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Current Strategy in the Treatment of Achilles Tendinopathy

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1. Introduction

Achilles tendinopathy is a common condition that has a relatively high morbidity in both the general population and in athletes. The most commonly used and accepted theory is that it is a degenerative tendinopathy caused by relative overuse in weightbearing activities, and this is especially the case in chronic cases. In the early stages of symptoms it is more likely that there will be no obvious degenerative changes and this has been termed tendinosis and is considered reversible. Achilles tendinopathy can be a difficult condition to treat, especially when it is chronic, and it is important to address causative factors in the management of this condition to ensure symptom reduction and minimize the risk of recurrence. In order to do this, a sound knowledge of anatomy and pathophysiology is required to accurately assess the musculoskeletal regions impacting on the loading of the Achilles tendon. As with all tendinopathies, the basic principles of evidence based exercise rehabilitation should be the mainstay of treatment. All other therapies should be used as adjuncts to this. Many medications can be trialed as therapy is more recalcitrant cases, but caution must be used to weigh the known risks and benefits and "first do no harm" ("primum nil nocere"). Achilles tendinopathy is a condition that can be frustrating to treat and requires careful management and patience to ensure optimal outcomes.

2. Anatomy

The Achilles tendon is a large strong tendon that joins the calf muscle to the posterior aspect of the calcaneus. The gastrocnemius muscle is formed from two heads that arise from the posterior femur above the knee joint and join in the calf with the soleus muscle, which arises from the posterior tibia and fibula below the knee joint. These two muscles merge to form the Achilles tendon in the mid-calf. The gastrocnemius and soleus muscles and their tendoAchilles are the primary ankle plantarflexors. The soleus component of the Achilles tendon twists medially as its approaches the Achilles insertion and, occasionally, an accessory soleus muscle is present and inserts onto the posteromedial calcaneus separate to the Achilles tendon.

At the insertion onto the posterior calcaneus there are two bursae associated with the Achilles tendon, the retrocalcaneal bursa that slides in the angle between the

posterosuperior calcaneal border and the anterior aspect of the distal Achilles tendon (Figure 1), and the retroAchilles bursa that slides between the posterior Achilles tendon at its insertion and the overlying skin and subcutaneous tissue. These bursae have a lubricating function to reduce friction during ankle movement, but can become involved in the pathologic process.



Fig. 1. MRI sagittal image demonstrating the Achilles tendon anatomy. The arrow indicates the Achilles tendon (black) inserting onto the posterosuperior calcaneus and also shows a small fluid collection in the retrocalcaneal bursa (white) between the anterior aspect of the Achilles tendon and the posterosuperior calcaneus.

The Achilles tendon is surrounded by a fine sheath, the paratenon, that is not a synovial sheath but has lubrication and friction reduced properties. It is recognized that the paratenon may be involved in the pathologic process of Achilles tendinopathy, and may contribute to loss of friction reduction and potentially increased tendon fibre strain and thus degeneration.

3. Pathophysiology

Tendons are relatively slow metabolically, with approximately 10-13% of the oxygen uptake of muscle. Tendons heal at a slower rate than muscle and take approximately 100 days (over 3 months) to form biomechanically strong collagenous scar tissue with enough tensile strength and elasticity to accept the forces applied to the Achilles tendon during heavy weightbearing activity such as running or jumping. This slow metabolic rate and slow healing rate have implications for treatment of the Achilles tendon in order to prevent injury recurrence.

Chronic Achilles tendinopathy is almost exclusively a degenerative tendinopathy with histopathologic features of collagen fibre disruption and disorientation (fibre tearing), mucoid degeneration, new blood vessel formation (neovascularisation), and an absence of inflammatory cells. It is due to the absence of inflammatory cells histopathologically that the

general descriptor "tendinopathy" is now preferred to the inflammatory descriptor "tendonitis". It should, however, be recognized that the histopathologic description above applies to chronic tendinopathy and it is possible, some may say highly likely, that is the acute stages of Achilles tendon injury there is an inflammatory component in the paratenon and possibly the tendon itself. There may also be symptoms with pathologic changes, this is termed "tendinosis", and is considered a reversible condition due to the lack of observed tendon changes.

Achilles tendinopathy is classified into two types, insertion Achilles tendinopathy that is commonly associated with bursitis, and non-insertional Achilles tendinopathy that is classically location in the relatively hypovascular "watershed" region of the tendon 4-6 cms from the calcaneal insertion. Non-insertional Achilles tendinopathy is rarely associated with bursitis but may have more of an element of paratenon involvement. Recent research has suggested a genetic association between the COL1A1 gene locus and the development of non-insertional Achilles tendinopathy in Caucasian populations in South Africa and in Australia. Other suggested risk factors for the development of Achilles tendinopathy include both intrinsic and extrinsic factors (Table 1). Many of these risk factors and their cause and effect relationship with Achilles tendinopathy remain to be scientifically tested and proven.

Intrinsic Risk Factors	Extrinsic Risk Factors
Genetics (COL1A1)	Training
	Insufficient warm-up/ stretching
Biomechanical abnormalities:	training frequency
leg length discrepancy	training intensity
subtalar hyperpronation	training duration
muscle asymmetry/ tightness	training volume
joint asymmetry/ stiffness	monotony of training
joint instability	cross-training
(e.g. lateral ankle laxity, peroneal	
subluxation)	Surfaces
	slopes/ hills/ cambered surfaces
Rheumatic disease:	change to hard or soft surface
rheumatoid arthritis	unstable surface (e.g. soft sand)
psoriatic arthritis	
	Equipment
Collagen disorders	footwear
	rackets in rackets sports
Metabolic disorders	
	Nutrition
	Inadequate carbohydrates/ protein
	Inadequate hydration during exercise
	Medications:
	- flouroquinolones (e.g. ciprofloxacin)
	- anabolic steroids

Table 1. Suggested intrinsic and extrinsic risk factors for Achilles tendinopathy.

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It may be that a combination of intrinsic risk factors such as muscle or joint asymmetry of subtalar hyperpronation, when combined with relative overuse of the tendon through repetitive loading and microtrauma, lead to the development of Achilles tendinopathy. Of course, in general, it is difficult to alter intrinsic risks and the major treatment modalities focus of alteration and correction of extrinsic risk factors. History and examination are critical in elucidating intrinsic and extrinsic risk factors and these factors should be corrected, wherever possible, to assist in alleviating symptoms and preventing injury recurrence.

Differential Diagnosis of Posterior Ankle/ Heel Pain
Bony
Fracture:
os trigonum
calcaneal stress fracture
Severe's disease (adolescent patients)
Joint
Tarsal coalition
Soft-tissue
Impingement syndromes
posterior ankle impingement
tarsal tunnel syndrome
entrapment medial calcaneal nerves
sural nerve entrapment
Musculotendinous injury
peroneal tendon tear/ tenosynovitis / subluxation/ dislocation
tibialis posterior tendon tear/ tenosynovitis
accessory soleus muscle (Figure 2)
Other
Referred pain from the lumbar spine
Rheumatic disease, especially rheumatoid arthritis or psoriatic arthropathy
Metabolic disease (gout, familial hypercholesterolemia)
Table 2. The differential diagnoses of patients presenting with posterior heel pain or ankle
pain.

4. Clinical features of achilles tendinopathy

Thorough history and examination is the cornerstone of diagnosis in medicine, and this must be the first step in assessing the Achilles tendon. This is to ensure that the correct diagnosis is made and that risk factors are addressed in the management program to optimize treatment outcomes.

4.1 History

Achilles tendinopathy is relatively common in weightbearing athletes with rates between 5-20% reported in runners. It is a common injury presentation in runners, track and field

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athletes, and all football players. The classic presentation of Achilles tendinopathy is the insidious onset of posterior heel or calf pain over a few months. There may be pain, swelling, and impairment of function. It may have a relapsing and remitting course with improvements during periods of rest from heavy weightbearing activity.



Fig. 2. MRI sagittal image of an accessory soleus muscle (green arrow) that is easily differentiated from the Achilles tendon (red arrow). The space occupying effect of this accessory soleus muscle can mimic symptoms of Achilles tendinopathy but also the features of posterior ankle impingement.

Referring to the work of Blazina et al, initially there may be pain in the tendon with the onset of weightbearing activity such as running, but the tendon pain resolves with continued running (the tendon "warms"). As the Achilles tendinopathy worsens, there may be aching after activity. With increasing use and tendon degeneration the pain may continue during the course of all weightbearing activity and may eventually preclude heavy weightbearing activity due to the level of pain. Morning pain and stiffness is a common feature of Achilles tendinopathy. Achilles tendon swelling may be noted by the patient, but it is rare to have mechanical symptoms or instability. History should ascertain if there have been any previous lower limb injuries, especially to the ankle, and also if the patient has other medical problems, is taking medication, or if there is a family history of rheumatic disease. Any treatment that the patient has had should be recorded and also if these treatments assisted with the symptoms or not.

4.2 Examination

On examination the examination should generally proceed in a structured manner to assess for the correct diagnosis, appropriate risk factors, and signs of differential diagnoses. Lower limb biomechanics should be assessed with standing and walking. In athletes it may e more

appropriate to assess this with video monitoring of treadmill running or on-field training. With the patient in a standing position the examination should include assessment of foot biomechanics such as pes planus or pes cavus and subtalar hyperpronation, weightbearing ankle range of motion (ROM) including ability to perform single leg heel raise, general lower limb alignment such as genu varum or valgum, dynamic pelvic stability with Trendelenberg testing, and lumbar spine ROM and symptoms referral. Gait examination should assess for antalgic gait, reduced ankle or knee ROM, and Trendelenberg gait. With the patient sitting, perform neural tension tests such as the slump test and look for hand and nail changes of psoriasis. The patient should then be positioned supine and assessed for leg length discrepancy, lower limb neurology, and joint ROM (this must include assessment of ankle joint, subtalar joint, and 1st metatarsophlangeal (MTP) joint, but should also include assessment of knee and hip joint ROM) laxity, especially lateral ankle laxity with anterior drawer test for anterior talofibular ligament (ATFL) laxity and talar tilt test for calcaneofibular (CF) ligament laxity. Ankle plantarflexion and dorsiflexion strength, as well as 1st MTP joint flexion and extension strength, and also foot eversion and inversion strength should be assessed. Tinel's test over the tarsal tunnel and medial calcaneal nerves can be performed in the supine position. The patient may then be positioned prone for inspection of calf muscle bulk and tone, as well as any Achilles or retroAchilles swelling. Swelling and tendon thickening may be obvious either at the Achilles insertion, where bursitis or calcaneal bony protruberences may occur, or 4-6 cms proximal to this at the classic site of non-insertional Achilles tendinopathy (Figure 3).



Fig. 3. The clinical manifestation of non-insertional Achilles tendinopathy is indicated by the arrow. There is obvious tendon thickening in the region 4-6 centimetres from the calcaneal insertion when compared with the other side, and this area will be tender to palpation. The tenderness may be reduced with the tendon placed under stretch by passive ankle dorsiflexion and re-palpation (London test).

Tenderness will generally be well localized to these two sites and tenderness may be reduced by placing the tendon under stretch with passive ankle dorsiflexion (London test). An assessment of tendon gliding function and crepitis may be performed in cases on noninsertional Achilles tendinopathy. Palpation of the retroAchilles space should be performed to assess for tenderness immediately deep to the distal Achilles tendon suggestive of

retrocalcaneal bursitis, fullness or tenderness within the space suggestive of posterior ankle impingement, accessory soleus muscle, or other space occupying lesion. Palpation should also be performed over the tibialis posterior tendons medially and peroneal tendons laterally and strength testing performed particularly with regard to assessing for peroneal tendon subluxation or dislocation. Posterior ankle impingement sign should be checked, and calcaneal squeeze test performed to exclude calcaneal stress fracture.

This examination should allow the examiner to determine with confidence the presence or absence of Achilles tendinopathy, the site of Achilles tendinopathy and any associated features, intrinsic and intrinsic risk factors, and also exclude the common differential diagnoses for posterior heel pain.

5. Investigations

Achilles tendinopathy is essentially a clinical diagnosis and investigations are generally only required to exclude other pathology around the ankle or calf. Xrays may delineate bony or joint problems such as acute or stress fractures, os trigonum, or tarsal coalition, and may show the presence of tendon calcification that should alert the clinician to suspicion or rheumatic or metabolic disease. Lateral ankle Xray may also be used to assess for Haglund's deformity which may be associated with insertional Achilles tendinopathy. Blood tests for rheumatic disease and urate levels may be considered. Magnetic resonance Imaging (MRI) is an excellent investigation for showing the presence or absence of soft-tissue pathology around the ankle and will generally delineate Achilles tendon pathology.

Asymptomatic tendon degeneration is common and should be considered when performing any investigation on the Achilles tendon. With either ultrasound (Figure 4) or MRI the Achilles tendon ultrastructure can be assessed for tendon fusiform thickening, fibre disruption, and fluid within or around the tendon, including bursitis. These investigation findings should be accurately correlated with the site of clinical examination findings before ascribing the diagnosis and classification of Achilles tendinopathy. Doppler ultrasound is an



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Fig. 4. Ultrasound image showing an Achilles tendon partial tear with hypoechoic region (green arrow).

extremely useful adjunct investigation for Achilles tendinopathy and may show neovascularisation, or active bursitis. Note that in the early stages of symptoms there may be no changes noted on investigation.

6. Non-surgical management of achilles tendinopathy

Treatment of all musculoskeletal injuries can involve non-surgical or surgical therapies. Non-surgical management of Achilles tendinopathy, whether insertional or non-insertional, is similar and generally involves symptom control, correction of risk factors, stretching and exercise rehabilitation, and therapeutic injections. Correction of intrinsic and extrinsic risk factors is a critical element of management to assist in decreasing abnormal load on the Achilles tendon, decreasing symptoms, and minimizing the risk of recurrence. Grading of tendinopathies is not generally clinically useful, however partial tears in the Achilles tendon will often take longer for symptoms to resolve. It is commonly stated that insertional Achilles tendinopathy is more recalcitrant to treatment, and this may be due to traction effects and calcification at the insertion (Figure 5).



Fig. 5. Insertional Achilles tendinopathy is often more recalcitrant to treatment than noninsertional Achilles tendinopathy, and this may be due to traction effects and calcification at the insertion (green arrow). Note the bony oedema adjacent to the tendon insertion. Discrete foci of calcification may require debridement surgery if symptoms persist despite nonsurgical therapies.

6.1 Symptom control

Pain is generally the dominant symptom in Achilles tendinopathy and may be managed using regular icepack application 15 minutes per time throughout the day. This may be particularly effective after exercise. Physical therapy treatment with electrotherapeutic modalities such as therapeutic ultrasound or interferential treatment may assist in decreasing pain, as may gentle transverse friction massage. Relative rest is preferred and this involves the avoidance of all aggravating activities, where possible. An aggravating

activity includes any activity that cause tendon pain during the activity, tendon pain after the activity, or increased tendon pain and stiffness the morning after an activity. To avoid aggravating activites it may be necessary to avoid weightbearing exercise and to cross-train with painfree non-weightbearing exercise such as cycling, rowing/ kayaking, or swimming. Analgesics can be used to control strong pain and paracetamol/ acetaminophen is the preferred analgesic. Non-steroidal anti-inflammatory drugs (NSAIDs) should only be used where the condition is inflammatory in nature. The use of NSAIDs should be considered in early symptoms of Achilles tendon pain (the initial 2-3 weeks of symptoms), although it is uncommon for patients to present for medical care at this stage of symptoms. NSAIDs are more commonly used where bursitis, retrocalcaneal or retroAchilles, is associated with insertional Achilles tendinopathy or where paratenonitis is demonstrated to be associated with non-insertional Achilles tendinopathy. Topical agents will be discussed later in the chapter.

6.2 Correction of intrinsic risk factors

6.2.1 Biomechanics of the lower limb

Intrinsic risk factors such as lower limb biomechanical issues can generally be corrected through the use of shoes or orthotics and referral for podiatric assessment may be required. The critical element of correcting biomechanical issues is to achieve sustained control of the hindfoot. Good quality supportive footwear is one aspect of this but orthotic correction is imperative in patients with pes planus and also those with functional hyperpronation (Figure 6).



Fig. 6. Photograph of a female with subtalar hyperpronation in stance phase. Note the prominence of the medial aspect of the arch. Also note the long second toe (Morton's foot type).

Taping the handfoot and assessing the response to a patient's pain is one way to determine if orthotic prescription is likely to be beneficial for the patient. Assessing the shoes for abnormal wear patterns and for friction over the Achilles tendon is also important to determine possible causative factors in the patient's symptoms. Wedged lift heel raise devices are advocated to unload the Achilles tendon through simple biomechanical processes, but these do not control the hindfoot. Heel lift wedges should be worn at all times early in the treatment of Achilles tendinopathy and must be worn in both shoes to avoid asymmetry in gait. Any recognized leg length discrepancy over 1 cm should be corrected through built up orthotics or shoes. Before correcting this it is suggested that a more accurate determination of leg length difference is performed and a single shot scout Computerised Tomography (CT) scan can better determine this using digital calipers.

6.2.2 Joint asymmetry

Restrictions in joint range of motion can also lead to disturbance of the kinetic chain and create force imbalance between sides. The major joints that require assessment are the 1st metatrophalangeal (MTP) joint, midtarsal joints, subtalar joint, ankle joint, knee joint and hip joint. Any restriction in joint range of motion can potentially increase the load on the Achilles tendon and range of motion exercises as well as active and passive joint mobilization is required as therapy. 1st MTP joint stiffness is commonly found in patients with Achilles tendinopathy and, again, may contribute to the onset of the condition or is caused by the condition. Stretching the 1st MTP and massage through the central plantar fascia can assist in normalizing joint range of motion, but toe flexion exercises should also be incorporated into the treatment program to ensure adequate 1st MTP joint strength and endurance as this is a critical aspect of the gait cycle with 1st MTP joint flexion largely contributing to the push-off phase of walking or running.

In athletes, especially those performing unilateral upper body movements, such as tennis players, volleyball players, or throwing athletes, it is important to assess shoulder range of motion, and also trunk and thoracic spine range of motion in rotation.

6.2.3 Joint instability

There are two causes of joint instability, hereditary such as with generalized liagmentous laxity, and traumatic. Generalized liagmentous laxity is a recognized hereditary predisposition to joint laxity and is characterized by knee recurvatum, lumbar spine ligamentous laxity (as can be demonstrated by the patient being able to stand and forward flex the lumbar spine to place their palms onto the floor), laxity in elbow joints with hyperextension, and laxity in finger and thumb metacarpophalangeal (MCP) joints (demonstrated by 5th finger MCP joint extension beyond 90 degrees, and by flexing the wrist and then touching the thumb to the volar aspect of the forearm) as per Beighton's criteria. Traumatic instability to joints distal to the Achilles tendon in the kinetic chain are generally more commonly identified contributing risk factors than joints proximal to the Achilles tendon. These joints and joint injuries include the 1st metatarsophalangeal joint and 1st toe flexor dysfunction, and the ankle joint and previous lateral ligament sprains and peroneal tendon weakness, or peroneal tendon subluxation/ dislocation. Essentially, both joint laxity and joint instability contribute to the causation of Achilles tendinopathy by increasing load

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onto the Achilles as a the primary ankle plantarflexor, or increasing the necessity of the Achilles tendon acting to assist as an ankle joint stabilizer. Often strengthening exercise is effective as therapy for joint instability to control joint motion, but in cases of gross laxity with functional instability, such as with peroneal tendon subluxation/ dislocation, surgery may be necessary to restore joint stability and function.

6.2.4 Rheumatic and metabolic disease

Patients with hereditary rheumatic diseases such as rheumatoid arthritis or psoriatic arthritis may have tendon abnormalities manifesting as part of the systemic disease process. In rheumatoid arthritis these tend to be nodular Achilles tendon abnormalities, whereas in psoriatic arthropathy the Achilles tendon manifestation is more likely to be exuberant tendon calcification, particularly adjacent to the Achilles tendon insertion. Metabolic diseases can also present with tendon manifestations such as the crystal deposition of gout, or the lipid tendon xanthomata associated with familial hypercholesterolaemia. These systemic diseases cannot truly be cured, and the management of the tendon manifestations is through control of the generalized disease process. Large tendon nodules or xanthomata may need surgical excision to restore appropriate gliding function of the Achilles tendon.

6.3 Correction of extrinsic risk factors

6.3.1 Training errors

One of the most common extrinsic risk factors for Achilles tendinopathy in active people is training errors. This often comes do to a lack of knowledge of basic training principles such as adequate warm-up and stretching before exercise, graded increases in training, adequate recovery time through rest days, and appropriate cross-training . A key aspect of preventing injury in sports is to have adequate warm-up and stretch prior to exercise. This ensures that the musculotendinous units are at an appropriate functional length and this should allow for exercise with minimal risk of injury. Specifically for the Achilles tendon it is essential that stretching is performed for both the gastrocnemius and soleus components of the calf musculature and this would normally take the form of a straight knee calf stretch and a bent knee calf stretch.

With any level of exercise or training it is generally rapid alterations to the training program that lead to overload or overuse injuries and this is no different for Achilles tendinopathy. Common changes would include increasing the duration or frequency of training sessions, and thus the total training volume or load, but it may also be that changes occur in the intensity of training. Cross-training with different forms of exercise, including nonweightbearing exercise, can be a useful aspect of training programs but must be performed with caution in athletes not familiar with weightbearing training (i.e. swimmers that beginning running for fitness). Starting training or exercise after being sedentary is a common history in patients with Achilles tendinopathy. For this same reason, resuming training after an absence, including an enforced absence due to injury or as part of the treatment for Achilles tendinopathy, requires careful planning to ensure that the training is not too much too soon. A recurrence Achilles tendinopathy can be avoided with simple advice and planning on resumption of exercise.

6.3.2 Surfaces and equipment

Weightbearing exercise on certain surfaces may contribute to the causation of Achilles tendinopathy. Hard surfaces increase the required shock absorption for the lower limb musculotendinous structures, including the Achilles tendon, whilst soft or unstable surfaces like soft sand require the Achilles and other stabilizing tendons to increase their workload during exercise. Certainly, it is recognized that changes in the surface on which weightbearing exercise is performed can contribute to the aetiology of Achilles tendinopathy. This can be from hard to soft surfaces or vice versa, from sprung flooring to unsprung flooring, or from treadmill running to road running. In the same way, changes to exercise on hills, or on slopes or cambered surfaces can also contribute to the development of Achilles tendinopathy. This may be caused by simple changes in running routes or by beginning to run on cambered roads rather than flat running tracks. Changes in running footwear may contribute to causation in Achilles tendinopathy due to different properties of the shoes such as level of foot control or shock absorption. All of these factors should be investigated in the history and addressed in management of Achilles tendinopathy.

6.3.3 Nutrition

Fatigue is often cited as a potential cause of tendinopathy and may contribute through loss of joint or muscle control leading to increased forces acting on the Achilles tendon. Ensuring adequate hydration and energy availability to working muscle during exercise is critical to minimizing fatigue. Again, simple preparation should ensure that adequate fluid intake and carbohydrate intake is maintained before, during, and after exercise.

6.3.4 Medications

Certain medications may affect tendon metabolism and it is important to address previous and current medications to ensure that these medications are used only if absolutely necessary. Flouroquinolones such as the antibiotic ciprofloxacin have been implicated in Achilles tendon ruptures and appear to have a detrimental affect on tendon metabolism. Anabolic steroids have also been suggested as a cause of tendon rupture, possibly due to the rapid increase in muscle strength before the tendon has completed its strength adaptations. Anabolic steroids are illegal in many countries but their use is widespread nonetheless and this needs to be addressed in taking a history from the patient.

6.4 Exercise rehabilitation for achilles tendinopathy

6.4.1 Stretching

Exercise rehabilitation through muscle stretching and eccentric exercise is accepted as the mainstay of therapy for Achilles tendinopathy. The timeframe of recovery for symptom resolution in Achilles tendinopathy is in the order of 2-3 months at a minimum and both the patient and the medical practitioner need to appreciate this timeframe and commit to treatment over this period. Although stretching is advocated by most medical practitioners as part of the treatment regime, very little evidence is available to support this, or to determine the best form of stretching in treating Achilles tendinopathy. Despite this, logic would suggest that the aims of treatment with stretching are to restore musculotendinous unit length and thus static prolonged stretching should best achieve this aim. Stretching

both the gastrocnemius muscle, through straight knee calf stretching, and also the soleus muscle component, through flexed knee calf stretching, is important to ensure that all components of the Achilles musculotendinous unit are incorporated in the stretching program. Stretching to the point of pain is generally discouraged and, especially in insertional Achilles tendinopathy, may be counterproductive and continue to irritate the symptoms and prolong recovery time. Any recognized muscle asymmetry also requires correction through stretching and a general lower limb stretching program should be considered. The kinetic chain relationship between the 1st metatarsophalangeal joint in toe flexion with toe "push-off" and the Achilles musculotendinous unit as an ankle plantarflexor and stabilizer in this gait phase is appreciated and requires that 1st metatarsophalangeal joint range of motion is optimal to assist in managing load through the Achilles tendon. Thus, stretching the 1st metatarsophalangeal joint is a critical part of management of Achilles tendinopathy.

6.4.2 Exercise rehabilitation

Again it is worth emphasizing that exercise rehabilitation through muscle stretching and eccentric exercise is accepted as the mainstay of therapy for Achilles tendinopathy. Numerous studies have demonstrated the symptom reduction achieved through eccentric exercise protocols in Achilles tendinopathy. Irrespective of other treatments used or considered in the treatment of Achilles tendinopathy, eccentric exercise must form an integral part of the treatment program in this condition.

Tensile loading of musculotendinous units stimulates tendon healing along appropriate lines and directions of force and allows the tendons to achieve the necessary strength and endurance required to accept loading with weightbearing activity. This tensile loading appears to be best achieved through eccentric exercise protocols. The type of eccentric exercise is similar for both insertional and non-insertional Achilles tendinopathy but the range of motion in the eccentric phase differs to achieve the best symptom reduction in each specific type of Achilles tendinopathy. Alfredson et al (Alfredson , 1998) demonstrated that eccentric exercise is effective in reducing pain in Achilles tendinopathy and is as effective as surgical treatment for symptom reduction (Figure 7). This group showed that heavy-load eccentric exercise could reduce pain levels form an average of 7 out of 10 to an average of 3 out of 10. It is important to recognize that few patients became completely asymptomatic with this treatment regime alone. The eccentric exercise involved heel-drop exercises off a step, with the forefoot only on the step and, as would be expected, two forms of eccentric exercise were used, one with the knee flexed and one with the knee straight in order to load both the soleus and gastrocnemius components of the Achilles musculotendinous unit. The protocol of heavy-load eccentric exercise used in this landmark study involved twice daily heel-drop exercises with three sets of 15 repetitions with a flexed knee and three sets of 15 repetitions with a flexed knee. This is a combined total of 180 heel drop exercises. This protocol is time consuming but is recognized as the appropriate level of eccentric exercise required to achieve a symptom reducing effect. It is critical to note that there is no active concentric, or muscle shortening, phase of the exercise and recovery of ankle position needs to be assisted either by weightberaing on the other leg or, preferably, by using the arms to pull up on a stair rail or other fixed structure to recover ankle position. Neisen-Vertommen et al (Neisen-Vertommen, 1992) further demonstrated that eccentric exercise alone was more effective the concentric-eccentric exercise and this is the reason that it is important to minimize, or.



Fig. 7. Eccentric heel drop exercises using a stair or step. The forefoot is on the step with the midfoot and hindfoot unsupported. The heel is then lowered and an eccentric exercises component will generated (tendon lengthening under load). This eccentric exercise is the core component of exercise therapy for Achilles tendinopathy.

abolish, any active concentric exercise component in the heel-drop exercise rehabilitation. Eccentric exercise rehabilitation for non-insertional Achilles tendinopathy should be performed is a slow and controlled manner and the heel should generally reach below the horizontal level of the step With the knee flexed, the heel will not reach far below this horizontal level. There is no benefit from holding the exercise at this end-range as the presumed benefit of eccentric exercise is the tensile lengthening of the musculotendinous unit under weightbearing load. Recent research suggests that in the treatment of insertional tendinopathy the heel drop exercises should be performed is a slow and controlled manner and the heel should not reach below the horizontal level of the step. This may allow for the tensile forces to act on the musculotendinous unit whilst also minimizing the traction effect on the Achilles tendon insertion.

6.5 Topical medications

6.5.1 Topical glyceryl trinitrate

Glyceryl trinitrate has a long history of therapeutic use, being discovered as an effective treatment of angina pectoris in miners handling the explosive nitroglycerin in the 1870's. The mechanism of action of topical glyceryl trinitrate is suggested to be through the biologically active metabolite nitric oxide, also termed endothelial derived relaxing factor, due to the vasodilatory action of this metabolite. In animal studies, the addition of nitric oxide to injured tendon was demonstrated to improve tendon healing, although the exact mechanism of this action is not clear. It may be an analgesic effect, an effect on global tendon bloodflow, or an effect on the tendon neovessels seen as a histopathologic response in degenerative tendinopathy. As a result of the basic science research into topical glyeryl trinitrate, studies were performed to assess the effect of topical glyeryl trinitrate in symptom reduction in non-insertional Achilles tendinopathy [Paoloni, 2004]. This randomized controlled clinical trial demonstrated significant pain reduction, increase in object tendon

strength measures, and an increased rate of patients being completely asymptomatic after six months of treatment. The topical glyeryl trinitrate was applied as a patch (Figure 8) over the symptomatic tender area of the Achilles tendon and was used in continuous dosage.



Fig. 8. The topical glyceryl trinitrate patch used in clinical trials to effectively treat the symptoms of non-insertional Achilles tendinopathy. This patch is one quarter of a 5mg/ 24 hour (0.2 mg/ hour) NitroDur® patch (manufactured by Schering-Plough Australia), howver similar drug dosages can be achieved with other glyceryl trinitrate patches or ointments.

The continuous dosage regime was designed to minimize the frequency and severity of side-effects through tolerance to the vascular side-effects of the patch, as opposed to the "drug holiday" regime used in cardiovascular medicine where there is a need to maintain vascular sensitivity to nitric oxide to ensure adequate vasodilatation. The side-effects of topical glyeryl trinitrate include headache and rash and approximately 5-10% of patients will not tolerate the patch usage to treat Achilles tendinopathy due to these problematic, but reversible, side-effects. It must be emphasized that the topical glyeryl trinitrate treatment is used in addition to exercise rehabilitation. Topical glyceryl trinitrate treatment, especially continuously, is contraindicated in patients with known cardiovascular disease, especially angina pectoris, due to the potential development of tolerance to topical glyceryl trinitrate vasodilatation effects.

6.5.2 Topical anti-inflammatory medications

As symptom relief is an important aspect of the treatment of Achilles tendinopathy, there may be a role for topical anti-inflammatory medications to treat Achilles tendinopathy, especially insertional Achilles tendinopathy associated with retrocalcaneal or retroAchilles bursitis. These topical anti-inflammatory agents may take the form of non-steroidal anti-inflammatory drugs (NSAIDs) such as diclofenac or piroxicam creams, or may be other medications such as traumeel®, an interesting semi-homeopathic preparation containing measurable amounts of multiple natural substances such as arnica and Echinacea, and having shown evidence of anti-inflammatory properties mainly in treating gastrointestinal disorders.

6.6 Injection therapies in achilles tendinopathy

6.6.1 Corticosteroid and anti-inflammatory injections

Corticosteroids are catabolic steroids that have anti-inflammatory properties and are widely used in musculoskeletal medicine. In weightbearing tendons, such as the Achilles or patellar tendon, there is a theoretical risk of tendon rupture after corticosteroid injections. This may be due to the catabolic effect of corticosteroids, with a transient weakening of tendon substance, it may be due to the incorrect placement of the injection into the tendon substance creating pressure necrosis and catabolic effects, or it may be due to the pre-existing degenerative process in the tendon that has already weakened the tendon substance. Few studies have assessed the effect of corticosteroids in the treatment of Achilles tendinopathy, however one study [Da Cruz, 1988] using ultrasound guided corticosteroid injection into the Achilles paratenon in non-insertional Achilles tendinopathy did not show any statistically significant improvement. This may be due to the degenerative nature of the pathologic process in chronic Achilles tendinopathy, although it is conceivable that this treatment may be effective in the first few weeks of symptoms of Achilles tendon pain, when an acute inflammatory response may be present. Corticosteroid injections may be more useful as therapeutic agents where retrocalcaneal bursitis (Figure 9) or retroAchilles bursitis is present with insertional Achilles tendinopathy. The indications for using corticosteroid injections are generally for decreasing severe pain to allow the patient to perform exercise rehabilitation relatively pain free, or when there is a plateau in any improvement with exercise rehabilitation. These injections are best performed guided to minimize the risk of intratendinous injection and thus potential tendon rupture. With ultrasound visualisation the fluid in the distended bursa may be seen, under Doppler ultrasound increased vascularity in the bursa may be appreciated, and the injection can also be seen to be in the bursa with bursal filling demonstrated.



Fig. 9. MRI sagittal image showing retrocalcaneal bursitis (arrow). This may be effectively treated using injected corticosteroid agents.

Where there is a suggestion that pain is potentially caused by inflammation, such as in noninsertional Achilles peritenonitis or where the pain is at the level 4-6 cms above the tendon insertion but there are nil ultrastructural changes or abnormal vascularity in this region, or at the Achilles insertion with associated retrocalcaneal or retroAchilles bursitis, and there is a concern about possible tendon rupture with the catabolic effect of corticosteroid injection, then other anti-inflammatory agents such as traumeel® injection may be considered. This may be the case where active patients or athletes will not be able to unload the tendon after injection. Any symptom relieving effect of traumeel® injection in this circumstance may be due to a dilution effect of any chemical irritation at the site of pain.

6.6.2 Polidocanol sclerotherapy injections

Polidocanol is a sclerosant medication that has predominantly been used to treat venous varicosities. However, studies have demonstrated a positive therapeutic effect in treating Achilles tendinopathy through the use of Doppler ultrasound guided polidocanol injections [Ohberg, 2002], and this effect appears to persist at 2 year follow-up[Lind, 2006]. Ohberg et al reported that the pain reducing effect was in the order of 80% pain reduction in approximately 80% of patients when using between one and five guided polidocanol injection sessions. The procedure involves assessing the Achilles tendon for increased vascularity under Doppler ultrasound, compared to normal tendon substance that does not show any vasuclarity under Doppler ultrasound (Figure 10). The vascularity is believed to be due to neovascularisation, part of the pathologic process of degenerative tendinopathy. It is relatively common to view a single vessel, or a few vessels, entering the tendon from the deep aspect and then branching more widely throughout the tendon substance. To avoid pressure necrosis it is best to attempt to sclerose the vessels at the tendon undersurface and use as small an amount of polidocanol sclerosant as is possible. The more accurately the needle tip can be visualized the better the sclerosant effect and the less infiltrate that will be



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Fig. 10. Colour Doppler ultrasound image showing the Achilles tendon (green arrow marks the Achilles tendon) with thickening of the tendon, a hypoechoic region suggestive of small partial tendon tear, and prominent vascularity. The vascularity often appears to enter from the undersurface of the tendon (pink arrow) and then branch through the tendon. Sclerosing the vascularity at the entry point to the tendon can be an effective treatment for symptom reduction.

required during the procedure. This takes experience but should allow immediate abolition of the vascularity in the tendon as visualized in real-time under Doppler ultrasound. The effect can be quite impressive, but is not always complete. Repeating the procedure may be required if pain is not decreased or abolished. As reported in the initial study on polidocanol sclerotherapy [Ohberg, 2002], it may take several injections to achieve adequate symptom resolution. There does not appear to be any negative effect on tendon structure with polidocanol injections and the tendon may be loaded normally within a few days after the injection without any apparent risk. If bursitis is found to be associated with increased tendon vascularity, then it is recommended that polidocanol injections be used to sclerose the neovessels and corticosteroid is infiltrated into the inflamed bursa during the same ultrasound procedure. This may then require 5-7 days of rest before returning to normal tendon loading, in order to allow the corticosteroid to have an effect on the bursal inflammation.

6.6.3 Prolotherapy injections

Prolotherapy, or "proliferation therapy", is a term that is generally applied to various nonpharmacologic injection types all of which are proposed to act to strengthen weakened degenerative connective tissue in tendon presumably by an irritant effect. There are mostly used at insertions of tendons or ligaments. The types of solutions vary but commonly used injection types include glucose, local anesthetics such as lidocaine, phenol, or glycerine. In most medical practices it is the relatively innocuous injections such as glucose and lidocaine that are used due to the low side-effect profile. Prolotherapy injections may be considered, after weighing the risks and benefits of these, for recalcitrant insertional Achilles tendinopathy. Again, a period of rest is probably required after the injection and then continuance of exercise rehabilitation program.

6.6.4 Autologous blood product injections

Autologous blood products have been used as injection therapy in musculoskeletal medicine for many decades. This may be in the form of autologous blood or as blood extracts such as platelet rich plasma (PRP) or isolated specific growth factors such as fibroblast growth factor or platelet derived growth factor. The therapeutic use of isolated growth factors for specific musculoskeletal injury is still in its infancy, and is currently prohibited for competitive athletes under the World Anti-Doping Authority (WADA) code January, 2011. The evidence for the use of autologous blood products, including platelet rich plasma, to treat Achilles tendinopathy or other tendinopathies is scant and most well conducted clinical trials do not show evidence of effect [de Vos, 2010 and Paoloni, 2010]. However many facets of the use of these injections have not been established, including; what is the optimal blood product injection type, what is the optimal volume of injection, what is the optimal timing of injection after injury, what is the optimal number and spacing of injections (single injection versus injection series, and what the optimal rehabilitation is after injection. These autologous blood product injections are probably best used for cases of recalcitrant insertional Achilles tendinopathy where they may have an irritant or proliferative effect similar to that of prolotherapy injections. The risks and benefits need to be weighed before deciding on these injections, but the side-effect profile is low, with postinjection pain and irritation for up to 1-2 weeks the most likely side-effect. The irritant side-

effects may be greater if these products are injected into the tendon substance rather than around the tendon.

6.6.5 Mesotherapy injections

Mesotherapy is widely practiced in sports medicine and refers to the use of multiple injections, often of anti-inflammatory medications such as piroxicam, into the subcutaneous fat overlying a region of musculoskeletal pain. The evidence for this treatment is also scant and the mechanism of effect appears unclear but may be due to localised tissue uptake of anti-inflammatory medication. This treatment may provide symptomatic relief especially in insertional Achilles tendinopathy associated with retrocalcaneal or retoAchilles bursitis. Mesotherapy is generally performed as a series of three sessions of multiple subcutaneous injections over a three week period.

7. Surgical treatment for achilles tendinopathy

Surgical treatment for Achilles tendinopathy is generally reserved for recalcitrant cases where non-surgical management has failed to provide effective symptom relief or ability to function in daily life or in sporting activities. It is often dictated by the patient who may become frustrated by continued symptoms despite long periods of exercise rehabilitation and also trials of injections and other therapies. It is reported that between 24% and 46% of patients with Achilles tendinopathy eventually require some form of surgical treatment, however this rates appears quite high and may represent the incidence of surgery in patients presenting to orthopaedic surgeons [Maffulli, 2002]. Many patients with Achilles tendinopathy may never consult a surgical specialist for their condition. It must be recognised that probably 10-20% of patients with Achilles tendinopathy continue to have symptoms in the longer term and it is this population that would be considered for surgical treatment. Conditions such as chronic bursitis, especially associated with Haglund's deformity, may require surgery (Figure 11).

7.1 Tendon surgery

The exact surgical procedure used to treat Achilles tendinopathy varies amongst surgeons and geographical regions but will generally involve; tendon debridement and bursal resection, if required. The tendon debridement may take the form of multiple longitudinal incisions into the Achilles tendon substance with or without curettage of any mucoid degenerative areas, or may be less invasive and involve debridement of the tendon surface and paratenon only. Any bursal involvement at the Achilles insertion should warrant bursal resection. The majority of patients will experience symptom reduction after surgery, 85% of patients report good results after surgery, and many patients will be able to return to previous level of activities or sport within six to twelve months post-surgery whether surgery is performed with open procedures [Saxena, 2003] or less invasive methods [Maffull, 1997].

7.2 Surgery procedures for bony abnormalities

There may be bony abnormalities that contribute to the development of Achilles tendinopathy and these may nee to be addressed surgically. Surgery may be only to rectify these bony abnormalities or it may be in combination with tendon debridement surgery.

Achilles Tendon



Fig. 11. Chronic fat pad inflammation, Haglund's deformity (arrow) and insertional Achilles tendinopathy. This case may require surgery for ongoing symptoms. Also note the osteopoikilosis, a benign condition manifesting as dark bone islands on MRI.

Haglund's deformity is a posterosuperior bony calcaneal protruberance that may predispose to both retrocalcaneal bursitis and to insertional Achilles tendinopathy due to a mechanical friction effect on the deep surface of the Achilles tendon (Figure 8). If Haglund's deformity is present and there is concomitant recalcitrant Achilles tendinopathy then it may be necessary to perform surgical excision of the Haglund's deformity. Sever hindfoot abnormalities with hindfoot varus may also predispose to Achilles tendinopathy through a twisting or "wringing out" mechanism of the collagen fibres and microvasculature. Correction of moderate flexible hindfoot varus caused by subtalar hyperpronation may be achieved through footwear and orthotics as discussed earlier, however severe hindfoot varus or fixed hindfoot varus may require surgical correction through calcaneal osteotomy or similar biomechanic realignment procedures.

8. Conclusion

The aetiology of Achilles tendinopathy is complex and multifactorial and the management of this condition requires a thorough understanding of the extrinsic and extrinsic risks factors, biomechanics or the lower limb and the concept of the kinetic chain. Management of Achilles tendinopathy, whether insertional or non-insertional, should always be nonsurgical initially, and must focus on elements of pain control, correction of intrinsic and extrinsic risk factors, and exercise rehabilitation through appropriate stretching and eccentric exercise protocols. Other modalities such as topical or injected medications may be required if pain is not controlled or if symptomatic improvement with exercise rehabilitation plateaus. Based on stratification of risks and benefits in patients with recalcitrant Achilles tendinopathy, and no cardiovascular disease, a sensible approach to

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adjunctive modalities would start with topical agents such as continuous topical glyceryl trinitrate treatment in addition to exercise rehabilitation. Further adjunctive treatment with injections differs depending on the site of Achilles tendinopathy. In both insertional and non-insertional Achilles tendinopathy, an assessment of the tendon is made using ultrasound and Doppler mode. For patients with either insertional or non-insertional Achilles tendinopathy who demonstrate increases in vascularity in the tendon substance at the site that correlates with symptoms, Doppler ultrasound guided polidocanol injections should be the first-line treatment to assist in alleviating symptoms. Should there be ultrastructural tendon abnormalities without increased tendon vascularity at the symptomatic tendon site in non-insertional Achilles tendinopathy, then the preferred injection treatment becomes less clear and options would include; autologous blood or platelet rich plasma (PRP) into the degenerative area of the tendon or around the tendon, or other injections around the tendon which would include traumeel®, prolotherapy type agents such as glucose or lidocaine, or indeed innocuous injection agents such as normal saline.

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Achilles tendon has always attracted a great attention. Its disorders include various problems from pain and swelling with bumps to functional impairment or even ruptures. Debates concerning aetiology and optimal treatment are still going on. A lot of efforts and research have already been put on to find the answers to unsolved problems and this book is an attempt to share (some of) these findings to the readers. If only one of the papers helps the therapists or patients in understanding and solving their problems, we will consider that the mission of the book was accomplished.

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