

Eccentric training in the treatment of tendinopathy

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ISBN: 978-91-7264-821-0

ISSN: 0346-6612 (1279)

Printed in Sweden by Print & Media, Umeå University, Umeå

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“No pain, no gain”
Benjamin Franklin (1758)

Dedicated to my family – Eva, Willy and Saga

Contents

Abstract	7
Abbreviations	8
Original papers	9
Introduction/Background	10
The normal tendon	11
Anatomy	11
<i>Collagen fibre orientation</i>	12
<i>Internal architecture</i>	12
<i>General innervation</i>	13
General biomechanical forces in tendons	14
Metabolism	15
Disuse/immobilisation	15
Exercise/remobilisation	15
The Achilles tendon	17
Anatomy	17
<i>The myotendinous junction (MTJ)</i>	18
<i>The osteotendinous junction (OTJ)</i>	18
<i>Tendon structure</i>	19
<i>Circulation</i>	19
<i>Innervation</i>	19
Biomechanics	20
Achilles tendinopathy	20
<i>Definitions</i>	20
<i>Epidemiology</i>	21
<i>Aetiology</i>	21
<i>Intrinsic risk factors</i>	21
<i>Extrinsic risk factors</i>	22
<i>Pathogenesis</i>	23
<i>Histology</i>	24
<i>Pain mechanisms</i>	24
<i>Clinical symptoms</i>	25
<i>Clinical examination</i>	25
<i>Differential diagnoses</i>	25
Treatment	26
The patellar tendon	28
Anatomy	28
<i>Tendon structure</i>	29
<i>Circulation</i>	29
<i>Innervation</i>	29
Biomechanics	29

Patellar tendinopathy	30
<i>Definitions</i>	30
<i>Epidemiology</i>	30
<i>Aetiology</i>	30
<i>Intrinsic risk factors</i>	31
<i>Extrinsic risk factors</i>	31
<i>Pathogenesis</i>	32
<i>Histology</i>	32
<i>Pain mechanisms</i>	32
<i>Clinical symptoms</i>	33
<i>Clinical examination</i>	33
<i>Differential diagnoses</i>	34
Treatment	34
The supraspinatus tendon	36
Anatomy	36
<i>Tendon structure</i>	37
<i>Circulation</i>	38
<i>Innervation</i>	38
Biomechanics	38
Supraspinatus tendinopathy	39
<i>Definitions</i>	39
<i>Epidemiology</i>	39
<i>Aetiology</i>	39
<i>Intrinsic risk factors</i>	39
<i>Extrinsic risk factors</i>	40
<i>Pathogenesis</i>	40
<i>Histology</i>	41
<i>Pain mechanisms</i>	41
<i>Clinical symptoms</i>	42
<i>Clinical examination</i>	42
<i>Differential diagnoses</i>	42
Treatment	43
General aims	45
Material and methods	46
Subjects	46
General inclusion and exclusion criteria	47
<i>Inclusion criteria</i>	47
<i>Exclusion criteria</i>	47
Ultrasound and magnetic resonance imaging	48
Treatment methods	48
<i>Coaching</i>	48
<i>Eccentric training program</i>	49
<i>Study I</i>	50
<i>Study II</i>	51

<i>Study III</i>	52
<i>Study IV</i>	53
<i>Study V</i>	54
Outcome measures	55
<i>Visual analogue scale (VAS)</i>	55
<i>Satisfaction with treatment</i>	55
<i>The Victorian Institute of Sport Assessment (VISA)</i>	55
<i>The Constant score</i>	55
Statistical methods	55
<i>Ethics</i>	56
Summary of papers	57
Paper I	57
Paper II	58
Paper III	59
Paper IV	60
Paper V	61
General discussion	62
Conclusions	68
Acknowledgements	69
References	70

Abstract

Chronic painful tendinopathies are common, not only in sports and recreationally active people, but also among people with a sedentary lifestyle. Both the lower and upper limbs are affected. There is lack of knowledge about the etiology and pathogenesis to tendinopathy, and many different treatments options have been presented. Unfortunately, most treatments have not been tested in scientific studies. Conservative (non-surgical) treatment has since long shown unsatisfactory results and surgical treatment is known to give unpredictable results.

The aim of this thesis was to evaluate new models of painful eccentric training for the conservative treatment of different chronic tendinopathies. After promising results in a pilot study, using painful eccentric calf muscle training in patients with chronic mid-portion Achilles tendinopathy, we investigated if these results could be reproduced in a larger group of patients with both mid-portion and insertional Achilles tendinopathy (study I). After 12 weeks, 89% of the patients with pain from the mid-portion were satisfied and back in previous activities. In the group with insertional Achilles tendinopathy the results were poor. A new model for eccentric training was designed for patients with insertional Achilles tendinopathy. The eccentric calf muscle training was done from tip-toe to floor level (study II). With this new regimen 67% of the patients were satisfied and back in previous activities. The next step was to investigate the effects of painful eccentric quadriceps training on patients with jumper's knee/patellar tendinopathy (study III). Two different training protocols were used. Eccentric training performed on a 25° decline board showed promising results with reduced pain and a return to previous activities, while eccentric training without the decline board had poor results. In a following prospective study, patients with jumper's knee/patellar tendinopathy were randomised to either concentric or eccentric painful quadriceps training on a 25° decline board (study IV). After 12 weeks of training, there were significantly better results in the group that did eccentric training. In a pilot study (study V), we investigated painful eccentric deltoideus and supraspinatus muscle training on a small group of patients on the waiting list for surgical treatment of subacromial impingement syndrome. After 12 weeks of training, 5 out of 9 patients were satisfied with the results of treatment and withdrew from the waiting list for surgery. In conclusion, the present studies showed good clinical results with low risks of side effects and low costs. Thus, we suggest that painful eccentric training should be tried in patients with Achilles and patellar tendinopathy before intratendinous injections and surgery are considered. For patients with chronic painful impingement syndrome, the results of our small pilot study are interesting, and stimulates to randomised studies on larger materials.

Keywords: eccentric training, Achilles tendon, patellar tendon, supraspinatus tendon, impingement, tendinopathy, tendinosis

Abbreviations

ACh	Acetylcholine
Ac-joint	Acromioclavicular joint
CD	Colour Doppler
CGRP	Calcitonin gene-related peptide
ChAT	Choline acetyltransferase
COL5A1	Alpha 1 type V collagen
CSA	Cross-sectional area
ESWT	Extracorporeal shock wave therapy
GAGs	Glycosaminoglycans
KN	Kilo newton
MMPs	Matrix metalloproteinases
MTJ	Myotendinous junction
MRI	Magnetic resonance imaging
N	Newton
NK-1 R	Neurokinin-1 receptor
NSAID	Nonsteroidal anti-inflammatory drug
OTJ	Osteotendinous junction
PFPS	Patellofemoral pain syndrome
PGE2	Prostaglandin E2
PGP 9.5	Protein gene product 9.5
SAB	Subacromial bursa
Sc-joint	Sternoclavicular joint
SD	Standard Deviation
SLAP	Superior labral anterior to posterior
SP	Substance P
SPSS	Statistical Package for the Social Sciences
TIMPs	Tissue inhibitor metalloproteinases
US	Ultrasound
VAS	Visual analogue scale
VEGF	Vascular endothelial growth factor
VISA	The Victorian Institute of Sport Assessment
WHR	Waist-to-hip ratio
X-ray	Plain film radiography

Original papers

This thesis is based on the following papers, which will be referred to by their corresponding Roman numerals:

- I. Chronic Achilles tendon pain treated with eccentric calf muscle training. Fahlström M, Jonsson P, Lorentzon R, Alfredson H. *Knee Surg Sports Traumatol Arthrosc.* 2003 Sep; 11(5):327–333.
- II. New regimen for eccentric calf muscle training in patients with chronic insertional Achilles tendinopathy: results of a pilot-study. Jonsson P, Alfredson H, Sunding K, Fahlström M, Cook JL. *Br J Sports Med.* 2008 Sep; 42:746–749.
- III. A pilot study of the eccentric decline squat in the management of painful chronic patellar tendinopathy. Purdam CR, Jonsson P, Alfredson H, Lorentzon R, Cook JL, Khan KM. *Br J Sports Med.* 2004 Aug; 38(4):395–397.
- IV. Superior results with eccentric compared to concentric quadriceps training in patients with jumper's knee: a prospective randomised study. Jonsson P, Alfredson H. *Br J Sports Med.* 2005 Nov; 39(11):847–850.
- V. Eccentric training in chronic painful impingement syndrome of the shoulder: results of a pilot study. Jonsson P, Wahlström P, Öhberg L, Alfredson H. *Knee Surg Sports Traumatol Arthrosc.* 2006 Jan; 14(1):76–81.

Introduction/Background

Treatment of patients with chronic painful tendinopathies constitutes a clinical challenge. Tendinopathy not only affects athletes and recreationally active people, but also people with sedentary lifestyles. Tendon disorders are common, and both lower and upper limbs are affected. Traditionally, it has been suggested that the condition is inflammatory. Hence, treatment with rest and immobilisation has been recommended in conjunction with nonsteroidal anti-inflammatory drugs (NSAIDs) and corticosteroid injections (Andres, et al. 2008). However, research has shown that there is an absence of a prostaglandin E₂-mediated inflammation (PGE₂) within the tendon in chronic painful tendinopathies (Alfredson, et al. 1999; Alfredson, et al. 2001; Alfredson, et al. 2003a). For a long time, conservative (non-surgical) treatment has been associated with relatively poor clinical results, and surgical treatment has been associated with unpredictable results (Maffulli, et al. 1999). In a long-term follow-up study, 53% of patients with patellar tendinopathy had ended their sports career due to this painful condition (Kettunen, et al. 2002). About 25–33% of athletes with lower extremity tendinopathy demonstrated poor results following conservative therapy, and required surgical treatment (Kvist 1994; Cook, et al. 1997). In patients requiring surgery, only 46–64% recover sufficiently to return to sports activities after a rehabilitation period of 6–12 months (Cook, et al. 1997; Coleman, et al. 2000; Chiara Vulpiani, et al. 2003). Many different treatment options have been presented. Unfortunately, the majority of these treatment modalities are not scientifically based, but instead empirically.

The aim of this thesis was to evaluate new models of painful eccentric training for the conservative treatment of different chronic tendinopathies.

The normal tendon

Anatomy

Tendons are interposed between muscles and bones, and transmit the force created in the muscle to the bone which, in turn, enables joint movement. Basically, each muscle contains two tendons, one proximal and one distal. The point where the tendon connects to a muscle is referred to as the myotendinous junction (MTJ), and the point where it connects to bone is referred to as the osteotendinous junction (OTJ) (Ippolito, et al. 1986;Kannus 2000). The place where a muscle's proximal tendon attaches to bone is referred to as the muscle origin, and the distal tendon attachment is referred to as an insertion. Healthy tendons are brilliant white in colour and fibroelastic in texture, and show great resistance to mechanical loads (Kannus 2000).

The tendon is covered by the epitenon, a fine, loose connective tissue sheath containing blood vessels, lymphatics, and nerves. The epitenon extends deeper into the tendon between the tertiary bundles and the endotenon. More superficially, the epitenon is surrounded by the paratenon, a loose areolar connective tissue consisting essentially of type I and type III collagen fibrils (Kvist, et al. 1985).

Collagen fibre orientation

It is well documented that the collagen fibrils are oriented not only longitudinally, but also transversely and horizontally; the longitudinal fibrils also cross each other, thus forming spirals and plaits (Chansky, et al. 1991; Jozsa, et al. 1991) (Fig. 1).

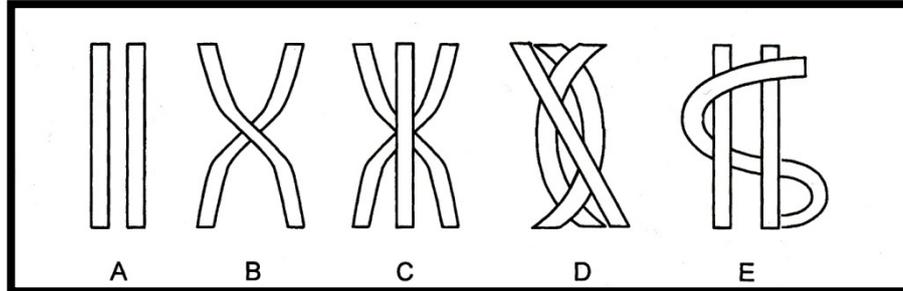


Figure 1. The types of collagen fibre crossing (Jozsa, et al. 1997).

A=parallel running fibres; B=simply crossing fibres; C=crossing of two fibres with one straight-running fibre; D=a plait formation with three fibres; E=up-tying of parallel running fibres.

This complex ultra-structure of the tendons provides good buffer capacity against longitudinal, transversal, horizontal, as well as rotational forces during movement and activity. There is great tendon-to-tendon variation, and within a tendon site-to-site variation, as regards collagen content and type distribution (Fan, et al. 1997).

Internal architecture

The basic elements of a tendon are collagen bundles, cells, and ground substance (a viscous substance rich in proteoglycans and glycosaminoglycans-GAGs). Collagen provides the tendon with tensile strength, whereas ground substance provides structural support for the collagen fibres and regulates the extracellular assembly of procollagen into mature collagen (Åström 1997). These elements are produced by tenoblasts and tenocytes, which are the elongated fibroblasts and fibrocytes that lie between the collagen fibres (Hess, et al. 1989).

Collagen is hierarchically arranged in levels of increasing complexity, beginning with tropocollagen (a triple-helix, polypeptide chain). Soluble tropocollagen molecules form cross-links to create insoluble collagen molecules, which then aggregate progressively into microfibrils and then into electron microscopically clearly visible units, i.e. the collagen fibrils (Kannus 2000). A bunch of collagen fibrils forms a collagen fibre, which is the basic

unit of a tendon. Then fibres (primary bundles), fascicles (secondary bundles), and tertiary bundles finally form the tendon itself (Jozsa, et al. 1997;Åström 1997) (Fig. 2).

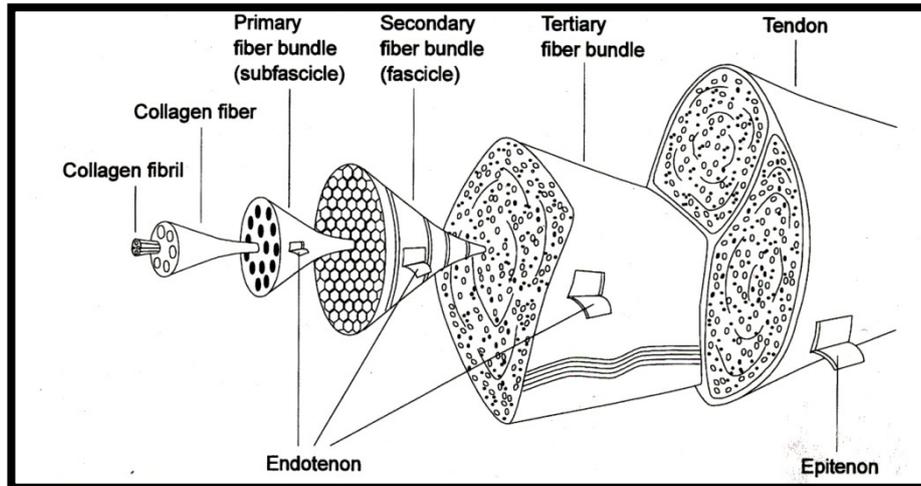


Figure 2. The hierarchial organisation of the tendon structure from collagen fibrils to the entire tendon (Jozsa, et al. 1997).

General innervation

Inside a tendon, the nerves, which are relatively few in numbers, follow the vascular channels that run along the axis of the tendon, and anastomose with each other via obliquely and transversally oriented nerve endings. Most of the nerve fibres are sensory nerve endings on the surface of the tendon (Jozsa, et al. 1997).

Based on anatomical and functional differences, nerve endings in tendons, ligaments, and joint capsules can be classified into four categories: type I Ruffini corpuscles (pressure receptors that are sensitive to stretch); type II Vater-Pacini corpuscles (activated by movement); type III Golgi tendon organs (mechanoreceptors); and type IV receptors (free nerve endings functioning as pain receptors) (Jozsa, et al. 1993).

General biomechanical forces in tendons

Tendons have a great capacity to withstand tensile and stretching forces, but are less tolerant to shearing and compressive forces transmitted by the muscles (Hess, et al. 1989). The stress-strain curve facilitates the understanding of the behaviour of the tendon during tensile load (Fig. 3).

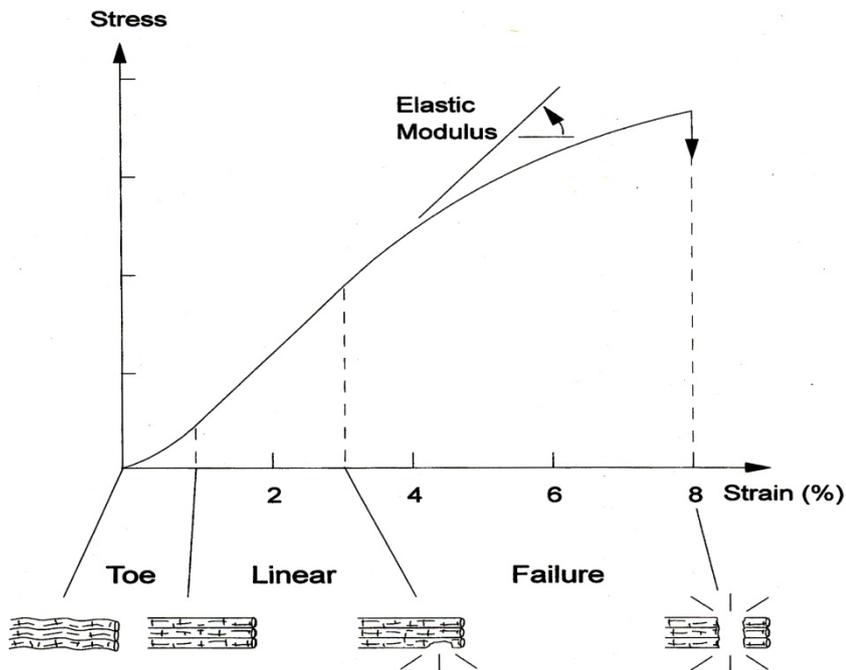


Figure 3. A schematic presentation of the stress strain curve (Jozsa, et al. 1997).

At rest, the tendon has a crimped or wavelike structure. When the tendon is stretched, the tendon progresses through three regions (toe, linear and partial failure). The first region is referred to as the toe region, and as the tendon is progressively loaded the fibres begin to straighten until reaching a 2% strain. This is the first part of the stress-strain curve, occurring due to the elastic properties of collagen. Beyond this point, tendons deform in a linear fashion (linear region) as a result of intramolecular sliding of collagen triple helices, and fibres become more parallel (O'Brien 1992). If strain does not exceed 4%, the tendon will return to its original length when unloaded (Loitz, et al. 1989). Microscopic failure of fibres occurs when the strain exceeds 4–8%. However, recently, strain values of up to 6–8% was said to be physiological (Magnusson, et al. 2003). The last region of the curve illustrates macroscopic tensile and partial failure of collagen fibres. This results in a complete tendon rupture when the strain has reached 8–10% of

its original length (O'Brien 1992). A healthy tendon is strong, its tensile strength being related to thickness and collagen content. A tendon with an area of 1 cm² is capable of resisting a weight of 500–1,000 kg (Jozsa, et al. 1997).

Metabolism

It is well established that tendon cells are metabolically active, both in energy production and in the biosynthesis of collagen and matrix components. Energy is derived mainly from three different sources: the Krebs cycle, the anaerobic glycolysis, and the pentose phosphate shunt. Over a life span, the metabolic pathway changes towards anaerobic glycolysis (Hess, et al. 1989; Kannus, et al. 1991; O'Brien 1997). Tendons have approximately eight times lower oxygen consumption than skeletal muscle. The low oxygen consumption makes it possible for a tendon to carry loads and remain tensioned without risk of ischemia and injury.

One negative aspect of the low metabolic rate in tendons is the slow recovery after activity, and healing after injury (Williams 1986).

Disuse/immobilisation

Few studies have investigated the effects of immobilisation on collagen synthesis rate in humans. One study showed decreased collagen synthesis in the patellar tendon after 10–21 days of immobilisation. However, there were no changes in the tendon cross-sectional area (CSA) (de Boer, et al. 2007). In a study on Achilles tendons, no difference in Achilles CSA was found following two weeks of immobilisation. The conclusion drawn was that the Achilles tendon seems to be more resistant to short-term immobilisation than muscle tissue (Christensen, et al. 2008). Animal studies have shown decreased stiffness, GAGs and water content after immobilisation (Woo, et al. 1982; Barnard, et al. 1987; Loitz, et al. 1989; Karpakka, et al. 1990).

Exercise/remobilisation

Compared to muscle tissue, the metabolic turnover in tendon tissue is slower due to poor circulation (Kannus, et al. 1997a). However, a study by Langberg et al. found that exercise increased the formation of collagen type I in the peritendinous tissue (Langberg, et al. 1999). The same research group later demonstrated increased collagen turnover (both synthesis and degradation) after four weeks of training (Langberg, et al. 2001), and they also presented a study with increased collagen synthesis rate after 12 weeks of eccentric training (Langberg, et al. 2007). Increasing age and disuse leads to a decrease in tendon stiffness, which in turn can be mitigated by resistance

exercise (Magnusson, et al. 2008). Physical activity seems to improve the tensile mechanical properties of tendons, in contrast to disuse. Effects like increased GAGs content, and better alignment of collagen fibres have been shown in animal studies (Woo, et al. 1982;Kellett 1986;Barnard, et al. 1987;Loitz, et al. 1989;Karpakka, et al. 1990).

The Achilles tendon

Anatomy

The triceps surae has two muscle bellies, the gastrocnemius and the soleus muscles. These are the strongest muscles of the calf, and they merge to form the Achilles tendon (O'Brien 1984) (Fig. 4). The Achilles tendon is the thickest and strongest tendon of the human body (O'Brien 1992). According to O'Brien, the CSA of the tendon is 0.8–1.4 cm². The gastrocnemius tendon begins as a broad aponeurosis at the distal margin of the muscle bellies, whereas the soleus tendon begins as a band proximal to the posterior surface of the soleus muscle (O'Brien 1992). The tendon becomes narrower and more rounded distally. The tendon length of the gastrocnemius tendon is 11–26 cm, and the length of the soleus tendon portion is 3–11 cm. As the tendon descends, it may spiral up to 90° laterally, so that fibres that were originally posterior become lateral, lateral fibres become anterior, anterior fibres become medial, and medial fibres become posterior at the distal end (Jozsa, et al. 1997). In this way, elongation and elastic recoil within the tendon is possible, and stored energy can be released during locomotion (Alexander 1977). Another important factor of the rotation is that a region of concentrated stress may be produced where the two tendons meet. This is most prominent at 2–5 cm proximal to the calcaneus insertion, and corresponds well with the region of the tendon that according to some authors has the poorest vascular supply (Curwin 1984; Reynolds, et al. 1991).

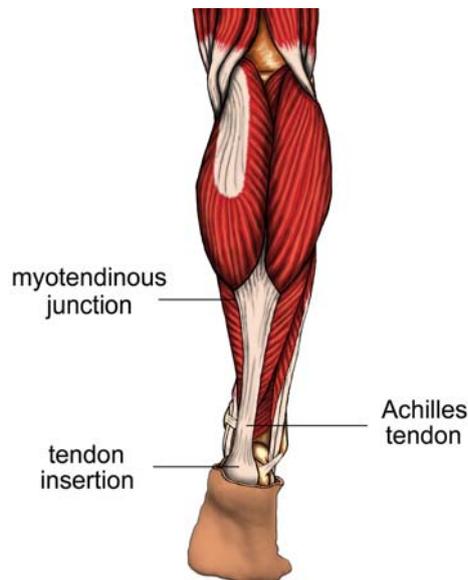


Figure 4. The Achilles tendon

The myotendinous junction (MTJ)

The MTJ is a specialised anatomic region in the muscle-tendon unit. Morphological studies have shown that at the MTJ, the collagen fibrils insert into the deep recesses that are formed between the finger-like processes of the muscle cells (Kvist, et al. 1991) (Fig. 5). This structure and composition increase the contact area between muscle fibres and tendon collagen fibres 10- to 20-fold (Tidball 1991). Although the MTJ can sustain high forces, it still remains the weakest structure of the muscle-tendon unit (Garrett 1990; Järvinen 1991).

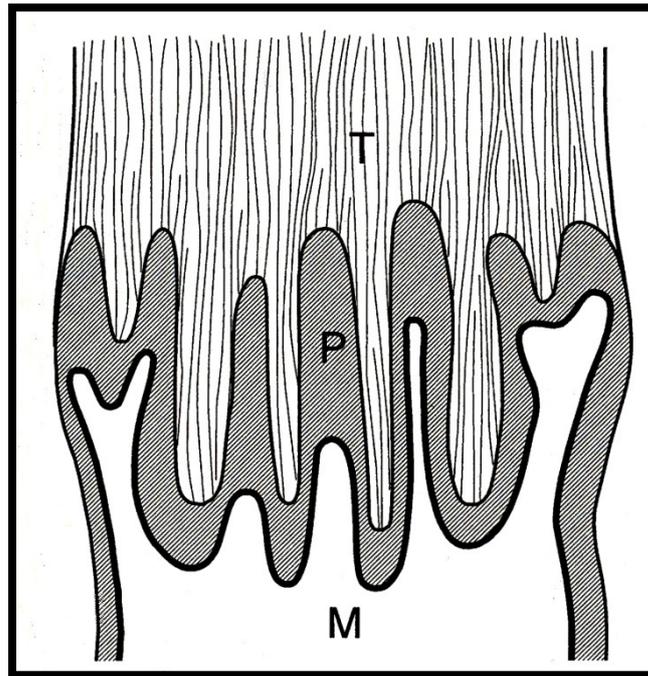


Figure 5. Myotendinous junction

T=Tendinous collagen fibrils; M=Muscle cell; P=Muscle cell processus.

The osteotendinous junction (OTJ)

The OTJ consists of tendon, fibrocartilage, and bone (Milz, et al. 2002). The insertion of tendons into bone involves a gradual transition from tendon to fibrocartilage, to lamellar bone. The tissue changes from soft to hard. The tendon attachment to bone consists of four zones: pure fibrous tissue, unmineralised fibrocartilage, mineralised fibrocartilage and bone (Åström, et al. 1995).

The enthesis organ concept was presented by Benjamin and McGonagle, who defined the enthesis organ as a collection of related tissues at and near the enthesis, which serve a common function of stress dissipation (Benjamin, et al. 2001). It is widely applicable at different entheses and is most clearly recognised where the Achilles tendon attaches to the calcaneus. Immediately above the Achilles insertion on the posterior surface of the calcaneus, in the space between the tendon and bone, the retrocalcaneal bursa is located. There is also a subcutaneous calcaneal bursa, between the skin and tendon. The bursae both decrease the friction and compression on the tendon (Rufai, et al. 1995). The concept of an enthesis organ is of particular significance for clinicians, as it can help explain the injury pattern and why symptoms associated with a particular enthesopathy are diffuse (Benjamin, et al. 2006).

Tendon structure

Unlike other tendons around the ankle (tendons with a synovial sheath), the Achilles tendon is enveloped by a paratenon, which is a membrane that consists of two layers: a deeper layer surrounding, and in direct contact with, the epitenon, and a superficial layer, the peritenon (Kvist, et al. 1980).

There are specific variations in proteoglycan content within the Achilles tendon. At the insertion site there is an increased amount of aggrecan, which protects the enthesis from compressive and shearing forces. In the mid-portion of the Achilles tendon there is more versican, which provides the tendon with tensile strength (Waggett, et al. 1998).

Circulation

The blood supply to the Achilles tendon comes mainly from the muscle and is usually divided into three regions: the musculotendinous junction, the length of the tendon, and the tendon bone junction. The blood vessels originate in vessels in the perimysium and periosteum, and run via the paratenon and mesotenon. The main blood supply to the middle portion of the tendon takes place through the paratenon (O'Brien 1997).

Innervation

The nerve supply to the Achilles tendon originates mainly from the sural nerve, via nerve fascicles that occur subcutaneously. The innervation within the Achilles tendon has been sparsely studied. However, recently, Bjur et al. defined the innervation pattern, and showed a general (PGP9.5), a sensory (SP/CGRP), and an autonomic nervous system in the Achilles tendon (Bjur, et al. 2005).

Biomechanics

It is difficult to determine the tendon tensile forces in vivo (Komi, et al. 1992; O'Brien 1992). Komi and co-workers used buckle transducers in the tendon, and studied a wide range of activities such as walking, running, jumping, and bicycling (Komi, et al. 1987; Komi 1990). The peak Achilles tendon force during running at 6 m/sec was estimated to 9 kilo newton (kN), corresponding to 12.5 times the bodyweight, (11.0 kN/cm²). Other studies show that an athlete can generate forces in the Achilles tendon during jumping and running activity of 6–14 times the bodyweight (Kader, et al. 2002; Paavola, et al. 2002).

Achilles tendinopathy

Definitions

The terminology relating to the chronic painful conditions in the Achilles tendon is somewhat confusing. Different terms have been used in the past to describe the conditions. Terms such as **Achilles tendinitis** and **tendonitis** have been widely used, assuming that there is an inflammation within the tendon. However, histopathological, biochemical, and molecular studies have shown an absence of a true prostaglandin-mediated inflammatory process inside the chronic painful tendon (Kannus, et al. 1991; Åström, et al. 1995; Alfredson, et al. 1999; Alfredson, et al. 2003a). Puddu et al. proposed the term **tendinosis** as a histological description of degenerative pathology with an absence of inflammatory changes (Puddu, et al. 1976). The term **achilodynia** has been used by Åström, as a symptomatic diagnosis (Åström 1997). The authors recommend that the terms **tendinosis** (tendon degeneration) and **peritendinitis** are reserved for conditions where the pathology has been verified by surgical exploration, imaging, histological analysis of biopsies, or a combination (Åström 1997). Maffulli et al. suggest that the combination of tendon pain, swelling, and impaired performance should be clinically labelled as **tendinopathy** (Maffulli, et al. 1998). This has been widely supported in the last decade (Khan, et al. 2002). The terms **insertional** and non **insertional Achilles tendinopathy** were proposed by Clain et al. (Clain, et al. 1992). Today, **tendinopathy** is used to describe a condition with tendon pain, swelling, and impaired function, and **tendinosis** is used when the pathology is objectively verified by ultrasound (US), magnetic resonance imaging (MRI) or examination of biopsies.

Achilles tendon injuries can be acute or chronic. The term chronic is widely used when symptoms persist for longer than three months.

Epidemiology

Tendinopathy is often seen in individuals in the age group of 30–60 years (Kvist 1991a). They may participate in middle- or long-distance running, badminton, or track and field activities (Kvist 1991b;Fahlström, et al. 2002). However, inactive individuals can also suffer from mid-portion Achilles tendinopathy (Kvist 1991a, 1994). In elite runners, the incidence of tendinopathy has been reported to be 7–9% (Lysholm, et al. 1987). Kvist showed that 66% of 698 competitive and recreationally active patients suffered from Achilles tendinopathy (Kvist 1991b). In badminton players, Achilles tendinopathy accounts for 10.5% of all overuse injuries (Jørgensen, et al. 1990).

The incidence of insertional Achilles tendinopathy has not been well established. In a surgical and histopathological survey of 163 patients with chronic Achilles tendinopathy, the prevalence of insertional tendinopathy was 20% (Åström, et al. 1995). Insertional tendinopathy is often diagnosed in older, non-active, and overweight individuals (Meyerson 1999).

Aetiology

The aetiology to Achilles tendinopathy is still unclear. Many different theories have been presented (Kvist 1991b;Åström 1997). The aetiology is believed to be multifactorial, involving intrinsic and extrinsic risk factors. In acute tendon injuries, extrinsic risk factors are predominating, and in chronic Achilles tendinopathy a combination of intrinsic and extrinsic factors is often seen (Kannus, et al. 1997b;Khan, et al. 1998). It should be stressed that scientific evidence of the role of intrinsic and extrinsic factors is still lacking.

Intrinsic risk factors

Age is related to the development of tendinopathy in the Achilles tendon, and an old tendon is more vulnerable than a young one (Kannus, et al. 1991;Kvist 1991a).

Anatomical factors like leg-length discrepancy and malalignment (genu valgum, forefoot varus), have by some authors been suggested as causative in the development of Achilles tendinopathy (Kvist 1991b, 1994). However, this is controversial and has been questioned by other authors (Kannus, et al. 1997b).

Decreased joint flexibility of the ankle will increase ground reaction force during landing. This finding has been linked to Achilles tendinopathy (Kaufman, et al. 1999).

Muscle weakness/imbalance of the gastrocnemius muscles could disturb and change the coordinated movements of the kinetic chain through the hip, knee, and ankle. This is a common finding in tendinopathy. However, it is unclear whether this is the cause to, or a result of, tendinopathy (Kountouris, et al. 2007).

High bodyweight and adipose tissue levels seem to be associated with tendinopathies in the lower limb (Gaida, et al. 2008).

Gender. Some studies on women indicate that oestrogen protects from tendinopathy (Cook, et al. 2007).

Genetics. There are reports showing a correlation between the incidence of Achilles tendinopathy and blood group o (Jozsa, et al. 1989). Furthermore, an association between the alpha 1 type V collagen (COL5A1) and Achilles tendinopathy has been found (Collins 2003).

Systemic diseases like Marfan's and Ehlers-Danlos Syndrome, and rheumatoid arthritis are well known to be associated with defects of the collagen metabolism, causing weakness of the tendons (Jozsa, et al. 1997).

Extrinsic risk factors

Training errors have been reported to be common among runners that have Achilles tendinopathy (Kvist 1994). Training errors were identified as primary aetiological factors in over 75% of the cases (Clement, et al. 1984).

Poor technique, improper footwear, and running on hard, slippery or uneven surfaces could be predisposing risk factors for Achilles tendinopathy (James, et al. 1978).

Overuse is believed to be the major cause to tendinopathy (Jozsa, et al. 1997). "Too much too soon" was stated by Brody (Brody 1987). However, it is not clear if overuse is solely responsible. In a study of 58 patients with Achilles tendinopathy, 31% were not active in sports or vigorous physical activity (Rolf, et al. 1997). Around 50% of patients presenting with mid-portion Achilles tendinosis at the Sports Medicine Unit in Umeå, Sweden are not active in any sports or recreational activity (Alfredson, personal communication 2009).

Underuse. As with tendinopathy in general, overuse and poor training habits are suggested to be the main aetiological causes of insertional Achilles tendinopathy (Benjamin, et al. 2000). However, a new idea concerning the aetiology of insertional Achilles tendinopathy has been presented (Almekinders, et al. 2003;Maganaris, et al. 2004). When there is a lack of tensile load on the ventral side of the Achilles tendon, there is a tendency to develop cartilage-like or atrophic changes on the stress-shielded side of the enthesis (Benjamin, et al. 1986;Rufai, et al. 1995). This may lead to disturbed remodelling and primary degenerative lesions in that area. Since tendinopathy is not always related to high loading activity, but is also age-related, the suggestion is that insertional tendinopathy results from underuse and stress-shielding, rather than overuse.

Pathogenesis

The pathogenesis in the chronic painful Achilles tendon is unknown, although several theories have been presented. Three theories dominate the discussion. The primary inflammatory theory, where inflammation leads to a degeneration, with at least six different states of collagen degeneration (hypoxic, mucoid, hyaline, fibrocartilaginous metaplasia, tendon calcification, and lipoid). Collagen degeneration is suggested to be irreversible, and is seen as an end-stage of the pathology (Jozsa, et al. 1997). The mechanical theory claiming that repeated load-bearing within the normal physiological stress range of a tendon causes fatigue, and eventually leads to tendon failure. Repeated and/or prolonged stress at higher levels of strain could also lead to microscopic degeneration within the tendon. Later the mechanical properties of the tendon can be altered and lead to a symptomatic tendon due to micro trauma. Some authors have suggested that the pathology is a state of failed healing, where the injured tendon is in a healing phase, including active cells, increased protein production, disorganisation of matrix, and neovascularisation (Clancy 1989;Khan, et al. 2002). The vascular theory, suggesting that as tendons are more metabolically active than previously believed and require a vascular supply, the compromise of such a vascular supply may cause degeneration. Some tendons like the Achilles tendon and the supraspinatus tendon are at higher risk than others. It has been suggested that there is a hypovascular region in the mid-portion of the Achilles tendon 2–6 cm proximal to the calcaneal insertion (Carr, et al. 1989), which makes this part of the tendon more prone to injuries. This theory has been questioned by Åström and Westlin, who showed a uniform blood flow in the Achilles tendon, except at the insertion where the flow was lower (Åström, et al. 1994). A recently published study supports the theory of a hypovascular weak area in the mid-portion (Chen, et al. 2009)

Histology

Histological evaluation of the pathological Achilles tendon has demonstrated that there is no evidence of an ongoing PGE₂ mediated inflammatory process inside the tendon. However, there could be a neurogenic inflammation (Hart, et al. 1998; Alfredson, et al. 1999; Alfredson, et al. 2003a). The histological findings in tendinopathy show four distinct structural changes within the tendon: increased cellularity, increased production of ground substance, (especially GAGs), separation of collagen bundles, and neovascularisation (Jozsa, et al. 1990). There are certain differences in the morphology of mid-portion Achilles tendinopathy and insertional Achilles tendinopathy. In the Achilles insertion there are several structures, e.g. the superficial and retrocalcaneal bursa, Kager's fat pad, and sometimes a prominent upper calcaneus Haglund's deformity, that all alone or in combination could give rise to symptoms in this area. The retrocalcaneal bursa and Kager's fatpad has an important function to promote free movement between tendon and bone. It is not uncommon with an inflammation in the retrocalcaneal bursa due to increased compression between tendon and bone (Canoso, et al. 1988; Theobald, et al. 2006). According to Shaw et al., the bursa contains sensory nerve endings, and may have a proprioceptive function in monitoring insertional angle changes between bone and tendon during foot movements (Shaw, et al. 2007).

Pain mechanisms

New methods like intratendinous microdialysis and molecular biology techniques have shown that there is no inflammation inside the chronic painful Achilles tendon (Alfredson, et al. 2001; Alfredson, et al. 2003a). These new techniques together with ultrasound (US) in combination with colour Doppler (CD), and immunohistochemical analyses of tendon tissue biopsies, have facilitated a better understanding of the pain mechanisms. By using US with CD, Öhberg et al. showed that neovascularisation is present in pathological and symptomatic, but not in normal pain-free Achilles tendons (Öhberg, et al. 2004a). Neovascularisation and accompanying nerves might be important factors related to the pain mechanisms of tendinopathies (Alfredson, et al. 2003b). It is worth noting that not all tendons with neovascularisation are painful (Zanetti, et al. 2003). Bjur et al. found nerves linked to the vascular structures in tendinosis tendons (Bjur, et al. 2005). Furthermore, the neurokinin-1-receptor, associated with the neuropeptide substance P (SP), has been found in the vascular wall (Forsgren, et al. 2005). Follow-up studies of patients with Achilles tendinopathy who have become pain-free after treatment with eccentric training have shown an absence of neovessels (Öhberg, et al. 2004b).

Clinical symptoms

Patients with chronic Achilles tendinopathy most commonly have a history of a gradual onset of tendon pain, often related to a change in activity level. However, as stated previously, non-active individuals may also suffer from Achilles tendinopathy. Initially, the patients often experience stiffness, pain or discomfort at the beginning of activity, followed by less pain during activity, and a return of the stiffness and pain afterwards (Rogers 1996). Later on, pain increases during activity, and patients have to stop the activity. The patients often complain about stiffness in the morning, and together with pain during activity, this is a characteristic of chronic Achilles tendinopathy. It is suggested that the amount of morning pain and stiffness a patient suffers serves as a good indicator of the tendon condition, the more symptoms, the poorer stage of the tendon condition (Cook, et al. 2002).

Clinical examination

Examination should include the biomechanics of the lower extremity, i.e. leg, ankle, and foot, during loading activity. Range of motion in the ankle joint should also be noted. Inspection and palpation of the surrounding structures must be included in a clinical examination, and tenderness and thickening of the tendon should be noted. The examiner should be gentle during palpation of the tendon, as there may be some pain during palpation even if there is no injury (Cook, et al. 2001).

Differential diagnoses

It is important to exclude total or partial ruptures (Maffulli, et al. 1998). If plantar flexion tonus is poor or nonexistent, a rupture should be suspected. Tonus of the muscle-tendon unit should be examined with the patient under resting conditions in prone position. Partial ruptures are difficult to diagnose clinically, and therefore the patient's history is important in these cases. Tenosynovitis of the medial flexor tendons, and dislocation of the lateral peroneal tendons, also needs to be excluded. Other possible differential diagnoses are os trigonum syndrome, tumours of the Achilles tendon (xanthomas), neuroma of the sural nerve, and an accessory soleus muscle (Cook, et al. 2002; Alfredson 2005; Alfredson, et al. 2007). Muscle strains in the MTJ are not uncommon (Kvist 1991a). Partial muscle ruptures are most common in the medial muscle belly, commonly referred to as tennis leg (Millar 1979). At the insertion of the Achilles tendon, an inflamed superficial and/or retrocalcaneal bursae could be co-existing as well as a Haglund deformity of the upper calcaneus (Vega 1984).

Treatment

The purpose of the treatment of patients suffering from Achilles tendinopathy is to decrease pain and improve physical activity, thereby making it possible for the patient to continue to participate in physical activities. During the last decade, several different treatment options have been presented. Unfortunately, many of these treatments lack scientific evidence.

Rest are often recommended, however rest is detrimental to the tendon causing weakness and impaired tendon properties (Kannus, et al. 1997a)

NSAIDs have not been shown to be efficient in the treatment of Achilles tendinopathy (Åström 1992). Interestingly, a study showed that NSAIDs blocked protein synthesis in skeletal muscles after exercise (Trappe, et al. 2002).

Corticosteroid injections have shown promising short-term pain relief results, but poor long-term results (Kleinman, et al. 1983; Jones 1985; Gill, et al. 2004). Also, there are reports of tendon ruptures following corticosteroid injections (Kleinman, et al. 1983; Jones 1985).

Extracorporeal shockwave therapy (ESWT). In patients with mid-portion Achilles tendinopathy, Costa et al. did not find clinical improvement after ESWT treatment (Costa, et al. 2005). These results were supported by Rasmussen et al., who for some reason suggested that treatment with ESWT anyhow should be used as a supplementary treatment (Rasmussen, et al. 2008). Rompe et al. demonstrated more pain reduction with ESWT compared to the wait-and-see approach (Rompe, et al. 2007), but no benefit compared to eccentric exercises (traditional method used by Alfredson et al) (Alfredson, et al. 1998). The same authors compared eccentric exercise (as above) with ESWT in patients with insertional Achilles tendinopathy, and eccentric exercise showed inferior results compared to the group receiving ESWT (Rompe, et al. 2008).

Low-level laser treatment in patients with mid-portion Achilles tendinopathy have shown better results in combination with eccentric exercises than alone (Stergioulas, et al. 2008).

Ultrasound is used to treat Achilles tendon pain, but there is no evidence of beneficial effects in controlled trials (Robertson, et al. 2001).

Heel pads were evaluated by Lowdon et al. who found them ineffective. This study did not separate insertional tendinopathy from mid-portion tendinopathy, and the heel-pads were not custom-fit (Lowdon, et al. 1984).

Topical glyceryl trinitrate patches treatment showed promising results in a randomised double-blind study on patients with mid-portion Achilles tendinopathy. One negative side effect was that 53% of patients reported severe headaches during the treatment period (Paoloni, et al. 2004).

Strength training. It appears that exercise is a positive stimulus to the collagen alignment (Kannus, et al. 1997a). Eccentric contraction seems to be more beneficial than concentric activation (Mafi, et al. 2001). In the mid 1980s, Curwin and Stanish presented the first eccentric exercise intervention program for tendinopathy, using a program with progressively increased moderate eccentric load, and changes in speed, where the patient's symptoms controlled the progression of load (Curwin 1984). The exercises were performed without tendon pain. More than a decade later, in a pilot study, Alfredson et al. used painful eccentric heel drops on a step as treatment for patients with mid-portion Achilles tendinopathy (Alfredson, et al. 1998). This study reported excellent results with a significant decrease in pain and a return to previous activity level after 12 weeks of training. There were some main differences between Curwin's and Alfredson's protocols (Curwin 1984; Alfredson, et al. 1998). Alfredson used painful training, heavier loads, and single leg exercises. The Alfredson protocol has been widely used, and the good clinical results have been reproduced by other groups (Roos, et al. 2004; de Vos, et al. 2007; Rompe, et al. 2007).

Sclerosing injections of polidocanol outside of the tendon, where the neovessels enter the tendon, have shown promising clinical results, including decreased pain and improved physical activity (Alfredson, et al. 2005b).

Traditional surgical treatment has consisted of a longitudinal tendon incision and excision of abnormal tendon tissue, followed by a period of immobilisation. A critical review by Tallon et al. on the outcome of surgery for chronic Achilles tendinopathy, found successful results in 70% of the cases. However, clinical results were better in studies with a poor scientific design, and less good in studies with a good scientific design (Tallon, et al. 2001). In the past, patients suffering from pain in the insertion of the Achilles tendon have generally been treated with surgery when conservative (non-operative) treatment has failed. However, the clinical outcome following surgery has shown great variations (35–59%) as regards the return to unlimited activity (Maffulli, et al. 1999; McGarvey, et al. 2002).

The patellar tendon

Anatomy

The patellar tendon is an elongation of the quadriceps tendon, which is the tendon of the quadriceps muscle. The patellar tendon runs from the patellar bone to the tendon insertion into the tuberositas tibiae (Fig. 6). The quadriceps muscle is the largest muscle in the human body and is composed of four muscle bellies. M. rectus femoris originates from spina iliaca anterior inferior and inserts via the quadriceps tendon and patellar tendon into the tuberositas tibiae. The vastus muscles (M. vastus medialis, M. vastus lateralis and M. vastus intermedius) originate in different locations on the femur, and insert at the patella and the patellar tendon at the tuberositas tibiae. The quadriceps muscle is the strongest extensor muscle in the knee joint. Besides extension in the knee joint, it assists in the hip flexion via M. rectus femoris. According to Peers et al. the width of the patellar tendon is approximately 30 mm (in the frontal plane) and the thickness is 4–5 mm (in the sagittal plane) (Peers, et al. 2005). During its course, there are variations in the width of the tendon from 31.9 mm at its attachment to the apex of patella and 27.4 mm at its attachment to the tibial tubercle (Andrikoula, et al. 2006).

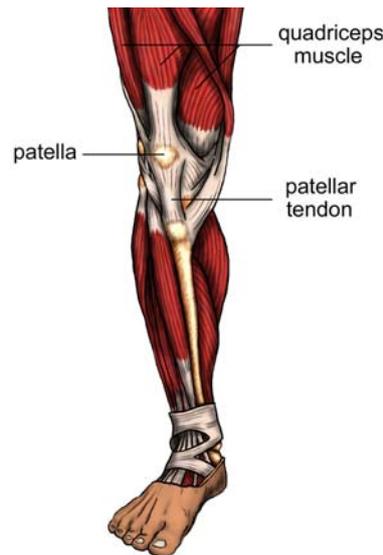


Figure 6. The patellar tendon

Tendon structure

The structure of the patellar tendon is described previously (page 12).

Circulation

The arterial supply of the human patellar tendon was systematised by Andrikoula et al. Three arterial pedicles were observed: superior, middle, and inferior, placed on each side of the patellar tendon. Medial pedicles originate in the descending and inferior medial genicular arteries. The lateral pedicles originate in the lateral genicular arteries and the recurrent anterior tibial artery. Two main vascular arches anastomose these pedicles: the retropatellar and the supratubercular (Andrikoula, et al. 2006).

Innervation

Recently, the nervous system in the patellar tendon was clarified by Danielson et al. (Danielson, et al. 2006a). In his thesis, Danielson presented that the loose paratendinous connective tissue of the patellar tendon was richly innervated by nerve structures. The paratendinous tissue was more innervated than the tendon tissue. Parts of the nerve structures were related to sensory afferents and parts to cholinergic and, especially, sympathetic nerve fibres. There are limited innervations within the patellar tendon. However, recently the tenocytes themselves were shown to produce signal substances, normally associated with neurons (Danielson 2007).

Biomechanics

The patellar tendon is able to transmit high muscle forces to the bone during movement. Particularly activities like running and jumping can put the tendon under high stress. The parallel fibre arrangement allows the tendon to tolerate high strains, but the tendon is not as good at tolerating shearing forces (Kirkendall, et al. 1997). The highest load on tendons occurs during eccentric muscle activation, up to three times higher than during concentric activation (Komi 1984;Fyfe, et al. 1992). It has been estimated that the forces within the patellar tendon can reach 0.5 kN during level walking, 8.0 kN when landing from a jump, up to 9.0 kN during fast running, and 14.5 kN (17 times the bodyweight) during competitive weight lifting (Zernicke, et al. 1977).

Patellar tendinopathy

Definitions

Overuse of the patellar tendon has historically been called ***jumper's knee*** or ***patellar tendinitis***. However, these terms are misnomers. The condition is found in many patients who do not participate in jumping sports (Khan, et al. 1996;Cook, et al. 1997;Lian, et al. 2005), and histopathological studies have consistently shown the underlying pathology to be degenerative (tendinosis) rather than inflammatory (tendinitis) (Khan, et al. 1996;Alfredson, et al. 2001;Maffulli, et al. 2004). Nowadays it is commonly agreed that the term ***patellar tendinopathy*** should be used for chronic pain symptoms in a tender area of the tendon (Maffulli, et al. 1998), and combination of chronic pain in a tender part of the tendon and US, MRI or biopsy evidence of corresponding changes in the tendon is commonly called ***patellar tendinosis*** (Movin, et al. 1997;Khan, et al. 1999).

Epidemiology

Patellar tendinopathy is most commonly seen in sports with high demands on speed and power from the leg extensors (Lian, et al. 2005). The highest prevalence (40–50%) has been reported among male volleyball players (Ferretti, et al. 1983;Ferretti, et al. 1990;Lian, et al. 1996). Other sports where the condition is seen include basketball, soccer, football, and track and field (Blazina, et al. 1973). Overuse of the patellar tendon is also common among army recruits, constituting 15% of all soft-tissue injuries (Linenger, et al. 1992). In one of the few epidemiological studies on elite athletes in different sports, the overall prevalence of patellar tendinopathy was reported to be 14%. The prevalence was lower in women (5.6%) compared to men (13.5%) (Lian, et al. 2005). Other clinical studies have also shown that the condition is more common in men (Myllymäki, et al. 1990). In junior basketball players the prevalence was shown to be 7% (Cook, et al. 2000a), and in Swedish elite junior volleyball players the prevalence was 11% (Gisslen, et al. 2005b). The long-term prognosis for male athletes has been shown to be poor, and 53% of athletes abandoned their sports career due to pain symptoms (Kettunen, et al. 2002).

Aetiology

The aetiology to patellar tendinopathy is unclear. The condition is associated with a variety of both intrinsic and extrinsic risk factors, and some individuals seem more prone to develop tendinopathy than others, despite participating at similar activity levels. Also, it is more common among

younger individuals, in contrast to Achilles tendinopathy, that often is seen in the middle-aged group (Lian, et al. 2005;Magra, et al. 2008).

Intrinsic risk factors

Poor flexibility in the ankle joint increases the ground reaction forces during landing, and is associated with patellar tendinopathy (Malliaras 2000). Further, poor flexibility of the hamstrings and quadriceps muscles is related to the development of patellar tendinopathy (Cook, et al. 2004a).

Weakness/imbalance of the quadriceps and gastrocnemius muscles could have a role in the development of tendinopathy according to (Gleim, et al. 1997), however, Witvrouw et al. didn't find any relationship between weakness in the thigh muscles and patellar tendinopathy (Witvrouw, et al. 2001).

Gender. There are a few studies that show that men are more prone to sustain patellar tendinopathy than women (Cook, et al. 1998;Lian, et al. 2005). This has been challenged by Witvrouw et al. who found no significant gender-based differences among patients with patellar tendinopathy (Witvrouw, et al. 2001).

Bodyweight. A few studies have highlighted high body mass as an important intrinsic risk factor for the development of patellar tendinopathy (Gaida, et al. 2004). A high waist-to-hip ratio (WHR) has been found in volleyball players with patellar tendinopathy (Malliaras, et al. 2007;Gaida, et al. 2008).

Extrinsic risk factors

Training errors such as increased frequency and duration of training sessions have been shown to be associated with patellar tendinopathy (Ferretti 1986;Gisslen, et al. 2007).

Overuse due to repetitive tendon loading during running and jumping is a strong aetiological factor associated with patellar tendinopathy (Jozsa, et al. 1997). Reports have shown that volleyball players who participated in more than three training sessions per week were more prone to sustain patellar tendinopathy than those who participated in less than three sessions per week (Ferretti 1984). This was supported by Gaida et al. who found a correlation between training hours per week and the development of patellar tendinopathy in volleyball players (Gaida, et al. 2004).

Underuse. Almekinders et al. investigated the strain in the inferior pole of the patellar tendon during range of motion while the tendon was loaded (Almekinders, et al. 2002). The authors reported that the highest tensile strain was distal to the inferior pole of the patella on the ventral side of the tendon. The lowest tensile strain was on the dorsal side of the tendon. Tendinopathy is commonly found on the dorsal side of the proximal tendon. Investigating the strain in the whole length of the tendon, Basso et al. found that the highest strain was at the dorsal aspect of the tendon (Basso, et al. 2002). However, this may not reflect what actually occurs at the insertion into the tip of the patella. It seems that the strain within the tendon is not uniform, and that there could be an alternative biomechanical explanation for the pathology found at the enthesis (Maganaris, et al. 2004).

Pathogenesis

Patellar tendinopathy is believed to be a degenerative condition caused by high loads on the patellar tendon (Cook, et al. 2000a). As in Achilles tendinopathy, three major causative theories have been suggested, i.e. a mechanical, a vascular, and a failed healing theory (Curwin 1998;Cook, et al. 2002;Fenwick, et al. 2002). Hamilton and Purdam have presented a new theory, where the authors proposed a histological adaption of the proximal patellar tendon, due to compressive loads, possibly leading to a weakness of the tensile properties of the dorsal aspects of the tendon. The remaining tensile properties of the tendon and surrounding tissue might be overloaded and nociceptors activated (Hamilton, et al. 2004). An impingement theory was presented by Johnson et al. suggesting that impingement between the inferior pole of the patella and the tendon occurs during knee flexion (Johnson, et al. 1996). This theory has been criticised by Schmid et al. who found that during knee flexion in both asymptomatic and symptomatic individuals, the angle between the patella and the patellar tendon was decreased. They concluded that the patellar tendon could not be impinged (Schmid, et al. 2002).

Histology

The histology in the chronic painful patellar tendon is the same as for the chronic painful Achilles tendon described previously (page 24).

Pain mechanisms

The source of pain in patellar tendinopathy is still unknown. Although histopathology is present within the tendon, some tendons are painful and some are not (Cook, et al. 1998;Cook, et al. 2000b). Recent research has shown that neovascularisation is present in painful patellar tendinopathy,

but rarely in the normal tendon. This has given new insight into the source of pain (Gisslen, et al. 2005a). More pain and deteriorated function is closely related to the presence of neovascularisation; this has been shown in a study using US and CD (Cook, et al. 2004b). Substance P (SP) positive nerve fibres, seen as free nerve endings, have been observed interspersed between collagen fibres in chronic painful patellar tendons in athletes with pain symptoms indicating patellar tendinopathy (Lian, et al. 2006). Recently, acetylcholine (ACh) and catecholamines, which are ordinarily found in the nervous system, were found to be produced by tendon cells in tendons with tendinopathy (Danielson, et al. 2006b). This highlights the fact that the tendon cells themselves could have a key role in the production of pain substances. This possibly supports the 'biochemical' hypothesis presented by Khan et al. (Khan, et al. 2000).

Clinical symptoms

The clinical diagnosis of patellar tendinopathy is based on the patients subjective reports of pain related to activity. The pain is often localised in the tendon insertion into the inferior pole of the patella, but pain can also be located at the tibial attachment of the tendon (Blazina, et al. 1973). Symptoms often start gradually, and relate to changes in sports activity (e.g. duration, intensity or frequency). Most often, patients complain of pain after strenuous activity, leading to impaired performance. In the early stages, patients only experience pain after activity. As the symptoms progress, patients may also report pain before and during sport activity. In severe cases, the patient may complain of pain during daily activity (e.g. ascending and descending stairs) (Cook, et al. 1998; Cook, et al. 2000b; Peers, et al. 2005).

Clinical examination

The medical history and clinical examination of patients with patellar tendon injuries are in most cases straight forward. The objective diagnostic tests for tendinopathy may however not be as valid and reliable as earlier believed (Cook 2001). Pain during loading and tenderness are always present in patients with patellar tendinopathy. However, a study by Cook et al. showed that a tendon may be tender even though no injury exists. During palpation of the tendon, the knee should be fully extended and the quadriceps muscles relaxed (Cook, et al. 2001). When the knee is flexed to 90°, the tension in the tendon increases and tenderness often decreases. A frequently occurring clinical finding is atrophy of the quadriceps muscles, resulting in weakness and pain during knee loading activities such as jumping and squatting. The decline squat is a valuable clinical tool, which increases the load on the patellar tendon during squats. Pain during squats could be estimated on a

visual analogue scale (VAS). The angles of knee flexion where pain occurs should also be established.

Differential diagnoses

There are several different anatomical structures close to the patellar tendon, and hence it is sometimes difficult to make a correct diagnosis. Tendon pain is often well localised in tendinopathy, but if the patient has diffuse pain symptoms, other diagnoses should be suspected. Pain from the patellofemoral joint should be excluded. Clinically, it is difficult to differentiate chondropathology originating in the inferior pole of the patella from tendon pain (Cook, et al. 2001; Alfredson 2005). Plica formations may cause symptoms like pain, snapping or popping related to the patellofemoral joint. This has also been associated with chondral lesions of the patella (Dupont 1997). Impingement of the infrapatellar fat pad (Hoffa's disease) is unusual, but could occur at full knee extension (Kumar, et al. 2007). Patellofemoral pain syndrome (PFPS) usually goes with pain in the antero-medial aspect of the patella (Witvrouw, et al. 2005). A partial patellar tendon rupture can be difficult to differentiate from patellar tendinopathy, but often appear in relation to high impact activities in athletes, or after a sudden blow to the tendon. The Osgood-Schlatter syndrome is an inflammation in the bone and/or cartilage where the patellar tendon attaches to the tibia. The condition is commonly seen in adolescents during growth spurt, usually in combination with high activity levels (Medlar, et al. 1978). The Sinding-Larsen-Johansson disease is also found in adolescents and is believed to be caused by traction from the patellar tendon in the region of the growth plate where the proximal patellar tendon attaches (Medlar, et al. 1978).

Treatment

The goal for the treatment of patients with patellar tendinopathy is the same as for other tendinopathies: to decrease pain and improve physical activity. Different treatment methods have been recommended, but there is sparse scientific evidence for most methods used. Corrections of intrinsic and extrinsic risk factors like training errors, flexibility, biomechanical abnormalities, and muscle weakness are often initiated.

Rest and NSAIDs are often recommended, however rest is detrimental to the tendon causing weakness and impaired tendon properties (Kannus, et al. 1997a). Furthermore, there is strong evidence to suggest that there is no inflammation inside the tendon, at least not in the chronic stage (Alfredson, et al. 2001). Therefore the use of NSAIDs is considered inappropriate.

Cryotherapy. Besides the analgetic effect, cryotherapy is used to reduce the metabolic rate in the tendon and decrease the extravasation of blood and protein from new capillaries found in tendon injuries (Rivenburgh 1992).

Ultrasound that often is used clinically has proven to have no effect in the treatment of tendinopathies (Stasinopoulos, et al. 2004). Warden et al. found low-intensity US no better than placebo treatment in a double-blind, placebo-controlled trial (Warden, et al. 2008).

ESWT has become a popular treatment option for patellar tendinopathy. However, there are few studies showing clinical effects. Peers et al. concluded that ESWT contributed to improvement with less pain and better function in the short-term perspective (Peers, et al. 2003).

Transverse friction massage. Manual therapy techniques are commonly employed. To our knowledge only one study has evaluated transverse friction massage in patients with patellar tendinopathy, and no effects on pain and function were found (Stasinopoulos, et al. 2004).

Strength training. A similar eccentric training protocol as described by Curwin for Achilles tendinopathy has also been used for patellar tendinopathy with encouraging results (Curwin 1984). As for Achilles tendinopathy, eccentric training is considered the cornerstone in a rehabilitation program for patellar tendinopathy (Peers, et al. 2005). Different groups have previously studied the effects of an eccentric training regimen, with varying results (Jensen, et al. 1989; Cannell, et al. 2001).

Injections of sclerosing polidocanol outside of the tendon, where the vessels enter the tendon, have shown promising results with decreased pain and return to previous physical activity levels (Alfredson, et al. 2005a).

Surgical treatment methods are similar to those used to treat Achilles tendinopathies, i.e. most often a longitudinal tendon incision and excision of abnormal tendon tissue, followed by a period of immobilisation, is used. The outcomes of surgery for patellar tendinopathy are unpredictable. In a review of 23 studies, Coleman et al. evaluated the success rate of surgical outcome, showing varying results (46–100% good results). Interestingly, clinical results were better in studies of poor scientific design, and less good in studies with a good scientific design (Coleman, et al. 2000). In a randomised study by Bahr et al. comparing the results of intra-tendinous surgery with painful eccentric quadriceps training, there were 45% good results after surgery and 55% good results after eccentric training (Bahr, et al. 2006).

The supraspinatus tendon

Anatomy

The rotator cuff consists of four separate muscles: the subscapularis, the supraspinatus, the infraspinatus and the teres minor muscle. They all arise from the scapula and are inserted into the tuberosities of the humerus (Fukuda, et al. 1990). The supraspinatus has a fleshy origin in the fossa supraspinatus, and inserts into the greater tuberosity (Fig. 7). Its tendinous insertion fuses with the infraspinatus posteriorly, and the coracohumeral ligament anteriorly. Because of its anatomical position, confined above by the acromion and the coracoacromial ligament, and below by the humeral head, the tendon is at risk for compression and attrition. The rotator cuff is considered to have three functions, to provide the gleno-humeral joint with stability, movement, and nutrition (Fukuda, et al. 1990).

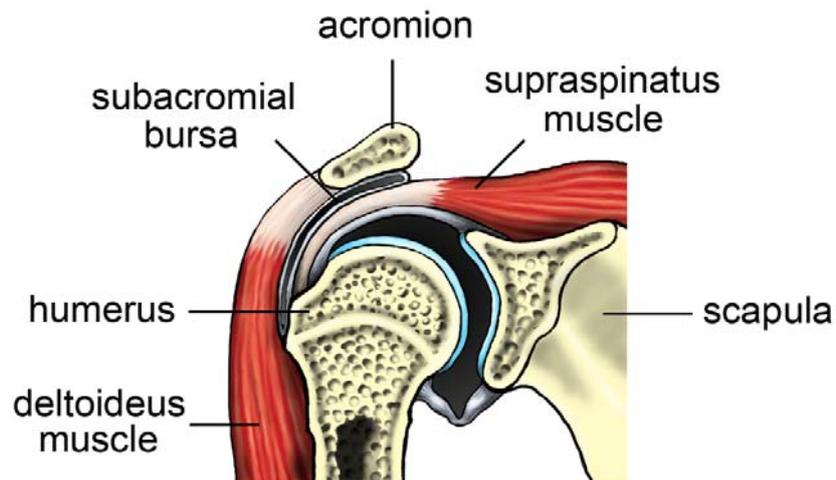


Figure 7. The shoulder joint

Tendon structure

The rotator cuff tendons are surrounded by an epitenon. This is the outermost layer, as the rotator cuff tendons do not have a paratenon. Near the insertion of the supraspinatus tendon into the great tuberosity, a five-layer complex has been described, that details the density and organisation of collagen and its associated elements (Malcarney, et al. 2003). It has been suggested that this intratendinous variation of collagen fibre density and orientation, may create shear forces within layers and lead to intrasubstance tears (Soslowsky, et al. 2000). Such variations in fibre orientation within the cuff/capsule complex, from superficial to deep, affect the biomechanical properties. In a complex way, the five-layer collagen complex can redirect the intratendinous strain in the rotator cuff during abduction (Fig.8). This may mask a tear during joint abduction. This also explains why partial-thickness tears may propagate into larger, full-thickness tears (Bey, et al. 2002).

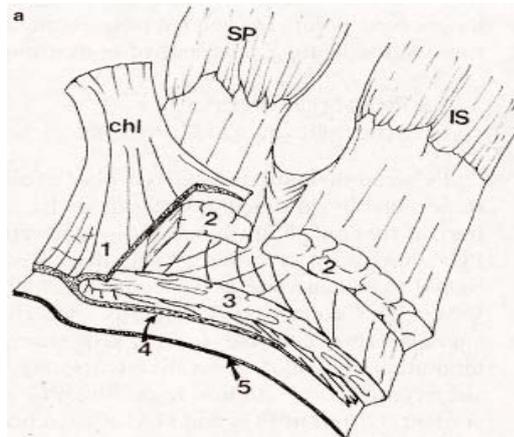


Figure 8. Schematic diagram of a dissection section vertically at various points along the supraspinatus and infraspinatus tendons and joint capsula. Layer 1 through 5 are labelled. Chl=coracohumeral ligament; IS=infraspinatus; SP=supraspinatus. (Reproduced with permission from Sports Medicine, 'The Rotator Cuff: Biological Adaption to its Environment' Malcarney et al, 2003)

Circulation

The rotator cuff receives its blood supply from several different branches of the axillary artery, for example the anterior and posterior circumflex humeral arteries, the thoracoacromial artery, and the suprascapular artery. The anterior circumflex humeral artery runs along the inferior border of the subscapular muscle, supplying the anterosuperior rotator cuff (Rathbun, et al. 1970;Malcarney, et al. 2003).

Innervation

The supraspinatus muscle is innervated by the suprascapular nerve (C4-6). The innervation patterns of the rotator cuff have not been clearly described. An electrophysiological experiment by Minaki et al. showed that there are nociceptors receiving pain sensation, and proprioceptors receiving position sense in the supraspinatus muscle-tendon unit (Minaki, et al. 1999). The subacromial bursa is positioned in very close proximity to the tendon. The bursa is innervated by the suprascapular nerve posteriorly and anteriorly by the lateral pectoral nerve (C5, 6), and they provide the bursa with proprioception and nociception (free nerve endings) (Ide, et al. 1996).

Biomechanics

Due to the anatomical structure, it is difficult to measure strain forces of the supraspinatus tendon in vivo. In vitro strain tests of the supraspinatus tendon during different angles of arm abduction have been reported. The cross-sectional area of the tendon fibres has been shown to be smaller on the joint side compared to the bursal side of the tendon, leading to a reduced ability to withstand strain at the joint side (Nakajima, et al. 1994). It seems that the strain is higher at the joint side, near the insertion of the tendon, compared to the bursal side (Bey, et al. 2002). The strain at the joint side increases in higher angles of arm abduction. There is also a difference in strain between the joint and bursal side during static loading and abduction of the humerus, implying a shearing effect between the two parts. It has been stated that this initiates intratendinous tears at the joint side (Reilly, et al. 2003;Huang, et al. 2005;Seki, et al. 2008). The tensile strength and stiffness of the supraspinatus tendon decreases with age. However, a specimen from a 65 year old human can still demonstrate a maximum tensile strength of about 900 newton (N), and is not necessarily ruptured or degenerately altered (Rickert, et al. 1998).

Supraspinatus tendinopathy

Definitions

Intrinsic ***supraspinatus tendinopathy*** is defined as tendon pathology that originates within the tendon, usually as a consequence of overuse or overload (including compression) (Lewis 2009). Hashimoto et al. have described similar degenerative changes as in other tendinopathies (Hashimoto, et al. 2003).

Epidemiology

Disorders of the shoulder rotator cuff are very common in the general population and in overhead athletes. The incidence of shoulder pain in the general population ranges from 6.6–25 cases per 1,000 persons, with a peak incidence in those aged 45–64 years (van der Windt, et al. 1995). The annual prevalence in the adult population in different countries is reported to range between 20% and 51% (Hasvold, et al. 1993;Pope, et al. 1997). The prevalence in swimmers has been shown to increase with the number of swimming years, ranging from 3% up to 65% (Kennedy 1974;Bak 1996). Recently, a study on elite swimmers showed that 69% suffered from supraspinatus tendinopathy (Sein, et al. 2008). Questionnaire studies by Fahlström et al. reported that among recreational and competitive badminton players, 52% had previous or present shoulder pain on the dominant side. Furthermore, the studies showed that persisting shoulder pain during the playing season was found among 20% of competitive players and among 16% of recreational players (Fahlström, et al. 2006;Fahlström, et al. 2007).

Aetiology

Rotator cuff disease is the most common cause of shoulder pain, and is often explained by chronic overuse. However, the aetiology to rotator cuff injuries has not been clarified and appears to be multifactorial, related to primary changes within the tendon (intrinsic cause) and other structures in its environment (extrinsic causes) (Uthoff, et al. 1997).

Intrinsic risk factors

Age, joint laxity, and bony impingement due to the shape of acromion, have been linked to supraspinatus tendinopathy and subacromial impingement (Neer 1972;Kannus 1997).

Muscle weakness of the rotator cuff muscles and pathological changes in the supraspinatus tendon due to overload (Nirschl 1989). Ogata et al, suggested that degeneration is the primary aetiological factor contributing to partial supraspinatus tears (Ogata, et al. 1990).

Extrinsic risk factors

Overuse, such as excessive physical activity in high arm positions, and with high loads, is an important factor contributing to the development of tendon injury (Stenlund, et al. 1993). In a recently published study on swimmers, Sein et al. found that more than 15 hours of swim training per week increased the risk of developing supraspinatus tendinopathy (Sein, et al. 2008).

Underuse or stress-shielding has lately been discussed as an aetiological factor in supraspinatus tendinopathy. There is ongoing remodelling in the normal tendon with degradation and rebuilding of tendon tissue. This process is believed to be mediated by matrix metalloproteinases (MMPs) and tissue inhibitor metalloproteinases (TIMPs). It has been suggested that underload possibly causes the development of supraspinatus tendinopathy by altering the remodelling process. Decreased expression of MMP3, TIMP2, and TIMP3 may contribute to tendon degeneration in sedentary individuals (Lo, et al. 2004).

Pathogenesis

Two main theories, one vascular and one impingement theory, have been used to explain why the supraspinatus tendon seems prone to degenerative changes. The vascular theory suggests that a compromise of the vascular supply may cause degeneration. Codman described a theory of a critical zone in the supraspinatus tendon, near the distal end of the tendons insertion into the humerus (ruptures were often seen in this area) (Codman 1934). Later, Rathbun reported poor vascularity in this critical zone, and hypothesised that this could be the reason for degenerative changes (Rathbun, et al. 1970). Other authors have supported this theory and also suggested that the vascularity is poorer on the joint side compared to the bursal side of the supraspinatus tendon (Lohr, et al. 1990).

It has been proposed that the high incidence of supraspinatus tendinopathy could be a result of impingement in and around the critical zone of the vascular supply (Luo, et al. 1998). Further, it has been suggested that this is the primary cause of tendinopathy in the shoulder (Neer 1983). In the shoulder, it is thought that the supraspinatus tendon impinges under the anterior acromion during forward flexion of the shoulder, leading to

degeneration. The shape of the acromion has been linked to impingement of the supraspinatus tendon. Three types of anatomical variations have been described (Bigliani 1986): type I (flat), II (curved) and III (hooked), where type III has been shown to be related to a high incidence of cuff tears (Neer 1972). The impingement theory has been challenged, Hyvonen et al. suggested that acromioplasty does not prevent the progression of the impingement syndrome into rotator cuff tears (Hyvonen, et al. 2003). Furthermore, if impingement itself is causing rotator cuff tears at the later stage of the disease, why do the majority of tears occur intrasubstance and/or on the joint side when the compression occurs on the bursal side (Ogata, et al. 1990).

Histology

The histopathology of chronic painful supraspinatus tendinopathy is similar to that of other insertional tendinopathies. The tendon shows mucoid degeneration and fibrocartilaginous metaplasia, along with loss of the characteristic parallel collagen bundles with separation and disorganisation (Fukuda, et al. 1990; Hashimoto, et al. 2003). Hypervascularity and neovascularisation of the degenerative rotator cuff have been reported by Chansky et al, who concluded that the hypervascularity was an attempt to repair or regenerate the lesion in the suprapinatus tendon (Chansky, et al. 1991).

Pain mechanisms

Pain in the shoulder often decreases shoulder strength and causes impaired function. The rotator cuff and subacromial bursa (SAB) are considered to be the primary pain-producing structures. The SAB reduces friction during shoulder movement and has been shown to provide the shoulder with kinesthetic sense and mechanoreception (Ide, et al. 1996). Individuals with pain during shoulder elevation have been shown to have high concentrations of inflammatory, pain-mediating and matrix-modifying proteins, in the bursa. This could have a catabolic effect on collagen (Voloshin, et al. 2005). Also, a high concentration of SP in the bursa has been shown to be correlated with pain (Gotoh, et al. 1998; Gotoh, et al. 2001; Yanagisawa, et al. 2001; Voloshin, et al. 2005). Using US and CD, high blood flow was found in chronic painful, but not in pain-free, supraspinatus tendons. Pain relief has been achieved from sclerosing polidocanol injections in the region with high flow (bursa wall just outside the tendon) (Alfredson, et al. 2006).

Clinical symptoms

Patients with supraspinatus tendinopathy often describe the onset of their pain symptoms as gradual. Pain after activity, especially overhead activity, weakness, and fatigue in the shoulder muscles are common complaints. The pain is usually dull and aching, commonly located at the anterior aspect of the shoulder, and sometimes laterally in the upper humerus.

Clinical examination

Based on the patient's medical history, a physical examination should help to clarify the diagnosis. It is important to also examine the neck carefully in order to exclude abnormalities such as degenerative disease or nerve entrapment. Examination should include an inspection of the scapular motion and position (winging). Test of the active and passive range of motion of the shoulder in all planes, as well as palpation of the rotator cuff, the acromioclavicular joint (Ac-joint) and sternoclavicular joint (Sc-joint) should always be included. Specific strength tests of the rotator cuff muscles should be carried out. The examination can disclose pain and weakness but also scapulothoracic dysfunction. Neers and Hawkins tests are examples of traditionally used subacromial impingement sign tests (Hawkins, et al. 1980; Neer 1983).

Differential diagnoses

Osteoarthritis in the Ac-joint could cause similar pain symptoms as subacromial impingement. Palpation of the joint and a cross-body adduction test meant to provoke could reproduce pain in this area. Injection of a local anaesthetic into the joint is helpful when trying to trace the pain. Cervical radiculitis should be ruled out by using provocative tests and nerve stretch tests. A superior labral anterior to posterior (SLAP) lesion could lead to secondary impingement. One important difference is that the symptoms often arise acutely, often following a trauma to the shoulder. Tests that are commonly used include the O'Brien and the biceps load II test (O'Brien 1998; Kim, et al. 2001). Biceps pathology could be tested using Speed's biceps test (Bennett 1998). Glenohumeral instability is common in overhead athletes and could lead to secondary impingement. Tests commonly used to provoke instability include the apprehension and relocation tests (Speer, et al. 1994; Tennent, et al. 2003). The Sulcus Sign for inferior instability is a general laxity test and posterior instability is evaluated using the posterior subluxation test (Tennent, et al. 2003). Partial or total ruptures of the supraspinatus tendon are not uncommon in traumatic falls in middle-aged and elderly individuals. These patients experience weakness and/or pain during abduction of the shoulder. An US or MRI confirms the clinical

diagnosis (Frost 2006). Adhesive capsulitis due to chronic inflammation and fibrosis in the subsynovial layer of the shoulder capsule reduces the passive and active range of motion (Frost 2006). Calcific tendonitis is a deposition of calcium salts within the intact supraspinatus tendon. The patients have severe pain anterolaterally, and movement of the shoulder joint aggravates the pain. A plain radiography (x-ray) is useful to confirm this diagnosis (Frost 2006).

Treatment

Many different conservative treatment methods for patients with supraspinatus tendinopathy are used, the purpose is usually to decrease pain and improve activity. However, many of these methods have not been studied in proper scientific studies (Almekinders, et al. 1998).

Rest is commonly used to avoid activities that aggravate symptoms (Almekinders, et al. 1998).

ESWT has shown promising results in patients with calcifying tendinitis, but not for supraspinatus tendinopathy (Gerdesmeyer, et al. 2003).

Ultrasound is used but there are no evidence for beneficial effects in randomised controlled trials (Robertson, et al. 2001).

Low-level laser is used as a treatment method, but has not been shown to be effective (Basford 1995).

Topical glyceryl trinitrate patches. A well designed double-blind study by Paoloni et al. showed promising results in patients with supraspinatus tendinopathy (Paoloni, et al. 2005).

Strength training and exercise programs are the mainstay for the conservative treatment of patients with supraspinatus tendinopathy. However, there is a lack of high quality research to support this (Michener, et al. 2004). Exercise programs may include the following interventions: range of motion, stretching and flexibility, and strengthening exercises. The general recommendation for rehabilitation exercise for cuff tendinopathy is that the exercise should be performed with a low range of motion, primarily focusing on the depressor muscles like the subscapularis, infraspinatus and teres minor, endeavouring to avoid exercising the deltoideus and supraspinatus muscles. The exercise should be carried out within a pain-free range of motion (Brewster, et al. 1993;Litchfield, et al. 1993;Morrison, et al. 1997). Some randomised controlled trials have investigated the effectiveness of exercises (Brox, et al. 1993;Conroy, et al. 1998;Bang, et al. 2000;Ludewig,

et al. 2003;Walther, et al. 2004;Haahr, et al. 2005;Senbursa, et al. 2007;Lombardi, et al. 2008). Results from these studies show that exercise is effective as pain reduction in patients with impingement syndrome. Furthermore, home-based exercise was comparable to supervised exercise. The study protocols vary a lot as regards to frequency, duration, load, and type of exercise (Brox, et al. 1993;Bang, et al. 2000;Ludewig, et al. 2003;Haahr, et al. 2005;Senbursa, et al. 2007). Unfortunately, the type of strength training used is often not described in detail (Haahr, et al. 2005;Senbursa, et al. 2007). Most studies used elastic bands as a resistant tool for strength training (Bang, et al. 2000;Ludewig, et al. 2003;Walther, et al. 2004;Senbursa, et al. 2007).

US and CD-guided sclerosing polidocanol injections targeting the region with high blood in the subacromial bursa wall just outside the supraspinatus tendon have shown promising results in a pilot study (Alfredson, et al. 2006).

Surgical reports from subacromial decompression (acromioplasty) therapy for impingement syndrome have shown a success rate of 80–90% (Burns, et al. 1992;Checroun, et al. 1998;Chin, et al. 2007). However, these results have been questioned. When comparing acromioplasty with physiotherapy exercises, surgery was not clinically beneficial at 6, 12 or 48 months (Brox, et al. 1993;Haahr, et al. 2005;Haahr, et al. 2006). According to Hyvonen et al. acromioplasty does not prevent from progression of impingement syndrome to a rotator cuff tear (Hyvonen, et al. 1998).

General aims

The general aims of this thesis were to evaluate and modify painful eccentric training as a treatment method for different chronic tendinopathies;

- Achilles tendinopathy (study I-II)
- Patellar tendinopathy (study III-IV)
- Supraspinatus tendinopathy (study V)

Material and methods

Subjects

The patients studied in this thesis were referred to the Sports Medicine Clinic at the University Hospital of Umeå in study I-IV. In study II patients were also recruited from the Capio Arthro Clinic in Stockholm. In study V, all patients were recruited from the waiting list for shoulder surgery at the Orthopaedic Department at the University Hospital of Umeå.

The majority of the Achilles patients were recreational athletes and people with sedentary lifestyle, the knee patients were sports active, and the shoulder patients were not active in strenuous shoulder activities. For more information about the patients, please see (Table. 1) and summary of papers.

	Study I	Study II	Study III	Study IV	Study V
Patients	*78 **30	27	17	15	9
Tendons	*101 **31	34	◇10 ◇◇12	●9 ●●10	9
Male/Female	*53/25 **24/6	12/15	◇8/1 ◇◇5/3	●6/1 ●●7/1	5/4
Symptom duration months (mean±SD)	*19.2±28.6 **32.0±42.4	26.5±21.1	◇14.1±6.6 ◇◇19.1±17.9	●19.6±20.3 ●●15.4±6	41±18.2

Table 1. Description of subjects

*Mid-portion **Insertional

◇Standard eccentric training on the floor ◇◇Eccentric training on decline board

●Concentric training on decline board ●●Eccentric training on decline board

General inclusion and exclusion criteria

Inclusion criteria

Study I-IV

- Pain during tendon loading for more than 3 months
- Clinical and ultrasound or MRI verified tendon and/or bursae , bone changes

Study V

- Pain during shoulder activity for more than 3 months
- Positive Neers and Hawkins test
- No mechanical impingement on dynamic ultrasound examination during abduction (0-100°)

Exclusion criteria

- Patients with chronic inflammatory diseases
- Previous surgical treatment
- Arthrosis or other disorders in nearby joints
- Disorders causing radiating pain in affected limb

Ultrasound and magnetic resonance imaging

The majority of chronic tendinopathies are diagnosed with clinical examinations. However, in order to confirm the diagnosis, imaging using grey scale US and MRI is helpful. In the past, US was considered a complement to MRI for the imaging of tendons. But today's modern high-resolution US is highly competitive. US is now the preferred choice when imaging tendon injuries (Rasmussen 2000), and it is proven to be accurate and cost-effective (Jacobson 1999). One important advantage of US is that the examination is dynamic. Recently, Warden et al. compared the accuracy of MRI and US when confirming clinically diagnosed patellar tendinopathy. The authors showed that US was more accurate than MRI (Warden, et al. 2007). In tendinosis, grey scale US reveals a widening of the tendon, irregular organisation of collagen fibres, and also focal hypoechoic regions (Khan, et al. 1996) (Alfredson, et al. 2005a). By using CD together with US, it is possible to study blood flow within the tendon (Weinberg, et al. 1998;Öhberg, et al. 2001). A common finding in tendinopathies when using MRI is a local widening of the tendon and high signal intensity in the affected region of the tendon (Johnson, et al. 1996;Schmid, et al. 2002).

Treatment methods

Coaching

All patients in study I–V were personally instructed on how to perform the training program by the same physiotherapist. They also received written instructions on how to perform the different exercises. The patients were told to call the physiotherapist during working hours, or e-mail if they had any questions relating to pain and/or load increases. At six weeks, all patients were followed up at the clinic (except in study II where 50% of the patients, due to practical reasons, were contacted by telephone and e-mail at two and six weeks to check training compliance) to make sure that patients did their exercises correctly and, when necessary, to adjust the load to a new painful level. Common complaints included muscle soreness, and increased morning stiffness, especially during the first two weeks. It was sometimes difficult for the patients to determine the correct progression of load to a new painful level of training. The patients registered the amount of discomfort and/or morning stiffness. Less discomfort and morning stiffness indicated that the load should be increased. During the time for these studies it was noticed that it is very important to support and coach patients through the first period of pain and discomfort. Poor coaching or no coaching seemed to be related to less good results.

Eccentric training program

Regarding the duration and frequency, the eccentric training program was based on the original eccentric training regimen by Alfredson et al. (Alfredson, et al. 1998), where the exercises were performed twice a day, seven days a week for 12 weeks. The patients performed three sets of 15 repetitions (3x15 repetitions). In study I–IV, the patients were recommended to wear stable footwear when exercising. Training was supposed to be painful, and when there was no pain during exercise, the patients increased the load to a new level of painful training. The level of tolerated pain varied between patients. The recommendation was that the subject should train with as much pain as could be tolerated, but it should be possible to perform the exercises in a controlled manner. In study I–IV, the load was increased using a backpack with weights, and in study V with a dumbbell or a weight attached to the wrist. In all studies, the patients were thoroughly informed that muscle soreness was to be expected during the first one to two weeks. In all studies the exercises were performed in slow speed. The patients were told to avoid concentric loading when returning to the start position in study I-III and V. In patients with unilateral problems, this was done by using the pain free side. In study IV, the patients were told to avoid either eccentric or concentric activation when returning to the start position depending on which group they were randomised to.

In study I, patients were allowed to perform light jogging after 4-6 weeks, if it could be done without pain. In study II, patients were allowed to slowly return to their previous sports/recreational activity after 6 weeks. In study III, patients were not allowed to continue their sports activity the first 8 weeks. In study IV, patients were not allowed to return to their sports activity for the first 6 weeks of treatment.

Study I

The patients performed the eccentric calf muscle training in an upright position, both with straight and with slightly bent knee (Fig. 9a-b); this ensured the activation of both the gastrocnemius and soleus muscles. Patients were standing on tiptoe on a step (stairs) with all the weight on the forefoot. From that position, they slowly lowered the heel below the step (Fig. 10 a-b). Patients were instructed to do their exercises twice a day, seven days a week, for 12 weeks. Each of the two exercises was done in three sets of 15 repetitions (3x15 repetitions). Patients were also instructed to avoid concentric loading when returning to the start position by using their non-injured leg and/or the arms.

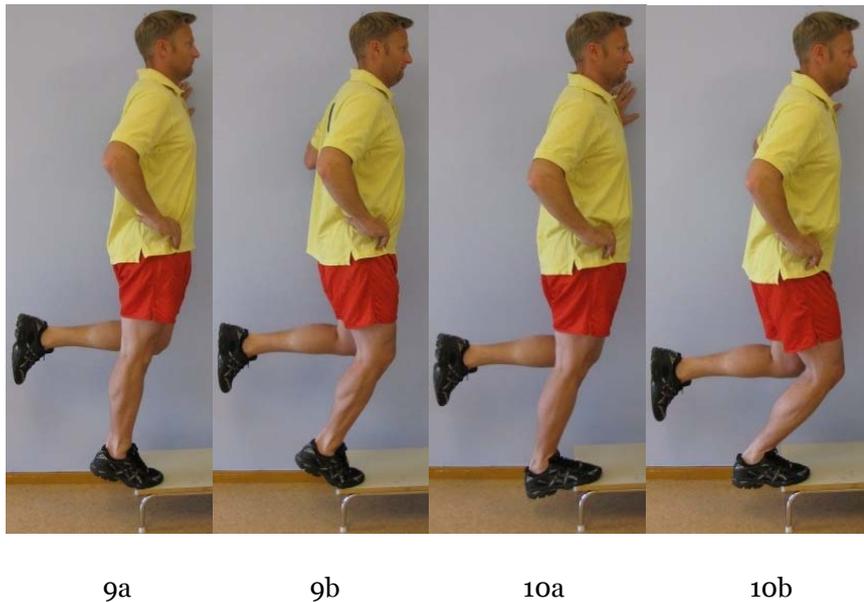


Figure 9a. Start position with straight knee

Figure 9b. Start position with slightly bent knee

Figure 10a. End position with straight knee

Figure 10b. End position with slightly bent knee

Study II

In this study, the patients performed the eccentric training in an upright position with straight leg. The patients were standing on tiptoe with their entire bodyweight on the forefoot at the floor. From that position, patients slowly lowered their heel to floor-level (Fig. 11a-b). No loading took place with the ankle in dorsiflexion. The patients performed the exercise in three sets of 15 repetitions (3x15 repetitions), twice a day, seven days a week for 12 weeks and patients with bilateral symptoms performed a leg press while standing on a box (stair) in order to reach the start position (Fig. 12). Patients with unilateral pain used the other leg to return to the start position, this was done to avoid concentric activation.



11a

11b

12

Figure 11a. Start position for insertional Achilles tendinopathy

Figure 11b. End position for insertional Achilles tendinopathy

Figure 12. Back to start position with the other leg

Study III

Two eccentric exercises were performed in study III. The patients were standing in an upright position with all their body weight on the affected leg. The first nine patients were prescribed eccentric quadriceps training with the foot placed on the floor (Fig. 13). The other eight patients were prescribed eccentric quadriceps training having the foot on a 25° decline board (Fig. 14). In both groups, the patients were instructed to perform the exercises slowly down to 90° knee flexion (Fig. 15 a-b). They were instructed to do the exercises in three sets of 15 repetitions (3x15 repetitions) twice a day, seven days a week for 12 weeks. The patients were instructed to as much as possible avoid concentric loading when returning to the start position by using their non-injured side. In cases with bilateral tendinopathy, both arms and legs were used.



13

14

15a

15b

Figure 13. Start position with the foot placed on the floor

Figure 14. Start position with the foot placed on a decline board

Figure 15a. End position with the foot placed on the floor

Figure 15b. End position with the foot placed on a decline board

Study IV

Eccentric and concentric quadriceps exercises were performed on a 25° decline board. The patients were standing in an upright position with all their body weight on the affected leg. The patients in the eccentric group slowly lowered their body to 70° knee flexion (Fig. 16 a-b). In the concentric group, standing on the 25° decline board, they started with the knee flexed to 70°, and from that position they slowly straightened the knee to full extension (Fig. 17 a-b). The patients were instructed to do the exercise in three sets of 15 repetitions (3x15 repetitions) twice a day, seven days a week for 12 weeks. Patients with unilateral symptoms used their non-injured side, in cases of bilateral tendinopathy, both arms and legs were used.



16a

16b

17a

17b

Figure 16a. Start position for eccentric quadriceps training

Figure 16b. End position for eccentric quadriceps training

Figure 17a. Start position for concentric quadriceps training

Figure 17b. End position for concentric quadriceps training

Study V

The eccentric training was performed with the patients sitting. In order to put high load on the deltoideus and supraspinatus muscles the patients performed the eccentric exercise with the shoulder in 30 degrees of horizontal abduction, with the thumb pointing towards the ground (the 'empty can' position) (Fig. 18). From that position the subjects slowly lowered the arm (eccentrically) to the end position (Fig. 19). To reach the starting position a device called an Ulla-sling was used, where the affected arm was elevated by the non-training arm; this method was used in order to avoid concentric loading (Fig. 20). The patients were instructed to do the exercise in three sets of 15 repetitions (3x15 repetitions), twice a day, seven days a week for 12 weeks. To increase the load to a new level of painful training, a dumbbell, or a weight was attached to the wrist (Fig. 21).



18

19

20

21

Figure 18. Start position for eccentric training

Figure 19. End position for eccentric training

Figure 20. Back to start position with the opposite arm

Figure 21. Increased load with a dumbbell, or a weight attached to the wrist

Outcome measures

The main goal with the treatment was to reduce pain during tendon loading activity and improve physical activity. The following tools were used;

Visual analogue scale (VAS)

The patients estimated the amount of pain during tendon-loading activity on a 100-mm long VAS, where 0-mm equalled no pain and 100-mm equalled maximal pain. This estimation was carried out at baseline and after 12 weeks of training. According to Williamson et al. (Williamson, et al. 2005), VAS is a valid, reliable, and precise scale.

Satisfaction with treatment

The patients recorded if they were “satisfied” (return to previous tendon-loading activity) or “not satisfied” (no return to previous tendon-loading activity) with the treatment. In Study V, being “satisfied” with the treatment equalled withdrawal from the waiting list for surgery.

The Victorian Institute of Sport Assessment (VISA)

In study IV, functional evaluation was carried out using the VISA score. The VISA score focuses on sports activity and has been shown to be valid and reliable in patients with patellar tendinopathy (Visentini, et al. 1998).

The Constant score

In study V, the Constant score was used. The Constant score is a widely used functional score for all types of pathological shoulder conditions (Constant, et al. 1987). The score assesses both subjective and objective measures, and calculates a maximum of 100 points (pain free and full function). The subjective measures, such as pain and daily living activities, are allocated 35 points, and the objective measures, such as shoulder strength and range of motion, are allocated 65 points.

Statistical methods

For statistical calculations, SPSS package version 9-11.5, (SPSS Inc, Chicago, IL, USA) was used in study I, II, IV and V. In study III, Statistica Release 6, 2002; (Statsoft Inc) was used.

The results are in general presented as means \pm SD, and range. As the number of patients in the majority of studies is relatively limited, non-parametric tests for independent samples (Mann-Whitney U-test) and for paired samples (Wilcoxon sign rank test) were used to calculate differences within groups before and after treatment (study I, III, IV, and V). Student's t-test was used to compare differences in study I and II. A p-value of less than 0.05 was considered significant.

Ethics

The Ethical Committee of the Medical Faculty, Umeå University, Sweden, has approved all investigations. The patients were informed verbally and in writing prior to consent.

Summary of papers

Paper I

Chronic Achilles tendon pain treated with eccentric calf-muscle training

Aims

The aim was to prospectively investigate if previous good clinical results with eccentric calf muscle training could be reproduced in a larger group of patients with chronic painful mid-portion tendinosis, and also to investigate the effects of eccentric training on a group of patients with chronic insertional Achilles tendon pain.

Subjects and methods

Seventy-eight patients with chronic painful Achilles tendinosis in the mid-portion (2–6 cm level) of a total of 101 tendons (55 unilateral and 23 bilateral), and 30 patients with chronic insertional Achilles tendon pain in 31 tendons (29 unilateral and one bilateral), were included. Most patients were recreational athletes. The patients were treated with eccentric calf-muscle training with straight and bent knee for 12 weeks. Most patients were recreational athletes. Evaluation of the amount of tendon pain during activity was recorded on VAS, before and after treatment. Patients satisfaction with treatment, and return to pre-injury activity levels were evaluated.

Results

After 12 weeks of eccentric training, 68 of 78 patients (89%, 90/101 tendons) were satisfied and had returned to their pre-injury activity level. Their amount of pain during activity had decreased significantly, from 66.8 ± 19.4 to 10.2 ± 13.7 on VAS. In the group with chronic insertional Achilles tendon pain, only ten patients (32%, 10/31 tendons) were satisfied and had returned to their pre-injury activity levels. In the satisfied patients the amount of pain had decreased significantly from 68.3 ± 7.0 to 13.3 ± 13.2 on VAS.

Conclusions

Treatment with painful eccentric calf-muscle training showed good clinical results in patients with chronic painful mid-portion Achilles tendinosis, but not in patients with chronic insertional Achilles tendon pain.

Paper II

New regimen for eccentric calf-muscle training in patients with chronic insertional Achilles tendinopathy: Results of a pilot study

Aims

The aim was to investigate whether a new model of painful eccentric training had an effect on chronic painful insertional Achilles tendinopathy.

Subjects and methods

Twenty-seven patients (12 men, 15 women, mean age 53 years) with a long duration (mean 26 months) of pain in 34 Achilles tendon insertions, were included. The patients performed a new model of painful eccentric training regimen without loading into dorsiflexion. This was done as 3x15 repetitions, twice a day, seven days a week for 12 weeks. Pain during Achilles tendon-loading activity on VAS and patients satisfaction (return to previous activity) was evaluated.

Results

At follow-up (mean 4 months), 18 patients (67%, 23/34 tendons) were satisfied and had returned to their previous tendon-loading activity. Their mean VAS was significantly decreased from 69.9 ± 18.9 to 21 ± 20.6 . Nine patients (11 tendons) were not satisfied with the treatment, although their VAS was significantly reduced from 77.5 ± 8.6 to 58.1 ± 14.8 .

Conclusions

In this short-term pilot study the new model of painful eccentric calf-muscle training showed promising clinical results in 67% of the patients with chronic insertional Achilles tendinopathy.

Paper III

A pilot study of the eccentric decline squat in the management of painful chronic patellar tendinopathy

Aims

The aim of this non-randomised pilot study was to identify differences in pain reduction and recovery of function, using eccentric single leg quadriceps training, on a flat surface and on a decline, in patients with chronic patellar tendinopathy.

Subjects and methods

Nine patients (10 tendons; 8 men, 1 woman; mean age 22 years) performed eccentric exercises with the ankle joint in a standard (flat foot) position. Eight patients (12 tendons; 5 men, 3 women; mean age 28 years) performed eccentric training standing on a 25° decline board, designed to increase the load on the knee extensor mechanism. The eccentric training was performed twice daily, with three sets of 15 repetitions, for 12 weeks. The patients were taken out from their regular sport activity for the first eight weeks. Outcomes were pain evaluation (VAS) during activity, and return to previous activity.

Results

Good clinical results were obtained in the group who trained on the decline board, with 6 patients (9 tendons) returning to sports and showing a significantly reduced amount of pain over the 12-week period (VAS from 74.2 to 28.5). At 15 months, 4 patients (5 tendons) reported satisfactory results (mean VAS 26.2). In the standard quadriceps training group the results were poor, with only 1 athlete returning to previous activity. Mean VAS in this group was 79.0 at baseline and 72.3 at 12 weeks ($p=0.144$).

Conclusions

In this small non-randomised pilot study, on patients with chronic patellar tendinopathy, painful eccentric quadriceps training on a decline board produced encouraging results in terms of reduced pain and return to function. Painful eccentric single leg quadriceps training on a flat surface appears to be a less effective form of rehabilitation in reducing pain and returning patients to previous levels of activity.

Paper IV

Superior results with eccentric compared to concentric quadriceps training in patients with jumper's knee: A prospective randomised study

Aims

The aim of this randomised study was to compare the results of painful eccentric quadriceps training on a decline board, with painful concentric quadriceps training on a decline board, in a group of athletes, with the diagnose jumper's knee/patellar tendinopathy.

Subjects and methods

Nineteen patellar tendons from 15 patients (13 men and 2 women, mean age 25 years) with chronic jumper's knee/patellar tendinopathy, were randomised to treatment with either painful eccentric or painful concentric quadriceps training on a decline board. Fifteen exercises were repeated three times, twice daily, seven days a week, for 12 weeks. All patients ceased sporting activity for the first six weeks of treatment. VAS and VISA scores, before and after treatment, and patients satisfaction, were used for evaluation.

Results

Age, height, weight, and duration of symptoms were similar between groups. In the eccentric group, for 9/10 tendons, (7/8 patients) were satisfied with the treatment, VAS decreased from 73 to 23 ($p<0.005$), and the VISA score increased from 41 to 83 ($p<0.005$). In the concentric group, 9/9 tendons (7/7 patients) were not satisfied, and there were no significant differences in the VAS (from 74 to 68, $p<0.34$) or VISA score (from 41 to 37, $p<0.34$). At follow-up (mean 32.6 months), the patients in the eccentric group were still satisfied and active in sports, but all patients in the concentric group had been treated surgically or with sclerosing injections.

Conclusions

Eccentric, but not concentric, quadriceps training on a decline board seems to reduce pain and allow for a return to previous sport activity in patients with chronic jumper's knee/patellar tendinopathy. The study aimed to include 20 patients in each group, but the study was stopped at the half-time control because of poor results in the concentric group.

Paper V

Eccentric training in chronic painful impingement syndrome of the shoulder: Results of a pilot study

Aim

The aim of this pilot study was to investigate if treatment with painful eccentric deltoideus and supraspinatus muscle training was effective in patients with a long duration of pain symptoms related to chronic subacromial impingement syndrome in the shoulder.

Subjects and methods

Nine patients (5 males and 4 females, mean 54, range 35–72 years) with chronic painful impingement syndrome, were included. All patients had a long duration of pain symptoms (mean 41 months), and were on the waiting list for surgical treatment (mean 13 months). The study prospectively investigated the effects of a specially designed painful eccentric training programme for the deltoideus and supraspinatus muscles, consisting of three sets of 15 repetitions, repeated twice daily, seven days a week for 12 weeks. The patients evaluated the amount of shoulder pain during horizontal shoulder activity on a VAS, and satisfaction with treatment (withdrawal from waiting list for surgery). Constant score was also assessed.

Results

After 12 weeks of treatment, five patients were satisfied with the treatment, their mean VAS had decreased (62–18, $p < 0.05$), and their mean Constant score had increased (65–80, $p < 0.05$). At the 52-week follow-up, the same five patients were still satisfied, and their mean VAS and Constant score were 31 and 81, respectively. Among the satisfied patients, two had a partial supraspinatus tendon rupture and three had a type III shaped acromion.

Conclusions

In this small pilot study with short term follow-up, it seems that painful eccentric training for the deltoideus and supraspinatus muscle might be effective in the treatment of patients with chronic painful impingement syndrome of the shoulder.

General discussion

Chronic tendinopathy is a common painful condition, often seen in response to overuse (Maffulli, et al. 1998). Certain tendons are more prone to injury than others, like the Achilles tendon, patellar tendon, and supraspinatus tendon (Rees, et al. 2009). Conservative (non-surgical) treatment is usually a difficult and longstanding process, which could be frustrating for both the patient and involved clinicians. Treatment of chronic tendinopathy has traditionally focused on controlling inflammation and pain (Speed 2001;Rees, et al. 2006;Kountouris, et al. 2007), however, histological studies do not support an inflammatory process in the chronic painful tendon (Åström, et al. 1995;Movin, et al. 1997;Almekinders, et al. 1998;Hashimoto, et al. 2003), and treatments such as corticosteroid injections and NSAIDs cannot be considered to be indicated. The main finding of this thesis is that treatment with eccentric training seems promising regarding reduction of pain and return to physical activity for patients with chronic painful tendinopathies.

In an attempt to reproduce the good clinical results from our pilot study (Alfredson, et al. 1998), we evaluated the eccentric exercise protocol on a larger group of patients with chronic painful Achilles tendons, both with mid-portion and insertional tendinopathy (**study I**). The study showed very good results for the group with mid-portion Achilles tendinopathy, 89% were satisfied and back to previous tendon-loading activity. However, only 32% of the patients with chronic insertional Achilles tendinopathy were satisfied and back to tendon-loading activity. The Alfredson protocol has been widely used in patients with chronic mid-portion Achilles tendinopathy, and good clinical results have been reported also by others (Mafi, et al. 2001;Roos, et al. 2004;de Vos, et al. 2007;Rompe, et al. 2007). However, the results vary in different parts of the world, and there might be several explanations to this. This type of protocol requires motivated patients that are going to perform painful exercises twice a day for 12 weeks. In certain countries, with different cultures, training, and especially with pain, is simply not accepted. Also, we believe that it is very important to provide the patients with support and coaching, and arrange for regular follow-ups to control that the exercises are done correctly and to adjust the load (load progression). If the patients are given a programme with painful exercises to follow on a daily basis for 12 weeks, without any follow-ups or contacts during this period, there might be a risk that the compliance will be poor. Another possibility to the different results in different studies might be that not all studies have confirmed the diagnosis with objective measures (Niesen-Vertommen 1992;Silbernagel, et al. 2001;Norregaard, et al. 2007). It might be that some patients have other diagnoses such as a partial rupture, accessory soleus muscle, retrocalcaneal

bursitis etc., not responding to eccentric training. A study by Silbernagel et al. showed favourable results with eccentric training in combination with concentric exercises and other exercises like stretching and quick rebounding toe-raises (Silbernagel, et al. 2001).

Based on the results from **study I**, where eccentric training showed poor results in patients with chronic insertional tendinopathy, a modified version of eccentric training was used in **study II**. The poor results in **study I** were believed to be due to that the eccentric exercises caused impingement between the tendon, bursa, and bone when the ankle was loaded in plantar flexion. Instead, in **study II** the eccentric exercises were done only to floor level, avoiding load in plantar flexion. The results, using this modified version of eccentric training, were much better, with 67% satisfied patients. Interestingly, there were no differences in the results between patients with tendon, bursae, or bone, pathology, alone or in combination. Even among patients with major bone pathology, like large bone spurs, seemingly causing mechanical problems, there were good clinical results after this eccentric training regimen. Since the results after surgical treatment are varying and complications not are uncommon (Nesse, et al. 1994;Maffulli, et al. 1999;Paavola, et al. 2000;Boberg, et al. 2002), the results with this relatively simple treatment regimen are of interest. However, again, it needs to be emphasized that these patients need coaching to be motivated to train with pain two sessions per day for 12 weeks.

Chronic jumper's knee/patellar tendinopathy is known to be difficult to manage. The prevalence of patellar tendinopathy is high, around 40–50% in sports with high demands on the leg extensor mechanism, like volleyball and basketball (Ferretti 1986;Cook, et al. 1997;Lian, et al. 2005). With the encouraging results from treatment with painful eccentric training on chronic Achilles tendinopathy, we decided to also evaluate eccentric training on patients with jumper's knee/patellar tendinopathy. Our Australian co-authors Craig Purdam and Jill Cook had new ideas about the importance of the foot positioning to maximize the load on the patellar tendon, during the exercise. To stand on a 25° decline board during the eccentric exercise, would theoretically put more loads on the tendon by decreased activity of the calf muscles. In a small, non-randomised pilot-study, painful single-leg eccentric quadriceps exercises performed standing on a 25° decline board, or standing flat on the ground, were evaluated in **study III**. Interestingly, the results were good in the majority of the patients using the decline board, but very poor among the patients that performed the exercises standing flat on the ground. The theories behind using the 25° decline board, stated by Purdam and Cook, have been tested and further evaluated by others. According to Kongsgaard et al. using a decline board mediates a relaxation of the gastrocnemius muscles, and also decreases the ankle joint moment (increased load on the patellar tendon) (Kongsgaard, et al. 2006).

Furthermore, Zwerver et al. found that the hip moment decreased and the knee extensor moment increased with 40% (Zwerver, et al. 2007). In following studies by others there are varying results using eccentric quadriceps training on a decline board. Visnes et al. showed poor results from eccentric training on a decline board on volleyball players performing the treatment regimen during the playing season (Visnes, et al. 2005). Young et al. found no differences between eccentric exercise done on a decline board and single leg squats on a 10 cm step (according to the Curwin and Stanish protocol) (Curwin 1984). Also in that study the exercises were done during the playing season (volleyball), in parallel with the regular training and playing (Young, et al. 2005). In our study the patients were taken out from their sports activity the first 8 weeks of the eccentric training regimen, and they were not allowed to participate in other strenuous knee loading activities during this period. This is an interesting difference possibly of importance for the outcome of the treatment. An observation favouring this is that in the study by Young et al. (Young, et al. 2005), the results in the decline board group improved at the 1 year follow-up, when eccentric training had been done during off-season (no volleyball playing). To optimize the effects of the eccentric training regimen, maybe other heavy quadriceps activities should be avoided, at least initially in the treatment period.

In **study IV**, in patients with chronic jumper's knee/patellar tendinopathy, we compared the clinical results after painful eccentric quadriceps training on a decline board with painful concentric quadriceps training on a decline board. For this randomised study the power analysis showed that 20 patients were needed in each group. However, at the mid-term control, the results in the group performing concentric exercises were very poor, almost a worsening in all patients, leading to that due to ethical reasons no more patients were included. This explains why the two groups are relatively small. Anyhow, the clinical results in the group randomised to eccentric training were good in the majority of the patients, clearly showing a significant difference between eccentric and concentric training on a decline board. A difference among studies evaluating eccentric quadriceps training as a treatment for chronic jumper's knee/patellar tendinopathy is that there are differences in the inclusion criteria. Our studies are the only that used an inclusion criteria with tendon pathology in the proximal tendon (inferior pole of the patella) alone, whereas in other studies also tibial insertional tendinopathy was included (Cannell, et al. 2001; Stasinopoulos, et al. 2004; Visnes, et al. 2005). One study looked at both proximal and distal patellar tendinopathy (Bahr, et al. 2006), and one did not distinguish where in the tendon the injury was found (Young, et al. 2005). This makes it difficult to compare the results between the studies.

The structural degenerative tendon changes found in the supraspinatus tendon in patients with chronic supraspinatus tendinopathy and chronic painful impingement syndrome are similar to what is found in chronic Achilles and patellar tendinopathy. Therefore, in a small pilot study (**study V**) we investigated if painful eccentric training for the deltoideus and supraspinatus muscles had good clinical effects on patients with severe (on the waiting list for surgical treatment) chronic painful impingement syndrome. A specially designed eccentric training regimen was used, aiming to try to maximise the load on the deltoideus and supraspinatus muscles. Opposite to what is considered as standard in shoulder rehabilitation, the “full can” position of the hand, we used the “empty can” position, (internal rotation of the arm and having the thumb pointing downwards) during the exercise. Altogether, five out of nine patients were satisfied and withdrew from the waiting list for surgery after three months of painful eccentric training. These results are interesting, especially since these patients had a long duration of pain symptoms, had tried multiple types of conservative treatments, and were waiting for surgical treatment. Even more interesting is that 3 out of 5 patients that were satisfied had a hooked acromion (Bigliani type III), a finding that is considered to be a strong predictor indicating the need for surgical treatment. The material in this pilot study was very small, and conclusions cannot be drawn. However, the results may serve as an indicator that this type of treatment might be worth testing in larger materials.

Shoulder pain is common in the general population, about 50% of the population annually suffers from periods with shoulder pain (Brox 2003), and it seems important to try to find methods for treatment. A problem when dealing with shoulder pain is that it is sometimes difficult to give a correct diagnosis. In our study, many patients couldn't be included because they had the majority of the pain problems from the Ac-joint, a condition likely not suited for treatment with this type of exercises. Also, some patients had multiple diagnoses, such as labral tears which caused secondary impingement symptoms. Consequently, the inclusion procedure must be as optimal as possible, including reliable and valid tests. In our study we used clinical impingement tests, like the Neers and Hawkins tests and we used dynamic US to exclude mechanical impingement during abduction and x-ray to exclude arthrosis in the acromioclavicular and humeroscapular joint. A close relation between weakness and pain in the supraspinatus muscle-tendon unit, and subacromial impingement syndrome, has been shown (Lewis 2009), where supraspinatus weakness is believed to cause migration of the humeral head upwards towards the subacromial space causing a secondary impingement syndrome. Exercise to decrease pain and improve strength and function of the supraspinatus might be of significant importance.

In this thesis we found good clinical effects in terms of significantly decreased pain during activity and return to previously activity levels, after treatment with painful eccentric training, in patients with chronic Achilles-, patellar- and supraspinatus tendinopathy. We cannot explain why treatment with painful eccentric training leads to these good clinical results. Multiple theories have been raised. Langberg et al, have shown an increased collagen synthesis in the peritendinous tissue of the Achilles tendon in response to eccentric exercises, leading to improved tendon strength (Langberg, et al. 2007). Rees et al, suggested that eccentric exercise created an oscillating activity inside the tendon, that is not seen during concentric exercise, leading to improved remodelling of the tendon (Rees, et al. 2008). There are also theories around eccentric exercise leading to increased tendon stiffness (Pousson, et al. 1990) and changes related to lengthening of the muscle-tendon unit (Mahieu, et al. 2008). Ourselves, we believe that there might be a traumatic effect, where the eccentric exercises traumatise nerves and vessels when these structures are coming from the soft fat tissue and enter the relatively hard and dense tendinosis tendon. That could possibly explain why these patients have increased pain levels initially during the training regimen, and also that some patients are cured already after 4-6 weeks. Also, eccentric training could stimulate to a normalisation of the tendon structure, this have been shown in patients with mid-portion Achilles tendinosis (Öhberg, et al. 2004b).

An interesting observation, accompanying the results of our studies with others, it seems that the results of the eccentric training regimen are better if the patients that are taken out from their sports or recreational activity during the first 6 weeks of treatment. Maybe the tendon and it's cells cannot cope with too much stimuli? Even though the eccentric exercises are demanding, especially for patients less used to physical activity, for some reason it seems that the tendon copes well with this type of loading. With the exception of **study I**, there are relatively few patients included in our studies. This is also noticed from other studies. In a review on eccentric training for patellar tendinopathy, Visnes et al. found that there were 7 trials and a total of 162 patients. Altogether, 112 patients were included to different eccentric training protocols (Visnes, et al. 2007). In another review on eccentric training for lower extremity tendinosis, Wasielewski et al. reported the need for multicenter studies due to difficulties to include larger study groups (Wasielewski, et al. 2007). Roos et al. reported that it took 3 years to include 44 patients in a randomised study on eccentric training for mid-portion Achilles tendinopathy (Roos, et al. 2004). Altogether, worldwide it seems that it is difficult to include a large material within an acceptable period of time, allowing for well designed randomised studies. Another problem, something we experienced in **study IV**, is that if the half time clinical results in a treatment group are very poor the study needs to be

stopped due to ethical reasons. It cannot be considered correct to include multiple patients for a treatment that has shown poor clinical effects or even worsening of the condition.

In summary, the present studies showed good clinical results with low risks of side effects and low costs. Thus, we suggest that painful eccentric training should be tried in patients with Achilles and patellar tendinopathy before intratendinous injections and surgery are considered. For patients with chronic painful impingement syndrome, the results of our small pilot study are interesting, and stimulates to randomised studies on larger materials.

Conclusions

- Treatment with painful eccentric calf-muscle training showed good clinical results in patients with chronic painful mid-portion Achilles tendinosis, but not in patients with chronic insertional Achilles tendon pain.
- A new model of painful eccentric calf-muscle training showed promising clinical results in 67% of the patients with chronic insertional Achilles tendinopathy.
- Painful eccentric quadriceps training on a decline board produced encouraging results in terms of reduced pain and return to physical activities in patients with chronic jumper's knee/patellar tendinopathy. Painful eccentric single leg quadriceps training on a flat surface appears to be a less effective form of rehabilitation in reducing pain and returning patients to previous levels of activity.
- Painful eccentric, but not concentric, quadriceps training on a decline board seems to reduce pain and improve physical activities in patients with chronic jumper's knee/patellar tendinopathy.
- It seems that painful eccentric training for supraspinatus and deltoideus might be effective in patients with the diagnose chronic supraspinatus tendinopathy.

Acknowledgements

This thesis has benefited from the assistance and encouragement of several persons, and I would especially like to express my warmest thanks to:

Håkan Alfredson, my supervisor and mentor, for your generous support over the years, patience and invaluable help.

Martin Fahlström, my co-supervisor, co-author, for outstanding support and enthusiasm.

Peter and Anna Nordström for all support during these years, your skill and knowledge about statistical analyses and help with the layout.

Ronny Lorentzon, co-author, for your scientific knowledge during our discussions and your valuable/critical thoughts.

Lars Öhberg, co-author for introducing me to the world of ultrasound.

Per Wahlström, co-author, for teaching me a proper shoulder examination.

Patrik Danielson for sharing your knowledge about pain mechanisms.

Gustav Andersson for the help with anatomy pictures.

Kerstin Sunding, co-author, for your friendliness at Capio Artro Clinic.

Jill Cook, co-author, for your research spirit, support and generosity.

Craig Purdam, co-author.

Karim Khan, co-author.

Torsten Sandström introducing me to the computer world.

Gunilla Solander for assistance with administrative work.

Suzanne Werner for your trust and valuable support.

Past and present staff at Sports Medicine Unit.

Eva, my wife, Willy and Saga our children, for their love and patience.

David and Majvor, my parents, for their love and support throughout my life.

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