ELSEVIER
SAUNDERS

Foot Ankle Clin N Am
10 (2005) 309–320

FOOT AND
ANKLE CLINICS

Insertional Achilles Tendinopathy

Murali Krishna Sayana, MBBS, MS, AFRCSI,
Nicola Maffulli, MD, MS, PhD, FRCS(Orth)*

Department of Trauma and Orthopaedics, University Hospital of North Staffordshire, Keele University School of Medicine, Thornburrow Drive, Stoke-on-Trent, ST4 7QB, UK

Achilles tendinopathy is one of the conditions that causes posterior heel pain. Clain and Baxter [1] introduced the terms insertional and noninsertional Achilles tendinopathy with a view to plan management better. Insertional tendinopathy had a prevalence of 20% in a surgical and histopathologic survey of 163 patients who had chronic Achilles tendinopathy [2]. In a consecutive series of 432 patients who had chronic Achilles overuse injury in Finland, 107 (24.7%) had insertional Achilles pathology. Of these, 5% (21 patients) had pure insertional tendinopathy, and 20% (86 patients) had calcaneal bursitis alone or in combination with insertional tendinopathy [3].

The incidence of Achilles insertional tendinopathy is not well established. It was reported as the most common form of Achilles tendinopathy in athletes who presented to an outpatient clinic [4]. Conversely, 5% to 20% of the Achilles tendinopathy were of the insertional variety [3,5]. Insertional tendinopathy often is diagnosed in older, less athletic, overweight individuals [6], and in older athletes [7].

In this article we specifically concentrate on Achilles insertional tendinopathy. We shall not discuss Haglund's deformity and retrocalcaneal bursitis in detail. We advocate the use of the term ''tendinopathy'' for a clinical diagnosis that is based on pain, swelling (diffuse or localized), and impaired performance. We use the suffix ''osis'' or ''itis'' only after histopathologic examination of the affected tendons has confirmed degeneration or inflammation [8]. Although, insertional tendinopathy of the Achilles tendon is often described as ''true inflammation'' within the tendon [7], the histology from 22 patients who had

* Corresponding author.
E-mail address: n.maffulli@keele.ac.uk (N. Maffulli).

1083-7515/05/$ – see front matter
doi:10.1016/j.fcl.2005.01.010

recalcitrant calcific insertional Achilles tendinopathy showed fibrocartilaginous or calcifying degeneration close to the area of calcific tendinopathy. There was disorganization of the tendon substance with no evidence of intratendinous inflammatory reaction [9]. We advocate the use of the term ''Achilles insertional tendinopathy'' in this context.

Anatomy

The gastrocnemius muscle merges with the soleus to form the Achilles tendon. It has a round upper part, and is mostly flat in its distal 4 cm. Its fibers spiral through 90° and increase the release of stored energy during locomotion [10]. The Achilles tendon inserts into the posterior surface of the calcaneus, which can be divided into three areas [11]:

- A triangular bursal area with superior apex. The tendon is not inserted in this area.
- A rough quadrilateral area inferior to the bursal area that provides insertion to the central part of the Achilles tendon.
- A triangular inferior area with inferior apex. This gives attachment to fascial structures that are continuous with the plantar fascia below and with the sheath of the Achilles tendon above.

The insertion of the Achilles tendon, the posterior aspect of the calcaneus, the retrocalcaneal bursa, and the pretendinous bursa constitute the posterior aspect of the heel. The enthesis, the bursa, and the bursal walls form a complex insertional region that protects the Achilles tendon and the posterior aspect of the heel.

Histology

The osteo-tendinous junction of the Achilles consists of tendon, fibrocartilage, and bone. Milz et al [12] used the distribution of type II collagen in sagittal sections of the Achilles tendon to reconstruct the three-dimensional (3-D) shape and position of three fibrocartilages (sesamoid, periosteal, and enthesis) that are associated with its insertion. A close correspondence between the shape and position of the sesamoid and periosteal fibrocartilages was found. The former protects the tendon from compression during dorsiflexion of the foot, and the latter protects the superior tuberosity of the calcaneus. The 3-D reconstructions that used the zone of calcified enthesis, fibrocartilage, and the subchondral bone showed complex interlocking between calcified fibrocartilage and bone at the insertion site; this is of fundamental importance in anchoring the tendon to the bone.

Merkel et al [13] studied 11 insertional tendinopathy (including two Achilles tendons) specimens using light and electronic microscopy, and enzyme histochemistry. The pathologic changes of insertional tendinopathy consisted of edema, mucoid degeneration, disruption of collagen bundles, necroses, small hemorrhages, and calcification. Acid mucopolysaccharides may be present in lakelike accumulations between collagen fibers, in contrast to neutral collagens that are seen in aging. Small bony particles lay within the cartilaginous portion of the insertion. Also, there were areas with proliferating blood vessels within tendon tissue with lymphocytes and histiocytes that suggested a reparative process. There was increased activity of NADP-diaphorase, lactate dehydrogenase (LDH) in these tendon samples. β-glucuronidase and alkaline phosphatase enzymes were also found in these samples, though their activity was lower compared to NADP-diaphorase, LDH. Electron microscopy showed marked submicroscopic calcification and fibrillar degeneration.

Etiopathophysiology

Classically, overuse and poor training habits are considered to be the main etiology of Achilles insertional tendinopathy. Spur formation and calcification at the Achilles insertion is attributed to gradual repetitive traction force. Benjamin et al [14] studied enthesophyte formation in rats compared with human specimens. Bony spurs can develop in the Achilles tendon without the need for preceding microtears or inflammatory reactions, and form by endochondral ossification of enthesis fibrocartilage. The increased surface area that is created at the tendon–bone junction may be an adaptive mechanism to ensure the integrity of the interface in response to increased mechanical loads [14].

Tight Achilles tendon, hyperpronation, pes cavus, and obesity can predispose to degeneration, attrition, mechanical abrasion, and chemical irritation that could lead to a chronic inflammatory response at the heel [6].

Lyman et al [15] studied the in vitro strain behavior of the anterior portion of the Achilles tendon as it is affected by the insertional tendinopathy. Relative strain shielding was noticed in this portion of the tendon; this suggests that the role of repetitive tensile loads in the causation of Achilles insertional tendinopathy is complex. These findings may explain the variable therapeutic response that followed measures that were aimed at decreasing tensile loads on the tendon.

A different etiology of insertional tendinopathy recently was suggested based on biomechanical studies (C.N. Maganaris, PhD, personal communication, 2004). There is a distinct tendency to develop cartilage-like or atrophic changes on the stress-shielded side of the enthesis as a response to the lack of tensile load [16,17]. Over long periods, this process may induce a primary degenerative lesion in that area of the tendon. Thus, tendinopathy is not always activity-related, and can be correlated with age; this suggests that insertional tendinopathy would result from stress-shielding, rather than overuse injury.

In many ways, the cartilage-like changes in the enthesis can be considered to be a physiologic adaptation to the compressive loads. Even cartilaginous metaplasia, however, may not allow the tendon to maintain its ability to withstand the high tensile loads in that region. In athletes, certain joint positions may place high tensile loads on the enthesis. As the stress-shielding may have led to tensile weakening over time, an injury may occur more easily in this region. In this manner, insertional tendinopathy could be considered to be an overuse injury, but is predisposed by pre-existing weakening of the tendon.

As the joint changes position, strains in one section of the tendon could produce changes in the opposite direction. Internal shear forces and heat could be generated that produce injury to the cellular or matrix components of the tendon [18]. Accumulation of these injuries could lead to the intratendinous degeneration that is seen in tendinopathy.

Clinical features

Achilles insertional tendinopathy is characterized by early morning stiffness, pain that is localized at the insertion of the Achilles tendon that worsens after exercise, climbing stairs, running on hard surfaces, or heel running. The pain may become constant. There may be a history of a recent increase in training, and poor warm-up or stretching habits. Examination reveals tenderness at the Achilles tendon insertion, thickening or nodularity of the insertion, and limited dorsiflexion of the ankle.

Differential diagnosis

Other causes of posterior heel pain should be considered. Achilles insertional tendinopathy is a localized manifestation of systemic conditions, such as gout, hyperlipidemia, sarcoidosis, diffuse idiopathic skeletal hyperostosis, or seronegative spondyloarthropathies. Systemic corticosteroids and oral fluoroquinolones may induce Achilles insertional tendinopathy. Local conditions, such as Haglund's deformity, retrocalcaneal bursitis, os trigonum, posterior impingement, posterior talar process fracture, flexor hallucis longus tendinopathy, peroneal tendinopathy, tibialis posterior tendinopathy, deltoid ligament sprain, and osteochondral lesions of talus should be ruled out.

Investigations

Radiographs may reveal ossification at the insertion of the Achilles tendon or a spur (fishhook osteophyte) on the superior portion of the calcaneum. Morris et al [19] classified the radio-opacities of the Achilles tendon into three types (Table 1).

Table 1
Radio-opacities of the Achilles tendon

Type I lesion	Etiology
Microtrauma	Shoe counter, work-related irritation
Macrotrauma	Insertion rupture, blunt trauma
Tendinopathy	Overuse, bursitis, calcaneus shape
Foot type	Cavus, rear foot varus, plantarflexed 1st metatarsal
Arthropathy	Gout, rheumatoid, Reiter's, ankylosing spondylitis, diffuse idiopathic skeletal hyperostosis
Metabolic	Renal failure, obesity, hyperparathyroidism, hemochromatosis
Infectious	Acute or chronic syphilis
Type II lesion	
Arthropathy	Articular chondrocalcinosis, pseudogout
Metabolic	Vitamin deficiency
Type III lesion	
Trauma	Burn injury, partial/total tendon rupture
Postsurgery	Primary repair, lengthening, recession
Ischemia	Inherent anatomy
Infectious	Chronic osteomyelitis
Systemic/metabolic	Wilson's disease, hemochromatosis
Congenital	Aperiosteal metaplasia, neural arch deficiency
CNS	Tabes dorsalis

Abbreviation: CNS, central nervous system.
Modified from Morris KL, Giacopelli JA, Granoff D. Classification of radiopaque lesions of tendo Achillis. J Foot Surg 1990;29(6):536.

Type I: radio-opacities at the Achilles insertion or superior pole of the calcaneus. The lesion is present within the tendon and is attached partially or completely to the calcaneus. Bony changes to the calcaneus often are seen in type I lesions. Insertional Achilles tendinopathy causes type I abnormality (Fig. 1).

Type II: intratendinous radio-opacities located at the insertion zone, 1 cm to 3 cm proximal to the Achilles insertion and separated from the calcaneal surface.

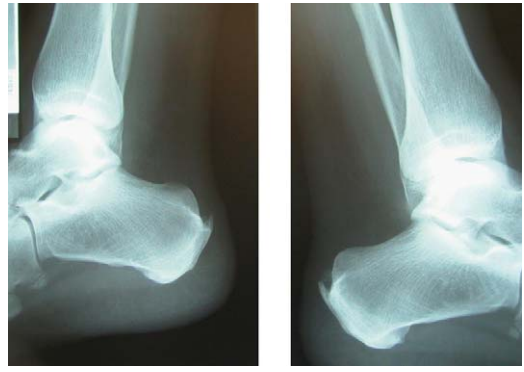

Fig. 1. Bilateral Achilles insertional tendinopathy. Type I.

Table 2
Ultrasonographic classification of insertional Achilles tendon abnormality

Classification	Insertional changes
No alteration	No calcification. Homogeneous fiber structure in the insertional area.
Mild abnormality	Insertional calcification, length 10 mm or less and thickness less than 2 mm. Homogeneous fiber structure in the insertional area.
Moderate abnormality	Insertional calcification, length more than 10 mm and thickness less than 2 mm. Slight alterations in the echo structure of tendon in the insertional area.
Severe abnormality	Insertional calcification, length more than 10 mm or thickness more than 2 mm. Moderate to severe variety in the echo structure of tendon in the insertional area.

Type III: radio-opacities located proximal to the insertion zone, up to 12 cm above the insertion zone. Type III is subdivided into IIIA (partial tendon calcification) and IIIB (complete tendon calcification).

MRI and ultrasound scan are not needed for diagnosis, but they may help to identify the extent of the intratendinous lesion. An ultrasonographic classification of the Achilles tendon abnormalities that is based on changes at the Achilles tendon insertion was introduced by Paavola et al (Table 2) [20].

Management

Conservative management produces an 85% to 95% success rate [6,21] with rest, ice, modification of training, heel lift, and orthoses. Nonsteroidal anti-inflammatory medications in Achilles tendinopathy may only provide an analgesic effect [22]. Piroxicam showed no benefit over placebo in a randomized controlled trial when it was combined with an initial period of rest followed by stretching and strengthening exercises [23]. Diclofenac reduced the accumulation of inflammatory cells only within the paratenon, but provided no biochemical, mechanical, or functional benefits to the rat Achilles tendon following injury when compared with placebo. Also, no reduction in the accumulation of neutrophils and macrophages was found in the core of the tendon [24].

Modification of training, stretching, and strengthening exercises also are effective; however, eccentric calf muscle training helped only 32% of cases of Achilles insertional tendinopathy compared with 89% of cases of noninsertional tendinopathy [25]. In athletes, nonweight-bearing activities can help to maintain fitness until symptoms improve. Immobilization of the ankle in a below-knee weight-bearing cast or a walker boot can be counterproductive, although it was suggested by some investigators [6,26]. Tendon loading stimulates collagen fiber repair and remodeling. Therefore, complete rest of an injured tendon is not advisable. Ultrasound treatment could be beneficial to control symptoms at the insertion site. We do not use local injections of corticosteroids.

Sclerosing therapy in insertional tendinopathy showed promising results in a pilot study [27]. Polidocanol was injected into local neovessels that were localized by ultrasound and color Doppler. Eight of eleven patients experienced good pain relief, and seven of them had no neovascularization at a mean follow-up of 8 months.

Surgery

Surgery is undertaken if conservative management fails. Various surgical procedures have been described. The principles of surgery include debridement of the calcific or diseased portion of the Achilles insertion, excision of the retrocalcaneal bursa, and resection of the superior prominence. We prefer to reattach the Achilles tendon using bone anchors if one third or more of the insertion is disinserted. Augmentation using tendon transfer is well-described.

Anderson et al [28] studied the surgical management of chronic Achilles tendinopathy in 48 patients that included 27 competitive athletes. Twenty-eight (58%) patients underwent surgery for Achilles insertional tendinopathy with tenolysis, excision of the bursa, or excision of the posterosuperior portion of the calcaneum through a 10-cm medial incision. The recovery in these patients was longer (31 weeks) when compared with an Achilles tenolysis group (22 weeks) with a success rate of 93%.

Calder and Saxby [29] reported their results of intervention in which less than 50% of the tendon was excised (49 heels); the operated ankles were mobilized immediately, free of a cast. There were two failures using this regimen: one patient who had psoriatic arthropathy and another who underwent bilateral simultaneous procedures. Kolodziej et al [30] reported a biomechanical study that concluded that superior-to-inferior resection offers the greatest margin of safety when performing partial resections of the Achilles insertion; as much as 50% of the tendon may be resected safely.

McGarvey et al [31] reported the results on 22 heels that had surgery using a midline-posterior skin incision combined with a central tendon splitting approach for debridement, retrocalcaneal bursectomy, and removal of the calcaneal bursal projection, as necessary. Twenty of 22 patients were able to return to work or routine activities by 3 months. Only 13 of 22 patients were completely pain-free and were able to return to unlimited activities. Overall, there was an 82% (18 of 22 patients) satisfaction rate with surgery.

Watson et al [32] reported that retrocalcaneal decompression in patients who had insertional Achilles tendinopathy with a calcific spur was less satisfactory when compared with retrocalcaneal decompression in patients who had isolated retrocalcaneal bursitis.

Den Hartog [33] reported the successful use of flexor hallucis longus transfer for severe calcific Achilles tendinopathy in 26 patients (29 tendons), in whom conservative treatment had failed and who also had failed tendon de-

bridement or Haglund's resection. These patients were sedentary, overweight, and had chronic symptoms. The American Orthopaedic Foot and Ankle Society ankle-hindfoot scale improved from 41.7 to 90.1. The time to maximum recovery was approximately 6 months. All patients lost flexor strength at the interphalangeal joint of the great toe.

Leitze et al [34] recently reported decompression of the retrocalcaneal space using a minimally invasive technique. Patients who had retrocalcaneal bursitis, mechanical impingement, or Achilles insertional tendinopathy who failed to respond to conservative management underwent endoscopic decompression; major calcific insertional tendinopathy of the Achilles tendon was considered to be a contraindication for endoscopic decompression. The advantages of the endoscopic procedure included quicker surgery and fewer complications, although the recovery time was similar to open decompression.

We reported on 21 patients who had recalcitrant calcific insertional Achilles tendinopathy who underwent bursectomy, excision of the distal paratenon, disinsertion of the tendon, removal of the calcific deposit, and reinsertion of the Achilles tendon with bone anchors. The outcome of surgical management was rated according to Testa et al [35], using a 4-point functional scale that was validated for the evaluation of long-term results following surgery for tendinopathy. Eleven patients reported an excellent result and five reported a good result. The remaining five patients could not return to their normal levels of sporting activity and kept fit by alternative means [9].

Authors' preferred surgical technique

Surgery is performed on a day case basis. Under general anesthesia, the patient is laid prone with a thigh tourniquet that is inflated to 250 mm Hg after the limb has been exsanguinated to provide hemostasis. The Achilles tendon is exposed through a longitudinal incision that is 1 cm medial to the medial border of the tendon, and is extended from the lower one third of the tendon up to 2 cm distal to its calcaneal insertion (Fig. 2). The incision can be extended transversely and laterally in a hockey-stick fashion, if necessary. Sharp dissection is continued to the paratenon, which is dissected from the tendon and excised, taking care to preserve the anterior fat in Kager's triangle and not to injure the mesotenon. The retrocalcaneal bursa is excised, if there is evidence of bursitis. The Achilles tendon is inspected for areas that had lost their normal shining appearance and palpated for areas of softening or thickening. The areas that have lost their normal shining appearance, and the areas that are softer or thicker are explored by way of one to three longitudinal tenotomies; areas of degeneration are excised and sent for histology. The longitudinal tenotomies are not repaired. The area of calcific tendinopathy is identified and its proximal, medial, and lateral edges are defined using the tip of a syringe needle. The calcific area is exposed starting from its proximal and medial aspect. Most patients have at least one third of the Achilles tendon that surrounds the area of

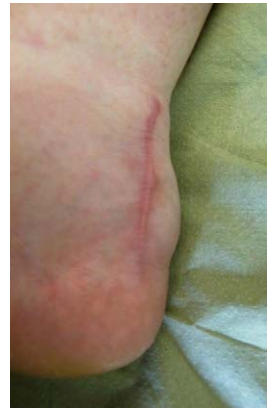

Fig. 2. Well-healed incision on the medial side 6 weeks after surgery. The posterior swelling would take a few months to subside.

calcific tendinopathy detached by sharp dissection; occasionally, a total disinsertion has to be performed. The area of calcific tendinopathy is excised from the calcaneus. The area of hyaline cartilage at the posterosuperior corner of the calcaneus may be degenerated macroscopically; it is excised using an osteotome and, if needed, its base paired off with bone nibblers. The tendon is reinserted in the calcaneus using bone anchors. Two bone anchors are be used if 33% to 50% of the Achilles tendon is disinserted; three soft bone anchors are used if 50% to 75% of the Achilles tendon is disinserted; four bone anchors are used if 75% or more of the Achilles tendon is disinserted (Fig. 3); and five bone anchors are used if the Achilles tendon is disinserted totally. The Achilles tendon is advanced in a proximal to distal fashion and reinserted in the calcaneum. We normally do not perform a tendon augmentation or a tendon transfer. After release of the tourniquet, hemostasis is achieved by diathermy. The wound is closed in layers using absorbable sutures.

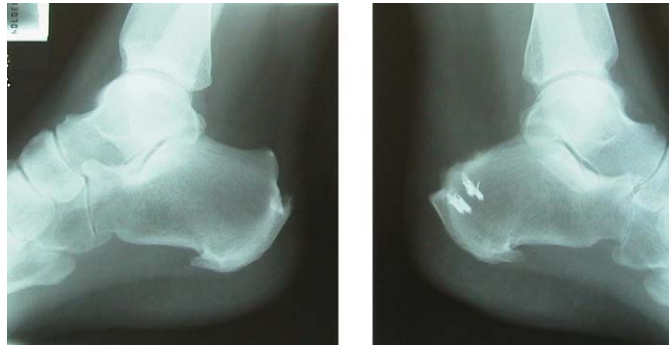

Fig. 3. Four suture anchors in a postoperative radiograph.

Postoperative management

The skin wound is dressed with gauze, and sterile plaster wool is applied. A synthetic below-knee cast, with the ankle plantigrade, is applied. Patients are discharged the day of surgery. Patients are mobilized with crutches under the guidance of a physiotherapist in the immediate postoperative period. Patients are advised to bear weight on the operated leg as tolerated, but are told to keep the leg elevated as much as possible for the first 2 postoperative weeks. The cast is removed 2 weeks after the operation. A synthetic anterior below-knee slab is applied, with the ankle in neutral. The synthetic slab is secured to the leg with three or four removable Velcro straps for 4 weeks. The patients are encouraged to continue to bear weight on the operated limb and to progress gradually to full weight bearing, if they are not doing so already. A trained physiotherapist supervises gentle mobilization exercises of the ankle, isometric contraction of the gastrosoleus complex, and gentle concentric contraction of the calf muscles. Patients are encouraged to perform mobilization of the involved ankle several times per day after unstrapping of the relevant Velcro straps. Patients are reviewed 6 weeks from the operation, when the anterior slab is removed. Stationary cycling and swimming is recommended from the second week after removal of the cast. We allow return to gentle training 6 weeks after removal of the cast. Gradual progression to full sports activity at 20 to 24 weeks from the operation is planned, according to the patient's progress. Resumption of competition is dependent on the patient's plans, but is not recommended before 6 months after surgery. Patients are reviewed at 3, 6, and 9 months from the operation, and every 6 months thereafter. Patients are advised to contact the operating surgeon should any problems ensue. Further physiotherapy, along the lines as described above, is prescribed if symptoms are still present; the patients are followed until they improve, and therefore, are discharged; need further surgery; or default.

Discussion

There has been significant progress in our understanding of Achilles insertional tendinopathy since Clain and Baxter [1] divided Achilles tendon disorders into noninsertional and insertional tendinopathy in 1992. Achilles insertional tendinopathy is a degenerative, rather than an inflammatory, lesion, although the accompanying bursitis may paint a picture of an inflammatory lesion.

The true incidence of Achilles insertional tendinopathy is not clear. Incidence varies from 5% to the most common presentation in athletes. Achilles insertional tendinopathy is recognized as being distinct from retrocalcaneal bursitis and Haglund's deformity, although they often can coexist. Further epidemiologic studies are needed, with a clear terminology, to identify the true incidence of this problem.

The in vitro biomechanical studies on the distal Achilles tendon have given us new insight into possible etiologies. Eccentric calf muscle training was not beneficial in Achilles insertional tendinopathy [16]. This reiterates the fact that the treatment strategies should be different in insertional and noninsertional tendinopathy of the Achilles tendon, because their etiologies are likely to be different. The in vitro strain studies on the distal Achilles tendon identified that the anterior portion of the Achilles insertion is stress-shielded or is underused. This stress-shielded area could be a site for primary degenerative lesion, or could be predisposed to injury because of pre-existing weakness. This can explain the occurrence of this lesion in older, less athletic, overweight individuals, and persons with poor warm-up and stretching habits, and a recent increase in training.

The diagnosis mainly is clinical and radiographs help to confirm the diagnosis as do ultrasound scan or MRI scan. Newer management measures will be introduced as the etiology of Achilles insertional tendinopathy becomes clearer. Various surgical techniques are aimed at debriding the degenerate area of the Achilles tendon; this is accompanied by excision of the retrocalcaneal bursa and resection of the superior prominence. We prefer to reattach the Achilles tendon using bone anchors if one third or more of the Achilles tendon insertion is disinserted.

References

[1] Clain MR, Baxter DE. Achilles tendinitis. Foot Ankle 1992;13(8):482–7.
[2] Astrom M, Rausing A. Chronic Achilles tendinopathy. A survey of surgical and histopathologic findings. Clin Orthop 1995;316:151–64.
[3] Paavola M, Orava S, Leppilahti J, et al. Chronic Achilles tendon overuse injury: complications after surgical treatment. An analysis of 432 consecutive patients. Am J Sports Med 2000;28(1):77–82.
[4] Benazzo F, Todesca A, Ceciliani L. Achilles tendon tendonitis and heel pain. Oper Tech Sports Med 1997;4(3):179–88.
[5] Kvist M. Achilles tendon injuries in athletes. Sports Med 1994;18(3):173–201.
[6] Myerson MS, McGarvey W. Disorders of the Achilles tendon insertion and Achilles tendinitis. Instr Course Lect 1999;48:211–8.
[7] Schepsis AA, Jones H, Haas AL. Achilles tendon disorders in athletes. Am J Sports Med 2002;30(2):287–305.
[8] Maffulli N, Khan KM, Puddu G. Overuse tendon conditions: time to change a confusing terminology. Arthroscopy 1998;14(8):840–3.
[9] Maffulli N, Testa V, Capasso G, et al. Calcific insertional Achilles tendinopathy: reattachment with bone anchors. Am J Sports Med 2004;32(1):174–82.
[10] Alexander RM, Bennet-Clark HC. Storage of elastic strain energy in muscle and other tissues. Nature 1977;265:114–7.
[11] Fowler A, Philip JF. Abnormality of the calcaneus as a cause of painful heel. Its diagnosis and operative treatment. Br J Surg 1945;32:494–8.
[12] Milz S, Rufai A, Buettner A, et al. Three-dimensional reconstructions of the Achilles tendon insertion in man. J Anat 2002;200(2):145–52.
[13] Merkel KHH, Hess H, Kunz M. Insertion tendinopathy in athletes. A light microscopic, histochemical and electron microscopic examination. Path Res Pract 1982;173:303–9.

[14] Benjamin M, Rufai A, Ralphs JR. The mechanism of formation of bony spurs (enthesophytes) in the Achilles tendon. Arthritis Rheum 2000;43(3):576–83.
[15] Lyman J, Weinhold PS, Almekinders LC. Strain behavior of the distal Achilles tendon: implications for insertional Achilles tendinopathy. Am J Sports Med 2004;32(2):457–61.
[16] Rufai A, Ralphs JR, Benjamin M. Structure and histopathology of the insertional region of the human Achilles tendon. J Orthop Res 1995;13(4):585–93.
[17] Benjamin M, Evans EJ, Copp L. The histology of tendon attachments to bone in man. J Anat 1986;149:89–100.
[18] Wilson AM, Goodship AE. Exercise-induced hyperthermia as a possible mechanism for tendon degeneration. J Biomech 1994;27(7):899–905.
[19] Morris KL, Giacopelli JA, Granoff D. Classification of radiopaque lesions of tendo Achillis. J Foot Surg 1990;29(6):533–42.
[20] Paavola M, Kannus P, Paakkala T, et al. Long-term prognosis of patients with achilles tendinopathy. An observational 8-year follow-up study. Am J Sports Med 2000;28(5):634–42.
[21] Clement DB, Taunton JE, Smart GW. Achilles tendinitis and peritendinitis: etiology and treatment. Am J Sports Med 1984;12(3):179–84.
[22] Almekinders LC. Breaking with tradition. Rehab Manag 2002;15(6):40–2, 45.
[23] Astrom M, Westlin N. No effect of piroxicam on Achilles tendinopathy. A randomized study of 70 patients. Acta Orthop Scand 1992;63(6):631–4.
[24] Marsolais D, Cote CH, Frenette J. Nonsteroidal anti-inflammatory drug reduces neutrophil and macrophage accumulation but does not improve tendon regeneration. Lab Invest 2003;83(7): 991–9.
[25] Fahlstrom M, Jonsson P, Lorentzon R, et al. Chronic Achilles tendon pain treated with eccentric calf-muscle training. Knee Surg Sports Traumatol Arthrosc 2003;11(5):327–33.
[26] Gerken P, McGarvey WC, Baxter DE. Insertional Achilles tendinitis. Foot Ankle Clin N Am 1996;1(112):237–48.
[27] Ohberg L, Alfredson H. Sclerosing therapy in chronic Achilles tendon insertional pain-results of a pilot study. Knee Surg Sports Traumatol Arthrosc 2003;11(5):339–43.
[28] Anderson DL, Taunton JE, Davidson RG. Surgical management of chronic Achilles tendonitis. Clin J Sport Med 1992;2(1):39–42.
[29] Calder JD, Saxby TS. Surgical treatment of insertional Achilles tendinosis. Foot Ankle Int 2003;24(2):119–21.
[30] Kolodziej P, Glisson RR, Nunley JA. Risk of avulsion of the Achilles tendon after partial excision for treatment of insertional tendonitis and Haglund's deformity: a biomechanical study. Foot Ankle Int 1999;20(7):433–7.
[31] McGarvey WC, Palumbo RC, Baxter DE, et al. Insertional Achilles tendinosis: surgical treatment through a central tendon splitting approach. Foot Ankle Int 2002;23(1):19–25.
[32] Watson AD, Anderson RB, Davis WH. Comparison of results of retrocalcaneal decompression for retrocalcaneal bursitis and insertional Achilles tendinosis with calcific spur. Foot Ankle Int 2000;21(8):638–42.
[33] Den Hartog BD. Flexor hallucis longus transfer for chronic Achilles tendonosis. Foot Ankle Int 2003;24(3):233–7.
[34] Leitze Z, Sella EJ, Aversa JM. Endoscopic decompression of the retrocalcaneal space. J Bone Joint Surg Am 2003;85-A(8):1488–96.
[35] Testa V, Maffulli N, Capasso G, et al. Percutaneous longitudinal tenotomy in chronic Achilles tendonitis. Bull Hosp Jt Dis 1996;54:241–4.