Achilles tendinopathy: new insights in cause of pain, diagnosis and management

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General introduction

BACKGROUND

The Achilles tendon was weak, small or absent in the great ape, but is one of the hallmarks of bipedal man. It possibly originated soon after the divergence of chimpanzee and human lineages[4]. Its origination may be related to the greater relative length of the tarsal bones in man. The name 'Achilles tendon' is derived from the Iliad by Homer. Achilles was made invulnerable in infancy by his mother the goddess Thetis, who plunged him into the river Styx. As he was held by one heel, this part was not bathed and so remained unprotected. This heel was the site where Achilles was mortally wounded by a poisoned arrow launched from the bow of Paris during the Trojan War. The 'Achilles heel' is currently used as an idiom to express one's principal weakness. In 1693 the Dutch surgeon Philip Verheyen changed the concept 'tendo magnus of Hippocrates', the anatomical connection between the calcaneus and triceps surae muscle, to 'Achilles tendon'[23].

The human body is built for movement. However, over the years we have become progressively less active in daily life. The prevalence of obesity in the Western World has been rising, and as a reaction keeping fit is increasingly advocated in recent years. Healthy nourishment, physical discipline, and fitness occupy a considerable part of the cultural space. Endurance sports like triathlons and long distance running have gained popularity. Although scientific evidence supports the notion that humans evolved to be runners, with the ability to outrun almost any animal when it comes to long distances[28], overuse injuries have become prominent in daily practice.

General inactivity during the week in an office-setting for many years, followed by extreme and extensive exercise in the weekend without decent build-up in training program allow for these types of injury to occur. Thirty to fifty percent of all sports injuries are due to overuse, and overuse tendon injury has become an important issue in both recreational and competitive athletes. Chronic Achilles tendon pathology is one of the most frequently occurring problems in sports involving running and jumping. In elite long-distance runners there even is a lifetime risk of 52% of sustaining an Achilles tendon injury[24]. However, the injury is not *always* related to excessive physical activity. Thirty percent of patients have a sedentary lifestyle, which may suggest that physical activity might exacerbate symptoms rather than cause them[3].

Recalcitrant Achilles tendons may cause pain for years and are often resistant to any form of treatment. In the end, chronic Achilles tendon complaints may be self-limiting, since they have been described to quench after an average of 8 years[43]. Doctors and athletes will not consent with this prospect. Unfortunately, the cause of pain has not yet been clarified and therefore its treatment is challenging and often unsatisfactory.

Terminology

The terminology of chronic Achilles tendon disorders has been confusing. Maffulli and co-workers proposed 'Achilles tendinopathy', the combination of Achilles tendon pain, swelling, and impaired performance as standard terminology for chronic midportion problems, including the histopathological entities tendinosis and peritendinitis[33]. Terminology is still confusing for problems around the insertion: retrocalcaneal bursitis, insertional spurs, Haglund's syndrome, - disease, - exostosis, pumpbump, cucumber heel and many more are used interchangeably.

Anatomy

The Achilles tendon consists of the fibers of two muscle units in the superficial compartment of the posterior leg: the gastrocnemius muscle (medial and lateral head) and the soleus muscle. The gastrocnemius muscle crosses the knee and ankle joints (ankle and subtalar); originating from the posterior surface of condylus medialis and -lateralis femoris and inserting onto the calcaneus. The soleus muscle lies anterior to the gastrocnemius muscle and originates from each side of the anterior aponeurosis attached to the tibia and fibula, and from the posterior surfaces of the head of the fibula and its proximal quarter, as well as the middle third of the medial border of the tibia. The soleus muscle only crosses the 2 ankle joints. Distally, both the gastrocnemius and soleus muscles form an aponeurosis, from each of which a tendon originates. At about the level where the soleus contributes fibers to the Achilles tendon, rotation of the tendon begins and becomes more marked in the distal 5-6 cm. The gastrocnemius fibers rotate to lateral and the soleus fibers are positioned medial to the insertion[41]. The Achilles tendon inserts crescent-shaped halfway the posterior tuberosity of the calcaneus[29]. Just anterior to the distal portion of the Achilles tendon and posterior to the posterosuperior calcaneal prominence the retrocalcaneal bursa is situated.

Unlike other tendons in the leg, the Achilles tendon lacks a synovial sheath. Instead, it has a paratenon, which is an array of thin, fibrous tissue containing blood vessels[49]. Together with the bone- tendon- and the muscle-tendon junction the paratenon forms the sole vascular supply of the Achilles tendon. Within the paratenon, the plantaris tendon runs with the Achilles tendon. The plantaris tendon is believed to be rudimentary. Absence in 7-20% human lower limbs has been reported[6, 7, 12, 39, 51].

The muscle of the plantaris muscle-tendon complex is triangularly shaped and lies posterior to the knee joint, originating from the inferior part of the lateral supracondylar line of the femur. The tendon travels inferomedially, posterior to the soleus muscle and anterior to the medial gastrocnemius muscle. The tendon crosses the calf relatively proximal, running medial from, and parallel with the Achilles tendon from the midportion of the calf; in the majority of cases inserting medially onto the calcaneus[7, 16]. The plantaris tendon is considered to function as a weak ankle- and knee flexor and ankle invertor.

Clinical assessment of chronic Achilles tendon pathology

The most common clinical diagnosis of chronic Achilles tendon problems is paratendinopathy with or without tendinopathy (55-65%) (midportion tendinopathy), followed by insertional problems such as retrocalcaneal bursitis and insertional spurs or calcifications (20-25%)[17-19, 27]. This thesis focuses on midportion Achilles tendinopathy and retrocalcaneal bursitis.

Midportion Achilles tendinopathy

General symptoms of midportion Achilles tendinopathy include painful swelling typically 2-7 centimeter (cm) proximal to the insertion, and stiffness especially when getting up after a period of rest. Pain is often most prominent on the medial side of the tendon[52].

Tendinopathy and paratendinopathy often co-exist. In isolated paratendinopathy, there is local thickening of the paratenon. Paratendinopathy can be acute or chronic. *Acute* isolated paratendinopathy manifests itself as peritendinous crepitation as the tendon tries to glide within the inflamed covering. Areas of increased erythema, local heat, and palpable tendon nodules or defects may also be present at clinical examination. In *chronic* Achilles paratendinopathy, exercise-induced pain is still the cardinal symptom while crepitation and swelling diminish. The area of swelling does not move with dorsiflexion and plantarflexion of the ankle, where it does in tendinopathy[35,55,60]. In addition, ankle instability and malalignment of the lower extremity, especially in the foot, should be looked for in patients with Achilles tendon complaints.

Retrocalcaneal bursitis

Retrocalcaneal bursitis is caused by repetitive impingement of the bursa between Achilles tendon and posterosuperior calcaneus. Differentiation between midportion and insertional tendinopathy can be made on thorough history taking and physical examination. In case of a retrocalcaneal bursitis, patients complain of pain after a day of strenuous activity or when starting to walk after a period of rest. Wearing shoes with rigid heel counters is often avoided. Physical examination reveals swelling on both sides of the Achilles tendon at the level of the posterosuperior calcaneal prominence. Pain is aggravated by palpating this area just medial and lateral to the Achilles tendon. Retrocalcaneal bursitis can be accompanied by insertional tendinopathy. In case of insertional tendinopathy, there is pain at the bone-tendon junction, which gets worse after exercise. The area of maximum tenderness is often located in the central part of the insertion.

In clinical practice overuse injuries often do have features of more than one pathophysiological entity, however in most cases thorough history taking and physical examination should provide with the correct diagnosis.

Imaging

Standard lateral and anteroposterior (AP) weight-bearing radiographs of the foot should routinely be made even when soft tissue pathology is suspected, to exclude bony pathology and assess foot deformities. Calcifications and bone spurs can be detected in case of insertional tendinopathy. A posterosuperior calcaneal prominence can be seen and may cause retrocalcaneal bursitis, but is not conclusive of the diagnosis. In case of a clinical diagnosis of midportion tendinopathy or retrocalcaneal bursitis, additional investigations such as ultrasonography (US) or magnetic resonance imaging (MRI) can be helpful.

In midportion Achilles tendinopathy, US may reveal discontinuity of tendon fibers, focal hypoechoic intratendinous areas, fusiform tendon swelling, and neovascularisation. In the acute phase of paratendinopathy, there is fluid surrounding the tendon. Peritendinous adhesions can be seen as thickening of the hypo-echoic paratenon with poorly defined borders. In case of a retrocalcaneal bursitis fluid can be detected in the retrocalcaneal space, and an insertional problem provides a hyperechoic signal.

US is a cost-effective and accurate measure to evaluate disorders of the Achilles tendon and has the advantage of dynamic assessment, but is operator-dependent and often not accessible by the orthopedic surgeon. Moreover, it is nowadays often superseded by contrast enhanced MRI. Although MRI is expensive and time-consuming, its ability to acquire images from multiple planes is an advantage, and is especially important for pre-operative planning. The tendon proper may show expansion on T1- weighted images and central enhancement of the signal within the tendon. In the acute phase of Achilles paratendinopathy, MRI is the most useful imaging modality and shows high signal around the Achilles tendon on the STIR- and T2 images. In the chronic phase the paratenon is thickened. In case of a retrocalcaneal bursitis a high signal is seen on T2 and STIR in the retrocalcaneal space.

Outcome measures

Many studies have been published on different treatment modalities for Achilles tendinopathy. Important in the evaluation of treatment outcome are standardized outcome measures[46]. A patient's subjective assessment of treatment outcome is increasingly appreciated. Criteria as pain, functional ability and satisfaction fulfill the criteria of being valid, reliable and sensitive to change if gathered by a correctly designed and tested patient-centered questionnaire[8]. Response bias is minimized since the investigator has minimal influence on the scoring[5, 56]. The Victorian Institute of Sports Assessment- Achilles (VISA-A) questionnaire was created in 2001. It is a self- administered valid and reliable questionnaire evaluating symptoms and their effect on physical activity[46]. Subjective scoring systems can be used in all countries after translation and validation for specific language and population[13, 14, 38].

Management

Midportion tendinopathy

A wide range of conservative treatment measures is available for Achilles tendinopathy. An eccentric training schedule is currently the treatment of first choice, preferably combined with avoidance of painful activities, correction of malalignments of the foot, stretching and use of NSAIDs when an acute element is present or just for pain relief. With compliance this program is described to render good results in up to 90% of patients[1,11,36,42,48,53]. If it fails, many other conservative measures can be applied, with varying results. An effective treatment algorithm has not been developed yet. Independent of the levels of evidence, at 6 months after the start of treatment, 75-80% of patients report to be able to return to their previous sporting activities. For the remaining patients surgical treatment can be considered. Surgical measures can be divided into minimally invasive and open procedures, but also into treatment for paratendinopathy, tendinopathy of the main body, and a combination of both.

Open debridement of the Achilles tendon is the most commonly performed procedure. After removal of pathological tissue often enough tendon remains to achieve side-to-side closure. When more than 50% of the tendon is degenerative, augmentation with a turn-down flap and/or plantaris tendon can be performed, but larger defects require tendon transfer. When the paratenon is involved, open adhesiolysis with or without resection of the paratenon can be performed[20,25,32,40,47]. Aftertreatment for open debridement often consists of immobilization in cast; modern postoperative strategies consist of bracing in walkers or the use of heel raises and functional treatment. Return to sport tends to be at around the 6- month mark but should be individualized. Because of the long time to recovery, minimally invasive procedures have been developed to reduce per- and postoperative morbidity, rehabilitation and time to normal activity. For isolated tendinopathy of the main body, percutaneous longitudinal tenotomies can be performed[34, 57]; for paratendinopathy percutaneous stripping of the paratenon and Achilles tendoscopy have been invented[31, 55].

Success rates for the surgical management of non-insertional Achilles tendinopathy are reported to be in the order of 75-100%[26, 40, 44, 50]. However, these results cannot be matched with day-to-day clinical practice. These long-term difficulties in assessing the methods and outcome of surgery are probably caused by a lack of understanding the pathology[37].

Retrocalcaneal bursitis

Multiple conservative treatment options have been described to manage retrocalcaneal bursitis, including avoidance of tight shoe heel counters, cast immobilization, NSAIDs, activity modification, padding, shock wave treatment, physical therapy and injection of corticosteroids into the retrocalcaneal space. When these measures fail, surgical treatment can be

considered. Mostly open procedures performed through a lateral, medial or transverse Achilles tendon incision. The retrocalcaneal area is debrided and an osteotomy of the posterosuperior calcaneus is performed. These open procedures are invasive, have high complication rates and rehabilitation is extensive[7,20,31,32,42,47]. Therefore, an endoscopic approach was developed by Van Dijk et al.[59]. In this 'endoscopic calcaneoplasty' two portals are created, just lateral and medial to the Achilles tendon, directly proximal to the superior part of the calcaneus. During the procedure, the retrocalcaneal bursa, posterosuperior calcaneus tuberosity and local synovitis are addressed.

Current concepts on the cause of pain in midportion tendinopathy

The cause of complaints in midportion Achilles tendinopathy has not yet been unraveled; however extensive research over the past three decades has led to the formulation of generally two different theories; (1) mechanical overuse and (2) neovascularisation. The first theory hypothesizes that repetitive tensional loading with repeated strains below acute tendon injury threshold induces microdamage. When matrix composition and organization is repaired and mechanical strain continues on transiently weak tissue, tendon damage accumulates. Since there is no time for proper repair due to overuse, degeneration develops. The poor vascularisation and low metabolism contribute to the pathology. However, this theory does not explain *pain* associated with tendinopathy, nor the cause of complaints in 30% of sedentary patients[45,54]. Degeneration is believed to be associated with aging. Tendons become stiffer, blood flow declines and responsiveness of cells to loading decreases[45,54]. Disuse is closely connected to ageing; a disused tendon has similar changes as an aged tendon[43]. Nevertheless, degeneration does not seem to be an inevitable consequence of aging. Synergism of overuse and ageing in the development of Achilles tendinopathy has been proposed[54,58].

Several studies have reported intratendinous changes in up to 34% of cadaver specimens and MRI images of patients *without* complaints[10,15,21,22]. A long-term follow-up study was published by Alfredson et al. revealing persistent structural abnormalities and thickening of the tendon 13 years after intratendinous surgery for Achilles tendinopathy, whereas all patients were satisfied with the results and went back to Achilles tendon loading activities without restrictions[2]. These studies imply that an area of intratendon abnormality of the Achilles tendon proper is not necessarily the cause of pain.

The second, more recent theory on the cause of tendinopathy is the increased irregular vascular and neurogenic ingrowth into the tendon proper as a contributory factor to the pain in patients with symptomatic midportion tendinopathy. Pain-free normal tendons are sparsely vascularised, which is even less at the two hypovascular regions at the insertion and midportion area, commonly associated with pathology. Blood flow declines with ageing and mechanical loading. Although the role of neovascularisation is poorly understood, and may even be part of a healing response, treatment with Doppler ultrasound guided injection of

Polidocanol (Ethoxysclerol) into these neovessels is described as a successful invasive conservative treatment modality in different publications[1-4,19,60].

AIMS AND OUTLINE OF THIS THESIS

This thesis aims at elucidating the cause of pain, and optimizing current diagnostic and management methods in midportion Achilles tendinopathy and retrocalcaneal bursitis. A better understanding will have positive consequences for the patients.

The terminology for chronic Achilles tendon pathology has become inconsistent and confusing. Eponyms have been introduced but their definitions vary. Some terms represent a disease, some an anatomic location, pathology, or a generic name. For proper research and assessment of diagnosis and management, a uniform and clear terminology is necessary. The objective of **Chapter 1** is to provide a clear and uniform terminology for Achilles tendinopathy. It comprises anatomic location, symptoms, clinical findings, and histopathology. **Chapter 2** reviews the currently available outcome measures and assessment tools for the evaluation of complaints and outcome of interventions.

Validated outcome measures are essential in the evaluation of complaints and effectiveness of treatment in both the clinical setting and research. The VISA-A has successfully been translated into Swedish, German and Italian[30, 38, 53]. The aim of **chapter 3** is to translate and validate the Dutch version of the VISA-A subjective outcome measure for Achilles tendinopathy (VISA-A-NL). It was hypothesized that the translated version would be a reliable and valid tool in the subjective evaluation of complaints in patients with Achilles tendinopathy. Another hypothesis was that the questions on activity could be deleted when applied to non-athletes.

In **Chapter 4** the aim was to evaluate the short- and midterm outcome of Ethoxysclerol injections as a conservative measure for Achilles tendinopathy. This treatment was introduced in 2003 by Alfredson et al. and was based on the findings of enhanced vascularisation around degenerative tendons. Ethoxysclerol obliterates neovessels by destroying the intima as is done in the treatment of varicose veins. We hypothesized that sclerosing Etoxysclerol injections would not yield good results in the majority of patients.

When conservative measures fail, surgery can be considered. Also for operative interventions many options are available. Minimally invasive treatment is preferable, and has proved to cause less postoperative morbidity, to facilitate shorter rehabilitation and quicker sport resumption when compared to open surgery. Amongst Achilles tendon endoscopy, 'Achilles tendoscopy', **chapter 5** describes the endoscopic approach and outcome of tendon problems around the ankle.

The exact pathophysiological mechanism causing midportion Achilles tendinopathy has not yet been clarified. Obviously this is the main reason that the optimal treatment modality has not yet been found. Pain is often located on the medial side of the Achilles tendon, where the plantaris tendon accompanies the Achilles tendon. Also, most ultrasonographically detected midportion disorders are found in the medial segment of the tendon[9]. In a healthy situation the plantaris tendon is attached to the calcaneus or Achilles tendon at its insertion, but is separate from the Achilles tendon at its midportion in 95%[7]. During Achilles tendoscopy it is found that the plantaris tendon is fixed to the Achilles tendon at the level of complaints[55]. The aim of **chapter 6** is to evaluate the anatomic relations of the plantaris muscle and distal tendon origins. We hypothesized that in chronic Achilles tendinopathy adhesions between Achilles- and plantaris tendon are formed and so could seriously contribute to patient's symptoms. In **chapter 7** the results of stripping the plantaris tendon in 3 patients with midportion Achilles tendinopathy are analyzed. It was hypothesized that stripping the plantaris tendon would relate to a good outcome. In **chapter 8**, the cause of pain in midportion Achilles tendinopathy is re-evaluated. We hypothesized that the tendon proper is not the cause of complaints, but the process of scarring and adhesion of the paratenon onto the Achilles and plantaris tendon that comes with the inflammatory response. This theory is elucidated using the available evidence on the cause and treatment of midportion Achilles tendinopathy.

As described earlier, chronic Achilles tendon problems also occur around the tendon's insertion onto the calcaneus. Chronic retrocalcaneal bursitis is diagnosed at physical examination as a prominent palpable swelling is palpable lateral and medial from the Achilles tendon just proximal to the calcaneus. Standard lateral and anteroposterior radiographs of the ankle are made to assess foot deformities or any bony abnormalities. In order to confirm the diagnosis chronic retrocalcaneal bursitis, ultrasound, MRI or even bone scans are performed. These are expensive and time consuming. **Chapter 9** evaluates the usefulness of a visible soft tissue swelling in the retrocalcaneal recess on standard lateral weight-bearing radiographs on the ankle in diagnosing a retrocalcaneal bursitis. It was hypothesized that the retrocalcaneal recess on radiography would be a useful and reliable tool in the diagnosis of a chronic retrocalcaneal bursitis.

Primary treatment of choice consists of a number of conservative treatment options including avoidance of tight shoe heel counters, cast immobilization, NSAIDs, activity modification, padding, shock wave treatment and physical therapy. When conservative treatment fails, endoscopic calcaneoplasty can be performed. In **chapter 10** this surgical procedure is described. Adequately determining the location of landmarks can be difficult due to the anatomy and swelling. Portals are positioned too far proximal and as a consequence the posterosuperior part of the calcaneus cannot be reached properly resulting in a suboptimal procedure or, when the problem is recognized, a need to re-incise or lengthen the incisions.

For this reason, we aimed to standardize portal placement in **chapter 11**. It was hypothesized that the landmark of the fibula tip in standard 2-portal hindfoot endoscopy was usable, and that in patients with flat feet portals should be placed more proximal to this landmark.

REFERENCE LIST

1. Alfredson H, Pietila T, Jonsson P, Lorentzon R. Heavy-load eccentric calf muscle training for the treatment of chronic Achilles tendinosis. Am J Sports Med 1998;26(3):360-366.
2. Alfredson H, Zeisig E, Fahlstrom M. No normalisation of the tendon structure and thickness after intratendinous surgery for chronic painful midportion Achilles tendinosis. Br J Sports Med 2009;43:948-949.
3. Ames PR, Longo UG, Denaro V, Maffulli N. Achilles tendon problems: not just an orthopaedic issue. Disabil Rehabil 2008; 30(20-22):1646-1650.
4. Bramble DM, Lieberman DE. Endurance running and the evolution of Homo. Nature 2004;432(7015):345-352.
5. Coleman BD, Khan KM, Maffulli N, Cook JL, Wark JD. Studies of surgical outcome after patellar tendinopathy: clinical significance of methodological deficiencies and guidelines for future studies. Victorian Institute of Sport Tendon Study Group. Scand J Med Sci Sports 2000;10(1):2-11.
6. Danforth CH. The Heredity of Unilateral Variations in Man. Genetics 1923;9:199-210.
7. Daseler EH. The Plantaris Muscle: An Anatomical Study of 750 Specimens. J Bone Joint Surg 1943;25:822-827.
8. Dawson J, Carr A. Outcomes evaluation in orthopaedics. J Bone Joint Surg Br 2001; 83(3):313-315.
9. de Vos RJ, Heijboer MP, Weinans H, Verhaar JA, van Schie HT. Tendon Structure is Not Related to Clinical Outcome Following Eccentric Exercises in Chronic Mid-Portion Achilles Tendinopathy. Unpublished results.
10. Emerson C, Morrissey D, Perry M, Jalan R. Ultrasonographically detected changes in Achilles tendons and self reported symptoms in elite gymnasts compared with controls - An observational study. Man Ther 2010; 15(1):37-42.
11. Fahlstrom M, Jonsson P, Lorentzon R, Alfredson H. Chronic Achilles tendon pain treated with eccentric calf-muscle training. Knee Surg Sports Traumatol Arthrosc 2003;11(5): 327-333.
12. Freeman AJ, Jacobson NA, Fogg QA. Anatomical variations of the plantaris muscle and a potential role in patellofemoral pain syndrome. Clin Anat 2008;21(2):178-181.
13. Guillemin F. Cross-cultural adaptation and validation of health status measures. Scand J Rheumatol 1995;24(2):61-63.
14. Guillemin F, Bombardier C, Beaton D. Cross-cultural adaptation of health-related quality of life measures: literature review and proposed guidelines. J Clin Epidemiol 1993; 46(12):1417-1432.
15. Haims AH, Schweitzer ME, Patel RS, Hecht P, Wapner KL. MR imaging of the Achilles tendon: overlap of findings in symptomatic and asymptomatic individuals. Skeletal Radiol 2000;29(11):640-645.
16. Harvey FJ, Chu G, Harvey PM. Surgical availability of the plantaris tendon. J Hand Surg Am 1983;8(3):243-247.
17. Järvinen M. Epidemiology of tendon injuries in sports. Clin Sports Med 1992;11(3):493-504.
18. Järvinen TA, Kannus P, Maffulli N, Khan KM. Achilles tendon disorders: etiology and epidemiology. Foot Ankle Clin 2005;10(2): 255-266.
19. Järvinen TA, Kannus P, Paavola M, Jarvinen TL, Jozsa L, Jarvinen M. Achilles tendon injuries. Curr Opin Rheumatol 2001;13(2): 150-155.
20. Jozsa L, Kannus P. Human Tendons: Anatomy, Physiology, and Pathology. Human Kinetics 1997.
21. Kannus P, Jozsa L. Histopathological changes preceding spontaneous rupture of a tendon. A controlled study of 891 patients. J Bone Joint Surg Am 1991;73(10):1507-1525.

22. Khan KM, Forster BB, Robinson J et al. Are ultrasound and magnetic resonance imaging of value in assessment of Achilles tendon disorders? A two year prospective study. Br J Sports Med 2003;37(2):149-153.
23. Kirkup J. Mythology and History. In: Helal B, Wilson D, editors. The Foot. Churchill Livingstone; 1988:1-8.
24. Kujala UM, Sarna S, Kaprio J. Cumulative incidence of achilles tendon rupture and tendinopathy in male former elite athletes. Clin J Sport Med 2005;15(3):133-135.
25. Kvist H, Kvist M. The operative treatment of chronic calcaneal paratenonitis. J Bone Joint Surg Br 1980;62(3):353-357.
26. Kvist H, Kvist M. The operative treatment of chronic calcaneal paratenonitis. J Bone Joint Surg Br 1980;62(3):353-357.
27. Kvist M. Achilles tendon injuries in athletes. Sports Med 1994;18(3):173-201.
28. Lieberman DE, Bramble DM. The evolution of marathon running : capabilities in humans. Sports Med 2007;37(4-5):288-290.
29. Lohrer H, Arentz S, Nauck T, Dorn-Lange NV, Konerding MA. The Achilles tendon insertion is crescent-shaped: an in vitro anatomic investigation. Clin Orthop Relat Res 2008; 466(9):2230-2237.
30. Lohrer H, Nauck T. Cross-cultural adaptation and validation of the VISA-A questionnaire for German-speaking Achilles tendinopathy patients. BMC Musculoskelet Disord 2009; 10(1):134.
31. Longo UG, Ramamurthy C, Denaro V, Maffulli N. Minimally invasive stripping for chronic Achilles tendinopathy. Disabil Rehabil 2008;30(20-22):1709-1713.
32. Maffulli N, Binfield PM, Moore D, King JB. Surgical decompression of chronic central core lesions of the Achilles tendon. Am J Sports Med 1999;27(6):747-752.
33. Maffulli N, Khan KM, Puddu G. Overuse tendon conditions: time to change a confusing terminology. Arthroscopy 1998;14(8): 840-843.
34. Maffulli N, Testa V, Capasso G, Bifulco G, Binfield PM. Results of percutaneous longitudinal tenotomy for Achilles tendinopathy in middle- and long-distance runners. Am J Sports Med 1997;25(6):835-840.
35. Maffulli N, Walley G, Sayana MK, Longo UG, Denaro V. Eccentric calf muscle training in athletic patients with Achilles tendinopathy. Disabil Rehabil 2008;1-8.
36. Mafi N, Lorentzon R, Alfredson H. Superior short-term results with eccentric calf muscle training compared to concentric training in a randomized prospective multicenter study on patients with chronic Achilles tendinosis. Knee Surg Sports Traumatol Arthrosc 2001; 9(1):42-47.
37. Matheson GO. Nonoperative treatment of midportion Achilles tendinopathy: a review. Clin J Sport Med 2010;20(1):72-73.
38. Mathias SD, Fifer SK, Patrick DL. Rapid translation of quality of life measures for international clinical trials: avoiding errors in the minimalist approach. Qual Life Res 1994;3(6):403-412.
39. Moss ALH. Is There an Association Between an Absence of Palmaris Longus Tendon and an Absence of Plantaris Tendon? Eur J Plast Surg 1988;11:32-34.
40. Nelen G, Martens M, Burssens A. Surgical treatment of chronic Achilles tendinitis. Am J Sports Med 1989;17(6):754-759.
41. O'Brien M. The anatomy of the Achilles tendon. Foot Ankle Clin 2005;10(2):225-238.
42. Ohberg L, Lorentzon R, Alfredson H. Eccentric training in patients with chronic Achilles tendinosis: normalised tendon structure and decreased thickness at follow up. Br J Sports Med 2004;38(1):8-11.
43. Paavola M, Kannus P, Paakkala T, Pasanen M, Jarvinen M. Long-term prognosis of patients with achilles tendinopathy. An observational 8-year follow-up study. Am J Sports Med 2000;28(5):634-642.
44. Paavola M, Orava S, Leppilahti J, Kannus P, Jarvinen M. Chronic Achilles tendon overuse injury: complications after surgical

treatment. An analysis of 432 consecutive patients. Am J Sports Med 2000;28(1): 77-82.
45. Rees JD, Wilson AM, Wolman RL. Current concepts in the management of tendon disorders. Rheumatology (Oxford) 2006; 45(5):508-521.
46. Robinson JM, Cook JL, Purdam C et al. The VISA-A questionnaire: a valid and reliable index of the clinical severity of Achilles tendinopathy. Br J Sports Med 2001;35(5): 335-341.
47. Rolf C, Movin T. Etiology, histopathology, and outcome of surgery in achillodynia. Foot Ankle Int 1997;18(9):565-569.
48. Roos EM, Engstrom M, Lagerquist A, Soderberg B. Clinical improvement after 6 weeks of eccentric exercise in patients with mid-portion Achilles tendinopathy -- a randomized trial with 1-year follow-up. Scand J Med Sci Sports 2004;14(5):286-295.
49. Saxena A, Bareither D. Magnetic resonance and cadaveric findings of the "watershed band" of the achilles tendon. J Foot Ankle Surg 2001;40(3):132-136.
50. Schepsis AA, Jones H, Haas AL. Achilles tendon disorders in athletes. Am J Sports Med 2002;30(2):287-305.
51. Schwalbe, Pfitzner. Varietäten-Statistik und Anthropologie. Deutsche Med Wchnschr 1894;XXV(3):459.
52. Segesser B, Goesele A, Renggli P. [The Achilles tendon in sports]. Orthopäde 1995; 24(3):252-267.
53. Silbernagel KG, Thomee R, Thomee P, Karlsson J. Eccentric overload training for patients with chronic Achilles tendon pain--a randomised controlled study with reliability testing of the evaluation methods. Scand J Med Sci Sports 2001;11(4):197-206.
54. Smith RK, Birch HL, Goodman S, Heinegard D, Goodship AE. The influence of ageing and exercise on tendon growth and degeneration--hypotheses for the initiation and prevention of strain-induced tendinopathies. Comp Biochem Physiol A Mol Integr Physiol 2002;133(4):1039-1050.
55. Steenstra F, van Dijk CN. Achilles tendoscopy. Foot Ankle Clin 2006;11(2):429-38.
56. Tallon C, Coleman BD, Khan KM, Maffulli N. Outcome of surgery for chronic Achilles tendinopathy. A critical review. Am J Sports Med 2001;29(3):315-320.
57. Thermann H, Benetos IS, Panelli C, Gavriilidis I, Feil S. Endoscopic treatment of chronic mid-portion Achilles tendinopathy: novel technique with short-term results. Knee Surg Sports Traumatol Arthrosc 2009; 17(10):1264-1269.
58. Tuite DJ, Renstrom PA, O'Brien M. The aging tendon. Scand J Med Sci Sports 1997;7(2): 72-77.
59. van Dijk CN, van Dyk GE, Scholten PE, Kort NP. Endoscopic calcaneoplasty. Am J Sports Med 2001;29(2):185-189.
60. Williams JG. Achilles tendon lesions in sport. Sports Med 1993;16(3):216-220.