**TABLE 6.** *Etiologic factors associated with Achilles tendinitis in 109 athletes*

| Factor | Cases |
|---|---|
| Training errors | 82 |
| Abrupt increase in mileage | 13 |
| Single severe session | 11 |
| Increase in intensity | 6 |
| Hill training | 4 |
| Return from layoff | 4 |
| Combination | 44 |
| Alignment factors | 67 |
| Varus with functional pronation | 61 |
| Valgus pes cavus foot | 6 |
| Gastrocnemius/gastrocnemius-soleus flexibility | 41 |
| Ineffective footwear | 11 |

Adapted from Clement DB, Taunton JE, and Smart GW. Achilles tendinitis and peritendinitis: etiology and treatment. *Am J Sports Med* 1984;12:179, with permission.

neus. Posterior heel pain is predominately activity induced, and at times any type of pressure over the posterior heel is painful. This may make it difficult for the patient to wear shoes in addition to performing the normal chores of daily living. Although tenderness may extend into the Achilles itself, the maximal point of pain is identified at the insertion of the Achilles. Deep, chronic pain between the calcaneus and the anterior tendon border in the region of the retrocalcaneal space is by definition retrocalcaneal bursitis.

Clinically, peritendinitis presents as a generalized edema and swelling in the region of the affected tendon. At times, this may be subtle, and comparison with the contralateral leg makes detection easier. With manipulation of the ankle and palpation with active and passive range of motion, tenderness is elicited, and crepitation may or may not be evident. Lesions within the tendon versus peritendinitis may be distinguishable on physical examination. With tendon lesions, the primary region of tenderness moves with the tendon as the ankle is put through a range of motion, demonstrating a painful arc of motion (485). Conversely, the target area in peritendinitis remains localized despite tendon motion.

## Imaging

MRI is a highly effective modality for evaluation of chronic inflammatory conditions of the Achilles tendon, especially when it is performed in the sagittal and axial planes (486). The tendon itself is normally free of signal intensity, except where it inserts into the calcaneus. Inflammatory fluid accumulates anterior and medial to the tendon and has high signal intensity in peritendinitis, whereas the normal tendon is black (no signal) in T2-weighted imaging. Sagittal plane imaging may demonstrate focal thickening in the watershed region in cases of chronic tendinitis. Signal alteration develops within the tendon after prolonged inflammation. T1 proton density imaging provides superior resolution in cases of partial tendon rupture.

The use of contrast medium enhancement, with gadolinium, for example, has been shown to demonstrate greater sensitivity than ultrasound for tendinopathy (487). Gadolinium enhancement accentuates regions of fluid accumulation and readily identifies edematous processes in both medullary bone and soft tissue structures. MRI has been shown to produce more accurate assessments of lesion size, better discrimination of the extent of tendinopathy, and identification of moderate degrees of degeneration than the ultrasound alternative (309,445,446). Other clinicians find MRI and ultrasound to have similar potential in Achilles tendinopathy and emphasize their use as prognostic tools (488). The use of low-field MRI (0.2 T) has been shown to have a higher sensitivity and better accuracy than ultrasound when correlated with the "gold standard" of histologic correlation (489).

Ultrasound is a first-line modality for approaching tendinopathy, whether acute or chronic, based on utility in diagnosis, noninvasive technique, and cost-effectiveness (331,490–492). It has been used successfully in evaluating Achilles tendinopathy with high sensitivity (493,494). For example, ultrasound findings include identification of rupture through the disappearance of the normal tendon path, in addition to the localization of hematoma formation (495). Ultrasound has demonstrated great specificity and accuracy in follow-up studies of surgical repair in Achilles rupture. Fibrillate restructuring of the tendon was a specific finding noted in these patients that may be responsible for the good clinical recovery of tendon function (330,496). However, ultrasound imaging is operator dependent and relies on skilled technicians and radiologists.

Although normal tendons show a network of well-organized, parallel, and linear fibrillar echoes, the spectrum of tendinopathy can include a variety of changes in the fibrillar pattern: increased fibril thickness, frank disruption of fibrillar bundles, various degrees of fragmentation, and decreased echogenicity depending on the extent of tendon disease (497,498).

The spectrum of tendon conditions consists of: (a) tenalgia without clinical or ultrasonic findings, (b) nodularity in the form of tiny hypoechoic lesions, (c) peritendinous edema, (d) circumscribed tendon swelling, and (e) extensive inhomogeneities. Grading of pathologic lesions of the Achilles has shown promise for early qualification and management of tendon disease (499), and it may be useful as a prognostic indicator supplementing the physical examination (334, 479,500). Ultrasound may be employed for preoperative tendon study as the modality can differentiate inflammatory states from partial rupture (494). In some instances, detection of tendinopathy can be accomplished before the development of clinical symptoms.

In patients with familial disease states, such as hypercholesterolemia, ultrasound has revealed intratendinous xanthoma formation within the Achilles tendon in advance of clinical dysfunction or pain, criteria essential for diagnosis (501–503). Clinical symptoms elicited by manual compression of the ultrasound transducer against an affected area have been shown to correlate often with the subjective complaint and aid in diagnosis and treatment of rheumatologic conditions (504).

Technical innovations in ultrasonography have refined the imaging technique. Conventional frequency probes may prove inadequate for identifying subtle changes associated with the initial phases of inflammatory and degenerative disorders of the Achilles tendon. When conventional ultrasound techniques fail to illustrate disease or when subjective complaints do not clinically correlate, a higher-frequency imaging probe may be employed. High-frequency probes, such as 13.0 MHz as opposed to the conventional 7.5 MHz, are more effective in detecting early phases of tendinopathy (490,497). Further, the use of an angulation technique has demonstrated utility for the identification of early tendinitis, a stage of disease associated with good prognosis (505).

Soft tissue imaging with either MRI or ultrasound has proven helpful in delineating the extent of tendon damage. The study by Astrom and Rausing compared the findings of ultrasound, MRI, and histologic examination in 163 patients with chronic Achilles tendinopathy. Ultrasound findings were positive in 21 of 26 cases, whereas MRI revealed 26 positive studies out of 27. Paratenon evaluation was unreliable regardless of technique. Unique to this study, biopsy specimens were taken from symptomatic and asymptomatic portions of the tendon and paratenon. The paratenon was mostly reported normal or with only slight changes noted. Histologically important features were the lack of inflammatory cells and poor healing response (468). Degenerative changes (abnormal pattern of tendon fibrils, vascular proliferation, and focal hypercellularity) were noted in 90% of symptomatic patients and in 20% of asymptomatic patients. Fibrinogen was identified in most biopsy specimens. Nineteen percent showed partial rupture, which was always associated with regions of tendinosis.

### Treatment

#### *Conservative Measures*

Some *conservative approaches* may be employed in an attempt to alleviate the symptoms of Achilles tendinopathy. First, therapeutic rest may need to be instituted, or in extremely active persons, an alternative form of conditioning or exercise. Oral antiinflammatory medications are also part of the initial treatment protocol for most patients. Any biomechanical or structural deformities that may predispose the patient to tendinopathy may need to be addressed as well. Stretching and strengthening exercises may also be employed as the patient progresses, but in some cases, aggressive exercises may exacerbate the symptoms. Other physical therapy modalities such as ultrasound, deep fiber and ice massage techniques, compression stockings, and orthotic support may be employed. Although the efficacy of orthotic devices has not been scientifically determined in this condition, 70% to 80% of runners have been reported to improve initially with orthotic management (506). Immobilization may also be helpful, especially in patients whose symptoms are resistant to other conservative modalities. Patients with insertional Achilles tendinopathy generally respond to immobilization from either a weight-bearing cast or a Velcro cast if early intervention is not successful.

Variable results have been reported with conservative treatment. Most clinicians appear to favor the use of conservative therapy for 4 to 12 months (475,507–510). Recurrent inflammation and dysfunction may predispose the patient to further Achilles tendon weakness and rupture. Biomechanical compensations and guarding of the affected part can affect the contralateral limb as well.

Anesthetic agents can be used to distend the tendon-peritenon interface, also known as *brisement,* in cases with an audible crepitance or squeaking of the tendon with ambulation. Using a long-acting anesthetic agent for this purpose has led to permanent resolution of symptoms in some patients (508). Steroid injection therapy is generally not performed in these conditions because their use may predispose the tendon to rupture, although injections delivered between the peritenon and the tendon have been reported to be beneficial in cases of peritendinitis (511,512). Read and Motto performed a 12-year retrospective study including 83 athletes with tendo Achillis pain. Steroid injections did not contribute to an earlier return to sport, nor did they have any apparent deleterious effect (513). To date, no well-defined prospective studies have been conducted to determine the actual benefit of local steroid use for this condition. Actovegin (deproteinized hemodialysate) has been investigated in Achilles tendinitis of relatively short duration (less than 3 months). Pforringer et al. reported a significant decrease in the inflammatory process and increased tolerance to full athletic activity with the use of this agent (514). This medication is thought to increase oxygen and glucose consumption in local cells and thus potentiate the healing process.

#### *Surgical Measures*

*Surgical treatment* has been reported as successful in some patients with chronic Achilles tendinopathy. Schepsis et al. reviewed a large series of patients treated surgically for chronic overuse tendinopathy. Out of 79 cases, 79% had a satisfactory outcome. The highest percentage of satisfactory results was noted within the paratenonitis group (87%). Those treated for tendinosis had a much lower percentage of satisfactory outcomes (67%) (515). These findings suggest that peripheral inflammatory conditions respond better to surgical intervention than those conditions causing degeneration of the tendon fibers directly.

Typically, a vertical incision is placed medial to Achilles tendon to avoid the sural nerve. Blunt dissection is carried down to the deep fascial layer. The paratenon should be inspected to determine the quality of the tissue. In cases of chronic inflammation, this layer may be thickened and at times adherent to the underlying tendon. Limited removal of affected paratenon eliminates stenotic redundant tissue. If the underlying tendon appears unaffected, this technique alone can provide good functional results (508,515,516).

Fibrotic tissue, adhesions, and calcifications within the Achilles can at times be visualized or appreciated with palpation of the usually fusiform tendon. Longitudinal incisions, in line with the fibers of the tendon, have been advocated as a means for access to degenerative elements and to allow closer inspection of the tendon while promoting vascular ingrowth for tendon healing (464,516,517). In cases of tendinosis, areas of mucinoid degeneration are débrided. Re-

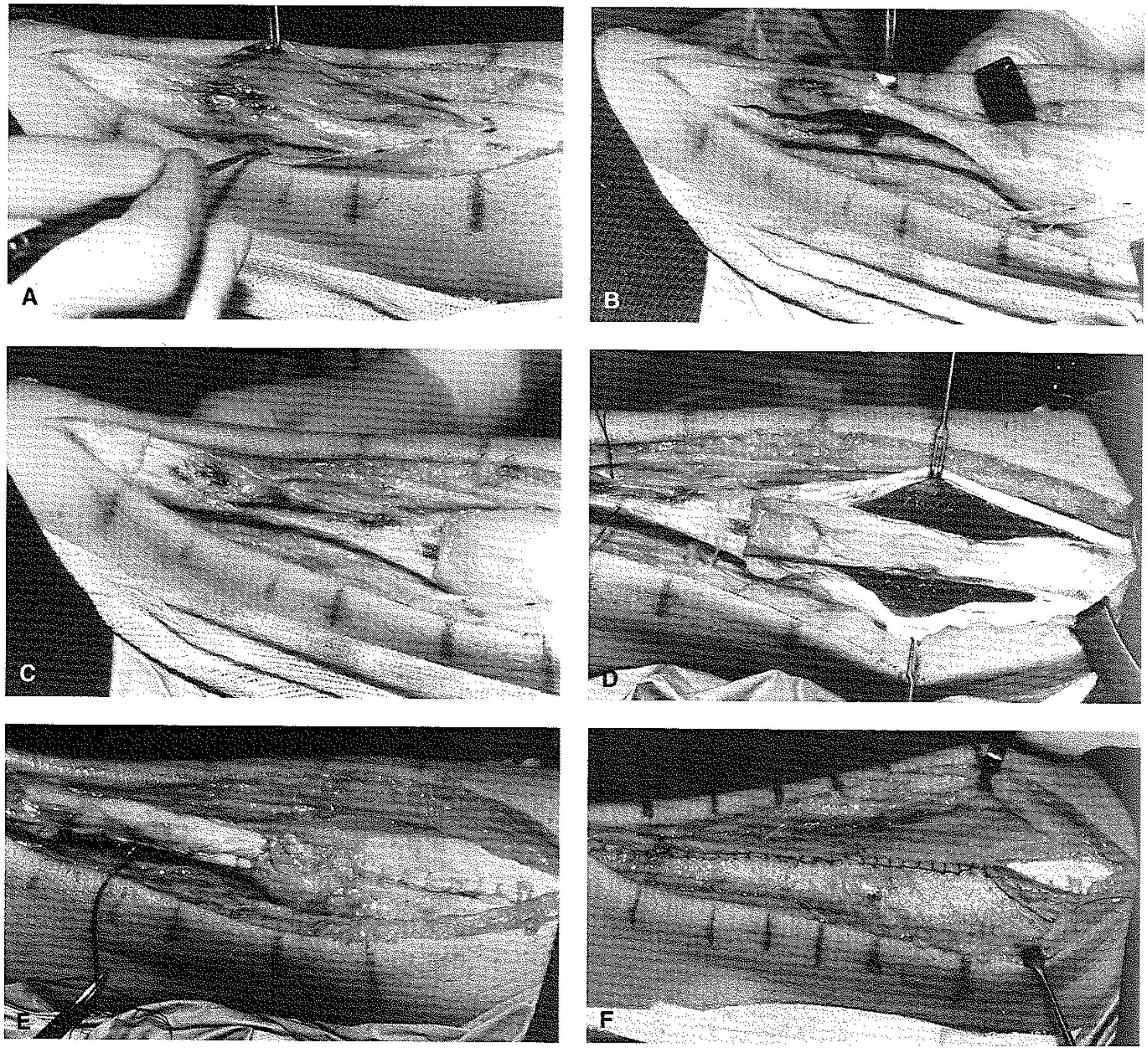

**FIG. 38.** In cases of tendinosis, areas of mucinoid degeneration are débrided. **A:** In this Achilles tendon, the peritenon is thickened and fibrotic, and a focus of intratendinous hemorrhagic synovitis is easily appreciated. The generalized discoloration and yellowing of the deep fascial elements are secondary to chronic inflammation and hemorrhage. **B:** The longitudinal tendon split is appreciated early in the tendon exploration. **C:** After ample débridement, the remnant tendon is insufficient for simple retubularization. **D:** A turn-down flap is designed to reinforce the distal tendon. The proximal aponeurosis is split in the frontal plane to afford a slip of fibers for the distal tendon, and the proximal aponeurosis has a more fibrous texture, whereas the turn-down flap appears more fat laden and glistening. **E:** After harvesting the aponeurosis flap, the donor site is reapproximated, and the distal tendon is retubularized and reinforced with the flap. **F:** Paratenon and deep fascial structures are closed in a single layer.

gions of attenuation are tubularized, and partial tears are repaired before closure. Peritendinitis may be addressed by releasing this tissue layer, whereas hypertrophic paratenon can be debulked. Subsequent closure of the paratenon is avoided to prevent restenosis while the fascia and subcutaneous tissue are closed as a single layer.

Severe cases of tendinosis may be addressed with supplementation by a turn-down flap (Fig. 38). Favorable results have been reported using this technique (484). In cases of significant paratenon injury or insufficient paratenon for coverage of the repair, this structure may be closed as a single layer with the deep fascia.

Benazzo et al. summarized that younger patients with tendon pain of shorter duration generally fared better functionally after surgery (469). The shorter duration of symptoms correlated with lower grades of tendon degeneration. A gradual return to normal passive dorsiflexion is desirable.

*Postoperative Course*

A sterile compressive dressing is applied at the completion of the surgical procedure. Elevation and rest optimize wound healing, most importantly during the first 3 days. The wound is usually evaluated within the next 5 to 7 days. Much of the later aftercare may be dictated by the pathologic features observed at the time of surgery and the repair required. If the paratenon was simply released or excised, the a faster return to range of motion may be instituted. However, if there was significant tendon compromise and subsequent repair, then a short leg cast can be applied and left in place for a period of 4 weeks. However, immediate mobilization has also been advocated (472). After that point, the cast may be bivalved, or the patient may be placed in a Velcro cast to facilitate gentle physical therapy measures. Weight-bearing activities on crutches are titrated to patient tolerance, with full weight bearing permitted at 4 weeks. A heel lift may be helpful through this transition period, and strengthening and stretching exercises may be instituted. Progress is variable and is dictated by the patient's ability to participate in the rehabilitation process. Additional non–weight bearing may still be required for patients with more significant tendon compromise.

## CHRONIC WEAKNESS AFTER ACHILLES TENDON RUPTURE

One of the potential adverse sequelae of any tendon rupture is *weakening,* the result of healing in a lengthened position. Acute rupture of the Achilles tenon is reportedly misdiagnosed in as many as 25% of the cases (518–520). Chronic weakness of the gastrocnemius-soleus complex may lead to an inadequate propulsion in gait, digital contracture resulting from flexor substitution, and pain within the foot as a result of the altered biomechanics.

In patients with untreated ruptures, active plantarflexion is primarily provided by the tendons of the deep posterior muscle compartment. Depending on the interval of time since the rupture, one may note a subtle gap or defect within the tendon itself or an irregular contour to the normally well-defined structures. Swelling is usually present involving the posterior ankle or inferomalleolar area. Pain within the calf or heel may be an indicator of spontaneous rupture or rerupture, but it is not necessarily a cardinal finding because variable presentations have been reported (521–524). Regardless of previous treatment, calf atrophy and an inability to perform a unilateral toe rise are commonly encountered findings (Fig. 39).

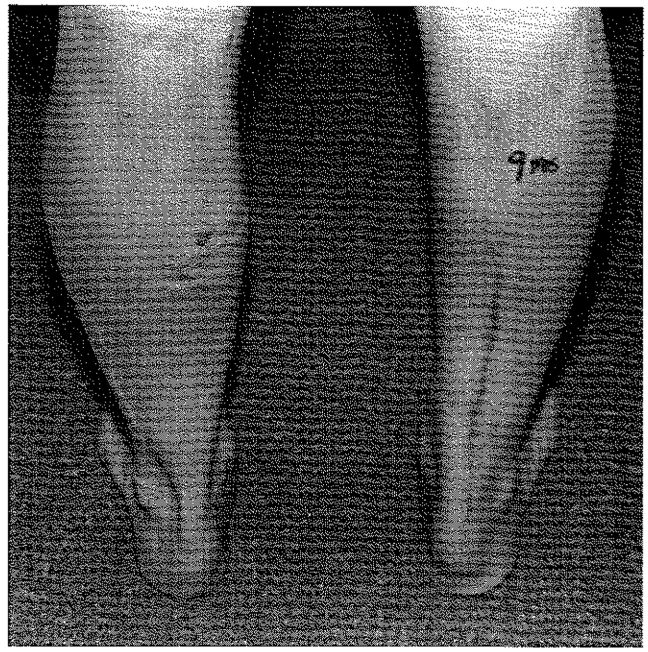


**FIG. 39.** Calf atrophy and persistent weakness are common after repair of neglected Achilles tendon ruptures. This patient is seen 9 months postoperatively, and although the patient is significantly improved, the difference in the bulk of the calf musculature is obvious.

Posttraumatic shortening is thought to occur early, within the first few days of injury, making the differentiation of acute versus chronic rupture an area of debate. In light of this finding, Dalton has suggested a definition for chronic Achilles tendon rupture based on the extent of the tendon defect, rather than on the time elapsed after the injury. Based on this rationale, a chronic rupture would include those tendons with defects that are not fully reducible by simple plantarflexion of the ankle (525). However, this classification does not take into account the potential for rehabilitation of the gastrosoleal complex that may be available if the surgical repair is performed early in the overall process.

Plain radiographs may demonstrate an increased soft tissue density and volume obliterating Kaeger's triangle. Calcifications may or may not be present. The insertional region of the calcaneus may show deformation and spurring. The tendon may appear thickened.

## Treatment

### *Conservative Measures*

*Conservative treatment* is usually reserved for those persons with low weight-bearing demands, patients with medical problems precluding surgery, or those who simply want to avoid invasive procedures. Heel lifts, high-top boots, and ankle-foot orthoses may be employed to assist in managing functional losses and residual biomechanical imbalance. Muscle-strengthening programs are typically employed, particularly in the early phases of rehabilitation. Historically, patients with neglected or dysfunctional Achilles tendon injuries do not respond favorably to conservative treatment, so continued dysfunction is common. Rerupture is reported in as many as 18% of patients undergoing conservative treatment (524).

### *Surgical Measures*

Simple end-to-end repair techniques are usually unsuccessful in patients with neglected Achilles tendon rupture resulting from retraction of the tendon ends. In some instances, the tendon ends are of poor quality, so that there is insufficient tendon volume for direct repair. *Surgical repair* can be challenging. Often, the extent of tendon atrophy, intratendinous degeneration, and calcifications make primary repair impossible. Kann and Myerson categorized procedure selection based on the extent of tendon defect and reserved lengthening procedures for those defects greater than 5 cm (480). These measures also have a place in the overall repair even if additional procedures are employed, particularly if it is believed that the gastrocnemius-soleus complex may be able to resume some level of function once more physiologic tone is applied to the Achilles tendon. Numerous grafting techniques have been suggested over time, with *in situ* tissue transfers dominating the field (526–533) (Table 7).

**TABLE 7.** *Chronic Achilles tendon rupture repair techniques*

| |
|---|
| Primary repair |
| End-to-end (often not feasible in chronic injury) |
| Autologous tendon transfers |
| Achilles |
| Posterior tibial |
| Flexor digitorum longus |
| Flexor hallucis longus |
| Peroneus longus |
| Peroneus brevis |
| Plantaris |
| Autologous grafts |
| Aponeurosis V-to-Y advancement flap |
| Achilles tendon "turn-down flap" |
| Free tendon graft |
| Patellar tendon |
| Rectus femoris |
| Free fascial graft |
| Fascia lata |
| Synthetic repair material |
| Carbon fiber |
| Collagen tendon prosthesis |
| Dacron vascular graft |
| Marlex mesh |
| Polyglycol threads |

#### *Autologous Tendon Transfers*

Addressing the Achilles tendon defect and contracture by tendon lengthening combined with various forms of end-to-end repair is a popular approach. These usually are variations of a gastrocnemius or tendo Achillis lengthening technique (532,534–536). The plantaris tendon has been harvested and transformed into a sheet that is applied across the area of defect (537). Alternatively, the plantaris tendon may be harvested proximally and ultimately weaved through the Achilles in a circular fashion, repeating the weave until sufficient tendon augmentation was achieved (538). Unfortunately, the plantaris tendon is congenitally absent in 30% of the population or often is ruptured along with the Achilles tendon.

The flexor hallucis longus tendon may be harvested and employed for augmentation of Achilles tendon repair. Incisional approaches vary from a medial midfoot approach (529), a posterior approach accessing the long flexor through the Achilles tendon incision, or a two-incision technique over the medial arch and posteromedial leg (526,530,539). Once the tendon defect has been supplemented, it may be necessary to prepare the distal tendon end for tenodesis into the posterior superior calcaneus.

Teuffer devised Achilles reconstruction by transferring the peroneus brevis tendon. All 30 of his patients had satisfactory results at an average follow-up of 5 years (540). Turco and Spinella used this transfer technique in addition to a direct end-to-end repair of the triceps surae with success; no incidence of rerupture was experienced, and all athletes had complete recovery of function in this series of 40 patients (541).

#### *Free Fascial Graft or Free Tendon Graft*

Free fascial grafts from the fascia lata have been used to provide coverage for extensive defects with good functional results (527). Rectus femoris and patellar tendon-bone grafts have been described for use in insertional Achilles tendon rupture repair. This approach provides a bone block for attachment into the posterior superior calcaneus and the tendon affords repair of the defect (542).

#### *Synthetic Materials*

The advantage of using a synthetic material for tendon repair is the avoidance of a second wound may that be necessary for tissue harvest. Dacron has been the material most often employed for the repair of chronic Achilles tendinopathy and rupture. This material is used to bridge the gap between the myotendinous junction and the calcaneus, and it ultimately allows for healing to occur in an end-to-end fashion. Good to excellent results have been reported (543,544), with Lieberman et al. encouraging use of the procedure to allow early return to function (544).

Polyglycol threads have been used for acute tendon rupture repair (545). When compared with autologous tendon graft procedures, similar functional results were obtained. Marlex mesh has been also been employed with satisfactory results, and in one study both improved functional outcomes and decreased soft tissue adhesions were reported when compared with autologous forms of repair (546). Other surgeons investigating the use of polyglactin mesh in animal studies found no functional benefit in repair for acute ruptures (547). Carbon fiber has also been used with favorable results (548,549).

#### *Postoperative Course*

Traditionally, a short leg cast is applied, and non–weight bearing is maintained for 4 to 6 weeks, followed by a similar course of full weight bearing in a short leg cast. Muscular atrophy is common requiring a substantial course of physical rehabilitation to achieve optimal strength. The site of a tendon repair is the weakest area during the first 4 weeks after cast removal. As such, this time interval is most commonly associated with rerupture. However, rerupture of the tendo Achillis more than 4 months after the initial injury is considered rare regardless of the type of initial therapy (521,529,550,551). After complete tendon healing, collagen bundles formed between ruptured tendon ends ultimately provide a tensile strength greater than that of the original tendon. This construction affords resistance against rerupture once sufficient reorganization and remodeling are completed. Nonetheless, elasticity and resiliency are not restored to 100% after a tendon injury. A comparison of functional outcome was conducted by Carden et al., who studied patients undergoing delayed treatment by conservative and surgical methods. Those in the delayed, conservative therapy group achieved an average 74% plantarflexory power versus those in the delayed, surgical repair group, who achieved an average of 91% plantarflexory power compared with the contralateral, uninjured limb (528).

#### ***Complications***

Complications for both conservative and surgical treatment efforts have been reported, and each approach may result in prolonged disability with an unpredictable time for return to function. The most common complication of conservative therapy, approximately 18%, is rerupture (524). Surgical complications have also been noted, including wound infections (519,521,531,552–556), skin slough (524,531,557), sural nerve damage (524,548), deep vein thrombosis, and pulmonary embolism (532,558).

More recently, the rate of surgical complications has been found to be lower than in earlier reports (518,524). Ingliss et al., for example, reported a 17% incidence of wound complications in a population of 150 patients treated for Achilles rupture. Five years later, a 4% incidence was reported in 48 patients. Ingliss and Scott et al. attributed the change in complication rate to refinements in surgical technique (521,553).

## TENDINOPATHY

### The Spectrum of Tendinopathy

Tendon degeneration can be initiated by overuse, direct trauma, or unusual stress. A direct blow or laceration creates an obvious site of tendon vulnerability. Sudden and severe stretch applied across a contracting muscle causes disruption because of unusual stress and strain. Similarly, an extreme force of contraction produces tendon injury. Perfusion defects of tendon, perhaps even microscopic defects, may play an important role in the process of tendon degeneration (559). McMaster investigated the cause and location of subcutaneous tendon ruptures and concluded that normal tendon does not rupture under stress. He demonstrated that applied linear tension across a tendon yields disruption at the myotendinous junction, muscle belly, or at the insertion (560). Normal activity did not cause rupture even when 75% of a tendons fibers were sectioned. In summary, a midsubstance tendon rupture is expected only in diseased tendons. From this realization, the concept of a spectrum of tendinopathy was developed and applies to an array of degenerative tendon changes.

Individual inflammatory processes that occur in or around a tendon unit typically represent a portion of the spectrum of tendinopathy (104,299,561). For this reason, it is important to underline those terms descriptive of progressive inflammatory processes affecting musculotendinous structures. A brief review of the medical terminology is provided herein.

*Tenosynovitis* (also known as tenovaginitis, peritendinitis, or paratenonitis) is an inflammatory process exclusively within the paratenon whether lined by synovium or not (562). Traditional definitions are based on De Quervain's tenosynovitis within the first dorsal compartment of the wrist (563). Predisposing factors for tenosynovitis are of particular importance in occupational health from a prevention standpoint. These predisposing factors may include inadequate tendon perfusion, excessive tendon tension (e.g., excessive gripping force), overuse, sudden overexertion of an otherwise sedentary muscle-tendon complex (often seen in weekend athletes), high-repetition activities that do not allow for adequate muscle recovery, and forceful motions made from positions of poor postural alignment (563). Optimal treatment for tenosynovitis must take into account functional goals and occupational needs in addition to these predisposing factors.

*Tendinitis* is distinctly different from tenosynovitis and represents a symptomatic degenerative, inflammatory process within the tendon itself. Chronic tendinitis can produce an inflammatory reaction within the paratenon and adjacent synovial structures, although these changes are secondary manifestations of the process. Therefore, the distinction between these two entities—tenosynovitis and tendinitis—is based on the primary tissue affected, that is, paratenon versus tendon. Both may ultimately disrupt normal tendon gliding function, but the initial clinical symptoms and progression of disability differentiate the diagnosis. In one, the process initiates within tendon fibers; in the other, it begins peripheral to the tendon.

*Tendinosis* is an asymptomatic process of tendon degeneration (564,565), and it is usually linked with repetitive microtrauma and noninflammatory intratendinous degeneration. Various factors can lead to tendon vulnerability, such as advanced aging, chronic overuse or overtraining, overuse, systemic disease, microtrauma, or a combination of these factors (566). Histologically, the tendon undergoes a spectrum of dysplastic changes over time, including progressive mucoid, fatty degeneration, or progressive calcification and osseous metaplasia. Long-standing conditions may advance into a symptomatic state because of chronic degeneration, dysvascularity, and the inflammation of repair. In this event, symptoms associated with continued tendon stress or overuse are considered tendinitis.

### Tenosynovitis

In the lower extremity, stenosing tenosynovitis represents a chronic degeneration and thickening of the synovial sheath constricting the underlying tendon. This stenosis is the result of persistent inflammation and fibrosis that creates adhesion and painful restriction of tendon movement by interrupting the tendons gliding mechanism. This occurs most commonly in regions where a tendon changes its course of direction. Severe pain may result from untreated or recalcitrant cases of tenosynovitis. In the ankle, this process is most commonly associated with the peroneal, tibialis posterior, and flexor hallucis longus tendons (3,53,111,483,565,567–571). Progressive biomechanical compensation in an attempt to offload the tendon may ultimately produce additional symptoms in other areas of the foot and leg.

Inflammation from systemic disease and infection can produce inflammatory tenosynovitis. This pain syndrome may be the first sign of an underlying systemic process. In rheumatoid arthritis, as many as 10% of patients suffer from some form of tenosynovitis, nearly half with bilateral involvement (572). The peroneal tendons and the tibialis anterior and tibialis posterior tendons seem to be affected, in order of descending frequency, in 50%, 25%, and 25%, respectively. Gout, collagen vascular disease, and infection can all produce a similar syndrome of pain and swelling.

#### *Peroneal Tendon Tenosynovitis*

The peroneal tendons are prone to the development of tenostenosis in four distinct anatomic regions; the retromalleolar sulcus, the peroneal tubercle, the plantar lateral cuboid (peroneal sulcus), and the region between the cuboid and the insertion of the peroneus longus tendon at the plantar lateral aspect of the first metatarsal base (3,573–575). Commonly injured as a result of inversion ankle strain, the peroneal tendons may be stressed by an extreme foot position with an excess of tension produced along osseous pulleys about the lateral ankle joint, that is, the retromalleolar groove and the peroneal tubercle (peroneal trochlea). These regions may demonstrate congenital hypertrophy or may develop osteophytic spurring with persistent overuse. Long-distance runners and endurance athletes are often affected (3,570,576–578). Hypertrophic bone formation with impingement about the lateral wall of the calcaneus would be a common finding in those with a history of calcaneal fracture (579).

The natural progression of recalcitrant peroneal tenosynovitis is more advanced tendon degeneration and dysfunction. Subsequent ankle weakness may ensue. Profound swelling is generally associated with the advanced synovitis of a torn tendon (299). A partial tear is suspected in patients treated for chronic peroneal tenosynovitis who fail to respond to an aggressive course of conservative therapy (292).

#### *Flexor Hallucis Longus Tenosynovitis*

Stenosing tenosynovitis of the flexor hallucis longus tendon is thought to occur almost exclusively in highly athletic patients, although one study demonstrated this problem primarily in middle-aged, nonathletic men (111). Although classically described in ballet dancers, this condition is also reported in association with a variety of sports including equestrian activity, endurance running, any sport requiring significant amounts of jumping, and tennis (580–589). These patients can present with a complex set of symptoms, flexor hallucis longus tendinitis, plantar fasciitis, and tarsal tunnel symptoms may overlap resulting in misdiagnosis. Therefore, the term *flexor hallucis longus dysfunction* has been suggested to describe these complicated cases in which the primary affliction is suspected to be within the flexor hallucis longus tendon (111).

A high degree of suspicion is required to identify this disease. Passive manipulation of the tendon is not an aggravating factor and can be misleading in the clinical assessment. Mechanical tenosynovitis of the flexor hallucis longus tendon may be described as pain within the plantar foot with tenderness to palpation along the course of the tendon. Often misdiagnosed as plantar fasciitis and heel spur syndrome, the target region of tenderness is not the medial calcaneal tuberosity. However, extreme dorsiflexion of the metatarsophalangeal joints accompanied with deep palpation within the superficial plantar medial arch will exaggerate the contour of the inflamed flexor hallucis longus tendon. Flexor hallucis longus tenosynovitis may result in tenderness spanning from ½ inch distal to the medial calcaneal tuberosity to the distal aspect of the medial longitudinal arch (572).

Hamilton reviewed the pathomechanics associated with this condition as it presented among classical ballet dancers and coined the term *dancer's tendinitis* because of its increased frequency among these athletes (96). Commonly, an insidious onset of symptoms is described, with posterior ankle pain radiating distally through the medial arch. Clinically, tenderness and crepitation are noted about the ankle and first metatarsophalangeal joint with both passive and active maneuvers. Triggering of the hallux may be appreciated (120,121,581,588,590–592). Although often mistaken for plantar fasciitis, the symptomatic proximal, plantar flexor hallucis longus tendon is much more superficial than the deep enthesitis of the plantar fascia.

#### *Anterior Compartment Tenosynovitis*

All structures of the anterior compartment are subject to injury, especially in field sports. This compartment includes tibialis anterior, extensor hallucis longus, extensor digitorum longus, peroneus tertius, associated tendon sheaths, and retinacular structures (transverse crural and cruciate crural), each of which can become affected by an inflammatory process. These structures are superficial and are subject to direct injury in many aggressive athletic activities such as football and soccer. The chronic irritation of repetitive ankle dorsiflexion and plantarflexion may result in an overuse phenomena producing local swelling, warmth, and redness.

Protection from repetitive trauma and friction is the primary goal in treatment. Rest, ice, compression, and elevation are the first steps in treating acute flares of chronic pain syndromes. Conservative measures include accommodative padding to protect vital structures such as the extensor tendons and anterior neurovascular bundle, which can be irritated by a shoelace pattern or excessive ankle motion. Antiinflammatory agents and physical therapy can be used.

### Tendinitis

Tendinitis is a symptomatic injury or degeneration of tendon that is associated with intratendinous disease and local inflammation causing secondary peritendinitis.

The clinical presentation of tendinitis is variable, depending on the duration of the inflammatory process. Therefore, the terminology is expanded. The classification of tendon injuries by Clancy et al. includes a chronologic spectrum for grading tendinitis (104,562). *Acute tendinitis* is that condition causing pain and local swelling present for less than 2 weeks. *Subacute tendinitis* is represented by those symptomatic conditions of 2 to 6 weeks' duration, whereas *chronic tendinitis* includes those conditions persisting for greater than 6 weeks. Chronic tendinitis can result from injury or systemic disease producing a gradient of disease that can be distinguished by gross examination of the tendon and surrounding soft tissue structures. Clancy et al. described the various subdivisions of chronic tendinitis based on surgical observations and summarized the findings into four principal categories of tendon degeneration (483). These include (a) interstitial microscopic tendon failure, (b) central necrosis, (c) frank partial rupture, and (d) acute complete rupture. Presenting symptoms and associated degrees of dysfunction increase in accordance with the level of tendon disease. Advanced stages result in structural deformity and progressive tendon insufficiency leading to tendon failure in the case of untreated or recalcitrant conditions.

#### *Peroneal Tendinopathy*

Specific interest in peroneal tendinopathy dates back to 1924, when Meyer first described what is known as the *longitudinal peroneal split tear* (593). Since that time, peroneus brevis tendon disease has been described in detail (260,299,310,559,594,595). The literature of the late 1980s reflects an increased recognition of peroneus brevis disease and suggests that peroneal tendon degeneration has previously been underreported (310,594). Sammarco and DiRaimondo identified 11 cases of peroneus brevis tendinopathy in a review of 47 lateral ankle reconstructions (595). Noting this disease in the face of ankle instability seems to call into question the actual origin of the dysfunction. If the peroneus brevis split was an additional culprit in these cases of ankle instability, then a lateral ankle stabilization alone would not be curative (276). Sobel et al., in some cadaveric studies, noted attritional changes within the peroneus brevis tendon in 21 of the 57 ankle specimens explored (310). These studies led to the development of a grading scheme for peroneus brevis splits (Table 8).

##### *Clinical Evaluation*

Lesions of the peroneus brevis tendon are generally insidious in onset, and chronic lateral ankle pain may be associated with subtalar or ankle instability. Tendon rents or tears are typically located at the distal tip of the fibula and usually extend 1 to 4 cm just proximal and distal from the center of the lesion (292,299,596,597).

Chronic lesions of the peroneus longus tendon arise in a similar fashion and may present as chronic lateral heel or midfoot pain. Often, pain and tenderness occur in the vicinity of the cuboid sulcus and may be associated with an os pero-

**TABLE 8.** *Sobel classification for split peroneus brevis tendonopathy*[a]

| |
|---|
| Grade I: Tendon is splayed |
| Grade II: Split; partial thickness < 1 cm |
| Grade III: Split; full thickness 1–2 cm |
| Grade V: Split; full thickness > 2 cm |

[a] Based on cadaveric sampling.

neum. Clinically, tenderness accompanies a subtle fullness where the tendon courses across an osseous pulley (the fibula or cuboid). In this region, the tendon changes direction and endures chronic shear stress. With muscle contraction, the tendon is compressed against bone.

Split tears of the peroneal tendons are so called because of their longitudinal orientation within the tendon substance, not unlike disease found in the posterior tibial and flexor hallucis longus tendon. Often, these tears are associated with overuse syndromes, and chronic injury in athletes is considered a common occurrence (594,598–600). Partial tears should be suspected in patients with a presumptive diagnosis of tenosynovitis that fails to respond to conservative care (292).

Chronic, persistent edema along the peroneal sheath is perhaps the most consistent clinical finding in peroneus brevis tendon disease. Popping and clicking sensations may be identified with active eversion of the foot. Actual subluxation of the peroneal tendons may not be evident because tendon degeneration and dysfunction can result from subclinical tendon luxation. The *peroneal compression test* reproduces the pain. Compression of the peroneus longus against the brevis tendon will force the anterior surface of brevis against the retrofibular groove (260).

Krause and Brodsky devised a classification strategy meant to guide surgical treatment (299). Grade I lesions involve less than 50% of the cross-sectional area of the tendon and merit débridement with direct repair. In grade II lesions, which involve greater than 50% of the tendon cross-section, débridement in addition to a peroneus brevis-longus anastomosis is advised. Many of these tendons had multiple longitudinal tears of variable thickness with fraying of the peroneus brevis tendon. Typically, pathologic features are confined to an area adjacent to the tip of the fibula, either just proximal or distal. The morphology of the tendon is more flattened than round as a result of the split tears. Rarely, tears extend toward the base of the fifth metatarsal. Here, splaying of the tendon is considered a sign of advanced tendon disease. Stabilization of these tendons in an appropriate position of function behind the fibula is necessary for optimal function. Krause and Brodskyed report much better functional results in cases that did not involve workers' compensation (91% versus 56%). Nonetheless, 15 of 20 patients reported satisfactory results at an average of 39 months of follow-up (299).

### *Pathomechanics in Peroneus Brevis Split Tears*

The peroneus brevis undergoes degenerative changes resulting from mechanical friction and shearing injury within the retrofibular groove. Dynamic mechanical injury can also occur in the region of the calcaneofibular ligament. Sobel considered each of the primary components of the peroneal tunnel as potential culprits for abnormal tendon wear, that is, the superior retinaculum, the prominent retrofibular spike, a shallow fibular groove, and compression from the peroneus longus. Laxity of the superior peroneal retinaculum leads to instability about the retrofibular pulley. This condition, in addition to a shallow retrofibular groove, lends to instability and tendon subluxation. Subsequent peroneal subluxation compresses the longus tendon against the brevis and imposes the anterior border of brevis against the fibular recess. Subclinical subluxations likely begin the process and develop over time into frank tendon luxation. These physical stresses result in repetitive trauma and excessive tendon wear. This inordinate wear is linked with degenerative changes that develop longitudinally within the brevis tendon.

Anatomic variants have also been related to peroneus brevis tendinopathy and include a hypertrophic fibular ridge, the presence of peroneus quartius, anomalies of peroneus brevis tendon, and the presence of accessory or anomalous peroneal tendon (259,285,305,306). Each of these factors imposes pressure and constriction on the tendons of the lateral compartment. This decreases the area available for normal tendon excursion and results in chronic friction and instability about the fibular pulley. Progressive degeneration and consequent subluxation of the tendon unit result from this intracompartmental stress. Ultimately, the peroneus longus tendon is affected by persistent dysfunction and inflammation. In severe cases, we have found that the split peroneus brevis rests superficial to the longus tendon, and the longus tendon actually herniates through the brevis split and displaces it to a more superficial position in the leg.

The longitudinal orientation of attritional changes within the brevis tendon underlines that a progressive degenerative process has been taking place, rather than a focal acute insult causing partial transection or rupture. Of the larger studies reported, these lesions appear on the anterior surface of the tendon consistent with the theory of direct mechanical friction within the retrofibular sulcus (310,594,601). Although some investigators believe that hypovascularity is a culprit for degeneration in regions of high tendon shear, other studies dispute this theory (313,324). Histologic studies discredit the concept that dysvascularity incites this specific tendon disease. A hyperprolific condition with increased vascular and fibroblastic ingrowth is commonly described in the literature (310,506,598).

### *Imaging of the Peroneal Tendons*

*Radiography.* Plain radiographs are the mainstay in initial evaluation of chronic ankle pain. Routine ankle views include AP, lateral, and mortise (15 degrees internally positioned) views. Signs of fleck fracture along the distal fibula, periostitis, or degenerative changes may correspond to clinical symptoms. Calcaneal axial views accentuate a hypertrophic peroneal sulcus and associated spur formation.

*Magnetic Resonance Imaging.* Acute lateral ankle injury is routinely managed on the basis of physical examination and plain radiographs. In contrast, painful chronic ankle conditions often require the aid of further ancillary imaging. MRI has been shown to demonstrate lesions of the peroneus brevis, but with a significant number of both false-positive and false-negative studies (299). MRI has been shown to have 100% specificity in imaging tendon tears, although in the same study, accuracy and sensitivity were disappointing (65.75% and 23.4%) (602). Because small tears and minimal degenerative changes can be missed, a normal or negative MRI scan of the peroneal tendons does not preclude surgical exploration.

Normal tendons have a smooth contour and appear purely black on both T1- and T2-weighted image sequences. Any signal emanating from within the tendon is suggestive of disease, although the magic angle effect is a frequent culprit for abnormality. Sequences with short echo times (T1 and proton density images) produce an increased signal intensity when the tendon is 55 degrees from the axis of the primary magnet. This magic angle effect does not persist after repositioning. This effect can be seen in normal tendon, particularly within the brevis tendon when the foot is neutral to the ankle. Positioning the patient with the foot plantarflexed best reduces the incidence of this imaging defect (597,603).

Just above the retromalleolar groove, the peroneus brevis tendon assumes a flat to crescent shape as it conforms to the shape of the posterior fibula. The peroneus longus tendon appears more well rounded or globular in this region, with the brevis tendon in anteromedial position. The superior peroneal retinaculum is delineated as a thin, low signal intensity from the lateral aspect of the retrofibular groove and inserts into the lateral border of the Achilles tendon and the lateral wall of the calcaneus.

Peroneus brevis splits emit an increased signal intensity on both T1- and T2-weighted images, with notable changes in girth and irregularity of contour. Typically, splits are found centered at the retrofibular groove, often with proximal and distal extensions. These splits are seen as clefts within the tendon and can be partial or complete (561,604). Rosenberg et al. imaged 10 asymptomatic patients and 31 with evidence of peroneus brevis split for comparison. Characteristically, the split brevis tendon appeared wrapped around the longus tendon in a C-shaped configuration, a hallmark appearance on axial images. Preoperative MRI scans exhibited a C-shaped tendon with high intratendinous signal intensity evident in all proton density images. This morphologic appearance is characteristic of peroneus brevis tear and is distinct from the chronic degeneration and tear of other ankle tendons. In partial tears, the peroneus brevis appears thinned centrally, with more globular extensions resting medial and lateral to the peroneus longus tendon. In a full-thickness tear, the peroneus brevis tendon may appear medial, lateral, or posterior to the longus tendon. Typically, a reconstituted tendon is identified along the lateral calcaneal wall. In one-third of the caases reported by Rosenberg et al., the peroneus longus was split as well, although these lesions did not have the characteristic flattened or C-shaped appearance of the peroneus brevis split. MRI data have been corroborated by surgical findings and include hypertrophic synovial proliferation, degenerative tendon, split tears, and a frayed tendon appearance (604).

Best demonstrated on T2-weighted or STIR images, a high signal intensity peripheral to the tendon is suggestive of fluid or hyperemic synovium. Thinning and tendon irregularity can easily be identified. Fluid within the sheath and adjacent soft tissues is common. A low to intermediate signal intensity on T2-weighted images is apparent in stenosing tenosynovitis (299,603). T2 and proton density images reveal low signal intensity consistent with fibrosis or scar within or around the tendon. Incidental degenerative changes within the brevis tendon have been noted in asymptomatic, elderly patients and may reflect chronic attrition and mechanical wear resulting from daily living (604). In a small study, chronic peroneal tendinitis was distinguished from longitudinal tendon splits using this technique (604).

Coincident ligamentous damage, anatomic variants, and peroneus longus tendon tears have also been noted (299,591,592,599). The peroneus longus tendon is thought to undergo degenerative change as the shock absorption provided by peroneus brevis is lost resulting in shear stress of the longus against the fibula. Frequently, the peroneus brevis may be present as a bifurcated tendon in the leg that becomes confluent at the ankle level. This finding can be misinterpreted as a pathologic tendon split (604).

Occasionally, a fibrocartilaginous ridge is noted, triangular and with a low signal intensity. This ridge has a well-defined marrow component and is a relatively common occurrence, appearing meniscoid and thickened (315). The morphology of the retrofibular sulcus is typically concave, although flattening or convexity may be associated with disease. The presence of a peroneus quartius or a distal extension of the brevis muscle belly can be depicted on axial images in the region of the distal fibula (292). Hypertrophy of the peroneal tubercle is also appreciable in this plane.

*Ultrasound Imaging.* Ultrasound may also be employed to evaluate peroneal tendon disease. For optimal tendon resolution and high-contrast imaging, a 7.5- to 10-MHz transducer is typically employed (449,491,602). Routinely, two orthogonal planes are imaged, such as longitudinal (sagittal) and axial (transverse). In longitudinal cross-section, tendon fibers appear as echoic fibrils in a parallel orientation. Disruption of this fibrillar arrangement is literal and is consistent with tendon fiber disruption. The anisotropic effect may cause fibers to appear hypoechoic because of their oblique orientation to the transducer sound beam (561). Similar to the magic angle effect seen in MRI, this defect can be differentiated from true tendon disease by changing the angular relationship between the sound beam and the subject. Because this maneuver is easily accomplished in real-time imaging, this defect poses no great interruption in imaging. With transverse plane imaging, fibers have a rounded or ovoid appearance. The tendon sheath is illustrated as a less echoic tissue immediately peripheral to the tendon fibers. The normal tendon sheath contains a small amount of hypoechoic fluid best appreciated distal to the malleoli. Ligamentous structures appear hyperechoic, and fibers generally are more compact than tendon. The sound beam must be perpendicular to the ligament for visualization because such deep ligaments may be difficult if not impossible to visualize. Muscle fibers appear hypoechoic in contrast to tendon or ligament. Osseous structures appear hyperechoic because of the interface between tendon and bone.

Peroneal splits are C-shaped or boomerang-shaped at the level of the fibular malleolus. In sagittal plane, longitudinal imaging, a fusiform swelling with a hypoechoic centroid is noted. Disruption of the fine, parallel echoic fibers of tendon accompanies a notable distention of tendon sheath by hypoechoic fluid. Transverse or axial plane imaging shows disruption of the bundles of rounded echoic fibers. Disruption of the fibrillar arrangement of tendon fibers is indicative of tendon disruption and must be confirmed by imaging in an orthogonal plane, that is, two views at 90 degrees to each other. Hypoechoic clefts or gaps extend from the periphery into the tendon substance. Rockett et al. compared the utility of ultrasound to MRI in a prospective study of tendon abnormality about the ankle. Peroneus brevis disease was identified in 9 of 13 tendons after an average 26.63 months of symptoms. Although MRI was obtained in 10 of these cases, only 1 was considered positive for disease. Overall sensitivity and accuracy were superior using ultrasound (100% and 94.41% ultrasound versus 23.4% and 65.75%, respectively). No false-negative studies and only 4 false-positive results were identified using the ultrasound technique. In contrast, the MRI studies included 13 false-negative and no false-positive results, with a specificity of 100% (602). True-positive studies using both modalities correlated accurately to the location of the tendon tear.

Simple fluid collections or water will appear anechoic, providing an imaging window to the structures behind it. More complex fluids collections may be loculated by fibrous septa, which are hyperechoic. Sediment or fluids containing solids show diffuse homogeneous hyperechoic signals. Elements of air and gas produce hyperechoic punctate signals that create a posterior shadow-effect "comet-tail" artifact (561). Hemorrhage, granulation tissue, heterotopic calcification, and hypertrophic synovium appear intense or hyperechoic.

Peroneus longus tears can easily be appreciated with ultrasound, although this disorder is considered rare in comparison with peroneus brevis tears (449,597). When an avulsion or fleck fracture of the posterolateral fibula is present, it may be readily identified using ultrasound. The frequency of occurrence for this bone fragment on plain radiographs is 15% to 50% in acute injuries. Ultrasound has the advantage of being able to identify tears or avulsions of the retinaculum in the absence of this avulsion fragment. The presence of a torn superior peroneal retinaculum is evidenced on axial (transverse) images as a hypoechoic space anterior to the tendons (449). Crowding of the retromalleolar peroneal tendons can be appreciated dynamically using real-time imaging. Performing active eversion activity or circumductory motion of the foot and ankle under ultrasonic guidance demonstrates dysfunction to crowding within the compartment.

### *Peroneal Tendinitis*

Tendinitis within the peroneal group is caused by irritation as the tendons function across the lateral malleolar pulley. Loss of tendon gliding function results as an oblique line of stress is applied across the tendon coursing from the leg, around the ankle, and into the foot. A regional degeneration results from mechanical drag or friction. Soccer players suffer from this process in the ankle as a result of direct or indirect injury. Often, the result of a direct kick to the peroneal tendon region this process can also be incited by the strain of kicking the ball, such as an outside kick. Concomitant contusion of the tendons, retinacular structures, and tendon sheaths is possible. Associated symptoms of pain and inflammation are typically noted proximal and distal to the lateral malleolus. Aggravating activity includes resisted eversion or passive inversion of the foot with the foot neutral to the leg. Crepitance may be present as the surrounding synovial structures become involved.

### *Peritendinitis Crepitans*

*Peritendinitis* (also known as tenosynovitis) is an inflammatory condition of the tendon sheath, *paratendinitis* refers to an inflammatory condition of the paratenon, and *peritendinitis crepitans* refers specifically to an inflammatory reaction in the region of a musculotendinous junction. Clinically, this entity can be identified with manipulation and deep palpation of the affected tendon. Passive and active tendon motion will produce tenderness and evoke crepitation in the region where muscle meets tendon (93,605–606). Differentiation of peritendinitis from tendinitis is determined in a similar manner. In peritendinitis, the target area of pain is a focal region of the paratenon; as the tendon glides within its sheath, this target area of pain remains unchanged. With tendon excursion, the target of tenderness does not change in peritendinitis. This is different from tendinitis, in which the area of tenderness moves with manipulation of the tendon; that is, as the tendon glides, the affected section of tendon, the target of pain, migrates in the direction of tendon excursion (Fig. 40).

### *Treatment*

Conservative therapy for tenosynovitis and tendinitis is the same and is generally considered successful (566, 578,592). Various methods are commonly used in combination to provide clinical improvement, including abstinence from the offending activity, limited immobilization, oral antiinflammatory medications, physical therapy modalities, accommodative padding, splinting, and injectable steroids.

For athletes in particular, a close review of the type of

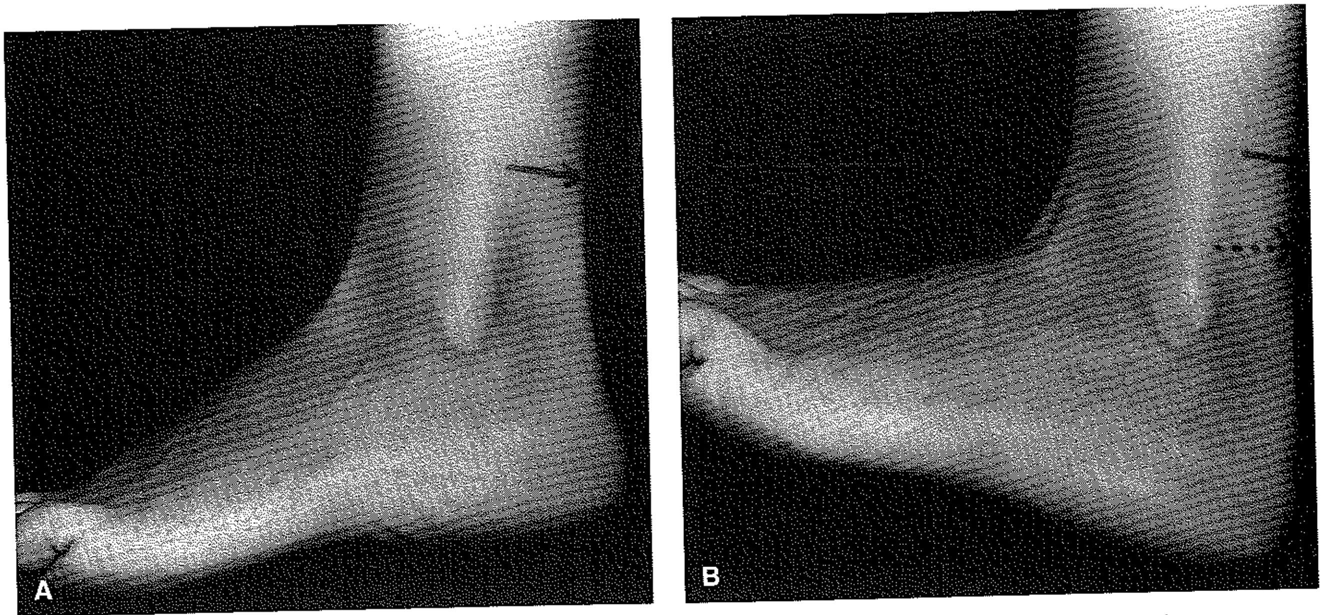


**FIG. 40.** Inflammatory conditions affecting the paratenon can be differentiated from tendinitis by virtue of its location, which remains unchanged despite tendon motion. In tendinitis, the point of maximum tenderness migrates as the tendon moves within its sheath. **A:** The *solid arrow* indicates point tenderness, the target area of pain within the tendo Achillis. **B:** The *dotted arrow* indicates that the target of pain has moved distally with dorsiflexion of the ankle, albeit modestly, indicating that the pathologic process is within the tendon itself. A lesion of the paratenon would remain fixed in position despite tendon motion.

training, to include frequency and endurance, should be evaluated. Extremes in conditioning may need to be modified, if only temporarily to reduce stress and strain, assisting in recovery. A long-term regimen of aggressive stretching may be maintained for the duration of the sport season or daily activity. Orthotic therapy, to include wedging techniques, is often employed for reducing stress within the affected tendon. Chronic tenosynovitis and tendinitis may require a prolonged course of conservative therapy (483).

The premise behind surgical treatment has been described as a restorative process that induces a wound repair cycle. Cell-matrix modification is thought to be responsible for the removal of residual degenerated fibers and tendon remodeling (607). Although scar replaces all injured or repaired tendon fibers, intervention is believed to optimize the process. Surgical treatment for chronic tendinopathy includes limited synovectomy and tendon débridement. All areas of fibrosis, adhesion, and calcification should be removed from the affected soft tissue (tendon or synovium), with augmentation procedures performed when necessary.

In the event that a peroneal anastamosis is performed, a deliberate functional advantage is given to the brevis tendon. The brevis tendon should be placed under tension while slack is maintained on the peroneus longus tendon. This is achieved by applying traction on the brevis tendon proximally while pulling the longus tendon distally. With the brevis tendon under tension and the peroneus longus relaxed, a side-to-side anastomosis is performed. A sufficient section of tendon is sutured approximately 4 to 5 cm proximal to the tip of the fibula and again at an equivalent position distal to the fibular tip. This technique provides strong support of the anastomosis with improved mechanical balance across the ankle favoring restriction of ankle inversion. Tendinous structures should be repaired or augmented with a nonabsorbable material.

The postoperative course varies in accordance with the procedure performed. In the event of a simple synovectomy, early return to range-of-motion exercises and progressive weight-bearing activity is instituted based on patient tolerance. Formal tenodesis, retinacular repair, or lateral ankle stabilizations require a non–weight bearing short leg cast for 5 to 6 weeks. Range-of-motion exercises and protected weight bearing in a castwalking boot are necessary for a commensurate period of time. Range-of-motion exercises can typically begin with conversion to protected weight-bearing status. Advancement of weight-bearing activity and frequency of range-of-motion exercises vary, based on the surgeon's discretion and the patient's tolerance of activity.

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