ACHILLES TENDON RUPTURES: A REVIEW OF THE LITERATURE

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Introduction

The Achilles tendon gets its name from Achilles, the ancient Greek hero of the Trojan War and Homer's Illiad (1). Achilles was the son of the nymph, Thetis, who attempted to make him immortal by dipping him in the river Styx. However, she was unable to dip his heel in the river because she had to hold it as she lowered his body; his heel therefore remained mortal. Achilles was then killed in war by the Trojan prince Paris, who shot a poisonous arrow in to Achilles' heel. This gave rise to the term the "Achilles heel," describing the weakest point of a person's body (2).

The Achilles tendon is the strongest and thickest tendon in the body. Lying in the posterior, superficial compartment of the gastrocnemius-soleus complex, it begins near the middle of the muscle mass and stretches down to the calcaneus. It is the conjoined tendon of the gastrocnemius and soleus muscles, with a small contribution from the plantaris. The average length of the tendon is 15 cm, but ranges anywhere from 11 to 26 cm. The tendon is broad close to its origin and receives muscle fibers from its soleus almost to its lower end (2). The Achilles tendon is not encased in a true synovial sheath, but instead is surrounded by the paratendon, composed of a single layer of cells. It functions as an elastic sleeve, permitting free movement of the tendon within the surrounding tissues. The paratendon is richly vascularized, supplying the Achilles tendon with much of its blood supply. The tendon also receives blood from the musculotendinous and osteotendinous junctions. The Achilles tendon is supplied by two arteries, the posterial tibial and peroneal arteries. The midsection is supplied by the peroneal artery while the proximal and distal sections are supplied by the posterior tibial artery (3). The "watershed area" of the Achilles tendon is an avascular area located 2 to 6

cm proximal to the Achilles tendon insertion. This area has poor healing potential and is associated with the pathology of multiple injuries to the Achilles tendon. The tendon is innervated from three main sources: the cutaneous, muscular and peritendinous nerve trunks. These nerve fibers do not actually enter the main body of the tendon, but terminate as nerve endings on its surface. Healthy tendons are brilliant white with a fibroelastic texture. Tenocytes and tenoblasts, constituting 90-95% of the cellular elements of the Achilles tendon, lie between the collagen fibers along the axis of the tendon (3). The fibers of the Achilles tendon are not aligned strictly vertically, but display a variable degree of spiraling or winding after the Achilles tendon forms at the fusion of the gastrocnemius and soleus muscles (2).

The Achilles tendon is considered a machine with multiple moving parts. The fibrils, fibers, and fassicles perform the basic function of force transfer to and from the skeleton during motion (4). The tendon is subject to the largest loads in the body, especially during running and jumping. The Achilles tendon can support as high as 9 kN during running, which is up to 12.5 times body weight. The fibers of the tendon spiral clockwise through 90 degrees during its descent, so the fibers that lie medially in the proximal portion become posterior distally. This provides a mechanical advantage to the Achilles tendon by allowing elongation and elastic recoil within the tendon to be possible. Stored energy can be released during the appropriate phase of locomotion (5).

Achilles tendon ruptures are common and can either be acute or chronic in nature. Acute ruptures, the more common injury of the two, occur when a sudden force is applied to the tendon. Chronic spontaneous ruptures are usually a degenerative process with no sudden trauma. Underlying factors, such as degeneration, can predispose the tendon to

an acute rupture (6). A number of factors influence the etiology of acute tendon ruptures including biomechanical, iatrogenic, and degenerative causes. Biomechanical influences can be intrinsic; a person having deformities such as tibia vara, an overly pronated foot, tight or underdeveloped hamstrings or a pes cavus foot can all predispose the individual to a rupture. Extrinsic factors include inadequate running shoes or training techniques. Diseases, such as gout, systemic lupus erythematosus, rheumatoid arthritis, and ankylosing spondylitis all predispose an individual to ruptures by affecting the elasticity and tensile strength of a tendon, both integral to its function. Corticosteroid use can be at risk for Achilles tendon ruptures. Although the anti-inflammatory properties of corticosteroids are beneficial for treating degenerative musculoskeletal disease, they may cause a delayed healing effect and direct necrotic action. Chronic degenerative changes and collagen alteration can also predispose an individual to Achilles tendon ruptures. The gastrocnemius-soleus complex contracts in an eccentric manner allowing fibers to lengthen as they contract. Lengthening allows the fibers to overcome an external force and dissipate the energy, advantageous when decelerating a plantar-flexed ankle. However, the elasticity of the tendon is dependent upon elastin and collagen fibers, which can degenerate (5).

The incidence of Achilles tendon ruptures has been increasing, particularly due to a recent increase in recreational sports participants being diagnosed with the injury. Participating in activities such as middle and long distance running, orienteering, track and field, tennis, and other ball games, are all risk factors for Achilles tendon ruptures. Previously, it was found that 80% of all acute ruptures were in high level athletes, particularly in sports demanding forceful plantarflexion or ‘push off.’ Now, Achilles

tendon ruptures are being increasingly found in individuals participating in just occasional sports and gym activities. It has been found that overuse and eccentric contraction during infrequent exercise can cause fatigue and microtrauma to the collagen in these individuals. Older age is also a risk factor for Achilles tendon ruptures. Elastin and collagen fibers degenerate with age. The degenerative process begins to play a role in the etiology of the disorder, especially in active middle aged individuals (30-35 years of age) (5). The incidence is highest in the 30-39 year age group and is 5.1 to 16.7 times more common in males than in females (7).

Achilles tendon ruptures are a commonly researched injury, and there is a vast amount of information on this topic.

My focus will be on literature that provides an overview of the injury and surgical repair of Achilles tendon ruptures. Topics I will cover will include history of the injury, risk factors, anatomy, etiology, diagnosis, prevention, treatments (both surgical and non-operative), rehabilitation, and long term effects.

History

Hippocrates was the first to record an injury to the Achilles tendon, claiming "this tendon, if bruised or cut, causes the most acute fevers, induces choking, deranges the mind and at length brings death." Due to lack of knowledge of infection and no clear distinction between nerve and tendon, the statement was widely accepted at the time (8). Even in the second century, Galen warned against poor results of tendon suture because he believed a tendon was a mixture of nerve and ligament; a suture within a nerve substance was likely to be followed by severe pain, twitching and convulsions. An

Arabian physician, Avicenna, performed the earliest procedures on tendons in the tenth century and in the twelfth century, the Italian surgeon Guglielmo di Faliceto stated that operative treatment was necessary to unite divided tendons (9). In the sixteenth century, Ambroise Pare performed the first recorded attempt to treat a subcutaneous Achilles tendon rupture. He strapped the Achilles tendon with bandages dipped in wine and spices, but found no true healing (8).

In the 17th century, Lanzweerde, with his experimental work on dogs, was able to perform successful Achilles tendon sutures after surgical division. Petit, also in the 17th century, described three cases of Achilles tendon ruptures; he advocated treatment using a bandage to keep the foot in plantar flexion; this treatment was widely accepted. Hunter, later in the 17th century, studied the natural process of tendon repair experimentally in dogs and concluded that tendon heals by formation of callus in a similar manner to the formation of bone callus. His case from 1776 where he used a plantar flexion bandage on a subcuatenous Achilles tendon rupture in himself resulted in excellent healing (9).

Haller, in the 18th century, demonstrated the specificity of tendon tissue; gradually operative treatment procedures on tendons became accepted. Billroth later asserted that the eliminated portion between tendon cuts hindered the growth of a scar, therefore delaying union. He asserted that absorption must first occur. The advances of antiseptics and anesthesia at the end of the 19th century finally made surgical treatment of tendon ruptures a viable alternative. Since then, numerous studies of tendon repair have been performed but conflicting views of tendon ends, tendon sheath, and surrounding connective tissue and blood remain (9).

Risk Factors

Achilles tendon ruptures have a multifactorial etiology and so it would make sense that there would be numerous factors that could put a person at risk for a rupture. Achilles tendon ruptures are most prevalent in middle and long distance runners, orienteers, track and field runners, tennis players, and other ball game players. Previously it was found that 80% of acute Achilles tendon ruptures were in elite athletes, particularly those participating in sport that required forceful plantarflexion or "push off." Now, Achilles tendon ruptures are increasingly found in individuals participating in simple occasional recreational sport and gym activities (around 62.3%). Achilles tendon ruptures are more prevalent in older individuals; elasticity of elastin and collagen fibers degenerate with age, predisposing a person to a rupture. This degeneration process begins between 30 and 50 years of age (5).

Both intrinsic and extrinsic risk factors have been found for Achilles tendon ruptures. Intrinsic factors include deformities such as tibial vara, an overly pronated foot, tight or underdeveloped hamstrings, and a high arched foot. Extrinsic factors include inadequate running shoes or training techniques. Diseases also can predispose a person to a rupture. Those with gout, systemic lupus erythematosus, rheumatoid arthritis and ankylosing spondylitis all are at risk for ruptures. These diseases affect viscoelasticity and tensile strength, both integral to tendon function (5). Hypertension, obesity, and hypercholesterolaemia also have been found to lead to ruptures (11). Taking certain antibiotics can also increase risk. Intake of fluroquinolones can increase risk by a factor of three to six (12). The taking of corticosterioids also increases risk; although the anti-inflammatory properties of corticosteroids are beneficial for treating degenerative

musculoskeletal diseases, they have a delayed healing effect and can have direct necrotic action on tendons (5).

There are a variety of potential risk factors for Achilles tendon ruptures that have been researched. A high serum lipid concentration is an intrinsic risk factor (13). It has also been speculated that Achilles tendon re-rupture is more likely if non-operative treatment was used initially. Because a mechanical disadvantage still exists, individuals are more likely to experience the injury again (14). Gender and race have also been noted as causes. It has been found that blacks (15) and men (11) are more likely to experience the injury, but it has not been discovered whether this injury is due to intrinsically being black or male, or that these populations are more likely to partake in the risk-related activities.

Incidence

There is a paucity of literature concerning the prevalence of Achilles tendon ruptures in the general population and even less so in the athletic population. However, it has been noted that the incidence of ruptured Achilles tendons, either spontaneously or not so, seems to be growing. It has not been determined whether this incidence is from an increasing percentage of the population or just a general growing population (16). Numbers have been reported as high as eighteen per 100,000 (8) and as low as two per 100,000 (7). It has been found that the peak incidence in men was from 30 to 39 years while in women the risk increased after the age of sixty years. By the age of 80, however, the incidence was greater in women than men (16). Other studies have demonstrated that Achilles tendon ruptures have occurred more often during sporting-related activities.

Postacchini and Pudu showed that 44% of their cases of rupture occurred during athletic activities (17). Cetti and colleagues reported that 83% of their patients in the study injured their tendons during activity (18). A Scandinavian study of badminton players showed that 52% of their patients with Achilles tendon ruptures were playing badminton at the time of injury (19). A Hungarian study showed that 59% of Achilles tendon ruptures happened during sport-related activities. This study also found that the sports that seem to have the highest incidence are recreational soccer, representing 33.5% of the cases, track and field, representing 16.2% of the cases, and basketball, representing 13.3% of the cases. There also seems to be a higher prevalence of Achilles tendon ruptures (53.7%) and re-ruptures (71%) in those with blood group O. Further, patients more commonly rupture their left Achilles tendon than their right, and most ruptures demonstrated histopathologic alterations on examination (20).

Anatomy

The Achilles tendon (AT), located in the posterior, superficial compartment of the gastrocnemius complex, begins near the middle of the gastrocnemius-soleus complex and stretches down to the calcaneus. The average AT length is 15 cm, but has been found to range between 11 and 26 cm. The AT is the conjoined tendon of the gastrocnemius and soleus muscles, with a small contribution from the plantaris. The relative contribution of the gastrocnemius and soleus ranges from person to person. The AT is very broad near the origin, but gradually becomes thinner in the mid substance and flairs out at the insertion. The average width at the origin, midsection, and insertion is 6.8 cm, 1.8 cm, and 3.4 cm respectively. The AT receives muscle fibers from the soleus almost to its

lower end. The fibers of the AT display a variable degree of spiraling. Tenocytes and tenoblasts make up 90-95% of the cellular elements of the AT and lie between the collagen fibers along the axis of the tendon. A healthy tendon is brilliantly white with a fibroelastic texture (2).

The AT is not encased in a true synovial sheath, but instead is surrounded by the paratendon. This single layer of cells is richly vascularized and supplies much of the blood supply for the AT. Blood reaches the tendon through a series of transverse vincula, which are passageways for the vessels. The paratendon functions as an elastic sleeve and allows free movement of the AT within the surrounding tissues. The AT also receives blood from vessels originating at the muscultendinous and osteotendinous junctions. There is an avascular area of the AT located 2-6 cm proximal to the insertion, known as the "watershed area." This section of the AT has poor healing potential and is associated with the pathology of multiple injuries (3).

The AT is innervated from three main sources: the cutaneous, muscular, and peritendinous nerve trunks. Most nerve fibers do not actually enter the body of the tendon but stop as nerve endings on the surface (3). The myelinated fibers' nerve endings function as specialized mechanoreceptors to detect changes in pressure or tension. The unmyelinated nerve endings act as nocioceptors, sensing and transmitting pain (5).

The AT is subject to the highest loads in the body, with the largest loads experienced during running, jumping, hopping and skipping. Its anatomy aids in its function. The calcaneal insertion has been described as the "enthesis organ" which aids in stress dissipation from the tendon to its bony attachment. This organ is formed by the

osteotendinous junction between the AT and the calcaneum, the sesamoid fibrocartilage near the dorsal deep surface of the tendon adjacent to the junction, a fibrocartilage layer covering the periosteum of the superior tuberosity of the calcaneum, and the tip of Kager's fat pad (2).

Etiology

The etiology of Achilles tendon ruptures is multifactoral, so therefore it is difficult to locate one specific cause. Achilles tendon ruptures can be traced back to degenerative changes, biomechanical influences, and iatrogenic causes (5). There are multiple cases of degenerative changes and collagen alteration in ruptured Achilles tendons. Chronic degenerative changes have been found at the site in majority of spontaneous ruptures (15).

Biomechanical changes associated with viscoelasticity and tensile strength have been linked to Achilles tendon ruptures. Elastin and collagen fibers and their metabolism are the primary contributors to these properties. Conditions and local diseases, such as gout, systemic lupus erythematosus, rheumatoid arthritis, and ankylosing spondylitis, affect fibers' metabolism. Elasticity of the elastin and collagen fibers is also important because elasticity provides a direct mechanical advantage to the Achilles tendon when overcoming an external force. The elasticity of the fibers is directly dependent on collagen density and maximum diameter, which naturally reduce with age starting around age 30 (5).

Taking of fluoroquinolone or steroid therapy can also contribute to Achilles tendon ruptures. Corticosteroids, although treating musculoskeletal disease, have a

delayed healing effect and can have direct necrotic action on tendons. Fluroquionolones can cause tendinopathy by altering the cytoarchitecture of tendons (21).

Acute vs. Chronic Ruptures

Achilles tendon ruptures are usually separated in to two broad categories: acute or chronic. Acute ruptures are the most common injury and are usually classified as a spontaneous complete rupture of the tendon. Acute injuries result from rapid force shifts to the lower limb. They generally present in sports such as football, basketball, track and field, volleyball, squash and badminton. Although labeled spontaneous, often times failed healing response changes are found in Achilles tendon ruptures, suggesting that there exists a pre-rupture phase and even a predisposition to rupture (22).

Chronic Achilles tendon ruptures, often called delayed ruptures, in the athletic population are extremely rare. Ruptures presenting after four to six weeks of the injury can be classified as chronic or delayed ruptures. Generally, an MRI is used for more precise evaluation of any patient where a chronic rupture is suspected for better understanding of the tendinous structure. In delayed ruptures, the tendons usually become a solid mass of scar tissue and it can be difficult to determine healthy or viable tendon and corresponding layers. Operative repair is almost always recommended for chronic ruptures; the goal of any ruptured tendon is to attempt an end-to-end anastomosis of the ruptures site, but with a rupture that has been neglected for a while, this becomes much more difficult (16).

Diagnosis

The diagnosis of Achilles tendon ruptures is generally simple and straightforward. Commonly, the rupture is diagnosed with a recording of the patient history and a clinical examination. The patient generally initially reports of pain and swelling in the posterior ankle and a traumatic event or feeling of being kicked in the back of the heel (8). The patients could also report having heard a popping sound. The patients may also complain of difficulty with generally walking, or more commonly walking uphill or up stairs (16).

During the examination, a common sign for Achilles tendon ruptures is calf atrophy compared with the contralateral leg. A clinician should also look for loss of Achilles tendon congruity or a palpable gap. The patient may also present with the inability to do heel raises or a weakness of ankle joint plantar flexion (16).

The most common clinical test used today was originally described by Simmonds and then made popular by Thompson and Doherty. The patient is first instructed to lie in prone with his/her feet hanging off the edge of the table. The clinician squeezes the largest muscle portion of the calf complex to simulate a contraction of the Achilles tendon complex. Normally, a patient should be able to produce plantar flexion of the foot. An “abnormal” reaction is observed when there is no plantar flexion response; this indicates a positive Thompson test (23).

Another diagnostic test was first described by Matles. Again, the patient is instructed to lie in prone on the table, but with the knees flexed at 90 degrees. The examiner evaluates the “resting tension” position of the feet. A patient with an Achilles tendon rupture will present with less plantar flexion or even a neutrally positioned or slightly dorsiflexed injured foot when compared with the uninjured leg (24).

Another diagnostic test is the Copeland test. With the patient prone and their ankle placed in passive plantar flexion by the clinician, a sphygmomanometer cuff is placed around the middle of the calf and inflated to 100 mm Hg. The examiner then passively dorsiflexes the ankle. With an intact normal Achilles tendon, dorsiflexion produces a rise in pressure of between 35 and 60 mm Hg. However, if the tendon is torn, there is little or no pressure rise. Often this test is performed while the patients are under anesthesia (25).

The O'Brien test is another test used for diagnosing Achilles tendon ruptures. The patients are put under either general or spinal anesthesia. The patient is placed in prone and a 21-guage needle is inserted at a right angle through the skin of the calf, just medial to midline, ten centimeters proximal to the superior border of the calcaneus. The needle is inserted until the tip is just within the tendon substance. The clinician then passively alternates placing the foot between dorsiflexion and plantar flexion. The normal response indicating that the tendon is intact is for the needle to swivel so that it points in the direction opposite to the motion of the ankle. So when the ankle is in dorsiflexion, the needle points distally. The abnormal response is that the needle does not move or moves slightly in the same direction as the motion of the ankle. When the ankle is in dorsiflexion, the needle does not move or points slightly proximally. This indicates that the Achilles tendon has lost its continuity between the needle and its insertion (26).

Maffulli went on to evaluate the sensitivity, specificity and predictive values of the Thompson test, palpable gap, Matles test, O'Brien needle test, and Copeland sphygomamanometer test. He concluded that all tests showed a high positive predictive

value. However, the Thompson test and Matles test were found to be significantly more sensitive than the other tests (27).

Other potential diagnostic modalities include radiographs, ultrasonography, and magnetic resonance imaging (MRI). Standard radiography is rarely used and usually not indicated. However, a lateral ankle view with radiography allows a clinician to rule out a posterior calcaneal avulsion fracture, possible distortion of Kager triangle, or evaluation of Toygar sign (27).

Ultrasonography and MRI are both useful diagnostic tools, especially when differentiating between a partial and complete Achilles tendon rupture. They give a more detailed evaluation of the tendinous structure with a chronic rupture. Ultrasonography is easy to use, inexpensive, allows dynamic imaging, and is able to measure the residual gap between tendon ends. An ultransonograhpic cimage of an uninjured Achilles tendon shows hypoechogenic bands of parallel fiber lines contained between two hyperechogenic bands in the longitudinal plane and round or oval shape in the transverse plane. The images of a ruptured tendon demonstrate discontinuity of the normal fiber pattern, a gap between torn ends, and an acoustic vacuum (16).

When Hartgerink evaluated twenty six suspected Achilles tendon ruptures with ultrasonography and compared the results with surgical results, he found that ultrasonography was accurate in distinguishing fullthickness tears from partial-thickness tears or tendinopathy with a sensitivity of 100%, a specificity of 83%, an accuracy of 92%, a positive predictive value of 88%, and a negative predictive value of 100% (28). The disadvantages of using ultrasonography include false diagnoses of high-grade tears if the plantaris tendon remains intact and difficulty in differentiating between all tendon

pathologies including partial ruptures of the Achilles tendon. In these cases, he argued MRI should be used (16).

Ultrasonography combined with Doppler examination has recently been used to help diagnose Achilles tendon ruptures, including partial tendon ruptures. Ultrasonography is used to evaluate tendon thickness and structure while Doppler examination is used to diagnose high blood flow. In a patient with an Achilles tendon rupture, ultrasonography shows an irregular dorsal tendon structure with a wavelike and disrupted superficial tendon line while the Doppler examination shows high longitudinal blood flow in the region with tendon changes. The region of high blood flow for the partial rupture is a sign of ongoing tendinous activity, such as after sustaining a partial rupture. Although in a chronic tendinosis condition, there is also high blood flow. This flow is often found inside and outside the ventral tendon, in close relation to the structural changes in the ventral tendon. Overall, the combination of ultransonography and Doppler examinations is a helpful tool in identifying partial ruptures that are in need of specific treatment (29).

An MRI uses sagittal and axial images with T1 and T2 weighted sequences recommended for the evaluation of Achilles tendon injuries. Normal tendons demonstrate low signal intensity (black) on all images. A ruptured tendon shows a signal disruption on T1 weighted images and high signal intensity on T2 weighted images, consistent with hemorrhage or edema with retraction of torn ends with a complete rupture of the tendon. MRI gives an adequate evaluation of the size of partial and intrasubtance tears, potential gapping of ruptured ends, and the amount of tendon degeneration or scar tissue (16).

Differential Diagnosis

Although Achilles tendon ruptures can be diagnosed with a simple clinical examination, 25% of ruptures are missed due to insignificant injuries, vague symptoms, and inadequate clinical assessment. Additionally, patients self-diagnose ankle sprains and delay presentation which may adversely affect treatment outcomes. There are some common diagnoses to be considered when suspecting acute Achilles tendon rupture, but key differentiating indicators that can help the diagnosis.

Patients with Achilles tendinopathy complain of pain and stiffness in both the morning and during exercise. Signs include focal tenderness, thickening and nodularity of the tendon. A simple Doppler with ultrasound scan can differentiate this condition. Medial gastrocnemius tears or tennis leg is another diagnosis to consider. Patients present with symptoms of pain and swelling and bruising in the medial calf. Clinicians usually notice a tender, palpable defect in the medial gastrocnemius. The combination of ultrasound and a Doppler examination can again rule out this condition. A calcaneal avulsion fracture should also be checked. The patient should report a history of trauma and present with swelling, pain, and difficulty weight-bearing. Plain radiographs and computer assisted tomography however should differentiate. Bursitis is another condition of the differential diagnosis. Patients experience pain with walking or putting on shoes. The clinician should check for pain with side-to-side compression anterior to Achilles tendon. Lastly, an ankle sprain should be ruled out. Throbbing pain with weight bearing, swelling, and focal tenderness are all signs of an ankle sprain. Simple radiographs can help investigate these conditions further (15).

Prevention

Although the exact etiology of Achilles tendon ruptures is unknown, there are some measures individuals can take to reduce the incidence. Avoiding degenerative changes within the tendon is the primary means of prevention. Collagen, the primary component of the tendon that helps resist applied forces, must retain necessary tensile strength, and production of the weaker type III collagen fibers must be avoided (21). Regular physical activity will help maintain the blood supply to the tendon, while irregular physical activity, such as for recreational athletes, increases one's risk for rupture. Maintaining regular physical activity levels with age also helps to promote tendon hypertrophy, increase nutrient delivery, and reduce fiber fatigue accumulation. Partaking in warm-up periods prior to exercise can also reduce the risk of Achilles tendon tears. Warming up increases temperature of the tissue and prepares the tendon for loading, increasing the extensibility of the fibers (2). Adding stretching in to the warm up period has not been demonstrated to prevent rupture, but may help enhance the extensibility of the fibers. Strengthening the ankle plantar flexors has also been shown to prevent rupture. Strengthening with eccentric exercises has been specifically shown to prevent rupture because of the force generated during eccentric contraction and with functional activity (30). Correcting foot misalignments with orthotics has also been shown to prevent rupture. Hindfoot and forefoot varus and valgus can be specifically targeted (30). Finally, avoiding fluoroquionlone antibiotics and corticosteroid abuse will prevent weakening of the tendon's extracellular matrix (31).

Non-operative vs. Operative Treatment

The optimal treatment for Achilles tendon ruptures is still controversial. Generally, it has been concluded that while surgical treatment results in a lower risk of re-rupture compared to non-operative treatment, the risk of infection and other complications of surgery is increased. Thirteen percent of Achilles tendon rupture cases with non-surgical treatment have resulted in re-rupture compared to about 2% with surgical treatment. However, surgery may result in infections, adhesions and disturbed skin sensibility (32). Non-operative treatment also eliminates the chance of wound complications and intra-operative sural nerve damage (33). As long as re-rupture and complications of surgery are avoided, conservative treatment and surgical treatment appear to produce the same functional results (34).

Non-operative Treatment

There are different recommended non-operative treatments. Cast immobilization has been shown to induce a delayed recovery, due to gastroc-soleus atrophy which results from prolonged immobilization of the ankle joint. Functional bracing does allow immediate weight bearing, reducing calf muscle weakness and enabling a faster recovery (33). However, casting is sometimes applied with a recommendation of weight-bearing activities from day one. This helps increase the strength of the healed tendon as fibroblasts and collagen fibers fill the tendon gap and orient themselves along the long axis of the tendon as a result of mechanical stress. Casts are commonly applied for eight weeks, with the ankle immobilized in plantar flexion for four weeks and in neutral for another four weeks. During the last weeks, orthosis can be used. The differences

between the results of non-operative treatments can be explained by the degree of plantar flexion in the cast, the use of the heel raise, and variations in instructions of the physiotherapist who may help with advising on how to ambulate and exercise during the immobilization period (34).

Open vs. Percutaneous Repair

Open repair of Achilles tendon ruptures has advantages over nonsurgical treatment since it results in a decreased rate of re-rupture, less residual tendon lengthening, less muscle atrophy, better ankle range of motion and faster resumption of higher level of sports activities (35). Open repair aims at restoring the anatomical length of the triceps surae through stump re-approximation (36). Open repair involves a variety of techniques ranging from Bunnel or Kessler sutures to more complex procedures including facial flaps or tendon grafts (37). Open repair can be performed under local, spinal, or general anesthesia (34). The disadvantage of open repair compared with nonsurgical treatment is the increased chance of complications. Most frequent complications include adhesive scars, infection, problems with wound healing, new rupture, sural nerve lesions, suture granuloma, deep venous thrombosis, and pulmonary embolism. Surgical wound healing is the most frequent of all these because the longitudinal incision, the most frequently used incision, is made on poorly vascularized skin (38).

Percutaneous repair of the Achilles tendon has become increasingly popular over the last few years (39). It has been proposed as an alternative to open repair based on the fact that there is a reduction in skin complications because the repair involves less invasiveness (40). Percutaneous repair is based on stump juxtaposition without exposure

of the rupture site, helping to spare the peritendinous sheaths and cutis and can be performed under local anesthesia using a wide variety of surgical techniques (41). Ma and Griffith described percutaneous repair initially using six skin incisions, three lateral and three medial to the Achilles tendon. The suture was criss-crossed through the tendon and tied on the tendon's surface. Patients experienced irritation from the subcutaneous knot which required late removal of the suture, but there was no injury to the sural nerve or re-rupture (39). Webb and Bannister later developed a technique of percutaneous repair of the Achilles tendon which minimized injury to the sural nerve and breakdown of the wound, while maintaining the re-rupture rate of the tendon similar with that achieved by open repair. The repair was carried out using three midline stab incisions over the posterior aspect of the tendon. A no. 1 nylon suture on a 90 mm cutting needle approximates the tendon with two box stitches. The result of the new technique was a low rate of complications (42). McCelland and Maffuli later modified this method by using stronger absorbable sutures and a Kessler suture (27). Percuatenous repair is increasing in popularity because it is performed on a day surgery basis and reduces cutaneous complications and surgery times, enabling a faster recovery (43).

Endoscopic Assisted Flexor Hallucis Tendon Transfer

Endoscopic assisted flexor hallucis tendon transfer has been recently introduced as an effective option for repair of chronic ruptures of the Achilles tendon. At the start of the operation, the patient is positioned prone with a pneumatic tourniquet applied to the thigh of the operated side (44). Patients can choose not to have the tourniquet (45). The posteromedial portal is then established at the intersection between the medial margin of

the Achilles tendon and a line joining the sustentaculum tali and the inferior border of the medial cuneiform and first metatarsal. The posterolateral portal is established one centimeter anterior to the lateral margin of the Achilles tendon insertion. Posterior ankle endoscopy is then performed. The flexor hallucis longus (FHL) is identified and the overlying fascia is released. The orifice of the fibro-osseous tunnel for the FHL identified and a Wissenger rod is placed underneath the sustenaculum tali. The plantar fascia is penetrated distal to the level of the navicular tubercle and the plantar portal is made. At this point, the FHL can be seen at both the plantar portal and the posteromedial portal; the tendon is cut with arthroscopic scissors just proximal to the knot of Henry and retrieved at the posteromedial portal. A stay stitch of No. 2 ethibond is applied to the free tendon end. In the next step of the procedure, a bone tunnel is made at the posterior calcaneal tubercle with a 4.5 mm drill bit through posterolateral portal wound. The exit point of the drill bit at the medial end is then cut open. The free end of the FHL tendon is passed from the posteromedial portal wound to the distal wound and then through the bone tunnel to the posterolateral portal wound. It is then passed through the posteromedial portal wound again (44).

The ankle is plantarflexed, the FHL tendon tensioned, and the free tendon end is sutured to the FHL tendon itself and to the distal stump of the Achilles tendon at the posteromedial portal wound. This reconstruction is helped by suturing the FHL tendon to the medial border of the distal stump of the Achilles tendon with a No. 2 ethibond through the posteromedial portal. Wounds are closed with 3.O ethilon and the patient is placed in an equine short leg cast (44).

The advantages of FHL transfer include using a long durable tendon with a stronger muscle than other tendon transfers (46), the axis of the FHL contraction closely reproduces the Achilles tendon (47), the FHL tendon offers stronger plantarflexion than other tendons (28), and the FHL is active in phase with the triceps surae (48). The advancement of the flexor hallucis tendoscopy allows for the flexor hallucis tendon to be harvested at the level just proximal to the knot of Henry without the need of extensive soft tissue dissection (44). Further, with the aid of posterior ankle endoscopy, the fascia overlying the flexor hallucis longus can be released, allowing the mobilization of the muscle during the transfer. With this endoscopic approach, the FHL transfer can be performed without the need of extensive soft tissue transfer (49).

Free Gastrocnemius Aponeurosis Flap

A new technique was initially performed in 1996 that involved a free gastrocnemius aponeurosis flap to cover the tendon gap after an end-to-end suture. During this treatment, the patient is initially placed in prone position under general or spinal anaesthesia. A 20-25 centimeter longitudinal slightly curved centrally incision is made extending from the middle third of the gastrocnemius muscle. The incision is medially curved distally in order to reduce the risk of injuring the sural nerve. Scar tissue is found between the tendon stumps so the tendon ends are carefully excised. A Kessler suturing technique with a 1-0 PDS is used for an end-to-end suture. The free gastrocnemius aponeurosis flap is then prepared. The length and the width of the flap depend on the size of the tendon gap, but usually the flap is around 3-5 cm x 5-10 cm. The free flap is placed over the ruptured site and secured with peripheral sutures using 3-

0 PDS. The flap covers the total tendon cap and 75% of the circumference of the tendon. Any defect in the aponeurosis is repaired using side to side sutures. After end-to-end tendon suture and flap reinforcement, the subcutaneous tissue and skin are carefully closed with low tension (50).

There were two reasons for developing this new method. One was to improve tendon augmentation while requiring only once incision and a fairly simple procedure. The second was to accomplish easier wound closure by avoiding thickening caused by a turn-down flap. The wound closure is easier when there is less thickening at the surgical site. This technique tended to cause less discomfort and pressure symptoms caused by the turn-down flap at the surgical site. The method also appears to allow more intrinsic tendon healing since the end-to-end suture is well covered by the flap. The limitations of the technique could involve the original size of the tendon gap, the extent of the scar tissue, and the time from index injury to treatment (50).

The "Pull-out" Wire Technique

The "Pull out" wire technique is an open repair surgical technique that provides rigid fixation, minimized tissue strangulation, no knots at the repair site, reapproximation of large gaps, and leaves no permanent foreign material in the body. The technique is performed under general or regional anesthesia. The patient first is positioned prone with a thigh tourniquet applied. The patient is given one gram of cephalosporin. Using an Esmarch bandage, the leg is exsanguinated and the thigh tourniquet is inflated to 275 mmHg (51). A posteromedial approach is used by the surgeon in order to avoid injuring the sural nerve (52). The surgeon makes a longitudinal incision over the posterior medial

aspect of the tendon directly through the paratenon. The wound is opened in one layer directly off the tendon so no flaps are created. At this time, the frayed ends of the tendon are debrided. The incision is made so that it extends proximally, exposing the confluence of the gastroc-soleus fascia. The wires are knotted over this dense tissue, providing the anchor to pull the tendon distally and close the tear (51). Two strands of 26-guage wire are woven through the proximal tendon with the Bunnell technique (53). The strands are threaded in parallel to the level of the os calcis and fastened distally around a partially threaded 4.0- mm AO cancellous screw that the surgeon inserts in to the calcaneal tuberosity. The surgeon makes sure the repair is tensioned, ensuring that the tendon is in continuity with the foot in neutral. The proximal strands are tied over the gastroc-soleus fascia and pulled out through the skin posterolaterally. Finally, the paratenon is repaired with 4-0 monofilament absorbable suture, the subcutaneous layer closed with interrupted 3-0 monofilament absorbable sutures, and the skin is closed with 4-0 simple nylon sutures (51).

Compressive plaster reinforcement is placed posteriorly with the foot in neutral. The patient elevates the foot for 48 hours and is instructed to be non-weight bearing with crutches. Six weeks after surgery, the screw is removed by the surgeon under local anesthesia. The distal loop is transected and the percutaneous wire is removed from its proximal exit site. The calcaneal wound is closed and the patient is placed in a short walking cast. One week later, the cast is replaced with a removable cast boot and the patient is allowed to weight bear as tolerated (51).

The advantages of this procedure include the advantages of an open operative repair. It provides the lowest rate of rerupture and a high level of function (18). Further,

the “Pull-out” wire technique provides a wire that is rigid and the foot can be immobilized in the neutral resting position without gapping the repair site. Also, the tendon’s normal resting length is maintained by rigid internal flexion for six weeks after surgery. The pull out wire technique also minimizes circumferential tendon constriction because it avoids multiple crossed locking sutures of traditional repairs. This helps to minimize the restriction on the tendon’s blood supply. Also, the strength of the wire in this technique allows re-approximation of tendon ends when extensive debridement is needed or shortening is encountered. There are no knots needed at the repair site. Finally, when the initial healing period is complete at six weeks, all hardware is removed. Therefore, no foreign material is left behind as in standard suture or grafting techniques. Chronic inflammation and irritation or late infection can result from foreign material left behind. The primary disadvantage of the “Pull-out” wire technique is that a second anesthetic and OR visit are necessary. Further, several women had difficulty with certain types of shoe wear post-operatively, relating to both the incision and a hypertrophic Achilles tendon. Patients should be aware of a possible sequel of a thickened tendon after repair and the fact that the incision can serve as a source of irritation as well (51).

Peroneus Brevis Transfer

The peroneus brevis transfer is a satisfactory option for a successful repair of the Achilles tendon. It overcomes the multiple factors that challenge a successful repair: (1) suturing of a shredded tendon (2) reestablishment of physiological tension (3) weakness associated with a lengthened tendon (4) revitalizing an ischemic injured tendon and (5) difficulty obtaining secure fixation when the insertion is avulsed from the calcaneal

tuberosity. A healthy musculotendinous transfer allows a secure suture of the shredded proximal and distal Achilles tendon fragments under normal physiologic tension. Further, suturing to a healthy tendon splint is very secure and the retracted proximal calcaneal tendon can be brought distally to be sutured also under normal tension to the peroneus brevis (54).

The operative peroneus brevis transfer procedure is performed under general anesthesia with the patient in prone position under tourniquet control. A straight incision lateral to the heel cord between the peroneal tendons and the lateral margin of the rupture is made so that the tear can be identified. The sural nerve is identified as well just beneath the skin so it can be protected. The nerve varies in location, but needs to be identified, isolated, and retracted before the peroneus transfer to the Achilles tendon. The incision should not be made directly over the Achilles tendon; a midline incision limits access to the peroneal muscle compartment, making it necessary to do more skin damage (54).

The peroneal tendons are identified in the lateral compartment and mobilized. The peroneus brevis is identified by its attachment to the fifth metatarsal, being more muscular distally than the longus, lying posterior and deep to the peroneus longus, and having a smaller excursion than the peroneus longus. A second incision is made at the base of the fifth metatarsal, and the broad attachment of the brevis to the metatarsal styloid is sharply detached. The distal portion of the peroneus brevis is cut subcutaneously and delivered into the proximal wound. The lateral intermuscular septum is excised between the peroneal compartment and the Achilles tendon before the tendon is transferred. Usually a good distal stump of the Achilles remains unless the tendon has

been entirely avulsed from the calcaneal tuberosity. The tendon is anchored distally to the remaining stump of the Achilles tendon (54).

A scalpel is used and guided in retrograde fashion through the distal ruptured fragment of the Achilles tendon. The peronus brevis is passed through the calcaneal tendon from lateral to medial and then sutured with physiologic tension to the lateral and medial aspects of the distal Achilles. In the case of a calcaneal avulsion, the brevis must be anchored to the calcaneus via a drill in the os calcis. The proximal shredded Achilles tendon is pulled distally and secured to the U-shaped tendon transfer in a neutral to five degrees of equine position. Special care is taken to suture the peroneus tendon in the mid-coronal plane of the Achilles tendon so the tendon transfer does not dislocate posterior to the ruptured tendon. The two ends of the Achilles tendon are now sandwiched between the peroneus brevis tendon. The new ends of the Achilles tendon are secured even further by direct end-to-end suture. At the end of the operation, the tendon sheath is closed if possible, and the sural nerve is placed in its anatomically superficial location. A routine wound closure is performed (54).

The primary advantage of the peroneus brevis surgery is that it minimizes calf atrophy. The physiologic tension is restored to the muscultodinous unit early to allow normal muscular tone. The soleus muscle is particularly sensitive to disuse because of its high proportion of type I fibers and its function in postural adjustments. The peronus brevis transfer has other advantages as well. It simplifies the repair technically, improves the biologic mileu for healing by using healthy tendon as scaffolding, and hastens complete rehabilitation (54).

Semitendinosus Graft Transfer

A free semitendinosus graft transfer is a surgical technique recommended for reconstruction of chronic Achilles tendon ruptures with large defects over six centimeters. The procedure is performed with the patient prone under spinal or general anaesthesia. The ankle is placed in plantarflexion as a longitudinal posterior midline incision is made over the heel cord, exposing the stumps associated with the rupture of the Achilles tendon. The scar tissue between the stumps and the ends of the tendon are excised until only viable tendon tissue remains, and the length of the tendon defect is measured. If the tendon gap is greater than six centimeters, the surgeon prepares for a semitendinosus graft transfer. The semitendinosus tendon is harvested with a vertical incision over the pes anserinus. The semitendinosus graft is passed through a small incision in the proximal stump of the Achilles tendon in a mediolateral direction. The graft is then pulled downward and passed through a small incision in the distal stump in the same direction. Finally, the graft is pulled upward in the same manner to form the figure eight. The semitendinosus tendon is sutured to the Achilles tendon at each entry and exit point. The wound is closed to complete the surgery (55).

The semitendinosus tendon graft is recommended in patients with large defects because of its long and strong form. While local tendons are insufficient in providing a strong enough graft, the semitendinosus graft provides a robust reconstruction to the ruptured Achilles tendon (55). The free semitendinosus graft transfer provides the foot with motor strength and power, is safe, and because of its length, can be used with large gaps (56). Harvesting the tendon is easy and is associated with no functional deficits. It also preserves the sural nerve and reduces damage risk (55). The largest advantage of this

technique is that the surgeon can perform a semitendinosus tendon augmentation in a minimally invasive fashion, preserving skin integrity. Preservation of skin cover during reconstruction procedures is a large advantage because the skin is not injured by the operation, and therefore can protect the reconstruction beneath. Disadvantages of the semitendinosus graft transfer include a technically demanding procedure and complications of surgery. Wound breakdown, infection, and DVT are more common complications of surgical repair (56).

Classifications of Achilles Tendon Ruptures with Recommended Surgical Techniques

Myerson and Kuwada have attempted to provide recommendations based on the resultant gapping in chronic ruptures. Myerson recommends an end-to-end anastomosis and a posterior compartment fasciotomy for a type 1 defect, which he defines as a defect no more than 1 to 2 cm in length. A defect that ranges from 2 to 5 cm in length is defined as a type 2 defect and can be repaired with a V-Y lengthening combined with a possible flexor hallucis longus (FHL) tendon transfer. Finally, a defect that is longer than five cm is a type 3 defect and can be bridged with an FHL tendon transfer with possible V-Y lengthening (46).

Kuwada recommends the following. Type I lesions are partial tears can be managed with conservative casting. Complete ruptures smaller than 3 cm are type II lesions and can be repaired with end-to-end repair. Ruptures of 3 to 6 cm are type III lesions and can be repaired with autogenesis turndown flaps and/or synthetic grafts. Finally, ruptures larger than 6 cm are type IV lesions and can be repaired with gastrocnemius recession, lengthening, and/or free tendon transfer (57).

Rehabilitation

Traditionally, methods of rehabilitation for Achilles tendon ruptures have involved immobilization within a below-knee non-weight bearing plaster cast for six weeks then subsequent ankle range of motion and strengthening exercises. More recently however, research has indicated that newer methods of rehabilitation involving immediate weight bearing as early as the first postoperative day can lead to better results. Early range of motion exercises and splinting in a walking boot or modified orthosis instead of a cast have led to a faster recovery (58).

Many researchers have modified, developed, and reported new rehabilitation techniques with functional bracing. The rehabilitation methods differ based on the design of the orthotic, the degree of plantarflexion in the orthotic, how long the orthotic was worn, and the type and frequency of range of motion exercises (59).

The first researchers to report of an immediate weight bearing functional bracing rehabilitation were Speck and Klaue in 1998. The researchers used a rigid rocker orthotic worn for six weeks. The orthotic was worn with the foot in neutral. The patients were weight bearing on the first day post operatively and performed ankle exercises four times a day. The patients had positive results and the researchers concluded that immediate weight bearing was safe (60).

Costa and his research team modified this method by using a carbon-fiber orthosis with three 1.5 cm heel raises. Range of motion was improved by removing one heel raise every two weeks until a plantigrade position of the ankle was achieved. The orthosis was removed at eight weeks. Patients were allowed to be weight-bearing from day one post-operatively. Costa and his research team found that with their functional bracing

rehabilitation program, patients returned to work and sport quicker, were able to walk and climb stairs quicker, and had greater strength than patients with a non-weight bearing rehabilitation program (61).

Majewski reported a different functional rehabilitation program using a special shoe following surgery. After surgery, patients were given a splint that holds the foot in 20 degrees of plantar flexion for 24 hours. On day two, a specially designed shoe was given to the patient to wear during the day while still wearing the splint at night. The shoe provided a high shaft for the injured side and offered anterior, medial, and lateral stabilizers, as well as a 3-cm heel wedge held within the shoe to keep the ankle at 0 degrees of flexion. The patient wears a similar shoe with a 3-cm wedge on the uninjured foot to prevent pelvic tilting. The patients were again allowed to perform full weight bearing activities. After four weeks, the height of the heel wedge was reduced from 3 cm to 1 cm in weekly intervals until week eight. Also, the patients began partial weight-bearing exercises with bare feet. After eight weeks, the patients followed the same protocol with the same amount of physical therapy and the stabilizers were removed. The researchers reported the patients of their rehabilitation program achieved greater range of motion, greater strength, greater calf circumference, less pain, and a quicker return to work and sport (62).

Functional rehabilitation in place of immobilization following surgery has presented with many advantages. Animal models have consistently shown the detrimental effects of immobilization on both tendon and muscle tissue following Achilles tendon ruptures and the potential benefits of early movement and controlled loading. Benefits include improved tendon characteristics through maturation and orientation of collagen

fibers as well as decreased muscle atrophy. Early functional rehabilitation has proved to improve range of motion faster, reduce re-rupture rate by making a greater force required for re-rupture to occur, and reduced atrophy of the gastroc-soleus complex. This last advantage is especially important in older patients, where atrophy of the gastroc-soleus complex usually leads to gait abnormalities (59). Patients having undergone functional rehabilitation tend to also have decreased complication rates, including scar adhesions and transient sural nerve deficits (63).

The key to the functional rehabilitation program is that progression of rehabilitation needs to be balanced against the clinical risks of re-rupture and tendon lengthening. Tendon lengthening can present if the rehabilitation method does not provide adequate restriction of movement during the early phases of rehabilitation. Tendon re-rupture will occur if too much load is applied to the tendon before it is healed (64). Restricted range of motion can be safely started at the end of fibroplasia, but early motion is recommended following tendon repairs to prevent adhesion formation. There has been concern that active rehabilitation following mini-open techniques would lead to wound problems or an increased re-rupture rate, but results with an open surgical technique and rehab are optimal. Early introduction of an active rehabilitation program appears to provide a rapid return to social and occupational activities (65).

Changes in Gastroc-Soleus Strength and Endurance Following Achilles Tendon ruptures

Gastroc-soleus muscle endurance and strength are common deficits observed after Achilles tendon ruptures. The strength deficit of the musculature is approximately 10-

30% on the injured side compared with the uninjured side, while the endurance deficit is approximately 12-48% after one year (1, 2). These deficits are not only related to everyday functioning, but they are very difficult to overcome. Muscle endurance and strength are related to the function of the gastrocnemius-soleus-Achilles complex which is crucial in gait, jumping, and running (66). Examining muscle endurance is an important outcome in the evaluation of a rehabilitation program aimed at the recovery of Achilles tendon ruptures repair and preventing possible future problems (67).

The heel-rise test is recommended to measure plantar flexion muscle endurance during recovery of an Achilles tendon rupture. It helps to find a weakness in end-range plantar flexion. In the heel-rise test, a linear encoder is used. A spring-loaded string is connected to a sensor inside the linear encoder unit. The spring-loaded string of the linear encoder is attached to the heel of the participant's shoe. When the string is pulled, the sensor outputs digital pulses that are proportional to the distance travelled. One pulse is approximate equal to 0.07 mm. By counting the number of pulses over time, the displacement as a function of time can be recorded and used to calculate force, velocity, and power. The patient performs the heel-rise test on one leg at a time standing on a box with an incline of ten degrees. The participants are allowed to place two fingertips with each hand, at shoulder height, against a wall for balance. A cassette player with a recorded voice says "up" every two seconds to maintain a frequency of thirty heel-rises per minute. The participant goes as high as possible on each heel-rise and then lowers his/her heel to the starting position and waits for the next "up" signal. The participant performs as many heel-rises as possible. The test is terminated when the patient stops, cannot maintain the frequency, or does not perform a proper heel rise (66).

The number of heel rises, the height of each heel rise, and the total work are recorded. These recorded values can be used to assess calf muscle endurance and strength. The injured side generally has a significant decrease in height and number of repetitions when compared with the uninjured side, indicating a loss in muscle endurance (66).

Researchers have made many speculations on the possible explanation for the incomplete recovery of muscle endurance after Achilles tendon ruptures. One possible explanation is there is an increased tendon length, causing a non-optimal length-tendon relationship in the muscle tendon unit. Researchers have found that patients, who are male, have Achilles tendon pain at three months, and present with lower physical functioning at six months tend to have a delayed muscle endurance recovery at one year. Achilles tendon pain at three months could be a potential early marker for patients and therapists of poor prognosis. If therapists notice the Achilles tendon pain at an earlier stage, they can modify rehabilitation in an attempt to help delays in recovery of muscle endurance. Being male could be a possible risk factor in delayed calf muscle endurance because males tend to place extra weight on the recovering tendon, increasing stretch of the tendon and causing a non-optimal length-tendon relationship in the muscle-tendon unit (67).

Changes in Gait Following Achilles Tendon Ruptures

Ankle plantar flexion is changed by Achilles tendon ruptures. Because plantarflexion is an important component to gait, this daily motor activity is affected by the injury. A patient with an Achilles tendon rupture generally does not recover normal

biomechanical properties of the calf-muscle in the first year, but they are still able to walk. Patients make adaptations in response to their limitations in plantarflexion that in many cases can be detrimental over the long term. For example, patients generally present with an eccentric strength impairment that is responsible for some adaptive changes that could be considered negative for the healed tendon over a long term. Although patients may be satisfied because they are able to mobilize after Achilles tendon ruptures, gait generally is affected in some way by the rupture (68).

There are two phases for the plantar flexor muscle-tendon unit during the gait cycle, one in which the plantar flexion muscle mass is lengthening and one in which this muscle unit is shortening. During the lengthening phase, the ankle performs dorsiflexion that is restrained by plantar flexor stiffness, but after activation by plantar flexor muscles' eccentric work. Although it would be expected that a low degree of passive stiffness and eccentric strength would determine a mild angular impulse with an increased ankle range of motion. In contrast, more passive stiffness may contribute to a stronger plantar flexor angular impulse and limited range of motion. During gait after Achilles tendon ruptures, patients generally have lower values of angular impulse and restrained ankle dorsiflexion movement for the involved side, associated with low soleus activity and talus-soleus co-activation. There is a reduced plantar flexor recruitment attributed to adaptive changes aimed at minimizing active resistance to ankle dorsiflexion that compensates for the increased passive stiffness. This adaptation may contribute to the development of an eccentric strength deficit by reducing calf-muscle recruitment during eccentric tasks. Also, later in recovery, after normal passive stiffness is achieved and there is an increase in ankle dorsiflexion, an increase of calf muscle activity may be responsible for placing

additional stress on the Achilles tendon. Overall, during the lengthening phase of the gait cycle, the different recovery of calf-muscle passive stiffness and eccentric strength may be the main factor in determining changes of motor strategy predisposing the patient to injury over the long term (68).

Increased gastroc-soleus extensibility in the early postoperative rehabilitation phase is recommended because of the relationship between extensibility and stiffness. Therefore, exercises such as early ankle mobilization can be encouraged to prevent the onset of anomalous muscular recruitment, the development of eccentric strength deficits and the increased stress placed on the Achilles tendon and ankle joint structures. A proprioceptive training program could enhance muscle activity modulation through afferent feedback and strength gain and from use of neuromuscular facilitation techniques, increase muscle compliance (68).

The gastroc-soleus muscle shortening phase of gait cycle is marked by plantar flexor muscles undergoing concentric contraction, providing energy for a leg forward acceleration. Plantar flexor muscle activity produces the angular impulse that determines ankle plantar flexion and contralateral step length. In this phase, neuromuscular coordination reduces the dorsiflexor muscle co-contraction to reduce plantar flexor mechanical output. Plantar flexor muscles concentric strength loss combined with neuromuscular impairment associated with the Achilles tendon rupture is responsible for reduced angular impulse, ankle range of motion and contralateral step length. Although restoration of concentric strength helps these parameters of gait, it does not completely restore the parameters to normal. The continued presence of tibialis anterior-soleus co-activation during walking reduces the plantar flexor moment and its mechanical effects.

The co-contraction pattern increases the effort required to deliver energy for push-off, decreases mechanical output of plantar flexor activity and places further stress on the Achilles tendon by requiring more gastroc-soleus muscle activity (68).

In rehabilitation, patients should be re-conditioned to selectively activate the muscles in the push off task. They may benefit from gait rehabilitation, electromyographic bio-feedback and proprioceptive neuromuscular facilitation techniques that include the theory of reciprocal inhibition (68).

Marked Pathological Changes

In ruptured Achilles tendons, the tissue in the ruptured area undergoes large changes, down to rearrangement at the molecular level. Tendon changes are not only localized at the site of the rupture, but can be observed also in the macroscopic intact proximal and distal tendon portion. However, pathological features are significantly more pronounced directly at the site of the rupture (22).

Fiber structure and arrangement are both affected by Achilles tendon ruptures. In normal tendons, fibers are arranged close and parallel to each other with a slight waviness. After ruptures, increased waviness and separation of the fibers accompany slight and moderate ruptures. More severe Achilles tendon ruptures show loss of finer fiber structure. Fiber arrangement is also impacted by Achilles tendon ruptures. In intact tendons, the fibers are arranged parallel to each other, but in ruptured samples, this parallel arrangement is completely lost (22).

The tenocyte nuclei of the tendon are also markedly different in ruptured Achilles tendons. The tenocyte nuclei of healthy tendons are flattened and spindle shaped,

sometimes even arranged in rows. In ruptured tendons, the tenocytes first decrease in number. Then, as pathologic changes progress, the nuclei become progressively rounded. In some instances, these tenocytes can even resemble chondrocytes (22).

Marked changes in cells can be observed in ruptured Achilles tendons. The degree of cellulairty is greater in ruptured tendons. Vascular bundles, which normally run alongside the collagen fibers in healthy Achilles tendons, increase in number. With more advanced changes in the tendon due to rupture, the number of vascular bundles increases. Collagen sustainability is also impacted by Achilles tendon ruptures. Normal collagen changes color to a deep pink-red when hematoxylin and eosin stain are added, indicating sustainable stronger collagen. However, with ruptured Achilles tendons, the collagen is considerably paler. This indicates degeneration of the collagen (22).

Deep Vein Thrombosis Following Achilles Tendon Ruptures

The incidence of asymptomatic and symptomatic deep venous thrombosis (DVT) is high after Achilles tendon ruptures. DVT is a feared effect following either surgery or conservative treatment of Achilles tendon ruptures because of its association with pulmonary embolism and cardiovascular events. It is a common complication following surgery to the lower leg and a common observed effect following immobilization for patients undergoing a more conservative treatment of Achilles tendon ruptures. DVT can present a major problem because after injury or surgery to the lower limbs, a clinical diagnosis of DVT is unreliable. This is because the pain and swelling caused by trauma, surgery and immobilization make the usual signs and symptoms of thrombosis difficult to interpret. Additionally, the majority of venous thrombi are asymptomatic, making it

harder to diagnose. In some patients, clots can resolve spontaneously, but in others, they can propose serious health risks and complicate asymptomatic distal thrombi (69).

Color duplex sonography (CDS) can screen for DVT. CDS is a good method because of the recent advancements in image resolution and transducer technology and the addition of color Doppler. Further, it is a non-invasive method, making it more comfortable for the patient. It is also less expensive and the examination can be performed quicker compared with the other testing method, venography. Thromboprophylactic measures have reduced the frequency of venous thromboemboolism and the use of thromboprophylaxis has become routine after orthopaedic surgery for Achilles tendon ruptures (69).

Changes in Running Following Achilles Tendon Injuries

Running is an important element that is affected by Achilles tendon ruptures. The changes resulting from healing present some difficulties to the patient when running. Most patients present with changes in both their kinematics and kinetics. Generally, most studies on the running of Achilles tendon ruptures base their study on comparison between the unaffected limb and affected limb because it is rare to have mechanical data on running that precede the injury.

It has been reported that most patients return to sports and running after Achilles tendon ruptures. However, the majority of these patients report residual weakness and decreased function in their affected limb after treatment. This has been hypothesized to be due to the tendon healing in an elongated position or the post injury calf muscle weakness that is reported (70).

Achilles tendon ruptures result in gait asymmetries such as increased ankle dorsiflexion and decreased plantar flexion moment of the involved limb. The elongation of the healed tendon is one explanation for increased dorsiflexion range of motion and peak dorsiflexion observed during running following injury. Plantar flexion weakness is another proposed explanation for increased dorsilexion, since the gastroc-soleus works eccentrically during stance to limit the amount of dorsiflexion. The inability of plantar flexors to generate power has also been shown to limit stability in the secondary planes of movement of the ankle, primarily in inversion and eversion. The differences observed in the involved limb are likely associated with a limited ability to generate plantar flexion power (70).

Reduction in peak moment and power generation at the ankle appears to be due to both the strength deficit and tendon elongation. The alteration of the length-tension relationship (due to greater tendon length) may also affect the ability for the person to utilize the stretch-shortening cycle. Although this defect is primarily observed with jumping, which requires significant power generation, running is also affected (70).

A large issue with abnormal running mechanics observed after injury is the possibility of re-injury. Alteration in the involved limb biomechancis may result in an increased loading on the uninvolved limb. Patients with Achilles tendon rupture have nearly a 200-fold increase in risk of a contralateral tendon rupture (71). Alterations in involved limb mechanics also place higher demand on other structures within the limb. For example, greater demands are placed on the posterior tibialis muscle because the plantar flexors in an injured Achilles tendon have limited ability to stabilize the rearfoot in the frontal foot. This places an individual at greater risk for injuries like tendinopathy.

Greater power absorption and power generation are observed in the knee of the involved limb compared to that of the uninvolved limb. Increased demand on the knee extensor mechanism place this individual at greater risk for overuse injuries, such as patellofemoral pain syndrome or patellar tendinopathy (70).

Following a rupture, patients have been found to have long-term deficits in strength, heel-rise height and tendon length. However, a majority of patients still return to physical activity, sports, and running. Therefore, it should be encouraged to return to activity after an Achilles tendon rupture, but alternative activities like bicycling may be advisable in the presence of running biomechanical alterations (70).

Conclusion

The incidence of total Achilles tendon ruptures has increased during the past decade in western countries. The rise is more prominent among young males than among any other population. The ruptures occur dominantly in recreational athletes participating in sports requiring bursts of energy like in jumping, running, or pivoting.

The exact etiology of Achilles tendon ruptures remains unknown. However, Achilles tendon ruptures can generally be traced back to degenerative changes, biomechanical influences, or iatrogenic causes. Degenerative characteristics are usually present in Achilles tendons presenting with rupture, but the amount of degeneration between individual tendons ranges. The degeneration has suggested to be from decreased arterial blood flow, resulting in local hypoxia and impaired nutrition and metabolic activity. A sedentary lifestyle has been the primarily proposed reason for poor circulation in the tendon.

Surgery has been the primary treatment choice since the 1990s, especially for the younger, high demand athletes and in chronic untreated injuries. Conservative treatment can be considered generally for older lower demand individuals. Which surgical treatment is superior has been under review, but generally, the type of surgical treatment depends on the condition of the Achilles tendon ruptures. Local tendon grafts have been proposed for ruptures presenting with a small gap, while larger gaps have been suggested to be treated with stronger grafts such as the semitendinosus graft.

Functional, weight bearing rehabilitation has been the primary postoperative choice since being proposed in 1998. Early movement prevents the negative side effects of immobilization, but should be used with caution. Range of motion exercises and weight bearing activities have been proposed to be implemented as early as the first post-operative day for Achilles tendon ruptures.

There are many detrimental effects of Achilles tendon ruptures. The primary result is tendon elongation which affects the gastrocnemius-soleus complex strength and endurance. There are also notable histopathological changes in the tendon after rupture. Fiber arrangement, fiber structure, collagen sustainability, and cellular structure are all impacted by an Achilles tendon rupture. The primary effects now being observed with respect to Achilles tendon ruptures are gait effects. During the gait cycle, the gastrocnemius-soleus complex is critical at push-off, and without adequate strength, gait can suffer. Adapting to the deficits in strength, patients can change their gait in ways that may be detrimental to them over a long period of time. Therefore, it has been suggested more recently that gait be inserted in to a rehabilitation program to monitor changes and keep a patient on track.

Running is also affected by Achilles tendon ruptures, but not as many studies have been reported. The primary results are increased dorsiflexion and decreased plantarflexion which result in abnormal running mechanics. These issues are thought to arise from calf muscle weakness and tendon elongation. However, it is common for a person to recover, and running and physical activity should be allowed after Achilles tendon rupture to return a patient to full functioning.

Overall, Achilles tendon ruptures are growing increasingly common and should be evaluated and treated as quickly as possible to promote healing. Although altered mechanics and changes in tissue often result, a near full recovery is possible. Therefore, patients should partake in functional activities during rehabilitation.

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