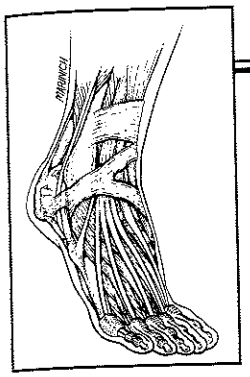


CHAPTER 35

Chronic Ankle Conditions

Molly Schnirring-Judge and Michael D. Perlman

SINUS TARSI SYNDROME

Clinical Presentation

Sinus tarsi syndrome is characterized by a deep aching, throbbing, or sharp pain within the sinus tarsi that may be associated with a sense of rearfoot instability. Although not always recalled, a previous sprain or other injury is often the inciting event (1–7). Patients most often present with a complaint of "ankle" pain, when in reality the pain is emanating from the subtalar area. However, when asked to identify the site of maximal tenderness, patients often point directly to the sinus tarsi region. At times, pain may involve the anterior and or lateral ankle as well, although this pain is less intense. The perception of ankle symptoms may be secondary to previous trauma, referred pain, or an altered gait in an attempt to reduce pain.

The symptoms are aggravated by weight-bearing activities, especially during ambulation on uneven surfaces, and they usually improve or resolve with rest (2,5,7). Swelling may or may not be present, and a subtle fullness may go unnoticed. A sense of hindfoot instability is often reported (8–16). This may be secondary to previous disruption of the ligamentous and neurovascular structures within the sinus tarsi and about the subtalar joint.

Anatomic Considerations

The sinus tarsi is a funnel-shaped channel located just anterior to the posterior subtalar joint facet, between the inferior surface of the talus and the superior surface of the calcaneus. The opening of the sinus tarsi is larger laterally, and the channel narrows as it progresses in a posterior and medial direction; it exits posterior to the sustentaculum tali and below the medial malleolus. Within and about this compartment lies a fenestrated pattern of vascular foramina, ligamentous and fatty structures, and proprioceptive elements (2,8–10,12–14,17–29). A large venous plexus exits from the talus and the anterior part of the posterior subtalar joint capsule to the lateral and medial venous systems of the foot. Also within this region is a fibroadipose plug (Hoke's tonsil), the artery of the tarsal canal, the deltoid artery, a bursal projection, and proprioceptive nerves (25,26,30) (Fig. 1).

The ligaments of the sinus tarsi work in conjunction with the lateral ankle ligaments to stabilize the lateral aspect of the ankle and rearfoot (1,27,31). These ligamentous structures include the cervical, bifurcate, anterior and lateral talocalcaneal, and interosseous ligaments, as well as the three roots of the inferior extensor retinaculum (8,9,21,27). Cadaver studies have been used to determine the stabilizing effect of the ligamentous structures of this region (9). Neither lesions of the cervical nor the interosseous ligament resulted in an increase in the total range of subtalar joint movement greater than 2.6 degrees in any of the triplane fields of motion. Sectioning of the interosseous talocalcaneal ligament produced the greatest loss of stability, approaching 43% of the total increase of motion appreciated. This increased motion may be sufficient for the patient to sense instability, without objective signs of instability evident clinically or radiographically. The inferior extensor retinaculum, composed of loose elastic fibers, is unlikely to contribute significantly to subtalar joint stability.

Etiology

Many patients with sinus tarsi syndrome recount a specific inciting traumatic event. An inversion injury of the ankle or rearfoot is common (1,2,5,6,12–14,20,32–34). Taillard et al. demonstrated a predictable pattern of ligament rupture in association with inversion ankle injuries (2). Increasing stress in the calcaneofibular ligament is followed by lateral talocalcaneal ligament attenuation, and ultimately stress is applied through the interosseous ligament. This sequence of injury may be a primary cause of sinus tarsi syndrome. The initial injury may have responded to conservative therapies, but recurrent pain or pain settling into the sinus tarsi may be experienced with activities of daily living.

Meyer et al. demonstrated a high correlation between subtalar and ankle disorders in 32 of 40 stress arthrograms per-

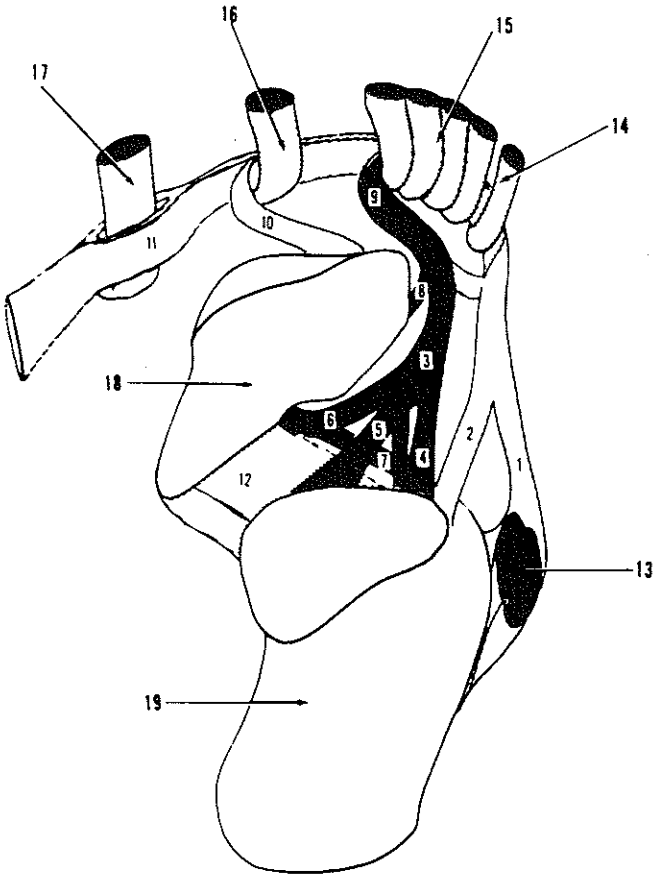


FIG. 1. Diagram with magnification after dissection. The oblique osteotomy of the talus in the direction of the tarsal canal. The posterior half of the talus has been removed. *1,* Lateral root of the inferior extensor retinaculum; *2,* intermediary root of the inferior extensor retinaculum; *3,* medial root of the inferior extensor retinaculum; *4,* lateral calcaneal component of the medial root; *5,* medial calcaneal component of the medial root; *6,* talar component of the medial root attached into the canalis tarsi; *7,* oblique talocalcaneal band of the medial root; *8,* talar body attachment of the medial root; *9,* loop formed by the medial root of the inferior extensor retinaculum turning around the extensor digitorum longus tendon; *10,* reflected component of the oblique superomedial band of the inferior extensor retinaculum forming a sling for the extensor hallucis longus tendon; *11,* tunnel for the tibialis anterior tendon; *12,* interosseous ligament of the canalis tarsi, oblique in direction, forming an *X* with the medial calcaneal component of the medial root of the inferior extensor retinaculum; *13,* tendons of the peronei; *14,* peroneus tertius tendon; *15,* extensor digitorum longus tendons; *16,* extensor hallucis longus tendon; *17,* tibialis anterior tendon; *18,* talus; *19,* calcaneus. (From Saraffian SK. *Anatomy of the foot and ankle.* Philadelphia: JB Lippincott, 1983:109, with permission.)

formed in patients presenting after ankle injury. These findings suggest that concomitant subtalar joint sprain may occur more commonly than previously suspected. Investigators believe that if these sprains are ignored or improperly treated, subsequent sinus tarsi syndrome or chronic ankle pain may develop (20,32,34,35).

The histopathologic features of tissue excised during surgery include scarring of the ligaments and hyperplasia of the synovium with obliteration of synovial recesses. Hemosiderin deposition may be noted, indicative of old hemorrhage. Chronic synovitis is perhaps the most common histologic finding (2,5,6,9). These soft tissue changes suggest a reparative process and are also consistent with a traumatic origin (2,5,6,20,34,36).

Posttraumatic pain may be attributable one of several sources. Disruption of the ligaments can result in pain from either instability or the injury itself. Trauma or chronic inflammation may result in compromise of the local tissues with a resultant decrease in venous outflow. This, in turn, may elevate pressure within the compartment of the sinus tarsi and may possibly lead to pain (23,37). Injury to the neurovascular structures of the sinus tarsi disturbs normal proprioceptive elements that supply the ligaments within this region. Consequently, the patient's perception of pain and instability leads to a decreased activity level and often to a modified lifestyle or altered gait.

Taillard et al. made the observation that 70% of these cases are posttraumatic, whereas the remainder can be attributed to a host of causes, including systemic disease, structural abnormalities, or soft tissue masses (2). Structural abnormalities include pes valgus, pes cavus (15,32), forefoot valgus (16), and tarsal coalitions. Systemic diseases such as seropositive or seronegative arthritis, crystal-induced arthritis, and infection may also create symptoms (2,5,6,38–40). Soft tissue masses such as ganglia (41,42) and lipomas have been reported in isolated case studies as sources of chronic sinus tarsi pain (15,17). In a study of 14 feet undergoing surgical procedures for a nonspecific diagnosis of sinus tarsi syndrome, intraoperative findings were significant for a variety of identifiable entities including 10 interosseous ligament tears, 2 cases of osteoarthritis, and 2 cases of arthrofibrosis (43).

Clinical Evaluation

The initial diagnosis of this condition is usually made by eliciting pain on compression of the lateral aspect of the sinus tarsi. The examination may be facilitated by supinating the patient's foot before palpation. Having the patient stand in an inverted position or performing stance maneuvers producing inversion and adduction across the subtalar and midtarsal joints may also produce pain (2). Local edema may be present, especially with more acute symptoms and when an active inflammatory process involved. However, in patients with the more typical chronic process, swelling is usually absent. Confirmation of the syndrome can be made when a diagnostic injection of local anesthetic into the sinus tarsi results in marked relief of pain (2,32,35). Although some degree of rearfoot instability may be described by the patient, in most instances clinical confirmation is lacking.

Taillard et al. described four consistent clinical signs associated with sinus tarsi disease: (a) tenderness with direct

palpation over the sinus tarsi or increased tenderness when standing on uneven surfaces or when supinating and adducting the subtalar joint, (b) instability on uneven terrain, (c) relief of pain with a diagnostic block of a local anesthetic, and (d) failure to identify clinical or radiographic evidence of instability (2).

Ancillary Studies

Ancillary studies are not required for most patients with sinus tarsi syndrome because the clinical evaluation is usually adequate to make the diagnosis. Furthermore, although helpful in supporting the diagnosis of sinus tarsi syndrome, these ancillary studies are not definitive diagnostic tests. Although standard radiographs may help to identify patients with foot types that may be more susceptible to inflammation at this level, there is generally a lack of degenerative arthritis or other remarkable findings noted. Stress radiography is often negative in patients with sinus tarsi syndrome as well (2,14,17,20,34,44–48).

Electromyography has demonstrated evidence of peroneal muscle dysfunction characterized by a reduction or complete absence of electrical activity during one or more walking cycles in gait. Investigators have proposed that this finding represents an insufficiency of the peroneal muscles with a subsequent failure to restrict inversion, a dysfunction resulting from the initial traumatic injury (2). When normal proprioceptive fibers are lost or disrupted, the neural feedback is inhibited, and inversion beyond an acceptable threshold is possible, with resulting instability. An anesthetic block has been shown to reverse this inhibition of the peroneal group for 10 to 20 minutes after the injection (5,23,49,50). Taillard et al. noted the return of proprioceptive input and peroneal response after surgical intervention or the use of anesthetic blockades (2).

Before the advent of magnetic resonance imaging (MRI), arthrography was an alternate means of assessing subtalar joint pathology (10,11,44,51,52). Typically, 2 mL of contrast material is injected into the posterior subtalar joint. Arthrograms of the normal joint demonstrate microrecesses within the sinus tarsi (2). Trauma to the subtalar joint and scarring and inflammation about the interosseous and cervical ligaments results in a loss of these microrecesses and correlates with the chronic pain syndrome (2,10,11,29,44,53). In this event, a flattening of the anterior border of the posterior subtalar joint facet may be noted (Figs. 2 and 3). However, both stress radiography and arthrography are seldom employed in the evaluation of sinus tarsi syndrome because the condition is usually readily identified with the clinical examination, and treatment is directed at the alleviation of symptoms.

With MRI, T1-weighted sequences of a normal foot produce high signal intensity within fatty structures of the tarsal sinus and canal contrasting the low signal intensity of the interosseous and cervical ligaments. In sinus tarsi syndrome, the normal fat signal intensity is replaced by fibrous-type tissue of low signal intensity on T1-weighted sequences (Fig. 4). Conversely, high signal intensities are found on T2-weighted sequences. This is believed to be a frequent imaging change noted in sinus tarsitis (11). STIR sequences are particularly useful in identifying fluid and the presence of marrow edema indicating areas of stress injury (Fig. 5). T2-weighted gradient echo sequences best reveal increased signal intensity within the sinus and canalis tarsi and are useful for elucidating the status of the ligaments. Normal ligamentous structures may be obliterated by ligamentous tear or inflammation. In patients without antecedent trauma who were undergoing MRI, the cervical ligament was identified in 69% and 88% of scans in the coronal and sagittal planes, respectively (28).

MRI has also demonstrated disruption of the lateral collat-

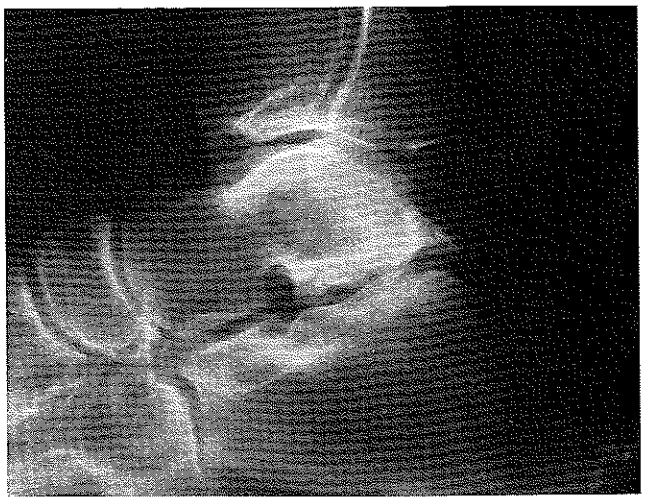


FIG. 2. This plain radiograph shows a flattened posterior subtalar joint facet and subchondral sclerosis in a patient with a history of chronic ankle sprains.

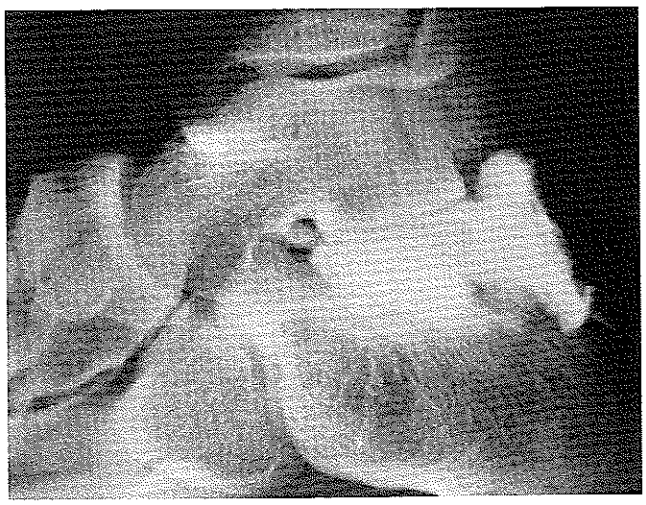


FIG. 3. A posterior subtalar joint arthrogram demonstrating the flattening of the anterior joint outline indicative of sinus tarsi syndrome.

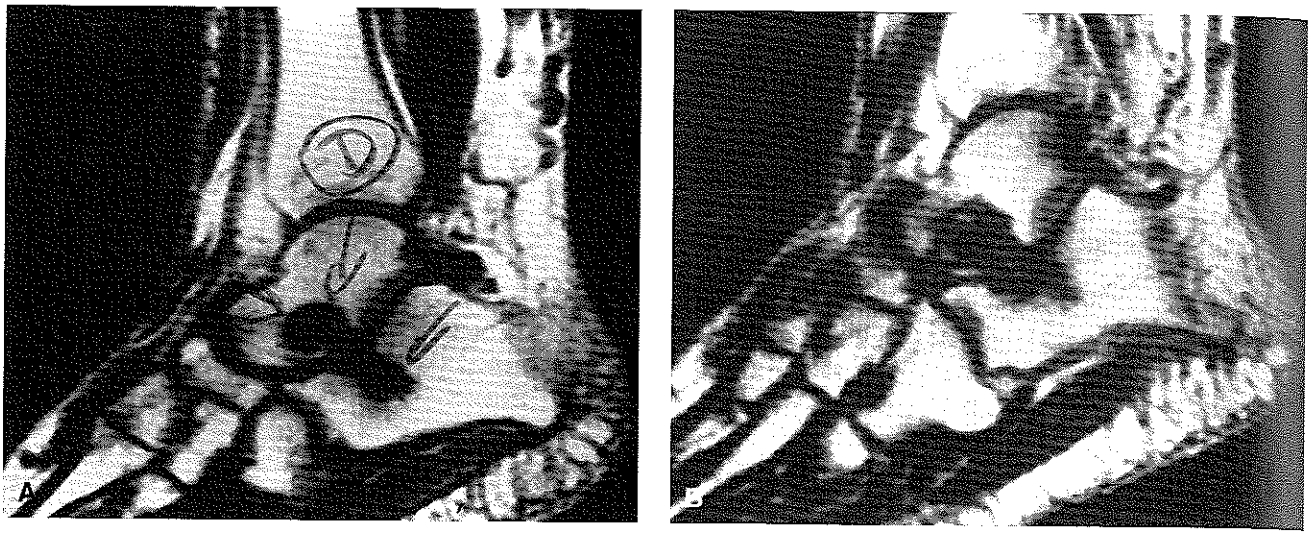


FIG. 4. A: This T1-weighted magnetic resonance imaging scan shows extensive changes within the sinus tarsi labeled *D*. Normal fat signal intensity of the sinus tarsi is replaced by tissue of low signal intensity. Decreased signal intensity is noted, obliterating the interosseous talocalcaneal ligament on this image. Additionally, significant subcortical erosion is identified within the critical angle of Gissane and the posterior subtalar joint facet. **B:** This demonstrates the interosseous ligament and subcortical erosions within the critical angle of Gissane. These findings, low signal intensity on both T1-weighted and T2-weighted images, indicate fibrotic changes within the sinus tarsi.

eral ankle ligaments in the majority of patients with sinus tarsi abnormalities. Three distinct MRI patterns were identified in Klein's study of 123 ankle MRI scans. In 85% of cases, a diffuse infiltration was seen within the sinus tarsi; 52% demonstrated a low T1 and T2 signal intensity consistent with fibrotic changes; 33% of patients demonstrated a low T1 signal with an increased signal intensity on the T2 phase consistent with chronic synovitis and nonspecific changes. Multiple abnormal fluid collections were identified in 15% of the scans, a finding consistent with synovial cysts (29).

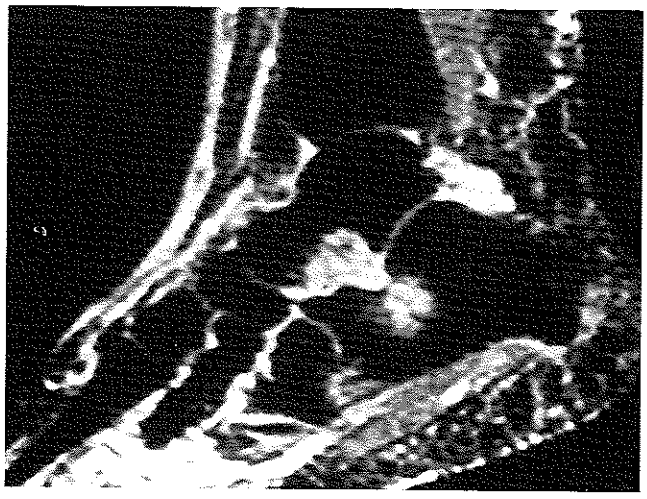


FIG. 5. This STIR magnetic resonance imaging scan illustrates the edematous changes within the sinus tarsi along with marked marrow edema within the critical angle of Gissane.

MRI may be considered in some patients with recalcitrant symptoms in whom traditional conservative treatment has failed. One may also consider this modality if the patient's symptoms are not well localized and if concomitant disease is suspected. However, because the MRI findings associated with sinus tarsi syndrome are usually nonspecific, the scan alone seldom influences treatment.

Treatment

Conservative treatment is usually adequate to alleviate symptoms in most patients and has been reported effective in 50% to 70% of cases (2,17,47,48). Typical measures consist of physical therapy modalities such as ultrasound and hydrotherapy, oral antiinflammatory medications, and local injections of anesthetic and steroids into the sinus tarsi. Various taping methods may allow for resolution of the inflammatory process if taping is applied in a manner that sufficiently restricts subtalar joint motion. Some compressive supports or orthotic devices have also been employed with success, depending on the patient and the mechanics of the foot. More aggressive immobilization with soft casting or a formal short leg cast may be considered.

Surgery

In the event of conservative treatment failure, surgical intervention may be required and has historically consisted of evacuation of the contents of the sinus tarsi (1,2,6,39,40, 45,54). Subtalar joint arthroscopy has also been advocated by some surgeons for subtalar joint decompression (55). The

goal of the surgical procedure is to decompress the sinus tarsi and to evacuate the ligaments and hypertrophic synovium. The extent of evacuation may vary among surgeons principally because of the controversy over removing the ligaments of the subtalar joint.

The incision is placed parallel to the relaxed skin tension lines over the sinus tarsi. At the more anterior aspect of the incision, one may encounter the intermediate dorsal cutaneous nerve. A communicating branch between the intermediate dorsal cutaneous nerve and the sural nerve may cross the sinus tarsi and may need to be sacrificed. The incision is deepened by sharp and blunt dissection to the level of the inferior extensor retinaculum. The peroneal tendons and sheaths may be noted in the lateral portion of the incision, and the extensor digitorum brevis muscle is visualized anteriorly. The deep fascia is then incised overlying the sinus tarsi, and the contents of the sinus tarsi are excised. The overlying portion of the extensor retinaculum may be repaired or simply excised, based on the surgeons' preference.

Some controversy exist about whether to excise the interosseous talocalcaneal ligament, and this decision is left to the discretion of the individual surgeon. Taillard et al. advocated excision of only the lateral half of the sinus tarsi, under the presumption that this controlled resection would spare the blood supply to the talus (2). However, in some patients, the ligament has been damaged, and this contributes to the inflammatory process. Considering this fact, as well as the failure to observe any potential adverse problem related to possible circulatory compromise of the talus, one may consider a more complete evacuation of the sinus tarsi. Once the tissues are removed, the posterior facet of the subtalar joint is inspected for degenerative changes. If significant osteoarthritis is present, then arthrodesis may be considered. Most often, intraoperative inspection often fails to elucidate meaningful disease. After closure, a sterile, compressive dressing is then applied. Protected weight bearing with a walking cast or surgical shoe is used for 2 to 4 weeks postoperatively.

In recalcitrant cases of sinus tarsi syndrome, a major rearfoot fusion may be required. Patients suffering from chronic pain may require a subtalar or triple arthrodesis to alleviate symptoms (2,5,6,33).

ADHESIVE CAPSULITIS

Adhesive capsulitis, pericapsulitis, or *frozen joint* is characterized by generalized periarticular pain and tenderness with severe loss of active and passive motion in all planes of the affected joint (56). Often described as a sharp, or tight, stretching sensation, patients may also recount a popping or cracking with motion. Classically, the process was described as an inflammatory manifestation affecting the glenohumeral joint. This intraarticular disease is thought to result from an active proliferation of fibrous exudate that accumulates over time, decreases the volume within the joint, and subsequently restricts motion (57). Few reports of ankle joint capsulitis have actually been reported, each the result of trauma (58–60), although some patients have been affected without antecedent injury. In this latter circumstance, some cases of inflammatory synovial lesions have been described as idiopathic (61), or they have been associated with systemic disease including the following: inflammatory arthropathy (56), diabetes (62,63) and other endocrinopathies, heart disease (62), connective tissue disease (64) and other seropositive arthropathies, infection, and autoimmune processes.

Although the pathologic features of adhesive capsulitis are the same regardless of the joint affected, the process as it affects the ankle affliction has been addressed by numerous authors (57–60,65–69). These patients present with chronic ankle pain and recurrent edema that may wax and wane over time and may contribute to a painful limited range of motion. Often associated with cast disease, symptoms may be evident immediately after cast removal or with a more insidious onset during progressive weight-bearing activities. Immobilization itself appears to contribute to this process and has been cited as an etiologic factor (56,57,70–72). Clinically, dorsiflexion and plantarflexion of the ankle joint are restricted, most notably at the extreme end range. Muscle splinting or guarding may be appreciated with these maneuvers. Tenderness with focal palpation of the ankle is usually present, particularly at the anterior and posterior ankle capsule. Calf muscle wasting secondary to limited function and chronic edema is common.

Pathophysiology

The characteristic pathologic features of adhesive capsulitis consist of fibrous thickening and secondary contracture of the ankle joint capsule. Because the entire joint capsule is affected by this process, the axis of motion becomes limited through all planes (56–60,70–72). Investigators have proposed that specific cytokines are responsible for the initiation and progression of the fibrotic process. Transforming growth factor-β, platelet-derived growth factor, and hepatocyte growth factor have also been specifically identified as contributing to the inflammatory and fibrotic process (73). In cases of limited adhesion, a localized fibrous band may bind the synovium or capsule in an isolated region resulting in ankle restriction to a lesser degree. These localized adhesions are commonly seen in the anterior ankle joint and are more easily remedied than a generalized adhesive ankle capsulitis (74).

The effects of adhesive capsulitis on bone mineral density have been evaluated, but not in the ankle. Studies in the shoulder demonstrate a significant decrease in bone mineral density of the proximal humerus when compared with the contralateral, unaffected limb. In patients with active disease, this decrease was significant (-5.6%; $p = .001$). The patients with a previous history of adhesive capsulitis showed differences in bone mineral density that were insignificant when compared with the contralateral, unaffected

limb (-1.5%; $p = .009$). Therefore, the loss of bone mineral density may be reversed over time with restoration of more normal motion (75). The reason for the reaction of the surrounding bone in adhesive capsulitis of the ankle is unknown.

Ancillary Imaging Techniques

The findings on plain radiographs may not be impressive, although diffuse osteopenia may be present (59,70,75). Traditionally, arthrograms have been used to diagnose adhesive capsulitis (10,11,20,44,73,76–78), and this is still a useful technique (Fig. 6). Arthrography, with or without computed tomography (CT), may demonstrate intraarticular foreign bodies, tears, or rents within tendon sheaths, as well as chondral or ligamentous lesions. A normal ankle joint can accept 9 to 16 mL of fluid. In the pathologic joint, only 3 to 5 mL of contrast material may be injected. Fibrosis and hypertrophic synovium add to the high intraarticular pressure, and a significant resistance to infusion can be anticipated in severe cases. The arthrogram typically reveals a diminished synovial space as well as hyperproliferative synovium, as evidenced by a scalloping margin about multiple loculated recesses. Normal anterior and posterior recesses of the ankle syndesmosis are obliterated. Lymphatic uptake of contrast, as evidenced by opacified lymph vessels, reflects the high pressure within the ankle joint (23,37). Arthrography itself can be therapeutic to a degree because the maneuver can disrupt small adhesions within the joint.

MRI is useful in delineating this generalized adhesive condition. Typically, T1-weighted, T2-weighted, and proton density images are obtained. Although T1, fat-weighted images best demonstrate marrow changes and lipid-laden structures, T2 and proton density images are best suited for delineating effusion, bursae, and pannus formation (66). With the advent of kinematic or dynamic MRI, the extent of adhesions and degree of ankle joint restriction can be more closely evaluated. Synovial thickening or pannus typically presents in a frond or leaflike distribution. This fibrous tissue echoes an intermediate or low signal intensity in chronic states. Pannus is typically surrounded by effusion, bursae, or adjacent tendon sheaths. In the presence of ongoing inflammation, hyperemia, and edema, signal characteristics are similar to those of fluid. Contrast enhancement aids in delineating effusion from pannus. This provides better contrast imaging for assessment of intraarticular structures and coincident disease such as osteochondral lesions or marrow edema (66).

Technetium bone scanning has been shown to produce abnormal patterns of uptake in patients presenting with the clinical features of adhesive capsulitis within the shoulder. Clunie et al. found this to be clinically significant and supported the reliability of this technique (64). However, the correlation with clinical disease and the reliability of this imaging technique for adhesive capsulitis in the foot and ankle have not been described.

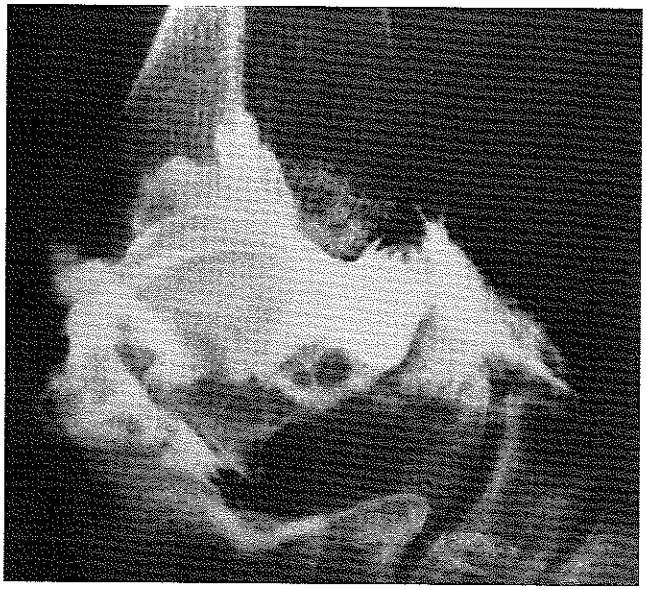


FIG. 6. Adhesive capsulitis of the ankle joint demonstrated by arthrography. The extreme irregularity of the joint outline indicates this condition.

Treatment

Physical therapy modalities are the primary means of conservative management (78–80). The goals of therapy are focused on reducing pain, releasing adhesions, and improving range of motion. These modalities can be used in combination or delivered in stages based on the individual response to therapy. The use of antiinflammatory agents in addition to electrical stimulation, hydrotherapy, ultrasound, or diathermy may be a good initial step. For recalcitrant pain and restriction of motion, hydraulic distention of the joint or intraarticular corticosteroid injection with continued range-of-motion exercise may provide benefit. In extreme cases, manipulation under general anesthesia may be required. Many, if not all, of these measures are unlikely to be totally successful. Residual stiffness and intermittent pain are to be expected to varying degrees and are likely to correlate with initial severity of the process.

Ankle arthroscopy may be employed for both diagnostic and therapeutic purposes (57,58,61,69,72,74,78–86). Exploratory ankle arthroscopy can be accompanied by synovectomy, removal of meniscoid or loose bodies, and arthroplasty for repair of transchondral lesions. The initial dilatory infusion and distention of the joint are themselves maneuvers that aid in releasing small adhesions. In cases of profound fibrosis and synovitis, penetration of the joint with the arthroscope may be accomplished, but one may not be able to visualize the joint adequately. At times, penetration of the joint may be impossible because of extensive disease or occult fibrocartilaginous overgrowth requiring conversion to an open surgical procedure. After adequate resection of hypertrophic synovium and adhesive lesions, copious lavage

is performed to filter out fibrous debris. Range-of-motion exercise may begin early in the postoperative period, and physical therapy modalities may also be employed to enhance restoration of motion.

OS TRIGONUM SYNDROME

Anatomic Review

The *os trigonum* is a triangular ossicle located at the posterior aspect of the talus adjacent to the lateral tubercle. The anterior surface articulates with the posterolateral process of the talus and the inferior surface articulates with the superior aspect of the calcaneus. In some patients, the ossicle is united to the posterior process of the talus by cartilaginous, fibrocartilaginous, or fibrous tissue. An os trigonum has been reported to be present in 1.7% to 15% of patients (87–99), and it is apparently more common bilaterally (91,100). It is rarely bipartite.

Numerous theories have been proposed regarding the origin of this ossicle (87,90–92,101–104). O'Rahilly indicated that these bones often have an ossification center separate and distinct from adjacent structures (90). This finding is consistent with McDougall's findings of a secondary ossification center for the posterior process of the talus that appears variably in patients between the ages of 8 and 11 years and usually unites with the main bone within a year (92). Whether the ossicle is, in fact, the result of a developmental anomaly or caused by a pathologic fracture, the clinical significance is of questionable value.

The os trigonum is a distinct entity, not to be confused with *Stieda's process,* a term that refers to the posterior extension of the lateral tubercle of the talus. Stieda described the ossicle as the result of an aberration in development (105), with the observation that it was indeed an ossicle, rather than a fracture of a bone as others would later suggest (102). When these ossicles are present, most remain asymptomatic (89). However, this anatomic variant is susceptible to fracture by virtue of its location. Fracture and chronic inflammatory conditions of this ossicle are common among athletes requiring forceful, repetitive ankle joint plantarflexion. Ballet appears to be the typical activity associated with this injury. However, numerous other sports have also been a source of injury, and this syndrome has also been noted to occur in the nonathlete (94,106–111).

A classification scheme has been described for the anatomic variations of the posterolateral talus (95) (Fig. 7). Type I is considered a normal tubercle without clinical consequence. Type II, Stieda's process, is an enlarged tubercle prone to injury when the ankle undergoes extreme plantarflexion. In this instance, the tubercle is compressed between the tibia and calcaneus, and the result is fracture. This fracture fragment can be the source of chronic ankle pain in the event of malunion or nonunion, which in the latter circumstance creates a loose body. Type III is an accessory bone, the os trigonum, which may be the source of discomfort because of repetitive microtrauma. Type IV is a coalition, or fused os trigonum, which forms a synchondrosis or syndesmosis with the talus (102,103,112). In some patients, an arthritic process may develop between the talar projection and the underlying calcaneus that leads to pain and loss of motion in the subtalar joint.

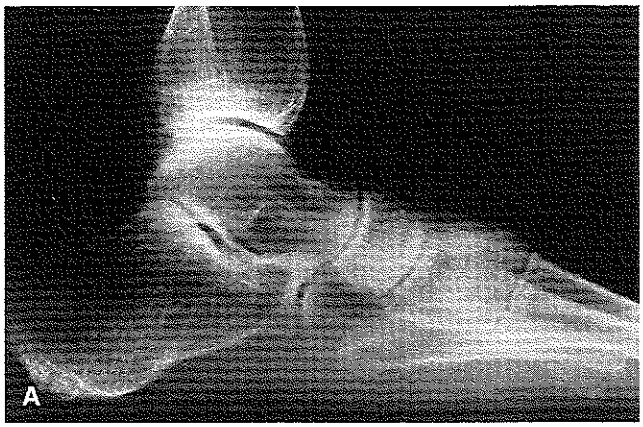
At the posterior ankle, the flexor hallucis longus tendon passes between the medial and lateral posterior talar processes, superior to the posterior talofibular ligament and inferior to the posterior talocalcaneal ligament. In extreme ankle dorsiflexion, the ossicle is compressed between the flexor hallucis longus tendon and the posterior talofibular ligament. With end-range plantarflexion, the superior aspect of the calcaneus directly compresses the ossicle against the tibia. The sheath of the flexor hallucis longus tendon is normally thickened in this region (107). Such thickening may predispose this segment of tendon to repeated traction injury in patients with high physical demands. Chronic inflammation of this and other local structures such as the posterior neurovascular bundle can create symptoms consistent with tarsal tunnel syndrome (110).

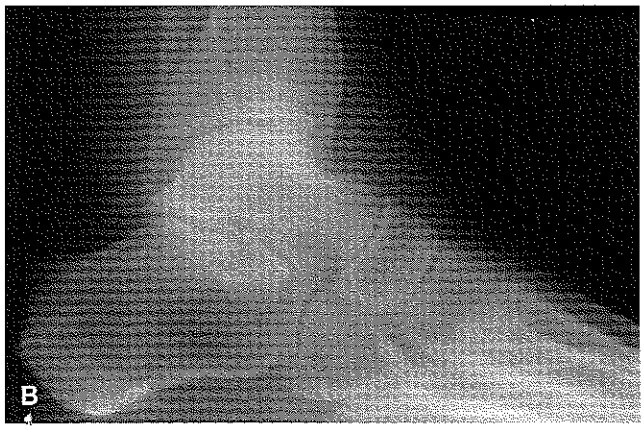
The posterior talocalcaneal ligament and the posterior talofibular ligaments attach to the posterolateral process of the talus. In the high-arched or supinated foot, traction is exerted on the posterolateral talar process by the posterior talofibular ligament. In the pronated foot, the posterior talocalcaneal ligament is the source of traction. As the calcaneus everts, the superior surface of the calcaneus is directed toward the tibia and possibly creates an impingement of the accessory ossicle. Extremes of motion in both plantarflexion and dorsiflexion may irritate the flexor hallucis longus tendon along the posterior talar tubercle.

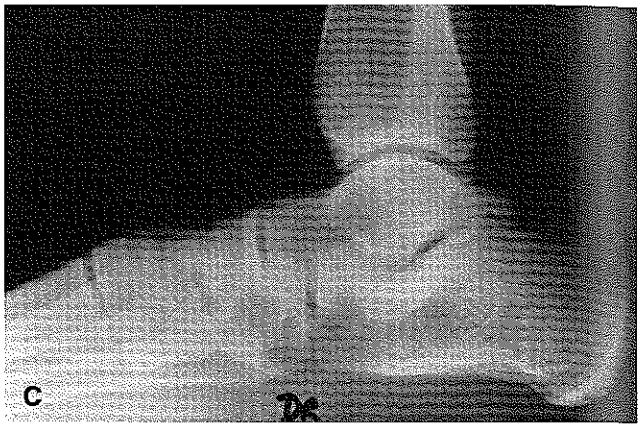
Clinical Presentation

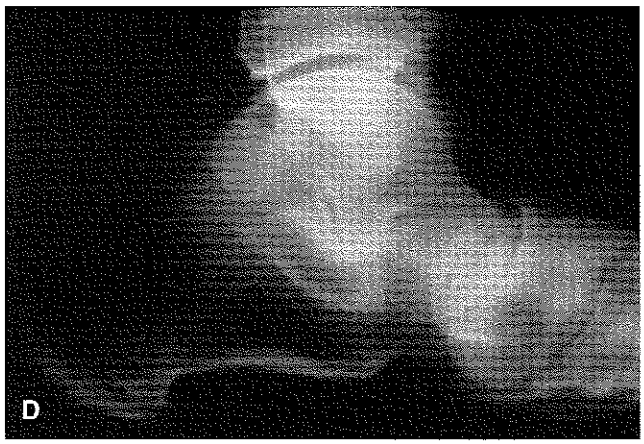
In *os trigonum syndrome,* the chief complaint may be one of poststatic dyskinesia, aggravated by weight-bearing activities. The pain is usually described in the posterior ankle as a deep, aching sensation. Decreased range of subtalar joint motion and peroneal spasm, similar to a tarsal coalition syndrome, have been described when the ossicle is fused to the posterior subtalar facet (109). Commonly, pain is greater at the end range of plantarflexion and dorsiflexion of the ankle, both weight bearing and non–weight bearing, and there may be an associated impingement of the flexor hallucis longus tendon (113–116). Manual muscle testing of the flexor hallucis longus tendon with dorsiflexion of the hallux may reveal tenderness in the posterior ankle. Pain may also be associated with subtalar joint motion and is usually greater during weight bearing on uneven terrain. Often, symptoms are relieved simply by rest. Ballet dancers best typify this patient profile, because their dance requires such extremes of ankle motion (23,96,107,111,114–122).

The onset of pain is often associated an ankle sprain that is recalcitrant to treatment. In the study by Fallat et al. of 639 ankle sprains, only 0.2% suffered from os trigonum syn-









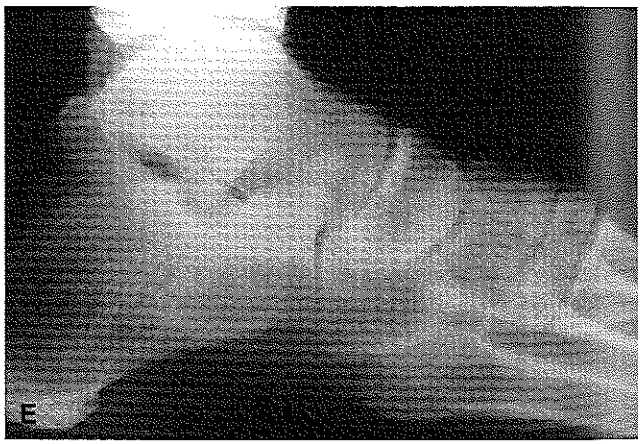


FIG. 7. A: Patient without evidence of an os trigonum for comparison of the morphology types defined by Watson and Dobas. **B:** Type I. A posterior process united to the talus by cartilaginous, fibrocartilaginous, or fibrous tissue. It is not overly prominent and is considered a normal tubercle without clinical consequence. **C:** Type II. Stieda's process is an enlarged or elongated posterolateral talar process. This anatomic variant is susceptible to fracture. **D:** Type III. An accessory bone, the os trigonum, which may be the source of discomfort because of repetitive microtrauma. **E:** Type IV. A coalition, or fused os trigonum, forms a synchondrosis or syndesmosis with the talus.

drome (123). However, the coincidence of this injury with ankle sprains is likely underreported because this syndrome is unfamiliar to most general physicians. An os trigonum does not mature or ossify until the second decade of life and usually fuses within 1 year after its appearance (between the ages of 8 and 11 years), and as such, the pain syndrome commonly affects an age group ranging from 20 to 35 years (107,124).

Some of the mechanisms that may be responsible for chronic pain in the region include repetitive microtrauma, Shepherd's fracture (fracture of the posterior talar process) (125), tendinitis or tenosynovitis of the flexor hallucis longus, direct compression of the ossicle between the tibia and calcaneus, and periostitis within the sulcus of the long flexor.

Diagnostic Imaging

Differentiation of a painful os trigonum from Shepherd's fracture has been the impetus for exploring the use of imaging techniques for diagnosis. Although considered an academic exercise by some investigators (35), the subject has been addressed and reconciled based on morphology of the bone on plain radiographs. A sharply defined fracture edge may be consistent with acute Shepherd's fracture (Fig. 8),

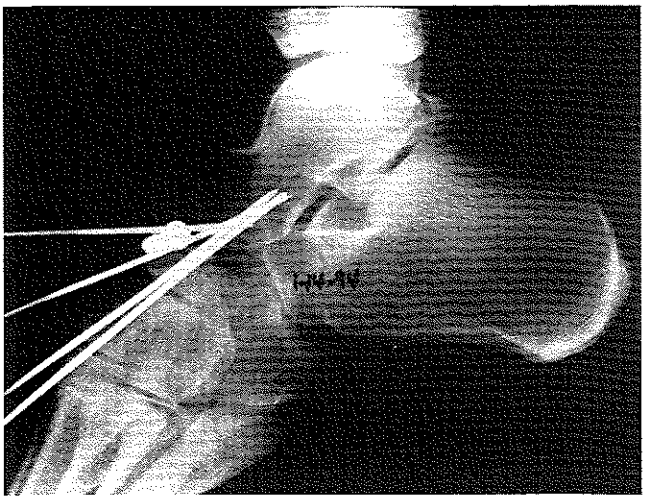


FIG. 8. A fractured os trigonum is identified coincident with a navicular fracture.

whereas the presentation of a smooth, well-rounded osseous body is indicative of an os trigonum. One can argue that an old Shepherd's fracture will have undergone a change in morphology over time, but in cases of chronic pain the issue is not of clinical significance. Additionally, an enlarged posterolateral process may incite chronic pain, and, once again, the importance of differentiation remains academic because the treatment remains the same. Stress lateral radiographs with the ankle in maximal dorsiflexion and maximal plantarflexion, or evaluating the patient under fluoroscopy, may also help to determine whether the ossicle is mobile.

Technetium scans may be of assistance for differentiating bone lesions from soft tissue disease as in posterior capsulitis or other inflammatory processes. Tenograms may aid in localizing stenosing tenosynovitis of the flexor hallucis longus tendon (126). CT and MRI findings may support the diagnosis of a symptomatic os trigonum or Shepherd's fracture, but these tests are not considered essential in the majority of cases. MRI is thought to be superior for investigating this pain syndrome (127,128).

Treatment

Conservative therapy may be initiated with some form of splinting or immobilization and antiinflammatory medications. Injections of steroid may also be employed. Foot strappings simulate the function of an orthotic device and may be useful as a test for those patients in whom orthotic therapy may be beneficial. A weight-bearing or non–weight-bearing short leg cast may also be instituted.

Surgical treatment may be necessary in patients with recalcitrant pain resulting from os trigonum syndrome or fracture. The ossicle can be resected through a curvilinear posterolateral incision. The architectural landmark for the incision plan is the posterior edge of the peroneal tendons, anterior to the sural nerve. The sural nerve and lesser saphenous vein may be identified, isolated, and retracted inferiorly from the field. Once the superior surface of the calcaneus is identified, blunt dissection medially provides the exposure necessary. The flexor hallucis longus tendon can be identified by placing the hallux through its range of motion. An incision is carried over the posterior subtalar joint, and the osseous fragment or prominence is resected. The bone of the superior calcaneus and posterolateral talus is then palpated to appreciate residual prominences or bony debris. Recontouring of these surfaces may be required. Hypertrophic or fibrotic tissues should be identified and resected. Any scarred or hypertrophic areas of the flexor tendon sheath may also be excised. The wound is closed in anatomic layers, and a compressive dressing is applied. Also of note is that the surgical procedure has been performed using arthroscopic techniques (76,83,113,114).

Some controversy exists on the necessity of absolute immobilization after this procedure, and the postoperative course may vary accordingly. Most authors employ a period of immobilization, usually 2 to 3 weeks, whereas some advise return to weight bearing and range of motion as soon as possible (83,111,114–118,120–122,124,129).

TIBIOFIBULAR DIASTASIS AND SYNDESMOTIC INJURY

Separation or *diastasis of the tibiofibular joint* may occur in some patients with ankle injuries, yet it often remains unrecognized. Whereas the lateral ankle is the site for 85% of all ankle sprains, syndesmotic sprains are the next most commonly encountered, approximately 10% of the time. In a study of 1,344 ankle injuries, diastasis was found in 15 patients, a prevalence of 1% (130). Other investigators have reported a greater incidence, but with patient populations that were comparatively smaller (131). As clinicians have become more aware of the entity, more recent studies have demonstrated an incidence as high as 17% to 20% (130–144). Nonetheless, the injury has garnished remarkably little interest in spite of its lingering effects in some patients. Given the complex arrangement of the ligaments at this level, it is easy to understand that injuries affecting the ankle could produce strain across the syndesmosis that results in variable disruption of the inferior tibiofibular joint.

Although this type of ligamentous injury is considered a relatively common complication of ankle fractures, it may remain undiagnosed in the absence of osseous damage. Because little swelling is associated with acute disruption of these ligaments, the injury may not be appreciated, possibly leading to a symptomatic chronic tibiofibular diastasis. A history of an ankle sprain that was slow to heal or recalcitrant to conservative therapies is typical (129), and the description of instability and pain may seem out of proportion to the inciting event.

Gerber et al. performed a prospective study of ankle injuries to provide updated data on the epidemiology and disability associated with ankle injury. In 104 ankle injuries, 96

were ankle sprains, including 16 syndesmotic injuries. At 6-month follow-up, all patients had returned to work, although 40% suffered from residual symptoms. A syndesmotic ankle sprain, regardless of grade, was predictably associated with chronic ankle symptoms (132). Ligamentous laxity and previous ankle sprains were not considered reliable predictors of chronic dysfunction or pain.

Anatomic Considerations

Sarrafian described three ligaments that function to support the distal tibiofibular syndesmosis. The anterior inferior tibiofibular ligament, the posterior inferior tibiofibular ligament, both deep and superficial components, and the interosseous ligament (Fig. 9). The anterior inferior tibiofibular ligament is a flat fibrous band originating from the anterior aspect of the lateral malleolus and coursing obliquely from distal lateral to proximal medial to insert onto the anterolateral tubercle of the tibia. The longer distal fibers approach 25 mm in length and pass over the lateral shoulder of the talus. The distal fibers extend to the origin of the anterior talofibular ligament. The anterior inferior tibiofibular ligament may be present in two or three bands or as one multifascicular unit.

The posterior inferior tibiofibular ligament is composed of a superficial and deep segment. The superficial component originates from the posterior border of the fibula just above the digital fossa. These fibers run proximally and medially as they insert on the posterolateral tibial tubercle. Some fibers may extend to the lateral border of the groove for the tibialis posterior tendon. The deep segment is the transverse ligament, which creates a labrum beneath the posterior tibial margin deepening the articular surface of the tibia. The posterior medial border of the fibula is nonarticular, but it is filled by the transverse ligament. The ligament contacts the talar dome in this region and creates a distinguishing beveled triangular facet on the posterior lateral border of the talar dome.

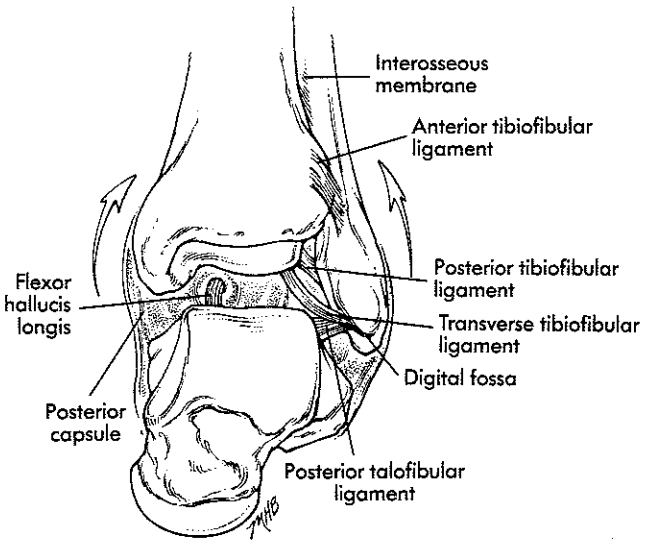


FIG. 9. A diagram of the ankle syndesmosis.

The interosseous ligament is composed of a thick layer of short fibers combined with adipose tissue and vessels. It forms a vault over the syndesmosis coursing from the anteroinferior aspect of the distal fibula and inserts at a similar level on the distal tibia. The fibula and tibia have a small, crescent-shaped surface area of articulation inferior to the interosseous ligament.

Cadaver studies have been used to determine the contribution of each ligament to the overall stability of the ankle based on resistance to a 2-mm diastasis (133). The anterior inferior tibiofibular ligament provided 35%, the deep posterior inferior tibiofibular ligament provided 33%, the interosseous ligament supplied 22%, and the superficial posterior inferior tibiofibular ligament provided 9% of overall ankle stability.

Pathology

Chronic symptoms may result from poorly reduced ankle fractures, soft tissue interposition or impingement syndromes, and occult ligamentous disruption. The syndesmotic ankle sprain has been contrasted with the lateral ankle sprain, and the former is considered the more substantial injury. Boytim et al. investigated this injury in a professional football team over a 6-year period and found that syndesmotic ankle sprains required significantly longer treatment and greater recovery times in comparison with players afflicted with lateral ankle sprains (134).

Chronic instability of the distal tibiofibular syndesmosis can develop after bimalleolar and trimalleolar ankle fractures (135). Clinical and functional recovery from these injuries largely depends on adequate reduction of the fibular malleolus. Reduction of the syndesmosis is an important step in attempting to minimize subsequent dysfunction and arthritic degeneration (135–143). Ankle diastasis without fracture, although an uncommon injury, has been reportedly associated with high-contact, collegiate, and professional sports (130,131,133,134,142,144–147). Although rare, tumor has been reported as causative (148), and congenital diastasis of the distal ankle syndesmosis has also been documented (149,150). Interposition of Tillaux's fracture between the tibia and the fibula can mimic syndesmotic separation (151).

Diagnosis

Syndesmotic injury may be noted after an "ankle sprain" (123,144,147,152–155). In acute settings, and in the absence of fracture, pain may be elicited over the tibiofibular syndesmosis, as opposed to the lateral ankle ligaments themselves. If the condition is identified early after the injury, the specific location of the edema may also indicate which structures may have been injured. However, after several hours, the swelling becomes more diffuse. Muscular splinting or reflex

spasm may obscure any clinical demonstration of laxity at this level.

In patients with chronic symptoms, the clinical findings may not be impressive. Pain in the ankle, edema, and tenderness overlying the syndesmosis are typical, and the absence of swelling in other anatomic areas may help to confirm the diagnosis. A snapping within the tibiofibular joint may at times be appreciated with manipulation. This response may best be elicited by stabilizing the patient's anterior tibia with one hand and the calcaneus in the other. The heel can then be manipulated in the frontal plane, by inverting and everting the calcaneus. The talar body then shifts in the medial or lateral direction and evokes the tell-tale snapping within the syndesmosis.

Pain may be present with dorsiflexion as the wider anterior portion of the talus is rotated into the ankle mortise and strain is produced across the anterior and posterior tibiofibular ligaments. Alternately, with external rotation of the ankle, the lateral shoulder of the talus rotates against the fibula and imposes a strain on the anterior inferior tibiofibular ligament. In some patients, the application of manual medial lateral compression of the malleoli elicits pain (147,156,157). This maneuver has been referred to as the *distal compression test.* Compression at the midleg level has also been reported to produce pain and is referred to as the *proximal compression test.* In this examination, the compression produces a separation of the syndesmosis rather than direct compression of the joint (157).

Imaging

Although numerous measurements have been described for determining tibiofibular diastasis with radiographs (158–166), three of the most common methods include the medial clear space, the syndesmotic clear space, and the amount of tibiofibular overlap. The *medial clear space* is measured at the level of the talar dome on the ankle mortise radiograph. It is defined as the straight-line distance from the lateral border of the medial malleolus to the medial border of the talar body (Fig. 10). This value should be within 1 to 2 mm of the uninjured, contralateral ankle (149), or less than 4 mm when it is evaluated individually (134,167).

Tibiofibular overlap is defined by the horizontal distance from the medial border of the fibula to the lateral border of the anterior tibial prominence on the anteroposterior (AP) or mortise radiograph (Fig. 11). Various interpretations of this measurement have been reported (139,155,167–171). Normal findings are subject to interpretation because a small amount of motion is available at this joint and varies among patients. Therefore, comparison films with the uninjured contralateral limb may be helpful in determining the normal values for the patient. Normal variant schemes for tibiofibular overlap include the following:

Greater than or equal to 10-mm overlap on the AP radiograph or greater than a 1-mm overlap on the mortise radiograph (138,167)

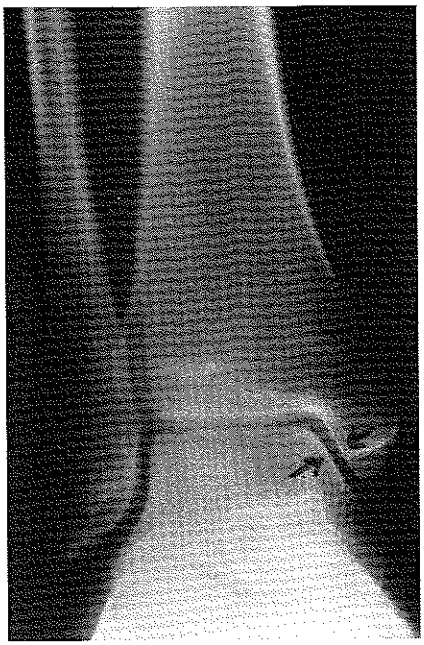


FIG. 10. The medial clear space is measured at the level of the talar dome and is defined as the straight-line distance from the lateral border of the medial malleolus to the medial border of the talar body. This value should be within 1 to 2 mm of the uninjured contralateral side.

Overlap of at least one-third the width of the fibula (133)
Overlap greater than 42% of the fibula on the AP radiograph or greater than 1 mm on the mortise view (169)
Overlap greater than 6 mm (155)

Many other more specific parameters have also been described for further interpretation (137–140,165,166,170–172).

The *syndesmotic clear space* is defined as the horizontal distance between the incisura fibularis (tibial notch) and the medial border of the fibula (Fig. 12). This may be assessed on both the AP and mortise views, and the measurement is made 1 cm proximal to the tibial plafond. A normal clear space is less than 6 mm on AP and mortise views. Measurements on the contralateral uninjured limb should be within 1 to 2 mm of the affected limb (135,160,162,164,170,172).

Stress testing may be helpful in demonstrating syndesmotic dysfunction and instability. The external rotation stress test involves applying external rotation force to the ankle, which will produce a widening of the mortise on plain radiographs in affected patients. Pain may also be noted during this test (129,141,147,173).

CT scans may prove to be more effective in evaluating the syndesmosis than plain radiographs. In cadaveric specimens, investigators have noted that all specimens with a 1- to 2-mm diastasis and 50% of specimens with a 3-mm diastasis could not be appreciated on plain films. However, these deviations were appreciated with CT scans (155). Ultrasound

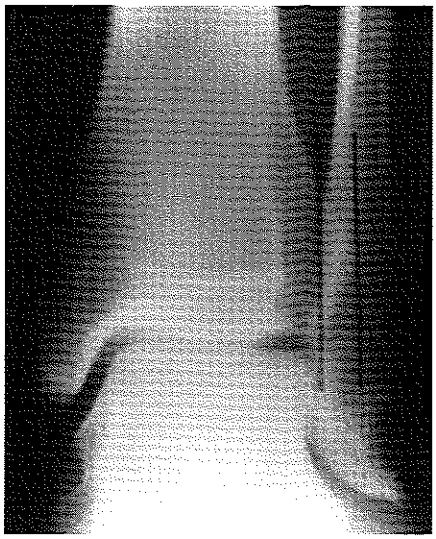


FIG. 11. The tibiofibular overlap is defined by the horizontal distance from the medial border of the fibula to the lateral border of the anterior tibial prominence on the anteroposterior or mortise radiograph. The measurement is taken approximately 1 cm proximal to the ankle joint.

has been used to diagnose injury to the anterior syndesmosis with specificity similar to that of arthrography (174). One prospective study of patients with acute ankle trauma had high interobserver agreement in the preoperative evaluation of the syndesmotic injury using MRI (175).

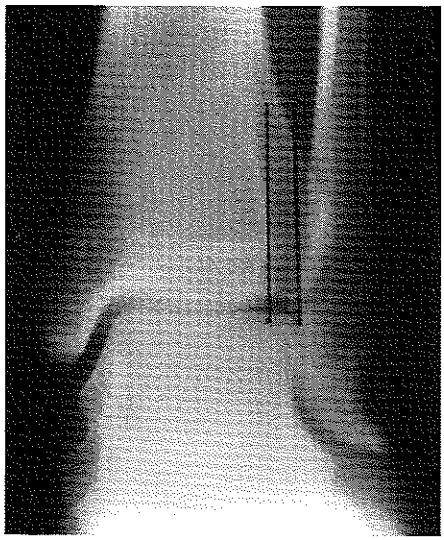


FIG. 12. The syndesmotic clear space is measured 1 cm proximal to the tibial plafond on the anteroposterior or mortise view. This distance, from the incisura fibularis (tibial notch) to the medial border of the fibula, is normally less than 6 mm. It should measure within 1 to 2 mm when compared with the uninjured contralateral limb.

Classification of Tibiofibular Diastasis

Edwards and DeLee categorized these injuries as either *latent diastasis* or *frank diastasis,* depending on the radiographic presentation (133). Frank diastasis was evident on plain radiographs, whereas the latent injury was identified only with the assistance of stress radiographs. It was proposed that latent diastasis required no closed reduction and would respond well to cast immobilization. Patients with frank diastasis were believed to require anatomic reduction of the ankle mortise before casting to achieve optimal functional outcome. Four types of diastasis without fracture have been identified based on radiographic changes in the ankle mortise, with treatment dependent on the severity of the findings (147).

Type I. This is a straight lateral fibular subluxation with an increased medial clear space. No plastic deformation of the fibula is appreciated. The recommended treatment consists of open reduction and internal fixation with the use of a transyndesmotic screw. The deltoid and anterior inferior tibiofibular ligaments may be interposed within the joint obstructing reduction of the ankle mortise.

Type II. This is a straight lateral fibular subluxation with plastic or angular deformation of the distal fibula. Open reduction and internal fixation often require a fibular osteotomy for adequate restoration of the ankle mortise accompanied by a transyndesmotic screw.

Type III. Posterior rotation and subluxation of the distal fibula is present. Because the posteroinferior tibiofibular ligament remains intact, the fibula becomes entrapped behind the talus and is rotated posteriorly.

Type IV: The talus is dislocated superiorly causing a wedge effect between the tibia and fibula resulting in an increase of the intermalleolar distance. Most authors believe that treatment of type III and IV injuries are best accomplished by open reduction and internal fixation with the use of a transsyndesmotic screw (141,142,176–184).

Alternatives for Treatment

Traditionally, open reduction and internal fixation have been considered the definitive treatment for frank tibiofibular diastasis. However, few alternatives have been suggested for patients with persistent pain or chronic instability. With the advent of arthroscopic techniques, diagnosis and treatment of ankle syndesmotic injury may be enhanced (179,185,186).

Primary repair of the anteroinferior tibiofibular ligament has been advocated using the plantaris or sections of the peroneal tendons. Kelikian recreated this ligament using the distal portion of the long extensor tendon to the fourth toe (186). Ankle arthrodesis may be considered in cases of significant pain or degenerative changes.

Patients with chronic diastasis and recalcitrant pain may require fusion of the syndesmosis. In chronic injuries with

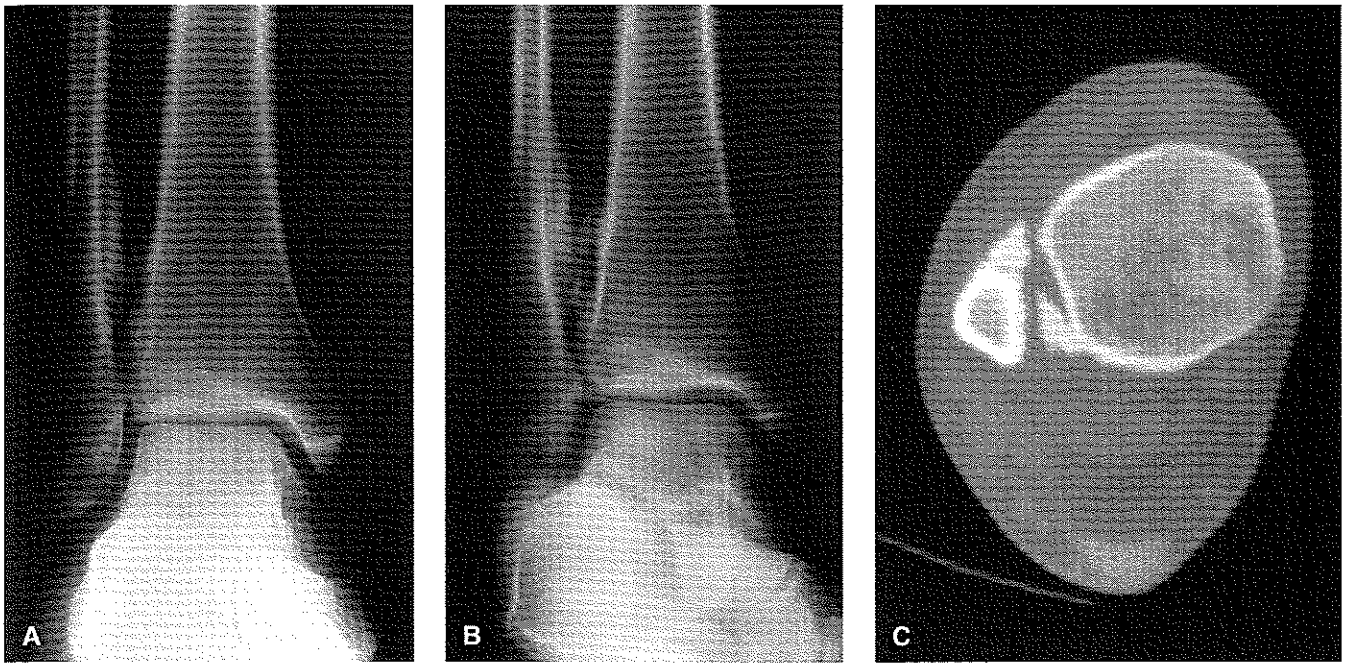


FIG. 13. Mortise **(A)** and internal oblique **(B)** radiographs and a computed tomography image **(C)** in a patient 18 months after a supination external rotation ankle fracture. Extensive calcification within the interosseous membrane, fibular shortening, and chronic tibiofibular diastasis are evident.

persistent dysfunction, substantial arthritic changes are probable and will likely prove difficult to resolve with conservative efforts (129,141–143,176,177,186) (Fig. 13). Controversy exists over the optimal position for fusion. Some authors believe that maintaining the ankle in a slight degree of dorsiflexion (5 degrees) will achieve a more stable mortise for function (129,141). Critics argue that if the fusion site is reduced with the ankle fully dorsiflexed, then the fibula will be shifted laterally and externally rotated, and the result will be persistent widening of the mortise and possible instability with motion. Other investigators believe that the most important goal in syndesmotic fusion is to avoid overtightening of the joint and subsequent restriction of motion. Therefore, a position of close to end-range dorsiflexion may be desired (176,177).

CHRONIC MEDIAL ANKLE PAIN

Chronic medial ankle pain is less common than chronic lateral ankle pain, and patients most often present with a history of significant ankle trauma. Medial ankle pain and instability after traumatic injury may result from overuse syndrome, rupture of the deltoid ligament, transchondral fracture of the medial shoulder of the talus, impingement after open reduction of the ankle, or residual ligamentous laxity. Although the clinical presentation is thought to be rare, the list of differential diagnoses is long (Table 1).

Chronic Medial Ankle Instability

The function of the deltoid ligament is to stabilize the medial ankle. Disruption of this complex is thought to constitute 15% of all ankle sprains (187,188). However, the true incidence has likely been obscured because the injury may go undetected in patients with concomitant trauma.

TABLE 1. *Differential diagnosis of chronic medial ankle pain*

Posttraumatic pain
Isolated medial ankle fractures involving deltoid ligament
Fibular fractures with interposition of the medial collateral ligaments
Unidentified malleolar fracture in presence of Achilles injury
Collicular fractures, anterior and posterior
Painful internal fixation device
Pseudarthrosis
Nonunion or malunion medial malleolar fracture or avulsion
Recurrent inversion ankle sprains
Chronic deltoid rupture
Medial meniscoid lesion of ankle
Anterior or medial ankle impingement
Ankle epiphyseal injury or congenital arrest
Tibialis posterior tendon dysfunction
Severe pes valgo planus
Varus deformity
Ligamentous laxity
Stress fracture
Tumor
Accessory ossification center

Over time, there has been considerable discussion regarding the necessity of primary repair of the deltoid ligament associated with ankle fractures (189–192). Traditionally, the emphasis has been placed on anatomic realignment of the fibula and stabilization of the syndesmosis when necessary. The medial ankle ligaments have been considered of lesser significance because complete deltoid ruptures, when left untreated, have reportedly not led to significant ankle instability (193–197). However, other investigators have found that patients suffer from chronic pain syndromes after such injuries, not because of instability, but rather from chronic overuse of the attenuated ligament (189,198,199). Insufficiency or rupture of the deltoid ligament has been shown to have a profound effect on the anterior and lateral shift of the talus, by decreasing total tibiotalar contact area (200,201).

TABLE 2. *Inventory of deltoid ligament function*

Superficial deltoid ligament: resists rear foot eversion
Tibiocalcaneal ligament: limits talar abduction
Anterior tibiotalar ligament: limits talar plantarflexion and external rotation
Posterior tibiotalar ligament: limits talar dorsiflexion and external rotation
Intermediate tibiotalar ligament: limit talar abduction and external rotation
Posterior and intermediate tibiotalar ligaments together with the anterior talofibular ligament: limits internal rotation of the talus
Deep deltoid ligament: resists external rotation of the talus
Deep anterior tibiotalar ligament: limits talar plantarflexion and external rotation
Deep posterior tibiotalar ligament: limits talar dorsiflexion and external rotation

Anatomy

The deltoid ligament is composed of two layers, superficial and deep, with the deep layer the stronger of the two. The superficial component is a wide, triangular ligament composed of the superficial anterior tibiotalar, tibionavicular, tibioligamentous, and tibiocalcaneal ligaments. The tibiocalcaneal ligament is the strongest of the group, approximately 2 cm in length and 1 cm in width at insertion. These superficial deltoid structures originate from the anterior colliculus of the medial malleolus (202,203). The superficial posterior tibiotalar ligament is considered a variable component originating from both the anterior and posterior colliculus and inserting at the posteromedial talar tubercle near the flexor hallucis longus tunnel (203).

The deep deltoid is shorter yet stronger than the superficial layer. The deep anterior tibiotalar ligament comprises the anterior compartment of the deep deltoid. It courses from the anterior colliculus and intercollicular groove to the medial aspect of the talus distal to the anterior border of the comma-shaped facet. The deep posterior tibiotalar ligament originates from the anterior colliculus, the intercollicular fossa, and the posterior colliculus, and it courses inferiorly, posteriorly, and laterally to insert on an oval elevation beneath the tail of the comma-shaped facet on the medial talus. Measurements of 1.5 cm width and 1.0 cm thickness have been reported at the origin, as well as a length of approximately 1.5 cm. This is the strongest element of the entire deltoid complex (203,204).

Rasmussen et al. conducted a functional analysis on cadaveric models to define the role of the deltoid ligament components (193). Function was assessed by the change in mobility about the ankle after transection of individual deltoid ligaments in varying sequences. These results are summarized in Table 2.

Evaluation

In patients with insufficiency of the deep deltoid ligament, the point of maximal tenderness is usually localized between the medial malleolus and the sustentaculum tali. The entire span of the deltoid may present with variable tenderness depending on the extent of initial injury. Induration or subtle edema may result from local chronic inflammation with an associated increase in local temperature. Concomitant fullness about the retromalleolar tendons, the tibialis posterior and flexor tendons, may be evident because of chronic irritation from an excessively lax medial ankle with persistent local inflammation. A valgus calcaneal stance position or valgus ankle may serve as a deforming force that may interrupt resolution of the acute process and may encourage prolongation of symptoms (198,205–207).

Manual manipulation of the ankle with valgus stress evokes pain. Comparison with the contralateral, uninjured ankle may reveal an increased range of motion because the talus is allowed to tilt excessively within the mortise, unopposed by normal ligamentous constraint. Muscular guarding of the retromalleolar tendons may be present as they splint against painful subtalar and ankle joint motion. Capsular defects, evident at the extreme anterior or posterior portion of the medial ankle joint capsule, can evoke a focal crepitance at end range of motion in the sagittal plane.

Medial ligament insufficiency may be visualized radiographically by creating a valgus stress across the ankle to assess for widening of the medial clear space and talar tilt (196,199,208) (Fig. 14).The unstable medial ankle shows an increase in talar tilt in comparison to the contralateral uninjured ankle. An increased medial clear space of greater than 3 mm has been correlated with deltoid ligament disruption. However, the position of the foot is important during the stress evaluation because the normal ankle shows an increased medial clear space when the foot is plantarflexed (209). In addition, some normal ankles have also been shown to exhibit a small amount of talar tilt on valgus stress radiography (199).

MRI may be helpful in identifying the deltoid ligament and the extent of damage (187,188,210–216). Coronal images may provide a full longitudinal view of individual components of the deltoid ligament. Axial slices provide in-

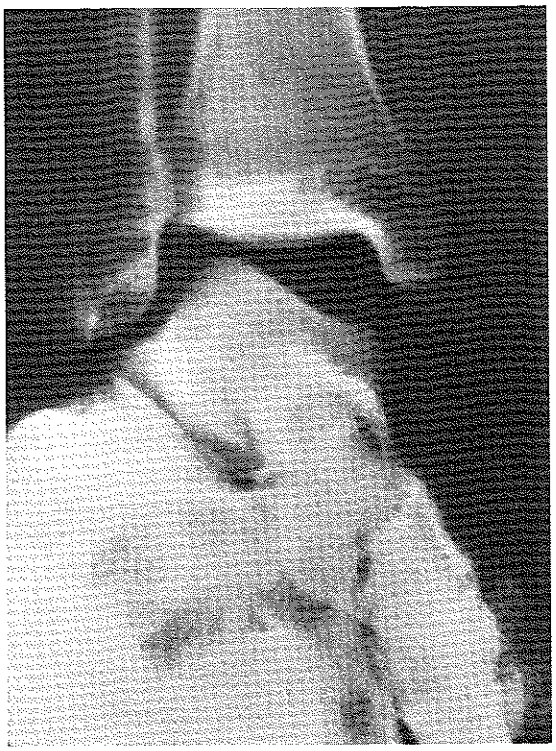


FIG. 14. A stress eversion radiograph showing positive findings.

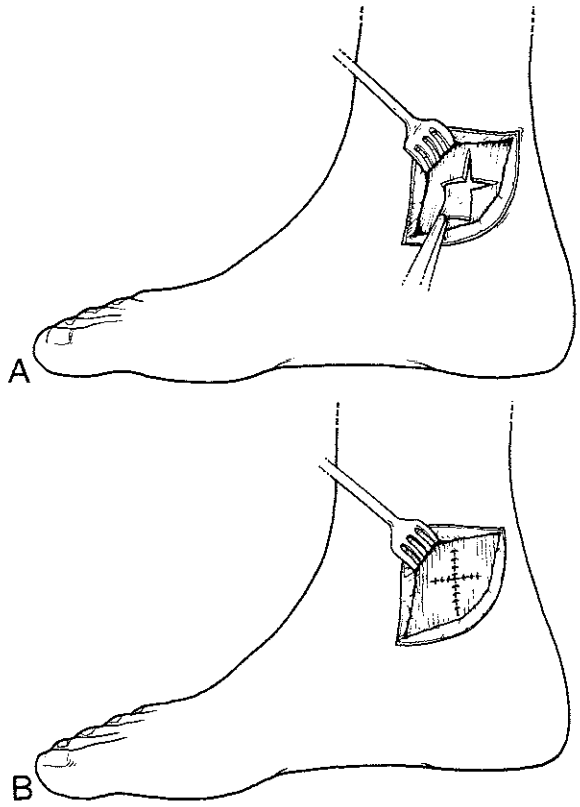


FIG. 15. DuVries repair of a lax deltoid ligament. **A:** The ligament is incised in a cruciate pattern. **B:** The incised margins are sutured, and the resulting scar tissue provides medial stability.

formation regarding associated tendinous structures and neurovascular elements. Specific pulse sequences such as T1-weighted, T2-weighted, STIR, gradient echo sequences, and fat-suppression imaging can be implemented on consultation with the radiologist. The appearance of deltoid injury has been noted to be strikingly similar to those changes noted in lateral ankle injuries (187). There is also a growing interest in the use of ultrasound for evaluation of the collateral ankle ligaments in both acute and chronic injuries. However, in one study comparing these two imaging modalities, ultrasound demonstrated injury in only 21% of the cases where deltoid ligament disease was previously identified by MRI (217).

Treatment

Minor degrees of medial ligamentous laxity compounded by malposition of the heel and ankle may respond to a combination of external stabilization such as taping, casting, orthotics, or ankle-foot orthotic devices with or without physical therapy. Should conservative measures fail, numerous different surgical approaches are available. Ligamentous repair techniques are grouped into three categories: imbrication, delayed primary repair, and ligamentous reconstruction with tendon transfers or grafting techniques. The selection of the specific procedure may depend on the condition of the deltoid ligament as determined by clinical evaluation and ancillary studies (218,219).

A transverse or hockey stick incision may be used for exploration and repair of the deltoid ligament and associated structures. Ligamentous imbrication may be employed to eliminate insufficiency by tightening the attenuated portion of the ligament or by removing excess ligament similar to a capsulorrhaphy. Repair of the ligament is then achieved under physiologic tension. Duvries described a simple method of achieving repair of deltoid laxity by incising the affected portion of the ligament in a cruciate pattern (220). The four arms are then repaired, eliminating excessive fibers, and closure is achieved resulting in four linear scars, presumably providing strength and, therefore, medial ankle stability (Fig. 15).

Modifications of pes valgus procedures have also been used to address ligamentous laxity (220–222). Schoolfield's technique would include detachment of the deltoid ligament at or near the tibial insertion. As the foot is held in an inverted position, the ligament is reattached to the tibia and periosteum proximal to the area of the initial detachment. This maneuver allows for elimination of redundancy within the deltoid and restoration of physiologic tension across the medial ankle. Delayed primary repair of the deltoid using an end-to-end method may also be performed.

Reconstruction or reinforcement procedures involve harvesting portions of local tendons for transfer to recreate the

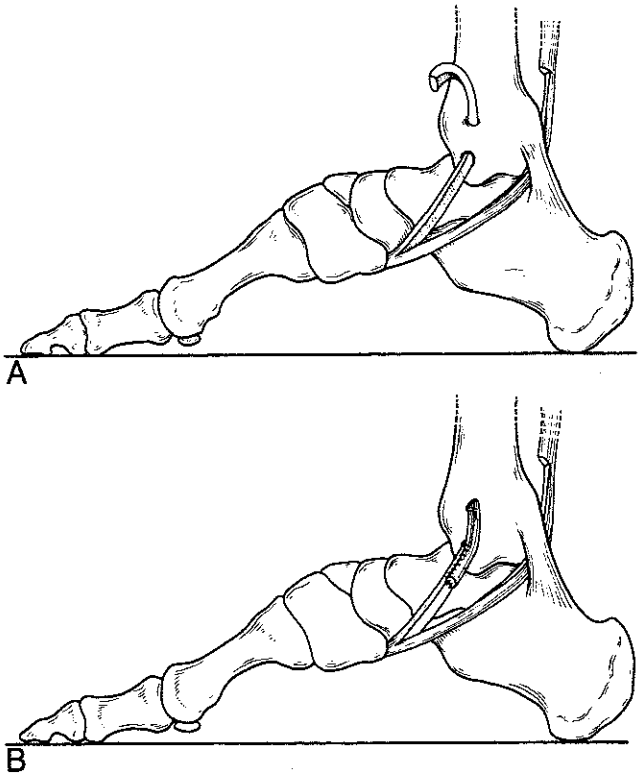


FIG. 16. Wittenberger and Mallory method for correcting chronic medial ankle instability using a tendon graft. **A:** Split portion of the tibialis posterior tendon is obtained, leaving the insertion intact. It is passed from inferior to superior through a drill hole in the medial malleolus. **B:** Split portion of the tibialis posterior is then sutured back down onto itself.

ligament. Free tendon grafts have been described and may prove beneficial when the integrity of the deltoid ligament is suspect. For example, Wittberger and Mallory detailed a technique transferring the anterior half of the posterior tibial tendon to support the medial ankle (223) (Fig. 16). The tendon is split longitudinally with the anterior slip passed through a hole on the medial malleolus, then folded back distally and sutured onto itself.

Medial Malleolar Stress Fractures

The diagnosis of *medial malleolar stress fractures* can be challenging because the complaint of pain may be vague and generalized. The history is typically devoid of overt trauma with an insidious onset of pain and dysfunction presenting over time. Stress fractures of the medial malleolus are uncommon (224–230). Traditionally, serial plain radiographs and clinical evaluation have been employed to identify occult or stress fractures. Pain is usually elicited by direct palpation. An additional diagnostic tool is the *tuning fork test.* When a stress fracture is present, applying the vibrating instrument to the symptomatic area generally exacerbates the pain.

Ultrasound applied directly to the affected area may result in similar amplification of pain. The ultrasound vibration modality has also been used in evaluating patients with acute ankle fractures (231). The sensitivity for detecting fracture was 39%, and the specificity for identifying nonfractured ankles was 83%, both with a confidence interval of 95%. Although there was only a positive predictive value of 26%, a negative predictive value of 90% was noted, suggesting that a negative result may rule out the presence of a fracture.

MRI can be used to diagnose stress fracture or to identify associated intramedullary edema and joint effusions (212,213,225,231–234). This modality is perhaps most valuable in cases of suspected occult or stress fracture recalcitrant to conservative therapy and with persistent negative findings on plain radiographs. The usefulness of MRI has been demonstrated in monitoring the healing of medial malleolar stress fractures as well (225). Radioisotope imaging reveals the hyperemia associated with bone healing in all three phases of the triphasic bone scan. Increased uptake during the third phase is diagnostic of a stress fracture.

Pseudarthrosis

Pseudarthrosis of the medial malleolus is a potential complication after ankle fractures (235–252). Some authors have concluded that only a mobile pseudarthrosis is likely to be symptomatic and of clinical significance. Symptoms have been noted to be most pronounced during the first 2 years after injury and to diminish slowly over time. The prognosis has been correlated with the width of the pseudarthrosis as well as its location. Wide lesions located proximally on the malleolus resulted in more substantial pain and disability (253,254).

Sneppen reviewed the long-term course in 119 cases of pseudarthrosis in 1,717 ankle fractures involving the medial malleolus (198). One hundred fifty-six of these 1,717 cases (9.1%) demonstrated radiographic signs of nonunion at greater than 9 months of follow-up; 119 patients were evaluated for long-term follow-up. Approximately 33% of the 119 patients with nonunion went on to spontaneous healing, a finding indicating that the pseudoarthrosis had a tendency to unite, although slowly, over time; 46% of the pseudarthroses united over a range of 16 to 18 years after the initial injury. The clinical symptoms associated with the pseudarthrosis subsided considerably over the first 2 years, and after more than 8 years complaints were interpreted as mild. The presence of a rearfoot valgus was shown to be significant. The number of patients with a pseudarthrosis and evidence of arthritic changes within the talocrural or subtalar joint was 45%, no greater than expected after conservative treatment in ankle fractures (198,255–257). Of those patients with pronounced arthritis, the incidence was higher in those with a united pseudarthrosis. Finally, no hallmark symptoms were identified, nor was an association found between width of the lesion and symptoms (Fig. 17).

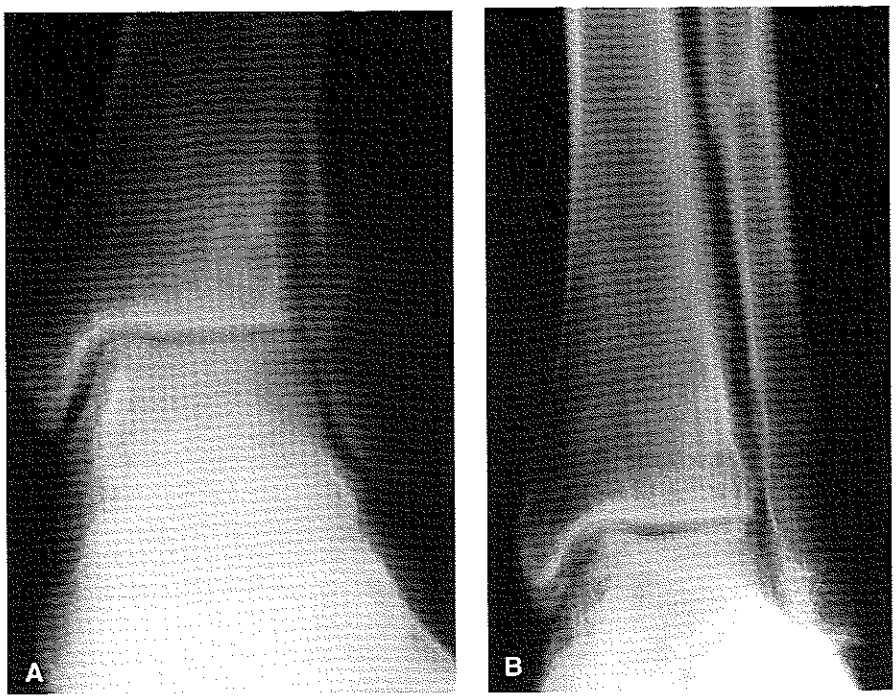


FIG. 17. A chronically symptomatic medial malleolar nonunion 2 years after open reduction of a pronation external rotation ankle fracture. **A:** An anteroposterior radiograph of the ankle shows an ununited collicular fracture of the medial malleolus. Notice the fine line lucency with a subtle sclerotic border separating the fracture from the medial malleolus. **B:** Internal oblique ankle view helps to delineate the ununited fracture fragments of the malleolus as well as degenerative changes and calcification within the medial gutter of the ankle, the syndesmosis, and along the interosseous membrane. The proximal fibular fracture is easily appreciated on this view.

PERONEAL TENDON SUBLUXATION AND DISLOCATION

Anatomy

The peroneal muscles originate from the lateral surface of the fibula. As the tendons course distally, a single synovial sheath begins approximately 4 cm superior to the lateral malleolus and ends approximately 4 cm distal. The confluent sheath bifurcates distal to the fibula, and each tendon is encircled with an independent sheath. These muscles are active during midstance, firing after forefoot loading and ceasing before toe off. Three key anatomic structures that are important for peroneal tendon function are the superior peroneal retinaculum, the retrofibular groove, and the fibroosseous ridge of the fibula.

The peroneus longus tendon changes direction at three points along its course: angulating behind the fibula through the retromalleolar sulcus (posterior and lateral to the peroneus brevis tendon), as it runs inferior to the trochlear process along the lateral wall of the calcaneus, and laterally as the tendon turns approximately 90 degrees over the lateral surface of the cuboid to cross the plantar arch.

The peroneus brevis tendon courses deep to peroneus longus in the leg and crosses anterior to the peroneus longus tendon within the retromalleolar sulcus. The peroneus brevis crosses the calcaneofibular ligament at approximately 30 degrees, courses above the peroneal trochlea, and inserts into the styloid process of the fifth metatarsal base.

The *peroneus quartus muscle* is an anomalous structure with a frequency of occurrence reported between 17% and 21.7% The most common origin (63%) is from the muscular portion of peroneus brevis in the distal one-third of the leg. This tendon inserts into the peroneal tubercle of the calcaneus and is thought to assist in pronation because persons with this muscle have a low occurrence of lateral ankle sprain (258,259). Sobel et al. identified this structure in 21.7% of 124 cadaveric specimens and in 18% of these found definite attrition of the peroneus brevis tendon within the fibular groove (260).

The peroneal tendons course distally into the leg, where they are covered by two major retinacula. The superior peroneal retinaculum originates from the lateral border of the retromalleolar groove and the tip of the fibula, and it courses obliquely and distally, superficial to the superior peroneal tunnel. It inserts onto the lateral surface of the posterior calcaneus and the aponeurosis of the Achilles tendon. Although there is considerable variability regarding the insertions of this structure (261), its biomechanical role remains undisputed. It functions to prevent subluxation or dislocation of the peroneal tendons about the fibular malleolus, and it stabilizes their position within the peroneal sulcus. Purnell found that tendon position was unaffected with manipulation

of the foot after sectioning of the inferior retinaculum (262). In contrast, after sectioning the superior peroneal retinaculum, the tendons were able to dislocate or sublux anteriorly with dorsiflexion and eversion of the foot. Because the calcaneal band of the superior retinaculum is parallel to the calcaneofibular ligament, it may play a secondary role in lateral ankle stability.

The inferior peroneal retinaculum is a continuum of the lateral root of the inferior extensor retinaculum. It takes origin from the posterolateral rim of the sinus tarsi with both superficial and deep fibers. Superficial fibers course plantarly and posteriorly, cross the peroneal trochlea, and insert superior to the calcaneal tubercle, whereas deep fibers insert onto the trochlear process. This structure forms two fibrous tunnels (inferior peroneal tunnel) over the trochlear process, a superior tunnel for the peroneus brevis and an inferior tunnel for the peroneus longus.

Acute Peroneal Tendon Dislocations

Acute peroneal dislocations can occur from a variety of athletic activities. Typically, subluxation injury is the result of indirect trauma, and although direct injury causing peroneal dislocation is rare, Alm et al. reported 2 such cases in 1979 (263). Although rarely reported, subluxations can occur strictly within the peroneal sulcus, with one tendon subluxating over the other, resulting in pain and dysfunction (264–266). Snow skiing injuries are perhaps the most commonly reported cause of acute peroneal dislocation. A frequency of occurrence approaching 1 in every 200 skiing accidents has been reported (267). However, many other athletic activities have been associated with this disorder including football, rugby, basketball, water skiing, dance, gymnastics, mountain climbing, and combat training (263,264,268–273).

Dislocation of the peroneal tendons from their retrofibular sulcus often results from a rapid dorsiflexory force across the ankle with simultaneous, violent, reflex contraction of the peroneal tendons and ankle joint plantarflexors (274–277). Although some controversy exists regarding the position of the foot and ankle at the time of injury, it stands to reason that a dorsiflexed ankle places linear tension on the peroneal muscles and tendons and forces them anteriorly against the fibula. With pronation, the subtalar joint everts, and the foot produces a laterally directed force that lends the peroneal tendons a mechanical advantage against the retinaculum. This lateral force promotes strain and ultimately insufficiency or failure of the superior peroneal retinaculum that results in tendon dislocation from the peroneal sulcus (272,278). In downhill skiing, if the ski tip becomes imbedded in the snow, the skier is pitched forward by sudden deceleration, and a dorsiflexory force is imposed on the ankle while the foot is in a pronated or valgus position (273,274,279). An alternate situation exists during the swift turns of downhill skiing maneuvers. These rapid, alternating motions direct the force of weight bearing onto the medial edge of the ski and put the lateral ankle ligaments and superior peroneal retinaculum under an enormous degree of tension, possibly leading to anterior dislocation of the tendons (270). Another mechanism that has been proposed is violent contracture of the peroneal tendons across a supinated subtalar joint and dorsiflexed ankle, which is believed to tighten the calcaneofibular ligament to such a degree that it compresses the tendons against the superior retinaculum resulting in strain and tendon luxation (270,272).

In the acute setting, these injuries are often misdiagnosed and regarded as ankle sprains. The patient typically presents for evaluation with the tendons in the reduced position (280). The patient may recall the sensation of a snapping or pulling around the lateral ankle, and in some instances a crack or a pop may have been heard during the injury. There may be an associated feeling of ankle weakness. Acute pain, although severe at the time of injury, subsides relatively swiftly and results in a generalized syndrome of pain and edema about the fibula and peroneal tendons. This can make accurate diagnosis more difficult. If the patient is seen within the first 2 hours after the injury, then edema may be visible overlying and just proximal to the lateral malleolus and extending along the tendons. Acute pain is usually elicited with palpation along the posterior margin of the retromalleolar groove. If the patient is seen more than 2 hours after the injury, significant edema, ecchymosis, and pain are usually noted at the retromalleolar sulcus. Tenderness and redislocation can be reproduced with ankle dorsiflexion and subtalar joint eversion against resistance. Concurrent injuries have also been reported with these proposed mechanisms of disruption, most notably lateral ankle instability, Achilles tendinopathy, fractures of the lateral talar process, subluxation of the posterior tibial tendon, and ankle fracture (267,270,280, 281).

Eckert and Davis described three patterns of injury based on observations noted in the surgical repair of dislocated peroneal tendons (274) (Fig. 18). A grade I injury was characterized by the separation of the retinaculum and periosteum from the fibrocartilaginous ridge of the lateral malleolus. The tendons were frequently found overlying the fibula and, when reduced, were unstable only under tension. This finding was noted in 51% of their patients. In grade II injuries, the fibrous lip of the fibula was elevated in addition to the retinaculum. This accounted for 33% of the injuries. Grade III lesions were evident in 16% of patients. In this instance, a thin fragment of bone and the fibrous lip avulsed from the underlying fibula with the deep fascia and retinaculum still attached. In all cases, the attachment of the superior peroneal retinaculum was ruptured at the interface between the fibula and the retinaculum. In grade II and III injuries, the tendons were usually found over the lateral malleolus and were unstable when reduced. Oden later added a fourth pattern of injury, seen when the retinaculum is torn from the posterior fibula and is found lying deep to the peroneal tendons within the peroneal sulcus (282).

Other authors have discovered a simple elevation of the

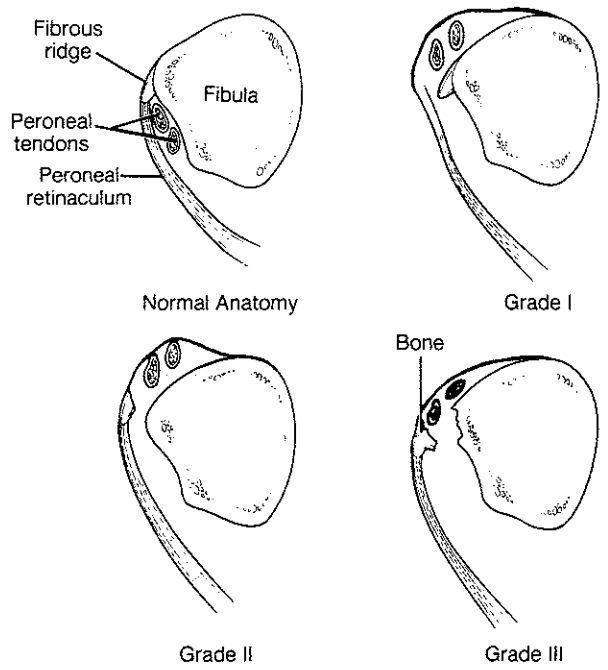


FIG. 18. Eckert and Davis classification of injuries to the peroneal retinaculum. Normal position *(upper left)* of the peroneal tendons. The sulcus is augmented laterally by a fibrocartilaginous ridge. Grade I injury *(upper right)* in which the retinaculum has separated from the fibrous ridge. It has not been torn. Grade II injury *(lower left)* demonstrates the fibrous ridge has detached from the fibula along with the retinaculum. Grade III injury *(lower right)* is a grade II injury with a small fragment of bone also detached.

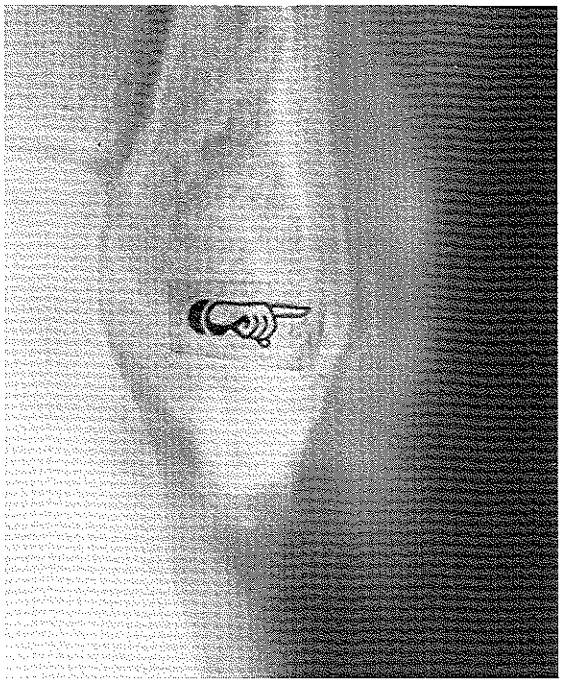


FIG. 19. This fleck fracture seen lateral to the lateral malleolus is produced from a peroneal retinacular avulsion of cortical bone and is indicative of peroneal dislocation.

retinaculum resulting from hematoma formation (263, 273,274,283). A dissecting hematoma can undermine the periosteum, deep fascia, and retinaculum and can allow subsequent tendon dislocation without frank retinacular tear or rupture. Despite these conflicting accounts of intraoperative findings, the classification scheme described by Eckert and Davis is still employed for intraoperative grading of peroneal subluxation or dislocation injuries.

Imaging

Radiographic evaluation with plain films may reveal a small avulsion or "fleck" fracture off the lateral aspect of the distal fibula. This small fracture fragment is oriented parallel to the long axis of the fibula and represents an avulsion of bone from the lateral ridge of the fibula. This is the hallmark of peroneal subluxation or dislocation injury and is recognized on the mortise or internal oblique ankle views (262,273,284–286). The presence of this avulsion fracture has been noted on radiographs in 10% to 50% of reported cases (263,270,273,275,281,287–289) (Fig. 19).

Treatment

Conservative treatment of acute peroneal dislocation generally consists of a below-knee cast for 6 weeks. However, conservative therapy is generally met with failure, rates ranging from 50% to 100% (268,269,273,290,291). In contrast, Stover and Bryan reported excellent results in 5 patients who were casted non–weight bearing and in plantarflexion, for 6 weeks (270). Eckert and Davis believed that this method of treatment to be futile in cases of chronic subluxations (274). McLennan reported 9 out of 17 good to excellent results using taping, J-pads, and crutch walking with 3 weeks of non–weight bearing (272).

Because of the poor results that may be encountered with conservative measures, surgical intervention has been employed for repair of the acutely dislocated peroneal tendons. A J-shaped incision is made just posterior to the lateral malleolus and extending about 1 cm distal to the tip of the fibula. The incision is deepened to the level of the peroneal retinaculum. The deep fascia is then incised just posterior ot the lip of the retromalleolar groove. From this point, the grade of the injury determines the specifics of the procedure.

In grade I injuries, the anterior edge of the incised fascia is tagged to the intact cartilaginous lip with nonabsorbable suture. The posterior fascial edge is then brought up over the anterior edge and is sutured to the cartilaginous lip. With grade II dislocations, the anterior edge of the fascia and the cartilaginous lip are repaired with nonabsorbable suture. This repair may be facilitated by placing drill holes into the edge of the fibula. The posterior fascial edge is repaired as previously described. The avulsed osteocartilaginous fragment in grade III lesions may be repaired by suture, pins, or screws, depending on the size of the fragment. Postopera-

tively, a below-knee cast is applied for 6 weeks. Weight bearing may be instituted after 3 weeks if desired.

Chronic Peroneal Subluxation or Dislocation

In the chronic disorder, the complaint of lateral ankle pain with activity is associated with a painful snapping of the lateral tendons and frequently a sense of lateral ankle instability. Concomitant lateral ankle ligament injury may also be present. When palpating the peroneal tendons, attention should be directed to the retrofibular sulcus. Manipulation of the foot and ankle may confirm the diagnosis by dorsiflexing the ankle and everting the foot against resistance, thus encouraging subluxation of the tendons. Palpation during this maneuver may reveal crepitus, possibly associated with lateral ridge fracture. In some patients, simple repetition of ankle dorsiflexion and plantarflexion or a circumductory motion of the foot can achieve the same results. Knee flexion in conjunction with external rotation of the foot also encourages subluxation. Visible anterior subluxation of the peroneals is pathognomonic. In some patients, an audible click is noted. Spontaneous relocation after this maneuver can be expected (273).

The *peroneal tunnel compression test* may help to elucidate whether the patient has concomitant peroneal tendon disruption (260). Recreating the force exerted by the superior peroneal retinaculum against the peroneal tendons is believed to elicit the pain associated with peroneus brevis split tears. Pain, crepitus, triggering, clicking, and anterior subluxation may be elicited with this maneuver. The patient is placed in a sitting position with the knee flexed over the edge of the examining table, the foot and ankle hanging in an equinus position. For the right foot, the examiner's right hand is placed behind the ankle with the fingertips grasping the posterior edge of the patient's fibula in the region of the superior peroneal retinaculum. The left hand then resists the patient during voluntary eversion of the foot. Pain during the maneuver is considered a positive examination. If the examiner's left hand is too far distal, the calcaneofibular ligament will be palpated, tenderness here representing ligamentous injury common in chronic ankle instability (276). The *wet leather sign* is noted as a palpable crepitance or squeaking of the injured tendon (260). In an alternate method, the examiner places the hand across the ankle anteriorly with the thumb extending across the posterior ridge of the fibula to the calcaneus. Compression of this area during dorsiflexion and eversion elicits similar symptoms.

Chronic peroneal subluxation or dislocation is often the sequela of an acute anterior dislocation of the peroneal tendons. In the acute injury, subluxation may cause mild longitudinal intratendinous tears that are mended by fibrotic tissue. The morphology of the tendon changes with chronic injury and results in a flattened, boomerang shape or a split tendon just distal to the fibula (265,266) (Fig. 20). Tears and tendon flattening are usually noted distal to the tip of the fibula and may extend proximally 2 to 5 cm (260). Recurrent subluxation can be significantly disabling, often associated with stenosing tenosynovitis and intratendinous defects responsible for the chronic pain syndrome.

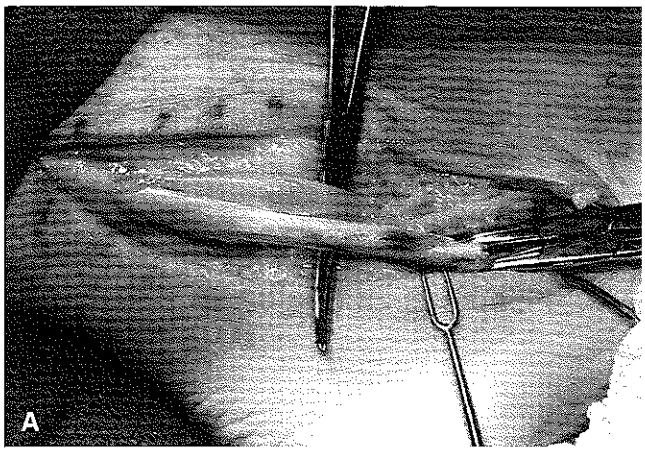
In patients presenting without antecedent trauma, anatomic variations of the retrofibular groove or insufficiency of the superior peroneal retinaculum are thought to be predisposing factors (261,270,292–295). The incidence of congenital peroneal tendon dislocation has been reported in neonates and infants, and spontaneous resolution can be expected in most of these patients (296).

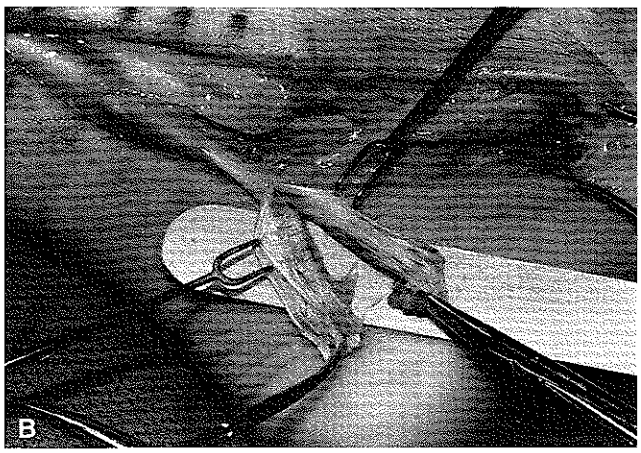
Special Anatomic Considerations

Anatomic variants in the distal fibula were first described by Edwards (297). He reported that 82% of the cadaver specimens demonstrated a definite posterior sulcus ranging from 5 to 10 mm in width. The morphology of the sulcus varied from flat to convex, 11% and 7%, respectively. In 70% of the fibulae, a bony ridge 2 to 4 mm high was noted about the lateral edge of the retromalleolar groove. This ridge, when present, was approximately 3 to 4 cm in length. Edwards believed that this ridge was insufficient for maintaining the tendons within the groove. Therefore, the peroneal tendons, when subluxating over this ridge, would suffer from chronic friction and inflammation resulting in tenosynovitis and insufficiency tears. Sobel et al. elaborated on the mechanism of this injury by citing four anatomic constraints possibly responsible for chronic peroneus brevis tendon wear: a sharp posterior edge of the fibula, compression against the fibula imposed by peroneus longus, a shallow posterior fibular groove allowing tendon subluxation, and a lax superior peroneal retinaculum. After cadaveric examination, Sobel graded split tears as follows: grade I, a splayed tendon; grade II, a partial-thickness split less than 1 cm; grade III, a full-thickness split 1 to 2 cm; and grade IV, a full-thickness split more than 2 cm (238).

Most surgical studies do not report rupture of the retinaculum, but rather an avulsion of periosteum or a thin cortical margin of bone often with associated intratendinous defects (267,273,274,279,280,282,284,287,298,299). With soft tissue avulsion from the fibula, a potential space of redundant fascia is created that allows the peroneal tendons to sublux with dorsiflexion and eversion of the foot. Overt rupture of the retinaculum is considered rare, although it has been reported (274).

Other causes and anatomic factors have been discussed, and many mechanisms for peroneal dislocation injuries have been proposed (270,272,273,300–303). Anomalies include the congenital absence of the retinacula or posterior fibular ridge and a bifid peroneus brevis muscle (285,300,304–307). Predisposing foot types of cavovarus and valgus are considered prone to this injury. Posttraumatic retinacular attenuation and prior inversion ankle injury are also considered factors contributing to peroneal subluxation and dislocation. Geppert et al. illustrated the deleterious effects of lateral ankle instability on the superior peroneal retinaculum (194).





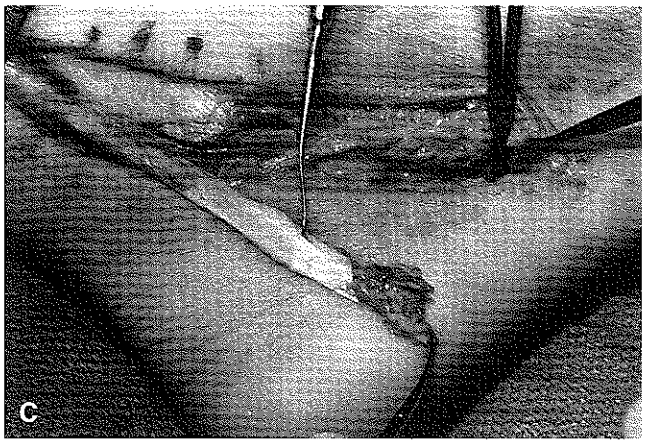


FIG. 20. Surgical exploration in a patient with chronic peroneal tendon symptoms. **A:** The peroneus longus tendon lies within the inferior aspect of the wound, and the peroneus brevis tendon is superior in this right ankle. Notice the discoloration of the tendons with areas of hemorrhagic synovitis most impressive distally. **B:** Intratendinous degeneration throughout the distal tendon; multiple longitudinal tears, mucoid degeneration, and hemorrhagic synovium throughout this "mop-end" tendon. **C:** Chronic intratendinous tears result in flattening of the peroneus longus at the inferior tip of the fibular malleolus that allows subluxation of the brevis tendon from beneath the peroneus longus. The distal tip of the fibula is visualized within the proximal incision, and its sharp posterior margin is easily appreciated. The skin hook illustrates the extent of an intratendinous longitudinal tear.

The strain of ankle instability causes laxity in the retinaculum that leaves the ankle susceptible to dysfunction. These investigators believed that this retinaculum serves as an adjunctive restraint to ankle inversion because the calcaneal band is in parallel alignment with the calcaneofibular ligament.

Rarely reported, *subluxation within the peroneal sulcus* has been described (264–266,306). In this disorder, gross tendon subluxation is not identified clinically because the tendons are simply rolling one over the other beneath the retinaculum. The mechanism of injury is thought to be traumatic, resulting from prior inversion ankle sprains or chronic tendinopathy. Chronic intratendinous tears result in flattening of the peroneus longus at the inferior tip of the fibular malleolus and may allow subluxation of the brevis tendon from beneath the longus (265,266,308). This phenomenon may be appreciated with manipulation and palpation inferior to the distal tip of the fibula. The subjective complaint is indistinguishable from that of traditional peroneal subluxation. The history reflects an insidious onset of a painful popping of the tendons that is aggravated by excessive weight-bearing activity. A high degree of clinical suspicion is required to identify this process.

In cases of chronic subluxations, the mechanism may be simple, repetitive inversion ankle injury (268–270,292). These recurrent strains of the lateral ankle ligaments and superior peroneal retinaculum lead to insufficiency and tendon dysfunction. Over time, this continual trauma leads to attenuation of the retinaculum and attrition of the peroneus brevis tendon (260,266,292,309–313).

According to the Eckert and Davis classification, any injury beyond stage I exposes the peroneal tendons to the sharp border of the fibular ridge and results in high mechanical friction and wear of the tendon. *Peroneal split syndrome,* characterized by pain and instability associated with longitudinal tears within the peroneus brevis tendon, is often accompanied by peroneal subluxation (260,276,292,299,309,310), and it is believed to be the result of overuse and wear (260,310,314). Acute plantarflexion and inversion of the ankle are thought to cause the injury. Often, such pathologic findings are accompanied by hypertrophy of the peroneus longus. T2-weighted MRI images show high intratendinous signal intensity within either the peroneus brevis or peroneus longus tendon in as many as 60% of patients with peroneal disease. Similar findings have been noted with or without actual peroneal splits (315). MRI evaluations have revealed that peroneal subluxations are slightly more common in patients with peroneal tendon splits (315). Chronic peroneal subluxation was noted in 25% of cases with peroneal splits, and it was believed to be accentuated by a flat or convex

fibular groove. Variations in tendon anatomy such as an extremely distal extension of the peroneus muscle belly, the presence of peroneus quartus, and regional osseous morphology such as an enlarged peroneal tubercle are readily identified using this technique (292). Yu and Shook maintained that MRI is not required in all cases of peroneal tendinopathy or subluxation; rather, they considered it a modality reserved for cases recalcitrant to conservative therapy or in patients suffering from chronic symptoms (276).

Lateral ankle effusions may represent tendon diseases such as tenosynovitis or intratendinous tears and may warrant ancillary studies to aid in determining the extent of injury and prognosis. Hutchinson and Gustafson found that chronic injury with an associated lateral effusion usually indicates significant tendinopathy (298). A diagnostic injection of a local anesthetic has been proposed as a means of differentiating the pain of tendinopathy versus recurrent subluxation (266,316).

Imaging

Tenography has been used in the past for evaluation of peroneal tendon sheath disorders, but generally it is unreliable for detecting peroneal splits because they are often obscured by synovial folds (267,317,318). However, with CT or MRI, an abnormal tendon position or deviant shape of the retrofibular groove may be identified (3,53,319,320). CT, using osseous and soft tissue windows, may help to delineate the anatomic variability of the distal fibula and surrounding soft tissue elements. As such, it may elucidate damage to the peroneal retinaculum, the lateral ridge of the fibula, and peroneal injury (319,321–323).

MRI may help to elucidate tendinous and retinacular defects, fluid within tendons or sheaths versus synovitis, abnormalities of the retrofibular groove, and posterolateral fibular spurring (263,321,322,324). Sobel et al. demonstrated the attritional changes of the peroneus brevis tendon by showing distinct thinning and wavy irregularity of the tendon contour using MRI (313,324). Consistent evidence of spur formation on the lateral ridge of the peroneal groove has also been noted (315). This fibular spur is distinguished from the normal peroneal retinaculum insertion (lateral ridge) because it has an independent marrow cavity. This spurring likely results from chronic subluxations and mechanical friction against the posterolateral fibula (261,263,284). As with any imaging modality, pitfalls in interpretation can lead to false-negative studies. Some investigators have suggested that this may result from the lack of specific criteria for diagnosing peroneal tendon tears (299,309,312,319). In light of this fact, a normal or negative MRI scan is not necessarily a contraindication to surgical exploration.

Ultrasound imaging is an inexpensive, noninvasive, reproducible technique that can provide considerable information regarding the integrity and continuity of tendon in addition to contiguous soft tissue and osseous structures. The identification of fluid and calcifications within and about tendons can be readily identified, in some studies providing guidance for fluid aspiration or surgical excision of calcifications (325,326). In the lower extremity, ultrasound has been useful for evaluating all forms of tendinopathy, perhaps most often reported for Achilles and tibialis posterior tendinopathy. The protocols and techniques for musculoskeletal imaging are similar for all muscle compartments (327–334). Ultrasound is operator dependent, and techniques for the lower extremity require specific expertise.

Treatment

Some patients with chronic peroneal tendon dislocation may have little pain or disability, although this would seem to be the exception rather than the rule. Investigators have proposed that this could ultimately lead to adaptive shortening of the peroneal tendons over time, possibly resulting in lateral foot and ankle weakness and a reduction in propulsive power (272,280). In patients with chronic subluxation, surgery may be preferred.

The correction of subluxating peroneal tendons has inspired much creative thought, and many procedures have been described to accomplish this purpose (195,201,262, 263,268,271,272,274,290,294,295,301,303,308,314,315, 335–348). The goals are to stabilize the peroneal tendons within their retromalleolar groove and to maintain their gliding function around the fibular pulley. These goals can be achieved by repairing the superior peroneal retinaculum or deepening the retrofibular groove. Repair of the retinaculum restores integrity to the lateral border of the superior peroneal tunnel. If the remaining retinacular tissue is insufficient, soft tissue or tendon transfer may be required for supplementation. Significant adaptive changes often take place in chronic injury. Repetitive trauma and hemorrhage set the stage for intratendinous degeneration that should be addressed in this setting. Attenuated retinaculum should be imbricated, whereas calcifications and adhesions should be excised or débrided at this time to obtain optimal results. The surgical approach is the same as that described for repair of the acute injury, although in some instances in which only the retinaculum is to be repaired, one may not need as much distal exposure.

Surgical procedures can be divided into five categories (280,349):

I. Anatomic soft tissue reconstruction (263,271,273,274, 350–352)
II. Tissue transfer (301,303,315,347,350,353–357)
III. Groove deepening (289,343,345,347)
IV. Bone block (289)
V. Rerouting (291,304,346,358)

Anatomic Soft Tissue Reconstruction

Primary repair of the superior peroneal retinaculum is perhaps the most popular approach and has been described by

many authors (254–256,263,271,273,274,286,359–365). If the retinacular remnant is sufficient for repair, reattachment to the periosteum and fibula can be performed using simple suture techniques through drill holes in the fibula. Anchoring devices are also an option for fixation. A periosteal flap, created to reconstruct the superior peroneal retinaculum, can be used alone or in combination with a primary retinacular repair for reinforcement (359–363). These flaps can be developed from both the fibular and calcaneal periosteum (353). In the case of subluxation within the retrofibular groove, tissue transfer using portions of the superior retinaculum can be interposed between the peroneus longus and brevis to stabilize them proximally. McConkey and Favero interposed two posteriorly based flaps from the superior retinaculum for this purpose (266). Exploration of the peroneal tendons may be performed if warranted by clinical and or surgical evidence of disease. A chronic lateral effusion may indicate an underlying tendinopathy. Significant hemorrhage and adhesions are resected. Tenosynovitis is addressed by tenosynovectomy, and tendon splits or tears are treated by tenoplasty.

Tissue Transfer

The primary tissue transfer repair consists of reconstruction of the superior peroneal retinaculum by tendon transfer or graft. In the event that the remaining retinacular tissue is insufficient or unidentifiable, soft tissue or tendon transfer

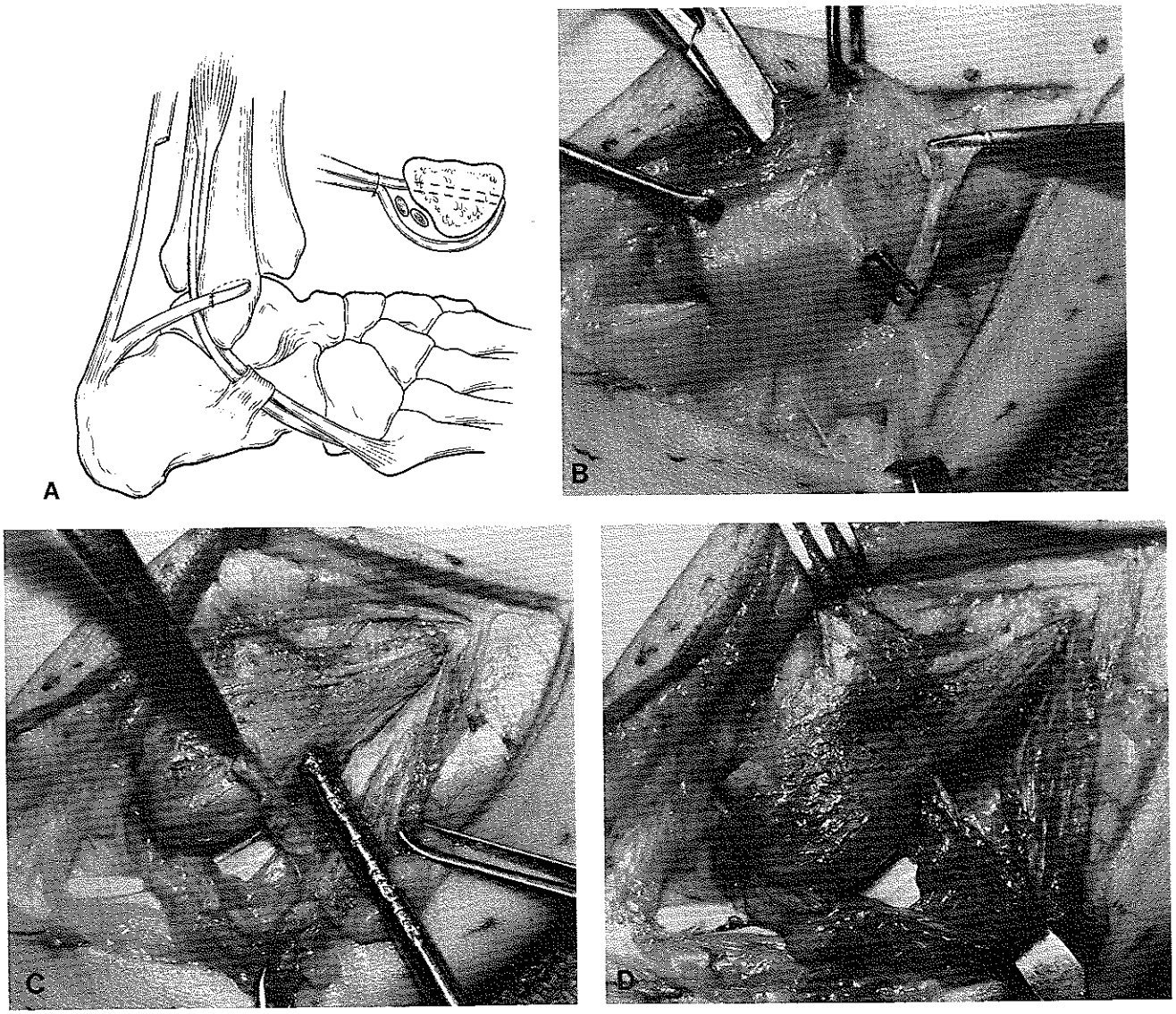


FIG. 21. Reconstruction of a new retinaculum by the method of Jones. **A:** A small strip of Achilles tendon is detached, routed from posterior to anterior through a drill hole in the fibula, and then is sutured onto itself. **B:** Intraoperative view of a Jones repair with fascial augmentation demonstrated before tendon translocation. **C:** A trephine hole is made to anchor the slip of Achilles tendon. **D:** Appearance of the Achilles tendon slip after suturing. (Courtesy of Thomas F. Smith, D.P.M.)

may be required for supplementation. Traditionally, a tendon transfer harvested from the distal lateral aspect of the tendo Achillis is attached to the fibula to simulate the stabilizing function of the superior peroneal retinaculum (303). A modification of this procedure includes attachment of the tendon slip to the lateral fibular ridge and periosteum (337). The original Ellis-Jones procedure for chronic dislocation of peroneal tendons is described later.

With exposure of the lateral malleolus, the peroneal tendon sheath is isolated and is retracted posteriorly. A tunnel is drilled from anterior to posterior through the fibula approximately 3 cm proximal to the distal tip. Dissection then is carried posteriorly to expose the distal aspect of the tendo Achillis. An *in situ* tendon graft is obtained by liberating a section of tendon proximal to the Achilles insertion. The desired length may be determined with the use of umbilical tape before sectioning the tendon. A tendon slip approximately 7 cm long is freed leaving the insertion intact. With the patient's ankle dorsiflexed and the foot in a supinated position, this tendon slip is transferred from posterior to anterior through the fibula and sutured onto itself (Fig. 21). The postoperative course typically requires 4 to 6 weeks of non–weight-bearing immobilization in a short leg cast. Good results have been reported with minimal complications or adverse effects using this approach (290,366).

Groove-Deepening Techniques

These procedures are deemed appropriate when the peroneal groove is shallow or convex (343,345,351,367). Zoellner and Clancey described a classic method for deepening the peroneal sulcus (343). This procedure would appear to maintain a smooth bed for the tendons and allow unrestricted gliding function. An osteoperiosteal flap is made within the retrofibular groove, and a "trap door" is created in the frontal plane with its hinge based medially (Fig. 22). This flap can be opened anterior to the peroneal tendons to allow for debulking of the underlying cancellous bone. This maneuver decreases the cubic content of bone beneath the peroneal groove and deepens the sulcus approximately 6 to 9 mm on closure of the trap door. In 10 cases, all were reported as sustaining an excellent result after a 2-year follow-up. Hutchinson reported 71% good results. Fair and poor results were associated with tendinopathy, typically longitudinal tears resulting in continued pain, swelling, and considerable disability (298).

Bone-Blocking Procedures

Kelly used a sliding bone block to enlarge the lateral lip of the fibula (289). A sagittal plane osteotomy is made in the distal 5 cm of the fibula. A transverse cut is then made at the most proximal extent of the osteotomy from anterior to posterior. The lateral segment of bone and intact periosteum is translocated posteriorly, to extend the lateral lip of the fibula to the desired position (316). This flap may be

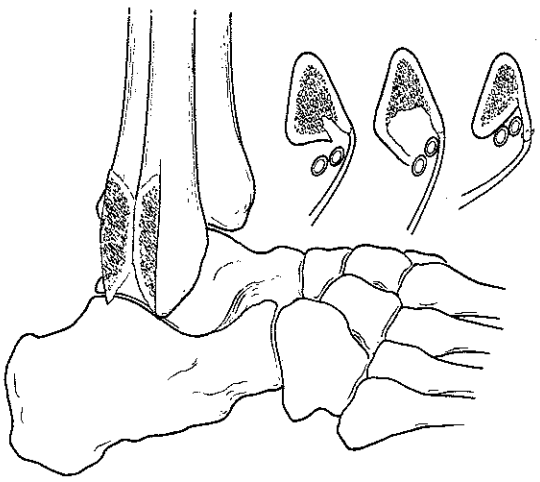


FIG. 22. By creating an osteoperiosteal flap within the retrofibular groove, a "trap door" is created in the frontal plane with its hinge based medially. This groove-deepening procedure was described in 1979 by Zoellner and Clancy. An osteoperiosteal flap is raised on the posterior surface of the fibula, medullary bone is curetted out, and the "trap door" is closed.

oriented either parallel or oblique to the long axis of the fibula and is stabilized with screw fixation (Fig. 23). Modifications of this approach have been employed with success (262,280). Larsen et al. (367a) used this technique in 36 patients; after 5 years, 26 patients achieved excellent results and 5 good results. Despite "excellent" and "good" results, 42% complained of painful internal fixation that required removal. Recurrent subluxations were noted in 3 patients, and complaints of instability persisted in 7 patients. Complications included failure of internal fixation devices, graft fracture, graft resorption, and penetration of the joint by fixation devices. DuVries described a posterior sliding bone

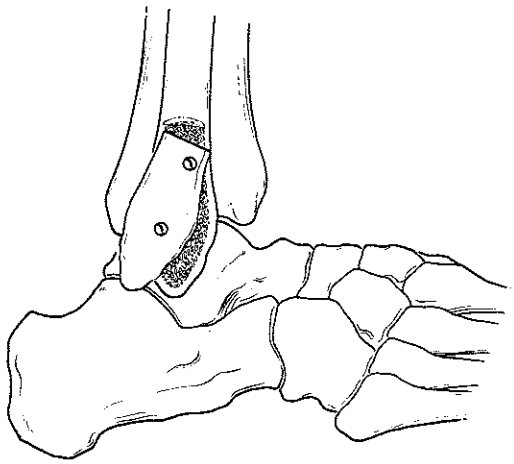


FIG. 23. Deepening of the peroneal groove first performed by Kelly in 1920. A sagittal plane osteotomy is made through the distal fibula. This section of bone is rotated posteriorly and is fixed with two bone screws.

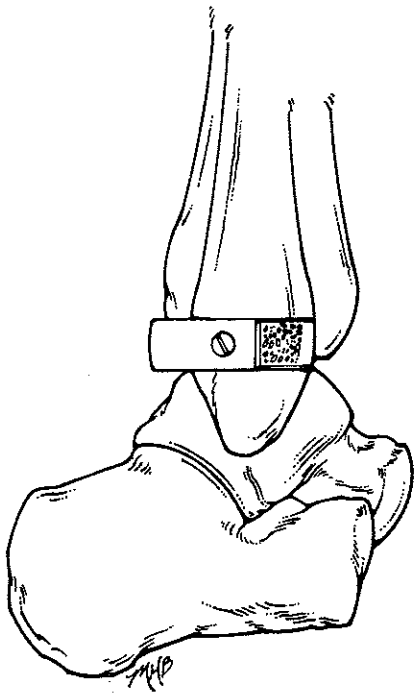


FIG. 24. DuVries posterior sliding bone graft used to deepen the peroneal groove. The graft is fixed with a single screw.

graft that involved a more proximal segment of the fibular malleolus for the same purpose (336) (Fig. 24).

One could argue that these procedures ultimately place the lateral aspect of the peroneal tendons against a bed of cancellous bone, the medial aspect of the osteotomy, and possibly encourage tenosynovitis and adhesion formation that impedes gliding function.

Rerouting of the Peroneal Tendons

Rerouting peroneal tendons beneath the calcaneofibular ligament has also been advocated to prevent subluxation (304,314,358), a maneuver that requires sectioning of the tendons. Alternatively, investigators have suggested that the calcaneofibular ligament be sectioned and then repaired over the peroneal tendons. To address associated lateral ankle instability, tendon transfer has been used to recreate the peroneal retinaculum using a split peroneus brevis tendon transfer. The tendon is harvested in the same manner as in a lateral ankle stabilization reconstructing the calcaneofibular ligament. The tendon graft is placed superficial to the peroneal tendons and is secured to the calcaneus, in contrast to the typical lateral ankle stabilization in which the tendon graft is transferred beneath the peroneal tendons (347).

Yu et al. proposed a split peroneus longus technique to reconstruct the lateral collateral ligaments (318), in contrast to the more traditional approaches that sacrifice a portion of peroneus brevis. This procedure is believed to stabilize the peroneal tendons in their retrofibular groove and to provide enhanced lateral ankle support while maintaining full functional integrity of the brevis tendon (358). By employing a portion of the peroneus longus tendon, any functional deficit is afforded by a tendon responsible for a supinating (inverting) force across the subtalar joint and leaving the integrity of the major evertor (peroneus brevis) of the rearfoot intact (276,297). The postoperative course for rerouting procedures in general requires 6 weeks of non–weight-bearing immobilization in a short leg cast.

CHRONIC LATERAL ANKLE INSTABILITY

Eighty-five percent of ankle sprains involve the lateral ankle, with syndesmotic sprains and isolated medial sprains occurring far less often, 10% and 5%, respectively (132,368). Although no specific patient profile exists for ankle sprains, an increased frequency has been noted in the population ranging from 15 to 35 years, with males predominating (367,369–371). After the age of 40 years, the incidence may be higher for women (372). *Lateral ankle sprains* are the most common injury among athletes (348,373–375), and these injuries may account for as much as 12% of trauma cases seen within the emergency room (369), with 25% to 50% of such sprains occurring in sports with a high degree of running and jumping action such as volley ball, basketball, soccer, rugby, and football (371,376). These sprains constitute 20% to 25% of all ''time-loss'' sports injuries (377).

The prevalence of chronic lateral ankle instability after lateral ankle injury has been reported in as many as 10% to 30% of these patients (371,378–380). This may be due in large part to inadequate treatment of the acute injury, with poor or insufficient healing of the ankle ligaments. Fallat et al. coined the term *sprained ankle syndrome* after conducting a prospective analysis of 639 acute ankle injuries. Most of these cases, 71.3% (390 patients), were diagnosed with a grade I ankle sprain. The most common pattern was injury to both the anterior talofibular ligament and the calcaneofibular ligament occurring in 34.2% (187 patients) (123). The authors identified myriad factors associated with ankle sprain that can be responsible for chronic pain if undiagnosed. Because residual pain syndromes are noted in as many as 40% of those affected by ankle sprain, the concept of the sprained ankle syndrome appears clinically relevant (123,381).

Anatomy

The lateral collateral ligament complex is a composite of three individual structures: the anterior talofibular ligament, the calcaneofibular ligament, and the posterior talofibular ligament. The primary functions of this complex are to provide proprioception as well as stabilization and limitation of pathologic motion about the lateral ankle. Proprioceptive fibers in this region supply an afferent sensory pathway that directs appropriate position for joint function in addition to stimulating the muscular reflex arc for stabilization

(381,382). The anatomic and geometric alignment of these ligaments provides for stability of the ankle and subtalar joint while permitting a full range of motion, guarding against pathologic extremes (383).

The lateral collateral ankle ligaments resist inversion stress across the tibiotalar joint. This stability is provided by passive and dynamic factors (384). Passive elements include the anatomic systems of bones, ligaments, and tendons that make up the ankle, that is, the congruency of the ankle mortise, the integrity of the lateral collateral and tibiofibular ligaments, and the retinacular structures and tendon tunnels crossing the ankle joint. The position of the foot relative to the weight-bearing surface, muscle function, and the influence of gravity on the extremity are among the dynamic elements stabilizing the ankle joint. The malleoli and the collateral ligaments provide frontal plane stability in the non–weight-bearing ankle, whereas tendons and tendon sheath attachments afford sagittal plane stability. The peroneal tendons laterally and the flexor group medially provide enhanced collateral ankle stabilization. The lateral collateral ligaments are not strained to any meaningful degree with sagittal plane motion about the ankle joint (385). In fact, when weight bearing, the ankle is most stable in the dorsiflexed position (386).

The anterior talofibular ligament provides anterior stability to the ankle joint, particularly with the foot plantarflexed. This ligament is an intracapsular structure and is the weakest of the lateral collateral ligaments. It is composed of two fibrous bands that are separated by vascular elements. Originating from the most distal portion of the anterior fibula, the superior band begins near the anterior inferior tibiofibular ligament, whereas the inferior band begins near the calcaneofibular ligament. Both bands course anteromedially and extend to the most anterior portion of the lateral talar body. This insertion is immediately anterior to the fibular facet of the talus.

Sagittal plane motion of the foot and ankle affects the alignment and position of this ligament. In plantarflexion, the anterior talofibular ligament is taut and more parallel to the tibia and serves as a primary restraint against inversion of the talus in the ankle mortise. The ligament is loosened as the ankle is dorsiflexed. With the ankle joint in the neutral position, the anterior talofibular ligament parallels the long axis of the foot and restricts the talus from excessive anterior displacement and medial rotation.

The posterior talofibular ligament provides stability to the ankle joint by securing the talus posteriorly and by limiting dorsiflexion of the foot and anterior displacement of the leg. The ligament takes origin from the inferior surface of the digital fossa of the fibula and courses laterally and posteriorly to the talus. Short and long fibers diverge as they approach the talus, with the short fibers inserting within a groove at the posteroinferior aspect of the lateral malleolar facet of the talus and the long fibers inserting on the posterior aspect of the talus. The medial extension of the ligament contributes to the floor of the flexor hallucis longus tunnel by sending fibers to the posterolateral tubercle of the talus. If an os trigonum is present, it is secured by these fibers. The posterior talofibular ligament is the strongest of the lateral collateral ligaments. The ligament is covered by the peroneal retinaculum laterally and the flexor hallucis longus tendon medially, a feature that makes it intracapsular but extrasynovial.

The posterior talofibular ligament assists in the translation of motion between the leg and the talus by conveying the force of internal leg rotation to the talus. It resists external rotation of the talus and internal rotation of the tibia and fibula. The fibers of this ligament are shorter anteriorly than posteriorly; as such, the posterior fiber become tense in dorsiflexion while the anterior fibers are tense throughout the entire sagittal plane range of motion.

The calcaneofibular ligament is an extracapsular structure that lies parallel to the subtalar joint axis and stabilizes both the ankle and subtalar complexes. Its origin is just inferior to the anterior talofibular ligament on the distal fibula. The calcaneofibular ligament courses beneath the peroneal tendons in a posterior, inferior, and medial direction to insert on the lateral wall of the calcaneus just posterior to the peroneal trochlea. Tension through this ligament is principally affected by sagittal plane motion of the ankle and frontal plane motion of the subtalar joint (Fig. 25).

The calcaneofibular ligament creates an angle with the bisection of the fibula that is variable, ranging from 0 to 90 degrees. Ruth studied this angle in 33 cadaveric specimens and in 55 surgical ankles and noted a range of 10 to 45

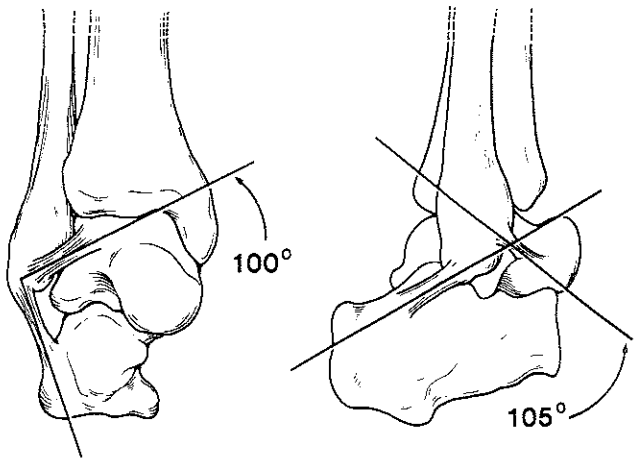


FIG. 25. The crucial relationship of the anterior talofibular ligament (ATFL) to the calcaneofibular ligament (CFL) is demonstrated. This complex relationship is difficult to recreate with tendon grafts and explains the limitation of subtalar joint range of motion that may follow stabilization surgery. An anteroposterior view of the ankle *(left)* shows that the CFL and ATFL are nearly at right angles to each other. The ATFL courses primarily from medial to lateral. The CFL lies on the subtalar joint axis. In the lateral view *(right)*, the CFL is angulated 20 to 45 degrees from the longitudinal bisection of the fibular and 105 to 130 degrees to the ATFL.

degrees in 74.66% (387). The relationship between the calcaneofibular and anterior talofibular ligaments is important to overall ankle stability. The angle formed between these ligaments has been reported to range from 70 to 140 degrees. The more obtuse the angle, the more unstable is the complex, especially in the sagittal plane (388). In an exaggerated angular construction, greater than 120 degrees, the ankle remains destabilized through the transition from dorsiflexion to plantarflexion. The calcaneofibular ligament becomes more horizontal and lax, unable to resist inversion stresses, whereas the anterior talofibular ligament remains incapable of stabilizing the ankle until a more vertical position is approached at plantarflexion. False-positive stress examinations may be explained by this variant in orientation. Inman suggested that this type of relationship between these ligaments was present in 10% to 15% of the population and considered this factor as the culprit for many ankle sprains (389).

Clinical Presentation

Joint instability may be the primary culprit for certain symptoms in chronic ankle disorders. Performance anxiety, pain, periodic swelling, recurrent injury, and the sense of "giving way" may all be components of the subjective complaint. The complaint of instability with weight bearing may be exaggerated on uneven surfaces, a finding that may be due to a delay in sensory input because poor ligament healing results in attenuation and loss of proprioceptive function (381,382).

Some disorders may predispose a patient to ankle instability. The history usually includes prior ankle trauma with inadequate functional recovery. However, the severity of the inciting event does not necessarily correlate with the intensity of residual symptoms (371,381,390). A pes cavus foot type, particularly in patients with neuromuscular diseases, places the patient at a greater risk for lateral ankle instability because of the structure and function of the foot and ankle. Table 3 provides a summary of predisposing factors.

Although mechanical instability may be defined as motion beyond the normal physiologic range, Freeman et al. coined the term *functional instability* to describe the subjective complaint of the ankle "giving way" (381). This does not necessarily require athletic activity because simple daily activities can aggravate ankle instability. Tropp defined functional instability as motion beyond voluntary control, which may not exceed the normal end range of motion (391). Although no direct correlation between mechanical and functional instability has been proven, Tropp found that functionally unstable ankles were often mechanically stable. Soccer players with functional instability were noted to possess a decreased ability to maintain postural equilibrium even in the absence of overt mechanical instability. It is possible that the degree of disability is related to numerous factors, mechanical or functional instability, and associated muscular insufficiency (392).

TABLE 3. *Predisposing factors for inversion ankle sprains*

Ligamentous laxity
Fixed calcaneal varus
Tibial varum
Rigid plantarflexed first ray
Forefoot valgus
Ankle varus
Uncompensated equinus
Peroneal muscle weakness
Previous inversion ankle trauma
Congenital weakness of the anterior talofibular ligament
Limb length discrepancy
Supinated subtalar joint
Phasic overactivity of tibialis posterior and anterior muscles

Imaging

Plain radiographs are often noncontributory, although one may note avulsion fragments about the fibula. In younger patients with open epiphyses, or in patients with equivocal results, contralateral views may need to be taken for comparison.

Stress radiographs may be obtained in an attempt to determine the mechanical integrity of the ligaments. Some controversy exists regarding which specific study is the most reliable, be it the anterior drawer test or the inversion stress test. The criteria used for determining what constitutes a positive finding are also controversial. The *anterior drawer test* is used to evaluate insufficiency or rupture of the anterior talofibular ligament. The test is performed with the patient supine or sitting up with the knees flexed over the table's edge. One hand is held against the anterior tibia and the other hand grasps the posterior aspect of the heel. The foot is internally rotated a slight degree to relax the deltoid ligament; otherwise, this structure may limit the true amount of anterior displacement that is available. The ankle is placed in a relatively neutral position. The calcaneus is then pulled forward while the tibia is stabilized simultaneously. In the event that the anterior talofibular ligament is compromised, the talus will displace anteriorly from beneath the tibia, and a dell may be appreciated in the skin of the anterolateral aspect of the ankle joint. Normal values for anterior displacement may range from 2.5 to 3 mm (393), and in general, anterior displacement of the talus greater than 4 mm is considered a positive result (Figs. 26 and 27). In questionable cases, evaluation of the contralateral, uninjured limb may be helpful to assess how much displacement is "normal" for that particular individual.

Performing the *stress inversion test* is valuable in assessing combined laxity of the anterior talofibular and the calcaneofibular ligaments (394). An isolated rupture of the anterior talofibular ligament may result in a small increase in talar tilt. *Talar tilt* is measured radiographically by the angle formed between the tibial plafond and the talar dome in the AP view when an inversion force is applied through the

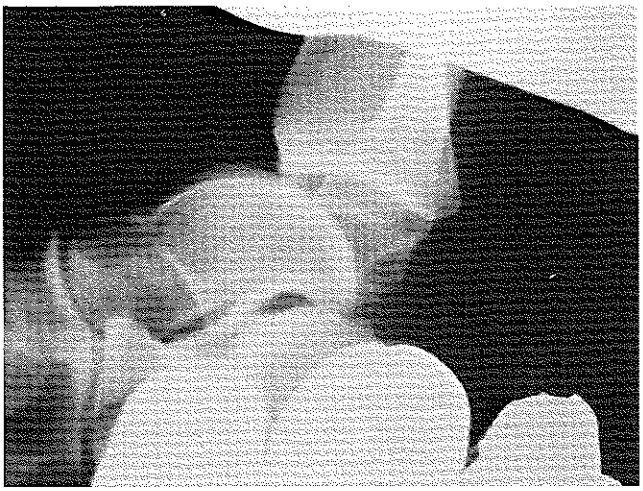


FIG. 26. A positive anterior drawer radiograph.

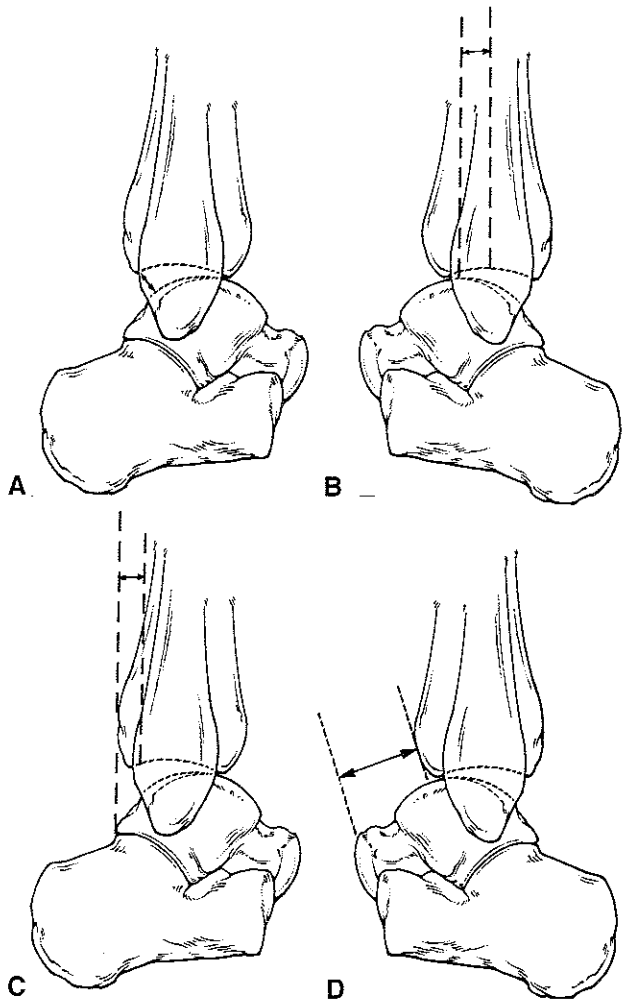


FIG. 27. Methods of measuring anterior drawer. **A:** The distance between the posterior lip of the tibia and the nearest point of the talar dome. **B:** The displacement index is demonstrated by the perpendicular distance between the center of the talar and tibial articular curvatures. This quantity is divided by the sagittal length of the tibial joint and is then multiplied by 1,000. **C:** The perpendicular distance between vertical lines drawn through the posterior ribial lip and the posterior lateral tubercle of the talus. **D:** Distance between the distal dorsal aspect of the talus at its articulation with the navicular and anterior margin of the articular surface of the tibia.

rearfoot and ankle (Figs. 28 and 29). The stress inversion test is performed with one hand grasping the heel while the opposite hand stabilizes the medial aspect of the leg just above the malleoli. The rearfoot and ankle are then forcefully inverted (395). An anesthetic block of the common peroneal nerve and sinus tarsi may be beneficial in reducing splinting and muscular guarding during the examination.

Leonard suggested that the optimal ankle position for the inversion stress test be based on the ligaments that are potentially injured. In patients with point tenderness at the anterior talofibular ligament, it was recommended that the ankle be stressed in plantarflexion. This position was found to produce maximum ankle instability with inversion stress based on cadaveric models (396). When the anterior portion of the talar dome rests within the mortise, the ankle is most stable, that is, in the dorsiflexed position. A plantarflexed talus within the mortise creates an ankle that is functionally and structurally more lax as the posterior aspect of the talus is more narrow. Therefore, the position of the talus within the mortise directly affects the available degree of talar tilt in both pathologic and normal ankles.

The normal value for talar tilt is 5 degrees or less in manually stressed ankles (397). Perlman et al. considered a disparity of 18 degrees or more between sides as diagnostic of injury to both the anterior talofibular and calcaneofibular ligaments. Hutchinson and Wardle found a 77% incidence of talar dome injury when the talar tilt exceeded 18 degrees (398). In contrast, Rubin and Witten reported a range of normal values from 0 to 23 degrees (399). Technical variables can affect this measurement, including method and duration of applying the inversion stress, ankle position, and the use of anesthesia. A host of physiologic factors can influence the inversion stress measurement; Table 4 provides a summary.

A technique for assessing subtalar joint instability has also been described (400). The leg is internally rotated 45 degrees, and with the ankle and foot in a neutral position, an inversion stress is applied. A notable loss of the parallel relationship between the subtalar joint facets is appreciated in patients with subtalar instability. Stress tomography has also been advocated as an option for evaluation of subtalar joint stability (401).

Stress testing is helpful in most cases, although a negative stress examination does not necessarily rule out instability. Patients with functional instability may not demonstrate overt mechanical evidence of laxity and, therefore, may have a negative stress examination. Because functional instability is a definitive process, a negative stress test does not necessarily rule out the need for stabilization.

Arthrography has also been employed to evaluate ankle

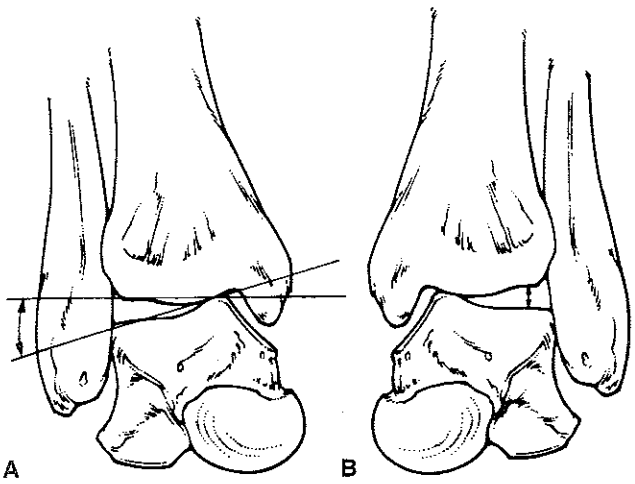


FIG. 28. Methods of measuring talar tilt. **A:** Measured in degrees. **B:** Measured in millimeters, the distance between the superolateral border of the talar dome to the inferolateral border of the tibia.

injuries for potential ligamentous disruption. This technique is likely to produce false-negative results if the study is performed later than 1 week after injury because of adhesion and hematoma organization among the periarticular structures (126). In chronic conditions, there may be a persistent communication between the ankle joint capsule and the channel of the common peroneal tendon sheath that can be visualized with arthrography (186).

MRI may also be used to assess patients with ankle instability. Current strategies have employed this technique to determine the extent of injury and the suspected chronicity of the disorder (187). Acute injuries have been associated with overt ligamentous defects and an obvious loss of normal tissue contours, with the ligament borders obscured by hypoplasia. Fluid within the joint and the adjacent peroneal tendons is typically noted. Chronic ligament injury has been described as providing a variable appearance ranging from a notable fibrous ingrowth about a hyperplastic ligament with poorly defined margins to an attenuated, hypoplastic ligament with more easily discernible edges. As the ligament undergoes repair, a notable hypoplasia within the defect is appreciated during the initial 3 to 7 weeks. The end stage of ligament recovery is characterized by hyperplasia resulting from an increased stress accepted through its fibers. Persistence of a hypoplastic ligament is reflective of residual attenuation. Older or long-standing injuries may be considered as having a lower potential for optimal functional recovery with conservative measures.

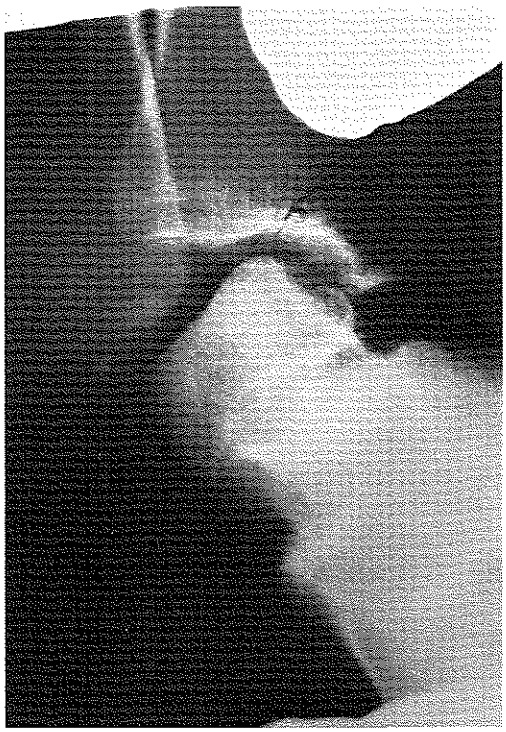


FIG. 29. A positive stress inversion radiograph.

TABLE 4. *Factors that influence increased ankle stress results*

Previous inversion trauma
Age < 15 years
Geometric orientation of lateral ligaments
Marfan's syndrome
Ehlers-Danlos syndrome
Ligamentous laxity
Osteogenesis imperfecta
Turner's syndrome

Treatment

Conservative Measures

Spontaneous resolution of ankle instability is not anticipated in patients with a history of chronic dysfunction. Less active patients with low physical demands and few symptoms may be adequately supported with conservative measures. However, conservative therapy for chronic ankle instability is unlikely to be successful in patients who desire a fairly active lifestyle or in those who are not active but whose symptoms interfere with activities of daily living. Traditionally, *conservative measures* have included external devices to support the ankle, ankle strapping, shoe-gear modifications, orthotics, and muscle-strengthening exercises with an emphasis on the lateral compartment (380). Ankle taping can provide good support, although this method has been reported to lose 40% of its stabilizing power within 10 minutes of use (300,301,309,310). A lace-up ankle brace worn in conjunction with athletic shoes may prove more effective than ankle strapping. Ankle braces are effective and commonly yield subjective improvement. The ankle brace may reduce the incidence of recurrent sprains and may allow the patient to resume more normal function. Physical therapy may be helpful in functional instability resulting

from proprioceptive defects, muscle weakness, and concomitant subtalar joint instability.

Lofvenberg et al. reported on the long-term outcome of conservative therapy in chronic lateral ankle instability after an average of 20 years (162). Although many of these patients had residual instability, only a few patients regarded their residual symptoms a significant interruption of daily living. There was no increased incidence of osteoarthritis within the ankles of these patients versus control subjects, a finding contrary to former belief (402–405).

Surgical Treatment

Surgery is recommended for patients where the instability creates a functional problem with their occupation or activities of daily living. Surgical repair of the lateral collateral ligaments can be categorized based on the ligaments being reconstructed (single, double, or triple), the type of repair (anatomic versus nonanatomic), and the types of grafts employed (autograft, allograft, or synthetic). Table 5 contains the classification for surgical repair of the lateral ankle ligaments as modified from Peters et al. (371).

The spectrum of surgical procedures for managing chronic lateral ankle instability includes more than 80 techniques, most of which are modifications of traditional approaches (255,266,275–278,280,281,307,313,314,406,407). A brief description of some of the more popular techniques is provided.

Primary Repair

Although many stabilization techniques are used for recreating the collateral ligaments, primary repair of the damaged ligaments is a viable option for many patients with chronic ankle instability. The procedure requires only a limited surgical exposure and provides for an anatomic restoration of ankle stability. Brostrom and Sundelin were early advocates of ligamentous repair and noted that the torn ligament ends do not atrophy and retract (378). Outcome studies for primary ligamentous repair to treat chronic ankle instability are rare. Janis et al. conducted a retrospective analysis of 26 chronic lateral ankle stabilizations to compare the outcome of delayed primary repair with a secondary reconstruction technique. In 23 patients (26 ankles), primary repair was performed when the remnant ligament was sufficient. At an average 1-year follow-up, subjective reports revealed an overall improvement of 90% in the primary repair group versus an 82% improvement in the secondary reconstruction group (408). However, if the patient has significant coexisting subtalar instability, then this form of repair may prove insufficient.

However, not all cases of chronic ankle instability are amenable to primary repair. Peters et al. consider generalized hypermobility and a delay of 10 years between injury and diagnosis to be relative contraindications to anatomic repair (371). Vissor et al. believed that primary repair in cases of

TABLE 5. *Reconstruction of lateral ankle ligaments*

- Nonanatomic reconstruction
  - Autogenous
    - Single ligament
      - Peroneus brevis
        - Calcaneofibular ligament
          - Guerevitz, 1979
          - Lee, 1957
        - Anterior talofibular ligament
          - Evans, 1953
          - Watson-Jones, 1952
          - Nilsonne, 1932
      - Peroneus brevis or tertius
        - Dockery et al., 1977
      - Peroneus longus
        - Pouzet, 1954
        - Jay, 1989
        - Zenni et al., 1977
      - Achilles split tendon
        - Storen, 1959 (stage I)
        - Jones, 1932
      - Ligament or bone block
        - Haig, 1950
    - Double ligament
      - Peroneus brevis
        - Zwipp and Oestern, 1981 (modified Evans)
        - Chrisman and Snook, 1969
        - Winfield, 1953
      - Peroneus longus
        - Yu et al., 1994
        - Hambly, 1945
      - Plantaris
        - Palladino et al., 1991
      - Periosteal flap
        - Rudert et al., 1997
        - Ahlgren and Larsson, 1989
      - Osteoperiosteal flap
        - Zwipp and Tscherne, 1984
        - Lee, 1957
  - Autograft transfer
    - Achilles split tendon
      - Storen, 1959 (stage I and II)
    - Free autograft plantaris
      - Kelikian and Kelikian, 1985
    - Free autograft fascia lata
      - Elmslie, 1934
    - Free autograft peroneus brevis
      - Dockery et al., 1989
  - Synthetic and allograft
    - Carbon fiber
      - Burri, 1985 & Jenkins, 1980
    - Bovine xenograft
      - Dockery, 1986
- Anatomic repair
  - Primary repair
    - Brostrom, 1966
  - Imbrication and repair to bone
    - Liu and Jacobson, 1995
  - Local tissue augmentation
    - Vammen et al., 1998
    - Saragaglia et al., 1997
    - Haig, 1950

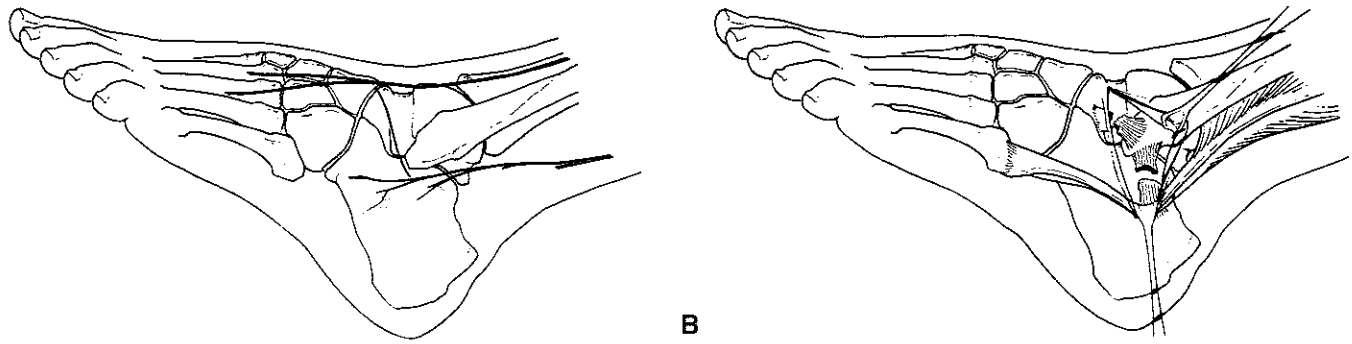


FIG. 30. A: Skin incision for delayed primary repair of the lateral collateral ligaments of the ankle. Note the location of the intermediate dorsal cutaneous and sural nerves at each end of the incision. **B:** The calcaneofibular ligament can be seen underneath the retracted peroneal tendons.

chronic ankle instability is inferior to tenodesis procedures (409). Zwipp suggested that patients with a long history of instability, greater than 10 years, in conjunction with a talar tilt of greater than 15 degrees and anterior drawer of 10 mm, are more suitable to the tenodesis reconstruction (402).

Delayed primary repair of the collateral ligaments is approached identically to primary repair in an acute rupture. The incision plans recommended vary based on the desired exposure. An oblique, curvilinear incision extending from the distal tip of the fibula to the lateral aspect of the extensor tendons at the talonavicular joint can provide adequate exposure of both the anterior talofibular ligament and calcaneofibular ligament while avoiding both the sural and intermediate dorsal cutaneous nerves (Fig. 30). The incision is deepened to the level of the joint capsule in the region of the anterior talofibular ligament. Because these structures are intimately associated, the deep incision will transect both elements granting exposure of the ankle joint. The talar dome can be evaluated at this time. The calcaneofibular ligament is appreciated with posterior and lateral retraction of the peroneal tendons, and it is often found attenuated or ruptured. The peroneal sheath may be explored in the event significant signs of tendon injury exist. Primary ligamentous repair or imbrication is completed most often using 0 to 2-0 gauge suture, absorbable or nonabsorbable.

In the event that exploration reveals remnants of injured ligaments insufficient for repair, various procedures have been described to recreate the lateral collateral complex (275,318,352,367,373,374,377,406,409–440). Although many types of repairs and reconstructions have been suggested, most include tenodesis. The most common donor tendons are peroneus brevis, peroneus longus, Achilles, and plantaris.

Other approaches have also be used to provide stabilization. The anterior talofibular ligament and calcaneofibular ligament have been released subperiosteally from the malleolus, and the ligaments have been anchored in a more proximal position to tighten these structures (441). A slip of the anterior inferior tibiofibular ligament with a segment of tibial bone has been transferred across the course of the anterior talofibular ligament (428). The deep fascia of the extensor digitorum brevis has also advanced and attached to the distal fibula as part of the primary repair (442,443). This procedure has also been combined with advancement of the entire capsular and ligamentous complex (437).

Single Ligament Replacement

Traditionally, the peroneus brevis tendon has been used as an autogenous graft for recreating the calcaneofibular ligament or the anterior talofibular ligament. Different configurations have been devised for use of the graft. The graft may be directed from the fibula to the calcaneus in a direction emulating the calcaneofibular ligament at the level of the superior peroneal retinaculum (347).

Watson-Jones inserted the graft through a drill hole created through the fibula from posterior to anterior, approximately 2 cm from the distal tip of the malleolus (421). The tendon was then passed through a vertical drill hole in the talar neck from dorsal to plantar and was returned through the fibula along the course of the anterior talofibular ligament, this time from anterior to posterior, so a tenodesis could then be performed posterior to the fibula (Fig. 31). Other surgeons have used the peroneus longus tendon in a similar manner (424).

Peters et al. summarized 9 long-term clinical series of the Watson-Jones tenodesis stabilization totaling 250 ankles, with stress radiographs used to analyze stability in 5 studies (371). The retrospective reviews over 1 to 7 years postoperatively revealed that 95% regained stability, with a good to excellent outcome in 80% to 85% of the patients. Residual ache was the difference between good and excellent results. The incidence of a sural neuroma ranged from 0% to 10%, whereas 10% to 30% of the patients had restricted inversion to dorsiflexion. Looking at the Watson-Jones procedure over a longer period postoperatively, Van der Rijt and Evans (443a) observed several treatment failures in patients initially considered stable. They reviewed 9 patients who had experienced instability or insecurity for an average of 22 years, 66% of whom also had a positive anterior drawer sign.

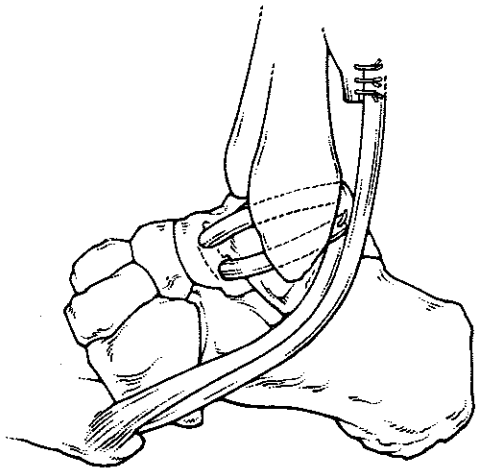


FIG. 31. Diagram of a Watson-Jones lateral ankle ligament repair.

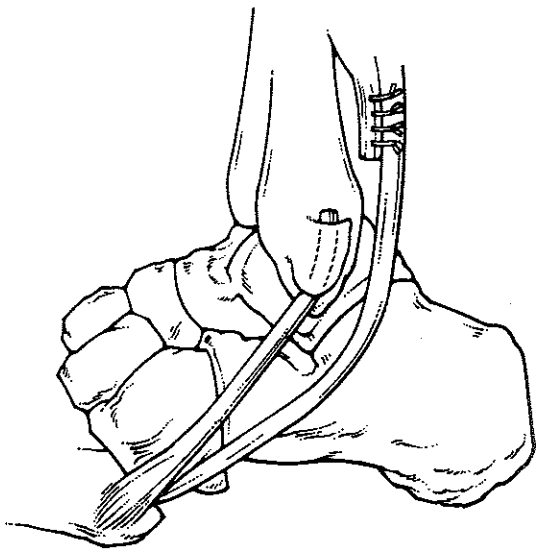


FIG. 33. Diagram of a Nilsonne lateral ankle ligament repair.

The Lee procedure is a modification of the Watson-Jones (422). Here, the entire peroneus brevis tendon graft is passed from posterior to anterior through the fibula. The graft is anchored distally by performing a peroneal anastomosis. A periosteal flap from the distal most anterior aspect of the fibula reinforces the new ligament (Fig. 32). This modification uses a shorter segment of tendon and requires only one drill hole through the fibula, thus reducing the technical difficulty. Similar modifications have been suggested (425).

Nilsonne described detachment of the peroneus brevis at the level of the musculotendinous junction. Proximally, the brevis muscle is sutured to the longus while the distal tendon is placed in a subperiosteal groove through the fibula oriented from posterosuperior to anteroinferior. The calcaneofibular ligament is then primarily repaired, and the brevis tendon is secured in the subperiosteal channel, approximating the course of the anterior talofibular ligament (374) (Fig. 33).

Evans modified the technique by placing a drill hole through the fibula in lieu of creating a subperiosteal tunnel (423). The osseous tunnel runs from the anteriormost and distalmost aspect of the fibula to a posterior and proximal location. The tendon is then fed through the fibula and is secured posteriorly at the proximal aspect of the superior peroneal retinaculum (Fig. 34). Although this procedure does not reconstruct either the anterior talofibular or the calcaneofibular ligament anatomically, it has been used because of its technical ease. Consequently, the procedure has been performed frequently, and some authors have reported successful results and supported its use, with 90% to 100% demonstrating stability and 80% to 95% reporting good to

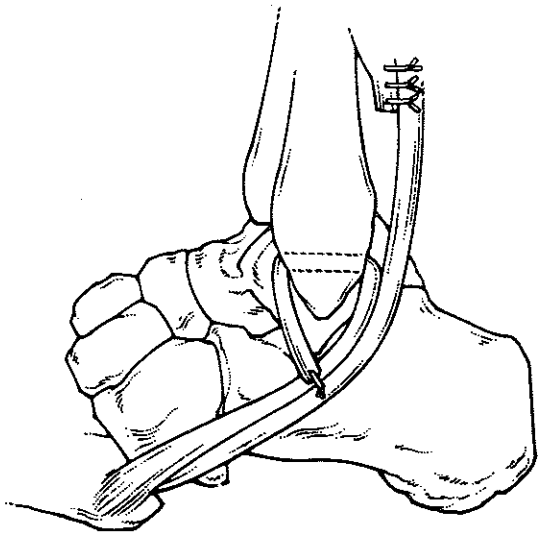


FIG. 32. Diagram of a Lee lateral ankle stabilization.

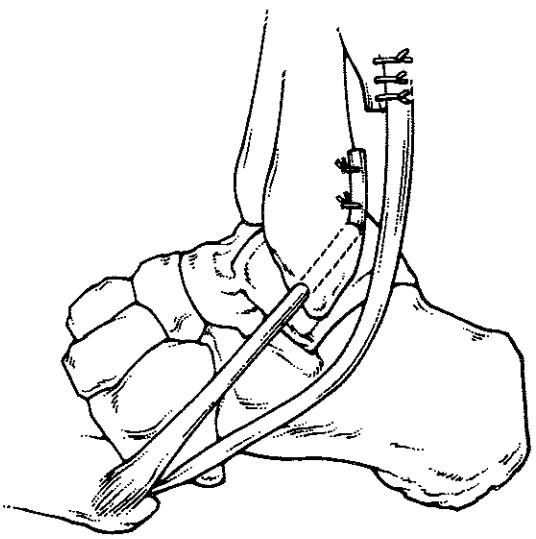


FIG. 34. Diagram of an Evans lateral ankle stabilization.

excellent results. These authors reported 4% with neuroma complications and 30% with restricted inversion (429–435). In one study, the authors believed that all their patients had restricted inversion (436).

Critics would argue that the Evans procedure restricts subtalar joint mobility and disrupts normal rearfoot motion (318,430,436,437). Some investigators have proposed that the Evans procedure does not provide optimal resistance against anterior displacement (especially when the ankle is plantarflexed), internal rotation, and adduction because it does not specifically reconstruct the anterior talofibular ligament or the calcaneofibular ligament (431,432). After a follow-up of 2 to 7 years, Orava et al. noted that approximately 50% of their patients demonstrated a positive anterior drawer after the Evans (434). Karlsson et al. reported on 42 patients 14 years after the procedure and found that 50% had fair to poor results, experiencing pain and instability symptoms. All patients with increased talar tilt postoperatively had local osteophyte formation (430).

Younnes et al. compared the results of 10 Watson-Jones procedures (100% good to excellent) to 10 Evans procedures (90% good to excellent) with an average follow-up of 5 years. Although the Evans was easier to perform, the Watson-Jones appeared to control the anterior drawer better (444).

A modification of the Evans was described by Zwipp and Oestern whereby the distal end of a split peroneus brevis tendon graft is routed from posterior proximal to anterior distal through the fibula and performing a tenodesis onto itself at the level of the fifth metatarsal base. Because the tenodesis completes a triangular configuration of the graft, it provides stabilization to both the tibiotalar and subtalar joints (438).

A partial Achilles tendon graft has also been employed for the repair of the anterior talofibular ligament (426). The lateral one-third of the Achilles tendon is resected proximally near the musculotendinous junction, thus leaving the insertional segment intact. A sufficient length is split from proximal to distal to provide a slip of tendon to course anteriorly through the distal fibula at its widest girth. This length can be measured precisely using umbilical tape before tendon harvest. Recreating the anterior talofibular ligament, the graft is directed through the talar neck from dorsal to plantar and secured to the bifurcate ligament.

Double Ligament Replacement

These procedures are designed to address chronic injury of both the calcaneofibular and anterior talofibular ligaments. Most involve routing a graft from either the peroneus longus or brevis through the fibula and into the calcaneus, modeled after the original Elmslie procedure (414). The Chrisman-Snook procedure is commonly considered the modern prototype for this category of repair (415). A split peroneus brevis tendon graft is harvested from the proximal aspect of the tendon and is routed through the talar neck and then the distal

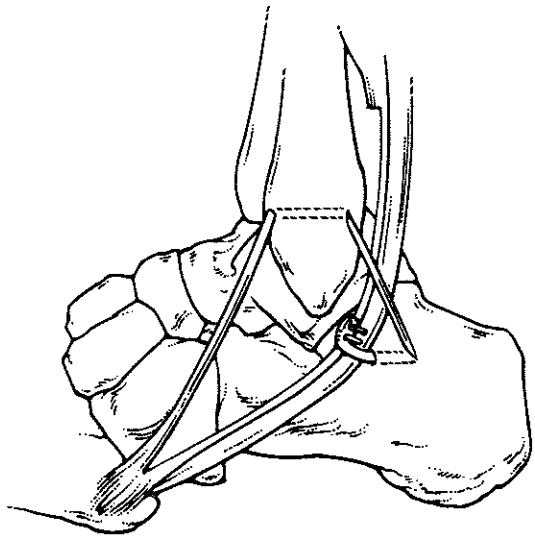


FIG. 35. Diagram of a Christman and Shook lateral ankle stabilization.

fibula through its widest girth from anterior to posterior. The graft is then sutured to a periosteal flap created at the level of the calcaneofibular ligament. Finally, the distal end is sutured onto itself (Fig. 35).

Some variations may be noted depending on the tendon used, the specifics of the anchor into the calcaneus and talus, and whether or not the tendon graft is routed subperiosteally. The peroneus longus tendon has also been used with good results (318,416,417).

Chrisman and Snook originally reported on their procedure performed on 7 athletic students and young adults. Not only did it suitably resolve the ankle instability, but for 3 patients it also resolved subtalar joint instability as well. All 7 had moderate limitation of inversion (415). Snook et al. subsequently reviewed 48 patients at an average of 10 years after they had performed their procedure. They had modified their the technique in that they used a long curvilinear incision over the peroneal tendons, took care to preserve the sural nerve, and harvested only one-half of the peroneus brevis tendon for the tenodesis reconstruction. They observed that 93% of their patients achieved good to excellent results. Although two-thirds had normal inversion, those left with less than 20 degrees were thought to have an outcome expected with the procedure and were not reported as a problem (439).

Successful clinical outcomes similar to those reported by Snook et al. have been reported in 5 modifications of the Chrisman and Snook procedure. Sammarco and DiRaimondo tried to improve the alignment of the graft in 43 patients by creating an osseous tunnel in the talus, slightly changing the direction of the tunnel within the fibula, and placing the graft deep to the peroneal tendons (445). Leach et al. modified the technique on 22 ankles fastening the graft to the calcaneus with a staple rather than through an osseous tunnel (446). Smith et al. routed the calcaneal portion to the

graft through an osseous tunnel and then vertically oriented it back across the subtalar joint and sutured it into the periosteum on the tip of the fibula in 18 patients. This helped to stabilize the subtalar joint further (447). Larsen modified the angle of the fibular canals within which to route the tendon graft in such a way as to also stabilize the subtalar joint (448). Finally, Sammarco and Idusuyi avoided drilling a hole in the fibula by using bone anchors to tenodese onto the fibula as well as onto the calcaneus and the talus with the anterior half of the peroneus tendon (372). In this study of 31 cases of chronic lateral ankle instability, a high degree of postoperative ankle stability was achieved; 94% good to excellent outcomes and 97% recovery of mechanical stability were achieved. Complications included wound infection, prolonged healing, irritation of internal fixation devices, dehiscence, sensory loss over the region of the sural nerve, chronic pain, limited ankle dorsiflexion, and overcorrection.

In one study comparing the Chrisman and Snook double ligament repair with the modified Brostrom primary lateral ankle ligament repair (440), both procedures improved the degree of talar tilt. Although more than 80% of patients were rated as sustaining either a good or excellent result regardless of the technique used, the complication rate was higher in the patients undergoing double ligament repair. Horstman et al. looked at postoperative results of the Chrisman-Snook, Evans, and Watson-Jones procedures (449). They were surprised to find that 63% of the patients with a Chrisman-Snook procedure had some degree of postoperative pain, with 5% describing it as severe. Although 70% of the patients demonstrated restricted inversion, only 12% were left with residual ankle instability, the lowest percentage in the study. Most patients who underwent the Evans procedure were satisfied with the results; however, 83% reported some degree of pain. Talar tilt was controlled equally well with the Chrisman-Snook and the Evans procedures, both better than the Watson-Jones. The overall satisfaction rate for all three procedures was 90% or better, but the Chrisman-Snook procedure appeared to be superior better it duplicated normal anatomy more accurately.

St. Pierre et al. analyzed the results of 5 different ankle stabilization procedures performed on a total of 53 ankles: Evans, Chrisman-Snook, Watson-Jones, Goldner, and Lee (450). Follow-up ranged from 1 to 7 years, with a mean of 2.1 years. Although the Watson-Jones and the Evans left the greatest percentage of ankles with recurrent instability, good to excellent clinical results were achieved in 91% of the surgical procedures overall.

Osteoperiosteal flaps have been described to augment ankle stability (163,404,422). Rudert et al. employed an incision running along the anterior aspect of the fibula coursing posteriorly at the distal tip of the malleolus (163). After deep dissection, the periosteum of the distal one-third of the fibula is exposed. Two periosteal flaps are created from the distal anterior and distal lateral aspect of the fibula. The flaps are then reflected from proximal to distal while maintaining the distalmost attachment. Each flap measures approximately 10 by 0.5 cm. A 4.5-mm drill hole is created in the distal fibula, running in the direction of the calcaneofibular ligament. The periosteal flap is pulled through the drill hole, and the site of the calcaneofibular ligament insertion is identified inferior to the peroneal tendons. A cortical window, approximately 5 by 5 mm, is resected from the calcaneus, and the periosteal flap is inserted into the window. The small bone block is then replaced. The same technique is used to run the anterior periosteal flap in the direction of the anterior talofibular ligament inserting into the talar neck. Excellent or good results were noted in 81% of patients. Complications included 4 superficial wound infections, 2 small areas of skin necrosis, and 1 neuroma.

Triple Ligament Replacement

Severe ankle sprain, with or without avulsion, may result in the rupture or dysfunction of all three lateral collateral ligaments. Although rarely discussed, the triple ligament repair or replacement is based on the same functional and physiologic principles as primary repair and single or double ligament reconstruction (419,420). Injuries resulting in this much damage are usually addressed in the acute and early phases of disability, rarely in the situation of chronic lateral ankle instability.

Exogenous Ankle Ligament Reconstruction

The use of carbon fiber as a material for ankle ligament substitute has been investigated. In a study of 40 patients implanted with such material, 82% were reported to have had excellent results with minimal complications (451). In a smaller series, however, 2 of the 5 grafts were removed because of chronic sinus formation and skin breakdown (407). Bovine xenografts were utilized for ligament reconstruction in 20 ankles by Dockery et al. using a modified Chrisman-Snook technique (452). These investigators observed enlargement in the fibular drill hole in 4 patients as well as long-term local inflammation overall and occasional skin breakdown. There was a reduction in talar tilt when measured by stress testing postoperatively.

Postoperative Management

Traditionally, the postoperative course has included non–weight bearing in a short leg cast from 4 to 8 weeks. However, protected weight bearing at an earlier phase may be instituted as well. Some surgeons have advocated shorter periods of casting with subsequent stabilization provided by braces or other devices (337,338). Athletic activity is typically restricted for a 3-month interval after cast removal.

ANTERIOR ANKLE IMPINGEMENT EXOSTOSIS

Anterior osseous ankle impingement is a common entity with the principal complaint usually being a generalized an-

terior ankle pain aggravated by dorsiflexion, especially to end range of motion. Typically, the history includes an activity that imposes repetitive stress across the ankle, rather than an acute traumatic event. Impingement exostosis is often described in the athlete such as the football lineman, although the condition is seen in nonathletes, but with lower incidence. The patient may relate a significant reduction in speed or push-off capacity, and the functional complaint of ankle restriction may exceed the complaint of pain (453–455). Limitation of ankle motion varies with the chronicity of the process. A more pronounced ankle joint restriction is appreciated in a more advanced process of hypertrophic bone formation and inflammation. Inflammation may be present in the affected area. Meniscoid lesions may be contributory factors and may account for minimally impressive radiographic changes in patients with chronic pain syndromes.

Diagnosis

Anterior impingement syndrome is a diagnosis that can be confirmed using plain radiography. The lateral ankle radiograph is the most helpful in identifying the exostosis, which may be focal or may span the entire breadth of the anterior joint. This growth may exist as a mixture of fibrous and osseous material that is prone to fragmentation after a prolonged period. Exuberant bone growth about the distal tibia, dorsal talar neck, or both, produces a "door jam" effect on ankle motion that results in a chronic pain syndrome unlikely to resolve with conservative treatment (Fig. 36). Generalized arthritic changes are a different entity resulting in osteophytic proliferation, osteochondral defects, and narrowing of the entire joint space. In an anterior ankle exostosis, the extraarticular bone results in the restriction of joint motion.

Radiographic parameters have been described for evaluating this condition. With the foot and ankle in a neutral position, the angle formed between the distal bevel of the tibia and the dorsal talar neck normally averages about 60 degrees (49). Any decrease in this angle indicates an osseous impingement at the anterior ankle. O'Donoghue proposed a grading system for these exostoses. Grade 1 represents a notable abnormal osseous contour; grade 2 is vaguely described as a sharply defined mass of bone; grade 3 consists of an extensive prominence from $\frac{3}{8}$ inch to $\frac{1}{2}$ inch, with good resolution of the base and apex of the spur; and grade 4 is an enlargement exceeding $\frac{1}{2}$ inch in height with poor visibility of the apex and base of the spur. Fragmentation may be noted in grade 4 conditions (453).

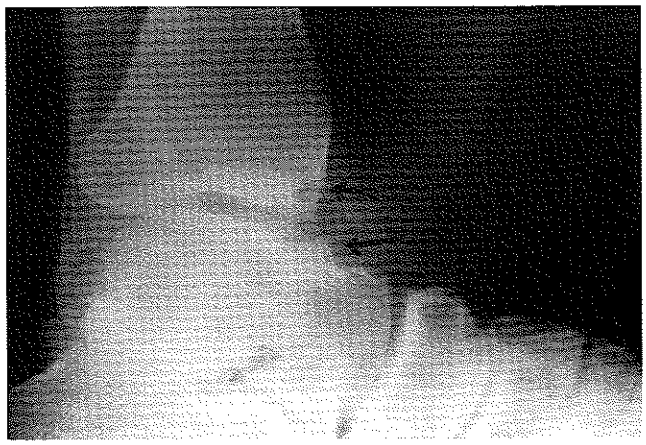


FIG. 36. An anterior impingement obviated by the osteophytic lipping at both the talar neck and distal tibia creating an acute angulation as indicated by the *arrows*.

Although many pastimes can be linked with the formation of an anterior ankle exostosis, activities that demand a high degree of propulsive activity are usually causative. Baseball, football, basketball, and soccer are sports causing excessive stress and strain on the foot and ankle, especially repetitive forced dorsiflexion. These sports require abrupt starts and stops with sudden bursts of acceleration that compress and apply strain to the anterior ankle. Although aggressive athletic activities such as these are typical, other activities are associated with the formation of an anterior ankle impingement, such as ballet and modern dance.

Treatment

The success of *conservative efforts* aimed at symptomatic relief varies with the extent of the impingement and amount of discomfort. Immobilization techniques may quell acute symptoms. However, they do not address the origin of the process and, therefore, are not considered curative. Other methods that may afford varying degrees of pain relief include heel lifts, antiinflammatory agents, and injectable steroid compounds. Perhaps the most successful conservative therapy is activity modification, although this is rarely possible for competitive athletes.

Open surgical techniques are usually required for cases with marked limitation of motion, large exostoses, recurrent pain, chronic swelling, and progressive dysfunction. An anterior medial approach affords optimal visualization of the distal tibia and dorsal talar neck while avoiding major neurovascular structures. The incision lies medial and adjacent to the tibialis anterior tendon. Sharp and blunt dissection is carried down to the level of the joint capsule. An arthrotomy is completed in line with the skin incision. Large prominences of fibrocartilaginous, osseous, and combination masses are resected using manual or power instrumentation. Hypertrophic synovium, osteophytic proliferation, loose bodies, and meniscoid degeneration may be present. Ultimately, all rough irregular surfaces are recontoured. Extreme plantarflexion of the ankle reveals the condition of the cartilage of the anterior talar dome. Extreme dorsiflexion confirms adequate osseous resection. Early range-of-motion exercises may help to minimize fibrosis during healing of the joint capsule to promote future mobility.

A Jones compression dressing is recommended for the

first 3 to 7 days postoperatively. A short leg walking cast is then employed, either non–weight bearing or with protected weight bearing. Active and passive ankle motion is encouraged, then a more aggressive physical therapy program can be prescribed approximately 3 weeks postoperatively. Return to athletic activities is variable and depends on the patient.

Alternatively, the *arthroscopic approach* to the anterior ankle allows for osseous resection, joint decompression, and lavage, with a minimum of postoperative disability and allowing an early return to function. Simultaneously, hypertrophic synovitis can be resected using this technique (456). Large spurs may be difficult to resect through the arthroscope. Protected weight bearing can be resumed almost immediately, although some surgeons encourage the patient to delay weight-bearing activity during the first 3 or 4 postoperative days to allow for intraarticular hemostasis as well as healing of the portal sites. Further weight bearing is advanced to tolerance and is guided by clinical recovery of the joint.

MISCELLANEOUS CHRONIC ANKLE CONDITIONS

With the advent of arthroscopy, an expanding vocabulary has been developed describing various fibrous and cartilaginous lesions found within the ankle such as hypertrophic synovium, hemorrhagic lesions, loose bodies, and plicae. Impingement of synovium and subsequent reactive synovitis are common sequelae of ankle trauma. Chronic pain, crepitance, and reports of ankle instability are typical ongoing complaints after injury. *Anterolateral impingement syndrome* is characterized by chronic pain and swelling of a posttraumatic origin. Intraarticular findings include anterior synovial hypertrophy, chronic synovitis, arthrofibrosis, chondromalacia, and occasionally loose bodies. Impingement of the lateral shoulder of the talus may be caused by aberrant fibers of the anterior syndesmotic ligament.

Stienstra produced a glossary of terms for intraarticular soft tissue lesions of the ankle and divided them them into discrete and nondiscrete lesions (74). Nondiscrete lesions include synovial impingement lesions, talar impingement syndrome, cicatrix, folds and fibrotic fatty tissue, anterolateral impingement syndrome, sports related synovitis of the ankle, and anterior soft tissue impingement of the ankle. Anatomically discrete lesions included meniscoid lesions, plicae, fibrous bands, and adhesions. Many posttraumatic entities are now readily visualized using arthroscopy.

Meniscoid Lesions

The *meniscoid lesion* is a complex of hyalinized tissue that can be found in areas of prior ligamentous injury as a result of traction, contusion, or strain. Any degree of trauma to a joint can result in intraarticular hemorrhage leading to hypertrophic synovitis and the development of exudate. Under usual conditions, this exudate and scar formation are cleared during the normal process of repair. When unresorbed, this exudate becomes hyalinized and forms a meniscoid body. First described by Wolin in 1950, the meniscoid lesion was noted as an aberrant component of the anterior talofibular ligament. Typically, it is a flattened, elastic mass with a tapered free end. The gross specimen is likened to a meniscus of the knee in both appearance and texture, hence the name meniscoid body (457). Some authors believe that this lesion represents partial tears of the anterior talofibular ligament that became trapped in the lateral ankle margin (458). Stone and Guhl made some observations and recommendations regarding these lesions. They suggested the term "meniscoid" was a misnomer and favored the term "synovial impingement lesion," which is not to be confused with the true meniscus of the posterior ankle recess (459).

Meniscoid lesions can be found in the region of the anterior talofibular ligament after lateral ankle sprains. They should not be confused with normal elements such as plicae, which are normal elastic soft tissue projections found throughout the ankle joint. These pliable connective tissue structures are differentiated from meniscoid bodies principally by their location. They are found about the syndesmosis, as well as the in the anterior and posterior areas of the ankle joint. They are not, however, seen in the talofibular region, nor will they be interposed within the ankle gutters as are meniscoid lesions. Further, the normal synovial fringe of the tibiofibular recess is a distinct pink soft tissue mass deep within the lateral ankle and should not be confused with either of these two lesions (78).

Ordinarily, these meniscoid lesions are attached to the anteroinferior talofibular joint capsule with a free end extending into the lateral gutter of the ankle (78). This dense mass generally has a firm fibrocartilagenous consistency approximately 5 mm in width. It is found anterior to the fibula and wedged between the lateral aspect of the talus and the distal medial fibula. This internal ankle derangement may result in a progressive loss of function with increasing pain during weight-bearing activities. These patients often complain of chronic anterior ankle pain, usually in the region of the distal medial fibula. Lahm et al. identified 19 meniscoid lesions in 59 athlete ankle arthroscopies, of which only 1 demonstrated lateral ankle instability (460). All lesions were compounded by synovitis. Chondromalacia and osteophytes were often coincident.

A history of recurrent ankle sprain and a sense of instability are common, although clinical ankle instability is not necessarily a concomitant finding. Meniscoid bodies have a texture similar to that seen in hypertrophic synovitis. Ankle joint arthrotomy and excision of the lesion are considered curative. Martin et al. reviewed 100 ankle arthroscopies resecting hypertrophic synovium and reporting 77% excellent or good results (461).

Fibrous Bands

Fibrous band formation typically presents in a limited fashion and causes adhesion between joint capsule and sy-

novium. These bands affect focal regions of the ankle synovium, in contrast to a generalized adhesive capsulitis. These lesions are yet another variant of chronic hypertrophic synovitis and are often found spanning the entire anterior ankle joint. Fibrous bands appear white or opalesque and are generally inelastic, fibrous cords. Distinct from meniscoid lesions, these hyalinized bands are loosely attached to degenerated articular structures and resemble mature scar. Histologically, they consist of fibrous tissue, heme, and a calyx of fibrocartilage that varies in thickness. A collagen matrix engulfs an abundance of fibrocytes and sparse chondrocytes, and frequently leukocytic infiltration is noted. Intraarticular fractures such as malleolar avulsion and osteochondral injury within the ankle mortise set the stage for hemarthrosis. The heme elements of coagulation within the joint may undergo metaplasia differentiating into elements of the fibrous band. Synovial fluid is known to impair blood clotting; however, its significance here has yet to be explained. Stone and Guhl noted fibrous bands associated with indentations or grooving on adjacent articular surfaces, presumably the result of mechanical pressure and friction. They believed that these lesions developed as a cellular response to intraarticular hematoma (459).

Fibrous bands are more commonly associated with coincident injury than are meniscoid bodies; as such, their presence should prompt a search for occult disease (78). Typically identified between the anterior lateral talar body and the distal anteromedial fibula, these fibrous bands are often associated with the pain of chronic ankle instability and are typically noted after trauma. Various complaints range from localized stiffness or tightness to a sharp, stabbing pain in the anterior ankle. Ankle stiffness most commonly occurs in the anterolateral joint capsule, although posterior pain can be experienced. Often a diagnosis of exclusion, fibrous band adhesion becomes asymptomatic with a block of local anesthetic. This finding confirms the intraarticular process; however, a more specific diagnosis is obtained by ruling out other disorders such as osteochondral lesions of the talar dome, avulsion fracture or loose body in the ankle gutters, osteophytic degeneration of the ankle, and ligament or tendon insufficiency.

Arthroscopic procedures have been described as curative after resecting fibrous bands from the ankle joint. McCarroll et al. reported on the incidence of these lesions in soccer players and described them as an adhesive soft tissue impingements within the anterior ankle (462). These investigators reported good long term relief of symptoms (average of 2 years after treatment) in 4 soccer players.

Posttraumatic Ligamentous Calcifications

Traumatic injury to soft tissues and ligamentous structures can produce hematoma with subsequent fibrosis and adhesion formation that may ultimately result in local calcification. Ankle fractures and severe ankle sprains may be complicated by such posttraumatic calcification. These dystrophic changes can also result in ectopic calcification within muscles (myositis ossificans), ligaments, and tendons (tenocalcinosis). As they develop within the substance of normal ligament fibers (i.e., intraligamentous calcifications), they tend to cause constriction and adhesion. A door-jam effect may then be produced as loose calcific bodies interfere with normal joint motion resulting in impingement, much like the anterior ankle impingement exostosis.

The process of ligamentous calcification has also been referred to as *heterotopic bone formation,* similar to the pathologic process previously described in the knee and known as Pellegrini-Stieda disease (463). This comparison was made in evaluating traumatic injury of the deltoid ligament, where heterotopic calcifications were found after extreme inversion and eversion ankle injuries. These lesions have been found within the medial or lateral collateral ligaments, the interosseous membrane, and the ankle joint capsule, and they cause marked limitation of motion and chronic pain. Anterior or posterior ankle impingement may be coincident because repetitive stress on the joint capsule provokes the development of exostosis formation (Fig. 37).

Conservative therapy includes prescribing orthotic devices that may provide symptomatic relief by limiting the extremes of frontal plane motion. Medications such as nonsteroidal antiinflammatories can help to alleviate painful symptoms. However, long-standing, exuberant calcifica-

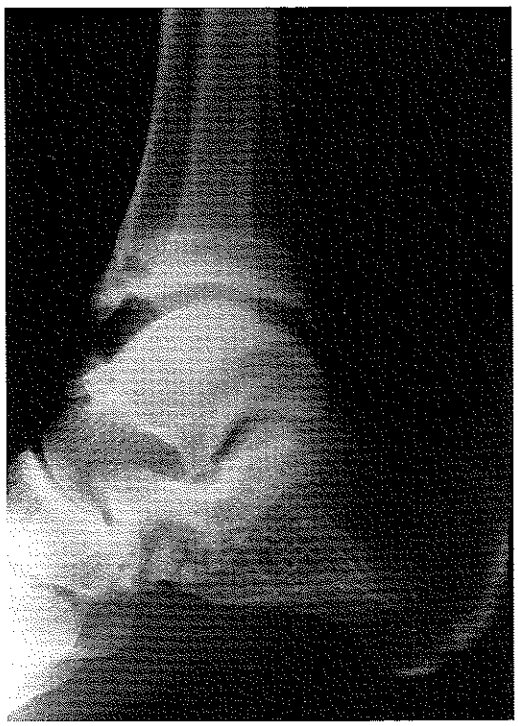


FIG. 37. Anterior or posterior ankle impingement may be coincident with the process of heterotopic bone formation as repetitive stress on the joint capsule provokes the development of exostoses. Exuberant heterotopic bone formation within the ankle capsule and osteophytic lipping of the distal tibia and dorsal talar neck are profound.

tions causing significant restriction of motion typically require more aggressive therapy.

Often the result of ankle fracture or ankle sprain, these calcifications can remain silent, fracture, and create pain, or they may ultimately produce an osseous bridging between closely approximated structures. In particular, coalitions within the syndesmosis and interosseous membrane of the ankle are not well tolerated. As a result, the functional deficit and resulting pain may be relieved by resection of the calcification. Depending on the location of the disease and the preference of the surgeon, arthroscopic or open *surgical resections* may be undertaken. Definitive stabilization techniques may also be necessary depending on the extent of calcification débridement and subsequent instability of the affected joint. Early physical therapy is recommended postoperatively to optimize joint mobility and return to function.

CHRONIC INFLAMMATORY CONDITIONS OF THE ACHILLES TENDON COMPLEX

Achilles tendinopathy is becoming a more common complaint, most likely because of the increased awareness of physical fitness and greater activity levels. Although *achillodynia,* or pain that develops at the Achilles region, has been generically termed *Achilles tendinitis,* Puddu et al. suggested a more specific nomenclature (464). Achilles tendinopathy may be described based on the location of the symptoms, with insertional tendinitis describing an inflammatory process affecting the tendon-bone interface. *Peritendinitis* denotes a generalized inflammation involving the tissues surrounding the Achilles tendon, yet without affecting the tendon itself. *Peritendinitis with tendinosis* describes patients with inflammation of the surrounding tissues and with an associated degenerative process in the Achilles tendon. *Tendinosis* refers to an asymptomatic degenerative process of tendon noted in cases of chronic overuse, cumulative microtrauma, or natural aging. With time and continued tendon wear, clinical symptoms may develop, at which time the term *tendinitis* becomes appropriate.

Anatomy

The *Achilles tendon* is the strongest and thickest tendon in the body. The tendon fibers rotate from medial to lateral as they extend distally. The degree of rotation is variable, although the site of maximal torsion appears to be fairly consistent, noted approximately 2 to 5 cm from the insertion into the calcaneus. The soleal contribution to the superficial tendon insertion increases with greater torsion; less extreme torsion results in a greater gastrocnemius contribution (465). Anterior to the calcaneal insertion is a predictable bursal projection located between the tendon and bone, the *retrocalcaneal bursa.* This bursa is a common site of local irritation and chronic inflammation among patients with a prominent posterior projection of the calcaneus. The Achilles tendon does not possess a synovial sheath, but it is surrounded by a distinct connective tissue envelope known as the *paratenon.* It is highly vascular and is one of the primary sources of blood and nourishment to the tendon. The paratenon is a sheer, gossamer layer of connective tissue matrix fortified with capillaries to help nourish the tendon. Further vascular supply to the tendon is provided by anterior muscular vessels, as well as those from the local periosteum and bone about its insertion. The paratenon has the potential to stretch as much as 2 to 3 cm to allow the Achilles tendon to glide without restriction. This tissue covering also provides for the gliding function between the Achilles and the surrounding tissues. If inflamed, the paratenon may create a significant vascular response, and in cases of chronic irritation, the normal gliding function of the tendon may be impaired. Of particular clinical interest is a relatively dysvascular area that has been noted in the Achilles tendon approximately 2 to 6 cm proximal to the Achilles insertion that is the most frequent site of rupture. In this same area, the tendon is almost perfectly round and at its smallest diameter, and coincidently it is the region of maximum tendon torsion.

Etiology

Inflammation in the paratenon and degenerative changes in the Achilles may result from various factors. *Direct trauma* to the Achilles tendon may begin the inflammatory process. Hematoma, subsequent inflammation, and fibrosis restrict the gliding function of the tendon and produce pain. Histologic specimens of chronic paratenonitis reveal hypertrophic proliferation of loose, poorly developed connective tissue, increased capillary infiltration, and degenerative changes in regional blood vessels (466,467). Fibrinogen deposition is a consistent finding. Other interesting features of these specimens include the lack of inflammatory cells and a poor immune response for healing (468). Benazzo et al. reported a notable increase in glycosaminoglycans, in particular chondroitin sulfate, in a study of surgical explorations in cases of Achilles tendinopathy (469). Disruption of local blood flow return results in a marked leakage of plasma proteins that contribute to the inflammatory process resulting in adhesion and fibrotic change of the paratenon.

Alterations in the normal biomechanical function of the foot and ankle have also been implicated as factors leading to chronic symptoms. In patients who pronate excessively, the Achilles tendon is subjected to rapid opposing forces of rotation. As the subtalar joint pronates in midstance, the calcaneus is everting. Simultaneously, the knee is extending. When both these structures are at the end range of motion, the leg and foot impose contrasting forces of medial and lateral rotation through the Achilles tendon (470). This chronic, repetitive stress may result in pathologic changes affecting the paratenon, the tendon, or both. Thus, patients who undergo excessive pronation from a supinated position, which may be characteristic of a high subtalar or midtarsal joint axis of motion, would appear to be at greater risk. Pa-

tients with this foot type have a greater proportion of transverse plane motion in the foot with the same degree of overall joint mobility. Additionally, compensation for forefoot varus has been correlated with paratenonitis (466,471,472).

Achilles tendinopathy may be aggravated in patients who possess ankle *equinus deformity* caused by triceps surae contracture, although this latter condition is capable of producing Achilles tendon disease in and of itself (470). Patients with such equinus deformity are constantly applying a higher degree of tension to the triceps surae and Achilles tendon, especially when the restricted ankle dorsiflexion is only partially compensated in other anatomic areas. Patients with gastrocnemius equinus who possess normal soleal flexibility would theoretically be more susceptible to inflammatory processes resulting from the different degrees of tension within the tendon fibers.

Patients with *congenital malformations about the retrocalcaneal region* may be subject to inflammatory conditions of the Achilles tendon and its insertion. Osseous prominence of the posterior projection of the calcaneus can promote irritation and inflammation of the tendon with ankle range of motion, perpetuating stress on the paratenon and the distal Achilles tendon. Insertional inflammation, or *Achilles enthesopathy,* is not uncommon in patients with Haglund's deformity, retrocalcaneal spurring, or calcinosis within the tendon. These mechanical irritants are the culprits for chronic inflammation and progressive degenerative change resulting in painful insufficiency of the distal tendon. Hypertrophic spurring at the insertion of the tendo Achillis may incite inflammation because of mechanical irritation. Those patients with recalcitrant symptoms associated with insertional spurring may have an osseous segment that has avulsed and is loose, further adding to the irritation. Rufai et al. found that specimens with a prominent superior tuberosity of the calcaneus had a modified appearance of this bursa. In these cases, a fibrocartilaginous wall surrounded the bursa and replaced contiguous calcaneal periosteum. Because specimens without this prominent superior projection did not exhibit this fibrocartilage, this modified bursal shell is thought to be responsible for retrocalcaneal bursitis (473).

Patients involved in high-intensity activities such as running have also been associated with chronic Achilles tendon disease. The incidence of Achilles tendinitis in runners ranges from 6.5% to 24% (429,430,432). Forces through the Achilles tendon during running approach 6 to 8 times body weight, with an average 800 foot strikes per mile (474). Whether this intense overactivity alone is the sole factor responsible for this process or whether the greater demands of running or other sports simply exacerbate other preexisting potential sources of inflammation has not been investigated.

Systemic arthropathy may also present with an inflammatory process affecting the Achilles tendon. In some reports, this type of process was associated with the HLA-B27 antigen (475), as well as with rheumatoid arthritis.

The *vascular idiosyncrasies* of the Achilles tendon have also been believed to be a potential source for the inflammatory process. As noted previously, the blood supply to the Achilles tendon is diminished between 2 and 6 cm proximal to the calcaneal insertion. Therefore, anoxic changes may be created or exacerbated by certain factors and may lead to degeneration within the tendon. Nuclear medicine imaging has been used to evaluate blood flow to the Achilles and has revealed that perfusion does diminish in this region, particularly after the third decade. This finding seems to be consistent with angiographic studies performed to date (476).

Astrom and Westlin challenged the belief that perfusion insufficiency was responsible for chronic Achilles tendon disorders. Patients with chronic Achilles tendinopathy were evaluated using Doppler flow studies (flowmetry), and perfusion was lower at the distal insertion than throughout the remaining tendon. There was an even distribution of blood flow throughout the tendon body, including the watershed area. Those suffering from chronic tendinopathy showed an increased blood flow for reasons unknown (477). Cerium-labeled microspheres have been used to detail perfusion patterns to the Achilles tendon in white rabbits. This study, conducted after promoting an exercise-induced Achilles paratenonitis and tendinosis, revealed an unaltered perfusion pattern from the paratenon to the tendon during exercise and suggests that tendinosis may not be explained by chronic circulatory dysfunction, in opposition to popular belief (478,479). After assessing 163 patients with chronic Achilles tendinopathy, Astrom and Rausing failed to identify significant disease of the paratenon (468). Histologically, the lack of inflammatory cells and a poor healing response were the most prominent features. Increasing age and male gender were typically associated with more pronounced lesions (480). Thus, the origin of these tendon lesions has yet to be fully explained. Degenerative changes are typically associated with abnormal tendon fiber morphology, focal hypercellularity, and vascular proliferation.

Clinical Presentation

Patients with Achilles peritendinitis typically complain of pain with weight bearing, but they may actually develop more acute pain of a poststatic nature. Commonly, patients have a history of an alteration in recent activity levels or a change in training conditions. In runners, a large percentage of these injuries may be associated a sudden increase in mileage (Table 6) (481–483). Pain at the onset of running may be followed by an interval of relief after a sufficient warm-up, but later as the activity increases, the pain will return. Periods of acceleration or sprinting have been correlated with tendinosis or partial rupture and have been corroborated by surgical exploration (484). Symptoms may also be associated with deceleration during running because of eccentric contraction of the muscle.

Insertional Achilles tendinitis may be associated with a deep pain at the tendon-bone junction of the posterior calca-