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An evaluation of tendon healing using magnetic resonance imaging and a standard physical assessment

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Yale University

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AN EVALUATION OF TENDON HEALING USING MAGNETIC RESONANCE IMAGING AND A STANDARD PHYSICAL ASSESSMENT

Victoria Lynette Barber

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AN EVALUATION OF TENDON HEALING USING MAGNETIC RESONANCE IMAGING AND A STANDARD PHYSICAL ASSESSMENT

A Thesis Submitted to the Yale University School of Medicine in Partial Fulfillment of the Requirements for the Degree of Doctor of Medicine

by
Victoria Lynette Barber
1990
To my parents, for giving me the courage to follow my dreams.
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# Table of Contents

Abstract i

Review of the Literature 1

Anatomy and Physiology of the Achilles Tendon 1
Pathologic Changes Affecting the Achilles Tendon 6
Diagnosis of Achilles Tendon Abnormalities 15
Treatment of Achilles Tendon Abnormalities 20

Methods and Materials 27

Patient Population 27
Methods 27
Surgical Technique 28
Postoperative Evaluation 29

Results 33

Subjective Evaluation 33
KINCOM Results 34
Magnetic Resonance Imaging 37

Discussion 41

Conclusions and Summary 55

Tables 58

Figures 65

Appendix 1 73

Appendix 2 77

References 78
ABSTRACT

An Evaluation of Tendon Healing Using Magnetic Resonance Imaging and a Standard Physical Assessment
Victoria Lynette Barber
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The healing process of Achilles tendon injuries was prospectively assessed with magnetic resonance imaging and an objective strength test. Nine patients were studied, six males and three females. Five of the patients sustained sudden complete rupture of the Achilles tendon, one patient was diagnosed as having a partial tendon tear and the remaining three patients presented with complaints suggestive of Achilles tendinitis. Each of these patients were further subdivided based on the treatment protocol followed (i.e. surgical versus nonsurgical intervention). Patients were assessed by established standards of physical examination. Intensive rehabilitation services were provided at the Gaylord/Yale New Haven Hospital Rehabilitation Center Incorporated, New Haven Connecticut.

The effectiveness of the rehabilitation process was evaluated utilizing three independent methods. First, the KINETIC COMMUNICATOR (KINCOM) device objectively analyzed the planter strength-generating-capabilities of individuals at various stages of rehabilitation. Maximal eccentric and concentric force output comparing the injured to the unaffected extremity was evaluated. Second, the Hewlett-Packard 5316-A
Universal Counter was utilized to study the return of normal Achilles tendon reflex response times. Third, serial Magnetic Resonance Imaging (MRI) was performed on individuals in each treatment group.

KINCOM data showed an equilibration of force production between the two extremities as tendon healing progressed. Universal Counter demonstrated a progressive decrease in the variation of response time between the injured and control extremity over time. MRI, which demonstrated increased signal intensity within injured tendon, showed progressively decreasing signal intensity with tendon healing.

Taken in conjunction, MRI, KINCOM, and the Plantar reflex data demonstrate the slow reparative process of injured tendon. Abnormal MRI signal persisted months after KINCOM and plantar reflex testing returned to normal indicating continued reorganization of the tendon substance. In conclusion, functional integrity of the injured Achilles tendon is restored months or years prior to completion of the healing process.
REVIEW OF THE LITERATURE

Anatomy and Physiology of the Achilles Tendon

The Achilles tendon is the common tendon of the gastrocnemius and the soleus muscles. These two muscles are often referred to as the calf muscles or the triceps surae. The Achilles tendon receives muscle fiber attachments from the soleus until a few centimeters from its insertion onto the calcaneus. It has been noted that these two muscles contribute separately to form the Achilles tendon, and that their individual contribution tends to vary among individuals. The gastrocnemius portion varies from twenty-six to eleven centimeters in length whereas the contribution of the soleus ranges from eleven to three centimeters (12).

The Achilles tendon which is initially flattened and fan-shaped, becomes rounded as it approaches its calcaneal insertion. There are two bursa - the subcutaneous bursa and the retrocalcaneal bursa - which serve to protect the tendon at its insertion. As it nears the calcaneal insertion, the Achilles tendon expands slightly coincidentally converting to fibrocartilage as it widens. The fibrocartilage results in increasing stiffness of the tendon as it progresses from origin to insertion. Barfred (1971) postulated that this stiffness functioned to protect the Achilles tendon from stresses of oblique traction (12).
Another interesting feature of the Achilles tendon is its tendency to twist as it descends. The tendon tends to rotate laterally. The rotation begins approximately twelve to fifteen centimeters above the calcaneal insertion, the region where the contribution of fibers from the soleus generally begins. The degree of rotation is dependent upon the amount of fusion between the gastrocnemius and the soleus muscles. The greater the fusion that exists between these two muscles, the less rotation observed in the tendon (12).

In 1946, Cummins et al. described three patterns of rotation: type I, where the gastrocnemius contributes two-thirds of the posterior part and the soleus one-third; type II, where the gastrocnemius and the soleus each contribute one-half; and type III, where the soleus makes up two-thirds and the gastrocnemius makes up the remaining third. Type I is the most common (52%) followed by type II (35%) and type III (13%) (12).

The twisting produces areas of stress in the tendon caused by a "sawing" action of one part of the tendon against the other. This effect was noted to be greatest in the region two to six centimeters above the tendon insertion. It should be noted that this portion of the tendon has the poorest blood supply and is also the most common site for Achilles tendinitis (12).

The Achilles tendon is surrounded by a sheath called the epitenon. This sheath continues into the interior of the tendon as the endotenon. The paratenon, which is a thin filmy layer of areolar
tissue covering the epitenon, acts as an elastic sleeve to allow free movement of the tendon within the surrounding tissue (12).

Investigation by Mayer (1916) suggested that the tendon receives its blood supply from three sources: 1) from muscular branches; 2) from vessels running in the surrounding connective tissue; and 3) from vessels of the bone and periosteum near the point of tendon insertion. Edwards (1946) noted that vessels supplying tendons were arranged in longitudinal channels surrounding the collagenous bundles making up the tendon. He proposed that each channel contained one artery and two veins. He additionally noted anastomotic connections between neighboring venous systems (46). In their study in 1959, Lagergren and Lindholm evaluated vascular distribution in the Achilles tendon utilizing angiographic and microangiographic techniques. They discovered that both the proximal and distal portions of the tendon were well supplied. The blood supply derived along the length of the tendon from the underlying mesotenon was noted to be most important. It was also discovered that the portion of the tendon two to six centimeters from the insertion is supplied by a smaller number of vessels as compared to the remainder of the tendon (46). A recent study by Carr and Norris (1989) supported these results. However, in addition to a reduction in the absolute number of vessels present in this mid-tendon region, Carr and Norris noted a decrease in the mean relative area of vessels in this location. This region has historically been the site of a predominance of injuries involving the
Achilles tendon, especially spontaneous tendon rupture and tendinitis. It seems likely therefore that decreased nutrition in the tendon compromises its ability to withstand stressful insult and thereby predisposes to injury (8).

Tendons must be able to withstand tensile forces. In order to fulfill this function, tendon fibers are aligned in the axis of the forces generated by contracting muscles. In areas where tendon must "absorb" large forces, fibers are usually oriented in a longitudinal direction in relation to their accompanying muscle bellies. These tendons are able to store some of the force generated by muscle contractions for brief periods of time (12). Generally, tendon is stronger than bone around its osseous insertion, and the weakest at the tendomuscular junction. This rule however does not apply to the Achilles tendon (10). Due to the twisted orientation of the Achilles tendon as it descends, areas of stress are generated as one part of the tendon saws across another despite the apparent longitudinal axis of tendon fibers. This sawlike action is compounded by the fact that the tendon is not homogeneous, but receives interdigitating fibers from the soleus.

Although the ankle is a hinge joint with motion only in the sagittal plane, forces applied to the Achilles tendon produce motion in more than one plane. Motion at the subtalar joint is directed in the frontal and transverse planes, producing inversion-eversion and abduction-adduction movements respectively. Movement in the transverse plane results in twisting and untwisting of the Achilles
tendon. Inversion and eversion at the subtalar joint may produce unequal forces across the tendon resulting in a bowstring deformity of the Achilles tendon (12).

Numerous authors have attempted to estimate the amount of force generated through the Achilles tendon under varying conditions. Estimates range from 1962 to 2354 Newtons in walking to between 3924 and 5886 Newtons in running and up to 8729 Newtons in fast running. Cronkite (1935) appreciated the existence of a large variation in the tensile strength of the Achilles tendon. This variation is evident upon examination of the varying magnitude of force creating identical injury in individual patients (47). The stress across the tendon can be calculated by dividing these values by the tendon's cross-sectional area. The maximum value for the amount of stress tolerated by the mammalian tendon is estimated to be 49 to 98 MPa (Elliot, 1965). McMaster, in his animal study, showed that half of the fibers of the gastrocnemius tendon had to be destroyed before the tendon breaks under violent tension (21).

The above values were largely derived from animal studies in which isolated tendon specimens were elongated at a constant rate until failure was evident. Stresses applied to the Achilles tendon during exercise are thought to exceed these estimates. Additional factors such as heel varus and valgus, tibial torsion or excessive tightness of the gastrocnemius or soleus may lead to increases in tensile forces generated across the Achilles tendon (12).
Pathologic Changes Affecting the Achilles Tendon

Given the great forces generated through the Achilles tendon, it is not surprising that pathological changes may develop within the tendon substance. Perhaps largely resulting from recent increases in participation in competitive and recreational sports, overuse syndromes are frequently noted. The most common overuse syndrome in the lower extremity involves the Achilles tendon (56). Tendinitis or peritendinitis, tendinosis and partial or complete rupture of the Achilles tendon represent the spectrum of the most commonly reported abnormalities.

Inflammation of the Achilles tendon and its contiguous structures is one of the most common overuse injuries seen in sports medicine clinics, accounting for eleven percent of all lower extremity complaints in one large runners' clinic (71). Lawrence et al (1955) noted that injury to the Achilles tendon comprised approximately one-fifth of all the large tendon injuries encountered (47).

Several etiologies for Achilles tendinitis have been described. The Achilles tendon itself may become involved as a result of repetitive mechanical stresses of running or other athletic endeavors. Schepsis and Leach (1985) proposed that these inflammatory changes within the tendon resulted from numerous microtears causing localized degeneration or fibrosis. Chronic inflammatory tissue often develops secondary to incomplete healing.
In some cases, areas of necrotic tissue culminated in the development of calcifications within the tendon (71).

The tendon sheath, or mesotenon, is also subject to inflammatory change with overuse. The mesotenon may become thickened and fibrotic as a result of repetitive stresses. This pathological variant, termed paratendinitis or tenosynovitis by many authors, can occur in isolation or in conjunction with tendon disease. Clement et al (1984) studied one hundred nine runners with complaints suggestive of overuse injury to the Achilles tendon. Within their series it was noted that the susceptibility of runners to Achilles tendon injury is related to:

- eccentric loading of fatigued muscle,
- excessive pronation producing whipping action of the Achilles tendon and or vascular blanching of the tendon by simultaneous internal and external rotary forces imparted to the tibia by pronation and knee extension (11).

Kvist et al (1988) in their study of eleven athletes with chronic Achilles tendinitis, examined the histological changes observed in this disease entity. The paratendineal fatty areolar tissue was found to be thickened and edematous in this patient population. Adhesions, which were present in the diseased tendons, were not present in the normal tendons studied. In addition, fat necrosis, connective tissue proliferation as well as a diminution of blood vessel were observed in the affected tendon. Histologic and
immunofluorescent examination further revealed the presence of fibronectin and fibrinogen within the proliferating connective tissue and vascular walls (42). Fibronectin, along with fibrin, functions as a scaffold for migrating cells during tissue repair. Kvist et al also noted an increased amount of ground substance, Type I and III collagen fibrils as well as an increased number of elastic microfibrils in their patients with Achilles tendinitis (42,77).

Nelen et al (1989) described three stages of Achilles tendinitis. In the first stage, inflammatory changes were confined to the peritenon with associated thickening of the peritenon tissue. During this phase, the actual tendon appears normal in contrast to the next stage in which inflammatory changes are evident within the tendon proper. Macroscopic examination of these stage two changes, termed tendinosis, may reveal nodular thickening or a glossy appearance of the tendon (56).

Histological abnormalities corresponding to tendinosis include peritendinous thickening as well as areas of mucoid or lipoid degeneration, fibrinoid necrosis and tearing of tendon fibers. Kvist et al (1979) in their study of biopsy specimens of patients with Achilles tendinitis noted the presence of certain enzymes in the degenerative tendon. Lactate, succinate, glucose-6-phosphate, glutamate dehydrogenases, NADH and NADPH-dehydrogenases, and, acid and alkaline phosphatases were among the enzymes discovered. These findings were suggestive of high catabolic activity and elevated energy metabolism via anaerobic pathways within the
diseased tendons studied (43,44). The presence of acid mucopolysaccharide-rich ground substance and haphazardly arranged cells and fibrils in the ground substance have also been described as early degenerative changes in diseased tendon (18). Evidence of proteoglycan modifications and variation in gycoprotein content observed in healing animal tendon have helped to further outline morphological and physical modification of the tendon tissue over time (9). Histochemical evaluation of the tendons demonstrated evidence of increased catabolism and decreased oxygenation in the inflamed paratenon. The second stage of tendon degeneration may also be marked by capillary and histiocyte proliferation and invasion into the tendon substance.

The final stage is distinguishable by the presence of a macroscopically noticeable disruption of the tendon. This final stage is especially common in association with prior use of local steroids (56). Collagen fibrils which compose tendons are arranged in a wavy course known as "crimping". It has been observed that anabolic steroids produce an increase in the crimp angle and a decrease in crimp length which alters normal tendon biomechanics and perhaps predisposes to tendon rupture (82).

Clinically, tendinitis and peritendinitis of the Achilles presents with pain which is often localized to the region two to six centimeters proximal to the calcaneal insertion. The pain is often increased by activity and may be associated with morning stiffness (49). Physical findings upon examination include; thickening of the
tendon or peritendinous structures, edema and crepitus (11,49). The crepitation is due to the fibrin that is precipitated from the fibrinogen-rich edema fluid (42). Limited dorsiflexion has been cited as a common variant in symptomatic patients (11).

Another injury affecting the Achilles tendon is partial or complete rupture of the tendon fibers. Only sixty-six cases of Achilles tendon rupture are reported in the literature prior to 1929. Indeed, Achilles tendon rupture continued to be an uncommon injury up to the 1950's. During the last few decades however, the diagnosis of Achilles tendon rupture has increased in most civilized countries (36).

Although antecedent complaints have been reported in ten to twenty percent of individuals, the majority of patients experience sudden rupture without prodromal symptoms. Haldemann and Soto-Hall stated that "the accuracy of the diagnosis one associates with fractures is lacking when the injury involves muscles and tendons" (10). Active plantar flexion produced by muscles other than those acting through the tendo calcaneous - namely the tibialis posterior, peronei, flexor digitorum and plantaris muscles - mimic the effects of an intact Achilles tendon complex and thereby can mislead examiners (10,30). It has been reported that the diagnosis of Achilles tendon rupture is missed about twenty-five percent of the time, leading to a delay in treatment (31,47).

The differential diagnosis of the injury is fairly extensive and includes ankle sprain, fractures of the malleoli or calcaneus,
tendinitis, peritendinitis, calcaneal bursitis, phlebitis and intermittent claudication. In one study the most frequent incorrect diagnosis was found to be a partial tear of the Achilles tendon or an ankle sprain in forty-two patients later diagnosed as having complete tendon ruptures (10).

Most published reports support the contention that Achilles tendon ruptures are most common in men between the ages of 30 and 50 years of age (2,36,47). Lawrence et al reported this mean as 40 years, while Jozsa found the mean age of patients with Achilles tendon ruptures to be 35.2 years. In addition it has been noted that there exists a high incidence of the blood group O among patients with Achilles tendon ruptures. In cases of multiple ruptures and reruptures, Jozsa quoted the frequency of blood group O as seventy-one percent (35). Hooker (1963) observed that the left side is affected much more than the right. He postulated that this finding is related to a predominance of pushing off on the left leg as opposed to the right (30).

The exact pathogenesis of Achilles tendon rupture remains obscure. Boyd (1938) proposed that syphilis, tuberculosis, pyogenic infections, tumors and cysts predispose to Achilles tendon ruptures (10). Beskin et al (1987) in their study of Achilles tendon rupture, noted a high incidence of gout in their population- 14.3% compared to 0.3% in the normal population. Two theories have however have been frequently propose in the recent literature. The first suggests that chronic degenerative changes in the tendon substance is the
mechanism by which rupture occurs (2). Fox (1975) supported the contention that devitalization and fibrosis of the Achilles tendon played significant a role in rupture (22). It has been noted however that the value of degenerative changes seen in ruptured tendon tissue at operation is minimal given the time elapsed between injury and suture in many cases (10). Additionally, given the fact that tendons degenerate as part of the normal aging process, one would expect a high incidence of rupture in older age groups. As already stated, the vast majority of Achilles tendon ruptures occur in individuals before the age of fifty years. Hooker (1963) noted that in some cases of tendon rupture no evidence of degeneration was observed (30).

The second theory suggests that the primary cause of Achilles tendon rupture is malfunction of the normal inhibitory mechanism of the musculotendinous unit. This inhibitory unit functions to prevent tendon failure from excessive or uncoordinated muscle contraction. The importance of coordinated muscular actions was appreciated by Christensen (1954) who also proposed that a constitutional weakness of the common tendon in conjunction with failure of coordination predisposed to rupture (10). Hypotrophy of the Achilles tendon may weaken the tendon substance and may also predispose to rupture (27).

Achilles tendon ruptures often occurred in recreational sports activities (59%), in contrast to other tendon injuries (2%) (36). This has been supported by numerous authors in the orthopaedic
literature (2,10,30,30,47,80). In a retrospective study of Achilles tendon injuries, it was reported that 592 out of 799 Achilles tendon ruptures (74%) resulted from participation in athletic activities. Christensen postulated that lack of training played an important role in the development of Achilles tendon ruptures. In his series of 26 ruptures sustained during exercise, 13 of the patients had recently resumed training after a long interval (36).

Three main causes of Achilles tendon rupture have been described by Hooker. The first involves the sudden stretch or strain of an already taut tendon as frequently seen in jumping, walking or running (30). The demand placed on the Achilles tendon are maximized at a point approximately 75% through the stance phase of gait or during running or jumping activities. Approximately five times body weight is necessary to equilibrate the forces at the ankle (2). During the last part of gait, extension of the knee results in a vigorous pull on the tense gastrocnemius which is transmitted to the ankle joint by way of the Achilles tendon (10,70). Sudden passive dorsiflexion stretching a relaxed tendon, as in slipping backwards over a ledge, may also result in Achilles tendon rupture. Finally, a direct blow over a taut tendon by a falling object has also been a frequently cited cause of Achilles tendon rupture (30).

Clinically, Achilles tendon rupture is described by most patients as a sudden snap in the lower extremity just above the heel. The injury is usually accompanied by pain and weakness in the ankle. Some studies have reported cases in which patients experienced
little or no pain despite the presence of complete tendon rupture (10,30). Those patients who do experience pain describe it as sharp or stabbing in nature.

Often a palpable gap develops secondary to retraction of the proximal end of the tendon. Usually this gap does not exceed six centimeters, but it may vary between one and twelve centimeters. The ruptured tendon ends are markedly frayed and have been compared to a torn rope (10). Following disruption of the tendon substance, hematoma develops which may obscure the detection of a tendon gap on physical examination. As hematoma and edema resolve, the gap may once again become visible to the examiner. Light and electron microscopic evaluation of the tendon gap has revealed the presence of newly formed tissue consisting initially of connective tissue cells of variable shape dispersed in an abundant intercellular matrix (64). Later this regenerative tissue becomes more compact, closely resembling the morphological features of the original tendon tissue (64,78).

Post injury, dorsiflexion of the foot produces pain and serves to emphasize the flattened contour of the tendon (10). Although the patient may be able to walk, great unsteadiness of gait is observed. Specifically, the patient experiences difficulty with the take off phase of walking as plantar flexion, if present at all, is minimal.


Diagnosis of Achilles Tendon Abnormalities

The diagnosis of Achilles tendon abnormalities are largely based on history and physical examination. In tendinitis and peritendinitis of the Achilles tendon, diffuse pain in or surrounding the tendon, inflammation and thickening of the peritenon and crepitus upon plantar flexion and dorsiflexion are often appreciated by the examiner (11). In addition, many patients complain that the pain is aggravated by walking or running on inclines and stairs. The complaint of morning stiffness of the affected ankle is also consistent with the diagnosis. In addition most symptomatic patients with tendinitis have some limitation of passive foot dorsiflexion (49).

Patients diagnosed with Achilles tendon ruptures present with the swelling and edema around the ankle seen in tendinitis, but there is an associated visible or palpable gap in the tendon substance. Other physical findings associated with Achilles tendon rupture include swelling of the calf, and loss of the push off phase of gait as a result of inadequate plantar flexion.

In 1938 Kager described what has since been termed Kager’s triangle or the Achilles fat pad. The space is filled with fatty tissue bounded by the margins of the Achilles tendon, the calcaneus and the deep flexor muscles. Kager noted that this triangular space loses its normal configuration when the Achilles tendon is ruptured.
Later Toygar suggested that the measurement of the angle of the posterior skin surface as observed on roentgenogram be used as a diagnostic tool. He proposed that Achilles tendon ruptures resulted in an anterior displacement of the fragmented tendon ends leading to an increase in the normal angle. Toygar considered a decrease in Kager's triangle and an increase in the posterior skin surface angle to between one hundred thirty and one hundred fifty degrees to be virtually diagnostic of Achilles tendon rupture. In their series of 39 patients however, Arner et al found that the deformation of Kager's triangle was very nonspecific and that Toygar's sign rarely was positive (10).

A more recent diagnostic test is the Thompson squeeze test first introduced by Thompson and Doherty in 1961. They found that when pressure is applied around the middle third of the calf in normal individuals, plantar flexion of the foot is observed. This plantar response was absent in cases of ruptured Achilles tendons (79). O'Brien (1984) described an additional nonoperative technique to diagnose acute Achilles tendon ruptures. The needle test involves the insertion of a 25-gauge needle into the substance of the Achilles tendon. The foot is then alternately plantar flexed and dorsiflexed. Observation of swiveling of the needle upon manipulation of the foot indicates that the tendon is intact throughout its distal ten centimeters. Absence of swiveling or only slight movement corresponding to the movement of the skin indicates a loss of
continuity of the Achilles tendon between its insertion and the position of the needle (61).

The history and physical examination form the most important basis for diagnosis, but when additional information is required, clinicians often rely on medical imaging. Sonography or ultrasound has been utilized to evaluate tendons for the presence of abnormalities. The extremities are particularly well suited to ultrasound evaluation because of the lack of interference from other tissue structures, relatively small amount of adipose tissue and the superficial location of structures of interest (37).

Based on ultrasound studies, the Achilles tendon in adults is estimated to be between four and six centimeters in thickness (21,28). Normally it has well defined echogenic margins with fine parallel internal echoes. Discontinuity of the tendon with focal swelling and changes in echogenicity can be appreciated by ultrasound in patients with complete or partial Achilles tendon ruptures. Focal enlargement and nodularity of the tendon may be observed in Achilles tendinitis. If peritendinous structures are also involved, ultrasound evaluation reveals thickening and decreased echogenicity of the peritendinous sheath or fluid in the peritendinous bursa. The distinction between marked Achilles tendinitis and a soft-tissue mass is often made with the aid of ultrasound techniques (21,28). Ultrasound can also be used to document calcification or effusions within the tendon substance. Tenosynovitis is the easiest diagnosis to appreciate with ultrasound
evaluation. Fluid accumulates in the subcutaneous bursa and continues cephalad to invest the tendon (3). Real time equipment allows dynamic evaluation of the tendon during contraction and relaxation of the muscle, which is useful in the diagnosis of tendon abnormalities.

Acute tendon ruptures viewed on ultrasound appear as focal lucencies in the tendon (3). Ultrasound is also useful in following Achilles tendinitis as well as in needle aspiration of the fluid surrounding the tendon. This latter function is used to determine if an area of necrotic tendon contains clear or hemorrhagic fluid. The former is more commonly seen in tenosynovitis whereas the latter may indicate tendon rupture (3).

Computed Tomography (CT) scanning has also been used to evaluate pathological variants in the Achilles tendon. Enlargement of the tendon or masses about the tendon often obliterate the Achilles fat pad. CT is capable of differentiation between swelling, calcification, granuloma and tears of the tendon. CT has also been utilized to follow the post-operative course of surgically repaired Achilles tendons. Achilles tendon sheath scarring and soft tissue neoplasms are sometime discovered through CT scanning (75). Disadvantages of Ct include radiation exposure, direct imaging limited to the axial plane, decreased resolution, and increased radiation dose with reformatted multiplanar images (1).

Like Ultrasound and CT, magnetic resonance imaging (MRI) more often functions to determine whether treatment should be
operative or nonoperative. MRI provides a high degree of contrast between different kinds of soft tissue. The dominant tissue type observed in magnetic resonance images of the extremity are muscle and adipose tissue which form a background for other tissue such as bone and tendon (15,67). T1-weighted images give excellent anatomical detail of high signal structures such as fat and bone marrow. T2-weighted images are highly sensitive for detecting pathological changes such as edema, hemorrhage and tumor (38).

Magnetic resonance imaging has been utilized by many investigators to study abnormalities of the Achilles tendon. The normal Achilles tendon has a flat to concave anterior margin and rounded medial and lateral aspects on MR images. Tendons usually demonstrate diminished signal intensity on all MR imaging pulse sequences and therefore contrast well against surrounding high intensity fat. Any increase in tendon signal is regarded as abnormal. Tendinitis is manifest by small focal areas of abnormally increased signal associated with slight widening of the tendon. Complete and partial tendon tears are characterized by gross areas of signal intensity, discontinuity of the tendon edges and retraction of the muscle-tendon junction up into the calf (14). Uncomplicated surgical repairs when seen on MRI appear as areas of tendinous continuity with inhomogeneous signal in the operative site (65).

The axial plane is used to compare the right to the left sides of the tendon and is valuable in detecting subtle differences in thickness. The sagittal plane is capable of demonstrating
longitudinal tendon tears. Several authors have noted the importance of positioning - namely dorsiflexion of the foot - during MR imaging. Failure to dorsiflex the foot during MRI evaluation will result in pseudothickening and buckling of the ruptured tendon (7,14).

Advantages of MR imaging over other available imaging techniques include excellent depiction of anatomical detail, superior contrast resolution, and the potential for multiplanar imaging (1). Disadvantages of MR imaging cited by Beltran (1987) were not inconsequential. The test is very expensive and is not yet widely available. The absence of signal from calcified soft tissue is another disadvantage. The characterization of different types of fluid is also not possible with MRI evaluation (1).

Treatment of Achilles Tendon Abnormalities

The vast majority of cases of Achilles tendinitis are managed by nonsurgical means. Initially many authors advocate a reduction in athletic activities or total rest to decrease heel pain. Cast immobilization is utilized in cases where complete rest of the tendon is necessary for successful treatment. However Leach and his colleagues did not recommend cast immobilization. They determined that limb immobilization leads to impaired function in all immobilized joints, tendons, ligaments and muscles (49). Decreases in muscular strength, increased fatigability during exercise, and increased rate of atrophy were commonly observed in
individuals following immobilization (5,74,81). Enwemeka et al also include joint stiffness, ulceration of the joint cartilage, osteoarthritis, skin necrosis, infection, tendocutaneous adhesions and thrombophlebitis among their list of complications of cast immobilization (16,17). Booth applied casts to the hind limbs of rats in order to duplicate joint immobilization in humans. He noted that atrophy began after a lag of one to three days in those skeletal muscles fixed at lengths equal to or less than resting lengths. He further observed that the loss of muscle mass and protein was greatest in the next five days (5).

Rothman investigated the effect of immobilization on the vascular bed in tendons. The average oxygen consumption of the healthy tendon was found to be 0.1 microliter of oxygen per milligram dry weight per hour and the average blood flow was estimated to be 0.10 cubic centimeters per gram per minute. The capillary bed in the tendon of the immobilized limb showed an average of $1.125 \times 10^{-3}$ milliliters per gram tendon versus $1.846 \times 10^{-3}$ milliliters per gram tendon demonstrated in the controls (69).

Adjunctive therapy such as heel lifts, oral antiinflammatory agents and ice massages are also advocated by many clinicians (71). In refractory cases, local steroid injection may become part of the conservative therapy regimen. However steroid injection into the inflamed or damaged tendon may retard the natural repair process leading to a weakening of the tendon substance and predisposing to tendon rupture(49,82).
Operative treatment for Achilles tendinitis is reserved for individuals who fail more conservative treatment options. In cases of Achilles tenosynovitis or tendinitis, the tendon sheath is usually found to be hyperemic, thickened, fibrotic and often is adherent to the underlying tendon. Discovery of a necrotic focus or partial tear within the tendon is not an infrequent occurrence in cases of longstanding tendinitis. When found these areas are debrided and the tendon fragments are reapproximated. Retrocalcaneal bursitis, which may mimic the symptoms of tendinitis, is usually associated with a prominent posterior superior tuberosity of the os calcis which is removed at the time of operation.(71).

Treatment of Achilles tendon ruptures was predominantly nonsurgical until the twentieth century. During this period various means of immobilization were used including strapping, wrapping and braces for various periods of time. The success of these conservative treatment measures was limited. Beginning in the 1920's, surgical treatment increased in popularity. The work of Arner and Lindholm (1959) and the development of modern surgical techniques contributed to increased use of this treatment modality (80). Despite the interest in surgical repair of Achilles tendon ruptures, many clinicians continued to strongly advocate a more conservative approach to this injury.

Proponents of a nonsurgical approach to Achilles tendon rupture state that the final outcome of a surgical versus a nonsurgical treatment protocol is not statistically different.
Gillies and Chalmers concluded that operative repair in fresh Achilles tendon ruptures, given the risk factors involved, offered results which were not significantly superior to those achieved by conservative management (24). Frequently cited complications of surgical repair include; anesthesia risk, pulmonary embolism, wound infection, skin necrosis and sinus tract formation (24,48). In one study, the complication rate for surgically treated ruptures was demonstrated to be 20% versus a 10% complication rate seen in patients managed conservatively (80).

Advocates of conservative treatment frequently cite data indicating the intrinsic healing property of tendon irrespective of the treatment modality employed (10). Tendon healing can be divided into two stages. The first stage, the connective tissue stage, is marked by proliferation of connective tissue on and surrounding the separated tendon ends forming a scar. This stage terminates the fourteenth to sixteenth day. The second stage, termed the formative stage, is dominated by conversion of tendon tissue callus into mature tendon substance. Evidence of tendon cell proliferation, though present of the fourth or fifth day, becomes dominant on the fourteenth day following injury (54).

Lea and Smith among others noted that this healing process occurred regardless of the treatment strategies utilized. Investigators have successfully demonstrated the ability of the tendon to heal itself using animal models. Lipcomb and Wakim discovered that the Achilles tendon of rats when sectioned, would
spontaneously heal itself and become almost completely normal. Niebauer, supporting this view, found that the Achilles tendon in dogs reconstituted itself after disruption of tendon fibers (48). It has been demonstrated during surgical repair that simply placing the foot in a full equinus position closely approximates the ends of the separated tendon. Thus supporting the contention that cast immobilization is an effective treatment for Achilles tendon ruptures (24). Proponents of conservative treatment have argued that the decrease in morbidity (ie. less time lost from work) as well as the avoidance of the expense of hospitalization make nonsurgical treatment the treatment of choice (48).

Generally conservative treatment of Achilles tendon ruptures involves cast immobilization in slight plantar flexion for six to eight weeks. This is followed by braces, heel wedges and/or crutches to decrease stress on the healing tendon. Rehabilitation also involves gradual stretching of the gastrocsoleus-Achilles apparatus to avoid permanent limitation in range of motion. It should be stated that the tendon tends to lengthen as it heels (48,60). Therefore care must be taken to avoid excessive dorsiflexion during immobilization. Numerous authors have also noted that following injury the healing Achilles tendon hypertrophies and can be visualized on physical examination to be wider than the tendon on the uninvolved side. This phenomenon however is not unique to nonsurgically treated ruptures but is also commonly demonstrated in tendons following surgical repair.
Finally, studies have demonstrated that excellent results from surgical repair can be obtained up to one year after injury (6). Therefore, initial nonsurgical measures can be instituted in cases of rupture and surgical intervention can still be attempted at a later date should the former treatment fail.

Various methods of surgical repair of the Achilles tendon have been described in the literature (2,23,26,50,51,52,55,57,60,68,72,80). Advocates for surgical repair of the Achilles tendon have found that the tendon is stronger following operative repair than after nonsurgical intervention. In 1976 Inglis et al used a Cybex II isokinetic dynamometer to evaluate planter flexion strength, power and endurance in 30 cases of early surgical repair, 17 cases of late surgical repair and 14 cases of total rupture treated nonsurgically. Comparing the surgically treated and nonsurgically treated groups, Inglis noted a statistically significant difference between the two groups. The strength of the surgical group was 101%, power averaged 88% and endurance 91%. The nonsurgically treated group had averages of 73%, 62%, and 64% respectively (31). The increase in strength in the operative group is believed to be related to the ability to maintain the appropriate musculotendinous tension and prevent significant muscle atrophy as well as the ability to accurately obtain the correct tendon length during surgery (39).

Advocates of surgical intervention also have demonstrated that 83% of patients treated surgically versus 69% of those treated
by cast immobilization were able to a return to their preinjury level of activity. Kellam et al noted that 94% of the surgically treated patients in their study returned to the same level of activity as before their injury (39).

Another factor supporting surgical versus nonsurgical treatment of Achilles tendon ruptures is based on comparisons of rerupture rates in these two groups. Nistor found that out of 2647 ruptures treated surgically, 2% had reruptures (60). The rerupture rate for the conservatively treated group in contrast was estimated to be between 10% and 30% (45). These results have been duplicated by Wills et al who demonstrated a 1.54% rerupture rate in the surgically treated group (12/777), versus a 17% rate (40/226) for conservatively managed patients (80).

Despite the controversy most authors support surgical intervention in Achilles tendon ruptures in the young, active and serious athlete (45). Nonoperative treatment, on the other hand, is given more consideration in a nonathletic person over 50 years of age. In addition, although a higher complication rate for surgical versus nonsurgical treatment exists, authors generally agree that these complications are often minor and resolve without compromise to overall tendon function (39).
METHODS AND MATERIALS

Patient Population

Nine patients, six males and three females, who had experienced sudden Achilles tendon rupture or chronic heel pain indicative of Achilles tendinitis were included in this study. At the time of injury patients ranged in age from twenty-five years to forty-eight years. Five of the patients sustained sudden complete ruptures of the Achilles tendon, one patient was diagnosed as having a partial tendon tear and the remaining three patients presented with complaints suggestive of Achilles tendinitis. The average follow-up for patients in the Achilles tendon rupture group was 17.8 months. This is inclusive of the one patient diagnosed with a partial tendon tear. The three patients with tendinitis were followed for an average of 25.0 months. Clinical information for patients included in this study is presented in Appendix 1.

Methods

In each patient the diagnosis was based on standard criteria. In suspected Achilles tendon rupture the diagnosis was confirmed by the presence of a palpable gap in the tendon and failure of pressure
on the calf (Thompson's test) to produce plantar flexion of the foot. Diagnosis of paratendinitis or tendinitis of the Achilles tendon was largely based on a chronic history of posterior heel pain, often aggravated by activity, seen in the absence of a palpable tendon gap or positive Thompson test. The above criteria were used to separate patients into two groups based on their diagnosis. Patients were further subdivided based on the treatment protocol followed (ie. surgical versus nonsurgical intervention). Six patients were treated surgically (4 patients with complete tendon rupture; 2 patients with chronic tendinitis) and three patients were managed conservatively.

**Surgical Technique**

**Achilles Tendon Rupture**

Three orthopaedic surgeons were involved in this study. A medial surgical approach was preferred to avoid injury to the sural nerve and short saphenous vein located laterally. After freshening the tendon ends absorbable suture was utilized to reapproximate the separated tendon stumps. One surgeon opted to reinforce the repair with a segment of tendon taken from the superior portion of the Achilles tendon and sutured to the distal segment thereby acting as an anastomotic connection (3 cases). Following surgery patients were placed in a long leg cast for six to eight weeks. Patients were then placed in an ankle hinge brace for added support after cast removal. Each patient began a physical rehabilitation program
designed to improve strength in the injured ankle. Patients were allowed to slowly increase exercise regimen.

**Achilles tendinitis**

Surgical repair in patients with Achilles tendinitis was reserved for those who failed more conservative treatment regimens. The surgeons used a longitudinal incision just medial to the Achilles tendon to avoid injury to the above mentioned neurovascular structures. The surgical technique used depended on the underlying lesion identified at the time of operation. Involvement of the tendon substance required more extensive debridement whereas peritendinitis alone often necessitated only minor debridement and release of fibrous adhesions. Postoperative care also depended upon the degree of surgical manipulation, with the minor procedures requiring a much shorter period of immobilization than more extensive procedures.

**Postoperative Evaluation**

Serial physical examinations and magnetic resonance imaging (MRI) were employed in order to assess each individuals progress. Intensive rehabilitation services were provided by the Gaylord Yale New Haven Hospital Rehabilitation Center Incorporated, New Haven Connecticut. Rehabilitation included exercises designed to increase strength in the gastrocsoleus- Achilles apparatus. In addition,
physical therapy was aimed at improving range of motion at the ankle joint to the preinjury level.

MR imaging protocol included spin echo and gradient echo scans (sagittal and axial) prior to therapeutic intervention and in the post intervention period at 3, 6 and 12 month intervals. Scans were performed using a transmit/receive extremity coil and a 1.5 T GE whole body imager. Transverse relaxation-time (T2) was calculated from a T2 weighted pulse sequence with repetition time (TR) of 2000 milliseconds and echo delay times (TE) of 20 and 80 milliseconds. The signal intensity of various anatomic structures was measured on both the 20 and 80 millisecond echoes. These values were then plotted to calculate the T2 value using a standard least squares fit and zero degrees of freedom (33,40). Specifically, the T2 value of the tendon was compared to the T2 value of the bone marrow. The difference in milliseconds (Δ msec.) of these values is given in Tables VII and VIII.

Achilles tendon strength was assessed post intervention on seven patients, the remaining three patients were lost to follow-up. The Kinetics Communicator (KINCOM), manufactured by the Chattecx Corporation, was employed to evaluate concentric and eccentric generation of plantar flexion force over a ten degree arc of motion. As the patient applied force to the arm of the KINCOM three signals, force, angle and velocity, are generated and fed back to the computer for evaluation. The force applied directly to the loadcell is representative of the actual force generated by the patient. The
group tested included: four patients with ruptures, three of which were treated surgically; one patient with a partial tendon tear, treated conservatively; and two cases of chronic Achilles tendinitis one of which required surgical intervention.

In each patient both ankles were evaluated by the KINCOM using the uninjured extremity as an internal control. In one patient with bilateral injury, the extremity with the most recent insult was compared to the other taken as the control. Specifically, the KINCOM was utilized to assess plantar flexion strength under isometric conditions. KINCOM data presented below represents the average of three trials in each of the following categories; eccentric force (control group), concentric force (control group), eccentric force (treatment group), and concentric force (treatment group).

Plantar reflex times were assessed in three patients with Achilles tendon rupture and two others with tendinitis after exercise on the KINCOM. Plantar reflex was tested utilizing the Hewlett-Packard 5316 A Universal Counter (Loveland, Colorado). This device, which was sensitive to both hammer strike and plantar reflex response, was set to trigger with contact of the Kynar Piezo film (Pennwalt Company; Valley Forge Pennsylvania). Two patients, one with Achilles tendon rupture and one with Achilles tendinitis, were unavailable for KINCOM or plantar reflex testing. Magnetic resonance imaging data is presented for these two patients in Tables VII and VIII.
Finally, patients were asked to complete a questionnaire to determine level of activity or limitations in function since the Achilles injury was incurred (see Appendix 2). This subjective analysis of function was compared to objective data derived from both physical examination and KINCOM strength testing results.
RESULTS

Subjective Evaluation

Patients were asked to complete a questionnaire assessing their level of function following therapeutic intervention compared to their preinjury level. The result was considered excellent if there were no residual symptoms and if the patient was able to return to his/her preinjury level of activity. Results were considered good if a full return to the preinjury activity was obtained, with only some stiffness after strenuous exertion. Results were classified as fair if there were improvements with regards to condition prior to medical intervention, with no more pain related to daily activity but persistent stiffness and aching related to exercise. A result was considered poor if no improvement was noted following medical intervention. Based on the above criteria, three patients reported an excellent result, four described their treatment outcome as good, the remaining two patients stated their recovery was fair. No patients described a poor outcome. It is important to note that three of the five patients judging their recovery as fair are less than a year from their Achilles tendon injury. As such, these individuals are expected to achieve excellent results in the future.
**KINCOM Results**

The KINCOM data is presented to assess the plantar strength in the various treatment groups following medical intervention. The distribution of results represented in Tables I-IV have been subdivided based on the type of injury to the Achilles tendon, rupture versus tendinitis. These groups are further divided into eccentric versus concentric force generated generated by patients in each group. KINCOM data is presented below for five patients assigned to the tendon rupture group and an additional two patients who were placed in the tendinitis group.

Table I outlines KINCOM results for the concentric plantar force production in the group treated for Achilles tendon rupture as compared to internal controls. Patient One was found to have a force output of 297.0 newtons on the involved side as compared with a 616.0 newton force output on the control side. Comparison of this data showed a fifty-two percent (52%) differential between the two test conditions. A ten percent (10%) difference between injured and control was noted in patient Two. This patient demonstrated a 467.0 newton concentric force generation on the side of rupture versus a 517.0 newton force production in the control. Patient Three had a 269.0 newton force output on the side of tendon rupture versus a 418.0 newton force output on the control side representing a thirty-six percent (36%) difference between the two conditions. Patient Four generated a 487.0 newton plantar force on the injured
side compared to a 635.0 newton force observed on the control side. This represented a twenty-three percent (23%) deficit in the injured extremity. The final patient showed a much larger variation in strength between the injured and unaffected ankle. This patient generated a force of only 191.0 newtons on the injured side versus a 434.0 newton output on the control side, a difference that represented a fifty-six percent (56%) deficit in the former.

Table II presents an evaluation of eccentric force (newtons) generated by patients who had sustained complete rupture of the Achilles tendon. These results are compared to the uninvolved ankle which was taken as an internal control. Patient One displayed a fifty-four percent (54%) differential between the injured side (286.0 N) and the control (627.0 N). Patient Two generated a force of 467.0 newtons on the injured side versus a 532.0 newton force output in the control. Thus the difference between the two data points indicate a twelve percent (12%) deficit in force generation of the injured side. KINCOM results in patient Three showed a forty-six percent (46.0%) deficit on the ruptured side (211.0 N) as compared to the control (391.0 N). The results from patient Four indicated a twenty-two percent (22%) difference in eccentric force generation between the injured (522.0 N) and control (671.0 N) extremity. The final patient in the rupture group had a 237.0 newton eccentric force output on the injured side as compared to a 474.0 newtons output seen in the corresponding control. This resulted in a fifty percent (50%) discrepancy between the two test conditions.
Plantar flexion strength was also assessed in the two patients in the Achilles tendinitis group. This data is displayed in Tables III and IV. Patient Six demonstrated an eccentric force output of 555.0 newton force output in the involve ankle as compared to a 570.0 newton output of 570.0 newtons in the control, a difference of only three percent (3.0%). Concentric force output in the same person was 447.0 newtons in the involved extremity versus a 527.0 newton force output generated in the control representing a fifteen percent (15%) difference. As noted below in Tables III and IV, this patient had previously been diagnosed with bilateral Achilles tendinitis. Based on the temporal relationship between the two diagnoses (4.5 years) the most remote injury was taken as the control in this patient. The final patient, patient Seven, demonstrated a forty-nine percent (49%) differential in eccentric force production between injured (129.0 N) and control (255.0 N). Concentric values for this patient were shown to be 86.0 newtons for the affected extremity versus 175.0 newtons for the unaffected side. These values represented a fifty-one percent (51%) differential in plantar force produced between the two test groups.

As a final phase of the examination, plantar reflex times were evaluated on five patients; four in the Achilles tendon rupture group, and the remaining patient in the tendinitis group (See Tables V & VI). Reflex times on the involved extremity were compared to results obtained from the control side. Patient Four displayed a plantar reflex response time of 17.8567 (S.D. 0.9709) milliseconds in the
control versus a 25.5482 (S.D. 2.7751) millisecond response on the injured side. Patient Two demonstrated a 11.6655 (S.D. 1.391) millisecond response time on the control side rupture as compared to a 11.2542 (S.D. 0.4680) millisecond response seen on the side of tendon rupture. Patient Five showed a 29.4157 (S.D. 0.3395) millisecond plantar reflex response time in the control versus a response time of 41.0167 (S.D. 5.3729) milliseconds observed on the side of injury. A 6.9666 (S.D. 0.2985) millisecond response time in the control contrasted with a 31.5539 (S.D. 1.0680) millisecond plantar reflex response time on the injured side observed in patient Three. Patient Six showed little variation between the two sides. Testing revealed a plantar reflex time of 33.3134 (S.D. 2.6657) milliseconds in the control as compared to a 34.2787 (S.D. 0.9599) millisecond response time in the injured extremity.

**Magnetic Resonance Imaging Results**

Magnetic Resonance Imaging data was compiled utilizing the protocol outlined above. T2 measurements were obtained at the site of the tear using axial T2 images. An exception exists in the case of Patient Three in Tables I-II in which one data point was obtained using sagittal T2 images. Imaging parameters included five millimeter thickness slices and a matrix of 256.0 x 256.0.

Table VII depicts serial MRI analysis of six patients following Achilles tendon rupture. This group includes two patients who were
treated nonsurgically and an additional four individuals who were managed by surgical means. Patient One was imaged 24 months after sustaining a partial rupture of the Achilles tendon. The injury in this patient was managed nonsurgically. T2 calculation revealed a tendon value of 45.5 milliseconds as compared to the 47.2 millisecond value produced by the bone marrow (Δ -1.70 msec). Patient Three in the same table describes MRI data for another patient treated nonsurgically for Achilles tendon rupture. Patient Three was imaged at two, four and seven months following injury as indicated in Table VII. At two months Patient Three showed T2 value of 43.8 milliseconds in the tendon substance as compared to 60.0 millisecond T2 value in bone marrow (Δ -16.2 msec). At four months the tendon T2 value was 36.0 milliseconds in contrast to 44.3 millisecond for bone marrow (Δ -8.30 msec). Seven months following injury, tendon T2 value was 31.4 milliseconds while bone marrow was 48.3 milliseconds (Δ -16.9 msec).

Table VII also describes changes in MRI T2 value for four patients with complete Achilles tendon ruptures managed with surgical intervention; patients 2, 4, 5 and 8. MRI evaluation of patient Two was performed at the time of injury and subsequently three months following injury. At the time of injury the T2 value was 42.7 milliseconds as compared to the 46.2 millisecond value in bone marrow (Δ -3.50 msec). Analysis of MRI data of the injured tendon at three months revealed a 42.8 millisecond T2 value and a 49.0 millisecond T2 in bone marrow (Δ -6.20 msec). Patient Four
demonstrated a tendon T2 of 63.7 milliseconds as compared to a bone marrow T2 value of 48.8 milliseconds ($\Delta +14.9$ msec) seven months after injury. The MRI data collected from patient Five showed a 87.2 millisecond T2 value in the tendon and a 49.0 millisecond T2 in the bone marrow at the time of injury ($\Delta +38.2$ msec). Three months after the injury patient Five was found to have a 48.7 millisecond T2 in the tendon in contrast to a 47.8 millisecond value in bone marrow ($\Delta +0.90$ msec). Patient Eight displayed a tendon T2 value of 62.3 milliseconds in contrast to a 46.2 millisecond T2 of bone marrow at the time of injury ($\Delta +16.1$ msec). Three months after rupture the same patient showed a 33.4 millisecond T2 value in tendon and a 47.4 millisecond T2 value in bone marrow ($\Delta -14.0$ msec).

Magnetic resonance imaging data presented in Table VIII represents the T2 value for two patients with Achilles tendinitis. Achilles tendinitis in patient Seven was managed surgically. MRI evaluation of this patient occurred at the time of injury and four and nine months following surgical intervention. Immediately after the injury the tendon T2 value was 48.7 milliseconds in contrast to 46.8 millisecond value in bone marrow ($\Delta +1.90$ msec). Four months after surgical intervention, tendon T2 was 38.3 millisecond as compared to a 47.0 millisecond T2 value in bone marrow ($\Delta -8.70$ msec). Nine months after surgical treatment, the patient identified as Seven displayed a 33.4 millisecond T2 in the tendon substance versus a 48.1 millisecond T2 value in bone marrow ($\Delta -14.7$ msec).
Patient 9 was treated nonsurgically and was evaluated 29 months following diagnosis. This patient demonstrated a 67.0 millisecond T2 value in the tendon substance in contrast to a 48.4 millisecond T2 value in the bone marrow (Δ +18.6 msec).

Figures I-IV depict MR images of two patients with Achilles tendon ruptures. The tendon rupture in patient Three was managed conservatively. The MR image presented in Figure I was taken four months after conservative treatment was initiated. Normal tendon appears black on MR imaging. An increase in signal intensity throughout the length of the tendon is appreciated in Figure I. A Follow-up MR image of the same patient three months later demonstrates decreased signal intensity as compared with the earlier MR image (refer to Figure II). Figures III and IV describe MR images of patient Two, whose rupture was managed operatively. Figure III demonstrates complete disruption of the continuity of the Achilles tendon diagnostic of a complete tear. The rupture site was determined to be six centimeters above the calcaneal insertion. A two to three centimeter gap was measured in this preoperative MR image. A follow-up axial view taken three months after surgery is presented in Figure IV illustrating the nonhomogeneous nature of the healing tendon.
DISCUSSION

Spontaneous Achilles Tendon rupture and Achilles tendinitis are becoming commonly reported injuries in the orthopaedic literature. Nine patients were included in the present study. Four patients who presented with complete rupture, one individual sustaining a partial tendon rupture and two others treated for complaints suggestive of tendinitis were assessed for plantar strength using the Kinetic Communicator (KINCOM). Three of the patients with complete tendon rupture were treated surgically while one underwent nonsurgical intervention. In the Achilles tendinitis group, one patient was treated surgically while the other was managed conservatively. These patients were also evaluated through use of magnetic resonance imaging (MRI) of the healing Achilles tendon. An additional two patients were assessed utilizing only MRI follow-up as they were unavailable for KINCOM testing. One of the patients in this group was treated surgically for a rupture of the Achilles tendon unit. The remaining patient complained of symptoms suggestive of Achilles tendinitis and was treated conservatively.

Previous authors have utilized the Cybex II device to evaluate the return of strength in patients following injury to the Achilles tendon. The Cybex II unit operates on the isokinetic principle of a controlled of muscular contraction. This device has a controlled rate
of motion and accommodating resistance. In this device resistance is equal to the immediate amount of effort applied against the input arm of the unit. Shields et al (1978) noted a 16.5% loss of plantar flexion strength and a 17.5% loss of plantar flexion power following injury to the Achilles tendon in their patient population (73). This study however omits discussion of the time from injury as a factor in the ability of individuals to generate a given force.

Stauber (1989) evaluated the KINCOM to assess the accuracy and the reproducibility of the results obtained. Analysis of the static response data indicated an error of 2.43 newtons present in the measurement of force by the strain gauge. This error appeared to be systemic and related to the orientation of the strain gauge and the lever arm grip attachment. Evaluation of dynamic response in the KINCOM revealed a more variable error that averaged 3.46 newtons (19).

In the present study, objective evaluation of the relative strength of the Achilles tendon was performed utilizing the Kinetics Communicator (KINCOM). This device has advantages over the Cybex II unit. Unlike the Cybex II unit, the KINCOM is computerized, thereby reducing the degree of human error in the measurements obtained. In addition, whereas the KINCOM is capable of interpreting eccentric force production, the Cybex II unit is not capable of analyzing this type of data. The former therefore is able to present data representing an isometric test (19). Isometric exercise denotes muscular contraction against a constant load which is basically immovable. The muscle is capable of generating near-maximum
tension, but its length remains constant with no physical work being performed (29).

The results of these KINCOM studies was compared to standard physical examination procedures and magnetic resonance imaging (MRI) assessment of the injured extremity. The KINCOM measurement was carried out with the patient lying prone on a table and the pelvis, legs and ankles were fixed in position with belts. Strength was assessed by measuring the plantar force output generated by each individual utilizing the uninvolved extremity as a control. Patients were grouped according to both their injury and the method of treatment employed in each case. Concentric and eccentric generation of force was evaluated bilaterally in each patient. Each patient was given three trials at maximum force production and an average value is reported in each case (Tables I-IV).

Concentric work describes exercise involving the shortening of actively contracting muscles. For example, lifting an object results in development of tension in the active muscles and a subsequent decrease in muscle length. In contrast, the activity of lengthening actively contracting muscles is termed eccentric contraction. In eccentric work the tension generated by the muscles is overcome and the muscles are forcibly stretched resulting in lengthening of the muscle unit (4,41). Previous research has demonstrated that greater tension per muscle fiber is generated under eccentric contraction conditions when compared to concentric conditions. As a result, relatively few fibers are recruited and relatively large forces are produced by eccentric contractions (12,41,58,59).
Table I displays the results of concentric plantar force generated in the Achilles tendon rupture population. One of the patients included in this table, patient One, was diagnosed with a partial tear of the Achilles tendon. This patient who was 38 months from injury at the time of testing exhibited a 52.0% deficit in the injured as compared to the control extremity. Upon evaluation of the three Achilles tendon rupture patients managed surgically, increasing strength is apparent in the involved extremity as compared to the control over time. Patient Five who had the most recent rupture also had the correspondingly greatest strength differential (56.0%) between the injured and control ankle. Patient Four was evaluated ten months after rupture and demonstrated a twenty-three (23.0%) deficit in the injured extremity. Likewise, patient Two, injured thirty-two months prior to KINCOM evaluation showed a ten percent differential in plantar strength between the injured and control leg. Given the above results it appears that there is a trend toward increased plantar strength as time progresses away from the point of tendon rupture. In addition, the measurement of plantar flexion force at the ankle joint in a previous study has demonstrated superior average result for the operatively treated compared to the conservatively treated group (32).

The remaining individual presented in Table I, identified as patient Three, also suffered a spontaneous Achilles tendon rupture but unlike the other patients described, this patient was treated nonsurgically 16 months prior to KINCOM evaluation. This patient generated a force differential of 36.0% between the involved extremity and the control. This value is greater than that of patient
Four who was 10 months status post surgical repair of an Achilles tendon rupture. Previous authors have reported the superiority of surgical intervention in this injury in terms of return of strength of the musculotendinous unit. Advocates for surgical repair of Achilles tendon ruptures have successfully documented that the tendon is stronger following operative repair than after nonsurgical intervention. Inglis et al (1976) observed that the plantar flexion strength generated by patients following surgical repair averaged 101.0% as compared to the uninvolved extremity. In the same study a 73.0% difference in plantar flexion strength was noted in patients with Achilles tendon ruptures treated conservatively. Similar findings were noted in the analysis of the power and endurance capabilities of the two groups (31). Although the patient population in the present study is small it indicates that the same phenomenon may be invoked to explain differences between these two groups. Kellam et al (1985) postulated that this difference in recovery of plantar strength between the two groups could be attributed an ability to maintain the appropriate musculotendinous tension within the involved tendon following operative repair of the rupture. Kellam et al postulated that the increased tensile strength in tendons after surgical repair related to the greater durability observed in the tendon substance (39).

Another related theory which might explain the KINCOM data for patient Three involves the fatiguability of muscles during exercise. Muscle fatiguability can be measured isometrically by assessing the rate of decline of maximal isometric force over a pre-selected time interval. It is important to note that fatiguability
during isometric contractions of 50% or greater is independent of blood supply since all blood vessels are collapsed. Therefore, many isometric tests rely heavily on oxidative pathways for energy supply - creatine phosphate, ATP and glycogen stores (76). As previously noted, return of plantar flexion strength in the injured extremity occurs more rapidly following operative treatment as compared to conservative therapy. Perhaps this trend relates to the greater endurance of healing tendon after surgical intervention. Although not evaluated in the present study, the KINCOM is capable of generating data to determine the affect of muscle fatigue on plantar flexion strength. Future investigation is required to examine the influence of muscle fatigue on the plantar force generating capabilities as related to the treatment protocols employed.

Table II depicts similar trends for eccentric force production in patients with Achilles tendon rupture. Patient One as noted above sustained a partial tear of the Achilles tendon substance. Data on this patient indicates a 54.0% deficit in the affected extremity 38 months after nonsurgical management of the injury. Patients are arranged in Table II from most remote to most recent time of injury. Patients Two, Four and Five exhibit force outputs of 12.0%, 22.0% and 50.0% respectively as time progressed from point of injury. As above this finding is representative of the time course necessary for the tendon to recover from trauma. Therefore, plantar strength testing is an ideal method to objectively assess an individual's progress during rehabilitation.

Regeneration of the tendons following insult has been extensively explored in the orthopaedic literature (6,16,18,63,64).
Postacchini et al (1978) examined an animal model to evaluate the microscopic changes of regenerating tendon. Immunofluorescence, light and electron microscopy were the methods used to evaluate tendon healing. The data indicated that 30 weeks following surgical repair of rupture, the newly formed tendon possessed the gross morphological characteristics of native tendon tissue. Despite acquiring the normal morphological characteristics, the typical texture of normal adult tendon was not evident at 30 weeks. Postacchini et al (1978) noted that the tenocytes were more numerous, less uniformly distributed and contained a greater amount of contractile proteins. The elastic fibers appeared immature resembling normal neonatal tendon in size (64). This observation likely relates to the initial decrease in plantar flexion strength noted in the injured extremity as compared to the control. Despite the appearance of healthy tendon the deficit in strength of the gastrocsoleus-Achilles mechanism documented in the present study illustrates the slow healing process characteristic of injured tendon substance.

The rate of increase in tendon strength has been documented in previous studies to be greatest between the fifth and ninth weeks of rehabilitation. This initial increase in tensile strength relates to the presence of increasingly greater numbers of newly formed collagenous fibers. Further increase in tensile strength of the healing tendon relates to the orientation of these newly formed cells in the direction of the longitudinal axis of the tendon (20). Fernando and Movat (1963) proposed that the direction of mechanical stress imposed on the healing tendon influenced the eventual longitudinal
alignment of the newly formed tendon fibers (18). As mentioned earlier, the period of immobilization following operative treatment of Achilles tendon rupture is shorter than that which is necessary after conservative treatment of this injury. It is possible that the earlier mobilization in the surgically treated patient exposes the injured extremity to mechanical stresses. This exposure allows the newly formed tendon fibers to assume a longitudinal arrangement thus increasing the tensile strength of the Achilles tendon.

Tables III and IV describe the results of KINCOM analysis of the tendon in two patients treated for Achilles tendinitis. Most authors agree that nonsurgical treatment of Achilles tendinitis should be attempted before surgical repair is undertaken. Initially patient Six was treated conservatively with casting, rest, and eventually steroids. However, conservative management in this patient failed to correct the pain and disability associated with Achilles tendinitis. Patient Six underwent surgical treatment 24 months prior to KINCOM evaluation of plantar strength. Concentric and eccentric data displayed a 15.0% and 3.0% deficit respectively in the injured extremity as compared to the control. In contrast, patient Seven was managed nonsurgically 11 months prior to KINCOM study. KINCOM results obtained on this patient indicated a much larger deficit in plantar strength on the affected side in comparison to the control. Concentric force output generated was 51.0% less than that observed in the control. Likewise, a 49.0% differential was determined through KINCOM analysis of eccentric force generation of the injured versus the unaffected ankle.
The increase in plantar flexion generating capabilities observed in the injured extremity with the progression of time can be explained by invoking previously described stages of tendon repair. The period of fibrillogenesis which begins within seven days of injury gives way to the stage of remodeling in which newly formed tendon fibers begin to assume the longitudinal alignment observed in normal tendon tissue. The longitudinal orientation of these newly formed fibers correlates with the increased tensile strength observed in the Achilles tendon throughout the repair process (16).

Surgical versus nonsurgical treatment in cases of Achilles tendinitis remains a controversial topic in the orthopaedic literature. In the present study it is apparent that the lifestyle of the injured patient deserves consideration when planning treatment strategies. Although both patient Six and Seven were diagnosed with tendinitis. However, patient Six was a marathon runner while patient Seven lived a more sedentary lifestyle. Both patients were able to return to their preinjury level of activity following therapeutic intervention.

Table V represents plantar reflex response times for patients with injury to the Achilles tendon. The Hewlett-Packard 5316 A Universal Counter was used to evaluate response times of the Achilles tendon reflex. In previous studies examining the Achilles tendon reflex, devices employed measured from the beginning of the stimulus artifact (produced by the movement of the foot caused by the tap of the reflex hammer) to the midpoint of the relaxation segment of the response curve (25). The device used in the present study has the advantage of recording time elapsed from the instant
of the reflex hammer strike through the entire plantar flexion response. Johnson et al (1963) found that the healthy subjects examined in their study had reflex times that ranged from 40 to 60 milliseconds (34). Early stages of muscle shortening require stretching of the series elastic component (SEC) in the muscle. This "taking up slack" requires time estimated by Stauber to vary from 20-100 milliseconds (76).

Patients 2-5 are in the Achilles tendon rupture group; the patient described as patient 6 is a member of the Achilles tendinitis group. In Patients Two, Four and Five surgical management of Achilles tendon ruptures was utilized. Achilles tendon rupture in patient Three was treated conservatively with cast immobilization. The plantar response time in patient Two thirty-two months after surgery reflected only a .4113 millisecond difference between injured side and control side. Patient Four was noted to have a 7.6915 millisecond difference between affected and unaffected extremity 10 months following surgical intervention. Patient Five also received operative repair of an Achilles tendon rupture. Plantar reflex response time five months after repair showed a 11.601 millisecond discrepancy between injured and uninjured side.

Although the patient population is small the data suggests that a return of plantar reflex response time occurs as time progresses away from the point of surgical intervention. Perhaps this trend relates to the continued migration of newly formed collagen fibers into the injured tendon throughout the healing process. The addition of these new cells in conjunction with resolution of edema fluid and hematoma may promote return of
normal reflex response time in the injured extremity. Additionally, it is possible that ingrowth of neural tissue during tendon repair may contribute to the decreasing plantar reflex time observed by decreasing nerve conduction time.

Patient Three sustained a rupture of the Achilles tendon 16 months prior to plantar reflex response time evaluation which was treated with cast immobilization. A 24.5873 millisecond differential in response time was noted between the injured extremity and the uninjured extremity taken as a control. Although a difference certainly exists the large value of the deficit in this patient may represent experimental error. It was noted during testing of this patient that Hewlett-Packard device recorded differences in strength based on the velocity of the reflex inciting hammer strike.

Table VI describes the results of plantar flexion response time analysis of a patient with Achilles tendinitis. Patient Six was treated surgically for Achilles tendinitis. Plantar reflex response times for patient Six seventy-three months after initial injury showed only a 0.9653 millisecond difference between the two extremities tested. These results when taken in conjunction with previously described plantar reflex response data further illustrate the normalization of injured tendon substance during the healing process.

The healing process was further evaluated with the aid of Magnetic Resonance Imaging (MRI) techniques designed to objectively access the intrinsic healing of injured tendon over time. Normal tendon typically appears hypodense on both T1 and T2 MR images (53,65). On sagittal images the Achilles tendon is seen as a thin linear structure coursing from the distal aspect of the
gastrocnemius-soleus complex to the posterior cortex of the calcaneus. On axial images the Achilles tendon has a flattened elliptical shape and is bordered anteriorly and posteriorly by fat (53). Ehman and Berquist (1986) noted the low intensity appearance of tendon related to the low mobile spin frequencies and very short T2 relaxation times noted in this substance (15). In contrast, bone marrow has high signal intensity on MRI providing an excellent background for the lower signal intensity of tendon. Previous authors have observed certain distinctive patterns in abnormal or injured tendon. Examination of MR images of injured tendon illustrated the capability of this modality to delineate between hemorrhage and soft tissue structures. Specifically T2-weighted images are highly sensitive for detecting pathologic changes such as edema, hemorrhage and tumor (38). In the present study, six patients who had sustained rupture of the Achilles tendon and two others with diagnosed Achilles tendinitis were evaluated utilizing MRI. Objective assessment of tendon healing over time was analyzed through periodic MRI follow-up.

Analysis of the MRI data outlined in Tables VII and VIII show a decrease in tendon T2 value over time. As the healing process progresses, an increasingly greater difference in T2 value is noted between the tendon and bone marrow. The increased T2 value of edema fluid and hematoma present in the tendon after injury closely approximates the relatively high T2 value normally described in bone marrow. During the healing process, edema fluid and hematoma slowly resolve and the T2 value of the injured tendon undergoes a corresponding decrease (67). Inspection of the data
presented in Tables VII and VIII support this contention. Initially, the T2 value of the tendon is greater than that of the bone marrow. However, the normally low intensity tendon signal returns as the inflammatory response resolves. Thus the difference between the tendon signal and the higher intensity bone marrow signal become more pronounced as tendon healing progresses.

Previous authors have described the unique appearance of hemorrhage on MR images of muscle, ligaments and tendon. The uniqueness of this material relates specifically to the diagnostic specificity of MRI for evaluating injuries of fibrous connective tissue. Ehman and Berquist noted that the T2 image of oxygenated liquid blood is relatively long because of the high free water content of blood relative to solid tissue. Prolongation of the T2-weighted signal also relates to the inflammatory reaction to interstitial blood observed in the surrounding muscular bed (15). MRI evaluation of previously disrupted or inflamed tendon substance observed on MRI shows evidence of resolution of hematoma and therefore a decrease in signal intensity as time progresses.

Magnetic Resonance Imaging results display a trend of decreased tendon signal intensity over time. An exception exists in the case of the patient identified as Two who showed a 0.1 millisecond increase between preoperative evaluation and three month MRI follow-up. As described above resolution of the hematoma partially accounts for decreased signal observed in tendon during the healing process. Another factor involved in the decreased T2 signal seen relates to the intrinsic healing of the tendon substance. The presence of increasingly greater amounts of low
intensity collagenous tendon scar corresponds to changes noted in tendon signal over time. Remodeling of newly formed collagen in healing tendon has been frequently described in orthopaedic the literature as part of the normal repair process (16,17,18,19,54). Tendon healing has been divided into two stages-a connective tissue stage and a stage of tendon callus formation. Mason and Allen described the first stage as a process in which connective tissue surrounding the healing tendon proliferates to form a fibroblastic cuff and scar about and between regenerative tendon. The second phase involves reorganization and alignment of newly formed tendon into callus resembling the original tendon substance (54).

The above described reparative process of injured tendon has been successfully examined in the present study utilizing Magnetic Resonance Imaging techniques. The percentage of low signal tendon observed in the healing tendon was found to increase with progression of the healing phase. Although the sample of patients involved in the present study is small, the MRI data presented supports the description of the normal repair process of tendon demonstrated in previous studies. MR images obtained in the present study reflect the slow return of homogeneous low intensity signal which typifies normal tendon.
CONCLUSIONS AND SUMMARY

The present study examined the rehabilitation of patients who had sustained either rupture of the Achilles tendon or who suffered with complaints of chronic Achilles tendinitis. Data obtained from KINCOM evaluation of five patients with Achilles tendon rupture who were managed surgically showed a decrease in percent difference between injured and uninjured extremity with progression of time. In contrast, the data obtained from the two patients with Achilles tendon rupture treated conservatively did not demonstrate an increase in plantar strength when compared to controls. An explanation for this discrepancy may be related to two factors. The first concerns the small number of patients included in this category. Secondly, both of the patients in this study approached the KINCOM evaluation with a great deal of hesitancy. Although subjectively these individuals had returned to many of their preinjury activities, they each reported being fearful of reinjury. Perhaps this hesitancy resulted in less than maximal force generated during KINCOM testing.

KINCOM analysis of two patients recovering from Achilles tendinitis revealed a significant decrease in the difference observed in eccentric force production between the injured and control extremities. Concentric data however did not demonstrate a corresponding pattern. The rationale for these differences is unclear but may relate to the level of physical fitness of the involved patients. Patient six is a marathon runner and worked to regain preinjury level of function. In contrast patient seven leads a more sedentary lifestyle. Further investigation will be required to
accurately evaluate the importance of preinjury level of function on treatment outcome.

Planter reflex response time was also utilized to objectively analyze return of function in the injured Achilles tendon. Evaluation of the patients treated surgically for Achilles tendon rupture demonstrated a progressive decrease in the variation of response time between injured and control extremity over time. This normalization between the two values presumably represents another modality by which healing tendon substance can be assessed and the rehabilitation followed. The data presented in Table V for the one patient managed conservatively shows a plantar reflex response differential that exceeds the value of surgically treated patients with more recent injury. Given the fact that only one patient in the rupture group presented was managed conservatively, no generalization on healing trends is possible in this instance. However, previous researchers have documented the superior outcome Achilles tendon ruptures treated surgically as opposed to those conservatively managed (31,39,80). Similarly, Table VI describes one data point for plantar response time of a patient with Achilles tendinitis operatively managed. Obviously, conclusions cannot be generalized to the population given one data point in isolation.

The MRI data presented as outlined above demonstrates a pattern of decreased tendon signal intensity over time suggestive of common stages in the repair process. These results illustrate progressive homogeneity in the healing tendon substance. Physical examination, in conjunction with the KINCOM and plantar reflex data
presented, suggest complete healing of the injured tendon. However, MRI data identified continued abnormality of the tendon in the majority of cases despite return of preinjury function. Further studies are needed in order to better correlate magnetic resonance imaging of injured tendon to more conventional methods of objective evaluation. Given the small patient population in the present study, and the expense of MRI evaluation, it is the belief of this author that such examinations be limited to cases in which physical examination fails to identify an abnormality despite continued symptomology.
### TABLE I: Kincom Results

**Comparison of Concentric Plantar Force Generated in Injured vs Control in Patients with Achilles Tendon Ruptures**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age at Injury years</th>
<th>Time Since Injury months</th>
<th>Medical Intervention</th>
<th>Force Output Control Newtons</th>
<th>Force Output Injury Newtons</th>
<th>% Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>30</td>
<td>38 months</td>
<td>nonsurgical</td>
<td>616.0 N</td>
<td>297.0 N</td>
<td>52.0%</td>
</tr>
<tr>
<td>2</td>
<td>32</td>
<td>32 months</td>
<td>surgical</td>
<td>517.0 N</td>
<td>467.0 N</td>
<td>10.0%</td>
</tr>
<tr>
<td>3</td>
<td>45</td>
<td>16 months</td>
<td>nonsurgical</td>
<td>418.0 N</td>
<td>269.0 N</td>
<td>36.0%</td>
</tr>
<tr>
<td>4</td>
<td>24</td>
<td>10 months</td>
<td>surgical</td>
<td>635.0 N</td>
<td>487.0 N</td>
<td>23.0%</td>
</tr>
<tr>
<td>5</td>
<td>29</td>
<td>5 months</td>
<td>surgical</td>
<td>434.0 N</td>
<td>191.0 N</td>
<td>56.0%</td>
</tr>
</tbody>
</table>

1 Patient sustained a partial rupture of the Achilles tendon. The author has elected to present this data in the Achilles tendon rupture group.
TABLE II: Kincom Results

_Eccentric Force generation in patient with Achilles Tendon Rupture_

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age at Injury years</th>
<th>Time Since Injury months</th>
<th>Medical Intervention</th>
<th>Force Output Control</th>
<th>Force Output Injury</th>
<th>% Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>30</td>
<td>38</td>
<td>nonsurgical</td>
<td>627.0 N</td>
<td>286.0 N</td>
<td>54.0 %</td>
</tr>
<tr>
<td>2</td>
<td>32</td>
<td>32</td>
<td>surgical</td>
<td>532.0 N</td>
<td>467.0 N</td>
<td>12.0 %</td>
</tr>
<tr>
<td>3</td>
<td>45</td>
<td>16</td>
<td>nonsurgical</td>
<td>391.0 N</td>
<td>211.0 N</td>
<td>46.0 %</td>
</tr>
<tr>
<td>4</td>
<td>24</td>
<td>10</td>
<td>surgical</td>
<td>671.0 N</td>
<td>522.0 N</td>
<td>22.0 %</td>
</tr>
<tr>
<td>5</td>
<td>29</td>
<td>5</td>
<td>surgical</td>
<td>474.0 N</td>
<td>237.0 N</td>
<td>50.0 %</td>
</tr>
</tbody>
</table>

1 Pt. sustained partial rupture of the Achilles Tendon. The author has elected to present this data in the Achilles tendon rupture group.
TABLE III: Kincom Results
Eccentric force output in two patients with Achilles Tendinitis

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age at Injury (years)</th>
<th>Time Since Injury (months)</th>
<th>Medical Intervention</th>
<th>Force Output (newtons) control</th>
<th>Force Output (newtons) injured side</th>
<th>% Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>6</td>
<td>48</td>
<td>24 months</td>
<td>surgical</td>
<td>570.0 N</td>
<td>550.0 N</td>
<td>3.0 %</td>
</tr>
<tr>
<td>7</td>
<td>34</td>
<td>11 months</td>
<td>surgical</td>
<td>255.0 N</td>
<td>129.0 N</td>
<td>49.0 %</td>
</tr>
</tbody>
</table>

1 Patient 6 had bilateral Achilles Tendinitis; both injuries were surgically treated. The most remote injury (73 months prior to present study) was taken as the control in this patient.
TABLE IV: Kincom Results

Concentric Generation of Force in two patients with Achilles Tendinitis

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age at Injury (years)</th>
<th>Time Since Injury (months)</th>
<th>Medical Intervention</th>
<th>Force Output (N) Control</th>
<th>Force Output (N) Injured side</th>
<th>% Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>6</td>
<td>48</td>
<td>24</td>
<td>surgical</td>
<td>527.0 N</td>
<td>447.0 N</td>
<td>15.0 %</td>
</tr>
<tr>
<td>7</td>
<td>34</td>
<td>11</td>
<td>surgical</td>
<td>175.0 N</td>
<td>86.0 N</td>
<td>51.0 %</td>
</tr>
</tbody>
</table>

1 Patient 6 had bilateral Achilles Tendinitis, both surgically treated. The remote injury (73 months ago at this writing) was taken as the control in this patient.
### TABLE V: Plantar Reflex Response Times (msec)

Plantar Reflex Response times in patients treated for Achilles tendon rupture. The uninjured side is taken as the control.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Time since injury (months)</th>
<th>Medical therapy</th>
<th>Response time msec ± S.D. control</th>
<th>Response time msec ± S.D. injured side</th>
<th>Δ msec</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>32</td>
<td>surgical</td>
<td>11.2542 ± 0.4680</td>
<td>11.6655 ± 1.3591</td>
<td>0.4113</td>
</tr>
<tr>
<td>3</td>
<td>16</td>
<td>non-surgical</td>
<td>6.9666 ± 0.2985</td>
<td>31.5539 ± 1.0680</td>
<td>24.5813</td>
</tr>
<tr>
<td>4</td>
<td>10</td>
<td>surgical</td>
<td>17.8567 ± 0.9709</td>
<td>25.5482 ± 2.7751</td>
<td>7.6915</td>
</tr>
<tr>
<td>5</td>
<td>5</td>
<td>surgical</td>
<td>29.4157 ± 0.3395</td>
<td>41.0167 ± 5.3729</td>
<td>11.601</td>
</tr>
</tbody>
</table>

### TABLE VI: Plantar Reflex Response Times (msec)

Plantar Reflex Response Time in one patient with Achilles tendinitis surgically repaired

<table>
<thead>
<tr>
<th>Patient</th>
<th>Time Since Injury months</th>
<th>Medical therapy</th>
<th>Response time ± S.D. (msec) control</th>
<th>Response time ± S.D. (msec) injured side</th>
<th>Δ msec</th>
</tr>
</thead>
<tbody>
<tr>
<td>6</td>
<td>73</td>
<td>surgical</td>
<td>33.3134 ± 2.6657</td>
<td>34.2787 ± 0.9599</td>
<td>0.9653</td>
</tr>
</tbody>
</table>

1 Patient 6 had bilateral Achilles tendinitis. The most remote injury (73 months ago at this writing) was taken as the control in this patient.
### TABLE VII: Magnetic Resonance Imaging Results

*T2 signals in Tendon and Bone Marrow (msec) in patients with Achilles tendon rupture. Data below represents involved extremity only*

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (years)</th>
<th>Medical Therapy</th>
<th>Time between injury &amp; MRI evaluation (months)</th>
<th>T2 (msec) Tendon/Bone Marrow</th>
<th>Δ msec</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>30</td>
<td>non-surgical</td>
<td>24.0</td>
<td>45.5/47.2</td>
<td>-1.7</td>
</tr>
<tr>
<td>2</td>
<td>32</td>
<td>surgical</td>
<td>0.0</td>
<td>42.7/46.2</td>
<td>-3.5</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>3.0</td>
<td>42.8/49.0</td>
<td>-6.2</td>
</tr>
<tr>
<td>3</td>
<td>45</td>
<td>non-surgical</td>
<td>2.0</td>
<td>43.8/60.0</td>
<td>-16.2</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>4.0</td>
<td>36.0/44.3</td>
<td>-8.3</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>7.0</td>
<td>31.4/48.3</td>
<td>-16.9</td>
</tr>
<tr>
<td>4</td>
<td>24</td>
<td>surgical</td>
<td>7.0</td>
<td>63.7/48.8</td>
<td>+14.9</td>
</tr>
<tr>
<td>5</td>
<td>29</td>
<td>surgical</td>
<td>0.0</td>
<td>87.2/49.0</td>
<td>+38.2</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>3.0</td>
<td>48.7/47.8</td>
<td>+0.9</td>
</tr>
<tr>
<td>8</td>
<td>39</td>
<td>surgical</td>
<td>0.0</td>
<td>62.3/46.2</td>
<td>+16.1</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>3.0</td>
<td>33.4/47.4</td>
<td>-14.0</td>
</tr>
</tbody>
</table>
**TABLE VIII: MAGNETIC RESONANCE IMAGING RESULTS**

*T2 signals in tendon and bone marrow (msec) in patients with Achilles tendinitis. Data below represents involved extremity only.*

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (years)</th>
<th>Medical Therapy</th>
<th>Time between injury &amp; MRI evaluation (months)</th>
<th>T2 (msec) Tendon/Bone Marrow</th>
<th>Δ msec</th>
</tr>
</thead>
<tbody>
<tr>
<td>7</td>
<td>34</td>
<td>surgical</td>
<td>0.0</td>
<td>48.7/46.8</td>
<td>+ 1.9</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>4.0</td>
<td>38.3/47.2</td>
<td>- 8.9</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>9.0</td>
<td>33.4/48.1</td>
<td>-14.7</td>
</tr>
<tr>
<td>9</td>
<td>35</td>
<td>non-surgical</td>
<td>29.0</td>
<td>67.0/48.4</td>
<td>+18.6</td>
</tr>
</tbody>
</table>
FIGURE I

Sagittal proton density magnetic resonance image the Achilles tendon. The arrows indicate diffuse increase in signal intensity within substance of the healing tendon following rupture. The diffuse pattern of inflammation resembles the MR image pattern of Achilles tendinitis. This MR image was taken of patient 3 four months after conservative management of an Achilles tendon rupture. Clinical history of this patient's injury revealed a several month history of posterior heel pain which perhaps predisposed to eventual rupture.
FIGURE II

Sagittal proton density MR image of the Achilles tendon in patient three months following MR image presented in Figure I. Note the continued region of increased signal in the tendon identified by the rectangle (1). The square indicates the region of bone imaged to obtain contrasting bone marrow T2 value.
FIGURE III

Sagittal proton density magnetic resonance image of the Achilles tendon. The arrow indicates complete disruption of the continuity Achilles tendon diagnostic of a complete tear. This is an MR image of patient six one week following rupture of the Achilles tendon. Interpretation of the MR image revealed a 2-3 centimeter separation of the tendon ends. Location of ruptured was measured as 6 centimeters above the calcaneal insertion.
Figure IV

MR image in the axial view through the distal lower leg. Note the nonhomogeneous nature of the tendon identified by square 1. Square 2 shows the region of bone utilized to contrast tendon versus bone marrow T2 values. This figure describes MR image results of patient six three months following surgical repair of Achilles tendon rupture.
APPENDIX 1

The information below contains a brief synopsis of the mechanism of Achilles tendon injury, treatment and follow-up for the patients included in the present study.

Patient One (refer to Tables I-II,VII): Patient is a 33 year old male who sustained a partial rupture of the right Achilles tendon as a result of sudden dorsiflexion when foot was caught in a pothole. Patient was treated conservatively with serial casting. Approximately seven months after this original injury the patient again sustained a partial tendon rupture on the right side via the same mechanism (i.e., sudden dorsiflexion). Patient was again managed conservatively with cast immobilization. Following both injuries rehabilitation included physical therapy at Gaylord/Yale New Haven Rehabilitation Center Incorporated. Patient is now 38 months away from initial injury and reports significant weakness in involved extremity, decrease level of physical activity.

Patient Two (refer to Tables I-II,V,VII): Patient is a 33 year old male who sustained a complete rupture of the right Achilles tendon 32 months prior to KINCOM evaluation. Patient was playing basketball at the time of injury. Surgical repair and subsequent long leg cast immobilization were utilized to manage the injury in this patient. Physical therapy occurred at the Gaylord/Yale New Haven Rehabilitation Center Incorporated. Patient has returned to jogging 3-4 miles per day.
Patient Three (refer to Tables I-II,V, VII): Patient is a 45 year old female who sustained a complete rupture of the right Achilles tendon 16 months prior to KINCOM evaluation while playing tennis. Patient was treated nonsurgically with serial casting for three months followed by eight months of physical therapy. Patient reports less frequent participation in athletic activity since injury.

Patient Four (refer to Tables I-II,V,VII): Patient is a 25 year old male who sustained complete rupture of the left Achilles tendon while playing indoor soccer. Patient injury occurred 10 months prior to KINCOM evaluation. Patient was treated with surgery followed by long leg cast for four weeks and four additional weeks of bracing. Patient also participated in physical therapy which included range of motion exercises and weight training to strengthen the injured extremity. Patient reports return of strength in left extremity but notes a decrease in flexibility.

Patient Five (refer to Tables I-II,V,VII): Patient is a 29 year old male who sustained complete rupture of the left Achilles tendon as a result of sudden dorsiflexion while jumping to avoid an oncoming car. Injury occurred five months ago and was managed surgically. Following surgical repair patient was immobilized in long leg cast for six weeks and an additional six weeks in range of motion brace. Patient reports return to preinjury level of function but notes persistent stiffness and occasional swelling in the involved ankle.
Patient Six (refer to Tables III-IV,VI): Patient is a 46 year old male who presented with a long history of bilateral chronic posterior heel pain often associated with running (patient is a marathon runner). Patient reports gradual increase in symptoms as time progressed until a decrease in physical activity was necessary to reduce symptoms. Initially patient was managed conservatively with rest, orthotics, antiinflammatory drugs and ultrasound. Conservative management failed and the patient eventually underwent surgical debridement and exploration of the inflamed tendon. Operative repair of the right ankle occurred 72 months prior to KINCOM testing while repair of the left ankle occurred 24 months prior to testing. For purposes of KINCOM evaluation in the present study the right extremity is taken as the control. Post surgical rehabilitation included range of motion exercises and progressive strengthening of the involved extremity. Patient reports complete return to preinjury level of activity and has continued long distance running without difficulty.

Patient Seven (refer to Tables III-IV,VIII): Patient is a 34 year old female who presented with an 11 month history of chronic Achilles tendinitis in left leg. Patient recalls presence of a "lump" in the region of the tendon associated with pain. Short leg casts were utilized on three separate occasions (9 months total) but only temporary relief was obtained. Operative debridement of the left Achilles tendon was undertaken 11 months prior to present study. Rehabilitation included range of motion and strengthening exercises. Patient reports occasional pain in involved extremity but has been able to return to preinjury level of activity.
APPENDIX 1 (cont.)

Patient Eight (refer to Table VII): Patient is a 39 year old male who sustained complete rupture of the right Achilles tendon while playing basketball. Patient is six months away from injury which was managed surgically. After surgical repair the patient was placed in cast immobilization for six weeks and subsequently braced for another six weeks. Patient reports little residual pain in tendon and notes steady progress toward preinjury level of function.

Patient Nine (refer to Table VIII): Patient is a 35 year old female with a history of congenital club feet. Operative repair to correct the deformity occurred during infancy. Patient sought medical assistance after noting a painful "lump" in the posterior aspect of the right heel. Magnetic resonance imaging of the right ankle was performed 40 months after presentation. Biopsy of the right heel indicated calcium deposits. Patient reports that the mass resolved without treatment one month after presentation. Patients notes recurrence mass (less painful) one month later. Patient reports decreased activity secondary to persistent tenderness.
Appendix 2
Achilles Tendon Study Questionnaire

Name: 
Age: 

1. Date of Injury: 
Side of involvement: 

2. Time between injury and seeking medical aid: 

3. Describe the circumstances surrounding your injury (at rest, during exercise, etc.): 

4. If injury occurred while participating in athletics please describe the frequency of your participation in sports. Had you recently started your exercise routine prior to your injury? 

5. Did you experience pain immediately following injury? If so, can you describe this pain (sharp, dull etc.)? 

6. Prior to injury had you ever injured your ankle? Did you experience pain of a chronic nature prior to your injury? 

7. How was your injury treated (surgery, casting, rest etc.)? 

8. Describe the rehabilitation required following your injury. 

9. Have you been able to return to your pre-injury level of function? If not, in what way have you limited your activity since your injury.
REFERENCES


REFERENCES (CONT.)


REFERENCES (CONT.)

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