Musculoskeletal Ultrasound

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Chapter 10

Inflammatory Joint Diseases

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INTRODUCTION

Ultrasound is increasingly used as a diagnostic modality in joint imaging by radiologists and rheumatologists. Several factors explain this development. Technical advances have improved image quality, simplified equipment, reduced cost, and improved diagnostic capability. New, highly effective treatments have become available that control joint inflammation and prevent or retard long-term joint damage, but early diagnosis of pre-destructive soft tissue changes is critical and best achieved by magnetic resonance imaging (MRI) or ultrasound. With the exception of pre-erosions (subcortical bone edema) and assessment of articular cartilage, MRI and ultrasound are equally effective in the diagnosis of inflammatory arthropathy.[1] Ultrasound is more operatordependent than MRI and cannot always access all parts of a joint (e.g., between metacarpal heads). However, ultrasound has better spatial resolution than MRI in superficial areas, can be used to scan multiple joints and sites quickly, and readily distinguishes effusion from synovitis without needing intravenous contrast. Ultrasound is now viable as a diagnostic tool for office-based practice and for this reason has been adopted by many rheumatologists in their routine practice.

In this chapter we will present the ultrasound technique and criteria for examining patients with arthropathies, and discuss initial diagnosis and follow-up. We will discuss the ultrasound findings in conditions that target the synovial membrane (e.g., rheumatoid arthritis, juvenile idiopathic arthritis, and crystal arthropathies); inflammatory arthropathies that target both entheses and synovial membrane (spondyloarthopathies); and other arthropathies (osteoarthritis, pigmented villonodular synovitis, synovial osteochondromatosis, amyloid, septic arthritis, and hemophiliac arthropathy). Finally, we will describe ultrasound-guided interventions.

Although the cost of ultrasound equipment has reduced considerably in real terms, high-quality equipment remains essential. High-frequency (>10 MHz) linear array transducers are essential, although larger joints may need curvilinear transducers and lower frequencies. Small footprint transducers are useful for the small joints of the hands and feet. A standoff is not required, but copious jelly helps. Beam steering and compound imaging are also useful. Joints should be relaxed when examined, and transducer pressure should be light or fluid may be displaced and missed or vascularity effaced.

Tip:

Use light transducer pressure or fluid or hyperemia may be missed.

New developments are being assessed for possible roles in joint disease: 3D imaging (possible quantification of the volume of inflamed synovium, regional vessels, or erosions); intravenous contrast agents (improved visualization of vascularity); elastography (compressibility of soft tissues); and fusion imaging (direct correlation of ultrasound to computed tomography [CT] and/or MRI). As yet, no practical applications for these techniques have been established in routine rheumatological practices.

Standardization of technique and diagnoses has been facilitated by the adoption of a number of consensus definitions[2]:

Synovial fluid: Intra-articular material that is displaceable and compressible and does not exhibit Doppler signal. It is usually anechoic (Fig. 10.1) or hypoechoic but may be hyperechoic.

Synovial hypertrophy: Abnormal intra-articular tissue that is not displaceable and is poorly compressible. It may/may not exhibit Doppler signal and is usually hypoechoic (Figs. 10.2 and 10.3).

Tenosynovitis: Thickened tissue (+/− fluid) within a tendon sheath seen in two perpendicular planes. It may/may not exhibit Doppler signal and is usually hypoechoic (Figs. 10.4 and 10.5).

Bone erosion: An intra-articular discontinuity in the bone surface that is visible in two perpendicular planes (Fig. 10.6).

Enthesopathy: Abnormally hypoechoic and/or thickened tendon or ligament at its bony attachment with loss

of the normal fibrillar pattern seen in two perpendicular planes. Echogenic foci due to calcification, Doppler signal, and bony changes (due to enthesophytes, erosions, or irregularity) may be present.

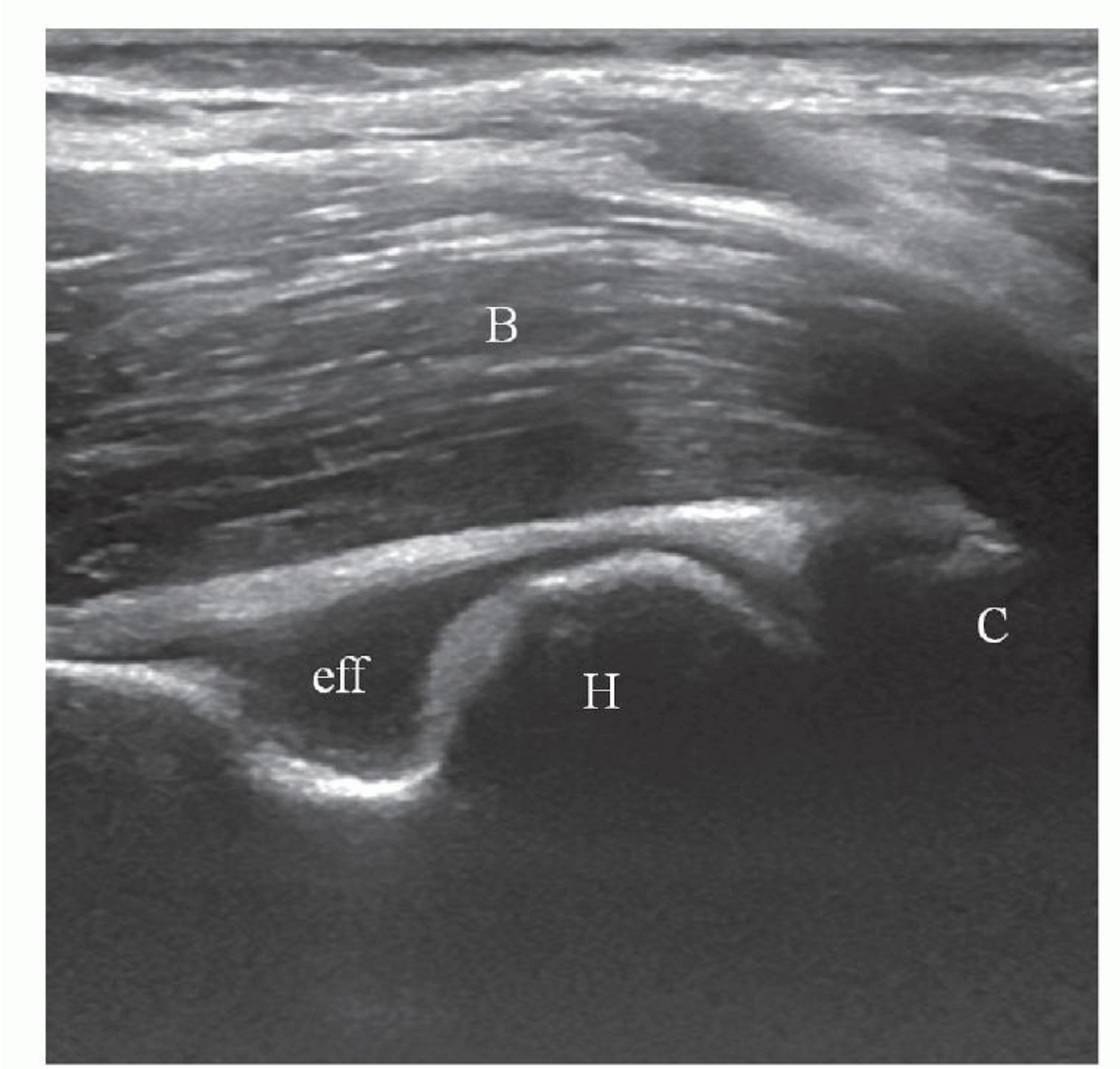

Figure 10.1. Longitudinal scan of anterior elbow. Anechoic effusion (eff) in anterior recess. B, brachialis muscle; C, coronoid process of ulna; H, humerus.

In clinical practice, the diagnosis of "active arthritis" (or clinical synovitis) is based primarily on clinical evaluation, but it is often difficult to determine whether

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joint pain, swelling, and/or limitation of mobility are caused by synovitis, tenosynovitis, enthesitis, or periarticular edema. Ultrasound has become a valuable adjunct in rheumatology. Ultrasound examination of joints demonstrates with high accuracy most of the anatomical structures involved: synovial recesses, articular/periarticular bone contours, ligaments, tendons, and tendon sheaths (including the bony insertions or entheses of ligaments and tendons), and bursae. Ultrasound can demonstrate inflammatory changes (hyperemia, synovial thickening, or effusion in joint recesses, bursae, or tendon sheaths) and destructive changes (bone erosions, joint destruction or ankylosis, cartilage loss, and tendon tears).

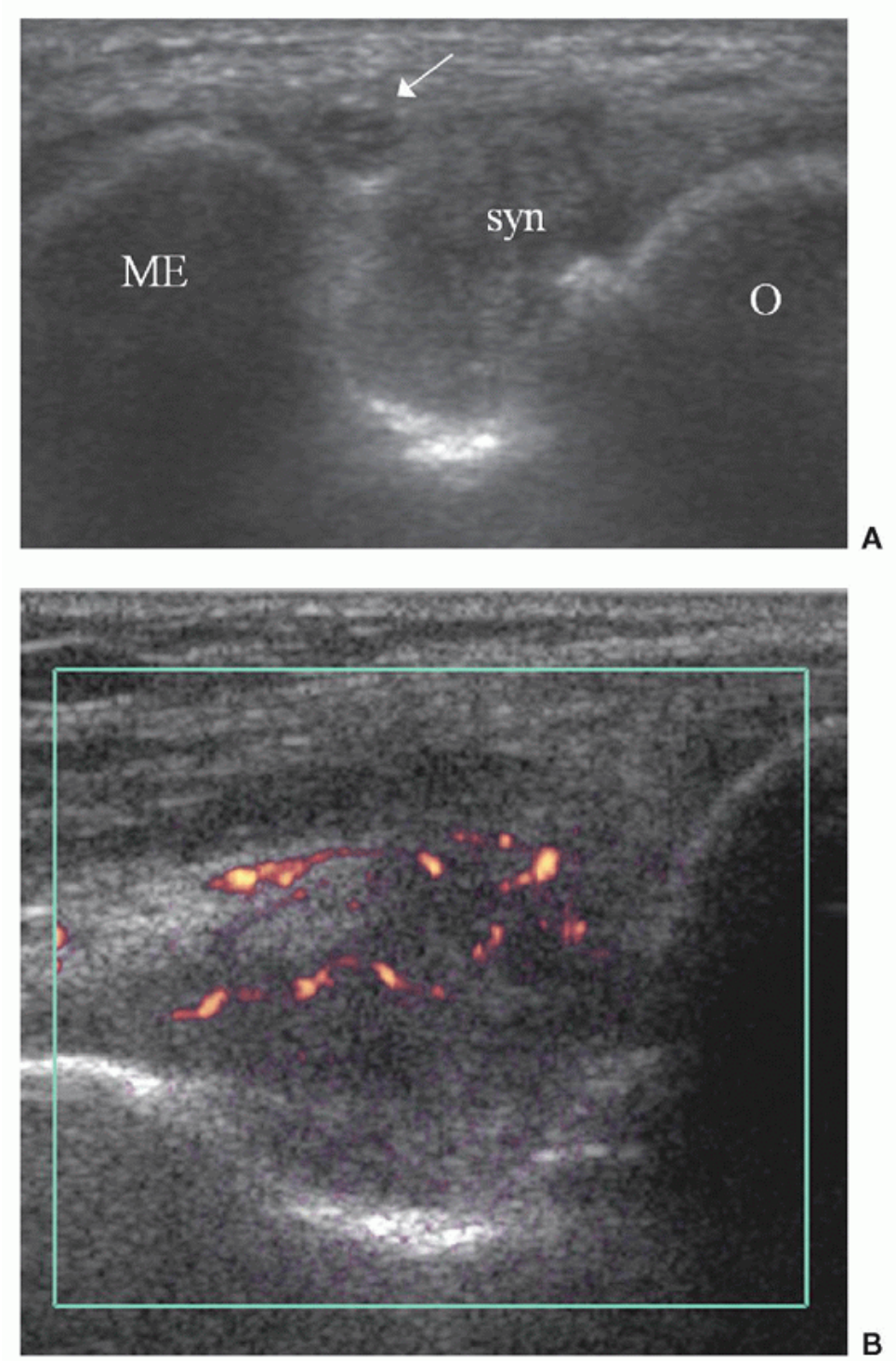

Figure 10.2. Patient with synovial hypertrophy (syn) in elbow. A: Transverse posteromedial ultrasound scan showing displacement of ulnar nerve (arrow). O, olecranon; ME, medial epicondyle of humerus. B: Power Doppler examination shows synovial hyperemia.

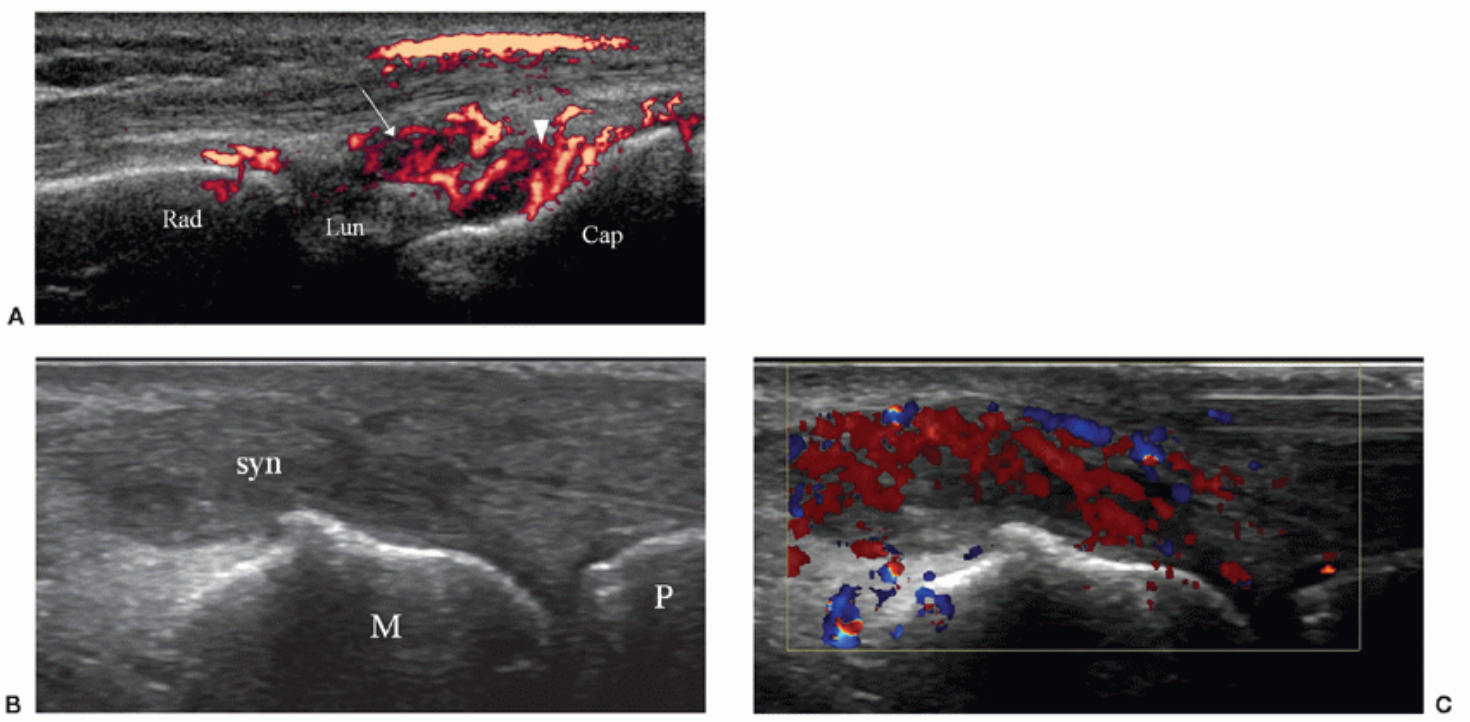

Figure 10.3. Rheumatoid arthritis patients. A: Dorsal longitudinal ultrasound scan of the wrist shows synovial thickening in the recesses of the radiocarpal (arrow) and midcarpal (arrowhead) joints. Synovial hyperemia on power Doppler examination confirms active synovitis. Rad, radius; Lun, lunate; Cap, capitate B: Dorsal longitudinal ultrasound scan of the third MCP joint shows synovial thickening (syn) extending from the joint line and along the metacarpal head (M). P, proximal phalanx. C: Synovial hyperemia on color Doppler examination confirms active synovitis.

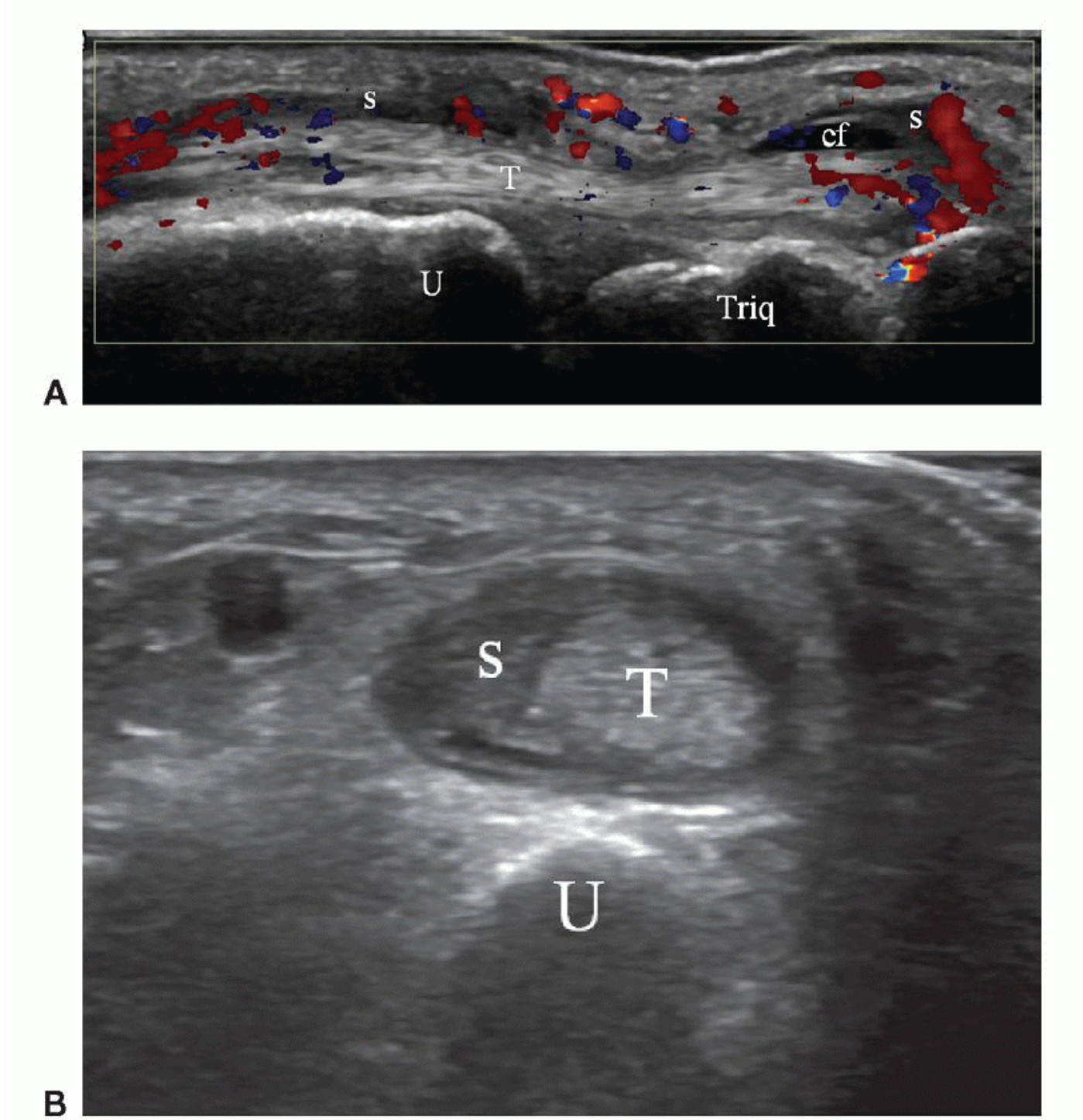

Figure 10.4. Longitudinal (A) and transverse (B) scans of the extensor carpi ulnaris tendon (T) at the wrist of a patient with RA and tenosynovitis. The tendon is slightly enlarged. Effusion (ef) and synovial thickening (S) in the tendon sheath. Color Doppler (A) shows tendon and synovial hyperemia. U, ulna; Triq, triquetrum.

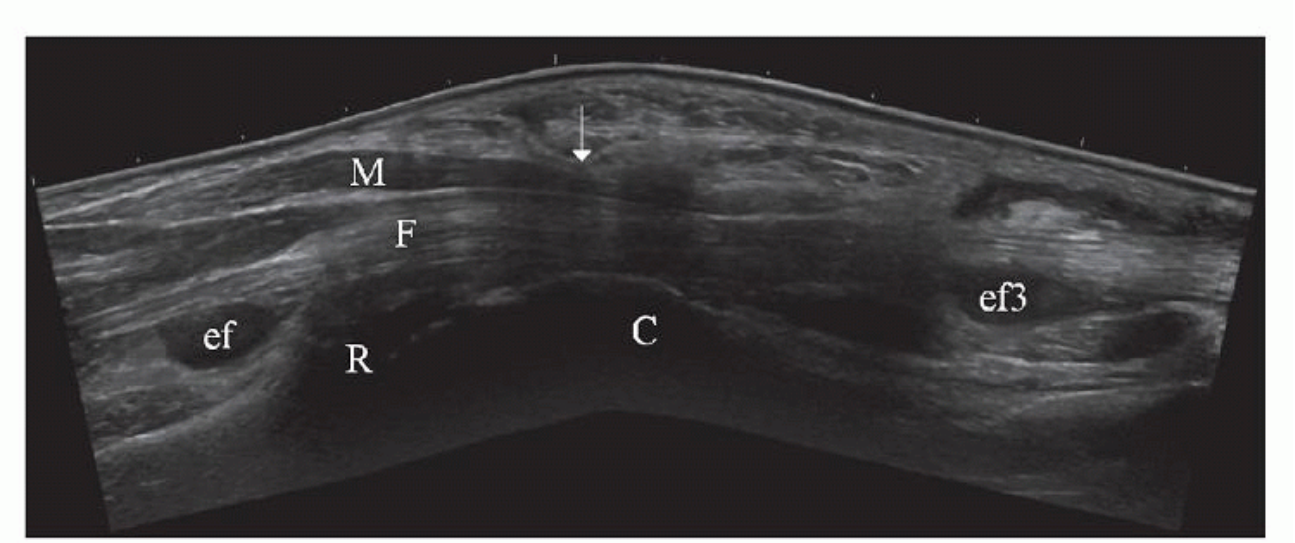

Figure 10.5. Tenosynovitis of the flexor digitorum tendons of the long finger (F). Longitudinal ultrasound scan shows effusion in the flexor tendon sheath proximal (ef) and distal (ef3) to the carpal tunnel. Median nerve is enlarged proximal to the carpal tunnel (M) and compressed under the flexor retinaculum (arrow). R, radius; C, carpus.

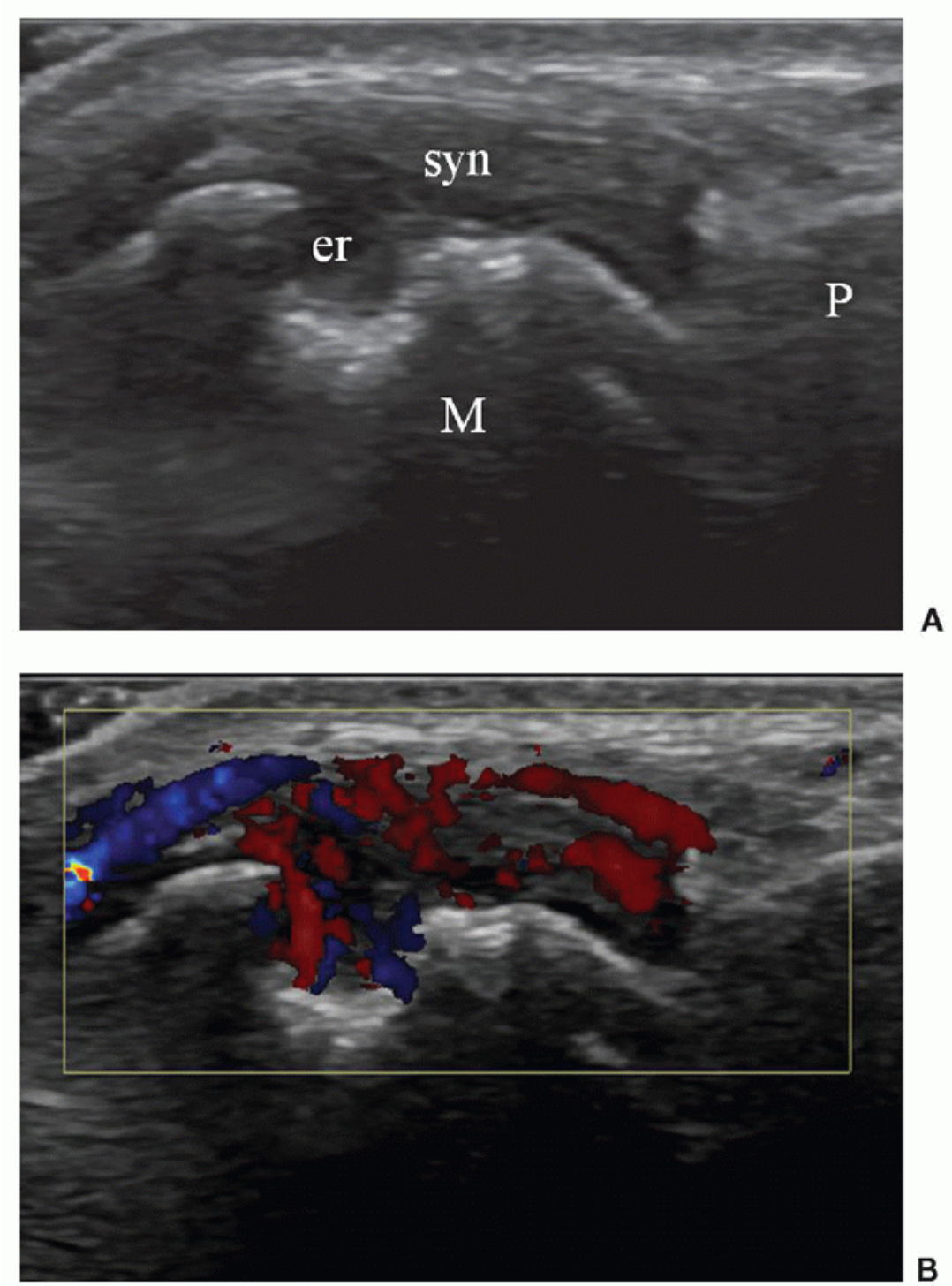

Figure 10.6. Dorsal longitudinal scans of second MTP joint in patient with RA. A: Synovial hypertrophy (syn) with erosion of metatarsal head (er). M, metatarsal head; P, proximal phalanx. B: Synovial hyperemia on color Doppler examination confirms active synovitis.

Most of the early studies that described the ultrasound findings in inflammatory arthropathy were performed on patients with rheumatoid arthritis (RA), but these findings are not specific and can be found in most types of joint diseases.

EXAMINATION TECHNIQUE

In general, joints are best examined with long-axis scans to show the joint recesses. Tendon sheaths are best studied in short axis when the transducer can be moved proximally and distally, employing an “elevator” technique. Bursae and Baker cysts are shown in two planes. Typical sites of synovitis include the ulnar aspect of the

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wrist, the metacarpophalangeal joints (MCPJs) especially the dorsal margins, and the suprapatellar recess of the knee.

Hand and Wrist

The hand/wrist is the area most frequently examined for inflammatory arthropathy. Ultrasound anatomy and technique have been described in Chapter 5 and will not be repeated in detail here, but a systematic approach to the examination is essential. The thumb

and interphalangeal joints are not usually included unless there is a specific indication. By moving the transducer slowly, the entire examination can be performed as a combined grayscale and Doppler procedure.

Start by examining the dorsal aspects of the second to fifth MCPJs with the hand pronated, fingers extended, and the transducer placed longitudinally (Fig. 10.7). Flexion at the wrist or MCPJs reduces the conspicuity of both grayscale and Doppler evidence of synovitis.[3] With grayscale and Doppler gain and focal zone suitably adjusted, examine each joint in turn by moving the transducer slowly backward and forward from medial to lateral. At the second and fifth MCPJs, respectively, continue the movement to examine the radial and ulnar aspects of the joints.

Tip:

The wrist and MCPJs should be extended when examined to optimize conspicuity of synovitis.

Now place the transducer transversely on Lister tubercle to examine the second to fifth extensor compartments before internally and externally rotating the wrist to examine the sixth and first extensor compartments, respectively. In each position, an “elevator” technique is employed by moving the transducer to and fro, proximally and distally. The transducer is then rotated through 90 degrees on the dorsal aspect of the carpals and again moved backward and forward, from radial to ulnar, to examine the wrist and midcarpal joints. The hand is then supinated and the transducer placed transversely across the carpal tunnel and moved proximally and distally looking for evidence of flexor tenosynovitis. Finally, the volar aspects of the second to fifth MCPJs are examined in similar fashion to the dorsal aspects.

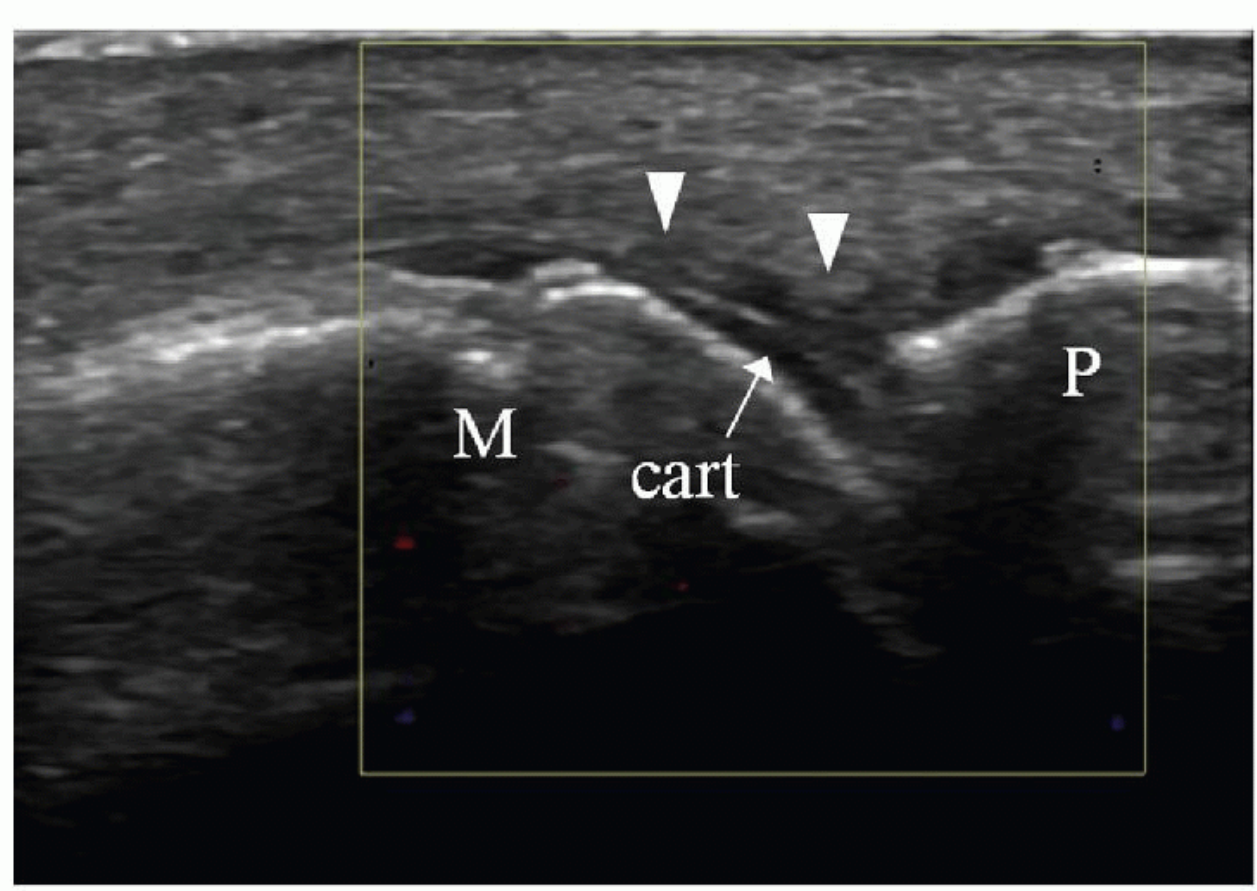

Figure 10.7. Dorsal longitudinal scan of normal second MCP joint showing normal capsule (arrowheads) and no synovial thickening. M, metacarpal head with anechoic cartilage (cart); P, proximal phalanx.

Foot and Ankle

A similar approach to the hand/wrist is used. The dorsal and plantar aspects of the metatarsophalangeal (MTP) joints and the dorsal midfoot are examined with the transducer placed longitudinally. The medial, peroneal, and anterior tendons at the ankle are examined transversely using a proximal and distal elevator technique, and the anteromedial and lateral gutters are also examined transversely.

Other Joints

Although long-axis scans of joints are usually preferred, short-axis scans can be useful, for example, in looking for fluid or synovitis in the medial and lateral pouches of the suprapatellar recess of the knee.

ULTRASOUND FINDINGS IN INFLAMMATORY JOINT DISEASES

Synovitis

Detection of synovitis is important because synovitis precedes bone erosions. Ultrasound is better than clinical examination and radiographs, and comparable to MRI in detecting synovitis.[1] Synovial hypertrophy and joint fluid are both frequently present (Fig. 10.8). Normal synovium is so thin it is not seen with ultrasound. Synovial hypertrophy produces solid, noncompressible, thickened hypoechoic tissue in joints and tendon sheaths.[2],[4],[5] The echogenicity of the thickened synovium is inversely proportional to its water content: the more fluid that is present, the less echogenic the synovium. A semiquantitative grading system

(normal/mild/moderate/large) for synovial hypertrophy may be employed.[6] Joint or tendon sheath fluid is usually anechoic or hypoechoic, and compressible. A complex or echogenic appearance is due to proteinaceous fluid, crystals, or debris. Even in large and easily palpable joints such as the knee, ultrasound is more sensitive in detecting fluid than clinical examination or radiographs.[7],[8] Volumes as small as 1 mL

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and interobserver agreement of 79% can be found in small joints of the hands and feet.[8]

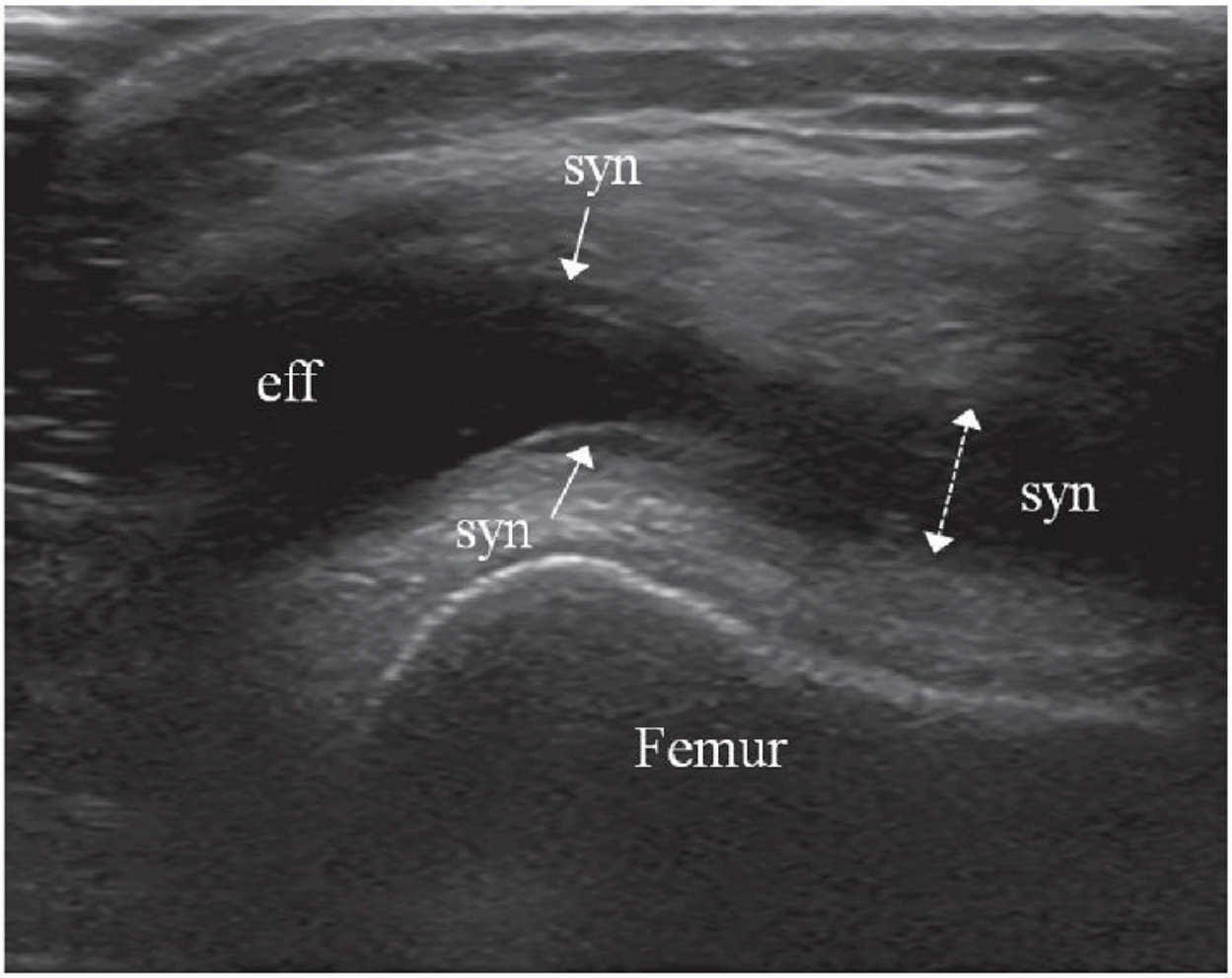

Figure 10.8. Transverse scan of suprapatellar recess of knee. Transducer pressure on right of image displaces fluid to left and distinguishes effusion (eff) from synovial hypertrophy (syn).

In patients with doubtful hypoechoic or anechoic areas in superficial joints, compression with the transducer[9] distinguishes between synovial hypertrophy (poorly compressible, non-displaceable) and effusion (compressible, displaceable) (Fig. 10.8). This does not work for deep joints like the hip.

Tip:

Joint fluid is compressible and displaceable. Synovial thickening is poorly compressible and non-displaceable.

Synovial hypertrophy occurs in both acute (inflammation) and chronic (fibrosis) stages of inflammatory joint disease, and it is not possible to distinguish between them using grayscale ultrasound. Doppler techniques (color or power Doppler) are therefore widely employed for diagnosis and follow-up of arthritic joints.

Tip:

Use Doppler to distinguish between active and chronic (inactive) synovitis.

Doppler techniques detect tissue hyperemia, which is associated with disease activity (Figs. 10.2 and 10.3). Some authors use power Doppler. Depending on the sensitivity of the ultrasound equipment, others prefer color Doppler. Synovial hyperemia on Doppler ultrasound correlates well with contrast enhancement on MRI[10],[11] and microvascular density on histology.[12] Synovial tissue may be seen extending into erosions (Fig. 10.6), sometimes with vascular signal,[13] probably related to erosion activity (Fig. 10.6). Fluid does not show Doppler signal and this helps to distinguish active synovitis from fluid. Light transducer pressure is essential as even moderate pressure may efface vessels and flow.

Intravenous injection of ultrasound contrast has been used to increase the sensitivity of Doppler examinations.[10],[14],[15] However, the sensitivity of current generation ultrasound machines has improved dramatically, even without contrast enhancement, and with high-end units it is now possible to display physiological joint flow.[16] The use of intravenous ultrasound contrast increases the cost

and time of the examination, and is invasive. Contrast is not used in routine clinical practice although it has been shown to improve the diagnosis of sacroiliitis.[17]

Doppler findings[5] can be reported in three different ways: qualitative assessment, semiquantitative grading, and quantitative assessment. Qualitative assessment is a simple report of the presence or absence of synovial flow. Semiquantitative scoring is the most often used technique in daily practice[10],[13] and employs a grading scale depending on the severity of findings.[1] Quantitative assessment[18],[19] is time-consuming but is more precise and is used for trials. It is performed by calculating the number of pixels showing power Doppler signal or by calculating the resistive index from spectral Doppler measurements. Future ultrasound systems may provide automatic measurements.

Bony Changes

Ultrasound of articular and periarticular bone surfaces can display erosions (Fig. 10.3), which are important signs in aggressive arthritis such as RA. Erosions are not disease-specific and can occur in other types of inflammatory joint disease. Small bone defects resembling erosions can be found in healthy controls.[20] Erosions typically occur in the bare area of a joint (i.e., the intracapsular cortex that is not covered by hyaline cartilage) and are seen as focal cortical defects identified in two perpendicular planes. Active erosions may show Doppler signal (Fig. 10.6).

Tip:

Erosions usually occur at the bare area of a joint.

Ultrasound is more sensitive than radiographs and comparable to MRI in detecting erosions in fingers[21],[22] or toes.[1] The specificity of ultrasound is lower than that of CT, probably because small cortical defects occur as normal findings. Some studies have found ultrasound to be less sensitive in detecting erosions than MRI,[23] possibly because of inaccessibility (e.g., on the lateral and medial margins of metacarpal and metatarsal heads or at the wrist joint).[1],[22] A drawback of ultrasound is that it cannot detect bone marrow edema, which has been

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shown to be an important prognostic factor on MRI. Bone erosions in early undifferentiated arthritis increase the risk of developing persistent arthritis.[23]

Cartilage Changes

Normal hyaline or articular cartilage forms a hypoechoic layer that covers the bright surface of the subchondral bone plate. Its thickness can be measured by ultrasound.[9] The superficial surface of normal cartilage is seen as a smooth, thin echogenic line. In inflammatory and degenerative joint diseases, nonspecific destructive cartilaginous changes can be detected with diffuse or focal thinning (Fig. 10.9), irregularity of the surface, or defects.

Tenosynovitis

In tenosynovitis, hypoechoic or anechoic fluid and/or synovial proliferation are seen around the tendon in the tendon sheath. Synovial hypertrophy may be nodular and show abnormal Doppler signal. Tendinopathy (with or without tenosynovitis) is defined by intratendinous changes: hypoechoic thickening best demonstrated on transverse scans, and disorganization of the fibrillar tendon structure best seen on longitudinal scans. Paratendinitis is an inflammatory thickening of the para-tendon, which can be pronounced in some arthritides (RA, crystal or infectious arthritides, and juvenile idiopathic arthritis). Tendon tears are defined by areas of interruption of tendon fibers and may be partial (transverse or longitudinal with tendon split) or complete (transverse tears). In aggressive inflammatory joint diseases such as RA, synovial tissue can enter tendon erosions, similarly to bony erosions.

Enthesitis

Enthesopathy refers to a pathological condition at the bony attachment of tendons, ligaments, capsule, or fascia, and can be mechanical or secondary to an inflammatory process (enthesitis). Enthesitis is characteristic of sero-negative spondyloarthropathies such as ankylosing spondylitis and psoriasis. Signs of inflammation in the spondyloarthropathies are more frequent at insertion sites than in the synovial membrane of joints or tendon sheaths. This is the opposite of RA[24] and is useful when considering the differential diagnosis, although a specific diagnosis cannot be achieved with ultrasound alone.

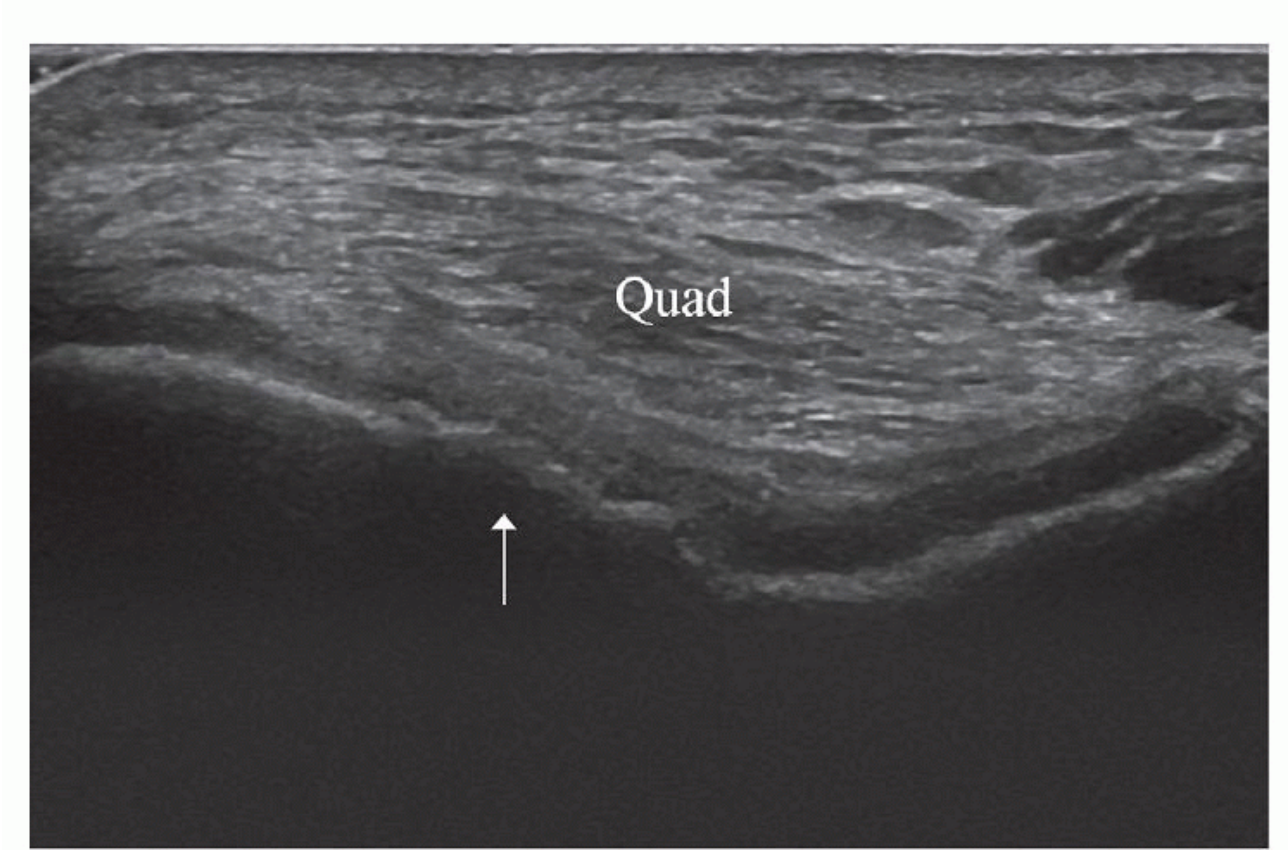

Figure 10.9. Transverse scan of femoral trochlea with knee in full flexion. Cartilage thinning and irregular hyperostosis are present on lateral aspect of trochlea (arrow). Quad, Quadriceps tendon.

Tip:

Enthesitis is characteristic but not pathognomonic of seronegative spondyloarthropathy.

The presence of inflammation at insertion sites is difficult to assess clinically. Radiographs show bony changes (enthesophytes, bony irregularity, and erosions) and tendon calcification at a late stage, often only after many years of symptoms.[25] Ultrasound and MRI can demonstrate the early soft tissue signs of inflammation. Ultrasound shows the soft tissue part of insertion sites in greater detail than MRI, especially at small structures, and is a helpful adjunct to clinical examination. Ultrasound shows focal hypoechoic thickening of the tendon insertion in two orthogonal planes, sometimes with hyperemia, calcifications, or bony changes.[2] Adjacent bursae may also be inflamed (e.g., enthesitis of the Achilles tendon and retrocalcaneal bursitis). As ultrasound can show only the surface of bone at an enthesis, local bone edema is not detectable, and this is a disadvantage compared with water-sensitive MRI sequences (STIR, fat-suppressed T2-weighted) or fat-suppressed contrast-enhanced T1-weighted sequences.[26] However, only large calcifications or spurs can be seen on MRI, whereas ultrasound shows even small enthesophytes.

Tip:

Enthesitis typically results in the thickening of a tendon/ligament +/− hyperemia on Doppler or hyperostosis.

Bursitis

Most peripheral bursae are accessible to ultrasound. Bursitis is diagnosed when a bursa is enlarged with fluid and synovial thickening or completely filled with synovial tissue (Fig. 10.10). Doppler signal may be present.

Bursal fluid is often anechoic but inflammation, infection, blood, and crystals may result in echogenic fluid. Bursae occur at many sites, for example, the olecranon bursa at the elbow, the retrocalcaneal bursa at the ankle, the subacromial bursa of the shoulder and around the knee.

In patients with swelling and/or pain of the knee and/or popliteal fossa, a Baker cyst is easily diagnosed at ultrasound by demonstrating a comma-shaped cyst with its characteristic neck between the medial head of

gastrocnemius and the semimembranosus tendon. The cyst may be filled with solid synovial tissue in inflammatory joint disease. Ultrasound identifies other causes of popliteal masses (tumor, aneurysm, ganglion cyst, hematoma, and abscess) and distinguishes between ruptured Baker cyst, when fluid is seen tracking along fascial planes, and deep venous thrombosis in patients with acute leg swelling.

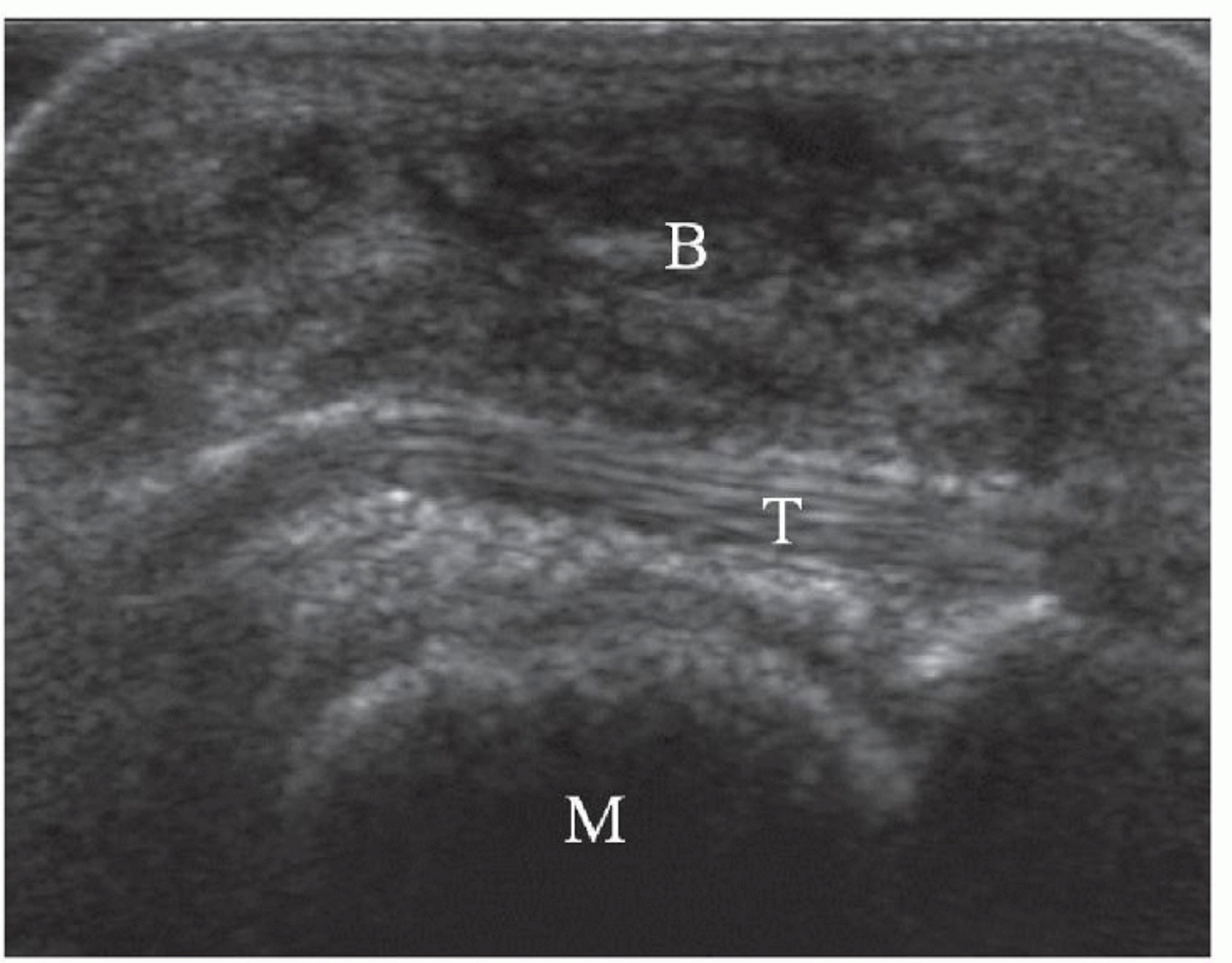

Figure 10.10. Rheumatoid bursitis superficial to first metatarsophalangeal joint. Longitudinal ultrasound scan shows enlarged hypoechoic bursa (B) superficial to flexor hallucis longus tendon (T) and first metatarsal head (M).

Adventitial bursae occur at sites of mechanical friction, often related to sports or occupation. They fill with fluid and do not have a synovial lining.

INFLAMMATORY JOINT DISEASES

Rheumatoid Arthritis

RA is a chronic, symmetric autoimmune disease characterized by joint inflammation, destruction of small and large joints, and extra-articular manifestations. Until 2010 it was diagnosed according to the American College of Rheumatology (ACR) 1987 Classification Criteria[27] based on clinical assessment, immunological markers (presence of rheumatoid factor), and conventional radiography.

The new 2010 European League Against Rheumatism (EULAR)/ACR Classification Criteria for RA[28] allows ultrasound to supplement the clinical examination. By showing the extent of joint involvement, the status of the disease may change from a mono- or oligoarthritis to polyarthritis,[29] thus potentially changing the diagnosis and instigating potent treatments. Only ultrasound signs of inflammation/synovitis play a role. No place is given to evidence of joint destruction although ultrasound has high sensitivity for the detection of bone erosions in small joints,[1,21,30] particularly in early disease.[22]

Typical symmetrical involvement of the wrist joints (most often around the styloid process of the ulna) (Fig. 10.11) and the small joints of the extremities can be easily detected with ultrasound (MCP-, MTP- and proximal interphalangeal [PIP]-joints). Tenosynovitis and possible tendon ruptures can also be detected (extensor carpi ulnaris and other extensor tendons, flexor digitorum tendons with carpal tunnel syndrome) (Fig. 10.12). Ultrasound shows all changes in joints affected by RA with the exception of bone edema and bone/cartilage damages in areas not accessible by ultrasound. Detection of joint effusion, synovial thickening, synovial hyperemia/inflammation with Doppler techniques, tendon pathology such as inflammation, fluid in tendon sheaths and bursae, tendon rupture, cartilage thinning, and bone erosions are all possible with ultrasound.

Rheumatoid Nodules

Rheumatoid nodules are areas of fibrinoid necrosis surrounded by fibrous tissue seen in up to 25% of patients with RA, usually with active disease and high levels of rheumatoid factor. Nodules are usually subcutaneous

and occur over bony prominences such as the olecranon process but can occur anywhere. Ultrasound shows a well-defined

relatively hypoechoic mass often with a hypoechoic central area due to necrosis.[31] Nodules in or adjacent to tendons may catch on retinacula or pulleys causing triggering.

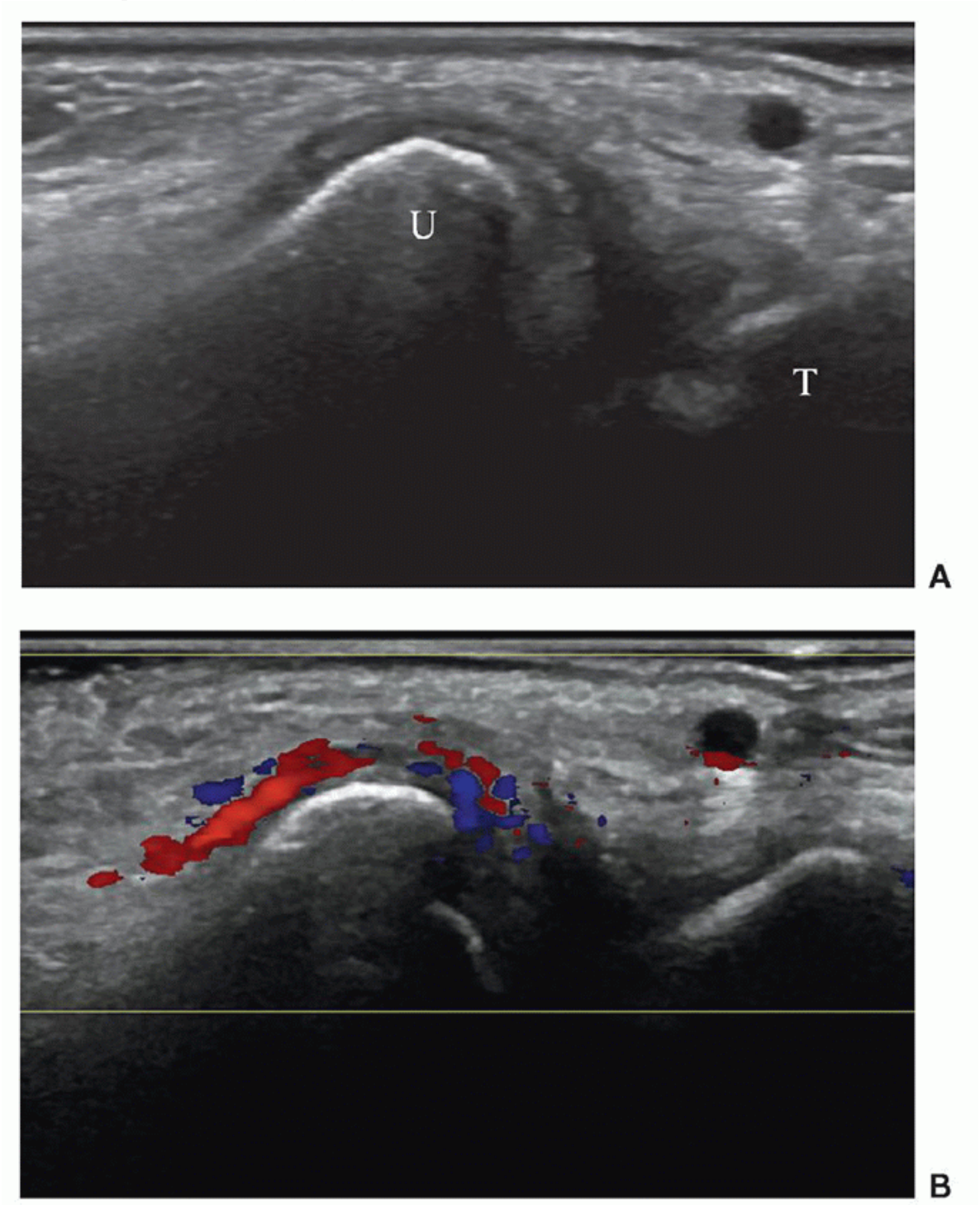

Figure 10.11. Rheumatoid arthritis patient. A: Hypoechoic synovial hypertrophy on ulnar aspect of wrist and around ulnar head (U). T, triquetrum. B: Synovial hyperemia around ulnar head on color Doppler examination is a sign of disease activity.

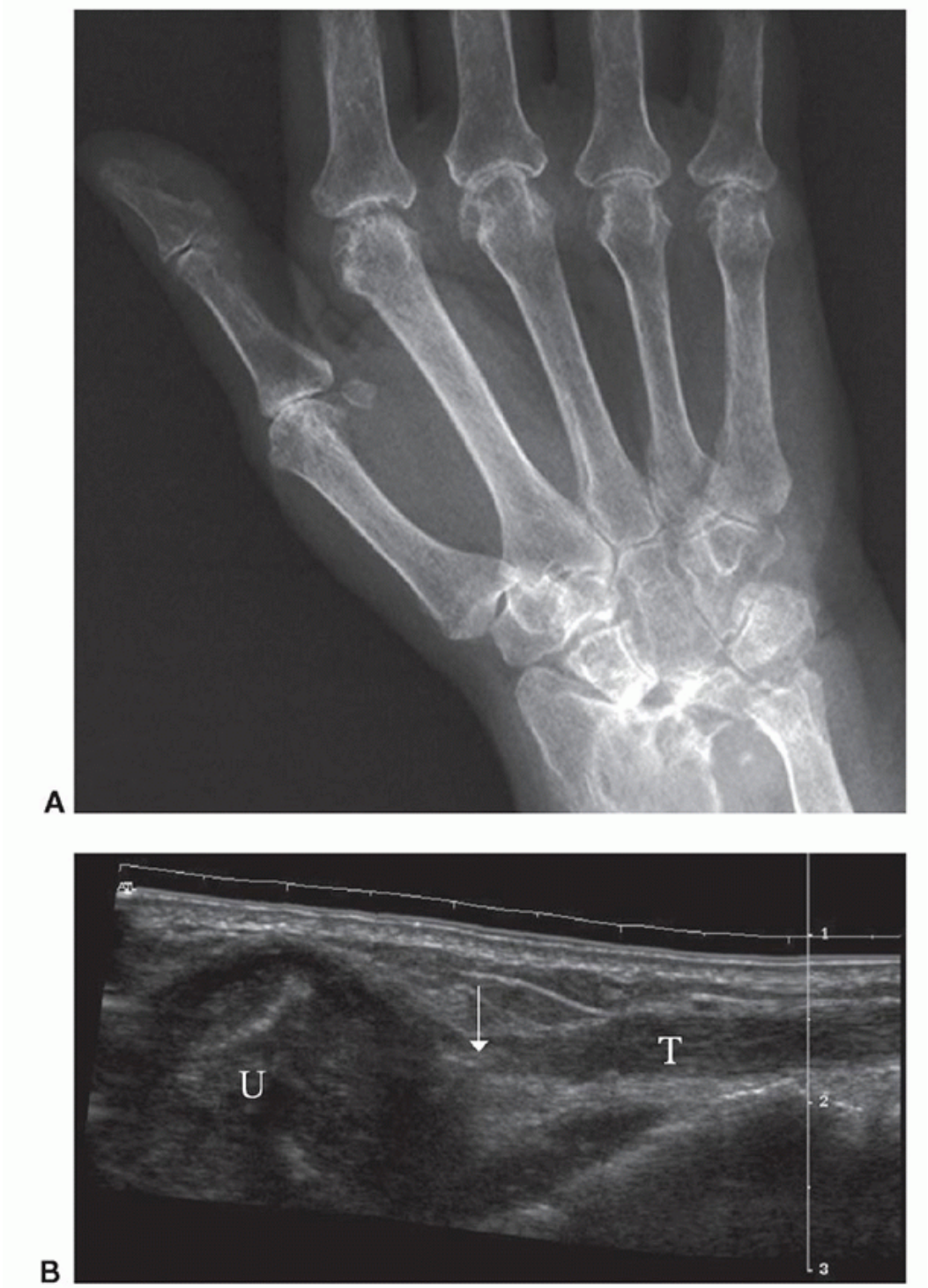

Figure 10.12. Patient with rheumatoid arthritis. A: radiograph of wrist shows advanced erosive arthropathy. B: Ultrasound shows complete tear (arrow) of extensor digiti minimi tendon (T). U, Ulnar head surrounded by hypoechoic synovitis.

Assessment of Response to Treatment

Ultrasound is widely used for follow-up of inflammatory arthropathy. Most studies have been on patients with RA. Clinical examination is insensitive at detecting low-level active synovitis[32] although subclinical synovitis is important and predicts subsequent radiographic deterioration[29],[33] if not treated. Therapeutic efficacy can be monitored by ultrasound, which shows reduced synovial thickening and Doppler signal after intra-articular steroid injections and systemic treatment with anti-TNF,[34],[35],[36],[37],[38] and the resistive index may increase.[19] Ultrasound also has potential for follow-up of erosive changes. Ultrasound detects more erosions than radiographs but fewer than MRI in the MCP and PIP joints of patients with RA.[21],[39] Ultrasound can detect complications during treatment (e.g., soft tissue infection, tendon tear, and deep vein thrombosis).

Prognosis

The role of ultrasound in the prognosis of RA has not been fully examined, but demonstration of synovial power Doppler signal in RA joints predicts radiographic progression.[40],[41] The grade of synovial hypertrophy, independent of Doppler signal, can play a role in predicting radiographic progression of RA.[33]

Spondyloarthropathies

Spondyloarthropathies are a group of diseases with common features characterized by chronic joint inflammation, strong association with HLA B-27, predilection for the axial skeleton (spondylitis, sacroiliitis) and insertion sites (enthesitis), and lack of association with rheumatoid factor (seronegativity). Conditions include ankylosing spondylitis, psoriatic arthritis, reactive arthritis, enteropathic arthritis, and undifferentiated spondyloarthropathy. Enthesitis is characteristic but not pathognomonic. Peripheral joint involvement is typically an asymmetric oligoarthritis with associated enthesitis, predominantly affecting the lower limbs. Extra-articular signs include anterior uveitis and mucocutaneous lesions. The prevalence of all types of spondyloarthropathies has been reported between 0.5% and 1.9%. The diagnosis of spondyloarthropathy, regardless of etiology, is generally based on the European Spondyloarthropathy Study Group (ESSG) criteria,[42] which include radiological sacroiliitis but not early ultrasonographic soft tissue changes.

Ultrasound of Spondyloarthropathy

The main sites of spondyloarthropathy, the spine and sacroiliac joints, are poorly visualized with ultrasound, although peripheral signs of enthesitis, synovitis, tenosynovitis, bone erosions, and cartilaginous changes can be displayed by ultrasound, especially inflammatory and destructive changes at the calcaneal insertion of the Achilles tendon (Fig. 10.13). Ultrasound monitoring of enthesitis during treatment can show significant improvement of the inflammatory changes.[43]

Ankylosing Spondylitis

Ankylosing spondylitis is the most common spondyloarthropathy, with a prevalence of up to 0.2%. Initial symptoms are insidious, typically in young adults, more often men than women, with axial involvement (sacroiliitis and spondylitis). The diagnosis is based on clinical,

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radiographic, and biochemical features but is often delayed. Ultrasound can show peripheral asymmetric oligoarthritis (hip, shoulder, and lower limbs) and enthesitis (heel, iliac crest, and anterior chest wall). The hand is not frequently involved. The prognosis is variable, but the disease has a tendency toward ankylosis of the spine and may lead to substantial functional loss and musculoskeletal or ophthalmologic complications.

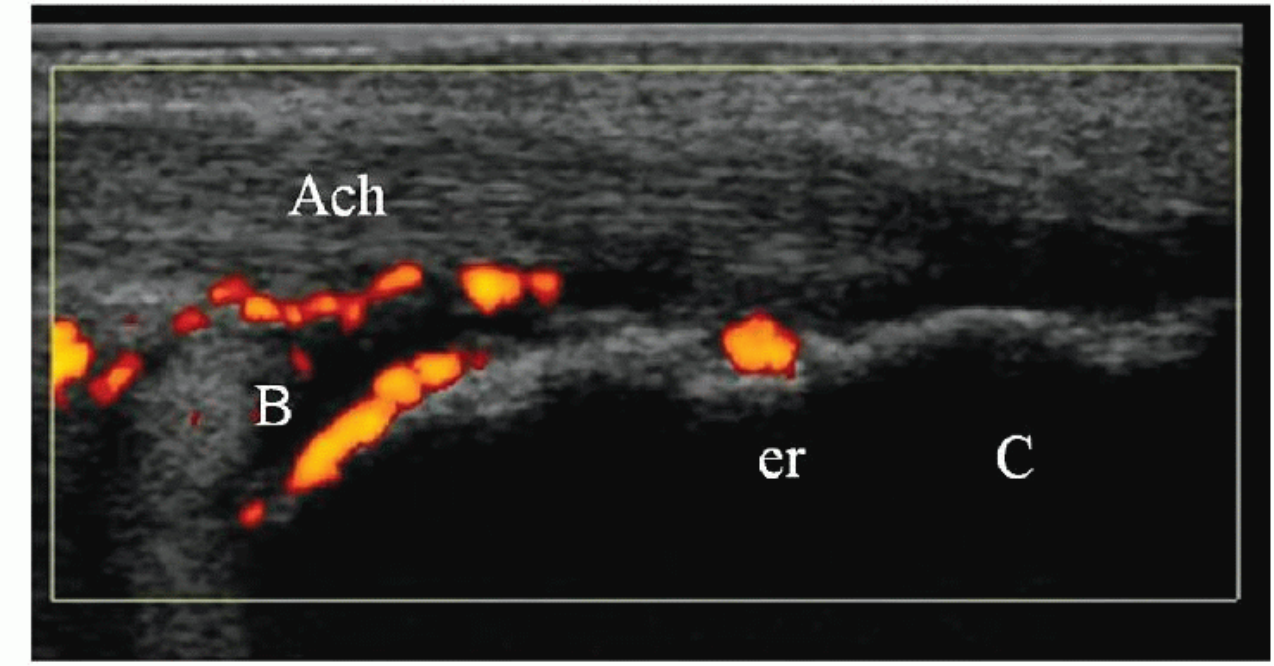

Figure 10.13. Longitudinal scan of the insertion of the Achilles tendon (Ach) of a patient with spondyloarthropathy. Enthesitis with slight focal hypoechoic tendon enlargement, retrocalcaneal bursitis (B), and bone erosion (er) of the calcaneus (C). Color Doppler examination shows hyperemia in bursa, anterior tendon, and erosion.

Psoriatic Arthritis

Psoriatic arthritis is a chronic inflammatory joint disease associated with psoriasis. Skin disease is generally present, but 10% to 15% of patients with joint involvement do not have cutaneous changes. The first symptoms often appear around 40 years of age, equally frequent in men and women. Any peripheral joint may be inflamed but an asymmetric oligo- or monoarthritis is typical, although polyarthritis is seen in 10% to 20% of patients. Sacroiliitis, spondylitis, enthesitis (Achilles tendon) (Fig. 10.14), synovitis of the small joints of the finger (especially the distal interphalangeal joints), and proliferative osseous changes are characteristic findings. Dactylitis is a sausage-shaped swelling of fingers or toes secondary to joint synovitis, tenosynovitis, periostitis, and enthesitis. The prognosis is variable. It is often better than RA, but erosive changes may lead to arthritis mutilans and significant incapacity.

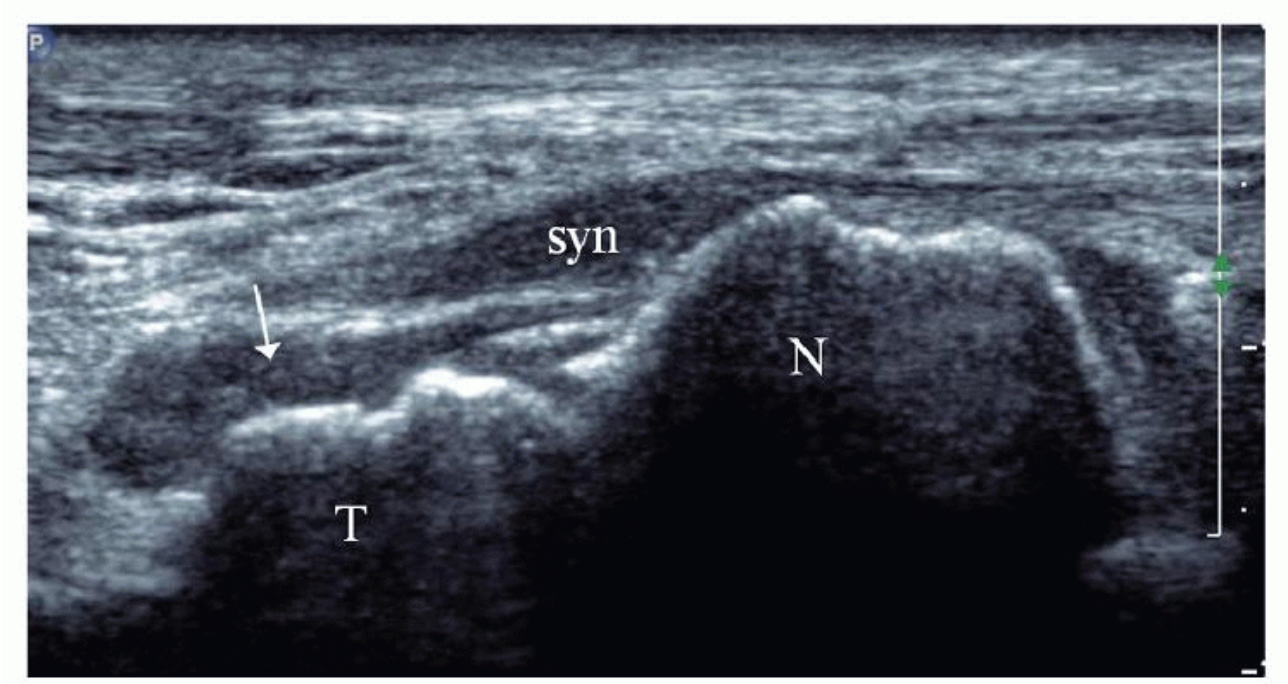

Figure 10.14. Psoriatic arthritis. Longitudinal ultrasound scan of dorsal aspect of talonavicular joint shows synovial hypertrophy (syn) and thickening of the proximal insertion of the dorsal talonavicular ligament due to enthesitis (arrow). T, Talus; N, Navicular.

Juvenile Idiopathic Arthritis

Juvenile idiopathic arthritis (JIA) is not one disease but encompasses all forms of arthritis that begin before the age of 16 years, persist for >6 weeks, and are of unknown etiology. This group of conditions is distinct in many ways from RA in adults. Seven subtypes are distinguished on the basis of clinical (intra-articular and extra-articular involvement) and laboratory findings: systemic JIA, rheumatoid factor positive polyarthritis, rheumatoid factor negative polyarthritis, oligoarthritis, enthesitis-related arthritis, psoriatic arthritis, and undifferentiated arthritis.[44] In high-income countries, the prevalence is 16 to 150 cases per 100,000 children.[45]

The distribution of joint involvement depends on the subtype. Any joint may be affected, frequently large joints. The largest group of patients with JIA present with oligoarthritis (25%) and frequently with involvement of knees and ankles. Small joints may also be affected, including hands and feet (especially in polyarticular disease), temporomandibular, cervical, thoracic, and lum-bosacral joints.

Clinical assessment of joint swelling is difficult. Ultrasound provides precise identification of the site of inflammation (synovial hypertrophy, joint effusion, tenosynovitis, enthesitis, and/or soft tissue edema) or the presence of destructive changes (bone erosions). Ultrasound findings in adult arthritis are not directly applicable to children because of differences in disease characteristics and the unique features of the growing skeleton such as age-related variations in articular cartilage thickness, incomplete ossification, and bone growth anomalies induced by the disease. It is more challenging to assess synovial hypertrophy in young children than in adolescents and adults in spite of better image quality, because synovial tissue and epiphyseal cartilage are both very hypoechoic and difficult to distinguish from each other (Fig. 10.15). Doppler examination is generally not a solution to this problem as vascularization is present in both inflamed synovium and cartilaginous epiphyses during growth. It is therefore important to know the normal ultrasound appearance of each joint at different stages of development and to avoid anisotropic artifacts (Fig. 10.15). A small amount of joint fluid is physiological in children. Tenosynovitis is commonly detected by ultrasound in swollen ankles and wrists of JIA patients, more than previously assumed.[46],[47] Hyperemia on Doppler studies is correlated with clinical activity as in adult arthritis.[48]

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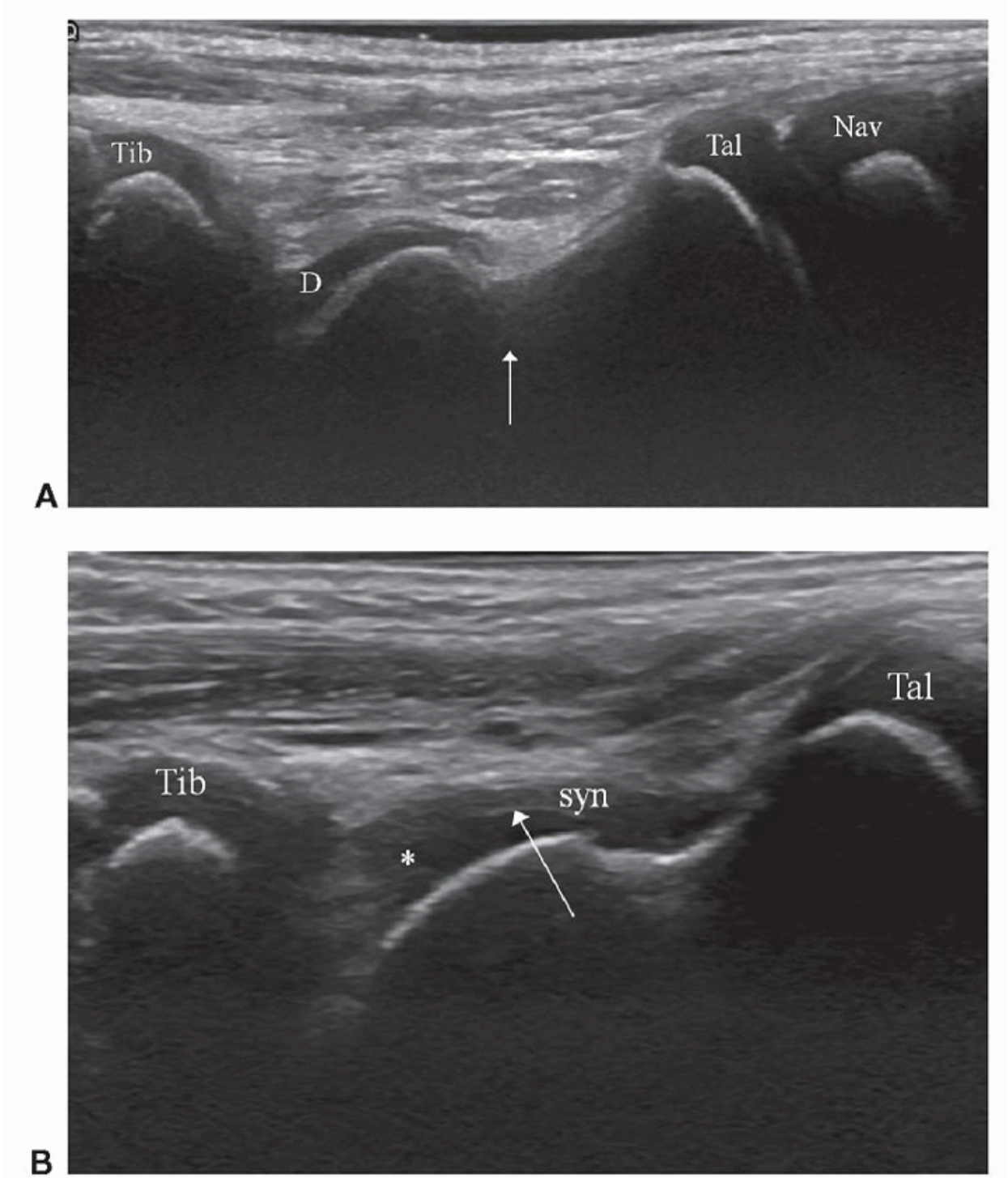

Figure 10.15. Dorsal longitudinal ultrasound scans of ankles of 4-year-old children. A: Normal ankle with thick hypoechoic cartilage on talar dome (D) and in growing epiphyses of distal tibia (Tib), distal talus (Tal), and navicular (Nav). No sign of synovitis. B: Juvenile idiopathic arthritis with hypoechoic synovial hypertrophy in anterior ankle joint recess. The interface between cartilage and synovium (syn) is seen when the ultrasound beam is perpendicular to the cartilage surface (arrow), but not identified when the ultrasound beam is angled due to anisotropy (asterisk).

Diagnosis with Ultrasound

MRI provides a more complete joint examination than ultrasound, but requires sedation of young children, an age group with a high prevalence of JIA. Consequently, ultrasound has a significant role in the assessment of disease activity. Young children can be seated on a parent's lap or play while being examined. The way a child communicates joint discomfort varies. Ultrasound is particularly useful if there are few verbal complaints, for example, in infants. Ultrasound is also more informative than clinical examination,[49] and subclinical synovitis is frequently detected by ultrasound, particularly in the hands and feet. There are no validated MRI- or ultrasound-scoring systems for evaluating inflammatory and destructive joint abnormalities in JIA.

Follow-up with Ultrasound

Ultrasound may detect decreases in joint effusion and synovial hypertrophy in patients treated systemically or with intra-articular steroid injections.[46],[47] Confirmation of remission cannot rely solely on clinical examination, but must include repeated imaging to

confirm the absence of subclinical inflammation,[50] although further studies are needed to evaluate the reliability of ultrasound assessment of treatment response.

A proportion of JIA patients who do not receive treatment will develop progressive joint destruction, growth disturbances, and serious functional disability, but the course of the disease is difficult to predict. One important prognostic factor is the JIA subtype. Early use of new highly efficacious treatments has improved the outcome in many patients with JIA. Ultrasound has the potential to detect subclinical synovitis or enthesitis, which might lead to change in disease categorization (e.g., from oligoarthritis to polyarthritis or enthesitis-related arthritis) and early change of therapy. Ultrasound detection of bone erosions early in the course of JIA is also an indicator of poor long-term outcome.

Crystal Deposition Diseases

In crystal deposition diseases, crystals are deposited in joints and periarticular soft tissues. The main crystals are monosodium urate, calcium pyrophosphate (CPPD), and calcium hydroxyapatite (HADD), although mixed crystal deposition may occur. Crystal deposition can cause inflammation or articular damage, although many patients are asymptomatic. Aspiration of joint fluid is needed for definitive diagnosis and may be facilitated by ultrasound-guided aspiration. Examination under polarized light microscopy distinguishes urate from pyrophosphate crystals. However, characteristic patterns of deposition may allow ultrasound differentiation between urate and pyrophosphate deposition.[51] Ultrasound can also show erosions and synovitis in established joint disease.

Gout

Gout results from deposition of monosodium urate crystals. Acute gout can occur at any joint, but preferentially involves the lower limb. Classically it involves the first MTP joint (podagra) and presents with severe pain, erythema, and swelling. Ultrasound shows synovial thickening, Doppler signal, and periarticular edema. Joint fluid during an acute attack may be anechoic, but pressure with the transducer may elicit a “snowstorm” appearance due to displacement of crystals, which are typically heterogeneous in size and shape.

Monosodium urate crystals are characteristically deposited on the surface of articular cartilage. The crystals are strongly reflective, even with low gain settings, and produce echogenic foci on the surface of the cartilage, resulting in a “double contour” appearance (Fig. 10.16).

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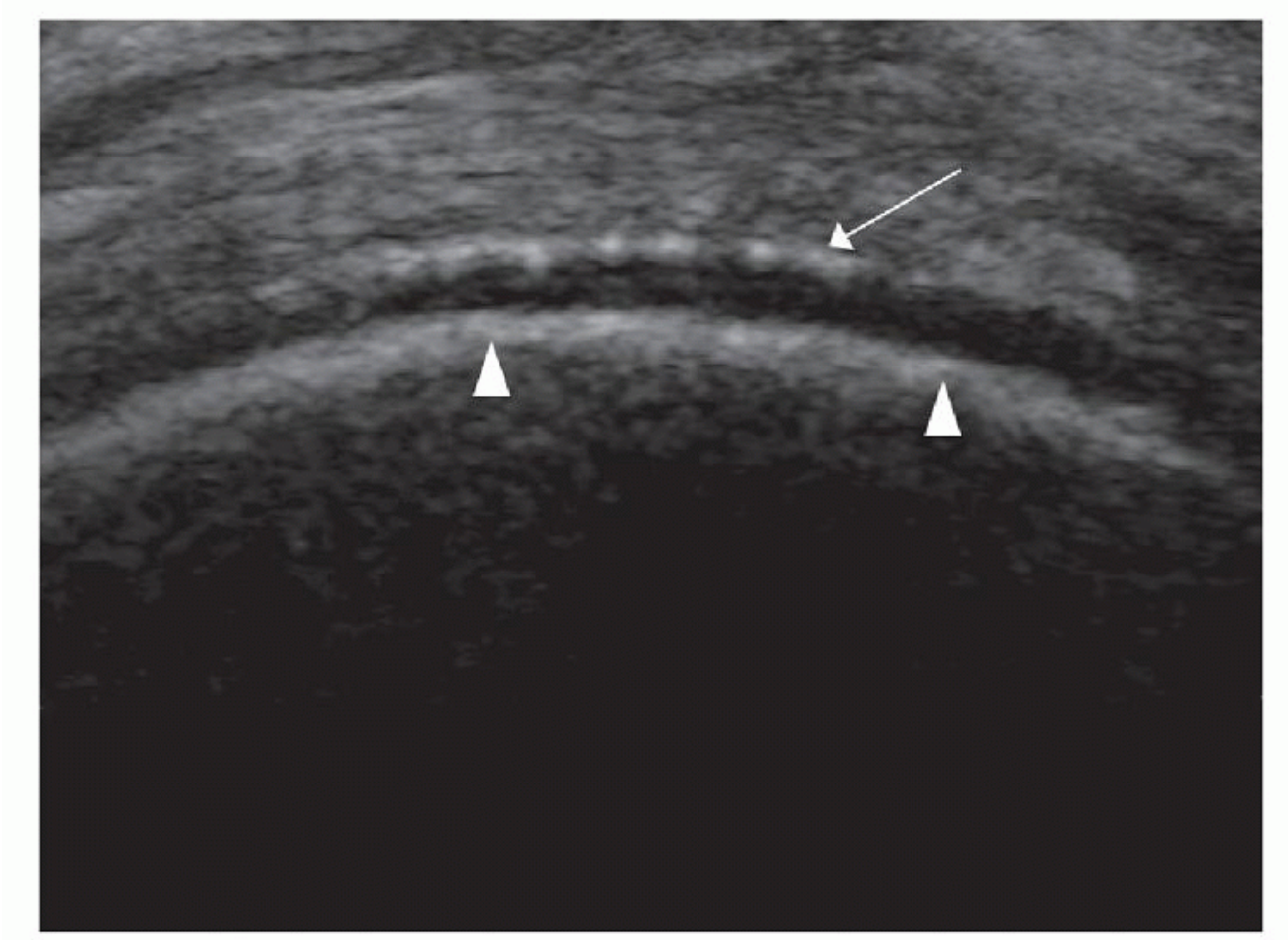

Figure 10.16. Ultrasound examination of the femoral articular cartilage of the knee of a patient with gout. The double contour appearance due to echogenic crystal deposition on the surface of the cartilage (arrow) is characteristic of gout. Note the subchondral bone (arrowheads).

Tip:

A “double contour” appearance due to deposition of echogenic crystals on the surface of articular cartilage is pathognomonic of gout.

Tophi are larger aggregations of monosodium urate crystals, most often seen in fingers, toes, and at the olecranon bursa at the elbow. They may be intra-articular or extra-articular and extend into tendons. Tophi may be soft, hard, or mixed. Soft tophi contain multiple small echogenic foci without distal acoustic shadowing. Hard tophi are echogenic and have distal acoustic shadowing (Fig. 10.17). Doppler signal may be prominent. Ultrasound may show intra-articular erosions and synovial thickening. The synovial thickening is uniform, whereas intra-articular tophi are nodular. Erosions in association with tophi are more common in the feet than hands.[52,53]

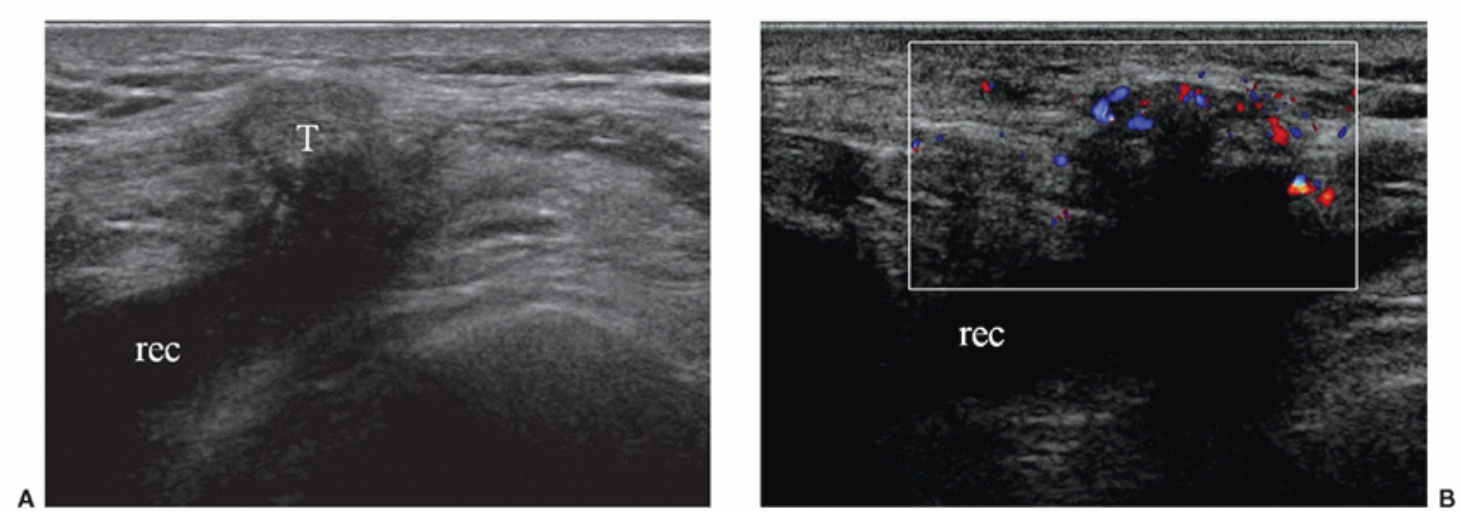

Figure 10.17. Transverse scan of the parapatellar recess (rec) of a patient with gout. A: A palpable tophus close to the parapatellar recess is inhomogeneous, hyperechoic, and casts strong acoustic shadowing (T). B: Peripheral hyperemia on color Doppler confirms the presence of inflammation.

Calcium Pyrophosphate Deposition Disease

Calcium pyrophosphate deposition in hyaline cartilage and fibrocartilage is often an asymptomatic incidental finding on radiographs. Pseudogout is an acute arthritis due to pyrophosphate deposition and usually affects large joints, typically the knee (Fig. 10.18). Pyrophosphate crystals are deposited within articular cartilage. This distinguishes CPPD from gout, in which the crystals are on the surface of the cartilage.

Tip:

Echogenic crystal deposition deep to the surface of articular cartilage is typical of CPPD.

Pyrophosphate crystals in fibrocartilage, such as the menisci of the knee, result in punctate, echogenic calcifications with or without posterior acoustic shadowing. Streaky calcification may be seen in tendons, synovium, and joint capsules.

Hydroxyapatite Deposition Disease

Hydroxyapatite crystals are characteristic of calcific tendinitis and may be seen in destructive arthropathies such as cuff tear arthropathy. Tendon deposits are frequently densely echogenic and have posterior acoustic shadowing. In acute calcific tendinitis, when the calcium softens and the patient presents with acute, severe pain, the calcium may be only faintly echogenic, difficult to distinguish from adjacent tendon, lack acoustic shadowing, and be surrounded by Doppler signal.

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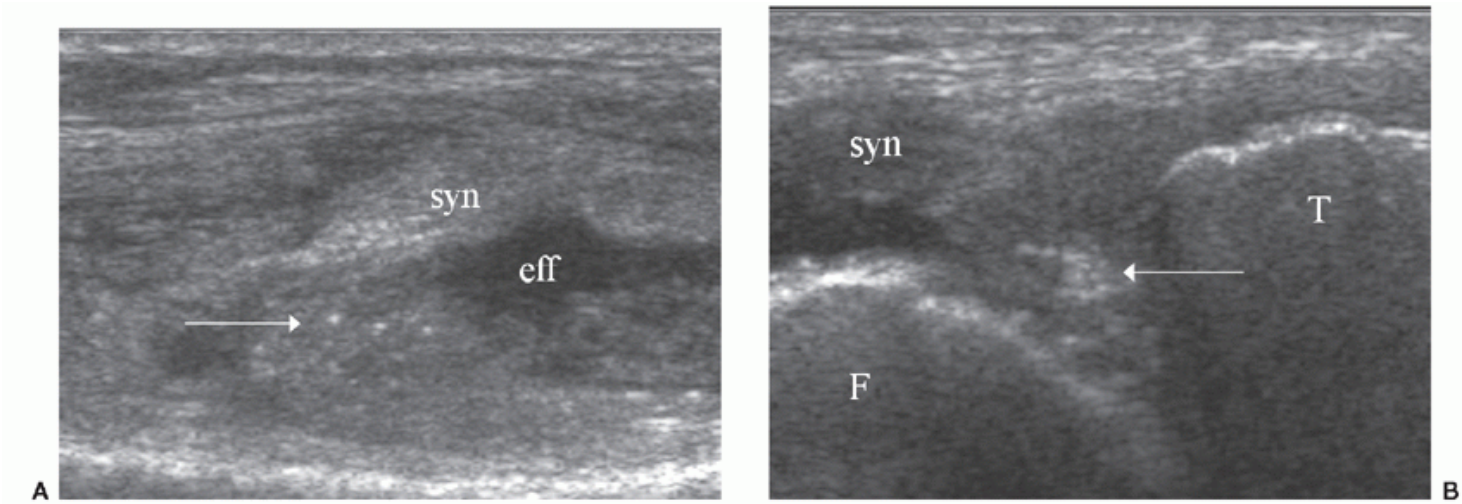

Figure 10.18. Patient with CPPD disease. A: Effusion (eff) and synovial thickening (syn) in the knee, with tiny echogenic foci of micro-calcification in synovium (arrow). B: Calcific deposits in the medial meniscus (arrow). F, femur; T, tibia.

OTHER JOINT DISEASES

Osteoarthritis

Nonspecific changes in osteoarthritis include joint fluid, synovial thickening, and thickening and bulging of the joint capsule.[54,55] Enthesopathic changes, such as thickening and loss of fibrillar architecture, may be seen at tendon and ligament insertions. Abnormal Doppler signal in joints or at entheses and intra-articular erosions may be present and are not exclusive to inflammatory arthropathy. Articular surface changes tend to be central, and this can be a problem because of limited ultrasound access. Improved visualization of articular surfaces can be obtained by examining some joints in flexion, for example, the MCP/MTP and interphalangeal joints. Knee flexion and plantar flexion at the ankle improve access to the trochlea of the knee (Fig. 10.9) and the talar dome, respectively. Articular cartilage may become ill-defined, heterogeneous, and thinned, and the articular cortex irregular. Osteophytes and enthesophytes appear as small bony prominences at the joint margin (Fig. 10.19), for example, Heberden and Bouchard nodes at the distal and PIP joints, respectively.

Pigmented Villonodular Synovitis, Synovial Osteochondromatosis, Amyloid

In addition to systemic arthropathies such as RA or gout, synovial proliferative conditions such as pigmented villonodular synovitis (PVNS), synovial osteochondromatosis, and amyloid[56,57,58] may cause intra-articular masses and large erosions. The synovial masses may be localized or diffusely involve a joint. PVNS is a benign neoplastic process that results in villous/nodular synovial hypertrophy. Repeated bleeding causes hemosiderin deposition. Localized intra-articular PVNS usually occurs at the knee. Another localized form of PVNS occurs in relation to tendon sheaths, particularly in the hand, and is also known as giant cell tumor of tendon sheath (GCTTS). Synovial osteochondromatosis occurs in joints, bursae, and tendon sheaths. It is thought to be a benign neoplastic process rather than a metaplastic process and results in synovial hyperplasia and cartilaginous nodules that may calcify or ossify and form loose bodies. Amyloid arthropathy occurs in patients on long-term hemodialysis, which results in the deposition of β2 microglobulin in synovium. Deposits also occur in tendons, bursae, and periarticular soft tissues including the carpal tunnel.

Ultrasound in all three conditions shows joint fluid and hypoechoic synovial masses that may be extensive or localized, nodular, or causing diffuse synovial thickening (Fig. 10.20). Large bony erosions may be present. The three conditions may be indistinguishable on ultrasound, and biopsy is usually required to confirm the diagnosis.

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PVNS tends to be hypervascular on ultrasound and is diagnosed on MRI if blooming artifact due to hemosiderin deposition is present on gradient echo images. GCTTS results in a hypoechoic mass that is intimately related to a tendon sheath (see Chapter 9). Synovial osteochondromatosis is hypovascular. Calcification or ossification of cartilage nodules results in echogenic foci (Fig. 10.21). Amyloid has no specific diagnostic ultrasound features, but the history of chronic hemodialysis is characteristic. Amyloid deposits cause tendon thickening, slightly hyperechoic masses in bursae and between muscles, and periarticular fluid collections.

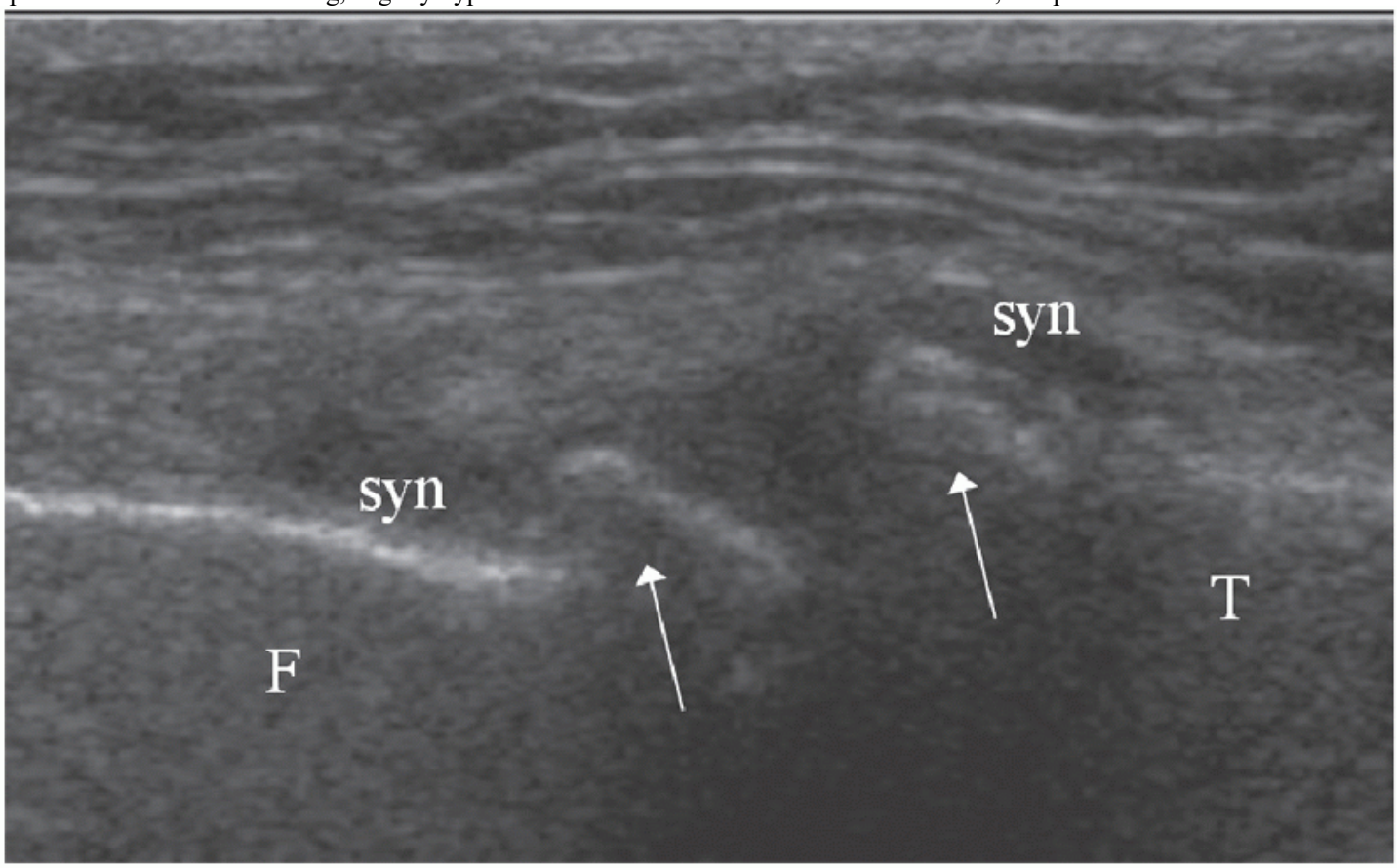

Figure 10.19. Osteoarthritis of the knee. Longitudinal scan of medial joint line shows osteophytes (arrows) and synovial thickening (syn). F, femur; T, tibia.

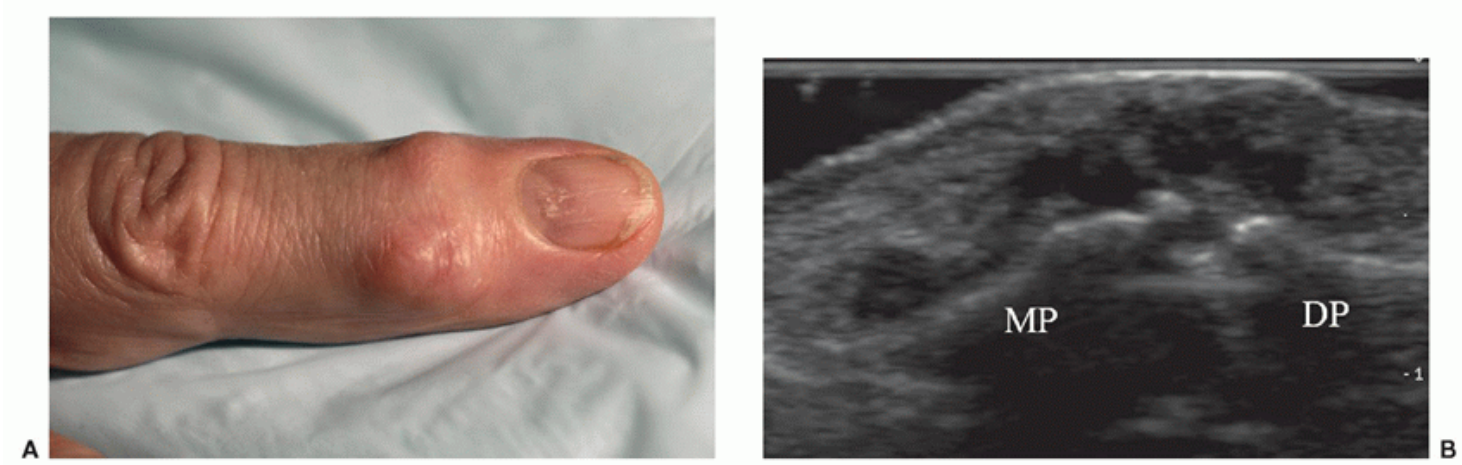

Figure 10.20. Patient with PVNS. A: Clinical photograph shows focal soft tissue swelling on ulnar aspect of distal interphalangeal joint of index finger. B: Longitudinal ultrasound on dorso-ulnar aspect of the distal interphalangeal joint shows a hypoechoic synovial mass communicating with the joint line. MP, middle phalanx; DP, distal phalanx.

Tip:

PVNS, synovial osteochondromatosis, and amyloid cause larger erosions than inflammatory joint disease.

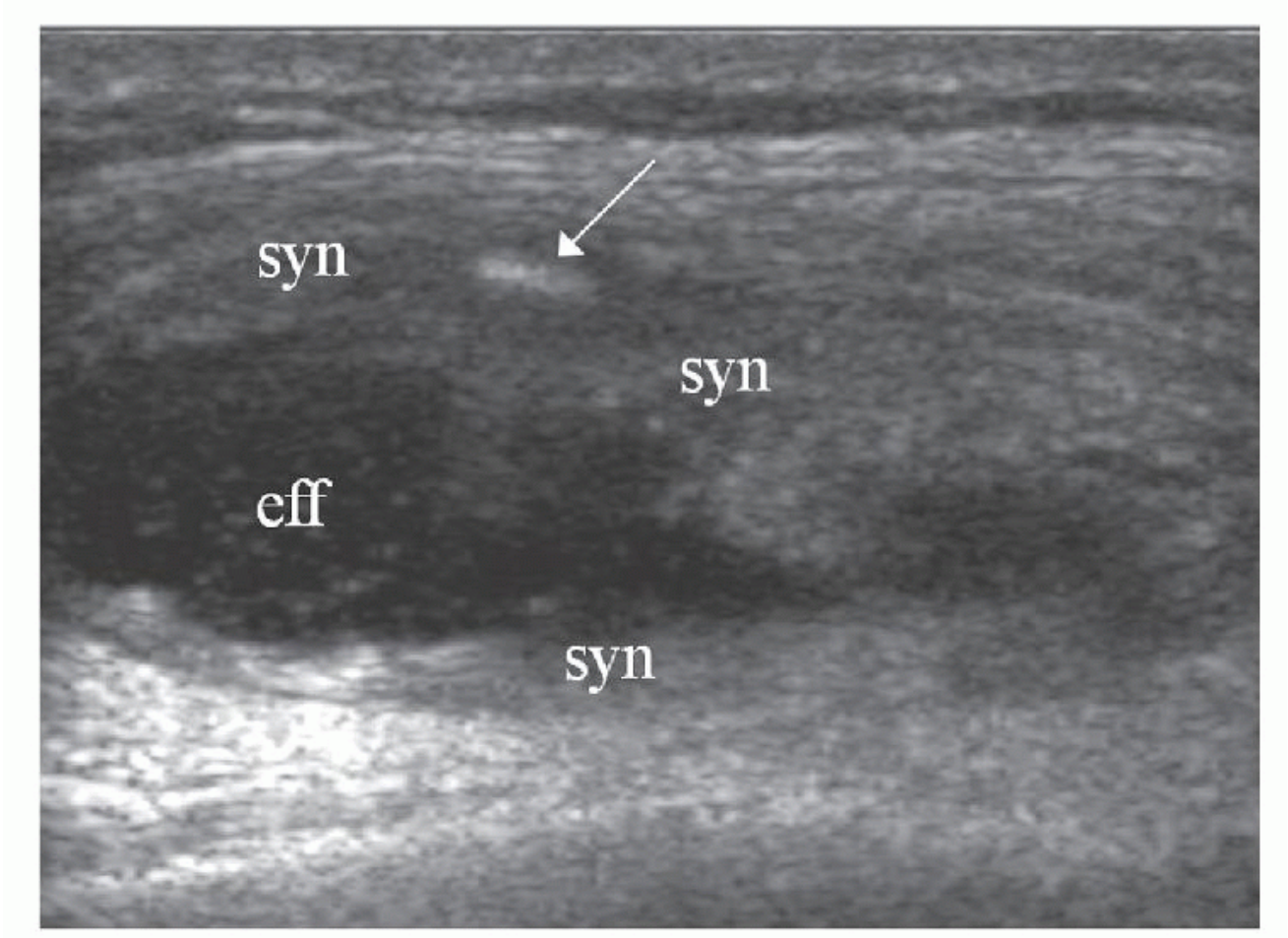

Figure 10.21. Osteochondromatosis of the knee. Ultrasound shows effusion (eff) and synovial hypertrophy (syn) in the knee recess. Calcification in the thickened synovial wall is seen (arrow).

Septic Arthritis

Joint infection rapidly destroys articular cartilage; therefore, urgent diagnosis and treatment are essential. Patients typically present with a painful, swollen joint, but a more indolent presentation may occur with less aggressive organisms or in immunocompromised patients. Inflammatory markers are not always elevated. To further confuse matters, inflammatory markers may be elevated in acute calcific tendinitis, which has a similar clinical presentation to septic arthritis at the shoulder. Radiographic changes in septic arthritis are delayed. Septic arthritis in children is discussed in Chapter 13.

The key ultrasound feature of septic arthritis is joint fluid, although synovial thickening and synovial and periarticular Doppler signal are also seen. Joint fluid is rarely anechoic and often contains low-level echoes. Echogenic fluid, debris, septations, and even intra-articular gas bubbles may be present. Echogenic fluid may simulate synovial thickening, but fluid can be distinguished from

synovial thickening by using the transducer to compress the joint and show typical fluid movement.[59] If fluid is demonstrated, ultrasound-guided joint aspiration should be performed to obtain fluid for an urgent Gram stain and culture, including for TB. Septic bursitis and tenosynovitis are also diagnosed by the presence of fluid in the appropriate clinical setting, and aspiration should be performed. Ultrasound is widely used to look for joint fluid in suspected septic arthritis. However, poor results have been recorded at the hip.[60] Some joints (e.g., the sacroiliac, sternoclavicular, and acromioclavicular joints) have non-distensible capsules, and the absence of fluid does not exclude infection. MRI should be performed if infection is not confirmed.[59] Ultrasound aspiration of prosthetic hips is often employed, although disappointing results have been reported.[61]
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Hemophilic Arthropathy

Hemophilic arthropathy is the result of repeated hemarthroses that often start within a few years of birth. The effect of intra-articular blood on the joint may be cartilage-mediated or synovial-mediated.[62] Synovial hypertrophy, inflammation, and hemosiderin deposition predispose to further bleeds. Chronic disease leads to cartilage and subchondral bone destruction with superimposed degenerative changes, and may finally result in ankylosis. Ultrasound can show very small hemarthroses in early disease and nonspecific effusions and synovial hypertrophy in later disease. Problems with standardization and interobserver variability mean that ultrasound is less suited than MRI to monitor disease progression.[63]

ULTRASOUND-GUIDED THERAPEUTIC TECHNIQUES

Local steroid injection is an important element in the treatment of arthritis and osteoarthritis. The clinical effect depends on placing the needle accurately. Ultrasound is increasingly used to guide aspiration or injection of joints, tendon sheaths, bursae, cysts, and around tendon insertions. It ensures accurate and safe needle placement, especially in anatomically complex areas such as the hip, ankle, and wrist. Aseptic technique is essential. The skin should be cleaned and sterile gloves and a transducer cover used. The needle (21G to 23G) should be inserted using a “free hand” technique with the needle inclined obliquely along the scan plane while scanning in real time. When the scan plane is longitudinal, the needle is inserted distal to the transducer and directed obliquely toward the target. Needle puncture of a joint recess or a tendon sheath is easier if it is distended by fluid or synovial hypertrophy. Injection of dry joints is more challenging, and may necessitate preliminary injection of a small amount of local anesthetic to ensure the needle is correctly positioned. Most joint injections are performed with a scan plane that crosses the joint line, and the needle tip is inserted into the joint recess or placed against articular cartilage. For injections in small joints or tendon sheaths of the wrist/hand and ankle/foot, a small “hockey stick” transducer is useful. The injected dose of steroid depends on the type of steroid and the size of the joint, for example, 20 mg of triamcinolone acetonide for small joints and 40 to 80 mg for large joints, usually with an appropriate volume of lidocaine or bupivacaine. The puncture site should be compressed after the procedure so as to minimize the risk of subcutaneous atrophy due to steroid leaking from the joint.

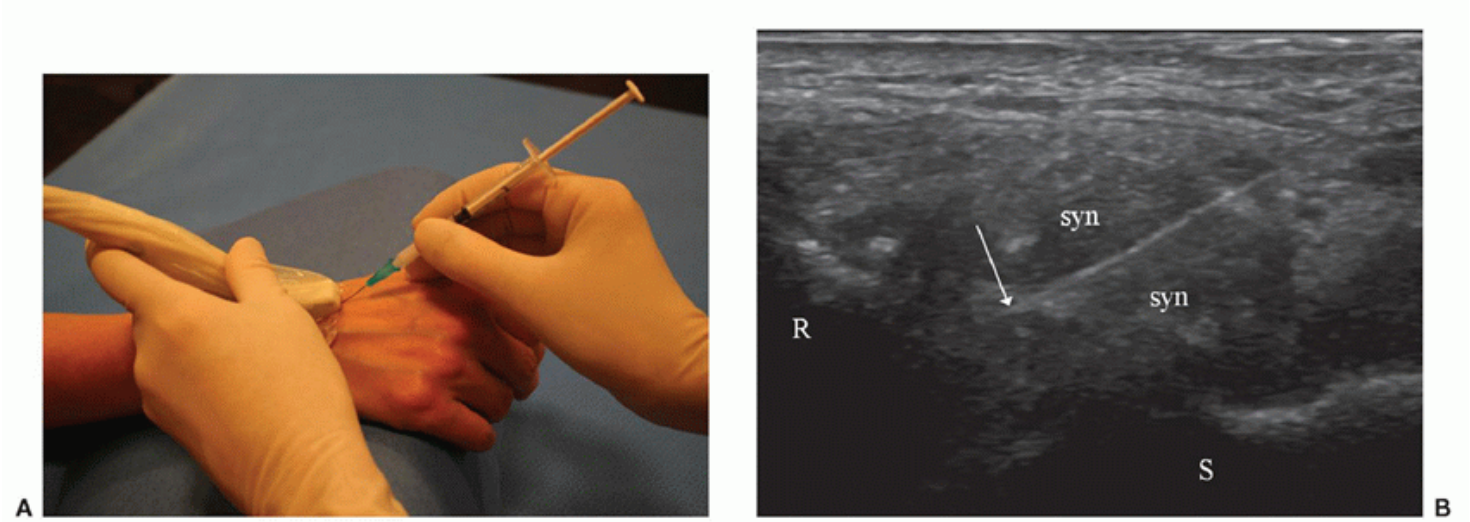

Figure 10.22. Ultrasound-guided injection in the dorsal recess of the radiocarpal joint with a longitudinal scan just distal to the Lister tubercle on the radius. A: Position of the transducer. B: Needle tip (arrow) in the radiocarpal recess enlarged by synovial hypertrophy (syn) in a patient with osteoarthrosis. R, radius; S, scaphoid.

We will here describe injection techniques in two difficult anatomical areas with frequent involvement in rheumatology; the wrist and hand, and the ankle and foot.

Ultrasound-Guided Injections in the Wrist and Hand

Puncture of the dorsal radiocarpal recess is performed with a longitudinal scan plane at the level of the Lister tubercle (Fig. 10.22). The dorsal midcarpal recess is punctured in a similar way, slightly distally, generally in a more ulnar longitudinal scan plane, avoiding the ulnar extensor tendons (Fig. 10.23). Puncture of the distal radioulnar joint is less frequently performed. A dorsal transverse scan plane and ulnar skin puncture avoid the extensor digiti minimi tendon. The needle is placed either just proximal to the joint in an enlarged superior recess or at the level of the joint line in contact with the cartilage of the ulnar head. Osteoarthritis of the first carpometacarpal
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joint is often treated by local steroid injection. Two puncture routes are possible: A dorsal longitudinal route with the needle inclined relatively perpendicular to access the joint while avoiding the branches of the radial nerves, or a palmar longitudinal route through the thenar muscles, with a more oblique approach.

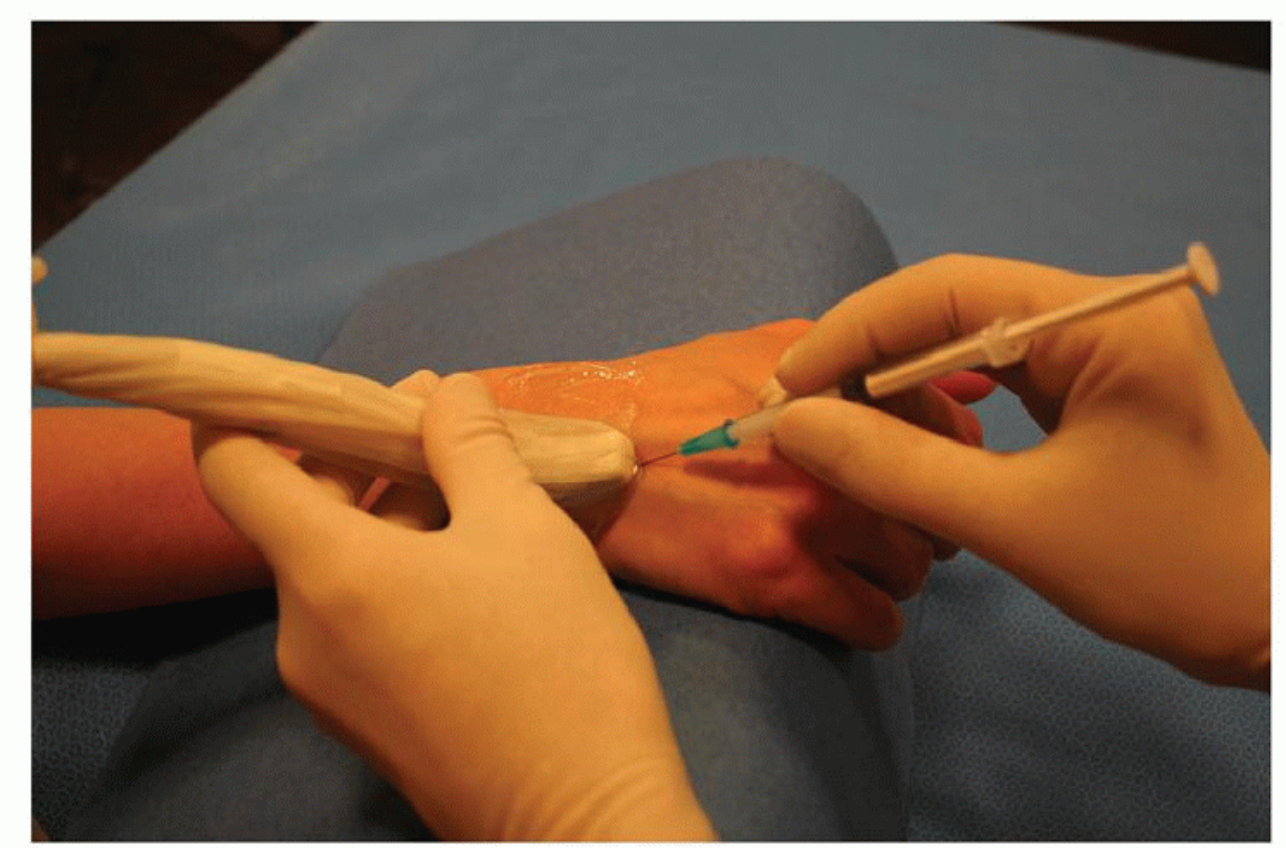

Figure 10.23. Ultrasound-guided injection in the dorsal recess of the midcarpal joint with a longitudinal scan just distal to the ulna. The finger joints are injected dorsomedially or dorsolaterally, avoiding the extensor tendon and the digital arteries and nerves. If the joint recess is distended, a longitudinal scan plane with a distal oblique approach is possible. If the recess is not prominent, it may be easier to visualize and mark the joint line with the help of a needle or paperclip placed between the skin and the transducer (Fig. 10.24). It is then generally easy to penetrate the joint line with a single perpendicular puncture using a hockey stick transducer only if the placement is doubtful (Fig. 10.24). Tendon sheaths can be injected in a longitudinal plane with a puncture route as tangential as possible and inserting the needle tip as far as possible into the sheath. If the sheath is minimally enlarged, a transverse scan plane may be easier.

Ultrasound-Guided Injections in the Ankle and Foot

The ankle joint is punctured with a longitudinal, anteromedial or anterolateral scan plane, avoiding the extensor tendons and the anterior tibial vessels. The needle tip is inserted into the anterior recess or placed against the cartilage of the talar dome. In osteoarthritis or JIA, injection of the posterior subtalar joint may be needed but can be difficult to perform, especially if the recesses are not bulging. An anterolateral oblique plane, anterior to the peroneal tendons, can be used by moving the transducer posteriorly from a coronal image of the tarsal sinus (Fig. 10.25). The anterior subtalar joint (talocalcaneonavicular) or other tarsal joints are punctured dorsally, with an appropriate scanning plane to avoid the tendons and

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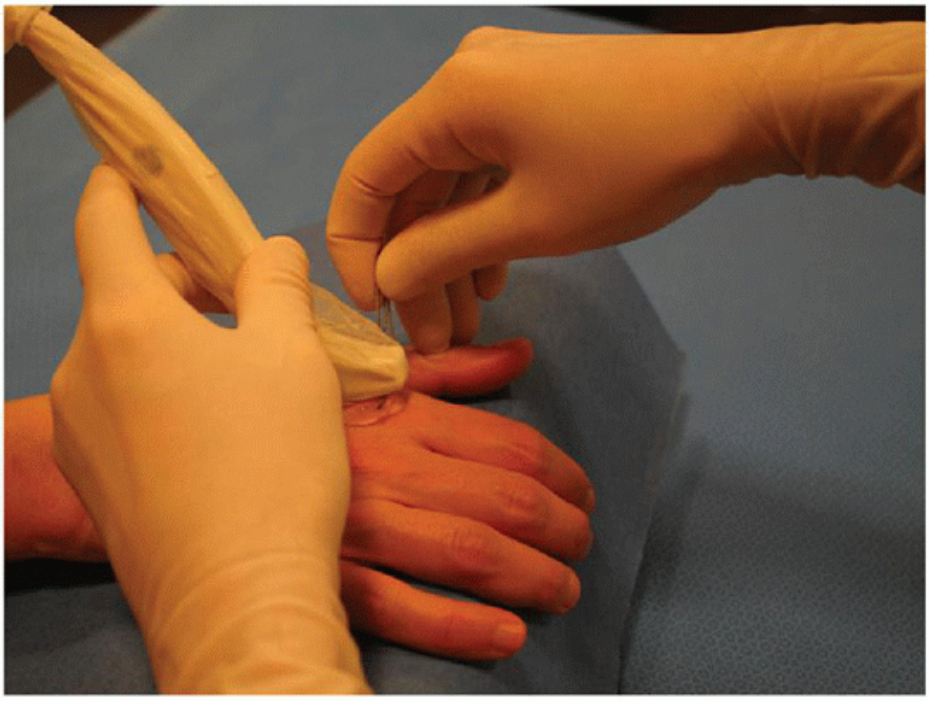
the vessels. Injections of periarticular tendons and toe joints are similar to the wrist and hand. The flexor hallucis longus tendon is deeply situated and is best accessed with an oblique approach, just proximal to the calcaneus, using a hockey stick transducer and avoiding the posterior tibial artery and the tibial nerve.



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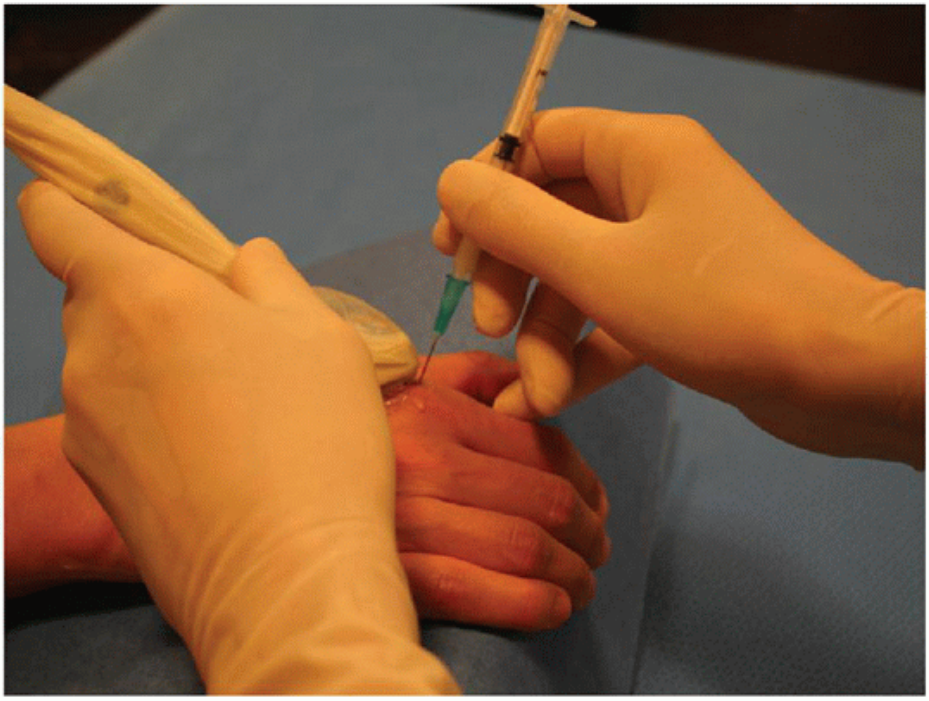

B

Figure 10.24. Steroid injection in the second MCP joint.
A: Marking the skin over the joint line with a paperclip.
B: Ultrasound-guided puncture.

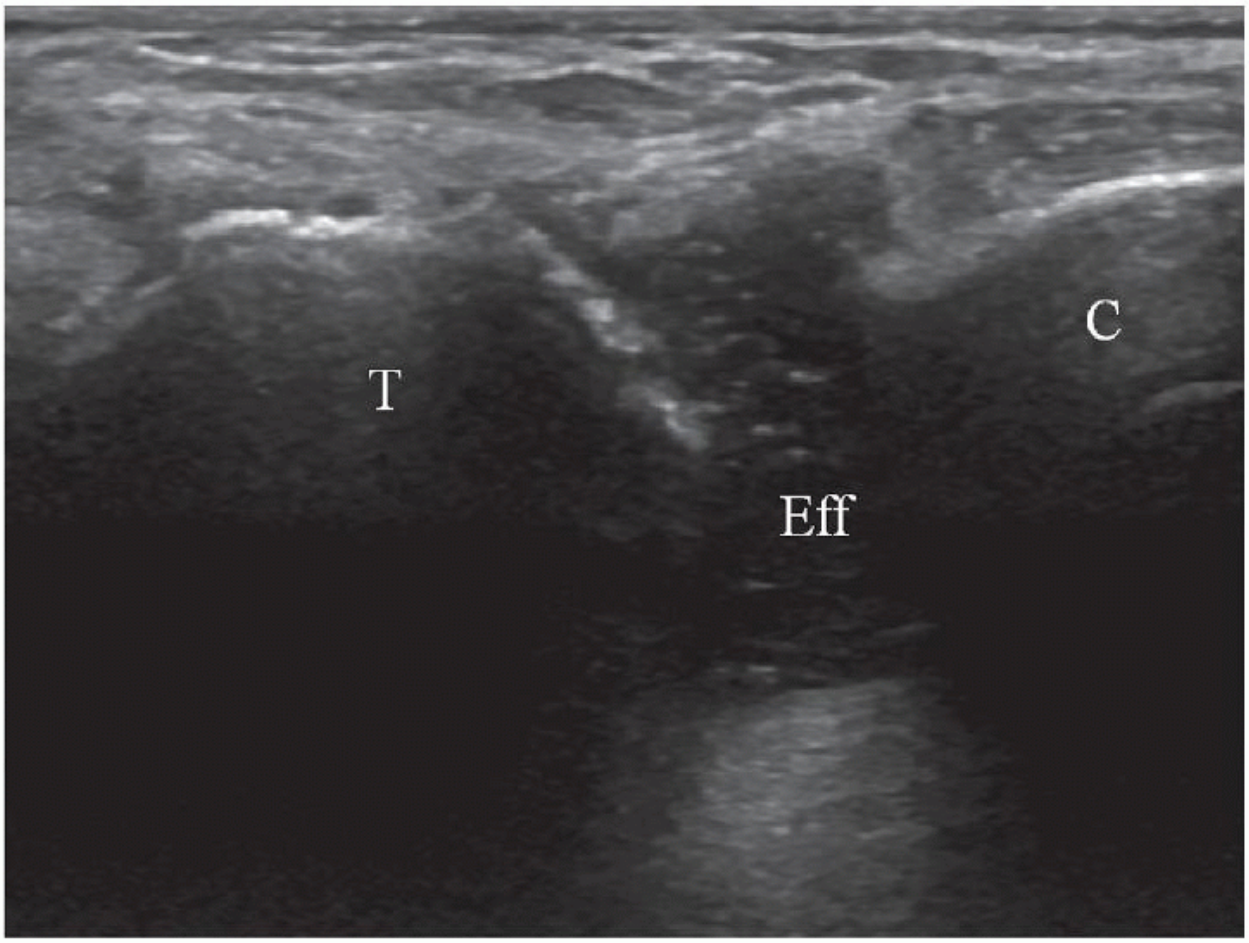

Figure 10.25. Osteoarthrosis of the posterior talocalcaneal joint. Lateral coronal ultrasound scan showing effusion (Eff) in the anterolateral recess.

CONCLUSION

Ultrasound is now established as an essential tool in the initial assessment, monitoring, and treatment of inflammatory arthropathy.

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