Surgical Treatment for Chronic Disease and Disorders of the Achilles Tendon

Sudheer S. Reddy, MD

David I. Pedowitz, MD, MS

Selene G. Parekh, MD, MBA

Imran M. Omar, MD

Keith L. Wapner, MD

Dr. Reddy is Sports Medicine Fellow, Department of Orthopaedic Surgery, University of California–San Francisco, San Francisco, CA. Dr. Pedowitz is Attending Physician, Department of Orthopaedic Surgery, Crystal Run Healthcare, Middletown, NY. Dr. Parekh is Assistant Professor, Foot and Ankle Surgery, Department of Orthopaedic Surgery, University of North Carolina School of Medicine, Chapel Hill, NC. Dr. Omar is Fellow, Department of Radiology, Thomas Jefferson University Hospital, Philadelphia, PA. Dr. Wapner is Clinical Professor, Department of Orthopaedic Surgery, University of Pennsylvania School of Medicine, Philadelphia.

Reprint requests: Dr. Parekh, Department of Orthopaedic Surgery, University of North Carolina School of Medicine, 3135 Bioinformatics Bldg, CB#7055, Chapel Hill, NC 27599.

Abstract

Chronic Achilles tendon disorders range from overuse syndromes to frank ruptures. Numerous forms of treatment have been used, depending on the nature of the disorder or injury. Ultrasonography and magnetic resonance imaging are commonly used for evaluation. The spectrum of disease comprises paratenonitis, tendinosis, paratenonitis with tendinosis, retrocalcaneal bursitis, insertional tendinosis, and chronic rupture. However, there is no clear consensus on what defines a chronic Achilles disorder. Nonsurgical therapy is the mainstay of treatment for most patients with overuse syndromes. Surgical techniques for overuse syndromes or chronic rupture include débridement, local tissue transfer, augmentation, and synthetic grafts. Local tissue transfer most commonly employs either the flexor hallucis longus or flexor digitorum longus tendon to treat a chronic rupture. Reports on long-term outcomes are needed before useful generalizations can be made regarding treatment.

Chronic Achilles tendon dysfunction and disease are challenging to manage. Numerous treatment options are available, with selection based on the nature of the disorder. At one end of the spectrum is a variety of overuse syndromes that continue to be further defined; at the other end is frank rupture of the tendon in the setting of chronic tendon pathology and chronic rupture. The confusing litany of terms used to describe chronic Achilles tendon disorders highlights the lack of consensus regarding which classification system best illustrates what is seen clinically and microscopically. For instance, what was once termed tendinitis is now known to be a condition in which inflammatory cells are not involved.[1] The term "chronic" has been used differently in various settings, leading to difficulty in interpreting results.[2] Further complicating any proposed treatment algorithm is that the assorted Achilles tendon pathology can and often does present in two distinct patient populations: the younger athlete and the older community ambulator, neither of whom may be an optimal candidate for any of the available treatment modalities. Surgical treatments are myriad and can involve local tissue transfer, augmentation procedures, and synthetic graft.

Figure 1
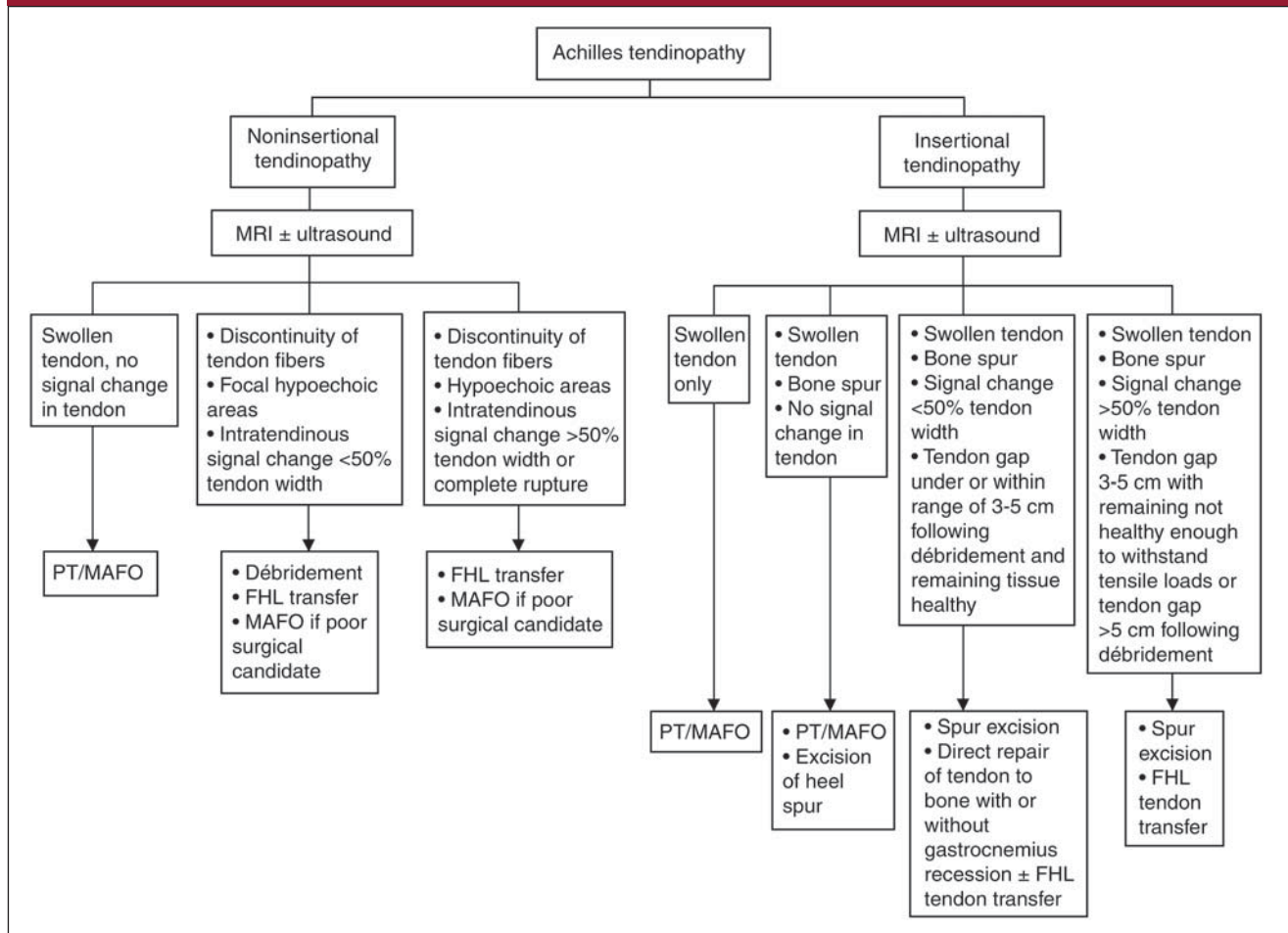
Algorithm for the treatment of Achilles tendinopathy. FHL = flexor hallucis longus tendon, MAFO = molded ankle-foot orthosis, MRI = magnetic resonance imaging, PT = physical therapy

Imaging

Imaging of the Achilles tendon is primarily limited to ultrasonography and magnetic resonance imaging (MRI). Standard radiographic imaging is useful for diagnosis of some conditions. Ultrasonography provides many advantages, including speed, safety, and low cost. It can verify the existence and location of intratendinous lesions. The results of ultrasonography have been found to be reliable, particularly in chronic cases involving adhesions around the tendon. In the acute phase of Achilles tendinopathy, ultrasonography can demonstrate fluid surrounding the tendon.[3,4] In the chronic form, peritendinous adhesions can be seen as a thickening of the hypoechoic paratenon with poorly defined borders.[4,5] Localized tendon swelling and thickening, discontinuity of tendon fibers, and focal hypoechoic intratendinous areas are the most characteristic ultrasonographic findings in the patient with a surgically verified Achilles intratendinous lesion.[4]

Ultrasonography has drawbacks, however. It is operator-dependent and is not as readily accessible as MRI.[4,6] Neither is it as accurate as MRI in delineating isolated paratenonitis unassociated with focal intratendinous disease. With ultrasound, it is difficult to differentiate a partial rupture from a discrete area of tendinosis. In evaluating Achilles tendon

disorders, ultrasound has a sensitivity of 0.80 and specificity of 0.49.[7]

MRI has been used extensively to visualize tendons. It provides extensive information on the internal morphology of the tendon and the surrounding structures. It is useful for evaluation of acute ruptures as well as various stages of chronic degeneration. It can aid in differentiating paratenonitis from tendinosis. As with ultrasound, excellent correlation has been reported between MRI results and intraoperative pathologic findings.[8] MRI has a sensitivity of 0.95 and a specificity of 0.50 in evaluating Achilles tendon pathology.[7,9] Unlike ultrasonography, it is not operator-dependent, and it allows multiplanar imaging.

Standard radiography has also been used in the evaluation of retrocalcaneal bursitis and Haglund deformity (ie, prominence of the posterosuperior angle of the os calcis). The prominence can cause a mechanical irritation of the retrocalcaneal bursa. On a lateral radiograph, the prominence of the posterosuperior calcaneal tuberosity can be measured using parallel pitch lines. This line can be measured by placing a line from the calcaneal tuberosity to the anterior calcaneal tubercle. A second line is drawn parallel to this at the level of the posterior lip of the posterior facet. Bony prominences extending above this line are abnormal.[10] Although a patient with a Haglund deformity has an element of retrocalcaneal bursitis, multiple studies have attempted to delineate Haglund deformity radiographically as related to calcaneal anatomy. However, no particular radiographic view has been consistently helpful in making a diagnosis or in planning treatment.[11]

After an exhaustive search of the literature, we have realized that there is no uniform classification or treatment scheme for Achilles tendon pathology. In regard to surgical planning, MRI is the most useful imaging tool as it allows for evaluation of the Achilles tendon in the sagittal plane

Figure 2
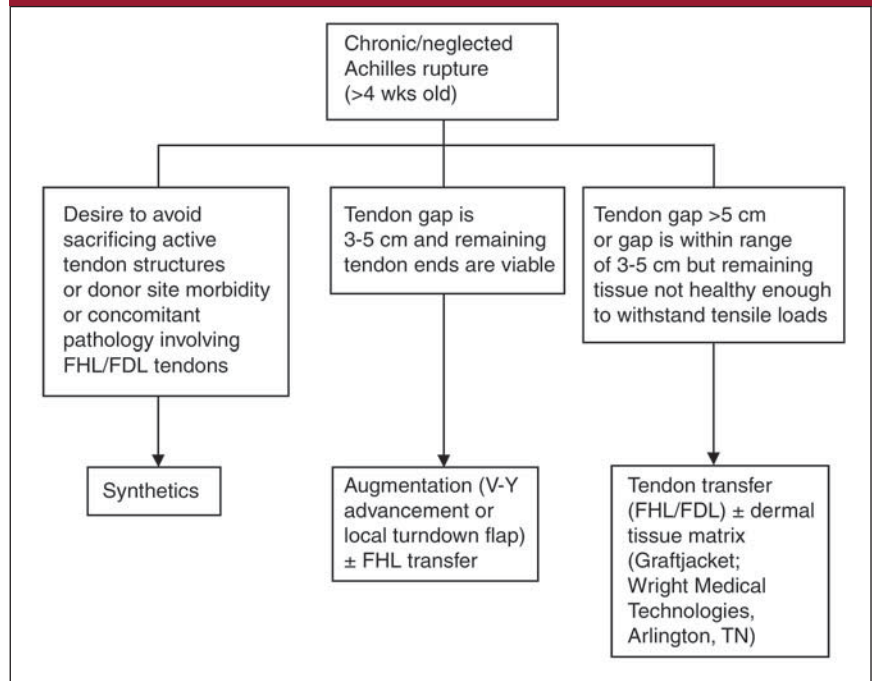
Algorithm for treatment of chronic Achilles tendon rupture. FDL = flexor digitorum longus tendon, FHL = flexor hallucis longus tendon

to determine the length of the injured or diseased tendon and subsequent surgical planning. We present two treatment algorithms based on imaging findings, albeit not on any direct evidence, to guide the surgeon in the treatment of chronic Achilles tendon disorders and disease (Figures 1 and 2).

Classification of Chronic Achilles Tendon Disorders

Clancy et al[12] proposed a scheme that classified Achilles tendinitis as being acute (symptom duration ≤2 weeks), subacute (symptom duration between 3 and 6 weeks), and chronic (symptom duration ≥6 weeks). A more descriptive and frequently used classification scheme based on clinical and anatomic-pathologic findings was created by Puddu et al.[13] Disorders are classified as pure peritendinitis (stage 1), peritendinitis with tendinosis (stage 2), and tendinosis (stage 3).

In this scheme, the peritendinous structures, which include the paratenon and the septum, are supported by loose connective tissue with abundant cellularity that can predispose them to an inflammatory process.[13] With regard to tendinosis, because of diminished vascularity and hypocellularity, the tendon is unlikely to undergo an inflammatory process and has been found histologically to undergo degeneration.[13] Because the tendon itself does not undergo true inflammation, the term tendinitis as it refers to the Achilles tendon is a misnomer. Paratenonitis would be a more descriptive term as it specifically refers to the tissue surrounding the tendon proper. Although Achilles paratenonitis and tendinosis are independent entities, they can coexist. Furthermore, disease in the retrocalcaneal bursa and the Achilles tendon insertion can result in retrocalcaneal bursitis and insertional tendinosis. Excluding rupture, the spectrum of

chronic Achilles tendon disease can be subdivided into five disorders: paratenonitis, tendinosis, paratenonitis with tendinosis, retrocalcaneal bursitis, and insertional tendinosis[11] (Table 1).

Achilles Paratenonitis

Familiarity with the anatomy of the Achilles tendon sheath is necessary to fully understand Achilles paratenonitis (Figure 3). The Achilles tendon is not encased in a true synovial sheath but rather in a single layer of paratenon made up of a single layer of cells. The paratenon is composed of fatty, mesentery-like areolar tissue that is highly vascularized and is responsible for a significant portion of the blood supply to the tendon. Perfusion of the tendon occurs through a series of vincula that serve as thoroughfares for blood vessels to reach the tendon. Most of the blood supply to the tendon is anterior. Angiographic studies have revealed that there is an area of tenuous blood supply approximately 2 to 6 cm proximal to the insertion to the calcaneus.[11]

Although uncommon in the older, more sedentary population, paratenonitis is commonly seen in athletes, especially long- and middle-distance runners.[14] Because of its vascularity, the paratenon is susceptible to inflammation, and the patient with paratenonitis commonly presents with diffuse discomfort and swelling of the tendon. Acutely, the Achilles tendon may appear sausage-like, with fusiform swelling (Figure 4). Tender nodules can often occur within the paratenon, reflecting localized hypertrophy of connective tissue.[11]

External pressure from poor-fitting shoes is thought to cause friction between the Achilles tendon and the overlying paratenon.[11] Addi-

Table 1

Classification of Chronic Achilles Tendon Dysfunction[3]

Disorder	Pathology
Paratenonitis	Inflammation of the peritendinous structures, including the paratenon and septum
Tendinosis	Asymptomatic degeneration of tendon without inflammation, with regional focal loss of tendon structure
Paratenonitis with tendinosis	Inflammation of the peritendinous structures along with intratendinous degeneration
Retrocalcaneal bursitis	Mechanical irritation of the retrocalcaneal bursa
Insertional tendinosis	Inflammatory process within the tendinous insertion of the Achilles tendon

Figure 3

A   B   C
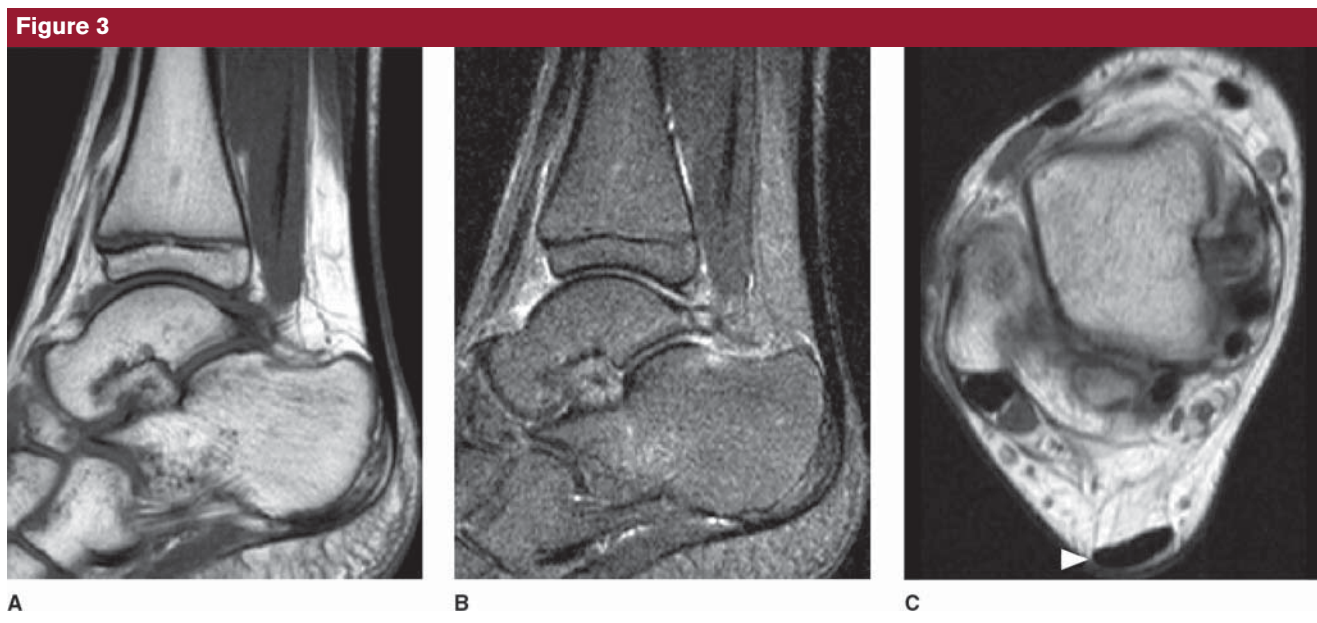
Magnetic resonance imaging (MRI) scans of a normal Achilles tendon in a 12-year-old girl with right ankle inversion. Sagittal T1-weighted non–fat-suppressed **(A)** and T2-weighted fat-suppressed **(B)** scans indicating the tendon is uniform in caliber and of homogeneously low signal, with no paratendinosis or edema in Kager fat. **C,** Axial proton density non–fat-suppressed MRI scan of the mid tendon (arrowhead) demonstrating a homogeneously low tendon signal with a flat or slightly concave anterior tendon margin.

Figure 4
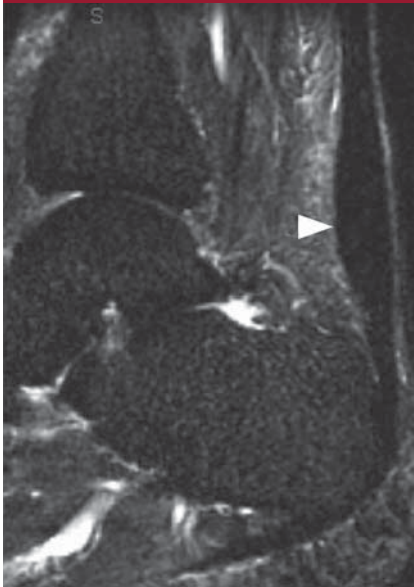
Sagittal T2-weighted fat-suppressed MRI scan demonstrating critical zone Achilles hypoxic tendinosis and fusiform enlargement of the tendon critical zone with uniform low signal (arrowhead) in a 40-year-old woman who presented with right posterior ankle pain.

Figure 5

Sagittal grayscale ultrasound image of a 61-year-old man with left posterior ankle pain demonstrating a tendon with fusiform enlargement (arrow) and surrounding hypoechoic fluid (arrowheads), indicating Achilles paratendinitis.

Figure 6

Axial T2-weighted fat-suppressed MRI scan of a 47-year-old man with right posterior ankle pain. The image demonstrates a convex anterior margin of the Achilles tendon, seen in critical zone tendinosis, with a circumferential rim of hyperintense signal (arrowhead) depicting paratendinitis.

tionally, fluid may accumulate adjacent to the tendon, and adhesions may develop. Macroscopically, the tendon is thickened and adherent to the normal surrounding tissue. In a patient with isolated paratenonitis, a histologic study will show capillary proliferation and inflammatory infiltrate confined to the paratendinous tissue.[15] Kvist et al[14] found that the blood vessels within the paratenon were often obliterated and degenerated, with fibronectin and fibrinogen commonly found in the proliferating connective tissue areas and in the vascular walls. It is thought that myofibroblasts in the peritendinous tissue synthesize abundant amounts of collagen in response to mechanical stress, resulting in scarring and shrinkage of the peritendinous tissue. This contracted state can also lead to vascular constriction, further impeding the circulation to the Achilles tendon.[3]

Given that Achilles paratenonitis often has a mechanical origin and frequently affects distance runners, a detailed history tailored to the activity of the runner should be sought. Pain is the cardinal symptom of Achilles tendinopathy.[3] In the early phase of paratenonitis, the patient may complain primarily of pain following strenuous exercise; if the condition is progressive, pain may accompany routine activities. For athletes, the pain may be disabling enough to curtail training regimens.[16]

Physical examination findings vary, depending on the degree of inflammation present within the paratenon. Decreased ankle dorsiflexion, often due to tightness of the gastrocnemius-soleus-Achilles tendon complex, and hamstring tightness are commonly found in patients with Achilles tendon pathology.[16,17] The Silfverskiold test can be used to measure tightness of the gastrocnemius-soleus complex by alternately relaxing and incorporating the muscle by flexing and extending the knee, respectively, while dorsiflexing the ankle.[10] A patient with paratenonitis will exhibit tenderness and thickness that remain fixed with active range of motion of the ankle.
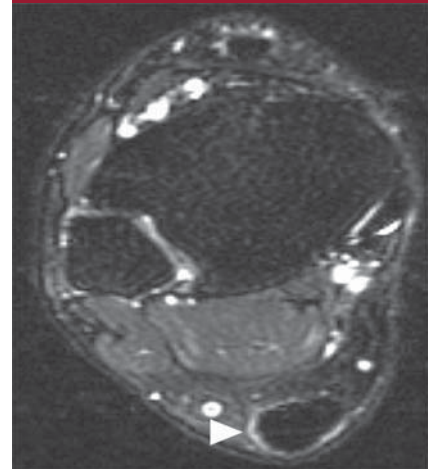
There is often palpable tenderness on both sides of the tendon, with the medial side being more tender than the lateral.[11] Imaging of paratenonitis primarily involves two modalities: ultrasonography and MRI.[15] Ultrasound frequently reveals fluid surrounding the tendon acutely and chronically and can reveal adhesions that can be visualized as thickening of the hypoechoic paratenon (Figure 5). T1-weighted MRI scans will show a thickened paratenon, with a high signal seen within the paratenon on T2-weighted imaging (halo sign)[18] (Figure 6).

Nonsurgical treatment is initially indicated for Achilles paratenonitis. For the less active, older patient, treatment generally focuses on immobilization with a nonarticulated solid molded ankle/foot orthosis, nonsteroidal anti-inflammatory drugs (NSAIDs), and possibly a short course of physical therapy. For the more active patient, conservative modalities typically consist of modification of training regimens (eg,

staged cross-training regimen), rest, ice, massage, and NSAIDs. A small heel lift or a shock-absorbing orthotic device may also help reduce acute symptoms but may allow the injured tendon to contract. Night splints may be added as needed to prevent this.

In a series of 41 patients, Johnston et al[19] found that 51% improved with an average of 18 weeks of nonsurgical therapy alone. Brisement can also be performed to break up adhesions in paratenonitis, particularly in patients with audible crepitus with ambulation. This procedure involves injecting slowly into the paratenon sheath approximately 5 to 10 mL of a dilute local anesthetic, such as lidocaine, or saline solution under pressure. Ultrasound guidance can be used, if available, to guide proper needle placement. Two or three injections can reduce symptoms approximately half of the time.[10,11,15]

Rarely, surgical treatment is considered for chronic paratenonitis that is resistant to nonsurgical measures. A medial longitudinal incision can be used, with development of full-thickness flaps of skin, subcutaneous tissue, and crural fascia. The anterior aspect of the paratenon is avoided to protect the blood supply to the Achilles tendon. Thickened paratenon can be excised posteriorly, medially, and laterally around the tendon.[15] For cases of paratenonitis only, postoperative therapy should focus on immediate range-of-motion exercises to prevent scarring and recurrence of adhesions. For the first 3 weeks, weight bearing is restricted in a removable boot to limit swelling and aid wound healing. Schepsis and colleagues[8,11] reported a satisfaction rate of 87% for surgical treatment of recalcitrant paratenonitis. Endoscopic methods, consisting of a proximal portal for visualization and a distal portal for instrumentation, have also been used to release, in a retrograde longitudinal fashion, and débride the constricting paratenon, by means of a shaver. Patients were allowed to

begin range-of-motion exercises and were progressed to full weight bearing by 3 to 6 days. Satisfactory subjective results were reported by Maquirriain et al[20] in using this technique for paratenonitis.
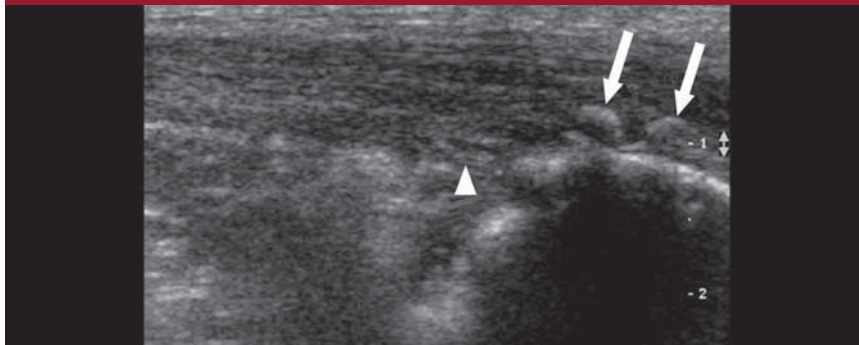
Achilles Tendinosis

Given that tendinosis is a degenerative process, the Achilles tendon with tendinosis on gross examination may appear thick, soft, and yellowish, owing to an accumulation of mucinoid material within the tendon.[13] The condition is often defined as an asymptomatic degeneration of the tendon without inflammation, caused by accumulated microtrauma, aging, or both. Because of its asymptomatic nature, tendinosis is often detected after frank rupture of the tendon.[1] Such frank ruptures are often preceded by partial ruptures, typically in middle-aged men who have suddenly increased their level of physical activity. The paratenon is rarely involved. Central to this pathologic process is the poor healing response following repetitive microtrauma.[3]

Although the pathogenesis of tendinosis is unknown, several theories have been proposed, including hypoxic degeneration, the effect of free radicals, and exercise-induced hyperthermia; however, all of these theories lack direct scientific evidence. Pathologic changes within the tendon matrix also occur, including calcification and accumulation of adipose tissue (ie, tendolipomatosis).[3] Systemic factors such as hypertension and hormone replacement therapy have been linked to tendinopathy in women, while obesity is an etiologic factor in both men and women because of diminution in local microvascularity.[21] Perhaps most important in determining the cause of this condition is defining the role that biomechanics plays in the disorder. Specifically, increased foot pronation has been proposed to be associated with Achilles tendinopathy and to result in tendinosis.[3] Prona-

tion of the foot generates a resultant internal rotation of the tibia that tends to draw the Achilles tendon medially. When the foot remains excessively pronated as the knee extends during the stance phase of gait, there will be a resultant "whipping" action of the Achilles tendon with repetitive walking or running that can lead to microtears in the medial aspect of the tendon, which can precipitate tendinosis.[9]

In their series of 109 runners treated conservatively for Achilles tendon overuse injuries, Clement et al[9] reported that most affected runners presented with a gradual evolution of symptoms, including pain and swelling approximately 2 to 3 cm proximal to the calcaneal insertion. A feature distinguishing tendinosis from paratenonitis is the mobility of the intratendinous nodule or thickening with the point of maximal tenderness during active range of motion (ie, painful arc sign). On ultrasonography, tendinosis can appear as a hypoechoic lesion with or without intratendinous calcification (Figure 7). MRI would also reveal tendon abnormalities, such as tendon thickening on sagittal imaging and altered signal appearance within the tendon tissue[3] (Figure 8).

As with paratenonitis, and for overuse injuries of the Achilles tendon in general, initial nonsurgical treatment should be directed toward relieving symptoms, correcting training errors in athletes, modifying limb malalignment with orthoses, and improving flexibility.[3] Physical therapy, concentrating on enhancing dorsiflexion (ie, eccentric training), is beneficial, given that most patients with chronic tendinopathy possess limited passive dorsiflexion. To treat the excess pronation often found in patients with Achilles tendinosis, a full-length, flexible or semirigid orthotic device has been found to work well; a shock-absorbing insole may also be helpful.[11] Although brisement can be used in the patient with chronic paratenonitis, it is not

Figure 7

Ultrasonographic image of a 52-year-old woman with right posterior heel pain near the Achilles tendon insertion, demonstrating intratendinous calcifications. The long-axis grayscale image demonstrates abnormal thickening, hypoechoic appearance, and disruption of the normal fibrillar arrangement of the tendon fibers (arrowhead). There are also at least two echogenic foci with posterior shadowing in the distal tendon from calcification (arrows), a pattern that is commonly seen in tendinosis.

Figure 8

Sagittal T2-weighted fat-suppressed MRI scan of a 60-year-old man with left posterior ankle pain demonstrating mucoid degeneration in Achilles tendinosis. The image shows marked fusiform enlargement of the Achilles tendon critical zone with abnormal, high intrasubstance signal, which is lower than the signal intensity of fluid (arrowhead).

used for tendinosis. It is also critical to note that the use of corticosteroids for chronic tendinopathy is contraindicated because of the risk of causing a frank rupture. When a patient fails to respond adequately to traditional nonsurgical treatment with NSAIDs, rest, bracing, and physical therapy within a 3- to 6-month period, attention should be directed toward managing the pathology surgically.[16]

In approximately 25% of cases, nonsurgical therapy is ineffective, and the ineffectiveness has been found to be correlated with patient age, duration of symptoms, and severity of tendinopathic change.[22] Surgical treatment of tendinosis consists of removing the areas of degenerated tendon. The extent of degeneration and the age of the individual can have a profound effect on postoperative outcomes. It has been recommended that if more than 50% to 75% of the tendon is involved, autogenous tendon transfer, such as with the flexor hallucis longus (FHL) or the flexor digitorum longus (FDL), or even allograft reconstruction should be done.[11] Patients older than age 50 years with a greater degree of tendon involvement also had less satisfying results with débridement alone. Den Hartog[23] re-

ported 88% good to excellent results with regard to improved function and pain in a series of 26 patients (mean age, 51.3 years) when using an FHL transfer for chronic Achilles tendinosis.

Achilles Paratenonitis With Tendinosis

Paratenonitis can coexist with tendinosis and is manifested as macroscopic tendon thickening, nodularity, softening, and yellowing of the tendon with fibrillation.[4] Pragmatically, Achilles tendinopathy regarding paratenonitis and tendinosis can be viewed as a spectrum, as originally classified by Puddu et al:[13] stage 1, pure paratenonitis; stage 2, paratenonitis with tendinosis; and stage 3, tendinosis.

Stages 2 and 3 are less likely to respond to nonsurgical treatment.[10] One of the earliest changes that can be seen histologically in tendinosis coexistent with paratenonitis is fragmentation of collagen fibers within the substance of the tendon.[3] Typically, it is the chronic form of paratenonitis that is associated with tendinosis.

With the onset of tendinosis, the physician should have a high index of suspicion for a partial rupture
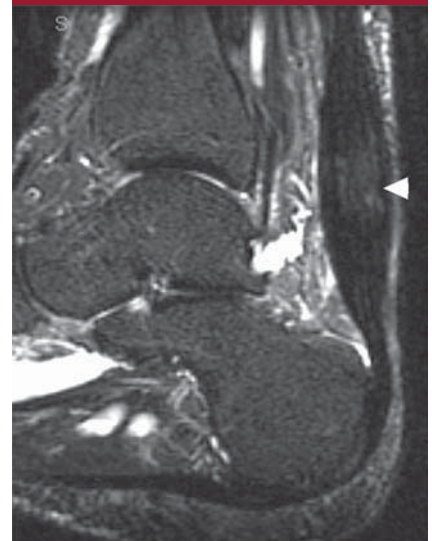
within the tendon. A cardinal sign of this would be a history of transient sharp pain or repeated episodes of sharp pain within the tendon while running.[8] Similar to the presentation of paratenonitis, in the acute phase of coexistent tendinitis, swelling and tenderness are usually found in the middle third of the tendon. As the injury becomes chronic, exercise-induced pain is the cardinal symptom, while crepitus and swelling are diminished. The development of focal, tender nodules heralds the onset of tendinosis in the setting of paratenonitis.[3]

Imaging and treatment must be tailored based on the existence of both tendinosis and paratenonitis. Imaging, in particular, can help identify those patients who are unlikely to respond to nonsurgical treatment. The patient with clinical signs of tendinosis with confluent areas of

intrasubstance signal changes on MRI is unlikely to respond to nonsurgical treatment. Earlier surgical intervention in these patients may lead to earlier return of function.[24] Surgical treatment involves excision of the diseased paratenon along with degenerated tendon. Schepsis and colleagues[8,11] reported a 70% satisfaction rate with débridement provided that <50% of the tendon was involved. When 50% of the tendon is involved, then augmentation consisting of a turndown flap or tendon transfer should be performed. Similar to Den Hartog,[23] Wilcox et al[25] reported 90% good to excellent results when using an FHL transfer for chronic Achilles tendinopathy.

Retrocalcaneal Bursitis

Unlike paratenonitis or tendinosis, retrocalcaneal bursitis is a distinct entity that is characterized by pain anterior to the Achilles tendon and that involves inflammation of the retrocalcaneal bursa. The bursa is roughly horseshoe-shaped and is 4 mm in width from anterior to posterior and 8 mm from medial to lateral. Its anterior surface is composed of fibrocartilage; the posterior aspect of the bursa merges with the paratenon of the anterior Achilles tendon. The bursa can become inflamed, hypertrophied, and adherent to the underlying Achilles tendon; this can lead to degenerative changes within the tendon. This condition is often associated with Haglund's deformity, which causes mechanical irritation of the bursa. Compression of the bursa between the calcaneus and the anterior aspect of the Achilles tendon occurs when the ankle is dorsiflexed. Commonly, the athlete who trains uphill is subject to this condition because of the extreme dorsiflexion of the ankles.[11]

Given the location of the bursa, perhaps the best method for detecting retrocalcaneal bursitis is the two-finger squeeze test, in which the pain response is observed on application of pressure medially and laterally anterior to the Achilles tendon insertion.[8] It is important to distinguish this entity from the condition known as "pump bump." Pump bump is a generic term that refers to any prominence in the subcutaneous Achilles tendon area. It can be attributed to an inflammation of the subcutaneous Achilles tendon bursa caused by an abrasive heel counter or from a bony protrusion.[11]

Routine radiographs can be helpful with the use of parallel pitch lines to measure the posterosuperior calcaneal tuberosity on a lateral radiograph. If changes within the Achilles tendon are present, ultrasound or MRI may demonstrate a partial Achilles tendon tear, peritendinous thickening, tendinosis, or ossification.

NSAIDs and modification of training regimens can also be helpful in the early phase of nonsurgical care. Additionally, abnormal external pressure, such as from a hard athletic shoe heel counter, should be avoided. When these measures fail, then a short period of immobilization in a short leg walking cast may reduce the acute symptoms. Corticosteroid injection is contraindicated because it may lead to tendon rupture. Occasionally a patient may fail to respond to these measures and require surgical treatment. Partial calcaneal ostectomy is a generally successful procedure in this population and can often restore patients to their original level of activity within 6 months.[11] An endoscopic approach has also been used. In a series of 28 patients (30 heels), there were 29 good or excellent results based on the American Orthopaedic Foot and Ankle-Hindfoot Scale, with one Achilles tendon rupture 3 weeks following surgery.[26]

Insertional Tendinosis

Insertional tendinosis is a true inflammatory process within the tendinous insertion of the Achilles. It is often associated with Haglund deformity and, in athletes, is commonly seen in those doing aggressive hill running and interval programs.[11] Like retrocalcaneal bursitis, insertional tendinosis causes posterior heel pain, thought to be due to bony impingement from the calcaneus, local bursitis, or both. Differentiating between the conditions that cause posterior heel pain can be difficult, and these conditions can be viewed as a spectrum of a single disease process. However, a diagnosis of insertional tendinosis requires that the patient have tenderness at the bone-tendon interface. There is also limited dorsiflexion. If the tendinosis becomes chronic, the tendon may also become palpably thickened.[27]

Radiographs can reveal a prominence of the posterior calcaneal tuberosity, possible calcification, or an intratendinous spur. MRI can be used to rule out abnormalities within the tendon and can demonstrate high signal intensity within the retrocalcaneal bursa, which is often best seen on T2-weighted imaging. It can also reveal degenerative or inflammatory changes within the tendon insertion[11] (Figure 9).

Most cases of insertional tendinosis improve with nonsurgical intervention aimed at relieving stress on the tendon insertion (85% to 90%).[27] Initial treatment should be guided toward Achilles tendon stretching, with use of a nonarticulated solid molded ankle-foot orthosis, heel-lift orthoses, and physical therapy. Should nonsurgical measures fail, surgical therapy may be necessary. McGarvey et al[27] reported on the use of a central-splitting approach, beginning with a skin incision 2 cm proximal to the Achilles insertion and extending 6 cm distally. The tendon insertion was then incised in its midline, and any calcific or degenerate regions were dissected free and removed. When excess débridement was required (>50% of the tendon insertion), then the plantaris tendon was used to augment the insertion; only one patient required this in a series of 22

Achilles tendons. Excision of the bursa was also done, followed by a posterosuperior calcaneal ostectomy. These authors reported an 82% satisfaction rate with this surgery.

Wagner et al[28] also evaluated complete detachment and reconstruction of the Achilles tendon attachment for insertional bursitis. In a series of 75 patients treated over 5 years for insertional tendinopathy, 49 patients underwent débridement and complete detachment, followed by reattachment with suture anchors and, for severe involvement, proximal V-Y lengthening. The authors reported satisfactory results in 92% of patients at a mean follow-up of 33 months, with no reruptures. Good to excellent results have also been reported with the use of an FHL transfer for insertional tendinosis.[23]

Chronic Rupture

Although chronic rupture is a distinct entity among the various chronic Achilles disorders discussed here, the methods of treatment, such as augmentation and tendon transfer, are pertinent to the treatment of degenerated, nonfunctional tendons. Gross observation of neglected human Achilles tendon ruptures has not demonstrated great healing potential, a finding similar to that with tendinosis and rupture in the setting of a chronic Achilles tendon disorder.

There is no single rationale for determining what is to be considered a late, chronic, or neglected rupture as authors have modified these terms and used them differently across study groups. Excluding acute rupture, a perusal of the English-language literature on the subject reveals minimum time points used, varying from 4 weeks to 2.5 months.[29] Chronic ruptures typically occur 2 to 6 cm above the calcaneal insertion in the vascular watershed area, with extensive scar tissue deposition between the retracted tendon stumps.[30] Porter et al[29] documented the presence of a highly vascularized collagen scar interposed

between the retracted tendon segments at 4 weeks. To the best of our knowledge, this is the earliest time point used that has demonstrated histologic evidence of a chronic healing process. For that reason, we use 4 weeks as the minimum period to consider a rupture neglected.

Surgical Techniques

In the neglected rupture, restoration of function is dependent on reestablishing appropriate resting length in the gastrocnemius-soleus complex. A variety of techniques has been developed for use, depending on the size of the tendon gap following débridement.[31] If the defect is smaller than 3 cm following débridement and is less than 12 weeks old, then often direct repair can be performed.[29] However, if the tendon gap is larger than 3 cm (more commonly observed), then additional techniques must be used; these techniques include local tissue transfer, tissue augmentation, synthetic biomaterials, and allograft.[30]

Local Tissue Transfer

To reestablish the functional integrity of the Achilles tendon, surgeons have developed a myriad of options for local enhancement of chronic tendon repair. These techniques can provide long, durable tendon grafts, which may augment the strength of the gastrocnemius-soleus contractile unit and supply vascularity to the relatively hypoxic frayed tendon ends. Local tissue transfer is particularly useful to span defects larger than 3 cm when the remaining Achilles tendon tissue is not healthy.[30]

Use of the FHL tendon was reported on by Wapner et al[32] and was described as having multiple advantages over previously recognized tendon transfers. In reporting on seven patients, the authors postulated that use of the FHL provided a long and reliable tendon source that was stronger than the FDL and peroneal

Figure 9
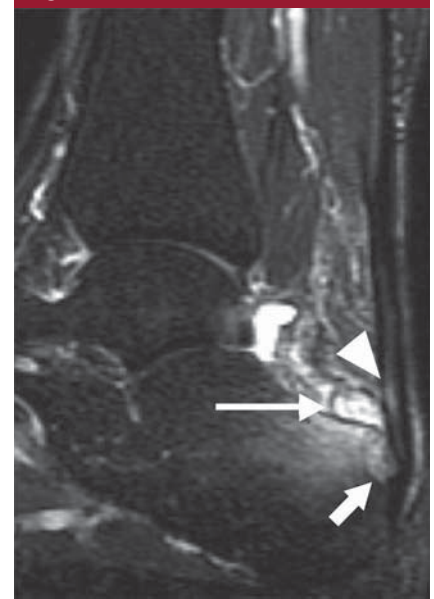
Sagittal T2-weighted MRI scan of a 61-year-old woman with rheumatoid arthritis as well as posterior right ankle pain and swelling, demonstrating retrocalcaneal bursitis. The image reveals fluid distention of the retrocalcaneal bursa resulting from bursitis (thin arrow) and reactive edema in the posterior calcaneus with erosion at the Achilles insertion (thick arrow). There is abnormally high signal in the tendon, likely related to mucoid degeneration (arrowhead).

tendons, contracted along an axis very similar to that of the native Achilles, and fired in phase with the gastrocnemius-soleus complex. The FHL also was closer than the FDL to the Achilles tendon, and it preserved the normal muscle balance of the foot. Wapner's technique involved harvesting the FHL through a medial arch incision (Figure 10). The tendon was then introduced into the posteromedial exposure (Figure 11, A) and passed superior to medial through a drill hole in the calcaneus (Figure 11, B) before being weaved proximally through the Achilles tendon (Figure 11, C and D). Using numerous outcomes measures, Wapner et al[32] reported good or excellent results with this technique in six of
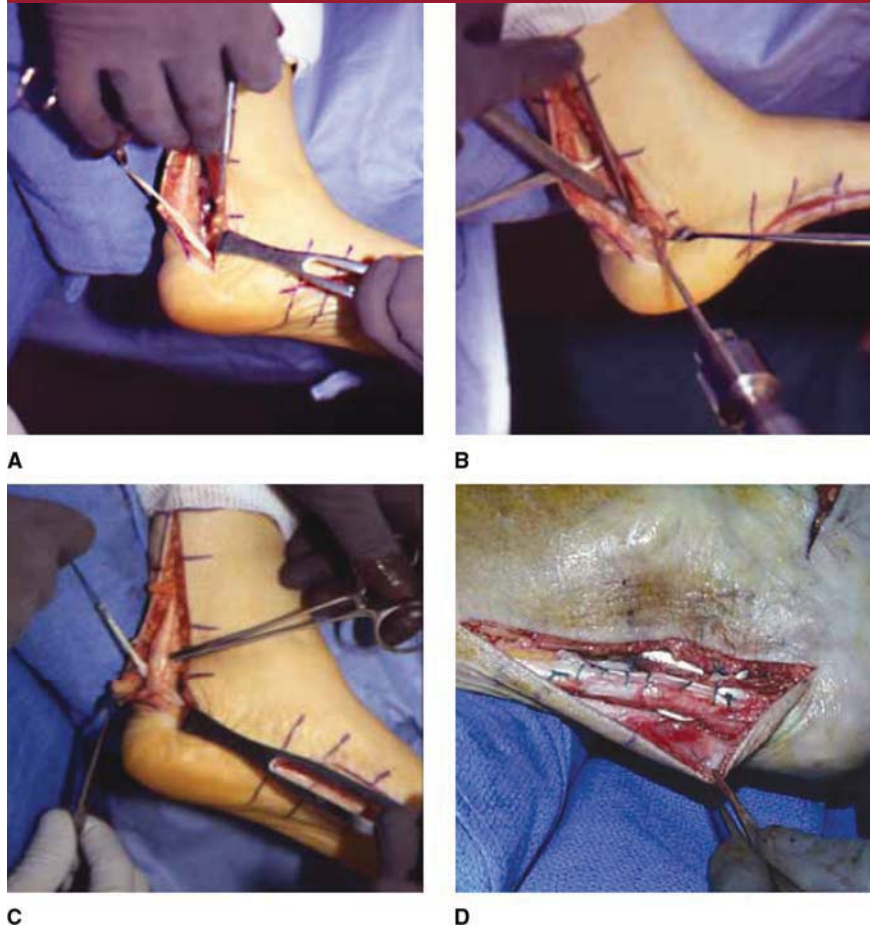
Figure 10

Intraoperative photograph demonstrating the medial arch incision used to release the flexor hallucis longus tendon of the patient in Figure 9.

Figure 11

Intraoperative photographs demonstrating the introduction of the flexor hallucis longus (FHL) tendon into the posteromedial exposure **(A),** preparation of the calcaneal drill hole for the FHL transfer **(B),** and weaving of the FHL tendon proximally through the Achilles tendon **(C)**. **D,** Completion of the FHL weave through the Achilles tendon.

seven cases. Den Hartog[23] used a technique similar to that described by Hansen,[33] in which the FHL is harvested from the posterior incision, and reported no functional deficit from loss of strength at the first interphalangeal joint.

Mann et al[34] used an FDL graft and central slip turndown to reconstruct and span large gaps in the Achilles tendon in seven patients. The FDL was harvested through a medial arch incision, after which the stump was sutured to the FHL tendon. The FDL was then passed medial to lateral through the calcaneus and sewn back on itself under appropriate tension. The authors postulated that this tendon transfer would help reestablish the more medial pull of the normal Achilles and would not create the imbalance thought to exist when a peroneal transfer is used. Although no standardized outcome survey or questionnaire was used, six of seven patients had excellent or good results at an average of 39 months. Two patients required additional soft-tissue procedures for wound complications. All patients retained active flexion of the lesser toes and had no hammer toe deformities. There were no reported reruptures in this series.

The peroneus brevis tendon may be routed lateral to medial through a drill hole in the calcaneus to bridge the gap between the ends of a neglected rupture. Once rerouted, the proximal part of the peroneus brevis is sutured to the proximal stump of the Achilles. The Turco modification involved routing the tendon through the distal stump as opposed to the calcaneus.[35] Excellent results were reported in four reruptures and four neglected ruptures.

Augmentation

The concept of Achilles tendon repair augmentation has expanded over time to include a variety of techniques and donor tissues. Arner and Lindholm[36] developed a technique of medial and lateral aponeurotic fascial turndown flaps. The indications for this method were later expanded by Inglis and Sculco[37] to include chronic ruptures with gaps. These authors used a double weave, creating four strands of augmentation across the gap. Additional augmentation sources include the plantaris, sliding V-Y advancement of the gastrocnemius-soleus complex aponeurosis, and fascia lata.[38,39] V-Y advancement and fascial turndown flaps are particularly useful for gaps of 3 to 5 cm when the remaining Achilles tendon tissue is healthy.[10,30]

Synthetics and Allografts

Various synthetic materials have been employed to address chronic ruptures, including carbon fiber, composite carbon fiber/absorbable polymer, polyester tape, and Marlex mesh (Phillips Sumika, The Woodlands, TX).[2,5] Recently, an acellular human dermal tissue matrix (Graftjacket; Wright Medical Technologies, Arlington, TN) was used as an augmentation material for chronic Achilles ruptures. Lee[40] reported on a series of nine patients with chronic Achilles ruptures treated with primary repair in conjunction with a dermal tissue matrix sewn around the repair. At a mean follow-up of 20 months, there was no rerupture, recurrent pain, or adverse reaction to the graft. However, none of the repair techniques using synthetic or biologic materials have found widespread use, primarily because of concerns regarding the introduction of a foreign body into an area with relatively poor healing capacity. As functional tissue engineering techniques become more refined and produce more reliable outcomes, new interest in the use of these materials is anticipated.

Achilles allografts have also been used to reconstruct large defects (approximately 10 cm) that are not suitable for a tendon transfer. Results are limited to case reports, and long-term clinical follow-up is lacking, but successful use of an allograft to reconstruct the Achilles tendon has been demonstrated in these situations.[41]

Summary

Surgically managing chronic Achilles tendon pathology, which ranges from paratenonitis to the neglected rupture, is neither simple nor straightforward. Whether a combination of turndown fascial flaps, tendon transfers, or the use of synthetic/biomaterials in the construction of a neotendon will yield superior long-term results is unknown. The prefer- ence for any one technique should be based on evidence-based medicine, review of current techniques, surgeon comfort, and the likelihood of a favorable outcome. The long-term follow up for many of these procedures must be studied and reported by comparative randomized studies. Additionally, a randomized prospective study design to compare the use of two or more techniques for repair of chronic Achilles disorders is required before generalizations concerning the efficacy of any specific surgical treatment can be made.

References

Evidence-based Medicine: References 7 and 14 are level II prospective randomized studies. References 1, 4, 8, 18, 19, 21, and 28 are level III case control studies. The remaining references include level IV case series (2, 5, 6, 12, 20, 23-27, 29, 31-36, and 38-41) and level V expert opinion (3, 9, 11, 13, 15-17, 22, and 30).

Citation numbers printed in **bold type** indicate references published within the past 5 years.

1. Aström M, Rausing A: Chronic Achilles tendinopathy: A survey of surgical and histopathologic findings. *Clin Orthop Relat Res* 1995;316:151-164.
2. Jennings AG, Sefton GK: Chronic rupture of tendo Achillis: Long-term results of operative management using polyester tape. *J Bone Joint Surg Br* 2002;84:361-363.
3. Paavola M, Kannus P, Järvinen TA, Khan K, Jósza L, Järvinen M: Achilles tendinopathy. *J Bone Joint Surg Am* 2002;84:2062-2076.
4. Paavola M, Paakkala T, Kannus P, Järvinen M: Ultrasonography in the differential diagnosis of Achilles tendon injuries and related disorders: A comparison between pre-operative ultrasonography and surgical findings. *Acta Radiol* 1998;39:612-619.
5. Ozaki J, Fujiki J, Sugimoto K, Tamai S, Masuhara K: Reconstruction of neglected Achilles tendon rupture with Marlex mesh. *Clin Orthop Relat Res* 1989;238:204-208.
6. Maffulli N, Regine R, Angelillo M, Capasso G, Filice S: Ultrasound diagnosis of Achilles tendon pathology in runners. *Br J Sports Med* 1987;21:158-162.
7. Khan KM, Forster BB, Robinson J, et al: Are ultrasound and magnetic resonance imaging of value in assessment of Achilles tendon disorders? A two year prospective study. *Br J Sports Med* 2003;37:149-153.
8. Schepsis AA, Wagner C, Leach RE: Surgical management of Achilles tendon overuse injuries: A long-term follow-up study. *Am J Sports Med* 1994;22:611-619.
9. Clement DB, Taunton JE, Smart GW: Achilles tendinitis and peritendinitis: Etiology and treatment. *Am J Sports Med* 1984;12:179-184.
10. Coughlin MJ, Schon L: Disorders of tendons, in Mann RA, Coughlin MJ, Saltzman CL (eds): *Surgery of the Foot and Ankle*, ed 8. Philadelphia, PA: Mosby Elsevier, 2007, vol 1, pp 1149-1277.
11. Schepsis AA, Jones H, Haas AL: Achilles tendon disorders in athletes. *Am J Sports Med* 2002;30:287-305.
12. Clancy WG Jr, Neidhart D, Brand RL: Achilles tendonitis in runners: A report of five cases. *Am J Sports Med* 1976;4:46-57.
13. Puddu G, Ippolito E, Postachini F: A classification of Achilles tendon disease. *Am J Sports Med* 1976;4:145-150.
14. Kvist MH, Lehto MU, Jozsa L, Järvinen M, Kvist HT: Chronic achilles paratenonitis: An immunohistologic study of fibronectin and fibrinogen. *Am J Sports Med* 1988;16:616-623.
15. Saltzman CL, Tearse DS: Achilles tendon injuries. *J Am Acad Orthop Surg* 1998;6:316-325.
16. Galloway MT, Jokl P, Dayton OW: Achilles tendon overuse injuries. *Clin Sports Med* 1992;11:771-782.
17. Schepsis AA, Leach RE: Surgical management of Achilles tendinitis. *Am J Sports Med* 1987;15:308-315.
18. Saxena A: Results of chronic Achilles tendinopathy surgery on elite and nonelite track athletes. *Foot Ankle Int* 2003;24:712-720.
19. Johnston E, Scranton P Jr, Pfeffer GB: Chronic disorders of the Achilles tendon: Results of conservative and surgical treatments. *Foot Ankle Int* 1997;18:570-574.
20. Maquirriain J, Ayerza M, Costa-Paz M, Muscolo DL: Endoscopic surgery in chronic achilles tendinopathies: A preliminary report. *Arthroscopy* 2002;18:298-303.
21. Holmes GB, Lin J: Etiologic factors associated with symptomatic achilles tendinopathy. *Foot Ankle Int* 2006;27:952-959.

22. Alfredson H, Cook J: A treatment algorithm for managing Achilles tendinopathy: New treatment options. *Br J Sports Med* 2007;41:211-216.

23. Den Hartog BD: Flexor hallucis longus transfer for chronic Achilles tendinosis. *Foot Ankle Int* 2003;24:233-237.

24. Nicholson CW, Berlet GC, Lee TH: Prediction of the success of nonoperative treatment of insertional Achilles tendinosis based on MRI. *Foot Ankle Int* 2007;28:472-477.

25. Wilcox DK, Bohay DR, Anderson JG: Treatment of chronic achilles tendon disorders with flexor hallucis longus tendon transfer/augmentation. *Foot Ankle Int* 2000;21:1004-1010.

26. Ortmann FW, McBryde AM: Endoscopic bony and soft-tissue decompression of the retrocalcaneal space for the treatment of Haglund deformity and retrocalcaneal bursitis. *Foot Ankle Int* 2007;28:149-153.

27. McGarvey WC, Palumbo RC, Baxter DE, Leibman BD: Insertional Achilles tendinosis: Surgical treatment through a central tendon splitting approach. *Foot Ankle Int* 2002;23:19-25.

28. Wagner E, Gould JS, Kneidel M, Fleisig GS, Fowler R: Technique and results of Achilles tendon detachment and reconstruction for insertional Achilles tendinosis. *Foot Ankle Int* 2006;27:677-684.

29. Porter DA, Mannarino FP, Snead D, Gabel SJ, Ostrowski M: Primary repair without augmentation for early neglected Achilles tendon ruptures in the recreational athlete. *Foot Ankle Int* 1997;18:557-564.

30. Wapner KL: Delayed repair of the Achilles tendon, in Kitaoka HB (ed): *Masters Techniques in Orthopaedic Surgery: The Foot and Ankle.* Philadelphia, PA: Lippincott Williams & Wilkins, 2002, pp 323-335.

31. Leitner A, Voigt C, Rahmanzadeh R: Treatment of extensive aseptic defects in old Achilles tendon ruptures: Methods and case reports. *Foot Ankle* 1992;13:176-180.

32. Wapner KL, Pavlock GS, Hecht PJ, Naselli F, Walther R: Repair of chronic Achilles tendon rupture with flexor hallucis longus tendon transfer. *Foot Ankle* 1993;14:443-449.

33. Hansen ST: Trauma to the heel cord, in Jahss MH (ed): *Disorders of the Foot and Ankle*, ed 2. Philadelphia, PA: WB Saunders, 1991, pp 2355-2360.

34. Mann RA, Holmes GB Jr, Seale KS, Collins DN: Chronic rupture of the Achilles tendon: A new technique of repair. *J Bone Joint Surg Am* 1991;73:214-219.

35. Turco VJ, Spinella AJ: Achilles tendon ruptures: Peroneus brevis transfer. *Foot Ankle* 1987;7:253-259.

36. Arner O, Lindholm A: Subcutaneous rupture of the Achilles tendon: A study of 92 cases. *Acta Chir Scand Suppl* 1959;116(suppl 239):1-51.

37. Inglis AE, Sculco TP: Surgical repair of ruptures of the tendo Achillis. *Clin Orthop Relat Res* 1981;156:160-169.

38. Schedl R, Fasol P: Achilles tendon repair with the plantaris tendon compared with repair using polyglycol threads. *J Trauma* 1979;19:189-194.

39. Bugg EI Jr, Boyd BM: Repair of neglected rupture or laceration of the Achilles tendon. *Clin Orthop Relat Res* 1968;56:73-75.

40. Lee DK: Achilles tendon repair with acellular tissue graft augmentation in neglected ruptures. *J Foot Ankle Surg* 2007;46:451-455.

41. Lepow GM, Green JB: Reconstruction of a neglected achilles tendon rupture with an achilles tendon allograft: A case report. *J Foot Ankle Surg* 2006;45:351-355.