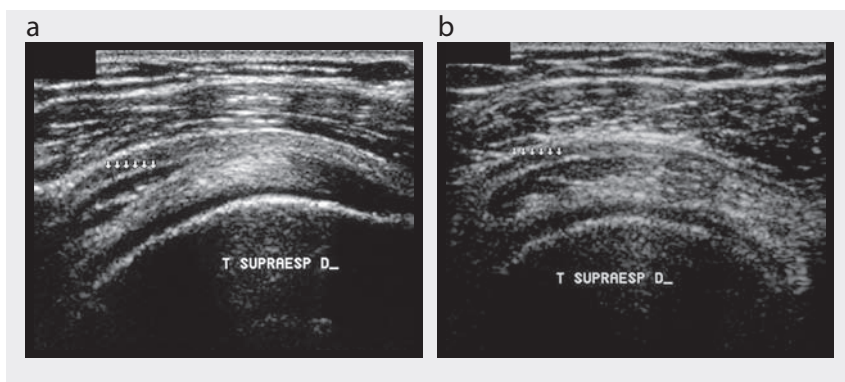


Fig. 6.3. Normal heterogeneity of the supraspinal tendon due to different spatial orientation of the layers of the tendon, generating a three-band aspect (stars)



Fig. 6.4. Fibrocartilaginous insertion of supraspinal tendon with an echo-poor aspect (calipers) adjacent to the osseous cortex

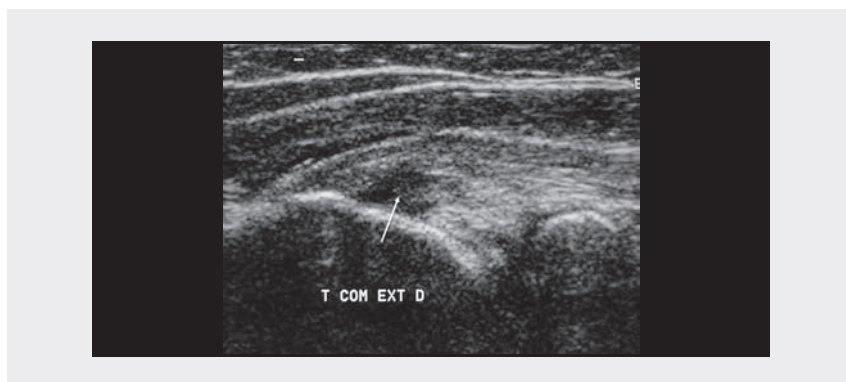


Equipment with transverse ultrasound beams significantly reduces the anisotropy generated by oblique arrival of the beam on the tendon surface, which forms echo-poor areas in the interior.

Alterations in tendinopathies start with a reduction in the echogenicity of the tendon (Fig. 6.5), sometimes accompanied by an increase in tendon thickness, secondary to the entry of water molecules into the triple-helix structure of the collagen between hydrogen bridges, which break up during tendinous degeneration. In chronic cases, calcifications can be seen as small, echo-rich foci, which is the main differential diagnoses from fibrosis and small partial ruptures.

During tendon degeneration, the process may remain stable or evolve to rupture, which can be partial or involve the entire thickness (transfixing).

Fig. 6.5. Tendinopathy of the forearm extensors, seen as a poorly defined, echo-poor area (arrow) inside the tendon

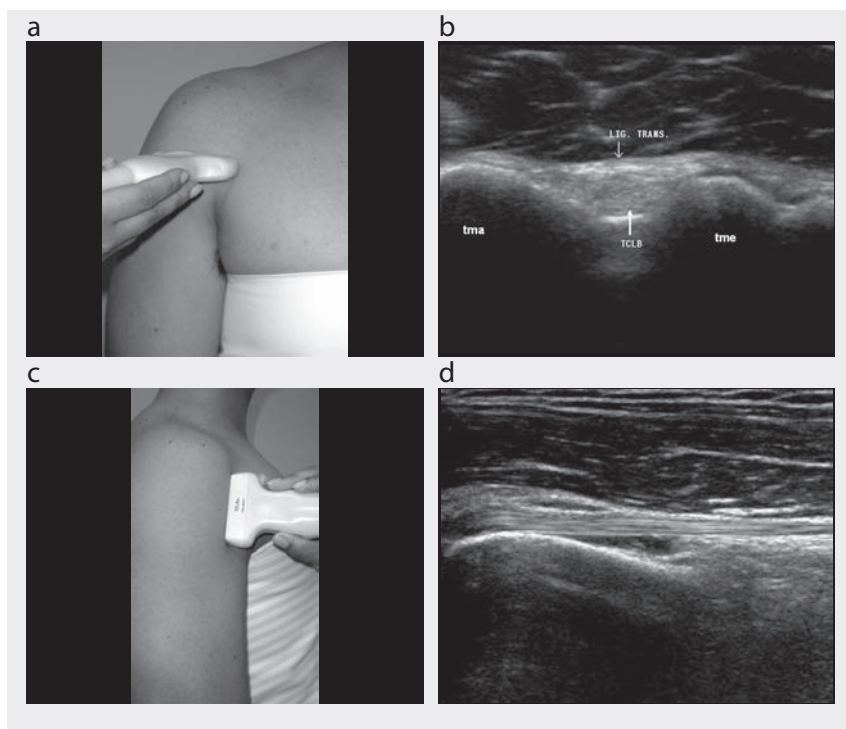


## Upper limbs

### Shoulder

About 60% of alterations of the shoulder are due to lesions of the rotator cuff, which is the deepest muscle group of the shoulder joint, forming a single functional unit involving the humerus head, which contributes to the stability of the glenohumeral joint and the movements of the upper member. It is composed of the supraspinatus (arm abductor), subscapularis (internal rotator), infraspinatus and teres minor (external rotators) muscles. The tendons join 15 mm proximal to the insertions at the larger and smaller tubercles of the humerus and cannot be separated by dissection. The thickness of the tendons varies from 5 mm to 12 mm. The difference from the contralateral side considered to be normal is 2 mm, and variations above this limit should be considered pathological. The function of the synovial bursae in the periscapular area is to reduce the attrition between soft tissue and bone structures. The largest is the subacromial-subdeltoid bursa, located below the acromion and the deltoid muscle venter, starting at the coracoid process and finishing some 3 cm from the larger tubercle of the humerus.

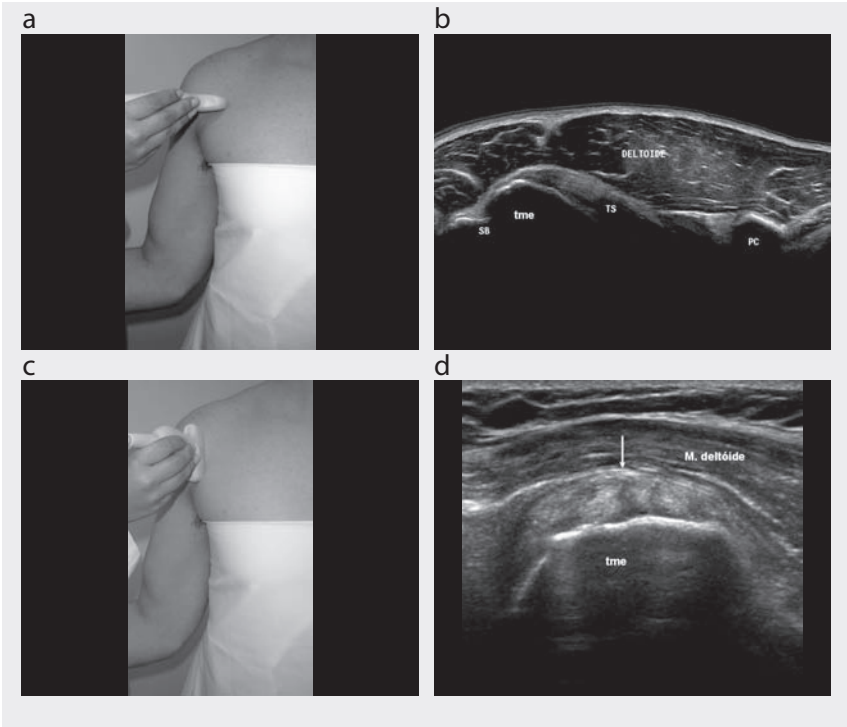
Fig. 6.6. Extra-articular section of the tendon of the long head of the brachial biceps (TCLB). (a), (c) Examination technique. Transversal (b) and longitudinal (d) scans. tme, smallest humerus tubercle; tma, largest humerus tubercle; lig trans, transverse ligament



The patient must cooperate during an ultrasound examination of the tendons of the rotator cuff, as external and internal rotation manoeuvres are necessary (Fig. 6.6, Fig. 6.7, Fig. 6.8, Fig. 6.9, Fig. 6.10). Both the infraspinatus and the teres minor tendon can be evaluated either by placing the hand on the contralateral shoulder or adopting the same position as for examination of the supraspinatus tendon. The pathological processes involving the rotator cuff usually affect the supraspinatus tendon, due to normal degeneration of the tendons, trauma, inflammatory arthritis or tendinosis due to excessive traction or impact syndrome.

Impact syndrome is the commonest cause of pain in the shoulder. It is defined as a group of signs and symptoms characterized by pain and progressive disabling caused by mechanical attrition of the elements of the coracoacromial arch with the structures of the subacromial soft tissues. Abduction (between 70° and 130°) associated with external rotation or anterior elevation with internal rotation of the arm are the commonest movements that cause secondary pain after subacromial impact.

Fig. 6.7. Subscapular tendon (arrow). (a), (c) Examination technique, with external rotation of the arm for better exposure of the tendon. Transversal (b) and longitudinal (d) scans. TS, subscapular tendon; PC, coracoid process; SB, bicipital sulcus; arrow, tendon of the long head of the brachial biceps; tme, smallest humerus tubercle



**Fig. 6.8.** Supraspinal tendon. (a), (c) Examination technique, with internal rotation of the arm, extension and adduction for better exposure of the tendon. Longitudinal (b) and transversal (d) scans. TS, supraspinal tendon; PC, coracoid process; bolsa, bursa subacromial sac-subdeltoid; GPS, peribursal fat; TI, infraspinal tendon; ACR, acromion; cabeca umeral, humeral head

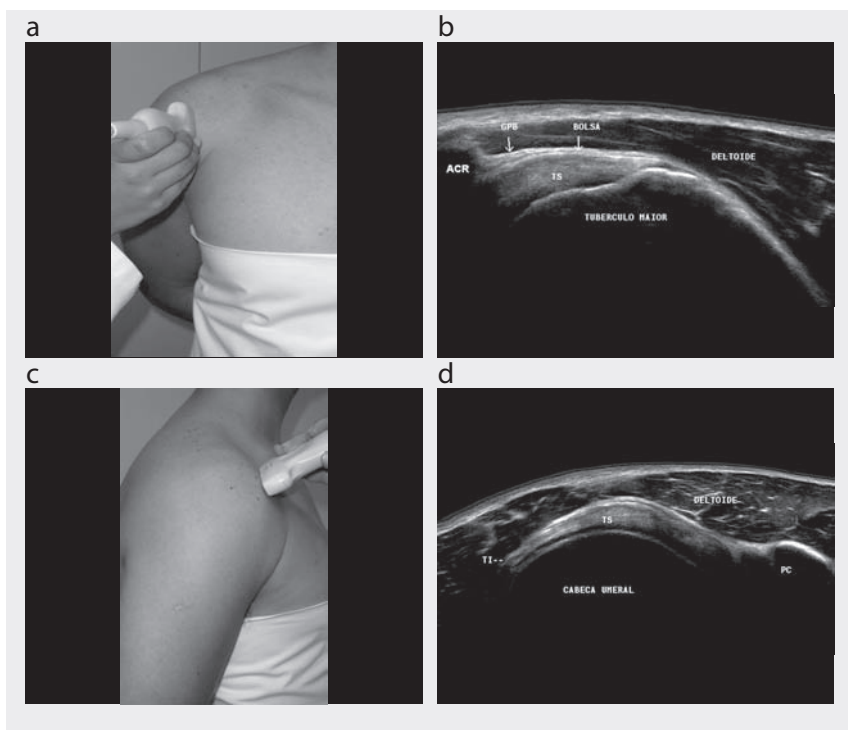




Fig. 6.9. Infraspinatus tendon (arrow). (a), (b) Examination technique. (c) Ultrasonographic examination. IF, infraspinatus muscle; glen, glenoid; t infraesp, infraspinatus tendon

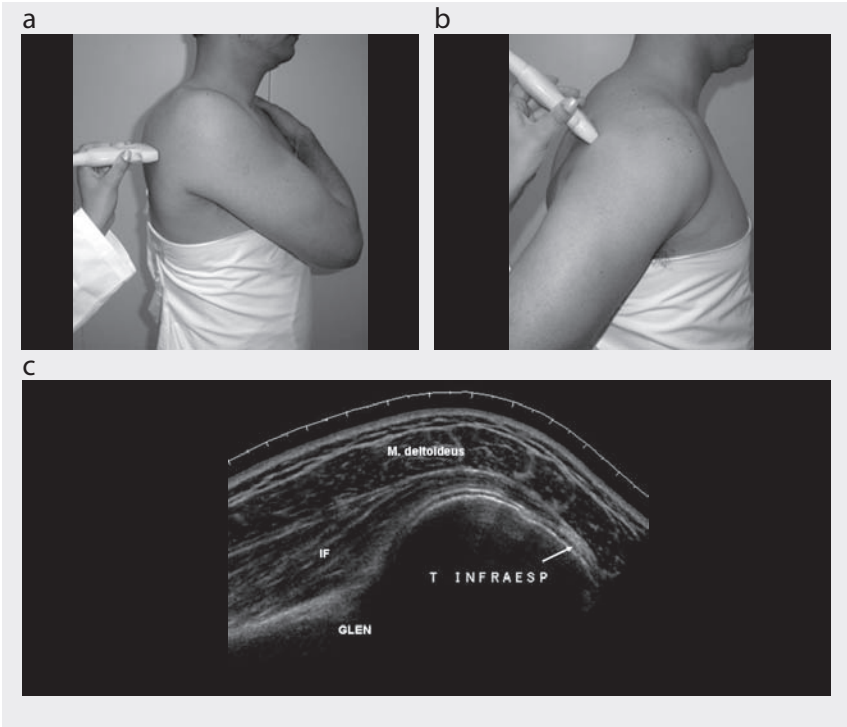
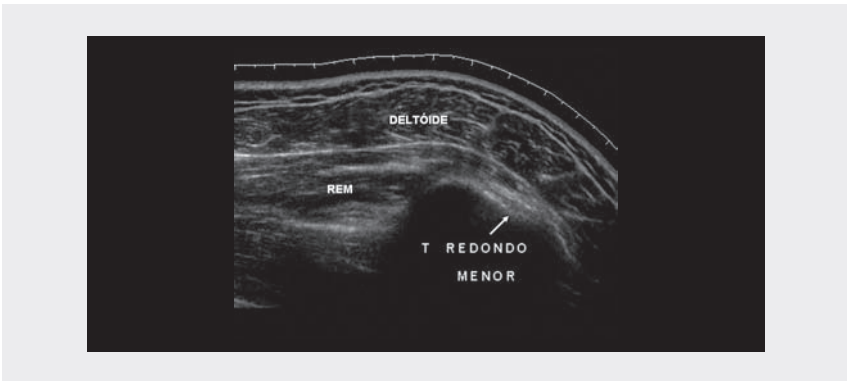


Fig. 6.10. Tendon (arrow) of the teres minor muscle (REM). Ultrasonography, showing more abrupt sharpening and less echogenicity than the infraspinatus tendon due to the presence of muscle fascicles among the tendon fibres



### Partial ruptures

Partial ruptures may have two distinct ultrasonographic patterns (Fig. 6.11). Echo-poor or echo-free lesions due to discontinuity of the fibres initially present linearly with delaminating of the tendon, especially if the trauma mechanism is secondary to eccentric contraction of the rotator cuff tendons. More commonly, a mixed lesion is seen, with an echo-rich centre surrounded by an echo-poor halo indicating perilesional fluid. The echo-rich centre is due to retracted tendon fibres or to a new acoustic interface generated by the rupture. Although these patterns predominate, they are not the only ones.

Some lesions are characterized by linear, echo-rich images along the tendon fibres. The continuity of this echo-poor image can be identified with high-frequency transducers (Fig. 6.12).

Fig. 6.11. Commonest ultrasonographic aspects of partial lesions of the rotator cuff.  
(a) Echo-free lesion (arrows) delaminating the tendon. (b) Mixed-type fracture, with echo-rich and echo-free areas inside (arrow)

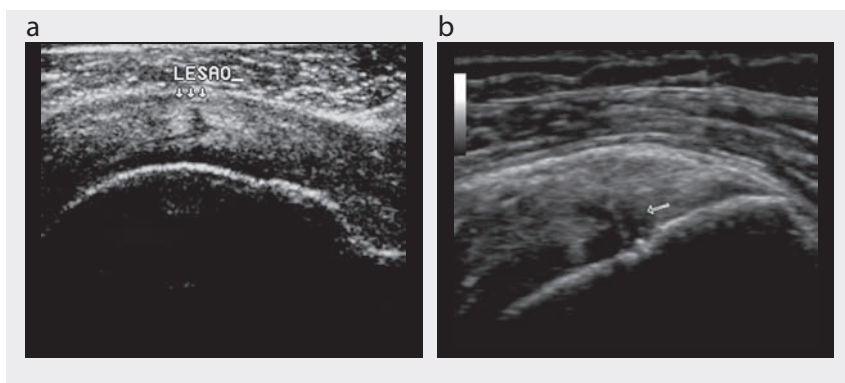
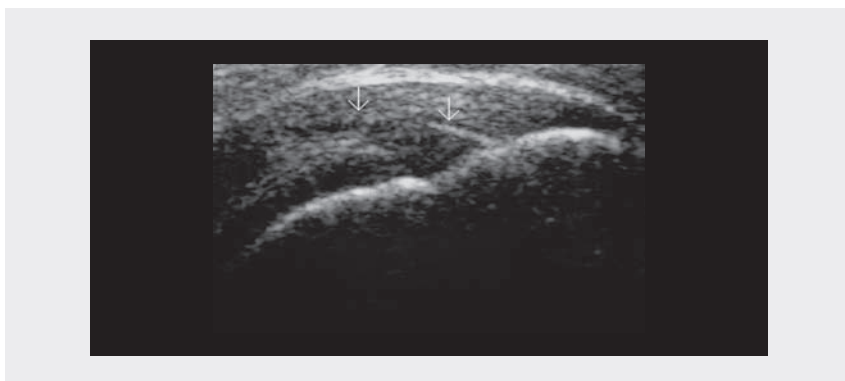


Fig. 6.12. Unusual partial rupture. Ultrasonograph showing that the echo-poor linear image is continuous with the echo-rich area (arrows)



### Complete rupture

Complete, transfixing ruptures of the entire thickness of the tendon are diagnosed from direct and indirect signs.

The **direct (primary) signs** can be divided into two large groups: alteration of the tendinous outline, including the absence and focal tapering of the tendon, and alterations of the echo texture, comprising heterogeneous echogenicity and an echo-free intratendinous focus or split.

When the tendon is not visible, the deltoid muscle touches the head of the humerus (bald humeral head sign), and a small echogenic strip can be seen between the two structures, indicating either thickening of the synovial bursa or repairing tissue (fibrosis) on the tendon. In the absence of the supraspinatus tendon, the deltoid muscle can act without an antagonist, resulting in subluxation of the humeral head with reduction of the subacromial space (Fig. 6.13).

Fig. 6.13. Bald humeral head sign. Unidentified supraspinal tendon (arrow) with reduction of the subacromial space. ACR, acromion



In the absence or focal tapering of the tendon, the usual convexity of the tendon is altered. In more severe ruptures, herniations of the synovial bursa and of the deltoid muscle itself represent the defect (Fig. 6.14). In less severe ruptures, tapering may be seen, with rectification of the bursal surface, and it is difficult to determine whether it is a complete rupture (transfixing) or a partial lesion. In these situations, it is useful to check the percentage of tapering, which corresponds to the depth of the concavity formed by the outline of the subacromial-subdeltoid bursa: if it is greater than 50%, it is a complete lesion; if it is less than 50%, it is a partial lesion.

Discontinuity of the fibres without alteration of the tendon outline indicates a connection between the glenohumeral joint and the subacromial-subdeltoid bursa.

Heterogeneous tendon echogenicity is the source of most faulty diagnoses, as an increase may represent a small partial or complete rupture, calcification or fibrosis (Fig. 6.15). Sometimes, the echogenicity can be increased by associated findings, such as a posterior acoustic shadow in a calcification or the linear form of the larger

Fig. 6.14. Absence of focus on the anterior portion of the supraspinal tendon (T) in both longitudinal (a) and transverse (b) views, accompanied by thickening of the subacromial-subdeltoid bursa (arrow). TSE, remnant of supraspinal tendon; TMA, largest humerus tubercle; TLCB, long head of brachial biceps tendon; ART AC, acromion-clavicle joint

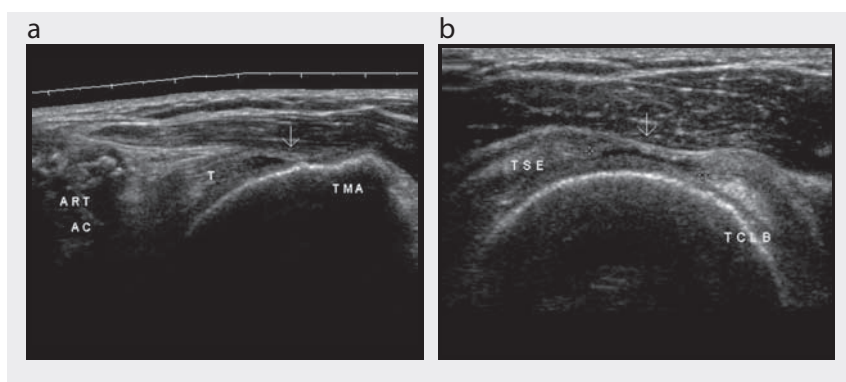
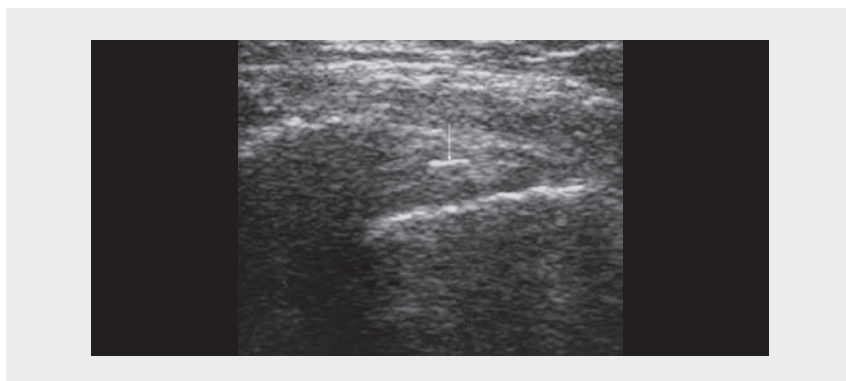


Fig. 6.15. Change in tendon echogenicity, with a small, linear, echo-rich, intratendinous image (arrow) with no posterior acoustic shadow and an unspecified aspect



tubercle of the humerus in ruptures. Calcifications sometimes have a slightly echo-rich aspect, with no acoustic shadow, surrounded by an artefactual linear, echo-poor image, simulating rupture in transition with the tendon. In such cases, a simple radiographic examination will confirm the presence of calcification.

In acute lesions, echogenic blood may fill the area of the rupture, impeding any change to the tendon and thus a diagnosis. As the echo texture of the tendon is heterogeneous, the transducer should be compressed on the tendon. In ruptures associated with tendinopathy, the usual convexity of the tendon may be lost (Fig. 6.16). Another manoeuvre that can be used to remove doubt is returning the arm to the neutral position, causing relaxation of the subacromial-subdeltoid bursa and mobilization of the fluid inside the lesion.

Fig. 6.16. Supraspinal tendon in longitudinal scan, before (a) and after (b) compression. (a) Heterogeneous tendon with a normal outline. (b) Rectification and tapering of the tendon close to its insertion (arrow), showing the presence of rupture

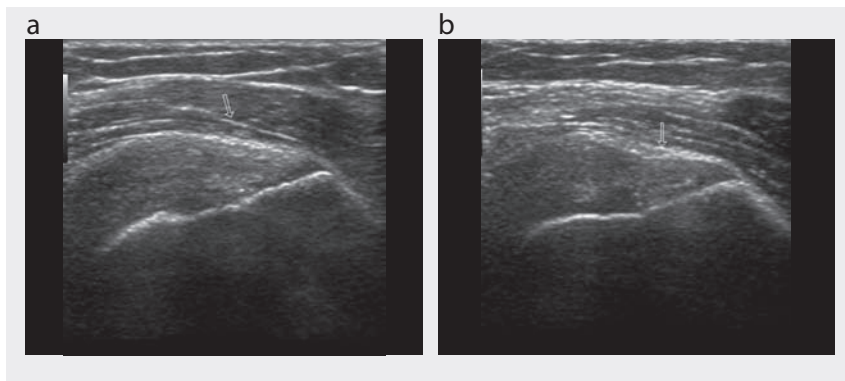
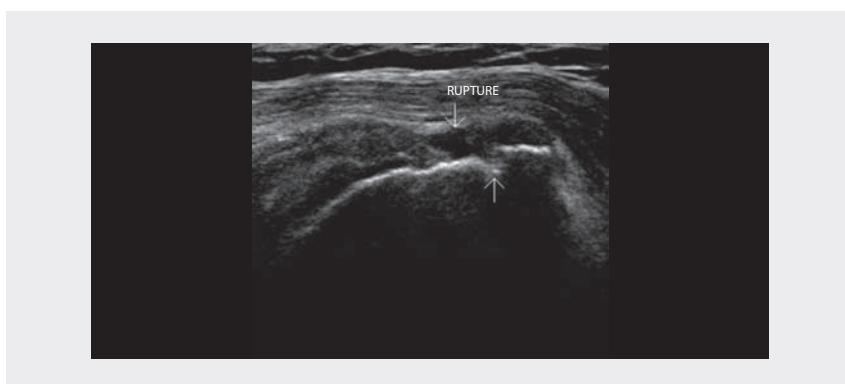


Fig. 6.17. Irregular outline of the largest tubercle of the humerus (arrow), associated with complete rupture (transfixing) of the supraspinatus tendon (rotura, arrow)



The **indirect (secondary) signs** include an irregular contour of the largest tubercle of the humerus. Most partial or complete ruptures of the tendon situated up to 1 cm from the insertion present some alteration on the bony surface of the largest tubercle. About 70% of partial lesions are accompanied by irregularity of the cortical bones, from small defects to bone fragments and exostosis. It may be caused by a posterosuperior impact or be secondary to traction of fixed tendinous fibres on the surface of the largest tubercle (Fig. 6.17).

Liquid is present in the acromion-clavicular joint (Geyser sign) only when the subacromial-subdeltoid bursa is connected to the acromion-clavicular joint. A periarticular cyst is formed, secondary to the passage of the glenohumeral to the acromion-clavicular joint through rupture of the rotator cuff.

Liquid in the glenohumeral joint is identified either from distension of synovial recesses of the joint or from the amount of fluid accumulated in the synovial sheath

of the long head tendon of the brachial biceps. In general, the synovial recesses are posterior, easy to access and located anterior to the tendinous muscle of the infraspinatus. Liquid accumulation occurs when the distance between the glenoid posterior labrum and the infraspinatus tendon is  $> 2$  mm. The synovial recesses may also be axillary, located below the inferior margin of the tendinous muscle of the teres minor (Fig. 6.18). External rotation during dynamic testing increases the sensitivity of the examination. They may also be approached through the axillary cavum; in this case, the diagnostic criteria are that the distance between the bone surface and the joint capsule must be  $> 3.5$  mm and the difference between the two sides must be  $> 1$  mm.

Liquid in the subacromial-subdeltoid bursa is suspected when the bursa presents a thickness  $> 1.5$ – $2$  mm. Although this phenomenon may also be seen in asymptomatic people, ultrasonographic detection of fluid in the bursa and the glenohumeral joint is highly specific for predicting rupture of the rotator cuff (Fig. 6.19).

Fig. 6.18. (a), (b) Glenohumeral articular haemorrhage (stars) below the inferior margin of the teres minor muscle (MRM) and anterior to the tendinous muscle transition of the infraspinatus (MIF). glen, glenoid

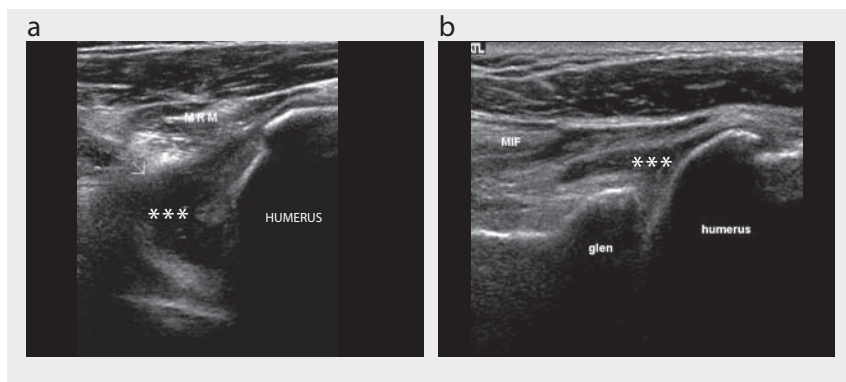
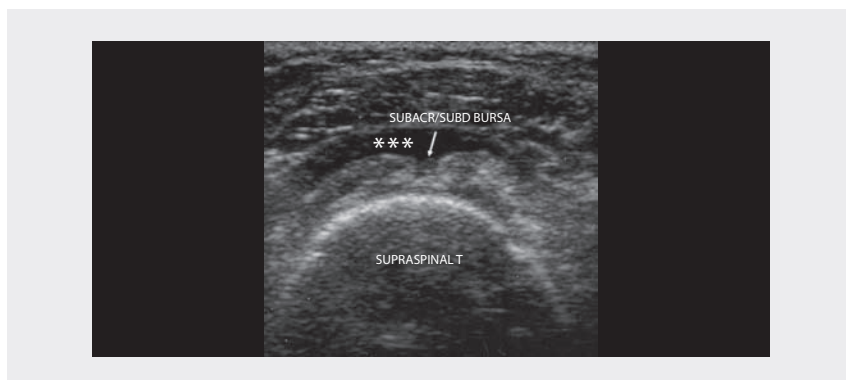


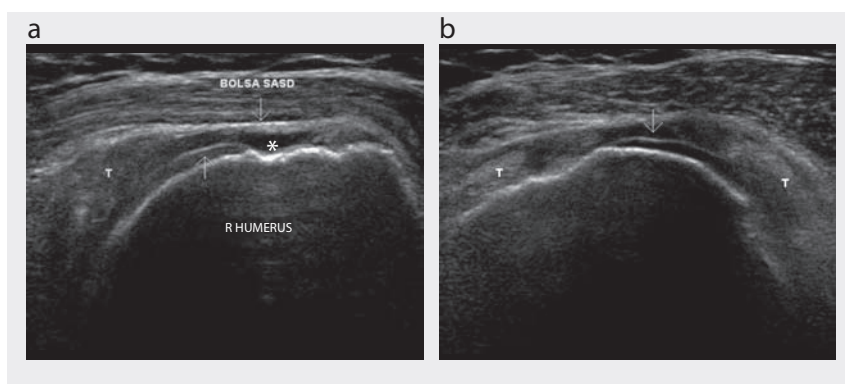
Fig. 6.19. Partial lesion of the supraspinatus tendon (arrow), containing fluid and distending the subacromial-subdeltoid bursa (stars)



The cartilage interface sign, also called the naked tuberosity sign, corresponds to linear hyperechogenicity below the lesion, representing the external outline of the hyaline cartilage that covers the humerus head. It is generated by posterior acoustic reinforcement due to the echoic rupture (Fig. 6.20).

Appropriate treatment should be based on an understanding of the type and dimensions of the tendinous rupture, the appearance of the glenohumeral joint on simple X-ray, the degree of muscle atrophy of the rotator cuff and the case history.

Fig. 6.20. Cartilage interface sign (arrow). (a) Longitudinal and (b) transverse scans of the supraspinal tendon (T), with two other signs: focal absence of the tendon (star) and irregular outline of larger tubercle of the humerus (umero); parietal thickening of the subacromial-subdeltoid bursa (bolsa SASD)

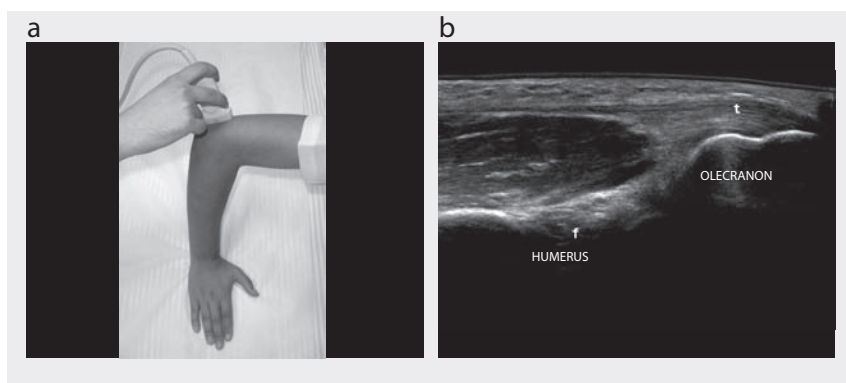


## Elbow

The musculotendinous structures of the elbow are made up of four groups of muscles: posterior, anterior, lateral and medial.

The largest posterior muscle is the brachial triceps, formed by three heads that merge to form a single tendon inserted into the upper margin of the olecranon and the antebrachial fascia (Fig. 6.21). Conjunction of small enthesophytes is common, but rupture is rare. In the periolecranon area, three synovial bursae can be identified, one subcutaneous, one intratendinous and one between the elbow joint capsule and the brachial triceps tendon.

Fig. 6.21. Brachial triceps tendon (t). (a) Examination technique and (b) longitudinal scan. f, olecranon fossa



The anterior group comprises the brachial and brachial biceps muscles. The two heads of the biceps join to form a tendon 6–7 cm long covered by a paratenon, with insertion into the posterior face of the radius tuberosity (Fig. 6.22). A hypovascularized area is seen close to the insertion, and the presence of tendinopathy is common. Two synovial bursae are found in the area: the bicipitoradial, between the radius and the brachial biceps tendon, close to its insertion, and the interosseous, between the ulna and the brachial biceps tendon.

The lateral group comprises the common extensor tendon, originating in the lateral epicondyle of the humerus, formed by the carpi radialis brevis extensor, finger extensors, digiti minimi extensor and carpi ulnaris extensor tendons (Fig. 6.23). This group also includes the brachioradial and supinator muscles and tendons.

The medial group is composed of the pronator teres muscle and the common flexor tendon, formed by the musculotendinous units of the palmaris longus, digitorum superficialis flexor, carpi radialis flexor and carpi ulnaris flexor, fixed in the medial epicondyle (Fig. 6.24).

Lateral and medial epicondylitis are overuse syndromes characterized by pain and increased sensitivity of the epicondyles, generally related to tendinopathy. The common tendon of the forearm extensors is involved in 80% of cases, initially affecting the deep portion, corresponding to the carpi radialis brevis extensor (Fig. 6.5). In medial epicondylitis, ulnar neuropathy is associated in 60% of cases.



Fig. 6.22. Brachial biceps tendon (t, arrow). (a) Examination technique; transverse scan in the axial plane, from the forearm proximal to insertion in the radius tuberosity. (b) Tendon positioned along the brachial artery, a, gradually going deeper (c), posterior to the bifurcation of the brachial artery, a. (d) Ulnar artery beside the tendon. Longitudinal scan. (e) Examination technique. (f) Ultrasound scan. L, lateral area; M, medial area; tuber. radio, tuberosity

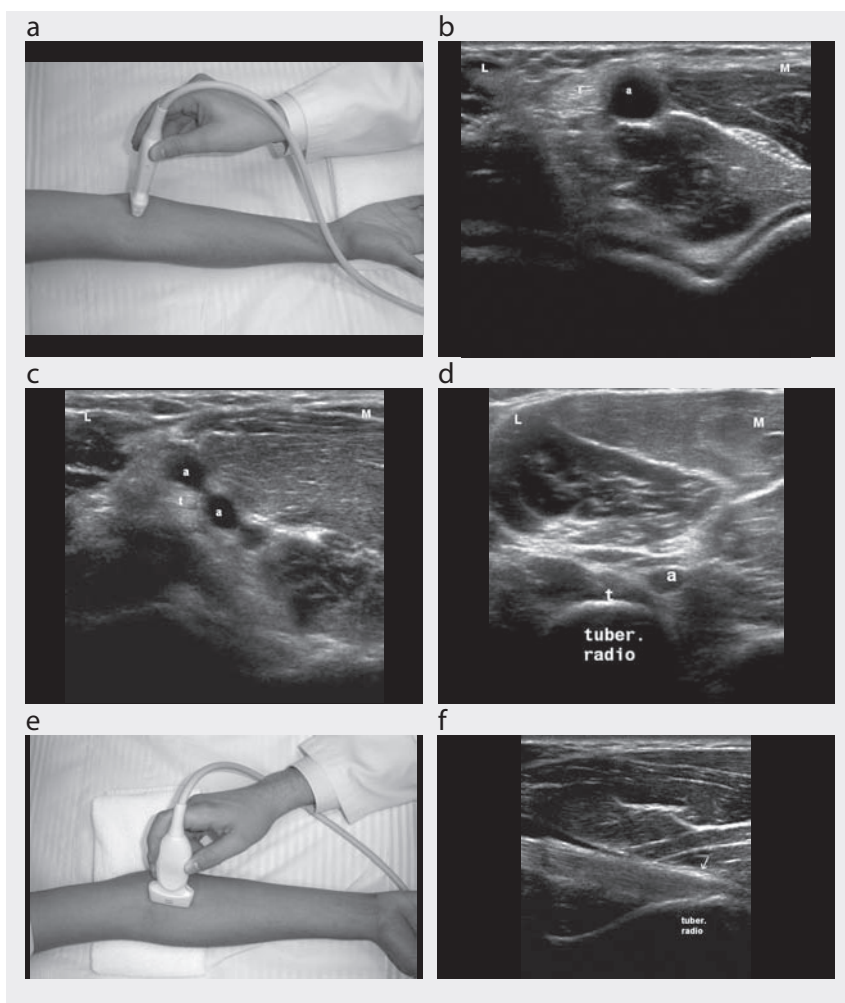


Fig. 6.23. Common tendon of the forearm extensors. (a) Examination technique. (b) Ultrasound scan; common tendon of the forearm extensors (arrow). br, brachioradial muscle; el, lateral epicondyle; cr, radius head

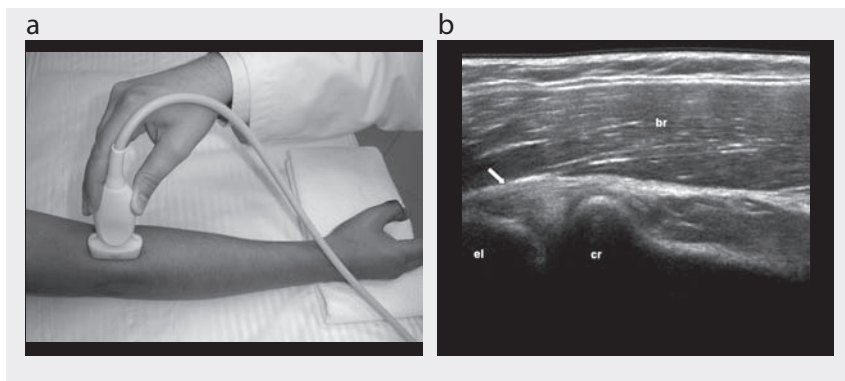
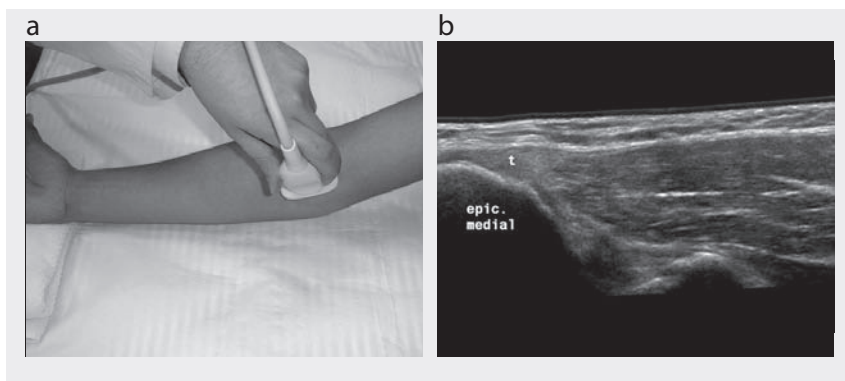


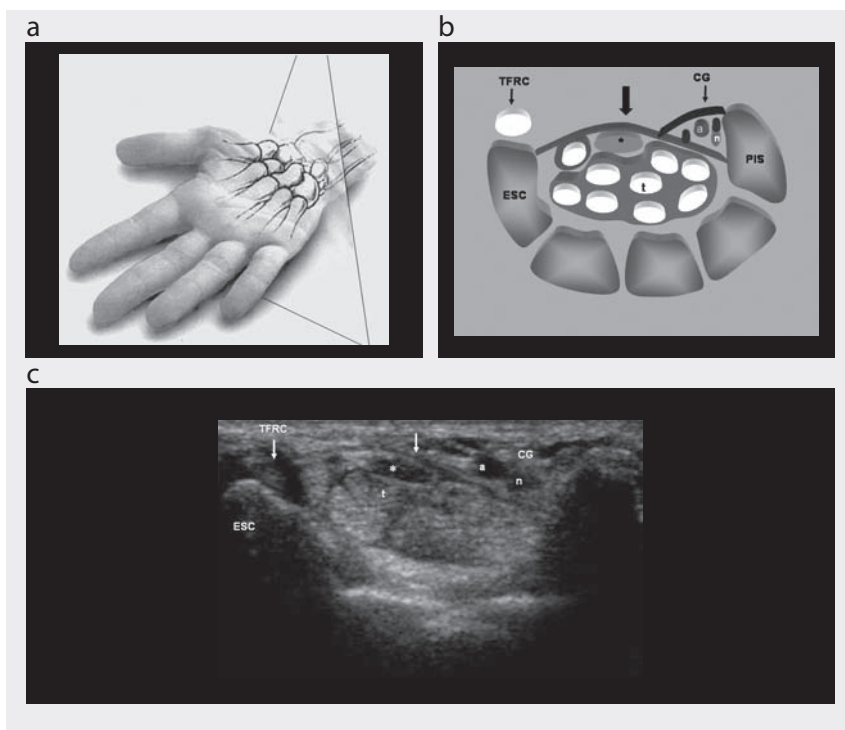
Fig. 6.24. Common tendon of the forearm flexors. (a) Examination technique and (b) ultrasound scan. epic. medial, medial epicondyle. t, common tendon of forearm extensors



## Wrist

The tendon groups of the wrist are flexors and extensors. The flexor tendons are located on the palmar face and comprise the digitorum flexor, carpi radialis flexor, carpi ulnaris flexor and pollicis longus flexor. The digitorum flexor tendons and the pollicis longus tendons pass through an osteofibrous tunnel—the carpal tunnel—bordered by the flexor retinaculum (anterior) and the carpus bones (posterior, lateral, medial). The other structures found inside the carpus, forming a kind of compartment, are fat, the median nerve and two synovial bursae: the radial, surrounding the long flexor tendon of the pollex, and the ulnar, involving the superficial and deep digital flexor tendons (Fig. 6.25).

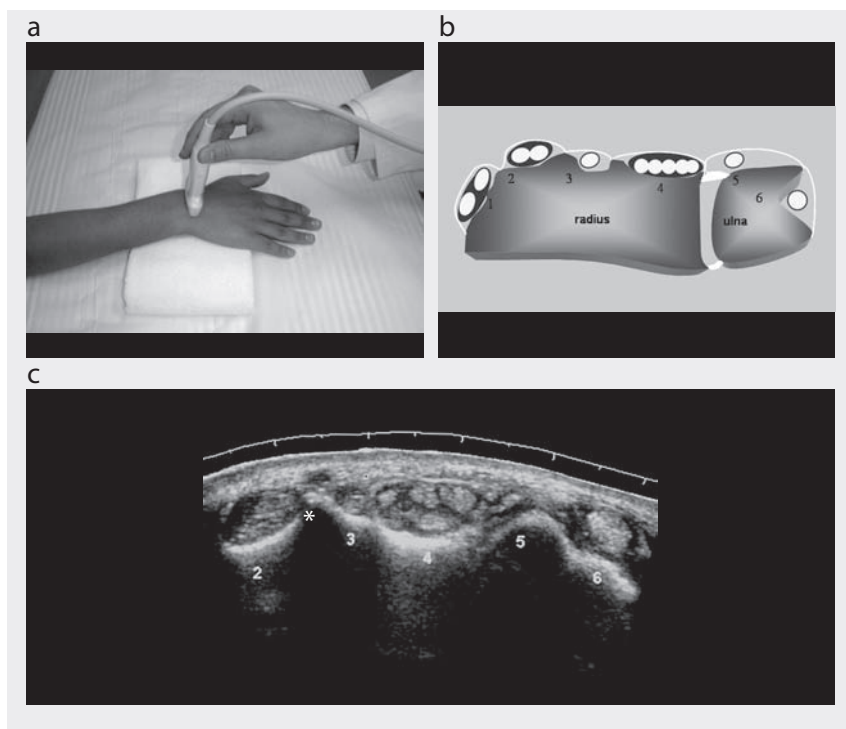
Fig. 6.25. Carpal tunnel (arrow). (a) Section plane at the proximal carpal tunnel with ultrasound beam. (b) Structures found inside the carpal tunnel. (c) Ultrasound scan. Dotted line, retinaculum of flexors; ESC, scaphoid; TFRC, carpal radial flexor tendon; CG, Guyon channel; \*, median nerve; t, finger flexor tendon; PIS, pisiform; t, trapeze; n, ulnar nerve; a, artery



The six synovial compartments of the extensors on the dorsal region of the wrist have individual synovial sheaths and are maintained in position by the retinaculum (dorsal carpal ligament; Fig. 6.26). The sheaths of the second, third and fourth compartments are connected; the presence of a small amount of fluid within them is normal, especially in the sheaths around the tendons of the second compartment.

The first compartment contains the abductor pollicis longus tendon and the pollicis brevis extensor in a single synovial sheath, situated on the lateral fascia of the wrist in contact with the radius stylohyoid process. It is the extensor compartment most frequently involved in stenosing tenosynovitis (De Quervain tenosynovitis; Fig. 6.27). This condition can be secondary to inflammatory arthritis, to acute or repetitive microtraumas due to gripping movements or to ulnar deviation of the wrist. It is more frequent in women and is bilateral in up to 30% of cases. Clinical examination reveals pain during palpation of the radial border of the wrist, and it may be difficult to differentiate from thumb carpometacarpal joint arthritis in the initial stages.

Fig. 6.26. Synovial compartments of the extensors of the wrist. (a) Examination technique. (b) Transverse view of the wrist at the level of the distal radio and ulna. (c) Ultrasound image. Sonography: star, tuber of Lister; 1, long abductor tendons and short extensor of the thumb; 2, radial extensor tendons of the carpus; 3, long extensor tendon of the thumb; 4, extensor tendons of the fingers; 5, extensor tendon of the fifth finger; 6, ulnar extensor tendon of the carpus

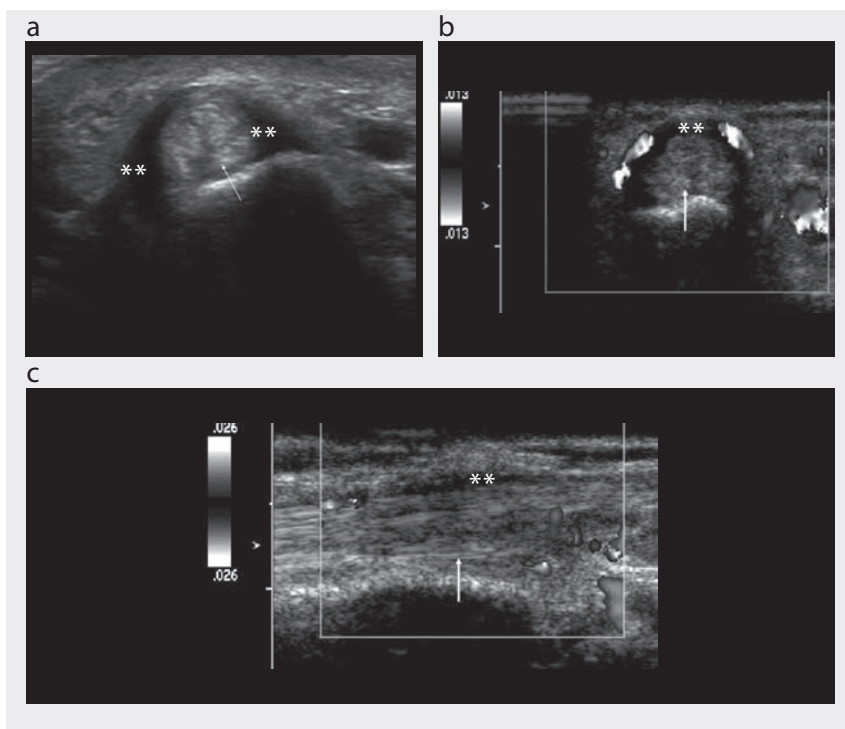


The second compartment contains the short and long radial extensor tendons of the carpus in the anatomical snuffbox. The long radial extensor tendon is situated at the base of the second metacarpal and the short tendon in the dorsal area of the third metacarpal.

The third compartment corresponds to the long extensor tendon of the pollex, medial to the tubercle of the radius (Lister tubercle). It borders the anatomical snuff-box medially, passing over the radial extensor tendons (posterior) and inserts into the dorsal region of the distal phalanx at the base of the thumb.

The fourth compartment is composed of the common tendons of the digital extensors and the indicis extensor. The common extensor tendon is inserted in the medial and distal phalanges of the second to the fifth fingers. The end of the indicis extensor is located at the proximal phalanx of the second finger.

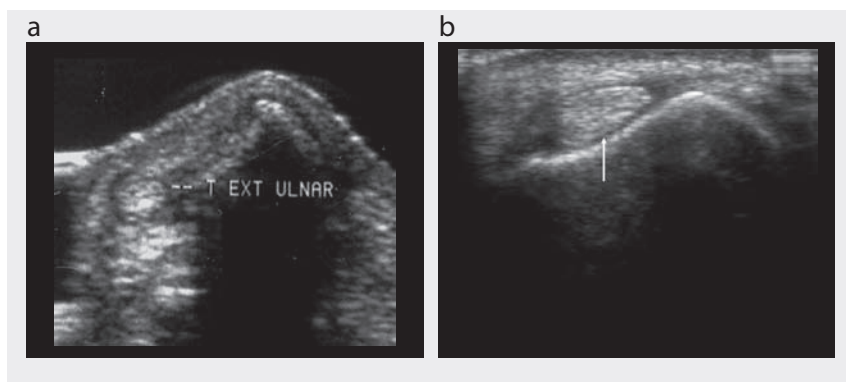
Fig. 6.27. De Quervain tenosynovitis. (a) B-scan showing thickening of the synovial compartment (arrow) and the retinaculum (echo-poor halo, stars) of the extensors. (b), (c) Colour Doppler showing increased flow in the retinaculum, sometimes the only alteration seen



The fifth compartment contains the extensor tendon of the fifth finger, seen posterior to the radioulnar joint, with insertion in the medial and distal phalanges of the fifth finger.

The sixth compartment corresponds to the carpi ulnaris extensor tendon, situated adjacent to the styloid process of the ulna and attached to the base of the fifth metacarpal. This is the second most common location of tenosynovitis, due to repetitive catching of an object. This wrist tendon is the most vulnerable to subluxation or luxation (Fig. 6.28).

Fig. 6.28. (a) Subluxation of the ulnar extensor tendon of the carpus, with deformation of the ulna head. (b) Topical tendon (arrow)



### Fingers

The tendinous anatomy of the fingers is different in the palmar and dorsal regions. A central tendon is inserted in the base of the medium phalanx on its dorsal face. Two tendinous bands meet near the base of the distal phalanx, medially and laterally to this tendon, forming the terminal tendon. Narrow strips of collagen, known as sagittal bands, link these structures to provide stability and allow harmonious extension. Because of this complex anatomy, the term 'digital extensor apparatus' is used rather than 'extensor tendon of the finger' (Fig. 6.29).

The flexor tendons are located in the palmar region of the hand and fingers. The superficial flexor tendon at the level of the proximal phalanx is anterior to the flexor digitorum profundus. In its distal course, it divides into two bands, with insertion in the medial phalanx posterior to the flexor digitorum profundus, which runs to the base of the distal phalanx (Fig. 6.30). In contrast to the extensor apparatus, the flexor tendons have a synovial sheath all along the phalanges.

In cases of tenosynovitis, there may be some parietal thickening, fluid or increased flow in the synovial sheath on colour Doppler (Fig. 6.31).

Fig. 6.29. (a–e) Digital extensor apparatus. TT, terminal tendon; tlub, lumbrical muscle tendon; bs, sagittal band; mtc, metacarpal bone; 1, tendon and interosseous muscle; 2, central tendon; 3, divisions of the central tendon; 4, collateral ligaments; 5, intermetacarpal transverse ligament

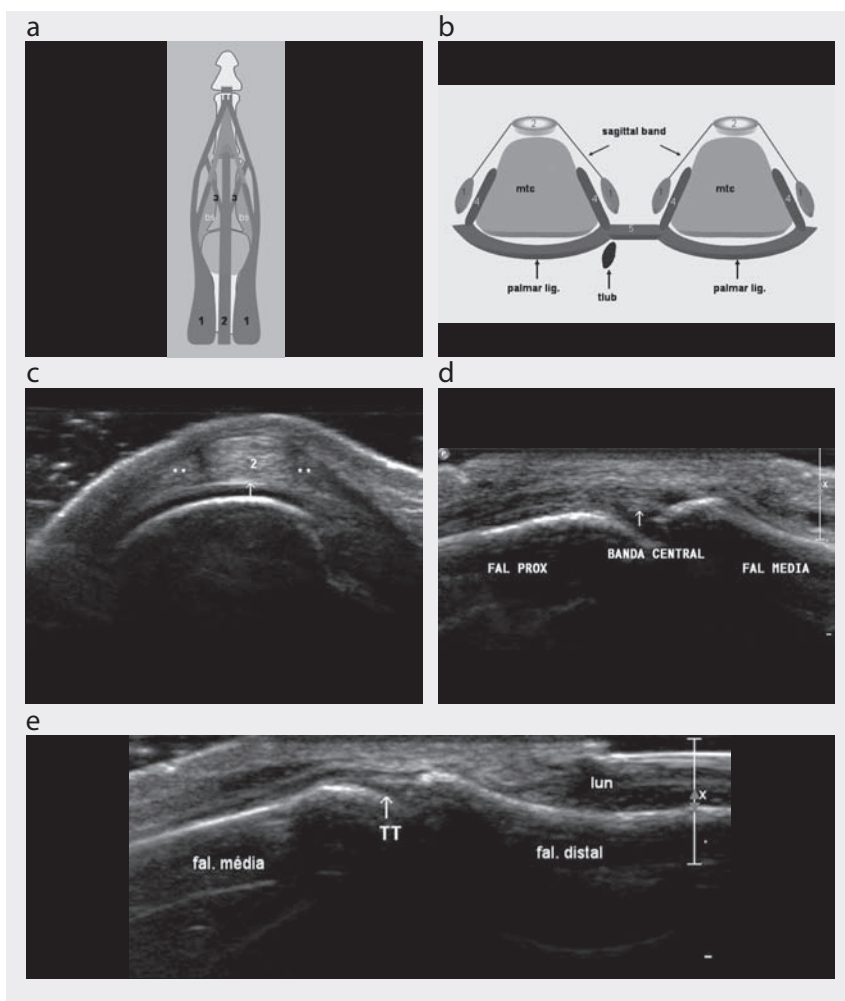


Fig. 6.30. Flexor tendons. (a) Surgical view. (b)–(e) Sections at which transverse scans of the flexor tendons were made. (f) Longitudinal scan of the flexor tendons of the proximal, medial and distal phalanges. FS and continuous arrows, superficial flexor tendon; FP and stars, deep flexor tendon; dotted arrow, flexor tendons

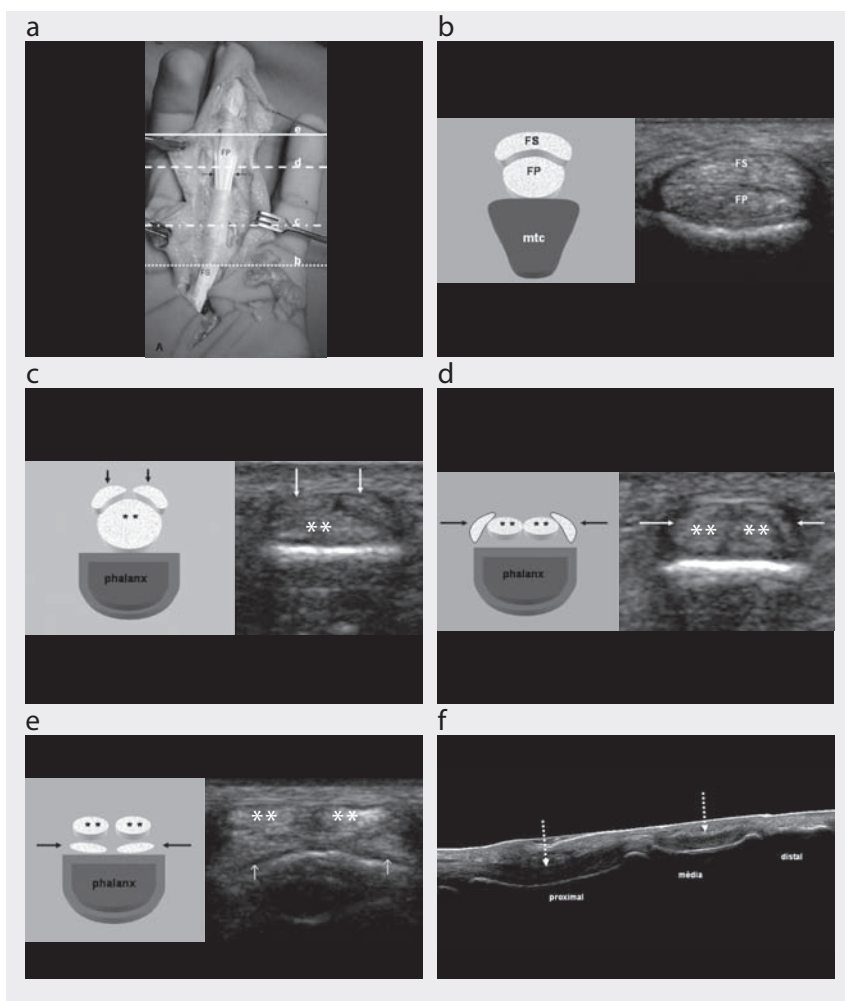
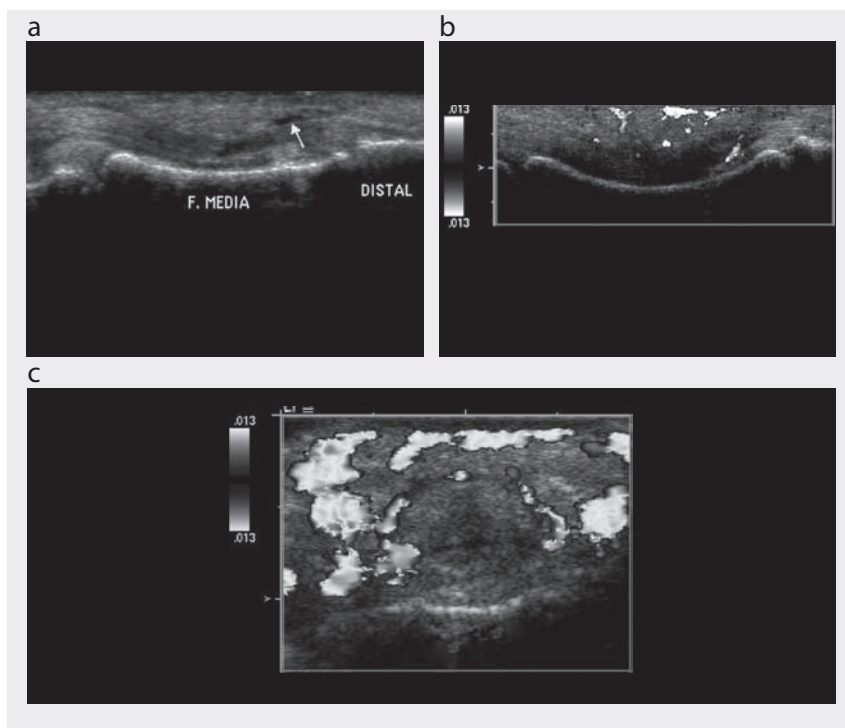




Fig. 6.31. Tenosynovitis of the flexors of the second finger. (a), (b) Longitudinal and (c) transverse scans of the tendon flexors, indicating thickening of the synovial sheath (arrow in (a)) with a large flow increase on colour Doppler (b), (c)



## Lower limbs

### Hip

The hip, like the shoulder, has a cuff made up of the musculotendinous units of the glutei minimus and medius, which are responsible for the internal rotation and abduction movements of the joint. The tendon of the gluteus minimus is situated in the anterior plane of the largest femoral trochanter, and the gluteus medius is in the lateral and posterosuperior planes, with intertwined fibres. There is a hypovascularized area, similar to that of the supraspinatus and infraspinatus tendons (Fig. 6.32).

Adjacent to the tendons, three synovial bursae are seen: the trochanteric bursa, the bursa of the subgluteus minimus and the bursa of the subgluteus medius. A bursa of the subgluteus maximus has been proposed.

A painful greater trochanter is a common condition. One of the main causes is tendinopathy of the glutei and trochanteric bursitis (Fig. 6.33, Fig. 6.34). These are not always readily diagnosed with ultrasound due to the oblique path of the tendons and patient characteristics, such as obesity.

Fig. 6.32. Tendons of the gluteus minima and media. (a) Insertions of the two tendons. (b) Examination technique. Longitudinal scans of the (c) gluteus minima tendon (arrow) and (d) the gluteus media tendon (arrow), with forms and echogenicity similar to that of the rotator cuff tendons of the shoulder. mi, insertion of gluteus minima tendon; me, insertion of gluteus media tendon

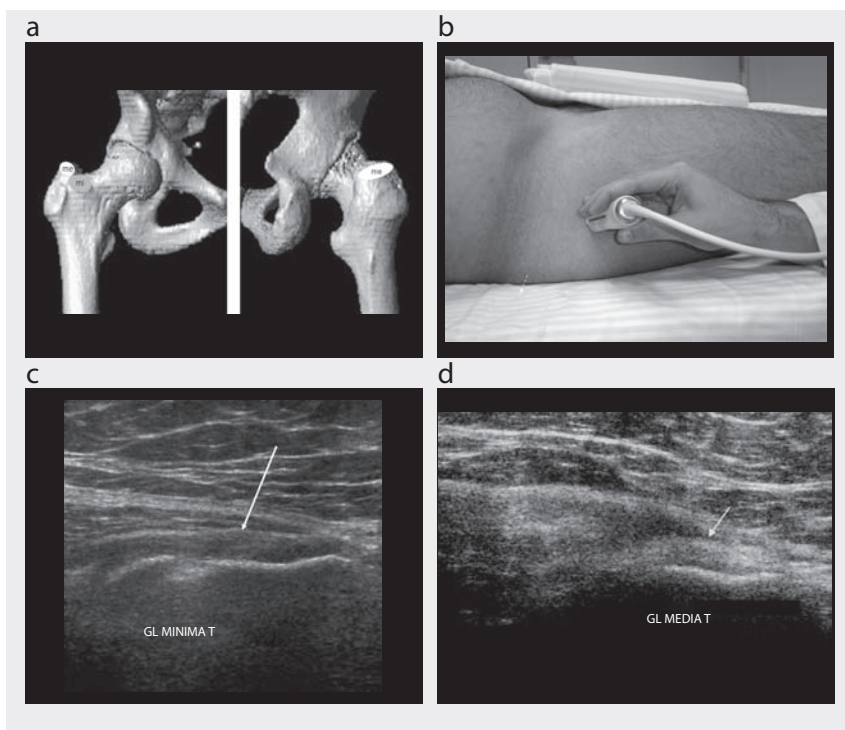


Fig. 6.33. Tendinopathy of the glutei enhanced by thickening and hypoechogenicity (arrow). (a) Transverse and (b) longitudinal scans

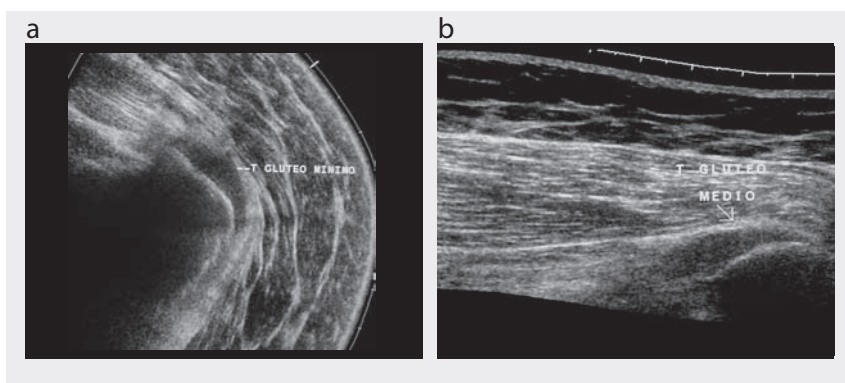
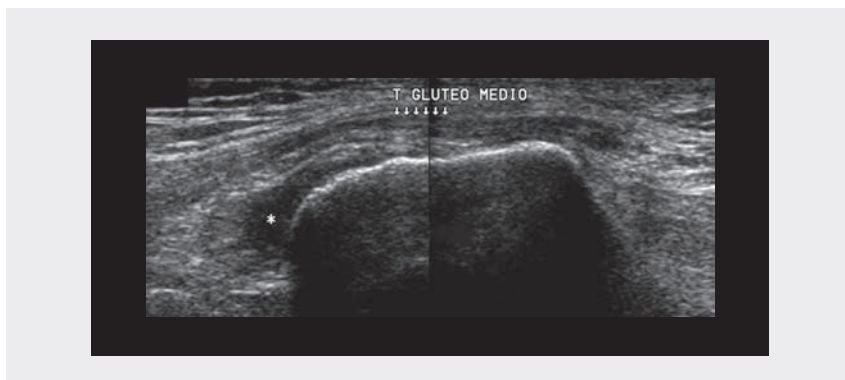
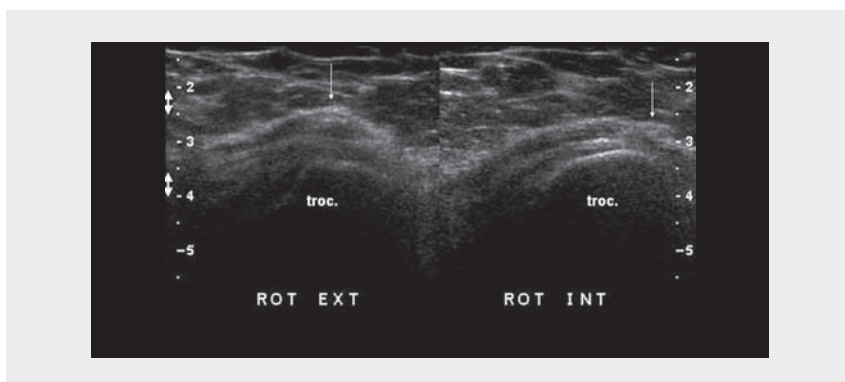


Fig. 6.34. Bursitis involving the synovial bursa of the medium subgluteus, containing a moderate amount of fluid (star)



Ultrasound examination is useful in cases of hips with a snapping, characterized by pain associated with an audible or tangible snap during movement of the hip. The cause may be intra- or extra-articular. The extra-articular factors are friction of the fascia lata against the largest femoral trochanter (Fig. 6.35) or of the tendon of the iliopsoas against the iliopectineal eminence.

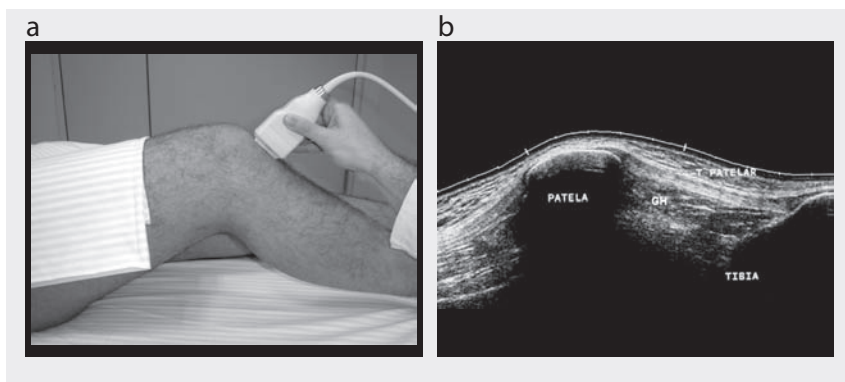
Fig. 6.35. Snapping hip. Transverse scan of the largest femoral trochanter (troc), indicating thickening of the fascia lata (arrow) situated lateral to the hip on internal rotation (rot int); on external rotation (rot ext), the fascia lata is in anterior position (arrow), producing a snap



## Knee

The patellar tendon in the periarticular area of the knee is that most frequently injured. It is situated between the subcutaneous tissue and the pretibial bursa (deep infrapatellar bursa), posterior to the inferior half of the tendon. The acoustic shadow of the cortical bone is used to identify its insertion into the patella and into the tuberosity of the tibia. Posterior to the tendon is a pad of fat known as the infrapatellar or Hoffa pad, which is joined to the articular synovia. The normal tendon is formed of parallel, homogeneous fibres, visualized as alternate echo-poor and echo-rich bands (Fig. 6.36). The average tendon is 4 mm thick and 21 mm wide. Sedentary people have thinner, ribbon-shaped tendons. Its function is to transmit the strength of the femoral quadriceps muscle to the tuberosity of the tibia.

Fig. 6.36. Patellar tendon. (a) Examination technique. (b) Ultrasound, indicating the patellar tendon and the Hoffa pad (GH), which is echo-poor with linear echo-rich images



The term 'jumper's knee' is used to describe a painful patellar tendon. The condition is common among athletes and young adults who practise sport regularly, secondary to excessive effort, especially in sports that require extension of the knee, such as running, basketball and football. Usually, the dominant side is affected. From the histopathological point of view, jumper's knee is characterized by the presence of tendinosis, usually beginning at the proximal insertion of the tendon into the apex of the patella.

Ultrasound may show not only echographic alterations of the tendon (Fig. 6.37), but also oedema of the infrapatellar pad and, in severe cases, thickening and irregularity of the tendinous envelope.

An important differential diagnosis of jumper's knee is Osgood-Schlatter disease, which consists of osteochondrosis or osteochondritis of the anterior tuberosity of the tibia. It is common in adolescent boys who practise sport frequently. Microtraumas due to functional activity of the tendon appear to be responsible for the lesion. Clinically, the complaint involves pain and local oedema. Simple X-ray is not sufficient in these cases, because it does not show the earliest alterations, which

are thickening and reduction of the echogenicity of the distal portion of the tendon, accompanied by oedema of the soft tissues around the anterior tuberosity of the tibia, sometimes associated with bone fragmentation (Fig. 6.38).

Fig. 6.37. Patellar tendon. Longitudinal scan, showing (a) proximal tendinopathy (arrow) and (b), (c) rupture

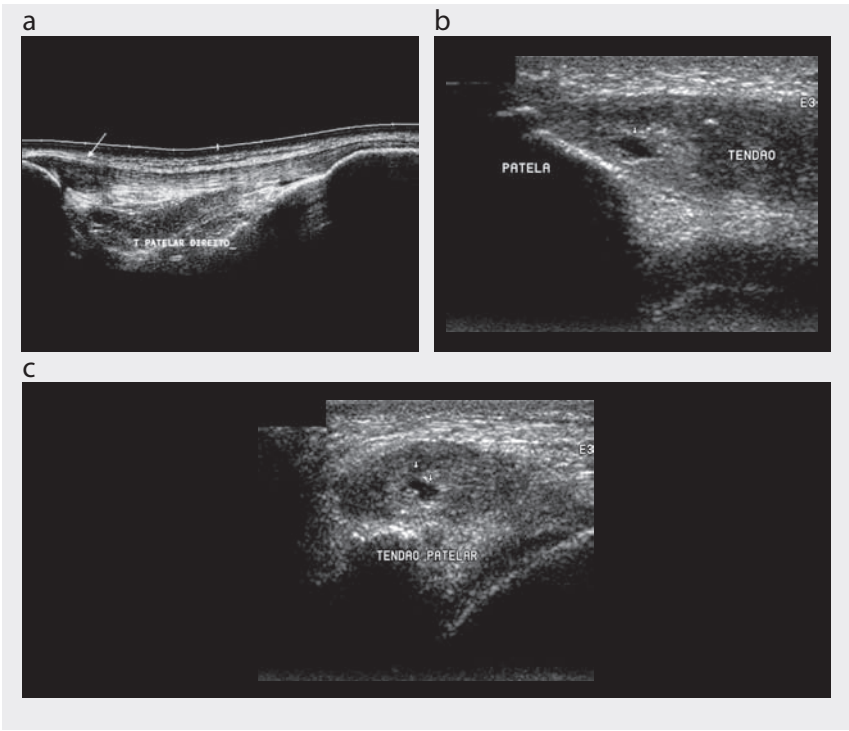
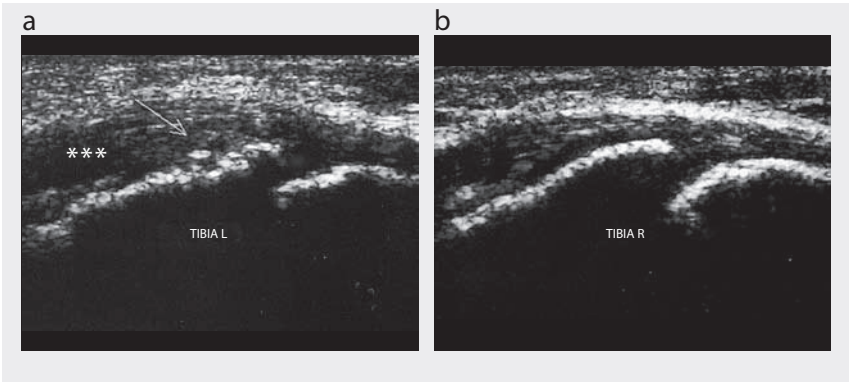
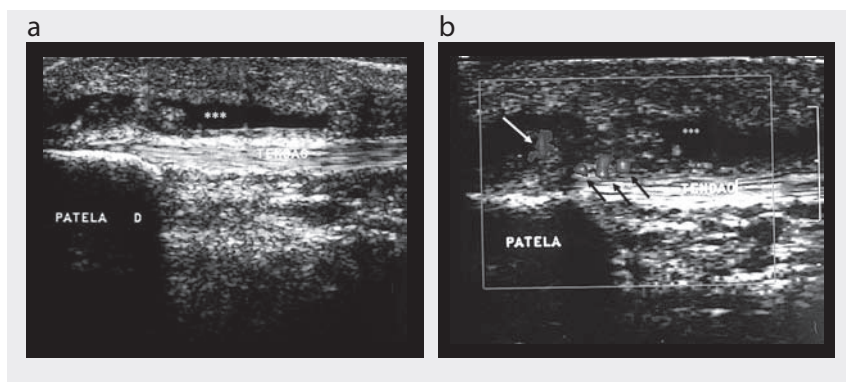


Fig. 6.38. (a) Osgood-Schlatter disease, with thickening of the insertion of the patellar tendon into the tibia, undefined, irregular contours (stars) and a small fragmented bone in the apophysis (arrow). (b) Normal contralateral side



The suprapatellar, prepatellar and pes anserinus tendon bursae are the main synovial bursae in the region. The suprapatellar bursa is used in research on joint effusion, which is in the joint cavity in about 90% of cases. Inflammatory processes are common in the synovial bursae situated anterior to the patella (Fig. 6.39) and adjacent to the pes anserinus tendons.

Fig. 6.39. Prepatellar bursitis. (a) Fluid (stars) and parietal thickening of the synovia. (b) Colour Doppler showing increased flow (arrows)



## Ankle

About 20% of lesions in runners involve the calcaneal (Achilles) tendon. This tendon is formed by the junction of the tendons of the gastrocnemius and soleus muscles in the middle third of the leg, with insertion into the superior tuberosity of the calcaneus bone. The tendon is about 15 cm long and 3.5–6.9 mm thick, and is larger in men and in tall and elderly people. The tendinous envelope is a paratenon. A retrotibial fat pad (Kager fat pad) is found anterior to the tendon, which may be affected in inflammatory processes. Between the Kager fat pad, the superior tuberosity of the calcaneus bone and the calcaneus tendon, there is a synovial bursa (retrocalcaneal), measuring less than 2 mm in the anteroposterior position; its function is to protect the distal portion of the calcaneus tendon from constant friction against the calcaneus bone. Posterior to the calcaneus tendon is another, acquired synovial bursa, which is superficial (subcutaneous) and may be seen when distended with fluid.

On ultrasound examination, the calcaneus tendon has a crescent appearance in the transversal plane, with its anterior concave and posterior convex faces distally rectified. Longitudinally, it presents a fibrillar echogenic pattern, although it may be echo-poor closer to its insertion (Fig. 6.40).

Alterations to the tendon can be either acute or chronic or be associated with a background disease, such as diabetes mellitus, collagenosis, rheumatoid arthritis, gout or familial hypercholesterolaemia. Tendinous xanthoma is a diagnostic criterion of heterozygous familial hypercholesterolaemia, the calcaneus tendon being the most frequently affected. Ultrasound is useful for demonstrating the xanthomatous deposition, which

occurs as fusiform thickening of the tendon associated with echo-poor foci (Fig. 6.41). As the ultrasonographic signs usually precede clinical manifestation of the disease, ultrasound is the recommended method for diagnosing and monitoring this condition.

Fig. 6.40. Calcaneus tendon. (a) Examination technique. Ultrasound examination in the (b) longitudinal and (c) transverse planes

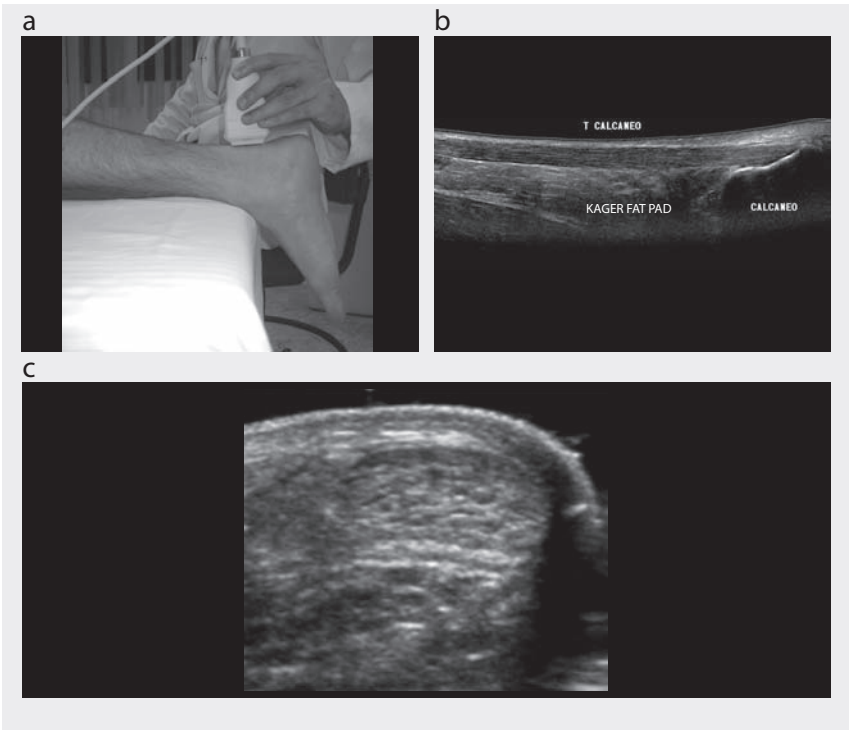
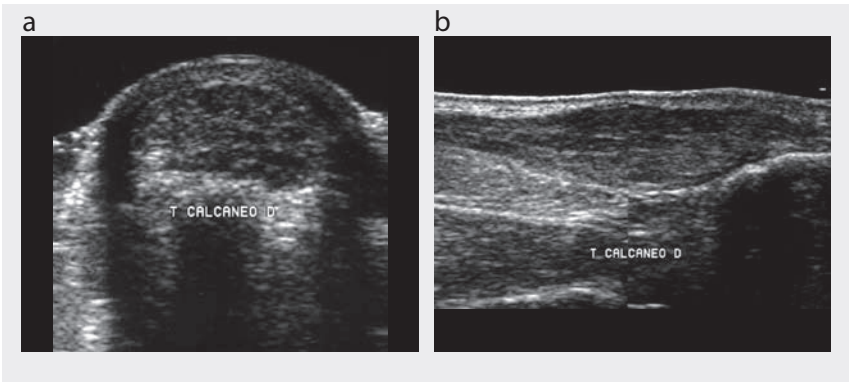


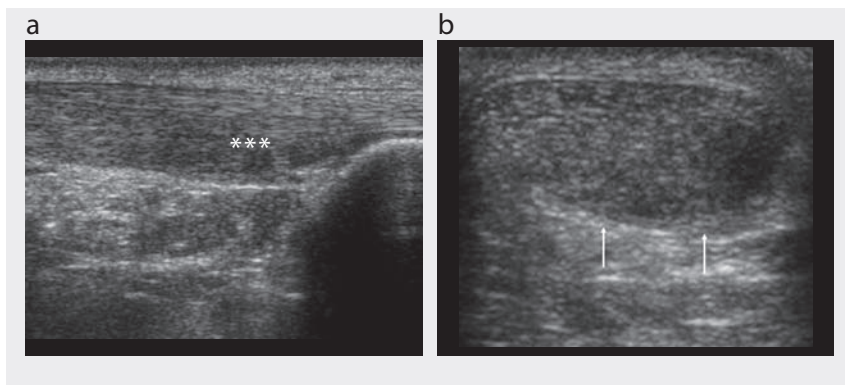
Fig. 6.41. Xanthoma of the calcaneus tendon: thickening associated with heterogeneous echo texture of the tendon due to xanthomatous deposition. (a) Transverse and (b) longitudinal scans





Other common conditions responsible for pain in the region are peritendinitis, paratendinitis and tendinopathies. The pathological processes involving the calcaneus tendon are usually situated in a hypovascularized region 2–6 cm proximal to insertion of the tendon into the calcaneus bone. In tendinopathies, the tendon is thickened, with altered echogenicity, which, in the subtlest cases, is seen as loss of the anterior concavity of the tendon in transversal (oblique) images (Fig. 6.42).

Fig. 6.42. Calcaneus tendinopathy, with thickening and alteration of the echogenicity ((a), stars) and contour ((b), arrows) of the tendon



In Haglund deformity, the calcaneus tendon is altered close to its insertion, with hypertrophy of the posterosuperior tuberosity of the calcaneus, affecting the retrocalcaneal bursa and the calcaneus tendon. Consequently, there is retrocalcaneal bursitis and tendinopathy (Fig. 6.43). Insertion tendinopathy may also be due to chronic overload (overuse) in athletes, seen as regions of calcification or intratendinous ossification associated with insertional osteophytes.

Paratendinitis is an inflammation of the paratenon. The echographic outline is blurred, corresponding to thickening (Fig. 6.44), which may extend to the adjacent soft tissue (peritendinitis). Although described separately, these two processes may represent spectra of the same disease.

Unsatisfactory evolution of the pathological process leads to rupture. When partial ruptures affect the anterior surface of the tendon, their diagnosis is facilitated by the inward invagination of the Kager fat pad (Fig. 6.45). Intrastance ruptures, especially small ones, can, however, be confused with severe tendinosis, which is difficult to differentiate by imaging. The presence of peritendinitis may suggest partial rupture, as these conditions coexist in up to 68% of cases.

Local oedema and limitation of plantar flexion in complete tendon ruptures may lead to an erroneous clinical diagnosis in up to 25% of acute cases. Ultrasound diagnosis of a complete rupture may be difficult, especially when the paratenon is intact. In diagnostic doubt, it is advisable to conduct plantar and dorsal flexion manoeuvres,



Fig. 6.43. Haglund deformity. Tuberosity of the calcaneus ((a), arrow; (b), (c), stars) associated with tendinopathy of the calcaneus tendon, resulting in retrocalcaneal bursitis and subcutaneous bursitis ((d), stars; (e), arrow) on colour Doppler (longitudinal and transverse planes) and MRI (f)

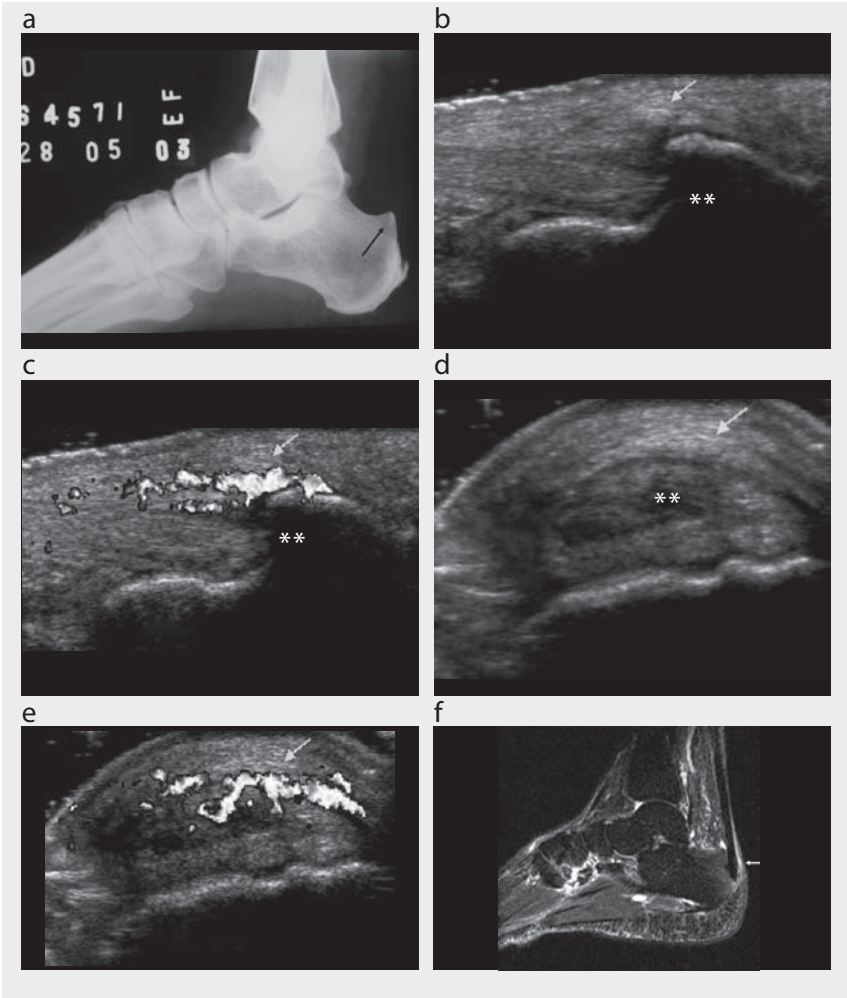


Fig. 6.44. Calcaneus paratendinitis. Thickening and hypoechogenicity of the posterior paratenon (arrow) in the longitudinal (a) and transverse (b) planes

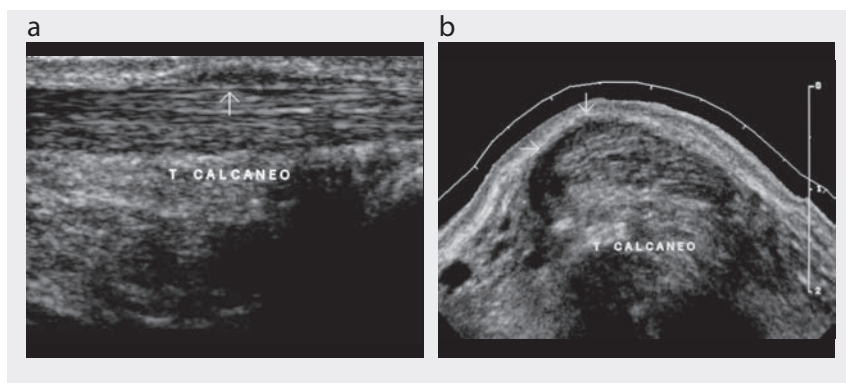
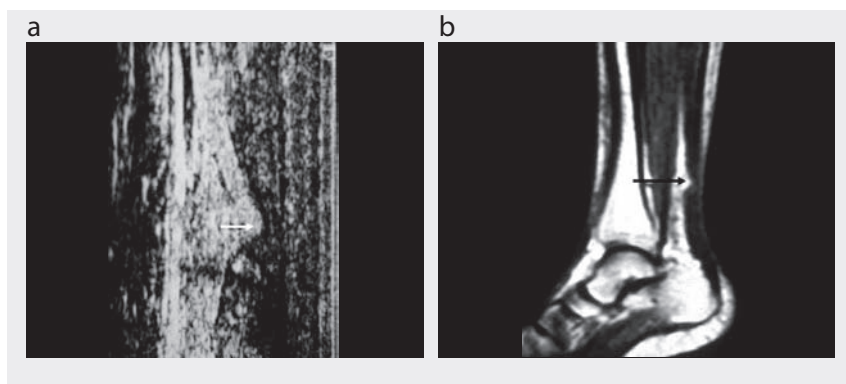


Fig. 6.45. Partial lesion with inward invagination of the Kager fat pad (arrows). (a) Ultrasound. (b) MRI



which not only confirm a clinical hypothesis but contribute to therapeutic choices by verifying the proximity of the tendinous stumps (Fig. 6.46).

Another useful sign of complete lesions is the presence of posterior acoustic shadow on the retracted tendinous stumps (Fig. 6.47), secondary to the oblique acoustic bundle on their surfaces, which have an irregular outline. Use of hyperflow in colour Doppler in chronic cases is controversial. In some descriptions, neovascularization is correlated with failed scarring; others correlate it with pain symptoms that are not related to the prognosis.

Fig. 6.46. Complete rupture of the calcaneus tendon ((b), arrows), characterized by dorsal flexion of the foot; haematoma (stars) in the unaffected paratenon ((a), (b), dotted arrow)

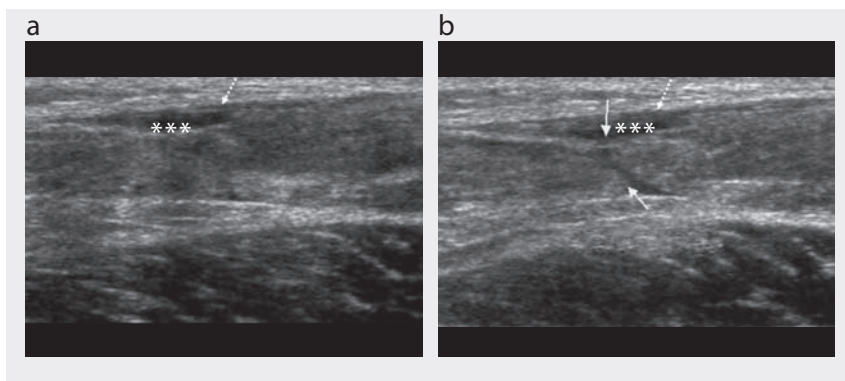
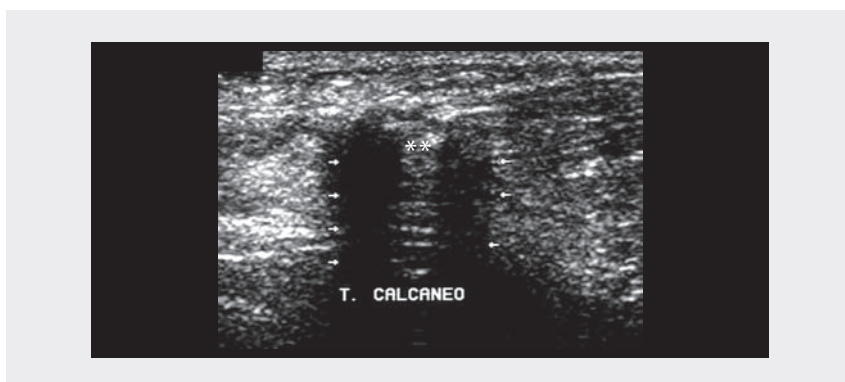


Fig. 6.47. Anisotropy characterized by acoustic shadow in the topography of the tendinous stumps (arrows). Stars, tendinous rupture

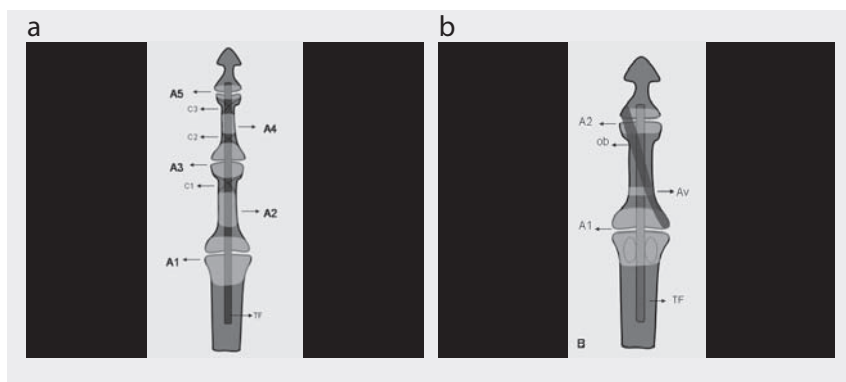


## Finger pulley systems

The flexor system of the second to fifth fingers is composed of five annular and three cruciform pulleys, corresponding to thickening of the synovial sheath of the flexor tendons. The odd annular pulleys are situated on the metacarpophalangeal (A1), proximal interphalangeal (A3) and distal interphalangeal (A5) joints, which are bound in the capsuloligamentous structures. The even pulleys are situated and inserted in the phalanges: A2 in the proximal two thirds of the proximal phalange and A4 in the middle portion of the middle phalange. The cruciform pulleys are interposed between the annular pulleys (Fig. 6.48).

The thumb is slightly different, with an annular pulley for each of the metacarpophalangeal (A1) and interphalangeal (A2) joints and one of variable position (Av) on the

Fig. 6.48. Pulleys of the flexors of the fingers (a) and the thumb (b). TF, flexor tendons; A, annular pulleys; C, cruciform pulleys; ob, oblique pulley



proximal half of the proximal phalanx. There is also an oblique pulley extending from the ulnar aspect of the proximal phalanx to the radial aspect of the distal phalanx (Fig. 6.48).

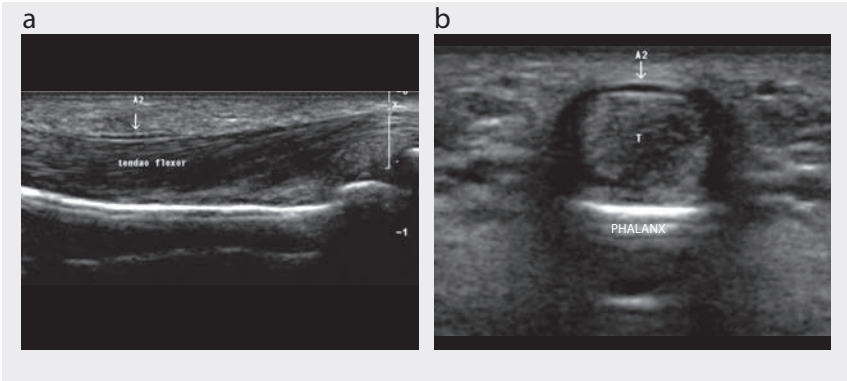
The main function of the pulleys is to maintain the flexor tendons in contact with the cortical bones of the phalanges and the metacarpophalangeal joints and interphalanges, transforming the movement of the flexor tendons during flexion of the fingers into rotation and torque at the level of the interphalangeal and metacarpophalangeal joints. The most important pulleys in terms of functionality are the annular ones, especially A2 and A4 for the second and fifth fingers and A2 for the thumb. The cruciform pulleys have a secondary role, allowing approach of the annular pulleys during flexion of the fingers while maintaining the effectiveness of the movement.

Lesions of the pulleys appear after vigorous flexion of the proximal interphalangeal joints at an angle wider than 90°, with extension of the distal metacarpophalangeal and interphalangeal joints, resulting in heavy mechanical overload on the A2 and A3 pulleys.

It is important to identify the type of lesion in order to guide treatment. In partial ruptures, the treatment is conservative; complete ruptures can be treated either conservatively or by surgery, depending on the patient's age and level of activity and on the number of pulleys involved. Lack of treatment of this type of lesion can lead to osteoarthritis and contractures in flexion of the proximal interphalangeal joints. In acute trauma, with oedema and local pain, known as tenosynovitis, displacements of the proximal interphalangeal joints and ruptures of the pulleys are not easily differentiated by physical examination, and diagnosis is based on imaging methods.

The cruciform pulleys cannot be visualized by ultrasonography. All the annular pulleys can be identified with high-resolution linear transducers with a frequency of 17 MHz. At a frequency of 12 MHz, only the A2 and A4 pulleys can be identified (Fig. 6.49), as the dimensions of the pulley are directly proportional to the size of the hand.

Fig. 6.49. Ultrasonography of the A2 annular pulley with a 17-MHz high-resolution transducer in the (a) longitudinal and (b) transverse planes. T, tendon



Diagnosis of lesions of the pulleys is based on the presence of two indirect signs. The first is peritendinous fluid, and the second is an increase in the distance between the phalangeal cortical bone and the posterior surface of the flexor tendons. The normal distance is 1 mm; in complete ruptures and ruptures of more than one pulley, the space between the phalange and the flexor tendons is as shown in Table 6.1. Measurements are made as shown in Fig. 6.50.

Table 6.1. Indirect signs of pulley lesions

Pulley	Place of measurement	Partial lesion	Complete lesion
A2	15–20 mm distal to the base of the proximal phalange	$1.0 \text{ mm} < D < 3.0 \text{ mm}$	$D > 3.0 \text{ mm}$
A4	Middle portion of the middle phalange	$1.0 \text{ mm} < D < 2.5 \text{ mm}$	$D > 2.5 \text{ mm}$

D, distance between phalangeal cortical bone and posterior surface of flexor tendons

The A2 pulley is that most commonly ruptured (Fig. 6.51). When the distance is greater than 5.0 mm, the A3 pulley is also involved. The pulleys, especially the A1 pulley, can become thicker (Fig. 6.52), and the finger resembles a trigger because the flexor tendons move with difficulty in the osteofibrous tunnel.

Fig. 6.50. (a), (b) Points for measuring the distance of the cortical bone in relation to the flexor tendons (dotted lines) for diagnosis of A2 and A4 annular pulley lesions

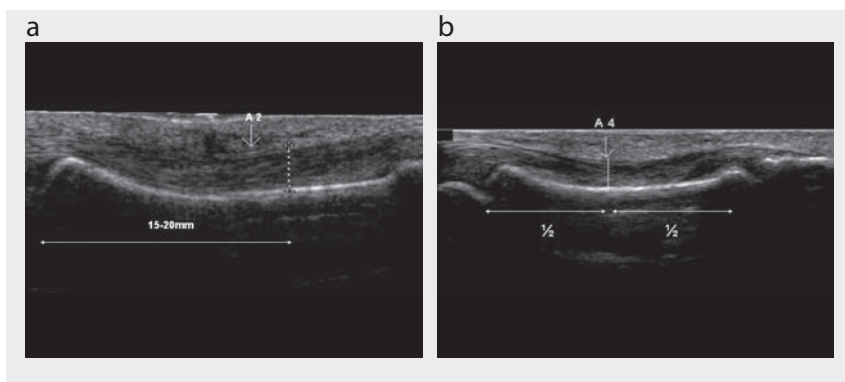


Fig. 6.51. Lesion of the A2 annular pulley. (a) Increased distance of the proximal phalangeal cortical bone in relation to the flexor tendons (arrow), which increases (0.4 cm) when the finger is flexed (b), (c)

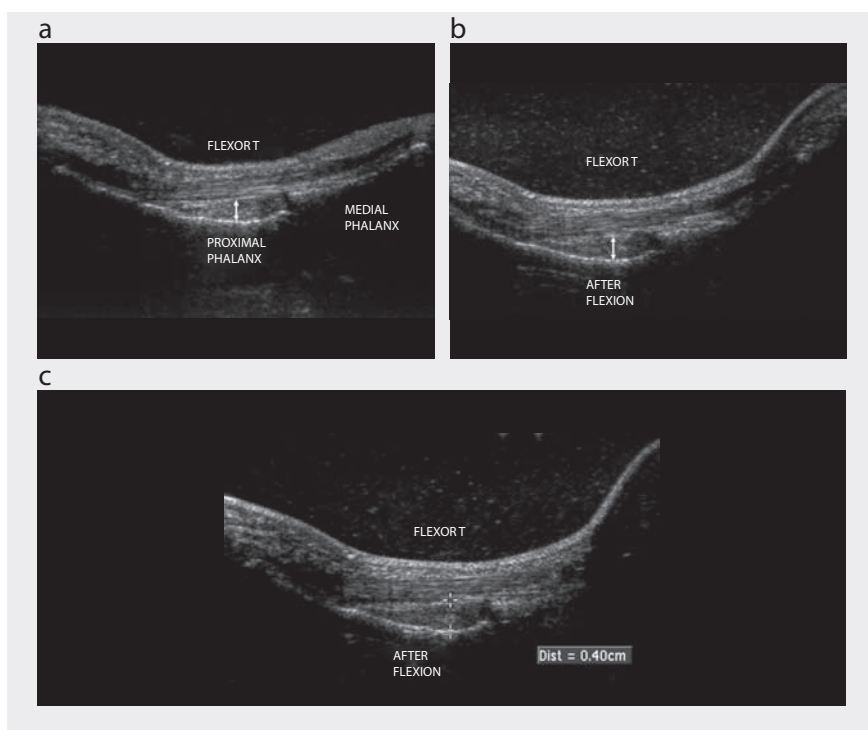
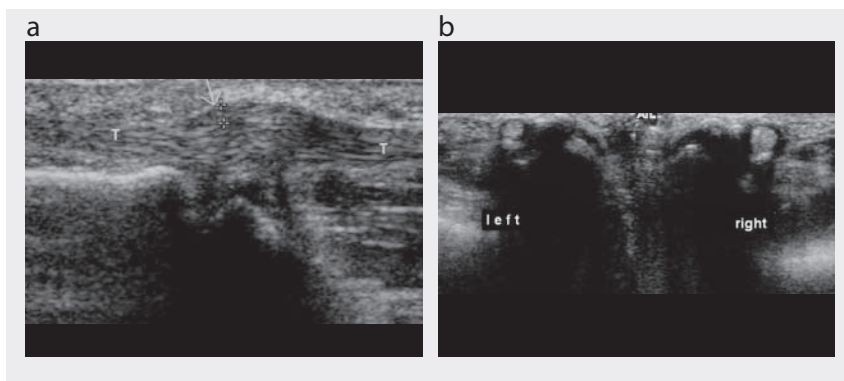


Fig. 6.52. Finger in trigger position: thickening of the A1 annular pulley ((a), calipers, arrow) and the thumb flexor tendons (T), seen (b) as an echo-poor halo in the tendons to the right



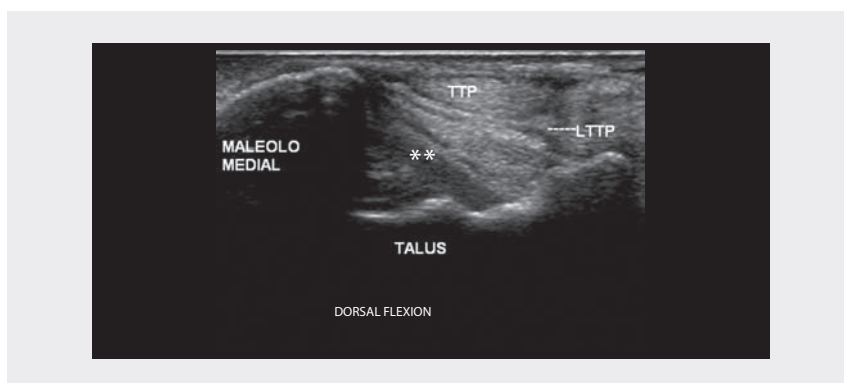
## Ligaments

### Structural features

Ligaments are made up of thick connective tissue, consisting mostly of type I collagen. The collagen fibres form bundles or fascicles, which are wavy and have a less regular, more heterogeneous histological aspect than tendons on ultrasonography. The presence of synovia or adipose tissue in the fascicles contributes to the heterogeneity of some ligaments, such as the deltoid (Fig. 6.53) and anterior cruciate ligaments.

Ultrasound is used mainly to study extra-articular ligaments in the diagnosis of acute ruptures and to monitor treatment or chronic lesions that result in instability of the joint.

Fig. 6.53. Heterogeneous echo texture (stars) of the deep portion of the deltoid ligament (posterior tibiotalar ligament) containing adipose tissue. LTTP, posterior tibiotalar ligament; TTP, posterior tibial tendon

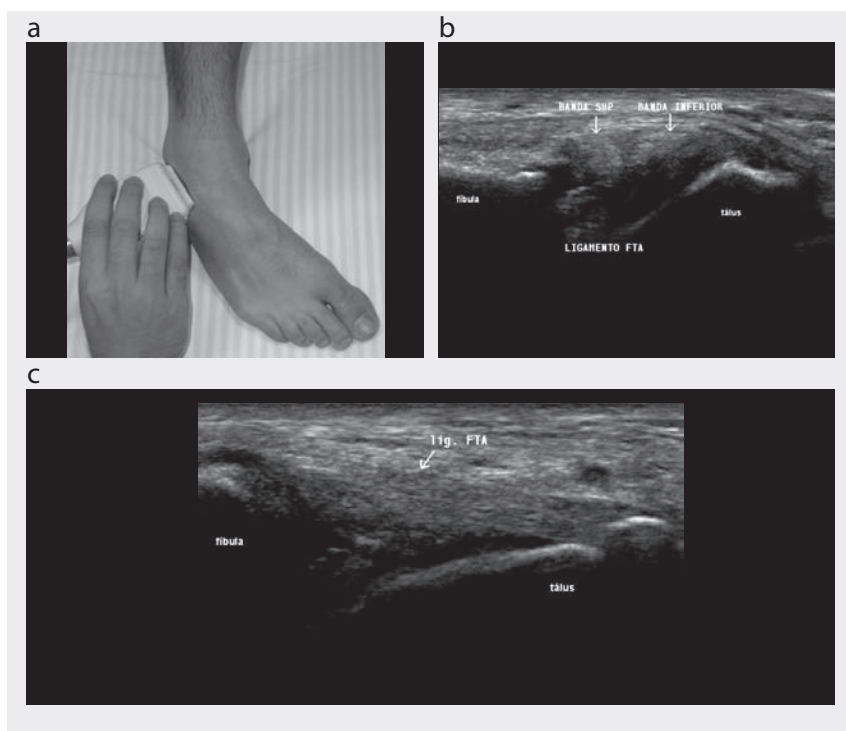


## Lateral ligament complex of the ankle

The commonest lesions associated with sport are of the lateral ligament complex of the ankle (16–21%). These become chronic in more than 40% of cases if not appropriately treated. The lateral ligament complex of the ankle is made up of three ligaments: the calcaneofibular and the anterior and posterior talofibular.

The **anterior talofibular ligament** reinforces the articular capsule, presenting either horizontally or with a discreetly inferior inclination ( $0$ – $20^\circ$ ) from the anterior border of the lateral malleolus to the lateral face of the talus body. A section parallel to the fibres shows a rectilinear trajectory and a uniform thickness of 2–3 mm, with a homogeneous or discreetly heterogeneous echo-rich texture. A transversal scan shows that the ligament is flat, with a concave–convex aspect composed of an upper, larger band and a lower one (Fig. 6.54). The upper band joins the fibular origin of the anterior tibiofibular ligament, while the lower one joins the fibular origin of the calcaneofibular ligament. In the neutral position, the fibres are relaxed and parallel to the long axis of the talus. Plantar flexion and inversion of the foot cause some stretching, generating tension in the fibres.

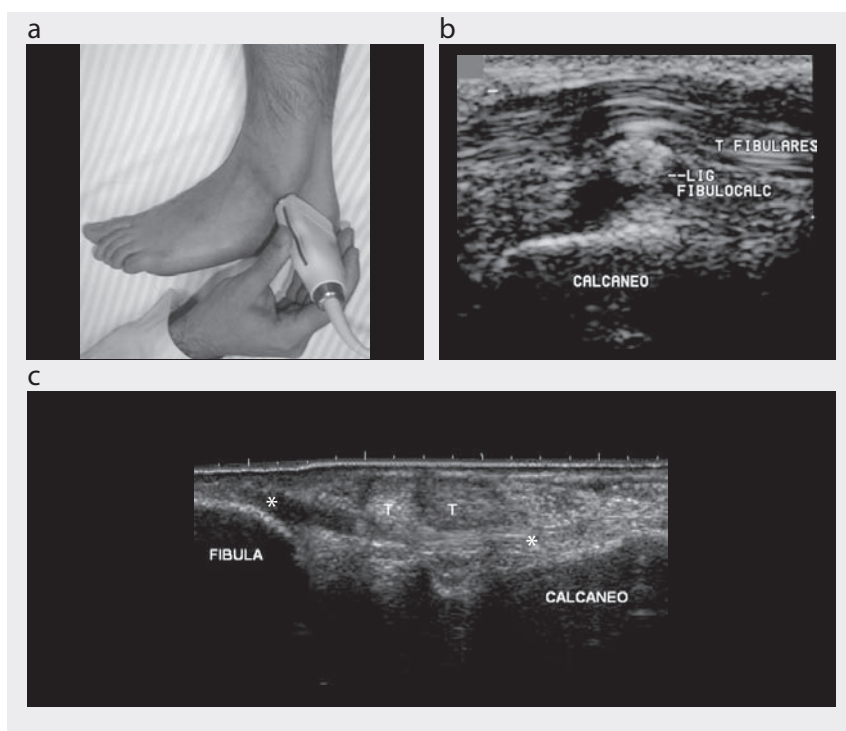
Fig. 6.54. Anterior talofibular ligament. (a) Examination technique. Ultrasound scan indicating the two sides of the ligament in the (b) transverse plane and its flat aspect in the (c) longitudinal plane3. FTA, anterior talofibular





The **calcaneofibular ligament** has a string-like aspect and runs in a coronal posteroinferior oblique plan, forming an angle of approximately 45° in relation to the fibular diaphysis, joining the lower aspect (but not the extremity) of the anterior margin of the lateral malleolus at a small tubercle situated on the lateral border of the calcaneus (Fig. 6.55).

Fig. 6.55. Calcaneofibular ligament. (a) Examination technique. Ultrasound scans in the (b) transverse and (c) longitudinal planes, showing the string-shaped ligament in close contact with the fibular tendons (T). Star, calcaneofibular ligament



The **posterior talofibular ligament** is difficult to examine by ultrasound. It looks like a bundle, with interposed bands of adipose tissue, and inserts into the internal concave margin of the distal malleolar fossa of the fibula and the lateral tubercle of the posterior process of the talus. The ligaments of the lateral complex are the most frequently injured in ankle sprains, usually due to plantar flexion and supination with inversion of the foot. If the force of the inversion is progressive, the lesions will occur in sequence, from the weakest to the most resistant ligament: the anterior talofibular (in 70% of cases); the calcaneofibular (20–25%), usually accompanied by a lesion of the anterior talofibular, making the hindfoot unstable; ligaments of the sinus tarsus; and the posterior talofibular ligament, which is injured only in ankle luxation.

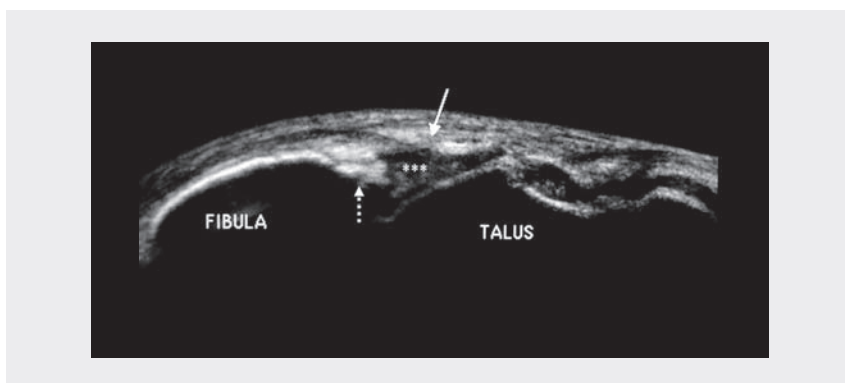
A diagnosis is frequently made solely by clinical evaluation; however, the accuracy of diagnosis of an acute lesion is reduced in 50% of cases by pain and local oedema, and imaging methods are recommended. MRI has been reported to be more accurate than ultrasound for the diagnosis of ligament lesions; however, the studies were conducted before the advent of high-resolution transducers, and there has been no recent comparison of the performance of ultrasound and MRI with current ultrasound equipment.

Ligament lesions can be classified according to the time since the trauma (acute and chronic lesions) and the extent or severity of the rupture (partial or complete). Ultrasound diagnosis is based on direct and indirect signs. The nonspecific, indirect signs in calcaneofibular ruptures are oedema or subcutaneous bruises on the lateral face of the ankle; articular effusion in the anterolateral talofibular recess; lesions of the anterior talofibular ligament; and fluid in the synovial sheath of the fibular tendons.

The direct signs are intrinsic alterations in the form, thickness and echogenicity of the ligament. Some are typical of partial lesions and others of complete lesions; some lesions present both situations, differing only in severity. In partial lesions, thickening and hypoechogenicity are seen. In lesions that are partial or complete, depending on how severely the ligament is affected, tapering, discontinuity and elongation with waving (looseness) of the contours are observed. Complete lesions, such as an absent ligament, complete discontinuity (Fig. 6.56) and amputation of the ligament with frayed stumps, are poorly defined or resemble a nodule (pseudotumour).

These signs are due either to intense oedema and haemorrhage (in partial or complete acute lesions) or to repairing tissue (in subacute or subchronic lesions). About 50% of ruptures of the anterior talofibular ligament are accompanied by fracture or avulsion of a talus bone fragment, and about 45% involve the middle third of the ligament. In the coronal plane, an echo-poor focus can be seen adjacent to the apex of the lateral malleolus.

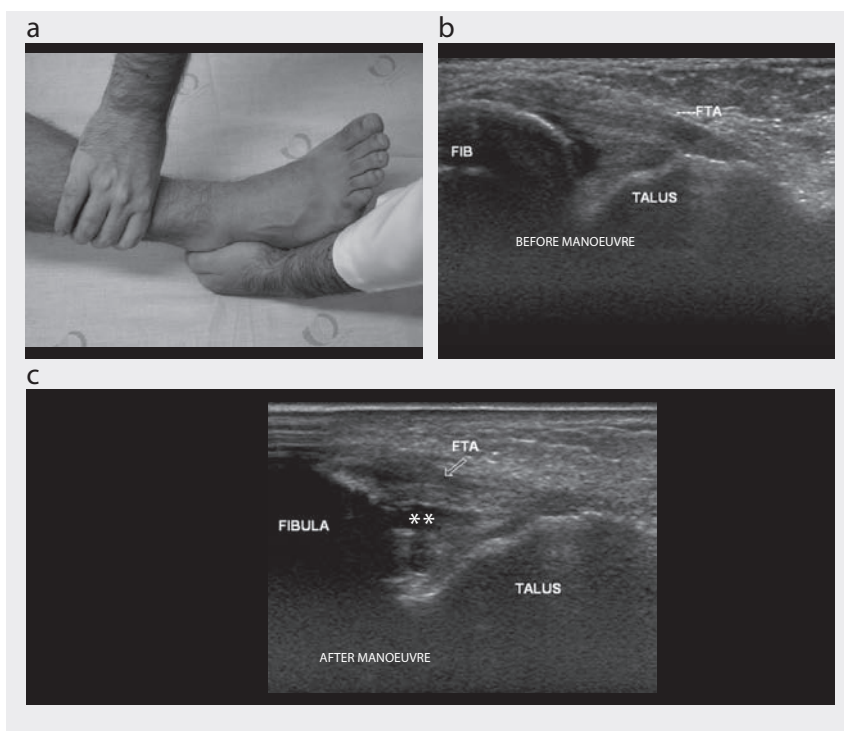
Fig. 6.56. Complete rupture (acute) of the anterior talofibular ligament (arrow) associated with fluid-debris (stars), with the remaining ligament stump adjacent to the fibula (dotted arrow)



Oedema of the soft tissue disappears during healing, which begins 7 days after a trauma. The ligament is always thickened; the first evidence of repair of a ligament, with visualization of echoes filling the discontinuities, appears about 5 weeks after a trauma. An echo-rich focus can be seen inside the scarred ligament, corresponding to calcifications, and bone irregularities are found adjacent to the insertions into the fibula and the talus as a consequence of bone avulsion.

If the scarring process does not take place appropriately, the lesion becomes chronic and may lead to instability, resulting in ligament inadequacy. Chronic lesions are characterized by lack of or significant tapering or stretching of the ligament and may be accompanied by small amounts of intra-articular fluid. In dynamic studies (drawing manoeuvre) of instability, the ligament is elongated (Fig. 6.57).

Fig. 6.57. Chronic lesion of the anterior talofibular ligament (FTA). (a) Drawing manoeuvre; ultrasonographic examination (b) before and (c) after the manoeuvre shows an elongated ligament and increased articular space, which is filled with fluid (stars)



# Muscle

Muscle is the largest individual mass of corporal tissue, corresponding to 40–45% of a person's weight. It is classified as elastic or nonelastic. Elastic muscle tissue is made up of muscle fibres joined into fascicles, which form the muscle. Nonelastic structures are made up of muscle surrounded by sheaths formed by connective tissues and muscle fasciae. The endomysium is an extensive network of capillaries and nerves involving all muscle fibres. Muscle fibres are bound into fascicles by perimysium, a fibroadipose septum made up of vessels, nerves and conjunctive and fat tissue. The epimysium, composed of dense conjunctive tissue, separates muscle venters and different muscles, such as the semimembranosus and the femoral biceps in the posterior thigh. The fascia is situated externally to the epimysium and contains a whole muscle.

Muscles may contain slow-twitch (type I) fibres rich in oxygen or fast-twitch (type II) fibres, with anaerobic metabolism. The proportion of each type of fibre inside the muscle venter is determined genetically, by type of physical training and by the location, form and function of the muscle. Posture muscles have linearly arranged fascicles, a prevalence of type I fibres and many mitochondria, allowing sustained low-energy contraction. The muscles in the superficial areas of the extremities, usually passing over more than one joint, have fibres with a pennate distribution and contain predominantly type II fibres. Muscles with these characteristics give more vigorous contractions and have a propensity to rupture.

Muscle contractions can be divided into isotonic and isometric. In isometric contractions, the length of the muscle fibre remains constant with changes in the applied load on the muscle. In isotonic contractions, the length of the muscle fibre changes, either shortening (concentric contraction) or lengthening (eccentric contraction). Usually, agonist muscles involved in a certain movement undergo concentric contraction due to the stability of the closest joint, which is determined by the eccentric contraction of the antagonist muscle, which is also responsible for slowing down the movement. This occurs, for instance, during a kick, when the stability of the knee joint is maintained by contraction of the ischiotibials, so that the femoral quadriceps can execute the movement.

## Muscle ruptures

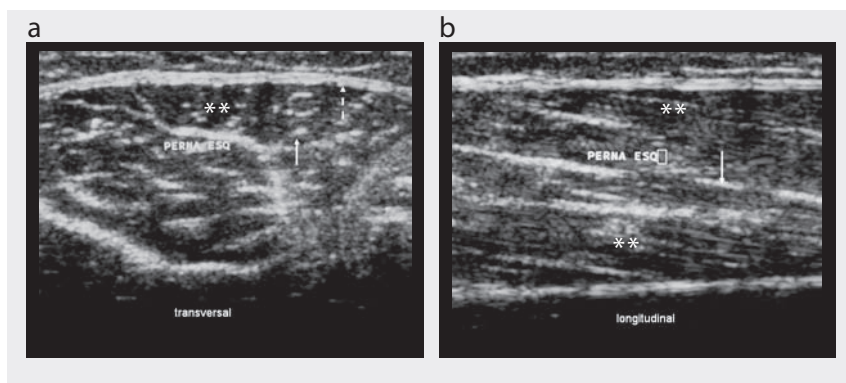
Muscle ruptures are secondary to direct or indirect trauma. Direct traumas, or contusions, involve compression of the muscle against a bone structure, so that the lesion is due to crushing. Indirect traumas are due to stretching of muscle fibres and can be generated by passive hyperextension of the fascicles, although they usually occur during eccentric contraction of the muscle.

Thus, both morphological and functional factors increase the risk for muscle lesion, the main ones being passing over more than one joint, eccentric contraction, predominance of type II fibres (quick contraction) and a superficial location at the extremities, mainly in the lower limbs. The site of the lesion depends on age and physical condition and is due to biomechanical particularities that determine weaker

areas. In the immature skeleton, lesions are usually found at the interface between tendon and bone, with a greater probability of fracture due to avulsion. In athletes and other young adults, lesions usually occur in the musculotendinous area, while in elderly people ruptures usually affect the tendon, resulting in tendinosis. When the lesion is of muscular origin, pain is restricted to the affected region, beginning immediately after the trauma. Sometimes, subcutaneous bruises can be seen 12–24 h after a trauma. If the alteration occurs in a tendon, the symptoms are diffuse and irradiated.

The approach described below is indispensable for correct interpretation of ultrasound findings. The elastic elements appear as elongated, echo-poor structures surrounded by nonelastic elements, which are echo-rich. In nonelastic structures, the endomysium is not seen on ultrasound, thereby preventing visualization of each muscle fibre. The perimysium is observed in a longitudinal section as multiple, parallel, linear, echo-rich images, separating the fascicles. Their orientation varies with the architecture of the muscle under study. In transverse section, the perimysium is seen as multiple points or irregular lines of varied lengths. The epimysium is seen as parallel, echo-rich lines external to the widest axis of the muscle and indistinguishable from the fascia (Fig. 6.58).

Fig. 6.58. Normal architecture of muscle venter. (a) Transverse and (b) longitudinal scans. Stars, muscle fascicles; arrow, perimysium; dashed arrow, epimysium and fascia



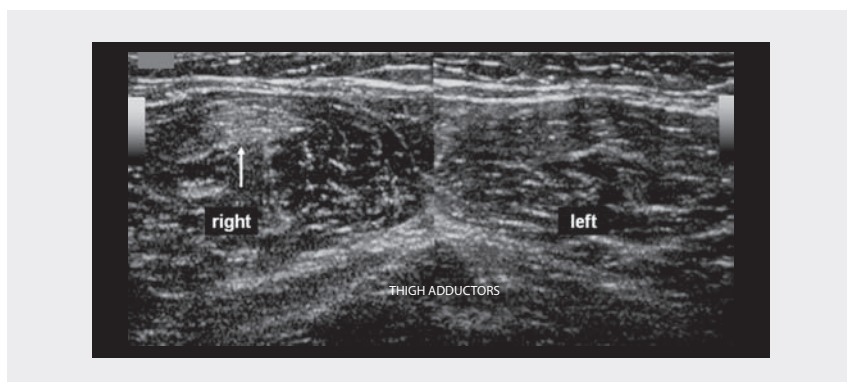
In post-trauma evaluation, ultrasound can be used for diagnosis, to identify the muscle involved, to grade the rupture or to monitor the healing process and possible complications, thus helping to predict the length of rest. A system for grading muscle lesions by ultrasound is illustrated in Table 6.2. Its clinical usefulness and inter- and intra-observer differences are, however, not yet established. In practice, the most important information for the orthopaedist is whether there is significant rupture of the muscle fibres or bruises.

In stretching and in bruises with no significant rupture of the muscle fibres, the only finding is a poorly defined echo-rich area, sometimes associated with a discreet increase in the volume of the muscle venter (Fig. 6.59). In these situations, the case

Table 6.2. Ultrasound grading of muscle lesions

Grade	Description	Ultrasound findings
0	Stretching	Normal
1	Stretching associated with lesion involving < 5% of muscle fibres	Small, striated, echo-poor images 3–7 cm long and 2–10 mm in diameter
2	Partial rupture	Discontinuity of fibroadipose septa and of muscle fascicles, associated with haematoma
3	Complete rupture	Retraction of muscle venter with formation of a pseudo-mass, accompanied by haematoma. The epimysium may be torn.

Fig. 6.59. Poorly defined, echo-rich zone (arrow) in the long adductor of the anterior right thigh, compatible with oedema secondary to stretching (grade I lesion)



history is important, as other conditions, such as denervation, myositis, late-onset muscle pain, compartmental syndrome, rhabdomyolysis and post-exercise condition, may present the same aspect.

The diagnosis must be made as soon as possible, because fluid (blood) may appear or accumulate after days or a few weeks. The earlier treatment is started, the less likely haematoma formation will be. Because the echogenicity may change in the post-exercise period, ultrasound evaluation should be conducted 2–48 h after the trauma. Examinations should be conducted during movement, at rest and during isometric contraction to help identify fibre discontinuity. Serial ultrasound examinations are used to monitor the evolution of grade II and III lesions (Fig. 6.60, Fig. 6.61), which are likely to have sequelae, especially if there is a large haematoma. Muscles have a high potential for regeneration, with cells originating in the endomysium; however, the process is slow, beginning 48 h after an acute event but taking from 3 weeks to 4 months to be completed. On ultrasound, regeneration is seen as slightly echo-rich tissue (Fig. 6.62) surrounding a haematoma, which is slowly reabsorbed. Fibroadipose septa gradually appear inside the tissue, taking the place of the rupture, so that the normal architecture of the muscle is restored.

Fig. 6.60. (a), (b) Partial muscle rupture (grade II lesion, arrow), associated with a small bruise

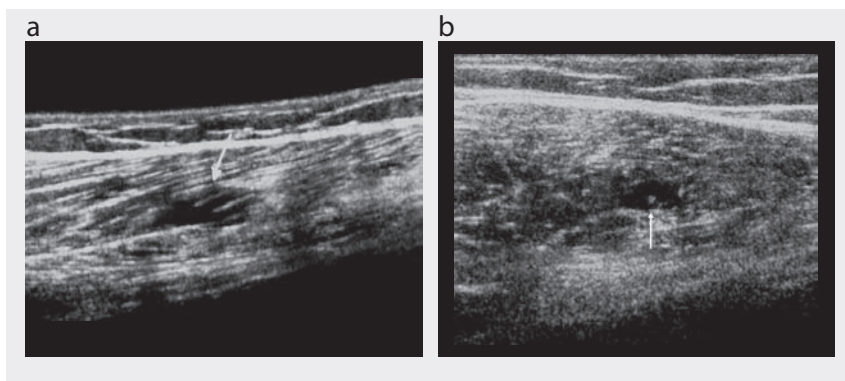


Fig. 6.61. Complete rupture (grade III lesion, arrow) in the musculotendinous transition of the long adductor of the thigh, filled with heterogeneous material (bruise). Dotted arrow, remaining tendon

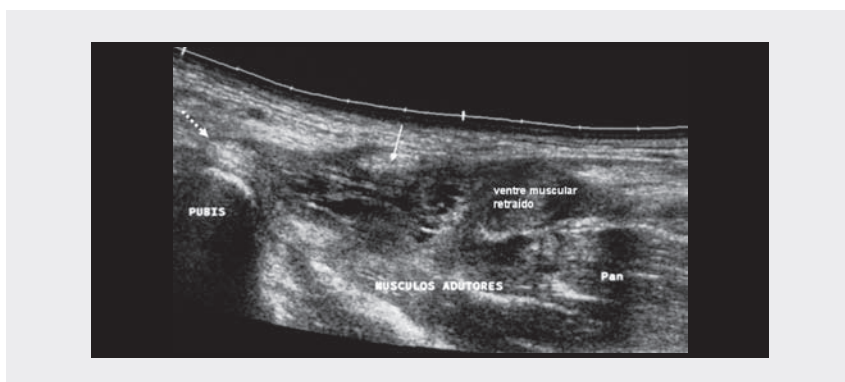
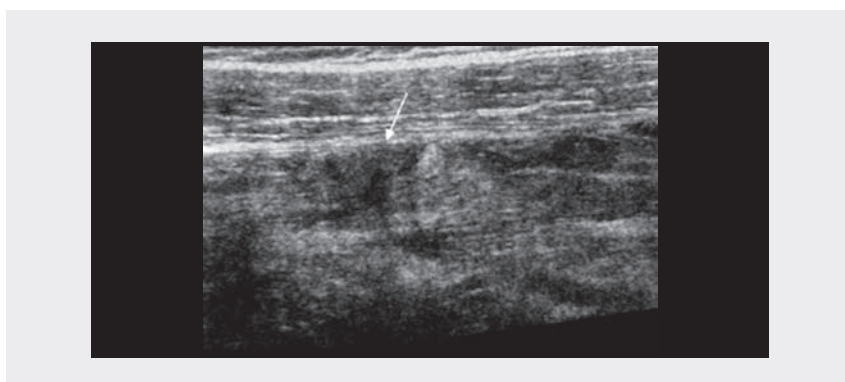


Fig. 6.62. Repairing tissue, characterized by discreetly echo-rich material (arrow), on the periphery of the subfascial partial lesion





## Rupture complications

### Acute

After severe ruptures or in patients with coagulation anomalies, **haemorrhages** may lead to compartmental syndrome.

**Rhabdomyolysis** may occur after serious trauma caused by crush, infection, hypoxia or drugs (e.g. cocaine) and secondary to metabolic alterations. It requires surgery. It is seen as an irregular, echo-poor area within the muscle, its volume being increased in areas of multiple necrosis.

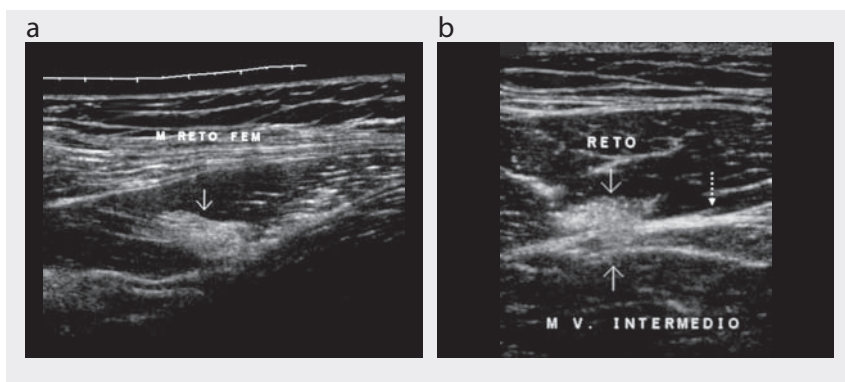
A haematoma is rarely infected to such an extent that **abscesses** are formed that require surgical drainage.

### Chronic

Most small lesions and intermuscular haematomas evolve without sequelae. Intramuscular lesions, larger lesions and recurrent lesions may lead to the appearance of fibrosis, cysts, myositis ossificans or hernia.

**Fibrosis:** In large ruptures, repair of the muscle involves two processes: regeneration of muscle fibres and formation of fibrosis. When the latter predominates, an irregular, focal, echo-rich or radiated area can be seen on ultrasound (Fig. 6.63), frequently adhering to the epimysium and sometimes resulting in focal retraction of the fascia. It remains unchanged during muscle contraction manoeuvres. The presence of fibrous scarring predisposes the muscle to recurrent ruptures.

Fig. 6.63. Ultrasonography of the femoral rectum muscle. (a) Longitudinal, (b) transverse plane. Fibrosis, characterized by an echo-rich zone (arrow) with partially clear edges, located inside the femoral rectum muscle venter and entering the vastus intermedius through a discontinuity of the muscle fascia (dotted arrow)



**Muscle cyst** is a rare complication and is due to incomplete resorption of a haematoma (Fig. 6.64). It also favours new muscle rupture.

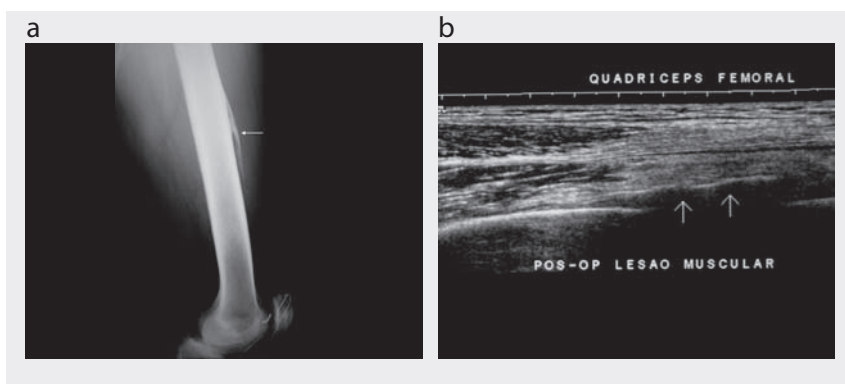
**Myositis ossificans** is usually the result of a lesion caused by direct trauma, with formation of an intramuscular haematoma, or by repeated microtraumas, mainly



Fig. 6.64. Intramuscular cyst (cisto) in the lateral vastus, secondary to previous rupture



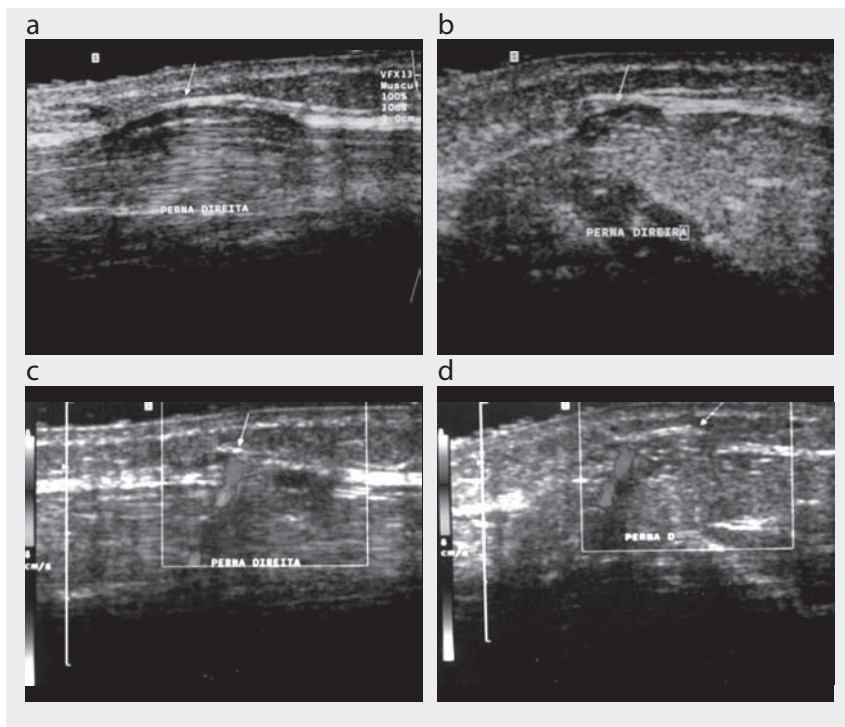
Fig. 6.65. Ossifying myositis. (a) X-ray and (b) ultrasound, showing lamellar calcification parallel to the femoral diaphysis cortex (arrow)



in athletes. The calcifications, which are initially lamellar, evolve to real heterotopic ossification, seen as linear, echo-rich images parallel to the adjacent cortical bone. Myositis ossificans is frequently situated inside the femoral quadriceps, particularly in the femoral rectum (Fig. 6.65).

**Muscular hernia** is a condition in which muscle tissue protrudes through a discontinuity or weakness of congenital or acquired fascia. The commonest causes are chronic compartmental syndrome, trauma and postoperative alterations. On ultrasound, the hernia is seen as a clearly defined nodular image in a mushroom form, its echogenicity depending on the stage of evolution. Initially, due to its proximity to fibroadipose septa, the nodule is echo-rich; afterwards, it becomes echo-poor (Fig. 6.66) due to the presence of oedema. A dynamic examination is essential, as the hernia may be fixed or intermittent, the latter being apparent only on isometric contraction of the muscle. Diagnostic sensitivity is also increased by conducting the

Fig. 6.66. Muscle hernia. (a), (b) Ultrasound and (c), (d) colour Doppler, showing an echo-rich nodular form (arrow) entering an existing defect in the fascia of the anterior leg, accompanied by a perforating vessel



examination after exercise: a muscle hernia is more obvious during exercise, with increased local blood flow and the consequent increase in muscle volume (10–15%).

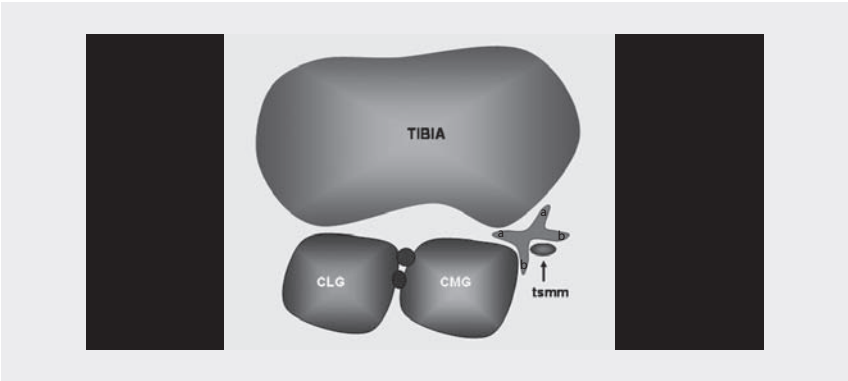
## Other disorders

### Baker cyst

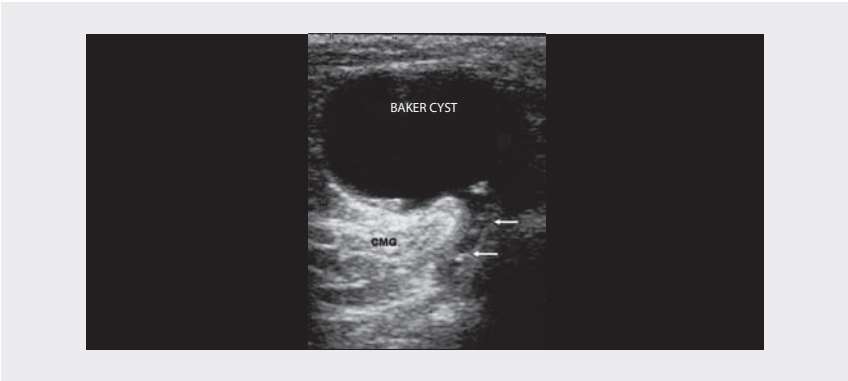
Baker cyst, initially described by Adams in 1840 and by W. Morant Baker in 1877, is the commonest synovial cyst in the human body. The synovial bursa of the gastrocnemius and semimembranosus connects with the knee joint in 50% of normal adults, and degeneration and reduced elasticity of the joint capsule in older people might explain the high prevalence of articular problems. Baker cyst arises from lesions of the synovia or any intra-articular process that leads to fluid formation, resulting in distension of the gastrocnemius and semimembranosus bursa. This condition, which is extremely common in people with rheumatoid arthritis, is characterized by a cystic body with echo-free contents, located medially in the popliteal fossa,

between the tendon of the semimembranosus muscle and the medial head of the gastrocnemius. The bursa of these two muscles has four horns—two anterior (medial and lateral) and two posterior (medial and lateral)—which may be filled with fluid, either separately or together (Fig. 6.67, Fig. 6.68). Thus, although a Baker cyst is situated in the medial area of the popliteal fossa, it can vary slightly in location and form, sometimes with extension into the muscle planes and even into the vastus medialis, and gastrocnemius muscles.

**Fig. 6.67.** Bursa of the gastrocnemius and semimembranosus muscles, with the two anterior (a) and the two posterior (b) horns. CLG, lateral head of the gastrocnemius; CMG, medial head of the gastrocnemius; tsmm, semimembranosus muscle tendon

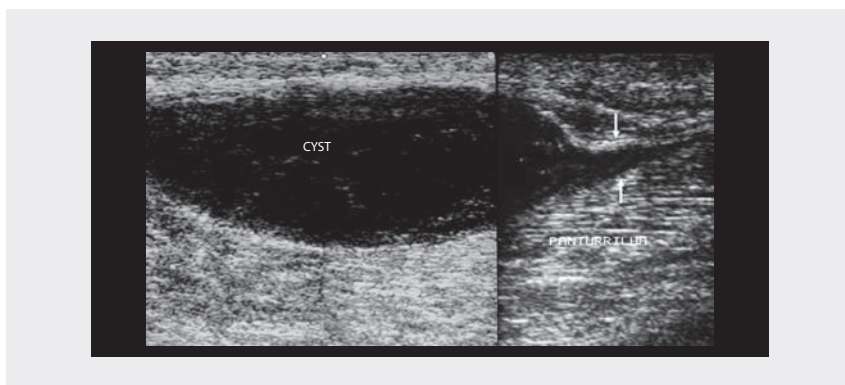


**Fig. 6.68.** Baker cyst, showing communication with the articular cavity (arrows); CMG, medial head of the gastrocnemius muscle



Parietal thickening, free bodies, septations and internal echoes are observed in cases of haemorrhage, infection or arthropathy caused by crystal deposits, sometimes with formation of a fluid–fluid level. The cysts may sometimes rupture, with acute pain, simulating deep-vein thrombosis. This can readily be diagnosed with ultrasound as loss of definition of the cyst wall, with fluid diffusing through the muscle and subcutaneous planes, associated with oedema of soft tissue (Fig. 6.69).

Fig. 6.69. Rupture of Baker cyst: heterogeneous content and septae due to undefined inferior wall, with perifascial free fluid (arrow)

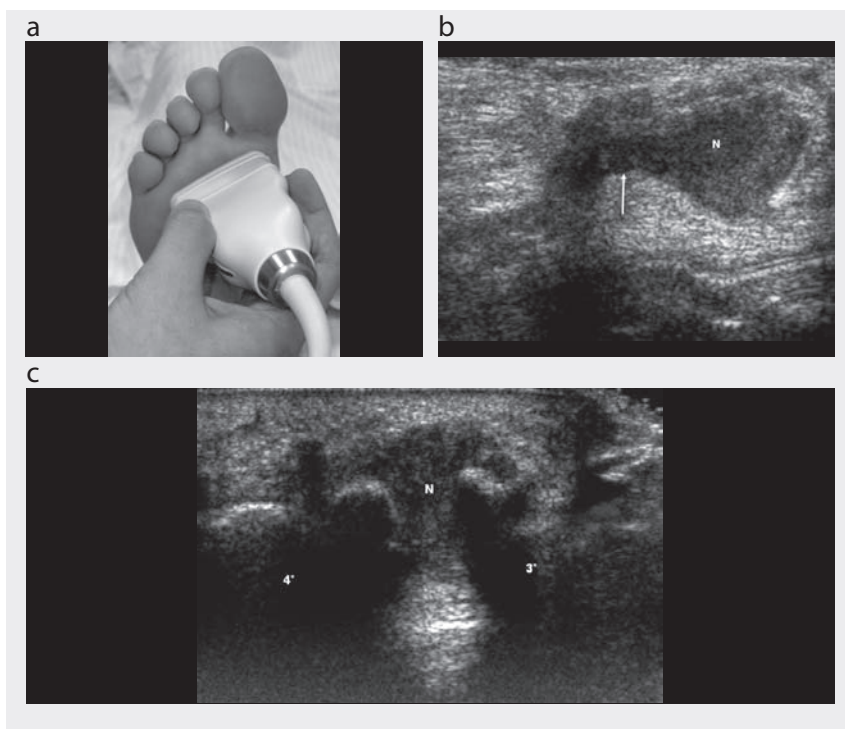


## Morton neuroma

Morton neuroma is a thickening of the interdigital nerve, usually in the third intercapitometatarsal space. Its cause is uncertain but is probably related to repetitive trauma or ischaemia resulting in neural imprisonment. It is prevalent in women aged 40–60 years and can be symptomatic or asymptomatic. When it is symptomatic, it leads to pain and paraesthesia, which worsens with walking. It is unilateral in 73–90% of cases.

On ultrasound examination, Morton neuroma is seen as an echo-poor nodule between the metatarsal heads, plantar to the transverse metatarsal ligament. Its diagnosis is confirmed when there is continuity with the interdigital nerve (Fig. 6.70), as other conditions, such as neurofibroma, schwannoma, angioliipoma or angioleiomyoma, may have a similar ultrasonographic aspect. Neuromas can be accompanied by intermetatarsal bursitis, which can also occur separately, characterized by increased fluid (> 3 mm), compressibility in dynamic manoeuvres and a location superficial to the deep transverse metatarsal ligament.

Fig. 6.70. Morton neuroma. (a) Examination technique. (b) An echo-rich nodule (N) seen in the longitudinal and (c) transverse planes, situated in the plantar area of the third space between the third and fourth metatarsal heads, with thickening of the interdigital nerve (arrow)



## Plantar fasciitis

Plantar fascia, or aponeurosis, originates in the posteromedial tuberosity of the calcaneus and has medial, central and lateral sections. The central section is the strongest and thickest (2–4 mm), with five bands in the middle part of the metatarsals. Inflammation or degeneration of the central section of the plantar fascia (fasciitis) is the commonest cause of pain in the plantar area of the calcaneus, corresponding to 7–9% of all lesions in runners. Other conditions can result in the same symptoms, including stress fractures of the calcaneus, tarsal tunnel syndrome, seronegative arthropathies and neuritis. Microruptures in the fascia are due to repetitive traction microtraumas, which lead to inflammation and angiofibroblastic proliferation, as observed in tendinosis. The predisposing factors include systemic diseases (rheumatoid arthritis, gout and spondyloarthropathy), splayfoot, concave foot and ill-fitting shoes.

Fig. 6.71. Plantar fascia. (a) Examination technique. (b) Ultrasound scan showing a striped, echo-rich image, starting in the inferior tuberosity of the calcaneus (calipers)

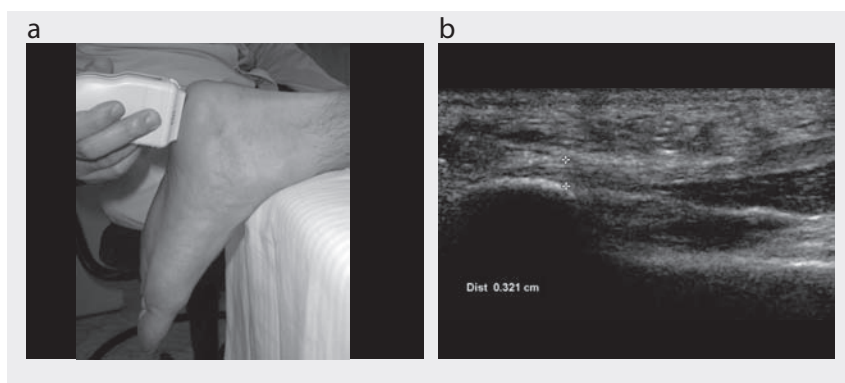
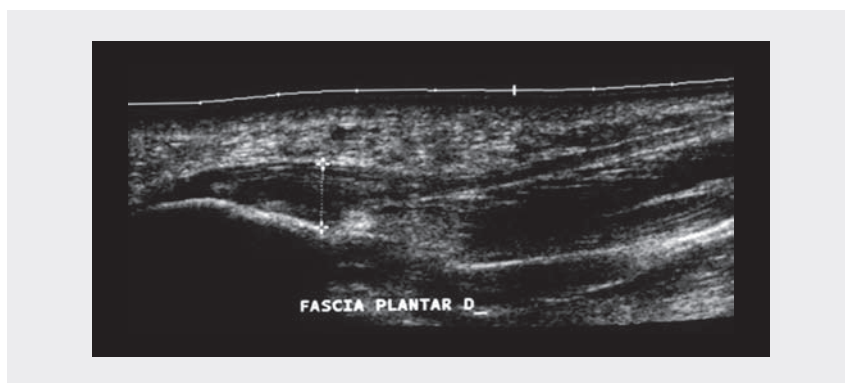


Fig. 6.72. Plantar fasciitis. Thickening and hyperechogenicity of the plantar fascia at its insertion into the calcaneus (calipers)



On ultrasound, the normal fascia presents a fibrillar aspect (Fig. 6.71), except in patients with discreet hypoechogenicity near the calcaneus due to an anisotropic effect. In fasciitis, there is some thickening ( $> 5$  mm) and reduced echogenicity of the fascia, usually close to its insertion into the calcaneus, and some calcification (Fig. 6.72). Bilaterality is not uncommon. Ultrasound can, however, lead to false-negative results.

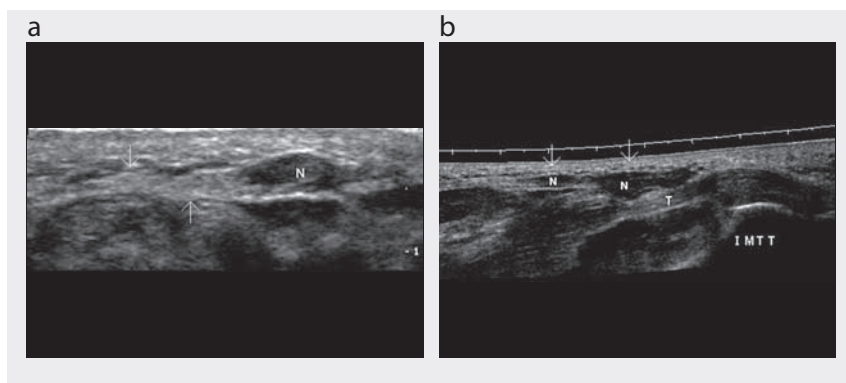
The commonest findings with MRI in suspected plantar **fasciitis**, in decreasing order of frequency, were perifascial oedema, oedema of the calcaneus medullary bone, signal alteration inside the fascia and thickening of the plantar fascia. Thus, if the **fasciitis** is slight, it is not seen by ultrasound; nor can bone oedema be seen by this technique.

## Superficial fibromatosis

Superficial fibromatosis is due to proliferation of benign fibrous tissue, with aggressive biological behaviour. Palmar (Dupuytren contracture), plantar (Ledderhose disease) and penile (Peyronie disease) fibromatoses are part of a spectrum of the same disease, although they may occur separately.

In Ledderhose disease, there is some thickening, with a nodular aspect and reduced echogenicity, beginning in the area of the plantar cavum (Fig. 6.73). Isolated nodules must be differentiated from granulomas and from rheumatoid nodules.

Fig. 6.73. Plantar fibromatosis. Echo-rich nodules (N) with well-defined outlines inside the plantar fascia (arrows) in (a) transverse and (b) longitudinal planes



The ultrasound aspect of Dupuytren contracture is similar to that of palmar fibromatosis, extending from the third to the fifth finger. It is prevalent in middle-aged or elderly men, alcoholics and patients with epilepsy who have taken phenobarbital for long periods. Patients report repeated microtrauma in the area. The nodules tend to converge over time, forming fibrous strings, with consequent retraction or palmar aponeurosis.

## Compressive neuropathies: Carpal tunnel syndrome

Compression of the middle nerve inside the carpal tunnel is the most frequent peripheral compressive neuropathy and that most easily treated. The syndrome is characterized by paraesthesia or pain on the palmar face, from the first to the radial half of the fourth finger, associated with weakness and atrophy of the thenar musculature in the most advanced cases.

More than half a century elapsed between Paget's description of its symptoms in 1854 and full understanding of the syndrome. The diversity of clinical aspects of compression of the middle nerve led to a certain confusion in characterization of this syndrome, which partly explains this relatively long period.



Usually, when peripheral nerves pass over a joint, they also pass over osteofibrous tunnels, with a risk for neural displacement during movement. As the tunnels are relatively inelastic, however, they are vulnerable to compressive neuropathy. The physiopathology of carpal tunnel syndrome has been the subject of much speculation. Nerve compression can be due to anatomical, intrinsic or mechanical factors.

The anatomical factors are related to conditions that determine a decrease in the dimensions of the carpal tunnel (acromegalia, wrist bone alterations and alterations of the distal radius) or an increase in the content of an osteofibrous tunnel (tumours, anomalous muscle venters, synovitis or haematomas). The intrinsic factors include neuropathy secondary to diabetes mellitus, alcoholism, amyloidosis, infections, gout or tenosynovitis and situations that alter the water balance, such as pregnancy, use of oral contraceptives, hypothyroidism or long periods of haemodialysis. The mechanical factors vary from repeated flexion and extension movements to excess weight on the extended carpal tunnel in patients who use a cane or a crutch.

The process starts with modification of the microcirculation, with a decrease in epineural capillary flow. As the pressure increases, epineural, endoneural and arteriolar capillary flow is reduced. This leads to endoneural oedema, associated with increased capillary permeability, resulting in macrophage migration. These inflammatory cells produce cytokines, which cause proliferation of the fibrous tissue, involving the neural sheath and the axon itself, culminating in axonal degeneration and demyelination.

If the causal factor is small and of short duration, the alterations are reversible; however, if the compression persists and becomes more intense, irreversible lesions can form, creating a vicious circle and resulting in persistent symptoms or symptoms generated by submaximum effort. The first symptoms are paraesthesia and hyperaesthesia, as the middle nerve is made up mainly (94%) of sensitive fibres. As the disease develops, motor fibres become involved, leading to weakness and atrophy of the thenar musculature.

Ultrasound criteria for diagnosis of carpal tunnel syndrome are a reduction in the echogenicity of the middle nerve due to oedema, accompanied by tapering in the distal carpal tunnel and an increase in its upstream area (Fig. 6.74).

These authors not only described qualitative alterations to the median nerve but also established quantitative criteria for the diagnosis of carpal tunnel syndrome. Hardening of the median nerve in the proximal carpal tunnel, at the level of either the distal radius or the pisiform bone, was evaluated by measuring the area of the nerve in transverse section. Tapering of the median nerve in the distal carpal tunnel in the hamate bone is measured in transverse section as the ratio between the largest and smallest axes of the median nerve (tapering ratio). Thin tapering corresponds to a ratio  $> 3$ . Cambering (incurvation, arching) of the retinaculum of the flexors is evaluated as the distance between the top of the flexor retinaculum and an imaginary line drawn between the trapeze and the hamate. Values  $> 4$  mm are considered abnormal. The most useful criterion for a diagnosis of compressive neuropathy is an



Fig. 6.74. Thickened, echo-rich median nerves, with a reduced number of neural fascicles to the right, replaced by echo-rich tissue corresponding to adipose tissue and fibrosis

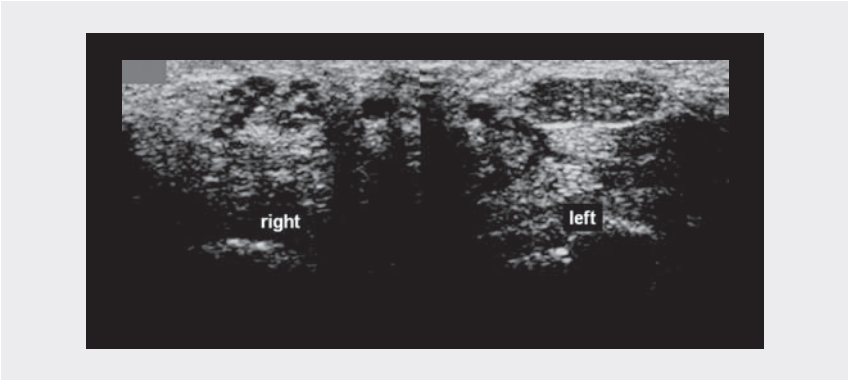


Fig. 6.75. Measurement of the area of the median nerve ( $0.07 \text{ cm}^2$ ) using (a) direct and (b) indirect methods. ESC, scaphoid; PIS, pisiform; CG, Guyon channel

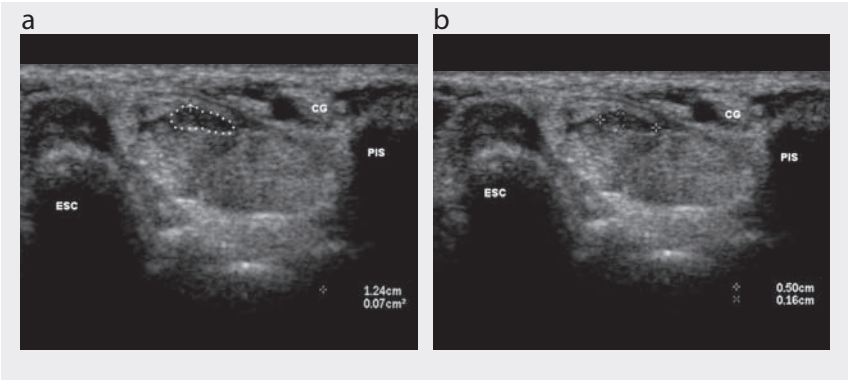
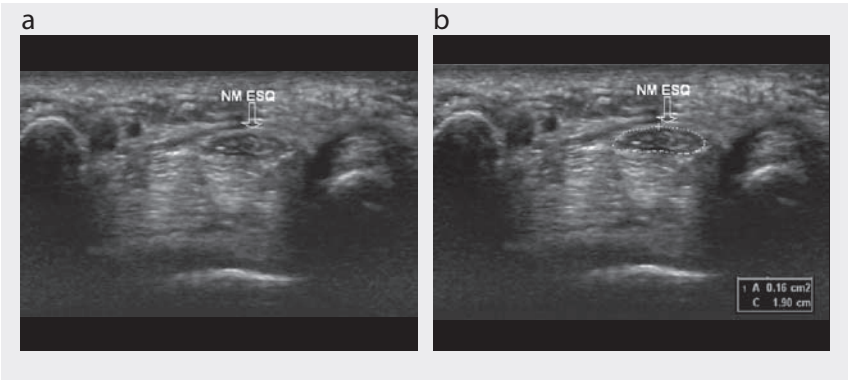


Fig. 6.76. Thickened median nerve (NM) inside a carpal tunnel (a) with a transverse section of  $0.16 \text{ cm}^2$  (b).



increase in the cross-sectional area of the median nerve. Distal tapering of the nerve and incurvation of the retinaculum of the flexors showed poor reproducibility in subsequent studies.

The cross-sectional area of the median nerve can be measured either indirectly or directly. In the indirect method, the formula for the area of the ellipse [ $\pi(D_1 \times D_2) / 4$ ] is used, in which  $D_1$  and  $D_2$  represent the transverse and anteroposterior diameters of the median nerve (Fig. 6.75 a). In the direct method, the area is calculated by ultrasound, from a continuous trace around the nerve (Fig. 6.75 b). Regardless of the method used, the neural sheath must always be excluded from the measure.

The cut-off point of the cross-sectional area for differentiating between normal and thickened nerves has been the subject of controversy in the literature, suggestions varying from 9 to 15 mm<sup>2</sup>. This wide variation is due to the use of different equipment, inclusion of people of both sexes in the same study, studies of people of different ages, different severity of disease and imprecise measurement area. Each unit should establish its own value on the basis of the population being studied. For women, we have adopted cross-sectional area cut-off points of 9 mm<sup>2</sup> measured by the indirect and 10 mm<sup>2</sup> measured by the direct method (Fig. 6.76).



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