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Editors

The Achilles Tendon

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To my newborn son
Giuseppe Darius Peter Maffulli:
—May he never have problems with his Achilles tendon!

Foreword

Professor Maffulli and Dr. Almekinders are to be congratulated not only for identifying a gap in the orthopedic literature and the need for a book on the tendo Achillis, but also for successfully producing a comprehensive text to cover the subject. They have had a long-standing interest in this anatomical region, and thus have been able to carefully select an international group of contributors to help them deal with the basic science and all aspects of the clinical problems including endoscopic treatment.

There are many reasons for a book on one superficial tendon in the leg. First, it is the largest and strongest in the body and its problems are related to all aspects of walking and running. It is a hallmark of the bipedal human, as it is not found in the great apes. Nowadays, with widespread interest in sports and physical activity, problems related to the tendon are common. Nevertheless, although much has been written about the tendo Achillis, there is still a lot to be learned. Even management of a ruptured tendon remains an area of controversy. This tendon has been a focus of interest since the early days of orthopedic surgery, when, to reduce the chances of complications, subcutaneous tenotomy was popularized by Stromeyer in Hanover in the 1830s for the management of clubfoot and later by Little in London.

The book will be invaluable for orthopedic surgeons, specialists in sports medicine, physiotherapists, and podiatrists who deal with patients who have tendon problems that are sometimes intractable, seriously affect sporting careers, and may result in prolonged absences from competitions. Professor Maffulli and Dr. Almekinders have started something that will keep them occupied for many years, because this text will need regular revision to keep it up to date with the rapid growth of collagen chemistry, biomechanics, imaging, and techniques of clinical management.

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Preface

It all started when, as a fourth year medical student, one of us suffered from Achilles tendinopathy as the result of too much running, and we realized how little science existed on the topic. It was the beginning of a wonderful, neverending relationship, always involving new materials, and never boring.

The tendo Achillis is the largest and strongest tendon in the body—and the most frequently injured. The fact that it causes much aggravation had long been recognized, and the amount of clinical and basic science work performed on it is phenomenal. Nevertheless, we still lack evidence-based guidelines for best practices. Some authors are very sanguine about their views. Others keep an open mind and continue to perform hypothesis tensing-based studies.

The tendon is subjected to overuse and acute injuries. We have put together a multi-continental team to tackle a variety of issues on the Achilles tendon. We believe that we have covered most of the bases from the gross anatomy to the more sophisticated vision of the future in which gene therapy and tissue engineering will be in the forefront.

We learned a lot, but much needs to be done. We enjoyed assembling this material, and we hope that readers will benefit from it.

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1
The History of the Tendo Achillis and Its Rupture

Leslie Klenerman

The story of the tendo Achillis is bound up with both ancient Greek mythology and palaeoanthropology.[1] The tendo Achillis does not occur in the great apes (Fig. 1.1), our immediate ancestors, and is a hallmark of bipedal man. Its presence may be related to the greater relative length of the tarsal bones in man. The name is derived from the *Iliad* by the Greek poet, Homer, written between 750 and 650 B.C.

Achilles was a magnificent warrior and, according to myth, made invulnerable in infancy by his mother Thetis, who plunged him into the river Styx, one of the five rivers of the netherworld. Since he was held by one heel, this part was not bathed in the waters, and so was the one part of him that was unprotected. It was here that Achilles was mortally wounded by a poisoned arrow launched from the bow of Paris during the Trojan war (Fig. 1.2), which was waged by a confederation of Greeks against the people of Troy to recover Helen, whom Paris had abducted from her lawful husband Menelaus, King of Sparta.

The confusion between Achilles' heel and Achilles' tendon, as pointed out by Kirkup[2] probably dates from 1693, when the Flemish anatomist Phillippe Verheyen (1648–1710) (Fig. 1.3), Regius Professor of Anatomy and later of Surgery at the University of Louvain, Belgium, first recorded the term *tendo Achillis* in place of the ancient *tendo magnus* of Hippocrates, and the *chorda Hippocratis* of later authors.

Ambrose Pare (1510–1590), the famous French war surgeon, described the first closed rupture of the tendo Achillis:[3]

> Rupture of the tendo Achillis: an affect of the large tendon of the heel.
>
> It oftimes is rent or torn by a small occasion without any sign of injury or solution of continuity on the outside as by a little jump, the slipping aside of the foot, the too nimble getting on horseback, or the slipping of the foot out of the stirrup in mounting into the saddle. When this chance happens, it will give a crack like a coachman's whip: above the head where the tendon is broken the depressed cavity may be felt with your finger; there is great pain in the part and the party is unable to go. This mischance may be amended by long lying and resting in bed and repelling medicines applied to the part . . . neither must we promise to ourselves or to the patient certain or absolute health. But on the contrary at the beginning of the disease we must foretell that it will never be so cured, and that some relics may remain. . . .

From his description it does not appear to have been a rare event, but the treatment was ineffective. He also wrote,[4]

> For the wounds of that large tendon which is composed in the leg by the concourse of three muscles, and goes to the heel, I have observed that when it has been cut by a Sword, that the wounds have been long and hard to cure, and besides, when at the last they have been healed, as soon as the patient got out of his bed and endeavoured to go, they have grown ill and broke open again. . . .

It has remained a difficult wound to treat.

In 1724, Jean Louis Petit (1674–1750), the foremost surgeon in Paris during the first half of the eighteenth century, reported three cases, one of which was bilateral.[5]

Figure 1.1. The insertion of the gastrocnemius and soleus into the calcaneum in a gorilla. Note the absence of a tendo Achillis. (By kind permission of Dr Rachel Payne of The Structure and Motion Laboratory, The Royal Veterinary College, Hawkshead Lane, Hatfield, Hertfordshire, UK.)

John Hunter (1728–1793) described "Broken Tendo-Achilles" as "case number 355" in his notebooks:

On Thursday morning at four o'clock the 20th of February 1766, I broke my Tendo-Achilles. I was jumping and lighting upon my toes without allowing my heels to come to the ground, by which means I supported the whole weight of my Body, joined with the velocity of it in falling, upon the Gastro-cnemii and Soloei muscles: these two joined was too much for the tendon, which gave way at once, by which my heel came to the Ground. The snap (or report) made by breaking of the Tendon was heard all over the room. I stood still without being able to make another spring; and the sensation it gave me was as if something had struck the calf of my leg, and that the noise was the body that had struck me, falling on the floor and I looked down to see what it was, but saw nothing. I walked to a Chair, but could not throw myself forwards on the toes on that foot: the Calf of the leg was extremely painful, and was in the state of a Cramp. I endeavoured to take off the Cramp by bending the foot, but found that the motion had no effect upon the muscles of the Calf of the leg, and upon further examination I found that the Tendo-Achilles was broken.

I bound it up at first with the foot extended, and the knee a little bent; with the ends of the Tendon about

Figure 1.2. The wounded Achilles by the French sculptor Jean-Baptiste Carpeaux, 1850. (By kind permission of Conway Library, Courtauld Institute of Art, London, UK.)

Figure 1.3. Professor Philippe Verheyen. (By kind permission of the Wellcome Library, London, UK.)

half an inch distant (asunder). This bandage remained for five days, when I got Monro's bandage. In shifting the bandage I had the opportunity of examining the parts but they were a good deal swelled, so that I could not now tell whether the ends of the tendon were closed together or not. I examined the parts every day, and when the swelling of the inflammation abated, which was in less than a fortnight, the parts were so smooth that I could not find any inequality: the only swelling that remained was of the oedematous kind, which only swelled at night and was down in the morning. . . .

It continued much the same for about three weeks after the accident, when my Foot slipt upon a Wet floor, which made me pitch on the toe of that foot; which gave me great pain at the time, and which continued for a considerable time. Whether the parts were torn asunder or not I could not tell; the inflammation from this accident was more than the former; was now in a good deal of pain upon the least motion of the parts; and the swelling was more considerable than before.

In about a fortnight after this last accident, the parts became again more easy, and the swelling abated. I now began to walk again; for this purpose I got an old shoe raised in the heel about an inch, with a strap behind the buckle to a laced bandage round the Calf; but this was principally when I went to bed that I wore it, which was to avoid the consequence that might arise from any involuntary motions in those muscles in my sleep.

Hunter's request that his injured tendo Achillis be preserved after his death was not complied with, but it was confirmed at autopsy that there was ossification at the site of rupture. This accident led him, in 1767, to perform an experiment in which he divided the tendo Achillis of several dogs using a couching needle (the type of needle used for lens dislocation in the treatment of cataracts), to simulate a ruptured tendon. The dogs were killed at different periods to show progress of union.[6]

During the following centuries, the injury was recorded by a number of authors, although usually in single instances. The first major series was published by Quenu and Stoianovitch in 1929.[7] They compared the operative results with those obtained by conservative treatment in two groups, each of 29 cases. This showed the superiority of tendon suture, and was the start of a long controversy that still persists today. Harry Platt, who later became president of the Royal College of Surgeons of England and lived to 100 years of age, recorded 11 cases in 1931.[8] For fresh ruptures he advocated a stout transfixion suture of kangaroo tendon inserted well above the line of suture to provide contact between the ends of the tendon. Additional security was provided by a number of catgut sutures of lesser caliber. Surgeons from the Massachusetts General Hospital wrote up a series of 31 cases treated during the period 1900 to 1954, and found that 25% of patients sought treatment from 2 to 14 months after injury.[9] Arner and Lindholm[5] noted that the total number of cases up to 1958 was between 300 and 400. The numbers described are small in contrast to modern series due to greater awareness of the injury and more careful examination, particularly the use of Simmonds' squeeze test for diagnosis,[10] in addition to more frequent occurrence.

One of the earliest descriptions of surgical tenotomy appears in the treatise, "On Surgery," by the Greek surgeon Antyllus (second century

A.D.), who performed the procedure subcutaneously for the treatment of ankylosis of the ankle.[1] Frederick Louis Stromeyer (1804–1876), in Hanover, using very fine knives, was the first surgeon in more modern times to use subcutaneous tenotomy for the treatment of clubfoot.[11]

Both traumatic and overuse injuries of the tendo Achillis have now become relatively common, and it is not infrequent to find surgeons who have operated on several hundreds of such patients in their professional career—and all of this from a mythological Greek warrior!

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2
The Anatomy of the Achilles Tendon

Michael Benjamin, P. Theobald, D. Suzuki, and H. Toumi

Introductory Comments

The Achilles tendon (tendo calcaneus) is the strongest and thickest tendon in the body and serves to attach the triceps surae (soleus and the two heads of gastrocnemius) to the calcaneus (Fig. 2.1). It is a highly characteristic feature of human anatomy and it has even been suggested that the tendon has helped to shape human evolution. The emergence of man is critically linked to his ability to run, and man's unique combination of moderate speed and exceptional endurance has been underestimated.[1] The Achilles tendon has been a key player in the natural selection process, and as in modern apes, an Achilles tendon was absent from *Australopithecus* (a genus ancestral to the genus *Homo*) and probably originated in *Homo* more than 3 million years ago.[1]

Several unique functional demands are placed upon the Achilles tendon that add to its vulnerability to injury:

1. The upright stance of the human dictates that the foot is at a right angle to the leg in the anatomical position and that the Achilles tendon approaches the back of the foot tangentially and generates heavy torque. The human thus has one of the largest angles between the long axis of the tibia and the calcaneus in any mammal.

2. The muscles contributing to the formation of the tendon have different functions and different physiological properties. The soleus plantar flexes the ankle joint and contains a high proportion of type I (slow-twitch) fibers, which facilitates its role as a postural muscle, preventing the body from falling forward when standing.[2] However, the gastrocnemius also flexes the knee joint, and contains a greater number of type IIB fibers (fast twitch). These promote the vigorous propulsive movements that occur in sprinting and jumping.

3. As the Achilles tendon attaches to the calcaneus, it acts on the subtalar as well as the knee and ankle joints. Because the axis of the subtalar joint typically passes upward and medially from the posterolateral corner of the calcaneus,[3] the triceps surae also supinates the foot.[4] Thus stress concentration between the medial and lateral sides of the Achilles tendon enthesis can be nonuniform.

4. The rotation of the limb bud that occurs during development implies that the adult Achilles tendon is twisted upon itself, so that the fibers derived from the gastrocnemius are attached to the lateral part of the calcaneal insertion site and those derived from soleus are attached medially.[5,6] Thus, when the tendon is under load, it is subject to a "wringing" action. Because the gastrocnemius crosses the knee joint and a flexed knee can rotate, the part of the Achilles tendon that is derived from the tendon of gastrocnemius can be variably twisted relative to the tendon of soleus (i.e., one tendon can exert a sawing action on the other).[4] This complex rotatory action is further compounded by the shape of the talus. This shape accounts for the fact that there is a subtle change in the position of the axis of the ankle joint relative to the Achilles tendon during dorsi- and plantar flexion. Slight passive rotation occurs.[7]

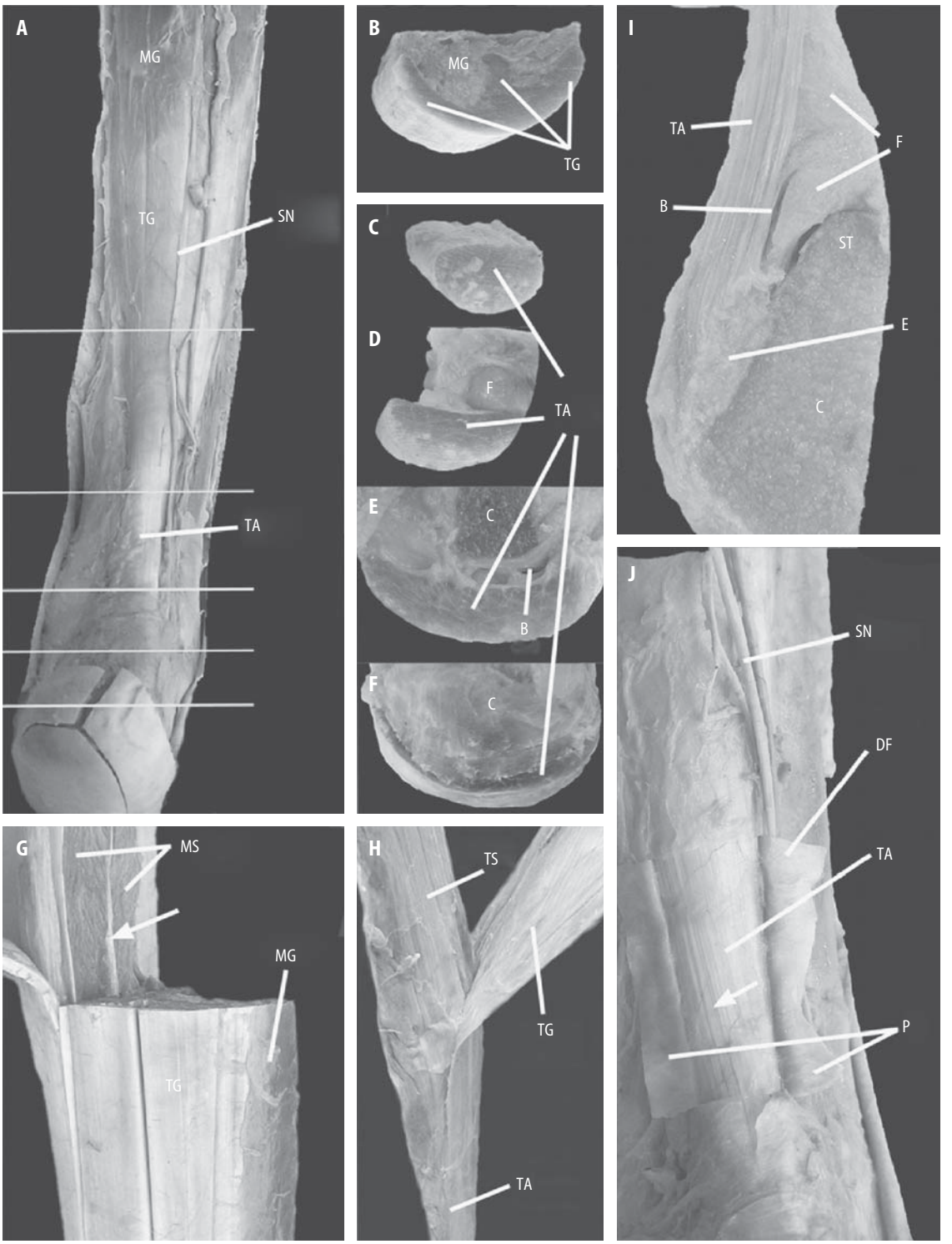
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Figure 2.1. Gross anatomy of the Achilles tendon. (**A**) A posterior view of the right Achilles tendon indicating with horizontal lines the levels at which the transverse sections featured in **B–F** are taken. Note the close relationship of the Achilles (TA) and gastrocnemius (TG) tendons to the sural nerve (SN). MG, muscle belly of gastrocnemius. (**B–F**) Transverse sections of the Achilles tendon to show the change in shape of the tendon from proximal to distal. Figures **B–F** inclusive correspond (from above down) to the 5 horizontal lines shown in figure **A**. Note that the gastrocnemius tendon is very broad and flat (**B**), that the Achilles tendon in the region vulnerable to ruptures is oval (**C**), and that the tendon flares out again (**D–F**) as it approaches the calcaneus (C). Sections taken at levels **D–E** pass through the pre-Achilles fat pad (F) and the retrocalcaneal bursa (B) into which the fat pad protrudes. At the enthesis itself (**F**), the extremely flattened Achilles tendon has a marked anterior curvature. (**G**) Here, both gastrocnemius and soleus have been partly removed so as to demonstrate the intramuscular tendon of soleus (arrow). MS, muscle belly of soleus. (**H**) The union of the tendons of soleus (TS) and gastrocnemius that form the Achilles tendon at mid-calf level. (**I**) A sagittal section through the calcaneus to show the Achilles tendon enthesis (E) and the prominent pre-Achilles fat pad (F). The tip of the fat pad is quite distinctive from the rest and protrudes into the retrocalcaneal bursa (B) between the Achilles tendon and the superior tuberosity of the calcaneus (ST). (**J**) A posterior view of the Achilles tendon to show its associated paratenon (P). A rectangular window has been cut into the paratenon exposing the underlying Achilles tendon in which a slight obliquity of the tendon fascicles can be noted (arrow).

5. The Achilles tendon transmits forces that are approximately seven times the body weight during running.[8] This represents an enormous increase on the forces that act during standing (which are roughly half the body weight).[8]

Gross Anatomy

The formation of the Achilles tendon from the gastrocnemius and soleus muscles has been described in detail by Cummins et al.[6] The medial and lateral heads of gastrocnemius arise from the femoral condyles and their contribution to the Achilles tendon commences as a wide aponeurosis at the lower ends of these muscular bellies (Fig. 2.1A). In 2.9–5.5% of people, there is a third head of gastrocnemius, most commonly associated with the medial head.[9] Occasionally plantaris can effectively form a third head (i.e., when it joins gastrocnemius at the point of convergence of its medial and lateral heads).[9] The lateral head of gastrocnemius can sometimes be reduced to a fibrous cord.[9]

The soleus arises entirely below the knee, largely from the tibia and fibula, and its tendinous contribution to the Achilles is thicker but shorter.[6] Occasionally, the tibial "head" of soleus can be absent or an accessory soleus muscle present between the soleus tendon and flexor hallucis longus.[9] An accessory soleus can contribute to the formation of the Achilles tendon, attach independently on the calcaneus, or fuse with the medial collateral ligament of the ankle joint.[9] Typically, a broad sheet of connective tissue begins on the posterior surface of the soleus muscle belly, at a position more proximal than the start of the aponeurosis of gastrocnemius (Fig. 2.1H). Consequently, where the soleus and gastrocnemius muscle bellies are in contact with each other (i.e., are subject to mutual pressure), the two bellies are separated by dense fibrous connective tissue on the surface of the muscles (Fig. 2.1H) and by a thin film of loose connective tissue between them. There is a similar arrangement in the quadriceps femoris, where the anterior surface of vastus intermedius is aponeurotic and overlain by the rectus femoris, but separated from it by areolar connective tissue. Such a tissue probably promotes independent movement.

The sheet of connective tissue on the posterior surface of soleus is attached to the gastrocnemius aponeurosis by fascia at a variable point near the middle of the calf (Fig. 2.1H). The combined aponeurosis continues to run distally over the posterior surface of the soleus, receiving further tendinous contributions from the muscle as it descends. In addition, there is a narrow intramuscular tendon within the soleus (promoting a bipennate arrangement of muscle fibers) that merges with the principal tendon distally (Fig. 2.1G).[10] Typically, full incorporation of the soleus and gastrocnemius tendons into the Achilles tendon is evident 8–10 cm above the calcaneal

attachment site, but occasionally the tendon of soleus can remain separate from that of gastrocnemius as far as the insertion itself.[11] Sometimes, the two heads of gastrocnemius remain separate, and the tendons that arise from them attach independently (both from each other and from the tendon of soleus) on the calcaneus.[9] Such anatomical variations can give a false impression of a pathologically thickened Achilles tendon. When viewed from behind, a typical soleus muscle belly is covered proximally by the gastrocnemius, but distally it protrudes on either side of the tendon of the gastrocnemius, making this a convenient site for biopsy or electromyography.[10]

As the tendon fibers derived from gastrocnemius descend, they converge so that the Achilles tendon narrows. However, the fibers also rotate around those of soleus, so that they ultimately come to be attached to the calcaneus laterally, whereas those of soleus (which also rotate) attach more medially.[6] The degree of rotation is variable, so that in addition to contributing to the lateral part of the calcaneal attachment site in all individuals, the gastrocnemius tendon contributes to its posterior part in some people and to its anterior part in others.[6] This rotation becomes more obvious in the terminal 5–6 cm of the tendon (Fig. 2.1J). Where the twisting of the tendon is marked, it is easier to trace the individual contributions of the soleus and gastrocnemius tendons to the Achilles tendon where rotation is slight.[4] The spiraling of the tendon fascicles results in less fiber buckling when the tendon is lax and less deformation when the tendon is under tension. This reduces both fiber distortion and interfiber friction.[12]

A variable proportion of the superficial fibers of the Achilles tendon do not attach to the calcaneus at all, but pass under the heel to become continuous with the fibers of the plantar fascia. Such soft tissue continuity is particularly marked in younger individuals[13] and is in line with a general principle that relatively few tendons attach to bone in isolation; most fuse with adjacent structures or attach at more than a single site, so as to dissipate stress concentration.[14] Myers[15] has greatly expanded on the related concept of myofascial continuities via an endless fascial "web" in the body.

The shape of the Achilles tendon varies considerably from proximal to distal (Fig. 2.1B–F). As with many tendons elsewhere in the body, the Achilles tendon flares out as it nears its bony attachment site. This contributes to the marked anterior-posterior flattening, and slight anterior concavity of the tendon, evident at the level of its enthesis (Fig. 2.1F). These features are also seen at imaging.[11] Typically, the distal part of the tendon does not exceed 7 mm in thickness and anything greater than that is suggestive of pathology.[16] At the insertion site itself, where the tendon is extremely flattened, it is approximately 3 cm wide and 2–3 mm thick.[17]

The Achilles tendon lacks a true synovial tendon sheath but has a false sheath or "paratenon" (Fig. 2.2A) that forms an elastic sleeve permitting the tendon to glide relative to adjacent structures.[18] The paratenon essentially consists of several closely packed, membranous sheets of dense connective tissue that separate the tendon itself from the deep fascia of the leg. It is rich in blood vessels and nerves and, together with the epitenon, which adheres to the surface of the tendon itself, is sometimes referred to as the peritenon. It can stretch 2–3 cm as the tendon moves.[19]

Relationships

The deep fascia of the leg is immediately superficial to the sheath of the Achilles tendon (Fig. 2.1J), fuses with the tendon sheath near the calcaneus, and serves as an unheralded retinaculum for the tendon. It thus contributes to the slight anterior curvature of the tendon[20,21] and prevents the tendon from bowstringing in a plantar flexed foot. We thus suggest that it plays an important role in minimizing insertional angle changes that occur at the enthesis during foot movements. This in turn reduces wear and tear.

The sural nerve lies in close contact with the Achilles tendon sheath (Fig. 2.1A, J) and commonly crosses its lateral border approximately 10 cm above the tendon enthesis.[22] The vestigial muscle belly of plantaris arises adjacent to the lateral head of gastrocnemius and its long tendon runs along the medial side of the Achilles tendon to end in a variable fashion. Usually, it attaches to the calcaneus on the medial side of the Achilles tendon (47% of cases according to Cummins

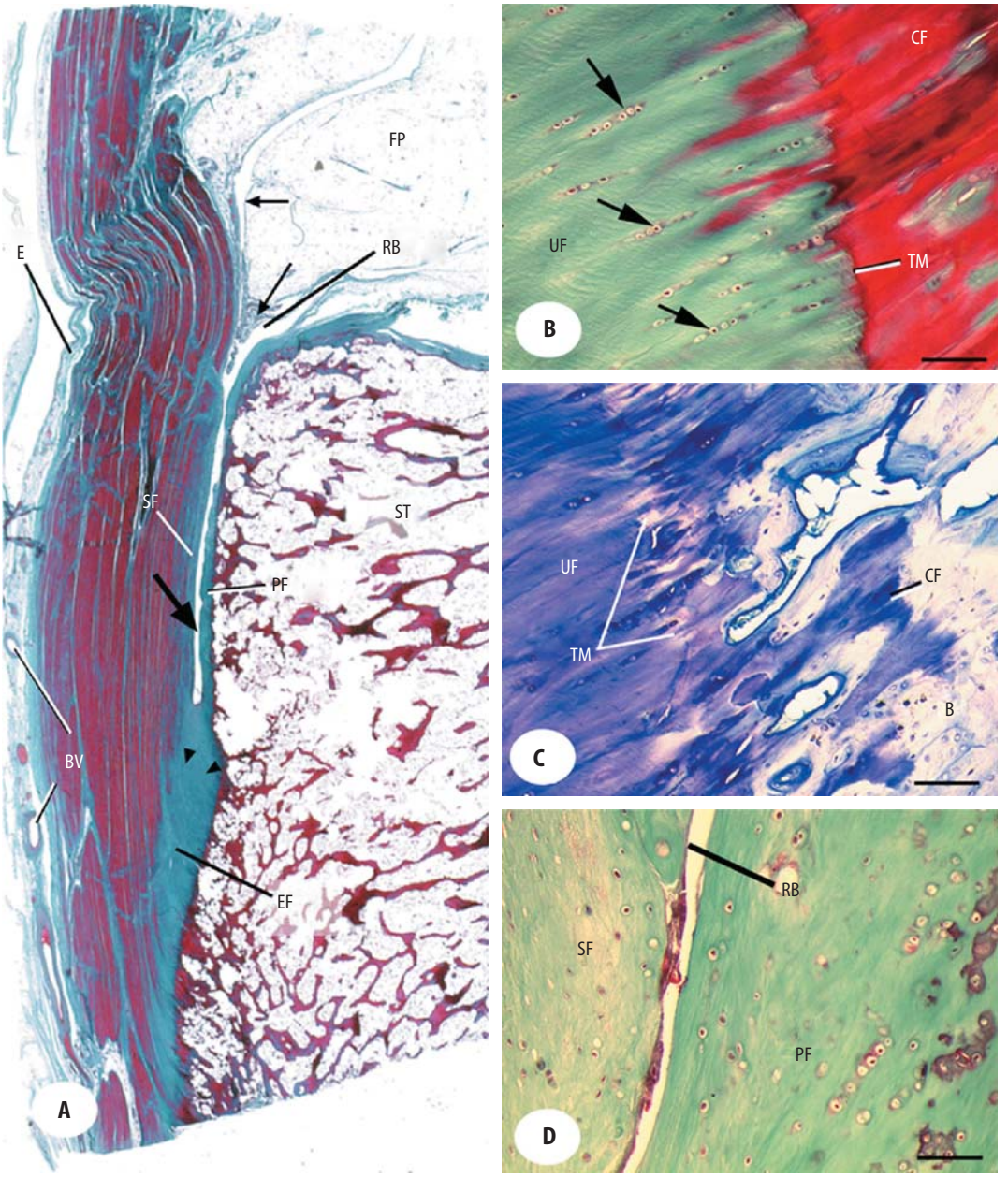

Figure 2.2. Microscopic anatomy of the Achilles tendon enthesis organ. (**A**) Low-power view of a sagittal section of the enthesis organ. The enthesis itself is characterized by a prominent enthesis fibrocartilage (EF), which is thickest in the deepest part of the attachment site (arrowheads). Immediately proximal to the osteotendinous junction, the deep surface of the tendon is related to the superior tuberosity (ST) of the calcaneus, but is separated from it by the retrocalcaneal bursa (RB). Protruding into the bursa is the pre-Achilles fat pad (FP), which is covered with a synovial membrane (arrows). The most distal part of the bursa is lined directly by sesamoid (SF) and periosteal fibrocartilages (PF). The former lies in the deep surface of the Achilles tendon, immediately adjacent to the enthesis, and the latter covers the superior tuberosity in a dorsiflexed foot. These fibrocartilages are shown in further detail in figure **D**. Note the epitenon (E) on the posterior surface of the tendon with several blood vessels (BV) visible within it and the paucity of a subchondral bone plate at the enthesis. (**B**) A high-power view of the enthesis fibrocartilage in the region either side of the tidemark (TM). Note the longitudinal rows of fibrocartilage cells (arrows) in the zone of uncalcified fibrocartilage (UF) and the zone of calcified fibrocartilage (CF) that lies immediately deep to the tidemark. (**C**) A high-power view of the enthesis fibrocartilage in the region either side of the tidemark, showing the complex interdigitations of the zone of calcified fibrocartilage with the underlying bone (B). (**D**) A high-power view of the fibrocartilaginous lining of the distal part of the retrocalcaneal bursa showing sesamoid fibrocartilage in the deep surface of the tendon and a periosteal fibrocartilage covering the bone. Note that neither fibrocartilage is covered with synovium. Scale bars: a = 2 mm; b–d = 100 μm. Figure **C** is of a specimen stained with toluidine blue; all the other sections are stained with Masson's trichrome.

et al.),[6] but in 36.5% of the 200 specimens with a plantaris tendon examined by these authors, the tendon inserts slightly anterior to the medial aspect of the Achilles. Intriguingly, in such individuals, the enthesis of the plantaris tendon serves to support the anteromedial part of the retrocalcaneal bursa. In the third variation of the plantaris insertion reported in 12.5% of cases by Cummins et al.,[6] the tendon fans out distally to invest the posterior and medial aspects of the Achilles tendon. Finally, in 4% of individuals, the plantaris tendon fuses with the Achilles tendon proximal to the calcaneal attachment site of the latter.[6]

Near its calcaneal insertion site, the Achilles tendon is flanked by two bursae.[23] There is a superficial bursa between the skin and the tendon that promotes skin movement and a deep (retrocalcaneal) bursa between the tendon and the superior calcaneal tuberosity that promotes tendon movement (Fig. 2.1I). Protruding into the retrocalcaneal bursa is a wedge-shaped, fatty, synovial-covered fold that represents the distal tip of Kager's fat pad, a mass of adipose tissue between the flexor hallucis longus muscle and the Achilles tendon (Fig. 2.1I). Intriguingly, the relative size of this fat pad differs between the foot of the newborn child and the adult,[24] though the significance of this is unclear. Latex molds of the bursa show that it is disc-shaped and has two extensions ("legs") directed proximally (see Fig. 4 in ref. 24). It is molded over the posterosuperior surface of the calcaneus, like a cap with an anterior concavity.[24] A healthy bursa has a smooth outline and 1–1.5 ml of contrast medium can be injected into it.[24] However, leakage of contrast material over time into the superficial bursa suggests that the bursae communicate with each other.[24] At magnetic resonance imaging (MRI), the retrocalcaneal bursa normally contains fluid, which gives a high-signal-intensity.[25] The bursa is filled with a clear, viscous fluid,[26] and in healthy individuals, the tip of Kager's fat pad moves in and out of the bursa in plantar and dorsiflexion respectively (M. Benjamin, P. Theobald, L. Nokes, and N. Pugh.[26A] This may influence the insertional angle of the Achilles tendon in different foot positions.[27] Although the retrocalcaneal bursa is enlarged in symptomatic patients, paradoxically, less contrast material can be injected into it.[23]

Blood Supply

The Achilles tendon receives part of its blood supply from vessels running in the paratenon that are largely derived from the posterior tibial artery.[12,28,29] The vessels enter the tendon via a structure that is comparable to a mesotenon.[4] The mid-region of the tendon is relatively poorly vascularized and this may contribute to the vulnerability of the tendon to rupture, 2–6 cm above the calcaneus. The proximal part of the tendon receives an additional supply from the muscle bellies that continues into the tendon via the endotenon, though this contribution is not believed to be significant.[12,30–32] The distal region of the tendon also receives vessels from an arterial periosteal plexus on the posterior aspect of the calcaneus.[33] This supply starts at the margin of the insertion and extends up the endotenon for approximately 2 cm proximally.[12,30,32,34] A healthy fibrocartilaginous enthesis is avascular so that vessels do not normally pass directly from bone to tendon at the osteotendinous junction.[35,36]

Innervation

There is no single comprehensive study of the innervation of the Achilles tendon from its myotendinous junction to its enthesis. Nevertheless, the sensory nerve supply of the tendon and its sheath is of nociceptive and proprioceptive significance. The integrity of the nerve supply to the tendon may also play a key role in promoting its repair, as peripheral denervation in rats reduces the load to failure of healing, transected Achilles tendons by 50% within two weeks.[37]

The Achilles tendon is supplied by sensory nerves from the contributing muscles and via twigs from neighboring cutaneous nerves, notably the sural nerve.[38] The paratenon is more richly innervated than the tendon itself, and it contains Pacinian corpuscles,[39] presumably important in proprioception. Both Golgi tendon organs and muscle spindles have been demonstrated in association with the Achilles tendon of the cat.[40] The former lie in the muscle itself, close to the myotendinous junction, but the latter are located more distally in the tendon.

There is an opioid system in the rat Achilles tendon that may contribute to a peripheral inhibi-

tion of pain.[41] Some of the sensory nerves (probably C fibers) immunolabel for the delta opioid receptor (DOR). Labeling is largely restricted to the endotenon and epitenon, where it typically occurs in association with blood vessels, and to the paratenon, where a vascular association is less obvious. The DOR labeling co-localizes with that for enkephalins, suggesting that the latter act as receptors. Enkephalins acting on DOR inhibit the nociceptive action and the pro-inflammatory response of sensory neuropeptides.[41] There is normally a fine balance between the expression of opioids in muscle-tendon units and the expression of sensory neuropeptides that could change with tendon pathology.[41]

It is difficult to reconcile what we know of the innervation of the Achilles tendon with the pain associated with tendinopathy.[42] Tendon pain may be linked to vascular changes. A common feature of tendinopathy is the proliferation of blood vessels either in the tendon itself or its sheath,[43–45] and injured tendons may show an ischaemic response.[42]

Structure of the Tendon Midsubstance

As with all tendons, the Achilles tendon is dominated by type I collagen, which accounts for its considerable tensile strength,[46] in the order of 50–100 N/mm.[46,47] However, this may well be an underestimate because of the general difficulty of clamping tendons, which by their very nature consist of large numbers of partly independent fibers.[48] Type I collagen is organized into heterotypic fibrils in association with types III and V collagens[46] and these minor collagens play a role in regulating fibril diameter.[49] Western blot analyses of Achilles tendons from elderly individuals show that the β and γ forms of type I collagen are conspicuous—probably reflecting the increased formation of crosslinks with age.[46]

Type I collagen fibrils are grouped successively into fibers, fiber bundles, and fascicles, so that a tendon is analogous to a multistranded cable.[49] Individual fibrils do not run the length of a tendon and thus stress must be transferred between them.[49] This is a function of the amorphous matrix in which the fibrils are embedded and it has been suggested that type VI collagen (a non-fibrillar collagen) and decorin (a leucine-rich repeat proteoglycan) are important. Both these molecules, along with fibromodulin, biglycan, lumican, and versican, are present in the Achilles tendon[46] and have a relatively high turnover.[50]

In general, fibrils within tendons run a wavy course (i.e., are "crimped") with an axial periodicity of approximately 100 μm.[49] Such "pre-buckling" is thought to contribute to their flexibility, along with the partial independence of fibrils and fascicles that derives from the low compressive stiffness of the extracellular matrix.[49] Of key importance here is the endotenon that separates adjacent fascicles and is continuous with the epitenon on the surface of the tendon. The endotenon forms vascularized and innervated layers of loose connective tissue that promote independent movement between fascicles.

The cells in the midsubstance of the Achilles tendon are fibroblasts that are arranged in longitudinal rows and have a highly complex shape. In the midsubstance of tendons, there are a number of broad, flat cell processes that extend laterally from the cell bodies and partition the collagen fibers into bundles.[51] There are also more elongated and thinner cell processes that extend longitudinally within a tendon. In both cases, where processes of adjacent tendon cells meet, the cells communicate by means of gap junctions.[51] Communication is established between cells both within and between rows. Consequently, there is a three-dimensional network of interlinking cell processes in the Achilles tendon that is as impressive as the better-known network of osteocytic cell processes permeating the extracellular matrix (ECM) of bone. Gap junctional communication (involving connexins 32 and 43) could form the basis for a co-coordinated response of tendon cells to mechanical load.[51] Connexin 32 junctions occur predominantly between cells within a row (and thus along the lines of principal tensile loading), while gap junctions characterized by connexin 43 link cells between rows as well.[51] Waggett et al.[52] have thus suggested that the two different gap junctions have distinctive roles in ECM synthesis when tendon cells are subject to mechanical loading. They have shown that connexin 43 gap junctional communication inhibits collagen synthesis, whereas that involving connexin 32 is stimulatory.

The Enthesis and the Enthesis Organ

The Achilles tendon attaches to a rectangular area in the middle third of the posterior surface of the calcaneus—with a greater surface area of the tendon attached medially than laterally.[53] The average height of the insertion (i.e., the distance between the superior and inferior limits of the tendon attachment) is 19.8 mm, and the average width is 23.8 mm superiorly and 31.2 mm inferiorly.[54] Thus, the tendon flares out considerably at its enthesis, dissipating the region of stress concentration. Although it is unlikely that the increased surface area of the tendon at this site is associated with a greater number of collagen fibers, the nature of the packing tissue has not been firmly established. In other tendons, fat accumulation near the osteotendinous junction is an important contributory factor.[55]

As with other tendons in the body, the direction in which the Achilles tendon approaches its insertion site is kept relatively constant in different positions of the foot and leg. When the foot is dorsiflexed, the superior tuberosity of the calcaneus (Fig. 2.2A) acts as a guiding pulley, but, during plantar flexion, simple inspection suggests that the deep crural fascia must be primarily responsible for controlling the insertional angle.[4] In pronation and supination movements of the calcaneus, comparable guiding control mechanisms for maintaining constancy of bone–tendon position are less obvious. Although continuity of the crural fascia with the periosteum on the medial and lateral aspects of the calcaneus is likely to be a factor, the fibrocartilaginous nature of the enthesis is probably also important. The "enthesis fibrocartilage" (Fig. 2.2A–C) balances the differing elastic moduli of the tendon and bone and reduces stress concentration at the insertion site.[56] Effectively, it stiffens the tendon at the hard–soft tissue interface and plays a role analogous to that of a grommet where a lead joins an electrical plug.[57] It ensures that any bending of the collagen fibers of the tendon is not all concentrated at the hard–soft tissue interface, but is gradually dissipated into the tendon itself, reducing the risk of wear and tear.

However, the task of reducing stress concentration at the Achilles enthesis does not all relate to mechanisms at the tendon–bone junction. In a dorsiflexed foot, the adjacent anterior surface of the tendon presses against the superior tuberosity of the calcaneus (Fig. 2.2A) and this reduces stress concentration at the enthesis itself. What never seems to be acknowledged in accounts of the surgical treatment of Haglund's deformity is the increase in stress concentration at the enthesis that inevitably follows any removal of bone from the superior tuberosity. The extent to which the stress concentration is increased depends on the prominence of the tuberosity. Such considerations may be particularly important when contemplating surgery on elite athletes in whom the Achilles tendon may periodically be heavily loaded.

The intermittent contact between the tendon and the superior tuberosity is associated with structural specializations at both surfaces because of the mutual compression of the tissues. Thus, the calcaneus is covered by a thick fibrocartilaginous periosteum and the deep surface of the tendon is lined by a "sesamoid fibrocartilage" (Fig. 2.2A, C, D).[58] The latter term was coined because this fibrocartilage lies within the substance of the tendon itself (i.e., it is comparable to a sesamoid bone). The free movement of the opposing surfaces is promoted by the retrocalcaneal bursa into which a tongue-like, downward extension of Kager's fat pad extends in a plantar flexed foot. The enthesis itself, the periosteal and sesamoid fibrocartilages, bursa and fat pad collectively constitute an "enthesis organ" (Fig. 2.2A).[14,36] This is a collection of tissues that all contribute to the common function of reducing stress concentration and the risk of failure at the osteotendinous junction.

At the distal tip of the retrocalcaneal bursa there is no synovial lining, for the walls of the bursa are formed directly by the sesamoid and periosteal fibrocartilages.[36,58] While it may surprise some readers to learn that part of the bursa is not lined by synovium, it is logical when one remembers that the bursa has much in common with a synovial joint.[36,59] The sesamoid and periosteal fibrocartilages serve effectively as articular cartilages and are thus subject to compression (in a dorsiflexed foot). Consequently, like classical articular cartilage, they cannot be covered with a vascular synovial membrane; this is therefore restricted to the more proximal parts of the bursa

(Fig. 2.2A). Degenerative changes paralleling those seen in osteoarthritic articular cartilage (in particular fissuring and chondrocyte clustering) are common in elderly people.[58] Detachment of tissue fragments into the bursa is also frequently seen. The inflammatory changes characteristic of retrocalcaneal bursitis may be a secondary consequence of what is primarily an issue of fibrocartilage degeneration.[58]

Four zones of tissue have been described at the enthesis itself: dense fibrous connective tissue, uncalcified fibrocartilage, calcified fibrocartilage, and bone.[36,58] Between the zones of calcified and uncalcified fibrocartilage is a tidemark, which marks the outer limit of calcification (Fig. 2.2B). In a healthy tendon, the tidemark is remarkably straight, for it serves as the mechanical boundary between hard and soft tissues. However, it is *not* the tissue boundary (i.e., the *exact* location of the tendon–bone junction). This boundary is the highly irregular interface between the zone of calcified enthesis fibrocartilage and the subchondral bone (Fig. 2.2C). The complex interdigitation of the two tissues in three dimensions is pivotal in securing the tendon to the bone, for little anchorage is provided by the direct continuation of collagen fibers from tendon to bone.[60] Thus, the mechanical and tissue boundaries of the tendon are spatially distinct. Conflicting functional demands means that they cannot coincide exactly. The mechanical boundary must be straight in a healthy enthesis so that the tendon is not damaged by jagged edges of bone as the tendon moves. However, the tissue boundary must be highly irregular to promote firm anchorage of tendon to bone. The mechanical paradox is solved in the adult tendon at least, by the presence of a thin coating of calcified fibrocartilage on the bone surface (Fig. 2.2C). This can be visualized as analogous to a layer of cement applied over rough cast brickwork. The presence of this layer accounts for the smooth marking left by the Achilles tendon on a dried bone. The soft tissues fall away from the bone at the level of the tidemark after maceration.[61]

As with other fibrocartilaginous entheses, Sharpey's fibers are not a prominent feature of the Achilles tendon insertion. This reflects both the development of the enthesis and the paucity of compact bone in the subchondral plate (Fig. 2.1A). In the rat Achilles tendon, the enthesis fibrocartilage develops by metaplasia of fibroblasts in the dense fibrous connective tissue of the tendon near its bony interface.[62] Thus the fibrocartilage cells are arranged in longitudinal rows (Fig. 2.2B) simply because the fibroblasts from which they develop also have this arrangement. The fibrocartilage probably develops in response to mechanical stimuli shortly after birth. The tissue acts as a "mini–growth plate" for the bone.[62] As tendon fibroblasts turn into fibrocartilage cells on one side of the enthesis (i.e., the border between the zones of dense fibrous connective tissue and uncalcified fibrocartilage), bone replaces fibrocartilage at the other, by a process analogous to endochondral ossification in the growth plate of a long bone.[62]

Enthesis fibrocartilage is not equally obvious over the entire osteotendinous junction. It is more conspicuous superiorly (i.e., in the deep part of the tendon; Fig. 2.2A) than inferiorly—where the enthesis is more fibrous. Curiously, bony spurs typically develop in the postero-inferior part. The wedge shape of the enthesis fibrocartilage may enable it to act as a soft-tissue pulley by virtue of its viscoelasticity.[60] This complements the action of the more obvious bony pulley that is formed by the superior tuberosity. However, such a soft-tissue pulley can compensate only slightly for the marked decrease in the moment arm of the Achilles tendon that inevitably occurs when the foot is dorsiflexed. Quigley and Chaffin[63] have calculated that the distance from the Achilles tendon to the axis of rotation of the ankle joint (i.e., the moment arm) decreases by 40% at 35° of dorsiflexion. This means that greater muscular effort is needed to rise onto the toes, and thus greater load is transferred from muscle to tendon and from tendon to bone.

Finally, little attention has been paid to the bone beneath the Achilles tendon enthesis. As stated above, there is a striking absence of any substantial layer of cortical bone (Fig. 2.2A). However, there is a highly ordered array of trabeculae orientated along the long axis of the Achilles tendon, linking the tendon enthesis to that of the plantar fascia.[60] The trabecular pattern suggests that there is a line-of-force transmission within the bone, linking these two soft tissues. In younger individuals, in particular, there can also

be soft tissue continuity between the Achilles tendon and the plantar fascia.[13] The situation is thus analogous to that in the patellar tendon where again there are parallel trabeculae in the anterior region of the patella, and tendon fibers that pass over the anterior surface to establish direct continuity between the patellar and quadriceps tendons (M. Benjamin).[64] In both cases, this presents a classic example of the "myofascial" continuity concept[15] that emphasizes the endless web formed by connective tissue throughout the body.

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3
Biomechanics of the Achilles Tendon

Constantinos N. Maganaris, Marco V. Narici, Louis C. Almekinders, and Nicola Maffulli

Tendons act as contractile force transmitters enabling skeletal movement. Fulfilling this role, however, tendons do not behave as rigid links between muscles and bones, but exhibit a viscoelastic behavior. This chapter reviews the main features and functional implications of this biomechanical behavior with specific reference to the Achilles tendon.

In Vitro Testing

The mechanical properties of tendons have traditionally been studied using methodologies involving stretching of isolated tendon specimens to failure, with both the specimen elongation and the applied force recorded throughout the test.[1–6] In such tests, four different regions can be identified in the tendon force–elongation curve produced (Fig. 3.1). Region I, referred to as the tendon "toe" region, is associated with nondamaging forces that reduce the resting crimp angle of collagen fibers without causing further fiber stretching. In the following "linear" region II, loading causes stretching of the already aligned fibers, and at the end point of this region some fibers start to break. Further elongation brings the tendon into region III, where additional fiber failure occurs in an unpredictable manner. Even further elongation brings the tendon into region IV, where complete failure occurs.[1–6]

Although regions I, II, III, and IV are always present when pulling a tendon until it breaks, the shape of the force–elongation curve obtained differs between specimens. To a great extent, these differences can be caused by interspecimen dimensional differences. To account for this, tendon forces are reduced to stress values (MPa) by normalization to the tendon cross-sectional area, and tendon elongations are reduced to strain values (%) by normalization to the tendon original length. The shape of the stress–strain curve is similar to the force–elongation curve, but it reflects the intrinsic material properties rather than the structural properties of the tendon. The most common material variables taken from a stress–strain curve are the Young's modulus (GPa), the ultimate stress (MPa), and the ultimate strain (%). Young's modulus is the slope of stress–strain curve in the "linear" region of the tendon (or the product of stiffness [N/mm], i.e., the slope of the force–elongation curve in the "linear" region, and the original length-to-cross-sectional area ratio of the specimen). It ranges between 1 and 2 GPa.[2,7–9] Ultimate tendon stress (i.e., tensile stress at failure) is ~100 MPa. Ultimate tendon strain (i.e., strain at failure) ranges between 4 and 10%.[2,4,5,7,8] The values for the human Achilles tendon properties in the *in vitro* state approximate the above figures.[10,11]

If a tendon is stretched, it does not behave perfectly elastically, even if the force applied does not stretch the tendon beyond its "toe" region. Due to the time-dependent properties of the tendon collagen fibers and interfiber matrix,[12,13] the entire tendon exhibits force-relaxation, creep, and mechanical hysteresis.[1–5] Force-relaxation means that the force required to cause a given elongation decreases over time in a predictable

curvilinear pattern (Fig. 3.2). Creep is the analogous phenomenon under constant-force conditions, yielding deformations that increase over time curvilinearly (Fig. 3.2). Mechanical hysteresis is evidenced as a loop formed by the force–elongation (or stress–strain) plots during loading and subsequent unloading of the specimen (Fig. 3.2). The area of the loop represents the amount of elastic strain energy lost as heat in the stretch–recoil cycle, and it is usually expressed as a fraction (%) of the total work done on the tendon during stretching. The average mechanical hysteresis value reported from in vitro tendon tests is ~10%.[7–9,14,15] This value, however, is obtained after the tendon is subjected to a few stretch–recoil cycles. In the first few cycles, the tendon does not recover its original length, resulting in the loading and unloading plots forming an open loop. This phenomenon is referred to as "conditioning," and it has been considered as an artifact caused by inadequate fixation of the in vitro specimen tested.[3] Recent results, however, in the human Achilles/gastrocnemius tendon show that conditioning occurs also in vivo,[16,17] indicating that it is an actual physical property of the tendon associated with viscoelastic creep—not an artifactual effect.

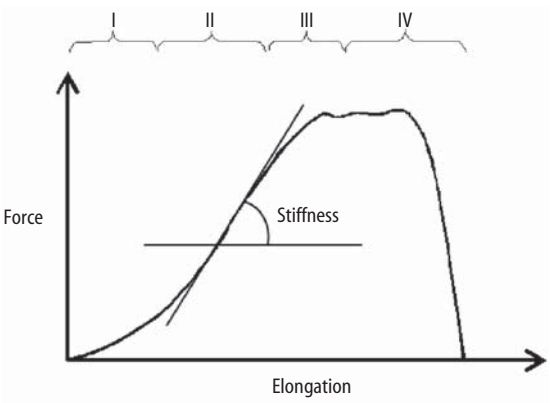

Figure 3.1. Typical force–elongation plot in a tendon tensile test to failure. I: "toe" region; II: "linear" region; III and IV: failure regions. Stiffness is the slope of the curve in the linear region.

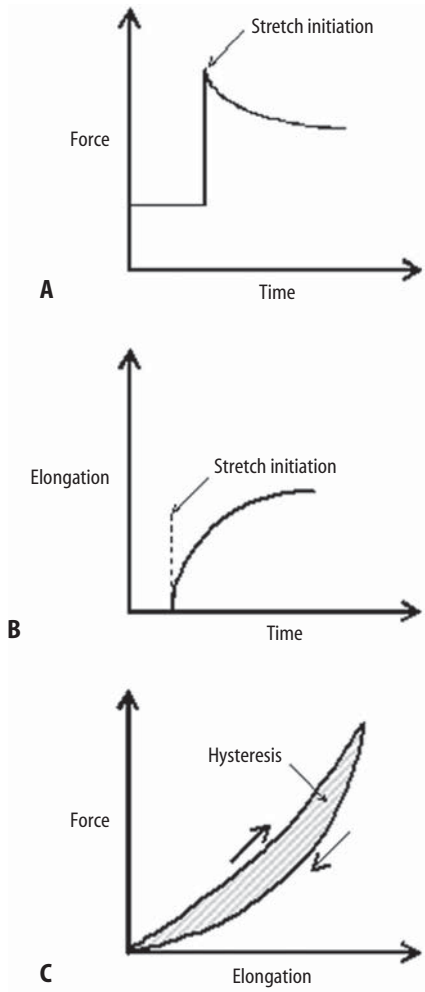

Figure 3.2. Force-relaxation (**A**), creep (**B**), and mechanical hysteresis (**C**). The arrows at the bottom graph indicate loading and unloading directions. In the first few loading–unloading cycles in a mechanical hysteresis test, the tendon resting length increases. This is referred to as "conditioning."

In Vivo Testing

The examination of tendon properties under in vitro conditions necessitates the use of donor specimens, which are not always readily available. Moreover, caution should be placed when using the results of the in vitro test to infer *in vivo* function for the following reasons: (1) The forces exerted by maximal tendon loading under in vivo conditions may not reach the "linear" region where stiffness and Young's modulus are meas-

ured under in vitro conditions. (2) Clamping of an excised specimen in a testing rig is inevitably associated with some collagen fiber slippage and/or stress concentration that may result in premature rupture.[3] (3) In vitro experiments have often been performed using preserved tendons, which may have altered properties.[18,19]

Recently, however, we developed a noninvasive method that circumvents the above problems to assess the mechanical properties of human tendons in vivo.[20–25] The in vivo method allows longitudinal investigations that could address important functional issues relating, for example, to the identification of effective training regimes for enhancing the mechanical properties of a tendon, and the duration of immobilization required to start inducing deterioration in the tendon properties. The in vivo method is based on real-time ultrasound scanning of a reference point along the muscle-tendon unit during an isometric contraction-relaxation (Fig. 3.3). The muscle forces generated by activation are measurable by dynamometry, and pull the tendon, causing a longitudinal deformation. This can be quantified measuring the displacement of the reference landmark on the scans recorded. On relaxation, the tendon recoils and the reference landmark shifts back to its original position (after the tendon has been "conditioned") (Fig. 3.3). The force–elongation plots obtained during loading-unloading can be transformed to the respective stress–strain plots by normalization to the dimensions of the tendon, which can also be measured using noninvasive imaging. Coefficient of variation values of less than 12% have been obtained in repeated measures using the in vivo method.[20–25]

Despite the advantages of testing a tendon in its physiological environment, the in vivo measurement has some inherent unavoidable problems. One inevitable problem relates to the incorporation of heat losses by the tendon–muscle and tendon–bone interfaces and by surface friction between the tendon and adjacent tissues, which would be reflected in the area of the hysteresis loop in the test. More important is the problem of nonhomogeneous stress application across the tendon by increasing or decreasing the intensity of muscle contraction to obtain the relevant force–elongation plot. This limitation would specifically apply to Achilles tendon testing, since this tendon is formed by two separate tendons (the gastrocnemius and soleus tendons) connected with collagenous links, which may allow some intertendon shearing.[26,27] Stress heterogeneity in the Achilles tendon at its calcaneal enthesis has also been considered as a potential factor implicated in chronic Achilles tendinopathy.[28]

Despite these limitations, the general principles of in vivo tendon testing have often been applied with the aim being to characterize the mechanical behavior of the human Achilles/gastrocnemius tendon in different situations and conditions.[22,24–26,29–35] The results obtained vary greatly. In young sedentary adults, for example, maximal tendon force and elongation values of ~200–3800 N and 2–24 mm, respectively, have been reported, with the corresponding stress and strain values being ~20–42 MPa and ~5–8%.[22,24–26,29–35] The tendon stiffness, Young's modulus, and mechanical hysteresis values obtained in the above studies are ~17–760 N/mm, 0.3–1.4 GPa, and ~11–19%, respectively. The immense variation in each mechanical parameter between experiments is most probably caused by interstudy methodological differences in (1) the way that forces are calculated (e.g., incorporation or nonincorporation of synergistic and antagonistic muscles) and (2) the location of the reference landmark traced by ultrasound (i.e., on the tendon, myotendinous junction, or muscle belly).

However, when comparing the human Achilles/gastrocnemius and tibialis anterior tendons of young adults using the same methodology, these two tendons have very similar Young's modulus (1.2 GPa) and mechanical hysteresis (18%) values.[20–23] This finding should be interpreted bearing in mind that the Achilles/gastrocnemius and tibialis anterior tendons are subjected to different physiological forces. The Achilles/gastrocnemius tendon is subjected to the high forces generated in the late stance phase, and the tibialis anterior tendon is subjected to the lower forces generated by controlling plantarflexion in the early stance phase of gait. In vivo measurements of tendon force indicate that the Achilles tendon may carry up to 110 MPa in each stride during running.[36] This stress exceeds the average ultimate tensile tendon stress of 100 MPa,[2,4,5,7,8] which highlights the possibility of Achilles tendon rupture in a single movement in real life.

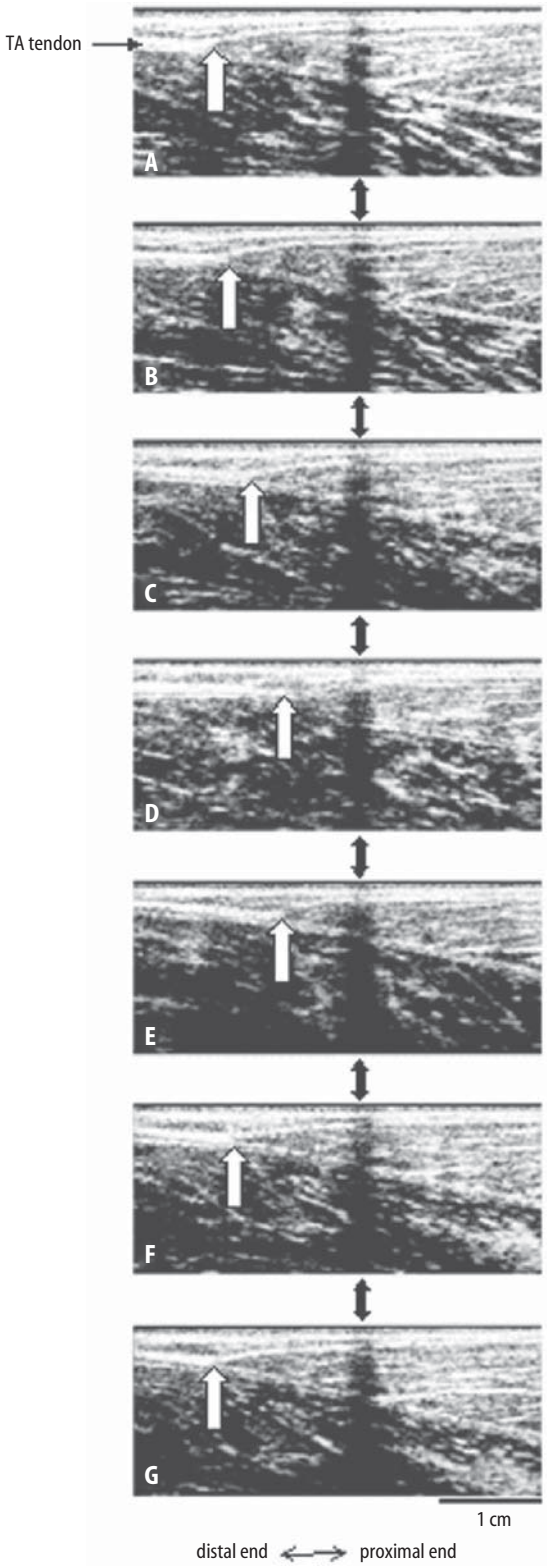

Figure 3.3. Typical in vivo sonographs of the human tibialis anterior (TA) tendon. (**A**) resting state; (**B**) 40% of maximal isometric contraction during activation; (**C**) 80% of maximal isometric contraction during activation; (**D**) 100% of maximal isometric contraction; (**E**) 80% of maximal isometric contraction during relaxation; (**F**) 40% of maximal isometric contraction during relaxation; (**G**) 0% of maximal isometric contraction at the end of relaxation. The white arrow in each scan points to the TA tendon origin. The black double arrows point to the shadow generated by an echo-absorptive marker glued on the skin to identify any displacements of the scanning probe during muscle contraction–relaxation. The tendon origin displacement is larger during relaxation compared with contraction at each loading level, indicating the presence of mechanical hysteresis in the tendon.[21]

Epidemiological studies of spontaneous tendon rupture verify these theoretical considerations.[37] Another difference between the two tendons relates to their ability to provide mechanical work. In contrast to the tibialis anterior tendon, the Achilles/gastrocnemius tendon acts as energy provider during locomotion. Most of the work done on the tendon by the initial ground reaction force is recovered as elastic strain energy during push-off, plantarflexing the ankle, and propelling the body forward at no energetic cost.[38] Notwithstanding the above differences between the two tendons, the Achilles/gastrocnemius tendon is neither intrinsically stiffer or more rebound resilient than the tibialis anterior tendon, in agreement with previous in vitro findings.[9,39] Thus, it seems likely that adjustments in the structural properties of the tendon to differences in physiological loading are accomplished by adding or removing material rather than altering the material intrinsic properties. Recent experimental results on horse tendons, however, indicate that during maturation the material properties of highly stressed tendons may change, with improvements related to increasing levels of a noncollagenous protein named *cartilage oligometric matrix protein* (COMP).[40]

The effect of altered mechanical loading on the mechanical properties of the human Achilles/gastrocnemius tendon in vivo has been examined in several cross-sectional and longitudinal studies. Cross-sectional studies have mainly compared young and older subjects[41,42] and sedentary and athletic subjects.[31] Longitudinal studies have typically employed exercise training over a number of weeks to increase mechanical loading[29,32] and immobilization over a number of weeks to induce disuse.[34,43] Consistent with most in vitro results, most of the above in vivo studies have shown that the human Achilles/gastrocnemius tendon complex becomes stiffer with chronic mechanical loading and more compliant with reduced mechanical unloading. Changes in tendon cross-sectional area may partly account for these effects, but changes in the tendon Young's modulus have also been reported, indicating alterations in the material of the tendon, potentially caused by factors such as changes in glycosaminoglycan content, reducible collagen cross-linking content, and alignment of collagen fibers.[2,5,6,44–46] Gender effects on the behavior of the Achilles/gastrocnemius tendon have also been examined: tendons in males are stiffer and more rebound resilient than tendons in females.[33] This may relate to intergender performance differences.

Functional Implications of the Mechanical Behavior of a Tendon

The tensile viscoelasticity of a tendon has several important functional implications for the in-series muscle.

First, having a muscle attached to a compliant tendon makes it more difficult to control the position of the joint spanned by the tendon.[47] Consider, for example, an external oscillating force applied to a joint at a certain angle. Trying to maintain the joint still would require generating a constant contractile force in the muscle. If the tendon of the muscle is very compliant, its length will change by the external oscillating load, even though the muscle is held at a constant length. This will result in failing to maintain the joint at the angle desired.

Second, the elongation of a tendon during a static muscle contraction is accompanied by an equivalent shortening in the muscle. For a given contractile force, a more extensible tendon will allow the muscle to shorten more. This extra shortening would cause a shortening in the sarcomeres of the muscle. According to the cross-bridge mechanism of contraction,[48] if the sarcomere operates in the ascending limb of the force–length relation, having a more extensible tendon would result in lower contractile force. In contrast, if the sarcomere operates in the descending limb of the force–length relation, having a more extensible tendon would result in greater contractile force. The sarcomeres in the triceps surae muscle operate in the ascending limb of the force–length relation.[49,50] Increases in Achilles tendon length would, therefore, produce a reduction in muscle force. As discussed earlier, "conditioning" is a physiological means to increase the tendon length transiently. In fact, we recently showed that, as a consequence of the increasing elongation of the Achilles/gastrocnemius tendon during conditioning, the gastrocnemius muscle fascicles shorten

by ~12%.[16] Calculations based on the cross-bridge model indicated that the resultant changes in myofilament overlap might reduce the force-generating potential of the muscle by ~10%,[16] a decrease that could be mistaken for evidence of neuromuscular fatigue. In a physiological situation involving repeated loading of the Achilles/gastrocnemius tendon after a period of unloading (e.g., in the first steps taken after awakening in the morning), the extra stretch needed to take up the elongation present could also be obtained by further dorsiflexing the ankle and/or further extending the knee at push-off. Calculations using relevant moment arm values indicate that each of these joint rotations would be ~6 degrees.[17]

Finally, stretching a tendon results in elastic energy storage. Since tendons exhibit low mechanical hysteresis, most of the elastic energy stored during stretching is returned on recoil. This passive mechanism of energy provision operates in tendons in the feet of legged mammals during terrestrial locomotion, thus saving metabolic energy that would otherwise be needed to displace the body ahead.[38] As discussed earlier, energy is also dissipated in the form of heat, but this effect is small and does not endanger the integrity of a tendon in a single stretch–recoil cycle. However, in tendons that stretch and recoil repeatedly under physiological conditions (e.g., the Achilles tendon), the heat lost may result in cumulative tendon thermal damage and injury, predisposing the tendon to ultimately rupture. Indeed, in vivo measurements and modeling-based calculations indicate that highly stressed, spring-like tendons may develop during exercise temperature levels above the 42.5°C threshold for fibroblast viability.[51] These findings are in line with the degenerative lesions often observed in the core of tendons acting as elastic energy stores, indicating that hyperthermia may be involved in the pathophysiology of exercise-induced tendon trauma.

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4
Imaging of the Achilles Tendon

Robert R. Bleakney, Lawrence M. White, and Nicola Maffulli

Imaging plays a critical role in the diagnostic evaluation and assessment of patients with problems at and around the Achilles tendon, both in the documentation and differential assessment of disease as well as in the staging of the extent and severity of disease present. Imaging may additionally provide important information regarding the status of the tendon and surrounding osseous and soft tissue structures following therapeutic intervention, and in some instances may provide prognostic information regarding ultimate tendon function. In this chapter, we review the normal and pathologic imaging features of the Achilles tendon, highlighting the potential utility and limitations of various imaging techniques in the noninvasive assessment of the tendon and the potential impact of imaging findings on clinical patient care.

Conventional radiography is currently the mainstay of bone and joint imaging, particularly in trauma. As it lacks soft tissue contrast, radiography provides limited information regarding the soft tissues. However, conventional radiography is fast, inexpensive, and readily available and may still provide important information regarding the Achilles tendon and adjacent structures.[1] On lateral projection conventional radiographs, the normal margination of the Achilles tendon and adjacent pre-Achilles fat pad (Kager's triangle) is seen as a sharp soft tissue interface along the anterior (volar) margin of the tendon (Fig. 4.1).[2] Rupture of the Achilles tendon, Achilles tendinopathy, or inflammation/hemorrhage within the pre-Achilles fat pad may obscure this sharp interface between the tendon and adjacent fat (Fig. 4.2).[2] These findings may be subtle without the use of specific high-contrast (low-kilovolt) radiographic technique.

Morphologically, the normal Achilles tendon should be no more than 8 mm thick in the anteroposterior (AP) dimension, being thickest proximally and tapering slightly along its distal third to its insertion on the calcaneal tubercle. The normal retrocalcaneal bursa should produce a radiolucency anterior to the distal insertional fibers of the Achilles tendon that extends at least 2 mm below the superior surface of the calcaneus (Fig. 4.1).[3] Bursitis or thickening of the tendon at its calcaneal insertion may obliterate this normal radiolucency on conventional radiography (Fig. 4.3). If adjacent erosions are seen to the posterior calcaneus in the region of the retrocalcaneal bursa, then an underlying inflammatory arthritis, such as rheumatoid arthritis or psoriatic arthritis, with inflammatory bursitis and pannus formation should be considered.[3] Conventional radiography promptly reveals avulsive fractures, calcification, or ossification of the tendon and adjacent soft tissues. Ossification of the Achilles tendon is rare, with an ossific mass contained within the substance of the tendon, usually seen in patients prior to Achilles tendon rupture or chronic Achilles tendinopathy (Fig. 4.4).[4] In contrast, ethesopathic ossification (spur formation) at the calcaneal insertion of the Achilles tendon is a fairly common finding of little clinical significance.

In contrast to conventional radiography, cross-sectional imaging techniques such as ultrasound and MRI have excellent soft tissue contrast imaging capabilities, and have thus become the

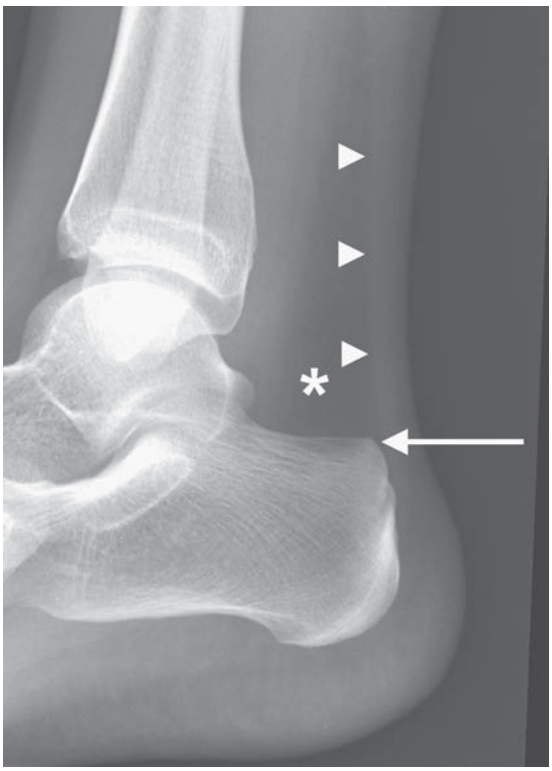

Figure 4.1. Lateral conventional radiograph of a normal ankle demonstrating a well-defined anterior margin of the Achilles tendon (arrowheads), the pre-Achilles/Kager's fat pad (*), and the retrocalcaneal bursal recess (solid arrow).

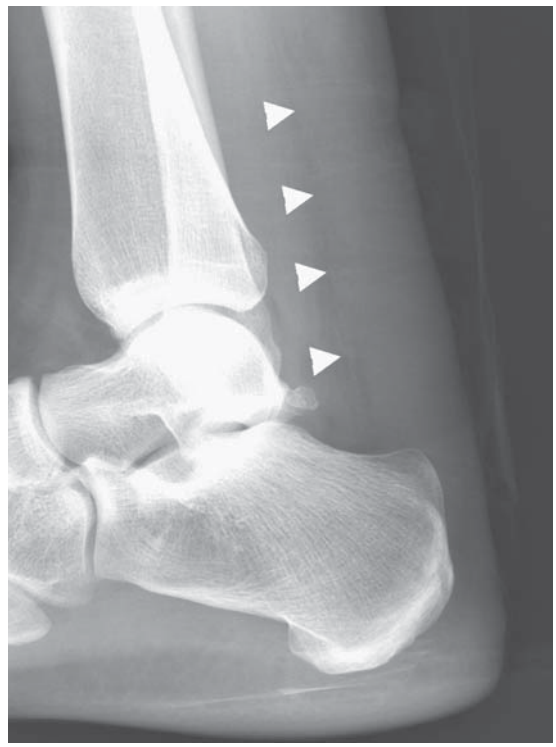

Figure 4.2. Lateral conventional radiograph of an ankle following rupture of the Achilles tendon. There is marked thickening of the Achilles tendon, loss of the normal sharp anterior border (arrowheads), and effacement of the pre-Achilles/Kager's fat pad.

modalities of choice for imaging assessment of the Achilles tendon. Musculoskeletal ultrasound (MSK US) is frequently utilized for assessment of myotendinous disorders, particularly in Europe where its use has been extremely popular and widespread. Constant improvement in technology, with higher frequency transducers (15 MHz), smaller footprint probes, power Doppler (Fig. 4.5), extended field of view capabilities,[5] 3D imaging, and tissue harmonics[6] have all contributed in part to this popularity. In comparison with MR imaging, MSK US has several advantages in assessment of the Achilles tendon: it is readily assessable, has a relatively quick scan time, and has better patient tolerability. MSK US also allows easy contralateral comparison. In addition, the personal interaction with the patient can produce a more patient-directed examination, tailored to the investigation of specific clinical complaints or symptoms. However, MSK US is operator

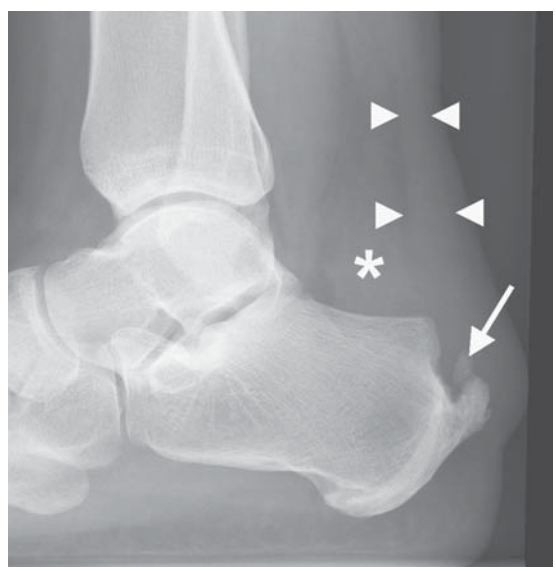

Figure 4.3. Lateral conventional radiograph of an ankle showing a thickened distal Achilles tendon (arrowheads), loss of the normal retrocalcaneal bursal recess, dystrophic insertional ossification (solid arrow), and a retrocalcaneal bursitis, replacing the normal low attenuation fat pad (*).

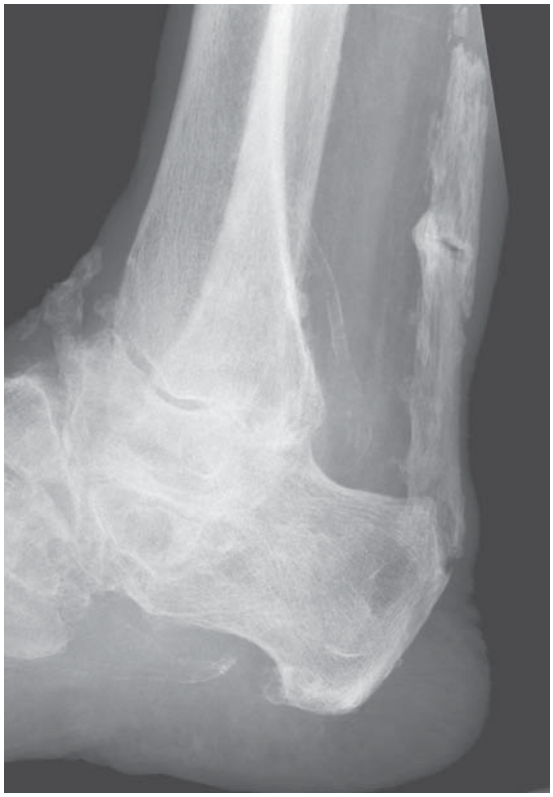

Figure 4.4. Lateral conventional radiograph showing extensive ossification of the Achilles tendon.

dependent, with a long learning curve. Appropriate training and experience are required for accurate and efficient use of this modality in clinical practice. Higher frequency musculoskeletal ultrasound transducers provide better spatial resolution, and thus more detailed delineation of normal and abnormal superficial soft tissues, but are of limited value in the assessment of deeper structures due to poor return of echoes. As a result, MSK US has been increasingly used in the evaluation of the superficial tendon, and in particular the Achilles tendon, in the assessment of tendinopathy and rupture to post-treatment follow-up.[7–18]

For optimal ultrasonographic evaluation, tendons should be interrogated/scanned along both their long and short axes,[9,19] orientating the ultrasound probe so that the ultrasonic waves reach the tendon perpendicularly. The highly ordered pattern of parallel collagen tendon fibers shows the highest echogenicity when examined perpendicular to the ultrasound beam (Fig. 4.6). If this is not the case, the majority of the reflected ultrasonic waves will not be received by the transducer and tendons will appear hypoechoic or anechoic (Fig. 4.7). This angle-dependent appearance of tissue structures is referred to as acoustic fiber anisotropy.[20] Imaging artifacts related to fiber anisotropy can mimic the ultrasonographic appearance of tendon pathology. Awareness of the normal curvature of tendons and proper

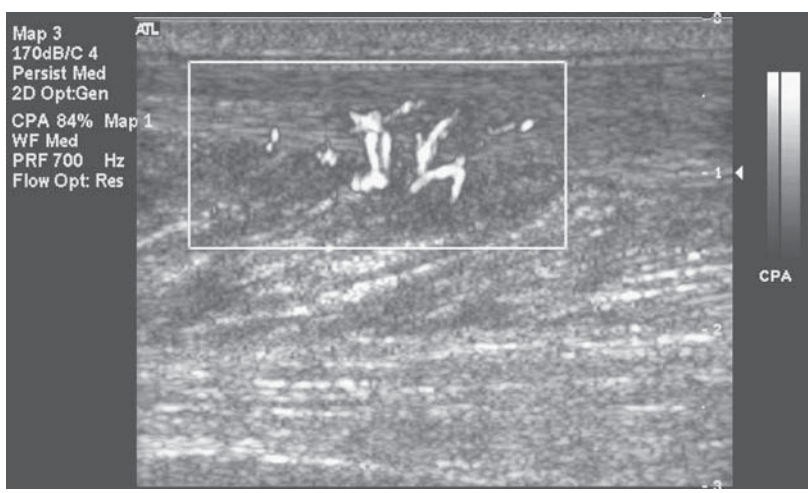

Figure 4.5. Longitudinal ultrasound image of the Achilles tendon with Power Doppler, demonstrating increased vascularity at the musculotendinous junction.

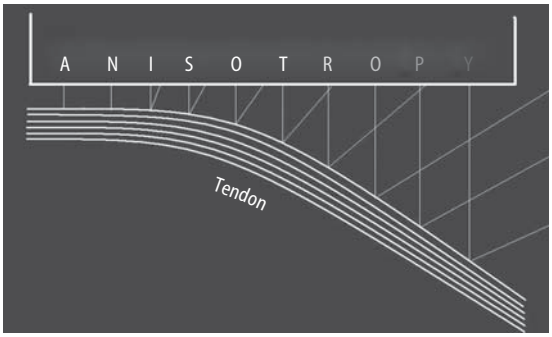

Figure 4.6. Diagrammatic representation of anisotropy. When the ultrasound beam is perpendicular to the tendon, reflected waves return to the transducer. If the tendon is off perpendicular, then the majority of the reflected waves will not be received by the transducer.

ultrasonographic investigation, including dynamic real-time imaging in more than one plane, are essential to avoid this potential imaging pitfall.[21] Except for its insertion on to the calcaneus, the Achilles tendon has a relatively straight course, compared with other ankle tendons, and is thus less susceptible to anisotropy at US evaluation. Nevertheless, careful examination of the Achilles, particularly at the tendon's calcaneal insertion, with cranial and caudal angulation of the probe is necessary to assess the inherent ultrastructural integrity and echogenicity of the tendon.

The normal Achilles tendon has an echogenic pattern of parallel fibrillar lines in the longitudinal plane and an echogenic round-to-ovoid shape in the transverse plane (Fig. 4.8). The number of echogenic lines visible in the tendon with ultrasound is simply a correlate of the ultrasound

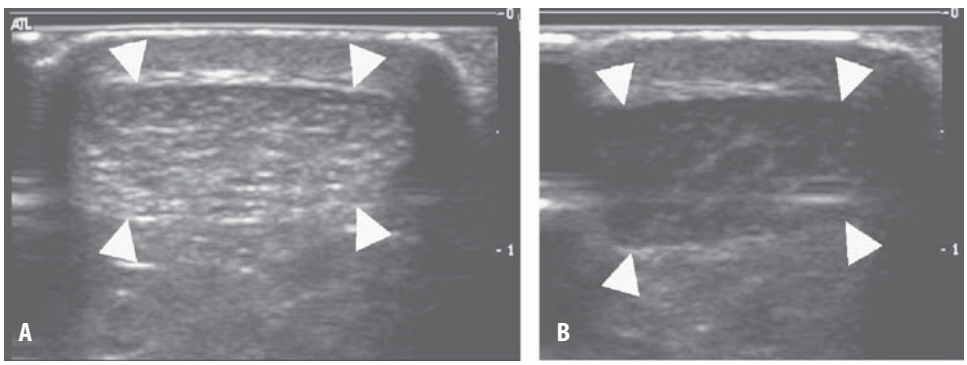

Figure 4.7. Transverse ultrasound images of a normal Achilles tendon. (**A**) Probe orientated at 90° to the tendon demonstrating a normal oval echobright tendon (arrowheads). (**B**) Probe angled off perpendicular to the tendon demonstrating a hypoechoic (dark) tendon (arrowheads).

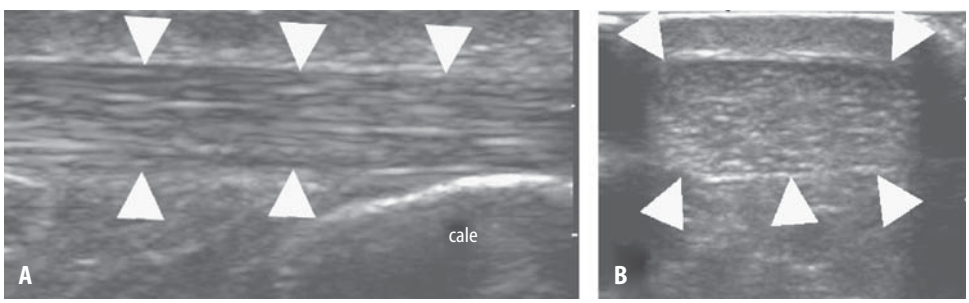

Figure 4.8. (**A**) Longitudinal ultrasonographic image of a normal Achilles tendon. Note the echogenic, parallel fibrillar pattern (between arrowheads). (**B**) Transverse ultrasonographic image of a normal Achilles tendon showing echogenic ovoid shape (arrowheads).

probe frequency.[22] On transverse imaging, the normal Achilles tendon has a flat to concave anterior surface and measures 4–6 mm in anterior to posterior (AP) diameter.[8,12,23–25] While 6 mm is generally accepted as the upper limit of normal for AP dimension, the measurement can be somewhat variable due to anatomical variation in the shape of the Achilles tendon.[25] Proximal to its calcaneal insertion, the Achilles tendon lies immediately superficial to the pre-Achilles fat pad, a triangular area of adipose tissue known as Kager's triangle. At ultrasound imaging, the pre-Achilles fat pad shows low mottled echogenicity relative to the normally echogenic tendon. Further anterior to this pre-Achilles fat pad is the deep flexor compartment of the calf, predominantly composed of the flexor hallucis longus muscle, which overlies the echobright acoustical interface of the posterior tibial and talar cortices (Fig. 4.9). Two bursae can be present around the insertion of the Achilles tendon onto the calcaneus. Both are well delineated at ultrasonography. The pre-Achilles bursa, also referred to as the retrocalcaneal bursa, lies deep to the Achilles tendon between the Achilles tendon and the subjacent calcaneus. This bursa is commonly seen in normal subjects, and may vary considerably in appearance and relative dimensions with flexion and extension of the ankle.[15] The superficial tendo-Achilles bursa or retro-Achilles bursa is an acquired bursa occurring in the potential soft tissue interval superficial to the distal tendon between it and the dorsal subcutaneous tissues. This bursa is not seen in normal individuals, and its presence is typically post-traumatic or inflammatory in etiology.[23]

Tendon thickening and hypoechogenicity are the most common abnormalities encountered in clinical ultrasonographic assessment of the Achilles tendon. Focal or diffuse thickening of the Achilles tendon is most commonly seen in

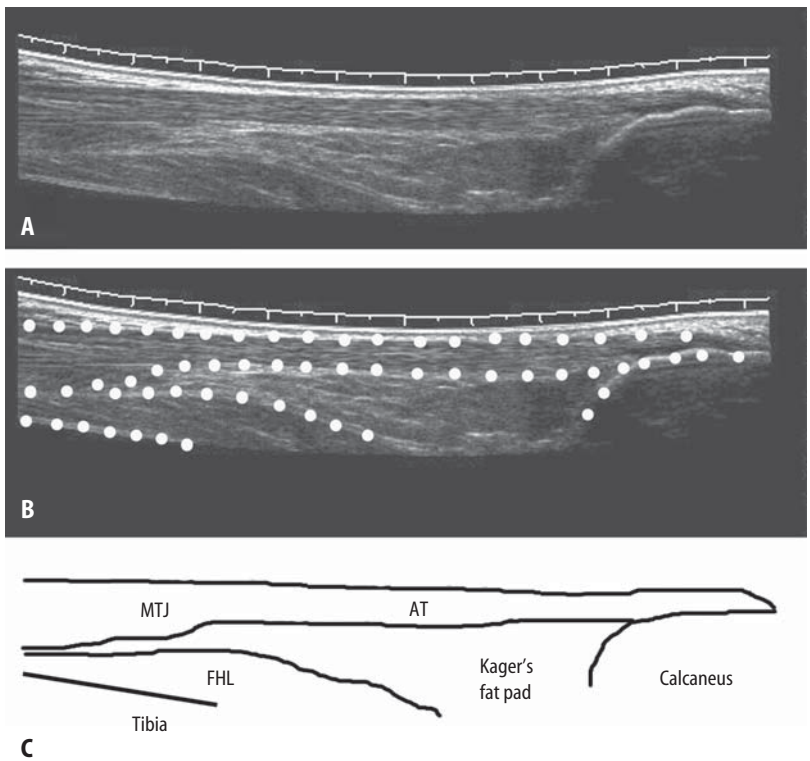

Figure 4.9. (**A**) Longitudinal ultrasonographic image of the Achilles tendon using extended field of view, from its soleal musculotendinous junction (MTJ) to its insertion on to the calcaneus. (**B**) Soft tissue interfaces marked by dotted line. (**C**) Line diagram of the same field of view. Deep to the Achilles tendon (AT) are the flexor hallucis longis (FHL), the echogenic (bright) tibial cortex, and Kager's fat pad.

association with tendinopathy (Fig. 4.10). Prior investigations have documented tendon thickening ranging from 7 to 16 mm in patients with a clinical diagnosis of tendinopathy.[8] Similarly, in athletes with a clinical diagnosis of Achilles tendinopathy, affected tendons were on average 78% thicker than the contralateral unaffected tendon.[14] Focal hypoechoic areas within the normally echobright tendon represent areas of tendinopathic lesions.[10,26] Many of the so-called spontaneous tendon ruptures are due to progressive degeneration of the tendon.[27] Thickening of the tendon and focal hypoechoic areas are seen with both tendinopathy and partial tearing, thus making the differentiation between the two difficult.[1,26,28] However, Åström proposed that thickening of the tendon >10 mm and severe intratendinous abnormalities indirectly suggested partial rupture.[26] However, true partial tears are rare at surgery, and potential differentiating imaging features can at times be misleading.

In contrast to potential difficulties encountered in the differential evaluation of partial tears versus tendinopathy, ultrasound is highly accurate in the diagnosis of full thickness tears of the Achilles tendon.[11,28] Paavola et al. correctly diagnosed 25 of 26 full thickness tears before surgery,[28] and Hartgerink et al. showed that ultrasound can be effective in the differentiation of full versus partial thickness tears or tendinopathy, with a sensitivity and specificity of 100% and 83%, respectively, and an accuracy of 92%.[11] Undectable tendon at the site of injury, tendon retraction, and posterior acoustic shadowing at the site of a tendon tear have been described as ultrasonographic signs of a full thickness tear (Fig. 4.11).[11,12] Posterior acoustic shadowing deep to the torn tendon margins is thought to occur secondary to sound beam refraction by frayed/torn tendon ends.[12] A potential pitfall in the ultrasound evaluation of a complete full thickness tear of the Achilles tendon is visualization of an intact plantaris tendon medial to

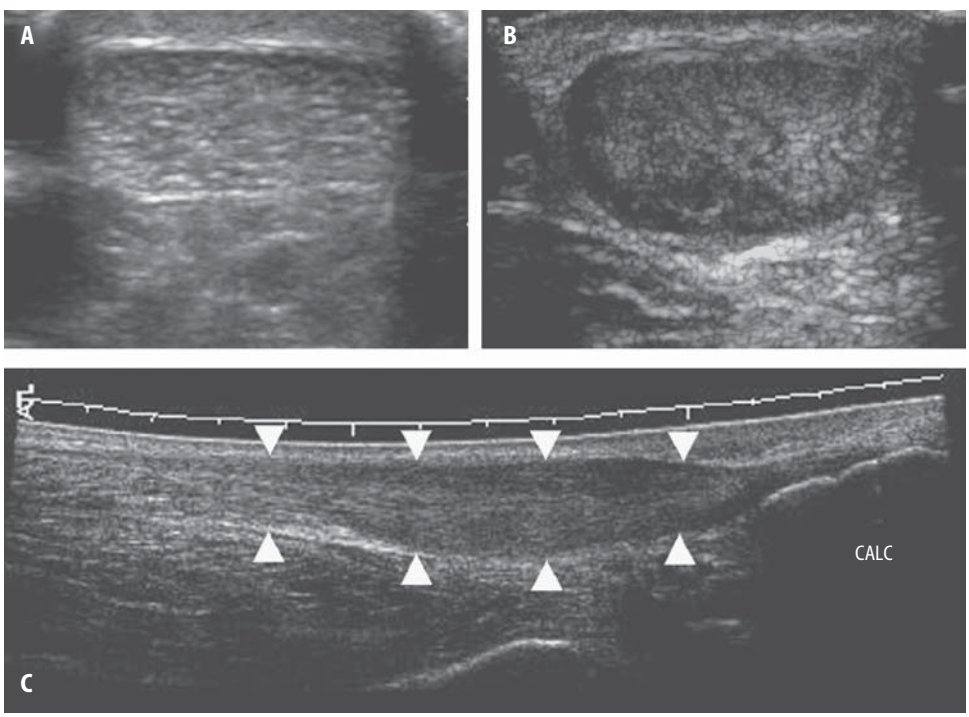

Figure 4.10. (**A**) Normal transverse ultrasonographic image of the Achilles tendon. (**B**) Transverse ultrasonographic image of an Achilles tendinopathy. The tendon is thickened, heterogeneous, and hypoechoic. (**C**) Longitudinal extended field of view ultrasonographic image of Achilles tendinopathy demonstrating fusiform thickening of the Achilles tendon (arrowheads).

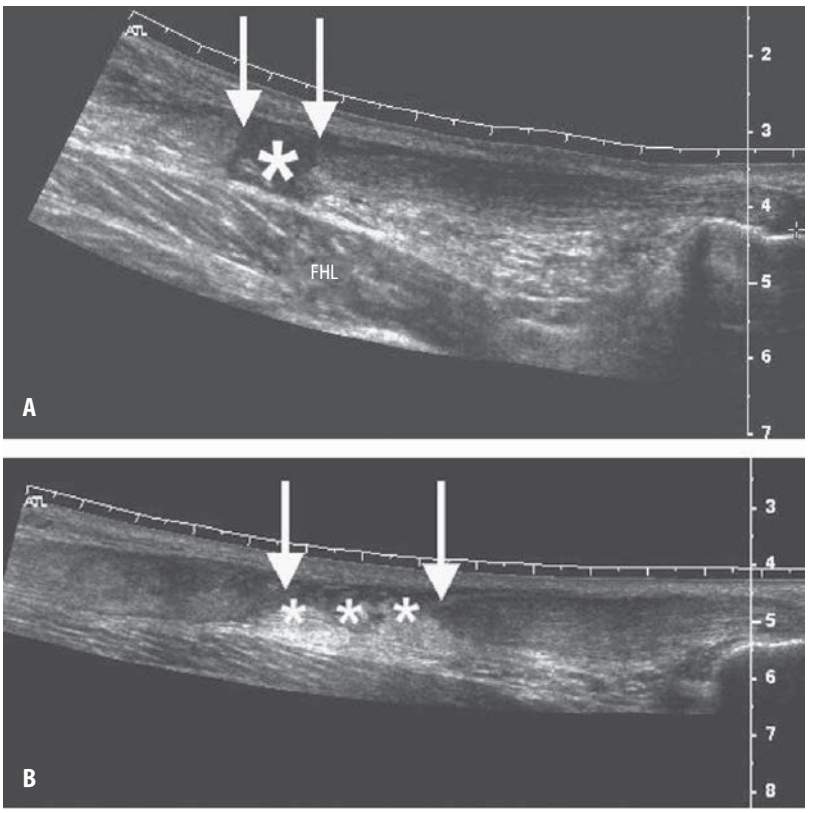

Figure 4.11. (**A**) Longitudinal extended field of view ultrasonographic image of a complete tendon tear showing the gap in the tendon (*) and the torn tendon ends (solid arrows). The muscle belly of flexor hallucis longus is well seen deep to the tear. (**B**) Longitudinal extended field of view ultrasonographic image of a complete tendon tear. Note more retraction, compared to case A, with a larger gap in the tendon, torn tendon ends (solid arrows), and echogenic (bright) fat herniating into the tendon gap (*).

the torn fibers of the Achilles (Fig. 4.12). The normal plantaris tendon may be mistaken for residual intact Achilles tendon fibers, and can lead to a false diagnosis of a high-grade partial tear rather than a complete tendon tear.[11,29] Dynamic ultrasound assessment of a complete Achilles tendon rupture can further reveal whether the retracted torn tendon ends can be approximated to one another on plantar flexion. This may be of use when deciding between conservative versus surgical treatment.

Following successful management of Achilles tendon rupture and tendinopathy, tendon abnormalities can persist on ultrasound despite resolution of patient symptoms. Following conservatively or surgically treated Achilles tendon ruptures, the tendon can continue to appear thickened and irregular with focal hypoechoic areas at ultrasound evaluation.[8,16,30–32] Rupp et al. tried to correlate long-term clinical outcome after surgery for Achilles tendon rupture with ultrasound morphology of the tendon, but found that, while ultrasound is able to reveal long-lasting changes of the morphology of the tendon, it was of only limited value in evaluation of the functional result.[16,32] Calcifications may also occur at the site of a prior tear: they are seen as hyperechoic areas casting acoustic shadowing (Fig. 4.13).[8,30,31] Despite the ability of ultrasound to accurately depict structural abnormalities of the Achilles tendon, only moderate correlation exists between ultrasound appearance and clinical assessment of chronic Achilles tendinopathy.[33] In addition, Khan and co-workers showed that the baseline ultrasound appearance of the tendon, in the setting of chronic tendinopathy, was a poor predictor of subsequent

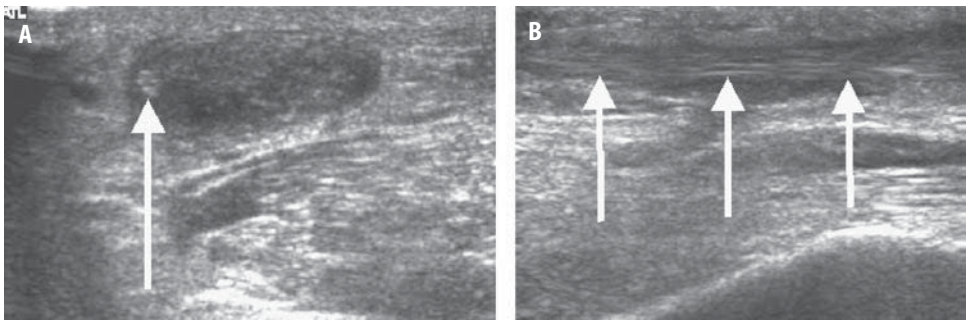

Figure 4.12. Transverse (**A**) and longitudinal (**B**) ultrasonographic images of a full thickness tear of the Achilles tendon, demonstrating an intact echogenic plantaris tendon (solid arrows).

clinical outcome.[33] Conversely, other investigators have shown that tendon inhomogeneity can be used to predict clinical outcome in painful Achilles tendons.[34,35] Nehrer et al. additionally reported that patients with a clinical diagnosis of Achilles tendinopathy, with a normal ultrasound appearance of the Achilles tendon, had a significantly better clinical outcome, compared to individuals with abnormal findings at ultrasound.[36] They also documented that patients with tendon thickening and focal hypoechoic areas had higher rates of subsequent spontaneous tendon rupture.[36]

The recent addition of color and power Doppler imaging to ultrasound has allowed for the noninvasive study of blood flow and vascularity within and surrounding the Achilles tendon (Fig. 4.5). In patellar tendinopathy, color Doppler has demonstrated increased vascularity in the setting of an abnormal tendon, suggesting neovascularization.[37] Zanetti et al. demonstrated that the presence of neovascularization is a relatively specific sign for a clinically painful tendon. However, the presence of neovascularization did not affect the patient's outcome adversely.[35]

The multiplanar imaging capabilities of MRI combined with its excellent soft tissue contrast characteristics make it ideally suited for imaging of the Achilles tendon. Sagittal and axial planes are most useful in the evaluation of the Achilles tendon commonly using a combination of T1 and T2 weighted imaging sequences.[38,39] In general, T1 or intermediate weighted sequences provide optimal delineation of anatomic detail, and T2 weighted sequences are most sensitive to the abnormal increase in fluid signal that accompanies most pathological conditions of the tendon.[40] Short tau inversion recovery (STIR) and fat satu-

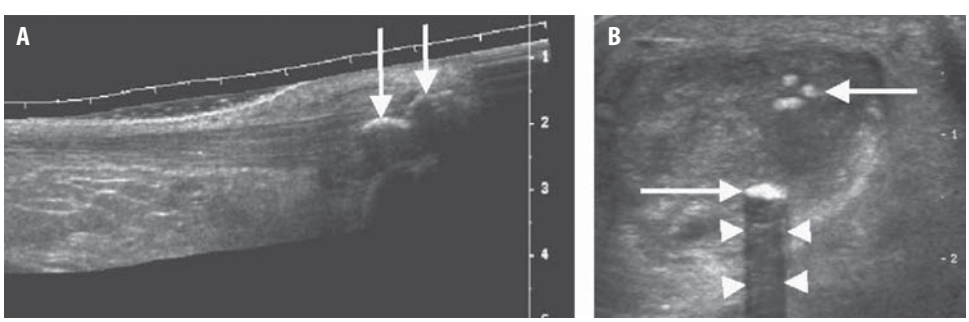

Figure 4.13. (**A**) Longitudinal extended field of view ultrasonographic image of an insertional Achilles tendinosis with echogenic (bright) calcification (solid arrows) in the thickened distal Achilles tendon. (**B**) Transverse ultrasonographic image of a previously torn Achilles tendon. There are focal echogenic (bright) areas of calcification (solid arrow) with posterior acoustic shadowing (arrowheads). The Achilles tendon is thickened, hypoechoic, and heterogeneous.

ration T2 weighted sequences may additionally serve to increase signal contrast between free water and the surrounding fat and adjacent tendon.

On sagittal MR images, the anterior and posterior aspects of the normal Achilles tendon should be parallel to one another distal to the soleus insertion (see reference 43). The normal average AP dimension of the Achilles tendon on MRI is 6 mm.[25] At axial imaging, the anterior aspect of the tendon should be flat to concave (Fig. 4.14). The length of the Achilles tendon is variable, ranging from 20 to 120 mm between the musculotendinous origin and the calcaneal insertion of the tendon.[41] The presence of an accessory soleus muscle produces an apparently shorter Achilles tendon, as the accessory soleus may have an insertion directly onto the anterior margin of the Achilles tendon, mimicking a more distal musculotendinous origin (Fig. 4.15).[42] The normal Achilles tendon is of low signal (black) on all MR imaging sequences, reflecting its ultrastructural composition of compact parallel arrangements of collagen fibers and its low intrinsic water content (Fig. 4.16).[25,40,43] However, the magic angle phenomenon can produce areas of increased signal within normal tendons, observed as tendon fibers approach an orientation angle of 55° relative to the main magnetic field.[44,45] While the Achilles has a relatively straight course, this effect can occur as

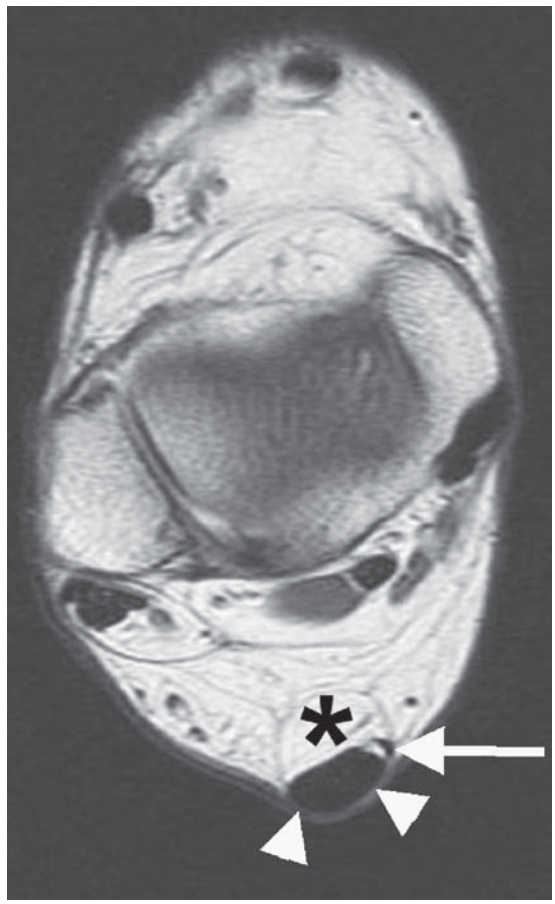

Figure 4.14. Axial T2 weighted sequence of a normal ankle, demonstrating a normal Achilles tendon (arrowheads), adjacent plantaris tendon (solid arrow), and Kager's fat pad (*).

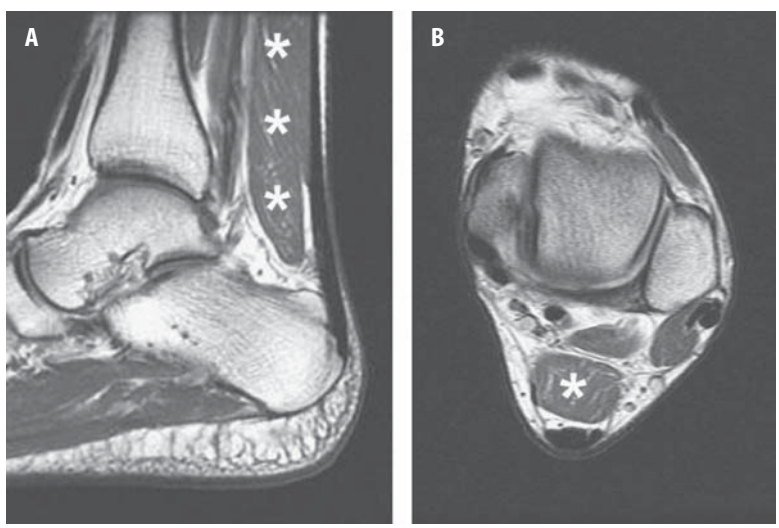

Figure 4.15. Sagittal T1 weighted image (**A**) and axial T2 weighted image (**B**) of the ankle showing an accessory soleus (*).

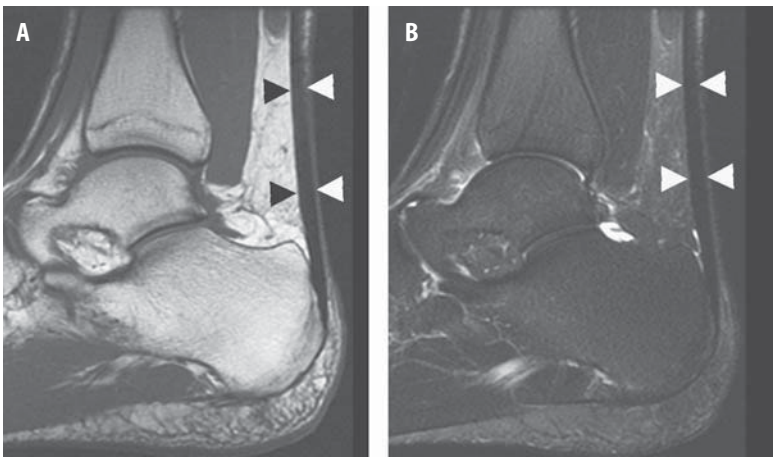

Figure 4.16. Sagittal T1 (**A**) and T2 with fat saturation (**B**) images, of a normal Achilles tendon. Note the parallel anterior and posterior tendon surfaces (arrowheads) and its low signal (black) appearance on both sequences.

the Achilles fibers spiral internally.[43] The magic angle effect is usually seen with echo times of less than 20 msec (e.g., T1 weighted and proton density/intermediate acquisitions), but the effect should not be present on T2 weighted (long echo time) acquisitions.[45] Recently MR magic angle imaging has been used to advantage in imaging of the Achilles tendon. With this technique the Achilles tendon is imaged at 55° relative to the main magnetic field rather than at the usual 0°. In this way, signal related to the magic angle phenomenon becomes detectable within the tendon.[46–48] Using this method, intratendinous STIR signal change was more apparent, and contrast enhancement was much more evident within the tendon.[47]

As with ultrasonographic assessment, a pre-Achilles/retrocalcaneal bursa can be seen in asymptomatic individuals at MR evaluation. The bursa normally measures up to 6 mm craniocaudally, 3 mm medial to lateral, and 2 mm anterior to posterior.[49] Achilles paratendinopathy manifests as linear or reticular areas of increased signal on T2 weighted images, paralleling the deep margin of the Achilles tendon, representing areas of edema or increased vascularity. However, both ultrasound and MRI evaluation are unreliable in the assessment of the paratenon.[26]

Classic MR imaging features of Achilles tendinopathy include morphologic findings of a fusiform tendon shape, AP tendon thickening, convex bulging of the anterior tendon margin, and areas of increased intratendinous signal on T1 and T2 weighted sequences (Fig. 4.17).[43,50] Areas of increased signal within the tendon on T2 weighted sequences are thought to represent more severe areas of collagen disruption[50,51] and partial tearing.[26] AP thickening of the tendon greater than 10 mm correlates with pathological findings of partial tendon tearing.[26]

Full thickness tearing of the Achilles tendon is manifest on MR imaging as complete disruption of the tendon fibers with tendon discontinuity and high signal intensity within the tendon gap acutely on T2 weighted images (Fig. 4.18).[52] MR imaging with plantar flexion, when feasible, may provide additional information regarding separation of the torn tendon ends and potential apposition of the tear margins. Potential diagnostic difficulties may arise in the MR imaging differentiation of chronic tendinopathic changes with a complete non-retracted tendon tear from cases of chronic tendinopathy with partial tendon tearing. As with ultrasound examination, MRI following Achilles tendon repair typically illustrates tendon thickening and intrasubstance imaging heterogeneity (Fig. 4.19).[32,51,52] A decrease in intrasubstance signal, together with an increased tendon size, may gradually progress over one to two years after surgery, possibly

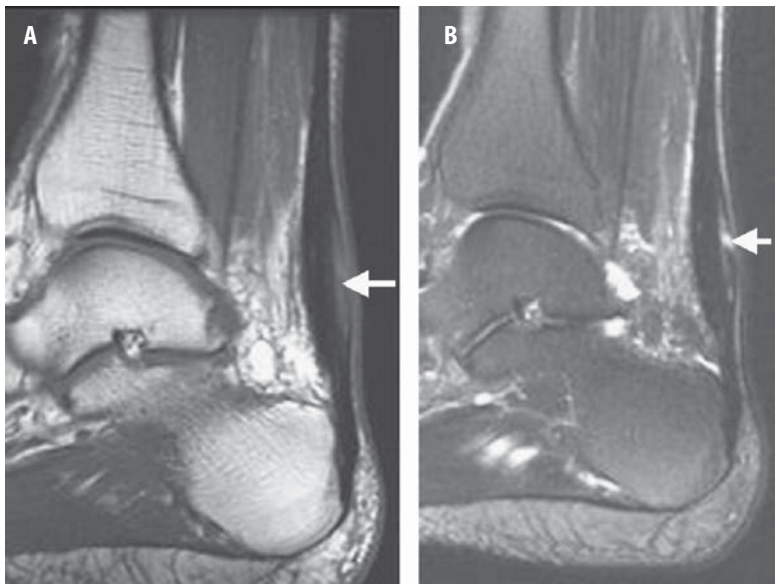

Figure 4.17. Sagittal T1 (**A**) and sagittal T2 with fat saturation (**B**) images showing fusiform thickening of the Achilles tendon and abnormal intratendinous signal dorsally (solid arrows).

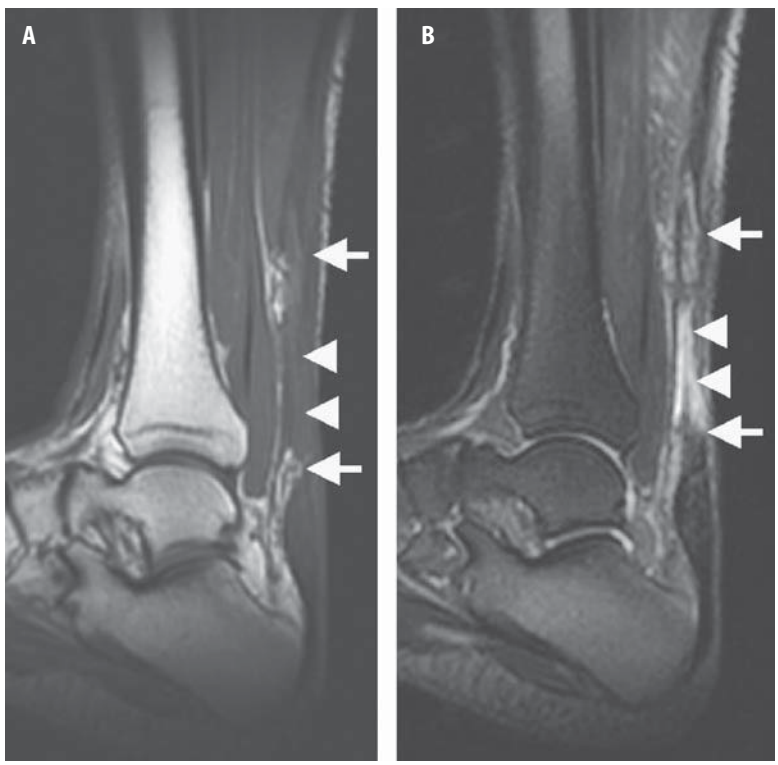

Figure 4.18. Sagittal T1 (**A**) and sagittal T2 with fat saturation (**B**) weighted images in a patient with an Achilles tendon rupture. Note discontinuity of fibers, high signal within the tendon gap (arrowheads), and the torn tendon ends (solid arrows).

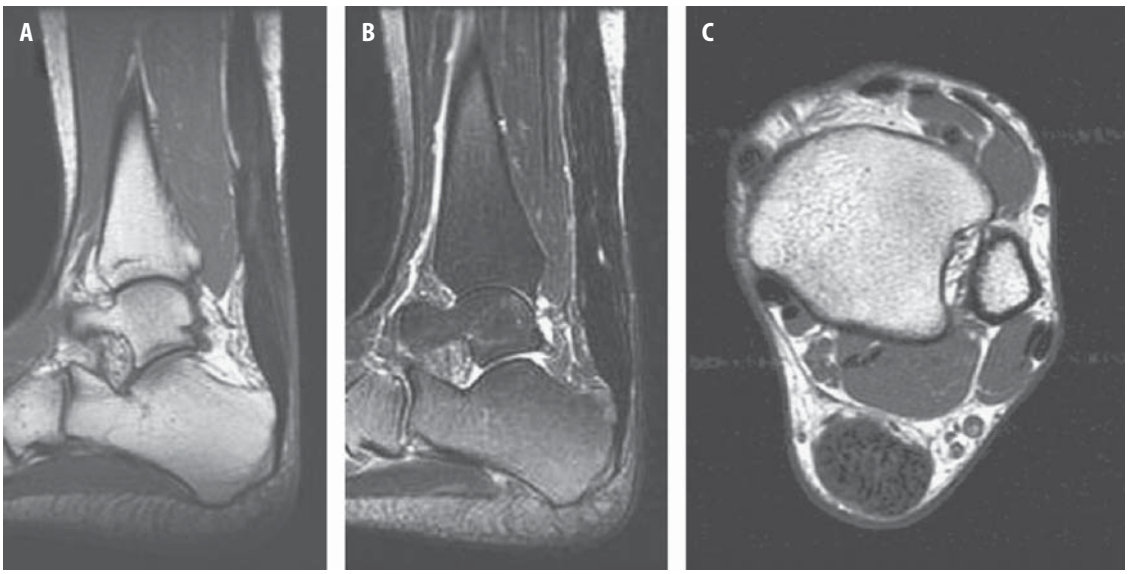

Figure 4.19. Sagittal T1 weighted image (**A**), sagittal T2 weighted image with fat saturation (**B**) and axial T2 weighted image (**C**), in a patient with prior Achilles tendon full thickness tear. Note persistent marked thickening of the Achilles tendon as well as abnormal intratendinous signal.

reflecting progressive scar maturation at the repair site.

Despite the acknowledged benefits and widespread use of MR imaging in the assessment of the Achilles tendon, there is some debate as to the utility of MRI in examination in this regard. Karjalainen et al. examined 117 Achilles tendons with MRI and documented the overall sensitivity of MR imaging in the detection of abnormalities in cases of painful Achilles tendon to be 94%, with a specificity of 81%, and overall accuracy of 89%. The interobserver agreement for the MR imaging findings evaluated was good in all categories.[50] However, several authors demonstrated an overlap of imaging findings in symptomatic and asymptomatic individuals.[41,53] Signal heterogeneity and subtle focal increases in intrasubstance signal with distal longitudinal striations or small punctate foci of increased T1 weighted signal may represent normal fascial anatomy or possibly small vessels in normal Achilles tendons.[41] This fascicular signal should be less apparent on STIR/T2 weighted images and the tendon should maintain a normal morphologic appearance with a concave anterior surface on axial imaging.[54] Areas of mild increased T2 weighted signal visualized within the Achilles tendon in asymptomatic patients have been described and postulated by Haims and coworkers to represent areas of subclinical tendinopathy/mucoid degeneration. In contrast, areas of intense T2 weighted signal and thickened tendons were associated with chronic symptoms and tendon tears.[53] Unlike ultrasound, Khan et al. did show that graded MRI did correlate with 12-month clinical outcome in patients with tendinopathy.[33]

In conclusion, the Achilles tendon is the most commonly injured tendon in the foot and ankle, with injuries commonly related to sports/athletic activities. Imaging modalities most commonly employed in the diagnostic assessment of the Achilles include conventional radiography, ultrasonography, and magnetic resonance imaging. Cross-sectional imaging, including ultrasound and MR imaging, plays an important role in the documentation and staging of disease of the Achilles, and provides a noninvasive means of assessing the tendon's response to therapy or progression of disease.

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5
Etiology and Epidemiology of Achilles Tendon Problems

Jonathan S. Young and Nicola Maffulli

Introduction

Rupture of the Achilles tendon is on the increase,[1,2] and appears more common in men in the white-collar professions.[3,4] Various hypotheses have been put forward as to why the Achilles tendon ruptures.[3,5–8] Tendinopathy of the Achilles tendon is common both in athletic and nonathletic individuals.[9–12] As in ruptures of the Achilles tendon, its etiology is likely to be multifactorial.[13,14]

Acute Ruptures

Two main theories are advocated, the "degeneration theory" and the "mechanical theory." According to the degeneration theory, chronic degeneration of the tendon leads to a rupture without excessive loads being applied. Degenerative changes can result from several factors, including age-related alterations in the tendon, chronic overloading with microtrauma, drug treatment, and in association with other diseases.

Kannus and Jozsa[5] evaluated specimens obtained from the biopsy of spontaneously ruptured tendons in 891 cases; 397 of these ruptures were of the Achilles tendon. They compared the histopathology of the 397 Achilles tendon ruptures with 220 control tendons using conventional and polarized light microscopy, and also scanning and transmission electron microscopy. The control specimens were age- and sex-matched for 445 tendons taken at the time of death from the cadavers of previously healthy individuals who died accidentally. A healthy structure was not seen in any spontaneously ruptured tendon, whereas two-thirds of the control tendons were structurally healthy. There were characteristic histopathological patterns in the spontaneously ruptured tendons. Most (97%) of the pathological changes were degenerative, with hypoxic (45%), mucoid (19%), tendolipomatous (6%), and calcifying tendinopathy (3%), either alone or in combination. These changes were also found in 31% of the control tendons. The findings indicated that, at least in an urban population, degenerative changes are common in the tendons of subjects older than 35 years and that these changes are associated with spontaneous rupture.[5]

Disruption of the homeostasis of extracellular matrix components such as the fibrillar collagens and the proteoglycans may predispose to rupture. Type I collagen comprises 95% of collagen in normal tendons. This parallel arrangement imparts high tensile strength to the tendon. Maffulli et al.[6] showed that Type I collagen is the main collagen in normal tendons with type III collagen being present in small amounts. However, in ruptured Achilles tendons there was a significantly greater proportion of type III collagen, which is less resistant to tensile forces and may predispose the tendon to spontaneous rupture.[6] An in vitro model was used to determine whether tenocytes from Achilles tendons that are ruptured, nonruptured, tendinopathic, and fetal exhibit different behavior. Samples of Achilles tendon were digested with collagenase and the released tenocytes were collected. Primary tenocyte cultures were established and subsequently cultured onto

glass coverslips. Once a confluent monolayer was obtained, the cell populations were "wounded" by scraping a pipette tip along the surface. The cultures were further incubated for either 1, 4, 8, 12, 16, or 24 hours, and production of the collagen types was assessed by immunostaining. Cultures from ruptured and tendinopathic tendons showed increased production of type III collagen.

Athletic participation places excess stress on the Achilles tendon, which could potentially lead to areas of localized microtrauma within the tendon. These areas may heal by the production of type III collagen, an abnormal healing response. Accumulation of such episodes of microtrauma could result in a critical point where the resistance of the tissue to tensile forces is compromised, and tendon rupture occurs.[6] This leads to the conclusion that ruptured tendons produce and exhibit significantly greater proportions of type III and reduced type I collagen, together with significantly higher degrees of degeneration, than nonruptured tendons.

Birk and Mayne[15] investigated the localization of collagen types I, III, and V during different stages of tendon development. The tendon fascicles and their connective tissue investments (endotendenium) were studied. The data show a changing pattern of type III collagen expression in the developing tendon. The increases in diameter are associated with a decrease in type III collagen reactivity. During all stages of tendon development there is a constant, small but detectable amount of type V collagen. However, no correlation between type V reactivity and fibril diameter was observed at any stage of development. These results indicate an inverse relationship between type III collagen reactivity, and fibril diameter in the developing tendon.[15]

Magnusson et al.[7] tested the hypothesis that collagen fibril diameter and crimp angle in ruptured human Achilles tendons differed from that of intact ones. Although crimp morphology is unchanged, there appears to be a site-specific loss of larger fibrils in the core and periphery of the Achilles tendon rupture site. Moreover, the lack of symptoms prior to the rupture suggests that clinical tendinopathy is not an etiological factor in complete tendon ruptures.[7]

The role of versican, the principal large proteoglycan expressed mid-tendon (with regard to tendon pathology), has recently been investigated. Corps et al. attempted to define the expression of versican isoform splice variant messenger ribonucleic acid (mRNA) in normal Achilles tendons, in chronic painful tendinopathy, and in ruptured tendons.[16] Changes in versican expression relative to that of collagen observed in ruptured Achilles tendons may have contributed to the changes in matrix structure and function and therefore contributed to the etiology of the rupture.

Decorin is a prototype member of the family of the small leucine-rich proteoglycans (SLRPs). It plays a significant role in tissue development and assembly, as well as playing both direct and indirect signaling roles. It modulates collagen fibrillogenesis, and is a vital player in maintaining tendon integrity at the molecular level.[17] The glycosaminoglycans bound to decorin act as bridges between contiguous fibrils, connecting adjacent fibrils every 64–68 nm. This architectural arrangement suggests a possible role in providing mechanical integrity of the tendon structure. Laboratory evidence suggests that fluoroquinolone antibiotics decrease decorin transcription,[18] which may alter the viscoelastic properties of the tendons and induce increased fragility. Bernard-Beaubois suggested that perfloxacin, a fluoroquinolone, does not affect transcription of type I collagen, but decreases the transcription of decorin at a concentration of only 10^{-4} millimoles.[18] The resulting decrease in decorin may modify the architecture of the tendon, leading to altered biomechanical properties and increased fragility.

Clinical painful tendinopathy is not common before complete Achilles tendon ruptures.[7] Most patients who sustain an acute Achilles tendon rupture are asymptomatic prior to injury. Nine (5%) of the 176 patients presenting with a rupture of the Achilles tendon in Aberdeen, Scotland, between January 1990 and December 1995 had had previous symptoms.[3] However, Nestorson et al. reported that among 25 Achilles tendon rupture patients over 65 years age, 11 (44%) had had Achilles tendon symptoms, and 7 of those had received local cortisone injections.[19] Although clinical painful tendinopathy seemingly is a risk factor, patients with chronic Achilles tendinopathy in reported nonsurgical and surgical series have had a long duration of symptoms (several months or years) without sustaining a rupture.[20,21]

On the other hand, at the time of Achilles tendon rupture, degeneration and necrosis were present in 47 of 50 and 42 of 50 of the contralateral asymptomatic Achilles tendons, respectively. Spontaneous rupture of the Achilles tendon seems to be preceded by widespread, bilateral tendon damage.[22] Additionally, the patients' asymptomatic contralateral Achilles tendons showed a greater prevalence of intratendinous alterations at ultrasonography.[23] Simultaneous bilateral ruptures without preceding factors are very rare.[24] However, ruptures of both Achilles tendons at different times have been reported in up to 6% of the patients with no preceding factor.[25]

Achilles tendon rupture may result from a drug adverse event. Fluoroquinolone antibiotics such as ciprofloxacin have been implicated in the etiology of rupture of the Achilles tendon during the last decade.[8,26] In France, between 1985 and 1992, 100 patients treated with fluoroquinolones had tendon disorders that included 31 ruptures.[26] Szarfman et al. demonstrated disruption of the extracellular matrix of cartilage as well as depletion of collagen in animals that received fluoroquinolones.[27] This may also apply to humans.

Local and systemic corticosteroids are administered for a variety of diseases and have been widely implicated as a risk factor for tendon rupture.[28] Both oral and peritendinous steroid injections have been associated with Achilles tendon rupture.[29,30] Balasubramaniam and Prathap injected hydrocortisone into the calcaneal tendons of rabbits, thus inducing necrosis at the site of injection 45 minutes after the injection.[31] Tendons injected with corticosteroid had a delayed healing response compared with those injected with saline solution. Newnham et al. reported a series of 10 patients attending a respiratory outpatient clinic taking oral corticosteroids, who subsequently ruptured their Achilles tendon in the course of 12 years.[29]

There has recently been some evidence to support peritendinous steroid injections, providing the needle does not pass directly into the Achilles tendon body. Gill et al., in a retrospective cohort study of 83 patients,[32] established the safety of low-volume injections of corticosteroids for the management of Achilles tendinopathy when the needle is carefully inserted into the peritendinous space under direct fluoroscopic visualization. In this study, although 23 patients (53%) did not report any improvement, only 3 patients (7%) felt that their condition was any worse. Alfredson found normal prostaglandin E2 (PGE2) levels in chronic painful tendinosis (Achilles and patellar) tendons, showing that there is no PGE2-mediated intratendinous inflammation in the chronic stage of these conditions.[33] The neurotransmitter glutamate (a potent modulator of pain in the central nervous system) was, for the first time, found in human tendons. Microdialysis showed significantly higher glutamate levels in chronic painful tendinosis (Achilles and patellar) tendons, compared with pain-free normal control tendons. A specially designed treatment, using ultrasound and Doppler-guided injections of the sclerosing agent Polidocanol, targeting the neovessels outside the tendon, has been shown to cure tendon pain in pilot studies in a majority of the patients. Many authors feel injections of corticosteroids in patients with established Achilles tendinopathy are to be avoided.[34] Speed reviewed the literature on corticosteroid injections involving tendinopathies and concluded that there was no good evidence to support corticosteroid injections.[35] It is possible that the anti-inflammatory and analgesic properties of corticosteroids may mask the symptoms of tendon damage, and individuals will continue to maintain high levels of activity even when the tendon is damaged.[36]

Spontaneous rupture of the Achilles tendon has been associated with many disorders, such as inflammatory and autoimmune conditions,[37] genetically determined collagen abnormalities,[38] infectious diseases,[39] neurological conditions,[3,40] and hyperlipidemia.[41,42] A disease process may predispose the tendon to rupture from minor trauma.[43] Blood flow into the tendon decreases with increased age[44] and the area of the Achilles tendon more prone to rupture is relatively avascular compared with the rest of the tendon.[45–47]

High serum lipid concentrations have been reported in patients with complete ruptures of the Achilles tendon.[41,42] Although there is uptake and excretion of sterols by the enzyme sterol 27-hydroxylase (CYP27A1) in the Achilles tendon,[48] histopathological evidence of lipomatosis was only found in 6% of specimens from Achilles tendon ruptures.[5] Further, patients with familiar hypercholesterolemia and Achilles tendon xan-

thomata do not appear to be at greater risk of ruptures.

When considering the "mechanical theory" in relation to acute rupture, McMaster proposed that a healthy tendon would not rupture even if subjected to severe strain.[43] Barfred (in three 1971 papers)[49–51] investigated this hypothesis, and noted that, if straight traction were applied to the tendon, the risk of rupture would be distributed equally to all parts of the muscle-tendon-bone complex. However, if oblique traction is applied, the risk of rupture is concentrated on the tendon. Subjecting a 1.5-centimeter-wide Achilles tendon to traction in 30° supination on the calcaneus leads to elongation of the fibers on the convex aspect of the tendon by 10% before the fibers on the concave side are strained.[49] This means that the risk of rupture would be greatest when the tendon is obliquely loaded with the muscle in maximum contraction, and when the initial length of the tendon was short. In sports requiring rapid push-off, the above factors are likely to be present.[52] Even if a tendon is healthy, there is still a chance of rupture if the strain on the muscle is strong enough, especially in the presence of certain functional and anatomical conditions.[3] Sports are commonly associated with problems of the Achilles tendon, and training errors will increase the risk of these problems.[9,12,53] Clement et al. investigated 109 runners with Achilles tendinopathy.[54] In this series, the three most prevalent causes were overtraining (82 cases), functional overpronation (61 cases), and gastrocnemius/soleus insufficiency (41 cases). Clement et al. speculated that runners would be susceptible to Achilles tendinopathy due to microtrauma produced by the eccentric loading of fatigued muscle.[54] Excess pronation produces a whipping action of the Achilles tendon. Vascular blanching of the Achilles tendon is produced by conflicting internal and external rotatory forces imparted to the tibia by simultaneous pronation and knee extension. Equipment can also be linked with Achilles tendon problems; the flared heel on some sports shoes can force the hindfoot into pronation when the heel strikes the ground.[29]

Intrafibrillar sliding is the process by which tendons are initially damaged at the submicroscopic fibrillar level. This may apply to tendons that rupture without previous degenerative changes. This process occurs a few seconds before macroscopic slippage of collagen fibers, implying that tendons unaffected by degenerative changes may rupture due to accumulation of fibrillar damage. This supports the theory of complete rupture being due to multiple microruptures and the tendon reaching a critical end point prior to rupture. Knorzer et al. illustrated this by using X-ray diffraction spectra to study the behavior of the structure of collagen during tendon-loading.[55] Not only slow or very fast elongation, but also very fast unloading of stretched fibers seems to be responsible for disseminated damage, which reduces the stability of a fiber.

Consideration of temperature in relation to rupture is important, as 10% of elastic energy stored in tendons may be released as heat.[56] Although research has not been performed on human tendons, Wilson and Goodship[14] used equine models to mathematically model tendon thermodynamics. They predicted that the temperature of the central core of the equine superficial digital flexor tendon would plateau at 11°C above the tendon surface temperature during a sustained gallop. Peak intratendinous temperatures in the range 43–45°C were recorded. Temperatures above 42.5°C may result in fibroblast death in vitro.[57] These in vivo recordings provide a possible etiology for the degenerative changes observed in the central core of tendons in both equine and human athletes, and a link with exercise-induced hyperthermia.

Epidemiology of Acute Rupture

Achilles tendon rupture usually occurs in middle-aged men working in a white-collar profession during sports activities.[58] Its incidence has increased during the last decades, at least in Northern Europe and Scotland.[3,59] Leppilahti et al.[2] found an increased incidence of Achilles tendon ruptures in Oulo, Finland from 2/100,000 in 1979–1986 to 12/100,000 in 1987–1994, and also demonstrated a bimodal age distribution. The incidence was highest in the age group 30–39 years with a smaller peak incidence between 50 and 59 years. The mean age was significantly lower for patients experiencing rupture during activities.

Patients with Achilles tendon rupture can be classified into two subgroups, namely young or middle-aged athletes and older nonathletes. Epidemiological data from Malmö, Sweden[59,60] have shown an incidence curve with two peaks:[60] a larger one in young, middle-aged individuals and a smaller one in patients in their seventies. Compared with the age-specific incidence in 1950–1973, there was a marked increase in both sports and nonathletic injuries, with patients in the latter group older than in the former period. Of all spontaneous tendon ruptures, complete Achilles tendon tears are most closely associated with sports activities, with 60% to 75% of all Achilles tendon ruptures related to sports.[61,62] In 430 tendon ruptures, the number of sports-related Achilles ruptures was approximately 62%, similar to the two studies mentioned earlier, while only few (2%) ruptures on other tendons were sports-related.[63] The increase in the athletic group is mostly explained by increased participation in recreational sports. The cause of increase in the elderly group is unknown, though 13% of ruptures occur in patients older than 65 years.[19] Patients with a spontaneous Achilles tendon rupture are at increased risk of sustaining a contralateral Achilles tendon rupture.[25]

The distribution of Achilles tendon ruptures in different sports varies considerably from country to country, according to the national sports traditions. In Scandinavian countries, badminton players are particularly at risk.[64] In a study of 111 patients, 58 (52%) had a rupture of the Achilles tendon while playing badminton.[65] In Northern and Central Europe, soccer, tennis, track and field, indoor ball games, downhill skiing, and gymnastics are the most common sports accounting for Achilles tendon ruptures.[3,66] In North America, American football, basketball, baseball, tennis, and downhill skiing predominate.[3,66]

Achilles tendon rupture is predominantly a male disease and the dominance of males is evident in all studies, with a male : female ratio of 2 : 1 to 12 : 1, probably reflecting the higher prevalence of males involved in sports.[3,67,68] Almost all studies report a dominance of left-sided Achilles tendon ruptures. In a review by Arndt,[69] 57% of 1,823 Achilles tendon ruptures were left-sided, probably because of the higher prevalence of right-side-dominant individuals who push off with the left lower limb.[3,58]

Hungarian and Finnish studies showed a higher prevalence of rupture of the Achilles tendon in patients with blood group O.[63,70] These findings have not been confirmed in another Finnish area and in Scotland,[2,71] probably due to peculiarities in the distribution of the ABO groups in genetically segregated populations.

Among U.S. military personnel who underwent repair of Achilles tendon ruptures between 1994 and 1996, blacks were at increased risk for undergoing repair of the Achilles tendon compared with nonblacks.[72]

Etiology of Achilles Tendinopathy

Overuse injuries of the Achilles tendon are well documented and fairly common.[34,73] Repetitive overload of the Achilles tendon to a level beyond its physiological threshold can lead to inflammation of its sheath, degeneration of its body, or a combination of both.[74]

To date, the etiology of Achilles tendinopathy remains unclear.[73,75] Tendinopathies have been linked to overuse, poor vascularity, lack of flexibility, genetic makeup, gender, endocrine or metabolic factors, and quinolone antibiotics.[76–78] Excessive loading of the tendon during physical exercise is currently thought to be a main pathological stimulus.[79] If the tendon is repetitively overloaded beyond its physiological threshold, it will respond by either inflammation of its sheath, degeneration of its body, or a combination of both.[80] Repetitive microtrauma to the tendon without adequate time for recovery and repair, even if within physiological limits, can also lead to tendinopathy.[81] Microtrauma is linked to non-uniform stresses between the gastrocnemius and soleus, due to their different individual contributions in force. This results in abnormal concentrations of load within the tendon, frictional forces between the fibrils, and localized damage to fibers.[82] It is likely to be multifactorial from a combination of intrinsic and extrinsic factors.[73,74]

Sports injuries can result from intrinsic or extrinsic factors, either alone or in combination.[83] Vascularity, dysfunction of the gastrocnemius-soleus complex, age, gender, body weight and

height, deformity of the pes cavus, and lateral instability of the ankle are considered to be common intrinsic factors.[34] Changes in training pattern, poor technique, previous injuries, footwear, and environmental factors such as training on hard, slippery, or slanting surfaces are the extrinsic factors predisposing to tendinopathy.[73,79] Extrinsic factors predominate in the acute trauma setting; however, overuse injuries and chronic tendon disorders commonly have multifactorial causation.[83]

The extrinsic and intrinsic factors associated with Achilles tendinopathy are listed in Tables 5.1 and 5.2. The basic etiology of Achilles tendinopathy is multifactorial.[83,84] In epidemiological studies, various patterns of malalignment of the lower extremity and biomechanical faults were identified in two thirds of the athletes with Achilles tendon disorders.[73,85] Kvist found malalignment of the lower extremity in 60% of patients with such disorders (Table 5.1).[73,85] However, the mechanisms by which these factors contribute to the pathogenesis of the Achilles tendinopathy remain unclear.[86] The most common malalignment in the ankle is hyperpronation of the foot. Limited subtalar joint mobility and limited range of motion of the ankle joint were more frequent in athletes with Achilles tendinopathy than in those with other complaints.[86] In addition, forefoot varus correlates with Achilles tendinopathy,[73,85,87,88] as does increased hindfoot inversion and decreased ankle dorsiflexion with the knee in extension.[88] Excessive movement of the hindfoot in the frontal plane, especially a lateral heel strike with excessive compensatory pronation, is thought to cause a "whipping action" on the Achilles tendon, predisposing it to tendinopathy.[79]

Table 5.1. Predisposing Intrinsic Factors Related to Achilles Tendinopathy in Sports

General Factors	Local (Anatomic) Factors on the Lower Limb
Gender	Malalignments
Age	Foot hyper- or hypopronation
Overweight	Forefoot varus or valgus
Constitution: weak or strong	Hindfoot varus or valgus
Blood group	Pes planus or cavus
HLA-types	Leg-length discrepancy
Predisposing diseases	Muscle weakness and imbalance
Blood supply	Decreased flexibility
Ischemia	Joint laxity
Hypoxia	
Hyperthermia	

Table 5.2. Predisposing Extrinsic Factors Related to Achilles Tendinopathy in Sports

General Factors	Sports-Related Factors
Therapeutic agents	Excessive loads on the lower extremities
Corticosteroids (local and systemic)	Speed of movement
Fluoroquinolone antibiotics	Type of movement
Weight-lowering drugs	Number of repetitions
Anabolic steroids	Footwear/sportswear
Narcotics (cannabis, heroin, cocaine)	Training surface
	Training error
	High-intensity training
	Fatigue
	Poor technique
	Poor equipment
	Environment (temperature, humidity, altitude, wind)

In addition to hyperpronation and the other malalignments noted earlier, leg-length discrepancy is another controversial potential contributing factor.[83] The traditional orthopedic view is that discrepancies of less than 25 mm are not clinically important.[86] In elite athletes, however, a discrepancy of more than 5 mm may be symptomatic and, consequently, for a discrepancy of 10 mm or more, a built-up shoe or shoe insert has been recommended to prevent overuse symptoms. However, the true occurrence of these proposed biomechanical alterations, their magnitude, and, above all, their clinical importance is not known.[83]

The importance of muscle weakness, imbalance, and decreased musculotendinous flexibility in the development of Achilles tendon disorders is also a matter of debate. Muscle strength, power, endurance, and flexibility are an important part of physical performance, and can thus be important in the prevention of tendon injuries.[83] If the muscle is weak or fatigued, the energy-absorbing capacity of the whole muscle-tendon unit is reduced and the muscle no longer protects the tendon from strain injury, subsequent inflammation, and pain.[83] Recently, excellent short-term

improvements have been reported in patients with chronic Achilles tendinopathy using a heavy-load eccentric training rehabilitation program based on increasing the length, tensile strength, and force of the muscle-tendon unit.[20,89–92] This concept, however, is open to speculation as the studies do not provide conclusive evidence on whether muscular weakness, imbalance, and musculotendinous tightness are the causes or consequences of injuries.

An overuse tendon injury is caused by repetitive strain of the affected tendon such that the tendon can no longer endure tensile stress. As a result, tendon fibers begin to disrupt microscopically, and inflammation and pain result.[83] Of the extrinsic risk factors, excessive loading of the lower extremities and training errors have been said to be present in 60–80% of the patients with Achilles tendon overuse injuries (Table 5.2).[73,84,85] The most common of these include running too long a distance, running at too high an intensity, increasing distance too greatly or intensity too rapidly, and performing too much uphill or downhill work.[73,84,85,88] Monotonous, asymmetric, and specialized training, such as running only (i.e., without cross-training), as well as poor technique and fatigue are considered further risk factors for Achilles tendon overuse injuries (Table 5.2). Poor environmental conditions, such as cold weather, hard ground surface, and slippery/icy surface may also promote Achilles tendon problems.[66,83,86,93,94] The lack of high-quality prospective studies limits the strength of the conclusions that can be drawn regarding these extrinsic risk factors.

The pathogenesis of Achilles tendon disorders includes many factors such as tissue hypoxia and resulting free radical changes to the tendon from ischemic reperfusion injury and exercise-induced hyperthermia.[13,14] If a tendon is strained to more than 4% of its original length, it loses its elasticity and is at an increased risk of a subsequent break in its collagen structure.[66]

Levels of type III collagen mRNA can be significantly higher in the tendinopathic Achilles tendon compared with normal samples.[95] The significance of this finding is still open for debate. It should be noted that most of the above factors should be associative, not causative, evidence, and their role in the etiology of the condition is therefore still debatable.

Epidemiology of Achilles Tendinopathy

In large studies, the most common clinical diagnosis with reference to Achilles disorders is tendinopathy (55–65%), followed by insertional problems (retrocalcaneal bursitis and insertional tendinopathy) (20–25%).[73,85,96–102] Kujala et al., in an 11-year follow-up cohort study,[103] found questionnaire-reported Achilles tendon overuse injury in 79 out of 269 male orienteering runners (30%), and in 7 of the 188 controls (4%). The age-adjusted odds ratio was 10.0 in runners compared with controls.[103]

There is a clear association with strenuous physical activities such as running and jumping.[34,66,93,96] In top-level runners, the annual incidence of Achilles tendon disorders is reported to be between 7% and 9%.[98,99] The occurrence of Achilles tendinopathy is highest among individuals who participate in middle- and long-distance running, orienteering, track and field, tennis, badminton, volleyball, and soccer.[73,85,96–100]

Kvist[73,85] studied the epidemiology of Achilles tendon disorders in a large group of competitive and recreational athletes with Achilles tendon problems. Following a review of 698 patients, running was the main sports activity in patients presenting with an Achilles tendon disorder (53%) and patients who were runners represented 27% of all patients studied in the sports medicine clinic where the study was performed.[73,85] Sixty-six percent had Achilles tendinopathy and 23% had Achilles tendon insertional problems.[73,85] In 8% of the patients, the injury was located at the myotendinous junction, and 3% of the patients had a complete tendon rupture. On review of the sex of the patient, only 11% were female.[73,85]

As athletes increase in age, chronic Achilles tendon disorders become more common.[104] In a report of 470 patients with Achilles tendinopathy and insertional complaints, only 25% of subjects were teenage or child athletes with 10% under 14 years of age. The majority of these younger patients were diagnosed with calcaneal apophysitis (Sever´s disease).[73]

Patients with unilateral Achilles tendinopathy have a relatively high risk of developing Achilles tendinopathy in the other leg. Initially,

the unaffected leg may show no clinical symptoms and signs of tendinopathy. However, in one study 41% developed symptoms of the Achilles tendinopathy in the contralateral leg by eight years' follow-up.[105]

Conclusion

Achilles tendon problems are increasingly common. Appropriate etiopathogenesis and epidemiological studies are lacking, and most studies do not adequately define their population.

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6
Paratendinopathy of the Achilles Tendon

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There has been a dramatic increase of Achilles tendon overuse injuries in the past four decades. This is in line with the increased prevalence of overuse sports injuries in Western countries due to increase in the demands on competitive and professional sports performances. Achilles tendon overuse injuries not only affect competitive athletes, but also affect recreational sports participants and individuals in occupations where the lower limbs are under excessive load.[1–6]

The terms Achilles *peritendinitis*, *tendonitis*, and *tendonitis* have been widely used, even though no inflammatory cell infiltration in the paratenon or tendon is found in biopsies of chronic Achilles tendon problems. These terms supposedly describe the painful condition at or around the Achilles tendon, are confusing, and most often do not reflect the underlying pathology. Biochemical mediators of inflammation such as prostaglandin E_2 are not more abundant in patients with chronic Achilles tendon pain than in controls.[1,2] However, we have to caution that absence of inflammatory cell infiltration in the chronic state does not exclude a prior inflammatory condition.

Terms like Achilles *tendinopathy*, *tenopathy*, *tendinosis*, *partial rupture*, *paratenonitis*, *tenosynovitis*, *tendovaginitis*, *peritendinitis,* and *achillodynia* have been used to describe the non-insertional painful conditions of the Achilles tendon. *Achillodynia* was a term suggested by Åström, to be used as a symptomatic diagnosis. He further recommended that *tendinosis* (tendon degeneration) and *peritendinitis* be reserved for cases where the pathology has been verified by surgical exploration, radiological imaging, histological biopsies, or their combination.[7] *Tendinopathy* is a clinical diagnostic term suggested by Maffulli et al. for a combination of tendon pain, swelling, and impaired performance and should include the histopathological entities *peritendinitis* and *tendinosis*[8] only after histological confirmation. This suggestion has a sound basis, since the clinical rationale to differentiate the histopathological entities of Achilles peritendinitis and tendinosis is uncertain, and there have been no randomized studies comparing outcomes of treatment or the natural history of these two conditions. The term *paratendinopathy* is derived from *tendinopathy* to describe activity-associated Achilles tendon pain combined with tenderness at palpation, swelling around the tendon, and no suspicion of intratendinous pathology on the grounds of patient history, clinical examination, or imaging. However, no studies have revealed unconnected appearance or compared the outcome of these two conditions.[9]

Anatomy and Function of the Paratenon

The *paratenon* is a thin, gliding membrane covering the whole extent of the Achilles tendon. The paratenon functions as an elastic sleeve (probably not as effective as a true tendon sheath) permitting free movement of the tendon inside the surrounding tissues.[1,10] The paratenon forms a thin space between the tendon and the crural fascia, which is covered by subcutaneous fat and skin.[10–12]

Under the paratenon lies the *epitenon*, which is a fine, smooth connective tissue sheath surrounding the entire Achilles tendon. The epitenon is in contact with the paratenon on its outer surface. The epitenon's inner surface is in continuity with the endotenon, which binds the collagen fibers and fiber bundles together and provides the neural, vascular, and lymphatic supply to the tendon.[1]

The paratenon is a vascular structure that provides blood supply to the Achilles tendon.[1,2,13,14] The Achilles tendon and its paratenon are innervated by nerves from the attaching muscles and by small fasciculi from cutaneous nerves, especially the sural nerve.[15] There are only a few nerves and nerve endings and these nerve fibers terminate on the tendon surface or in the paratenon.[1] These nerves follow the vascular channels within the long axis of the tendon, anastomose with each other via obliquely and transversely oriented fibers, and finally terminate in sensory nerve endings.[1] In patients with Achilles tendon overuse injury, the sensory nerve endings follow the paratendinous neovascularization and may be a cause of Achilles tendon pain.[16]

Epidemiology

The annual incidence rate for running injuries varies between 24% and 65%.[17,18] About 50% to 75% of all running injuries are overuse injuries from constant repetition of the same movement mostly in the Achilles tendon and in the tendons surrounding the knee joint.[4] The occurrence of Achilles tendon overuse injuries is highest in middle- and long-distance running, orienteering, track and field, tennis, and other ball games,[4,6,19–25] with an annual incidence of between 7% and 9% in Achilles tendon overuse injuries in top-level runners.[20,21] The most common clinical diagnosis of Achilles overuse injuries is paratendinopathy and/or tendinopathy (55% to 65%), followed by insertional problems (retrocalcanear bursitis and insertional tendinopathy) (20% to 25%).[4,6,24,25] However, on the basis of epidemiological studies of patients with Achilles tendon overuse injury, the actual incidence of pure paratendinopathy without any alterations in the tendon itself is not known.

Kujala et al.,[26] in a cohort study with an 11-year follow-up, noted Achilles tendon overuse injuries in 79 of 269 male orienteering runners (30%) and 7 of 188 controls (4%), the age-adjusted odds ratio being 10.0 in runners compared with controls.

In another large cohort of 698 sports patients, Kvist studied the epidemiologic factors associated with Achilles tendon injuries. Paratendinopathy and/or tendinopathy were found in 66%, and Achilles tendon insertional problems accounted for 23%. The injury was located at the myotendinous junction in 8%, whereas 3% of them had a total tendon rupture.[4,19] The male : female ratio of patients with Achilles tendon injury was 9 : 1. Running was the main sporting activity in patients with Achilles tendon injury (53%), while running sports patients accounted for 27% of all patients studied in that clinic.

Etiology and Pathophysiology

Intrinsic or extrinsic factors, either alone or combination, can cause overuse injuries.[1] In acute trauma, extrinsic factors predominate while overuse injuries are generally multifactorial. In chronic tendon disorders, an interaction between these two types of factors is common.[1,2] The causes of Achilles tendon overuse injuries and the factors leading to the predominance of paratendinous pathology without any changes in the Achilles tendon itself are incompletely known.

The etiology of Achilles tendon overuse injuries is multifactorial.[24,27] These studies are mainly retrospective, and report statistical association between the condition and several factors. A direct cause–effect relationship has been demonstrated by very few studies. Training errors like running too long, at too high an intensity, increasing the training distance too greatly or the intensity too rapidly, and performing too much uphill or downhill work[4,20,27] have been reported in two-thirds of the running injuries.[21,28] Monotonous, asymmetric, and specialized training, such as running only (i.e., without cross-training), as well as poor technique and fatigue are further risk factors for Achilles tendon overuse injuries.[27] Poor environmental conditions such as cold weather, hard ground surface, and slippery/icy surface have also been suggested to promote Achilles tendon problems.[28–30]

Two epidemiological studies have associated various alignment and biomechanical faults with Achilles tendon disorders in two-thirds of the athletes.[4,19] However, the mechanism by which the overuse injuries occur remains controversial.[31] The most common and perhaps most important malalignment implicated is hyperpronation of the foot. Limited subtalar joint mobility and limited range of motion of the ankle joint were more frequent in athletes with Achilles tendinopathy than in those with other complaints.[19] In addition, forefoot varus is associated with Achilles tendinopathy.[4,19,31,32] Recently, Kaufman et al. observed that increased hindfoot inversion and decreased ankle dorsiflexion with the knee in extension is associated with Achilles tendinopathy.[33]

In addition to hyperpronation and the other malalignments, leg-length discrepancy is a further controversial potential contributing factor.[27] Traditionally, discrepancies of less than 25 mm were not considered to be clinically important.[27] In elite athletes, however, a discrepancy of more than 5 to 6 mm may be symptomatic and, consequently, for a discrepancy of 10 mm or more, a built-up shoe or shoe insert has been recommended to prevent overuse symptoms.[27] However, the exact occurrence of these proposed biomechanical alterations, their magnitude, and, above all, their clinical importance is not well known.[27]

Muscle weakness, imbalance, and impaired musculotendinous flexibility in the development of Achilles tendon disorders are also debated. Muscle strength, power, endurance, and flexibility play an important role in physical performance and therefore are important in the prevention of sports injuries, particularly tendon injuries.[27] A weak or fatigued muscle no longer protects the tendon from strain injury as the energy absorbing capacity of the muscle-tendon unit is reduced.[27] Also, the elastic properties of the Achilles tendon are correlated to muscle strength.[34] Very good short-term improvements have been reported in chronic Achilles tendinopathy with heavy-load eccentric training, a rehabilitation program based on increasing the length, tensile strength, and force of the muscle-tendon unit.[35–39] The beneficial effect of eccentric training has been demonstrated with MRI, as abnormal intratendinous signal intensity and tendon volume decreased after three months' eccentric training in patients with chronic Achilles tendinopathy.[38]

Histopathology of Paratendinous Alterations

Inflammatory cell reaction, circulatory impairment, and edema formation occur in the acute phase of Achilles paratendinopathy.[1,2] Movement of the Achilles tendon within a paratenon filled with fibrin exudates may result in clinically palpable crepitus. If the management or the natural healing process of this acute condition fails, the fibrin may organize and form adhesions that interfere with the normal gliding-movement between the tendon, paratenon, and crural fascia.[4,19] In chronic Achilles paratendinopathy, the paratendinous tissue becomes thickened due to fibrinous exudate, prominent and widespread proliferation of fibroblasts, and formation of new connective tissue, and the adhesions between tendon, paratenon, and crural fascia occur.[40–43]

Normal fibroblasts and myofibroblasts have been identified in the paratenon of chronic Achilles paratendinopathy.[44] Heavy mechanical strains imposed on the tendon stimulate the fibroblasts to secrete transforming growth factor-β (TGF-β), which in turn acts in an auto- or paracrine manner on tenocytes, which acquire a myofibroblast phenotype.[45,46] Myofibroblasts have cytoplasm fibers of α–smooth muscle actin, and are thus capable of producing the forces required in physiological processes such as granulation contraction.[45,46] In Achilles paratendinopathy, myofibroblasts are especially present at the sites of scar formation,[44] and about 20% of peritendinous cells are myofibroblasts in chronic paratendinopathy.[47] Myofibroblasts synthesize abundant collagen I and III,[40,41] and are probably responsible for the formation of permanent scarring and the shrinkage of peritendinous tissue around the tendon.[43,44] These cells most probably also play an important role in producing clinical symptoms, as they can induce and maintain a prolonged contracted state in the peritendinous adhesions around the tendon.[43,44] This may lead to constriction of vascular channels and to impaired circulation and further contribute to the pathogenesis of Achilles tendinopathy.[43,44] The proliferating connective tissue around the Achilles tendon causes increased intratendinous tension and pressure, resulting in increased friction between the tendon, paratenon, crural fascia, and the overlying skin.[43,44]

Diagnosis of Achilles Paratendinopathy

Patients with Achilles paratendinopathy present with pain in the Achilles tendon region or posterior heel region. The severity of pain is used to classify the severity of the disorder and is also used as an outcome measure.[1] Clinical examination reveals that the tendon is diffusely swollen on palpation, and is tender in the middle third of the tendon. A crepitus may be occasionally palpable in the acute phase;[4,9] however, the swelling and tenderness does not move when the ankle joint is dorsiflexed. Areas of erythema, increased local warmth, and palpable tendon nodules or defects may also be present. Ankle instability and malalignment of the lower extremity, especially in the foot, should be actively sought for in patients with Achilles tendon complaints.[1–3]

In chronic Achilles paratendinopathy, exercise-induced pain is still the cardinal symptom while crepitation and swelling diminish.[1–3] A tender, nodular swelling usually indicates tendinopathy of the main body of the tendon[28,48] and these focal tender nodules move as the ankle is dorsiflexed and plantarflexed.[49] However, there is a marked overlapping of the findings in history and physical examination, and, in clinical practice, overuse injuries have features of more than one pathophysiological entity (e.g., patients with tendinopathy or with partial rupture usually have additional peritendinous pathology).

Ultrasonography (US) is a useful diagnostic modality only if adhesions are present around the Achilles tendon in paratendinopathy.[50] Ultrasonography is unreliable when it fails to detect adhesions, and patients with few adhesions may also have a false negative result on US.[50] In acute form, US can detect fluid around the tendon,[50] whereas paratendinous adhesions are visualized as thickening of the hypoechoic paratenon with poorly defined borders in the chronic form (Fig. 6.1A, B).[51]

Magnetic resonance imaging (MRI) has been used extensively to visualize tendon pathology.[52,53] However, it has relatively high cost, limited availability in some countries, a time-consuming procedure, and slow and often incomplete resolution of signal changes after operative intervention.[2] Soila et al. have shown that the normal anatomy of an asymptomatic Achilles tendon may vary, causing diagnostic misinterpretation with MRI.[54] However, in patients with pure chronic Achilles paratendinopathy (without tendinopathy of the main body of the Achilles tendon), MRI infrequently reveals any pathological changes around the tendon.

Management of Achilles Paratendinopathy

Alleviation of pain and prevention of progression of acute to chronic paratendinopathy are the main goals of management in the acute phase. In chronic cases, the alleviation of pain predominates the management strategies, while predisposing factors and etiological causes are actively sought and corrective measures are taken. Little reliable experimental or clinical scientific work has been performed on the pathophysiology, etiology, natural course, and management of Achilles tendon overuse injuries.[2,3] Both conservative and surgical regimens vary considerably among countries, clinics, and physicians. Most management regimens are based only on what empirically seemed to work without much scientific support.[1–3]

In the early phase of Achilles paratendinopathy, conservative, nonoperative management is pursued.[2,55] Identification and correction of the predisposing factors mentioned earlier are addressed initially.[55] Correction of malalignment is addressed with orthotics, and if problems are identified with the training program, it is modified to place less strain on the Achilles tendon. In patients with severe symptoms, the lower extremity (or just the ankle joint) is completely rested for a short time.[2] The use of nonsteroidal anti-inflammatory drugs (NSAIDs) did not positively affect the outcome of Achilles tendinopathy in a randomized clinical trial;[56] however, they are often used for early management of pain from Achilles paratendinopathy.[55] The corticosteroid injections around the Achilles tendon should be used with extreme caution,[55,57] and should be administered only by experienced physicians.[57] Failure to adequately respond after 3 to 6 months of conservative management is the indication for surgery.[4,11,58–66] However, no prospective randomized studies comparing operative and conservative management of Achilles paratendinopathy have been published, and most of our knowledge

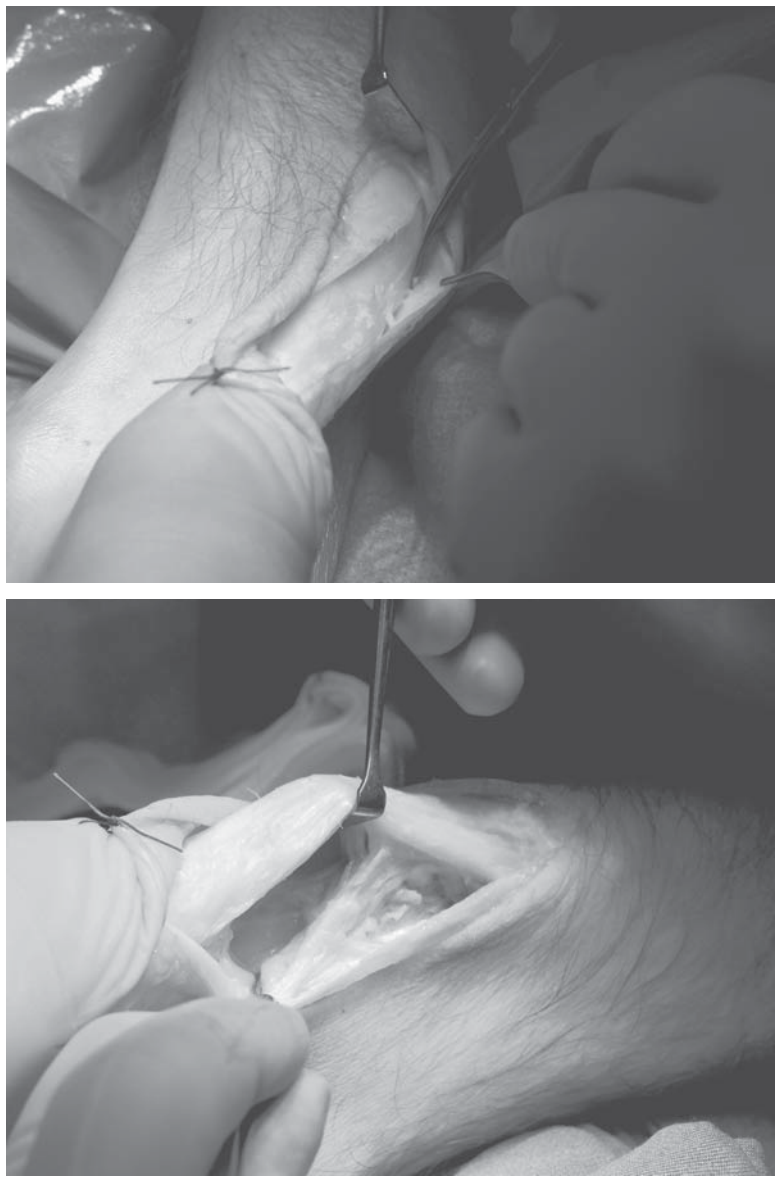

Figure 6.1. (**A**) The crural fascia has been incised and the adhesions around the thickened paratenon are divided. The tight adhesions between the paratenon and the crural fascia as well as between paratenon and tendon were carefully excised, but as many layers of the true Achilles paratenon as possible were left intact. (**B**) The Achilles tendon was freed from adhesions including its anterior aspect. There were a considerable amount of paratenon closed to the Achilles tendon.

on management efficacy is based on clinical experience and descriptive studies.[2,3]

Tallon et al. reviewed studies that reported surgical outcomes in the management of chronic Achilles tendinopathy and/or paratendinopathy.[67] These studies had low scientific validity, as methodology scores of these studies were low.[67] A negative correlation was found between reported success rates and overall methodology scores, but the positive correlation between year of publication and overall methodology score suggests that the quality of studies is improving.[67] In

paratendinopathy of the Achilles tendon, many authors recommend that, after the longitudinal division of the crural fascia, the paratenon is incised and any macroscopic adhesions are excised.[11,58,60–63] Some authors have reported that the adhesions were found mainly between the Achilles tendon and paratenon,[58,60] while others have found that the paratenon was adhered mainly to the crural fascia, or even to the skin (Fig. 6.2).[11,68]

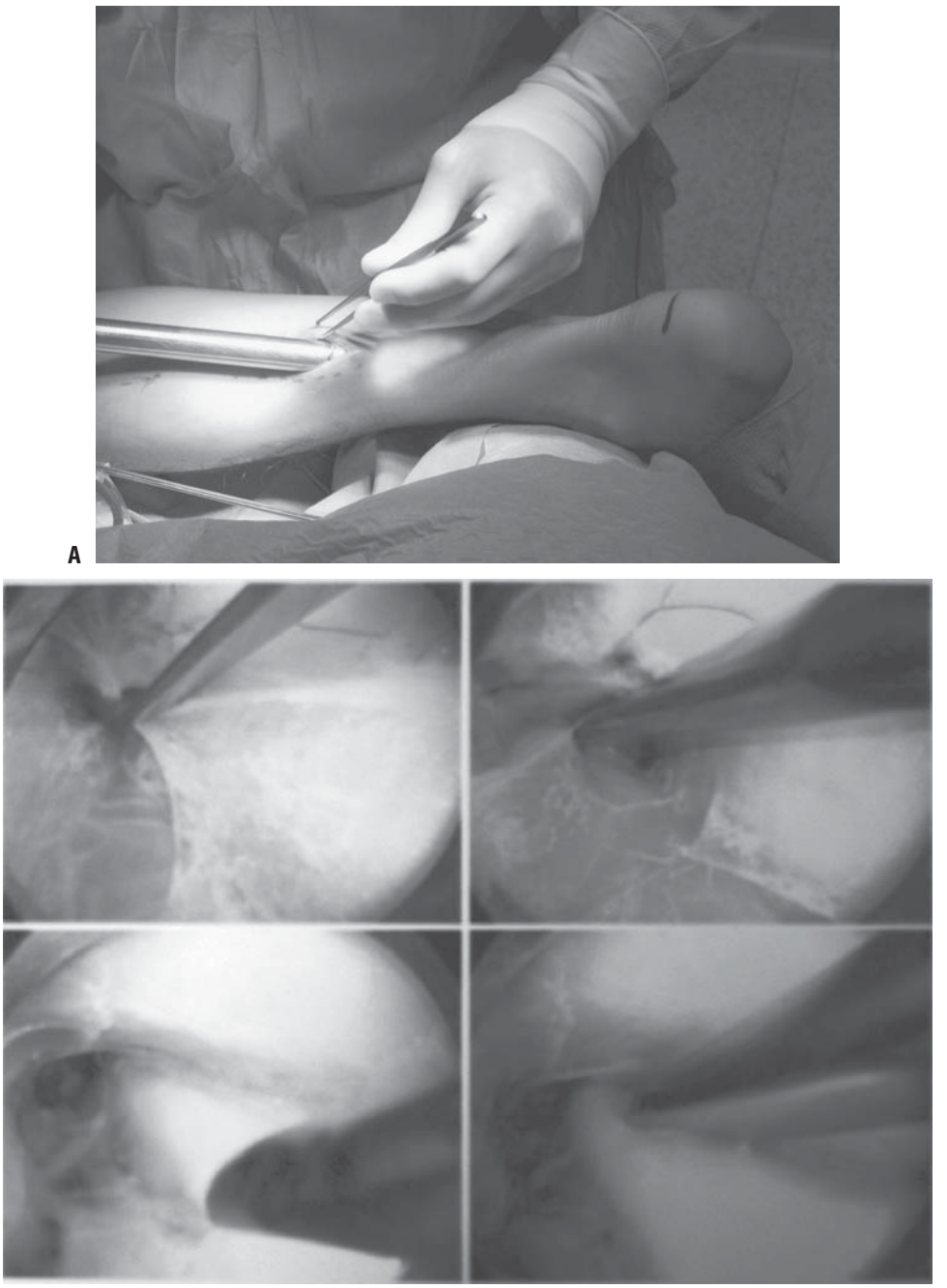

Figure 6.2. (**A**) Through proximal portal incision, the scope is introduced below the crural fascia, which is released longitudinally. (**B**) Via endoscope, the adhesions are released with retrograde knife blade or blunt dissector.

A few studies of an endoscopic-assisted surgical release of adhesions around the Achilles tendon have been done.[69–71] The preliminary results have been good,[70,71] and endoscopic technique may have advantage of reducing postoperative morbidity (Fig. 6.2). However, there are no studies comparing different operative methods in treatment of Achilles paratendinopathy.

Most studies report satisfactory results in 75% to 100% of patients who underwent surgery for Achilles tendon overuse injury. Most of these investigations are retrospective, and in only a few of them the results are based on objective evaluations, such as range of motion of the ankle. Moreover, the underlying pathology has usually been heterogeneous.[11,58–66] In a report by Paavola et al.,[72] operative management of Achilles paratendinopathy with or without tendinopathy of the main body of the Achilles tendon yielded good and acceptable short-term results using subjective, clinical, and functional tests as outcome criteria. A lower complication rate of the operative management and a better trend to recovery was observed in patients with pure paratendinous adhesions than in those with paratendinous adhesions combined with an intratendinous lesion.[72]

An overall complication rate of 11% was reported in a series of 432 consecutive patients.[73] The most common complication in that study was compromised wound healing (54%), which affected more frequently patients operated on for a partial Achilles tendon rupture than for Achilles tendinopathy only.[73]

Prognosis

Although we do not have complete knowledge regarding the natural course of Achilles tendon paratendinopathy, an 8-year follow-up study[74] showed good long-term prognosis. At the 8-year follow-up, there was a definite side-to-side difference between the involved and the uninvolved sides in the performance tests, clinical examination, and US findings.[74] However, 70 of the 83 patients (83%) were able to return to full levels of physical activity at 8 years, and 78 patients (94%) were asymptomatic or had only mild pain on strenuous exercise.[74] Delay of up to six months between the onset of symptoms and initiation of conservative management did not compromise long-term outcome.[74] Nevertheless, 24 of the 83 patients (29%) failed to respond to conservative management and underwent operative management.[74] Approximately half of the patients (41%) developed some overuse symptoms (exertional pain with or without swelling and stiffness) in the initially uninvolved Achilles tendon.[74]

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7
Tendinopathy of the Main Body of the Achilles Tendon

Adam Ajis, Nicola Maffulli, Hakan Alfredson, and Louis C. Almekinders

Introduction

In the past three decades, the incidence of Achilles tendinopathy has risen as a result of greater participation in recreational and competitive sporting activities.[1,2] A 10-fold increase in Achilles tendon injuries has been reported in runners compared to age-matched controls. The incidence of Achilles tendinopathy in top-level runners has been estimated at around 7–9%. Achilles tendinopathy is also common among athletes participating in racquet sports, track and field, volleyball, and soccer. However, Achilles tendinopathy does not exclusively affect athletes. In a recent study, 31% of 58 Achilles tendinopathy patients did not participate in vigorous physical activity.[3]

Etiology and Pathophysiology

Tendon injuries can be acute or chronic and are caused by intrinsic or extrinsic factors, either alone or in combination. In acute trauma, extrinsic factors predominate. Overuse injuries generally have a multifactorial origin.[4] In chronic tendon disorders, interaction between intrinsic and extrinsic factors is common.[5] There is sparse scientific knowledge about the etiology and pathophysiology of chronic painful tendon conditions, and most hypotheses put forward have not been substantiated by sound scientific research.

Tendon vascularity, gastrocnemius-soleus dysfunction, age, gender, body weight and height, pes cavus, and lateral ankle instability are common intrinsic factors. Excessive motion of the hindfoot in the frontal plane, especially a lateral heel strike with excessive compensatory pronation, is thought to cause a "whipping action" on the Achilles tendon, and predispose it to tendinopathy. Also, forefoot varus is frequent in patients with Achilles tendinopathy. Changes in training pattern, poor technique, previous injuries, footwear, and environmental factors such as training on hard, slippery, or slanting surfaces are extrinsic factors that may predispose the athlete to Achilles tendinopathy.[2,6]

Excessive loading of tendons during vigorous physical training is regarded as the main pathological stimulus for tendinopathy.[2] Tendons respond to repetitive overload beyond physiological threshold by either inflammation of their sheath, histological changes of their body, or a combination of both.[7] It remains unclear whether different stresses induce different responses. Active repair of fatigue damage must occur, or tendons would weaken and eventually rupture. The repair mechanism is probably mediated by resident tenocytes, which continually monitor the extracellular matrix. Failure to adapt to recurrent excessive loads results in the release of cytokines leading to further modulation of cell activity.[8] Tendon damage may even occur from stresses within physiological limits, as frequent cumulative microtrauma may not allow enough time for repair.[2] Microtrauma can also result from nonuniform stress within tendons, producing abnormal load concentrations and frictional forces between the fibrils, with localized fiber damage.[8]

The etiology of tendinopathy remains unclear, and many factors have been implicated.[1] Free radical damage occurring on reperfusion after ischemia, hypoxia, hyperthermia, and impaired tenocyte apoptosis have been linked with tendinopathy.[9] In animal studies, local administration of cytokines and inflammatory agents such as prostaglandins has resulted in tendinopathy.[10] Fluoroquinolones have also been implicated in the pathogenesis of tendinopathy. Ciprofloxacin causes enhanced interleukin-1β mediated MMP3 release, inhibits tenocyte proliferation, and reduces collagen and matrix synthesis.[11]

Degenerative tendinopathy is the most common histological finding in spontaneous tendon ruptures. Tendon degeneration may lead to reduced tensile strength and a predisposition to rupture. Indeed, ruptured Achilles tendons have more advanced intratendinous changes than tendinopathic tendons.[12] In Achilles tendinopathy, changes in the expression of genes regulating cell-cell and cell-matrix interactions have been reported, with down-regulation of matrix metalloproteinase 3 (MMP 3) mRNA.[13] Significantly higher levels of type I and type III collagen mRNAs have been reported in tendinopathic samples compared to normal samples.[13] In tendinopathic Achilles tendons, up-regulation of MMP 2 and VEGF has been reported, while MMP 3 was down-regulated compared to control samples.[14] Imbalance in MMP activity in response to repeated injury or mechanical strain may result in tendinopathy.

Pain is the main symptom of Achilles tendinopathy, but the underlying mechanism causing pain is not fully understood. Traditionally, pain has been thought to arise through inflammation, or via collagen fiber separation or disruption.[15,16] However, chronically painful Achilles tendons have no evidence of inflammation, and many tendons with intratendinous pathology detected on MRI or ultrasound are not painful.[15–18] As tendinopathies are not inflammatory conditions, pain may originate from a combination of mechanical and biochemical causes:[17] chemical irritants and neurotransmitters may generate pain. Microdialysis sampling revealed a twofold increase in lactate levels in tendinopathic tendons compared to controls.[19] High concentrations of the neurotransmitter glutamate, with no abnormal elevation of the pro-inflammatory prostaglandin PGE_2, have been found in patients with Achilles and patellar tendinopathy.[18]

Several studies have confirmed the occurrence of sensory neuropeptides in both animal and human tendons, and Substance P (SP) has been found in tendinopathic Achilles tendons.[20–22] Endogenous opioids provide a peripheral antinociceptive system, and morphine inhibits the release of SP from peripheral sensory nerve endings.[23] Under normal conditions, a balance probably exists between nociceptive and antinociceptive peptides. However, this balance may be altered in pathological conditions.

Histopathology

The pathologic label *tendinosis* is used to describe the disorganized impaired healing response in tendinopathy. Despite that, most clinicians still use the term *tendonitis* or *tendinitis*, thus implying that the fundamental problem is inflammatory. We advocate the use of the term *tendinopathy* as a generic descriptor of the clinical conditions in and around tendons arising from overuse, and suggest that the terms tendinosis, tendonitis and tendinitis only be used after histopathological examination.[24]

Histologically, Achilles tendinopathy is characterized by an absence of inflammatory cells and a poor healing response. Other changes include: noninflammatory intratendinous collagen degeneration, fiber disorientation and thinning, hypercellularity, scattered vascular ingrowth, and increased interfibrillar glycosaminoglycans.[4,8,25] Frank inflammatory lesions and granulation tissue are mostly associated with tendon ruptures.[26]

Various types of so-called degeneration may be seen in tendons, but in the Achilles tendon "mucoid" or "lipoid" degeneration is usually seen.[26] In mucoid degeneration, light microscopy reveals large mucoid patches and vacuoles between fibers. In lipoid degeneration, abnormal intratendinous accumulation of lipid occurs, with disruption of collagen fiber structure.[25]

Paratendinopathy may occur alone or in combination with degeneration of the tendon body.[27] Histologically, mucoid degeneration, fibrosis, and

vascular proliferation with a slight inflammatory infiltrate have been reported.[5,28,29] Clinically, edema and hyperemia of the paratenon are seen. A fibrinous exudate accumulates within the tendon sheath, and crepitus may be felt on clinical examination.[17]

Metalloproteases in Achilles Tendinopathy

Disorganized healing is constant in chronic tendinopathy. Normal tendon is mostly composed of Type I collagen, while tendinopathic tendons have a greater proportion of Type III collagen, which is associated with tendon rupture.[30] Matrix metalloproteases (MMPs) are involved in remodeling of the extracellular matrix (ECM) of tendons, being either up- or down-regulated in tendinopathy. A balance between MMPs and tissue inhibitors of metalloproteases (TIMPs) is probably necessary to maintain tendon homeostasis. The mechanism of activation of MMPs is poorly understood, and their precise role in tendinopathy is still unclear.

Degradation of collagen and other ECM compounds is initiated by matrix metalloproteases (MMPs).[31] These are zinc and calcium-dependent endopeptidases secreted from cells in proenzyme form.[32] MMPs are the major enzymes involved in remodeling of ECM because of their efficacy at neutral pH and their broad proteolytic capability against the ECM.[33]

The MMP family comprises 23 members,[33] subdivided into four main classes: collagenases, gelatinases, stromelysins, and membrane-type MMPs.[34] MMPs are involved in many other physiological remodeling processes, including wound healing, menstruation, uterine involution, bone growth and development, and angiogenesis.[35–37] They also play a role in pathological processes such as tumor invasion and metastasis,[38–42] multiple sclerosis,[43] periodontal disease,[44,45] hypertension,[46] and arthritis.[47–54]

The activity of MMPs is inhibited by tissue inhibitors of metalloproteases (TIMPs).[55,56] The balance between the activities of MMPs and TIMPs regulates tendon remodeling. An imbalance in MMPs and TIMPs is associated with collagen disturbances.[57] Cytokines such as interleukin-1 (IL-1) and tumor necrosis factor α (TNFα) enhance the production of MMPs,[58–60] whereas transforming growth factor β (TGFβ) and IL-6 enhance the production of TIMP-1.[61,62]

MMPs can be up- or down-regulated, locally and systemically, in tendinopathy and complete tendon tears. A balance exists between expression of MMPs and TIMPs to maintain tendon homeostasis. More research is required to determine the mechanism of action and regulation of MMPs in tendinopathy to promote the development of specific therapeutic strategies in these patients.[63]

Clinical Presentation

Pain is the cardinal symptom of Achilles tendinopathy. Generally pain occurs at the beginning and end of a training session, with a period of diminished discomfort in between. As the pathological process progresses, pain may occur during exercise, and, in severe cases, it may interfere with activities of daily living. In the acute phase, the tendon is diffusely swollen and edematous, and on palpation tenderness is usually greatest 2 to 6 cm proximal to the tendon insertion. Sometimes, fibrin precipitated from the fibrinogen-rich fluid around the tendon can cause palpable crepitation.[1,25,64] In chronic cases, exercise-induced pain is still the cardinal symptom, but crepitation and effusion diminish.[25] A tender, nodular swelling is usually present in chronic cases, and is believed to signify tendinosis.[64]

The diagnosis of Achilles tendinopathy is mainly based on a careful history and detailed clinical examination. Diagnostic imaging may be required to verify a clinical suspicion or to exclude other musculoskeletal disorders, such as os trigonum syndrome, tenosynovitis or dislocation of the peroneal tendons, tenosynovitis of the plantar flexors, an accessory soleus muscle, tumors of the Achilles tendon (xanthomas), and neuroma of the sural nerve.[65]

Imaging

Ultrasonography is commonly employed in Europe to examine tendon disorders. It is readily available, quick, safe, and inexpensive. However,

ultrasound is very operator dependent, has somewhat limited soft tissue contrast, and is not as sensitive as MRI.[25,66] However, the recent addition of power Doppler US imaging has made the use of US much more pertinent to the functional imaging of Achilles tendinopathy,[67] as it gives physiologically functional imaging that is not available with MRI.

In acute cases, ultrasound reveals fluid accumulation around the tendon. In chronic cases, peritendinous adhesions may be shown by thickening of the hypoechoic paratenon with poorly defined borders. A simple grading system has been devised for tendinopathy. Grade 1 represents a normal tendon; grade 2 an enlarged tendon; and grade 3 a tendon containing a hypoechoic area.[68] Hypoechoic areas can be nodular, diffuse, or multifocal, and they correlate well with macroscopic findings at surgery.

MRI provides information on the internal morphology of the tendon and the surrounding structures. It is useful to evaluate chronic tendinopathy and to differentiate between tendinopathy of the main body of the tendon and paratendinopathy. Excellent correlation between MRI and pathological findings at surgery has been reported.[69]

A recent longitudinal ultrasound study showed that mild-to-moderate changes were observed frequently in both involved and uninvolved Achilles tendons, but the occurrence of these changes was not clearly related to patients' symptoms.[70] Given the high sensitivity of these imaging modalities, an abnormality should be interpreted with caution and correlated to the patient's symptoms before formulating management recommendations.[4]

Management

In the early phase of Achilles tendinopathy, various forms of conservative management are normally used.[5,70,71,109] Seeking medical attention at an early stage may improve outcome, as treatment becomes more complicated and less predictable when the condition becomes chronic.[11,23] Surgical management is recommended for patients who do not adequately respond to a conservative program over 3 to 6 months.[5,28,69,71]

Conservative Management

Conservative management is recommended as the initial strategy,[72–81] with identification and correction of possible etiological factors, at times using a symptom-related approach. There are few scientific prospective studies, and only in the last few years studies have compared different types of conservative management regimens in the chronic painful Achilles tendon using a randomized controlled design.

The initial management most commonly consists of a multi-oriented approach, with combined rehabilitation models. There are many different regimes, most often including a combination of rest (complete or modified), medication (NSAIDs, corticosteroids), orthotics (heel lift, change of shoes, corrections of misalignments), stretching and massage, and strength training,[73,74,76,77,81–83] and the most widely used initial approach is eccentric strength training of the gastro-soleus complex.[84]

Decreasing the intensity, frequency, and duration of the activity that caused the injury, or modification of that activity, may be the only necessary action to control symptoms in the acute phase. Collagen repair and remodeling is stimulated by tendon loading, and complete rest of an injured tendon can be detrimental. Modified rest, which allows activity in the uninjured body parts and reduces activity at the injured site, has been recommended.[25,84]

Despite the absence of scientific evidence for an ongoing chemical inflammation inside the tendon,[79,85] nonsteroidal anti-inflammatory drugs (NSAIDs) are often being used as one part of the initial management.[76,77,82,86–90] In a randomized double-blind placebo-controlled study of 70 patients with chronic painful Achilles tendinopathy, oral piroxicam gave similar results to placebo.[91] We do not recommend NSAIDs.

Management with corticosteroid injections is being debated.[74,92,93] Most authors suggest that corticosteroid injections should be placed outside the tendon to avoid damage to the tendon tissue, but there are groups that suggest intratendinous injections.[94,95] Partial ruptures are found not infrequently after steroid injections[83,96–98] and management with steroid injections has been shown to predict a partial rupture in patients with chronic

Achilles tendinopathy.[96] Also, a relatively large number of patients operated on for chronic Achilles tendinopathy had previously received corticosteroid injections, indicating a poor effect of corticosteroids on the condition. Overall, peritendinous injections with corticosteroids are still controversial, evidence for their effectiveness is missing, and there are no good scientific reasons to support their use.[99] Intratendinous injections of corticosteroids are to be avoided. We do not recommend the use of corticosteroids, either intra- or peritendinously.[99]

Modalities such as cryotherapy,[77] heat,[100] massage,[101] ultrasound,[102] electrical stimulation,[103] and laser therapy[104] are sometimes included in the management regimen. These have been reported to be effective but there are no well-planned scientific clinical studies confirming their effects.

Cryotherapy has been regarded as a useful intervention in the acute phase of Achilles tendinopathy, as it has an analgesic effect, reduces the metabolic rate of the tendon, and decreases the extravasation of blood and protein from new capillaries found in tendon injuries.[25] However, recent evidence in upper limb tendinopathy indicates that the addition of ice did not offer any advantage over an exercise program consisting of eccentric and static stretching exercises.[105]

Therapeutic ultrasound may reduce the swelling in the acute inflammatory phase and improve tendon healing.[106] Ultrasound also stimulates collagen synthesis in tendon fibroblasts and stimulates cell division during periods of rapid cell proliferation.[107]

Deep friction massage has been advocated for tendinopathy and paratendinopathy. In chronic cases, this should be accompanied by stretching to restore tissue elasticity and reduce the strain in the muscle-tendon unit with joint motion.[108]

Augmented soft tissue mobilization (ASTM) is a noninvasive soft tissue mobilization technique successfully used in chronic tendinopathy patients. This is thought to work through controlled application of microtrauma, which would increase fibroblast proliferation.[109]

If there is foot malalignment, orthoses that place the hindfoot in neutral may prove beneficial. A heel lift of 12–15 mm is classically used as an adjunct to the management of Achilles tendon pain.[110] Orthotics correction can alter the biomechanics of the foot and ankle and relieve heel pain. Therefore, orthotics are commonly used, especially in runners, with up to 75% success.[111]

Several drugs, such as low-dose heparin, wydase, and aprotinin, have been used in the management of peri- and intratendinous pathology.[112,113] Although widely used and promising, evidence of their long-term effectiveness is still unclear.

The importance of eccentric training as a part of the rehabilitation of tendon injuries was noticed in the early 1980s.[73] Eccentric strengthening of the triceps surae muscle and Achilles tendon are important to preserve function of its musculotendinous unit, possibly by restoring normal ankle joint mobility and decreasing the strain of the Achilles tendon with normal motion. Painful eccentric gastrocnemius training is effective for patients with chronic painful mid-portion Achilles tendinosis.[80] Heavy-load eccentric training is superior to concentric training in decreasing pain in chronic Achilles tendinopathy affecting the main body of the Achilles tendon. Results have been obtained using an intensive heavy-load eccentric muscle training regimen.[114,115] However, this regimen gave poor results when applied to patients with insertional Achilles tendinopathy.[116]

When painful eccentric training was compared with painful concentric exercises in patients with tendinopathy of the main body of the Achilles tendon, eccentric training was clearly superior.[114] Follow-up of patients who underwent eccentric training showed that most were satisfied and back to their previous tendon loading activity level. Interestingly, the Achilles tendon thickness had decreased significantly, and the tendon structure looked ultrasonographically more normal.[116]

Explanations for the good clinical results achieved with painful eccentric calf-muscle training are as yet uncertain. Relief of pain may result from the increased tensile strength in the tendon or possibly from stretching with "lengthening" of the muscle-tendon unit, causing less strain during ankle joint motion. Also, the eccentric training regimen is painful to perform. This type of painful loading may be associated with some kind of alteration of the pain perception from the tendon.[116]

Sclerosing Injections and Neovascularization

In Achilles tendons with chronic painful tendinopathy, but not in normal pain-free tendons, there is neovascularization outside and inside the ventral part of the tendinopathic area.[118] During eccentric calf-muscle contraction, the flow in the neovessels disappears on ankle dorsiflexion. The good clinical effects with eccentric training may be due to action on the neovessels and accompanying nerves. Also, local anesthetic injected in the area of neovascularization outside the tendon resulted in a pain-free tendon, indicating that this area is involved in pain generation.[119] The above findings are the bases for a novel management modality whereby the sclerosing substance polidocanol under ultrasound and color Doppler-guidance is injected, targeting the area with neovessels and nerves outside the tendon. Rehabilitation after sclerosing injections includes a short period of rest (1–3 days), followed by gradual increase of tendon loading activities, but no maximum loading (jumping, fast runs, heavy strength training) for the first two weeks. After this period, normal tendon loading is allowed. Most patients with good clinical results after treatments with sclerosing injections had no residual neovessels. Patients with poor results showed residual neovascularisation.[120] The results of this new intervention are promising, but longer follow-ups of clinical status and sonographic findings are needed for further evaluation.

Surgical Management

In 24% to 45.5% of patients with Achilles tendinopathy, conservative management is unsuccessful, and surgery is recommended after exhausting conservative methods of management, often tried for at least six months.[64,70,73] However, long-standing Achilles tendinopathy is associated with poor postoperative results, with a greater rate of reoperation before reaching an acceptable outcome.[121]

The objective of surgery is to excise fibrotic adhesions, remove areas of failed healing and make multiple longitudinal incisions in the tendon to detect intratendinous lesions, and to restore vascularity and possibly stimulate the remaining viable cells to initiate cell matrix response and healing.[3,7,29] Recent studies show that multiple longitudinal tenotomies trigger neoangiogenesis at the Achilles tendon, with increased blood flow.[122] This would result in improved nutrition and a more favorable environment for healing. Patients are encouraged to weight-bear as soon as possible after surgery.

Most authors report excellent or good results in up to 85% of cases, although this is not always observed in routine nonspecialized clinical practice.[121,123] It is difficult to compare the results of studies as most investigations do not report their assessment procedure.[123] Also, no prospective randomized studies comparing operative and conservative treatment of Achilles tendinopathy have been published; thus most of our knowledge on treatment efficacy is based on clinical experience and descriptive studies.

It is still debatable why tendinopathic tendons respond to surgery.[124] For example, we do not know whether surgery induces long-term revascularization, denervation, or both, resulting in pain reduction. It is also unclear exactly how longitudinal tenotomy improves vascularization.

Conclusion

Although Achilles tendinopathy has been extensively studied, there is a clear lack of properly conducted scientific research to clarify its etiology, pathology, and optimal management. Most patients respond to conservative measures, and the symptoms can be controlled, especially if the patients accept that a decreased level of activities may be necessary.[125] As the biology of tendinopathy is being clarified, more effective management regimes may come to light, improving the success rate of both conservative and operative management.

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8
Custom Foot Orthotic Management in Achilles Tendinopathy

R. Neil Humble

Introduction

Foot orthoses have long been used to manage musculoskeletal problems, including Achilles tendinopathy.[1,2] The lesion occurs when progressive or repeated microtrauma stresses the tendon beyond its ability to absorb that stress. There is probably a narrow threshold between the amount of healthy activity or exercise for adaptation, and harmful excessive loading that stresses the tendon beyond a point of normal cellular homeostasis (Fig. 8.1).

Custom foot orthotic intervention can be beneficial to these patients by decreasing the frequency and severity of the injury and the long-term disability from it. The overall success rate of foot orthotic treatment in Achilles tendinopathy is between 70% and 80%,[3,4,5] though randomized controlled trials are lacking. Recent research performed at the University of Calgary Human Performance Laboratory has begun to substantiate the biomechanical effects of custom foot orthotics on the musculoskeletal system of runners and how they may affect the loads placed on these patients.[6,7]

Many authors have made suggestions for how and why foot orthotics help control the symptoms and the poor biomechanics in Achilles tendinopathy. The most common hypothesis is that controlling the excessive compensatory pronation of the rearfoot results in a reduction of the excessive twisting of the tendon, and produces subsequent improvement.[8] A knowledge of the biomechanical stresses in the foot caused by the excessive foot pronation often seen in walking and running will allow us to understand why these stresses may cause the Achilles tendon to fail, and why controlling these stresses with custom foot orthotics will help raise the tendon's threshold for breakdown. Appropriately manufactured custom foot orthotics, coupled with other interventions, will assist in the functional rehabilitation of Achilles tendinopathy.

Biomechanics

Musculoskeletal ailments have multifactorial etiologies, including intrinsic and extrinsic factors. Biomechanically, these etiologies can be classified in three categories:

1. Training errors[9]
2. Muscle tightness and/or imbalance[10]
3. Malalignment and/or abnormal biomechanical movement[8,11]

Orthotic and footwear intervention will address the extrinsic etiologies of muscle imbalance, skeletal malalignment, and the associated abnormal biomechanics.

Muscles with their associated tendons control or produce movement. For the Achilles tendon, the movement producer is the gastrocnemius-soleus complex. This includes the independent gastrocnemius and soleus muscle origins with their combined Achilles tendon insertion distally. The anatomy of this muscle-tendon unit allows it to affect the knee joint, ankle joint, subtalar joint,

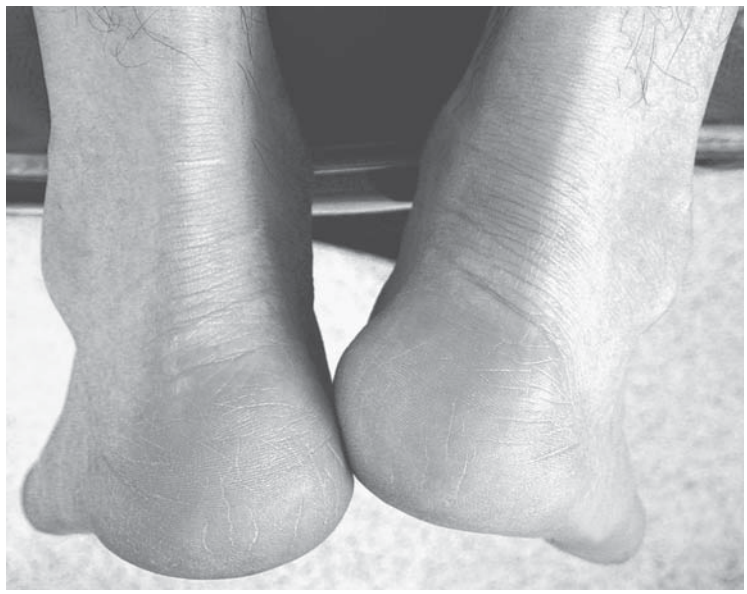


Figure 8.1. Clinical presentation of right Achilles tendinopathy.

and indirectly the midtarsal joints of the foot. (Fig. 8.2).

The ankle and subtalar joint axes allow triplanar range of motion. Practically, their predominant planes of motion with respect to the Achilles tendon are first in the sagittal plane and second in the frontal plane. The Achilles tendon muscle complex can act around these axes and planes of motion in three different modes of contraction:

1. *Concentrically:* the muscle tendon complex shortens as it develops tension.
2. *Isometrically:* the muscle tendon complex does not change in length while it develops tension.
3. *Eccentrically:* the muscle tendon complex lengthens or stretches while it develops tension.

Tendons, including the Achilles tendon, are subjected to more tension during an eccentric

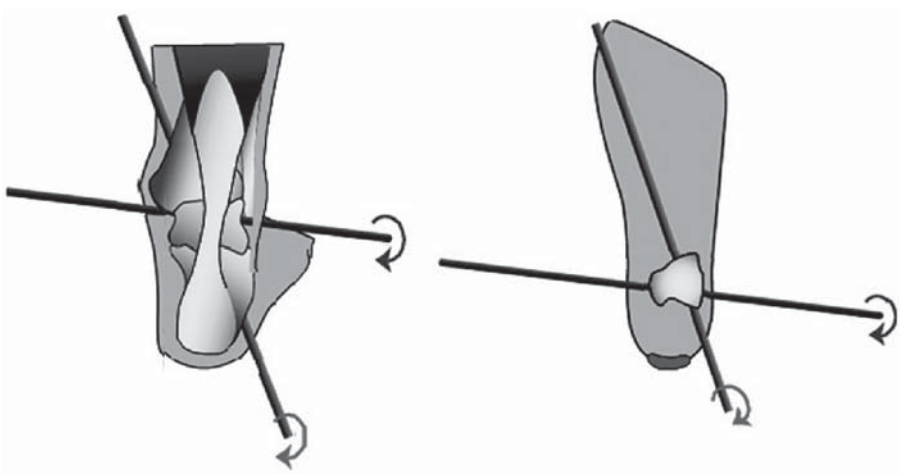


Figure 8.2. Relationship of Achilles tendon to ankle joint and subtalar joint axes.

muscle contraction and are more likely to fail while undergoing this mode of contraction.[12] Because of this, the discussion of foot orthotics and their effects on lower extremity biomechanical stress should focus primarily on the eccentric function of the gastrocnemius-soleus muscle tendon complex in the sagittal and frontal planes.

The primary collective function of the gastrocnemius-soleus complex is to restrain the forward motion of the tibia and to moderate the extension of the knee that occurs while the body transits over the planted foot in walking and running. It therefore stabilizes the leg during the stance phase of gait.[13,14]

Individually, the gastrocnemius muscle acts during the swing and stance phases of gait at the knee, ankle, and subtalar joints. It works initially during the beginning of the swing phase at the knee joint, where it contracts concentrically to flex the knee and aid in foot clearance before leg extension. At the ankle joint, it works eccentrically during the early swing phase, while dorsiflexion of the foot occurs for toe clearance. Later in the swing phase, before the upcoming contact phase, it again functions eccentrically, maintaining flexion at the knee before heel contact.

With stance-phase placement of the foot, the gastrocnemius functions to decelerate, stabilize, and then accelerate the leg during locomotion.[15] At the beginning of the stance phase of gait, it has a short-lived, modest, concentric tension to oppose the eccentric activity of the dorsiflexors as they control foot descent. After initial ankle plantar flexion at early stance phase, the ankle joint dorsiflexes, and the muscle again functions eccentrically. In the second half of the stance phase, the angle of the tibia is unchanged in walking, and the gastrocnemius-soleus complex undergoes isometric contraction.[16] As the shift from eccentric to isometric function occurs, the heel is lifted from the ground aided by forward momentum.

At forefoot loading, the gastrocnemius proximally works at the knee to decelerate internal rotation of the femur. At late midstance, it works eccentrically as the knee begins to extend, and initiates external rotation of the femur. At the end of midstance, it flexes the knee, which lifts the heel to initiate propulsion, and contributes to concentrical plantar flexion of the ankle. At the subtalar joint, its likely function is supination at late midstance, depending on the axis location.

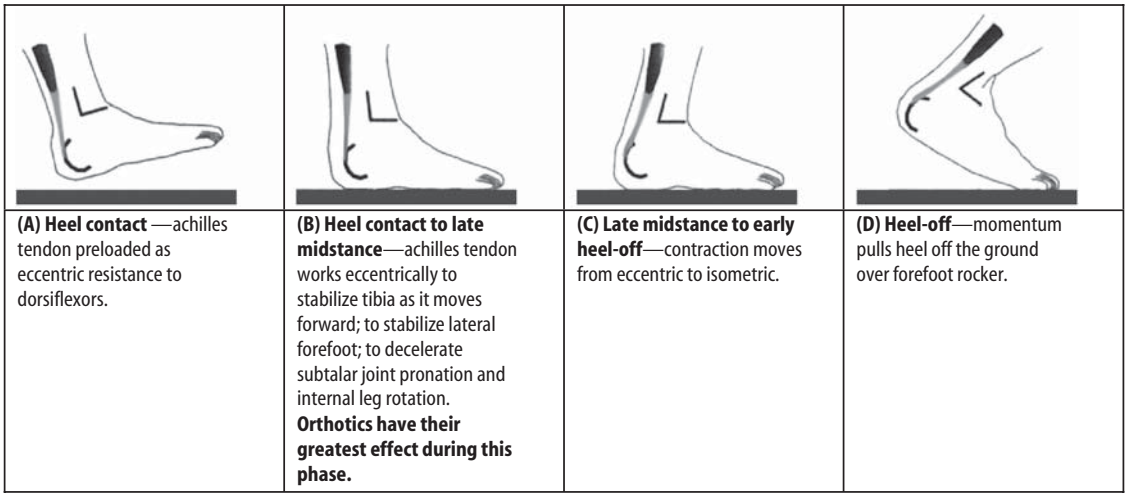
The soleus muscle works with the gastrocnemius to stabilize the lateral forefoot to the ground in midstance and to decelerate subtalar joint pronation and internal leg rotation.[15] At this moment in midstance, orthotics assist the most in decreasing eccentric stresses imposed on the muscle-tendon complex. The soleus then extends the knee indirectly by decelerating the tibia, and contributes to heel lift during propulsion by stopping ankle joint dorsiflexion as it decelerates the forward momentum of the tibia.

Electromyographic studies indicate increased activity of the gastrocnemius-soleus muscle complex from 5% to 45% of the total gait cycle.[15] The primary stress and the primary eccentric function is therefore from just before heel contact to late midstance, which is consistent with the clinical timing of failure and injury (Fig. 8.3).

Heel lift is ultimately a combination of the forward momentum of the trunk, deceleration of the forward momentum of the tibia, and active concentric knee flexion by the gastrocnemius. Because of the progressive movement of the center of gravity and the concentric contraction, at heel lift there is less strain on the Achilles tendon complex. Despite the decreased strain during this phase of gait, forefoot orthotic modifications can help facilitate smooth sagittal plane motion and decrease strain.

The gastro-soleus complex works only slightly differently during running.[16] With a heel-contact to toe-off pattern, it initially undergoes a stronger eccentric contraction before heel strike to counterbalance the sudden pull of the tibialis anterior. During stance phase, it maintains a more exertive and longer eccentric-versus-isometric contraction to stabilize the lower extremity. It also acts as a more integral part of the shock-absorbing mechanism by mediating the rate of dorsiflexion of the ankle and flexion of the knee during the stance phase of gait. Propulsion in running is aided even more than in walking with forward momentum and the use of muscles of the low back and hip.

Runners with different running styles who display different ground contact patterns, and especially midfoot to forefoot strikers, can be more predisposed to Achilles tendon injuries.



(A) Heel contact —achilles tendon preloaded as eccentric resistance to dorsiflexors.	(B) Heel contact to late midstance—achilles tendon works eccentrically to stabilize tibia as it moves forward; to stabilize lateral forefoot; to decelerate subtalar joint pronation and internal leg rotation. **Orthotics have their greatest effect during this phase.**	(C) Late midstance to early heel-off—contraction moves from eccentric to isometric.	(D) Heel-off—momentum pulls heel off the ground over forefoot rocker.

Figure 8.3. Eccentric function of Achilles tendon in heel-to-toe gait.

This change in strike pattern increases the moment arm of the forefoot with respect to the ankle joint axis, and increases the eccentric torque on the Achilles tendon. Forefoot strikers would have comparatively more Achilles tendon strain than rearfoot strikers, and will require more attention to forefoot control in their orthotic devices.

Furthermore, increasing stride length and speed can also increase Achilles tendon injuries. With increased stride length, the eccentric force on the Achilles increases from contact to forefoot loading to control forward momentum. Overstriding does not allow for a smooth gait, and there is a transient braking action at the beginning of each contact phase. Increasing speed decreases the base and angle of gait, and increases the varus striking position of the foot. This foot plant position increases the vectors of force that cause the foot to pronate more rapidly, putting more twist on the Achilles tendon, causing the gastro-soleus complex to fire eccentrically against a stronger pronation moment around the subtalar joint axis. This eccentric load can be lessened with orthotic positive cast modifications that will help to invert the end product orthotic with respect to the foot plant position. Forefoot strikers training at increased speed with an excessive stride length are predisposed to Achilles tendon injury that can be lessened with orthotic intervention.

Where malalignment and associated abnormal movement patterns are considered part of the etiology of Achilles tendinopathy, custom foot orthotics can be helpful. These devices work primarily through the midstance phase of gait to stabilize the foot, ankle, and knee and decrease the eccentric load placed on the muscle-tendon complex around these joint axes.

Shoe Modifications

Apart from custom foot orthotics, there are several appliances and shoe modifications that can be used both in and outside footwear to assist with Achilles tendinopathy. Most of these devices are used in the early stages of conservative management while waiting for a custom foot orthotic but can be added permanently to the shoe or foot orthotic as needed. These can include heel lifts, metatarsal pads or bars, shoe modifications, and night splints.

Sagittal plane assistance can be accomplished by adding a heel lift of modest height under the heel of both feet in symptomatic patients.[3] Various shock-absorbing materials of ¼ inch are routinely used to elevate the heel and decrease the strain on the tendon. In a clinical office setting, purchasing bulk sheets of such material can be cost effective. The material can then be cut to shoe size, ground

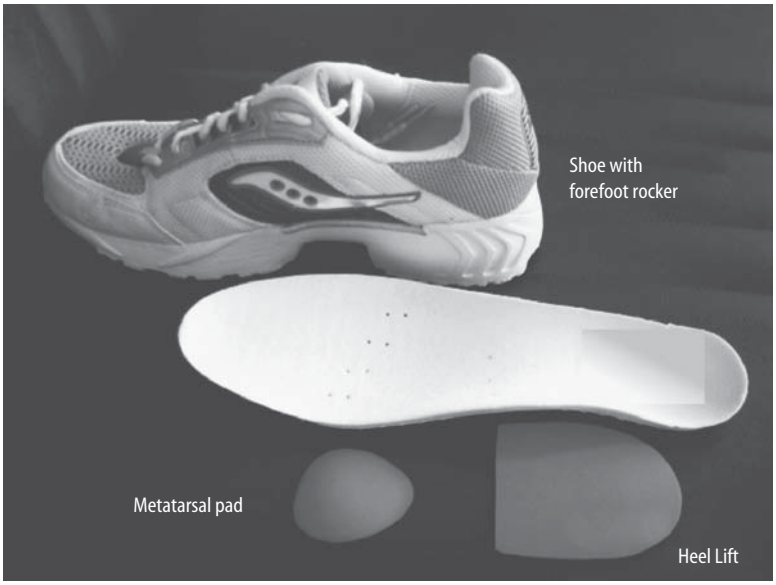


Figure 8.4. Traditional shoewear modifications.

with a taper distally, and put in the shoes under the stock insole. The lift must go in both shoes to avoid a limb-length discrepancy, producing other functional problems. The actual lift accomplished will vary depending on the material used and its compression with varying body weights and activities. A clinician should consider no more than a ¼-inch lift bilaterally.

Metatarsal pads and metatarsal bars can also be added to the stock insoles of shoes to assist in the fluid sagittal plane motion from forefoot loading to heel-off. These modifications can be purchased commercially and added to the undersurface of the conventional shoe insole just behind the metatarsal heads with contact cement or tape. The thickness tolerated can be patient specific, so there is often a need to add or subtract from a pad's thickness.

Certain shoe features and modifications to those features can assist with Achilles tendinopathy in both the long and the short term. First, shoes that are overly stiff or controlling in the midsole can exacerbate symptoms. Stiff or rigid shoes create strain on the Achilles tendon by increasing the velocity or "slap" experienced from heel contact to forefoot loading. A more compliant, neutral shoe allows a smoother transition from heel contact to forefoot loading. To further decrease sagittal plane stress on the Achilles tendon, shoes with increased heel height and good forefoot rocker will assist in the fluid sagittal plane movement of the weight-bearing extremity.

Night splints, although not a shoe modification, can be used in patients with Achilles tendinopathy. These splints hold the ankle at 90°, and are used primarily in the evening or during any period of inactivity to prevent natural gravity-assisted ankle joint plantarflexion, and thus prevent tightening of the Achilles tendon. This dorsiflexory stress may help improve symptoms in patients with plantar fasciitis.[17] These splints can be purchased commercially or simply fabricated in the office out of a 4-inch roll of fiberglass casting material, stockinet, and an elastic wrap.

Achilles tendinopathy can have a lengthy clinical course. It is important to include many short-term and long-term interventions both inside and outside the shoe to improve clinical outcomes (Fig. 8.4).

Custom Foot Orthotics

Functional custom foot orthoses were initially developed between 1954 and 1966.[18] These devices have long been prescribed to active individuals in their exercise shoes to increase comfort, to prevent

complaints of injury, or to rehabilitate from injuries. The potential benefits of functional foot orthotics as an intervention between the foot and the ground reaction forces on the lower extremity allow individuals to pursue their sports and remain functionally active.

The success rate of custom foot orthotics in lower extremity problems were first reported in 1978.[19] In that study, 46% of 180 injured runners were prescribed custom foot orthotics. Of those, 78% were able to return to their previous running program. Eggold reported that 40% of 146 runners with overuse injuries that resulted from abnormal function of the foot experienced complete relief with custom foot orthotics, and another 35% of patients reported partial relief.[20] Blake and Denton prescribed custom foot orthotics to 86 runners, and 75% reported that foot orthotics helped improve their running, 79% showed a reduction in injury rate, 43% were able to increase their mileage, 41% had decreased fatigue, and 27% were able to increase their speed.[21] Between 1978 and 1983, D'Ambrosia prescribed approximately 200 custom foot orthotic devices for runners, with improvement in 73% to 90% of them.[22] In another study, Dugan and D'Ambrosia found that 84% of 152 runners showed some improvement using custom foot orthotics.[23] Lohrer et al. reported excellent results for 34% and good results for 42% of 50 runners treated with foot orthotics.[24] Gross et al. showed a 75% success with custom foot orthotics.[25] In summary, the reported success rates of foot orthotics as a treatment for running injuries are between 70% and 90%.

Despite the reported clinical success of custom foot orthotics in patients with Achilles tendinopathy and the abovementioned positive effects on lower extremity injury, there has to date been no double-blind quality-controlled trial study or prospective outcome study performed. How, then, do custom foot orthotics act? Unfortunately, the actual insert or orthotic device used in many studies is somewhat questionable, as many of the tested devices are not those used in current practice. Few researchers have described either the design of the orthoses or the mechanism of fabrication. The term "orthotic" or "insert" has been used indiscriminately in the literature to describe everything from an over-the-counter manufacturer's insole, heel pad, or heel cup to a truly custom foot orthosis produced from a non-weight-bearing neutral suspension cast.

Recent studies have examined more closely not only the effects of a well-defined custom foot orthotic on lower extremity kinematics, kinetics, and muscle activity but also the effects of the various components of a foot orthotic on these biomechanical variables.[6] These studies have compared the effects of custom-molding, posting, and the combination of molding and posting, as well as touching on the possible effects of material intervention next to the foot in the form of top covers on the orthotics.[7,26] These recent studies have focused on populations that are homogeneous and asymptomatic. The effects on gait can be inferred in a symptomatic population with a clear understanding that further work needs to be performed to confirm these inferences in a clinical population such as those with Achilles tendinopathy.

A foot orthotic is used to maintain the foot at or near its neutral subtalar joint position throughout the walking and running gait cycles.[21] Vertical ground reaction forces are 1.3–1.5 times body weight during walking and 2–3 times body weight during running.[27] An orthotic should prevent excessive or prolonged pronation or supination of the foot and provide alignment during dynamic activities so as to affect how these forces are distributed around the subtalar joint. Excessive subtalar joint pronation causes the tibia to move forward and internally rotate as the talus adducts and plantar flexes with calcaneal eversion. Excessive speed, duration, or poor timing of this pronatory motion will stress the Achilles tendon primarily in the frontal and sagittal planes as it tries to stabilize the forward momentum of the tibia. The net effect of these factors is more pronounced while running. While running, the foot and ankle are subjected to high vertical forces, especially when the plantar aspect of the foot is positioned in a mechanically disadvantageous position in the frontal plane. The Achilles tendon is in its most efficient position only when the subtalar joint is neutral, placing the heel directly under the tibia.

Several functional effects of foot orthotics have been proposed, including aligning the skeleton, reducing impact loading, reducing joint loading, reducing muscle activity, and improving comfort.[28–32]

Of the proposed effects of foot orthotics, aligning the skeleton has been extensively studied. Earlier investigations found small and frequently insignificant differences in kinematic variables when comparing different foot orthotics.[31,33–38] These studies used different foot orthotics on a range of subjects at differing stages of foot orthotic intervention. Without accounting for the type of intervention and where it fits clinically in a homogeneous study population, it is difficult to draw conclusions on the effects of orthotic intervention.

A more recent measure of the functional success of footwear and footwear interventions such as custom foot orthotics is comfort.[28,39] Comfort is related to muscle activity, and thus can be correlated to consequent fatigue and performance.[39] Although not measured, comfort is a practical requirement used by clinicians to determine the appropriateness of foot orthotics. Thus, comfort seems to be an important functional quality of foot orthotics, and a comfort scale and protocol have been described as a possible measure of orthotic efficacy.[7,39]

There is a lack of consistency of the types of orthotics defined in both the scientific world and the clinical world. Before discussing the most recent custom foot orthotic research results, it is important to define a functional custom foot orthotic commonly used in patients with Achilles tendinopathy.

There are four essential steps in orthotic fabrication. The first is a thorough history and physical exam that discovers justification for a functional custom foot orthotic as part of a treatment program for a multifactorial problem such as Achilles tendinopathy. The second is the casting of a negative impression of the foot, followed by making a positive foot model, and finally the selection of materials and modifications often used to make an end product of a functional custom foot orthotic.

Orthotic devices are produced from a plaster cast (negative cast) of the foot that captures the foot position with the subtalar joint and midtarsal joint in their neutral positions (Fig. 8.5). Creating a proper negative cast is the most essential step when a functional custom foot orthotic is fabricated. This method was pioneered by Merton Root, DPM, in the 1960s.[40] This step in orthotic fabrication will accurately capture the angular relationship of the forefoot to the rearfoot and determine the orthotic shell shape. The practitioner is providing a model of the foot in its most functional stable position and the common beginning point for positive orthotic cast modifications including intrinsic posting.

This casting technique is accomplished by having the patient lie on a casting table either supine or prone. After applying wet plaster splints to the foot, the subtalar joint is maintained in neutral position. This is accomplished with a loading force being applied to the plantar surface of the fourth and fifth metatarsal heads in a dorsal direction producing a pronatory force around the midtarsal joint axis and locking the lateral column of the foot against a stable rearfoot. Though other methods have been used to capture the negative impression of the foot, the non-weight-bearing

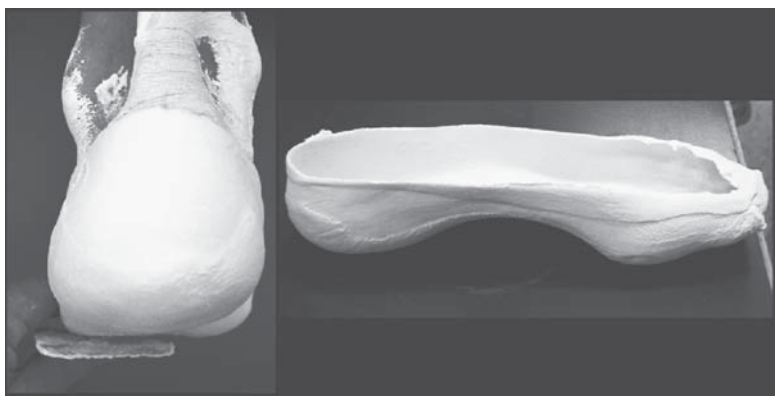


Figure 8.5. Non-weight-bearing neutral suspension cast.

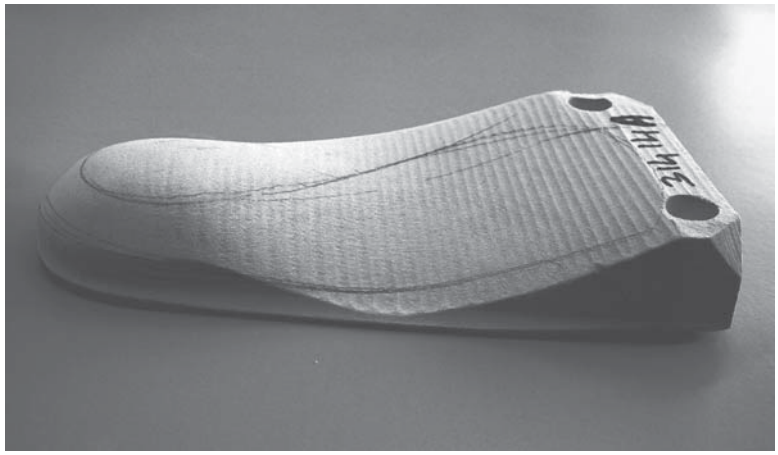


Figure 8.6. Positive model of the foot.

neutral suspension cast has been the gold standard clinically.[41]

Once the negative cast has been made, two fabrication streams can be used to produce the positive models: manual or automated. In the traditional manual stream, the positive model is produced by pouring liquid plaster into the negative cast mold. The resulting positive models are "hand-dressed" or modified applying further plaster with a putty knife. The automated stream uses a laser cast scanner to produce a computer image of the plantar surface of the foot from the plaster negative foot models, and software-created digital cast corrections are performed on a computer. Next, a computer-based numerically controlled milling machine can mill direct shells from a block of polypropylene, or can mill a positive model from a block of medium-density fiberboard (Fig. 8.6). These positive molds are used in the same fashion as traditional manually dressed models. Automated technology is thought to improve the accuracy and efficiency of the mold-dressing process, and, in some cases, the shell-fabrication process, given its high level of precision and reproducibility.

Standard cast modifications are then performed to the positive model. This includes what is thought to be the primary frontal plane controlling intervention of the foot, the anterior platform. This anterior plaster platform is added to the positive cast as a manual or digital change to intrinsically balance any congenital or acquired forefoot-to-rearfoot deformities. In the presence of any such deformities, this platform creates an orthotic shell that works to stabilize first the midtarsal joints, and second the subtalar joint by accommodating the forefoot-to-rearfoot deformities. This platform and consequent orthotic shell can be either inverted or everted (intrinsically posted) for varus and valgus forefoot deformities. The orthotic shell can often be posted up to 5° with this method, and additional posting can be performed extrinsically on the shell. Further cast modifications allow for the arch fill and heel expansion seen in weight bearing.

As Achilles tendinopathy often occurs in runners, there are two other positive cast modifications specific for these patients, who may need enhanced control. With increased speed of locomotion, the feet strike the ground in a more linear fashion directly under the body's center of mass, creating a functional running limb varus with the base of gait narrowing to zero.[42] This foot strike position increases hip adduction and results in an increased frontal plane varus attitude of the plantar plane of the rearfoot and forefoot with respect to the ground. The net effect of these biomechanical factors in running is that not only are the foot and ankle subjected to increased ground reaction forces, but the plantar aspect of the foot is positioned in a mechanically disadvantageous position, increasing the velocity of pronation. This effect may add an additional 5° of functional tibial and rearfoot varus while running as

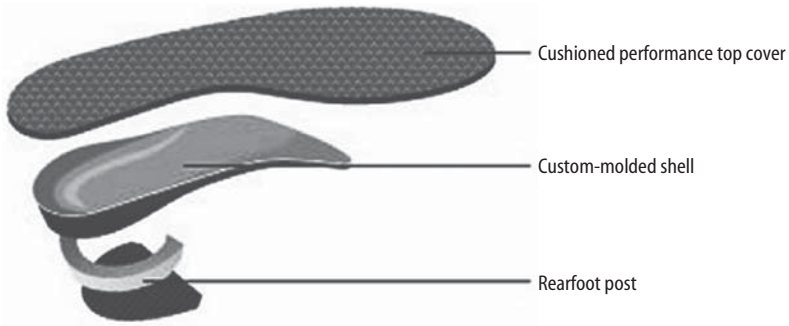


Figure 8.7. Functional anatomy of a custom foot orthotic.

compared to walking, depending on the speed. The functional varus needs to be addressed in the design of a custom foot orthosis.[42]

To accomplish the needed changes in the biomechanics of running, two cast modification approaches can be effective: the medial heel skive technique and the inverted functional foot orthotic. The medial heel skive technique involves selectively removing small amounts of the medial portion of the plantar heel of the positive cast of the foot to create a unique varus wedging effect within the heel cup of the foot orthoses.[43] The inverting technique described employs an aggressive plaster dressing of the positive cast models to accentuate shell contour.[44] This inversion is usually 10° to 20° for a moderate amount of abnormal pronation, and up to 25° for marked control.

Once the positive model has been fabricated, the actual custom foot orthoses can be constructed in many different ways. They can be modified in material and shape, and different components can be added.

In Achilles tendinopathy, the first decision in design is the selection of material and shape. Several materials can be used, including polypropylene, copolymer, and composite compounds of graphite and fiberglass. The most common material is semi-rigid polypropylene, the thickness of which is adjusted according to the patient's weight. This thermoplastic material is either digitally milled or more commonly vacuum formed over the positive model to produce the shell. The shells are then ground to shape, and a slight medial flange shape is often accommodated into the orthotic to allow better contouring to the plantar surface of the foot. The heel cup can be variable, but given the need for control in Achilles tendinopathy, a deeper shell heel cup of 18 mm is often used.

Finally, any extrinsic posts, additions, extensions, and covers are added. The type of cover material can depend on patient preference and their shoe gear type. Commonly a full-length shock-absorbing neoprene-type cover is used so that the orthotic can replace the standard insole in the shoe (Figs. 8.7 and 8.8).

The sagittal plane inefficiencies of Achilles tendinopathy may require other orthotic modifications such as heel lifts to decrease the strain at heel contact, metatarsal pads or bars to facilitate

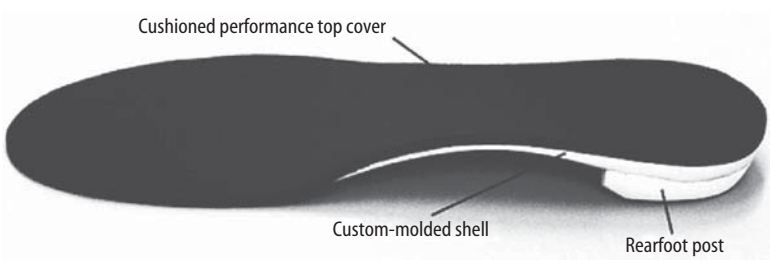


Figure 8.8. Finished custom foot orthotic.

forefoot loading, and rockers to assist with heel lift. Dananberg popularized the use of a kinetic wedge to help facilitate sagittal plane motion in patients with functional hallux limitus.[45] This application to sagittal plane motion enhancement should be considered in selected patients with Achilles tendinopathy (Fig. 8.9).

The discussion to this point has centered on the deficiency of consistent biomechanical studies of custom foot orthotics, as well as the definition and design of a proper custom foot orthotic. Recent studies on orthotic function have tried to account for these inconsistencies with an appropriate research design.[6,7]

Mundermann et al. studied 20 injury-free recreational runners classified as pronators. Each subject underwent a rigorous clinical assessment with strict inclusion and exclusion biomechanical information to make a homogeneous study population. Each subject was then casted prone using a non-weight-bearing neutral suspension casting technique. Each subject received four inserts, namely a control insert, a posted insert, a custom-molded foot orthotic, and a custom-molded and study-specific posted foot orthotic. The custom foot orthotic was consistent with what was previously described as a common active orthotic. It consisted of a polypropylene shell with a medial flange whose rigidity was adjusted to the patient's weight. Minimal cast modifications were done, and all posting was extrinsically done with ethylene vinyl acetate (EVA). The covers were in neoprene. The testing procedure included a running sandal within which one of each of the insert conditions were placed. Each subject had a 2-week adjustment time, and then participated in nine experimental sessions running at 4 m/s for a total of 430 trials per subject. Kinematic studies were performed with seven cameras and skin markers. Kinetic studies were performed with a Kistler

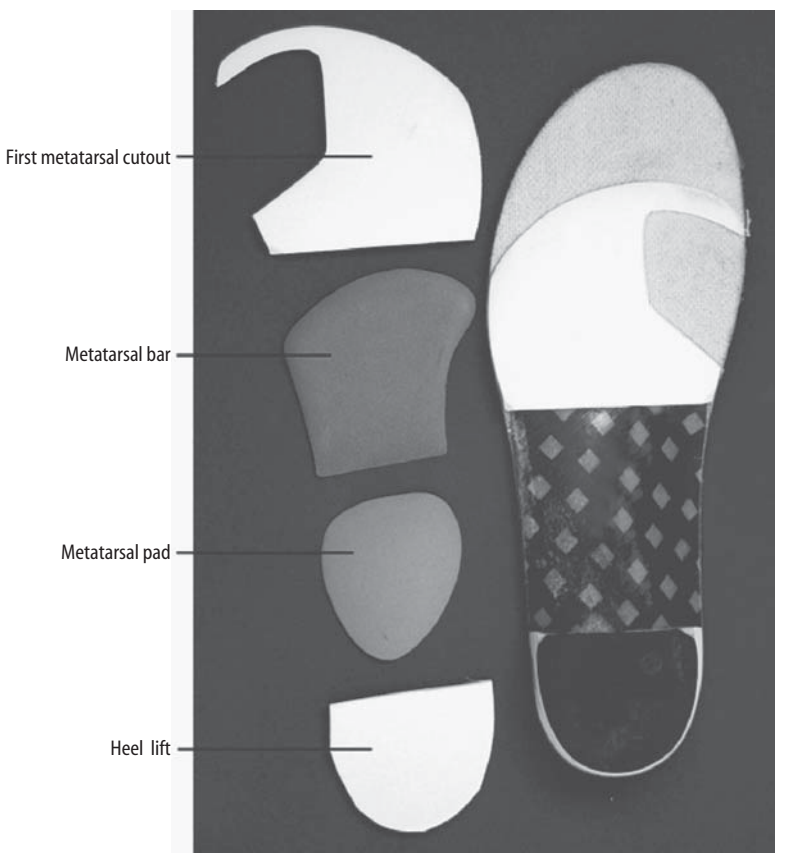


Figure 8.9. Possible custom foot orthotic additions for Achilles tendinopathy.

Table 8.1. Summary of Mundermann et al. Custom Foot Orthotic Study Results

	Posting	Molding	Molding & Posting
Max. foot eversion	↓	=	=
Max. foot eversion velocity	↓	=	=
Max. foot inversion	↓	↑	↑
Impact peak	↑	↓	↓
Vertical loading rate	↑	↓	↓
Ankle moments	↓	↓	↓
Knee moments	↑	↑	↑
Tibialis anterior intensity	=	↑	↑
Peroneus longus activity	↑	↑	↑
Comfort Rating	**−1**	***2**	**0.5**

force plate, and analyzed using inverse dynamics. EMG was performed with surface electrodes on seven lower extremity muscles and analyzed with wavelet analysis (Table 8.1). Extrinsic posting showed an improvement in frontal plane motion by decreasing maximal foot eversion and maximal foot eversion velocity. Custom molding and posting decreased impact peak, vertical loading rate, and ankle moments. The most consistent result over all subjects was the overriding effect of custom-molding in all the kinematic and kinetic data. With respect to comfort rating, custom molding was by far the superior insert.

Custom foot orthotics do affect lower extremity kinematic variables, with the greatest effects on vertical loading rates in a homogeneous asymptomatic running population.[6] Custom molding overrides the effects of posting alone, and the effects of molding can be increased by combining with posting.[6]

These recent studies on custom foot orthotics have focused on patient populations that are homogeneous and asymptomatic. The ground work that this study provided on the effects that a well-defined orthotic will have on gait can be inferred for a symptomatic population. However, further work with similar orthotics should be performed on specific clinical populations such as those with Achilles tendinopathy.

A general prescription of a custom foot orthotic that would assist in Achilles tendinopathy includes:

1. Proper diagnosis with excessive foot pronation as part of the etiology
2. Non-weight-bearing neutral suspension cast of the foot
3. Inverting cast modifying techniques for runners
4. Semi-rigid polypropylene shell
5. Extrinsic and intrinsic forefoot and rearfoot posts as needed, to help control frontal plane motion
6. Heel lifts and forefoot accommodations to assist sagittal plane motion
7. Neoprene top covers to the toes

This orthotic design will assist in improving the kinematics and kinetics of gait and reduce the eccentric load on the Achilles tendon in midstance, thereby decreasing the insult to the tendon and raising the threshold of injury.

Conclusion

Achilles tendinopathy has multifactorial etiology, and requires a multifactorial approach to management. In Achilles tendinopathy, custom foot orthotics can be part of the management plan if excessive foot pronation is part of its etiology.

Practitioners can improve their orthotic results by paying particular attention to negative casting techniques, positive cast modifications, and appropriate orthotic additions. Further research needs to be performed to ascertain how orthotics provide the clinical improvements seen.

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9
Percutaneous Surgical Management of Achilles Tendinopathy

Vittorino Testa, Murali K. Sayana, Jonathan S. Young, and Nicola Maffulli

The Achilles tendon is a common source of disability, due to continuous prolonged intense functional demands imposed on it. Athletic tendinopathy is characterized by pain and swelling in and around tendons, arising from overuse.[1] The prevalence of Achilles tendinopathy is about 11% in runners, 9% in dancers, and less than 2% in tennis players.[2–5] The condition is not only restricted to athletes: 25%–30% of patients are nonathletes who may lose a significant number of working days, producing marked financial impacts on society by adding substantially to workers compensation costs.[6] The management of Achilles tendinopathy lacks evidence-based support, and tendinopathy sufferers are at risk of long-term morbidity with unpredictable clinical outcome.[7]

Diagnosis

Patients can complain of burning pain in the posterior aspect of the calf and ankle, often worse at the beginning of a training session, and after exercise. Some patients have difficulty taking the first few steps in the morning. Pain is experienced during activities of daily living, including prolonged walking and stair climbing. Clinical diagnosis is mostly based on palpation and on the use of the painful arc sign.[8] In paratendinopathy, the area of tenderness and thickening remains fixed in relation to the malleoli when the ankle is moved from full dorsiflexion into plantarflexion. If the lesion lies within the tendon, the point of tenderness and any swelling associated with it move with the tendon as the ankle is brought from full dorsiflexion into plantarflexion. In mixed lesions, both motion and fixation of the swelling and of the tenderness can be detected in relation to the malleoli.[9,10]

Ultrasound Scan

Ultrasound (US) scan is a diagnostic aid. A real-time US machine, equipped with at least a 7.5 MHz sectorial transducer, should be used. Commercially available soft polymer echo-free material provides adequate contact between the skin and the probe, and improves the image quality by placing the tendon in the optimal focal zone of the transducer. The variables considered in the evaluation of the tendon and of the peritendinous tissues are tendon size and borders, intra- and peritendinous ultrasonographic pattern, and possible surgical sequelae.[11,12] An ultrasonographic diagnosis of tendinopathy can be made when the tendon presents altered intratendinous structure, at times with a well-defined focus. An ultrasonographic diagnosis of paratendinopathy is made when the peritenon is thickened or shows altered echogenicity.

Conservative Management

There are few scientific prospective studies, and few studies comparing different types of conservative management regimens in a randomized manner, on management of the chronic painful Achilles tendon. Conservative

management is recommended in the initial phases,[13–17] with identification and correction of possible etiological factors, at times using a symptom-related approach.

Training errors, muscle weakness, decreased flexibility, biomechanical abnormalities, and poor equipment have all been suggested as important etiological factors.[14,15,18–22] Again, these suggestions are resting on poor scientific grounds, and are to be considered as unsubstantiated hypotheses. Despite the absence of scientific evidence for an ongoing chemical inflammation inside the tendon,[23] nonsteroidal anti-inflammatory drugs (NSAIDs) are most often being used as part of the initial management.[17,18] Consequently, the use of NSAIDs to decrease existing chemical inflammation in the chronic painful Achilles tendon can be questioned. Indeed, in a randomized double-blind placebo-controlled study of 70 patients with chronic painful Achilles tendinopathy, oral piroxicam gave similar results as placebo.[24]

Peritendinous or even intratendinous corticosteroid injections are hotly debated.[15,25] Koenig et al. investigated the effects of intratendinous glucocorticoid injection for acute Achilles tendinopathy in five patients.[26] They hypothesized that the intratendinous hyperemia seen with ultrasound (US) color Doppler represented an inflammatory response. Six tendons in five patients were evaluated with grayscale US and color Doppler before and after US-guided intratendinous glucocorticoid injection. Pain and color Doppler activity decreased during a mean follow-up of 182 days. Intratendinous glucocorticoid injections seem to have a marked effect on both symptoms and color Doppler findings, which may be taken as an indication of an inflammatory component in the condition. The patients in that study presented acutely, and are not representative of chronic Achilles tendinopathy patients commonly seen in tertiary referral practice.

Partial ruptures are found after steroid injections,[27,28] and corticosteroid injections predict a partial rupture in patients with chronic Achilles tendinopathy.[27] Gill et al., in a retrospective cohort study, established the safety of low-volume injections of corticosteroids in Achilles tendinopathy when the corticosteroids are carefully injected into the peritendinous space under direct fluoroscopic visualization.[29]

Modalities such as cold therapy, heat, massage, ultrasound, electrical stimulation, and laser therapy are used.[17,30–34] These modalities are reported to be effective, but there are no well-planned scientific clinical studies that confirm their effects. The initial management most commonly consists of a multi-oriented approach, using combined rehabilitation modalities, including a combination of rest (complete or modified), medication (NSAIDs, corticosteroids), orthotics (heel lift, change of shoes, corrections of malalignments), stretching and massage, and strength training.[14,15,17,18,28,35,36]

Operative Management

Surgery is recommended for patients in whom nonoperative management has proved ineffective for at least six months; 24% to 45.5% of the patients with Achilles tendon problems fail to respond to conservative treatment and eventually require surgical intervention.[37–39] Paavola et al., in a prospective long-term follow-up study, showed that the prognosis of patients with acute-to-subchronic Achilles tendinopathy managed nonoperatively is favorable. At an average of 8 years, 94% of the patients were asymptomatic or had mild pain with strenuous exercise.

There are minor variations in surgical technique for tendinopathy.[40–47] The principles of surgical management are to excise fibrotic adhesions, to remove degenerated nodules, to make multiple longitudinal incisions in the tendon to detect intratendinous lesions, to restore vascularity, and possibly to stimulate the remaining viable cells to initiate cell matrix response and healing.[48,49] Most authors report excellent or good results in up to 85% of cases.

Management of paratendinopathy includes releasing the crural fascia on both sides of the tendon. Adhesions around the tendon are then trimmed; the hypertrophied adherent portions of the paratenon are excised.[38] In tenolysis, classically longitudinal tenotomies are made along the longitudinal axis of the tendon in the abnormal tendon tissues, excising areas of mucinoid degeneration. Reconstruction procedures may be required if large lesions are excised.[50]

When an open surgical approach is necessary, a longitudinal, at times curved, incision, with the concave part toward the tendon, is centered over the abnormal part of the tendon. A medial incision avoids injury to the sural nerve and short saphenous vein, and the curvature of the incision prevents direct exposure of the tendon in case of skin breakdown.[51,52]

The paratenon and crural fascia are incised and dissected from the underlying tendon. If necessary, the tendon is freed from adhesions on the posterior, medial, and lateral aspects. The paratenon should be excised obliquely as transverse excision may produce a constriction ring, which may require further surgery.[8] Areas of thickened, fibrotic, and inflamed tendon are excised. The pathology is identified by the change in texture and color of the tendon. The lesions are then excised, and the defect can either be sutured in a side-to-side fashion or left open. Open procedures on the Achilles tendon can lead to difficulty with wound healing due to the tenuous blood supply and increased chance of wound breakdown and infection. Hemostasis is important, since the reduction of postoperative bleeding speeds up recovery, diminishes the chance of wound infection, and diminishes any possible fibrotic inflammatory reaction.

Patient Selection

In patients with isolated Achilles tendinopathy, with no paratendinous involvement and a well-defined nodular lesion less that 2.5 cm long, multiple percutaneous longitudinal tenotomies can be used when conservative management has failed. An ultrasound scan confirms the precise location of the area of tendinopathy.

Preoperative Planning

An appropriate workup for theater should be instituted. Full history and examination and the diagnosis of Achilles tendinopathy should be established. Any relevant co-morbidity should be highlighted and managed. Although we undertake the techniques reported in this chapter under local anesthesia, there is a small chance that general anesthesia may be necessary, and therefore baseline investigations such as blood tests, ECG, and chest radiographs should be undertaken if deemed necessary. Patients should have DVT prophylaxis. Valid informed consent should be achieved prior to the operation, and the patient should be aware of risks of infection, bleeding, wound and scar problems, operation failure, and that further surgery may be required.

Percutaneous Operative Techniques

We advocate the use of two techniques for the percutaneous management of Achilles tendinopathy.[9,10]

Multiple Percutaneous Longitudinal Tenotomies

Patients are operated as day cases. The patient lies prone on the operating table with the feet protruding beyond the edge, and the ankles resting on a sandbag. A bloodless field is not necessary. The tendon is accurately palpated, and the area of maximum swelling and/or tenderness marked, and checked again by high-resolution US scanning. The skin and the subcutaneous tissues over the Achilles tendon are infiltrated with 10 to 15 ml of plain 1% Lignocaine (Lignocaine hydrochloride, Evans Medical Ltd., Leatherhead, England).

A number 11 surgical scalpel blade (Swann-Morton, England) is inserted parallel to the long axis of the tendon fibers in the marked area(s) with the cutting edge pointing cranially. Keeping the blade still, a full passive ankle dorsiflexion movement is produced. After reversing the position of the blade, a full passive ankle plantarflexion movement is produced. A variable, but probably in the region of 3-cm-long, area of tenotomy is thus obtained through a stab wound. The procedure is repeated 2 cm medial and proximally, medial and distally, lateral and proximally, and lateral and distally to the site of the first stab wound. The five wounds are closed with Steristrips (3M United Kingdom PLC, Bracknell, Berkshire, England), dressed with cotton swabs, and a few layers of cotton wool and a crepe bandage are applied.

Ultrasound-Guided Percutaneous Tenotomy

Patients are operated as outpatients. The patient lies prone on the examination couch with the feet protruding beyond the edge, and the ankles resting on a sandbag. A bloodless field is not necessary. The tendon is accurately palpated, and the area of maximum swelling and/or tenderness marked, and checked by US scanning. The skin is prepped with an antiseptic solution, and a sterile longitudinal 7.5-MHz probe is used to image again the area of tendinopathy. Before infiltrating the skin and the subcutaneous tissues over the Achilles tendon with 10 ml of 1% Lignocaine (Lignocaine hydrochloride, Evans Medical Ltd., Leatherhead, England), 7 ml of 0.5% Lignocaine are used to infiltrate the space between the tendon and the paratenon, to try and distend the paratenon and break the adherences that may be present between the tendon and the paratenon.

Under US control, a number 11 surgical scalpel blade (Swann-Morton, England) is inserted parallel to the long axis of the tendon fibers in the center of the area of tendinopathy, as assessed by high-resolution US imaging (Fig. 9.1). The cutting edge of the blade points caudally, and penetrates the whole thickness of the tendon (Fig. 9.2A, B). Keeping the blade still, a full passive ankle flexion is produced (Fig. 9.3A, B). The scalpel blade is then retracted to the surface of the tendon, inclined 45° on the sagittal axis, and the blade is inserted medially through the original tenotomy (Fig. 9.4). Keeping the blade still, a full passive ankle flexion

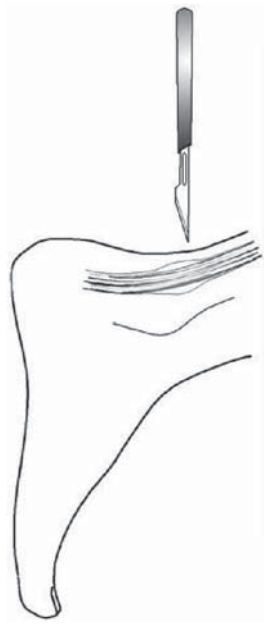


Figure 9.1. A no.11 scalpel blade inserted into the predetermined area with the sharp edge pointing caudally.

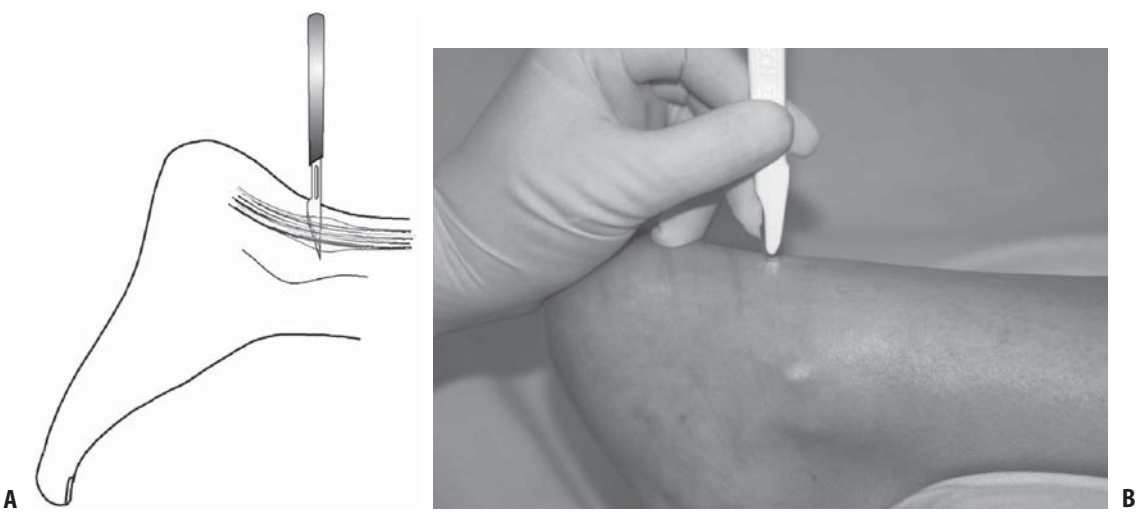


Figure 9.2. (**A, B**) The blade penetrating the whole thickness of the Achilles tendon.

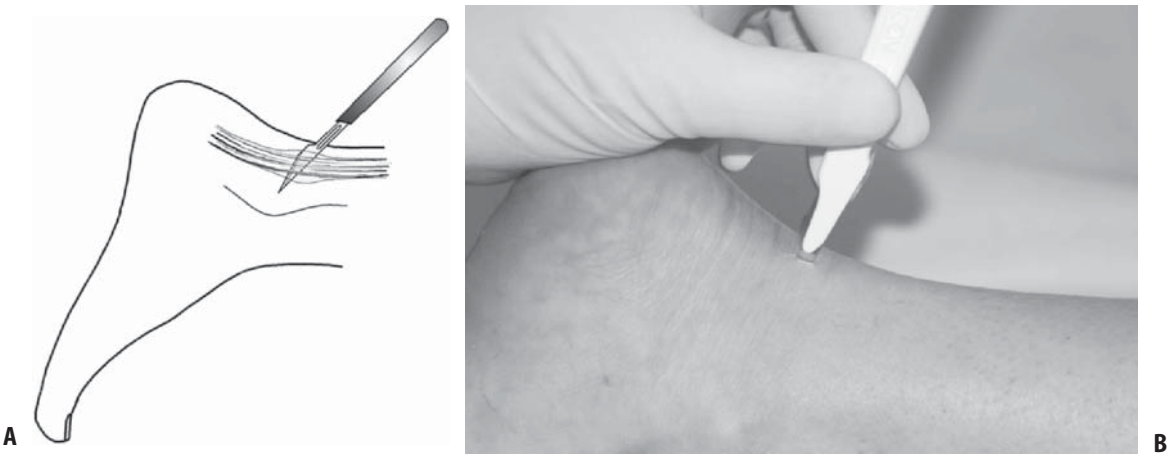
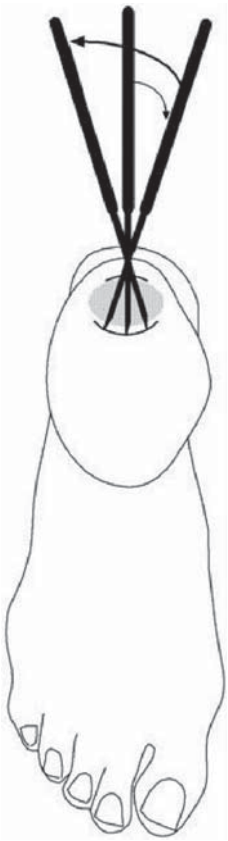


Figure 9.3. (**A, B**) Passive ankle flexion is produced.



is produced. The whole procedure is repeated inclining the blade 45° laterally to the original tenotomy, inserting it laterally through the original tenotomy (Fig. 9.4). Keeping the blade still, a full passive ankle flexion is produced. The blade is then partially retracted to the posterior surface of the Achilles tendon, reversed 180°°, so that its cutting edge now points cranially, and the whole procedure repeated, taking care to dorsiflex the ankle passively (Figs. 9.5A, B and 9.6A, B). Preliminary cadaveric studies showed that a tenotomy 2.8 cm long on average is thus obtained through a stab wound in the main body of the tendon.[9] Steristrips (3M United Kingdom PLC, Bracknell, Berkshire, England) can be applied on the stab wound, or the stab wound can be left open.[53] The wound is dressed with cotton swabs, and a few layers of cotton wool and a crepe bandage are applied.

Figure 9.4. The procedure is repeated with the blade inclined 45° medially and 45° laterally to the original tenotomy.

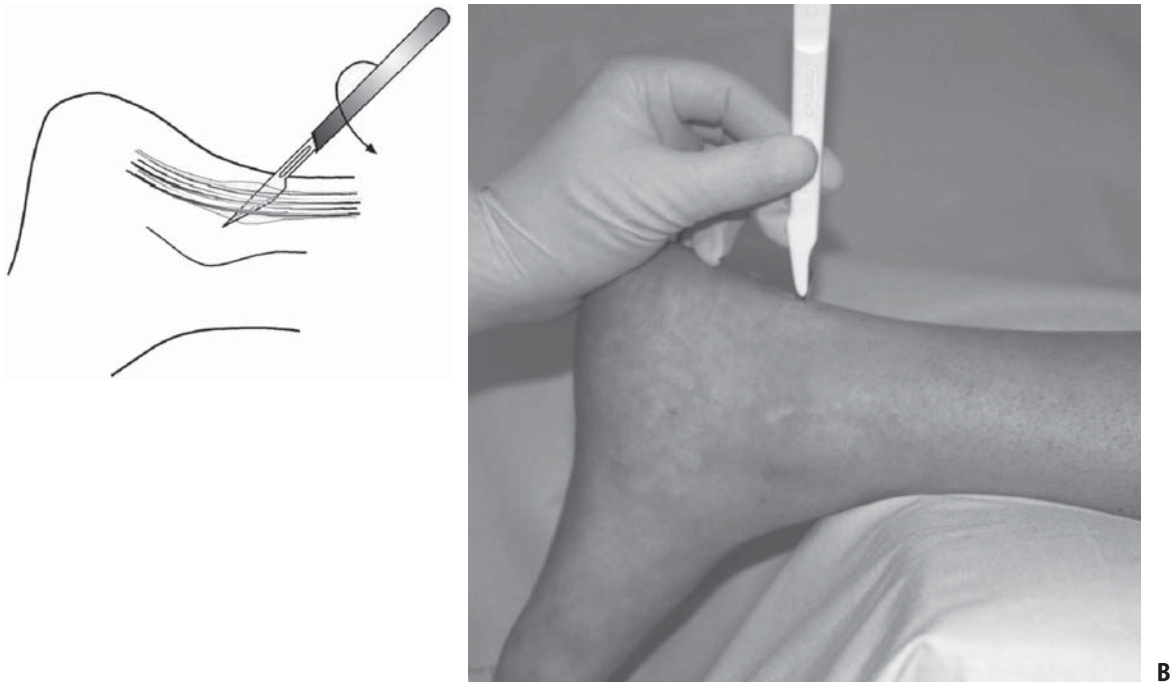


Figure 9.5. (**A, B**) The blade is reversed 180°.

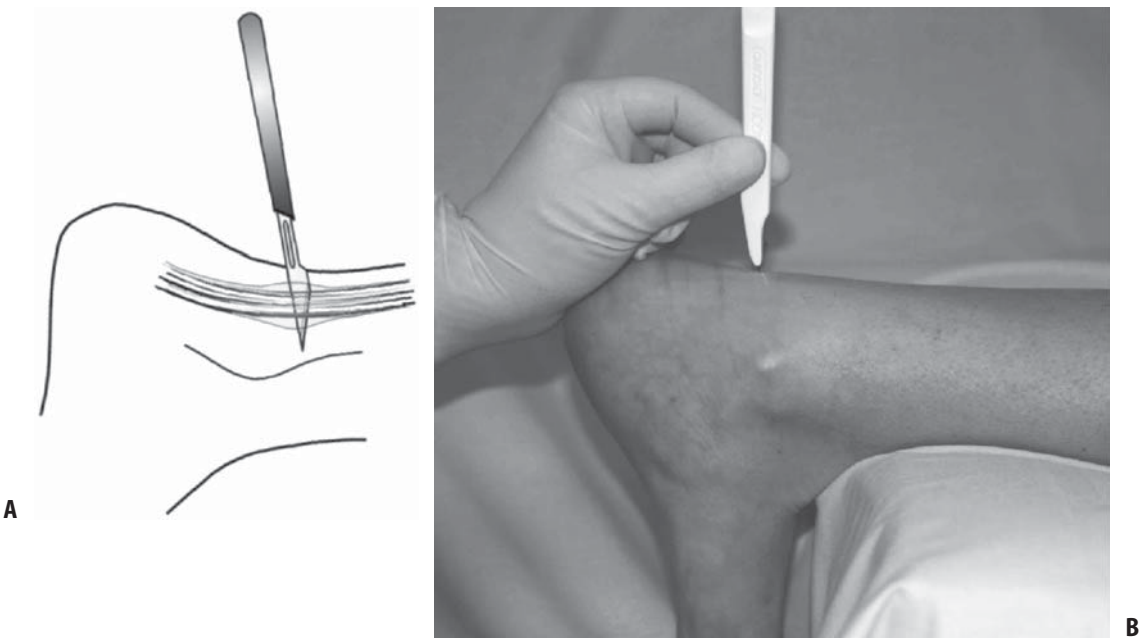


Figure 9.6. (**A, B**) The sequence of tenotomies is repeated with ankle dorsiflexion and the 45° medial and 45° lateral inclination to the initial tenotomy.

Postoperative Management

On admission, patients are taught to perform isometric contractions of their triceps surae. Patients are instructed to perform the isometric strength training at three different angles, namely at maximum dorsiflexion, at maximum plantarflexion, and at a point midway between the two.

The foot is kept elevated on the first postoperative day, and oral analgesics are given for pain control. Early active dorsi- and plantarflexion of the foot are encouraged.[54] On the second postoperative day, patients are allowed to walk using elbow crutches, weight bearing as able. Full weight bearing is allowed after two or three days, when the bandage is reduced to a simple adhesive plaster over the wounds. Stationary bicycling and isometric, concentric, and eccentric strengthening of the calf muscles are started under physiotherapy guidance after four weeks. Swimming and water running are encouraged from the second week. Gentle running is started four to six weeks after the procedure, and mileage gradually increased. Hill workouts or interval-training are allowed after a further six weeks, when return to normal training is allowed. Patients normally discontinue physiotherapy by the sixth postoperative month.

Results

Multiple Percutaneous Longitudinal Tenotomies

The procedure was performed in 52 Caucasian athletes who were training regularly, and competed up to international standards. All patients were able to weight bear on the operated limb by the third postoperative day.

At final review, at an average of 22.1 ± 6.5 (range 18–60) months from the operation, 47 patients attended. Of these, 27 patients rated themselves as excellent, 12 as good, seven as fair, and four as poor. Of the 11 patients in whom the procedure was not successful, two patients with a poor result and one with a fair result underwent a formal exploration of their Achilles tendon 9, 10, and 12 months, respectively, after the index procedure. In patients with poor results, after the peritenon was stripped through sharp dissection, a small intratendinous nodule was found and excised through a longitudinal tenotomy. The tendon was not repaired. Comparing the stab wound with the position of the nodule, either we had missed it in the first operation, or, less likely, it had developed subsequently. In the patient with a fair result, a chronic paratendinopathy with fibrous peritendinous adhesions was found. The tendon was freed by sharp dissection. In all but one of the 11 patients with a fair or poor result, the tendinopathy was associated with paratendinopathy. The three patients who underwent formal exploration resumed their sports after the open procedure. Of the remaining eight patients, five patients gave up their sports, and three were able to undertake occasional jogging.

Ultrasound-Guided Percutaneous Tenotomy

Seventy-five athletes with unilateral Achilles tendinopathy were managed with this technique.[10] In four patients, we realized at the time of the procedure that the area of tendinopathy extended beyond the length that could be covered by a single tenotomy. In another three patients, clinical and US examination of the Achilles tendon just before the procedure revealed another area of localized tendinopathy. In these seven patients, another stab wound was produced, and the tendinopathic areas were addressed.

All patients were able to weight bear on the operated limb by the second postoperative day. At final review, 51 (SD 18.2) (range 36–102) months from the operation, 63 patients attended: 35 patients were rated excellent, 12 good, 9 fair, and 7 poor. Of the 16 patients in whom the procedure was not successful, 8 had a pantendinopathy, 13 were runners (either middle distance or sprinters), and 3 were soccer players. Also, although the average interval between beginning of symptoms and operation in these patients was not significantly different from the whole group (21.6 vs. 19.2 months), these patients had received more peritendinous injections (group average: 1.3; average in the patients with a fair or poor result: 2.7), and had been less compliant with their preoperative conservative management. Nine of these 16 patients underwent a formal exploration of

the Achilles tendon 7 to 12 months after the index procedure.[10] A nodular area of tendinopathy was identified by palpation, and excised by sharp dissection. Five patients who underwent formal exploration had given up their original sports by the time of the latest review, and were able to undertake occasional jogging, swimming, and cycling.

With reference to symptomatic benefits, at the time of their best outcome, 62 subjects (83%) reported symptomatic benefit from surgery and had returned to sports. The median time to return to sports was 6.5 months (range 11 weeks to 14 months), with only two of the subjects who had returned to sports doing so after 10 months. At final follow-up, 55 of the 63 patients followed up at an average of 51 months from the operation continued to report symptomatic benefit, and 47 of 63 were still able to practice sports.

Complications

Multiple Percutaneous Longitudinal Tenotomies

Four patients developed a subcutaneous hematoma from one of the stab wounds, and a further patient suffered from a superficial infection of one of the stab wounds. This was treated by oral antibiotics for five days, and healed uneventfully. Three patients complained of hypersensitivity of the stab wounds. They were counseled to rub hand cream over the stab wounds several times a day, and were asymptomatic by six postoperative weeks. One patient developed a hypertrophic painful scar of three of the five stab wounds. These were injected with corticosteroids, and, when last interviewed, the patient reported a good functional and cosmetic result. At final review, only three patients were not pleased with the appearance of the operation scars.

Ultrasound-Guided Percutaneous Tenotomy

Five patients developed a subcutaneous hematoma, probably due to a cut in one of the superficial veins crossing the posterior aspect of the ankle. All such hematomas resolved with a pressure bandage, which was removed three to seven days later. In another patient, a superficial infection developed one week after the percutaneous longitudinal tenotomy, and was treated by oral administration of 500 mg of erythromycin three times a day for one week, recovering uneventfully. At the six-week follow-up appointment, eight patients complained of hypersensitivity of the stab wound scar when kneeling down. They were counseled to rub hand cream over the scar several times a day, and became asymptomatic three to six weeks from the prescription. No hypertrophic or keloid scars were noted at the latest follow-up. No patient complained of the appearance of the scar. Eleven patients complained of morning stiffness of the ankle in the early postoperative period, but they did not report the complaint at the six-month evaluation.

Conclusions

The management of Achilles tendinopathy aims to return the patient to a level of activity similar to that prior to acquiring tendinopathy in the shortest possible time without significant residual pain. Physiotherapy and conservative measures should constitute the first form of management.

If conservative measures fail, percutaneous longitudinal tenotomy is simple, requires only local anesthesia, and can be performed without a tourniquet. If postoperative mobilization is carried out early, preventing the formation of adhesions, this will allow the return to high levels of activity in the majority.

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10
Open Surgery for Achilles Tendinopathy

Murali K. Sayana, Louis C. Almekinders, and Nicola Maffulli

Introduction

Tendinopathy can affect the Achilles tendon in several regions of the tendon. One particularly common site is the main body of the tendon. Midsubstance or intratendinous lesions were first recognized in the 1970s.[1] Both surgical and nonsurgical management of Achilles tendinopathy have been emphasized. Cohort studies in the past 30 years have indicated that the etiology and response to various management modalities varies depending on the type of Achilles tendinopathy.[2–5] Therefore, outcome studies have generally reported the results of management for intratendinous tendinopathic lesion separately from paratendinous and insertional problems. Finally, intratendinous lesions and partial tears may represent similar problems on different ends of the tendinopathy spectrum.[6,7] There is no uniform agreement on the definition of a partial tear. However, descriptions of partial tears often suggest that they may result from intratendinous lesions that progressed to the surface of the tendon.

Evaluation and Indications for Management

The initial evaluation of Achilles tendon pain should include a careful history and physical examination of the character and location of the pain. Frequently, this will allow determination of the type of Achilles tendinopathy responsible for the pain. Pain and tenderness directly at the Achilles tendon insertion on the calcaneus is indicative of insertional tendinopathy. Pain 2–5 centimeters proximal to the Achilles tendon insertion is consistent with midsubstance tendinopathy and/or paratendinopathy. Since management and outcome are different for these two conditions, it is important to determine which is present and responsible for the pain. Paratendinopathy can be associated with a more diffuse swelling of the tendon sheath and occasionally some crepitus. Midsubstance tendinopathy frequently creates a more focal, firm swelling of the tendon that is tender on direct palpation (Fig. 10.1). Occasionally it can be difficult to determine the presence of both conditions on physical examination. In addition, both conditions can at times coexist.[8]

Imaging studies can help to diagnose midsubstance tendinopathy. Both MRI and ultrasound can show the intratendinous lesions.[9,10] However, intratendinous lesions on imaging studies may not necessarily correlate with clinical symptoms, as such tendon lesions can exist in nonsymptomatic tendons.[11] Therefore, one should correlate imaging findings with history and physical examination in each patient. Based on this, it could be argued that imaging studies are not required to make the diagnosis of midsubstance tendinopathy. However, if surgical management is contemplated, these studies can be helpful. The goal of surgery is to identify and, in most instances, excise the pathologic tissue to elicit an acute healing response. In midsubstance Achilles tendinopathy, the pathologic tissue is generally surrounded by normal tendon. Preoperative localization of the lesion through imaging studies will allow the

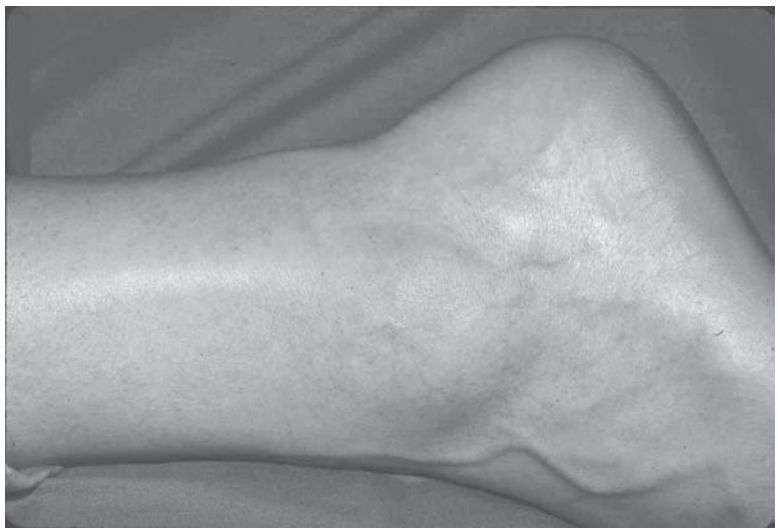

Figure 10.1. Swelling of the Achilles tendon 4 cm proximal to the insertion.

surgeon to minimize surgical dissection, and ensure that all pathologic tissue is addressed.

Most patients with confirmed midsubstance tendinopathy should have a trial of nonsurgical management as described previously. The results of nonsurgical management have traditionally been somewhat unpredictable. Angermann[12] reported a 35% failure rate at longer-term follow-up with nonsurgical management. Paavola[4] found that 29% of patients eventually had surgical management in a cohort of patients with 8-year follow-up. More recently, new concepts in the nonsurgical management of Achilles tendinopathy have been introduced.[13,14] These methods may be more effective, and it is possible that the failure rate will be reduced. However, failure of nonsurgical management remains a possibility. The definition of failure is not universally agreed upon. Most authors suggest at least 3 to 6 months of nonsurgical management should be attempted. Even if symptoms are still present at that point, surgical management may not be indicated. Frequently, pain is at least reduced.[15] Some patients may have developed strategies to manage their symptoms by activity-level modifications. Athletes frequently have adapted their training schedules to minimize symptoms. If the patients are willing to accept these changes, there appears to be no medical risk in allowing them to continue to train and compete with their current symptoms. In particular, the risk of complete rupture is low, as most patients with ruptures have no prodromal symptoms.

Patients will also need to understand the risks, length of recovery, and success rate following surgical management to make an informed decision about further surgical management for their midsubstance Achilles tendinopathy. Saxena[16] reported that return to activity in patients undergoing surgery for tendinopathy of the main body of the Achilles tendon was longer than in those who required isolated excision of the paratenon, with mean return-to-activity in patients with surgery for tendinopathy of the main body of the Achilles tendon of 13.2 weeks. Schepsis[17] indicated that return to full activities usually takes 5 to 6 months postoperatively.

Outcome and success rates have been reported in several cohort studies. However, the scientific quality of these studies is frequently low, making a true assessment of the value of surgical management for this problem more difficult.[18] Failures of surgery are not uncommon. Schepsis[19] reported a success rate of 67% in the surgical management of midsubstance tendinopathy. The same report includes success rates of 87% for paratendinopathy and 86% for insertional problems. Nelen[20] reported overall 80% good and excellent results with surgical management of midsubstance problems.

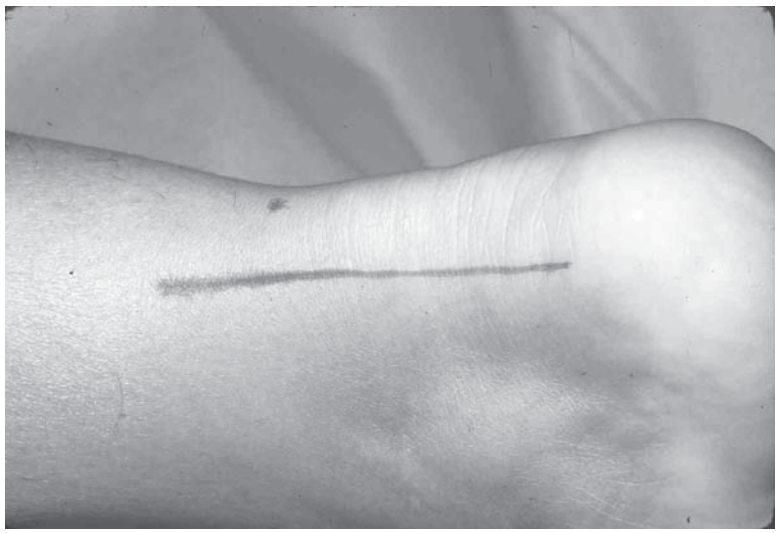

Figure 10.2. Incision for open surgery: 1 cm medial to the midline. Avoids sural nerve, short saphenous vein, and the scar is away from the shoe counter.

Surgical Technique

The prone position allows excellent access to the affected area. Alternatively, the patient can be positioned supine with a sandbag under the opposite hip and the affected leg positioned in a figure-of-four position. Open surgery for tendinopathy of the main body of the Achilles tendon involves a longitudinal incision. Generally the incision is made on the medial side of the tendon to avoid injury to the sural nerve and short saphenous vein (Fig. 10.2). A straight posterior incision may also be more bothersome with the edge of the heel counter pressing directly on the incision. Preoperative imaging studies can guide the surgeon in the placement of the incision. The skin edge of the incision should be handled with extreme care throughout the procedure, as wound healing problems are possible and potentially disastrous. The paratenon is identified and incised (Fig. 10.3).

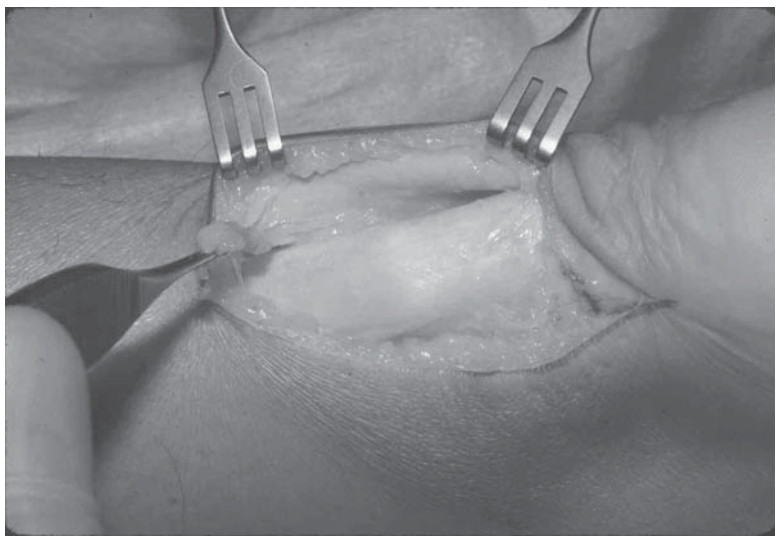

Figure 10.3. Paratenon and the Achilles tendon exposed.

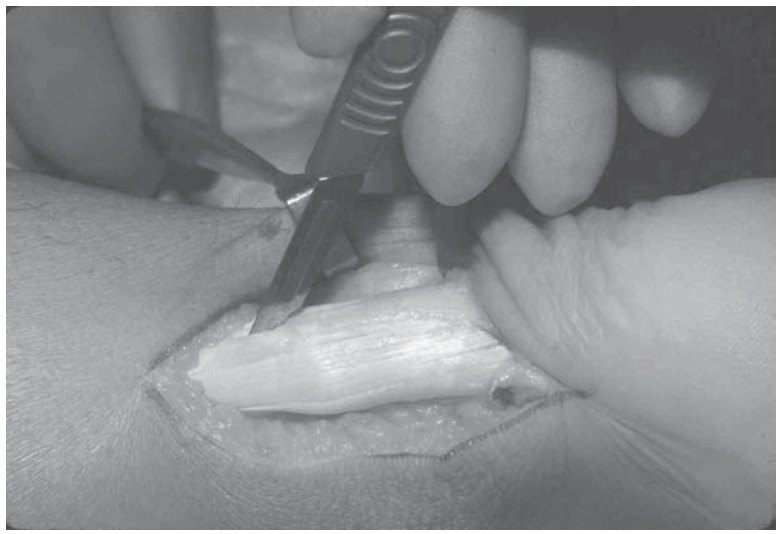

Figure 10.4. Incision in the tendon along the lines of tendon fibers.

In patients with evidence of coexisting paratendinopathy, the scarred and thickened tissue is generally excised. Care should be taken to minimize dissection and excision on the anterior side of the tendon. The fatty tissue anteriorly is thought to contain much of the vascular supply to the tendon. Based on preoperative imaging studies, the tendon is incised sharply in line with the tendon fiber bundles (Fig. 10.4). The tendinopathic tissue can be identified as it generally has lost its shiny appearance, and frequently contains disorganized fiber bundles that have more of a "crabmeat" appearance. This tissue is sharply excised (Fig. 10.5). The remaining gap can be repaired using a side-to-side repair. If significant loss of tendon tissue occurs during the debridement, consideration could be given to a tendon augmentation or transfer. A tendon turn-down flap has been described for this purpose.[20] With a turn-down procedure, one or two strips of tendon tissue from

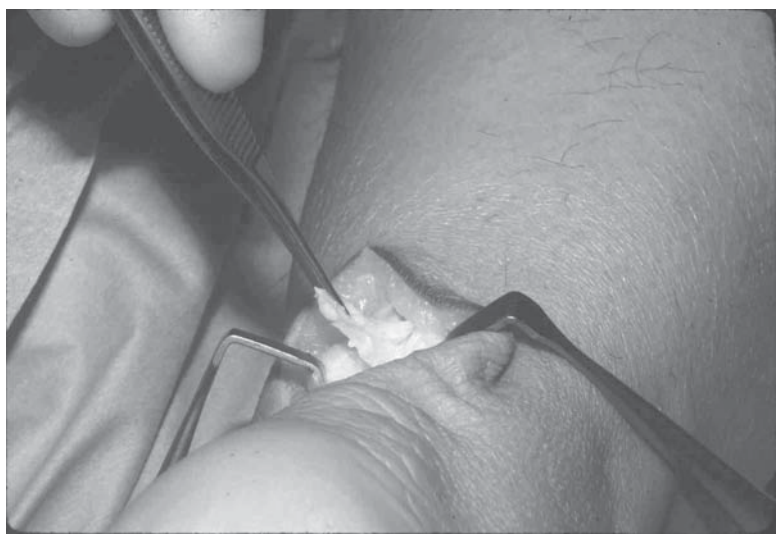

Figure 10.5. Tendinopathic tissue being sharply excised.

the gastrocnemius tendon is dissected out proximally while leaving the strip attached to the main tendon distally. It is then flipped 180° and sewn in to cover and bridge the weakened defect in the distal tendon. A plantaris weave has also been reported for this purpose.[19] The plantaris tendon can be found on the medial edge of the Achilles tendon. It can be traced proximally as far as possible and detached as close as possible to the muscle tendon junction to gain as much length as possible. It can be left attached distally to the calcaneus, looped and woven through the proximal Achilles tendon, and sewn back onto the distal part to the tendon. Alternatively, the plantaris can be detached distally as well and used as a free graft. Finally, transfer and augmentation with the flexor hallucis longus tendon has been reported.[21]

Postoperative Regimen

The rehabilitation program following open surgery for midsubstance tendinosis has not been studied in a controlled manner. Therefore, the regimen mainly depends on the experience and preference of the surgeon. Most reported programs emphasize early motion and avoidance of prolonged immobilization. A period of initial splinting and crutch walking is generally used to allow pain and swelling to subside. In addition, wound healing complications are difficult to manage and an initial period of immobilization may promote skin healing. After 14 days, the wound is inspected and motion exercises are initiated. Alfredson[22] studied the effect of more prolonged immobilization on the calf muscle strength after Achilles tendon surgery. No significant effects were found when comparing 2 weeks versus 6 weeks of immobilization. However, he did not report on the overall outcome with regard to the tendinopathic pain. Many basic science studies have shown the beneficial effects of motion on soft tissue healing. Therefore, the patient is encouraged to start daily active and passive ankle range-of-motion exercises. The use of a removable walker boot can be helpful during this phase. We do not limit weight bearing according to the degree of debridement needed at surgery, and encourage early weight bearing. However, extensive debridements and tendon transfers may require protected weight bearing for 4 to 6 weeks postoperatively. Ultrasound and scar massage are frequently used, although the exact value of these modalities is unclear. After 6 to 8 weeks of mostly range-of-motion and light resistive exercises, initial tendon healing will have been completed. More intensive strengthening exercises are started, gradually progressing to plyometrics and eventually running and jumping. However, most patients do not tolerate sports-specific exercises until 4 to 6 months postoperatively. It can take as much as 6 to 12 months before athletes feel fully recovered from this procedure.

Summary

Open surgery for midsubstance tendinopathy of the Achilles tendon can be considered if prolonged nonoperative management fails. However, patients should be informed of the potential failure of the procedure, risks of wound complications, and sometimes prolonged recovery period. The surgical procedure is relatively straightforward, but on occasion may require concomitant transfer of tendon tissue to reinforce the weakened tendon. Rehabilitation is focused on early motion and avoidance of overloading the tendon in the initial healing phase.

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11
Flexor Hallucis Longus Augmentation for Insertional or Noninsertional Achilles Tendinopathy

Gregory P. Guyton

Simple surgical debridement of the Achilles tendon, either for insertional or noninsertional tendinopathy, inevitably gives rise to a quandary: What if there is very little normal tissue left (Fig. 11.1)? In these cases, augmentation of the remaining tendon should be considered. A wide variety of potential options are available for this purpose, including the use of free allograft or autograft, local graft from the plantaris, reshaping of the Achilles itself by a V to Y transfer or turn-down procedure, a peroneus brevis tendon transfer, or a flexor hallucis longus (FHL) tendon transfer. Transfer of the FHL is attractive: the tendon is almost invariably free from tendinopathy itself, is anatomically convenient, and can be harvested with only minimal functional loss in nonathletic patients. We point out that the same technique can be used in the management of chronic ruptures of the Achilles tendon.

Indications

When Should Augmentation Be Considered?

No clear consensus exists as to the precise degree of tendon debridement that should warrant augmentation. In insertional tendinopathy, debridement of up to 50% of the tendon insertion is considered unlikely to result in major mechanical compromise of the tendon insertion. Kolodziej et al. performed a biomechanical study involving progressive release of the Achilles tendon insertion in 25% increments followed by application of a cyclic load of three times body weight in a testing jig. Debridement of 50% or less was not associated with rupture if performed in a superior-to-inferior direction.[1]

Mechanics aside, the addition of more plantarflexion strength may be appropriate even in cases of milder tendinopathy. Just as the threshold for determining when augmentation is appropriate is nebulous, so is the limit of debridement. A variety of subjective approaches have been described, the most radical of which was recently advocated by Martin et al.[2] In an effort to make insertional Achilles debridement more predictable, they adopted a technique of complete excision of the distal 5 cm of the Achilles, followed by midfoot harvest of the FHL and spanning of the defect. Despite the dramatic nature of the surgery, patient satisfaction was remarkably high, with 38 of 44 patients fully satisfied.

In the nonathletic patient, the clinical deficit resulting from the harvest of the FHL seems to be remarkably limited. Most patients in all the published series are over 50; it is unclear if the loss of hallucal push-off would be more noticeable in a younger population to whom the ability to sprint or jump is more highly valued. Coull et al. analyzed morbidity following FHL transfer including harvests both from the midfoot and the posterior aspect of the ankle.[3] A trend toward decreased loading of the hallux on the operative side was noted on force plate pressure measurements, and a clinically apparent decrement in flexion strength of the hallux was noted on physical exam. Nevertheless, clinical symptoms were essentially absent, and no transfer metatarsalgia was present. This result has been borne out

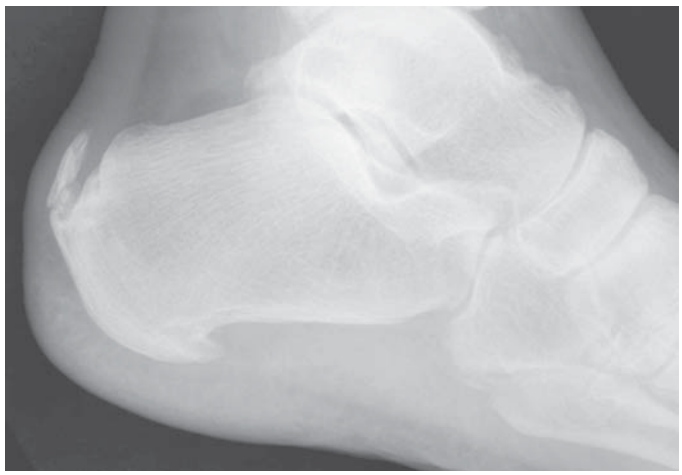

Figure 11.1. A chronic ossific insertional Achilles tendinopathy.

in other follow-up studies of the procedure; the success of the surgery may well outweigh any perceived deficits with the toe.

Surgical Technique

Harvest Site

Two anatomic options are available for harvesting the FHL for transfer, a long harvest at the knot of Henry and a short harvest behind the ankle through the same posterior incision used to debride the Achilles. The original descriptions of FHL augmentation of the Achilles utilized a medial midfoot incision to harvest the FHL at the level of the knot of Henry as the tendon passes over the flexor digitorum longus. This approach provides a substantial length of tendon that can then be doubled back upon the Achilles to bridge any defects. Tashjian et al. estimated that approximately 3 cm more tendon length can be obtained from the midfoot, but careful dissection can yield even more.[4] From a surgical standpoint, harvesting the tendon in the midfoot provides both advantages and disadvantages. It does involve an extra incision and the knot of Henry can rest remarkably deep within the foot. Nevertheless, the approach is along a natural fascial plane and is well-tolerated. Many patients will have one or more large juncturae connecting the flexor digitorum longus to the FHL as the two tendons cross each other. These usually have to be dissected free and released to free the FHL for transfer.

Surgically approaching the midfoot and the Achilles simultaneously can present intraoperative positioning challenges. It can be difficult to work on the medial side of the midfoot with the patient prone. As a compromise, most patients have sufficient external rotation of the leg to allow the Achilles to be approached with the patient in a semilateral position with the contralateral pelvis elevated. Because of the positioning issues, harvesting the FHL in the midfoot should be included as a possibility in the preoperative plan when considered.

The additional length of the FHL available through the midfoot harvest allows multiple options regarding its insertion into the calcaneus, including straight bone tunnels, convergent bone tunnels, suture anchors, or interference screws. Obtaining enough length from a posterior FHL harvest can be a challenge, and the FHL sheath must be incised as distally as possible to cut the tendon as it begins to pass underneath the sustentaculum tali. It is rare that sufficient length can be obtained posteriorly to double the tendon back upon itself, and fixation into the calcaneus is usually limited to suture anchors or interference screws. Additionally, the neurovascular bundle runs just superficial to and crosses over the FHL as it passes behind the ankle. No neurovascular injuries have been reported with the technique,

but its proximity remains a concern for surgeons learning the technique. The anatomic margins of safety are greater in the midfoot.

The posterior approach does not afford the option of tenodesing the distal stump of the FHL to the FDL, whereas this is easily accomplished, if desired, in the midfoot. Advocates of the technique argue that the juncturae connecting the FDL and FHL more distally provide a natural tenodesis in most patients. Some have advocated that tenodesis does not correlate with function in any case and routinely omit it as part of the procedure.

There is no clear consensus on the optimal harvest location for the FHL. The only comparative series of any kind is the previously mentioned analysis of hallux morbidity. Coull et al. did distinguish between patients who underwent harvest behind the midfoot and those who had posterior ankle harvest procedures, but it was not a primary outcome variable and the two patient populations represented a change in routine technique rather than randomization. No discernable differences in complications between the techniques were noted. Outcomes with regard to the Achilles itself were not analyzed. Hopefully, ongoing studies will address the issue and provide some guidance, but, for the time being, both points of view have merit and come down to the surgeon's choice. The advantages and disadvantages of the two harvest sites are summarized in Table 11.1.

Stepwise Procedure

The Posterior Approach

1. The patient is positioned according to the surgeon's preference. For an anticipated harvest from the midfoot, supine positioning with a large sandbag bump under the contralateral hip allows the leg to be externally rotated to access the medial aspect of the foot (Fig. 11.2).

2. A direct midline incision is usually utilized (Fig. 11.3). This incision follows the natural border between the angiosomes, or arterial vascular territories, of the leg.[5] The risk of leaving a small segment of devascularized skin is minimized by this technique. If it is anticipated that the distal Achilles is to be completely split and detached, a simple direct midline incision is used all the way down to the Achilles insertion (Fig. 11.4). If, however, the case involves a noninsertional tendinopathy or detachment of the Achilles is not anticipated, the distal 3–4 cm may veer across to the medial side to allow improved access to the medial aspect of the calcaneal tuberosity (Fig. 11.5).

3. Debridement of the Achilles tendon is then undertaken. If a supplementary length procedure is necessary, such as a V–Y lengthening or a central third turn-down, these can be accomplished at this time.

4. If a Haglund's deformity or insertional ossification is taken down, care should be taken to

Table 11.1. Comparison of Midfoot versus Posterior Ankle Harvest of Flexor Hallucis Longus Tendon

	Midfoot (Knot of Henry) Harvest	Posterior Ankle Harvest
Posterior Ankle Harvest Advantages	Midfoot harvest is difficult with the patient in a prone position.	Accomplished through the same incision as the Achilles debridement.
	Requires a separate midfoot incision, although it is generally well-tolerated.	Single incision technique.
	Requires the tendinous juncturae connecting the FHL and FDL to be released.	No juncturae are present behind the ankle.
Midfoot Harvest Advantages	Allows easy tenodesis of the distal FHL stump and the FDL if desired.	Tenodesis of the tendons is not possible.
	Dissection is carried out well away from the neurovascular bundle.	The tibial nerve and branches cross superficially over the FHL behind the ankle.
	Between 4 and 6 cm of additional tendon is available to double back and make up defects in the distal Achilles.	Sufficient tendon is available only to provide motor augmentation of the Achilles. No direct grafting of the Achilles can be achieved.
	A variety of fixation methods of the FHL into the calcaneus can be used, including simple bone tunnels.	Interference screws or suture anchors are usually required to secure the short length of FHL to bone.

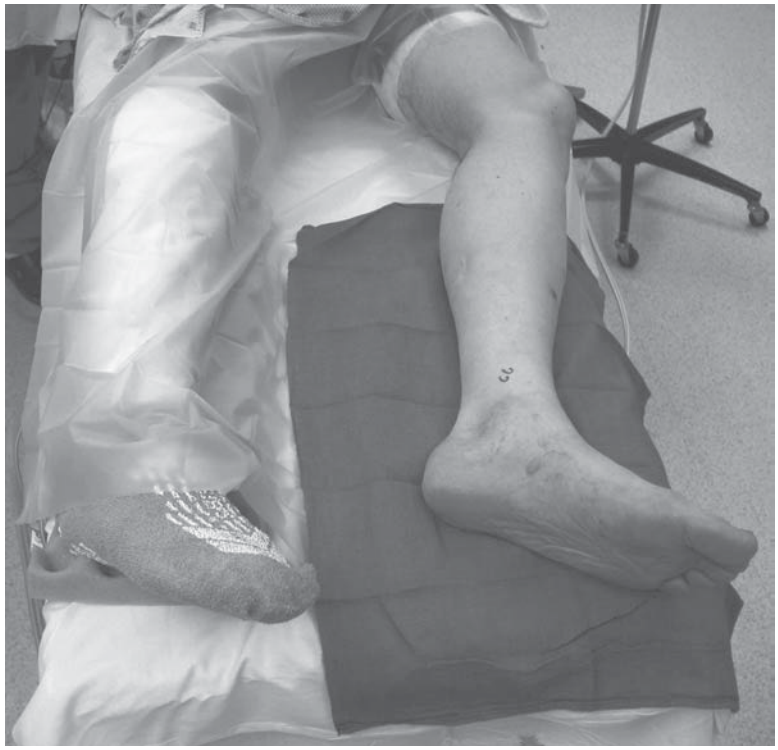

Figure 11.2. The patient is positioned supine with a large sandbag under the contralateral hip to allow simultaneous access to the Achilles and medial midfoot.

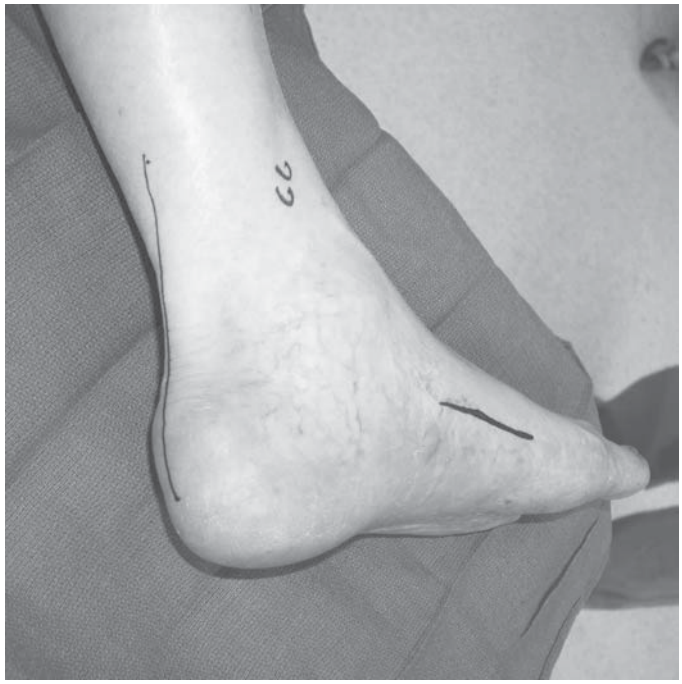

Figure 11.3. A direct midline posterior incision is used. The midfoot incision can be only 4 cm long, and is centered at the plantar border of the first tarsometatarsal joint.

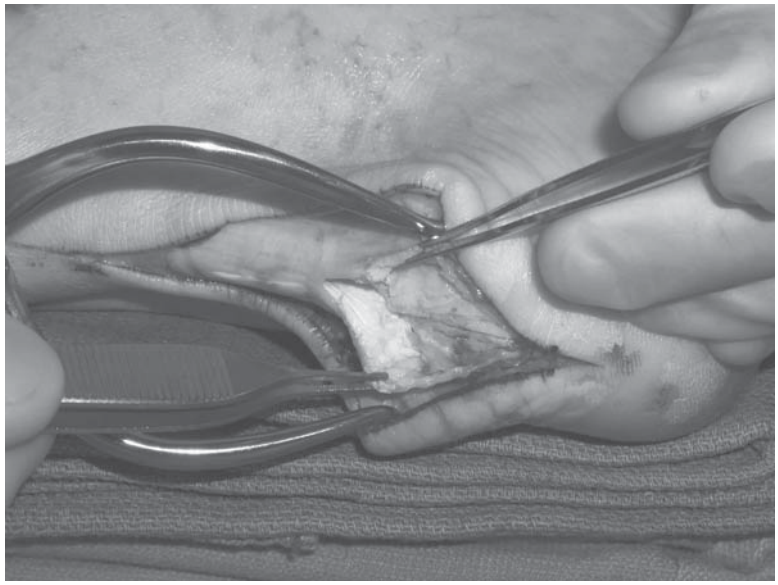

Figure 11.4. The tendon itself is split in the midline and the two halves are elevated off the ossific spur.

avoid such excessive resection that bone tunnels for the FHL augmentation could be compromised (Figs. 11.6 and 11.7).

5. The fat pad anterior to the Achilles is split directly in the midline, and the fascia investing the deep posterior compartment of the leg is exposed. This is then split beginning approximately 5–7 cm above the ankle mortise (Fig. 11.8). The FHL at this level comes well across the midline, and, provided that the dissection remains strictly on the

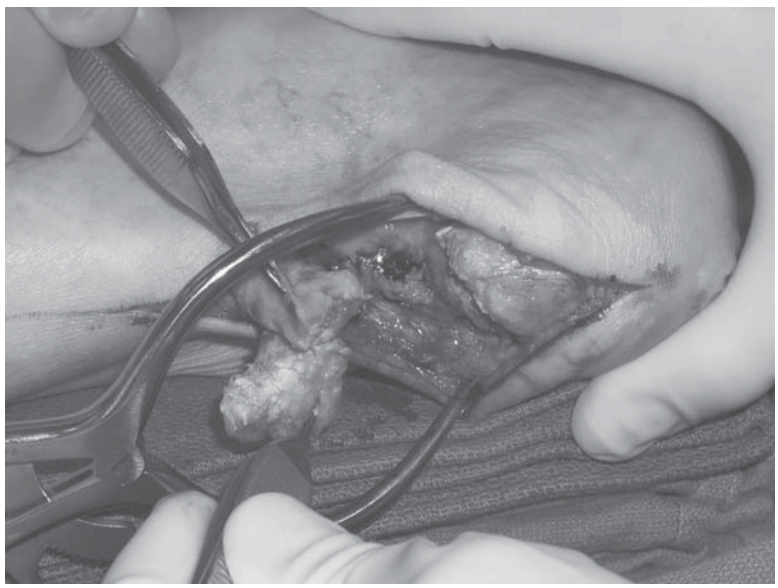

Figure 11.5. Most residual pain complaints appear to be related to underresection. If complete detachment of the Achilles is necessary, it should be undertaken without hesitation.

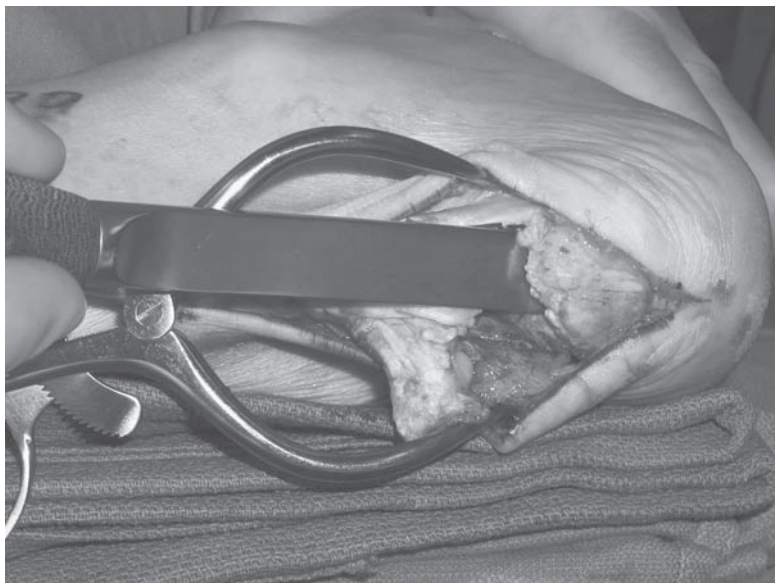

Figure 11.6. An osteotome directed from the top between the spur and the calcaneal tuberosity will restore the normal contour of the bone without excessive resection that could compromise the bone tunnels necessary for the FHL augmentation.

midline, the muscle belly immediately encountered below the fascia will be the FHL (Fig. 11.9). The peroneus brevis muscle belly originates off the fibula and the intermuscular septum far to the lateral side; it is usually easily avoided (Fig. 11.9).

6. A right-angle hemostat is useful for getting around the tendon at the level of the musculotendinous junction. Before pulling vigorously, considerable care should be taken to ensure the tibial nerve has not also been inadvertently pulled up. The nerve at this level can

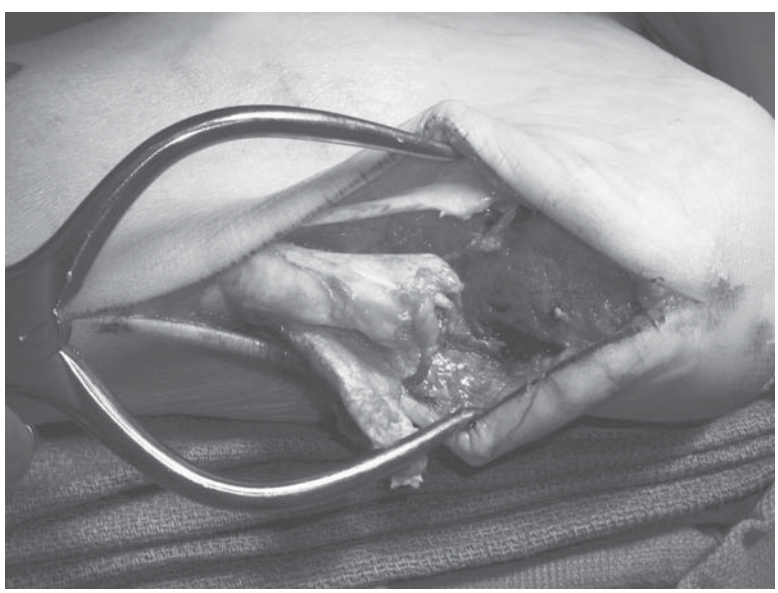

Figure 11.7. The calcaneus after resection. Note the still-intact plantaris on the medial (upper) portion of the wound.

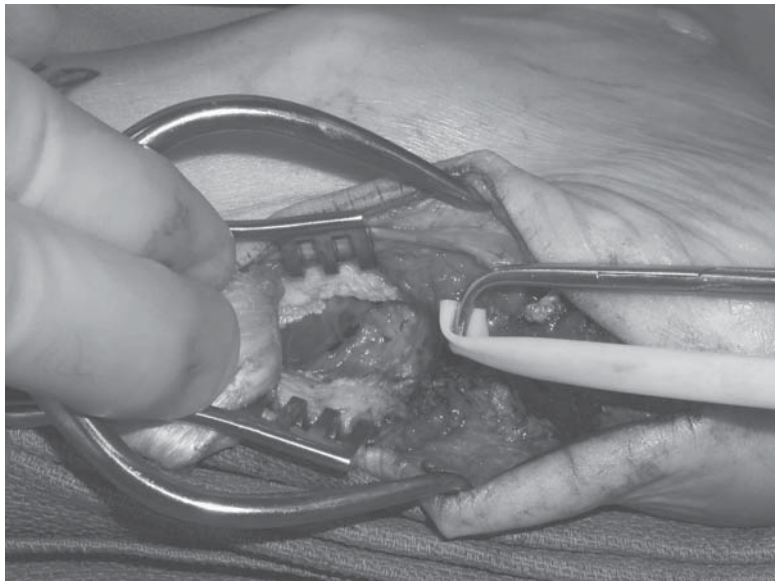

Figure 11.8. The fascia is split directly anterior to the Achilles tendon in the midline. The FHL muscle belly is readily apparent. A Penrose drain is placed around the tendon; care is taken to ensure it does not pass around the tibial nerve.

be roughly the same dimension as the FHL tendon itself.

7. The tendon is then traced as distally as possible behind the sustentaculum and carefully severed or harvested in the midfoot (see below).

Fixation

1. If a posterior harvest has been made, the FHL is then placed into the calcaneus using suture anchors or with a tenodesis screw using a blind tunnel technique.

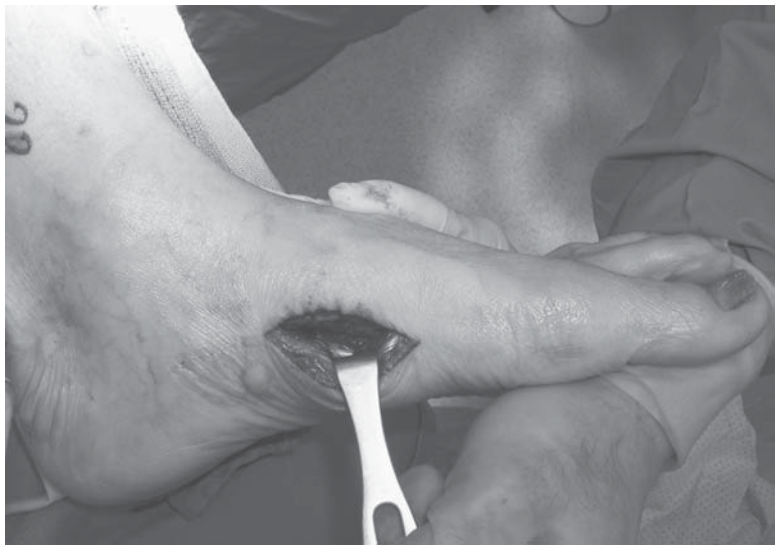

Figure 11.9. The midfoot incision. The abductor hallucis muscle belly is reflected inferiorly.

2. If a midfoot harvest has been made, a 4.0-mm burr is used to make convergent bone tunnels in the calcaneus. As originally described, the procedure utilized a direct medial-to-lateral bone tunnel, but this unnecessarily wastes a considerable length of tendon. A bone tunnel directly on the dorsal surface of the calcaneus can be made with the ankle dorsiflexed. A second, converging bone tunnel from the medial side allows a relatively short bone tunnel but with a strong bone bridge. The bone tunnels should be widened to approximately 6 mm using the burr, depending on the size of the FHL tendon.

3. The tendon end is then whip-stitched using suitable lead suture, and passed from dorsal to medial through the bone tunnels. It is then turned back upon itself. Under appropriate moderate tension, approximately 6–8 cm of length can usually be reflected proximally up the Achilles. It is then sutured in position.

The Midfoot Harvest

1. A 6-cm incision is made along the medial border of the foot centered over the medial aspect of the first tarsometatarsal joint.

2. The abductor hallucis muscle belly is reflected inferiorly. This area can be highly vascular in some patients and meticulous electrocautery is essential.

3. A fascial plane can be readily identified that courses deep into the foot. Access to the knot of Henry at this level is blocked by the tendinous origin of the flexor hallucis brevis. This origin occasionally is so well-defined that it can be mistaken for the FHL itself. The medial plantar nerve at this level is usually more plantar and lateral than the dissection plane, but care should still be exercised (Figs. 11.10 and 11.11).

4. The tendinous origin of flexor hallucis brevis is taken down usually by passing a right-angle hemostat deep to it and cutting against it. Loose fatty tissue deep to it encompasses the knot of Henry.

5. At the distal end of the knot of Henry the FDL and FHL can be tenodesed using a 2-0 vicryl suture. The FHL is then severed and whip-stitched (Fig. 11.12).

6. Using the whip-stitch to pull up on the FHL, tenotomy scissors are used to take down the tendinous juncturae between the FDL and FHL at the proximal end of the knot of Henry. This is always

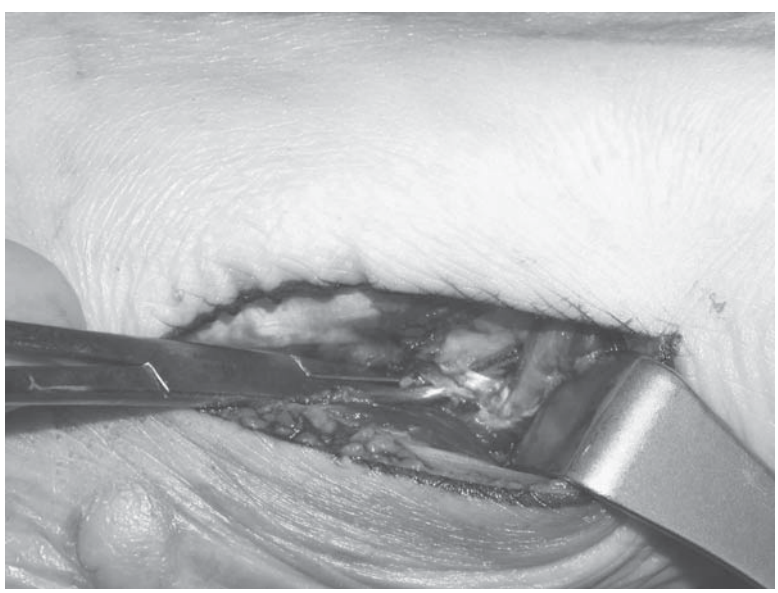

Figure 11.10. The tendinous origin of the flexor hallucis muscle belly can mimic the appearance of the FHL. It must be released to access the knot of Henry at this level.

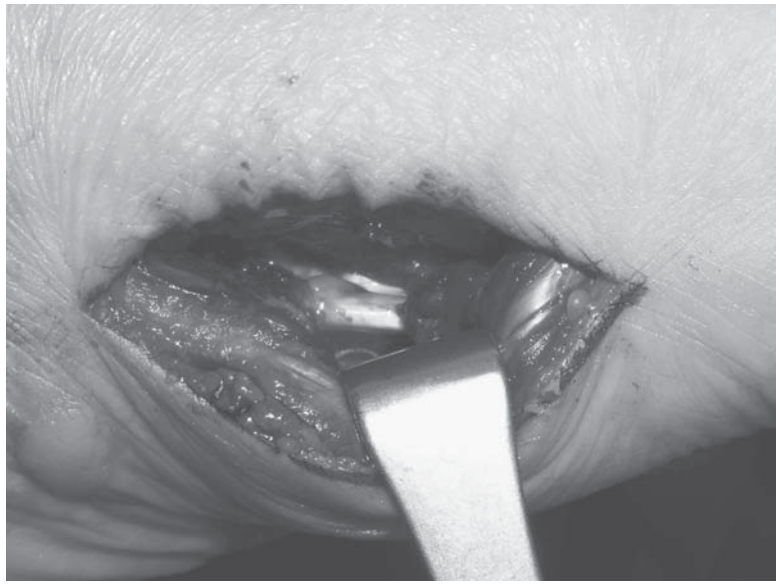

Figure 11.11. The knot of Henry.

easier to accomplish at this point than before an attempt is made to pull the tendon into the posterior ankle wound. Whip-stitching the tendon at this point rather than later facilitates easy retrieval if the juncturae are only partially released and the tendon gets held up during passage.

7. The tendon is pulled into the posterior wound (Fig. 11.13).

Results

The concept of using the flexor tendons of the foot to augment the Achilles tendon began with the flexor digitorum longus (FDL), described in 1991.[6] The more convenient anatomy of the FHL was subsequently recognized, and the technique of FHL augmentation for chronic Achilles tendon

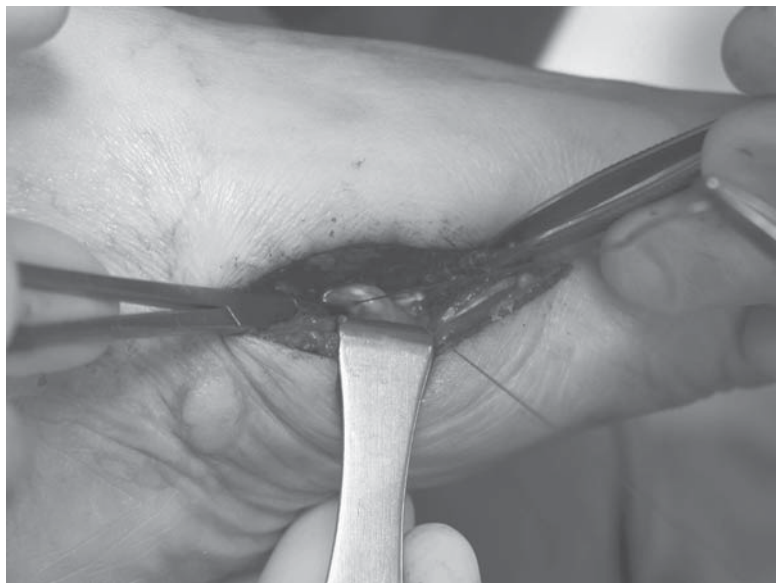

Figure 11.12. The FHL and FDL are tenodesed prior to release of the FHL. This is followed by whip-stitching the FHL and release of the juncturae between the two tendons.

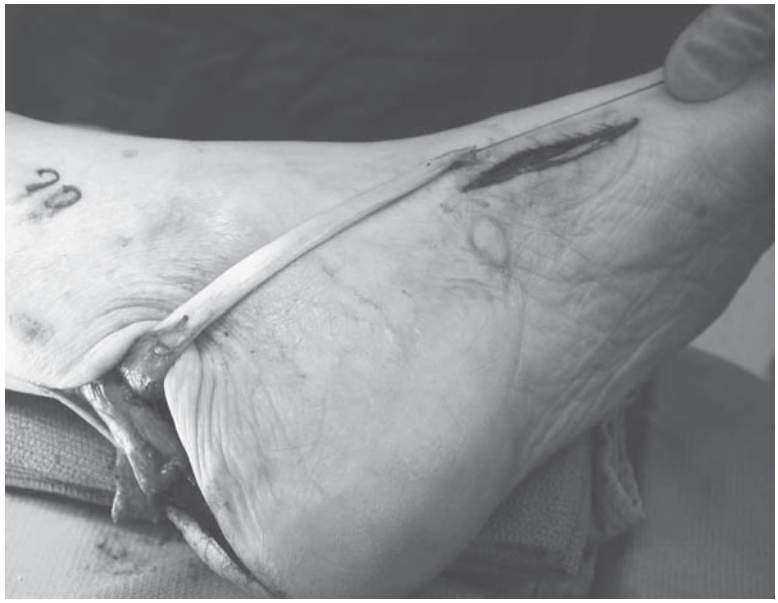

Figure 11.13. The FHL is pulled into the posterior wound.

rupture was first described in 1993 using a midfoot harvest and a transverse bone tunnel in the calcaneus.[7] The initial report contained seven patients with reportedly satisfactory clinical results. A mild limitation of motion was noted and one patient continued to use an ankle foot orthosis (AFO) (Figs. 11.14–11.19).

A subsequent series in 2000 followed 20 patients who underwent the procedure for a mean of 14 months.[8] Again, good subjective clinical results

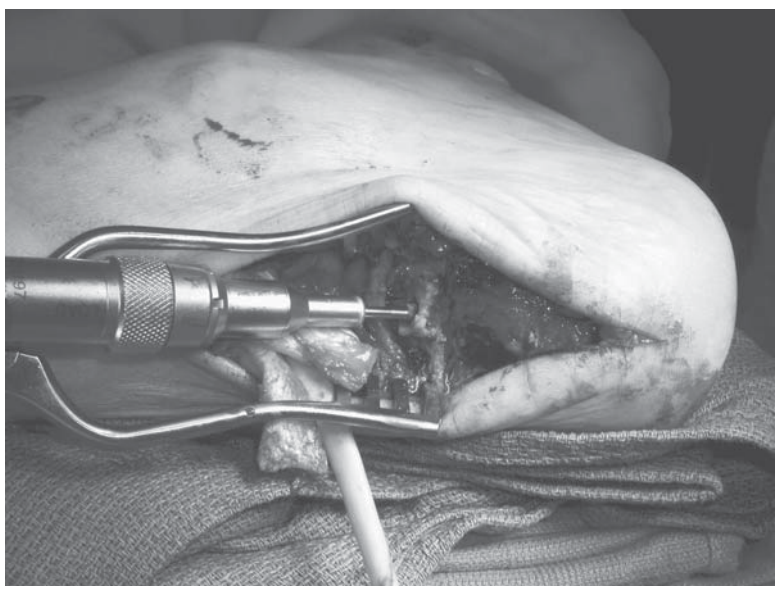

Figure 11.14. Convergent bone tunnels are placed in the calcaneus. The ankle is first dorsiflexed, and a 4.0-mm burr is used to make a tunnel on the dorsal half of the calcaneus.

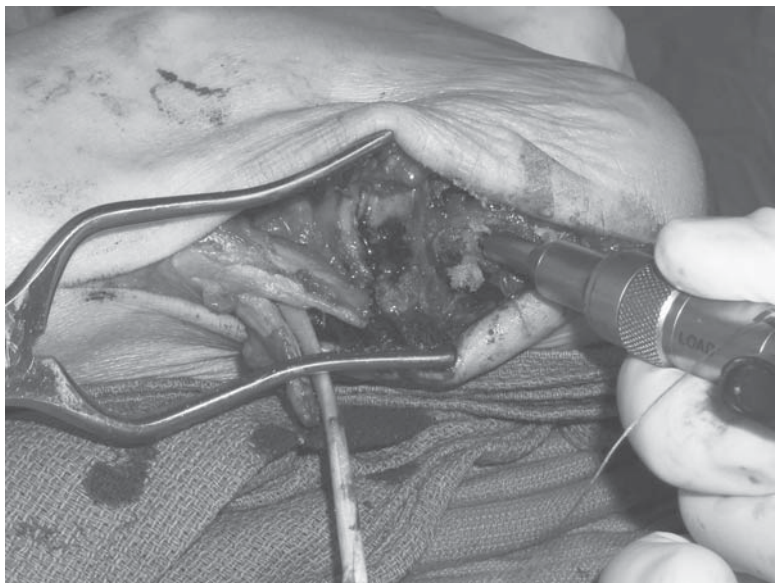

Figure 11.15. In this patient, the tendon has been completely detached, and a direct posterior tunnel can be made to intersect the dorsal one. If the insertion of the Achilles tendon is intact, a medially based tunnel is made.

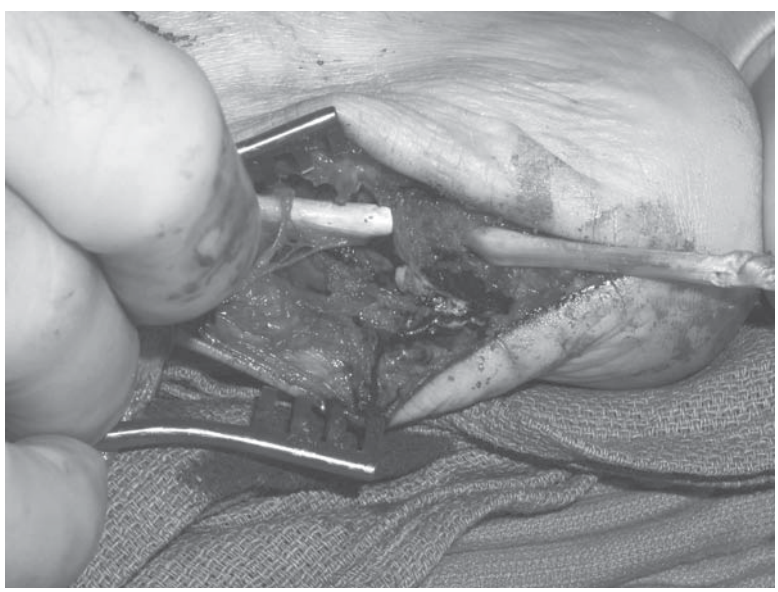

Figure 11.16. The FHL tendon is passed from dorsal to plantar/posterior.

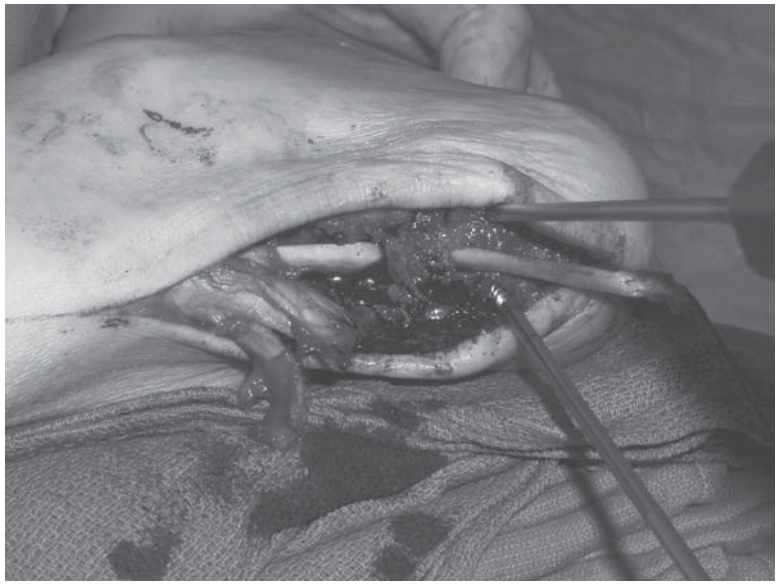

Figure 11.17. The 3.5-mm suture anchors are placed on either side of the posterior tunnel to reattach the Achilles tendon to the calcaneus.

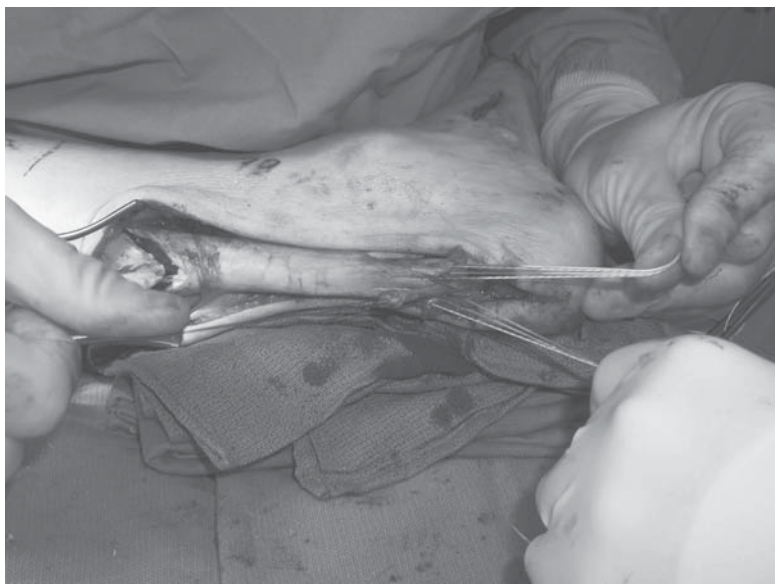

Figure 11.18. A V–Y lengthening at the musculotendinous junction is undertaken in this case because the amount of resected tendon distally resulted in a deficit. The FHL augmentation can be easily combined with any of the lengthening procedures for the Achilles.

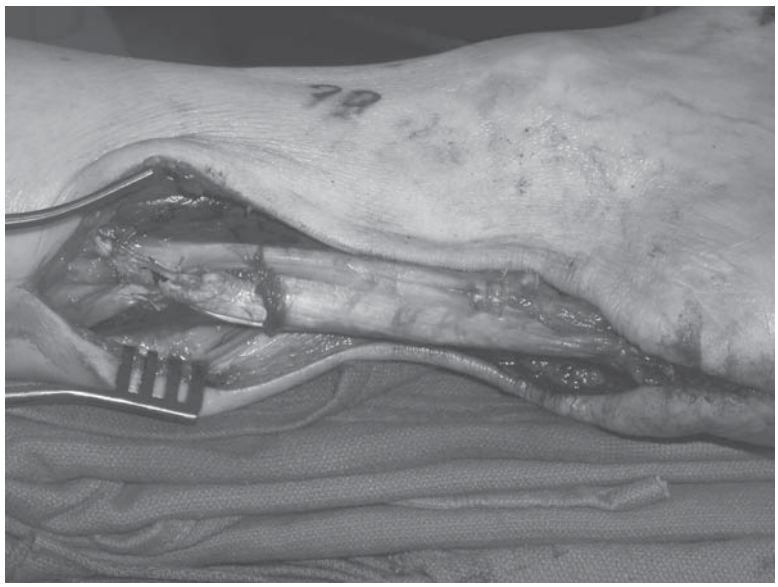

Figure 11.19. The V–Y lengthening is repaired. The FHL is woven back through the distal Achilles and sutured in position.

were achieved. The AOFAS Hindfoot scoring system, an unvalidated region-specific clinical score, demonstrated significant increases in function following intervention. Notably, however, the physical function section of the SF-36, an extensively validated general health measure, continued to demonstrate significant deficits.

Den Hartog subsequently provided a longer-term review with 100% follow-up of 26 patients at a mean of 26 months.[9] Twenty-three of 26 patients achieved subjectively good or excellent results. Again using the AOFAS Hindfoot scoring system, significant improvements were noted from the preoperative to postoperative status. The most notable finding of the study, however, was the long time to maximal improvement: 8.2 months, with a range of 3 to 20 months.

Isokinetic testing documented the return of plantarflexion torque following an Achilles rupture or tendinopathy reconstruction in nine patients.[10] Despite good subjective clinical results, marked torque deficits persisted after a mean of 19 months following surgery. The mean deficit compared to the unoperated limb was 20% at 120° per second and 26% at 30° per second.

Two recent reports have stressed subjective unhappiness with the clinical results of partial Achilles debridement for insertional ossific tenidinosis. Both have argued for complete excision of the distal Achilles in conjunction with the use of the FHL as a bridge to fill the gap. Martin et al. followed 56 of these procedures performed in a patient population with a mean age of 58.[11] Follow-up was limited and physical examinations were available on only 19 patients. Of those who responded to the mail survey, 86% expressed satisfaction with their result. Plantarflexion strength was noted to have a significant residual deficit of approximately 30%. A study of five elderly patients undergoing an identical procedure reported 100% patient satisfaction.[12] All patients were able to perform single-leg stance, but strength deficits of approximately 10% were noted on Cybex testing.

In summary, the FHL transfer has become a useful adjunct for addressing deficits in the Achilles tendon following a wide variety of pathologies including noninsertional tendinopathy, insertional tendinopathy, and neglected rupture. Except on the point that the FHL transfer has minimal morbidity, however, the literature on the topic remains confusing. No randomized controlled trials comparing the technique to nonaugmented repair exist. It is not clear whether the use of midfoot harvest provides utility when greater FHL length is weighed aganist the more tedious surgical procedure required. Most important for the patient considering the surgery, however, is that, despite the generally good pain relief

associated with the procedure, the final surgical result is not apparent for 6 to 12 months. Also notably, significant plantarflexion torque deficits of between 10% and 30% can be expected even at long-term follow-up.

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12
Generalities of Insertional Tendinopathy

Jonathan S. Young, Murali K. Sayana, and Nicola Maffulli

Common causes of posterior heel pain are insertional tendinopathy of the Achilles tendon, retrocalcaneal bursitis, Haglund's deformity, or pretendon bursitis. Insertional tendinopathy of the Achilles tendon, retrocalcaneal bursitis, and Haglund's deformity, the prominent posterosuperior calcaneal process, constitute the Haglund's triad. Clain et al.[1] introduced the terms *insertional* and *noninsertional Achilles tendinopathy* with a view to better plan management. Insertional tendinopathy had a prevalence of 20% in a surgical and histopathological survey of 163 patients with chronic Achilles tendinopathy.[2] In a consecutive series of 432 chronic Achilles overuse injury patients in Finland, 107 (24.7%) had insertional Achilles pathology. Of these, 5% (21 patients) had pure insertional tendinopathy, and 20% (86 patients) had calcaneal bursitis alone or in combination with insertional tendinopathy.[3]

The incidence of Achilles insertional tendinopathy is unclear. It is reported as the most common form of Achilles tendinopathy in athletes presenting to outpatient clinics.[4] However there are reports that 5% to 20% of Achilles tendinopathy is of the insertional variety.[5,6] Insertional tendinopathy is often diagnosed in older, less athletic, and overweight individuals as well as in older athletes.[7–9]

Histology

The osteotendinous junction of the Achilles consists of tendon, fibrocartilage, and bone. The distribution of type II collagen in sagittal sections of the Achilles tendon has been used to reconstruct the three-dimensional (3D) shape and position of three fibrocartilages (sesamoid, periosteal, and enthesis) associated with its insertion.[10] A close correspondence between the shape and position of the sesamoid and periosteal fibrocartilages was found. The sesamoid protects the tendon from compression during dorsiflexion of the foot, and the periosteal fibrocartilage protects the superior tuberosity of the calcaneus. The 3D reconstructions of the zone of calcified enthesis, fibrocartilage, and the subchondral bone showed complex interlocking between calcified fibrocartilage and bone at the insertion site.[10]

Histopathology

Light and electronic microscopy and enzyme histochemistry have been used to study a series of 11 insertional tendinopathy (including two Achilles tendons) specimens.[11] The pathological changes of insertional tendinopathy consisted of edema, mucoid degeneration, disruption of collagen bundles, necroses, small hemorrhages, and calcification. Acid mucopolysaccharides may be present in lake-like accumulations between collagen fibers, in contrast to neutral collagens seen in aging. Small bony particles lay within the cartilaginous portion of the insertion. Also, there were areas with proliferating blood vessels within tendon tissue with lymphocytes and histiocytes, suggesting a reparative process. There was increased activity of NADP-diaphorase, LDH, β-glucuronidase, and alkaline phosphatase (lower activity of the latter two enzymes) in these tendon

samples. Electron microscopy showed marked submicroscopic calcification and fibrillar degeneration.

Recent work by Maffulli et al. (unpublished data) has shown increased levels of type II and III collagen and decreased levels of type I collagen present in calcific insertional tendinopathy. Periodic acid Schiff staining showed the constant presence of chondroitin sulphate, suggesting a metaplastic change of the distal portion of the Achilles tendon in calcific insertional tendinopathy.

Etiopathophysiology

Classically, overuse and poor training habits are considered the main etiological factors of Achilles insertional tendinopathy. Also, a tight Achilles tendon, hyperpronation, pes cavus, and obesity can predispose to degeneration, attrition, mechanical abrasion, and chemical irritation that could lead to chronic inflammatory response.[7]

The in vitro strain behavior of the anterior portion of the Achilles tendon and how it is affected by the insertional tendinopathy has been investigated.[12] This suggested a role for repetitive tensile loads in the causation of insertional tendinopathy of the Achilles tendon, due to relative strain shielding noticed in this portion of the tendon. The variable response following measures aimed at decreasing tensile loads on the tendon may be explained by these findings.

The spur formation and calcification at the Achilles tendon insertion is attributed to gradual repetitive traction force. Benjamin et al.[13] investigated enthesophyte formation in rats, and compared it with human specimens. Bony spurs can develop in the Achilles tendon without the need for preceding microtears or inflammatory reactions, and form by endochondral ossification of enthesis fibrocartilage. The increased surface area at the tendon–bone junction may be an adaptive mechanism to ensure the integrity of the interface in response to increased mechanical loads.[13]

Alternative Biomechanical Theories in Insertional Tendinopathy

The traditional view of a tendon overuse injury as a result of tensile overload appears plausible. However, although this view is widely accepted, without a prospective design and adequate control groups, any conclusion regarding the etiologic role of factors such as training errors, poor technique, inadequate equipment, inflexibility, and muscle imbalance remains speculative. Only a few studies have attempted to examine these factors in a controlled, prospective manner and these have shown conflicting results.[14–20]

Epidemiological studies have also made some interesting observations. For example, overuse injuries, including tendon problems, are significantly more common in elderly athletes compared with young athletes.[21,22]

Biomechanical studies on failure modes of muscle–tendon units have clearly shown that failure will occur within the muscle near the muscle–tendon junction,[23,24] not in the tendon. Although these load-to-failure studies do not investigate repetitive submaximal loads, they do point out how a healthy tendon is biomechanically "overengineered" compared with its attached muscle. In insertional tendinopathy, the pathological tendon lesion lies at or very close to the insertion site of the tendon, the enthesis. Insertional Achilles tendinopathy is generally found at the calcaneal side.[25] Thus, the pathology is predominantly found at the joint-side of the enthesis.

The enthesis transfers the mechanical tensile loads generated by the muscle–tendon unit onto the bone through a thick tendon, which inserts at a varying oblique angle depending on the position of the joint. The architecture of these tendon insertion sites is complex, with a cartilaginous transition zone most pronounced on the joint side of the tendon.[26] Recent work on the increase of proteoglycans within the posterior tibialis tendon indicates that cartilaginous metaplasia can occur as an adaptive response to mechanical compression on the tendon.[27] These histological findings question whether the tendon insertion site is uniformly subjected to tensile loads. This has led to various biomechanical studies on the strains near the tendon insertion sites.

In a study on the Achilles tendon insertion site,[28] the tendon was instrumented with strain gauges just proximal to the calcaneus. Again, the lowest strains were found on the calcaneal side, where the pathological changes of Achilles insertional tendinopathy are generally found.

Although not all biomechanical studies report the same results, a consistent pattern appears to emerge: the strains within the tendons near their insertion site are not uniform. If we assume that the material properties are similar throughout the tendon, this would mean that any muscle force transferred through the insertion site preferentially loads the side of the tendon that is usually not affected initially in tendinopathy. In that case, the side affected by tendinopathy is generally "stress shielded." Thus, the presence of differential strains opens the possibility of alternative biomechanical explanations for the pathology found in these regions of the tendon. The traditional concept of tensile failure may not be the essential feature of the pathomechanics.

The recent biomechanical data reported above suggest a different biomechanical etiology of insertional tendinopathy. The stress-shielded side of the enthesis shows a distinct tendency to develop cartilage-like and/or atrophic changes in response to the lack of tensile load.[25–27] Over long periods, this process may induce a primary degenerative lesion in that area of the tendon. This may explain why the tendinopathy is not always clearly activity related, but is sometimes more strongly correlated with age. In this manner, tendinopathy would result from stress shielding rather than increased, overuse injury.[29]

The cartilage-like changes in the enthesis in many ways can be considered a physiologic adaptation to the compressive loads. However, even cartilaginous metaplasia may not allow the tendon to maintain its ability to withstand the high tensile loads in that region. It seems possible that in athletes occasionally certain joint positions will still place high tensile loads on the enthesis. As the stress shielding may have led to tensile weakening over time, an "injury" may occur more easily in this region. In this manner, insertional tendinopathy could be considered an overuse injury, but predisposed by preexisting weakening of the tendon.[29]

Finally, as the joint changes position, strains in one section of the tendon could be changing in opposite directions. Internal shear forces and heat could be generated, producing injury to the cellular and/or matrix components of the tendon. Accumulation of these injuries could lead to the intratendinous degeneration seen in tendinopathy.[29]

Presentation and Diagnosis of Insertional Tendinopathy

The symptoms of insertional Achilles tendinopathy are specific, and are related to pain at the bone–tendon junction, frequently worse after exercise, but which ultimately become constant. Although this is a fairly common finding in athletes, other conditions and medications that cause pain in the posterior aspect of the heel should be considered, including the various causes of insertional enthesopathy, seronegative spondyloarthropathies, gout, systemic corticosteroids, orally administered fluoroquinolones, familial hyperlipidemia, sarcoidosis, and diffuse idiopathic skeletal hyperostosis.[7] Insertional Achilles tendinopathy can be aggravated by running uphill or by activities performed on a hard surface. Frequently, the patient reports a history of poor stretching, of running on the heels or over an excessive distance, or a sudden increase in training intensity. Examination reveals tenderness at the Achilles tendon insertion, thickening or nodularity of the insertion, and at times limited, painful dorsiflexion of the ankle. Swelling may be present if there is accompanying retrocalcaneal bursitis.[30] The tenderness is specifically located either directly posterior or posterolateral to the insertion of the Achilles tendon. Dorsiflexion is limited compared with that of the uninvolved ankle because of the relative tightness of the triceps surae. Pain in the heel is the cardinal symptom, and it is increased by prolonged standing, walking, running uphill, or running on a hard surface.[7]

The pain generally emanates from the posterior aspect of the heel and is aggravated by active or passive motion. Haglund's deformity can lead to a prominent posterosuperior tuberosity of the calcaneum, although Haglund's triad is more common than isolated insertional tendinopathy of the Achilles tendon. Radiographs help demonstrate Haglund's deformity. The ossification is in the most proximal extent of the insertion of the tendon or as a spur off the superior portion of the calcaneus.[7] The size of the osteophyte cannot be accurately determined on radiographs because the structure has a very broad surface that extends across the central half of the insertion of the tendon. Although radiographically the osteophyte

appears to be located in the tendon that envelops it, the tendon is not actually attached to the spur, and the insertion of the tendon is continuous with the posterior wall of the calcaneus. Secondary imaging studies, such as magnetic resonance imaging and ultrasonography, are not necessary to make the diagnosis or to plan treatment. Rarely, magnetic resonance imaging may be helpful if there is extensive degeneration because the extent of the degeneration may have some bearing on the choice of the reconstructive procedure.[7]

Management

Most patients can be successfully managed nonoperatively.[1,31,32] The various nonoperative modalities produce an 85% to 95% success rate.[7,33] Even the more active or competitive athletes should persevere with nonoperative management.[7] Modifications in training and the use of ice, nonsteroidal anti-inflammatory medication, and heel-lifts in conjunction with stretching and strengthening exercises can be effective for athletes. Nonsteroidal anti-inflammatory medications may only provide analgesia.[34] Piroxicam showed no benefit over placebo in a randomized controlled trial when combined with an initial period of rest followed by stretching and strengthening exercises.[35] Diclofenac reduced the accumulation of inflammatory cells only within the paratenon, but provided no biochemical, mechanical, or functional benefits to the rat Achilles tendon following injury when compared with a placebo group. Also, there was no reduction in the accumulation of neutrophils and macrophages in the core of the tendon.[36]

Other simple measures, such as widening or deepening the heel-counter of the shoe or distribution of pressure with use of a silicone sleeve or pad, may also be effective for active or athletic patients. Various pads should be used to take pressure off the insertion of the Achilles tendon. A quarter-inch to half-inch (0.64- to 1.3-centimeter) felt heel-lift can be incorporated inside the shoe, although this tends to lift the heel out of the shoe. When this happens, a heel-wedge is added to the sole of the running shoe. A horseshoe-shaped felt pad is most effective and may be applied to either the shoe or the posterior aspect of the heel. If these modalities are not effective, more intensive stretching of the Achilles tendon should be performed with use of a night splint to hold the foot in maximum dorsiflexion. However, eccentric calf muscle training helped only 32% of patients with insertional tendinopathy, compared with 89% of patients with noninsertional tendinopathy of Achilles tendon.[37] If the condition is refractory, some authors suggest immobilizing the leg in a below-knee weight-bearing walking cast or a walker boot for six weeks.[7,38] This can be counterproductive; tendon loading stimulates collagen fiber repair and remodeling. Therefore, complete rest of the injured tendon is not advisable.[30] After a short period of rest from formal exercise, activities are gradually resumed with incorporation of a good flexibility program that includes correction of any biomechanical abnormalities. Cross-training may be useful after the acute phase subsides. The introduction of non-loading-type activities, such as swimming, bicycling, and aqua jogging, or open-chain kinetic-type weight-lifting exercises to the management program permits the athlete to stay conditioned. An added advantage is better compliance on the part of the athlete because he or she has a training alternative.[7] Ice, compression, and elevation, as well as nonsteroidal anti-inflammatory medication for a short period, may be helpful in the early phases of the condition. Physical therapy focuses on flexibility of the hamstrings and the gastrocnemius-soleus complex as well as the use of modalities such as ultrasound and contrast baths to help to control pain and inflammation at the site of the insertion. Ultimately, when activity (particularly running) is resumed, the distance should be decreased compared with that before the injury, and the running surface should be soft. Occasionally, a biomechanical abnormality, such as hyperpronation, is identified. A semirigid orthosis can help to control such a problem. Slight undercorrection of a pronated foot with use of an orthosis is better tolerated by runners than is complete correction and overcorrection is frequently intolerable. Again, the injection of corticosteroids is contraindicated.[7] In a pilot study,[39] polidocanol (a sclerosing agent) was injected into local neovessels localized by ultrasound and color Doppler. Eight of 11 patients experienced good pain relief,

and seven of them had no neovascularization at a mean follow-up of eight months. Two of the three patients who had poor results had bony spurs. Therefore, it appears that patients with severe bone pathology and mechanical problems are less suitable for sclerosing therapy.[40]

Surgery

Only when conservative management is exhausted is surgery undertaken. The principles of surgery include debridement of the calcific or diseased portion of the Achilles insertion, excision of the retrocalcaneal bursa, and resection of the Haglund's deformity. Sayana and Maffulli[30] prefer to reattach the Achilles tendon using bone anchors if one-third or more of the insertion is disinserted. Augmentation using tendon transfer is also described.

Anderson et al.[41] studied the surgical management of chronic Achilles tendinopathy in 48 patients. Twenty-eight patients (58%) underwent surgery for Achilles insertional tendinopathy with tenolysis, excision of the bursa and/or excision of the posterosuperior portion of the calcaneum through a 10-cm medial incision. The recovery in these patients was longer (31 weeks) when compared with patients with tendinopathy of the main body of the Achilles tendon who underwent tenolysis only (22 weeks). The success rate was 93%.

Calder et al.[42] reported the results in patients who had less than 50% of the tendon excised (49 heels), and whose ankles had been immediately mobilized free of a cast. There were two failures using this regimen: one patient with psoriatic arthropathy and another who underwent bilateral simultaneous procedures.

Kolodziej et al.[43] reported a biomechanical study that concluded that superior-to-inferior resection offers the greatest margin of safety when performing partial resections of the Achilles insertion, and as much as 50% of the tendon may be resected safely.

McGarvey et al.[44] reported on 22 heels that had surgery using a midline-posterior skin incision combined with a central tendon splitting approach for debridement, retrocalcaneal bursectomy, and removal of the calcaneal bursa projection as necessary. Twenty of 22 patients were able to return to work or routine activities by three months. Only 13 of 22 were completely pain free and were able to return to unlimited activities. Overall, there was an 82% (18 of 22) satisfaction rate with surgery.

Watson et al.[45] reported that retrocalcaneal decompression in patients with insertional Achilles tendinopathy with calcific spur was less satisfactory when compared with retrocalcaneal decompression in patients with retrocalcaneal bursitis.

Den Hartog et al.[46] reported successful use of flexor hallucis longus transfer for severe calcific Achilles tendinopathy in 26 patients (29 tendons) in whom conservative treatment failed and who also had failed tendon debridement and/or Haglund's resection. These patients were sedentary, overweight, and had chronic symptoms. The AOFAS ankle-hindfoot scale improved from 41.7 to 90.1. The time to maximum recovery was approximately 6 months. All patients lost flexor strength at the interphalangeal joint of the great toe.

Leitze et al.[47] recently reported decompression of the retrocalcaneal space using minimally invasive techniques. Patients with retrocalcaneal bursitis, mechanical impingement, and/or Achilles insertional tendinopathy who failed to respond to conservative management had an endoscopic decompression performed. However, major calcific insertional tendinopathy of Achilles tendon was considered a contraindication for endoscopic decompression. The advantages of the endoscopic procedure included quicker surgery and fewer complications, although the recovery time was similar to open decompression.

Maffulli et al.[48] reported a series of 21 patients with recalcitrant calcific insertional Achilles tendinopathy who underwent bursectomy, excision of the distal paratenon, disinsertion of the tendon, removal of the calcific deposit, and reinsertion of the Achilles tendon with bone anchors. The outcome of surgical management was rated according to Testa et al., using the 4-point functional scale validated for evaluation of long-term results following surgery for tendinopathy. Eleven patients reported an excellent result and five a good result. The remaining five patients could not

return to their normal levels of sporting activity and kept fit by alternative means.[48]

Conclusions

Much progress has occurred in our understanding of Achilles insertional tendinopathy since Clain and Baxter classified Achilles tendon disorders into noninsertional and insertional tendinopathy in 1992.[1] Insertional tendinopathy of the Achilles tendon is a degenerative rather than an inflammatory condition, although the accompanying bursitis may paint an inflammatory picture. The etiology of tendinopathy is likely to be multifactorial, with factors including some of the traditional ones such as overuse, inflexibility, and equipment problems. However, other factors need to be considered as well, such as age-related tendon degeneration and biomechanical considerations as outlined in this chapter. Recent in vivo and in vitro studies have shed some light on the biomechanics of the main body of tendon, but more research is needed to determine the significance of tensional loads, stress shielding, and compression in tendinopathy. The current biomechanical studies indicate that certain joint positions are more likely to stress the area of the tendon commonly affected by tendinopathy. These joint positions seem to be different from the traditional positions advocated in stretching exercises used for prevention and rehabilitation of tendinopathies. Incorporating different joint position exercises may exert more controlled stresses on these affected areas of the tendon, possibly allowing better maintenance of the mechanical strength of that region of the tendon, and therefore prevent injury. Alternatively, it could stress a healing area of the tendon in a controlled manner, and thus stimulate healing once an injury has occurred. Newer management measures will be introduced as the etiology of insertional tendinopathy of the Achilles tendon becomes clearer. Various surgical techniques are aimed at debriding the degenerate area of the Achilles tendon, accompanied by excision of the retrocalcaneal bursa and resection of the superior prominence. Endoscopic procedures shorten the operating time, and may decrease the complications and morbidity associated with open procedures.

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Insertional Tendinopathy of the Achilles Tendon

Murali K. Sayana and Nicola Maffulli

Introduction

Posterior heel pain can be caused by insertional tendinopathy of the Achilles tendon, retrocalcaneal bursitis, Haglund's deformity, or pretendon bursitis. Insertional tendinopathy of the Achilles tendon, retrocalcaneal bursitis, and Haglund's deformity, the prominent posterosuperior calcaneal process, constitute the Haglund's triad.

The exact incidence of Achilles insertional tendinopathy is unclear. On the one hand, it is reported as the most common form of Achilles tendinopathy in athletes presenting to outpatient clinics.[1] On the other hand, 5–20% of the Achilles tendinopathies were insertional.[2,3] Insertional Achilles pathology was seen in 24.7% of 432 chronic Achilles overuse injury patients. Of these, 5% (21 patients) had pure insertional tendinopathy, and 20% (86 patients) had calcaneal bursitis alone or in combination with insertional tendinopathy.[2] Thus, insertional tendinopathy of the Achilles tendon seems to present more often as a triad rather than as a solitary pathology. In another surgical and histopathological survey of 163 patients with chronic Achilles tendinopathy, insertional tendinopathy of the Achilles tendon had a prevalence of 20%.[4] Insertional tendinopathy is often diagnosed in older, less athletic, and overweight individuals as well as in older athletes.[5,6]

The triad of pain, swelling (diffuse or localized), and impaired performance constitutes *tendinopathy*. The suffix *osis* or *itis* should be used only after histopathological examination of the affected tendons has confirmed degeneration or inflammation.[7] Although insertional tendinopathy of the Achilles tendon is still often described as "true inflammation" within the tendon,[6] the histology from 21 recalcitrant calcific insertional Achilles tendinopathy patients showed fibrocartilaginous or calcifying degeneration close to the area of calcific tendinopathy. There was disorganization of the tendon substance with no evidence of intratendinous inflammatory reaction.[8]

Histology

The osteotendinous junction of the Achilles tendon is made up of bone, fibrocartilage, and tendon. The type II collagen distribution in sagittal sections of the Achilles tendon has been used to reconstruct the three-dimensional (3D) shape and position of three fibrocartilages (sesamoid, periosteal, and enthesis) associated with its insertion.[9] Close correspondence between the shape and position of the sesamoid and periosteal fibrocartilages was noted. The former protects the tendon from compression during dorsiflexion of the foot, and the latter protects the superior tuberosity of the calcaneus. Complex interlocking between calcified fibrocartilage and bone at the insertion site was noted on the 3D reconstructions, which used the zone of calcified enthesis, fibrocartilage and the subchondral bone. This interlocking is of fundamental importance in anchoring the tendon to the bone.

Histopathology

Merkel et al. used light and electronic microscopy, and enzyme histochemistry to study 11 insertional tendinopathy (including two Achilles tendons) specimens.[10] Edema, mucoid degeneration, disruption of collagen bundles, necroses, small hemorrhages, and calcification were noted in these specimens. Acid mucopolysaccharides may be present in lake-like accumulations between collagen fibers, in contrast to neutral collagens seen in aging. Small bony particles lay within the cartilaginous portion of the insertion. Also, there were areas with proliferating blood vessels within tendon tissue with lymphocytes and histiocytes suggesting a reparative process. There was increased activity of NADP-diaphorase, LDH, β-glucuronidase, and alkaline phosphatase. Electron microscopy showed marked submicroscopic calcification and fibrillar degeneration.

Calcific insertional tendinopathy samples harvested from an area proximal to the insertion on the calcaneus have increased levels of type II and III collagen and decreased levels of type I collagen. Periodic acid Schiff staining showed the constant presence of chondroitin sulphate, suggesting a metaplastic change of the distal portion of the Achilles tendon in calcific insertional tendinopathy (unpublished data).

Etiopathophysiology

Repetitive Traction Forces

The etiological factors implicated in Achilles insertional tendinopathy have traditionally been overuse and poor training habits. Gradual repetitive traction force leads to spur formation and calcification at the Achilles tendon insertion. Enthesophyte formation in rats was compared with human specimens by Benjamin et al.[11] Bony spurs can develop in the Achilles tendon by endochondral ossification of enthesis fibrocartilage. There is no need for preceding microtears or inflammatory reactions to cause insertional Achilles tendinopathy. The increased surface area at the tendon–bone junction may be an adaptive mechanism to ensure the integrity of the interface in response to increased mechanical loads.

Abnormal Anatomy

A tight Achilles tendon, hyperpronation, pes cavus, and obesity can predispose to degeneration, attrition, mechanical abrasion, and chemical irritation that could lead to a chronic inflammatory response at the heel.[5]

Strain Shielding

The anterior portion of the Achilles tendon is affected by the insertional tendinopathy. Lyman et al.[12] studied the in vitro strain behavior of the Achilles tendon and noticed relative strain shielding of this portion of the tendon, suggesting a role for repetitive tensile loads in the causation of insertional tendinopathy of Achilles tendon. These findings may explain the variable response following measures aimed at decreasing tensile loads on the tendon.

Stress Shielding

A distinct tendency to develop cartilage-like and/or atrophic changes on the stress-shielded side of the enthesis as a response to the lack of tensile load was noted.[13,14] Over long periods, this process may induce a primary degenerative lesion in that area of the tendon. Thus, tendinopathy is not always activity-related, but can be correlated with age, suggesting that insertional tendinopathy would result from stress shielding rather than overuse injury.[15]

Overuse Injury on a Weak Tendon

The cartilage-like changes at the enthesis can be considered a physiological adaptation to the compressive loads. However, even cartilaginous metaplasia may not allow the tendon to maintain its ability to withstand the high tensile loads in that region. In athletes, certain joint positions may place high tensile loads on the enthesis. As the stress shielding may have led to tensile weakening over time, an injury may occur more easily in this region. In this manner, insertional tendinopathy could be considered an overuse injury, but with predisposition caused by preexisting weakening of the tendon.

Thermal Injury

Strain in one section of the tendon could produce changes in the opposite section, as a tendon moves the joint. Cellular and/or matrix components of the tendon could be injured by the internal shear forces, and heat could be generated.[16] Intratendinous degeneration seen in tendinopathy could be due to accumulation of these injuries.

Clinical Features

Early morning stiffness, pain at the insertion of the Achilles tendon that deteriorates after exercise or climbing stairs, running on hard surfaces, or heel running are the typical features of Achilles insertional tendinopathy. This pain, which is initially intermittent, may later become constant. Patients may also give a history of recent increase in training, and poor warmup or stretching techniques. Achilles tendon insertion is tender on palpation and is often accompanied by thickening or nodularity of the insertion. Range of motion of the ankle, if limited, is due to painful dorsiflexion of the ankle. Swelling, if present, may be due to accompanying retrocalcaneal bursitis.

Haglund's deformity, a prominent posterosuperior tuberosity of the calcaneum, may also present as a diffuse swelling or prominence in the posterior heel region. Haglund's triad is more common than isolated insertional tendinopathy of the Achilles tendon. Hence, the clinical picture may be a mixture of the clinical findings described above. Plain radiography can help confirm the Haglund's deformity and/or ossification or calcification of the Achilles insertion.

Differential Diagnosis

Both systemic and local conditions can mimic symptoms produced by insertional tendinopathy of the Achilles tendon. Systemic affections include gout, hyperlipidemia, sarcoidosis, systemic corticosteroids, oral fluoroquinolones, diffuse idiopathic skeletal hyperostosis, and seronegative spondyloarthropathies.

Haglund's deformity, retrocalcaneal bursitis, os trigonum/posterior impingement, posterior talar process fracture, flexor hallucis longus tendinopathy, peroneal tendinopathy, tibialis posterior tendinopathy, deltoid ligament sprain, and osteochondral lesions of talus are some of the local conditions that may have a similar presentation as insertional tendinopathy of Achilles tendon.

Investigations

Blood tests for biochemistry and immunology will either confirm or rule out conditions such as gout, hyperlipidemia, and seronegative spondyloarthropathy.

Imaging (MRI scan and US scan) can help to confirm the diagnosis and identify the extent of the lesion if a surgical procedure is being planned. Radiographs help identify ossification of insertion of the Achilles tendon or a spur (fishhook osteophyte) on the superior portion of the calcaneum. Radiopacities of the Achilles tendon were classified into three types by Morris et al. (Table 13.1):[17]

Table 13.1. Radiopacities of the Achilles Tendon

Type I	Lesion
Microtrauma	Shoe counter, work-related irritation
Macrotrauma	Insertion rupture, blunt trauma
Tendinopathy	Overuse, bursitis, calcaneus shape
Foot type	Cavus, rearfoot varus, plantarflexed first metatarsal
Arthropathy	Gout, rheumatoid, Reiter's, ankylosing spondylitis, diffuse idiopathic skeletal hyperostosis
Metabolic	Renal failure, obesity, hyperparathyroidism, hemochromatosis
Infectious	Acute or chronic syphilis
Type II	**Lesion**
Arthropathy	Articular chondrocalcinosis, pseudogout
Metabolic	Vitamin deficiency
Type III	**Lesion**
Trauma	Burn injury, partial/total tendon rupture
Postsurgery	Primary repair, lengthening, recession
Ischemia	Inherent anatomy
Infectious	Chronic osteomyelitis
Systemic/metabolic	Wilson's disease, hemochromatosis
Congenital	Aperiosteal metaplasia, neural arch deficiency
CNS	Tabes dorsalis

Modified from Morris et al. (see reference 17).

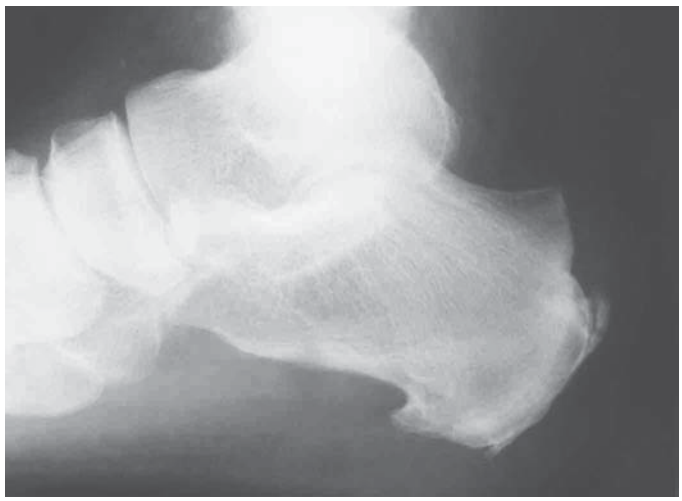

Figure 13.1. Plain radiograph demonstrating fishhook osteophyte and Haglund's deformity.

Type I. Radiopacities at the Achilles insertion or superior pole of the calcaneus. The lesion is present within the tendon, and is attached partially or completely to calcaneus. Bony changes to the calcaneus are often seen in type I lesions. Insertional tendinopathy of Achilles tendon causes type I abnormality (Fig. 13.1).

Type II. Radiopacities are intratendinous and are located at the insertion zone, 1–3 cm proximal to the Achilles insertion, and are separated from calcaneal surface.

Type III. Radiopacities are located proximal to the insertion zone, upward to 12 cm above the insertion zone. Type III is subdivided into IIIA (partial tendon calcification) and IIIB (complete tendon calcification).

Table 13.2. Ultrasonographic Classification of Insertional Achilles Tendon Abnormality

Classification	Insertional Changes
No alteration	No calcification. Homogeneous fiber structure in the insertional area.
Mild abnormality	Insertional calcification, length 10 mm or less and thickness less than 2 mm. Homogeneous fiber structure in the insertional area.
Moderate abnormality	Insertional calcification, length more than 10 mm and thickness less than 2 mm. Slight alterations in the echo structure of tendon in the insertional area.
Severe abnormality	Insertional calcification, length more than 10 mm or thickness more than 2 mm. Moderate to severe variety in the echo structure of tendon in the insertional area.

Classification of the Achilles tendon abnormalities based on ultrasonographic changes at the Achilles tendon insertion was introduced by Paavola et al. (Table 13.2).[18]

Management

Success rates of 85% to 95% have been reported with simple measures like rest, ice, modification of training, heel lift, and orthoses.[5,19] The use of nonsteroidal anti-inflammatory medications is controversial, given their mechanism of action in this condition. They probably only provide analgesic effects.[20] A randomized controlled trial has shown no advantage of piroxicam over placebo when combined with an initial period of rest followed by stretching and strengthening exercises.[21] Diclofenac reduced the accumulation of inflammatory cells only within the paratenon, but provided no biochemical, mechanical, or functional benefits to the rat Achilles tendon following injury when compared with a placebo group. Also, there was no reduction in the accumulation of neutrophils and macrophages in the core of the tendon.[22]

Modification of training, and stretching and strengthening exercises can also be effective.

However, eccentric calf muscle training helped only 32% of patients with insertional tendinopathy, compared to 89% of patients with noninsertional tendinopathy of Achilles tendon.[23] In athletes, non-weight-bearing activities can help to maintain fitness until symptoms improve. Immobilization of the ankle in a below-knee weight-bearing cast or a walker boot can be counterproductive, although suggested by some authors.[5,24] Tendon loading stimulates collagen fiber repair and remodeling. Therefore, complete rest of the injured tendon is not advisable. Ultrasound treatment could be beneficial to control symptoms at the insertion site. We do not use local injections of corticosteroids.

Sclerosing therapy in insertional tendinopathy showed promising results in a pilot study.[25] Polidocanol was injected into local neovessels localized by ultrasound and color Doppler. Eight of 11 patients experienced good pain relief, and seven of them had no neovascularization at a mean follow-up of eight months. The two of the three patients who had poor results had bony spurs. Therefore, patients with severe bone pathology and mechanical problems are less suitable for sclerosing therapy.[26]

Surgery

Surgical options are considered after 3 to 6 months of conservative management fail to produce satisfactory symptomatic relief. The principles of surgery specific for this condition are debridement of the calcific or diseased portion of the Achilles insertion, excision of the retrocalcaneal bursa, and resection of the Haglund's deformity, if present. Various surgical procedures have been described that adhere to the above principles. However, they differ in terms of reconstruction, if the Achilles tendon is disinserted or extensively debrided. We prefer to reattach the Achilles tendon using bone anchors if one-third or more of the insertion is disinserted. Augmentation using tendon transfer techniques have also been described.

Anderson et al.[27] studied the surgical management of chronic Achilles tendinopathy in 48 patients including 27 competitive athletes. Twenty-eight patients (58%) underwent surgery for Achilles insertional tendinopathy with tenolysis, excision of the bursa, and/or excision of the posterosuperior portion of the calcaneum through a 10-cm medial incision. The recovery in these patients was longer (31 weeks) when compared with patients with tendinopathy of the main body of the Achilles tendon who underwent tenolysis only (22 weeks), with a success rate of 93%.

Calder et al.[28] reported only two failures in 49 heels, where less than 50% of the tendon was excised. These ankles had been immediately mobilized free of a cast. One patient had psoriatic arthropathy, and a second patient underwent bilateral simultaneous procedures.

Kolodziej et al.[29] concluded that superior-to-inferior resection offers the greatest margin of safety when performing partial resections of the Achilles insertion, and as much as 50% of the tendon may be resected safely, based on a biomechanical study.

McGarvey et al.[30] reported the use a midline-posterior skin incision combined with a central tendon-splitting approach for debridement, retrocalcaneal bursectomy, and removal of the calcaneal bursal projection as necessary. Twenty of 22 patients were able to return to work or routine activities by three months. Thirteen of 22 were completely pain free and were able to return to unlimited activities. Overall, there was an 82% (18 of 22) satisfaction rate with the approach.

Watson et al.[31] reported that retrocalcaneal decompression in patients with insertional Achilles tendinopathy with calcific spur was less satisfactory when compared with retrocalcaneal decompression in patients with retrocalcaneal bursitis.

Den Hartog et al.[32] reported successful use of flexor hallucis longus transfer for severe calcific Achilles tendinopathy in 26 patients (29 tendons) in whom conservative treatment failed and who also had failed tendon debridement and/or Haglund's resection. These patients were sedentary, overweight, and had chronic symptoms. The AOFAS ankle-hindfoot scale improved from 41.7 to 90.1. The time to maximum recovery was approximately 6 months. All patients lost flexor strength at the interphalangeal joint of great toe.

Decompression of the retrocalcaneal space using a minimally invasive technique was reported by Leitze et al.[33] Indications for this procedure

were patients with retrocalcaneal bursitis, mechanical impingement, and/or Achilles insertional tendinopathy, who failed to respond to conservative management. Major calcific insertional tendinopathy of the Achilles tendon was considered a contraindication for endoscopic decompression. The advantages included quicker surgery and fewer complications although the recovery time was similar to open decompression.

Figure 13.2 Incision: 1 cm medial to the medial border of the lower one-third Achilles tendon.

Authors' Preferred Surgical Method

Patients are operated under general anaesthesia, with a thigh tourniquet inflated to 250 mmHg after exsanguination of the limb. A longitudinal incision, 1 cm medial to the medial border of the Achilles tendon, is extended from the lower one-third of the tendon to up to 2 cm distal to its calcaneal insertion (Fig. 13.2). The incision can be extended transversely and laterally in a hockey-stick fashion, if necessary. A Cincinnati-type incision could also be used. The Achilles tendon is exposed and sharp dissection is continued to the paratenon, which is dissected from the tendon and excised, taking care to preserve the anterior fat in Kager's triangle and not to injure the mesotenon (Fig. 13.3). The retrocalcaneal bursa is excised, if there is evidence of bursitis. The Achilles tendon is inspected for areas that have lost their normal shining appearance, and palpated for areas of softening or thickening. These areas are explored via one to three longitudinal tenotomies, and areas of degeneration are excised and sent for histology. The longitudinal tenotomies are not repaired. The area of calcific tendinopathy is identified and edges defined using the tip of a syringe needle (Fig. 13.4). The calcific area is then exposed starting from its proximal and medial aspect. Most patients will have at least one-third of the Achilles tendon surrounding the area of calcific tendinopathy detached by sharp dissection, and occasionally total disinsertion of the Achilles tendon is necessary. The area of calcific tendinopathy is excised from the calcaneus (Fig. 13.5).

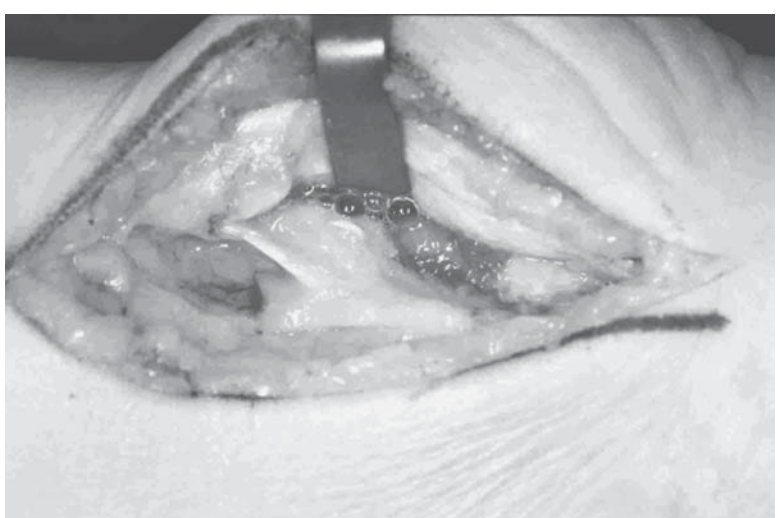

Figure 13.3 Deeper dissection.

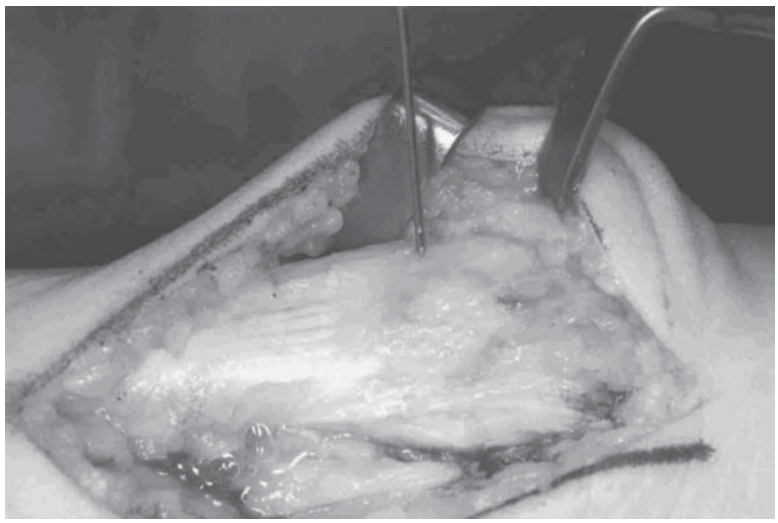

Figure 13.4 Calcified areas being probed with needle.

The area of hyaline cartilage at the posterosuperior corner of the calcaneus may be macroscopically degenerated. In this instance, it is excised using an osteotome and, if needed, its base paired off using bone nibblers. The tendon is reinserted in the calcaneus using bone anchors (Figs. 13.6 and 13.7). Two bone anchors are used if one-third to 50% of the Achilles tendon is disinserted. Three bone anchors are used if 50% to 75% of the Achilles tendon is disinserted. Four bone anchors are used if 75% or more of the Achilles tendon is disinserted, and five bone anchors are used if the Achilles tendon had been totally disinserted. The Achilles tendon is advanced in a proximal to distal fashion and reinserted in the calcaneum (Fig. 13.8). We do not usually perform a tendon augmentation or a tendon transfer. After release of the tourniquet, hemostasis is achieved by diathermy. The wound is closed in layers using absorbable sutures (Figs. 13.9 and 13.10).

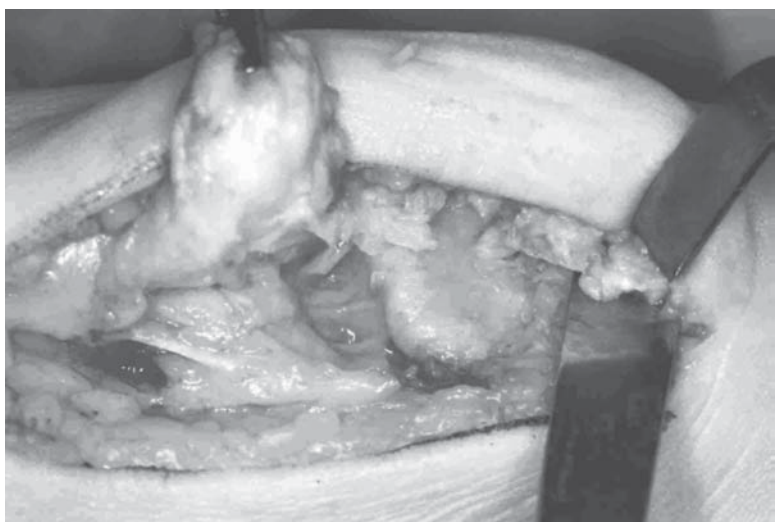

Figure 13.5 Disinserted Achilles tendon and Haglund's deformity.

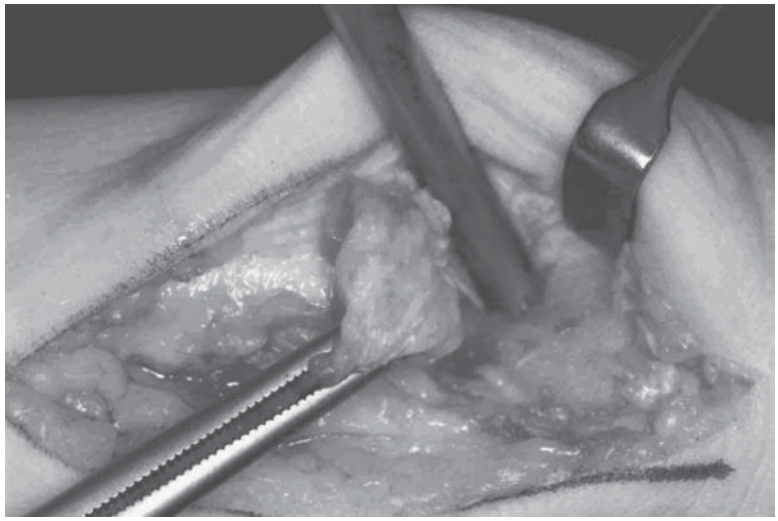

Figure 13.6 Haglund's deformity is excised, and drill holes are made to receive bone anchors.

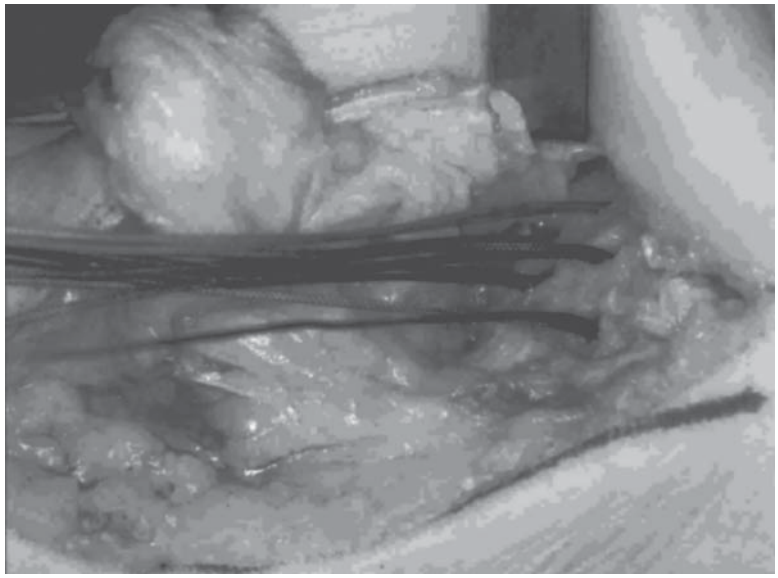

Figure 13.7 Bone anchors in situ.

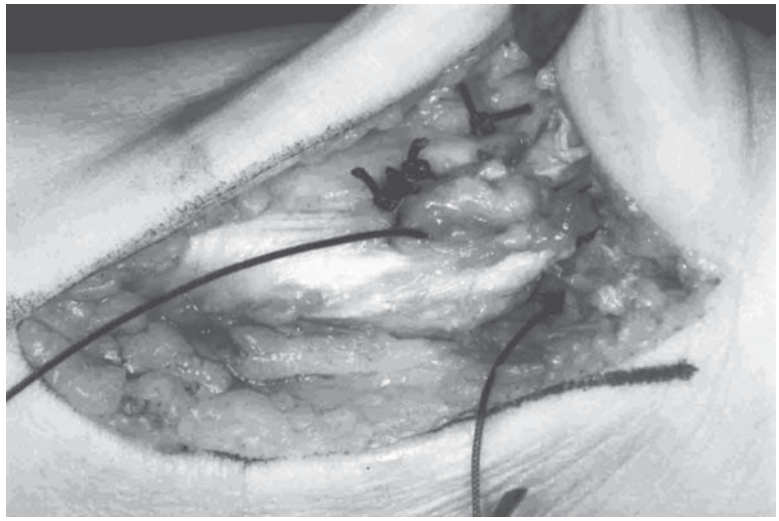

Figure 13.8 Achilles tendon being reinserted.

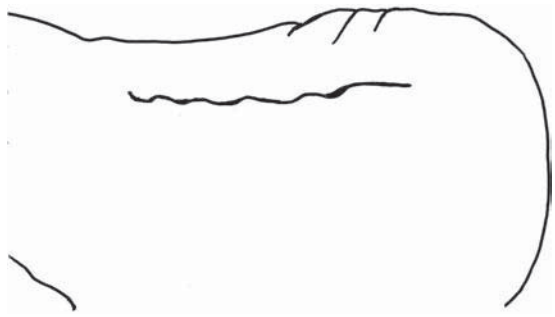

Figure 13.9 Line diagram of the surgical wound after subcuticular closure.

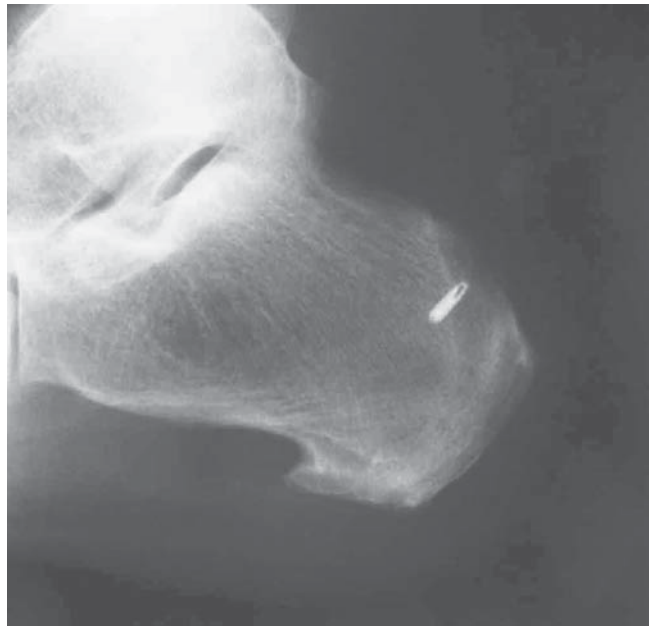

Figure 13.10 Postoperative radiograph with bone anchor in situ.

Postoperative Management

The skin wound is dressed with gauze, and sterile plaster wool is applied. A synthetic below-knee cast with the ankle plantigrade is applied. Patients are discharged home on the day of surgery, after mobilizing with crutches under the guidance of a physiotherapist. Patients are advised to bear weight on the operated leg as tolerated, but are told to keep the leg elevated as much as possible for the first 2 postoperative weeks.

After 2 weeks, the cast is removed. A synthetic anterior below-knee slab is applied, with the ankle in neutral, and secured to the leg with three or four removable Velcro straps for 4 weeks. The patients are encouraged to continue to bear weight on the operated limb and to gradually progress to full weight bearing, if they are not already doing so. A trained physiotherapist supervises gentle mobilization exercises of the ankle, isometric contraction of the gastrosoleus complex, and gentle concentric contraction of the calf muscles. Patients are encouraged to perform mobilization of the involved ankle several times per day after unstrapping of the relevant Velcro strap(s).

After 6 weeks, the anterior slab is removed. Stationary cycling and swimming are recommended from the second week after removal of the cast. We allow return to gentle training 6 weeks after removal of the cast. Gradual progression to full sports activity at 20 to 24 weeks from the operation is planned according to the patients' progress. Resumption of competition will depend on the patients' plans, but is not recommended before 6 months after surgery.

Patients are reviewed at 3, 6, and 9 months from the operation, and at 6-month intervals thereafter. Further physiotherapy along the lines described above is prescribed if symptoms are still present, and the patients are followed up until they have improved and are therefore discharged, or need further surgery.

We operated on 21 patients with recalcitrant calcific insertional Achilles tendinopathy who underwent bursectomy, excision of the distal paratenon, disinsertion of the tendon, removal of the calcific deposit, and reinsertion of the Achilles tendon with bone anchors. The outcome of surgical management was rated according to Testa et al., using the 4-point functional scale validated for evaluation of long-term results following surgery for tendinopathy. Eleven patients reported an excellent result and five a good result. The remaining five patients could not return to their normal levels of sporting activity and kept fit by alternative means.[8]

Discussion

Significant progress has occurred in our understanding of Achilles insertional tendinopathy since Clain and Baxter classified Achilles tendon disorders into noninsertional and insertional tendinopathy in 1992.[34] Insertional tendinopathy of the Achilles tendon is a degenerative rather than an inflammatory lesion, though the accompanying bursitis may paint an inflammatory picture.

The true incidence of Achilles insertional tendinopathy is still not clear. Incidence varies from 5% to the most common presentation of Achilles tendon in athletes. Insertional tendinopathy of the Achilles tendon is distinct from retrocalcaneal bursitis and Haglund's deformity, and can coexist with them. Further epidemiological studies are needed with a clear terminology to identify the true incidence of this problem.

Presence of type II and III collagen, chondroid metaplasia, and decreased type I collagen was found in histological specimens proximal to the Achilles insertion in insertional tendinopathy. Type I collagen contributes to the tensile strength in tendons, allowing them to resist force and tension and to stretch. Therefore, tendons with an increased type III and a reduced type I collagen content are less resistant to tensile stresses.

The in vitro biomechanical studies on the distal Achilles tendon have produced new insight into possible etiologies. Eccentric calf muscle training is not beneficial in Achilles insertional tendinopathy.[23] This reiterates the fact that the management strategies should be different in insertional and noninsertional tendinopathies of Achilles tendon, as their etiologies are likely to be different. In vitro strain studies on the distal Achilles tendon have identified that the anterior portion of the Achilles insertion is stress shielded or is underused. This stress-shielded area could be a site for primary

degenerative lesions, or could be predisposed to injury because of preexisting weakness. This can explain the occurrence of this lesion in older, less athletic, and overweight individuals, who may have poor warmup and stretching habits or have undergone recent increase in training.

The diagnosis is mainly clinical, and radiographs help in confirming the diagnosis as do ultrasound scan or MRI scan.

Newer management measures will be introduced as the etiology of insertional tendinopathy of the Achilles tendon becomes clearer. Various surgical techniques are aimed at debriding the degenerate area of the Achilles tendon accompanied by excision of the retrocalcaneal bursa, and resection of the superior prominence. Endoscopic procedures may shorten the operating time. This may decrease the complications and morbidity associated with open procedures.

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14
Endoscopic Techniques

Ferry Steenstra and C.N. van Dijk

Introduction

Endoscopic surgery around the Achilles tendon provides easy access to the retrocalcaneal space and the narrow space around the Achilles tendon and the paratenon. The diagnostic process and the choice for surgical versus a nonsurgical management are the same as for conventional open surgery. With the advances in technique the surgeon should become familiar with endoscopic procedures and with the endoscopic anatomy around the hindfoot and the Achilles tendon.

Insertional Problems

Haglund's Deformity

Haglund[1] described in 1928 a single patient with a painful hindfoot caused by an enlarged posterosuperior border of the calcaneus rubbing against low-back shoes. The mainstay of management of Haglund's deformity was removal of the posterosuperior corner of the calcaneus. Classically, patients with Haglund's condition report pain and tenderness at the posterolateral aspect of the calcaneus, where a prominence, the "pump-bump," can be palpated.

Distinction should be made between Haglund's condition and other conditions such as superficial Achilles tendon bursitis.

Haglund's condition must also be differentiated from Haglund's syndrome. The latter involves painful swelling of the inflamed retrocalcaneal bursa, sometimes combined with Achilles tendinopathy. The retrocalcaneal bursa is located posterior and superior of the calcaneus and just ventral of the Achilles tendon. It provides a smooth gliding surface in dorsiflexion and plantar flexion.[2–4] Impingement of the retrocalcaneal bursa between the ventral aspect of the Achilles tendon and the posterosuperior process of the calcaneus can be the cause of this retrocalcaneal bursitis.

Haglund's syndrome is a complex of symptoms involving the retrocalcaneal bursa and the superodorso-lateral calcaneus.[4–9] Haglund's syndrome is rarely seen acutely. There typically is pain when starting to walk after a period of rest. Operative management consists of removal of the bursa, and when there is a bony prominence this is excised as well.

In insertional tendinopathy, there is thickening of the Achilles tendon at its insertion on the calcaneus. Pain is often located in the midline. On plain radiography, intratendinous calcifications or an area of ossification can be seen at the site of insertion. In patients who also have retrocalcaneal bursitis, this can be seen as a white shadow in the black Kager's triangle.

Operative management consists of excision of the ossicle and adjacent tissue.

Diagnosis

Physical Examination

Patients report local pain in rest, worsening on weight bearing. Hindfoot ailments can derive

from intra- and extra-articular problems. In arthritis of the tibiotalar or subtalar joint, motion will usually be painful. Anterior ankle impingement is painful at palpation. The posterolateral aspect of the ankle and the subtalar joint can be palpated with the ankle at 15–20° of plantar flexion between the Achilles tendon and the peroneal tendons. The posteromedial aspect of the ankle joint cannot be palpated because of the overlaying neurovascular bundle and tendons (Fig. 14.1).

Posterior ankle impingement syndrome is diagnosed by forced plantar hyperflexion of the ankle. Recognizable pain on forced plantar hyperflexion is pathognomic.

All insertional and noninsertional Achilles tendon problems can be palpated. There may be subtle swelling and local tenderness. The patient recognizes the pain on palpation. It is important to differentiate between tendinopathy and paratendinopathy. In insertional tendinopathy, the pain on palpation is typically located in the midportion of the insertion at the calcaneus. In retrocalcaneal bursitis, the thickened bursa can be palpated just medial and lateral from the Achilles tendon and directly proximal of the dorsal aspect of the calcaneus.

In tendinopathy of the main body of the Achilles tendon, there is a nodular tender swelling 4–6 cm proximal to the insertion onto the calcaneus. The nodular swelling moves up and down on passive plantar- and dorsiflexion of the ankle (Fig. 14.2). In paratendinopathy, the swelling does not move on passive plantar- and dorsiflexion of the ankle.

Radiology

On plain lateral radiographs, Kager's triangle, the fat pad just ventral to the Achilles tendon, is easily detectable. In chronic conditions, the triangle is disturbed. In retrocalcaneal bursitis, lateral radiographs often show abnormality in the posterosu-

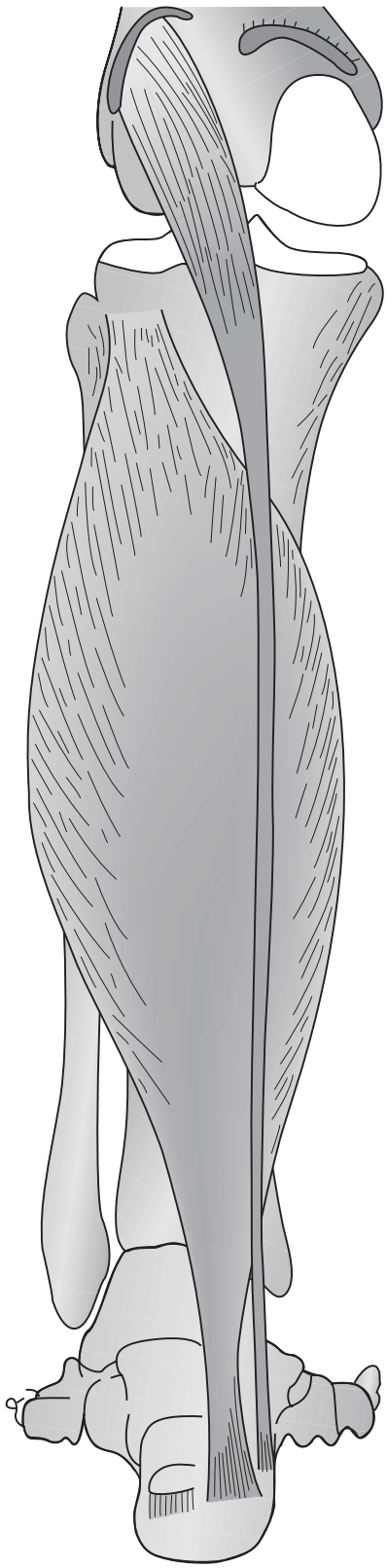

Figure 14.1. Dorsal view of a left lower leg; the gastrocnemius muscle has been removed. Note the medial insertion of the soleus muscle; in the course of the tendon there is a lateral twist of 90°. Location of the plantaris muscle: it runs from the lateral femurcondyl downward to the medial side of the Achilles tendon where it inserts on the calcaneus.

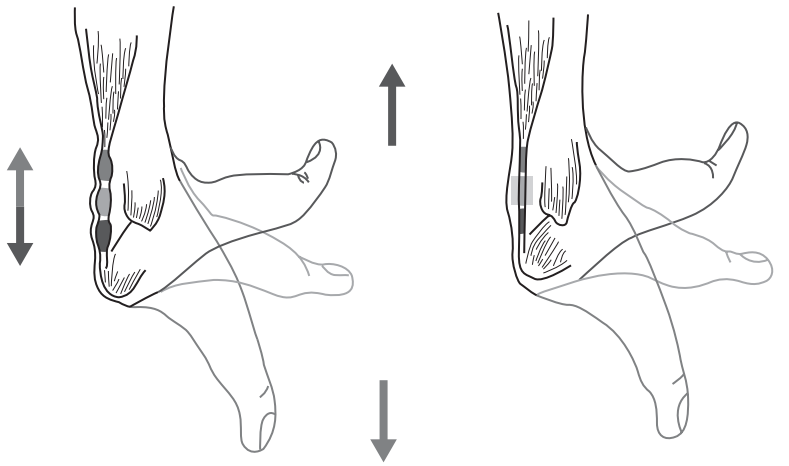

Figure 14.2. If the nontender nodular swelling moves when passive plantar and dorsal flexion is performed, then there is a tendinopathy. When it is not moving there is (also) a paratendinopathy.

perior border of the calcaneus. Parallel pitch lines on weight-bearing lateral radiographs allow one to assess the potential for retrocalcaneal impingement (Fig. 14.3).[10] In retrocalcaneal bursitis, lateral radiographs can show changes in Kager's triangle, which becomes less black and more gray because of infiltration of water.

Sometimes, the bursa becomes visible (Fig. 14.4). A calcification in the distal Achilles tendon can also be seen on lateral radiographs (Fig. 14.5).

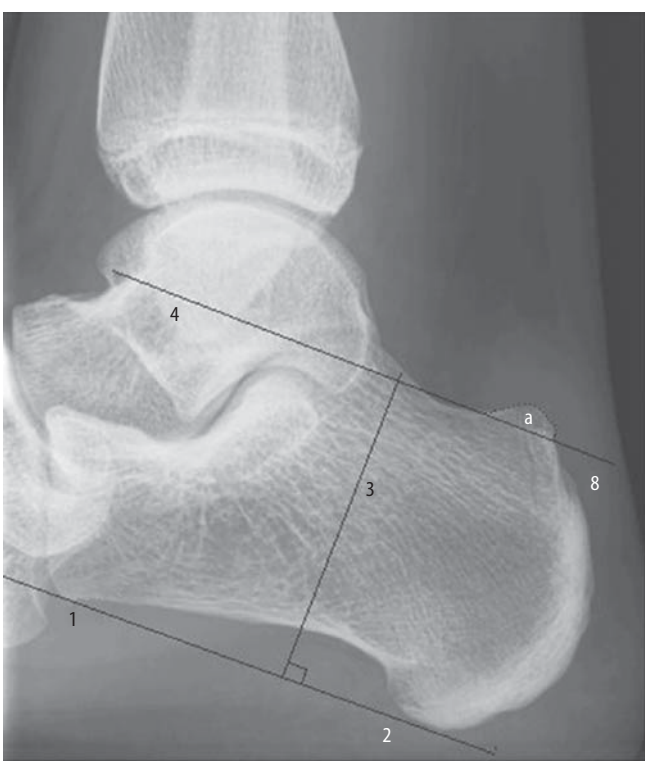

Figure 14.3. Parallel pitch lines on a weight-bearing lateral X-ray of the foot are described by Pavlov to assess the potential for retrocalcaneal impingement. The first line joins the anterior and medial calcaneal tuberosities (1–2). Then a perpendicular line is drawn through the lip of the posterior facet of the subtalar joint (3). In this manner a parallel line (4–5) can be drawn with line 1–2. The piece of bone (a) is supposed to cause the impingement on the retrocalcaneal bursa and should be removed.

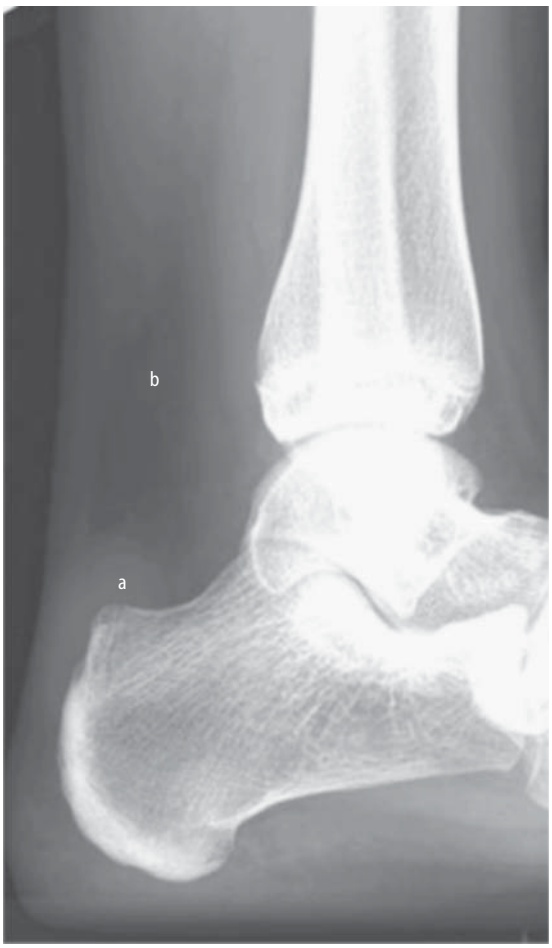

Figure 14.4. Lateral radiograph of the foot and ankle. Kager'triangle (b) is highly visible; note also the bursa retrocalcaneare (a).

Ultrasonography beautifully depicts the Achilles tendon, though it is operator dependent. A local nodule can be identified. Also, the retrocalcaneal bursa can be identified with ultrasound. Power Doppler can be used in combination with ultrasonography to ascertain whether neovascularization is present. We use MRI if we are still unsure of the diagnosis.[11]

Differential Diagnosis

Intra-articular pathology of the ankle joint and subtalar joint, and tendinopathy of peroneus longus and brevis, flexor hallucis longus, or the tibialis posterior tendons should all be considered in the list of differential diagnoses.

Management

Operative Techniques

Operative Setup for Endoscopic Procedures

The procedure is carried out on an outpatient basis under local, epidural, spinal, or general anesthesia. The patient is placed prone. A tourniquet is placed around the upper thigh. The affected foot is placed at the end of the table, to allow the surgeon to move the foot in all directions.

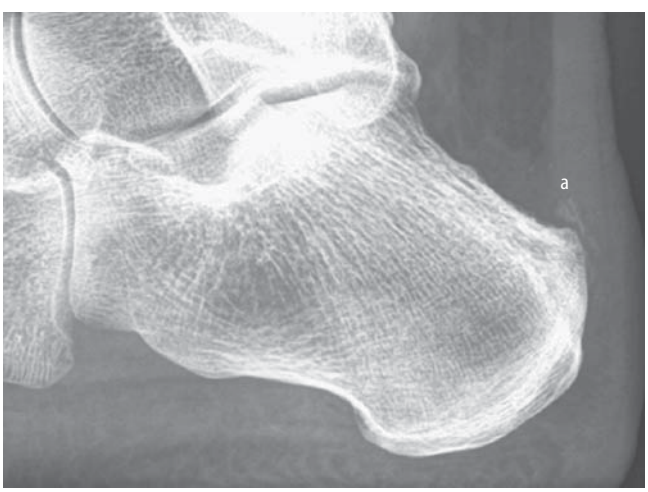

Figure 14.5. Lateral radiograph. A symptomatic calcification in the distal portion of the Achilles tendon is evident.

Arthroscopic Equipment

A 30° 2.7- or 4-mm arthroscope is used. The small-diameter, short arthroscope yields excellent vision comparable to that of a standard 4-mm scope. The small-diameter arthroscope sheath, however, cannot deliver the same amount of irrigation fluid per time as the standard 4-mm sheath. This is important in a procedure in which a large diameter shaver is used. For Haglund's syndrome, therefore, we use a 4-mm arthroscope. For endoscopic management of tendinopathy or paratendinopathy, we use a 2.7-mm arthroscope.[12]

Irrigation

Different fluids can be used at endoscopy: Ringer's lactate, normosaline, or glycine. When a 4-mm arthroscope is used, gravity inflow is usually adequate through the arthroscope sheet. With the 2.7-mm arthroscope, a pressurized bag or pump device may be necessary.[12]

Instrumentation

With the standard endoscopic equipment, a probe and an endoscopic shaver system are used.

Noninsertional Achilles Tendinopathy

With the patient prone, both feet are positioned at the end of the operating table. Epidural, spinal, or general anesthesia can be used. The short saphenous vein is marked on the lateral side of the Achilles tendon, the leg is exsanguinated, and the tourniquet inflated.

The distal portal is located on the lateral border of the Achilles tendon, 2–3 cm distal to the thickened portion (nodule) of the Achilles tendon.

The cranial portal is located 2–4 cm above the nodule on the medial border of the Achilles tendon. It is thus usually possible to visualize and work the complete circumference of the tendon over a length of approximately 10 cm (Fig. 14.6). The distal portal is produced first. After making a stab wound on the skin, a mosquito clamp is introduced, followed by the blunt 2.7-mm trochard in a cranial-lateral direction. The 30° 2.7-mm arthroscope is introduced in the same direction looking ventrally over the edge of the tendon on the superolateral aspect. Identification of the Achilles tendon is easy at the level of a healthy part of the tendon. We advise keeping the arthroscope on the tendon to minimize the risk of iatrogenic damage to neurovascular structures. The proximal portal is made in the same manner. The plantaris tendon is identified just medial from the Achilles tendon (Fig. 14.7). Typically, in patients with paratendinopathy the plantar tendon, the Achilles tendon, and the paratenon are adherent to each other. The goal of the endoscopic procedure is to remove the local thickened paratenon and to release the plantaris and Achilles tendons (Fig. 14.6).

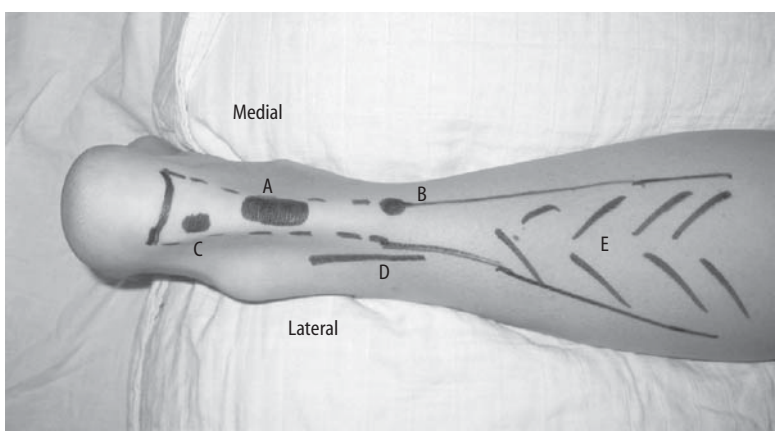

Figure 14.6. Schematic drawing in endoscopic release of a combined tendinopathy and paratendinopathy (A) of the Achilles tendon. The cranial portal (B) is on the medial side, the distal portal (C) on the lateral side of the Achilles tendon. Be aware of the sural nerve (D) on the lateral side; more proximal is the belly of the medial (m), gastrocnemicus (E).

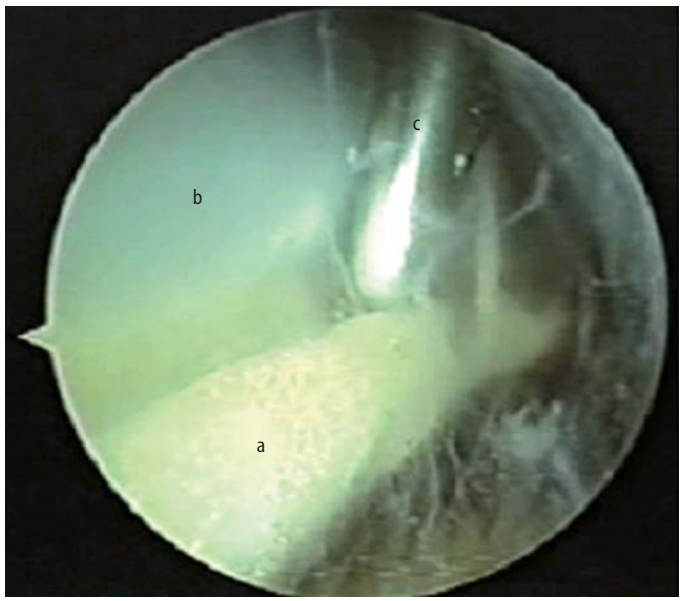

Figure 14.7. Intraoperative photographs during an endoscopic release of a noninsertional paratendinopathy of the Achilles tendon of the left leg. The arthroscope is introduced in the distal portal looking proximal. The probe (c) is introduced in the proximal portal. The plantaris tendon (a) and the Achilles tendon (b) are clearly seen.

On the ventral side of the Achilles tendon at the level of the tendinopathy, the paratenon is resected. In this area, neovascularization is accompanied by small nerve fibers. These neurovascular endings can be removed by blunt dissection with a blunt trochar followed by removal of tissue with the full radius resector 2.8-mm shaver.

Changing portals can at times be helpful. At the end of the procedure, one should be able to move the arthroscope without any problems over the tendinopathic area.

Endoscopic Calcaneoplasty for Haglund's Syndrome[13]

The lateral portal is made first at the corner of the insertion of the lateral border of the Achilles tendon and the contour of the posterolateral calcaneal border. The medial portal is produced at the corner of the insertion of the medial border of the Achilles tendon and the contour of the medial-posterior aspect of the calcaneus.

The sural nerve and saphenous vein should be marked and preserved. The medial and lateral branches of the posterior tibial artery and peroneal artery provide blood supply to the posterior aspect of the calcaneus. Structures further away from the operating site include the tibial artery and nerve, medially and ventrally the flexor hallucis longus tendon, and laterally the peroneal tendons.[14,15]

A vertical skin incision is made. A blunt trochar and blunt dissection of the retrocalcaneal space follow. The 4-mm arthroscope with a 30° angle facing distally is introduced. Under direct vision, the medial portal is made with introduction of a spinal needle under vision, and incision under vision. A probe is introduced and the retrocalcaneal bursa is inspected. A 5.5-mm full radius resector is introduced for removal of the bursa and reduction of the superior posterior calcaneal rim. Bringing the foot into maximal plantarflexion creates more working space between the Achilles tendon and the calcaneus. Changing portals can be necessary to remove the complete rim. It is important to remove enough bone at the posteromedial and posterolateral corner and to move the resector over the posterior edge of the calcaneus (Fig. 14.8). In full plantarflexion, the

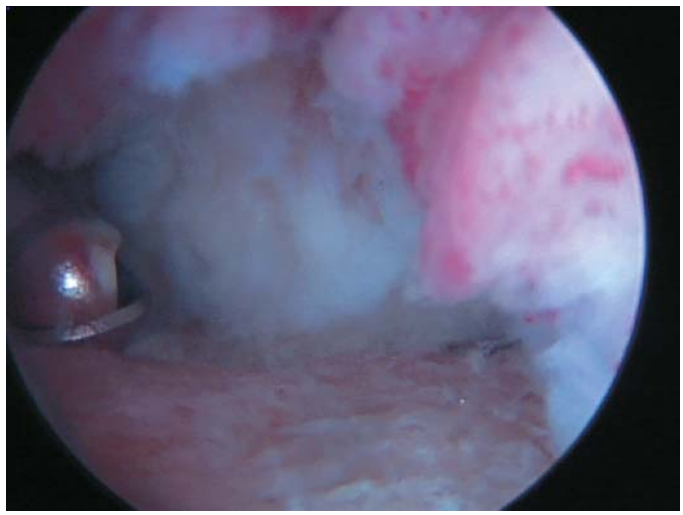

Figure 14.8. Intraoperative picture in the case of an endoscopic calcaneoplasty. Be sure to take away enough bone on the calcaneal side, but leave the insertion of the Achilles tendon intact.

insertion of the Achilles tendon can be identified. During the procedure, the closed end of the resector points toward the Achilles tendon. In this way, the Achilles tendon is protected. If the cortical bone at the insertion of the Achilles tendon is too hard for the resector, a bone bur can be used. Fluoroscopy can be used during the procedure to verify the amount of resection. At the end of the procedure, the skin portals are sutured. A compressive dressing is used. A postoperative radiograph gives immediate feedback on the amount of bone resected (Fig. 14.9).

Postoperative Care and Rehabilitation

A compressive dressing is applied for two to three days. The patient is instructed to fully weight bear as tolerated. Initially, the foot must be elevated when not walking. Active range of motion of the ankle is encouraged.[16]

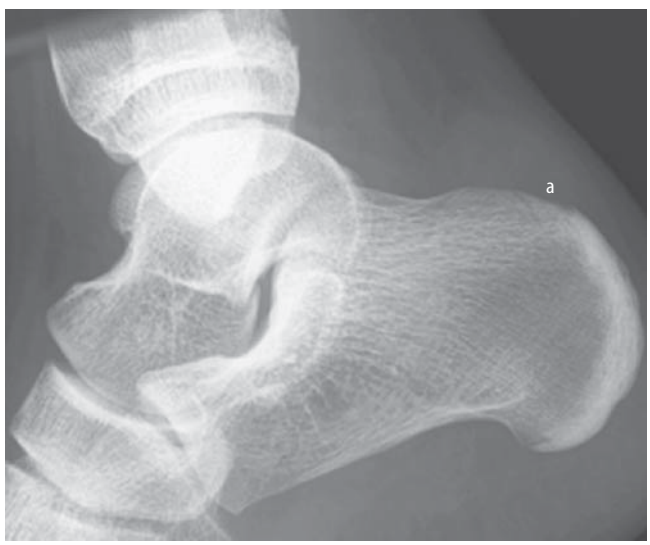

Figure 14.9. Postoperative picture after an endoscopic calcaneoplasty in which a piece of bone has been removed (a) (same patient as the preoperative picture in Figure 14.3).

Outcome

We operated on 20 successive patients using endoscopic release for noninsertional tendinopathy. All patients had complaints for more than 2 years. We reported results with a follow-up of 6 years.[2–9] Sixteen patients were seen for follow-up, which included the AOFAS and the SF-36 questionnaires.

There were no complications. Most patients were able to resume their sporting activities after a short period of time. All patients had significant pain relief. The AOFAS and SF-36 scores were almost normal comparing with a cohort of people without Achilles tendon complaints.

A comparable study on seven patients undergoing endoscopic release in chronic Achilles tendinopathy reported similar results. The mean score of this group on a scale of 100 improved from 39 preoperatively to 89 postoperatively with no complications.[17]

Concerning endoscopic calcaneoplasty, we reported the results of our first 20 patients in 2001.[12] All patients had typical complaints of inflammation of the retrocalcaneal bursa unresponsive to nonoperative management for more than 6 months. All patients presented positive parallel pitch lines (Fig. 14.3). The mean follow-up was 3.9 years (range 2–6.5). There were no surgical complications. One patient had a fair result, 4 patients had good results, and the remaining 15 patients had excellent results. Endoscopic calcaneoplasty for chronic retrocalcaneal bursitis is a minimally invasive technique that can be performed in an outpatient setting combined with a functional rehabilitation program in which patients have a short recovery time and quickly resume work and sports.[18]

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15
Achilles Tendon Rupture Generalities

Jonathan S. Young, Tomas Movin, and Nicola Maffulli

History

Achilles was the famous warrior who played a heroic role in Homer's *Iliad*. Made invulnerable by being immersed in the river Styx, he was a fearsome warrior, won many great battles, and defeated his rival, Hector, outside the gates of Troy. However, he did have one weakness: his heel, which had missed being dipped in the Styx. Paris, Hector's brother, eventually killed Achilles after firing a poisoned arrow into his heel (Homer's *Iliad*).

Anatomy

The tendinous portions of the gastrocnemius and soleus muscles merge to form the Achilles tendon, the largest and strongest tendon in the human body.[1,2] The gastrocnemius tendon emerges as a broad aponeurosis at the distal margin of the muscle bellies, whereas the soleus tendon begins as a band proximally on the posterior surface of the soleus muscle. Regarding the distal component, the Achilles tendon becomes progressively rounded in cross section until about four centimeters from its calcaneus insertion, where it flattens out prior to inserting into the proximal calcaneal tuberosity.[3] The calcaneal insertion is specialized: it is composed of an attachment of the tendon, a layer of hyaline cartilage, and an area of bone not covered by periosteum. There is a subcutaneous bursa between the tendon and the skin, and a retrocalcaneal bursa between the tendon and the calcaneus.[4]

Strength

The Achilles tendon has a high capacity to withstand the tensional forces created by the movements of the human body.[5] Approximately 95% of the collagen present in tendons is type 1 collagen, with a small amount of elastin. Ruptured Achilles tendons contain a substantial proportion of type 3 collagen. Type 3 collagen is less resistant to tensile forces, and therefore is predisposed to spontaneous rupture.[6]

The tenocytes within the tendon contain both actin and myosin, allowing for the contraction–relaxation mechanism of the tendon. In vivo peak force of the Achilles tendon has been measured at 2,233 newtons.[7]

At rest, due to crimping of the collagen fibrils, the tendon has a wavy conformation. When the tendon is subjected to tensile stresses the wavy configuration disappears. Collagen fibers respond linearly to increasing tendon loads. At strain levels less than 4%, the fibers retain their original configuration on removal of the load. At strain levels between 4% and 8%, collagen fibers slide past one another and intermolecular crosslinks fail. Strain levels greater than 8% lead to macroscopic rupture due to tensile failure of the fibers.[8]

Pathobiomechanics

Achilles tendon ruptures occur commonly in the midsubstance of the tendon, usually two to six centimeters proximal to the insertion to the calcaneus. Other less common locations are the

musculotendinous junction and the insertion into the calcaneus. The injury can be open or closed, and may be caused by a direct blow or an indirect force. Most injuries tend to occur when pushing off with the weight-bearing foot while extending the knee. Some Achilles tendon ruptures occur following sudden ankle dorsiflexion or violent dorsiflexion of a plantarflexed foot.[9,10] Most ruptures occur during sports. The sport that dominates as the cause of Achilles tendon rupture depends on the country where the study is performed. Basketball was the most common sport in the U.S. military;[11] in Scandinavian countries, badminton players sustain most of the Achilles tendon ruptures;[12–19] and in Germany it is soccer.[20,21]

Clinical Examination

Generally, most people who present with rupture of the Achilles tendon give a history of sudden sharp pain in the back of the calf of the affected leg. It is often described as if they had been kicked.[2,22] An audible snapping sensation has also been described. Patients then may have difficulty weight bearing on the affected side, and may describe ankle stiffness. Patients can also present with a chronically ruptured Achilles tendon, and they will often recall a minor trauma with pain in their heel. It then is apparent that routine day-to-day tasks, including climbing stairs, become difficult.[23] A direct injury mechanism to the Achilles tendon is rare.[24]

At examination there may be swelling and sometimes bruising in and around the region of the Achilles tendon. If the swelling is not severe, a palpable gap can often be felt along the line of the tendon. The gap is most often felt approximately two to six centimeters proximally from the insertion of the tendon. The average location of rupture has been measured to be 4.78 centimeters proximal to the calcaneal insertion.[25]

In the majority of patients, the clinical diagnosis of Achilles tendon rupture does not pose a problem. When sports participation is not involved, the injury may be missed or misjudged either by the patient or the doctor. Further difficulties to reach the correct diagnosis may occur in elderly patients if the patients consult a doctor some days after the injury: Achilles tendon ruptures are missed in up to 20% of patients.[26,27] In elderly patients with Achilles tendon rupture, nine (36%) of 25 patients had a delay of more than one week to definitive management.[28] In chronic rupture of the Achilles tendon, the pain and swelling may have subsided, and the gap between the tendon ends may have filled with fibrous tissue.[2,29] If there is uncertainty or clinical confirmation is required, several tests can be undertaken.

The calf squeeze test is relatively straightforward. It is often described as Thompson's[30] or Simmonds' test,[31] though Simmonds described the test five years before Thompson. The patient lies prone on an examination couch with both feet hanging over the edge. The examiner squeezes the calf; if the Achilles tendon is intact, the foot will plantarflex against gravity, and should be compared against the other leg. This occurs because on calf squeezing the muscle is deformed, thus causing the overlying Achilles tendon to bow away from the tibia. Matles' test[32] is carried out with the patient lying prone on a bed. Both legs are flexed at the knee to 90°. On the side without the rupture, the ankle will remain in plantarflexion. On the side with the rupture, the ankle will dorsiflex or remain in the neutral position. There is a theoretical risk of a false positive result if the plantaris tendon is intact.

Imaging

If there is clinical doubt about rupture of the Achilles tendon, imaging may be of help. For the trained eye, lateral radiographs of the ankle can be used to aid the diagnosis of rupture. The triangular fat-filled space between the anterior aspect of the Achilles tendon, the posterior aspect of the tibia, and the superior aspect of the calcaneus is called Kager's triangle.[33] This area loses configuration and can be distorted if there is a rupture. Deformation of the contours of the distal segment of the tendon from loss of tone is the most likely radiographic change associated with rupture.[9]

Real-time high-resolution ultrasonography is another useful diagnostic aid, though it is user dependent. Ideally, a linear array transducer

probe with spacer gel should be held at 90 degrees to the tendon, ensuring the optimum amount of ultrasonic energy is returned to the transducer.[34,35] The longitudinally arranged collagen bundles of the Achilles tendon reflect the ultrasound beam. High-frequency probes provide the best results, although they have a short focusing distance.[36] Men have slightly thicker tendons than women,[37] and, when the Achilles tendon ruptures, an ultrasound scan reveals an acoustic vacuum with thick irregular edges.[38]

Magnetic resonance imaging is the gold standard of imaging. Both T1 and T2 weighted images should be used to evaluate the tendon for rupture. The images should be in the axial and the sagittal planes. A normal Achilles tendon is viewed as an area of low signal intensity on all sequences. High signal intratendinous intensity is viewed as abnormal.[39] In T1 weighted images, a complete rupture is visualized as disruption of the signal within the tendon. T2 images are not as specific, and generalized increased signal intensity represents the edema and hemorrhage within and around the ruptured tendon.[40]

Management

The goals of management of Achilles tendon ruptures are to minimize the morbidity of the injury, optimize rapid return to full function, and prevent complications.[24] To measure whether the management of Achilles tendon rupture has been successful, one needs to examine different variables. The variables most frequently studied in modern outcome studies after Achilles tendon rupture include complications, calf muscle strength, endurance, tendon configuration, patient satisfaction, and the impact of Achilles tendon rupture on absence from work and sports participation. Overall outcome measurements by 100-point scoring systems have been used.[19,41–43] A major limitation for their common use has been that some scores include dynamometry testing, which is not widely available in routine clinical practice.

Despite the developments of the last few decades, there is still ongoing debate on how to deal with an early acute rupture of the Achilles tendon. The management options are either surgical or nonsurgical, with surgical management involving open or percutaneous methods. Management to some degree depends on the time of presentation, and the patient's degree of athleticism, age, fitness, and personal preference. The preference of the surgeon will also be a factor. There is as yet no established protocol for the management of ruptures.[2,44] Management of Achilles tendon ruptures should allow the tendon to heal and enable the patient to return to an acceptable functional level. Factors such as age, occupation, and recreational activities should be taken into account when counseling patients.

Comparing Open Surgical versus Nonsurgical Management

Möller et al.[19] performed a multicenter prospective randomized controlled trial with two years follow-up on 112 patients comparing open surgical repair and nonsurgical immobilization. More patients in the nonsurgical management group (11/53) sustained a re-rupture than did those in the surgical treatment group (1/59) ($p < 0.001$). Patients who had surgery reported a better quality of life during the 8-week treatment period ($p < 0.001$) and rated the result of treatment more highly than did patients in the nonsurgical management group ($p < 0.001$). While there was no significant difference between both groups for length of time before return to work, patients with jobs requiring mobility returned to work sooner after surgery ($p = 0.03$).

Cetti et al.[14] found that open surgery with simple end-to-end sutures followed by cast immobilization resulted in a better outcome and patient satisfaction than nonsurgical treatment in a cast. The prospective randomized trial included 111 patients, with a re-rupture rate of 5.4% (3/56) in the surgical group and 12.7% (7/55) in the nonsurgical group ($p = 0.19$). There was no significant difference in the mean length of time off work or major complications. Nonsurgical management resulted in lower rates of minor complications ($p = 0.004$). At 12 months' follow-up, the surgically managed patients had a significantly higher rate of resuming sports activities at the same level, a lesser degree of calf atrophy, better ankle movement, and fewer complaints.

In 105 patients, nonsurgical management was favored as there were fewer complications,[13] with a 4.5% re-rupture rate in the surgical group and 8.0% in the nonsurgical group (NS). The patients were evaluated clinically and with static and dynamic measurements of plantarflexion strength. Only minor, insignificant differences were noted between the final results in the two groups.

Nestorson et al.[28] examined the functional ability after Achilles tendon rupture in 25 patients older than 65 years. Fourteen were managed surgically and 10 conservatively, with one patient receiving no treatment. Only 9 patients returned to their previous activity level, and 11 patients had at least one complication. Achilles tendon rupture in this age group reduces lower limb function, and complications were common following surgical and nonsurgical management. Surgical repair may not be beneficial on average in the over-65 age group.

In general, however, there is an increased risk for re-rupture following nonsurgical management and an increased risk for minor complications following surgical management. For physically active patients most surgeons would suggest operative management. It is still controversial which surgical technique gives the best outcome.

Percutaneous Repair versus Open Repair

Lim et al.[45] performed a prospective randomized multicenter controlled trial with a minimum of six months' follow-up in 66 patients comparing open and percutaneous repair of closed ruptured Achilles tendons. The difference in infective wound complications between the two groups was statistically significant ($p = 0.01$). The authors advocated percutaneous repair on the basis of the low rate of complications and improved cosmetic appearance.

Majewski et al.[46] compared the treatment of ruptured Achilles tendon by operative end-to-end surgery, percutaneous repair, or conservative therapy in 73 patients. After 2.5 years, there were no differences, with patients obtaining an excellent or good result using a 100-point score. Patients in whom a percutaneous repair had been performed resumed work and sports activities sooner than the two other groups.[46]

Halasi et al.[47] in a retrospective report of 144 percutaneous repairs from one center registered a re-rupture rate of 4.2%, no wound or sural nerve complications, and a rate of general complications of 10.4% (partial rupture 2.8%, delayed healing 5.6%, and deep vein thrombosis 2.1%).

Hockenbury and Johns[48] compared in vitro percutaneous repair[49] with open repair with use of a Bunnell suture in transverse sectioned Achilles tendons in 10 human cadavers. The tendons repaired with an open technique resisted almost twice the amount of ankle dorsiflexion before a 10-millimeter gap appeared in the repaired tendon compared to the percutaneous technique. However, the results of that study should be interpreted with caution, as the number of suture threads passing through the rupture site was greater in the open repair group than in the percutaneous repair group. Modifications of the percutaneous Ma and Griffith technique have improved the strength of the repair.[50] Overall, most studies on percutaneous repair demonstrated that the rate of repeat rupture is higher than after open operative repair, but are based on older percutaneous repair methods. More modern configurations have produced extremely low re-rupture rates.[51]

End-to-End Suture versus Tendon Augmentation

Augmenting an Achilles tendon repair aims to provide a stronger repair and decrease the chance of re-rupture. However, comparisons between simple end-to-end sutures and augmentation for the management of acute uncomplicated ruptures have detected no clinically significant differences.[52–54] Thus, a nonaugmented end-to-end repair is the management of choice.[55] The strength of the repair will differ according the suture techniques,[56–59] although this does not always predict a better outcome.[60] Mortensen et al.[60] compared in a randomized trial a weaker Mason suture technique with a stronger reinforced continuous six-strand suture technique in end-to-end repair. No difference was found between the two techniques, suggesting no advantage for more complicated suture techniques.[60]

Postoperative Treatment in a Cast versus Functional Rehabilitation

The issue of complete casting compared with functional rehabilitation on the Achilles tendon with relation to the stresses on the tendon is a difficult one. Complete immobility without stress leads to tissue atrophy and adhesions, whereas too much stress too early is likely to jeopardize the tendon repair and lengthen the repair or provoke a re-rupture.

The standard European orthopedic practice was surgical repair followed by below-knee cast immobilization with a gradual reduction of equinus to plantarflexion during six to eight weeks.[2] During the last few decades, many reports of good results following surgical repair and early motion and/or early weight bearing have been published.[18,19,53,57,61–72]

Functional rehabilitation may include the use of an orthosis, a splint, or a modified shoe. Many different functional rehabilitation protocols are advocated for six to eight postoperative weeks, and it is difficult to compare such studies. The general trend from the functional rehabilitation regimens indicates no apparent increase in re-rupture rate. Pooled data from five prospective comparative studies investigating cast immobilization versus functional brace regimens[18,69,71–73] revealed an incidence of re-rupture rate of 5.0% in the cast immobilization group and 2.3% in the functional brace group ($p = 0.26$).[74] Further, a postoperative functional brace seems to shorten the time needed for return to work and rehabilitation to sports.[74]

Nonsurgical Treatment: Immobilization in Plaster versus Functional Rehabilitation

Functional nonsurgical management has gained increasing interest in parallel with the increased use of postoperative functional treatment. Saleh et al.[75] in a randomized trial used a dorsiflexion limiting splint and compared it to immobilization in plaster. The functional treatment was appreciated by the patients, and mobility was restored more rapidly. Petersen et al.[76] reported on 50 patients with a first-time rupture of the Achilles tendon that were randomized to either a cast or a CAM walker. Both groups were treated for eight weeks. They found five re-ruptures in 29 patients treated with a cast (17%). No re-ruptures occurred in 21 patients treated with a CAM walker. The difference, however, was not statistically significant ($p = 0.07$).

Both surgical and nonsurgical management with functional treatment with a boot (Variostabil™) have been prospectively compared.[77] In 50 patients (22 of whom underwent surgery and 28 conservative management), there were no significant differences either in the functional results or in the course of healing. No re-rupture was registered in either of the treatment groups. Functional treatment in both groups allowed shorter periods of rehabilitation, and acceptance of the boot was particularly high in all patients.[77] The same boot (Variostabil™) was used in a prospective study in Braunschweig, Germany, in 161 patients.[78] Complications included seven cases of re-rupture (5.3%), and, in the course of treatment, four patients (3%) suffered deep vein thrombosis of the leg, which in one patient developed into post-thrombotic syndrome.

Nonsurgical management by a combined protocol including the use of casts and a removable orthosis has shown excellent results.[79] In a series of 140 patients,[80] the overall complication rate was 8%, with three complete and five partial tendon re-ruptures, two deep vein thromboses, and one temporary dropfoot.

Limited conclusions can be drawn from the current literature on nonsurgical functional management.[74]

Complications

Complications of Achilles tendon rupture are not uncommon and can be related to the type of treatment (i.e., conservative) or surgery (open repair or percutaneous repair). There are further general complications that will affect patients regardless of treatment.

Complications of Surgery

Surgical management significantly reduces the risk of Achilles tendon re-rupture, but increases the risk of infection when compared

with conservative management.[81] Arner and Lindholm report a 24% complication rate in 86 operative repairs.[82] Open repair caused 20 times more minor-to-moderate complications than conservative management, but there were no significant differences between open surgical and conservative management regarding major complications.[83] The results of open repair vary markedly.[82,84] These differences are likely to be multifactorial and may well result from subtle variations in technique, degree of experience of the operating surgeon, the type of suture material used, and the location of the incision.

Percutaneous repair is a compromise between open surgery and conservative management, though the early reports outlined an increased risk of re-rupture and of damage to the sural nerve.[49] Ma and Griffith reported an excellent success rate with no re-ruptures and two minor complications.[49] Some studies have demonstrated that the rate of re-rupture after percutaneous repair is higher than that after open operative procedures.[85,86] More recent studies comparing the two repair techniques show similar results, with no difference in re-rupture rate between percutaneous and open repair.[45] That study showed a significantly higher rate of infective wound complications using open repair.[45]

Compartment syndrome is rare following ruptured Achilles tendon.[87] Many surgeons use a tourniquet in the repair of Achilles tendon. While relatively safe, there have been cases of compartment syndrome following tourniquet use for lower limb surgery.[88]

If a longitudinal incision is used in open repair of the Achilles tendon, it passes through poorly vascularized skin,[89] with the potential of poorly healing wounds. Even defects less than one square centimeter take a long time to heal. Wounds that break down need coverage, as tendons left exposed undergo desiccation and secondary adhesions.[90] Local or free flap coverage may achieve this if the lesion is not responsive to conservative management.[91]

Less common complications of Achilles tendon rupture include peritendinous calcifications after open repair.[92] These can be managed either conservatively or operatively.

Complications of Conservative Management

Conservative management may lengthen the tendon, altering its function.[93] Many authors feel that, aside from the functional problems of conservative management, there is also a higher re-rupture rate.[94] This may need surgical correction,[95] and can be avoided in the first place if surgery is performed.[2] Haggmark et al.[96] highlighted the functional problems associated with Achilles tendon rupture. They followed 23 patients for three to five years; 15 had an open repair and eight were managed nonoperatively. The latter had significantly impaired dynamic calf muscle function in comparison with operatively managed patients, in whom no such impairment was shown. Wong et al.[94] reported a re-rupture rate of 10.7% for conservative management of rupture. Lo et al.[83] reported an overall re-rupture rate of 2.8% for operatively managed and 11.7% for nonoperatively managed patients ($p < 0.001$). Persson and Wiedmark showed that 7 of 27 patients had a re-rupture, and a further 7 patients were not satisfied with the result of conservative management.[97]

Conservative management is still in use and advocated by some authors. Wallace et al.[79] reported excellent results with conservative management using a hard cast for one month before switching to a functional brace for one more month.

General Complications

Re-rupture complicates both surgical and conservative management, but it is generally more common with conservative management.[83] Early careful ankle mobilization and full weight bearing after primary Achilles tendon repair does not increase the risk of re-rupture.[67]

Deep vein thrombosis may follow surgical and conservative management,[98] and has a number of documented risk factors. It may be difficult to diagnose as the patients are often in equinus cast for some weeks. It is not common following Achilles tendon repair. Arner and Lindholm reported two cases of deep vein thrombosis in 86 patients following open repair of Achilles tendon.[82] One of these patients developed a pulmonary embolism and subsequently died. Deep vein thrombosis and

associated pulmonary embolism can be minimized by early mobilization, making sure that the patient receives appropriate prophylaxis.[98] Achilles tendon operations can be performed under regional nerve blocks, which would avoid potentially prolonged immobilization and the risks of general anesthesia, including deep vein thrombosis.

Conclusion

Although it is the strongest tendon in the human body, Achilles tendon ruptures are common. The cause of rupture is still hypothesized and the incidence of rupture is increasing. The evidence for best management is still controversial, and, in selected patients, conservative management and early mobilization achieves excellent results. Surgery will decrease the chance of re-rupture but is classically associated with an increased risk of superficial skin breakdown. Percutaneous repair performed under local anesthesia and followed by early functional rehabilitation is becoming increasingly common, and has a decreased superficial infection rate. The strength of percutaneous compared to open repair is under question but the re-rupture rate is comparable. Appropriate randomized, controlled trials are lacking, and efforts should be made to run such studies to clarify the issues highlighted in this chapter.

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16
Conservative Management of Acute Achilles Tendon Rupture

Adam Ajis and Richard G.H. Wallace

Introduction

Hippocrates stated, "The tendon, if bruised or cut, causes the most acute fevers, induces choking, deranges the mind and at length brings death."[1] He was probably referring to injuries, some sustained in battle. Today such dire consequences are not seen after Achilles tendon rupture. Nevertheless it can result in significant morbidity and disability if not well managed. Such management can be either surgical or conservative. The case for conservative management of spontaneous rupture of the Achilles tendon will be discussed in this chapter and a well-tried protocol for conservative management will be described.

Background

There is a never-ending debate about the best treatment for acute Achilles tendon rupture, with strong views held about both surgical and conservative management. Those supporting the surgical approach feel that the correct tendon tension can be achieved only by direct visualization, and further suggest that surgical repair results in a lower rate of re-rupture.[2–5] It has also been said that open repair will allow earlier ankle mobilization. However, surgery is not without disadvantages, including hospital admission, anesthesia, wound healing problems, and increased cost.

Most publications on the treatment of Achilles tendon rupture refer to surgical management and generally deal with relatively small numbers of patients.[6–8] There have been only three prospective randomized studies and in these and other studies classical immobilization in a plaster \cast was used rather than functional bracing.[6,7,9] The concept of functional bracing was developed by McComis et al.,[8] but again the numbers were small.

The lack of a universally accepted scoring system makes comparison of reports on surgical and conservative management difficult. The criteria proposed by Boyden et al.[10] were modified by Lippilahti[11] and later used to compare the results of a large study of conservative and orthotic management of acute Achilles rupture, which found as good if not better overall results than those published for surgical management.[12]

Nonoperative treatment of Achilles tendon rupture is not a new concept. John Hunter ruptured his Achilles tendon while dancing and treated himself with strapping. Pels-Leusden advocated this method in the early 1900s.[13]

The author observed that, when operated on within 24 hours, it was clear that as the Achilles tendon was exposed and the ankle well plantarflexed, the tendon ends came together anatomically without further assistance. At this stage, hematoma did not prevent the tendon ends approximating very well. Attempts to suture two ragged tendon ends inevitably led to some bunching up of the tendon, sort of like trying to suture two strands of wet spaghetti end to end! It was not thought that the suture had any real functional strength; rather it was simply holding the tendon ends in good anatomical position while natural healing and repair took place. If this could be achieved reliably by conservative means, then surgical intervention would surely not be required.

Table 16.1. Physiotherapy

Stage 1	Cast removed; Pneumatic Walker fitted • Gradual progression to full weight-bearing, wearing Pneumatic Walker with initial protection of crutches • Regular removal of Pneumatic Walker to perform active ankle and subtalar exercises
Stage 2	Pneumatic Walker removed Normal footwear; +/−1-cm heel lift if required • Increase active range of movement exercises • Stretching exercises against floor resistance • Gradual strengthening exercises • Balance exercises

Furthermore, older patients with Achilles tendon rupture who were unfit for anesthetic were treated conservatively and found to do very well. If the older individual, who could be expected to have poorer healing, still recovered well without surgery, then the younger and fitter patient should do at least as well.

The author therefore developed a protocol (see Table 16.1) for conservative management of acute Achilles tendon rupture. Only those patients who had been placed in an equinus cast within 24 hours of rupture were included in the initial trial. The equinus position was maintained in a cast for four weeks with the patient strictly non-weight-bearing. A removable, custom-made double shell patellar-tendon-bearing orthosis with 20° of plantarflexion was then provided with overshoe, and weight bearing commenced (Figs. 16.1 and 16.2). Patients were advised to remove the orthosis in bed at night and also during the day while seated to perform regular active ankle exercises.

Figure 16.2. Overshoe with heel lift.

After 140 patients had been managed successfully using this protocol, three patients were given the usual custom-made orthosis but also provided with an InCare Pneumatic Walker, including appropriate heel lifts within the splint (Fig. 16.3).

Figure 16.1. Double shell PTB orthosis.

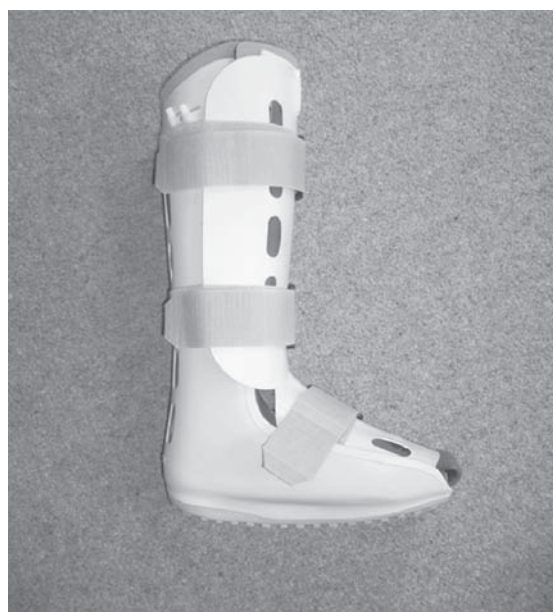


Figure 16.3. InCare Pneumatic Walker.

They were asked to alternate these splints on a daily basis. At subsequent review, all three stated that the double shell orthosis was comfortable and caused no problems. However, they expressed a strong preference for the Pneumatic Walker because of convenience and even better comfort. Since then, over 400 more patients have been treated using the Pneumatic Walker for the second month of treatment. The rocker sole of the brace further protects the healing tendon by reducing the lever effect of the foot during walking. At an early stage in the development of the treatment protocol structured physiotherapy was introduced with good effect.

A preliminary study of the first 32 consecutive patients presenting with acute Achilles tendon rupture was very encouraging,[14] and this lead to further work in which an independent physiotherapist carried out a detailed study of the next 140 consecutive patients.[12] Over 550 consecutive patients have now been treated using the conservative protocol. The re-rupture rate has remained at less than 4%. All those suffering a re-rupture were again treated using the same conservative regime with excellent results in all but three, who required surgery after which they did very well. One patient, an orthopedic surgeon, who sustained an early complete re-rupture was able to return to highly competitive international tennis following a second course of conservative treatment. Many patients treated conservatively have returned to their previous sporting level and one patient was observed skiing barefoot on a choppy lake 18 months after his rupture.

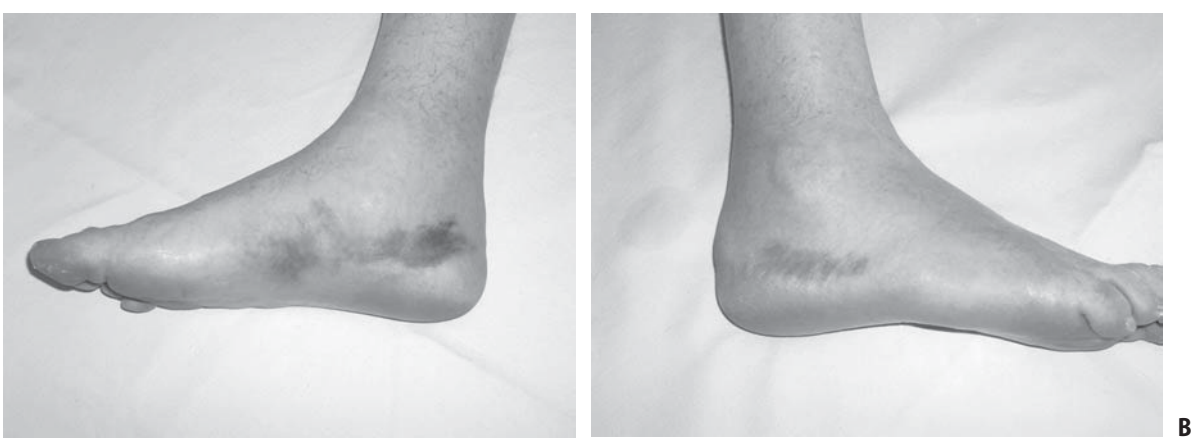
Protocol

Patients presenting at A&E departments with suspected acute TA rupture are placed in an equinus plaster of Paris cast and referred to a weekly central TA rupture clinic. Ideally they will be seen at this clinic between one and two weeks after the rupture. The cast is removed and the patient is assessed.

Observation of the patient with a recent TA rupture will reveal classical bruising on both medial and lateral sides of the heel (Fig. 16.4). This bruised area will not be tender and there will be no bony tenderness about the ankle. A calf muscle tear is excluded and the tendon itself is examined. With tenderness over the Achilles tendon and a palpable gap one can be confident of the diagnosis. The absence of plantarflexion on calf squeeze adds further reassurance to this diagnosis.

The ankle is then well plantarflexed and the tendon palpated to ensure that the tendon ends are felt to oppose well. This is a reliable clinical test in experienced hands and it is rare that further tests such as ultrasound will be required.

A suitably padded synthetic cast is applied with sufficient plantarflexion to achieve satisfactory apposition of the tendon ends as per the clinical assessment (Fig. 16.5). The patient is then instructed to use elbow crutches and remain strictly non-weight-bearing until the next appointment at the clinic. The conservative management of the rupture, expected progress, and outcome are all explained in appropriate detail to patients



A B

Figure 16.4. (**A, B**) Classical bruising of acute Achilles tendon rupture.

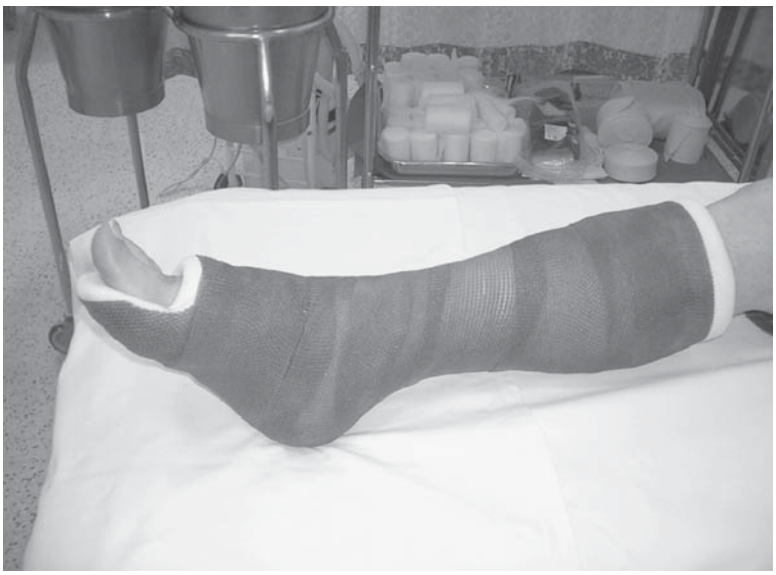


Figure 16.5. Lightweight equinus cast.

and those accompanying them. An advice leaflet giving all these details is also provided.

The next review is timed to ensure a total of four weeks in the equinus cast. At this review the cast is removed and the integrity of the Achilles tendon is assessed by an experienced clinician. The tendon is first palpated to ensure continuity. The patient is then asked actively to plantarflex the foot against resistance of a single finger of the examiner while the examiner's other hand palpates the tendon, feeling for any "fiber tearing." The examiner gradually applies an increasing resistance while still palpating the tendon. By this means it is possible to ascertain if the tendon has healed sufficiently to progress to the next stage of the treatment. If not, a further week or two in an equinus cast may occasionally be required—such as in a rheumatoid patient on steroids.

Where the healing progress is considered satisfactory, an InCare Pneumatic Walker of suitable size is fitted (Fig. 16.6). Heel lifts are added to allow comfortable weight bearing on the heel (Fig.

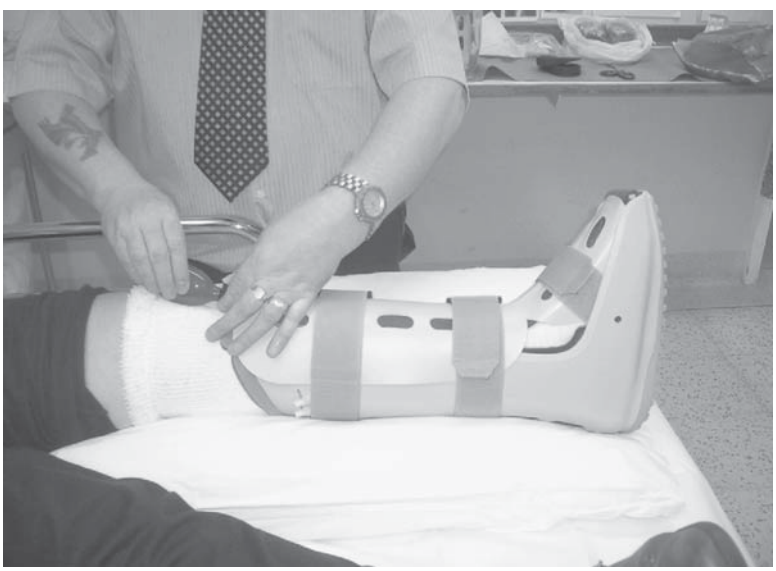


Figure 16.6. Fitting of Pneumatic Walker.

16.7). The height of the lift varies from one patient to another. The Pneumatic Walker is to be worn at all times when standing or walking for four weeks. It is stressed that standing or trying to hop without the brace is absolutely forbidden. Patients are advised that they may weight bear fully in this brace, but usually require the support of crutches for the first day or two (Fig. 16.8). The splint may be removed in bed with strict instructions that it must be reapplied before getting out of bed for any reason. Bathing is allowed with advice to have a chair beside the bath to avoid any temptation to

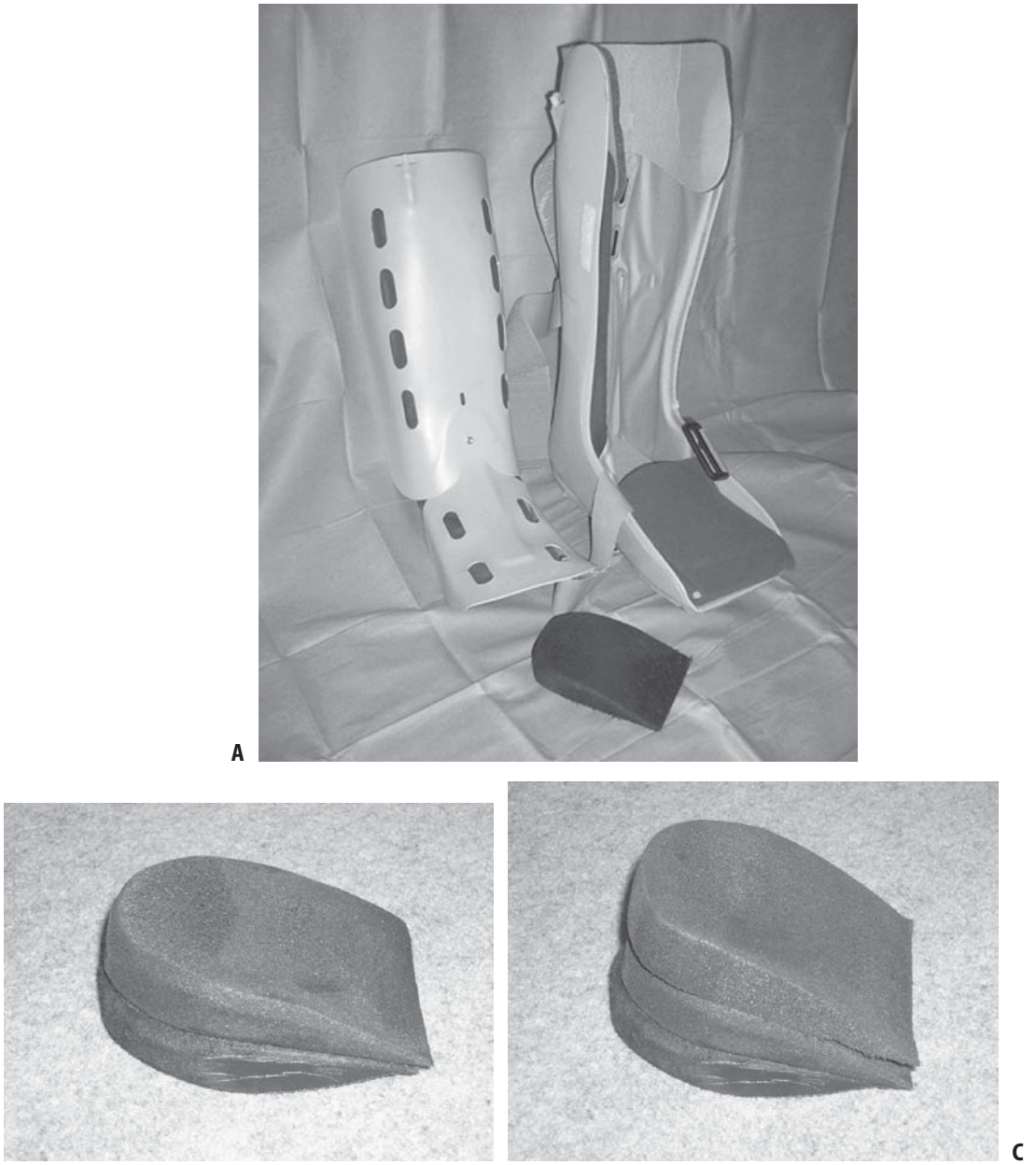


Figure 16.7. (**A–C**) Pneumatic Walker and heel lifts, which can be increased as required.

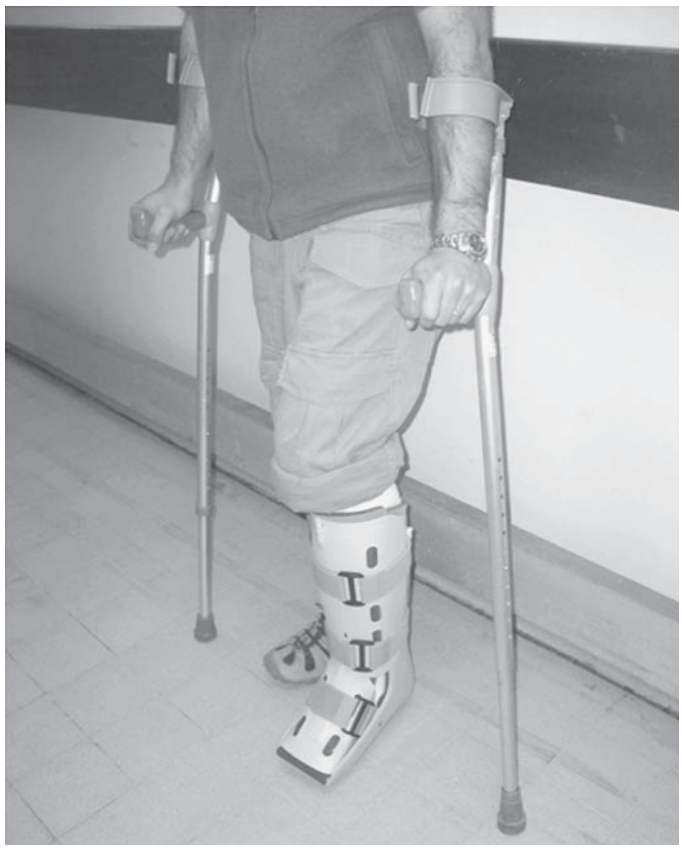


Figure 16.8. Patient weight bearing in Pneumatic Walker.

stand without the splint. Unless there is a shower seat patients are told not to use a shower. Through the day patients are encouraged to remove the brace as much as possible while seated to facilitate active ankle and subtalar exercises. Immediately following fitting of the brace, patients attend the physiotherapy department for instruction on active exercise. There is an open access policy so that any problems or anxieties may be addressed promptly. Where appropriate, the heel lift in the Pneumatic Walker is reduced after two weeks.

Management Problems

Diagnostic difficulty can arise with a calf muscle tear as the presenting history is often very similar to that of an Achilles tendon rupture, including a description of the sensation of a kick or blow to the back of the ankle. Careful examination will usually resolve the diagnosis as there is no palpable gap in the tendon and the tenderness is usually located either in the medial or lateral head of gastrocnemius. The tendon itself may be tender but rarely is it as severe as with a tendon rupture. There is also a different distribution of bruising, although this takes some time to develop and is not evident at the acute presentation (Fig. 16.9). Such patients are best treated with simple elastic support and crutches. Gradual weight bearing is allowed as symptoms permit, and ankle exercises are encouraged with the assistance of the physiotherapists.

Incomplete rupture will present with a similar history to that of a complete rupture and there is a palpable defect. However, plantarflexion power is greater than could be explained by action of the

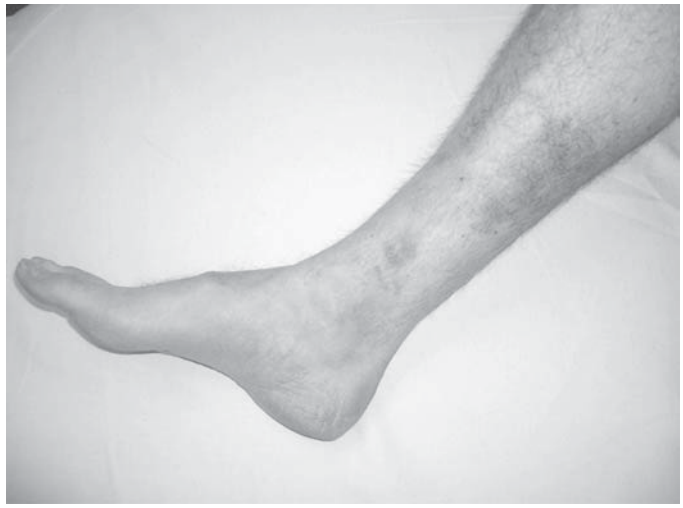


Figure 16.9. Typical bruising of calf muscle tear.

other calf muscles alone. Nevertheless, it is the author's view that these patients should still be managed using the protocol as for a complete rupture of the tendon. Patients with an incomplete rupture can present late and this may lead to difficulties with management. In these cases an ultrasound examination can help to clarify how much of the tendon remains intact. In the author's experience any decision to manage surgically is made on the basic clinical assessment rather than relying on diagnostic imaging. The patient's age, level of activity, expectations, and general medical status all have to be taken into account.

Simultaneous bilateral Achilles ruptures can be managed conservatively and there is no need to operate on these patients. A wheelchair is required while both legs are in short-leg equinus casts, and, if good family support is not available, hospital admission will be required. Staged removal of the casts and provision of the Pneumatic Walker one week apart is advised. This is again adopted when the splints are to be removed and free weight bearing commenced.

Patients presenting late are in general not suitable for conservative management. At surgery the tendon ends are separated by organizing hematoma and frequently have become adherent to the adjacent tissues. Postoperatively these patients are then managed using the standard conservative protocol.

Conclusion

This protocol of conservative management can be adopted successfully for routine management of acute Achilles tendon rupture at all ages and levels of athletic activity. However, the supervising clinician must be experienced in this method of treatment and be able to decide when surgical intervention is required. Furthermore, the supervising clinician should be experienced in such surgery.

Conservative management must not be regarded as an easy management option. To achieve good results, which can be reasonably expected, close supervision by a senior and experienced clinician is essential. The author has, on a number of occasions, had to deal with poor results arising from care by clinicians who thought that an Achilles tendon rupture could be easily treated by just leaving it in a plaster cast for a few weeks. The inexperienced often take shortcuts and make inappropriate alterations to the protocol, resulting in an inevitably poor outcome for their patients.

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17
Open Surgery for Achilles Tendon Ruptures

Louis C. Almekinders and Nicola Maffulli

Introduction

Acute Achilles tendon rupture is the most dramatic injury that affects the Achilles tendon. Although it is generally considered the strongest tendon in the body, the Achilles tendon is also one of the most common tendons affected by spontaneous complete rupture. As previously discussed, the injury typically affects middle-aged "weekend athletes." Generally, in these patients the Achilles tendon first develops a pathologic alteration approximately 2 to 5 cm proximal to the calcaneal insertion.[1] This weakening is frequently described as a degenerative tendinopathy, but it is not clear whether this is similar to the midsubstance tendinopathy that develops in other athletes associated with overuse. The pre-rupture tendinopathy is generally not associated with pain, whereas the typical "overuse" tendinopathy is painful, and generally does not lead to complete rupture. This suggests that these conditions are different entities with some overlap in the clinical features.

Many theories have been advanced explaining the pre-rupture degenerative changes that lead to tendon weakening. Poor tendon vascularity, age-related tendon cell apoptosis, disuse atrophy, poor conditioning and flexibility, training errors, and poor foot wear have all been proposed as etiologic factors in tendinopathy.[2]

Clinical Evaluation

Patients who sustain an acute Achilles tendon rupture usually describe a sudden sharp pain in the posterior aspect of the ankle. Many will insist that they felt they were "stepped on" or were "kicked in the back of the heel." However, generally this is a noncontact injury, and a sudden push-off and landing is responsible for the mechanical load that causes the rupture. Following the initial acute pain, pain decreases dramatically to a point where many patients do not seek immediate medical attention. Some persistent pain and diffuse ankle swelling may be noted in the subsequent days. Medical evaluation requires a certain degree of clinical suspicion to reach the correct diagnosis. Active plantarflexion is still possible through the peroneal and posterior tibial tendons, although it is weaker than the contralateral, and repeated toe raises are usually not possible. A defect in the tendon can usually be palpated, and the calf squeeze test is positive. During the calf squeeze test, the patient is generally prone on the examining table with both feet hanging freely off the end. The examiner squeezes the proximal muscular half of the calf, avoiding direct pressure on the tendon. On the unaffected side, plantarflexion of the foot is evoked during the squeezing maneuver. This is a negative test. On the ruptured side, no movement of the foot is noted during the squeezing maneuver. Another simple, no-touch test is the knee-flexion test. The patient lies prone on the examination table, and is invited to flex both knees so that the tibia comes perpendicular to the floor. On the injured side, the angle between the anterior aspect of the shin and the dorsal aspect of the foot is more acute than on the uninjured side (Fig. 17.1).

Imaging studies are generally not needed unless there is some clinical suspicion for concomitant injuries. Both ultrasound and MRI can allow

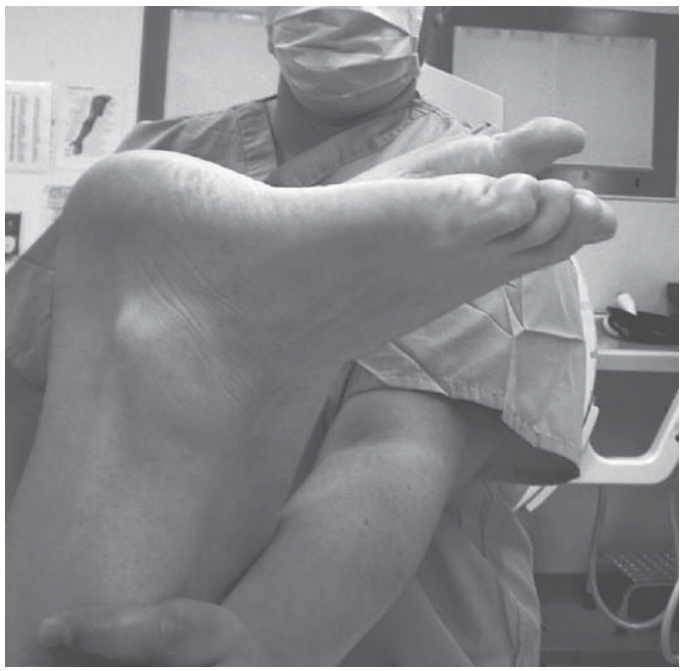

Figure 17.1. The knee flexion test for diagnosis of Achilles tendon tear. The patient lies prone on the examination table, and is invited to flex both knees so that the tibia comes perpendicular to the floor. On the injured side, the angle between the anterior aspect of the shin and the dorsal aspect of the foot is more acute than on the uninjured side.

excellent visualization but the clinical implications of these imaging tests with regard to treatment are minimal.

Once the diagnosis has been made, treatment decisions will have to be made based on the patients' expectations and wishes. The acute Achilles tendon rupture can be managed by nonoperative and operative means. Controversy persists as to the ideal approach to this injury. In the past decade or two, the prevailing opinion has been that nonoperative treatment is better suited for the elderly, low-demand patient. This was based on cohort studies that suggest that operative treatment allowed a more aggressive rehabilitation with a lower re-rupture rate once vigorous activities were resumed.[3] Operative treatment carries the risk of complications such as infection, tendon calcifications, and other wound healing problems that can result in poor outcomes.[4,5] The rate of wound complications has been reported at over 10% in one large cohort study.[4] Conversely, nonoperative treatment was thought to require more prolonged and more extensive immobilization in order to allow the tendon to heal and minimize re-ruptures. Particularly in elderly patients, nonoperative treatment avoids the risk of infection and wound breakdown. This approach has been challenged by comparative studies as well as meta-analysis comparing surgical repair with a nonoperative approach.[6–8] Early, protected activities and aggressive rehabilitation resulted in outcomes that were similar to operative treatment.

At this point, many surgeons continue to recommend operative treatment for acute Achilles tendon ruptures. However, it is important to explain to patients that the nonoperative approach can be successful, and to allow the patient to participate in the treatment decision. Once surgical management has been chosen, the surgical approach needs to be considered. Classically, a surgical exposure of the rupture site is used to perform the repair. The following section will describe in detail the standard open approach. However, a percutaneous approach or limited open approaches can yield satisfactory results.[9–11] In the percutaneous approach, the rupture site is generally not exposed. The sutures used to repair the rupture are placed and tied in a percutaneous fashion. The specifics of this technique are described in another chapter in this book.

Surgical Technique

The patient is placed in the prone position under general or regional anesthesia. If the feet are left on the operating table, there is a tendency to push the ankle joint in hyper-plantarflexion due to the weight of the leg. During the repair, this can lead to overtightening the repair. Instead, it can be helpful to bring both feet down off the end of the table or to place a soft roll underneath the distal tibia. Both feet will assume an unforced, more neutral position. Prior to the repair, the affected foot is in neutral or slight dorsiflexion. During the repair, this approach will tend to make it easier to judge the tension of the repair and the normal balance between plantar- and dorsiflexors by comparing it with the natural equinus position of the unaffected leg. If a tourniquet is used, it should be applied to the thigh. A calf tourniquet may make mobilization of the muscle difficult. The foot and leg are prepped and draped in the usual manner. The site of rupture is often readily identified through direct palpation.

The initial incision is centered over the rupture site. Most often a posteromedial approach is recommended in order to minimize potential injury to the sural nerve, and to allow easy access to the plantaris tendon if needed. A straight posterior approach may create a tender scar on the heel counter of the shoe. It may be reasonable to start with a relatively small, two-inch incision. The amount of tendon fraying and damage on each side of the rupture is somewhat variable. If intact, firm tendon can be exposed through a small incision, and no additional dissection is needed. If the tendon is frayed and of poor quality over a longer segment, generally a longer incision will have to be made to expose healthy tendon.

Following the skin incision, the edges of the wound should be handled with great care. The skin and subcutaneous tissue are relatively thin and without an abundant blood supply. Initially, the dissection should be taken straight down to the fascia overlying the tendon (Fig. 17.2). Subcutaneous dissection with development of skin flaps should be avoided. Frequently, the crural fascia is intact even at the site of rupture. An attempt can be made to incise this sharply and use this layer for a separate closure when the repair is completed. Following incision of the fascia in line with the skin incision, the rupture site is usually immediately evident. Blood and blood clots are evacuated and rinsed from the rupture site. At that point, both ends of the tendon are gently dissected free to a level where relatively healthy, non-frayed tendon is visible. The amount of dissection can vary from patient to patient. Care should be taken to minimize dissection of the anterior aspect of the tendon. This is thought to minimize chances of additional vascular insult to the tendon, as this is the location of feeding vessels into the tendon. Frequently, the ends of the ruptured tendon are

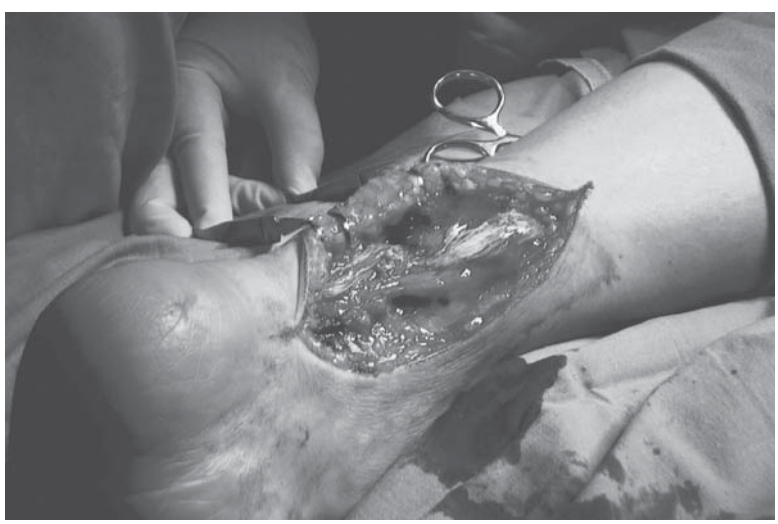

Figure 17.2. A torn Achilles tendon at open surgery. Note the swollen, injected paratenon.

sharply debrided, but it is not known whether this will improve healing. However, given the studies showing that this is an area of preexisting tendon degeneration,[12] it seems reasonable to debride the area prior to repair. In addition, not infrequently, long, thin strands of disrupted collagen bundles mark the frayed end of the tendon, making suture placement difficult. On the other hand, extensive debridement is not recommended since this may force the surgeon to overtighten the muscle tendon unit in an attempt to bring the resected tendon ends together.

Suture choice and methods of suture placement have been the source of much debate. Generally, most reports describe the use of two to six strands of large absorbable or nonabsorbable suture according to the surgeon's preference.[13–15] Some reports have also shown good results with the use of a pull-out wire.[16] The core sutures can be placed in several ways. Bunnell-, Kessler- and Krackow-type suture placement have been described.[13,17] No controlled clinical studies are available that conclusively show the superiority of one configuration over the other. Studies on sutures generally measure the initial holding strength, but do not address clinical outcome. One biomechanical study suggested that a locking suture has superior holding strength.[18] We favor a Kessler-type suture, possibly with a Tajima locking modification. This involves a locking loop and the knots are buried within the repair site rather than on the surface of the tendon. The locking feature also allows the assistant to coapt the tendon by the two untied ends while the surgeon ties the other two ends. Again, some care must be taken not to overtighten the repair (Fig. 17.3).

Once the core sutures are tied, some smaller peripheral sutures can be placed to augment the repair and improve the coaptation of the tendon. Most authors do not use any augmentation at the time of initial open repair of an acute Achilles tendon rupture, as there is an increase in complication rate in some augmented repairs.[19] Occasionally, an augmentation can be considered if the suture repair has poor holding strength and yields poor coaptation. Artificial materials such as polyester tape[20] and a braided polypropylene interposition[21] have been used for augmentation with acceptable results. Possible autogenous sources of augmentation are the plantaris tendon and a section of proximal Achilles tendon. If the plantaris tendon is present, the tendon is usually not injured (Fig. 17.3), and is in the operative field as an available graft option. The plantaris tendon can be detached proximally and distally as far as possible. Conversely, the tendon can be left attached distally, since the rupture is generally close to the distal attachment. The tendon is then woven through the ruptured

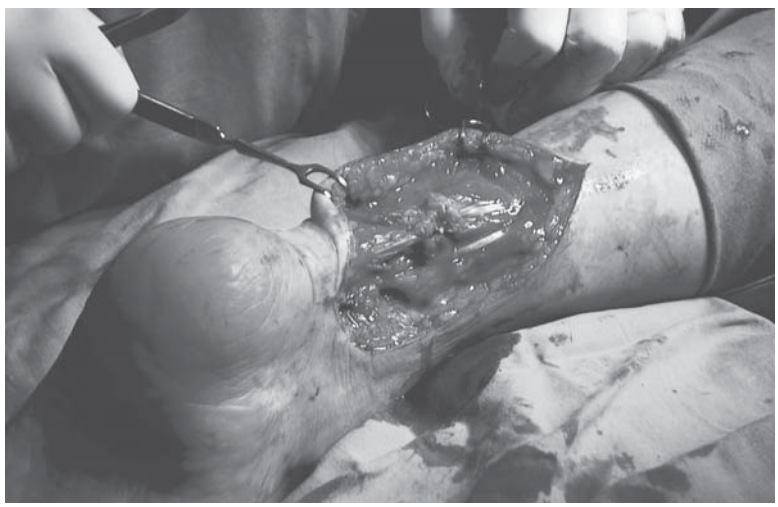

Figure 17.3. The tendon ends have been sutured to each other with a modified Kessler suture. Note the intact plantaris medial to the Achilles tendon.

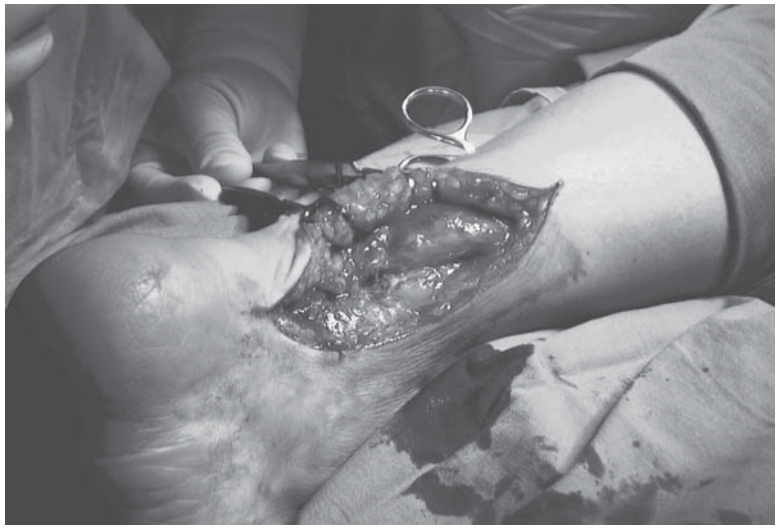

Figure 17.4. The paratenon has been sutured over the Achilles tendon. A suture of the soft tissues in layers will now take place.

ends and sutured in place. An alternative is one or two proximal strips of tendon tissue harvested from the proximal tendon and gastrocnemius aponeurosis.[22] The strips are detached proximally, turned down onto the repair site, and sutured in place. Both methods of augmentation generally require a large incision and more dissection than isolated repair only.

Once the repair is completed, an attempt is made to close the surrounding fascia. This may minimize adhesions of the tendon to the surrounding tissue. However, the fascia is frequently thin and may not hold sutures well (Fig. 17.4). Following closure of the skin, a short leg cast is applied in equinus.

Rehabilitation following open repair of the Achilles tendon has been variable. Often, a fairly conservative program is used with initial static splinting in an equinus position. After 4 to 6 weeks, the foot is gradually brought up in a plantigrade position, and weight bearing is initiated. Range-of-motion exercises are generally initiated early after the initial splint is removed and sutures are removed. More recently, studies have shown that early weight bearing does not adversely affect the outcome following open repair.[23] In addition, early motion as opposed to immobilization has resulted in improved outcome in calf strength[24] and does not increase the risk of re-rupture or wound healing.[25] Based on these reports, there is a trend toward gentle mobilization following surgical repair.

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18
Chronic Ruptures of the Achilles Tendon

Jonathan S. Young and Nicola Maffulli

Introduction

Complete rupture of the Achilles tendon can be experienced both by sedentary patients and athletes.[1] It is especially common in middle-aged men who occasionally participate in sports.[2–5] Chronic rupture of the Achilles tendon is troublesome for both patient and surgeon, and can be associated with functional operative morbidity. While the role of operative and nonoperative treatment of acute ruptures continues to be debated,[2,5,6–10] most authors agree that neglected ruptures should be treated operatively unless there are significant contraindications to surgery or the patient has minimal functional demands. Platt in 1931 wrote, "In untreated ruptures the thickened sheath becomes adherent to the tendon ends and acts as a feeble bond of union . . . the power of plantar flexion is permanently impaired."[11]

The definition of chronic rupture is variable. The most commonly used timeframe, which will also be used in this chapter, is four weeks from the time of injury.[4,12–29]

Pathophysiology

Even at the beginning of the twentieth century it was noted that chronic rupture of the Achilles tendon caused difficulty and impairment of plantarflexion.[11] The sheath may become thickened or adhere to the retracted tendon ends, and will act as a weakened plantarflexor.[11] Often there is no tendinous tissue inside the sheath at the site of the defect due to tendon retraction.[29] Zadek describes the retracted tendon ends as a conical-shaped proximal tendon stump, and a bulbous distal stump.[29] The proximal tendon stump is often adherent to the fascia posterior to the flexor hallucis longus muscle belly.[30] The tendon of plantaris, if present, may be hypertrophied. Another common finding is thick scar tissue bridging the site of rupture,[10,24,31,32] due to partial regeneration of the tendon.[33] After 56 days following calcaneal tendon resection in rabbits, a well-organized connective tissue was formed; by 240 days it still did not display the fascicular arrangement of a tendon. This new tissue is not as strong as normal tendon, and will elongate with time.[10,34] Whatever the pathological findings at the site of rupture, there is usually a gap between the tendon ends. Retraction of the proximal stump results in shortening of the gastrocnemius-soleus complex, and weakness of plantarflexion of the ankle. The tension that the muscle fiber can produce decreases as the fiber shortens, until it becomes zero when the fiber is approximately 60% of its resting length.[35]

Diagnosis

Clinical Examination

Most patients with acute rupture of the Achilles tendon give a history of sudden sharp pain in the back of the calf, as if they have been kicked.[9,36] Patients with a chronically ruptured Achilles tendon may recall a minor trauma with pain in

their heel. Later, routine day-to-day tasks such as climbing stairs becomes difficult.[5] Older series report 18–20% initial rates of misdiagnosis.[2,13,32]

In acute ruptures, providing the swelling is not severe, a palpable gap can often be palpated along the tendon. The gap is most often two to six centimeters proximally from the insertion of the tendon. On average the rupture site lies 4.78 centimeters proximal to the calcaneal insertion.[37]

In most patients, the clinical diagnosis of Achilles tendon rupture does not pose a problem. However, in the absence of sports participation, or an additional painful event, the injury may be missed or misjudged either by the patient or the doctor. Further difficulties to reach the correct diagnosis may occur in elderly patients and if the patients consult a doctor some days after the injury: Achilles tendon ruptures are missed in up to 20% of patients.[13,38] In elderly patients with an Achilles tendon rupture, nine (36%) of 25 patients had a delay of more than one week to treatment.[39] For chronic rupture of the Achilles tendon, the pain and swelling may have subsided and the gap between the proximal and distal ends of the Achilles tendon may have filled with fibrous tissue.[9,40,41] Therefore, a less apparent or absent gap exists between the tendon ends. Subtle evidence of pain and swelling around the proximal and distal stumps of the ruptured tendon may be present.[17] Active plantarflexion is also possible by the action tibialis posterior and the long toe flexors, contributing to a delayed diagnosis.[42] Active plantarflexion will, however, be weak and associated with a limp.[17] If there is uncertainty or confirmation is required, a series of special tests can be undertaken.

Special Tests

The calf squeeze test is a straightforward test. It is often described as Thompson's[43] or Simmonds'[44] test, though Simmonds described the test five years before Thompson. The patient lies prone on an examination couch with both feet hanging over the edge. The examiner squeezes the calf. If the Achilles tendon is intact, the foot will plantarflex against gravity, and the results of the test should be compared against the other leg. The plantarflexion occurs because on calf squeezing, the muscle is deformed, thus causing the overlying Achilles tendon to bow away from tibia. Matles' test[45] is carried out with the patient lying prone on an examination couch. Both legs are flexed at the knee to 90°. On the side without the rupture, the ankle will remain in plantarflexion. On the side with the rupture, the ankle will dorsiflex or remain in the neutral position. There is a theoretical risk of a false positive result if the plantaris tendon is intact.

Simmonds' and Matles' tests are probably the two most common tests used to aid diagnosis, but for reinforcement of the diagnosis one can employ O'Brien's needle test[46] or Copeland's sphygmomanometer test.[47] To perform the needle test a hypodermic needle is inserted through the skin of the calf, approximately half a centimeter off the midline and approximately 10 cm proximal to the insertion of the tendon. The needle should be inserted until its tip is just within the substance of the tendon. The ankle is then dorsiflexed and plantarflexed. If on dorsifexion the needle points distally, the portion of the tendon distal to the needle is presumed to be intact. If the needle points proximally, there is presumed to be a loss of continuity between the needle and the site of insertion of the tendon.[46]

For the sphygmomanometer test, a sphygmomanometer cuff is wrapped around the middle of the calf with the patient lying prone. The cuff is inflated to 100 mmHg (13.3 kilopascals) with the foot in plantarflexion. The foot is then dorsiflexed. If the pressure rises to around 140 mmHg (18.7 kilopascals), the musculotendinous unit is presumed to be intact. If the pressure remains at or around 100 mmHg, then it is likely the patient has an Achilles tendon rupture. If two of the aforementioned tests are positive, the diagnosis of Achilles tendon rupture is certain (Maffulli 1998).[40]

Imaging

For the trained eye, lateral radiographs of the ankle can be used to aid the diagnosis of rupture. The triangular fat-filled space between the anterior aspect of the Achilles tendon, the posterior aspect of the tibia, and the superior aspect of the calcaneus is called Kager's triangle.[48] This area loses its configuration and can be distorted if there is a rupture. Deformation of the contours of the distal segment of the tendon from loss of tone is the most likely radiographic change associated

with rupture.[49] Radiographs also assist by ruling out a calcaneal avulsion and prior foot and ankle difficulties.[42] In a series of chronic ruptures, calcification in the distal portion of the proximal stump of the Achilles tendon was present in three of seven patients.[17] Additionally attritional changes due to old athletic injuries are often apparent on plain radiograph.[42]

Real-time high-resolution ultrasonography is another useful diagnostic aid. It is relatively inexpensive, fast, repeatable, and has the potential for dynamic examination.[50] It is user dependent, and may require substantial experience to operate the probe and interpret the images correctly.[50] Ideally, a linear array transducer probe (as sector-type transducers produce excess obliquity at the edges) with a spacer gel should be held at right angles to the tendon. This ensures that the optimum amount of ultrasonic energy is returned to the transducer, thus enabling the production of good dynamic and panoramic images of the tendon.[51,52] The longitudinally arranged collagen bundles of the Achilles tendon reflect the ultrasound beam. High-frequency probes provide the best results.[53] A normal Achilles tendon appears as a hypoechogenic, ribbon-like image contained within two hyperechogenic bands that are separated when the tendon is relaxed and more compact when the tendon is under tension. When the Achilles tendon ruptures an ultrasound scan reveals an acoustic vacuum with thick irregular edges.[54] Ultrasound of Achilles tendon rupture has greater sensitivity than ultrasonic examinations for other traumatic soft tissue injuries of the lower limb.[55]

Magnetic resonance imaging allows determination of the extent and nature of the condition of the tendon ends in complete Achilles tendon tears.[56] Both T1 and T2 weighted images should be used to evaluate the tendon for rupture. The images should be in the axial and the sagittal planes. Subtle thickness changes are detected in the axial plane and the longitudinal extent of the tear on sagittal images. A normal Achilles tendon is viewed as an area of low signal intensity on all sequences. The tendon will taper smoothly and show no focal defects. The dark band of the tendon is well contrasted from the high signal intensity of the pre-Achilles fat pad.[56] Any high-signal intratendinous intensity is viewed as abnormal.[56,57] For a T1 weighted image, a complete rupture is visualized as disruption of the signal within the tendon, mixed with hemorrhage and edema that localizes in the pre-Achilles fat pad. Older complete tears display hemorrhage as low signal intensity on T1 weighted images. A T2 image will show generalized increased signal intensity representing the edema and hemorrhage within and around the ruptured tendon.[56] Discontinuity, fraying of the tendon, widening of the tendon edges, the abnormal orientation and condition of the fibers, and retraction of the tendon edges into the calf are more comprehensively seen on T2.[56]

Injury Guidelines and Classification with a View to Treatment

The size of the Achilles tendon defect is likely to affect management. Both Myerson[10] and Kuwada[58] have noted this and each have provided a scheme for management.

Myerson treats ruptures of the Achilles tendon based on the size of tendon defect:[10]

- Defects of 1 to 2 cm are treated with end-to-end anastomosis and posterior compartment fasciotomy.
- Defects between 2 and 5 cm are repaired using V-Y lengthening, and occasionally augmented with a tendon transfer.
- Defects greater than 5 cm are repaired using tendon transfer alone or in combination with V-Y advancement. Due to the bulk of the tendon at the point of which it is passed inferiorly, he prefers not to use a turn-down flap, but acknowledges that it does have a role.

Kuwada grades Achilles tendon injuries I to IV:[58]

- Type I injuries are classified as partial tears treated with cast immobilization.
- Type II injuries are complete ruptures with a defect up to 3 cm. These are treated with end-to-end anastomosis.
- Type III injuries have a 3–6-cm defect after debridement of the proximal and distal ends of the Achilles tendon to healthy tissue. This grade of defect requires a tendon graft flap, possibly augmented with synthetic graft.
- Type IV injury is a defect that is greater than 6 cm and requires gastrocnemius recession, a free tendon graft, and/or synthetic graft.

The Kuwada classification scheme for tendo Achillis rupture is based on 28 repairs and 102 gastrocnemius recessions.[58]

Management

The management of chronic and neglected rupture of the Achilles tendon is usually different from that of acute rupture, as the tendon ends have retracted. The blood supply to this area is poor, and the tendon ends have to be freshened to allow for anastomotic healing. Due to the increased gap, primary repair may be difficult.[41] Subsequently the repair requires reinforcement (augmentation); this is can be done by the use of a turn-down flap, the use of a tendon transfer, tendon graft, or by using synthetic materials.[17,18,21,42,59,60]

Nonoperative Management

Christensen[61] reported a series of neglected ruptures treated conservatively: 18 of 51 patients with 57 ruptures (nearly two-thirds of which were neglected) were treated conservatively, either because the operation was contraindicated or refused, or because the rupture was several months old and the triceps surae showed signs of regaining strength, and hence the injury was managed "expectantly" (11 patients). What were deemed satisfactory results (i.e., normal gait, return to previous occupation, and slight or no discomfort) were obtained in 75% of operated and 56% of nonoperated cases. In addition he reported that improvement in all nonoperated cases occurs slowly, sometimes over several years. Brace management should be considered in patients without functional deficit and in those with potential wound healing problems or anesthetic contraindications to surgery. An ankle-foot orthosis, with or without ankle hinge, may be used.[10]

Operative Management

V-Y Tendinous Flap

This procedure was reported by Abraham and Pankovich in 1975,[62] and was applicable to neglected, chronic ruptures of the Achilles tendon. The aim of this procedure was to achieve end-to-end anastomosis of the Achilles tendon. This is made possible by a sliding tendinous flap developed over the proximal portion of the tendon, by making an inverted V incision that is then repaired in a Y fashion. Abraham and Pankovich reported on four patients,[62] with three of the patients regaining full strength of the triceps surae muscle and able to raise their heels from the floor equally when on tiptoe. One patient continued to have slight weakness of the triceps surae muscle, and the heel-to-floor distance on the operated side was two centimeters less than the operated side. The only complication was one sural nerve neuroma. Leitner et al.[20] reported on three patients with tendon defects of 9 to 10 cm managed successfully using this technique. Kessel et al.[63] used the same technique, augmented with plantaris weave and pullout suture in 14 patients. Parker and Repinecz[25] described a similar technique in which a tongue-in-groove advancement of gastrocnemius aponeurosis was used to close a 6.5-cm defect in one patient. They reported this technique as easier than V-Y advancement, and up to 50% more length can be accomplished.

Turn-Down Flaps

Turn-down flaps of the Achilles tendon have been reported to repair or augment chronic ruptures of the Achilles tendon. Christensen's technique was used for chronic and acute ruptures of the Achilles tendon.[61] A distally based 2-by-10-cm flap was cut in the proximal tendon fragment, turned down to cover the tendon defect or previous repair, and then the defect created by the flap was closed. Two re-ruptures occurred, and 75% of patients were classified as satisfactory. Using Silfverskiold's technique the graft was rotated through 180°, hence the smooth surface faced posteriorly. Arner and Lindholm[32] follow on from Silfverskiold's, but two flaps, one medial and one lateral, are rotated. The functional results of these techniques were reported as being approximately equal,[32] but Arner and Lindholm's technique produced less tethering of the scar. Gerdes[64] reported a cadaver study showing that flap augmentation repair had 41% higher ultimate tensile strength than simple suture repair alone.

Rush's operation to reconstruct a neglected rupture of the tendo Achillis uses the aponeurosis

of the gastrocnemius-soleus muscle fashioned into a tube.[24] The repair was felt to be strong and effective, and produced good results in five patients. Bosworth[34] reported on seven patients, five of which had chronic ruptures. He used a strip of the superficial part of the tendinous portion of the proximal stump of the Achilles tendon to augment the repair. He made a posterior longitudinal incision and dissected a half-inch by 7- to 9-inch strip of tendon on a distal pedicle. He then threaded this through the trimmed ends of the ruptured tendon and sutured it to them with the foot in plantarflexion. No complications occurred. Other authors have used V-Y advancement and flap turn-down in combination[65] or as isolated techniques[16] with good results.

Peroneus Brevis Transfer

Peroneus brevis tendon transfer for rupture of the Achilles tendon was popularized by Perez-Teuffer,[66] who operated on 30 patients. In the original technique, the peroneus brevis tendon is harvested from the base of the fifth metatarsal and passed through a transosseous drill hole in the calcaneum. The tendon was then passed back onto itself and sutured over the Achilles tendon. The ruptures in this series were acute with 28 of 30 patients able to return to their original level of sports. Turco and Spinella[42] augmented end-to-end repair of the Achilles tendon with a modification of Teuffer's technique, by passing the peroneus brevis through the distal tendon stump rather than the calcaneus. Although excellent results were reported, the criteria by which these results were obtained were not identified. McClelland and Maffulli[41] approach the Achilles tendon medially, and deliver the Achilles tendon through the posteromedial wound. The tendon is gently pulled through the inferior peroneal retinaculum; thus the blood supply is retained from the intermuscular septum. The peroneus brevis tendon is then woven through the ends of the ruptured Achilles tendon, passing through small coronal incisions in the distal stump, and then through similar incisions in the proximal stump. The tendon of plantaris, if present, can also be harvested to augment the repair if there is a large gap. A concern about the peroneus brevis technique is that there is the potential for eversion weakness.[10,17,67] This may not be a problem due to the retained peroneus longus having more than twice the strength of eversion of the peroneus brevis.[68] St. Pierre et al.[69] showed no significant loss of eversion strength following Evan's lateral ligament reconstruction. Also, if the tendon of peroneus brevis is placed distally in a lateral-to-medial direction, it does not duplicate the medial pull of the normal Achilles tendon.[17]

Flexor Digitorum Longus (FDL)

Mann et al.[17] described a technique using flexor digitorum longus as a graft in 7 patients. He used a medial hockey-stick incision for the Achilles tendon, and a second medial incision on the foot inferior and distal to the navicular extending toward the first metatarsophalangeal joint to allow access to the flexor digitorum longus. The FDL was then cut proximally to its division into separate digital branches. The distal stump was sutured to the adjacent flexor hallucis longus. The proximal stump of flexor digitorum longus was delivered into the wound. They also included a proximal fascial turn-down flap in all cases and, when length allowed, the proximal stump was reattached to the calcaneus with a pullout technique. They had six good or excellent results, no reruptures, and no functional disability secondary to loss of FDL.

Flexor Hallucis Longus (FHL)

Flexor hallucis longus has a long tendon that will allow bridging of large Achilles tendon defects. In athletic individuals, though, the loss of push-off from the hallux may cause difficulty when sprinting.[41] Hansen[70] advocates that the flexor hallucis longus muscle belly augments the strength of triceps surae and also improves the tendon's blood supply. Wapner et al.[71] reported the results of a small series of 7 patients treated using a flexor hallucis longus graft. Once harvested, the tendon was passed through a drill hole in the calcaneus and woven through the ruptured Achilles tendon ends. The distal end of the FHL was tenodesed to the tendon of flexor digitorum longus of the second toe. Three patients had an excellent result, three a good result, and one a fair result. Each patient developed a small but functionally

insignificant loss in range of motion in the involved ankle and great toe. Cybex testing revealed 29.5% decrease in plantarflexion power compared with the nonoperative ankle. No functional disability was noted secondary to FHL harvest. This is in agreement with Frenette and Jackson,[72] who reported 10 cases of FHL tendon laceration in young athletes, four of which were not repaired, with no disability evident. The theoretical advantages of FHL transfer include a long, durable tendon with a stronger muscle than other tendon transfers,[73] and the fact that the axis of FHL contraction most closely reproduces the Achilles tendon. The FHL fires in phase with the gastrocnemius-soleus muscle. Also anatomic proximity makes the surgical technique easier and avoids the need to disturb the neurovascular bundle or lateral compartment muscles. Harvesting of FHL allows maintenance of normal muscle balance of the ankle (i.e., plantarflexor to plantarflexor). Finally, this technique adds 10–12 cm of tendon compared with Hansen's technique, allowing weaving of the tendon through the Achilles. This technique is similar to that used by Dalal and Zenios,[19] who reported excellent results following reconstruction of three chronic ruptures in two elderly patients. Wilcox et al.[18] treated 20 patients with chronic Achilles tendinopathy, with a similar technique.

Gracilis

More recently, Maffulli and Leadbetter[74] have harvested the tendon of gracilis to aid repair of chronic ruptures of the Achilles tendon. After trying to reduce the gap of the ruptured Achilles tendon, if the gap produced is greater than 6 cm despite maximal plantarflexion of the ankle and traction on the Achilles tendon stumps, the gracilis tendon is harvested. This is accomplished through an incision centred over the distal insertion of the pes anserinus. The tendon of gracilis is then harvested with a tendon stripper. When present, the tendon of plantaris can be harvested with the tendon stripper, left attached distally, and used to reinforce the reconstruction.[74] Most patients were satisfied with the procedure; only two were classified as having an excellent result, although 15 of 21 patients achieved a good result.

Synthetic Materials

The advantage of use of synthetic materials is that the technique is relatively simple and lacks donor site morbidity. Howard et al.[75] used carbon fiber to repair five neglected ruptures. After a follow-up period from 4 to 19 months, the average plantarflexion strength was 88% compared with the opposite limb. All patients had excellent results, but some complications resulted, with stiffness in two patients and one delayed wound healing. Parsons et al.[76] used an absorbable polymer carbon fiber composite ribbon in 48 patients with Achilles tendon ruptures, 27 of which were chronic. The ribbon was woven through the proximal and distal stumps with 6 to 8 passes to bridge the defect. A proximal tendon flap was used "at the surgeon's discretion." From their own devised score, 86% had a good or excellent result; 29 patients had at least one year follow-up from the original 48 and we do not know if these were for acute or chronic ruptures. Complications included two re-ruptures, two deep infections, and three superficial infections. Investigations have been carried out on carbon fiber in sheep tendons.[77] Carbon fiber fragmentation has been shown in sheep calcaneal tendons associated with a poor collagen response. With polyester implants, the neotendon was denser, more collagenous, and closely adherent.[77]

Ozaki et al.[28] used three layers of Marlex mesh (polypropylene) to reconstruct neglected ruptures. The gaps ranged between 5 and 12 cm in a series of six patients. The minimum follow-up in this series was 2.4 years, with all patients showing satisfactory function, and averaging 94% plantarflexion strength compared with the uninjured side. No complications were noted in the series. Dacron vascular grafts have been used to augment Achilles tendon rupture[78,79] and have shown good or excellent results in acute rupture. Jennings and Sefton[15] used polyester tape with a Bunnel-type suture in 16 chronic ruptures. The tape was tensioned so the ankle could just dorsiflex to neutral. One patient required removal of the tape from around the calcaneum, one had a sural nerve injury, and three had superficial wound infections. No re-ruptures occurred.

Fascia Lata

Using fascia lata to repair and augment Achilles tendon rupture has produced good results.[11,29,80] Bugg and Boyd[23] reported 21 Achilles tendon ruptures or lacerations, 10 of which were chronic. They bridged the gap in the Achilles tendon with three strips of fascia lata, with a sheet of fascia lata sutured around these grafts in a tube-like fashion, with the serosal surface outward and the seam placed anteriorly and sutured to the proximal distal stumps. A wire pullout suture was also used. No formal results were given, but two case reports were provided stating that the technique has given satisfactory function and cosmetic results.

Allografts

This form of repair is not that commonly reported on in the literature, and as with synthetic materials, does not require a donor site. Nellas et al.[81] used two strips of freeze-dried Achilles tendon allograft to reconstruct a 4.5-cm tendon defect, following debridement of an infected primary repair. The patient had a good functional result, although had lower peak torque compared with the uninjured side. More recently, Haraguchi et al.[82] has performed Achilles tendon allografts for both chronic rupture and extensive tendinosis. The cortical bone in this procedure is removed from the patient's heel, allowing room for the allograft, which is secured in position with two 4.0-mm screws. The graft is then tensioned and repaired to the native Achilles tendon. No formal results have been published as yet for this series, but no rejection of allograft has been observed and neither has transmission of disease to the host occurred.

Conclusions

Chronic Achilles tendon ruptures are uncommon but potentially debilitating. The choice of management is partly guided by the size of the tendon defect with the optimal management being surgical. There are many different techniques that can be used to repair or reconstruct the rupture. Comparison of different techniques is difficult, due to the studies involved being retrospective and generally small. Every patient is different, and can present with varied co-morbidity, varied time of presentation, and different lengths of Achilles tendon retraction gap. Postoperative management varies from immediate mobilization, to up to three months in plaster. Functional outcome measures are also diverse, given the highly variable outcome criteria applied.

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19
Fascial Turn-Down Flap Repair of Chronic Achilles Tendon Rupture

S. Ghosh, P. Laing, and Nicola Maffulli

Introduction

Fascial turn-down flaps can be used for an anatomic repair of chronic Achilles tendon rupture. This technique allows one to strengthen the suture line and diminish the formation of adhesions between the sutured site and the skin.[1–5]

Historical Perspective

Christensen[1] and Gerhardt[2] separately described similar techniques. After suturing the tendon ends, they raised a distally based flap from the gastrocnemius aponeurosis and turned it over itself across the suture line and sutured it to the distal part of the Achilles tendon. Silfverskiold[6] twisted the gastrocnemius flap through 180 degrees before suturing it distally. This resulted in the smooth surface of the flap coming in contact with the skin, thereby decreasing the chance of adhesion between the flap itself and the overlying tissue. Toygar[7] described a technique for chronic ruptures, where it is difficult to regain continuity between the two ends. The gap is bridged by two flaps, raised from the two ends of the tendon, one from the medial side and the other from the lateral side. Weisbach[8] described another technique to address the same problem. Along with the gastrocnemius flap, he raised another flap from the distal stump of the Achilles tendon, and sutured these two flaps in order to bridge the gap.

In this chapter we describe two of the most commonly used techniques using the principle of turn-down flaps: first one that was described by Christensen in 1931 and Gerhardt in 1937, followed by the technique described by Lindholm in 1959.

Operative Technique (Central Flap)

The patient is placed in prone position. Anesthesia should ensure maximal muscle relaxation. Make a linear/curvilinear medial incision (to minimize the risk of injuring the sural nerve) from the midcalf to the calcaneus, taking care not to cross the midline in the distal part, in order to avoid scarring the tendon (Fig. 19.1). Incise the deep fascia in the midline after freeing it from the skin, thus making sure that the fascial incision lies fully under the skin flap. The site of the Achilles tendon rupture and the proximal gastrocnemius muscle are thus exposed (Fig. 19.2). Debride the tendon stumps as necessary, excising any fibrous tissue that may have formed in between the torn edges (Fig. 19.3). Then appose the refreshed ends with a box type of mattress suture, if possible, using heavy absorbable sutures. Place the foot in as much plantarflexion as required for proper apposition. Next, raise a flap approximately 2.0–2.5 cm broad and 7–8 cm long (depending on the gap to be bridged in case of neglected ruptures) from the middle of the proximal tendon and the gastrocnemius aponeurosis (Fig. 19.4), thus creating a central flap. Make sure that the flap is long enough to bridge the gap and it can be sutured securely to the distal tendon. Leave the flap attached for at

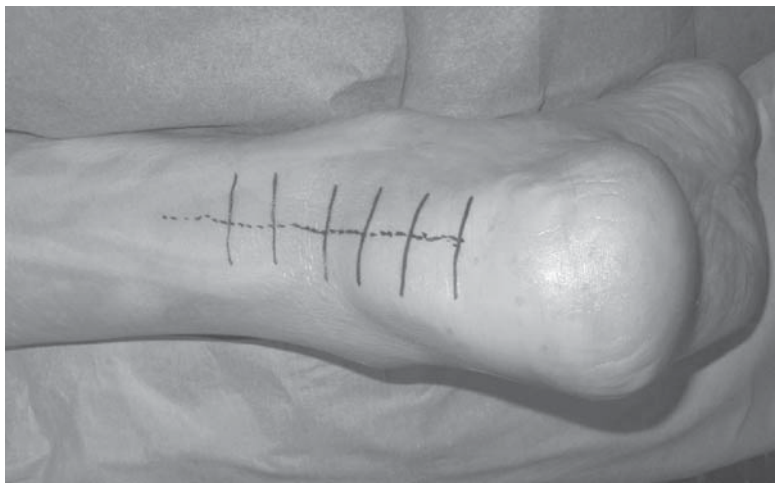

Figure 19.1 Medial skin incision.

least 3 cm proximal to the rupture. Next, turn the central flap upside down on itself and suture it to the distal stump with interrupted sutures (Fig. 19.5). Close the defect in the proximal tendon and gastrocnemius muscle belly with interrupted sutures. Close the tendon sheath and the deep fascia, followed by skin closure. Apply a plaster cast with the foot in gravity equinus.

Aftertreatment

At two weeks remove the cast, check the wound, and remove the sutures. Apply another short leg cast, with the foot in gravity equinus, for two weeks. After four weeks from surgery, bring the foot gradually to the plantigrade position over the next two weeks by serial changes of cast. The

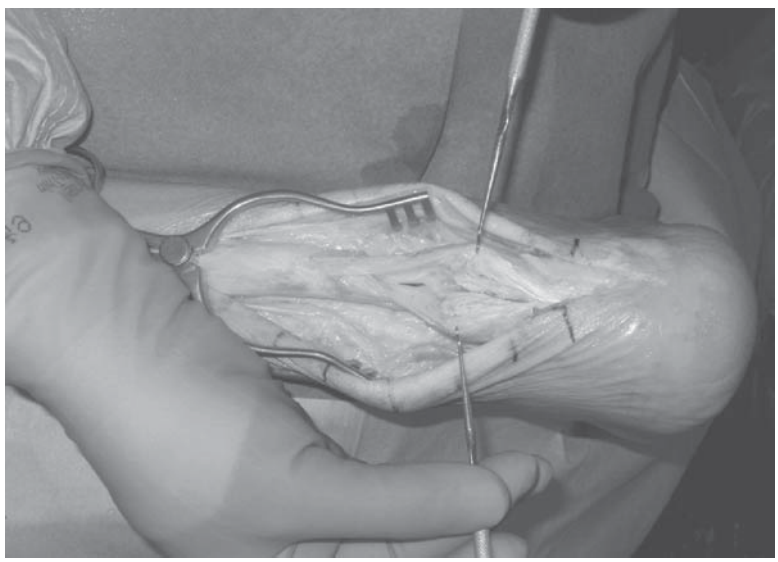

Figure 19.2 Tendon sheath exposed and fibrous tissue (which is bridging the defect) visualized.

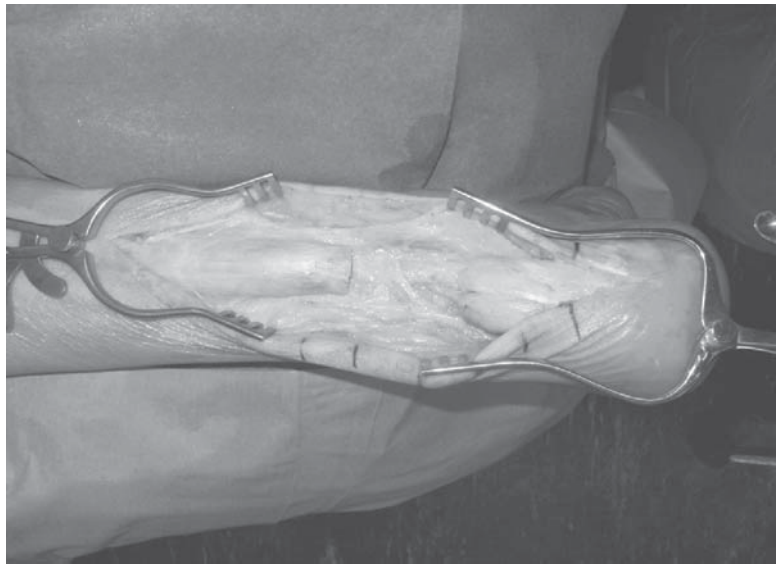

Figure 19.3 Refreshed tendon ends after adequate excision of fibrous tissue.

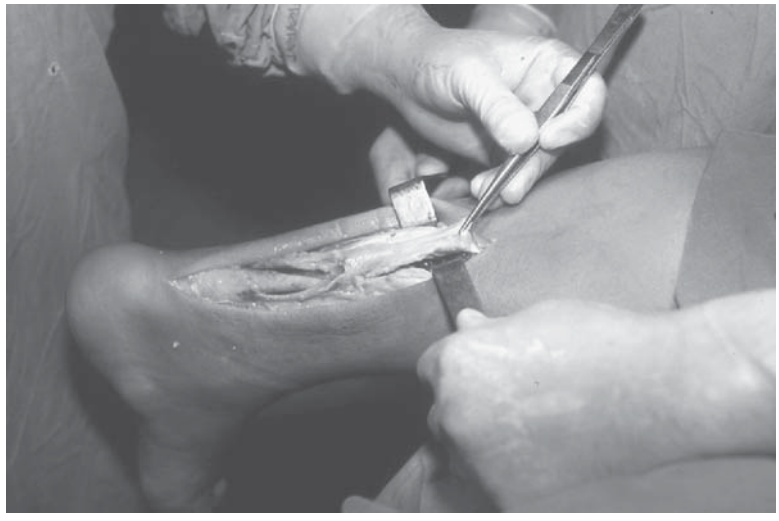

Figure 19.4 A central flap raised from the proximal tendon and gastrocnemius muscle.

Figure 19.5 The central flap is turned down on itself to bridge the gap.

patient can gradually resume walking with partial weight bearing on crutches during a two-week period. At six to eight weeks, apply a short leg walking cast with the foot in the plantigrade position, and allow full weight bearing. Alternatively, use a removable brace allowing only plantarflexion. Begin gentle active range-of-motion exercises and isometric ankle exercises. Toe raises, progressive resistance exercises, and proprioceptive exercises, in combination with a general strengthening program, constitute the third stage of rehabilitation. At 12 weeks, start using a reverse-90-degree ankle stop brace or similar device (if not already in use) and continue until a nearly full range of motion and strength 80% that of the opposite extremity has been obtained, usually within 6 months. In reliable, well-supervised patients with good tissue repair you may accelerate this program, with earlier use of dorsiflexion-stop orthoses and active range-of-motion exercises.

Discussion

This technique is easy and simple to carry out. However, there is a risk of adhesion as the raw inner surface of the flap comes in contact with the subcutaneous tissue. This can be overcome by the modification proposed by Silfverskiold and discussed earlier. Lindholm noted avascular necrosis of the turned-down flap, and proposed his technique to avoid a possible injury to the vascularity of the flap.

Operative Technique (Medial and Lateral Flaps)

The initial steps, until the tendon is exposed, are the same as described above.

The Achilles tendon rupture and lower part of the gastrocnemius muscle bellies are thus exposed (Fig. 19.3). Next, raise a flap approximately 1.0 cm broad and 7–8 cm long (depending on the gap to be bridged in chronic ruptures) from either side of the proximal tendon and the gastrocnemius aponeurosis about 0.5–1 cm from the midline (Fig. 19.6). Leave these flaps attached at a point 3–4 cm proximal to the tendon suture. Try to use the superficial layer of the aponeurosis only, though at times this is difficult. However, the detachment of the superficial layer is easier if you make the longitudinal incisions first, and then undermine the flap, before dividing it proximally. After creating the flaps, twist them 180 degrees backward on themselves so that the smooth external surface lies next to the subcutaneous surface as you turn it distally over the rupture. Close the

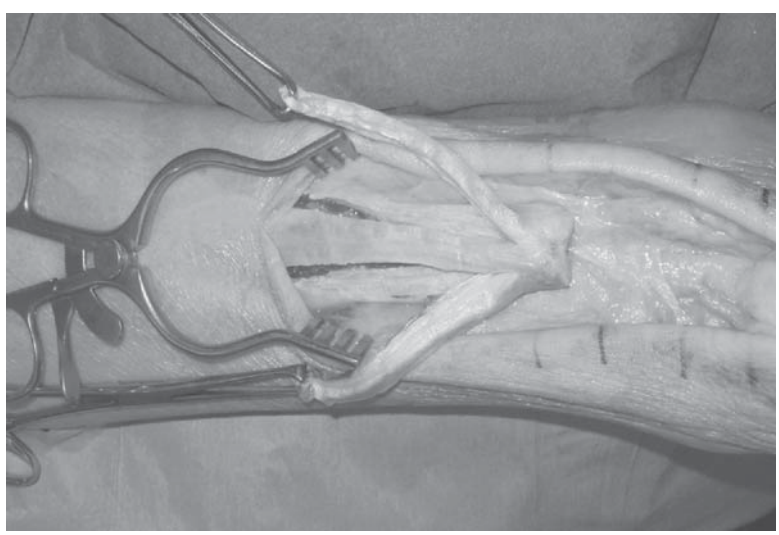

Figure 19.6 The two flaps are raised from the proximal end.

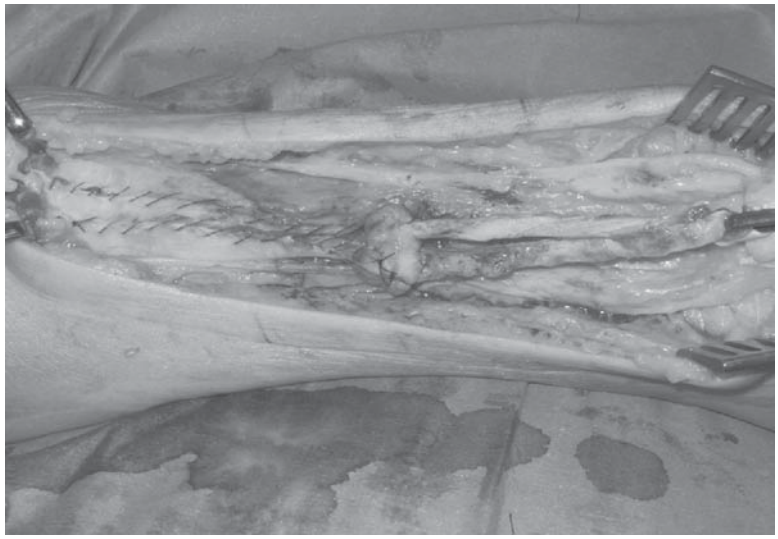

Figure 19.7 The flaps are turned down on themselves through 180° to reach the distal end and the proximal defects are sutured.

proximal defect in the gastrocnemius aponeurosis with interrupted sutures (Fig. 19.7). Suture the flaps to each other in the midline and to the distal stump of the tendon using interrupted sutures (Figs. 19.8 and 19. 9). Close the tendon sheath and the deep fascia carefully (Fig. 19.10). Appose the subcutaneous tissues together and close the skin wound. After suturing the stumps of the Achilles tendon, gradually reduce the plantarflexion of the foot during the rest of the operation, by careful, continuous pressure against the sole of the foot. This ensures the tendon is progressively stretched, so that at the end of surgery the foot can be placed with only 5–10 degrees of plantarflexion. Apply a plaster cast with the foot in gravity equinus.

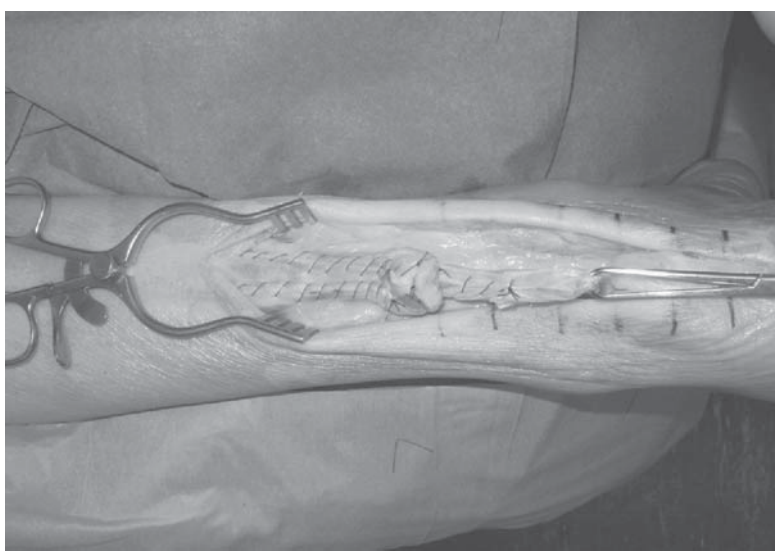

Figure 19.8 The flaps are sutured to each other.

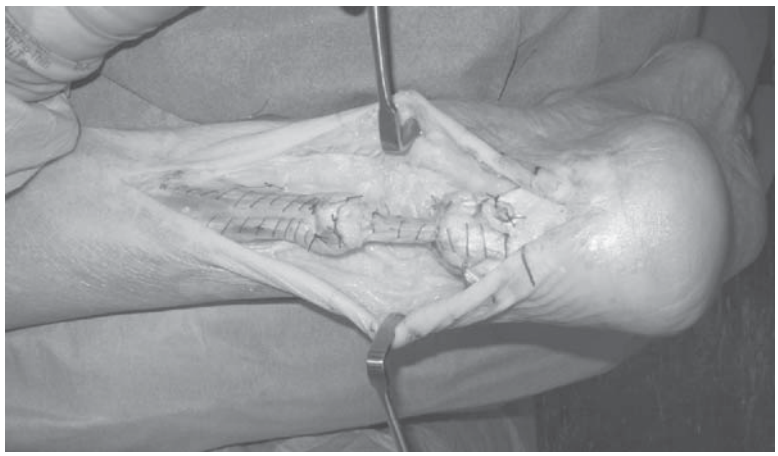

Figure 19.9 The combined flap is sutured to the distal stump.

Aftertreatment

This is the same as in the previous technique.

Discussion

Lindholm published this technique with results on 20 patients. He described better cosmetic results and lack of adhesion, while maintaining good functional outcome.[3] He described one patient with superficial infection of the wound, one with re-rupture, and one with a mild degree of skin fixity. Lindholm primarily described his technique only for acute ruptures and did not recommend its use in chronic ruptures, as he believed that "a free gliding surface forms against the subcutaneous tissue" in old ruptures and "plastic procedures in these cases greatly impede suture of the skin." However, we use it in cases of neglected ruptures only.[5] The rationale behind fashioning two flaps instead of one central flap, as described by Silfverskiold, was to preserve vascularity of the tendon, which is mostly centrally distributed, thus preventing necrosis of the flaps.[3]

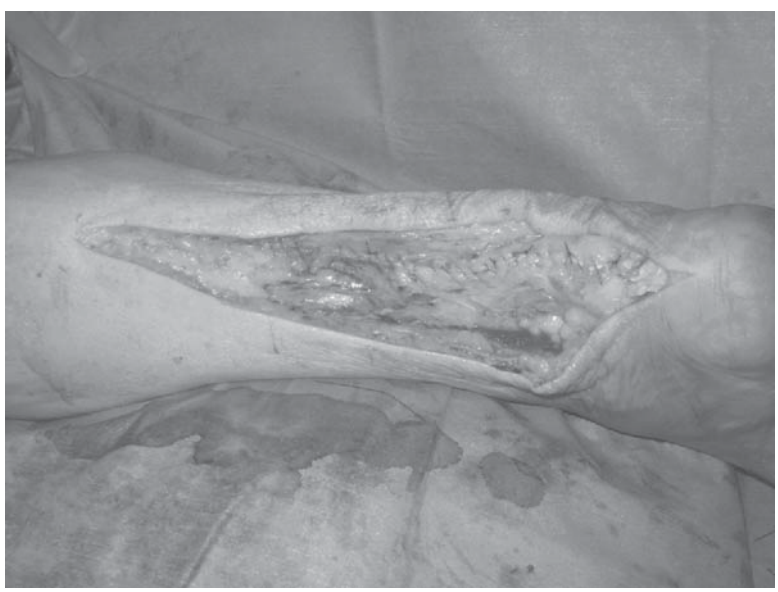

Figure 19.10 The paratenon is closed over the tendon.

20
Peroneus Brevis Tendon Transfer for Chronic Achilles Tendon Ruptures

Jonathan S. Young, Murali K. Sayana, D. McClelland, and Nicola Maffulli

Introduction/Historical Perspective

The Achilles tendon is the most commonly ruptured tendon in the human body.[1] Complete rupture of the Achilles tendon can be experienced both by sedentary patients and athletes,[1] and is especially common in middle-aged men who occasionally participate in sports.[2–5]

In acute ruptures, often a snapping sensation is felt in the posterior aspect of the ankle, with the patient then experiencing difficulty with weight bearing on the affected side. There is often a palpable gap between the ruptured tendon ends.[6] However, this may not be the case with chronic ruptures that present after a delay, which can prove more difficult to diagnose and manage.[6]

The management of chronic Achilles tendon ruptures is usually different from that of acute rupture, as the tendon ends have retracted. The blood supply to this area is poor, and the tendon ends have to be freshened to allow healing. Due to the increasing gap, primary repair is generally not possible as oppose to primary repair is generally possible.

Peroneus brevis tendon transfer for rupture of the Achilles tendon was popularized by Perez-Teuffer.[7] In the original technique, the harvested peroneus brevis tendon was passed through a transosseous drill hole in the calcaneus. Subsequently, Turco and Spinella[8] modified the technique by passing the peroneus brevis tendon through the distal stump of the Achilles tendon.

McClelland et al.[6] described approaching the Achilles tendon medially, and delivering the Achilles tendon through the posteromedial wound. The distally transsected peroneal tendon is gently pulled through the inferior peroneal retinaculum, thereby retaining the muscle's blood supply from the intermuscular septum. The peroneus brevis tendon is then woven through the ends of the ruptured Achilles tendon, passing through small coronal incisions in the distal stump, and then through similar incisions in the proximal stump. The tendon of plantaris, if present, can also be harvested to augment the repair if there is a large gap. This technique is described in detail below.

Indications and Contraindications

A delay in presentation of Achilles tendon rupture of greater than *8 weeks* results in filling up of the gap between the ruptured ends with fibrous nonfunctional scar that needs excision. If the gap with the ankle in maximum plantarflexion is between 5 and 9 cm, peroneus brevis transfer can be utilized.

We do not use this technique if the gap between the ruptured ends of the Achilles tendon is greater than 8 cm, or the ipsilateral peroneus brevis has been used for other reconstructive procedures around the ankle (for example, for reconstruction in lateral instability).

Preoperative Assessment

The diagnosis of chronic, delayed rupture can be difficult.[6,9] Scar tissue may have replaced the gap between the proximal and distal ends of the Achilles tendon, thereby obscuring the gap

typically palpable in acute ruptures. Moreover, pain and swelling associated with acute ruptures may be absent.

Clinically, the Simmonds[10] and Matles[11] tests may assist in making the diagnosis in delayed Achilles tendon rupture. Occasionally, even these tests may be of dubious interpretation, and imaging may need to be considered.[12–14] Ultrasonography of a delayed rupture typically demonstrates an acoustic vacuum with thick irregular edges.[12,13] Magnetic resonance imaging shows generalized high signal intensity on T2 weighted images. On T1 weighted images, the rupture will appear as a disruption of the signal within the tendon substance.[14]

Clinical examination is performed, encompassing the abovementioned clinical tests, fitness for anesthesia, and neurovascular status of the limb, paying particular attention to the sural nerve. Once the diagnosis is confirmed, the patient is counseled regarding the technique and risks involved and informed consent is obtained. At our institution, general anesthesia is preferred for this procedure.

Operative Technique

The patient is placed prone with feet protruding over the edge of the operating table. Both legs are prepped and draped, allowing intraoperative comparison to adjust the tension of the reconstruction. If considered necessary, a tourniquet is applied to the thigh of the affected leg, the leg exsanguinated, and the tourniquet inflated to 250 mmHg. A 10- to 12-cm longitudinal skin incision is made just medial to the medial border of the Achilles tendon, and sharp dissection is carried out through the subcutaneous fat layer (Fig. 20.1). The Achilles tendon is exposed using a longitudinal incision of the paratenon in the midline for the length of the skin incision. Scar tissue is debrided from the ends of the Achilles tendon, which are freshened by sharp dissection, defining the defect between the freshened ends. The proximal and distal stumps are gently dissected out and mobilized.

Through the base of the wound, the deep fascia overlying the deep flexor compartment and the lateral compartment containing the peronei muscles can be seen. The internervous plane lies between the peroneus brevis (supplied by the superficial peroneal nerve) and the flexor hallucis longus (supplied by the tibial nerve). The muscle belly of the peroneus brevis passes from the midline medially and under the tendon of the peroneus longus to lie anterior to it and adjacent to the posterior aspect of the lateral malleolus. The tendons of the peroneus longus and brevis can be distinguished from each other at this level as the peroneus brevis muscle extends more distally than that of the peroneus longus. The deep

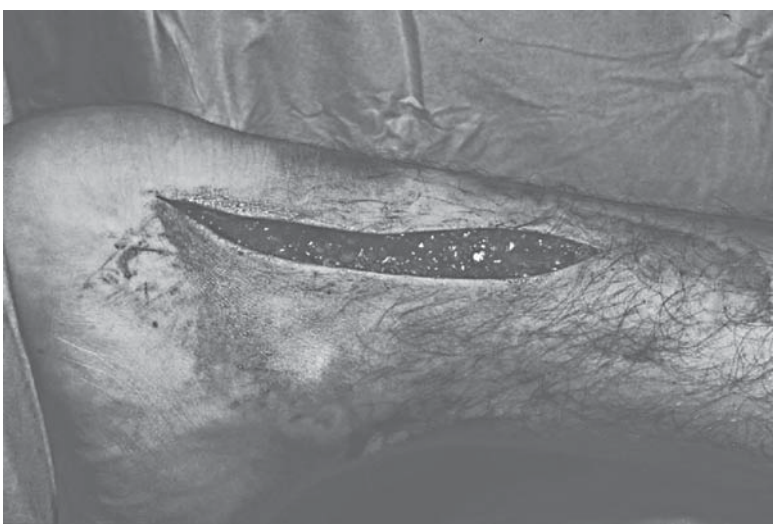

Figure 20.1 Incision over the medial edge of the Achilles tendon.

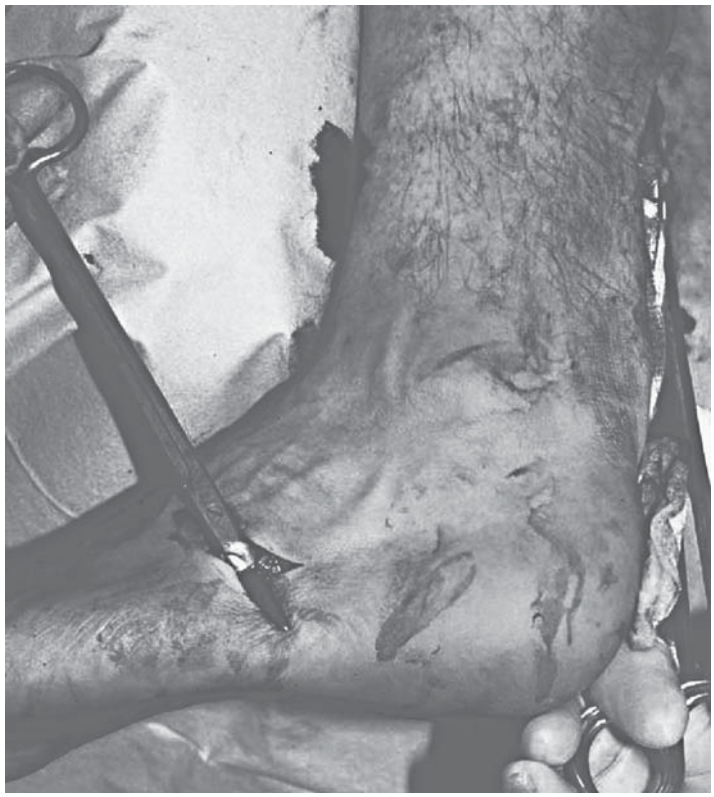

Figure 20.2 Incision over the base fifth metatarsal enabling identification of the distal end of the peroneus brevis tendon.

fascia overlying the peroneal tendons is incised, and the peroneal tendons are mobilized.

The peroneus brevis passes around the posterior aspect of the lateral malleolus and above the peroneal trochlea to insert onto the styloid process of the base of the fifth metatarsal. Both peroneal tendons are tethered both at the lateral malleolus and the peroneal trochlea by the superior peroneal retinaculum and the inferior peroneal retinaculum, respectively. A 2.5-cm longitudinal incision is made over the base of the fifth metatarsal (Fig. 20.2). The peroneus brevis tendon is identified, and a stay suture is placed in the distal end of the peroneus brevis tendon, which is then detached from its insertion and mobilized proximally. The tendon is then delivered through the posteromedial wound using gentle continuous traction as it is pulled through the inferior peroneal retinaculum (Fig. 20.3). In this fashion, the tendon of the peroneus brevis retains its blood supply from the intermuscular septum.

The peroneus brevis tendon is woven through the Achilles tendon ends. It is first passed from lateral to medial through the distal Achilles tendon stump via coronal incisions medially and laterally in the Achilles tendon (Fig. 20.4). The edges of the coronal incisions in the Achilles tendon are sutured to the peroneus brevis tendon to prevent the transferred peroneus brevis tendon from pulling out of the Achilles. The tendon is then passed through the proximal stump from medial to lateral with the ankle plantarflexed to achieve the correct tension relative to the uninvolved extremity. A comparison is made with the contralateral limb to confirm tension is equal. The peroneal tendon is sutured to the Achilles tendon stumps using 3/0 Vicryl (Ethicon, Edinburgh, UK, EH11 4HE) (Fig. 20.5). This is usually sufficient, but, if there is a large defect, the plantaris tendon may be utilized to reinforce the reconstruction (Fig. 20.6).

Hemostasis is achieved. In most patients with delayed ruptures of the Achilles tendon, the

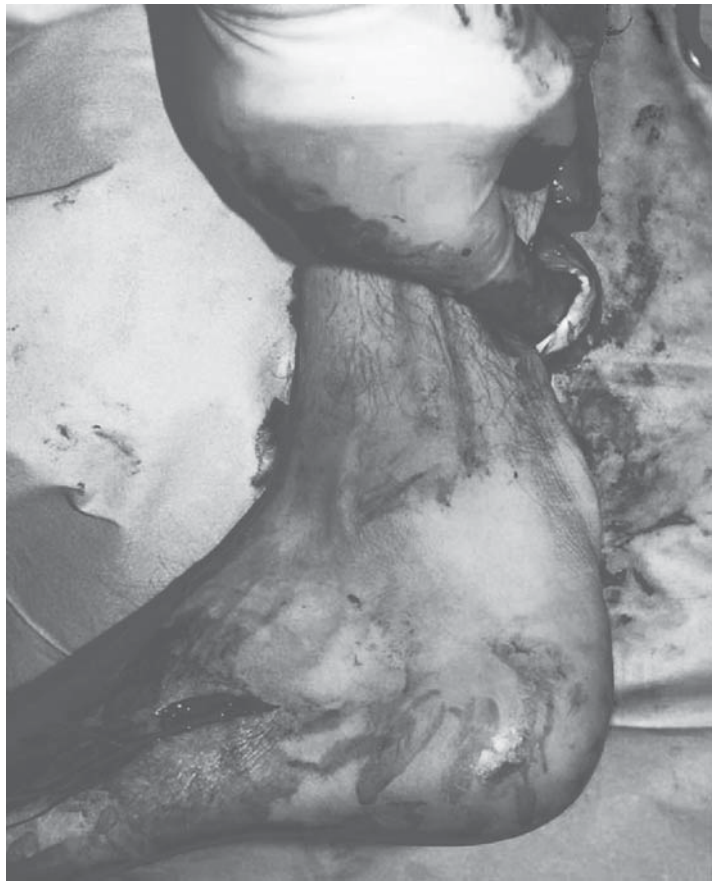

Figure 20.3 Peroneus brevis tendon being delivered into the posteromedial wound with gentle traction.

Figure 20.4 Passing peroneus brevis tendon through the ends of the Achilles tendon.

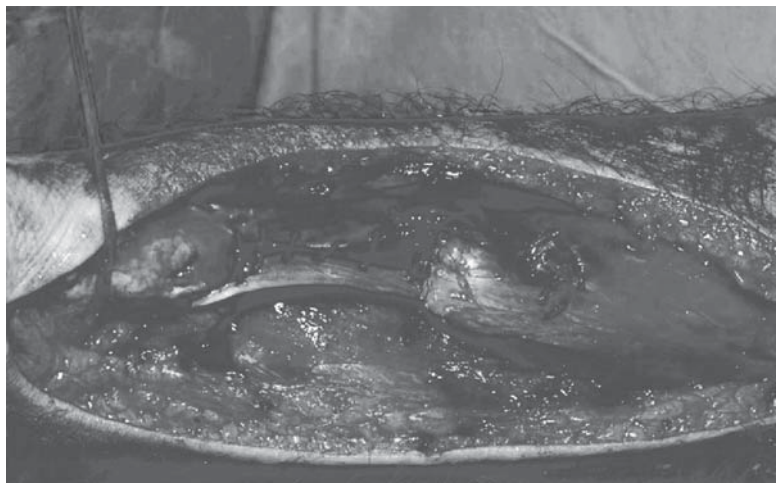

Figure 20.5 End result of sutured peroneus brevis graft in situ.

paratenon is either not present or not viable. If present, one can generally manage to close it over the proximal stump using 2/0 Vicryl. Reapproximation of the paratenon over the distal portion of the reconstruction is often not possible. The skin is closed with a continuous 2/0 subcuticular Vicryl suture. Steristrips (3M Healthcare St Paul, MN, USA) are applied, and the wound is dressed.

Postoperative Management

Elevation of the operated limb on a Braun frame is recommended overnight, and the patient should have regular neurovascular observations. Following review by a physiotherapist, the patient is generally discharged the day following surgery. A full below-knee cast is applied with the ankle in physiological equinus and retained for two weeks until review in an outpatient clinic. Patients are advised to keep the operated limb elevated as much as possible to minimize swelling. They are allowed to weight bear on the operated leg as tolerated, for the first two weeks until seen in the clinic, when the cast is split and the wounds are inspected. A synthetic anterior below-knee slab is fitted with the ankle in physiological equinus. The slab is secured to the leg with three or four removable Velcro (Velcro USA Inc., Manchester, NH, USA) straps for four weeks. At this stage, the patients

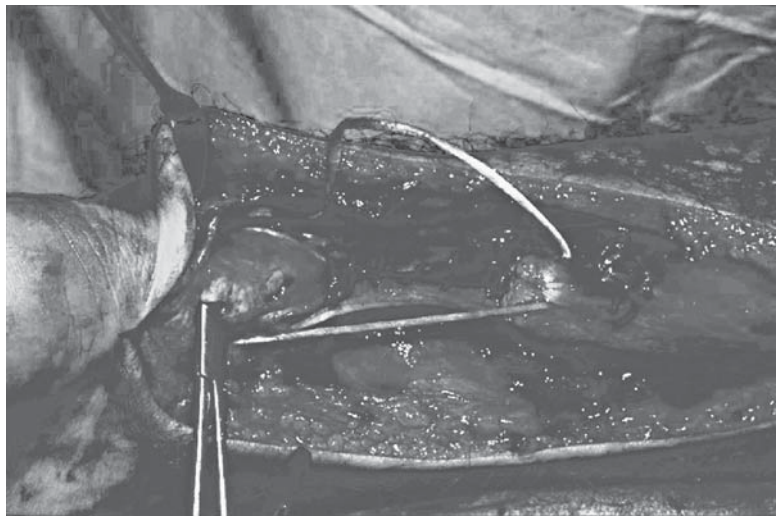

Figure 20.6 Plantaris tendon used to augment the reconstruction.

are encouraged to weight bear on the operated limb as tolerated, gradually progressing to full weight bearing. The patients are seen by a trained physiotherapist and taught to perform gentle mobilization exercises of the ankle, isometric contraction of the gastrocsoleus complex, and gentle concentric contraction of the calf muscles. Patients are encouraged to perform mobilization of the involved ankle several times per day after unstrapping the two most distal Velcro straps.[15] Patients are given an appointment six weeks from the operation, when the anterior slab is removed and the wound is again inspected.

Once the cast is removed, patients are referred for more intensive physiotherapy. They are allowed to begin gentle exercise such as swimming and cycling at eight weeks following surgery but are restricted from running for an additional four to six weeks and limited from return to sports until four to five months from time of surgery.

Results

We have treated 22 Achilles tendon ruptures with delayed presentation using this technique.[16] All of these patients were satisfied with the procedure. Despite subjective patient satisfaction, objective evaluation demonstrated greater loss of isokinetic strength variables at high speeds and greater loss of calf circumference when compared with patients undergoing open repair of fresh Achilles tendon ruptures.

Complications

Damage to the sural nerve, wound complications, and re-rupture are all risks with surgical reconstruction of chronic rupture of the Achilles tendon. The medial longitudinal incision helps avoid damage to the sural nerve.[6]

Longitudinal incisions used in open repair of the Achilles tendon are typically performed through poorly vascularized skin,[17] introducing the potential for poor wound healing. This risk can be reduced by careful handling of tissues and by maintaining thick skin flaps throughout the procedure.[6] Even minor areas of wound dehiscence may take a long time to heal. Wounds that break down need coverage, as tendons left exposed undergo desiccation and secondary adhesions.[18] Occasionally, local or free flap coverage may be required. Local flap coverage can be in the form of medial plantar flap, posterior tibial reverse flow flap, or peroneal reverse flow island flap, depending on the site of the defect.[19] This is advantageous in restricting the morbidity of the leg originally operated on, and, if a local flap fails, a free flap can still be considered.[18] Wound complications over the Achilles tendon warrant aggressive, early management.

Re-ruptures are rare: Perez-Teuffer[7] and Pintore et al.[16] reported no re-ruptures in their series of peroneal reconstructions of chronic Achilles ruptures. Patients must be cautioned about the risk of re-rupture and should allow the reconstructed tendon adequate time to heal in the duration of postoperative recovery.

Possible Concerns/Future of the Technique

This technique generally provides a good functional outcome, with minimal complications as long as good postoperative care is administered. The postoperative management in these patients is the same as that we have recently described following open repair of acute Achilles tendon ruptures.[20] Accelerated rehabilitation programs have been advocated,[21] but the priority in these challenging patients is to restore adequate muscle-tendon unit function without compromising skin healing.[22]

As the peroneus brevis is utilized to reconstruct the Achilles tendon, the peroneus longus becomes the sole evertor of the foot, and continues to maintain the transverse arch. Gallant et al. assessed eversion and plantarflexion strength after repair of Achilles tendon rupture using peroneus brevis tendon transfer and found mild objective eversion and plantarflexion weakness. However, subjective assessment revealed no functional compromise.[23] We have not come across any hindfoot varus deformities in our patients following this reconstructive procedures. However, in the long term, hindfoot varus is a theoretical (though unencountered) possibility.

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21
Free Gracilis Tendon Transfer for Chronic Rupture of the Achilles Tendon

Jonathan S. Young, Wayne B. Leadbetter, and Nicola Maffulli

Introduction

Chronic ruptures of the Achilles tendon are associated with both functional and operative morbidity.[1] Various methods are described to manage this condition.[1–4] The tendon of gracilis has recently been used to reconstruct the Achilles tendon.[5,6]

Diagnosis

Although diagnosis is straightforward for experienced surgeons,[7] and most Achilles tendon ruptures are promptly diagnosed,[1,7,8] first examining physicians may miss up to 20% of such injuries.[9] The diagnosis of chronic rupture can be more difficult,[1,10] as fibrous scar tissue may have replaced the gap between the proximal and distal ends of the Achilles tendon, and therefore the gap palpable in acute ruptures is no longer present. There may also be less pain and swelling.

Clinically, the Simmonds[11] and Matles[12] tests help aid the diagnosis in both acute and delayed rupture, but even these tests may be of dubious interpretation, and imaging may have to be used.[8,13,14] Ultrasonography of a neglected rupture will reveal an acoustic vacuum with thick irregular edges.[8,13] Magnetic resonance imaging will reveal generalized high signal intensity on T2 weighted images. On T1 weighted images, the rupture will appear as a disruption of the signal within the tendon substance.[14]

Management

Management of a chronic Achilles tendon rupture is more difficult than acute rupture. Generally, an open procedure must be carried out.[1,8] Surgery requires the tendon edges to be freshened, and, as they will be retracted, a large gap will thus be produced. Various techniques have been described to bridge the gap.

A strip of the superficial part of the tendinous portion of the proximal stump of the Achilles tendon has been used.[15] A proximal-to-distal V-Y advancement of the gastrocnemius tendon has also been described.[3]

Mann et al.[2] described the use of a flexor digitorum longus (FDL) graft in seven patients. Six of the seven patients had an excellent result, and one a fair result. There were no re-ruptures at an average follow-up of 39 months.

More recently, the tendon of flexor hallucis longus (FHL) has been used. FHL has a long tendon that allows bridging of large Achilles tendon defects.[4] Wapner et al.[4] reported 7 patients managed with this technique. The tendon of FHL was woven through the ruptured Achilles tendon ends. The distal end of FHL was tenodesed to the tendon of FDL of the second toe. Three patients had an excellent result, three a good result, and one a fair result. Each patient developed a "small but functionally insignificant loss in range of motion in the involved ankle and great toe." This may be important in athletic individuals, in whom the loss of push-off from the hallux may cause difficulty when sprinting.

Fascia lata grafts,[16] plantaris,[17] and synthetic materials[18] have also been used in the management of neglected ruptures of the Achilles tendon.

Perez-Teuffer[19] popularized the use of the peroneus brevis. Turco and Spinella[20] used a similar technique, but passed the peroneus brevis tendon through the distal stump of the Achilles tendon. McClelland and Maffulli[1] used a similar technique, but they approached the Achilles tendon through a curvilinear medial incision, thus minimizing the risk of sural nerve injury.

More recently, we used the tendon of gracilis as a free graft to bridge the gap in chronic ruptures.[5,6] We report the details of this technique.

Preoperative Assessment

The patient is assessed, a full history is collected, clinical examination is carried out, and fitness for anesthesia and the neurovascular status of the limb are assessed, paying particular attention to the sural nerve. The diagnosis of chronic rupture may be difficult and require further imaging. Written informed consent is taken. The patient should be aware of wound problems, neurovascular damage, altered sensation around the gracilis harvest site, calf wasting, weakness of ankle flexion, and the risk of failure of surgery and of anesthesia.

Operative Technique

With the patient prone and both feet dangling from the end of the operating table, the affected leg and ankle is prepped and draped. A single dose of a first-generation cephalosporin is administered at induction of anesthesia. The limb is exsanguinated and a thigh tourniquet is inflated to 250 mmHg. A 12- to 15-cm longitudinal, slightly curvilinear skin incision is made medial and anterior to the medial border of the tendon. The paratenon, if not disrupted, is incised longitudinally in the midline for the length of the skin incision. The Achilles tendon is thus exposed. Gentle continuous traction is applied so that the proximal stump of the ruptured tendon is further delivered into the wound, allowing the lowest possible residual gap. Scar tissue in both the proximal and distal stumps is excised to reach viable tendon.

If the gap produced is greater than 6 cm despite maximal plantarflexion of the ankle and traction on the Achilles tendon stumps, we proceed to harvest the tendon of gracilis. A vertical 2.5- to 3-cm longitudinal incision is made over the tibial tuberosity, and should be centred over the distal insertion of the pes anserinus (where the gracilis tendon inserts). There is a constant venous plexus lying at the distal end of the wound, and care should be taken to diathermy this. Using a small swab attached to an artery clip, dissection deep to the fat is carried out both medially and superiorly. A curved retractor is inserted, and a curved incision, 1 cm in length, is made along the superior margin of the pes anserinus into the sartorious fascia. Care is taken to avoid damage to the saphenous nerve. Through this incision, Mackenrodt scissors are introduced and opened so as to split and produce a window within the superior border of the sartorious, allowing for access to the tendon of gracilis.

The gracilis tendon lies more superiorly than the neighboring tendon of semitendinosus. It can be retrieved with the aid of a curved Moynihan clip (Fig. 21.1). The tendon is brought into the wound and distal traction on the tendon is imposed. An open-ended tendon stripper is used to harvest the tendon (Fig. 21.2).[21]

Once the tendon is freed of fat and muscle fibers (Fig. 21.3), it is passed through a small transverse incision produced by a number 11 scalpel blade in the substance of the distal stump of the Achilles tendon in a medial-to-lateral direction. The gracilis tendon is then pulled proximally and through a small incision in the substance of the proximal stump of the Achilles tendon in a lateral-to-medial direction through the proximal stump (Fig. 21.4). The gracilis tendon is sutured to the Achilles tendon at each entry and exit point using 3-0 Vicryl (Polyglactin 910 braided absorbable suture, Johnson & Johnson, European Logistics Centre, 66 Rue de la Fusee, B-1130 Bruxelles, Belgium). The repair is tensioned to greater than the physiological equinus present in the opposite ankle. When present, the tendon of plantaris can be harvested with the tendon stripper, left attached distally (Fig. 21.5), and used to reinforce the reconstruction (Fig. 21.6).

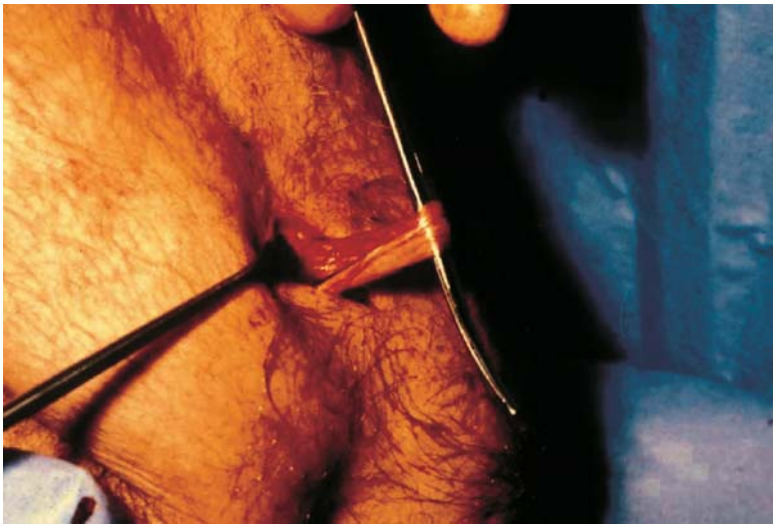

Figure 21.1 Gracilis tendon prior to removal from pes anserinus.

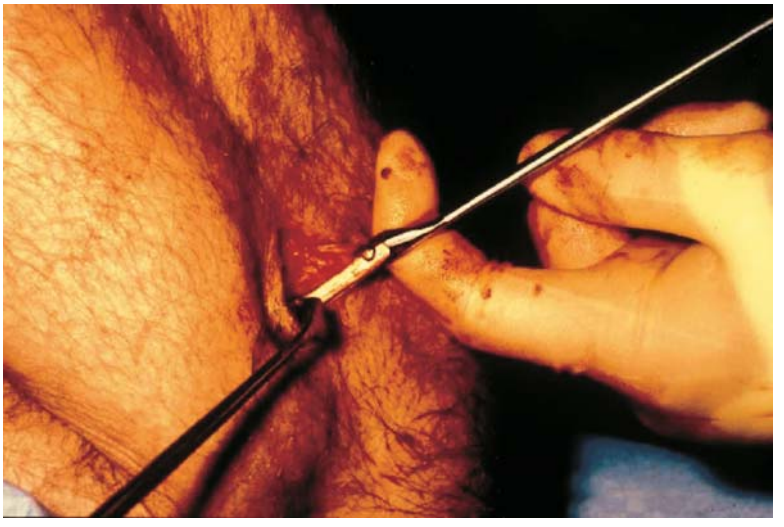

Figure 21.2 Extraction of gracilis with tendon stripper.

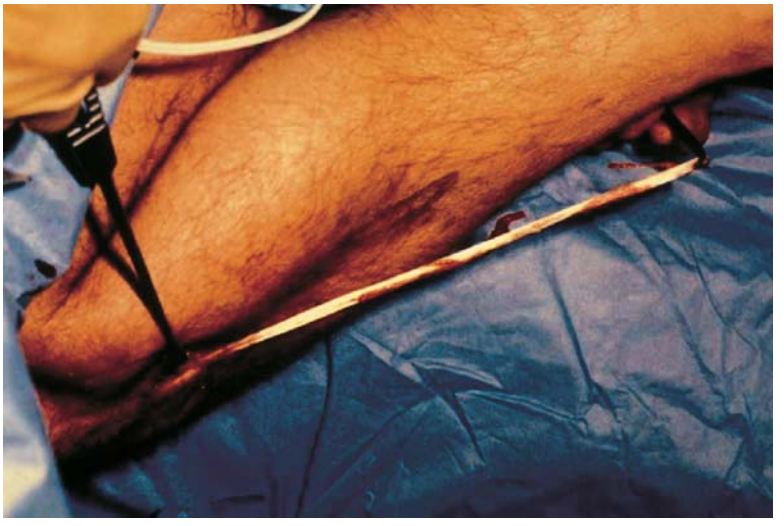

Figure 21.3 Free gracilis tendon.

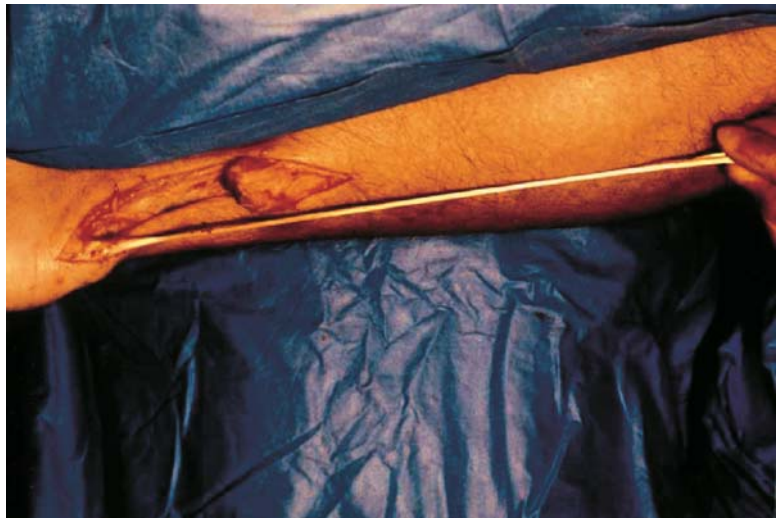

Figure 21.4 Plantaris tendon.

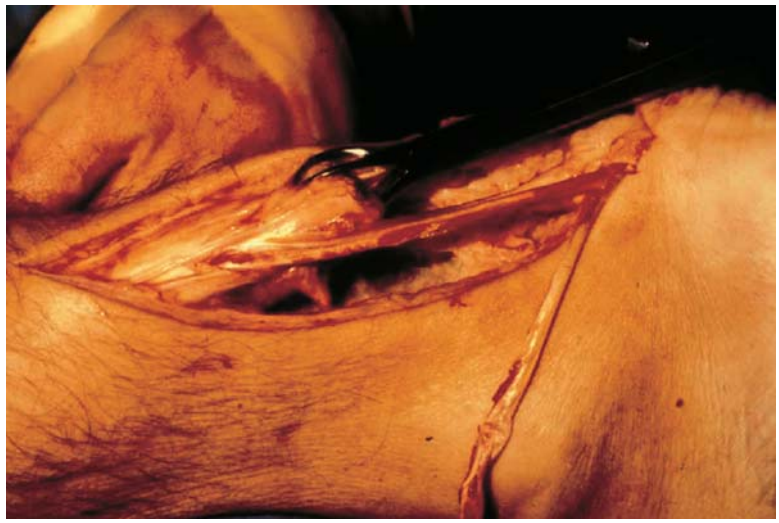

Figure 21.5 Weaving of gracilis tendon through the Achilles tendon stumps.

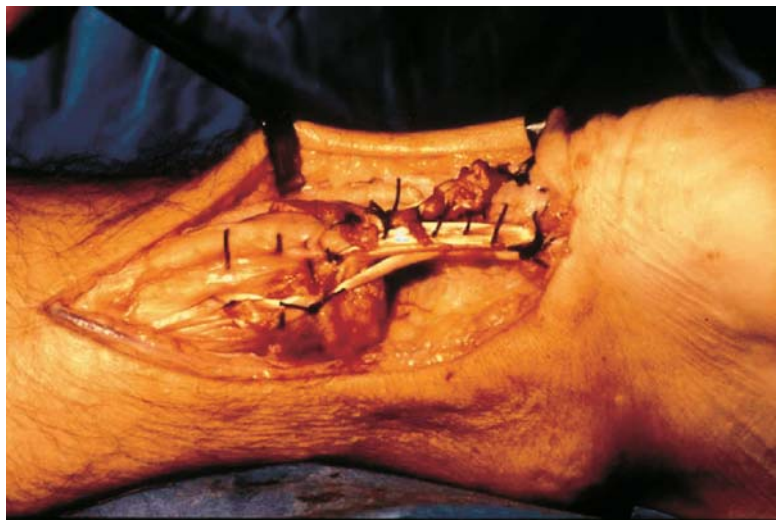

Figure 21.6 Repaired Achilles tendon using gracilis and plantaris.

Interrupted 4-0 Vicryl reabsorbable sutures are used for the subcutaneous fat, and the skin is closed with interrupted 4.0 Ethilon (Ethicon, Johnson & Johnson, European Logistics Centre, 66 Rue de la Fusee, B-1130 Bruxelles, Belgium), or with subcuticular 3-0 Vicryl. The tourniquet is deflated, the wound is dressed, and a below-knee plaster-of-Paris cast is applied with the patient prone.

Postoperative Care

Postoperatively, the injured leg is elevated until discharge. Patients are discharged the day after surgery, after having been taught to use crutches by an orthopedic physiotherapist.[8] Thromboprophylaxis is provided with Fragmin, 2,500 units (Deltaparin Sodium, Pharmacia and Upjohn, Roma, Italy) subcutaneously once daily, or with 150 mg of acetylsalicylic acid orally daily, until removal of the cast. When the cast has dried, patients are encouraged to mobilize with the use of crutches, under the direction of a physiotherapist. Patients are allowed to bear weight on the operated leg as tolerated, but are told to keep the operated leg elevated as much as possible for the first two postoperative weeks.[22]

The cast is removed two weeks after the operation, and a synthetic anterior below-knee slab is applied, with the foot in gravity equinus.[1] The synthetic slab is secured to the leg with three or four removable Velcro (Velcro USA Inc., Manchester, NH, USA) straps for four weeks. Patients are encouraged to weight bear on the operated limb as soon as comfortable, and to gradually progress to full weight bearing. The patients are seen by a trained physiotherapist, who teaches them to perform gentle mobilization exercises of the ankle, isometric contraction of the gastrocsoleus complex, and gentle concentric contraction of the calf muscles. Patients are encouraged to perform mobilization of the involved ankle several times per day after unstrapping the two most distal Velcro straps. Patients are given an appointment six weeks from the operation, when the anterior slab is removed.

Patients mobilize the ankle with physiotherapy guidance. They are allowed to weight bear as able, and perform gradual stretching and strengthening exercises.[22] Cycling and swimming are started eight weeks after surgery if the wound is healthy. Patients are prompted to increase the frequency of their self-administered exercise program, and are allowed to return to their sports in the fifth postoperative month.

Complications

Wound infection, breakdown, and scar problems are a documented risk in open repairs of the Achilles tendon,[1,8] given the tenuous blood supply in this area.[23] There is also the theoretical risk of infection and wound breakdown to the donor gracilis tendon site. In our series,[5] five patients had a superficial infection of the Achilles tendon surgical wound. They were managed conservatively with oral antibiotics following a microbiology swab to ascertain sensitivity, were asked to keep the leg elevated at all times, and healed uneventfully by the 18th postoperative week. At the sixth postoperative month, two patients complained of hypersensitivity of the surgical wounds. They were counseled to rub hand cream over the wounds several times a day, and all were asymptomatic by the next visit. One patient developed a hypertrophic scar in the area of the Achilles tendon surgical wound as it rubbed against the shoe, and was not pleased with the appearance of the operative scar. Other early complications include wound hematoma and sural nerve sensory deficit from intraoperative injury. Medial positioning of the incision helps to reduce sural nerve injury.[1] Re-rupture is one of the most important late complications.[1,19] Deep vein thrombosis is also a documented risk. Arner and Lindholm[24] reported two DVTs in 86 patients following open repair of the Achilles tendon. No patients in our series[5] sustained a re-rupture or developed a DVT. Also, functionally all patients were able to walk on tiptoes, and no patient used a heel lift or walked with a visible limp.

Results

Twenty-one patients were managed with this technique. The delay in presentation varied from 2 to 9 months following the rupture.[5] The outcome

of surgical management was rated using a four-point scale.[25] Most patients were satisfied with the procedure; only two were classified as having an excellent result, although 15 of our 21 patients achieved a good result.

The maximum calf circumference was significantly decreased in the operated leg both at presentation and at latest follow-up.

Patients were able to perform at least 10 single-leg heel lifts on the affected leg by discharge, and four patients were able to perform at least 60 single-leg heel lifts on the affected leg. All patients had returned to their pre-injury working occupation. Of the 21 patients included in this study, 15 had returned to their leisure activities. Of these 15, five of the seven patients who played tennis returned to playing doubles. Three patients who played squash were able to return to training, but did not plan to return to competition. Four patients returned to bowling, and the remaining three returned to golf, although not with the same frequency as before the injury. Of the whole group of 21 patients, six were sedentary and only walked their dogs and performed gardening. They reported no problems in these activities.

The operated limb showed a lower peak torque than the nonoperated one, but the patients did not perceive this decrease in strength as hampering their daily or leisure activities.

Conclusions

The management of chronic subcutaneous tears of the Achilles tendon by free gracilis tendon grafting is safe but technically demanding. It affords good recovery, even in patients with a chronic rupture of two to nine months' duration. Such patients should be warned that they are at risk of postoperative complications, that the wasting of their calf is not likely to recover, and that their ankle plantarflexion strength can remain reduced.

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22
Chronic Ruptures of the Achilles Tendon: Management with Synthetic Material

Graham Keith Sefton

Introduction

Chronic rupture of the tendon Achilles can be a very debilitating injury that may be difficult or even impossible to treat adequately. A rupture is classified as being chronic if it has been present for longer than approximately four weeks. Some patients can and do cope with the functional problems that arise in relation to a chronic rupture of the Achilles tendon. They refrain from sports, they know that they cannot run (even to catch a bus), and they feel as if they have no spring in their step. However, some patients find that the resulting disability from a chronic rupture is so great that they seek management. Examination reveals inability to stand on tiptoes on the injured side, an increased range of dorsiflexion at the ankle, wasting and weakness of the calf muscles, palpable gaps or thickening (which can be 6–8 cm in length) at the site of the rupture, and a positive calf squeeze test. Failed conservative or surgical management after acute Achilles tendon rupture presents a similar technical problem to the treating orthopedic surgeon.

Various techniques have been described to address this problem; most of these involve the use of autogenous material as an augment. This obviously is associated with donor-site morbidity. Most of the procedures described are also technically demanding.[1–9]

We use a polyester tape to repair the chronic rupture of the Achilles tendon,[10] a technique similar to that reported by Jennings et al.[11] in the management of acute injuries to the Achilles tendon. An open-weave polyester (ethylene terephthelate) tape (CTP 10 × 800 mm) is employed as a biological scaffold to aid repair of the tendon.

Polyester has been widely used for many types of permanent or temporary implants, including sutures, vascular grafts, intra-orbit eye implants, hernia repair patches, and over the past two decades, for ligament reconstruction and tendon repairs. It has a long history of safety in human surgical applications.

Postmarket surveillance over nearly 20 years and about 97,000 implantations of the Leeds-Keio polyester mesh material (55,000 ligaments and 42,000 tapes) has not produced any clear evidence of patient adverse effects that have been linked to a biological reaction to the material in clinical use. The incidence of adverse reactions is apparently very low and similar to that associated with autogenous tissue repairs. Adverse tissue reactions have sometimes been reported in the literature where artificially generated wear particles are introduced into the intra-articular space of *test animals*, but we are not aware of any such reports arising from the use of the polyester used for ligament or tendon repair. Various commonly used biological safety tests were used to demonstrate the safety of these polyester implants, using tests such as those suggested in ISO 10993-1, *Biological Evaluation of Medical Devices Part 1: Evaluation and Testing*.[12]

The implant has an open-weave structure that affords space for tissue ingrowth. This tissue is expected to remodel and mature into a tendon-like tissue and thereby augment the strength of the implant in isolation. The tape in current use has a tensile strength of 850 N, which is much

greater than the strength of sutures used in other surgical procedures for approximating the disrupted TA. The reconstruction with this tape therefore allows a much faster rehabilitation regimen for the patient. Furthermore, the usefulness of this strong implant becomes even more manifest when used for reconstruction of chronic rupture cases, where a deficiency in tissue exists, and approximating the disrupted TA with sutures is ineffective if not impossible.

With regard to applications, this implant is more effective in extra- than in intra-articular applications such as anterior or posterior cruciate ligament reconstruction. The problems encountered with these, however, were largely technique-related, and consequent to abrasions from intense early rehabilitation regimens rather than to implant design or material. The tape-type implants have proven suitable and effective in reconstructions of the patellar tendon, medial and lateral collateral ligaments, the quadriceps extensor mechanism, the ankle anterior talo-fibular ligament, and in the reconstruction of the Achilles tendon.

There are some important points to consider before performing this type of surgery:

- *Maintain the continuity of the Achilles tendon/scar/Achilles tendon axis.* It is very tempting to excise the scar tissue and freshen the ends of the torn tendon. This is counterproductive. Excision of the scar tissue and some of the tendon makes the gap much wider. While the polyester tape does act as a scaffold for the ingrowth of collagen, the smaller the gap the sooner healing occurs and the stronger the stability of the repair.
- *Make use of the scar tissue.* In most instances, once the polyester tape has been passed and tensioned, the scar tissue can be sutured to the tendon proximally and distally, and it can be brought around to cover the knot in the polyester tape. Hence, at the end of the procedure, the implant is not exposed. The surface of the repair appears smooth, and it glides easily within the tendon sheath.
- *Meticulous surgical technique is a prerequisite of this type of surgery.* Every effort should be made to minimize the amount of damage the surgeon causes to the surrounding tissues, the tendon sheath, and the skin. Even the presence of a hematoma or a small amount of necrotic tissue can have serious adverse effects on outcome. Delay in healing results in adhesions, pain, swelling, and limitation of movement.
- *Minimally invasive surgery.* While it is possible to repair an acute rupture of the Achilles tendon using a percutaneous approach,[11] this is not possible when trying to repair a chronic Achilles tendon rupture. However, compared to the very long extensive incisions used previously in, for instance, the gastrocnemius fascial flap,[1] the incision used for implanting the polyester tape is relatively small at 6–9 cm. This incision should, however, be long enough to be able to mobilize the tendon and also to cause as little damage as possible to the surrounding soft tissues.
- *A drain should always be inserted.* A small hematoma can cause major problems. A plaster back slab must be used to prevent the ankle going into plantarflexion, as this has a detrimental effect on skin healing. Once the wound is sound, the plaster can be removed and physiotherapy can be started. To prevent stretching the repair, initial attention should be paid only to increasing the range of movement and the strength of plantarflexion. Dorsiflexion of the ankle is not allowed until 6 weeks after the operation.

Operative Details

As a prosthesis is used, intravenous antibiotic prophylaxis is necessary. General anesthesia is normally used and the operation is always undertaken under tourniquet control. The patient is placed prone on the operating table with a pillow under the mid-shin area with the foot and ankle sufficiently free of the end of the table to allow accurate assessment to be made of the range of dorsiflexion and plantarflexion at each stage of the operation. This is especially important when the polyester tape is tensioned and then tied.

An incision is made on the posteromedial aspect of the Achilles tendon, essentially at the site of the rupture. The incision needs to be longer than the one used for repair of an acute Achilles tendon rupture (6–9 cm), extending proximally beyond the site of the rupture, but avoiding, if possible, going too far distal to the rupture.

The incision is deepened to expose the tendon sheath. A vertical incision is made through the sheath to expose the tendon. The numerous adhesions usually present need to be carefully excised so that the tendon can be properly mobilized. Sometimes it is just not possible to satisfactorily excise these adhesions without removing some or all of the tendon sheath itself. Once the adhesions have been removed, the site of injury can be adequately visualized (Fig. 22.1A). Scar tissue fills the gap between the torn ends of the tendon. The scar tissue is slightly gray and dull as compared with the normal tendon. There is wasting and thinning in the region of the scar tissue as compared with the normal bulk of the tendon proximally and distally. There is, in addition, always wasting of the muscles of the calf; even though there is a successful outcome to surgical repair of the chronic rupture, patients never regain the normal bulk, though the strength of the gastroc-soleus complex can be restored.[11]

The proximal portion of the Achilles tendon and, if necessary, the gastroc-soleus muscle complex is mobilized to bring these structures down to their normal position. Similarly, the distal stump of the Achilles tendon needs to be freed and brought as far proximally as possible. The distal stump is often small, and tends to retract distally. It can, therefore, be extremely difficult to fully release it and bring it up to its normal position. At this stage of the operation, it is not possible to adequately assess how much gap will be present. An accurate assessment can be made only once the polyester tape has been passed through, and the tape fully tensioned to bring the proximal tendon distally and the distal tendon proximally.

A stab incision is made on the lateral aspect of the os calcis. The incision should be made in the line of the tendon and should be made as far proximally and posteriorly on the side wall of the os calcis (Fig. 22.2A) as possible to minimize the amount of tape lying subcutaneously on the surface of the os calcis (Fig. 22.3A, B). A 3.5-mm drill is used to produce a horizontal tunnel through the os calcis. There must be adequate cortical

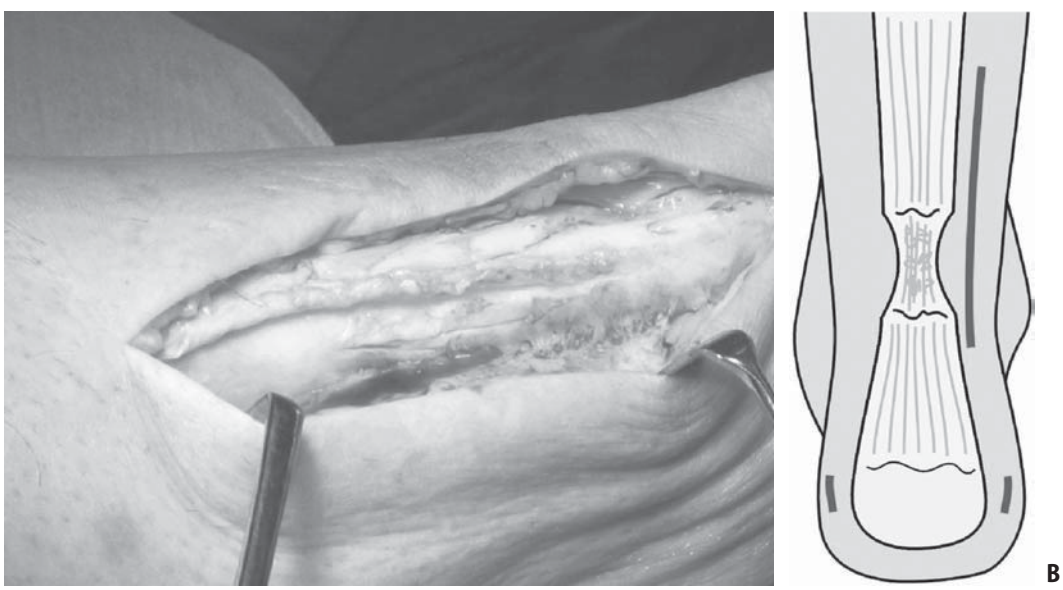


Figure 22.1 (**A, B**) The area of a chronic rupture of the Achilles tendon gives the appearance of an atrophic wasting on surgical exposure. The preferred approach is through a vertical posteromedial incision along the line of the tendon. Two stab incisions are made on the medial and lateral aspects of the calcaneum for passing the connective tissue prosthesis.

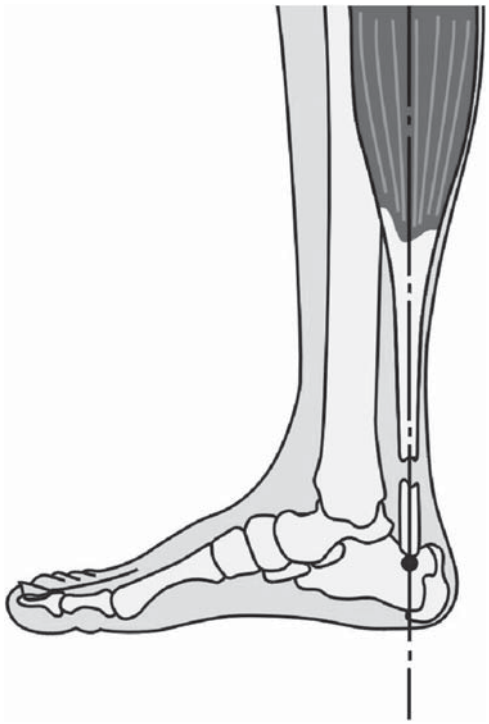


Figure 22.2 A drill hole is made in the body of the calcaneum as far superiorly and posteriorly as to maintain the mechanical axis of the Achilles tendon–gastrocnemius complex.

cover proximally so that the polyester tape does not cut out. A second stab incision is made on the medial aspect of the os calcis for the drill to exit through the skin. A malleable probe (Fig. 22.4A, B), blunt at one end and with a needle eye at the other, is used to pass the polyester tape through the tunnel from the lateral side to the medial (Fig. 22.5A, B). The two ends of the tape are then tunneled subcutaneously using the malleable probe, along the bony borders of the os calcis and into the distal stump of the tendon. Very little of the polyester tape should be left uncovered between the tunnel and the Achilles tendon (Fig. 22.6A, B). Most of the tape should be buried within the bone or the tendon. This reduces the incidence of postoperative irritation and discomfort. The malleable probe exits from the distal stump at the junction between the tendon and scar tissue. It is desirable to try to cross the two ends of the tape within the body of the distal stump (Fig. 22.7A, B). This gives better security and fixation of the tape within the distal stump and it also helps considerably to close the gap at the rupture site, because with tensioning the distal stump is brought proximal.

Tissue-holding forceps are used to pull the proximal tendon and muscle as far distally as possible. Further freeing of the adhesions may help

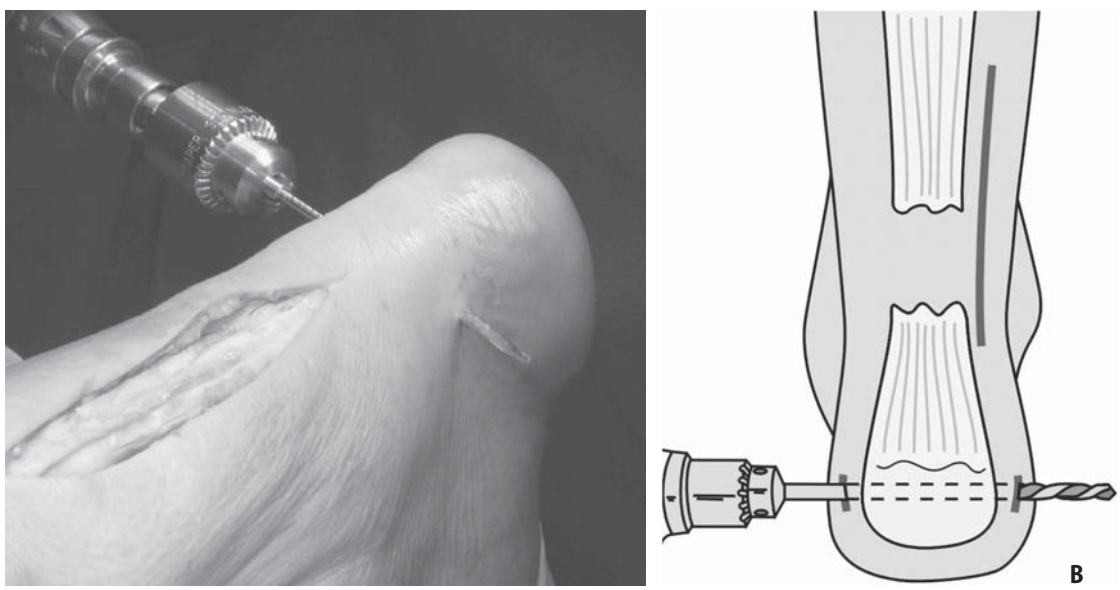


Figure 22.3 (**A, B**) A single drill hole is made from the lateral to the medial aspect of the calcaneum to accept the Leeds-Keio connective tissue prosthesis (CTP).

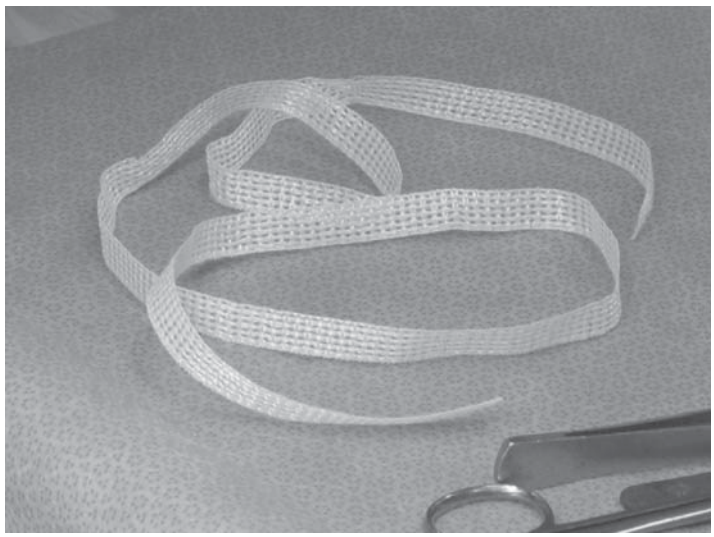
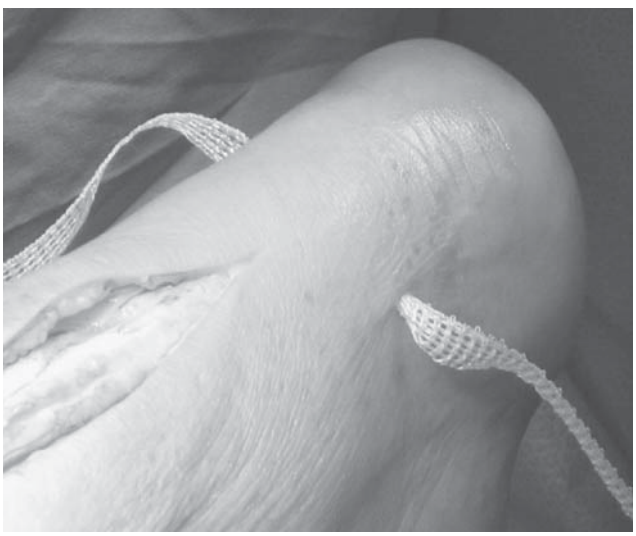
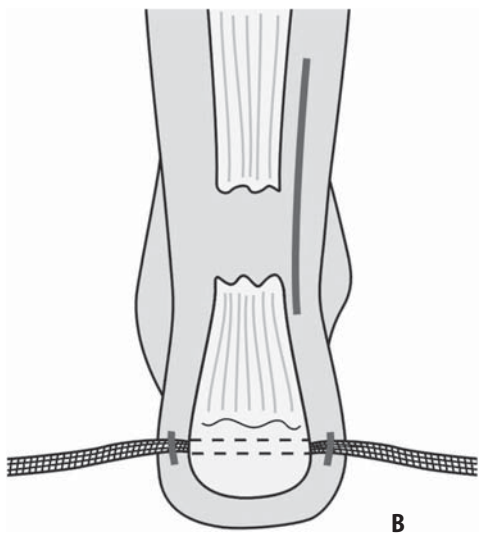


Figure 22.4 The Leeds-Keio connective tissue prosthesis (CTP) measuring 10 mm × 800 mm. (Manufactured by Xiros plc.)



A



B

Figure 22.5 (**A, B**) The connective tissue prosthesis is passed through the drill hole in the calcaneum from lateral to medial.

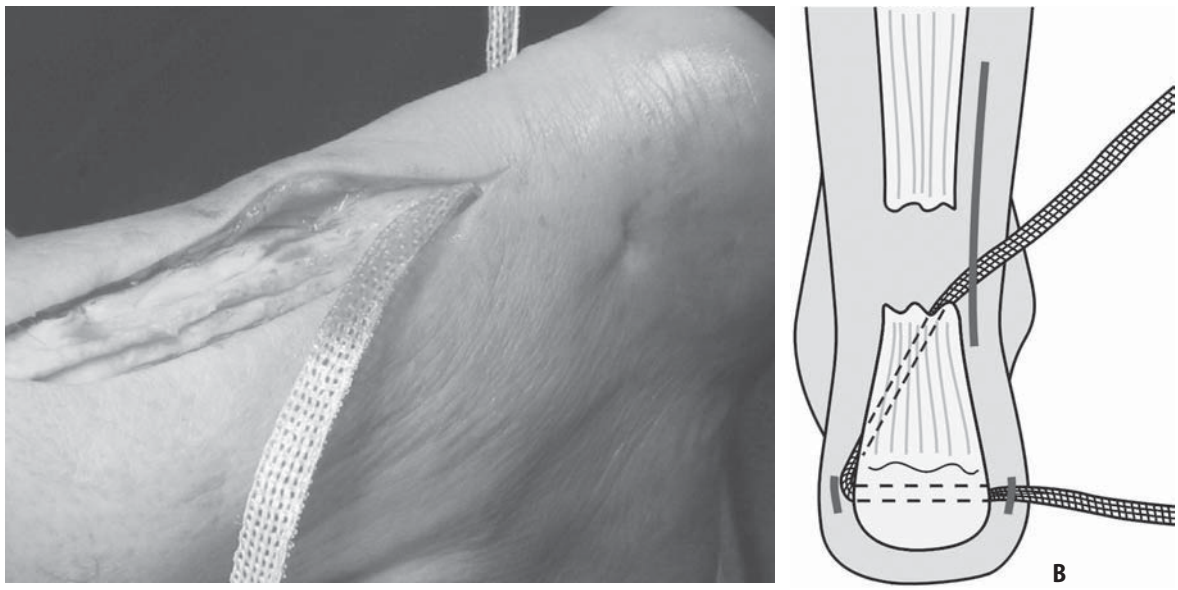


Figure 22.6 (**A, B**) Threaded onto a blunt malleable suture passer, the lateral end of the prosthesis is passed through the distal stump of the tendon to exit from the medial part of the distal ruptured end.

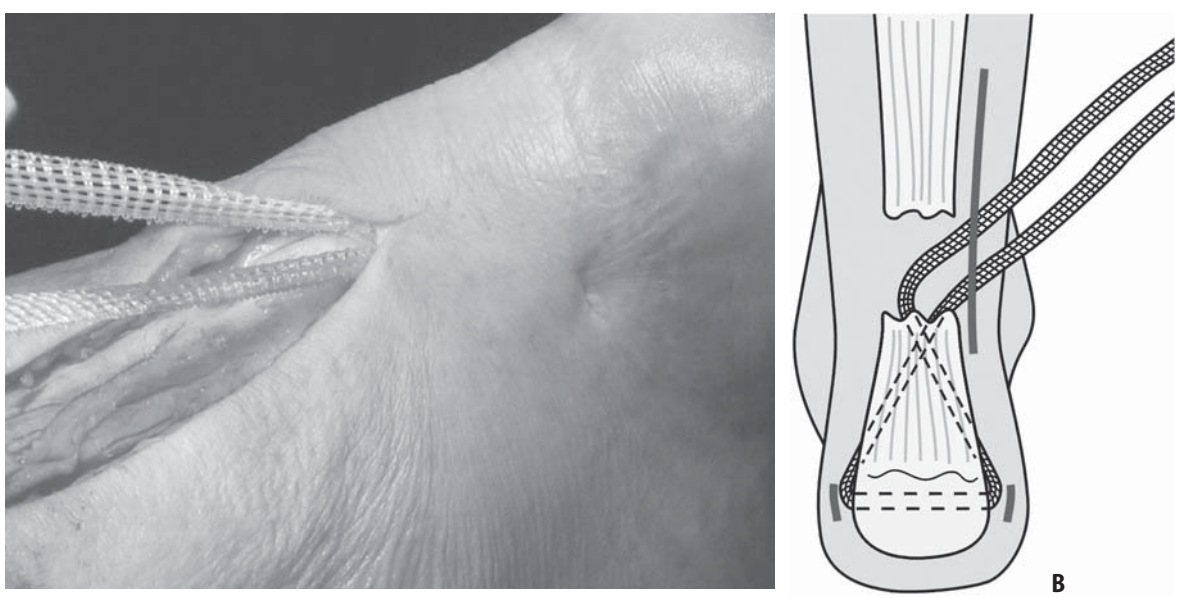


Figure 22.7 (**A, B**) The medial end of the prosthesis is passed through the distal stump of the tendon to exit from the lateral part of the distal ruptured end. The two ends of the prosthesis cross each other inside the substance of the distal tendon stump.

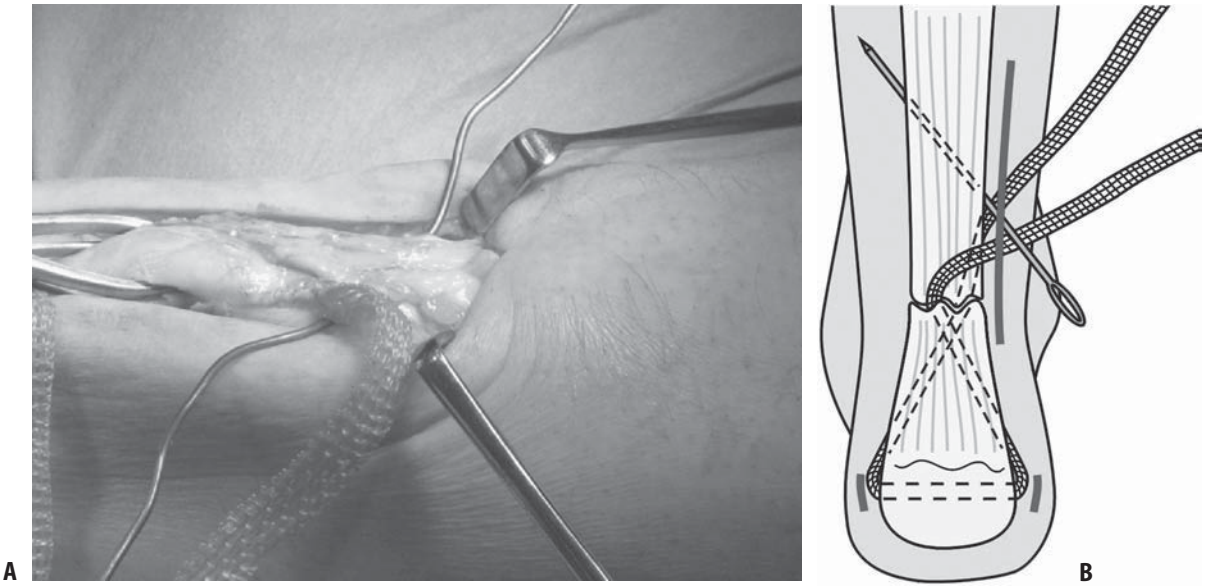


Figure 22.8 (**A, B**) The end of the prosthesis exiting from the medial part of the distal tendon stump is passed through the medial part of the proximal tendon stump again to exit medially at a proximal level.

in this maneuver. The long end of the polyester tape is threaded on to the probe and passed through the proximal tendon and muscle to create a Bunnell-type suture (Fig. 22.8A, B). In a chronic rupture, because the incision has been longer, it is possible to, at times with some difficulty, visually identify the exit and entry points of the probe. This helps to minimize the amount of polyester tape left on the surface of the tendon and it also means that the tape is not passed through the tendon sheath (Fig. 22.9A, B). It is essential, with each passage of the polyester tape through the

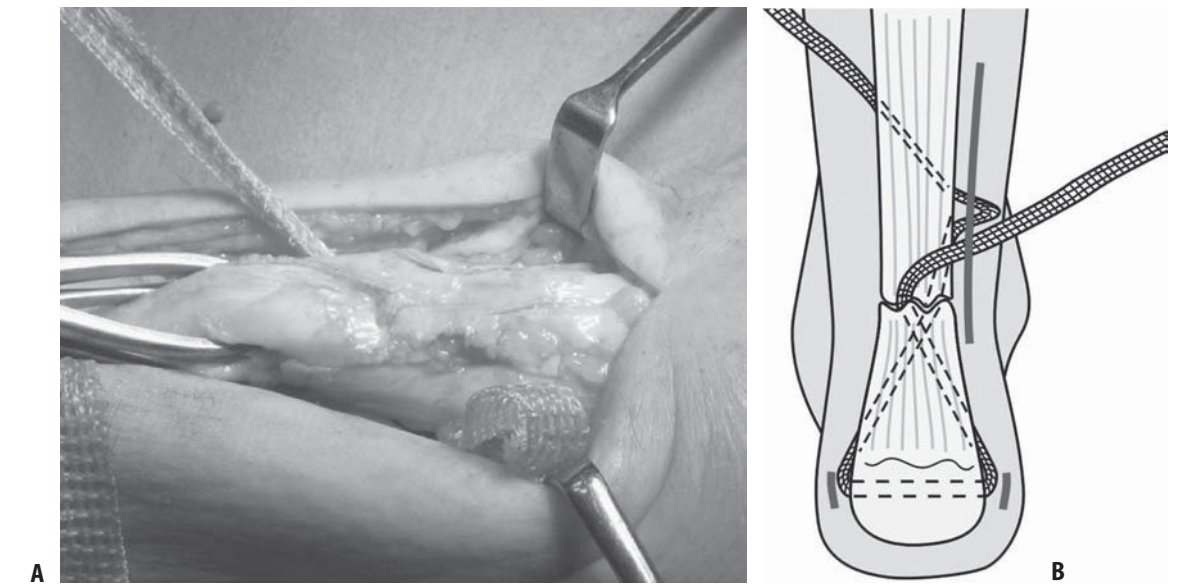


Figure 22.9 (**A, B**) The medial end of the prosthesis is passed through the proximal tendon in a superolateral direction. The loop of the prosthesis lying outside the tendon will be pulled in.

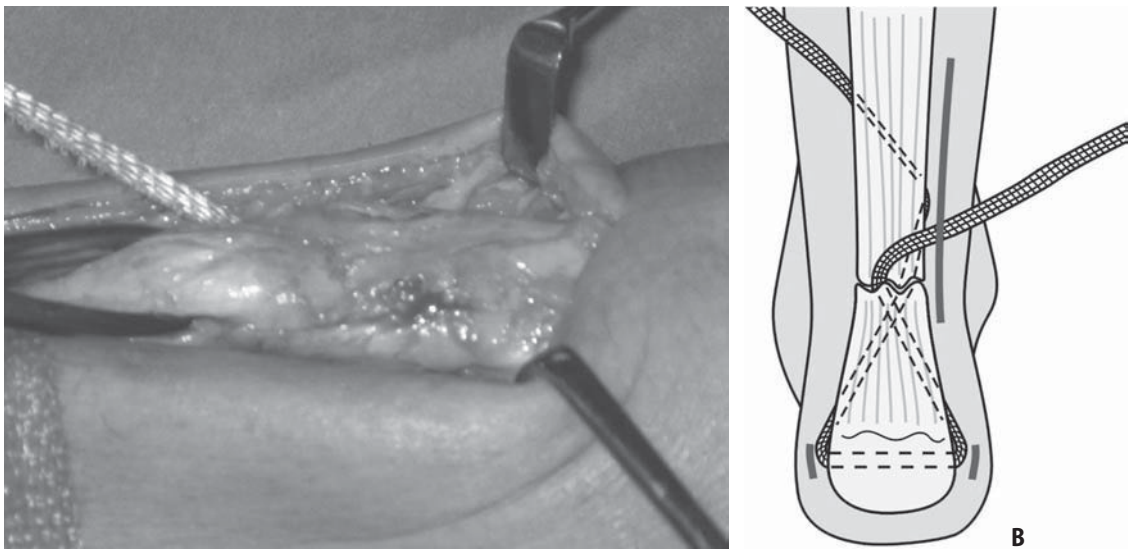


Figure 22.10 (**A, B**) The loop of the prosthesis seen outside the tendon in Figure 22.9A and B has been pulled into the tendon. It can be clearly seen that very little of the prosthesis remains outside the tendon.

tendon and muscle (Fig. 22.10A, B), first that the tendon is pulled distally and second that the tape is tensioned so that there is no laxity of the implant. It is not possible or indeed desirable to try to tension at the end of doing the Bunnell's suture (Fig. 22.11A, B). If an attempt is made to do this then, either the tape does not pull through properly or the tape cuts through the muscle. The tape should enter and exit at the proximal junction between the scar and the tendon.

Care must be taken when tensioning and knotting the tape. The greatest danger lies in leaving a "loose" repair. The assessment can and always has to be made carefully by evaluating the position of

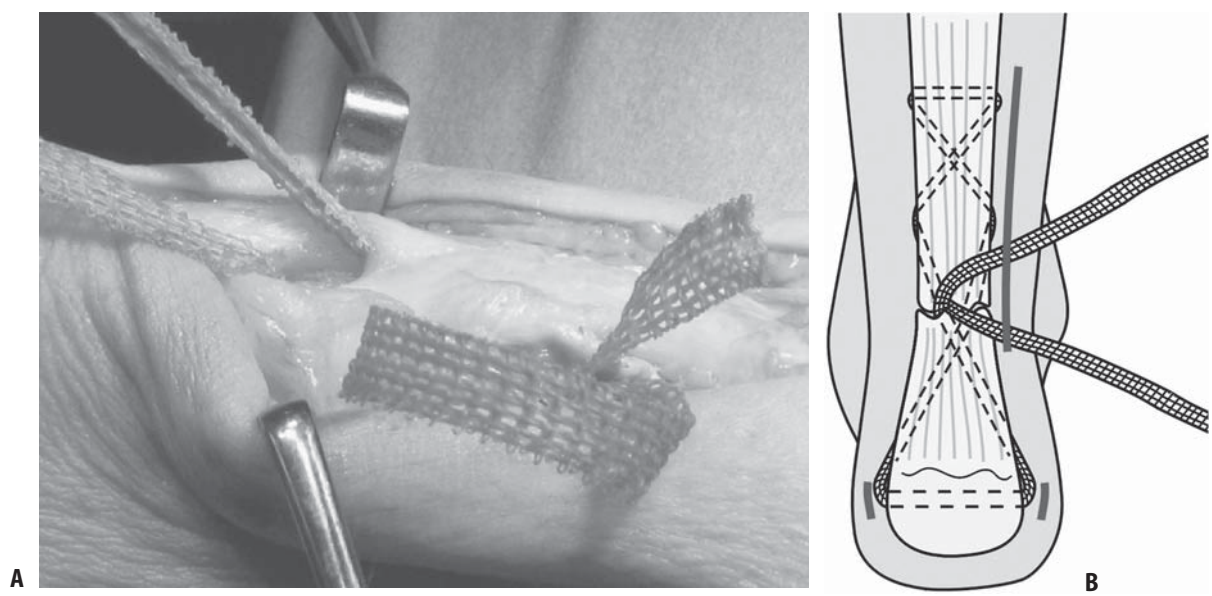


Figure 22.11 (**A, B**) The prosthesis is passed through the tendon in a criss-cross fashion to exit from the lateral end of the proximal tendon stump.

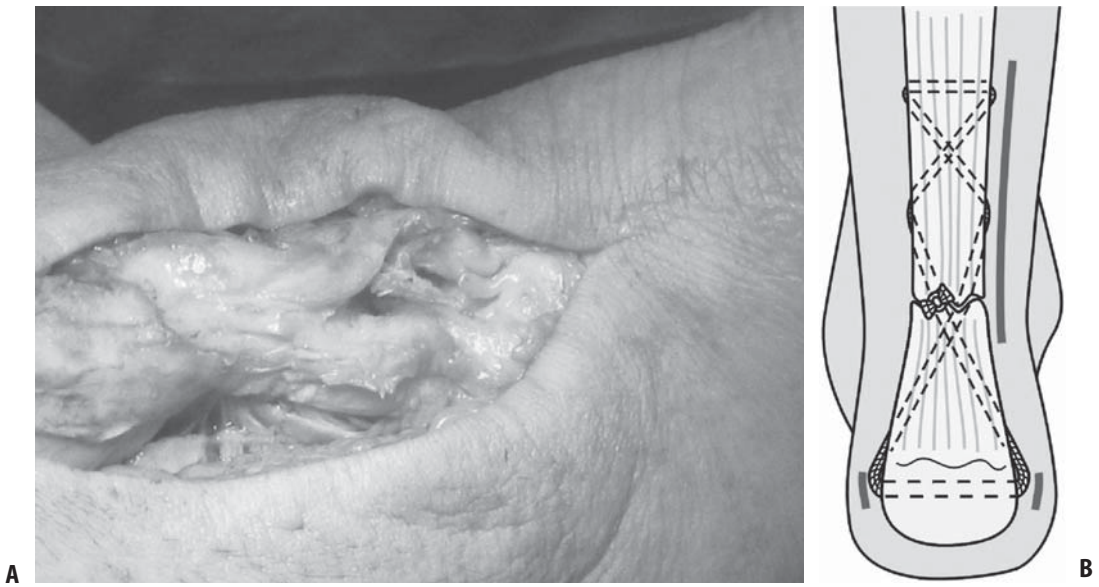


Figure 22.12 (**A, B**) The two ends of the prosthesis are tied using a reef knot. The ankle is held in some equinus and tension is maintained on the two ends of the ruptured tendon in order to close, as far as possible, the gap that is present.

the tendon both when the knee is flexed to 90° and when the knee is extended. Tension is applied and the gap closed. If, however, a gap is still present, this should not be of great concern, because the polyester tape is designed to act as a scaffold for the ingrowth of collagen. The tape should be tied with a reef knot (Fig. 22.12A, B) with the ankle at 5–10° of plantarflexion. In that position, the tape/tendon should be tight enough to just allow the foot to be brought to neutral (Fig. 22.13A). It

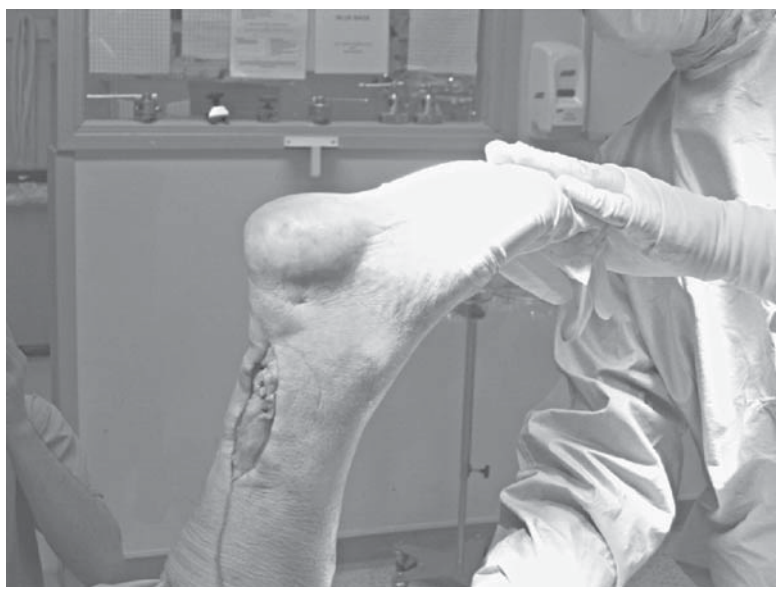


Figure 22.13 The stability of the repair is demonstrated by testing dorsiflexion of the ankle.

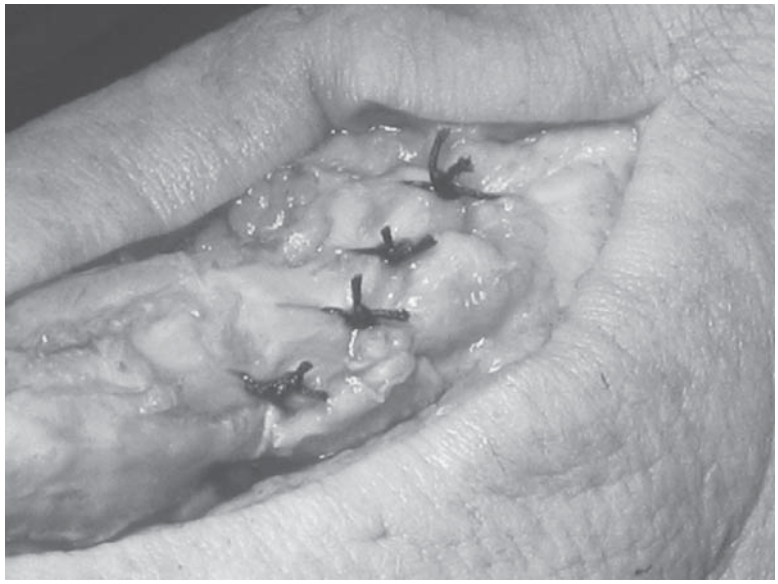


Figure 22.14 Sutures are used to approximate the two ends of the tendon so that the knot in the prosthesis is covered and therefore not exposed.

should not allow the foot or the ankle to go into dorsiflexion. There will, of course, be no limitation of plantarflexion. The long ends of the tape can then be cut about 1 cm away from the knot. The knot should lie within the confines of the tendon and scar tissue. To minimize the amount of synthetic material exposed, the scar tissue can be mobilized, split, and brought over and sutured so that it covers the knot and remnants of the polyester tape (Fig. 22.14A, B). The bulk of the tape and scar tissue adds somewhat to the size of the tendon at the repair site and gives it a slight fusiform shape. A running suture of absorbable Vicryl is used to close the tendon sheath, though, given the preexisting abnormalities of the sheath, it may not be possible to achieve this. With the larger incision needed in chronic rupture and with the amount of dissection necessary, it becomes imperative to use suction drainage. Bleeding does occur, and the presence of a hematoma and the possibility of infection will have an adverse effect on wound healing.

The rest of the wound is closed in layers. The position of the ankle at the time of suturing is important. The ankle should be kept close to a neutral position as possible because, if the ankle is allowed to go into plantarflexion and the skin "wrinkles," accurate skin apposition is not possible (Fig. 22.15A, B). In addition, if the ankle is not kept in this ideal position over the next two weeks, there is poor wound healing with oozing from the skin edges. A plaster back slab is used to maintain the ankle just short of neutral.

The day after surgery the wound is inspected and the drain removed. Patients are allowed to mobilize with crutches non-weight-bearing. At 12 days from surgery, the plaster is removed.

The plaster that is applied is not to be thought of as a splint or a protection for the repair of the Achilles tendon. The plaster back slab is used to hold the ankle in a relatively neutral position. By doing that, there is a greater likelihood of primary wound healing.

If the wound is healed, the sutures are removed and physiotherapy is started. Physiotherapy should consist of passive and active plantarflexion. Initially patients should not be allowed to actively dorsiflex. Similarly the therapist should not try to passively bring the ankle beyond the neutral position. Partial weight bearing is allowed, though a raise to the heel of the shoe may be necessary. The raise should be 1 or 2 cm, half within the shoe and half on the outer heel of the shoe.

Recovery from a repair of a chronic rupture is somewhat slower than that from a repair of an

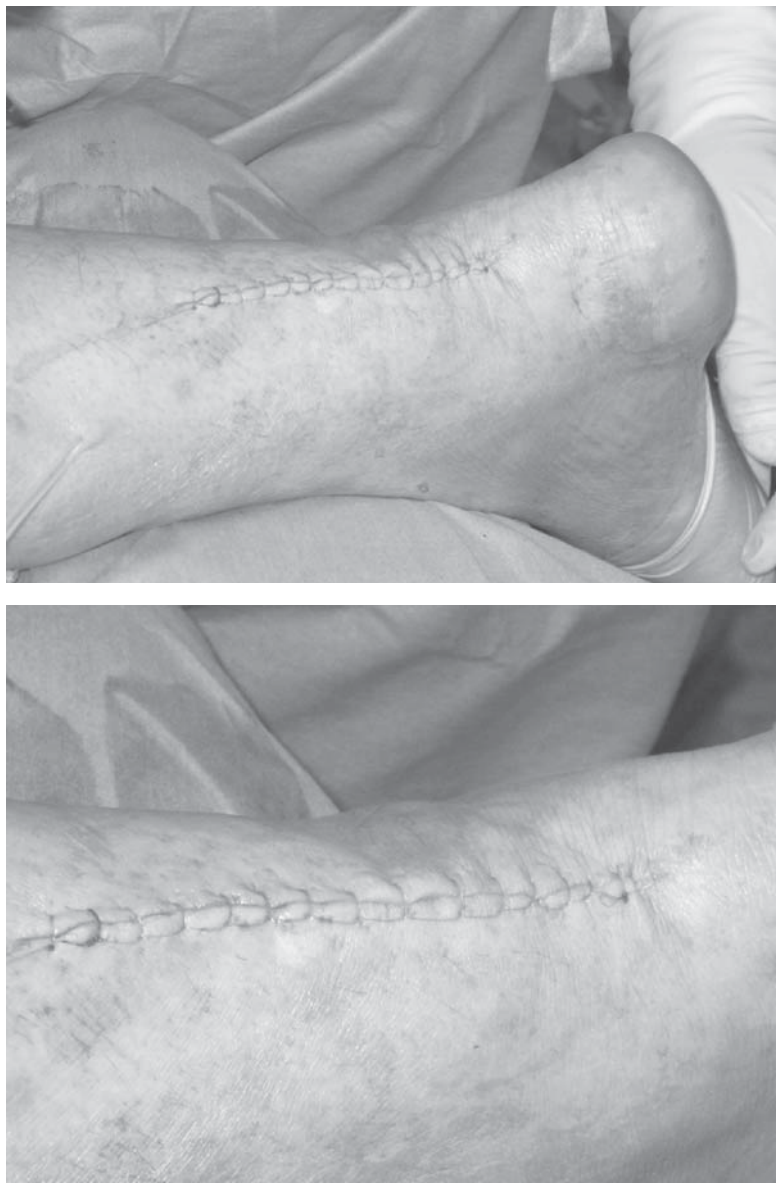


Figure 22.15 (**A, B**) In equinus, the skin over the tendon appears wrinkled. However, when the foot is put into neutral, the wrinkles tend to disappear, leaving the skin smooth.

acute rupture, though progression to normality is relatively rapid. Full weight bearing is allowed at 4–6 weeks from the operation. Patients may start driving a car at that time. At 6 weeks, the physiotherapist can initiate active and passive dorsiflexion. By 3 months from surgery, return to normal activities including sports can be encouraged. Early results[10] of the use of the polyester tape in repair of chronic Achilles tendon rupture has led us to adopt a slightly more aggressive approach to early rehabilitation. In addition, early movement facilitates a more rapid and stronger healing of the tendon without the development of adhesions.

Complications

There are specific problems and complications that arise when one uses an artificial substance to repair Achilles tendon rupture. Superficial infection can, and does, occur and can be treated by a combination of oral and intravenous antibiotics plus rest. The greater worry for most surgeons, however, is what to do if a deep infection arises. No matter how much care the surgeon takes to prevent this complication, the risk of deep infection is always present. Prophylactic antibiotics, operating in a clean air enclosure, and meticulous surgical techniques will help. It is perhaps the way in which the polyester tape is incorporated into the tendon and muscles (i.e., by using a type of weave) that raises doubts in surgeons' minds. How can you remove the polyester tape if there is deep infection? What will happen to the repair? It should not be necessary, however, to remove all of the implant. If a deep infection does occur, then at exploration the surgeon can safely remove the part of the tape that is exposed, leaving the rest of the implant embedded in the tendon and muscle. If the correct surgical technique has been used, very little of the tape should be exposed, and therefore, the integrity of the repair will not be affected. With adequate surgical exploration, debridement, and washout, plus intravenous antibiotics, a deep infection can be controlled and the polyester tape can be left in situ. There is a direct comparison to be made with the management of aortic aneurysms using a Dacron graft. In those cases, if infection occurs, then the graft is left in situ and the management is along the lines mentioned above.

The cut ends of the polyester tape can cause irritation and superficial infection. This problem can be eradicated simply by snipping off parts of the tape that are exposed and giving the patient oral antibiotics.

Localized pain sometimes occurs where the tape exits from the os calcis tunnel and is then taken proximally into the distal stump of the tendo Achillis. With time and with the ingrowth of tissue into the polyester tape, irritation and pain present at this site usually settle. Scar tissue forms over and into the exposed polyester tape on the surface of the os calcis. This essentially buries the tape and prevents irritation. If the symptoms do not settle, local injections of Depomedrone and Marcain are beneficial. Rarely is an exploration necessary, but, if that were the case, surgery would be delayed until the Achilles tendon repair was sound. Consequently, by that stage, it would be safe to remove all the exposed implant material without affecting the repair.

Chronic ruptures of the Achilles tendon are associated with marked wasting and weakness of the calf muscles. While it is to be expected that there will be some increase in bulk of the calf muscles, there will never be complete restoration in the shape of the calf. There will, however, be some restoration of strength, and in our recent studies, patients have been shown to regain 90–95% of normal calf muscle strength.[11]

Clinically, the Achilles tendon appears thicker after the repair. This is to be expected and is a consequence of the presence of the polyester tape and the subsequent ingrowth of collagen. The broader the tape used, the thicker is the resulting Achilles tendon. A 10-mm-wide polyester tape is actually stronger than a normal Achilles tendon, and can, therefore, resist the stresses put across the tendon during the early phase of rehabilitation when there has not been full biological healing of the repair. In our first few patients, we used a 20-mm tape, but it was difficult to close the tendon sheath, and the repair produced a thicker tendon than one would wish. The wider tape did not result in increased adhesions or more pain in the postoperative period, and did not limit the movement of the Achilles tendon.

With complex surgery to repair a chronic rupture of the Achilles tendon, a decreased range of movement would, perhaps, be expected. This, however, has not proved to be the case. Indeed, an essential part of the operation is to tension the polyester tape with the foot in plantarflexion so as to limit dorsiflexion. Despite great emphasis being placed on that point, patients do regain normal dorsiflexion, and some may have a minor degree of increased dorsiflexion. Some stretching of the polyester tape is to be expected during the aggressive early rehabilitation phase. Hence, the range of movement always increases postoperatively; it never decreases. It is, therefore, important at surgery to achieve correct tensioning of the polyester tape, placing the foot in slight plantarflexion.

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23
Management of Complications

Daniel N. Ronel

Complex Wounds

Complications from failed surgery, severe infection, or massive trauma may leave a complex wound involving the soft tissue around the Achilles tendon. A tendon defect in the setting of large tissue loss is a more difficult problem than simple repair of a ruptured or lacerated tendon. Successful reconstruction must include three essential elements: (1) tendon repair sufficient to provide strength, durability, and tension; (2) a soft tissue cushion thick enough to protect the area but thin enough to permit normal foot wear; and (3) a skin cover strong enough to withstand the repetitive friction and shearing forces of ambulation.[1] The defects are often contaminated with bacteria, and require viable healthy vascularized tissue so that components of the immune system and antibiotics can be delivered to the infected site. In a few cases, limited surgical treatment simply with skin flap coverage and controlled scar formation may achieve acceptable functional results, and this may be the safest option for those patients who cannot tolerate extensive surgery.[2]

The availability of local muscle, skin, or fascia to cover tissue defects in the lower leg is limited. The early surgical management of complex injuries to the Achilles tendon region involved multiple staged procedures, multiple prolonged hospital stays, and a relatively high incidence of postoperative complications, especially the formation of fibrous adhesions impairing gliding of the reconstructed tendon. The arcs of rotation and vascular supply of the gastrocnemius and soleus muscles usually preclude their use in the distal third of the foreleg. Intrinsic foot muscles are too small to cover large amounts of tissue loss. Plantar flaps of adequate size would hinder weight bearing and walking. A dorsalis pedis flap would require a skin graft on the donor site defect, which itself would be a fragile area (although friction from shoe wear could be minimized by wearing an open-toed sandal). Flaps that require the sacrifice of either the dorsalis pedis or posterior tibialis artery circulation would place at great risk the remaining blood flow to the foot.[3]

Despite these limitations, procedures have been developed that combine functional tendon repair and skin coverage for small and moderately sized defects. They are particularly suitable for hospitals where the infrastructure for microsurgery is not available. A small defect can be simply closed with a V–Y musculocutaneous advancement flap of the gastrocnemius over the soleus, attaching the proximal stump of the Achilles tendon to the calcaneus.[4] Another option for a small defect combines the thick skin of the medial plantar flap with a tensor fascia lata tendon graft, and has an acceptably low amount of friction from shoes on the repaired area.[5] A somewhat complex procedure replaces the Achilles tendon with a free fascia lata graft, covers it with a gastrocnemius flap turned down from above, and provides skin coverage with a local anteromedial fasciocutaneous flap.[6] A series that reported the successful treatment of small soft tissue defects in combination with tendon repair used peroneus brevis tendon covered by peroneal reverse flow island flaps or posterior tibial reverse flow flaps: the choice of flap was determined by available uninjured and

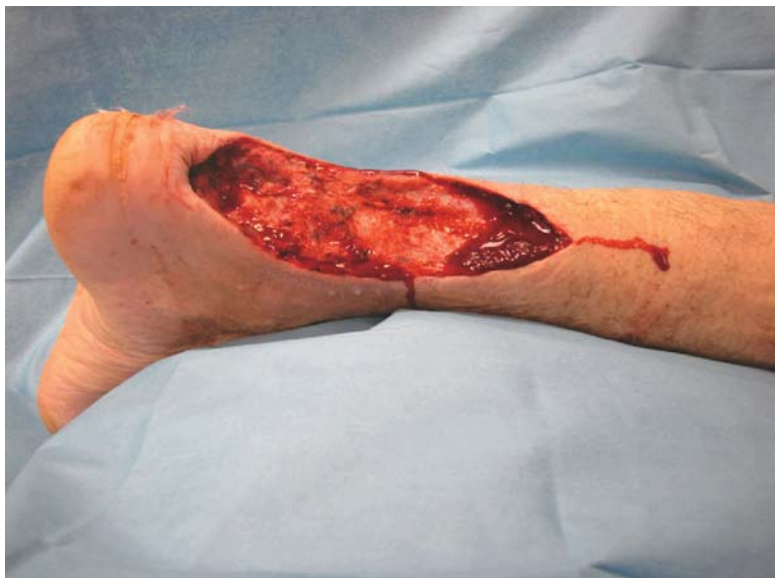

Figure 23.1. Large soft tissue defect that required free tissue transfer. (Reproduced with permission from Newman et al., 2005, Reference 58.)

scar-free tissue on the donor site and by the location of the defect.[7] The donor sites were skin grafted and one of the major arteries of the lower leg was sacrificed.

A potential problem with the procedures that use an avascular fascial graft for tendon repair is the formation of uncontrolled scarring and adhesions. There are reports of successful reconstruction using local vascularized tissue: a pedicled medial plantar flap with vascularized plantar aponeurosis was used for the reconstruction of a relatively small defect.[8] Another vascularized tendon graft used a peroneal cutaneotendinous flap from the leg adjacent to the Achilles wound.[9] This graft is based on a cutaneous perforator from the peroneal artery that also invests the peritendinous areolar tissue around the peroneus longus tendon. The donor site was skin grafted. A recently reported composite graft seemingly as complex as its name is the venoneuroadipofascial pedicled distally based sural island myofasciocutaneous flap. It is a modified neurocutaneous flap based on the vascular axis around the sural nerve, the short saphenous vein, and the lowest two septocutaneous perforators of the peroneal artery. Three cases have been reported, one of which included reconstruction of the Achilles tendon with the lateral belly of the gastrocnemius muscle.[10] These non-microsurgical vascularized repairs may be technically difficult, and are likely to have a high rate of failure.

Microsurgical Free Flaps

The development of microsurgical techniques made free tissue transfer possible to cover large defects in the Achilles region (Fig. 23.1). Free tissue transfer supplies vascularized tissue to enhance wound healing and to protect against infection. Skin, fascia, muscle, bone, and nerve can be transferred in various combinations to replace the function of lost tissues. The earliest microvascular free flaps were reported in 1971 and 1973: an abdominal dermis-fat graft was first transferred to a facial defect by anastomosing the superficial epigastric artery and a cuff of the femoral artery to the external carotid.[11] Two years later a defect in the skin and soft tissue of the ankle was covered with a skin island supplied by the superficial inferior epigastric and superficial circumflex arteries, which were anastomosed to the long saphenous vein, posterior tibial artery, and a vena comitans.[12] The first musculocutane-

Table 23.1. Microsurgical Flaps for Achilles Tendon Reconstruction

Donor Site	Advantages	Disadvantages
• Groin flap + external oblique aponeurosis, iliac bone, and/or abdominal fascia[27–29]	• Can reconstruct calcaneus • Donor scar usually hidden under clothing	• Short, thin pedicle • Need for donor site repair • Bulky
• Tensor fascia lata + lateral femoral cutaneous nerve[30–32]	• Can be sensate	• Bulky
• Lateral/anterolateral thigh flap + rolled fascia lata[1,34,35]	• Long, large pedicle • Large skin paddle • Direct closure of donor site if < 8 cm • Can include muscle • Can be sensate • Subcutaneous fat for tendon gliding	• Donor site weakness • Anatomic variation of perforators • Skin graft donor site if large skin paddle needed
• Gracilis muscle and tendon[37]	• Good soft tissue contour	• Skin graft recipient site
• Infragluteal flap with ischio-cutaneous ligament, posterior thigh cutaneous nerve[38]	• Hidden donor site, can be closed directly • Large skin paddle available • Can be sensate	• Anatomic variation of pedicle • Difficult nerve dissection
• Dorsalis pedis flap + extensor digiturum longus (EDL) strips, superficial peroneal nerve[39]	• Can be sensate	• Limited toe excursion • Donor site skin graft • Patent posterior tibial artery required
• Radial forearm flap + extensor carpi ulnaris, palmaris, or tendon of brachioradialis + superficial radial nerve or lateral antebrachial cutaneous nerve[16,42–46]	• Can be sensate • Moderately large donor site • Long, large consistent pedicle • Thin subcutaneous fat layer	• Donor site skin graft • Patent ulnar artery required • May not adequately replace large tendon defect
• Lateral arm flap + posterior cutaneous nerve, triceps tendon and muscle, or brachioradialis tendon + olecranon, portion of radius, or fascia lata[47–52]	• Can be sensate • Moderately large donor site • Long, large consistent pedicle • Thin area near lateral epicondyle • Can reconstruct calcaneus	• Donor site skin graft or scar • Possible radial nerve injury • Bulky unless use area near epicondyle
• Latissimus dorsi[56,57]	• Can be sensate • Thin subcutaneous fat layer	• Difficult nerve dissection • Frequent donor site seromas

ous free flap was reported in 1975, a de-epithelialized gluteus maximus transfer in a patient with aplasia of the breast.[13] Free flap coverage of posterior lower leg soft tissue defects with muscle or fascia quickly became popular, with donor tissue taken from many parts of the body.[14–20] Several groups reported success rates of 92–96%.[3,21–24] In addition to the benefits provided by the importation of vascularized tissue, early free flap design often included a cutaneous paddle or a gliding surface for the Achilles tendon.[25]

The simple coverage of soft tissue defects by free tissue transfer evolved into the reconstruction of complex defects with composite grafts made from different tissue types. Most composite grafts for reconstruction of Achilles region defects have been developed from leg and arm donor sites. These reports elucidate several variables that should be considered when designing a reconstruction plan for a particular defect (Table 23.1):

- Topographic anatomy of the defect: requirements for soft tissue volume and the amount of tendon replacement
- Requirement for a sensate flap
- Donor site characteristics: availability, associated morbidity, requirements for closure with mesh or skin graft
- Characteristics of the donor tissue vascular pedicle: required length and caliber to create an anastomosis without tension and outside the zone of injury, ease of dissection, requirement for interpositional vein grafts
- Need for further debulking procedures

It is not possible to choose a microsurgical technique for complex Achilles reconstruction based on outcome studies, as the reported series are too small. There is no standardized method of evaluating the outcomes of Achilles tendon repairs, making it difficult to compare results among the techniques. The most useful and simple

classification scheme groups outcomes into four categories, excellent to poor.[26] An excellent result is full function with no residual disability; a good result has the presence of slight weakness, an adherent scar, or a minor sensory deficit, but with no real limitation of activities and a full return to function as in the pre-injury period; a fair result has a definite weakness, some limitation of activities, and a slight limp; a poor result has a re-rupture or complete failure with severe weakness and a marked limp.

Lower Extremity Composite Free Flaps

Early composite grafts included free groin flaps with a sheet of external oblique aponeurosis, iliac bone, or abdominal fascia.[27–29] Advantages of these groin flaps included the ability to transfer iliac bone with the graft for a calcaneal defect, as well as the location of the donor scar, which was usually hidden by underclothing. Disadvantages included a short and thin superficial circumflex iliac artery pedicle, the need for repair of the external oblique in the donor site with foreign material, and the frequent need for further debulking procedures for a flap that was too thick for the posterior ankle region. Although there was limited dorsiflexion, the results were satisfactory.

Many constructs from thigh donor sites have been developed for reconstruction in the region of the Achilles tendon. One of the first reported is a fascial composite flap that transfers the tensor fascia lata with the lateral femoral cutaneous nerve to create a neurovascular, sensate free flap.[30] Another report described a tensor fascia lata fasciocutaneous perforator flap in five patients: the lateral femoral cutaneous nerve was included to provide sensation, and the donor site was closed primarily in most patients.[31] Several patients underwent further debulking procedures at the ankle. A tensor fascia lata myocutaneous flap has also been described in a patient who required little soft tissue replacement other than the Achilles: the entire iliotibial tract was used to replace the tendon, and skin coverage was cosmetically acceptable.[32] A disadvantage of the tensor fascia lata flap is its bulk, which is often too excessive for normal shoes in the narrow distal posterior leg region. The consistency of its vascular pedicle has also been questioned.[33]

Other procedures involve a free lateral thigh or anterolateral thigh flap with fascia lata sheet (Fig. 23.2).[1,34,35] The fascia lata in these flaps is rolled into a cylinder to replace the missing tendon segment. Advantages include a long and relatively large vascular pedicle (the descending branch of the lateral femoral circumflex artery), a large skin paddle of up to 800 cm^2, a donor site that can be closed directly if the defect is less than approximately 8 cm, ample subcutaneous fat to permit tendon gliding, and the possibility of including the rectus femoris or vastus lateralis muscle.[29] Inclusion of these muscles may be difficult, however: splitting the vastus lateralis longitudinally may jeopardize the blood supply to part of the muscle, and in the case of the rectus femoris, the takeoff of its pedicle is very close to the site of anastomosis on the lateral femoral circumflex. Other disadvantages of the thigh flaps are the anatomic variation and small size of many of the perforators from the lateral femoral circumflex and the profunda femoris, as well as the need for skin grafting the donor site if a large skin paddle is needed.[35,36] Although not yet described, it could be possible to construct a sensate flap using the lateral cutaneous nerve of the thigh.

The only composite flap reported from the medial thigh is a gracilis free flap that that was used in one case to reconstruct the Achilles tendon and provide vascularized soft tissue.[37] The flap was fitted to the tendon defect by folding the gracilis tendon on itself and suturing it to the muscle belly. A skin graft covered the muscle. There was excellent functional restoration of the tendon and an acceptable soft tissue contour.

A successful technique using a fasciocutaneous infragluteal flap has been reported in five patients with very good to excellent results.[38] Partial and small complete tendon defects were repaired with the ischiocutaneous ligament of the gluteal crease, and a branch of the posterior cutaneous nerve of the thigh was used to provide sensation. The arterial supply is somewhat variable, and dissection of the nerve can be difficult, but a large amount of soft tissue is available and the hidden donor site can be closed directly.

A dorsalis pedis free flap from the same foot has been reported, which included tendon strips of

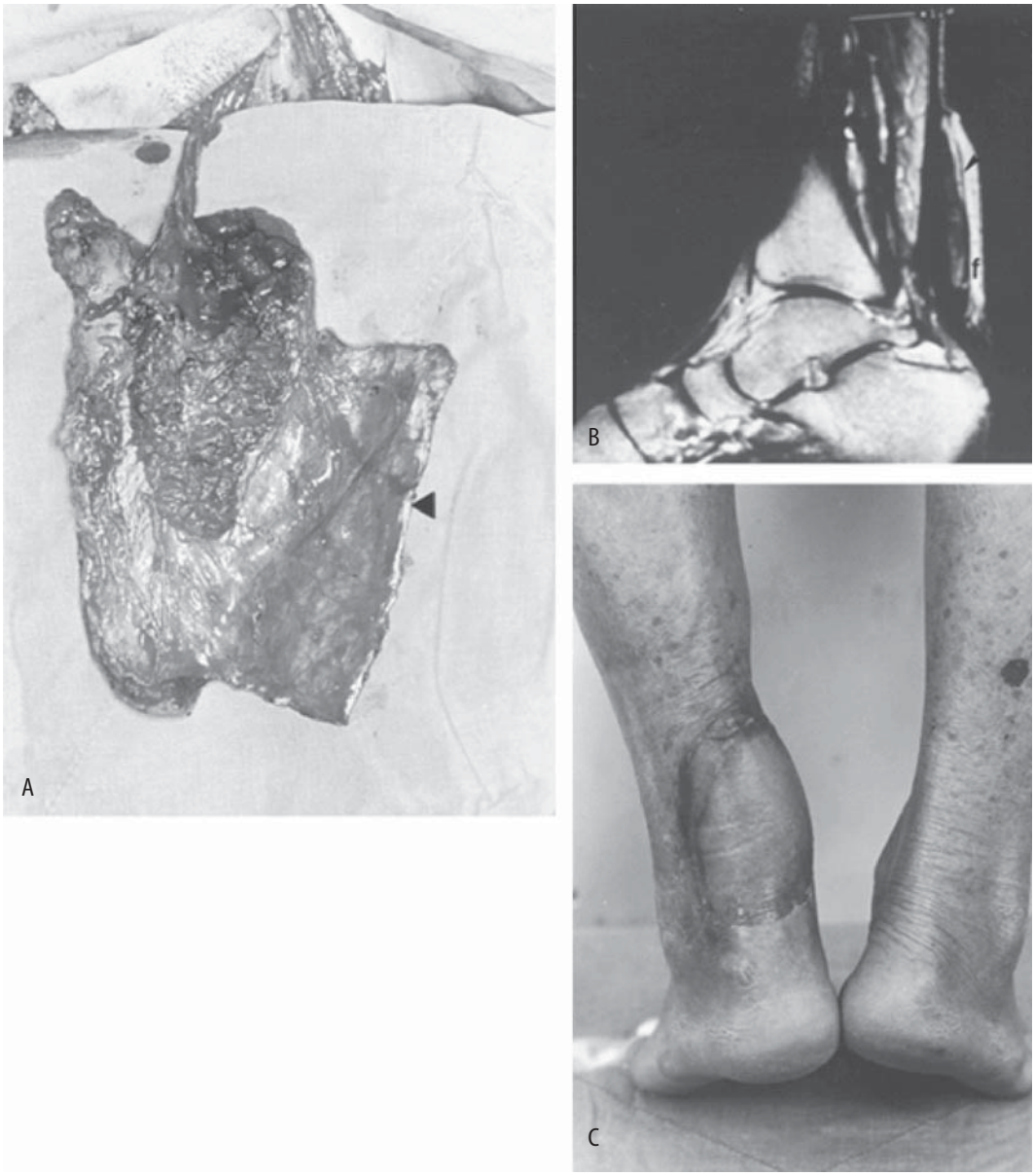

Figure 23.2. (**A**) Design of a 12- by 7-cm composite anterolateral thigh flap including a 6- by 8-cm strip of fascia lata (arrowhead) at the right thigh. (**B**) Magnetic resonance images demonstrating continuity of the reconstructed Achilles tendon 3 months postoperatively (arrowhead). f = fascia lata graft. (**C**) Left heel with stable coverage 2 years postoperatively. (Reproduced with permission from Kuo, et al., 2003, Reference 35.)

extensor digitorum longus and the superficial peroneal nerve. Excellent results were obtained after a second lengthening procedure and protective sensation was restored to the region, but there was limited toe excursion and the dorsalis pedis donor site required a skin graft.[39] A robust posterior tibial artery would be required to preserve blood flow to the foot.

An anterior rectus sheath fasciocutaneous free flap was successfully used to reconstruct the Achilles tendon and provide skin cover in two patients with infected recipient sites.[40] Musculo-

cutaneous flap coverage is superior to fasciocutaneous flap coverage in the presence of infection.[41]

Upper Extremity Composite Free Flaps

Radial forearm and lateral arm constructs have also been developed to address the problem of complex Achilles region reconstruction. A radial forearm flap raised with extensor carpi ulnaris and palmaris longus tendons was used to reconstruct an area of the posterior lower leg that did not require much tissue bulk.[19] Other radial forearm composite flaps have included the tendons of the brachioradialis and palmaris longus as well as the superficial radial nerve or the lateral antebrachial cutaneous nerve.[42–46] One group augmented the vascularization of the recipient site by harvesting wide areas of fascia from the free flap donor site and wrapping it circumferentially around the reconstructed tendon; the results of their four patients were excellent, with no evidence of peritendinous fibrosis.[44] Advantages of the forearm flap include a moderately large area of donor skin, a long pedicle of relatively good caliber and consistent anatomy, the potential for incorporating nerve, and a thin subcutaneous layer of fat that can provide a contour similar to the normal lower leg. Disadvantages include the unappealing cosmetic result of a skin graft on the donor site, the requirement for a patent ulnar artery, and inadequate tendon volume for replacing a long Achilles tendon segment.[35]

Lateral arm flaps based on the profunda brachii vessels have been reported.[44,47–50] Composite combinations may include the posterior cutaneous nerve, the triceps tendon vascularized with a portion of the triceps muscle, and the brachioradialis tendon vascularized with fasciocutaneous perforators. The olecranon or a portion of the radius can be included to secure the distal triceps or brachioradialis tendon anchors, respectively (Fig. 23.3).[49,50] If more tendon strength is required than can be supplied from the arm, a lateral arm flap can be wrapped around a sturdy section of fascia lata from the thigh: the fascia is interposed into the Achilles defect and the vascularized fascia of the lateral arm flap is wrapped around the tendon to provide a gliding surface. Results of such a sensate flap were good in five patients, although there were two donor sites and mesh was required to prevent herniation of the vastus lateralis.[51] Advantages of lateral arm flaps include consistent vascular anatomy, a long pedicle of

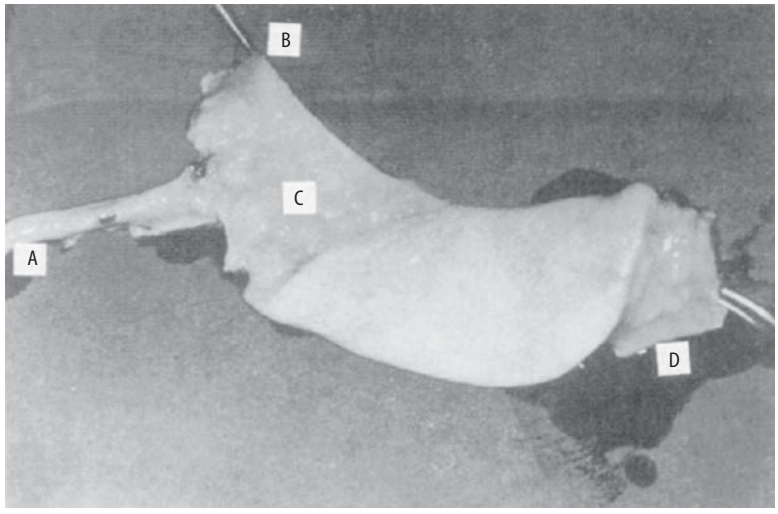

Figure 23.3. Free composite left forearm flap including the brachioradialis tendon and radial bone fragment. (A) Vascular pedicle. (B) Brachioradialis tendon graft. (C) Septofascial layer. (D) Fragment of radial bone. (Reproduced with permission from Stanec, et al., 1999, Reference 50.)

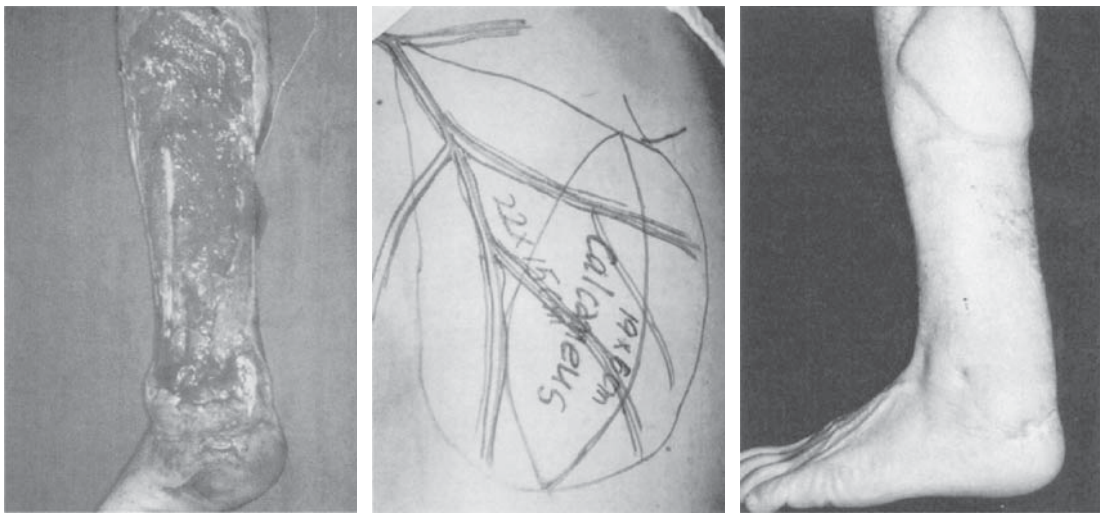

Figure 23.4. (**A**) Total elimination of the Achilles tendon and gastrocnemius-soleus muscle. (**B**) Design of the latissimus dorsi myocutaneous flap; the donor site was closed primarily without tension. (**C**) Four years after surgery. (Reproduced with permission from Lee, et al., 1999, Reference 56.)

relatively good caliber, an area of thinness near the lateral epicondyle, and a relatively large area of donor skin. Disadvantages include the noticeable donor site scar, occasional dysesthesia from radial nerve injury, and soft tissue bulkiness, although the flap has been extended distally to an area of thinner subcutaneous tissue.[52]

A donor site particularly suitable for complex Achilles tendon region reconstruction is the latissimus dorsi. The latissimus dorsi is a large, versatile muscle (Latin: *latissimus*, superlative of *latus*, wide). It can be removed completely or in part, and when necessary it can be transferred with an overlying skin paddle.[53] Although it contributes to arm extension, adduction, and internal rotation, and to scapular medial downward movement, its removal produces only minimal functional deficit. The thoracodorsal pedicle is relatively large and can be skeletonized from 9–15 centimeters, sufficient length to allow up to a 180° arc of rotation.[54,55] One or two venae comitantes and the thoracodorsal nerve accompany the artery. Atrophy of the muscle over 6–12 months usually leaves a thin subcutaneous layer. The latissimus dorsi was first used to reconstruct the Achilles and to provide soft tissue coverage in four patients in 1999. Split-thickness skin grafts covered the flaps—in one patient, the latissimus was raised as a myocutaneous unit but was de-epithelialized and folded over on itself to provide additional bulk for bony protection. The patients had fair to good plantarflexion and dorsiflexion and were walking without difficulty after 36–54 months of follow-up. This is the first description of the latissimus as a force-bearing conduit in Achilles tendon repair, and capitalizes on the observation that denervated muscle becomes fibrotic like a tendon (Fig. 23.4).[56] Others have used a myocutaneous latissimus dorsi flap to provide tendon, soft tissue fill, and skin coverage in a single stage with excellent results.[57,58] A sensate latissimus dorsi flap for Achilles tendon reconstruction has not been reported, although it would be possible to use the posterior branch of the seventh intercostal nerve.[59]

Conclusions

Repair of complex injuries to the Achilles tendon and surrounding tissue, especially in the presence of infection, requires the importation of vascularized tissue to restore function and sustain healing. Microsurgical free flaps are versatile and can replace tendon transfer procedures or the use of allograft or foreign material. Many techniques have been developed to provide adequate tendon

function, an appropriate amount of soft tissue coverage, and abundant vascularized tissue to enable rapid wound healing and to protect against infection. The particular requirements of each patient will dictate which donor site is most appropriate. In general, upper extremity donor sites are thin and useful when little soft tissue bulk is required. Lower extremity flaps with large and reliable vascular pedicles provide excellent tendon replacement but may result in excessive soft tissue bulk in the narrow region of the Achilles. Single-step procedures involving the latissimus dorsi or other muscles are applicable to a wide range of defect sizes, have low donor site morbidity, and achieve a cosmetically satisfactory contour in the posterior lower leg. Such definitive procedures can bring to a close the frustrating cycle of multiple debridements and attempts at closure of complicated wounds of the Achilles tendon.

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24
The Achilles Tendon, Ankle Equinus, and Athletes

Amol Saxena and Christopher W. Di Giovanni

Ankle equinus, with contracture of the Achilles tendon, could have a devastating effect on foot and ankle function and morphology in the long term if left unevaluated and untreated. The gastroc-soleus complex easily overpowers the other musculotendinous units and ligamentous constraints in the foot when pathologically tight. This can cause ulceration, midfoot breakdown, ankle pathology, and potential gait derangement (Fig. 24.1).[1–20] With repetitive contact between the ground and the foot, any structure responsible for abnormal loading across the foot during the gait cycle, most often the Achilles tendon, hastens foot breakdown.

In the short term, a powerful, even tight, Achilles tendon can give an athlete the extra performance burst needed for certain sports requiring strong push-off or jumping ability. However, in the long term, this advantage might also have the undesirable impact of producing wear and tear of surrounding structures, with detrimental abnormal impacts and eccentric loading. This is possibly one of the reasons why some elite and high-performance athletes may not be able to "do what they do" at a high level indefinitely.

Although the association of Achilles tendon tightness and Achilles tendinopathy has been reported in several association studies, the actual management of the contracture with lengthening and transfer procedures has not been adequately studied in athletic patients.[4,11,21–25] Also, none of the studies adequately explain why some patients with symmetrically decreased ankle dorsiflexion are asymptomatic or present with only unilateral pathology. Furthermore, activity levels of patients with Achilles tendon pathology in general have not been well documented. This chapter aims to review present concepts of ankle equinus, what procedures are available to manage it, and when and whether to consider them.

Traditionally, normal ankle range of motion is defined as 10° of ankle dorsiflexion with the foot in neutral (which is also subject to variable definitions) and the knee extended. The foot is in neutral when it is neither pronated nor supinated, based on the position of the talar head reduced on the navicular. With the knee flexed at 90° (the Silverskiold maneuver), ankle dorsiflexion should increase due to decrease of the tension of the gastrocnemius. Recently, studies have shown ranges of ankle dorsiflexion with the knee extended of 0–10°, and of more than 5° with the knee flexed in "normal" subjects.[3–5,11,19,21,26,27] Many of the studies have used no standardized reference points, foot position, or measuring devices. When assessing ankle equinus, one must also exclude confounding variables such as hamstring tightness, anterior ankle exostoses (visualized radiographically), posterior capsular contracture of the ankle and subtalar joints, and neuromuscular conditions. These associated conditions also can be causative factors in patients showing limited ankle dorsiflexion with the knee both flexed and extended.

DiGiovanni et al.[3] studied asymptomatic individuals and patients who were symptomatic due to forefoot/midfoot pathology. They used a sophisticated device, the "Equinometer," and a consistent dorsiflexion torque (10 newton-meters). Their reference points were the distal fibula and the second metatarsal axis, as a reliable

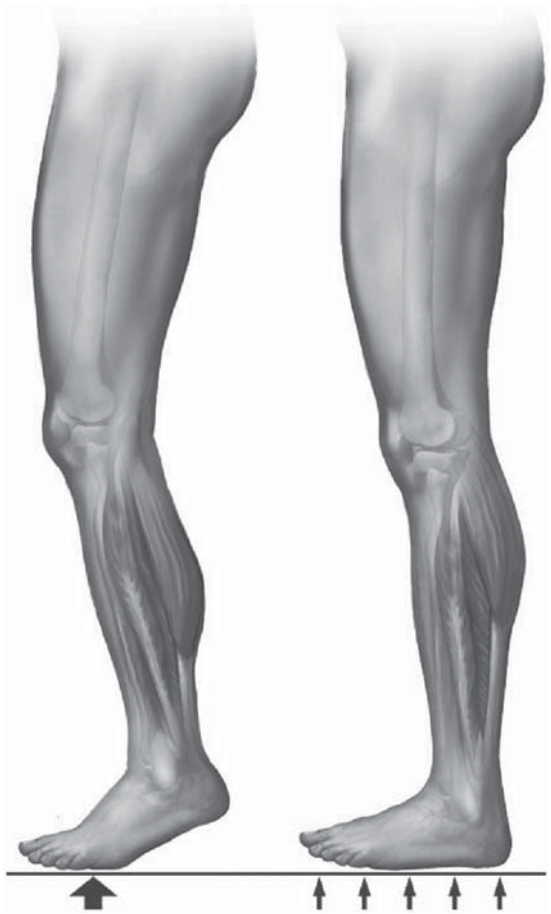


Figure 24.1. Artist's depiction of the effect of a tight gastrocnemius on the foot.

indicator for the central axis of rotation of the ankle joint. Their study showed that those subjects with less than 5° of dorsiflexion with the knee extended and less than 10° of dorsiflexion with the knee flexed were statistically more likely to be diagnosed with pathological equinus. The authors did not stratify their data according to athletic ability or activity level of their subjects. DiGiovanni et al. labeled those with limitations only with the knee extended as having gastrocnemius tightness (Fig. 24.2A, B), and those with limited dorsiflexion with the knee both flexed and extended as having Achilles tendon tightness.[3]

Saxena and Kim, in 40 adolescent athletes (average age 15 years) with no history of ankle pathology, found that the average ankle dorsiflexion was 0° with the knee extended, and 5° with the knee flexed.[28] These results may indicate that some degree of tightness of equinus may be beneficial in sports, particularly when athletes are encouraged to run on their forefoot.

Tabrizi et al. studied children who sustained a unilateral lower limb injury, measuring the contralateral limb's ankle dorsiflexion without placing force on the forefoot, and keeping the heel in varus. They used their patients who sustained upper extremity injuries as a control. They found 5.7° of dorsiflexion compared with 12.8° in the uninjured group. With the knee in flexion, the values were 11.2° and 21.5°, respectively. The interobserver measurement error was approximately 2.5°.[19] The subjects in this study were younger than in Saxena and Kim's. It also may be that the patients (in Tabrizi et al.'s study) sustaining lower extremity injuries were in sports in which less ankle dorsiflexion was beneficial. Those with upper extremity injury may not participate in these types of sports.

Using DiGiovanni et al.'s definition, all the subjects in Saxena and Kim's cohort would be defined as having Achilles tendon and gastrocnemius equinus/tightness. Tabrizi et al.'s cohort would also have borderline gastrocnemius equinus. This may pose the question whether these individuals, based on meeting arbitrary criteria defining them as having abnormally tight gastroc-soleus complexes, should undergo a lengthening procedure. However, it is difficult to find a study on neurologically normal adolescents and children undergoing isolated procedures for ankle equinus.

Kaufman et al. studied 449 Navy Seal recruits with an average age of 22.5 years. They found limitations in ankle dorsiflexion with the knee extended in those recruits with Achilles tendinopathy (measured as <11.5°) together with increased hindfoot inversion.[11] Unfortunately, no longitudinal studies have been conducted in athletic individuals to ascertain whether they are likely to develop long-term Achilles tendinopathy based on their limited ankle dorsiflexion. This relationship, however, has been strongly suggested in the diabetic population, as these patients are more commonly studied and almost always have Achilles tendon tightness as part of their disease process. In diabetic patients, however, pathology resulting from a tight Achilles tendon becomes more easily manifest because the patients are often neuropathic. As opposed to elite athletes

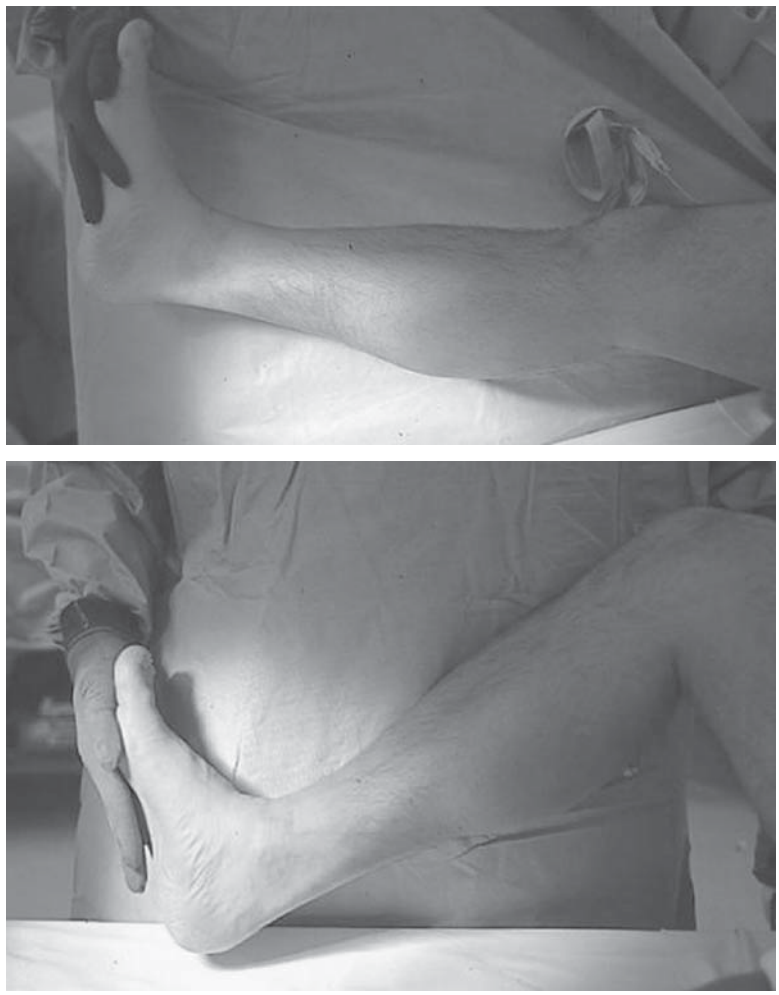


Figure 24.2. (**A**) Silverskiold's test. With the knee extended, ankle dorsiflexion does not allow the foot get to a 90° relationship to the leg. (**B**) With the knee bent, the foot is able to achieve 10° of dorsiflexion with respect to the leg.

with normal neurologic function, diabetic patients cannot identify nor protect themselves from this chronic, repetitive wear and tear process on surrounding structures (skin, ligaments, tendons, bone, joints). As a result, ulcers, Charcot breakdown, deformity, and instability are far more common in diabetic patients.[1–4,6,9,12,13]

Asymmetrical ankle dorsiflexion does require evaluation.[19] For athletes to be able to return to full function, ankle dorsiflexions should be within 5° of each other, or nearly symmetric.[24,29–32] Perhaps the best reason to consider treatment for ankle equinus is post-traumatic contracture.

Initial treatment of ankle equinus has traditionally been stretching (Fig. 24.3A, B). Although it remains unclear whether a chronic stretching regimen can effectively lengthen the gastroc-soleus complex, Grady and Saxena found an approximately 2° increase in ankle dorsiflexion over six months when subjects engaged in a stretching program. Their cohort consisted of nonathletic subjects, with an average age of 25 years, with initial dorsiflexion of 3° and 9° with the knee extended and flexed, respectively.[21] Stretching of the Achilles tendon in athletes is considered beneficial by many authors, but few outcome studies have been performed. Contractures do not generally occur through the tendons themselves, but rather within the muscle belly.[3] This is true for the Achilles tendon and gastroc-soleus complex

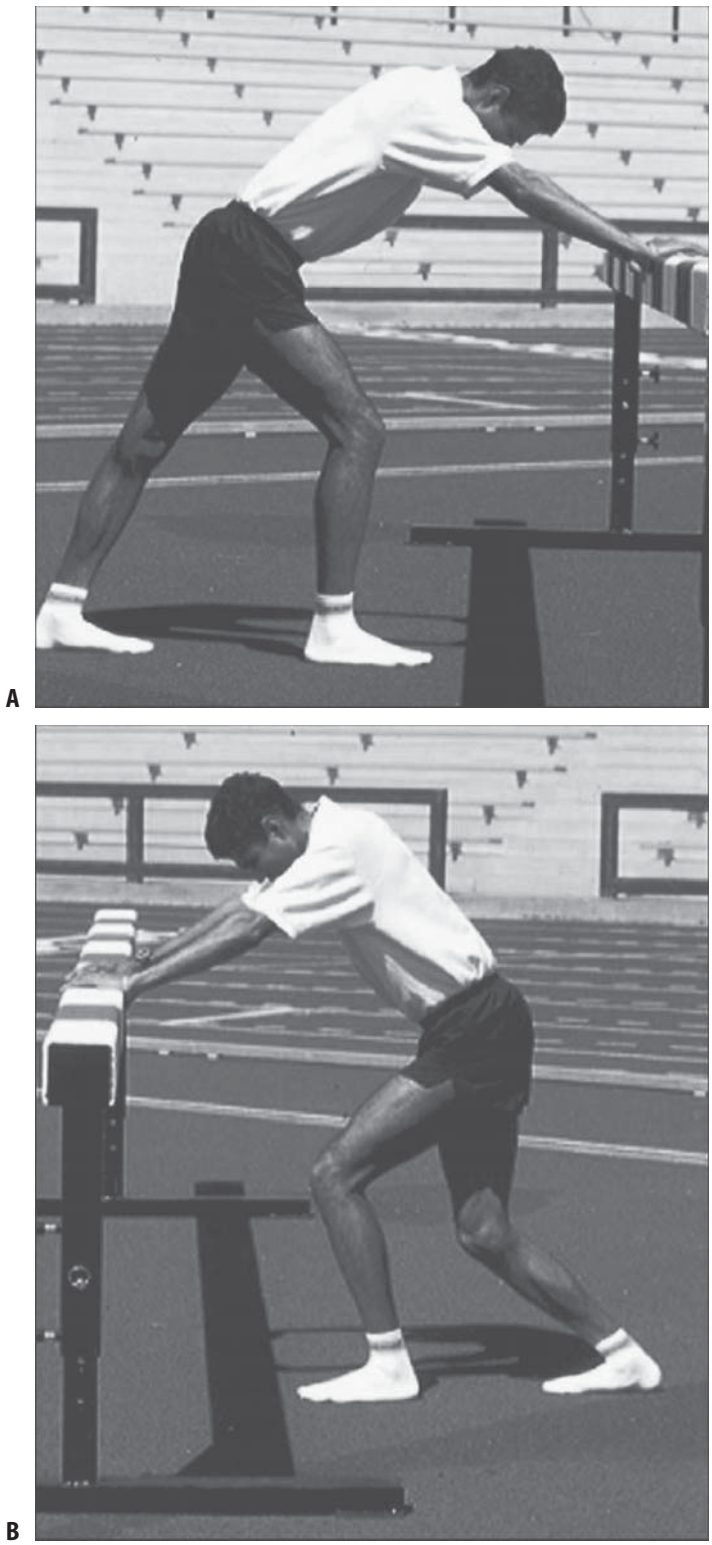


Figure 24.3. (**A**) Stretching with the knee straight creates tension on the gastrocnemius. (**B**) Stretching with the knee bent creates tension on the soleus and deep posterior muscle compartment.

as well. Commonly prescribed stretching regimens designed long term to "eliminate contracture" are probably more effective at preventing further contracture than at decreasing the tightness already present.

Eccentric strengthening for up to one year is beneficial in reducing pain and allows for increased function in patients with Achilles tendinopathy.[33] Strengthening of the anterior leg muscles may also improve ankle dorsiflexion.

Elongation of the Achilles tendon produces disruption at 3% and complete loss of integrity at 8%.[22] Stretch likely occurs mostly in the muscle, and only a small amount in the tendon.[22,23] Other modalities of nonsurgical lengthening have been casting and immobilization, though this has been predominantly studied in diabetic populations.[12]

Recently, injections with botulinum toxin A have become popular for cerebral palsy patients. A large multicenter clinical trial showed improvement in gait after botulinum injections, but the gains in ankle dorsiflexion were not reported. To date, none of the studies using botulinum injections have included athletic individuals or neurologically normal patients.[34]

Surgical lengthening of the Achilles tendon complex was first reported by Delpech in 1816.[35] William Little, a physician with infantile paralysis and equinovarus deformity, had an Achilles tenotomy performed by Stromeyer and became a proponent of this procedure for Achilles tendon contracture.[5,36] Throughout the twentieth century, other lengthening procedures were described. Variations of Achilles tenotomies were described by Hatt and Lampier, and Hoke and Sgarlato.[4,18,37] Hatt and Lampier described a triple hemisection of the Achilles tendon, attributed to Hoke. Two medial and one lateral evenly interspaced hemitenotomies were performed (Fig. 24.4).[4] Sgarlato described an open Z-plasty, distally severing the anterior two-thirds of the lateral tendon and proximally severing the posterior two-thirds of the medial Achilles tendon.[18] Posterior ankle and subtalar releases, as performed in deformities such as clubfoot, may also need to be considered as adjunctive procedures in patients with severe equinus deformity, as well as anterior ankle arthroplasty for patients with osseous equinus.

Hansen advocates open gastrocnemius recession for gastrocnemius tightness, percutaneous Achilles tendon lengthening for Achilles tendon tightness, and open Achilles tendon lengthening when precise lengthening is desired or when previous surgery has been performed on the Achilles tendon and a complete rupture of the Achilles tendon using percutaneous lengthening is a potentially greater risk.[7] Hansen's percutaneous technique is recommended for diabetics and elderly patients in which wound healing can be an

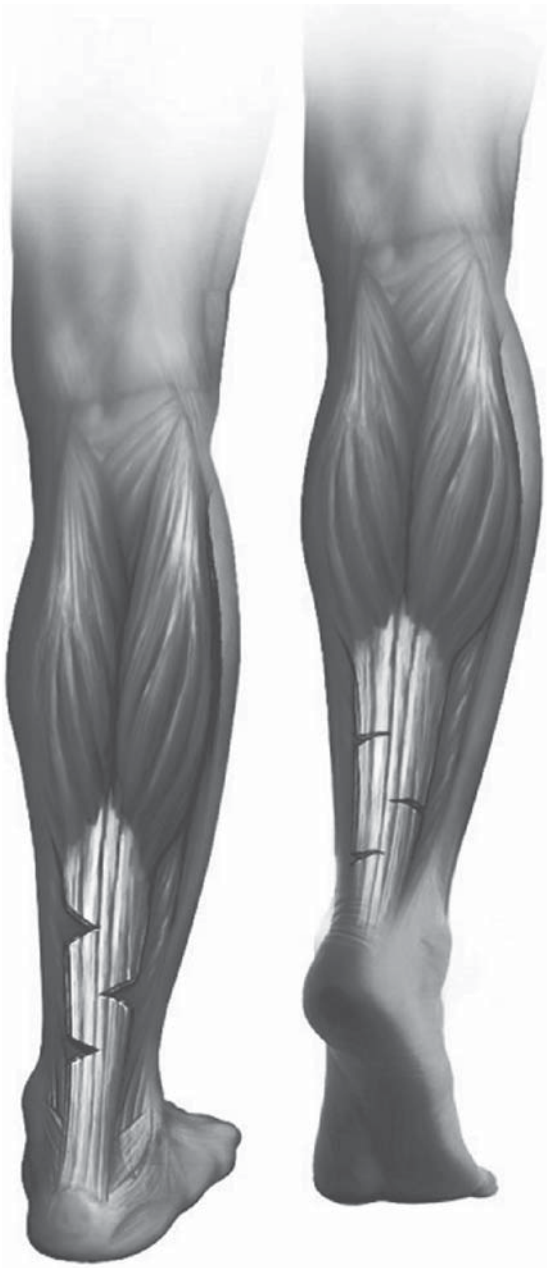


Figure 24.4. Triple hemisection of Hoke; two medial interspaced by one lateral hemisections are made in the Achilles tendon.

issue. The first incision (distal) is made 1 cm proximal to the Achilles tendon insertion, and, applying a dorsiflexion force, the anterolateral portion of the tendon is divided. A second more proximal incision is made about 3–4 cm proximal to the first one. A vertical midline incision is made, transecting the medial portion of the Achilles tendon (Fig. 24.5A–C). An open Achilles tendon lengthening may be performed with a sagittally based "Z-lengthening" incision. A dorsiflexion force is

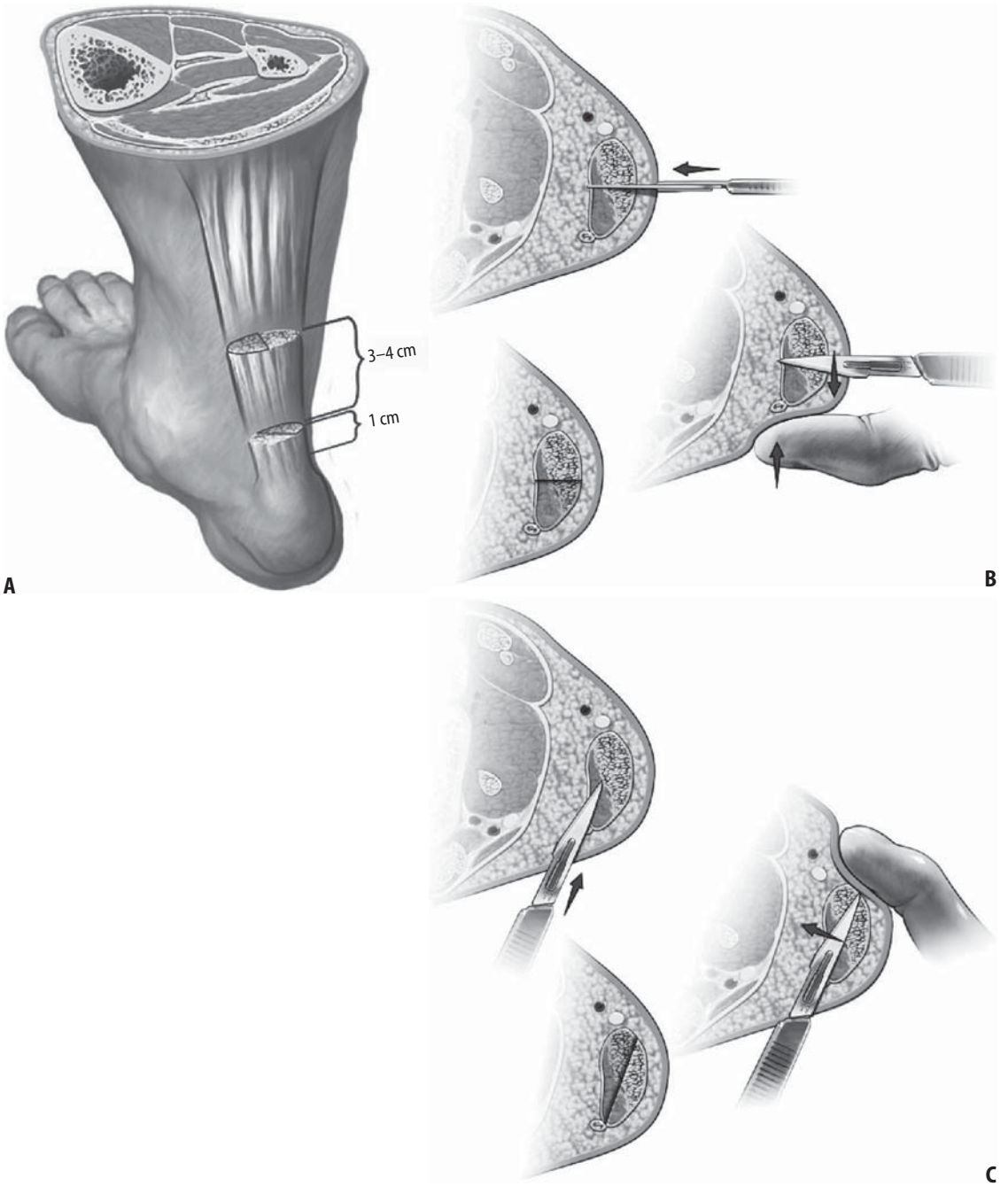


Figure 24.5. (**A**) Percutaneous Achilles tenotomy. (**B**) Distal incision for percutaneous Achilles tenotomy. (**C**) Proximal incision for percutaneous Achilles tenotomy.

applied to bring the foot out of equinus to the desired amount (Fig. 24.6). Hansen and others, over a period of almost 100 years, have recommended that Achilles tendon or gastrocnemius lengthening be performed for reconstructive foot procedures.[4,5,7,10,16–18,20,37] Unfortunately, none of these studies document patients' activity level such as participation in sports or even the ability to propulse on their toes, which would be an issue for athletes.

Postoperative regimens for Achilles tendon lengthening vary according to the procedure undertaken. However, generally the foot and ankle are protected with some form of immobilization for 4–6 weeks. Some authors allow diabetic patients to walk without any splint, and others state that protection with a below-knee cast depends on whether other concomitant procedures are performed.[1,2,4,5,9,13,16]

Though tenotomy is commonly performed on the Achilles tendon, some have noted that excessive weakening or "calcaneal" deformity can occur. Delp and Zajac advised against Achilles tendon lengthening for patients with isolated gastrocnemius contracture due to excessive weakening of propulsion. One centimeter of lengthening reduces propulsive forces by almost 30%,[38] a significant amount for athletic individuals.

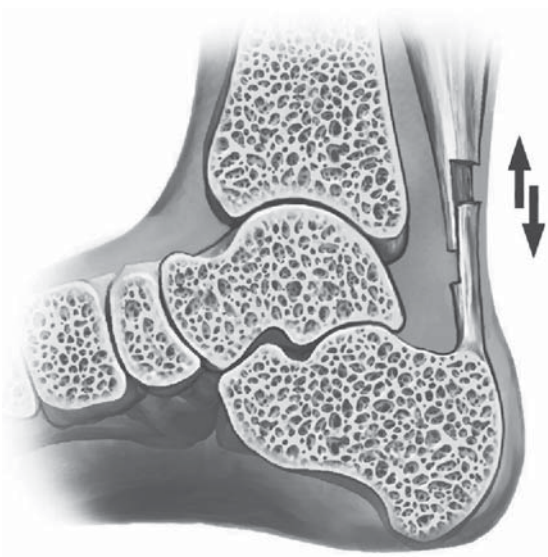
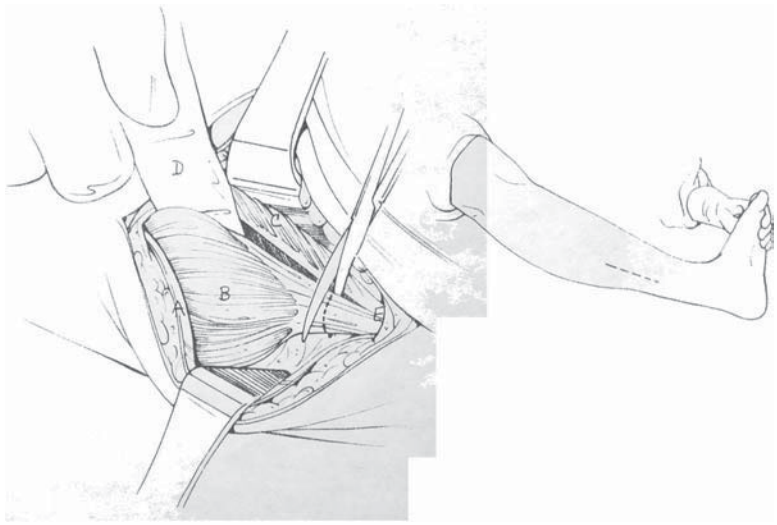


Figure 24.6. Open Achilles tendon lengthening.

Other procedures to reduce Achilles tendon contracture have been described. Strayer described an open gastrocnemius recession or tenotomy as a variation of Vulpius and Stoffel's distal gastrocnemius recession that avoided the main body of the Achilles tendon, as well as "tongue-and-groove" slide techniques.[4,5,39–41] In Strayer's procedure, the distal portion of the gastrocnemius aponeurosis is transected (Fig. 24.7A). A posterior midline incision is made, and the sural nerve is identified and protected (Fig. 24.7B). The medial and lateral margins of the gastrocnemius aponeurosis are identified and then transected. Improvement in ankle dorsiflexion is then noted (Fig. 24.7C). This procedure is occasionally performed in patients with neglected Achilles tendon ruptures to span a tissue loss of up to five centimeters. Studies on athletic individuals undergoing an open gastrocnemius recession with concomitant Achilles tendon repair are lacking.

Some of the largest studies on Achilles tendon rupture repair and surgical management for chronic tendinopathy fail to yield any authors advocating a lengthening procedure at the time of surgery.[24,29–32,42] However, if a tight Achilles tendon is considered a potential contributing factor to the chronic tendinopathy or tear of the Achilles, consideration may be given to a more proximal gastrocnemius recession.[43] This technique can facilitate easier reapproximation or repair of the Achilles tendon at the site of rupture, and results in little loss of Achilles tendon strength clinically. Approximately one centimeter of length can be gained with a proximal gastrocnemius recession in such patients, which can be helpful to avoid repair under tension in the event of tissue loss after debridement or a chronic defect. This situation, however, is generally not seen in patients with an acute tear of the Achilles tendon.

For acute tendon repair, one is advised to maintain the foot in a gravity equinus position, only gradually dorsiflexed to neutral in the following weeks postoperative.[44–45] In fact, an overlengthened position can cause deficits in athletic patients, and one paper describes surgical shortening for this situation. Cannon and Hackney recently described good results with surgical shortening on five athletic and active patients with dysfunctional Achilles tendons with prior treatment for rupture. The average age of their patients was 46



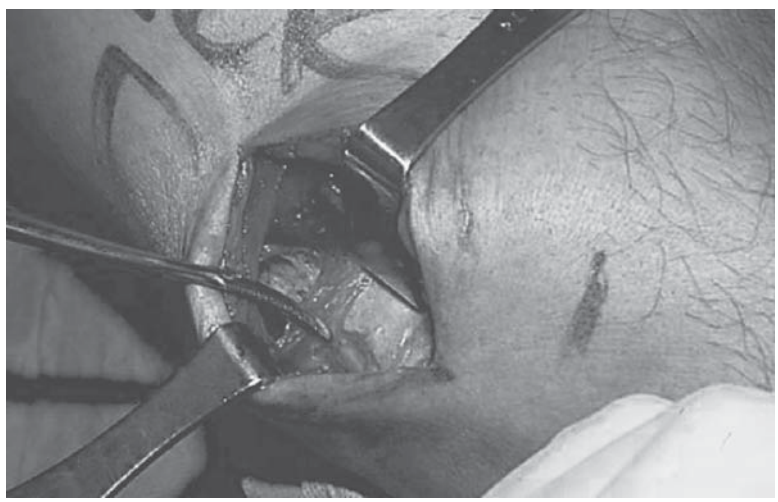


Figure 24.7. (**A**) Artist's depiction of open gastrocnemius recession (Strayer). (**B**) Intraoperative view of sural nerve during an open gastrocnemius recession. (**C**) Postoperative view of a patient; note incision length.

years, the tendon was shortened approximately 1 cm, and patients were protected from dorsiflexion postoperatively. They cite only a few papers dealing with this subject.[46] Alternatively, a deep posterior compartment release can be added to this procedure to decrease tension of the overlying paratenon or skin repair when necessary, and can also theoretically improve vascular inflow to the repair from the well-vascularized FHL muscle belly immediately adjacent when tenodesed to the Achilles tendon. To our knowledge, neither Achilles tendon lengthening nor gastrocnemius recession have been described in the context of surgery for chronic Achilles tendinopathy. Recently, Vulpiani et al. reported on 76 patients with 13-year follow-up for chronic Achilles tendinopathy, and do not mention any type of lengthening.[42] Another, larger study of 91 surgeries with average 4-year follow-up also does not describe the need for lengthening in active and athletic individuals. Furthermore, postoperatively, patients are kept in an equinus position for variable periods, and some even maintain a heel raise in the shoe for several months.[29]

Similar to the undocumented prevalence of contracture of the Achilles tendon in athletes, Achilles tendon lengthening for athletic individuals has also not been studied. As a result, this procedure is understandably uncommon in athletes.

Gastrocnemius recession essentially creates a surgically induced gastrocnemius tear.[47,48] This injury often is incurred without long-term sequelae, as treatment is supportive with rest, elevation, and rehabilitation. In this regard, gastrocnemius recession would appear to be better tolerated than an Achilles tendon lengthening by athletic patients. Indeed, we suggest that Achilles tendon lengthening be used with much greater caution than the gastrocnemius recession in these patients. Its direct effect on the muscle–tendon unit is probably much greater, and therefore its impact on direct activity of the loaded Achilles tendon can be expected to be similarly high.

Studies report the results of gastrocnemius recession and the amount of ankle dorsiflexion achieved. The 15 patients undergoing an open gastrocnemius recession had an increase of 18° two months after the procedure.[15] The patients are positioned prone, a 6–10 centimeter midline incision is made, and the medial and lateral aspects of the gastrocnemius fascia and aponeurosis distal to the muscle belly are transected. The relevant anatomy for the open technique of Strayer from a medial approach was recently studied by Pinney et al., who used a 7-cm medial incision.[40] The sural nerve was located in the superficial fascia in 42.5% of the legs operated, and deep to the fascia in the remaining 57.5%. The release site was on average 18 mm distal to the distal muscle belly of the gastrocnemius, and the gapping of the release site averaged 3 cm. Although they did not report actual data on appearance, they state, "Cosmesis of the incision can also be compromised by tethering of the skin to the underlying tissue," and report that this is a "relatively frequent complication." They also relate that sural nerve injury can occur by direct trauma or excessive stretch, but do not report the actual rate of this complication. Webb et al. noted the sural nerve courses over the proximal aspect of the Achilles tendon from lateral to medial, and surgical incisions should take this into account.[49] In general, gastrocnemius recession, as compared with Achilles tendon lengthening, may result in fewer complications such as calcaneal deformity, but the larger incision and nerve injury may be a drawback.

Endoscopic techniques include gastrocnemius recession, avoiding larger incisions, and visualizing the sural nerve.[16,50–52] Saxena and Widtfeldt reported on 18 patients undergoing endoscopic gastrocnemius recession. Their patients' increase in ankle dorsiflexion with the knee extended statistically improved from −8.7° to 2.6° after a year ($P < .00001$). This is generally performed supine with the heel on a bulky towel to allow passage of instruments (Fig. 24.8). Although activity levels were not reported, patients were able to perform a single-leg heel-raise on average at 13 weeks. They generally used a two-portal technique with the patients supine and the heel on a sterile roll, a 4.0-mm endoscope, and a cannulated endosopic blade to transect the gastrocnemius in a medial-to-lateral direction.[16]

Using endoscopic gastrocnemius recession, the medial incision is produced first, inferior to the medial gastrocnemius muscle belly and posterior to the great saphenous vein and saphenous nerve. A hemostat and then a fascial elevator are used to

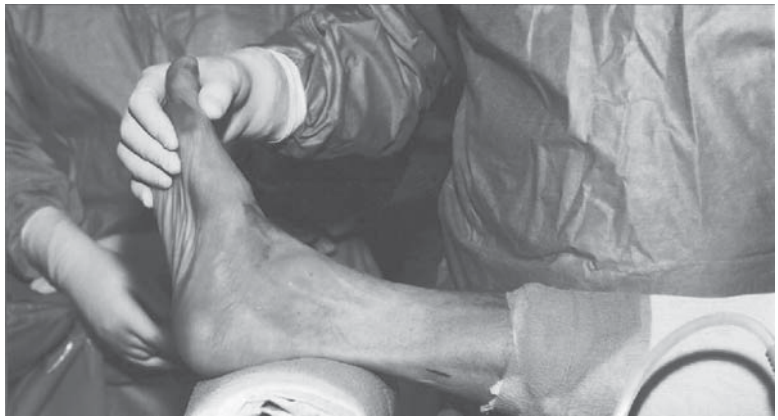


Figure 24.8. Actual patient before endoscopic gastrocnemius recession.

create a channel deep to the subcutaneous tissue directly posterior to the gastrocnemius aponeurosis (Fig. 24.9). A cannula is introduced with an obturator, which then is replaced with a 30° 4.0-mm endoscope. The aponeurosis is visualized above (Fig. 24.10A–C). The slotted cannula then should be rotated posteriorly to identify the sural nerve, which is generally located approximately 1 cm from the lateral border of the gastrocnemius (Fig. 24.11A, B). After the nerve has been protected posteriorly by the cannula, a lateral portal can be created by transillumination and tenting of the skin over the cannula. A small suction-tip device can be placed within the cannula to improve visualization, which is helpful during transection (Fig. 24.12). The endoscope is then temporarily removed to apply a cannulated knife (AM Surgical, Smithtown, NY USA). The knife/scope assembly is carefully inserted into the cannula, temporarily rotating the opening to align with the skin incision (Fig. 24.13A, B). With the knife/scope repositioned perpendicular to the gastrocnemius, the aponeurosis is transected from medial to lateral while dorsiflexing the foot. Hemorrhage

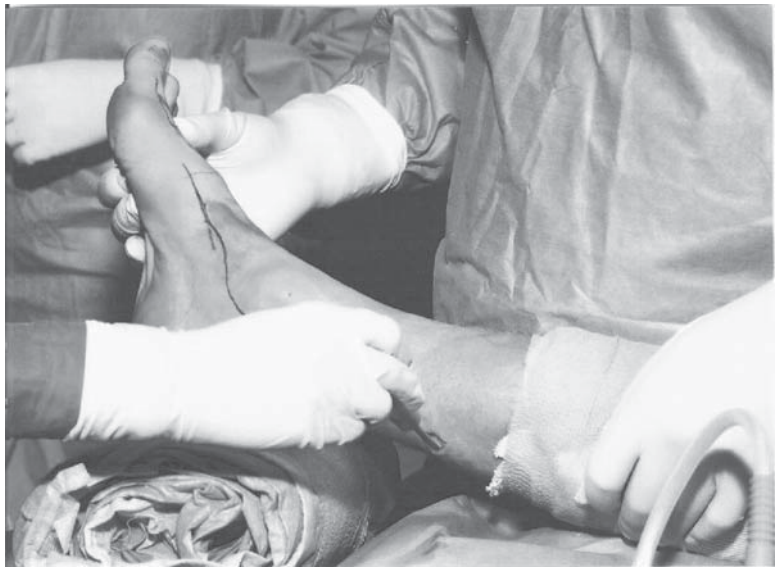


Figure 24.9. Creating a fascial pathway with elevator for the cannula.

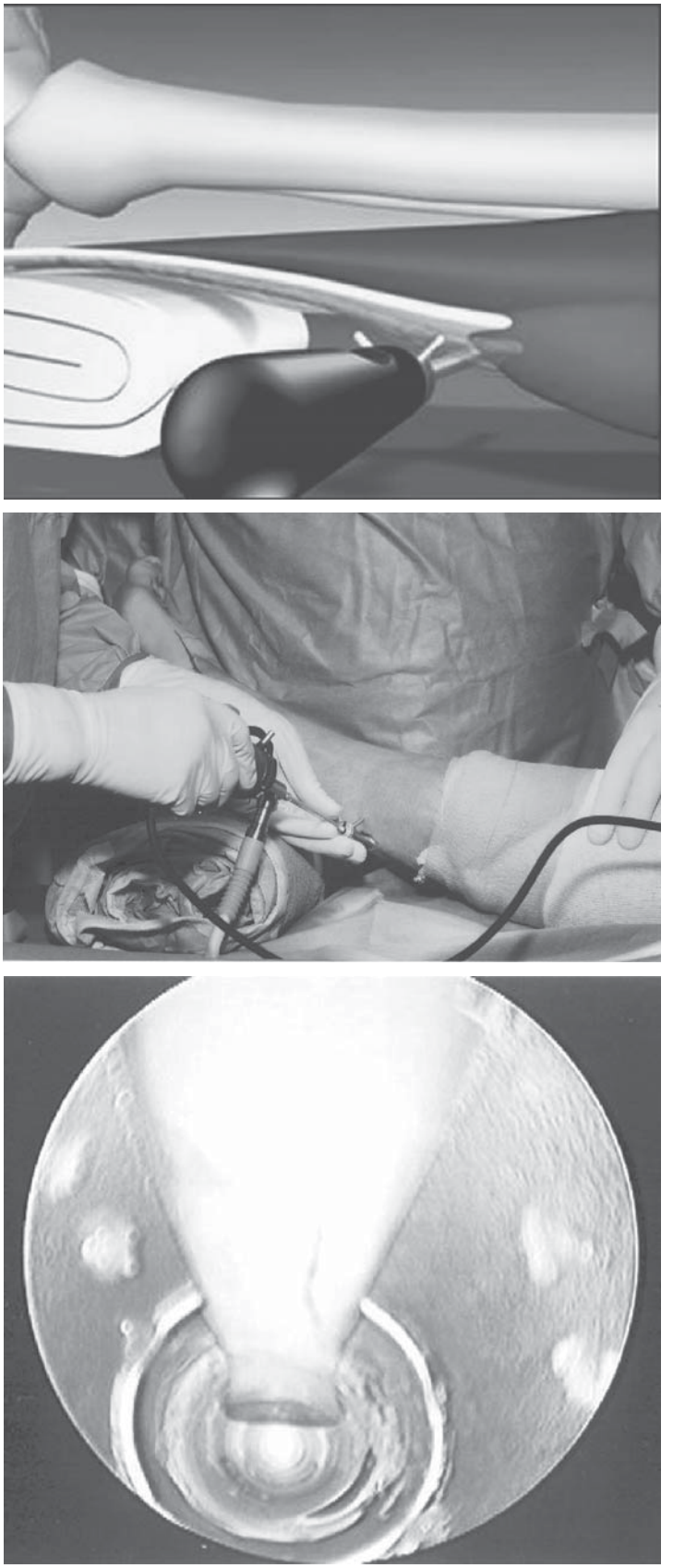
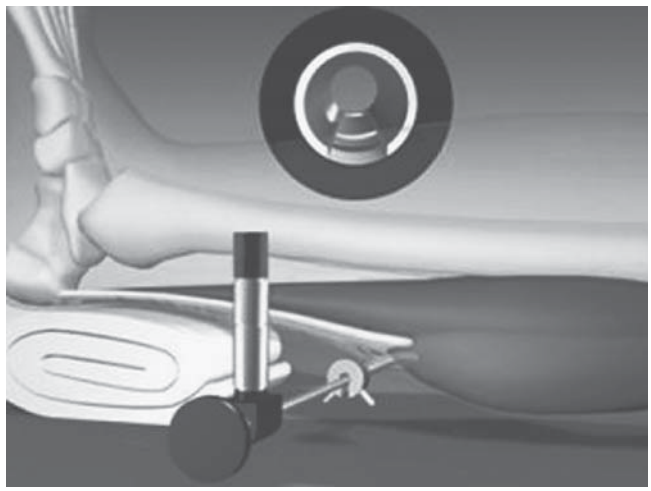


Figure 24.10. (**A**) Schematic of insertion of obturator/cannula assembly. (**B**) Obturator removed, and replaced by 4-mm endoscope. (**C**) Endoscopic view of gastrocnemius aponeurosis/fascia.



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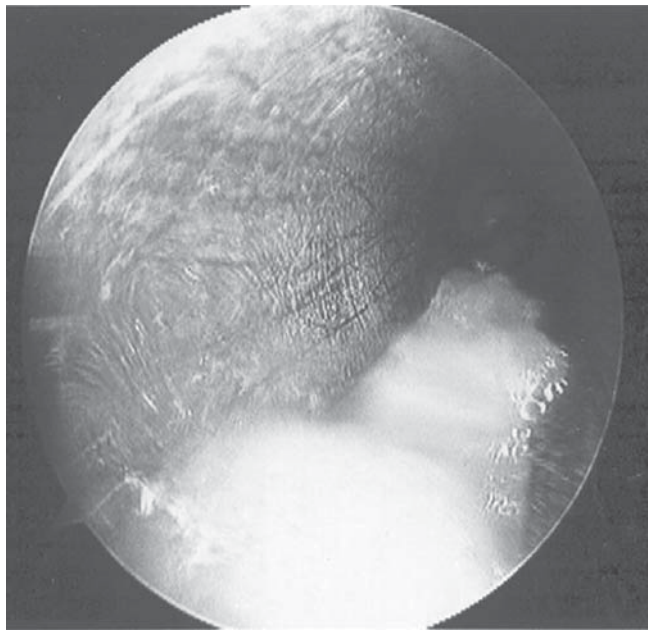


B

Figure 24.11. (**A**) Schematic of rotating the endoscope 180° posteriorly to visualize the neurovasculature. (**B**) Endoscopic view of the sural nerve and adjacent vein.

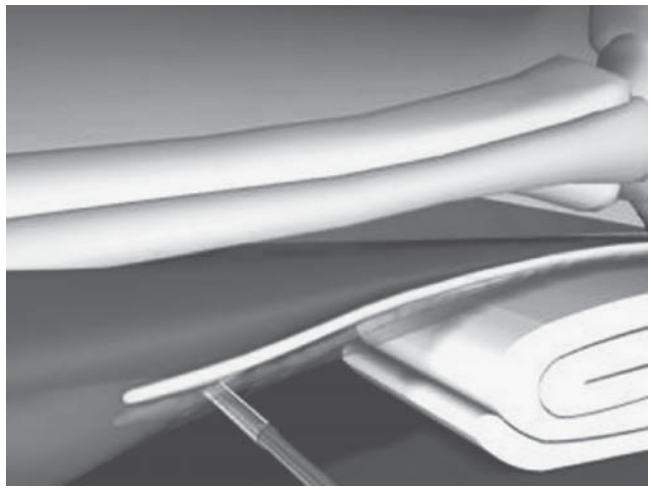


Figure 24.12. Transillumination to create a lateral portal to introduce suction.

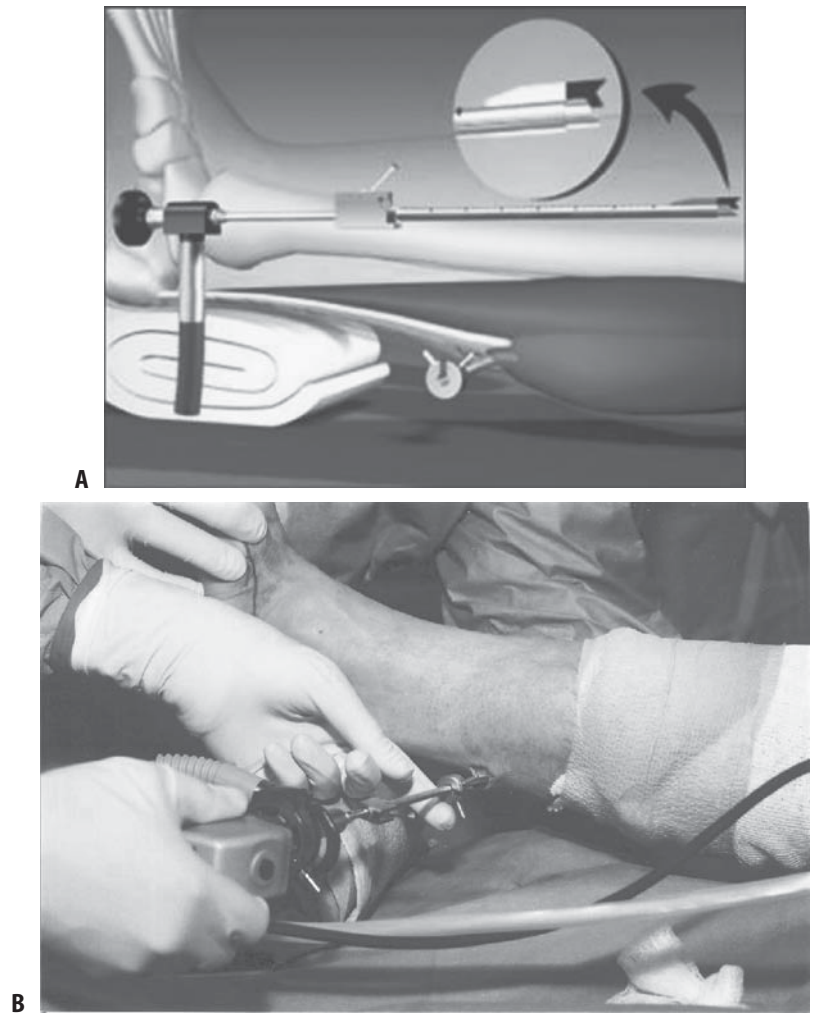


Figure 24.13. (**A**) Application of cannulated knife onto the endoscope. (**B**) Reinsertion of endoscope with knife into the cannula, requires a temporary rotation of 90° (in line with the incision). Additional protection with a small retractor can be beneficial while inserting.

may occur when transecting the intramuscular septae, making the suction device useful. Endoscopic confirmation of transection and visualization of exposed muscle is needed (Fig. 24.14A–D). Dorsiflexion should improve by at least 10°. The tendon of plantaris may also have to be transected (Fig. 24.15A, B). Skin sutures of 3–0 nylon are used to close the skin incisions (Fig. 24.16). To avoid tenting of the skin due to the exposed muscle tissue, the foot can be gradually mobilized out of the equinus position. Patients are maintained in a below-knee cast boot for at least 4 weeks postoperatively at 90°; longer immobilization for up to 12 weeks can be necessary if other procedures are associated. Muscle relaxants are sometimes needed postoperatively. Physical therapy to decrease fibrosis at the surgical sites and improve strength and gait is used. If endoscopic gastrocnemius recession is performed in isolation, physical therapy is started at two weeks. Otherwise, physical therapy is delayed according to additional procedures. In any case, patients are advised to massage the surgical sites as soon as they can access them.

Endoscopic gastrocnemius recession may produce better cosmesis and allows visualization and protection of the sural nerve. Researching endoscopic techniques on cadavers, Tashjian et al.

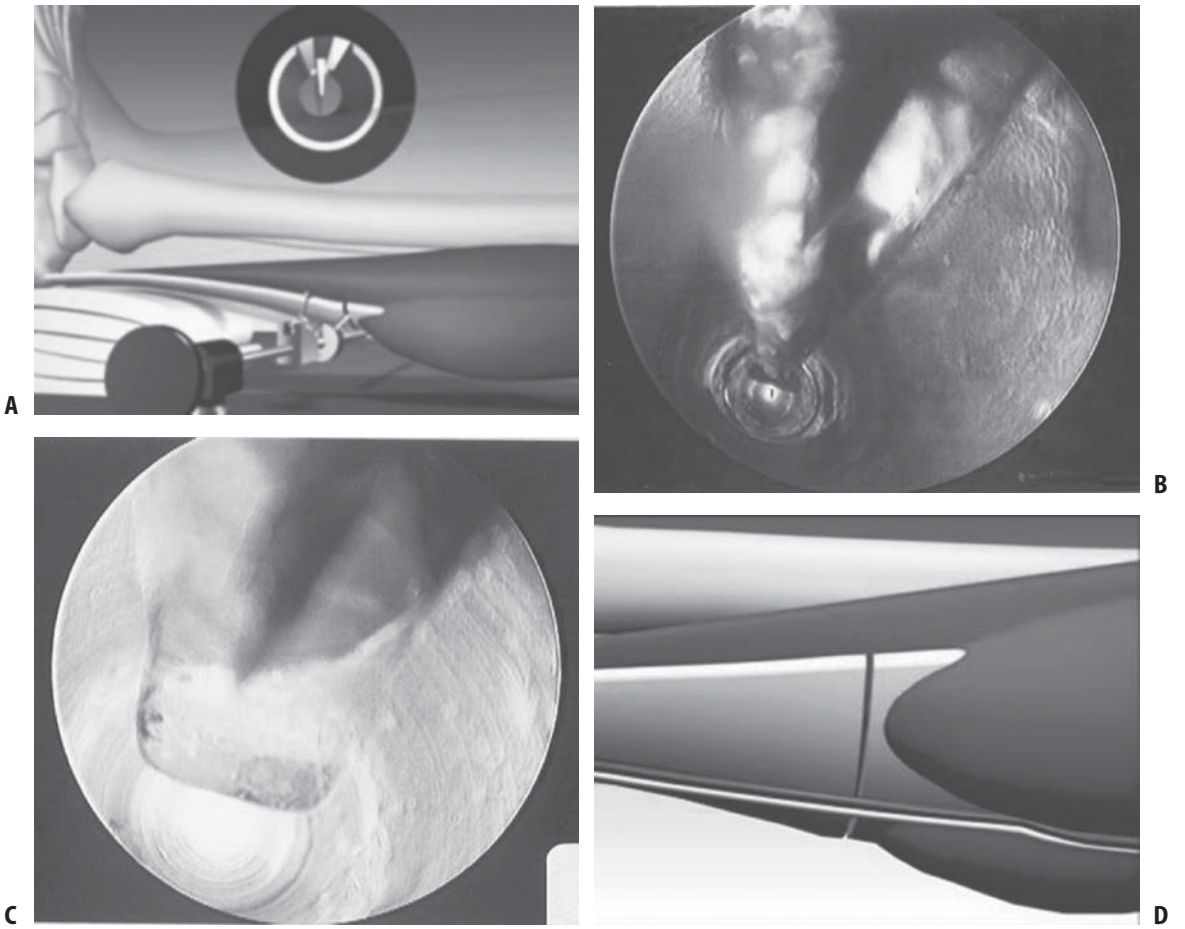
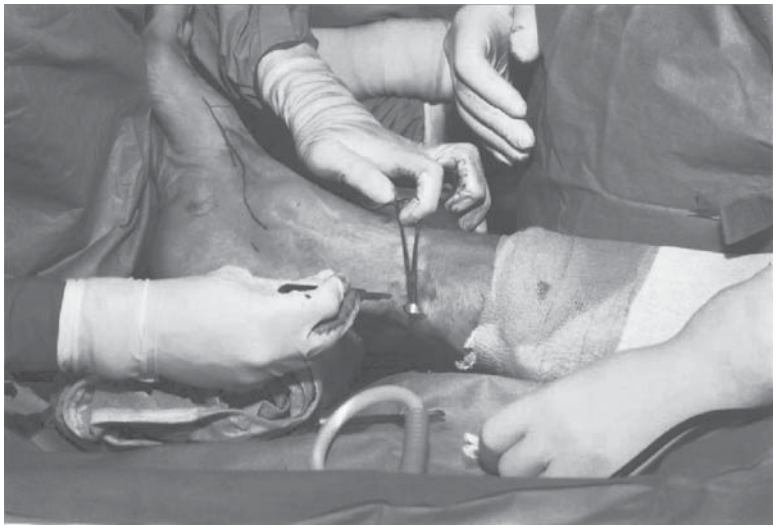


Figure 24.14. (**A**) Schematic of endoscope/knife rotated back to perpendicular to the gastrocnemius to initiate transection. (**B**) Endoscopic view of initial transection. The foot is being dorsiflexed during this maneuver to create tension on the gastrocnemius. (**C**) Additional transaction. Note further muscle exposure. (**D**) Schematic of preservation of neurovascular structures: the cannula is anterior and therefore protects them.

found that the sural nerve courses within 1 cm medial to the lateral border, or about 20% of the total width of the gastrocnemius aponeurosis.[52] When a medial percutaneous gastrocnemius approach is employed, this nerve is subject to injury. A larger medial incision allows visualization of the nerve.[40] Observation and protection of this nerve during open approaches or endoscopy should reduce neural injury, which could be more likely with percutaneous gastrocnemius recession techniques.[50]

Another procedure to correct an equinus deformity is anterior advancement of the Achilles tendon to the dorsum of the calcaneus.[52] Posterior lengthening of the Achilles tendon and gastrocnemius in neurologically impaired patients results in a high rate of recurrence of the equines deformity.[53,54] Therefore, anterior advancement has been recommended for this type of patient, and particularly for patients with spastic conditions.[4,5,55,56] The entire Achilles tendon is transected at its insertion and reinserted on the superior portion of the calcaneus proximal to the posterior subtalar joint (Fig. 24.17). It is unlikely that this procedure is performed in athletic individuals. However, with the increased popularity of the Special Olympics and Paralympics, it is possible that these athletes may have undergone anterior advancement of their Achilles tendon to produce a plantigrade foot.



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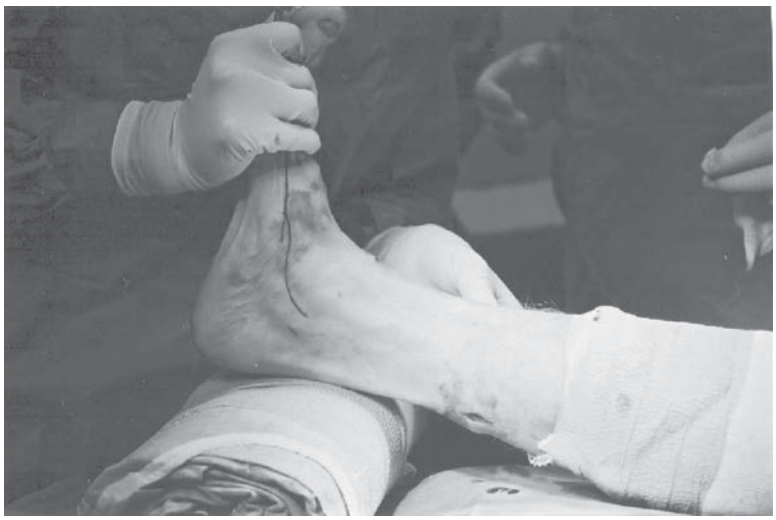

B

Figure 24.15. (**A**) Open transection of the plantaris tendon improves dorsiflexion. (**B**) Dorsiflexion should improve by at least 10 degrees.

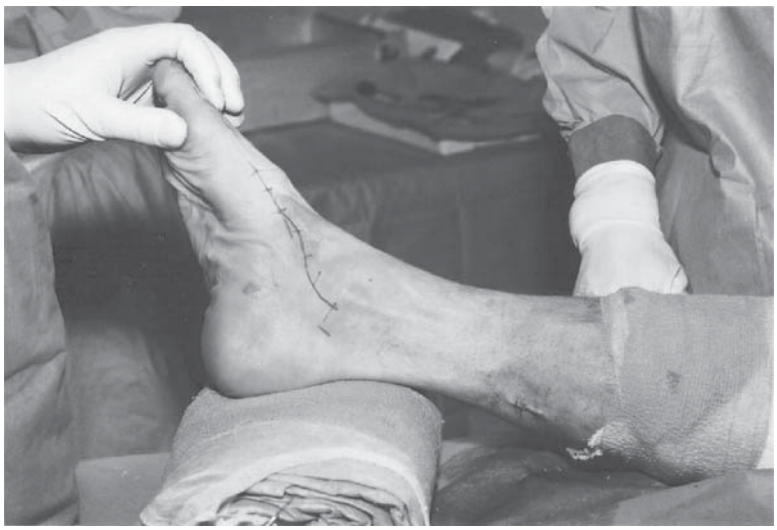

Figure 24.16. Skin incision after closure.

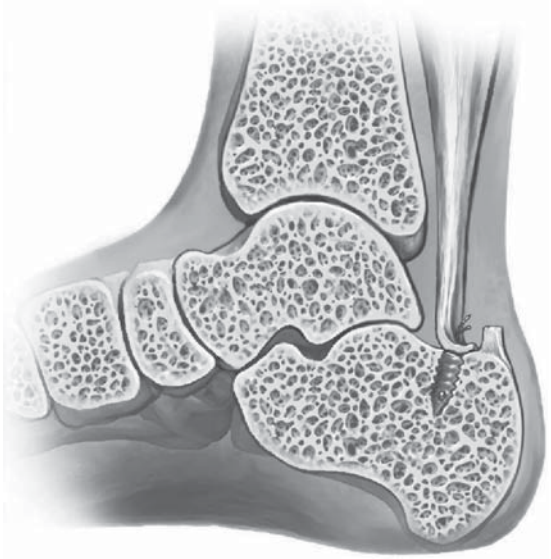

Figure 24.17. Murphy's procedure with anterior advancement of the Achilles tendon for spastic conditions.

Generally, Achilles tendon or gastrocnemius lengthening should be reserved for patients requiring significant foot reconstruction related to a tight gastroc-soleus complex, and for those needing to avoid forefoot ulceration such as diabetics. Diabetic patients benefit from Achilles tendon lengthening, probably due to their different metabolic state and altered configuration of collagen crosslinking, but can be susceptible to overlengthening. Perhaps, if these patients are athletic or are motivated to exercise to help control their diabetes, they could be studied for their altered biomechanics, and subsequent long-term benefits of the procedure.

Based on a current lack of data unequivocally linking a tight Achilles tendon to long-term pathology of the foot and ankle, it remains difficult to justify any lengthening of this structure in asymptomatic athletes or neurologically normal individuals without foot pathology. Future studies of such patient populations will likely shed light on this subject, and longitudinal evaluation of these groups may change these recommendations, particularly if any long-term pathological effects of a tight Achilles tendon left unchecked are not outweighed by any "advantages" of that tightness in the preceding years.

Repair of an Achilles tendon rupture and avulsion is typically performed with the foot in a neutral or equinus position. Scientific in vivo studies have not been performed with the Achilles tendon dorsiflexed. Therefore, until this is done, with long-term results sufficiently known, athletically active patients should proceed with caution when contemplating having an Achilles tendon positioned in a dorsiflexed position. Gastrocnemius recession is occasionally performed in conjunction with Achilles tendon rupture repair. Again, the ankle is generally maintained in an equinus position in this situation during surgery and postoperatively.

In summary, significantly asymmetric posttraumatic contracture is a consideration for athletic patients to undergo posterior lengthening, although only anecdotal reports are available. Isolated gastrocnemius recession for Achilles tendinopathy has not been documented. This may cause one to ponder whether the athletic foot may actually benefit from having an equinus position, and hopefully this will be better assessed in the future.

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25
Achilles Tendon Involvement in Pediatric Conditions

Ramanathan Natarajan and W.J. Ribbans

Introduction

The Achilles tendon is involved in a variety of pediatric conditions. Intrinsic tendon abnormalities such as tendinopathy and rupture are rare in the pediatric population, but the tendon is often secondarily involved in some common pediatric conditions that can be categorized under the following headings:

- Congenital
- Developmental
- Traumatic
- Neurological

Congenital Conditions

Contracture of the Achilles tendon is a constant feature of congenital conditions such as clubfoot and congenital vertical talus. The tendon can fully regenerate after complete tenotomies in infancy. This unique property is utilized in the currently popular nonoperative management programs for congenital clubfoot.[1]

Developmental Conditions

Achilles tendon shortening is also a feature of developmental conditions such as flatfoot, and it is still unclear whether the tightness of Achilles tendon in this condition is a primary or secondary abnormality. Another developmental condition associated with dynamic or static tightness of the Achilles tendon leading to an abnormal gait is idiopathic toe walking (ITW).[2]

Traumatic Conditions

A common painful condition in children causing heel pain is the so-called calcaneal apophysitis described by Sever in 1918, associated with overuse. Insertional and noninsertional Achilles tendon disorders are not infrequently encountered in adolescents, often from overuse.[3]

Neurological Conditions

Achilles tendon shortening or weakness is a feature of many neurological conditions affecting the central or peripheral nervous system (cerebral palsy, poliomyelitis, spina bifida, and hereditary neuropathies) and muscles (muscular dystrophy). Achilles tendon spasticity, weakness, or contracture in these conditions leads to gait abnormalities. Understanding the role played by the triceps surae in the unique coupling of ankle and knee motion is essential in the management of gait abnormalities from tightness of the gastro-soleus–Achilles tendon complex.

Lengthening of Tendo Achillis

As tightness of the Achilles tendon features in many pediatric conditions, its lengthening is commonly undertaken. Excessive lengthening of the tendon and the resulting weakness of the gastro-soleus–Achilles tendon complex may lead to deterioration of gait, particularly in neurological conditions. Understanding of the complex relationship between hip, knee, and ankle joint kinematics in spastic neurological conditions is

necessary to avoid the pitfall of surgically weakening the Achilles tendon by lengthening. Three relatively large muscles power the tendon. The two gastrocnemei are bi-articular muscles spanning the knee and ankle joints. Selective gastrocnemius aponeurotic release may be preferable to lengthening of the whole tendon to avoid permanent weakness of the gastro-soleus–Achilles tendon complex. Permanent weakness of this musculotendinous complex will cause an abnormal gait with weak push-off, and is not uncommon after open Achilles tendon lengthening in congenital clubfoot.[4]

Congenital Disorders with Achilles Tendon Abnormalities

Talipes Calcaneovalgus

This benign foot positioning abnormality of the newborn is relatively common (1 in 1,000 live births),[5] caused by hyper-dorsiflexion of the foot against a tight uterus. Silent hip dysplasia may be associated with this condition[6] and needs to be ruled out by ultrasound examination of the hip. The structure of the foot is normal, and the deformity resolves spontaneously. Passive stretching is always successful but serial casting may be necessary occasionally. The deformity usually resolves by the sixth month of life and residual Achilles tendon sequelae are seldom seen.

Some investigators believe that talipes calcaneovalgus and symptomatic hypermobile pes planus seen in an older child may be etiologically linked and recommend active treatment of the more severe deformity.[7,8]

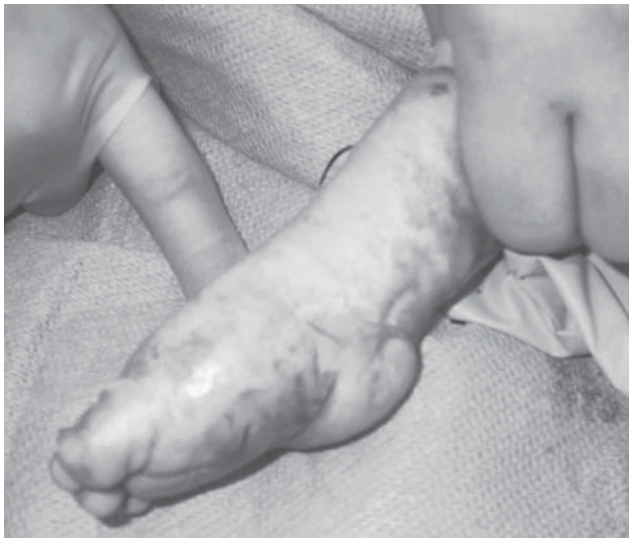
Congenital Clubfoot (Congenital Talipes Equinovarus)

Congenital clubfoot causes a fixed ankle and foot deformity often identified by the 20-week antenatal scan. The incidence of the idiopathic condition is 1 to 2 in 1,000 live births. Secondary talipes equinovarus deformity is seen in a variety of syndromes, arthrogryposis and spinal dysraphism, secondary to muscle imbalance. The idiopathic type results in localized musculoskeletal abnormalities affecting the leg-foot unit in an otherwise normal child.

Abnormal shortening of the Achilles tendon in clubfoot is secondary to the intrinsic foot deformity. The abnormally shaped talus in clubfoot results in medial talonavicular subluxation[9,10] and internal rotation of the calcaneum underneath the deformed talus. Calcaneal rotation manifests as heel varus and equinus. The soft tissue contracture of tendons and ligaments, including the Achilles tendon, maintains this deformity.

Surgical release or lengthening of the Achilles tendon is often necessary to correct the equinus deformity in clubfoot. The currently popular manipulative treatment methods also employ Achilles tendon tenotomy for correction of the equinus deformity. Serial weekly casting to correct all components of the deformity except equinus followed by Achilles tendon tenotomy to achieve 15° of dorsiflexion at final casting is widely practiced (Fig. 25.1). This approach is popularly referred to as the "Ponseti method."[11] A 30-year follow-up of 71 patients treated[12] shows retention of excellent gastro-soleus function in over 78% of feet, a significant improvement over the results of surgically managed clubfeet. Coupled with the remarkable lack of stiffness in the feet managed by using the Ponseti method, this has led to a great resurgence of interest in manipulative treatment of clubfoot. Gait abnormalities secondary to weakness of lengthened Achilles tendon is almost universal in clubfoot treated by open surgery.[4,13]

Insufficiency of the gastro-soleus secondary to open surgical release of clubfoot is common, and notoriously difficult to treat. Attention to detail during the primary procedure may prevent overlengthening. Coronal plane Z-plasty to produce two wide strips of tendon for correct tensioning of the lengthened tendon is important. Vigorous postoperative manipulation under anesthetic during cast change should be avoided to prevent tendon rupture.[14] Early diagnosis of gastro-soleus weakness can be made by gait observation and radiographical appearance of the calcaneus. Management of symptomatic gastro-soleus weakness involves tendon transfer. Peroneus brevis, flexor hallucis longus, and tibialis posterior tendons can be transferred to the calcaneus using tendon-bone fixation where possible. Tibialis anterior transfer is best avoided to prevent disabling foot drop



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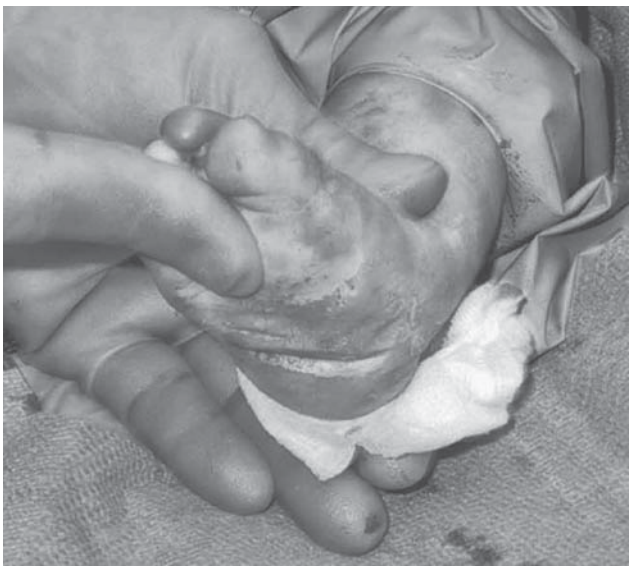

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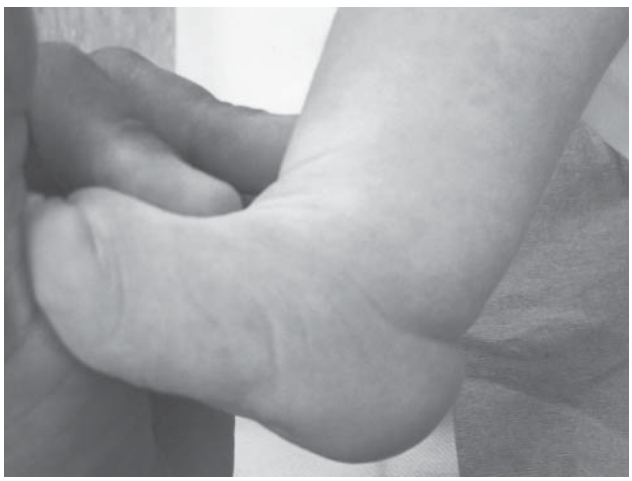

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Figure 25.1. Tenotomy of Achilles tendon in a 3-month-old infant to correct clubfoot deformity by Ponseti method. Complete tenotomy of the Achilles tendon in an infant does not cause long-term weakness. (**A**) Appearance of the foot before tenotomy. Note full correction of forefoot deformities. (**B**) Full correction of equinus after tenotomy. (**C**) Correction maintained at 6 months of age.

postoperatively, but has been used with some success in clubfoot.[15] In older children, calcaneal posterior displacement osteotomy may be necessary to increase the lever arm of the transferred tendons. Multiple combined tendon transfers to the calcaneum may not restore normal plantarflexion strength but several patients have developed toe-up ability, especially when the surgery was performed prior to age 6.[14]

Congenital Vertical Talus (CVT)

Congenital vertical talus or congenital convex pes valgus is a rigid deformity of the foot with a rocker-bottom appearance. CVT is a teratological dorsolateral dislocation of the talocalcaneo-navicular joint.[16] This condition is rare and often associated with other congenital limb, visceral, and neuraxis abnormalities. This condition can be differentiated from severe flatfoot by rigidity of the foot and resistance to passive restoration of the longitudinal arch in CVT. The lateral radiograph in CVT shows the talus in plantarflexion almost parallel to the distal tibia. In forced plantarflexion, the navicular fails to reduce on the talus and stays dorsal to the talar head.[17]

The Achilles tendon is invariably contracted, maintaining the calcaneum in equinus. Surgical single-stage correction is the mainstay of treatment and passive stretching and casting can be used to loosen soft tissues before surgery. Surgery involves dorsal capsulotomy and relocation of the talonavicular joint, dorsal extensors and peroneal tendon lengthening if indicated, and lengthening of the tendo Achillis. The relocated talonavicular joint is secured with a wire and held for 4 weeks. The results of one-stage surgery for CVT are generally good, but residual forefoot abduction, midfoot sag, and joint stiffness are to be expected.[18]

Post-traumatic Achilles Tendon Disorders in Children

Sever's Lesion

This is a common cause of heel pain in athletic older children and adolescents between the ages of 8 and 12. The etiology is likely to be post-traumatic due to a micro-injury of the metaphyseal bone adjacent to the calcaneal apophysis.[3] Achilles tendon stretches have been traditionally advocated for this self-limiting condition, although there is no evidence of pathological shortening of the Achilles tendon.

Overuse Injuries of the Achilles Tendon in Children

Insertional and noninsertional Achilles tendon problems have been reported in adolescent athletes. Achilles tendon rupture from athletic activities is extremely rare under the age of 20.[19] Clinical examination may identify predisposing factors such as foot pronation in stance, plantar fascia and Achilles tendon tightness, cavus foot, and obesity. Faulty running shoes and technique may also contribute to the symptoms, but a common underlying cause is excessive physical activity.[19] Ultrasonography[20] and magnetic resonance imaging are helpful in evaluation of these overuse injuries. The pathology of Achilles tendon pain in young athletes includes tendon sheath inflammation (paratendinopathy), occasional tendinopathy of the main body of the Achilles tendon, and retrocalcaneal bursitis. In adolescents with intractable Achilles tendinopathy, the possibility of juvenile chronic arthritis should be entertained and appropriate investigations performed. Modified rest, anti-inflammatory medication, and very short immobilization of the foot and ankle in a below-knee cast are successful in relieving pain in the majority of cases. A talar-neutral orthosis to prevent foot pronation and regular Achilles tendon stretching are generally successful in alleviating the symptoms. Surgical management for insertional and noninsertional Achilles tendon disorders is seldom necessary in this age group.

Developmental Foot Disorders with Achilles Tendon Tightness

Hypermobile Flat Foot with Short Tendo Achillis

In a study of 3,600 Canadian army recruits, Harris and Beath[21] found that the presence or absence of longitudinal foot arch did not correlate with dis-

ability. They also described a small group of subjects with symptomatic flat feet in whom there was associated Achilles tendon contracture. They coined the phrase "hypermobile flatfoot with short tendo Achillis" to describe this condition.[21] Up to 25% of subjects with flat feet may have an associated Achilles tendon contracture.

Older children with hypermobile flatfoot associated with Achilles tendon contracture tend to have callosities over the plantar medial prominence of the talar head, and may not tolerate rigid inserts. The heel valgus in these children may be considered a secondary compensation to accommodate the tight Achilles tendon, although it is equally possible that the Achilles tendon contracture is secondary to long-standing heel valgus. Examination of feet with this condition should reveal normal or excessive painless subtalar joint mobility, restoration of the longitudinal arch when non-weight-bearing, and Achilles tendon tightness. Achilles tendon tightness should be assessed with the heel locked in inversion.

Management should initially be directed toward passive stretching of the Achilles tendon. Older children can be given home stretching exercises.[22] Rigid inserts may not be tolerated due to pressure against the unyielding medial arch eminence of the insert.[23] Soft inserts or running shoes and regular stretching should reduce the symptoms in most children and adolescents with this condition. Surgery should be the last resort in a few selected patients. Persistent pain over the prominent talar head and medial arch pain on activity despite prolonged nonoperative treatment may constitute an indication for surgery.

Several surgical procedures have been described. Insertion of expansible sinus tarsi implants to limit subtalar joint mobility is advocated for flexible flatfoot, particularly in the podiatry literature, but there are no long-term outcome studies.[22] Evan's osteotomy of the anterior process of the calcaneum to restore the physiological talocalcaneal and talonavicular relationship has withstood the test of time.[24] Additional surgery on the medial aspect to reef the spring ligament and shorten the tendon of tibialis posterior may be necessary in conjunction with Evan's osteotomy.[23] A gastrocnemius slide should be an integral part of any surgery when Achilles tendon tightness is marked. Lengthening of the Achilles tendon should be avoided to prevent weakness.

Idiopathic Toe Walking (ITW)

Idiopathic toe walking should be considered a possible diagnosis in any child who toe walks after the age of two. ITW is a diagnosis of exclusion. Neurological conditions that cause toe walking, such as cerebral palsy, muscular dystrophy, spinal cord abnormalities, and poliomyelitis, should be excluded. A comprehensive developmental history and clinical examination should establish the diagnosis, although additional gait analysis and EMG studies may occasionally be necessary to differentiate mild cerebral palsy from ITW.[25]

Children with ITW typically walk on their toes, but can put their foot flat on request or when concentrating on their gait. A positive family history is found in 35–71% of cases.[26,27] Developmental delays relating to speech, language, gross or fine motor skills, and visual-motor development have been found in a significant proportion of children with ITW. Some investigators have suggested that ITW should be viewed as a marker for developmental problems and recommend that children with ITW should be referred for a developmental assessment.[2]

The etiology of ITW is not fully understood. The condition was originally described as "congenital short tendo Achillis," and this may well be the case in some patients.[28] EMG studies on ITW patients have shown changes similar to those seen in cerebral palsy patients with equinus deformity. Biopsy specimens of calf muscles in operated patients have shown features suggestive of a neuropathic process, indicating a possible neurogenic basis for this condition.[29]

The natural history of untreated ITW is not fully understood. In one study, 14 children with ITW were followed up for 7 to 21 years, and 11 of these were subjected to clinical examination and their gait videotaped. Three showed some evidence of toe walking when unobtrusively observed, but the others walked with a heel strike. The authors concluded that toe walking eventually resolved spontaneously in most children, and recommended that surgical treatment of ITW should be reserved for the few cases with a fixed ankle

joint contracture.[30] Serial casting has been advocated, but appears to be ineffective on its own.[31] To stretch the gastro-soleus–Achilles tendon complex, the foot needs to be supinated and dorsiflexed, and casted in that position, which is not amenable to walking.[32]

Botulinum toxin A (BTx-A) is being increasingly used to treat ITW but the long-term outcome of this intervention is not known. BTx-A has been shown to significantly improve ankle kinematics and normalize foot-strike pattern in ITW patients. This improvement appears to be maintained at 12 months after injection of BTx-A.[33]

Surgery has been advocated for ITW in the form of closed lengthening followed by a cast. This procedure appears to produce uniformly good results.[31,32] There is a risk of overlengthening the tendon, which can be minimized by undertaking percutaneous lengthening.

Achilles Tendon in Childhood Neurological Conditions

Cerebral Palsy

Movement disorders in cerebral palsy result from ante-, peri- or post-natal insult to the immature brain. Although the primary neurological injury is nonprogressive, secondary musculoskeletal pathology deteriorates in untreated children. Depending on the level and extent of the brain lesion, the child may suffer from hemiplegic, spastic diplegic, or total-body-involvement cerebral palsy. This chapter focuses on ankle and foot abnormalities in cerebral palsy, and refers to the spastic rather than dystonic type of cerebral palsy.

The management of movement disorders in cerebral palsy should involve multiple disciplines including physiotherapy, pediatric neurology, orthotics, occupational therapy, and gait analysis in addition to the orthopedic surgeon. Detailed clinical examination to determine strength, selective control and tone of muscles, deformity of joints, torsion of bones, and evaluation of balance and equilibrium is essential. In a walking child, gait analysis is an essential tool to make recommendations for management.[34]

Role of Ankle and Foot in Gait[35]

Human gait consists of a stance and swing phase. The action of the foot in the stance or support phase is described in terms of three rockers (Fig. 25.2).[36] During the first rocker, the heel makes ground contact, and the ground reaction force (GRF) with its fulcrum at the heel produces a plantarflexion moment. This force moment is restrained by the eccentric contraction of anterior tibial muscles that dampen the GRF and plays a shock-absorbing role.

The GRF passes anterior to the ankle joint during the second rocker in the mid-stance. This

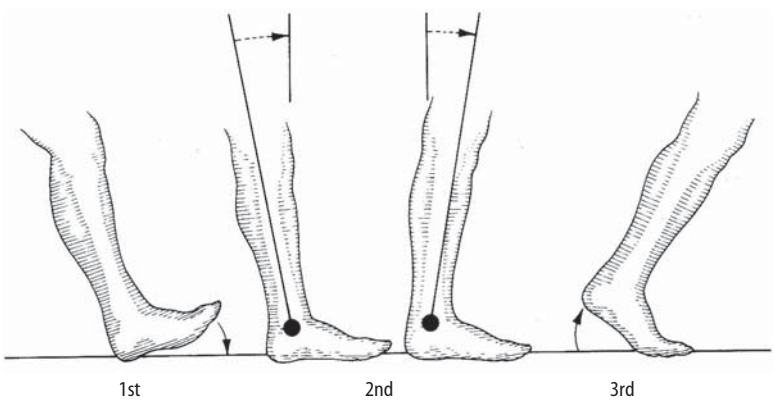

Figure 25.2. The three foot rockers in stance phase. The dorsiflexors and plantarflexors contract eccentrically (lengthening contraction) in the first and second rockers and are called deceleration rockers. The third rocker is an acceleration rocker where plantarflexors contract concentrically to achieve a push-off. (Illustration taken from Gage, 1991; see References 34, 35, 39. Reprinted with permission from Mac Keith Press.)

produces a dorsiflexion moment with its fulcrum now at the ankle joint level. This moment is resisted by the eccentric contraction of triceps surae. This restraint comes chiefly from the slow-twitch soleus muscle with later assistance by the gastrocnemius and long toe flexors. The tibia is held back by the contraction of the soleus, thereby keeping the GRF in front of the knee joint. By remaining anterior to the knee, the GRF maintains limb stability, as the knee is inherently stable in full extension even without quadriceps contraction. This extension moment on the knee generated by the GRF to achieve knee stability in mid-stance even without muscle contraction is called the plantarflexion/knee extension couple (Fig. 25.3).

Toward the end of the mid-stance phase, the combined action of plantarflexors raises the heel, and the fulcrum for this action moves to the metatarsal heads. This is the third rocker, and the action of the plantarflexors now switches from eccentric or lengthening contraction to concentric or shortening contraction, thereby achieving the push-off and forward acceleration.

In cerebral palsy, the first rocker can be absent due to dynamic or static plantarflexion at the terminal swing phase. In spastic diplegia, initial toe contact in stance may occur even without a static or dynamic ankle plantarflexion due to knee flexion deformity.[37] Excessive lengthening of the Achilles tendon without addressing the knee and hip problems may lead to a dramatic deterioration of gait due to weakness of plantarflexion causing migration of GRF posterior to the knee, resulting in a crouch gait. Children with cerebral palsy possessing walking potential should undergo gait analysis and electromyographic studies to precisely identify the abnormal kinematics and kinetics of their gait before surgery. Surgical procedures on triceps surae in cerebral palsy should be undertaken as part of a multilevel surgery to address pelvis, hip, and knee problems identified by gait analysis.[38]

The triceps surae, with other bi-articular muscles such as psoas, hamstrings, and rectus femoris, are mainly involved in cerebral palsy, probably because the strength and timing of these muscles have to be far more precise than mono-articular muscles.[39] Lengthening of the Achilles tendon as a whole will weaken the triceps surae across both ankle and knee joints, but isolated muscle lengthening of the gastrocnemius will achieve selective weakening without affecting the soleus, which, as discussed above, is important for stance phase knee/ankle stability. Further, contracted triceps surae dealt with by tendo Achillis lengthening may shorten with bone growth, as a lengthened muscle is not subjected to stretch with bone growth. Muscle stretch due to bone growth has been shown to be an impetus for muscle growth.[40]

Hemiplegia

In a hemiplegic child, treatment has traditionally focused on the ankle to address the commonly encountered equinus deformity in this condition. Gait analysis in hemiplegics has shown a spectrum of limb involvement of increasing severity. Based on gait analysis, four basic patterns of motor disorders have been identified in hemiplegia: types I to IV.[41] Dynamic or static equinus is seen due to plantarflexor overactivity in types I and II, without significant involvement of the knee and hip. Inadequate swing phase knee flexion is seen in type III due to co-spasticity of the hamstrings and rectus femoris. Type IV hemiplegics have additional involvement of the hip flexors and adductors. It is therefore important when managing a hemiplegic gait to identify coexisting proximal limb involvement and address all issues simultaneously through orthotics or surgery.

Spastic Diplegia and Total Body Involvement

In spastic diplegia, the lower limbs are predominantly involved with relative upper limb sparing. Ankle involvement is invariably associated with knee, hip, and pelvic abnormalities. Gait analysis is essential before surgery to identify the abnormal kinematics and kinetics at each level. In diplegics, initial toe contact at the first rocker can be secondary to knee flexion deformity, and patients may excessively dorsiflex their ankle to maintain a foot flat gait. Since the soleus spans only the ankle joint, it is not shortened, but may in fact be long. Overlengthening of the Achilles tendon in such patients will worsen the knee and hip flexion, leading to a crouch gait (Fig. 25.4). Secondary tightness of the gastrocnemius occurs when the

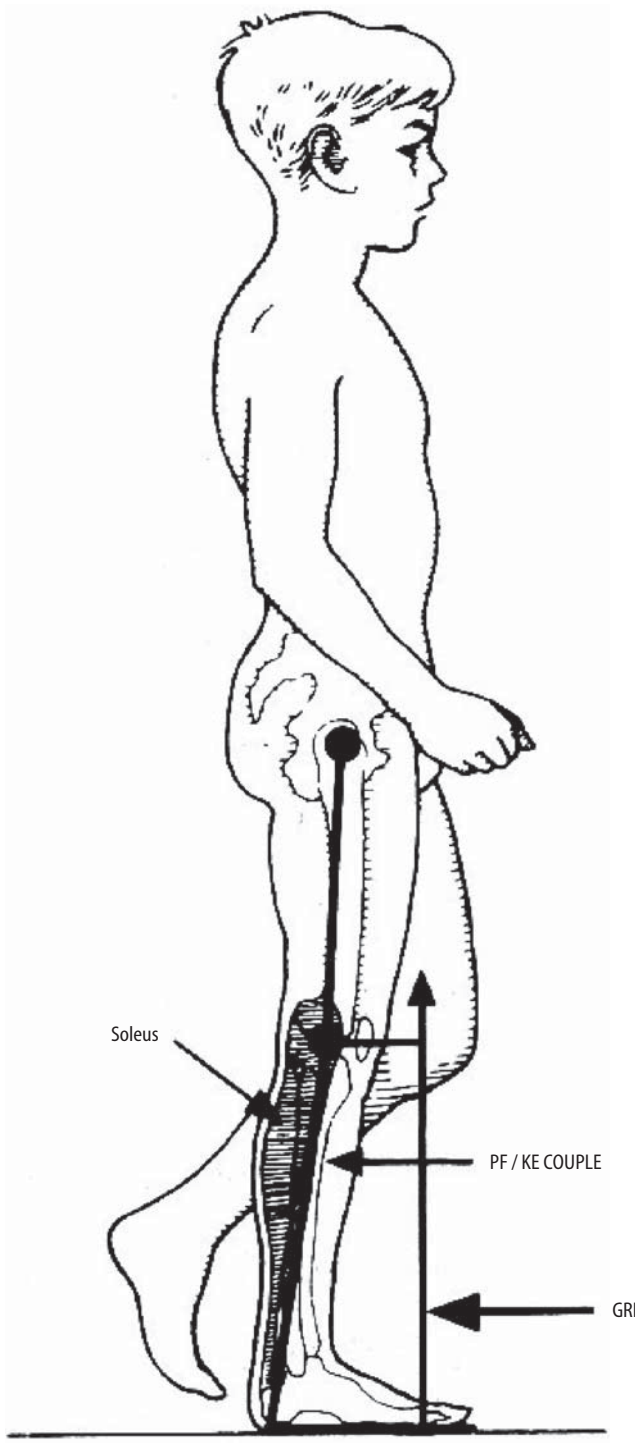

Figure 25.3. Plantarflexion/knee extension couple. The contraction of soleus during the second rocker restrains the tibia and keeps the knee joint behind the ground reaction force (GRF). The knee joint is inherently stable in extension even without the contraction of quadriceps. (Illustration taken from Gage, 1991; see References 34, 35, 39. Reprinted with permission from Mac Keith Press.)

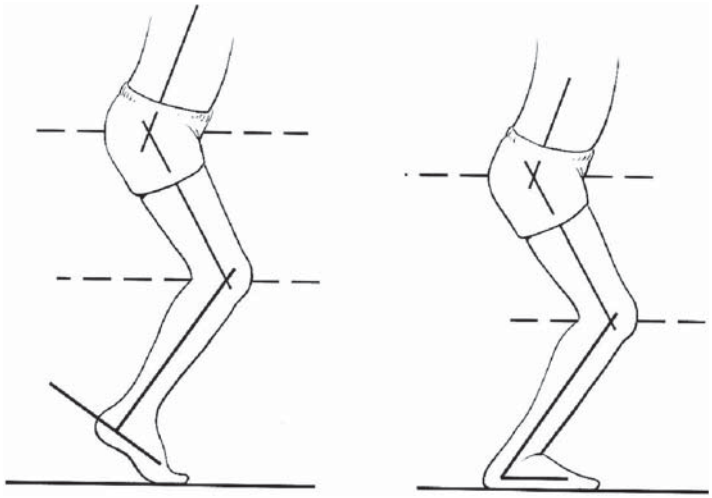

Figure 25.4. In a spastic diplegic child with crouched knee and toe-to-toe gait, the ankle is actually in a neutral position. Excessive lengthening of Achilles tendon causes excessive ankle dorsiflexion, leading to a calcaneus gait. (Illustration taken from Herring JA, ed., *Tachdjian's Pediatric Orthopaedics,* Vol. 2. Philadelphia: W.B. Saunders Company, 2002. Reprinted with permission from Elsevier.)

knee is surgically straightened by hamstrings lengthening: this will necessitate gastrocnemius release. Again, surgery should address hip, knee, ankle, and foot problems simultaneously.

Botulinum Toxin A (BTx-A) in Cerebral Palsy

Botulinum toxin A (BTx-A) is widely used to control spasticity in cerebral palsy.[42] BTx-A acts by blocking neurotransmission to muscles, reducing spasticity, and improving muscle balance. BTx-A injections in the calf muscle can significantly delay surgery in children with cerebral palsy, thereby reducing the incidence of repeat surgery.[43] Recurrence of Achilles tendon contracture and need for repeat surgery is higher in children with hemiplegia and diplegia undergoing open Achilles tendon surgery under the age of 6 years.[44] BTx-A administration to the calf muscles increases the compliance of orthotics wear in children with cerebral palsy.[45] Multilevel BTx-A injections after gait analysis reduces the frequency of surgery in cerebral palsy up to the age of nine in comparison with children not injected with BTx-A.[45]

Achilles Tendon Lengthening in Cerebral Palsy

Isolated Achilles tendon surgery and multilevel surgery in cerebral palsy is best delayed until children are at least 8 year old: by that time, gait is well established.[45] Equinus deformity in cerebral palsy can be corrected by selective gastrocnemius recession or by Achilles tendon lengthening. The Silverskiold test differentiates tightness of the Achilles tendon from isolated tightness of gastrocnemius component alone. If full correction of the ankle equinus is achieved by flexing the knee, the gastrocnemius is involved, and a selective release is indicated. The Silverskiold test performed under anesthetic can be used to determine the appropriate release.[46] Gastrocnemius recession may be performed using a variety of techniques, and the techniques of Vulpius, Strayer, and Baker are commonly used (Fig. 25.5).[47] The gastrocnemius aponeurosis is exposed in the midcalf through a longitudinal incision, and released transversely or in a chevron fashion. The underlying soleal aponeurosis is not disturbed, but the soleal septum is released. Care should be taken to avoid the sural communicating nerve in the vicin-

ity of the incision. Gastrocnemius recession preserves the power of the Achilles tendon during push-off,[48] and carries no risk of overcorrection and calcaneus deformity. A greater recurrence of equinus has been reported after gastrocnemius recession compared with Achilles tendon lengthening.[49]

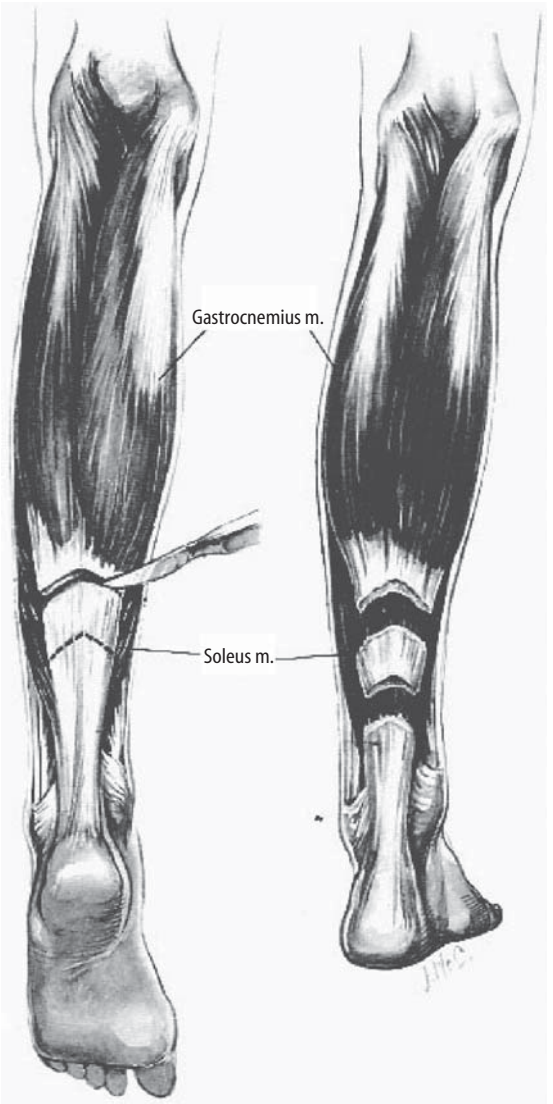

Figure 25.5. Gastrocnemius aponeurotic release by Vulpius method. A single-level transverse division of the gastrocnemius aponeurosis is sufficient. The underlying soleus and its aponeurosis are left intact. (Illustration taken from Herring JA, ed., *Tachdjian's Pediatric Orthopaedics,* Vol. 2. Philadelphia: W.B. Saunders Company, 2002. Reprinted with permission from Elsevier.)

Achilles tendon lengthening can be performed using open or percutaneous techniques. Open Z-lengthening should be undertaken with great caution in walking children, and sutured under appropriate tension to prevent overlengthening and calcaneus postoperatively. Closed techniques involve two or three hemisections of the tendon proximally and distally, followed by manual stretching of the tendon with heel locked in varus. Neutral ankle position is achieved with the knee in extension and a cast applied. A popular technique is the Hoke method, where the Achilles tendon is released proximally and distally by medial hemisections and a lateral hemisection performed in the middle (Fig. 25.6).[50] A rare complication of closed percutaneous release is complete severance of the tendon. If complete tendon severance is suspected, the calf squeeze test should be performed.[51] If there is no passive plantarflexion on calf squeeze, the tendon may be severed, and an open repair is indicated. This complication is extremely rare after percutaneous release.

Tendo Achillis in Spina Bifida

Foot deformity is found in 75–90% of patients with spina bifida,[52] depending on the level of the lesion. Lower lumbar and sacral lesions result in calcaneus deformity, while in upper lumbar and thoracic lesions equinus is more common. Equinus, calcaneus, equinovarus, vertical talus, and valgus ankle deformities are all seen in this condition.[3] The loss of sensation makes children with this condition prone to pressure sores, particularly when casting is used. For the same reason, fractures of the lower tibia and fibula secondary to manipulation of foot deformities may be missed. Although manipulation of foot deformities is recommended as the first line of management in spina bifida, it should be gentle to avoid fractures.

Equinus Deformity

Pure equinus deformities are common, and are mostly a positional deformity.[52,53] Equinus deformity may in some patients be secondary to posterior transfer of the tendon of tibialis anterior for calcaneus deformity. Passive manipulation is

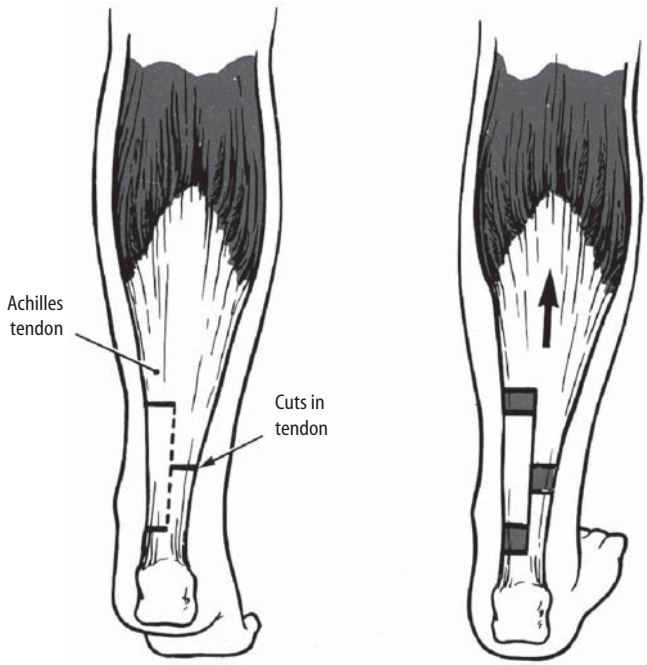

Figure 25.6. Hoke method of Achilles tendon lengthening. This method can be performed closed or open. The limb should be dorsiflexed to achieve neutral position and no more to avoid over-lengthening and calcaneus. (Illustration taken from Bleck, 1987; see References 47, 50. Reprinted with permission from Mac Keith Press.)

effective in the early stages, but care should be exercised to avoid distal tibial fractures secondary to manipulation, particularly in infancy. Often surgery is needed, and this should be timed to coincide with lower limb bracing for standing and ambulation. Closed or open Achilles tendon lengthening is performed, and a well padded below-knee cast applied. Long-term bracing is essential to prevent recurrence.

Calcaneus Deformity

Calcaneus deformity (Fig. 25.7) is often secondary to muscle imbalance in lower lumbar lesions due to unopposed tibialis anterior action.[54,55] Calcaneus deformity may also occur due to positioning in utero or post-natally. Positional deformities can be gently manipulated, but those due to muscle imbalance often need surgical correction. Calcaneus deformity in spina bifida is difficult to control with orthotics. Calcaneus deformity predisposes to persistent, difficult-to-treat heel ulcers, which may lead to calcaneal osteomyelitis. For this reason, early correction of calcaneus deformity is essential as delay in surgery increases the prevalence of calcaneal ulcers significantly.[55] Surgery consists of anterior capsular release and posterior transfer of the tibialis anterior to the calcaneus with the ankle in neutral position. This transfer can never substitute for the power of the gastro-soleus–Achilles tendon complex, and hence long-term bracing is essential. The foot should not be casted in equinus postoperatively, as this may lead to an equinus deformity. Further, passive manipulation to achieve a neutral position may cause a pathological distal tibial fracture.

Equinovarus Deformity

Secondary clubfoot deformity is often associated with spina bifida irrespective of the level of lesion.[56] Clubfoot deformity in this condition is always severe and rigid, and is described as teratological. Management is similar to the idiopathic type, but extensive soft tissue and bony surgery is required more frequently. Initial management should be gentle serial manipulations and well-padded casts.

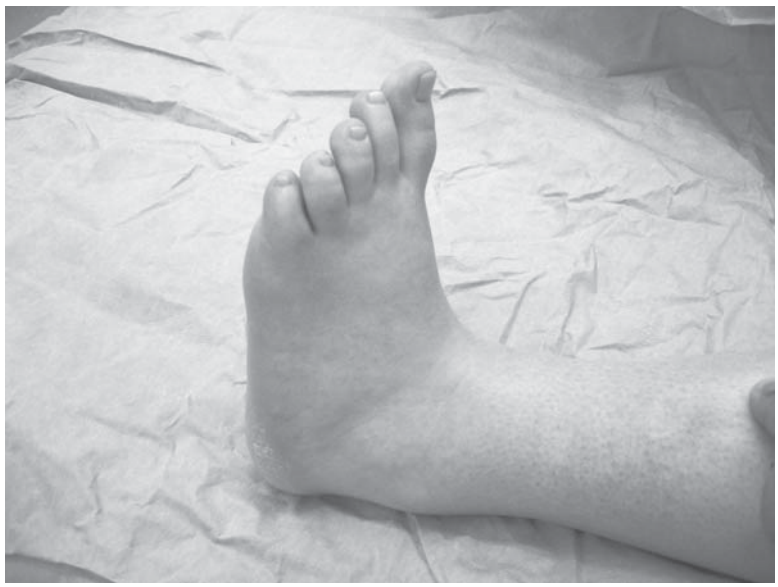

Figure 25.7. Calcaneus foot in spina bifida. Note the shortened tibialis anterior tendon.

These feet are insensate, and prone to pressure sores within the plaster casts. Posteromedial and lateral soft tissue releases for correction of this teratological deformity needs to be radical, and talectomy may be necessary for full deformity correction.[57] Unless function of the lower extremity is well preserved, as in sacral or very low lumbar lesions, tendon resections rather than lengthening or transfer are preferred to correct deformities. Talectomy or triple arthrodesis may sometimes be required to manage recurrent equinovarus deformity in spina bifida patients, but weight-bearing forces are not evenly distributed after either of these procedures, leading to neurotrophic ulcers.[58,59] Surgical correction of clubfoot in this condition should be delayed until the child is neurodevelopmentally ready for orthotics and ambulation.

Congenital Vertical Talus

This deformity is much less common in spina bifida than equinus, calcaneus, and equinovarus deformities.[53] The rigid planovalgus foot makes manipulation difficult, and orthotics are usually unsuccessful. One-stage surgery is necessary, followed by prolonged splintage in orthotics. Achilles tendon tenotomy is an integral part of this procedure to correct the equinus.

Ankle Valgus Deformity

Valgus deformity of the ankle is common in ambulatory patients with spina bifida, and may arise at the level of the ankle or hindfoot at the subtalar joint. A standing ankle anteroposterior radiograph is necessary to identify the site of valgus deformity.[60] Distal tibial osteotomy, distal medial tibial hemi-epiphysiodesis, or tendo Achills–fibular tenodesis can be used to treat ankle valgus deformity depending on the age of the patients, extent of the deformity, and the effectiveness of orthotics.

Mild to moderate ankle valgus deformity in a young child is amenable to Achilles tendon–fibular tenodesis. A portion of the Achilles tendon, still attached distally, is transferred laterally and sutured to the fibula above its distal physis. With weight bearing and ankle dorsiflexion, the fibula is pulled distally by the tenodesis, gradually correcting the deformity. The treatment is based on the concept that distal fibular growth is deficient in spina bifida from paralysis of the muscles in the lateral compartment. Improvement in talar tilt and relative fibular length was noted in most children managed by this method, with some improvement in the orientation of the hindfoot. External tibial torsion remained unaffected.[60]

Paralysis of Triceps Surae Muscle in Poliomyelitis

Flaccid lower motor neuron paralysis of triceps surae is very disabling. The patient lacks push-off, and walks with a calcaneus limp. The lack of push-off during the third foot rocker leads to an upward displacement of the anterior end of the calcaneum and head of talus. There is increase in the range of dorsiflexion and the normal posterior heel prominence disappears. Compensation by other plantarflexors such as tibialis posterior, long toe flexors, and the peronei result in equinus of the forefoot and eventually a calcaneo-cavus deformity. Contracture of the short toe flexors and plantar fascia increases the cavus deformity.

When the triceps surae is completely paralyzed, all available posterior muscles should be transferred to the calcaneum. The peronei, tibialis posterior, and flexor hallucis longus should be transferred posteriorly. In older children with fixed deformity, a triple arthrodesis is necessary in addition to tendon transfer. The os calcis may need to be shifted posteriorly and the bony deformity corrected at arthrodesis. In younger children, if tendon transfers restore sufficient plantarflexion strength, the calcaneus deformity may correct spontaneously by remodeling. The tibialis anterior may need lateralization to centralize it over the dorsum to prevent a dorsal bunion. When all plantarflexors are paralyzed, the tendon of tibialis anterior is transferred to the calcaneum, and the foot braced until skeletal maturity. An additional extra-articular subtalar arthrodesis may be necessary in younger children to achieve hindfoot stability after tendon transfer. A triple arthrodesis will be necessary in an adolescent.[61]

When no motors are available to substitute for triceps surae, consideration should be given to ankle arthrodesis, providing there is adequate knee stability and muscle control.

Muscular Dystrophies

Duchenne muscular dystrophy (DMD) is the most common form of muscular dystrophy, occurring in 1 in 3,500 boys. DMD usually manifests before the age of 3. Becker's muscular dystrophy (BMD) is less common, with disease onset at 7 years of age. Other forms of muscular dystrophy are much less common. Both DMD and BMD are X-linked recessive genes affecting only boys. The life expectancy in BMD is significantly better than in DMD, and patients with BMD may retain independent ambulation into early adulthood.

The earliest clinical manifestation of DMD may be ankle equinus, causing children to toe walk (Fig. 25.8). Muscular dystrophy should be considered in any young boy presenting with toe walking. The gastrocnemius muscle appears hypertrophic due to fibro-fatty replacement of muscle fibers: this appearance is termed pseudohypertrophy.

Examination reveals proximal muscle weakness. The child stands on his toes with his knees in hyperextension, locking the posterior capsule, thus achieving stability despite weak quadriceps. Hip extensor weakness leads to anterior pelvic tilt

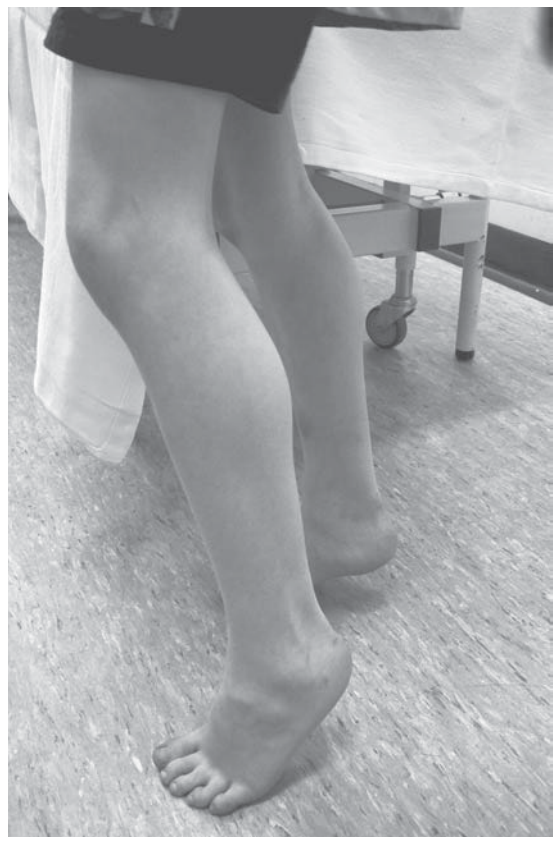

Figure 25.8. This 4-year-old child with Becker muscular dystrophy was referred for toe walking. Note the pseudo-hypertrophy of calf muscles.

with lumbar hyperlordosis during gait. The hips are stable because of the ground reaction force vector falling posterior to the hip.[62] When asked to stand from a sitting position without assistance, the child will "walk" his hands up the lower limbs to gain stability and achieve an upright position (Gower's sign).

As the disease progresses, ankle equinus or equinovarus deformities develop in addition to other lower limb joint contractures. Patients with muscular dystrophy can maintain their functional ambulation for longer if lower limb contractures are anticipated and managed early.[63] Physical therapy to stretch the ankle deformities, and provision of ankle-foot orthoses to maintain normal position, are important in the early stages. As the ankle and foot deformities get fixed despite regular stretching, surgery becomes necessary. Surgical correction of deformities by tenotomy and tendon transfers in the hip, knee, ankle, and foot can prolong independent ambulation for up to 2½ years longer in patients with DMD.[64]

The equinus is managed by percutaneous lengthening of the Achilles tendon. The varus deformity is managed by anterior transfer of the tendon of tibialis posterior through the interosseous membrane to the base of the second metatarsal, anchored through a bone tunnel. Below-knee casting is preferable postoperatively even after knee and hip surgery to maintain ambulation. Knee-ankle-foot orthoses (KAFO) can be used instead in an older child. Success of lower limb surgery in maintaining functional ambulation depends on the motivation of the child, absence of obesity, and an effective postoperative physical therapy regime.[65]

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26
Medico-Legal Aspects of Achilles Tendon Disorders

W.J. Ribbans and Ramanathan Natarajan

Introduction

Achilles tendon disorders present to a range of health-care professionals, including primary care physicians, physiotherapists, triage nursing staff, emergency care physicians, rheumatologists, and orthopedic surgeons.

Recent epidemiological studies have demonstrated a significant increase in the prevalence of Achilles tendon pathology,[1,2] with some authors reporting a 400% increase in the incidence of Achilles tendon ruptures in their personal practice over 20 years.

Tendon pathology accounts for between one-third and one-half of all sports-related injuries with the Achilles tendon most commonly involved.[1,3–5] To a large extent, this can be explained by the increasing numbers of people in their fourth decade and beyond who continue to enjoy aerobic exercise as a means of promoting cardiovascular fitness—the "weekend warrior" syndrome. Achilles problems are the third most common site of injuries in runners in the United States, accounting for 11% of all injuries and generating approximately 825,000 injuries every year.

Other chapters in this book describe the pathology and management strategies for specific forms of Achilles pathology. This chapter brings together some of the more controversial issues related to assessment and management. Where possible, it presents the evidence, or lack of it, for selecting particular pathways, and presents facts on outcomes that patients frequently ask.

While duplicating some of the information found elsewhere in this book, this chapter will:

- Help health-care professionals seek the evidence they require and provide signposts to the background work that underpins their management of these disorders.
- Provide objective information upon which patients can arrive at informed consent relating to treatment and outcome.

Acute Ruptures of the Achilles Tendon

Acute ruptures of the Achilles tendon present frequently and with increasing regularity as general participation in recreational sports increases. The history and examination usually lead to a clear diagnosis on clinical grounds alone. Considering the frequency with which the condition presents, the management of such injuries remains controversial with a surprising paucity of "gold standard" randomized prospective studies.

How Often Do Acute Achilles Tendon Ruptures Get Missed?

Acute Achilles tendon ruptures may present to a variety of health-care professionals with significantly different levels of clinical experience. Frequently the patient does not appreciate the significance of the injury sustained.

Overall, the diagnosis is missed in up to 25% of patients on initial assessment.[6,7] However, in the older age group, the delay is frequently greater due to a combination of reduced patient awareness and lowered clinical suspicion. In

Nestorson's series,[8] 36% of the over-65-year age group were not diagnosed for at least one week.

Medico-Legal Implications

- A careful history and examination should be taken and recorded in all patients with an account of sudden pain, snap, or pop in the calf. The findings on conducting the following tests should be recorded:
  - Palpable gap
  - Calf squeeze test (Simmonds, Thompson and Doherty)
  - Knee flexion test
  - Tiptoe test (may be too painful)
- Have a heightened suspicion in an elderly patient with a history of a snap or pop in the calf. This group forms a second peak for ruptures, albeit smaller than for the younger sportsman or -woman.
- After a few days, an initial palpable gap may be obscured by hematoma and early granulation tissue.
- If there are any doubts as to the presence of an acute rupture or whether it is total or partial, you should either seek more experienced advice or arrange for objective information in the form of either real-time high-resolution ultrasonography or an MRI scan.

How Accurate Is Imaging of a Suspected Acute Achilles Tendon Injury?

The diagnosis of an acute rupture of the Achilles should remain a clinical diagnosis based on a sound history and examination. There are, however, occasions when additional imaging may be helpful.

Conventional Radiography

Plain radiographs of the hindfoot and distal leg contribute little in the diagnosis of acute Achilles ruptures. Occasionally, a lateral radiograph, especially after a couple of days, may show loss of the usually sharp interface between the anterior border of the tendon and the pre-Achilles fat pad (Kager's triangle) due to swelling or hemorrhage. However, chronic tendinopathy may give a similar appearance.[9]

Ultrasound

The accuracy of ultrasound examination of the Achilles following rupture is still at least partially dependent on the experience of the radiologist.[10]

In experienced hands, ultrasound is very accurate at diagnosing a full-thickness tear as a complete gap between the retracted torn ends and posterior acoustic shadowing. The gap between the torn ends can be measured and act as a guide to decision making on surgical or conservative management. Because of its dynamic nature, ultrasound can determine whether torn ends will appose and if so the ankle position required to fully appose the torn ends.

There is some controversy over the usefulness of ultrasonography in postoperative follow-up,[11–13] although it has been advocated for patients treated conservatively.[14]

Ultrasound can offer unique advantages when trying to distinguish between full- and partial-thickness tears. There is always a caveat, as a plantaris tendon may manifest itself as an intact medial part of the Achilles tendon itself.

Distinction between a partial- and full-thickness tear could be made with 92% accuracy in Hartgerink et al.'s paper[15] with a sensitivity of 100% and specificity of 83%.

Ultrasonographically detectable tendon abnormalities persist long after the tendon has successfully healed and the patient has returned to normal activities of daily living.

MRI

MRI is increasingly used to image the Achilles tendon for chronic tendinopathy. The same imaging modality can be used to evaluate acute injuries.

MRI will accurately locate and determine the size of the gap following acute injury. Compared with ultrasound, it is less easily adaptable as a dynamic tool in determining the ability to close the gap between torn ends in different ankle positions.

It can be helpful in distinguishing between partial and complete tears. However, in patients with longstanding severe tendinopathy, distinction between a partial tear and tendinopathy is not always possible. Marked tendon enlargement and significant alteration in signal intensity from

within the tendon should be considered to be a partial tear unless proven otherwise.[16]

Medico-Legal Implications

Imaging may be useful in certain cases of suspected or proven acute Achilles tendon ruptures:

- To determine whether the tear is a partial or complete rupture
- In cases of delayed presentation, when the gap may have filled with hematoma and granulation tissue, to confirm diagnosis
- To determine whether the gap between torn ends closes as the ankle plantarflexes
- To monitor progress toward healing—especially if nonoperative treatment has been instituted
- Occasionally, to monitor satisfactory healing later in the rehabilitation process, especially for elite/professional athletes prior to return to sports

However, the limitations of ultrasound and MRI should be appreciated and the primacy of history and clinical examination acknowledged.

Conservative or Surgical Management for Acute Rupture of the Achilles Tendon?

When counseling a patient diagnosed with an acute rupture of the Achilles tendon, a significant proportion of the time will be spent discussing the merits of conservative versus surgical management, and the potential ensuing complications of each management modality.

The debate over conservative versus surgical repair of acute ruptures continues to rage and is hampered by the lack of a number of prospective randomized "gold standard" studies. Cetti,[17] Nistor,[18] and Möller[19] are the three studies that have attempted to prospectively examine these issues.

Medico-Legal Implications

- Patients need to be informed that there are no absolutes in decision making for management of acute ruptures of the Achilles tendon.
- Each patient needs to be individually assessed, and a final decision on management made according to elements such as the individual's age, occupation, level of activity, and medical history. Other factors such as the surgeon's own experience and facilities for rehabilitation need to be taken into account.

What Are the Likely Complications after an Acute Rupture of the Achilles Tendon?

Numerous studies tackle this subject. Kirkley[20] performed a quantitative review of the literature (Table 26.1) Others authors have produced similarly useful review publications on complications and re-rupture rates (Tables 26.2 and 26.3).

These studies combine many authors' experience using both conservative and several forms of surgical techniques on a large number of patients.

Table 26.1. Summary of Complications from the Management of Acute Achilles Tendon Ruptures

Complications	Nonoperative	Operative	Statistical Significance Using Unpaired t-Test
Major Complications			
Death	0	0	
Pulmonary embolus	1	0	
Deep vein thrombosis	4	1	
Pneumonia	0	2	
Skin slough	0	6	
Sinus formation	0	3	
Fistula formation	0	0	
Tendon lengthening	2	0	
Second operation	0	9	
Total	7/248	21/701	
Percentage	**2.8%**	**3.0%**	p = 0.296
Moderate Complications			
Delayed healing	0	18	
Granuloma formation	0	11	
Infection	0	4	
Sural nerve injury	1	42	
Total	1/248	76/701	
Percentage	**0.4%**	**10.84%**	**p = 0.0296**
Minor Complications			
Adhesions	2	99	
Total	2/248	99/571	
Percentage	**0.8%**	**17.3%**	**p = 0.0003**
Re-rupture Rate	29/248	21/742	
Percentage	**11.69%**	**2.83%**	**p = 0.001**

Adapted from Kirkley et al.[20]

Table 26.2. Review Articles Comparing the Complications Following Surgical and Nonsurgical Treatment of Acute Achilles Tendon Ruptures

Authors	Number of Articles Reviewed	Number of Tendon Ruptures	Nonsurgical Complication Rate	Surgical Complication Rate	Nonsurgical Re-rupture Rate	Surgical Re-rupture Rate
Wills et al.[21]	20	1003	2/20 (10%)	155/777 (19.9%)	40/226 (17.7%)	12/177 (1.5%)
Cetti et al.[17]	66	4597	24/514 (4.7%)	425/4083 (10.4%)	69/514 (13.4%)	58/4083 (1.4%)
Kirkley et al.[20]	19	990	10/248 (4%)	196/742 (26.4%)	29/248 (11.7)	21/742 (2.8%)
Popovic et al.[22]	16	5046	27/569 (4.7%)	492/4477 (11.0%)	76/569 (13.3%)	69/4477 (1.5%)
Wong et al.[23]	125	5056	59/645 (9.4%)	976/4411 (22.1%)	63/645 (9.8%)	103/4411 (2.3%)
Bhandari et al.[24]	6	248	0/210 (0.0%)*	10/221 (4.7%)	29/233 (12.4%)	7/225 (3.1%)
Kocher et al.[25]	32	1893	12/365 (3.3%)	306/1487 (20.6%)	29/347 (8.4%)	32/1437 (2.2%)
Khan et al.[26]	4	356	5/183 (2.7%)	59/173 (34.1%)	23/183 (12.6%)	6/173 (3.5%)

*Bhandari's paper limited to infections for complications.
Adapted from Movin et al.[32]

It is likely that different reviews will have used a similar cohort of papers upon which to base their findings. This makes it difficult to summate the findings and produce average complication and re-rupture rates. Similarly, the criteria for counting complications will vary widely between studies. However, the findings from these numerous authors provide a useful resource upon which to base patient counseling.

Medico-Legal Implications

- A discussion regarding potential complications following an acute Achilles tendon rupture should form a central part of the patient counseling process and help the patient and surgeon arrive at an informed decision over management strategy.
- Patients should be left in no doubt that the rupture of the most powerful musculotendinous unit in the body is not a benign injury. Significant complications may ensue, and some degree of long-term disability is possible.
- Patients should be informed that any management strategy has hazards or potential complications.
- Patients should be aware of the increased risks of wound breakdown and infection following surgery to this area. Sural nerve injury at 6% incidence[19] should be specifically mentioned.
- Overall, complication rates following conservative treatment are on average about 3–5%. Conversely, the overall complication rates for all types of surgical procedures combined is about four to five times greater at about 12–25%. This excludes re-rupture rates, which will be dealt with separately.

How Likely Is Re-rupture of the Achilles Tendon?

Kirkley's[20] literature review reported an overall re-rupture rate of 11.69% (29/248) in nonoperatively managed patients compared with a re-rupture rate of 2.83% (21/742) in those managed surgically (Table 26.1).

In Table 26.2, the re-rupture rate following surgical repair ranges from 1.4% to 3.5% in the papers quoted. Conversely, the re-rupture rate in nonoperatively managed patients ranged from 8.4% to 17.7% from the same set of authors.

Table 26.3. Summary of Complication Rates from Wong et al.'s Review with Respect to Management Techniques[23]

Management Techniques	Minor Wound Complications	Major Wound Complications	Minor General Complications	Major General Complications	Re-rupture Rate
Conservative ($n = 645$)	0.5%	0%	8.5%	0.6%	**9.8%**
Percutaneous + Immobilization ($n = 247$)	4.9%	0%	8.5%	0.8%	3.6%
Percutaneous + Early Mobilization ($n = 122$)	6.6%	**3.3%**	**14.8%**	0.8%	6.6%
Open + Immobilization ($n = 3718$)	**12.3%**	2.3%	12.3%	**2.3%**	2.2%
Open + Early Mobilization ($n = 283$)	4.9%	0.4%	5.3%	0.4%	1.4%
External Fixation (K-wire fixation) ($n = 41$)	7.3%	0%	7.3%	0%	0%

However, in some of these series, patients systemically unsuitable for surgery may have been offered conservative treatment only. Their underlying medical health (e.g., obesity, vascular disease, steroid medication) may increase their risk of re-rupture as well as making them unsuitable for surgery in the first instance. Also, 80% of re-ruptures occur within the first 3 months.[2]

Medico-Legal Implications

- All patients with acute Achilles ruptures should be counseled about the possibility of re-rupture.
- Overall the risk of re-rupture is about four times higher in patients managed conservatively.

Historically, patients managed surgically have about a 1:33 chance of re-rupturing compared with about a 1:8 chance for those managed conservatively.

- It is probable that modern methods of management—both surgical and conservative—will reduce re-rupture rates as techniques of repair, protection, and rehabilitation develop.
- The most dangerous time for re-rupture is within the first 3 months following injury.

What Is the Risk of Rupture on the Contralateral Side Following Initial Acute Rupture?

The reported risk of sustaining a rupture on the opposite side following an initial acute rupture is 6%.[27]

Medico-Legal Implications

The risk is at a sufficiently high level to suggest that, as part of the counseling process for patients following treatment for an acute rupture, the possibility of a rupture of the other Achilles should be discussed and advice given regarding warning signs.

How Complete Is the Recovery Following an Achilles Tendon Rupture?

The degree of recovery following an Achilles tendon rupture depends on a number of factors, including:

- Co-morbidity
- Level of pre-rupture activity
- Age of patient
- Type of occupation and pre-injury recreational activities
- Method and timing of initial treatment
- Rehabilitation regime
- Absence of complications

Despite changes in initial management, changing views on early weight-bearing status, and more proactive functional rehabilitation, the very fact of sustaining a rupture of the Achilles constitutes a potential significant long-term disability for the patient.

In 1986, Haggmark[28] reviewed the dynamic calf muscle status following rupture. At 3–5 years, those treated surgically were functioning significantly better than those treated conservatively.

More recent studies have indicated that, despite instituting more modern management strategies, some degree of residual weakness, muscle atrophy, and plantarflexion deficits can persist in the long-term.[19,29,30] Möller[19] found a gradual improvement in triceps surae strength and endurance during the first year post-injury.

A trend toward gradual, continuing improvement during the course of the first 12 months post-injury mirrors the serial ultrasound changes noted during the same period. Healing is noted within the first 6–8 weeks, and the tendon reaches its maximum width (15–20 mm) after 3 months. As remodeling progresses, the tendon gradually thins over several years. In the long-term, the tendon is approximately double (12 mm) normal width.[31]

A return to pre-injury occupation is dependent to a great extent on the type of work performed. A sedentary form of occupation is associated often with a return to employment within one month if the patient is motivated and the employer sufficiently understanding. Kirkley et al.[20] found that, in over 500 operated patients, the average time to return to work was 1.95 months compared with 2.3 months for those managed conservatively. This difference did not reach statistical significance ($p = 0.611$). However, in heavy manual occupations, periods absent from work are likely to exceed 3–4 months.[32] For the latter, health and safety issues at the workplace often govern the

willingness of the employer to allow the patient to return to work.

An overt limp should disappear by 3 months, but ballistic activities such as running and jumping will usually be undertaken after six months.[19]

A return to pre-rupture formal recreational activities is by no means guaranteed. Many patients declined to return, despite being able to, for fear of further injury (30% at one year), while 15% were unable to return at one year, leaving just over one-half (54%) returning to their pre-rupture recreational activities at 12 months.[19] Of 545 patients managed surgically, 73.39% returned to sports compared with 69.48% for the 200 patients managed conservatively. This difference did not reach statistical significance ($p = 0.581$).[20]

Medico-Legal Implications

The patient should be warned that following rupture:

- The Achilles tendon will remain permanently thickened—approximately double the healthy contralateral tendon.
- The thickening will reduce gradually over the course of the first year, in concert with improvement of strength and endurance of the triceps surae.
- Despite modern rehabilitation techniques, some degree of permanent calf wasting and weakness may persist, and be associated with some degree of ankle stiffness and loss of proprioception.
- 70–84% of patients will be able to return to the same level of recreational activities within the year, but less than two-thirds of those able will choose to do so.
- Return to work is largely determined by the pre-accident employment. Employed patients should liase at an early stage with their employees over any relevant health and safety issues governing a return to work.

Chronic Ruptures of the Achilles Tendon

Chronic ruptures of the Achilles tendon occur through either initial misdiagnosis or late presentation on behalf of the patient.

The patients usually present with calf atrophy, plantarflexion weakness, limp, and an inability to increase activity (e.g., to run, play sports, or stand on single-stance tiptoe). However, in some patients, their disability may be more subtle. There may have been some connective tissue regeneration between the tendon ends and the plantaris tendon may have hypertrophied. Therefore, the gap will not be so apparent and the patient may be able to perform activities such as single-stance tiptoe—although usually not repetitively.

Do All Patients with Chronic Ruptures of the Achilles Tendon Require Operative Repair?

There is no doubt that all patients with chronic Achilles tendon ruptures need appraising of their management options (i.e., surgical or conservative regimes).

Prospective, randomized trials of surgical and conservative treatment are not available, are probably unethical, and are likely to cause difficulty with matching patients on account of varying medical co-morbidity in many of the patients presenting in this group.

Many orthopedic surgeons with an interest in trauma and foot and ankle disorders will have encountered elderly patients with delayed Achilles ruptures, who appear to be performing adequately for their required level of functioning.

The largest series on outcome following conservative management of chronic ruptures was published over 50 years ago by Christiansen.[33] In a series of 51 patients, 18 patients (35%) were treated conservatively for a variety of reasons. Satisfactory results were obtained in 75% of surgically treated patients and 56% of the conservative group. A satisfactory result was judged by normal gait, return to work, and slight or no discomfort. Considering that some of the patients managed conservatively were "preselected" as surgery may have been contraindicated because of nonorthopedic considerations, the results are surprisingly similar. In addition, improvement in function in the nonsurgical group could continue for several years following diagnosis.

The late presentation of patients is often due to an underlying sedentary or infirm state. The patients are frequently elderly with little understanding of the significance of the injury, which may have seemed relatively trivial.

Among those conditions that might preclude surgical intervention would be:

- Peripheral vascular disease
- Chronic infection
- Systemic disease (e.g., diabetes mellitus, rheumatoid arthritis)
- Morbid obesity
- Advanced age
- Pre-senile dementia or dementia
- Sedentary or bed-bound existence
- Inability to comply with the postoperative regime

Surgeons should carefully evaluate the patient for all of these conditions before recommending operative intervention. Conservative options in the form of ankle-foot orthoses (AFO), physiotherapy, and pain relief can be employed to improve comfort and function.

What Should Patients Be Told Prior to Surgery for Chronic Achilles Tendon Ruptures?

Surgical intervention for chronic Achilles tendon ruptures involves increased and additional risks compared with primary repair.

All of the complications outlined following acute Achilles ruptures managed surgically still stand. However, the incision is likely to be considerably longer, the procedure more demanding, and the surgical time prolonged. Such patients are likely to be older than those presenting with acute ruptures, and the risks of delayed wound healing, wound dehiscence, and infection are all greater.

There are certain risks attached to each of the potential surgical solutions (e.g., autogenous versus allograft versus synthetic repairs). In addition, if an autogenous repair is employed, there are differences between different forms of gap bridging (e.g., direct repair, V-Y advancement, and functional augmentation, for example, using the tendon of flexor hallucis longus (FHL), flexor digitorum longus (FDL), peroneal, or plantaris).

Patients need to be aware that the surgical plan will be finalized only once the tendon has been exposed, the diseased stump ends "freshened," the surgical bed inspected, and the final gap produced measured.

No adjacent transferred tendon can ever be as strong as the Achilles it is augmenting. Calf strength and endurance will always be deficient. Loss of the transferred tendon from its normal point of insertion may cause disability, particularly for the peroneus brevis, with some loss of eversion strength, and with loss of hallucal flexion strength with FHL tendon transfer.

Medico-Legal Implications

- Chronic Achilles ruptures are rare. Consequently, reported series of particular surgeons' experiences are often small.
- Presenting patients may be elderly with significant co-morbidity. In such circumstances, strenuous efforts should be made to determine the viability of the soft tissues and their ability to heal.
- Not all patients need surgical intervention, with continuing improvement in function likely to occur over several years.
- Patients should be made aware of the potential for complications following surgery, which will exceed those of acute repair (i.e., in excess of 20%).
- Surgeons undertaking such surgery should be fully aware of and experienced in different techniques for repair and reconstruction. Options need to be kept open until the damaged area is exposed and prepared. This is not surgery for the inexperienced operator.

Chronic Achilles Tendinopathy

Attempts to ameliorate the disabling symptoms arising from various forms of Achilles tendinopathy have led to many different surgical, mechanical, and chemical modalities trialed with variable efficacy. In some instances, the management may endanger the health of the tendon and surrounding soft tissues.

What Should a Patient Be Told about the Prognosis of Chronic Achilles Tendinopathy?

Longitudinal epidemiological studies on the long-term outcome in patients with chronic Achilles tendinopathy are sparse. Paavola et al.[34] reported the long-term outcome after eight years of follow-up:

- 84% had returned to full levels of exercise, with 94% of the total group pain-free or experiencing only mild pain on strenuous exercise.
- A delay in commencing conservative treatment for up to six months did not adversely affect treatment outcomes.
- 29% of patients failed to respond to conservative management, and required surgery.
- Despite an overall good outcome in terms of comfort and return to sports, there still remain clear differences in the affected side in terms of clinical assessment and ultrasound examination.
- 41% of the group developed contralateral symptoms in the previously asymptomatic other Achilles during the study period.

Patients should be made aware that a degree of responsibility for ensuring a good outcome is dependent on them. Many presenting patients have developed problems as a result of training errors and high-impact exercise that they are reluctant to change, and frequently return to ill-advised running schedules before the problem is fully rectified.

Medico-Legal Implications

- Most patients will make a good recovery.
- However, patients need to be fully involved in the rehabilitation process and aware that early return to sports can significantly affect outcome.
- Surgical intervention following a failure of conservative management is required in about 3 out of 10 patients.

What Should Patients Be Told about the Management Options for Chronic Achilles Tendinopathy?

The scientific literature is replete with papers on different management options for chronic Achilles tendinopathy. As in many aspects of medicine and surgery where a condition has many potential remedies, it usually reflects an uncertainty of correct management. There is little reliable scientific evidence to support the many management strategies.

When considering the varying options, both the health-care professional and patient should consider the likely efficacy and potential side-effects.

The various nonsurgical options described include:

- Rest
- Physiotherapy, including heat, ultrasound, deep frictions, ultrasound
- NSAIDs
- Orthotics
- Eccentric stretches
- Cold packs
- Laser management
- Topical ointments (e.g., glyceryl trinitrate)
- Injections:
  - Steroid
  - Heparin
  - Aprotinin

The various surgical options described include:

- Needling
- Coblation
- Percutaneous (ultrasound-guided) tenotomy
- Arthroscopic debridement (tendoscopy)
- Percutaneous paratenon stripping
- Open tenotomy and paratenon stripping
- Open tendon debridement
- Tendon grafting
- Tendon reconstruction

Medico-Legal Implications

- Management protocols vary from unit to unit.
- There is no unanimity over the most effective strategy.
- The development of management strategies has usually arisen as a result of local empirical experience and available resources.
- Patients should be appraised of the potential side-effects of any proposed management.

Much controversy surrounds the use of steroid injections, nonsteroidal anti-inflammatory

medication, and the type and timing of any surgical intervention.

Do Corticosteroid Injections for Achilles Tendinopathy Increase the Risk of Subsequent Achilles Tendon Rupture?

The use of corticosteroid injections in the management of soft-tissue injuries is widespread. Corticosteroids have been injected peri- or intratendinously for many decades, particularly by rheumatologists.[35] Already in 1976, Clancy[36] reported on the safe use of intratendinous Achilles injections for the management of early tendinopathy in athletes. However, the study was confined to 5 runners. More recently, orthopedic surgeons have tended to be more circumspect with regard to the use of corticosteroid injections in this area—specifically within the tendon itself. Debate continues over its use in terms of both efficacy and potential complications.

Published data have failed to accurately establish the exact risk of rupture following injection[4,37] or its likely efficacy on the underlying pathology.[37–40] Partial Achilles ruptures have been reported after steroid injections.

One problem in reviewing the literature on this subject is that patients would not receive a steroid injection unless they had underlying Achilles pathology with an inherently increased risk of rupture. Possibly, one of the adverse effects of corticosteroids is to mask the pain and pathology, allowing the patient to return to activity on an incompletely healed tendon, precipitating a rupture.[41]

Another issue relating to intratendinous steroid injections is that any randomized double-blind prospective study in humans is unlikely to receive ethical approval given the understandable concerns of physicians and surgeons based on empirical experience.

Gill et al.[40] reported the safety of peritendinous steroid injections for chronic Achilles tendinopathy when carefully undertaken using low volumes under fluoroscopic control. In this large retrospective study of 83 patients, 3.6% felt that their condition deteriorated, and 27.7% unchanged following injection. Similar guidelines had been reported by Fredberg.[42] However, Unverferth and Olix[43] warned of the danger of rupture following peritendinous injections to the Achilles. Speed[37] could detect no significant difference in results compared with a placebo injection.

Other authors have confirmed that, if steroid injections are to be placed in the peritendinous space, the injection should be carefully undertaken by experienced physicians using low volumes of injectable material.[3,41,44] It would appear that if the injection is to be administered in the peritendinous space, it should be done in the early stages of the history and using short-acting corticosteroids.[4,40,42]

Local steroid injections have been reported experimentally by McWhorter[45] as having damaging effects at both the cellular and matrix level. Collagen synthesis by fibroblasts appears to be inhibited. A recent basic science article[46] demonstrated that a direct steroid injection in the Achilles tendon of a rabbit diminished its strength, increasing the risk of rupture in the following weeks.

Conversely, two recent articles suggest that intratendinous injections are safe and efficacious.[47,48] While Koenig's study[47] reports a decrease in the ultrasonographic features suggestive of inflammation, the study was small (6 tendons in 5 patients) and the injections administered in the acute phases of a tendinopathy.

Patients contemplating undergoing a corticosteroid injection into the soft tissues should be counseled regarding possible local complications, including infection, subcutaneous fat atrophy (Fig. 26.1), skin pigmentation changes, and risk of delayed wound healing if the area subsequently undergoes surgery.

Medico-Legal Implications

- The evidence for the benefit of local corticosteroids by injection to manage Achilles tendinopathy is unproven. Peritendinous injections undertaken by experienced medical practitioners using low injectable volumes preferably under imaging control in the early stages of the condition may be beneficial.
- Local steroids have been reported to increase the risk of spontaneous tendon rupture. Animal studies attest to the deleterious effect on the tendon of intratendinous injections. Human

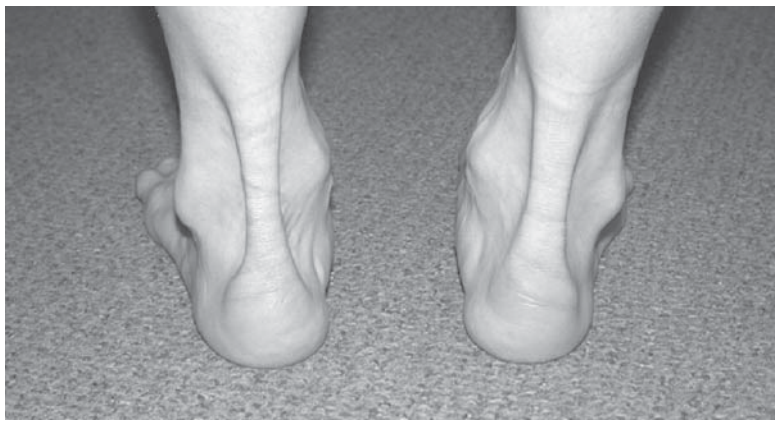

Figure 26.1. Subcutaneous fat atrophy around the Achilles tendon following repeated steroid injections.

studies reporting beneficial effects of direct tendon injections have not been without criticisms, and at the moment there does not seem to be any strong support for such a management regimen.

- Patients undergoing corticosteroid injections have an increased risk of wound healing problems in the area of the Achilles tendon, and should be appraised of such risks. Previous local steroid injections to the area can have a deleterious effect on the skin and subcutaneous tissues, increasing subsequent surgical wound healing risks.

Does the Use of NSAIDs Affect Achilles Tendon Healing?

Nonsteroidal anti-inflammatory drugs (NSAIDs) are widely used in soft-tissue disorders. Many different drugs exist within this category and their use is not without potential side-effects, such as gastrointestinal disturbances and renal damage. What is the evidence that they are helpful in the management of Achilles disorders, and is there any evidence that their use might be potentially harmful?

Physicians have frequently used nonsteroidal anti-inflammatory drugs (NSAIDs) in Achilles tendinopathy, especially in the clinically acute phases.[49–51]

Various forms of NSAIDs acted in a less-than-uniform manner when evaluated by Riley.[52] Indomethacin and naproxen inhibited cell proliferation and glycosaminoglycan synthesis in the tendon, suggesting that they should be avoided in clinical situations where relief is required from pain and swelling after tendon trauma. Conversely, no such effects could be ascribed to diclofenac or aceclofenac.

However, the evidence from drug trials and histological examination of pathological tissue does not support their use in the more chronic situation.

Åström and Westlin[53] undertook a randomized study on the effect of piroxicam in the management of Achilles paratendinopathy and could not detect a positive effect.

Histological specimens of Achilles tendon have failed to demonstrate an inflammatory cell infiltration in patients with chronic tendinopathies.[54]

Li[55] reported that NSAIDs may exert a detrimental effect on the tendon by increasing the levels of leukotriene B4 within the structure and enhance the likelihood of a tendinopathy. Almekinders[56] suggested that the effect of NSAIDs may be negative in the proliferative phase of healing by their inhibition of DNA synthesis, while acting in a more positive fashion later as the tissue matures and remodels by stimulating protein synthesis.

Medico-Legal Implications

- In the acute phases of Achilles tendinopathy, a short course of NSAIDs may help relieve symptoms.

- In chronic tendinopathy, NSAIDs are of little benefit, and may prove harmful.
- It seems likely that the only secure indication for the use of NSAIDs in patients with chronic Achilles tendinopathy is when it is associated with an underlying inflammatory arthropathy.[57,58]

Surgery for Chronic Achilles Tendinopathy

Nonoperative management fails to relieve symptoms in a sizeable minority of patients. In most patients, the treating surgeons will observe the outcome of natural healing and conservative management for a reasonable period before advocating surgery. A period of three to six months of adequate nonoperative management is common.

What Are the Likely Outcomes after Surgery for Chronic Achilles Tendinopathy?

Advising patients considering surgery on the likely outcomes is fraught with difficulties. The reasons for this are numerous, including:

- There are no randomized, prospective studies comparing conservative versus surgical management.
- Surgical management is inevitably followed by a period of rest and physiotherapy.
- There are several different surgical techniques described in this condition.
- There is a spectrum of pathologies included in the term *chronic Achilles tendinopathy*, and it is thus difficult to compare outcomes.

Despite these drawbacks, most studies on surgical outcome, albeit usually retrospective with varying outcome evaluations, report satisfactory results in excess of 75% of patients.

Watson[59] reported the outcomes of surgery for insertional Achilles problems. They stressed the more prolonged recovery time for calcific insertional problems (average 10.6 months) compared with an isolated retrocalcaneal bursitis (average 5.9 months). Additionally, patient satisfaction was higher in the isolated bursitis (93%) compared with calcific problems (74%).

Schepsis and Leach[60] reported better outcomes with noninsertional problems (92% good or excellent results) compared with insertional bursitis problems (71%).

Patients need to be appraised that recovery from surgery is prolonged, and rehabilitation is critically important.

Medico-Legal Implications

- Patients need to be appraised that recovery from surgery is prolonged and have stressed the critical importance of the rehabilitation process.
- A good outcome from surgery should occur in greater than 75% of patients.

What Are the Possible Complications Following Surgery for Chronic Achilles Tendinopathy?

Paavola et al.[61] studied 432 consecutive patients following surgery for Achilles tendinopathy. They reported an overall complication rate of 11%, with the majority (54%) due to problems with wound healing. However, other complications observed included sural nerve problems, scarring, rupture, and thrombo-embolic disorders. Overall, 3% of patients required further surgery to deal with complications. However, the authors stressed that, despite the complications, the long-term prognosis for pain relief and return to activity was good.

The Effect of Concomitant Medication of the Achilles Tendon

Steroids

The effect of steroids on Achilles tendon pathology should be considered according to whether they are corticosteroids or anabolic steroids.

Oral Corticosteroids

Oral corticosteroids are prescribed for a wide range of diseases. An increased risk of Achilles tendon rupture following the use of oral corticosteroids has been recognized for some time.

Newnham[62] reported on 10 patients who received corticosteroids for respiratory problems who experienced spontaneous Achilles tendon ruptures.

Inflammatory disorders such as rheumatoid arthritis, ankylosing spondylitis, giant cell arteritis, and gout are frequently controlled by prescribing corticosteroids. These conditions have been associated with an increased risk of Achilles tendon rupture per se. This raises the question of whether the disease process itself, the use of steroids to manage it, or a combination of both elements is important in the pathogenesis of the rupture.

Similarly, patients receiving corticosteroids as part of their immunosuppression therapy following major organ transplantation are at risk of suffering spontaneous Achilles ruptures.[30] The concomitant use of oral corticosteroids and fluoroquinolone antibiotics increases the risk of spontaneous Achilles tendon rupture[63] especially in the elderly.

Most patients on corticosteroid therapy and sustaining spontaneous Achilles tendon ruptures have been on the medication for some time, suggesting that the cumulative damage to the tendon may well be duration dependent. The majority of bilateral, simultaneous ruptures of the Achilles have been in patients taking oral corticosteroids for a variety of conditions.[64–66]

However, a few patients experience a spontaneous rupture following a short duration of therapy with high doses of steroids.[67] This suggests, in certain susceptible individuals, that dose dependency can be important.

Anabolic Steroids

The abuse of anabolic steroids has been associated with increased risk of tendon rupture.[68]

Medico-Legal Implications

- Oral corticosteroids can produce spontaneous rupture of the Achilles tendon. The benefits of these drugs for a widespread range of conditions are well-documented, and concern over the possibility of ruptures should not preclude their prescription based on sound clinical grounds. However, physicians should be aware of this potential risk, and patients should be sensitive to any symptoms from the tendon.
- Anabolic steroids increase the risk of spontaneous Achilles tendon rupture.

Rupture of the Achilles Tendon and Antibiotic Usage

An association between the use of certain antibiotics and tendinopathy has been acknowledged for some time. In particular, the use of fluoroquinolone antibiotics, such as ciprofloxacin, enoxacin, ofloxacin, and norfloxacin, has been associated with such injuries. This is a serious effect as it may progress to spontaneous tendon rupture. The number of such reports is increasing,[63,69–72] with the Achilles tendon most often implicated. Ruptures of the Achilles tendon were first reported in 1992.[73]

In 1996, the FDA issued directives to manufacturers to include warning statements for all fluoroquinolone products to alert to the potential for tendinopathy and tendon rupture.

Similarly in 1996, the Sri Lanka Drug Evaluation Sub-Committee decided that the product information of fluoroquinolone antibiotics should include a warning stating, "The onset of tendon pain calls for immediate withdrawal of fluoroquinolone antibiotics." Achilles tendon rupture was shown to occur even after withdrawal of the drug.

The Adverse Drug Reactions Advisory Committee (ADRAC) of Australia has recorded 25 reports of tendinopathy in association with these antibiotics. Most (88%) have been associated with ciprofloxacin, and three with norfloxacin. Most patients involved were elderly. The daily dose of ciprofloxacin ranged from 750 mg to 2,250 mg and for norfloxacin the usual daily dose was 800 mg. The average time of onset of reaction was within the first week.

A number of risk factors have been identified with regard to this adverse reaction, including age, renal dysfunction, and simultaneous corticosteroid therapy.

Van der Linden et al.[63] reported a large epidemiological study from the UK. The adjusted increased odds ratio (OR) for Achilles tendon rupture compared with a large control group was

4.3 for current exposure to quinolones, 2.4 for recent exposure, and 1.4 for past exposure. The OR of Achilles tendon rupture was 6.4 in patients aged 60 to 79 years and 20.4 in patients aged 80 years or older. In persons aged 60 years and older, the OR was 28.4 for current exposure to ofloxacin, while the ORs were 3.6 and 14.2 for ciprofloxacin and norfloxacin. Approximately 2–6% of all Achilles tendon ruptures in people older than 60 years can be attributed to quinolones, with increased problems in patients taking oral corticosteroids simultaneously, and a doubling of the risk of spontaneous rupture.

Pathologically, ultrastructural changes have been found in tendinocytes. As in other cases of fluoride poisoning, studies in animals show that magnesium deficiency aggravates the induced tendinopathy.

There is evidence that fluoroquinolone antibiotics decrease the transcription within the tendon of the proteoglycan, decorin,[74–76] which modulates collagen fibrillogenesis. A reduction in its synthesis would be expected to alter the viscoelastic properties of the tendon, and hence its propensity to rupture.

The reported numbers are too small to demonstrate whether antibiotic-implicated Achilles ruptures have an outcome different from spontaneous ruptures.

Medico-Legal Implications

- Fluoroquinolone antibiotics have a wide spectrum of antimicrobial activity and are widely used. The incidence of such injuries following prescription of one of these drugs remains small in the population overall.
- However, treating orthopedic surgeons should be aware of its potential for causing spontaneous rupture and, if found, such patients should be advised concerning repeat prescriptions to reduce the risk of a similar contralateral occurrence.
- An acute Achilles tendinopathy is more likely to occur in association with the risk factors mentioned above. The antibiotic should be withdrawn immediately to reduce the risk of tendon rupture in such circumstances. Problems are most likely to occur early in the period of administration of the drug.
- When assessing an elderly patient with an Achilles rupture, information on the recent use of fluoroquinolone antibiotics should be sought.
- Where possible, a combination of corticosteroid and fluoroquinolone antibiotic therapy in elderly patients should be avoided.

Genetic Predisposition to Achilles Tendon Disorders

The background to Achilles tendon disorders is multifactorial and recognizing any genetic predisposition has not been straightforward.

An association between the blood group O and Achilles tendon rupture was first reported in the early 1980s.[77,78] However, further surveys could not substantiate this trend.[79,80]

In equine studies, it has been demonstrated that a noncollagenous protein, cartilage oligomeric matrix protein (COMP), is particularly abundant during growth in the superficial digital flexor tendon (SDFT), the equine equivalent of the Achilles tendon. COMP has an organizational role in the formation of healthy collagenous matrix in the SDFT.[81] The presence of low levels of COMP, possibly secondary to certain inherited genotypes, may be associated with an increased risk of later tendon injury. The identification of a COMP mutation in humans in association with pseudoachondroplasia suggests that it has a similarly important role in human connective tissue development.[82]

As more is discovered of the finer detail of the human genome, work has centered on specific gene expression areas. Specifically, the production of a particular member of the matrix metalloprotease (MMP) family, MMP-3, may influence an individual's susceptibility to Achilles tendinopathy and rupture. MMP-3 has an important function in the normal maintenance and repair mechanisms that all tendons experience. Downregulation in certain individuals may diminish their ability to repair damaged tissue,[83,84] and this may be most important at the cellular level of the tenocyte.[85] In addition, an increase in the synthesis of MMP inhibitors such as TIMP-3 would adversely alter the ability of a tendon to repair itself as part of the normal homeostatic mechanisms.

Mokone[86] identified a genetic variant associated with Achilles tendon injuries. The overrepresentation of some genetic alleles was associated with a significantly increased risk of chronic Achilles pathology.

However, other inherited characteristics, such as somatotype and muscle fiber typing, may influence a person's propensity to exercise. Similarly, many environmental factors conspire to alter the level of sedentary or active lifestyles experienced by each of us and, hence, affect risk factors for Achilles tendon disease.

Medico-Legal Implications

- There is increasing evidence that certain individuals have an inherited predisposition to develop insufficiently strong tendons during growth and/or lack the ability to repair early damage to their tendons.
- However, the use of ABO blood typing to counsel patients as to the risk of chronicity of Achilles tendinopathy, healing capacity following injury, and the development of contralateral pathology, appears unproven.
- The cost and frequent lack of availability makes the accessing of genetic typing for an individual with Achilles pathology difficult. The work at genetic level remains, for the near future, a research tool rather than a widespread and helpful diagnostic tool.

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27
Genes and the Achilles Tendon

Adam Ajis and Nicola Maffulli

Gene therapy delivers genetic material to cells to alter synthesis and function, and can be achieved via viral vectors or liposomes.[1,2] Several animal studies have investigated the feasibility of gene transfer to tendons. Liposome constructs have been used to deliver β-galactosidase to rat patellar tendons.[3] In vivo and ex vivo adenoviral transduction of the lac Z gene into rabbit patellar tenocytes has been reported (Fig. 27.1). Gene expression lasted for 6 weeks, possibly long enough for clinical applications.[4,5] Apart from direct injection of vectors, gene transfer has been achieved via intra-arterial injection of liposomes.[6]

Animal studies have demonstrated that gene therapy can be used to alter the healing environment of tendons. Adenoviral transfection of Focal Adhesion Kinase (FAK) into partially lacerated chicken flexor tendons resulted in an expected increase in adhesion formation.[7] Although this study reports an adverse outcome, it proves the feasibility of gene therapy as a management modality.

Genetic Susceptibility to Achilles Tendon Injury

Genetic factors may be associated with an individual's susceptibility to Achilles tendon injury.[8–10] This was originally proposed in studies reporting an association between the ABO blood group and Achilles tendon ruptures or chronic Achilles tendinopathy.[8,11,12]

In a retrospective study, Jozsa et al.[11] compared the frequencies of the ABO and Rh blood groups of 292 Hungarian patients with primary Achilles tendon ruptures or re-ruptures and 540 patients with various other tendon ruptures with a control group consisting of 1.2 million subjects representative of the Hungarian population. The frequency of blood group O was significantly higher (53.1%) in the patients with Achilles tendon ruptures than for the general Hungarian population (31.1%). The O blood group was also higher in all the other investigated tendon ruptures, which included rupture of the long head of biceps (48.2%), extensor pollicis longus (53.9%), and the quadriceps (49.0%). Interestingly, 68.7% of the patients with multiple tendon ruptures (48) or re-ruptures (35) were of blood group O. No association was found in this study between the rhesus group and any of the tendon rupture groups.

Blood group O was still significantly higher in the combined tendon rupture group (54%) when 443 additional subjects were recruited and analyzed.[8] Kujala et al.[12] studied a group consisting of 917 Finnish patients diagnosed with a variety of musculoskeletal soft-tissue injuries. This group included 86 patients diagnosed with Achilles tendon ruptures and 63 with chronic Achilles tendinopathy. The frequency of the blood group O among the patients diagnosed with Achilles tendon ruptures (31.2%) was not significantly higher than in the control group (31.4%). The blood group O frequency was, however, higher in the patients diagnosed with Achilles tendinopathy (42.9%). The A/O ratios for the groups with rupture (1.0) or tendinopathy (0.7) of the Achilles tendon were lower than for the control population (1.42). The ABO blood group was not associated

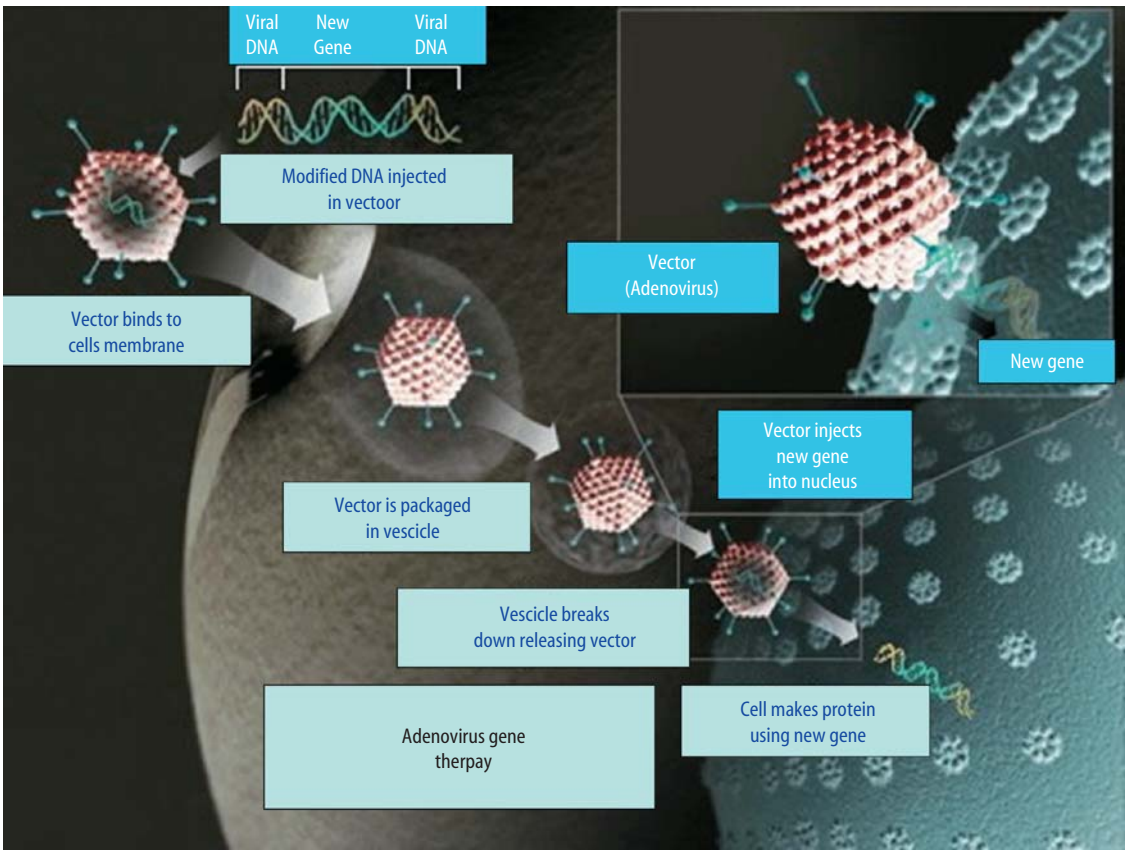

Figure 27.1. Schematics of adenovirus gene therapy.

with any of the other soft-tissue injuries. These soft-tissue injuries included rotator cuff impingement ($n = 142$), patellar dislocation ($n = 92$), anterior cruciate ligament rupture ($n = 205$), spondylolisthesis ($n = 177$), and intervertebral disc herniation ($n = 152$).

Årøen et al.[13] have suggested that there may be a genetic predisposition toward tendon ruptures as they observed that 9 of the 10 subjects who suffered a contralateral Achilles tendon rupture were of blood group B. The investigators, however, did not put forward suggestions why blood group B instead of blood group O was associated with Achilles tendon rupture in their study.

Contrary to these findings, other studies investigating Finish, German, and Scottish populations have not found an association between the ABO blood groups and Achilles tendon ruptures.[14,15]

Since the ABO gene encodes for transferases, some investigators have suggested that the different enzymes produced by the ABO gene determined not only the structure of the glycoprotein antigens on the red blood cells but also the structure of some of the glycoproteins found in the ground substance of tendons.[11] Others have, however, suggested that the association of the ABO gene with tendon injuries is not directly linked to the ABO blood group antigens. These investigators have proposed that other genes, closely linked to the ABO gene on the tip of the long arm of chromosome 9q32-q34, which encode for components of the extracellular matrix, are more likely to be associated with Achilles tendon pathology.[8,16]

There are examples of other pathologies, such as the nail-patella syndrome, where the ABO blood group was initially shown to be associated with the condition. It was subsequently discovered that the LMX1B gene, closely linked to the ABO gene, encoded for a protein responsible for the pathology.[17]

Since the ABO blood group has been shown to be associated with Achilles tendon pathology in some studies, possible candidate genes were identified, located on the tip of the long arm of chromosome 9, (9q32-q34), closely linked to the ABO gene that might be associated with Achilles tendon pathology. Two of these genes, namely tenascin-C gene and COL5A1, encode for structural components of tendons.[10]

The COL5A1 gene encodes for the pro alpha 1(V) collagen chain, found in most of the isoforms of type V collagen. The major isoform of type V collagen is a heterotrimer consisting of two pro alpha 1(V) chains and one pro alpha 2(V) chain. Trace amounts of type V collagen are found in tendons, where it forms heterotypic fibers with type I collagen.[18,19] Most investigators have speculated, based on the function of type V collagen in the cornea, that it plays an important role in regulating fibrillogenesis and modulating fibril growth in tendons.[19,20]

Dressler et al.[21] reported an age-dependent increase in the content of type V collagen, with a decrease in fibril diameter and biomechanical properties in the rabbit patellar tendon. In addition, Goncalves-Neto et al.[22] demonstrated an increase in collagen types III and V, together with a reduction in the content of type I collagen, in biopsy samples of tendons from patients with posterior tibial tendon dysfunction syndrome. A BstUI restriction fragment length polymorphism within its 3′-untranslated region of the COL5A1 gene has been shown to be associated with Achilles tendon pathology and more specifically chronic Achilles tendinopathy ($p = 0.0009$).[23] In addition, individuals with the A2 allele of this gene were less likely to present with symptoms of tendinopathy (odds ratio of 2.6; 95% CI 1.5–4.5, $p = 0.0005$).[23]

Although the COL5A1 gene is an ideal candidate gene for Achilles tendon pathology and more specifically chronic Achilles tendinopathy, the findings discussed above do not prove that type V collagen is involved in the etiology. It is possible that another gene closely linked to the COL5A1 and ABO genes on the tip of the long arm of chromosome 9 encodes for a protein directly involved in the pathogenesis of Achilles tendon injuries.

One such gene, the tenascin-C or hexabrachion gene, is expressed in tendons.[24–26] Since tenascin-C is able to bind to various components of the extracellular matrix and to cell receptors, it may play an important role in regulating cell–matrix interactions.[27] In normal adult tendons, tenascin-C is localized predominantly in regions responsible for transmitting high levels of mechanical force such as myotendinous and osteotendinous junctions.[24–26] Tenascin-C is also localized around the cells and the collagen fibers.[28] In addition, Järvinen et al.[26,28] have shown that expression of the tenascin-C gene is regulated in a dose-dependent manner by mechanical loading in tendons. Isoforms of tenascin-C with distinct functions are produced by alternative splicing of the primary transcript.[29,30] Riley et al.[31] have shown that healthy tendons express a small 200 KDa tenascin-C isoform, while degenerate tendons express a functionally distinct, larger 300 KDa isoform. In support of this finding, Ireland et al.[32] but not Alfredson et al.[33] have reported an increase in tenascin-C expression in biopsy samples of chronic Achilles tendinopathies using cDNA arrays.

The GT dinucleotide repeat polymorphism within intron 17 of the tenascin-C gene is also associated with Achilles tendon injury.[10] Alleles containing 12 and 14 GT repeats were overrepresented in individuals with the injury, while the alleles containing 13 and 17 repeats were underrepresented. Individuals who were homozygous or heterozygous for the underrepresented alleles (13 and 17 repeats), but did not contain an overrepresented allele (12 and 14 repeats), had a lower risk of developing Achilles tendon injury (odds ratio of 6.2, 95% CI 3.5–11.0).

A single gene, or a group of genes, on the tip of the long arm of chromosome 9 are highly unlikely to be exclusively associated with the development of the symptoms of Achilles tendon injury. It is more probable that this condition is polygenic, and other genes that encode for important structural components of tendons are also associated with Achilles tendon injury. Since the COL5A1 and tenascin-C genes have been shown to be associated with Achilles tendon injury, any gene that encodes for proteins involved in the same biological processes as type V collagen and tenascin-C within tendons would also be ideal genetic markers for tendon injury.

As mentioned above, type V collagen is involved in the formation of type I collagen–containing fibers. Several other proteins are also involved in

fibrillogenesis, including collagen types XI, XII, and XIV, the proteoglycans decorin, lumican, and fibromodulin, as well as the matricellular protein, thrombospondin 2.[34–37] In addition to tenascin-C, other proteins such as type XII collagen and type XIV collagen are also expressed in both tendons and ligaments and regulated by mechanical stretch.[38,39] It has also been postulated that collagen types XII and XIV play an important role in the regulation of fibril assembly due to their ability to interact with proteoglycans such as decorin, lumican, and fibromodulin.[40–42]

In addition, at immunoelectron microscopy, both these collagen types were associated with the surface of collagen fibrils, suggesting that they might be able to form interfibrillar connections and mediate fibril interaction with other extracellular and cell surface molecules.[43–46] The gene (COL12A1) that encodes for the alpha chain found in type XII collagen has been mapped to chromosome 6q12-q14. In addition, the COL9A1, COL10A1, and COL19A1 genes have also been mapped to chromosome 6, while the COL14A1 gene has been mapped to chromosome 8q23. Both collagen types XII and XIV are homotrimers and belong, together with collagen types IX, XVI, XIX, and XX, to the subfamily of fibril-associated collagens with interrupted triple helices (FACIT).[47–49]

Although only two specific genetic elements, namely the COL5A1 and tenascin-C genes, have been shown to be associated with Achilles tendon injury to date, neither these nor any other genes have been shown to be associated with any other overuse tendon or acute ligament injuries. Therefore, any genes located on the tip of the long arm of chromosome 9 (COL5A1, tenascin-C gene, COL15A1, COL27A1, and LAMC3) and on chromosome 6 (COL9A1, COL10A1, COL12A1, and COL19A1) that encode for a protein found in tendons could be ideal candidate genetic markers for tendon and ligament pathologies. In addition, genes that encode for proteins involved in the structure of tendons and ligaments (type I and III collagens, elastin, and fibronectin), fibrillogenesis (type V and XI collagens, decorin, lumican, fibromodulin, and thrombospondin 2), and are regulated by mechanical loading (tenascin-C and type XII and XIV collagens), could also be ideal candidate genetic markers for tendon and ligament pathologies.

Experimental Applications of Gene Therapy

During healing, levels of collagen type V increase, and persistently elevated levels have been found up to 52 weeks after injury in the rabbit medial collateral ligament. Elevated levels of collagen type V may favor the formation of smaller type I collagen fibrils, which in turn results in reduced mechanical strength.[50,51] Human patellar tenocytes transfected with specific antisense oligonucleotides synthesized reduced amounts of collagen type V.[52]

Complementary deoxyribonucleic acid (cDNA) for platelet-derived growth factor B was transfected into rat patellar tendons using liposomes.[2] The medial half of the patellar tendon was transected. Platelet-derived growth factor B resulted in an early increase in angiogenesis, and collagen deposition and matrix synthesis was greater at 4 weeks. However, there were no differences between the treated and control groups by 8 weeks.

Bone morphogenetic protein 12 (BMP-12) is the human analog of murine GDF-7.[53] BMP-12 increases gene expression of procollagen types I and III in human patellar tenocytes and is found at sites of tendon remodeling.[54] BMP-12 increased collagen type I synthesis by 30% in chicken flexor tenocytes, and, when tenocytes transfected with the BMP-12 gene were applied to a chicken flexor tendon laceration model, a twofold increase in tensile strength and load to failure was seen after 4 weeks.[55]

Conclusion

There is a high incidence of tendon and ligament injuries during exercise activities, but the exact etiology of this condition is not fully understood. Some studies have suggested that there is, at least in part, a genetic component involved in susceptibility to Achilles and other tendon injuries. Polymorphisms within the COL5A1 and tenascin-C genes are associated with Achilles tendon injuries in a physically active population, but further research is needed to determine which other genes are involved.

Gene therapy can be used to manipulate the healing environment for up to 8–10 weeks. This may be long enough to be clinically significant. Many genes may prove beneficial to tendon healing, and further research is required to establish the most advantageous genes to transfer. Many of the studies reviewed above have been conducted in tendon transection models, but gene therapy may also improve healing in tendinopathy.

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