Current Concepts Review - Rupture of the Achilles Tendon

NICOLA MAFFULLI

This information is current as of November 1, 2010

Reprints and Permissions

Click here to order reprints or request permission to use material from this article, or locate the article citation on jbjs.org and click on the [Reprints and Permissions] link.

Publisher Information

The Journal of Bone and Joint Surgery
20 Pickering Street, Needham, MA 02492-3157
www.jbjs.org
Achilles, the warrior and hero of Homer's Iliad, lends his name to the Achilles tendon, the thickest and strongest tendon in the human body. Thetis, Achilles's mother, made him invulnerable to physical harm by immersing him in the river Styx after learning of a prophecy that Achilles would die in battle. However, the heel by which he was held remained untouched by the water and thus Achilles had a vulnerable point. Achilles led the Greek military forces, which captured and destroyed Troy after killing the Trojan prince Hector. However, Hector's brother Paris killed Achilles by firing a poisoned arrow into his heel.

Hippocrates, in the first recorded description of an injury to the Achilles tendon, stated that "this tendon, if bruised or cut, causes the most acute fevers, induces choking, deranges the mind and at length brings death." A mbroise Paré, in 1575, recommended that a ruptured Achilles tendons be strapped with bandages dipped in wine and spices, but advised that the result was dubious. Operative repair of a ruptured Achilles tendon was advocated in 1888 by another Frenchman, Gustave Polaillon, although an Arab physician performed such procedures as early as the tenth century A.D. In the twelfth century, an Italian surgeon, Guglielmo di Faliceto, believed that nature was unable to unite divided tendons and that operative treatment was necessary.

Much research has been performed to elucidate the etiology of a rupture of the Achilles tendon, but its true nature still remains unclear. Aiso, the best method of treatment is still fiercely debated. Some physicians advocate operative repair, whereas others insist that an operation is unnecessary and poses an unacceptable risk.

The Achilles Tendon

The tendinous portions of the gastrocnemius and soleus muscles merge to form the Achilles tendon. The plantaris muscle, which was present in 93 percent (752) of 810 lower extremities in one study, is medial to the Achilles tendon and is distinct from it. The gastrocnemius tendon originates 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. The length of the gastrocnemius component ranges from eleven to twenty-six centimeters and that of the soleus component, from three to eleven centimeters. Distally, the Achilles tendon becomes progressively rounded in cross section, to a level four centimeters proximal to the calcaneus, where it can become relatively flatter, before inserting on the superior calcaneal tuberosity. The fibers of the Achilles tendon spiral through 90 degrees during its descent, such that the fibers that lie medially in the proximal portion become posterior distally. In this way, elongation and elastic recoil within the tendon are possible, and stored energy can be released during the appropriate phase of locomotion. Also, this stored energy allows the generation of higher shortening velocities and greater instantaneous muscle power than could be achieved by contraction of the triceps surae alone.

The calcaneal insertion of the Achilles tendon is highly specialized, as it is composed of the attachment of the tendon, a layer of hyaline cartilage, and an area of bone not covered by periosteum. A subcutaneous bursa may lie between the tendon and the skin to reduce friction between the tendon and the surrounding tissues. A retrocalcaneal bursa lies between the tendon and the calcaneus.

Structure of the Tendon

Tendons act as transducers of the force produced by muscle contraction to bone. Collagen accounts for 70 percent of the dry weight of a tendon. Aproximately 95 percent of tendon collagen is type-I collagen, with a very small amount of elastin. Elastin can undergo as much as 200 percent strain before failure. If it were present in the tendon in high proportions, there would be a decrease in the magnitude of force transmitted to bone.

Collagen fibrils are bundled into fascicles containing blood and lymphatic vessels as well as nerves. The fascicles are grouped together, surrounded by epitenon, and form the gross structure of the tendon, which is further enclosed by paratenon, separated from the epitenon by a thin layer of fluid to allow tendon movement with reduced friction.

Although the normal Achilles tendon consists almost entirely of type-I collagen, a ruptured Achilles tendon also contains a substantial proportion of type-III collagen. Fibroblasts from ruptured Achilles tendons produce both type-I and type-III collagen on culture.
Type-III collagen is less resistant to tensile forces and may therefore predispose the tendon to spontaneous rupture.

The normal Achilles tendon shows a well organized cellular arrangement, in stark contrast to one that is ruptured. Tenocytes, which are specialized fibroblasts, appear in transverse sections as stellate cells and are arranged in rows in longitudinal sections. This orderly arrangement probably is due to the uniform centrifugal secretion of collagen around the column of tenocytes, which produce both the fibrillar and the nonfibrillar components of the extracellular matrix and may also reabsorb collagen fibers.

Blood Supply

Tendons can receive their blood supply from vessels originating from three sources: the musculotendinous junction, the surrounding connective tissue, and the bone-tendon junction. The blood flow of the Achilles tendon depends on age, with a higher blood flow in younger individuals. The Achilles tendon is poorly vascularized, especially in its midportion, with blood vessels running from the paratenon into its substance. There is a dispute concerning the distribution of blood vessels in the tendon. Some investigations have shown that the density of blood vessels in the middle part of the Achilles tendon is low compared with that in the proximal part. Others have shown, with use of laser Doppler flowmetry, that blood flow is evenly distributed throughout the Achilles tendon and may vary according to age, gender, and loading conditions.

Biomechanics of the Tendon

Actin and myosin are present in tenocytes, and the tendon itself may have an active contraction-relaxation mechanism, which could regulate the transmission of force from muscle to bone. Fukashiro et al. measured a peak force of 2233 newtons within the human Achilles tendon in vivo. Komi et al. used buckle-type force transducers attached to the ankles of volunteers to show that, during walking, force builds up within the tendon before the heel strikes the ground. The force is then suddenly released for ten to twenty milliseconds during early impact. Thereafter, force builds up relatively fast until it reaches a peak at the end of the push-off phase, in a pattern similar to that observed during running. More recently, Arndt et al. showed that the Achilles tendon can be subjected to nonuniform stresses through modifications of individual muscle contributions. An injury therefore can be produced by a discrepancy in individual muscle forces caused, for example, by asynchronous contraction of the various components of the triceps surae or by uncoordinated agonist-antagonist muscle contraction due to impaired transmission of peripheral sensory stimuli.

At rest, a tendon has a wavy configuration, a result of crimping of the collagen fibrils. Tensile stresses cause the loss of this wavy configuration, accounting for
the toe-region of the stress-strain curve (Fig. 1). A s col-
lagen fibers deform, they respond linearly to increasing
tendon loads93. If the strain placed on the tendon re-
mains at less than 4 percent — that is within the limits of
most physiological loads128 — the fibers regain their
original configuration on removal of the load. A t strain
levels between 4 and 8 percent, the collagen fibers start to
slide past one another as the intermolecular cross-
links fail. A t strain levels of greater than 8 percent,
macroscopic rupture occurs because of the tensile fail-
ure of the fibers and interfibrillar shear failure128.

The compliance of the tendon is dependent at least
in part on intratendinous waviness98, which may affect
the ability of the gastrocnemius-soleus muscle complex
to generate force at the extremes of joint motion99. UL-
nately, it may also influence the forces exerted by
muscle contraction on the tendon and, hence, the pro-
penisty of the tendon to rupture.

**Epidemiology**

A lthough ruptures of the A chilles tendon are rela-
tively common, the incidence in the general population is
difficult to determine but has probably increased dur-
ing the past decade109. L epplihalti et al.120 estimated that
the incidence of ruptures of the A chilles tendon in the
city of Oulu, Finland, in 1994, was approximately eigh-
teen per 100,000. M ost ruptures of the A chilles tendon
(range, 44 percent [twelve of twenty-seven]121 to 83 per-
cent [ninety-two of 111]131) occur during sports activities.
In the Scandinavian countries, badminton players ap-
ppear to be at particular risk122; in a large study, fifty-eight
(52 percent) of 111 patients who had a rupture of the
A chilles tendon were playing badminton at the time of
the injury123. A rupture of the A chilles tendon is more
common in males, with a male-to-female ratio ranging
from 1:7:1 to 12:124,125,126, possibly reflecting the greater
prevalence of males than females who are involved in
sports, although there may be other as yet unrecognized
factors. The left A chilles tendon is ruptured more fre-
quently than the right127,128, possibly because of a higher
prevalence of individuals who are right-side-dominant
and thus push off with the left lower limb. Typically,
acute ruptures of the A chilles tendon occur in men who
are in the third or fourth decade of life, work in a white-
collar profession, and play a sport occasionally122,123.

The prevalence of rupture of the A chilles tendon
has been shown to be greater in patients who have
blood group O, at least among H ungarians15 and in some
Finns15. These findings have not been confirmed in other
studies126 even when the same ethnic groups were in-
volved128. We were not able to prove an association with
blood group in our area of Scotland, which has a high
incidence of rupture of the A chilles tendon127.

**Etiology**

Spontaneous rupture of the A chilles tendon has been
associated with a multitude of disorders, such
as inflammatory and autoimmune conditions96, geneti-
cally determined collagen abnormalities97, infectious dis-
eases7, and neurological conditions126. However, there is
little agreement with regard to its etiology.

A disease process may predispose the tendon to
spontaneous rupture from minor trauma128. Blood flow
in the tendon decreases with increasing age129, and the
area of the A chilles tendon that is typically prone to
rupture is relatively avascular compared with the rest of
the tendon129,130,131,132.

Histological evidence of collagen degeneration was
found in all seventy-four ruptured A chilles tendons de-
scribed in a study by A rner et al.120. However, nearly
two-thirds of the specimens were obtained more than
two days after the rupture. D avidsson and S alo122 re-
ported marked degenerative changes in two patients
with a rupture of the A chilles tendon who had an oper-
ation on the day of the injury. The changes, therefore,
should be regarded as having developed before the
rupture. In a study by Waterston127, performed at our
center, all tendons that were operated on within twenty-
four hours after the injury showed marked degenerative
changes and collagen disruption, in accordance with the
findings in another recent study18.

A lternating exercise with inactivity could produce
the degenerative changes seen in tendons12. Sports in
addition to daily activity places additional stress on the
A chilles tendon, leading to the accumulation of trauma,
which, although below the threshold for frank rup-
ture13, could lead to secondary intratendinous degener-
ative changes12.

Corticosteroids and Rupture of the Tendon

Corticosteroids are administered for a variety of
diseases and have been widely implicated in tendon
ruptures. Injection of hydrocortisone into the calca-
neal tendons of rabbits caused necrosis at the site of
injection forty-five minutes after the injection, and the
tendons that had been given an injection of corticoste-
roids showed a delayed healing response compared with
those that had received an injection of saline solu-
tion13. The anti-inflammatory and analgesic properties
of corticosteroids may mask the symptoms of tendon
damage13, inducing individuals to maintain high levels
of activity even when the tendon is damaged. Cortico-
steroids interfere with healing, and intratendinous in-
jection of corticosteroids results in weakening of the
tendon for as many as fourteen days13. The disruption is
directly related to collagen necrosis, and restoration of
the strength of the tendon is attributable to the forma-
tion of an acellular amorphous mass of collagen. For
these reasons, vigorous activity should be avoided for at
least two weeks after injection of corticosteroids in the
vicinity of a tendon13. U nverferth and O lix130 reported a
subcutaneous rupture in five athletes who had been
given injections of corticosteroids in the region of the
A chilles tendon for the treatment of tendinopathy. R e-
sidua of the corticosteroids were found at the site of the rupture in four of the five patients. A meta-analysis recently has shown that corticosteroid injections do not seem to play a beneficial role in the treatment of Achilles tendinopathy.

Orally administered corticosteroids also have been implicated in the etiology of tendon rupture. At our center, twelve patients who were managed with long-term oral therapy with corticosteroids for the treatment of chronic obstructive small airways disease were diagnosed with a rupture of the Achilles tendon during a ten-year period. Four of them sustained a bilateral injury.

It still is not possible to pinpoint the etiological role of corticosteroids, and some studies have not demonstrated deleterious effects of these agents. For example, McWhorter et al. demonstrated that a single injection of hydrocortisone acetate in a traumatized calcaneal tendon of a rat had no important biomechanical or histological adverse effect. However, given the present evidence, prolonged oral administration and repeated peritendinous injection of corticosteroids probably should be avoided.

Fluoroquinolones and Rupture of the Tendon

Fluoroquinolone (4-quinolone) antibiotics such as ciprofloxacin recently have been implicated in the etiology of rupture of the tendon. In France, between 1985 and 1992, 100 patients who were being managed with fluoroquinolones had tendon disorders, which included thirty-one ruptures. Many of these patients also had received corticosteroids, so it is difficult to implicate only the fluoroquinolones. Szarfman et al. noted that studies have shown that animals that received fluoroquinolone in doses close to those administered to humans had disruption of the extracellular matrix of cartilage, which exhibited as fissuration and chondrocyte necrosis, as well as depletion of collagen. The abnormalities seen in animals might also occur in humans. Szarfman et al. recommended that the labeling on packaging for fluoroquinolone be updated to include a warning about the possibility of tendon rupture. In its recommendations on the use of this class of antibiotics, the British National Formulary suggested that “at the first sign of pain or inflammation, patients should discontinue the treatment and rest the affected limb until the tendon symptoms have resolved.”

Recently, Bernard-Beaubois et al. found laboratory evidence of direct deleterious effects of fluoroquinolones on tenocytes. They suggested that pefloxacin, a fluoroquinolone, does not affect transcription of type-I collagen but decreases the transcription of decorin at a concentration of only 10^-4 millimoles. The resulting de-
crease of decorin may modify the architecture of the
tendon, altering its biomechanical properties, and pro-
duce increased fragility.

**Hyperthermia and Rupture of the Tendon**

As much as 10 percent of the elastic energy stored
in tendons may be released as heat\(^8\). Wilson and Good-
ship\(^9\) evaluated the temperatures generated in vivo
within equine superficial digital flexor tendons dur-
ing exercise. A peak temperature of 45 degrees Celsius
(a temperature at which tenocytes can be damaged\(^9\))
was measured within the core of the tendon after just
seven minutes of trotting. Exercise-induced hyperther-
mia therefore may contribute to tendon degeneration.
A good blood supply to tissues should help to cool
overheating, tissues such as the A chilles tendon, which
has relatively avascular areas, may be more susceptible
to the effects of hyperthermia.

**The Mechanical Theory**

McMaster\(^114\) proposed that a healthy tendon would
not rupture, even when subjected to severe strain. How-
ever, Barfred\(^13\) demonstrated that, if straight traction
were applied to a tendon, such as in McMaster’s experi-
ments, the risk of rupture would be distributed equally
to all parts of the muscle-tendon-bone complex. If oblique traction were applied, the risk of rupture would
be concentrated on the tendon. He calculated that, if a
1.5-centimeter-wide A chilles tendon in a human were
subjected to traction with 30 degrees of supination on
the calcaneus, the fibers on the convex aspect of the
tendon would be elongated by 10 percent before the
fibers on the concave side would be strained. Therefore,
the risk of rupture would be greatest when the tendon
was obliquely loaded, when the muscle was in maxi-
mum contraction, and when the initial length of the ten-
don was short. Such factors are probably all present in
movements occurring in many sports that require rapid
push-off. Barfred’s theory is largely supported by that
of Guillet et al. (reported in a study by Postacchini
and Puddu\(^10\)), who proposed a purely traumatic theory
for rupture of the tendon in young healthy patients. A
healthy tendon may rupture after a violent muscular
strain in the presence of certain functional and anatom-
ic conditions. These include incomplete synergism of
agonist muscle contractions, a discrepancy in the thick-
ness quotient between muscle and tendon, and ineffi-
cient action of the plantaris muscle acting as a tensor of
the A chilles tendon.

Participation in sports plays a major role in the de-
velopment of problems with the A chilles tendon, and
training errors are a major factor\(^33,39,62,69\). The flared heel
on most sport shoes forces the hindfoot into pronation
when the heel strikes the ground\(^33\), and the heel
tabs on some shoes may play a similar role. Clement et
al.\(^16\), in a study on the etiology of A chilles tendinopathy,
found that sixty-one (56 percent) of 109 athletes dis-
played a so-called functional overpronation of the foot
on heel-strike, with a whipping action of the A chilles
tendon. Exaggeration of this whipping action may lead
to intratendinous microtears. Poor flexibility of the
gastrocnemius-soleus unit was also considered to con-
tribute to overpronation\(^136,138\). Unequal tensile forces on
different parts of the tendon may produce a so-called
torsional ischemic effect — that is, transient vasocon-
striction of the intratendinous vessels — and therefore
contribute to the vascular impairment already present
within the A chilles tendon\(^16\).

Inglis and Sculco\(^16\) proposed that malfunction or
suppression of the proprioceptive component of skeletal
muscle predisposes athletes to rupture of the A chil-
les tendon. They believed that athletes who resume
training after a period of rest are particularly susceptible
to rupture of the A chilles tendon as a result of this
malfunction.

Knörzer et al.\(^15\) used x-ray diffraction spectra to
study the behavior of the structure of collagen during
tendon-loading. Tendons that rupture without previous
degenerative changes are damaged initially at the sub-
microscopic fibrillar level because of a process of in-
tratendinous fibrillar sliding, which occurs a few seconds before
macroscopic slippage of collagen fibers. Therefore, rup-
ture of tendons unaffected by degenerative changes
may result from the accumulation of fibrillar damage.
Such findings support the theory that a complete rup-
ture is the consequence of multiple microruptures and
that tendon damage must reach a critical point, after
which failure occurs.

**Mechanism of Rupture**

Aner and Lindholm\(^14\) classified the trauma resulting
in the rupture in ninety-two patients into three main
categories. The first category was pushing off with the
weight-bearing forefoot while extending the knee. This
movement is seen in sprint starts and in jumping in
sports such as basketball. This mechanism accounted for
53 percent of the ruptures in their series. The second
category was sudden, unexpected dorsiflexion of the
ankle, such as that occurring when the foot slips into a
hole or the individual falls down stairs. This mechanism
accounted for 17 percent of the ruptures. The third cat-
gegory was violent dorsiflexion of a plantar flexed foot,
such as may occur after a fall from a height. This mech-
anism was reported by 10 percent of their patients. The
exact mechanism of injury could not be identified for
the rest of their patients.

**Pathological Characteristics**

In 1976, Puddu et al.\(^10\) proposed a system to classify
abnormalities of the tendon. The major categories were
paratendinitis, paratendinitis with tendinosis, and pure
tendinosis. The term tendinosis describes the degener-
avative processes occurring within the tendon. Tendinosis
includes a number of pathological processes, such as
hyaline degeneration with a decrease in the normal cell population, mucoid degeneration with chondroid metaplasia or fatty degeneration of tenocytes, lipomatous infiltration of large areas of tendon, an increase in matrix mucopolysaccharides, and fibillation of collagen fibers. A rupture of a tendon may be the result of this process. In the opinion of Puddu et al., tendinosis is symptomless and is discovered only on rupture of a tendon. Patients who have symptoms before the rupture of a tendon commonly have a combination of peritendinits and tendinosis, and it is possible that a patient who has tendinosis may become symptomatic because of paratendinopathy, which may accompany tendinosis. Kannus and Józsa noted that only one-third of the 891 patients in their study had symptoms before rupture of the tendon. We found that only nine (5 percent) of the 176 patients managed because of a rupture of the Achilles tendon at our center between January 1990 and December 1995 had had previous symptoms.

Auner and Lindholm reported that all ninety-two ruptured Achilles tendons that they examined histologically had degenerative changes, including edematous disintegration of tendon tissue, patches of mucoid degeneration, and a marked inflammatory reaction. They also noted that approximately one-quarter of the larger-caliber arteries in the peritendinous tissue exhibited pathological hypertrophy of the tunica media and narrowing of the lumen.

Kannus and Józsa noted pathological alterations, 97 percent of which were degenerative changes, in all of the 891 spontaneously ruptured tendons from all of the sites that they studied. The most common degenerative lesion was hypoxic degeneration, with alterations in the size and shape of mitochondria, abnormal tenocyte nuclei, and occasional intracytoplasmic or mitochondrial calcium deposition. In advanced degeneration, hypoxic or lipid vacuoles and necrosis may be observed. A barrant collagen fibers also can be seen, with abnormal variations in the diameter, angulation, splitting, and disintegration of the fibers. Kannus and Józsa also noted vascular changes, mostly luminal narrowing due to hypertrophy of the arterial intima and media, in vessels of the tendon and paratenon. In 62 percent of the 891 ruptures, A iterations in blood flow, subsequent hypoxia, and impaired metabolism may have been factors in the development of the degenerative changes observed in the ruptured tendons. The interval between the rupture and the repair was short enough to suggest that the degenerative changes were preexisting.

Failure of the cellular matrix also may lead to intratendinous degeneration. Józsa et al. observed fibronectin on the torn surfaces of ruptured Achilles tendons. Fibronectin normally is located in basement membranes, is present in a soluble form in plasma, and binds more readily to denatured collagen than to normal collagen, indicating preexisting denaturation of collagen.

**Presentation and Diagnosis**

Patients who have a rupture of the Achilles tendon typically are first seen with a history of sudden pain in the affected leg, and they often report that, at the time of the injury, they thought that they had been struck by an object or kicked in the posterior aspect of the distal part of the leg. Some patients report an audible snap. They often are unable to bear weight and notice weakness or stiffness of the affected ankle. A rupture of the Achilles tendon may be associated with insufficient warm-up exercises before sports, with the injury occurring late in a game. Patients who have a chronically ruptured Achilles tendon also tend to have a fairly typical history; they often recall only very minor or perhaps no trauma and that they first noticed the injury because of an inability to complete everyday tasks such as climbing stairs.

Examination may reveal diffuse edema and bruising, and, unless the swelling is severe, a palpable gap may be felt along the course of the tendon. The site of the rupture is usually two to six centimeters proximal to the insertion of the tendon. Krueger-Franke et al. measured the location of the rupture intraoperatively in 303 patients and ascertained that, on the average, it was 4.78 centimeters proximal to the insertion of the tendon on the calcaneus.

In general, rupture of the Achilles tendon does not pose diagnostic problems. However, even in teaching centers, it is not uncommon to find that more than 20 percent of such injuries (sixteen of seventy-three in one study) are missed by the first doctor to examine the patient. There are a number of diagnostic signs and tests, both clinical and radiographic, that the examiner may use to aid in diagnosis.

**Clinical Tests**

**Calf-squeeze test:** The description of the calf-squeeze test is often credited to Thompson, who described the test in 1962; five years after Simmonds. With the patient prone on the examining table and the ankles clear of the table, the examiner squeezes the fleshy part of the calf. Squeezing the calf deforms the soleus muscle, causing the overlying Achilles tendon to bow away from the tibia, resulting in plantar flexion of the ankle if the tendon is intact. The affected leg should always be compared with the contralateral leg. A false-positive finding may occur in the presence of an intact plantaris tendon, although this has not been proved scientifically.

**Knee-flexion test:** The patient is asked to actively flex the knees to 90 degrees while lying prone on the examining table. During this movement, if the foot on the affected side falls into neutral or dorsiflexion, a rupture of the Achilles tendon can be diagnosed.
N. Needle test: A hypodermic needle is inserted through the skin of the calf, just medial to the midline and ten centimeters proximal to the insertion of the tendon. The needle is inserted until its tip is just within the substance of the tendon. The ankle is then alternately placed in plantar flexion and dorsiflexion. If, on dorsiflexion, the needle points distally, there is presumed to be a loss of continuity between the needle and the site of the insertion of the tendon

Sphygmomanometer test: For this test, a sphygmo-

Plain radiography: Lateral radiographs of the ankle have been used to diagnose a rupture of the Achilles tendon. When the Achilles tendon is ruptured, Kager’s triangle, the fat-filled triangular space anterior to the Achilles tendon and between the posterior aspect of the tibia and the superior aspect of the calcaneus, loses its regular configuration. Toygar’s sign involves measurement of the angle of the posterior skin-surface curve seen on plain radiographs, as the ends of the tendon are displaced anteriorly after a complete tear. The posterior aspect of Kager’s triangle then approaches the anterior aspect, and the triangle decreases or disappears. A normal Achilles tendon appears as an area of low signal intensity on all pulse sequences. The tendon is well delineated by the various imaging sequences that are more long-standing may be harder to diagnose because of associated tissue swelling. Real-time high-resolution ultrasonography and magnetic resonance imaging provide an adjunct to clinical diagnosis, and they are more sensitive and less invasive than soft-tissue radiography or xeroradiography.

Ultrasonography: Ultrasonography of the Achilles tendon with linear probes produces a dynamic and panoramic image of the tendon, the appearance of which varies with the type of transducer that is used and the angle of the ultrasound beam with respect to the tendon. High-frequency probes of 7.5 to 10.0 megahertz provide the best resolution, but they have a short focusing distance. The Achilles tendon is composed of longitudinally arranged collagen bundles, which reflect the ultrasound beam. The probe should be held at right angles to the tendon to ensure that an optimum amount of ultrasonic energy is returned to the transducer, avoiding the production of artifacts. Linear-array transducers, therefore, are better suited than sector-type transducers, which produce excess obliquity of the ultrasound beam at the edges. It also may be necessary to use a synthetic gel spacer or stand-off pad, which increases the definition of the surface echoes and allows a suitable support. A normal Achilles tendon appears as a hypoechoic, ribbon-like image that is contained within two hyperechoic bands. Tenon fascicles appear as alternate hypoechoic and hyperechoic bands that are separated when the tendon is relaxed and are more compact when the tendon is strained.

Men have slightly thicker tendons than women. Rupture of the Achilles tendon is seen on ultrasonography scans as an acoustic vacuum with thick, irregular edges. Campani et al. conducted 170 ultrasonographic examinations for various types of traumatic injury of the lower limb. The Achilles tendon had the highest percentage of positive findings (75 percent); in comparison, only 38 percent of the findings in the thigh were positive for a lesion. In a study that I did with two colleagues, we showed that ultrasonography is a suitable tool for assessing the structure of the tendon after operative repair. Ultrasonography also can be used to study the elastic properties of the tendon; this is done by measuring the distance between an intratendinous hypoechoic or hyperechoic point and the calcaneus and by assessing how this distance changes with different forces exerted by the gastrocnemius-soleus complex.
The many techniques and procedures described for the treatment of an acutely ruptured Achilles tendon can be grouped under three headings: open operative, percutaneous operative, and nonoperative. A s there is no agreed-on protocol, the choice of treatment regimen is still based largely on the preference of the surgeon and the patient. Nonoperative treatment has its supporters, but operative treatment has been the method of choice in the last two decades for athletes and young people and for patients who have a rupture for which treatment has been delayed. A cute ruptures in nonathletes may be treated nonoperatively. For example, in a prospective, randomized trial, forty patients who had an acute complete rupture of the Achilles tendon were allocated either to immobilization in a cast for eight weeks or to immobilization in a cast for three weeks followed by controlled early mobilization in a Sheffield splint, which is an ankle-foot orthosis that holds the ankle in 15 degrees of plantar flexion but allows some movement at the metatarsophalangeal joints. The splint allows controlled motion of the ankle during physiotherapy. Patients managed with the splint regained mobility notably faster and preferred the splint to the plaster cast. The range of dorsiflexion of the ankle improved more rapidly after treatment with the splint, and patients were able to return to normal activities sooner. Recovery of plantar flexion power was similar in the two groups, and no patient had excessive lengthening of the tendon. One repeat rupture occurred in each group.

Open Operative Repair

The many operative techniques used to repair ruptured Achilles tendons range from simple end-to-end suturing, with Bunnell or Kessler-type sutures, to more complex repairs with use of fascial reinforcement or tendon grafts. A tificial tendon implants, with use of materials such as absorbable polymer-carbon fiber composites, Marlex mesh (monofilament knitted polypropylene), and collagen tendon prostheses, have been used. End-to-end suturing, which can be performed with local anesthe sia, has been modified by use of materials such as Dacron vascular graft, which is passed through the tendon in a Bunnell-type fashion. Studies of dogs have shown that Dacron supports the growth of fibrous tissue and facilitates the approximation of the ends of the tendon, causing less tension at the repair site than standard sutures. However, maturation of collagen may be favorably influenced by cyclical tensional stimuli. Therefore, lack of tension on the repair site may not be advantageous.

Several authors have opposed operative repair, noting that the high rate of complications is the main disadvantage. A nner and Lindholm, in a series of eighty-six operative repairs of ruptured Achilles tendons, reported a 24 percent rate of complications, including two instances of deep-vein thrombosis, one of which resulted in pulmonary embolism and death; three wound infections; eleven instances of wound necrosis; and four repeat ruptures. More recent studies have demonstrated a much lower rate of complications. Soldatis et al., in a study of twenty-three patients who had operative repair, reported only two complications, both delayed wound-healing. The explanation for this low rate of complications may be greater operative experience combined with improved technique. However, wound problems should not be unexpected when open repair is used, as the most commonly used longitudinal incision passes through poorly vascularized skin. Photographic showing breakdown of the wound after operative repair of a rupture of the Achilles tendon. The patient had removed the cast against medical advice at four weeks after the operation and had returned to playing volleyball. At his first jump, the skin broke down.

Photograph showing breakdown of the wound after operative repair of the Achilles tendon. The patient had removed the cast against medical advice at four weeks after the operation and had returned to playing volleyball. At his first jump, the skin broke down.

Sutured Achilles tendons range from simple end-to-end suturing, with Bunnell or Kessler-type sutures, to more complex repairs with use of fascial reinforcement or tendon grafts. Artificial tendon implants, with use of materials such as absorbable polymer-carbon fiber composites, Marlex mesh (monofilament knitted polypropylene), and collagen tendon prostheses, have been used. End-to-end suturing, which can be performed with local anesthesia, has been modified by use of materials such as Dacron vascular graft, which is passed through the tendon in a Bunnell-type fashion. Studies of dogs have shown that Dacron supports the growth of fibrous tissue and facilitates the approximation of the ends of the tendon, causing less tension at the repair site than standard sutures. However, maturation of collagen may be favorably influenced by cyclical tensional stimuli. Therefore, lack of tension on the repair site may not be advantageous.

Several authors have opposed operative repair, noting that the high rate of complications is the main disadvantage. A nner and Lindholm, in a series of eighty-six operative repairs of ruptured Achilles tendons, reported a 24 percent rate of complications, including two instances of deep-vein thrombosis, one of which resulted in pulmonary embolism and death; three wound infections; eleven instances of wound necrosis; and four repeat ruptures. More recent studies have demonstrated a much lower rate of complications. Soldatis et al., in a study of twenty-three patients who had operative repair, reported only two complications, both delayed wound-healing. The explanation for this low rate of complications may be greater operative experience combined with improved technique. However, wound problems should not be unexpected when open repair is used, as the most commonly used longitudinal incision passes through poorly vascularized skin. In a study of forty patients, Aldam used a transverse incision just distal to the gap in the tendon and reported only one wound breakdown.

Several authors have proposed primary augmentation of the repair with the plantaris tendon, the peroneus tendon, a single central gastrocnemius fascial turndown flap, or two such flaps (one medial and one lateral). However, there is no evidence that primary augmentation of a repair of an acute rupture of the Achilles tendon is any better than nonaugmented end-to-end repair. I prefer to use augmentation in delayed repairs and in the treatment of repeat ruptures. After the operation, the leg is immobilized in a cast for four to six weeks. Some surgeons have advocated the use of a functional orthosis after several days of immobilization in a cast. This orthosis allows plantar flexion but restricts dorsiflexion and is designed to help prevent atrophy of the triceps surae. Some surgeons allow free motion of the ankle but no weight-bearing after operative repair.

There is a never-ending controversy regarding the use of open operative techniques to manage subcutaneous ruptures of the Achilles tendon, with some investi-
gators reporting a high overall rate of complications and others noting few complications and a low rate of repeat rupture.

**Percutaneous Repair**

Ma and Griffith developed a method for percutaneous repair as a compromise between open operative methods and nonoperative treatment. The technique involves producing six small stab incisions along the medial and lateral borders of the tendon and then passing a suture through the tendon with use of these incisions. In a small series of eighteen patients managed with this technique, Ma and Griffith reported only two minor, noninfectious skin complications and no repeat ruptures. Rowley and Scotland described twenty-four patients at our center who had a rupture of the Achilles tendon; fourteen were managed with immobilization in a cast alone, with the ankle in the equinus position, and ten were managed with percutaneous repair. One patient who had percutaneous repair had entrapment of the sural nerve, but no other complications were encountered. The patients who were managed with sutures were more likely to regain nearly normal plantar flexion strength, and they also returned to activity sooner than the group managed with a cast alone. Other authors have reported a much lower success rate with this technique. Klein et al. reported sural nerve entrapment in 13 percent of thirty-eight patients. Hockenbury and Johns compared in vitro percutaneous repair of the Achilles tendon and open repair of the Achilles tendon with use of a transverse tenotomy of the Achilles tendon and then passing a ten-millimeter gap appeared in the repaired tendon compared with those that were repaired with a percutaneous technique (a mean of 27.6 degrees compared with a mean of 14.4 degrees; p < 0.05). Entrapment of the sural nerve occurred in three of the five specimens that had percutaneous repair. The tendon stumps sutured with use of the percutaneous technique were malaligned in four of the five specimens. On the basis of the findings in that study, it appears that percutaneous repair of a ruptured Achilles tendon provides approximately 50 percent of the initial strength afforded by open repair and places the sural nerve at risk for injury.

**Nonoperative Treatment**

The most commonly used form of nonoperative treatment is immobilization in a plaster cast, usually for a period of six to eight weeks. Immobilization has been advocated by those who think that it produces results similar to those achieved with operative treatment. When the Achilles tendon ruptures, the paratenon generally remains intact. Stripping of the paratenon during an operation reduces the amount of reactive tissue that is produced later at the site of the injury. Those authors have therefore suggested that operative repair of a ruptured Achilles tendon should be avoided, as the paratenon provides a valuable blood supply to the damaged tendon. Lea and Smith reported in a study of fifty-five spontaneously ruptured Achilles tendons treated with eight weeks of immobilization in a plaster cast, reported that seven (13 percent) of the patients had a repeat rupture and only three patients were dissatisfied with the result. These results contrast sharply with those of Persson and Wredmark, who reported on twenty patients who were managed nonoperatively. Seven patients had a repeat rupture, and seven patients—not necessarily those who had a repeat rupture—were not satisfied with the result. Although function after nonoperative repair is generally good, the high prevalence of repeat rupture is considered unacceptable. A primary goal of the treatment of a rupture of the Achilles tendon is to avoid lengthening of the tendon, and this cannot be achieved with nonoperative treatment.

Recently, on the basis of results reported with use of functional postoperative bracing, McCormis et al. managed fifteen patients nonoperatively with a functional bracing protocol to repair a rupture of the Achilles tendon. They achieved good functional results, proving that, for selected patients, nonoperative functional bracing may be a viable alternative to operative intervention or to use of a plaster cast for the treatment of an acute rupture of the Achilles tendon.

Some patients, especially those who are elderly, may be seen with a long-standing rupture that has been discovered by chance. These patients often adapt well to the disability, but they are warned that an operation may be necessary if the symptoms caused by the rupture of the Achilles tendon worsen. Such patients are followed at regular intervals, but they usually do not need additional treatment.

**Physiological Effects of Immobilization**

After a Rupture of the Achilles Tendon

The cross-sectional area of a muscle is directly related to the muscle force that the muscle can develop.
and immobilization results in a profound alteration of the morphological and physiological characteristics of a muscle\(^{102}\). The soleus muscle appears to be particularly susceptible to the effects of immobilization, whereas the gastrocnemius, a biarticular muscle, is able to move when a below-the-knee cast is used and thus is less affected. The human soleus contains a high proportion of type-I muscle fibers\(^6\), which are particularly susceptible to atrophy if immobilized, as they are responsible for postural tone and are continually activated while the person is standing\(^{36}\). When the leg is immobilized, the muscle spindle relaxes and afferent impulses to type-I fibers cease, causing them to atrophy. The Achilles tendon is susceptible to the effects of prolonged immobilization\(^{102}\). Problems due to immobilization occur after open operative repair as well as after percutaneous repair, but not to the same extent. Häggmark et al.\(^{65}\) reported on fifteen subcutaneous ruptures of the Achilles tendon that were treated operatively and eight that were treated nonoperatively. They found a significant decrease in the circumference of the calf in the group that was treated nonoperatively (\(p < 0.01\)), whereas the group that was treated operatively showed no significant difference between the injured and the uninjured calf. Patients who have an open operative or percutaneous repair wear a plaster cast for less time\(^{21}\) than do those managed nonoperatively and on the whole are often more serious athletes who comply well with postoperative management regimens. The lack of tension on the immobilized musculotendinous unit is a major factor in the development of atrophy in the calf\(^{21}\). If optimum results from a repair of a ruptured Achilles tendon are necessary, the site of the repair should be put under as much tension as possible as early as possible\(^4\) and the cast should be changed regularly, decreasing the angle of plantar flexion as much as possible each time.

A viable alternative is immobilization of the foot and ankle in a plantigrade position\(^{102}\). This minimizes the number of changes of the plaster cast as well as the discomfort at the time of the changes when the ankle must be progressively dorsiflexed. It requires a sufficiently strong repair that can be subjected to early tensile stresses.

**Operative Compared with Nonoperative Treatment**

The results after operative treatment of ruptured Achilles tendons often have been compared with those after nonoperative treatment, but only a few well controlled, prospective, randomized trials have been conducted\(^{31,12,120}\). A major problem is the lack of a valid, reliable, reproducible protocol for subjective and objective evaluation of the results of repair of a ruptured Achilles tendon\(^{27}\).

Gillies and Chalmers\(^{4}\) measured plantar flexion strength after operative and nonoperative treatment of a rupture of the Achilles tendon. Little difference was found between the two groups, and the results of the operation were not sufficiently superior to warrant the risk associated with use of anesthesia and an operation. Inglis et al.\(^{44}\) followed forty-four patients who had been managed operatively and twenty-three who had been managed nonoperatively. No repeat ruptures occurred in the patients who had been managed operatively, but nine (39 percent) of the patients managed nonoperatively had a repeat rupture. Strength-testing revealed superior strength, power, and endurance after operative treatment, and the authors advocated the use of this type of management. Nistor\(^{120}\) conducted a prospective, randomized trial that included 105 patients; forty-five had operative management, and sixty-two had nonoperative management. The rate of repeat rupture in the group managed operatively was 4 percent compared with 8 percent in the group managed nonoperatively. However, there were a large number of secondary complications in the group that had an operation. The patients who were managed nonoperatively were found to have less absence from work, less ankle stiffness, and similar strength compared with the operatively managed group. Nistor recommended nonoperative management, as there were only minor functional differences between the two groups and the operation caused more complications in a study by Carden et al.\(^{9}\), seventy-six patients were managed nonoperatively and fifty-six were managed operatively. The patients who were seen less than forty-eight hours after the injury were compared with those who were seen more than forty-eight hours after the injury. The overall rate of complications was 4 percent in the group managed nonoperatively compared with 17 percent in the group managed operatively. The subjective results were also better in the group managed nonoperatively. The authors concluded that patients who are seen less than forty-eight hours after the injury should be managed nonoperatively, with eight weeks of immobilization in a cast, whereas patients who are seen one week or more after the injury should be managed operatively.

In a randomized trial, twenty-two patients who had operative management were compared with twenty-eight who had nonoperative management; both groups had functional treatment with a newly developed boot\(^{21}\). No significant differences were found between the groups with respect to either the functional results or the course of healing. The functional treatment in both groups allowed shorter periods of rehabilitation.

Kellam et al.\(^{90}\) performed a review of the literature and identified 609 patients who had been managed operatively and 208 who had been managed nonoperatively. The rate of repeat rupture was 1 percent for the patients who had had an operation compared with 18 percent for the patients who had had nonoperative management. Aiso, 83 percent of the patients managed operatively and 69 percent of those managed with immobilization in a cast returned to the preinjury level of activity. In addition, 93 percent of the patients man-
aged operatively and 66 percent of those managed non-operatively were satisfied with the result of treatment.

Cetti et al.\textsuperscript{31}, in a prospective, randomized study, reported on fifty-six patients who were managed operatively and fifty-five who were managed nonoperatively. The rates of complications and repeat ruptures were 9 and 5 percent, respectively, for the patients managed operatively compared with 16 and 15 percent, respectively, for the patients who were managed nonoperatively. The differences between the groups, however, were not found to be significant. The authors also conducted a review of the literature and identified 4597 ruptures of the Achilles tendon. Operative treatment of the ruptures was associated with lower rates of complications and repeat ruptures than was nonoperative treatment. Cetti et al. concluded that operative treatment was the method of choice, but nonoperative treatment was an acceptable alternative.

Recently, Lo et al.\textsuperscript{111} performed a quantitative review of all of the studies on the treatment of rupture of the Achilles tendon in the English-language literature published between 1959 and 1997 to determine the optimum treatment of acute ruptures. All of the identified articles were reviewed independently by at least three of the four authors to decide the eligibility of each study on the basis of predetermined criteria. Eligible studies were reviewed independently, and the data were extracted with use of standardized coding forms. Inconsistencies in data extraction were settled by discussion and majority vote. The main outcomes extracted were strength, time until the patient returned to work, frequency of return to sports, rate of repeat rupture, and complications. Complications were classified as major, moderate, or minor. Lo et al. identified a total of 742 patients who were managed operatively and 248 patients who were managed nonoperatively. The overall rate of repeat rupture was 3 percent for those managed operatively and 12 percent for those managed nonoperatively ($p < 0.001$). Although the rate of repeat rupture after operative treatment was lower than that after nonoperative treatment, the rate of minor and moderate complications associated with operative treatment was twenty times greater in some reports (Fig. 4).

In summary, treatment should be individualized according to the concerns and health of the patient. If optimum performance is necessary, operative management is probably the treatment of choice. Operative management should be used in athletes and in patients who have a high level of physical activity. Percutaneous repair should be considered for patients who do not wish to have open repair, possibly for cosmetic reasons or perhaps because they view an open operative repair as a more serious procedure\textsuperscript{160}. Nonoperative treatment should be reserved for older patients who are unlikely to derive any major benefit from an operative procedure and for patients who view an operation as an unnecessary risk.

**Treatment of an Older Rupture of the Achilles Tendon**

When patients are operated on within seventy-two hours after the injury, it is usually possible to suture the
stumps of the tendon in an end-to-end fashion\textsuperscript{121}. However, more than 20 percent of patients who have a rupture of the Achilles tendon may be diagnosed late\textsuperscript{122}. In these instances, young and middle-aged patients generally are first seen with a limp and severe impairment in the performance of day-to-day activities. When, after trimming, the tendon stumps cannot be approximated without undue tension (Fig. 5), the gap can be bridged with a single central gastrocnemius fascial turndown flap\textsuperscript{21} or with two such flaps (one medial and one lateral)\textsuperscript{110}. If it is available, the tendon of the plantaris longus can be used as a reinforcing membrane\textsuperscript{112}.

When a patient has an extreme rupture, in which the gap resulting from the rupture of degenerated tissue does not allow direct suture, the tendons of the tibialis posterior and peroneus brevis can be split longitudinally and used as pedicled transplants. The plantaris tendon is used as reinforcement, and the construct is augmented by a turndown flap of the superficial aponeurosis of the gastrocnemius\textsuperscript{52}.

More recently, Wapner et al.\textsuperscript{185,186} used a transfer of the flexor hallucis longus muscle and tendon to provide a dynamic repair. The procedure was performed on seven patients with a mean age of fifty-two years. After a mean of seventeen months (range, three to thirty months), there were no postoperative infections, loss of skin, or repeat ruptures. Each patient had a small loss in the range of motion of the involved ankle and hallux, but it was of no functional importance. All patients were satisfied with the functional result, although one needed a molded foot-ankle orthosis for prolonged walking\textsuperscript{119}.

When the gap between the ends of the tendon is such that the ends cannot be juxtaposed, the gap can be bridged by Marlex (monofilament knitted polypropylene) or Dexon (polyglycolic acid) mesh\textsuperscript{72,140}, and tendon graft can be used to cover the mesh. Postoperatively, the patients should be managed more conservatively, with restricted weight-bearing and immobilization in a plaster cast for eight weeks instead of six weeks.

Postoperative Care

Normally, patients are discharged on the day of the operation or the next day, after an orthopaedic physiotherapist has instructed them regarding the use of crutches\textsuperscript{111,125}. Patients are allowed to bear weight on the involved leg as tolerated, but they should be told to keep the affected leg elevated for as long as possible to prevent postoperative swelling\textsuperscript{121}. Patients are followed on an outpatient basis at two-week intervals, and the cast is removed six weeks after the operation\textsuperscript{119,121,125}. If the cast has been applied with the ankle in the equinus position, the cast is changed at two and four weeks, with the ankle placed in gradually increasing dorsiflexion until a plantigrade position is reached. The cast is removed altogether six weeks after the operation\textsuperscript{119}.

Patients are allowed partial weight-bearing and gradual stretching and strengthening exercises, increasing the frequency as tolerated, only after removal of the cast\textsuperscript{121}. Gradually, patients proceed to full weight-bearing at eight to ten weeks after the operation.

During the period of immobilization in the cast, patients are instructed to perform gentle isometric contractions of the gastrocnemius-soleus complex after weight-bearing has become comfortable\textsuperscript{121}. After removal of the cast, patients mobilize the ankle under the guidance of the physiotherapist. Two weeks after re-

\textsuperscript{1030} NICOLA MAFFULLI
moval of the cast, cycling and swimming are started and the mobilization exercises of the ankle are continued. Patients are prompted to increase the frequency of the self-administered exercise program. Patients are normally able to return to sports activities in the third or fourth postoperative month.

Postoperative Management of Achilles Tendons

Treatment that avoids immobilization of the ankle should be considered for athletes and well motivated, reliable patients. These patients are managed with an anterior below-the-knee plaster-of-Paris slab applied with the ankle in gravity equinus. Patients are discharged on the day of the operation or the day after, and they are allowed toe-touch weight-bearing on the involved limb as tolerated. They are instructed to keep the involved leg elevated for as long as possible. At fourteen to twenty-eight days after the operation, by which time the postoperative swelling (if any) has substantially decreased, the anterior below-the-knee plaster-of-Paris slab is changed to an anterior below-the-knee synthetic slab with the ankle in gravity equinus. The slab is kept in place by an elastic bandage, which allows plantar flexion of the ankle, while dorsiflexion is limited by the foot-piece of the slab. Patients are allowed weight-bearing as tolerated, with use of crutches. The slab is changed at the second and fourth postoperative weeks, so that the ankle can dorsiflex to neutral by the fourth postoperative week. The limitation of dorsiflexion is continued for a total of six weeks, at which time the slab is removed. High-level, well motivated athletes who comply with the postoperative protocol normally are able to return to sports activities six to eight weeks after the removal of the anterior slab. However, it is more expensive than a simple synthetic cast, although it is reusable.

Solveborn and Moberg prospectively studied seventeen consecutively managed patients (fifteen men and two women) who had operative repair of a subcuteaneous, complete acute rupture of the Achilles tendons. The patients were managed with a new postoperative regimen that allowed free motion of the ankle in a patellar ligament-bearing cast that had a protective frame under the foot to permit weight-bearing immediately after the operation. Early free motion of the ankle after repair of a ruptured Achilles tendon proved to be safe, with satisfactory clinical results.

Overview

The etiology of what would appear to be a simple condition still has not been completely clarified. Despite extensive investigation, few definitive answers have been found. The lack of study of human tissue is an important problem, but it is difficult to see how this could be overcome. It is notable that animal models of rupture of the Achilles tendon have relied mostly on tenotomy and. The model developed by Barfred, hardly reflects clinical practice and has not found wide application.

The treatment of acute rupture of the Achilles tendon depends on the preference of the individual surgeon. Open operative repair probably produces better functional results than nonoperative management does, but it may lead to a higher rate of postoperative complications. Nonoperative management may result in a poorer functional result, but the problems of postoperative complications are avoided.

A major problem has been the lack of a universally accepted system for scoring results of treatment of a ruptured Achilles tendon. Leppilaiti et al. proposed a scoring scale that included subjective assessment of clinical factors and isokinetic evaluation of strength. However, it has been used only by those authors, and isokinetic dynamometry is time-consuming, expensive, and not widely available.

If the reports that have described a rising incidence of rupture of the Achilles tendon are accurate, the field of operative repair of the Achilles tendon will become an increasingly important one for orthopaedic surgeons. Future developments may include the use of adhesives in operations on the tendon, an understanding of the role that cytokines play in tendon-healing also may lead to new treatments, possibly based on gene therapy. However, such novel interventions are unlikely to be in routine clinical use for some time.

References


RUPTURE OF THE ACHILLES TENDON 1033


RUPTURE OF THE ACHILLES TENDON


VOL. 11-A, NO. 7, JULY 1999