Achilles Tendon Injuries

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Abstract

As the number of persons who participate in athletic activity into their later years has increased, so has the incidence of overuse injuries to the Achilles tendon. The etiology of these problems is multifactorial and includes biomechanical factors and training errors. Use of a histopathologic scheme for classification of these injuries facilitates a logical approach to treatment. Conservative care is a mainstay of treatment for inflammatory conditions. Satisfactory outcomes may be obtained with either nonoperative or operative treatment of acute ruptures, although surgically treated patients appear to recover better functional capacity. Treatment of neglected injuries to the Achilles tendon continues to be a challenging problem.

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Insufficient preparation, overstrain, lack of general conditioning, and the pressure to succeed in sports all contribute to injury of the tendon named after the seemingly invincible Greek warrior Achilles. Participants in any sport involving repetitive impact loading associated with jumping are at an increased risk for Achilles tendon difficulties. In a prospective study of serious runners, approximately 10% had Achilles tendon problems within the 1-year observation period.[1] However, a fourth of all patients who present with Achilles tendon injuries give no history of athletic involvement or antecedent trauma.

Etiology

Most Achilles tendon problems are related to overuse injuries and are multifactorial in origin. The principal factors include host susceptibility and mechanical overload. The primary host factors are biomechanical malalignments in the lower extremity and increasing age. Both hyperpronation and cavus foot have been associated with Achilles tendon problems. Marked forefoot varus has been found to be more common in athletes with Achilles paratenonitis and insertional complaints.[2] The cavus foot has also been associated with a high rate of insertional difficulties. The cavus foot is thought to absorb shock poorly and to place more stress on the lateral side of the Achilles tendon.

Advancing age has been definitely shown to correlate with Achilles tendon overuse injuries. It has been hypothesized that decreased tendon vascularity associated with aging is the basis for the association of tendinopathy with aging. However, recent studies using laser Doppler flowmetry have brought this commonly espoused theory into question.[3]

Several mechanical factors have been implicated as part of the multifactorial etiology of Achilles tendon problems. Inappropriate footwear with insufficient heel height, rigid soles, inadequate shock absorption, or wedging from uneven wear can magnify the stresses exerted on the tendon during activity.[2] Training errors include sudden increases in training intensity, excessive training, training on hard surfaces, and running on sloping, hard, or slippery roads. A change in training schedule shortly before injury has been recorded in as many as 50% of running injuries.

Anatomy

The Achilles tendon is the largest tendon in the body. It is composed of tendinous fibers contributed by the gastrocnemius and soleus muscles (Fig. 1). As these fibers coalesce, they spiral toward their insertion on the calcaneal tuberosity.

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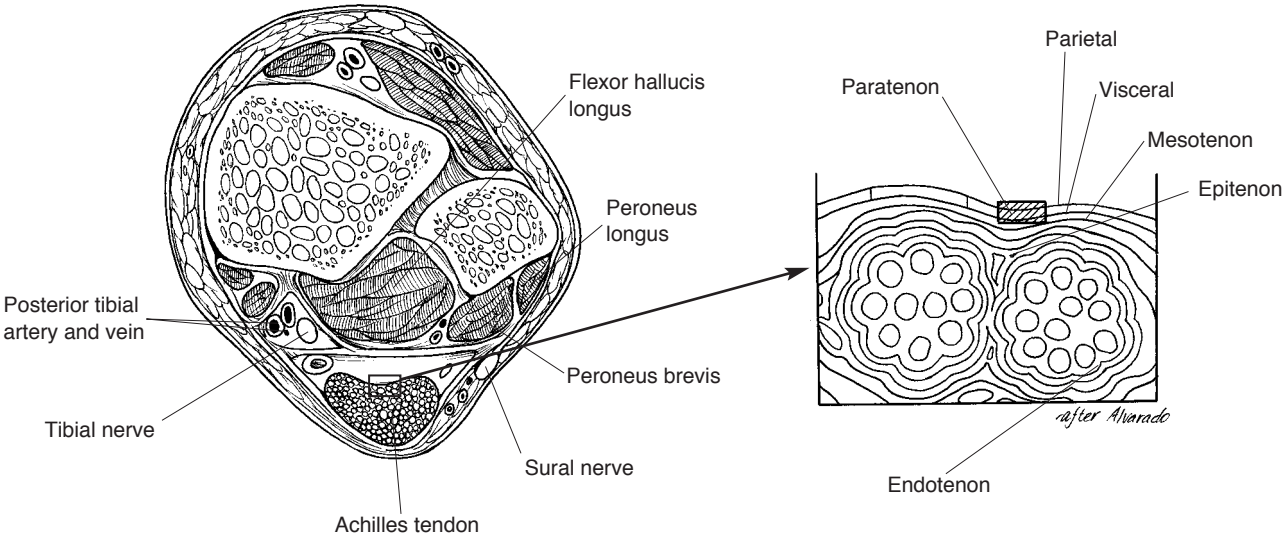

Fig. 1 Cross-sectional anatomy of the leg at the level of the Achilles tendon **(left)** with a magnified view of the peritendinous structures **(right)**. The double-layered paratenon surrounds the tendon. The mesotenon connects the outer, parietal layer to the inner, visceral layer and serves as a passageway for vessels nourishing the tendon. The density of these vessels is highest along the anterior tendon.

The Achilles tendon lacks a true synovial sheath; rather, it has a paratenon with visceral and parietal layers, allowing approximately 1.5 cm of tendon glide. In the dorsal, medial, and lateral regions, the paratenon consists of multiple thin membranes, rich in mucopolysaccharides, that function as a well-lubricated gliding layer. On the ventral side, the paratenon contains richly vascularized fatty tissue.

The blood supply to the Achilles tendon arises from three sources: the musculotendinous junction, the osseous insertion, and multiple mesotenal vessels. The mesotenal vessels are a series of transverse vincula that serve as conduits through which blood vessels can reach the tendon. Injection and nuclear imaging studies have shown that the mesotenal arteries are fewest at a level 2 to 6 cm proximal to the osseous insertion. Similarly, the number of intratendinous vessels and the relative area occupied by vessels are lower 4 cm from the calcaneus.[4]

Physiology and Biochemistry

The Achilles tendon is composed of mature fibroblasts (tenocytes) imbedded in an extracellular matrix consisting of collagen, elastin, mucopolysaccharides, and glycoproteins. Tenocytes and collagen fibrils align and form regular compact bundles invested in layers of collagen (the endotenon) surrounded by a connective tissue layer rich in blood vessels (the epitenon). It has been shown that with normal aging, the Achilles tendon undergoes substantial morphologic changes, including decreased cell density, decreased collagen fibril diameter and density, and loss of fiber waviness. These natural changes may contribute to the higher injury susceptibility of older athletes.[5]

A healthy Achilles tendon has a remarkable capacity to adjust to local mechanical stimuli. In response to exercise, the diameter of the tendon thickens; in response to inactivity or immobilization, it atrophies. Studies in animals have shown that controlled training influences tenocyte activity, resulting in increased matrix-collagen turnover and thickening of collagen fibrils and fibers. Biomechanically, tendon tensile strength and stiffness increase with continuously repeated loading. The natural time course for plasticity of this tissue explains why gradual changes in athletic training are much better tolerated than abrupt changes.

Biomechanics

The gastrocnemius-soleus-Achilles complex is a myotendinous unit spanning three joints. Although we tend to think of the Achilles tendon as a flexor of the tibiotalar joint, active gastrocnemius-soleus muscular contraction will also flex the knee and supinate the subtalar joint. During normal ambulation, subtalar joint pronation imparts an internal rotation force to the tibia, whereas passive knee extension imparts an external rotation force through the tibia. These opposing

rotational movements will translate into unusually high stress levels within the tendon. These forces are related to body weight and activity level. During running, for example, forces up to ten times body weight have been measured in the Achilles tendon.

Classification of Achilles Tendon Problems

In recent years, a standardized consensus terminology has emerged for classifying tendon inflammation and degeneration[6,7] (Table 1). This histopathologic scheme facilitates comparison of results of therapeutic interventions from different centers. The three stages of tendon injury are paratenonitis, paratenonitis with tendinosis, and tendinosis.

Paratenonitis is inflammation limited to the paratenon. Macroscopically, the paratenon is thickened and typically adherent to normal tendon tissue. Histologic findings include capillary proliferation and inflammatory infiltration confined to paratenal tissue. Paratenonitis with tendinosis combines elements of paratenonitis with focal intratendinous degenerative changes. Areas of tendinosis appear thickened and yellowish and have lost the normal luster and linear striations associated with healthy tendon tissue. Under the microscope, these areas have a noninflammatory histologic appearance, with collagen fiber disorientation, scattered vascular ingrowth, hypocellularity, and occasional areas of necrosis or calcification. These areas typically occur 2 to 6 cm proximal to the calcaneus.

Pathologic studies of partially and completely ruptured tendons have consistently revealed the characteristic changes of tendinosis. To some extent, these findings are related to age. As age increases, morphologic changes in the Achilles tendon include a decrease in the number of organelles within tenocytes, a diminution in the levels of mucopolysaccharides and glycoproteins, and a decrease in the maximum diameter and density of collagen fibrils. A large body of evidence from pathologic studies implicates reduced intratendinous vascularity as a primary cause of focal tenocyte destruction. In theory, reduced vascularity decreases the potential for mechanically induced collagen formation, resulting in less tensile strands and eventually a downward spiral of degeneration and rupture. However, as mentioned previously, this theory has recently been called into question because of studies using laser Doppler flowmetry to measure intratendinous blood flow within normal and diseased tendons. In a case-control study of patients with Achilles tendinopathy, Åström and Westlin[3] reported increased flow at rest within diseased tendons. Further studies involving the use of other technologies with better spatial resolution will be needed to confirm these provocative findings.

Table 1
Classification of Tendon Inflammation and Degeneration[7]

Stage	Definition	Histologic Findings	Clinical Signs and Symptoms
Paratenonitis	Inflammation of only the paratenon, either lined by synovium or not	Inflammatory cells in paratenon or peritendinous areolar tissue, local tenderness, warmth	Cardinal inflammatory signs: swelling, pain, crepitation, local tenderness, warmth, dysfunction
Paratenonitis with tendinosis	Paratenon inflammation associated with intratendinous degeneration	Same as for paratenonitis, with loss of tendon collagen, fiber disorientation, scattered vascular ingrowth, but no prominent intratendinous inflammation	Same as for paratenonitis, with palpable tendon nodule, swelling, and inflammatory signs
Tendinosis	Intratendinous degeneration due to atrophy (e.g., aging, microtrauma, vascular compromise)	Noninflammatory intratendinous collagen degeneration with fiber disorientation, hypocellularity, scattered vascular ingrowth, occasional local necrosis, or calcification	Often palpable tendon nodule that is asymptomatic; swelling of tendon sheath is absent

Diagnostic Techniques

Most Achilles tendon problems can be diagnosed simply on the basis of a thorough history and physical examination. Sophisticated imaging modalities generally are not necessary. The physical examination of a patient with an Achilles tendon problem should be conducted with the patient prone with the feet hanging off the edge of the examining table. The entire substance of the gastrocnemius-soleus myotendinous complex should be palpated while the ankle is gently put through active and passive ranges of motion. Calf atrophy, a common finding with chronic Achilles disease, can be recognized by comparing maximal girth measurements on the involved and noninvolved sides. Tenderness, crepitation, warmth, swelling, nodularity, and substance defects should be noted. The resting position of the forefoot with the ankle and talonavicular joints held in neutral position should also be noted. Forefoot varus (medial border of the foot elevated with respect to the lateral border) has been associated with the occurrence of paratenonitis in athletes, but can be readily treated with accommodative orthotics. Ankle and subtalar mobility are often reduced in patients with overuse injuries of the Achilles tendon.

With paratenonitis, the patient typically first complains of a well-localized tenderness and burning pain after engaging in strenuous sporting activities. Later, symptoms start when exercise commences. As the condition becomes more chronic, the local tenderness increases, and the pain is provoked by less intense activity. On examination, patients have diffuse tenderness, swelling, and warmth. Acute cases sometimes present with crepitation. Partial rupture may be superimposed on chronic paratenonitis and/or tendinosis and can present as an acute episode of focal pain and swelling. In this circumstance, the area of tenderness will be well localized and reproducible by side-to-side squeezing of the involved region.

Tendinosis is frequently painless. Often the only sign is the development of an asymptomatic but palpable tendon nodule. In some cases there will be a gradual thickening of the entire tendon substance. Patients who have activity-related pain and diffuse swelling of the tendon sheath with tendon nodularity usually have paratenonitis with tendinosis. The intratendinous lesion can become a partial rupture, which can cause marked pain in an area of previous tendinosis.

With either a partial or a complete rupture, patients typically experience a sharp pain, often described as feeling like being kicked in the leg. On occasion, the orthopaedist will encounter a patient who gives no history of an acute episode but clearly has sustained a tendon rupture. With a partial rupture, the physical examination will reveal a localized, tender area of swelling that occasionally involves an area of nodularity. With a complete rupture, the examination will typically reveal a palpable depression in the tendon. The Thompson test is positive (i.e., squeezing the calf does not cause active plantar flexion), and the patient is usually unable to perform a single heel raise.

In some cases, an accurate diagnosis of a complete rupture is difficult to establish on the basis of the findings from the physical examination alone. The tendon defect can be disguised by a large hematoma. Plantar-flexion power of the extrinsic foot flexors is retained, and the Thompson test can be false-positive if the accessory ankle flexors (posterior tibialis, flexor digitorum longus, and flexor hallucis longus muscles) are squeezed together with the contents of the superficial posterior leg compartment.

Delayed or missed diagnosis of Achilles tendon ruptures by primary treating physicians is a relatively common occurrence. In a study by Inglis and Sculco,[8] 38 (23%) of 167 Achilles tendon ruptures were initially misdiagnosed by the primary treating physician. When the distinction between partial and complete ruptures is unclear on clinical grounds, and that distinction will have an impact on the choice of treatment, further imaging studies are indicated.

Imaging

The two modalities that can best image the Achilles tendon are sonography and magnetic resonance (MR) imaging. Recent refinements in both technologies have tremendously improved our ability to image pathologic changes in tendons. Each technique has its inherent advantages and disadvantages.

Sonography is relatively inexpensive, is fast and repeatable, and has the potential for dynamic examination. It does, however, require substantial experience to learn how to operate the probe and interpret the images correctly. It is most reliable in determining the thickness of the Achilles tendon and the size of a gap after a complete rupture.

In contrast to sonography, MR imaging is relatively expensive and is typically not used for dynamic assessment. It is superior in the detection of incomplete tendon ruptures and the evaluation of various stages of chronic degenerative changes (Fig. 2). It can also be used

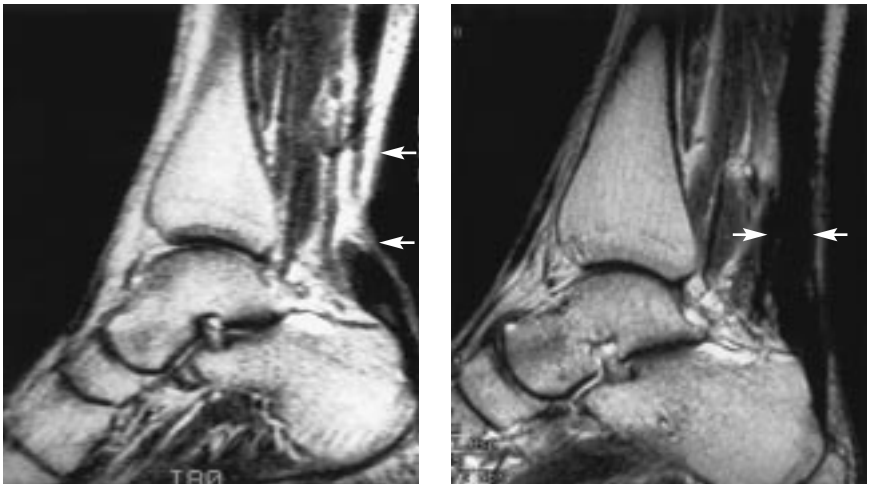

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Fig. 2 T2-weighted sagittal MR images of a chronic Achilles tendon tear. **A,** Image obtained before V-Y repair. Note retracted tendon ends (arrows). **B,** Image obtained 9 months after V-Y repair. The tendon is thickened (arrows) and has homogeneous low signal intensity throughout.

to monitor tendon healing when recurrent partial rupture is suspected (Fig. 3).

Most orthopaedic surgeons have access to adequate MR imaging facilities. As more experience is gained with the use of sonography, many orthopaedists will have a choice regarding imaging of a suspected Achilles tendon lesion. The recommended protocol is to first evaluate the tendon with sonography because of its inherent ease of use, potential for a dynamic examination, and lower cost; if the ultrasound findings are equivocal, an MR study can then be performed[9] (Fig. 4).

Treatment

Paratenonitis

Acute inflammatory conditions of the paratenon surrounding the Achilles tendon usually respond to simple conservative measures. Rest should always be a part of the initial treatment. The duration of rest is determined by the severity and duration of pain. Ice massage helps relieve acute pain and inflammation. Nonsteroidal anti-inflammatory medication may also ameliorate the acute symptoms. A small heel lift or a custom shock-absorbing orthotic may further reduce acute symptoms.

Most patients who present for treatment have chronic unremitting pain. An initial period of complete rest followed by a gradual and structured return to activities is often required.[10] A close examination of recent training conditions should be performed to identify training errors or schedule changes that may have contributed to the onset of symptoms. Many patients with paratenonitis have a tight triceps surae and some degree of calf weakness.

Heel cord tightness is treated with stretching exercises and use of a 5-degree dorsiflexion ankle-foot orthosis worn while sleeping for 3 months. Most athletes, especially runners, benefit from developing a staged cross-training program that first involves aqua-jogging and swimming, then stationary cycling, and, eventually, exercise on stair-climbing and cross-country skiing machines. The use of a custom orthosis that absorbs the shock of heel strike and controls excessive pronation may have long-term benefits for selected patients.

Corticosteroid injections around the tendon have been advocated in

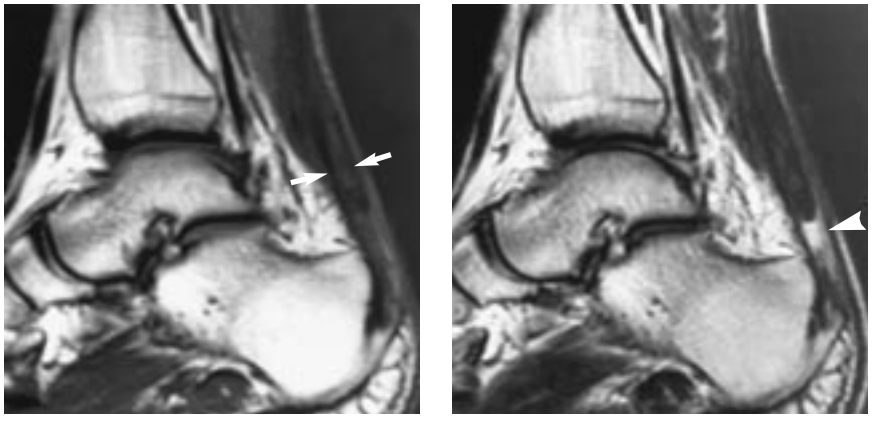

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Fig. 3 T2-weighted sagittal MR images of the Achilles tendon. **A,** This image shows increased signal intensity (arrows) within the substance of the thickened tendon, consistent with tendinosis. **B,** Image obtained after the acute onset of pain shows a partial rupture (arrowhead).

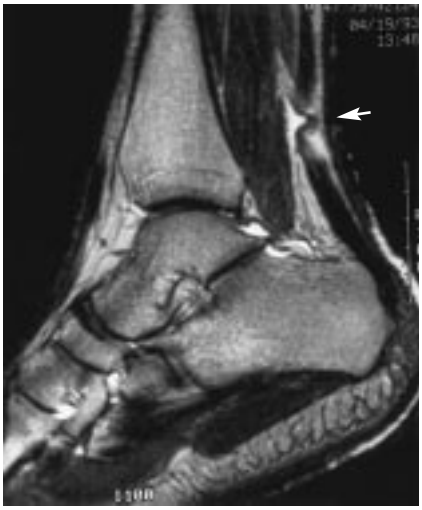

Fig. 4 T2-weighted sagittal MR image of an acute tear (arrow).

recalcitrant cases, in order to inhibit inflammation and scar formation. However, steroid injections carry the risk of adverse effects on the mechanical properties of the tendon if injected into the tendon or if used repeatedly. Therefore, steroid injections in the area of the tendon are not recommended because of the lack of proven efficacy and concerns about the deleterious effects on tendon integrity.

Brisement can be helpful in treating paratenonitis. With this technique, a dilute local anesthetic is slowly injected into the paratenon sheath to break up adhesions. This may be performed with ultrasound guidance to ensure proper placement of the needle.

Surgical treatment is considered for chronic cases resistant to an exhaustive conservative program. Through a medial longitudinal incision (Fig. 5, A), full-thickness flaps of skin, subcutaneous tissue, and crural fascia are developed. Thickened paratenon is excised posteriorly, medially, and laterally where thickened[11] (Fig. 5, B). The blood supply of the tendon within the anterior mesotenon is carefully avoided. The crural fascia is closed, to decrease subcutaneous scarring of the tendon.

Postoperatively, motion is initiated immediately. Swimming and aqua-jogging can be started when it is comfortable for the patient and the wound is sealed. Weight bearing is permitted when pain and swelling allow, usually in 7 to 10 days. The patient is instructed to walk as tolerated for 2 to 3 weeks. During this time, a progressive-resistance strengthening program involving the use of bands or tubing is initiated. When the patient can walk without pain, the rehabilitation program is expanded to include use of a stationary cycle and a stair climber. Running is gradually introduced 6 to 10 weeks postoperatively. A return to competition may take 3 to 6 months.

Tendinosis

While degeneration within the substance of the Achilles tendon is typically not symptomatic, patients may have tendinosis in conjunction with paratenonitis, which produces activity-related pain and swelling. Acute onset of pain with a thickened tendon nodule is consistent with a partial tendon rupture.

The treatment of symptomatic tendinosis is initially conservative. Should symptoms be resistant to the program described for chronic paratenonitis, surgery is recommended. The surgical technique consists of an initial evaluation of the paratenon. If the paratenon is hypertrophic and adherent to the tendon, it is excised. More typically, the sheath is split with fine scissors. A longitudinal incision is created within the body of the tendon over the thick or nodular regions. Degenerative areas are excised, and the defects are repaired[12] (Fig. 6). After debridement of the tendon, it is repaired side to side with absorbable suture.

Postoperatively, a period of protection in a removable walking boot with an adjustable heel and rocker sole (Fig. 7) is usually required. The patient is allowed to bear weight fully and typically wears the boot for 2 to 4 weeks, depending on the extent of debridement. Range-of-motion exercises are performed several times a

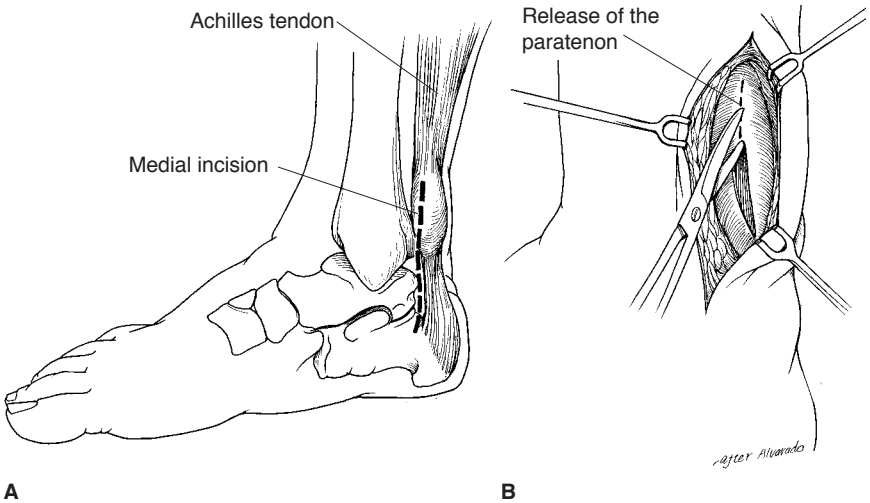

Fig. 5 **A,** The medial longitudinal incision minimizes risk to the sural nerve and short saphenous venous system. **B,** After creation of full-thickness flaps, the paratenon is released, and any thickened areas are excised.

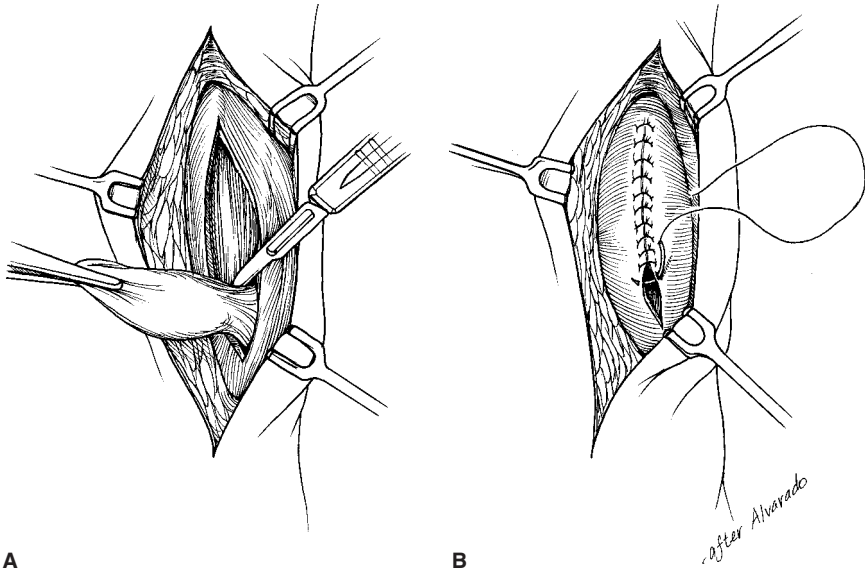

Fig. 6 Treatment of tendinosis. **A,** Diseased areas are excised through a longitudinal incision in the tendon. **B,** The paratenon is repaired to prevent subcutaneous scar formation.

day. For the athlete, a gradual return to sport is permitted after completion of a thorough strength rehabilitation program, as described for chronic paratenonitis.

Acute Rupture

The goals of treatment of a ruptured Achilles tendon are to restore length and tension and thereby to optimize ultimate strength and function. There continues to be controversy as to whether operative or nonoperative treatment best achieves these goals. Proponents of surgical repair point to lower rerupture rates (0% to 2% vs 8% to 39%) and improved strength, with a high percentage of patients returning to sport.[13] Those favoring nonoperative treatment stress the higher surgical complication rate due to wound infections, skin necrosis, and nerve injury. With careful operative technique, these complications can be minimized. When major complications, including reruptures, are compared, both forms of treatment have similar complication rates.

Nonoperative treatment begins with an initial period of immobilization. Ultrasonography can be used to confirm that tendon apposition occurs with 20 degrees or less of plantar flexion of the ankle (Fig. 8). Should a diastasis remain with 20 degrees of plantar flexion, operative treatment is indicated. Initially, the leg is immobilized in a splint for 2 weeks to allow hematoma consolidation. Immobilization can then be maintained in a short leg cast or a removable boot with an elevated heel. An open-back walking boot can facilitate sonographic monitoring during the course of treatment.

Typically, the short leg cast or boot is worn for 6 to 8 weeks, after which the patient is weaned from its use, and gentle range-of-motion exercises are begun. A heel lift is used in the transition to wearing normal shoes. Initially, a 2-cm lift is used. The heel height is decreased by 1 cm after 1 month and is removed after 2 months. Progressive-resistance exercises for the calf muscles are started at 8 to 10 weeks, with a return to running at 4 to 6 months. Patients should be informed that attainment of maximal plantar-flexion power may take 12 months or more and that some residual weakness is common.

Surgical treatment is often preferred when treating younger and more athletic patients and those in whom adequate tendon apposition is not obtained through closed means. The surgical technique utilizes a medial approach to expose the tendon ends. The stumps are approximated with two to four slow-absorbing sutures in a modified Bunnell technique (Fig. 9).

The recent literature has suggested that early gradual return to function after surgical repair is effective and may not increase the rate of rerupture.[14,15] For the elite athlete, range-of-motion exercises can be started as early as 3 to 7 days after surgery. These consist of passive plantar flexion and active

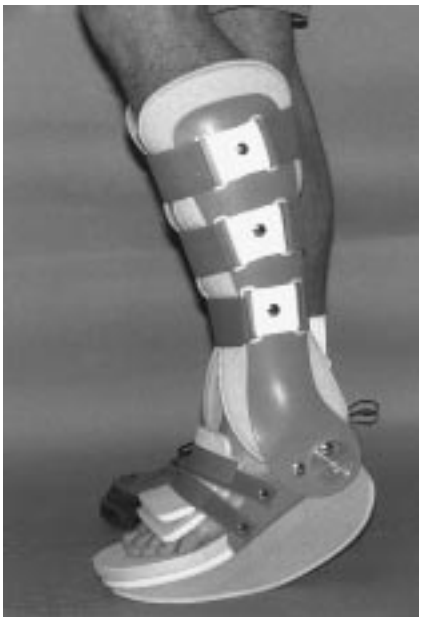

Fig. 7 The "Rock Boot" (Röck Orthopädie, Schopfloch, Germany) has an interchangeable elevated heel rocker and an open posterior aspect.

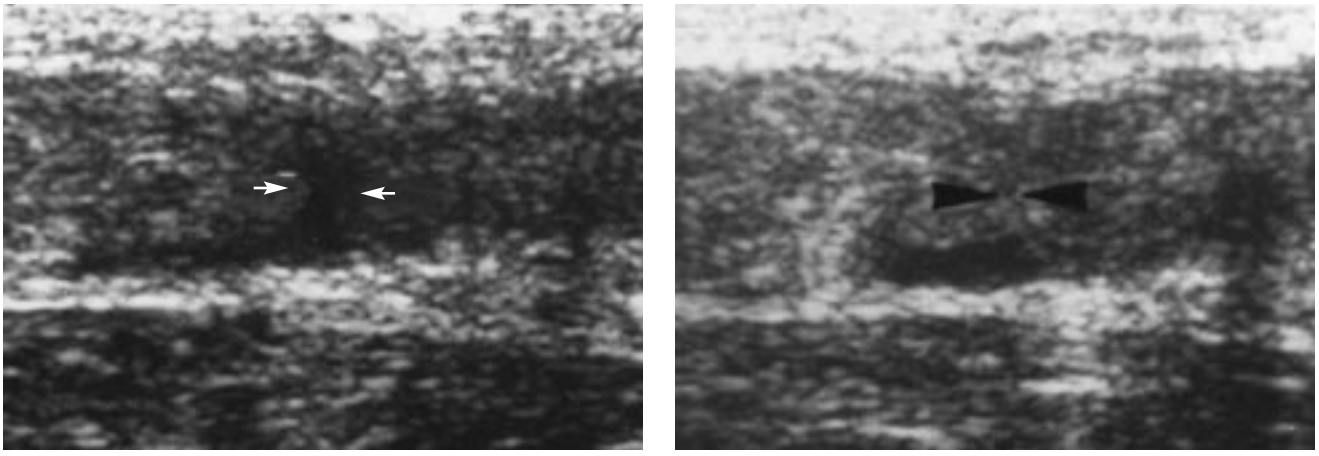

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Fig. 8 Tendon apposition may be confirmed with ultrasonography. **A,** Diastasis (arrows) is present with the foot in neutral position. **B,** Tendon ends are apposed with 20 degrees of plantar flexion (arrowheads). (Courtesy of Hajo Thermann, MD, Hannover, Germany.)

dorsiflexion limited to 20 degrees. A walking boot should be used for 6 weeks, with progression to sport on a schedule similar to that followed after nonoperative treatment. For the less demanding athlete and for the general population, use of a short leg cast for 6 to 8 weeks is preferred, followed by use of a 1-cm heel lift for 1 month. As with nonoperatively treated patients, progressive-resistance exercises are started at 8 to 10 weeks, with a return to running at 4 to 6 months.

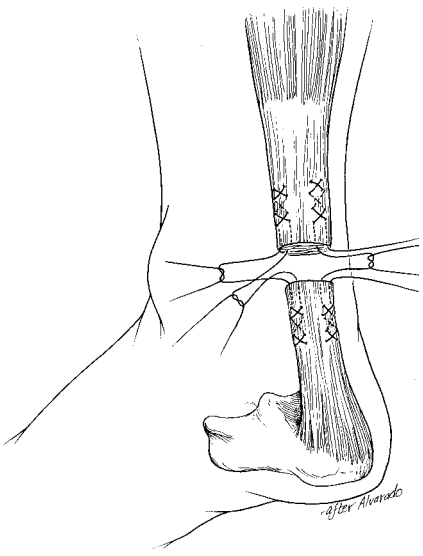

Fig. 9 Four-strand suture technique for repair of acute ruptures.

Outcomes after surgical treatment consistently show a slight advantage in isokinetic strength and a return to preinjury activity levels compared with nonoperative treatment. Clearly, both techniques provide satisfactory outcomes.

Chronic Rupture

Treatment delay after complete rupture of the Achilles tendon can result in substantial plantar-flexion weakness. When there is a significant gap, a good result can be obtained only by surgically approximating the musculotendinous unit near its normal resting length. The choice of surgical strategy depends somewhat on the level of rupture and the amount of stump separation.

The incision for treatment of chronic ruptures is extended proximally to identify the retracted tendon stump. After debriding scar and freshening tendon ends, a 1- to 2-cm-wide window is created in the fascia over the flexor hallucis longus muscle to allow the potential for improved vessel ingrowth to the repaired area. Defects less than 3 cm may be repaired with a turned-down flap.[16] For gaps up to 8 cm, a V-Y lengthening of the triceps surae may be required (Figs. 2, 10).

Treatment of difficult neglected tendon injuries and insertional avulsion is particularly challenging. For these problems, reconstructions with use of the flexor digitorum longus[17] or the flexor hallucis longus[18] have been reported to provide satisfactory results.

Summary

Overuse injuries to the Achilles tendon are frequently encountered in orthopaedic practice, especially as the interest in athletic activities increases. Conservative management is successful in most cases of acute paratenonitis and often ame-

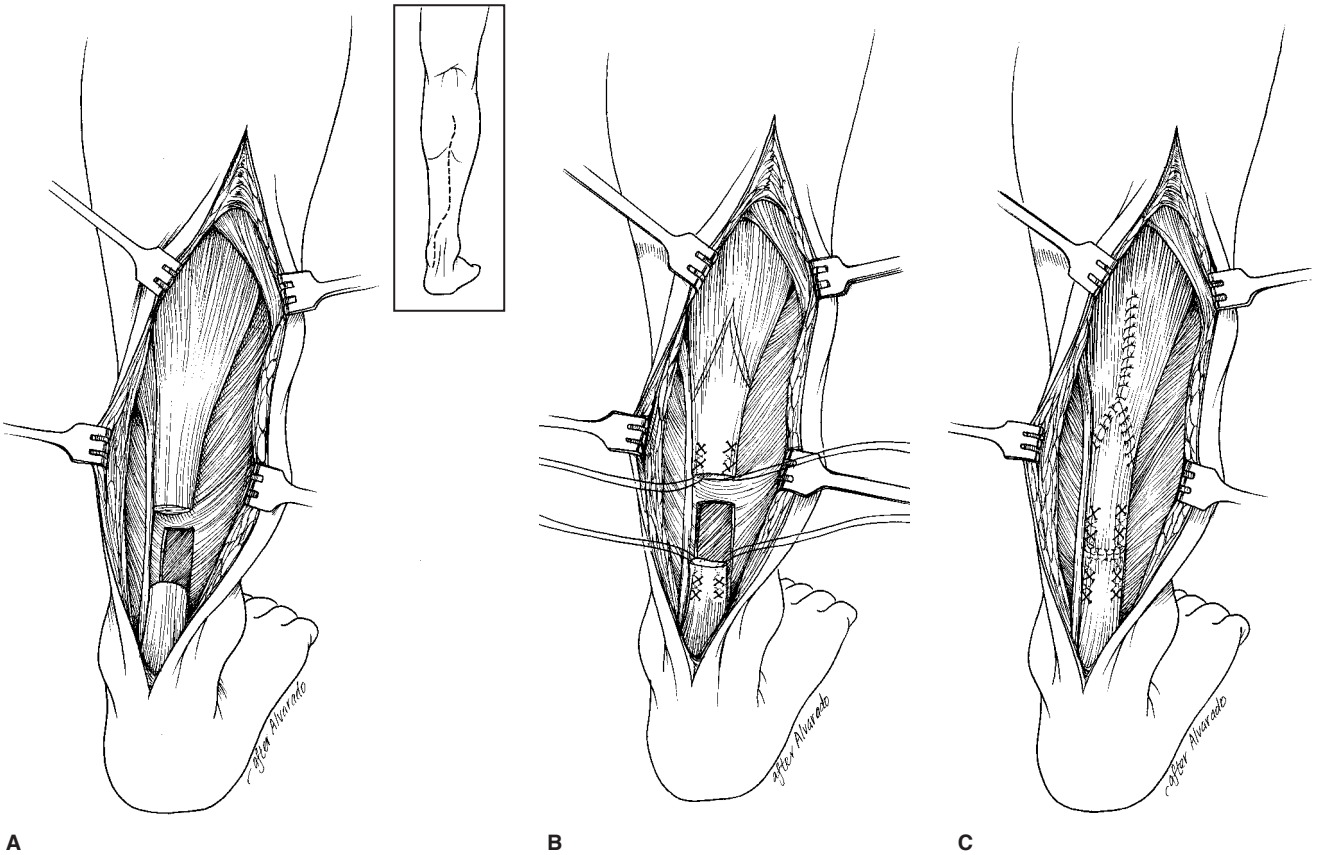

Fig. 10 Technique for V-Y lengthening of the triceps surae. **A,** A medial incision is extended proximally in a gently curving S (inset). The tendon ends are debrided, and the repair site is prepared by windowing the deep posterior fascia. **B,** A V cut is made in the triceps surae aponeurosis. **C,** After approximation of the tendon ends, the aponeurosis is closed.

liorates symptoms when paratenonitis accompanies tendinosis. Acute ruptures of the Achilles tendon can be treated by either nonoperative or operative means. Decreased rerupture rates and slightly improved strength and functional ability may be expected with surgical treatment; however, the rate of minor complications is higher than with nonoperative treatment.

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