Chapter 1
General introduction
Introduction

“In his unfinished epic the Achilleis, the Roman poet Publius Papinius Statius described the life of the Greek warrior Achilles, who was foretold a young death [124]. To prevent this, his mother Thetis took him to the mystic River Styx and immersed his body into the water. However, she held the young Achilles by the heel; this spot remained vulnerable. Ultimately, this weakness resulted in his death, as legend states he was shot in the heel by Prince Paris. We cannot state with certainty which anatomical landmark was penetrated by Paris’ (poisonous) arrow. Given the limited capacity of arrows in penetrating bone [70], it is thought the tendon known by the Romans as Tendo magnus (great tendon) was struck. In 1693 the Dutch anatomist Philip Verheyen coined this tendon the Achilles tendon (‘Chorda Achilles’) [151]. To this day, the Achilles tendon remains vulnerable to damage and disease. This thesis discusses the most significant injury of this tendon in modern-day: Achilles tendon rupture (ATR).

Injury definition

“This tendon, if bruised or cut, causes the most acute fevers, induces choking, deranges the mind and at length brings death” – Hippocrates

The first case of ATR was described by the French Surgeon Ambroise Paré in 1575 [8]. Several case descriptions followed including those of French surgeon Jean Louis Petit in 1736 [120] and Swedish surgeon Olof af Acrel in 1759 [3]. In 1959 Arner & Lindholm described an exceptionally large series of 92 cases of Achilles tears diagnosed as “partial” ruptures [15]. All cases were explored surgically and only one could truly be classified as a “partial” rupture [15]. The existence of the partial rupture was further questioned by Austrian surgeon Schönbauer in 1960 [134], who observed 240 cases of Achilles tendon tears and concluded the rupture is always complete. It is therefore thought that partial ruptures of the Achilles tendon are a different clinical entity, not the result of trauma, but perhaps purely associated with the process of degeneration. This thesis focuses on total ATRs and defines this injury as a complete loss of continuity of the Achilles tendon.

Epidemiology and etiology

“It is pointed out that the degenerative process described as occurring in the supraspinatus tendon and the tendon of the long head of the biceps muscle probably occurs in varying degrees in other similar structures in other parts of the body.” [158] – C. Laird Wilson & G. Lyman Duff, 1943
The incidence of ATRs seems to be increasing in the Western world [49, 59, 63, 83, 138]. The earliest and only epidemiological figures in the Netherlands date back to the doctoral dissertation of Johannes de Jong, defended at the University of Groningen in 1966 [65]. De Jong surveyed all large \( n = 17 \) Dutch hospitals on the frequency of ATR treatment between the years 1955-1965. The responding centers stated that they treated 1-2 ATR patients per year. In the same dissertation it was concluded that ATR occurs predominantly in middle-aged males and usually present during recreational physical activity [65]. These findings are confirmed by subsequent international studies: there is a male predominance and the largest increase in incidence is seen in the middle-aged [32, 49, 59, 63, 83, 86, 94, 108, 126, 138]. The most recent figures in Canada and Scandinavia show an incidence of 29.3/100,000 and 31.2/100,000 respectively [49, 138]. The average age of rupture is around 42 years [55] and there is a bimodal age distribution with a peak in the fourth decade and a lesser peak between the sixth and eighth decade [87, 94, 103]. Patients can therefore be classified into two subgroups: young or middle-aged athletes and older non-athletes [69, 103, 126]. The increasing incidence in the former is likely the result of increased participation in recreational sporting activities. Of all tendon ruptures, ATR is the most closely associated with sport [69], especially explosive sports are implicated (basketball, volleyball, badminton, netball etc.) [59, 94, 112, 126].

Several predisposing factors to ATR have been proposed and these include higher body mass index, middle-age, medications (fluoroquinolones, corticosteroids), and decreased tendon fibril size [7, 30]. Overall, two theories on the etiology of the ATR exist: the degenerative and the mechanical theory. The degenerative theory states that chronic degeneration eventually leads to tendon rupture under normal physiological loads [40]. This theory is supported by early experiments that showed that ruptures mostly occurred in the areas with limited blood supply [14, 82]. Additionally, in the histological analysis of ruptured Achilles tendons various stages of degeneration and tendinopathy can be identified [28, 68, 80, 93]. The mechanical theory states that tendon failure is the result of biomechanical forces and uncoordinated muscle contractions [40]. Most likely, the etiology is multifactorial resulting from the combination of degenerative pathology and uncoordinated biomechanics as a result of inconsistent training. This explains the population at risk: the “weekend warriors” who sustain an ATR during recreational activity as a sequel to a sedentary lifestyle. ATR thus has consequences for the population seeking to maintain functional ability and improve health in the context of healthy-ageing.

**Theoretical framework of this thesis**

“Lifestyle, food patterns and environmental factors influence the development of health. However, new knowledge is required about the influence of these factors, and how they interact with one another.” – University Medical Center Groningen on Healthy ageing

According to the current definition of the World Health Organization (WHO) proposed in 1948, health is defined as “a state of complete physical, mental, and social well-being
and not merely the absence of disease of infirmity” [50]. This definition emphasizes the biomedical aspects of disease or disability but disregards the concept of human functioning as a result. Several alternative definitions have been proposed such as: “the definition of health as the ability to adapt and self-manage, in light of the physical, emotional and social challenges of life” [61]. Disease or disability itself is not necessarily a predictor of work/activity impairment, social integration, and performance/complaints in daily living. By adapting to illness, people are able to feel healthy, despite biomedical limitations [61]. To provide a more holistic, comprehensive, framework to classify health and health-related domains, the WHO proposed the International Classification of Functioning, Disability and Health (ICF, Figure 1) [159]. The ICF is a classification method that provides an integration of medical and biopsychosocial models and synthesizes various perspectives of health (biological, individual and social), appreciating the interaction between health conditions (i.e. diseases and disorders) and contextual factors (i.e. environmental and personal factors) [159]. The ICF expands upon typical medical classifications and focuses on functioning. According to this model, the impact of a health condition is influenced by three interacting components: body structure and functions, activities, and participation. In this thesis the theoretical framework provided by the ICF is used to describe and structure the management and recovery after ATR, focusing on the components tendon structure, body functions and activities and participation.

Structure of the Achilles tendon

“In validissimum & crassum degenerat tendinum” (In very strong and thick it [parts of the triceps surae] degenerates from the tendons [152] – Andreas Vesalius, 1543

Anatomy of the Achilles tendon

The Achilles tendon is the thickest and strongest tendon in the human body. Its origin begins in the midline of the calf, as a common tendon connecting the triceps surae (the soleus and gastrocnemius muscles) with the calcaneal bone [38, 146]. The tendon consists of fascicles that begin proximal, originating from the medial head of the gastrocnemius on the posterolateral side (superficial) and the lateral head of the gastrocnemius on the anterior side (deep) [146]. These gastrocnemius fibers surround the fascicles originating from the soleus which join the tendon-fiber complex halfway, connecting centrally and medially [146]. As the fibers descend to the insertion, the individual fascicles twist around each other; the resulting tendon torsion is thought to effect biomechanical properties and risk of injury [119].

The Achilles tendon generally has a poor vascularity throughout its length [5]. The posterior tibial artery is the predominant artery, providing blood supply mostly to the proximal and distal portions by means of the surrounding tissue and the (highly vascular) paratenon [5]. Small contributions to blood supply are made by the peroneal artery, which supplies the middle (mid-portion) [38]. This mid-portion (2-6 cm above the insertion) is considered to be the least vascularized, leading clinicians and researchers to assume it is most prone to injury [5, 147]. It is indeed also the most frequent site of ATR [9].
Travelling along with the blood vessels are the nerve fibers of the Achilles tendon [10, 143]. Most of these nerve fibers do not penetrate into the tendon itself, but instead terminate at surrounding connective tissues (paratenon, epitenon, endotenon) [1]. The sensory innervation is supplied by nerves of the attaching muscles and cutaneous nerves [144]. Specifically the sural nerve on the lateral aspect of the tendon is involved, with a minor contribution coming from the motor nerve of the triceps surae, the tibial nerve [38, 144].

The nerve, blood and lymphatic vessels are contained by bundles tightly packed parallel arrays of mainly type I collagen and elastin encompassing the Achilles tendon [67, 92, 113]. The collagen fibrils are grouped together into a collagen fiber [67]. These fibers are bundled by endotendon to form the construct (macroscopically) termed as the tendon. This in turn is enclosed by epitenon, which is further enveloped by paratenon [38]. The paratenon functions as a sleeve allowing free movement against resistance and surrounds the Achilles tendon on the dorsal, lateral and medial sides [67].
**Tendon failure**

The well-developed Achilles tendon is thought to have played a key role in the evolution of human locomotion [22, 96]. The tendon does not occur in our immediate ancestors, great apes [76], and gives us a biological advantage. It is said the lengthening of the Achilles tendon facilitated energy storage and bipedal terrestrial locomotion in humans [22]. The Achilles tendon allows for storage of elastic energy, facilitating the process of propulsion by recoil, required for running [72]. This elastic energy storage increases running speed and reduces the (metabolic) costs associated with running [135]. As a result of its essential role in running, the human Achilles tendon developed into an exceptionally thick tendon, capable of tolerating large stress [73]. During activities such as running and jumping the Achilles tendon is subjected to repetitive high loads of up to 12 times body weight [77, 78]. The Achilles tendon responds and adapts to this mechanical load [111]. The mechanoresponsive tenocyte senses changes in tendon load and initiates a cascade of responses [17, 33, 111]. Literature shows the collagenous matrix ‘loosens’ [46, 95, 122, 136] and inflammatory markers are increased [48, 81, 85, 89, 99], resulting in a more pathological tendon structure as a result of overuse. On the other hand, understimulation (chronically low mechanical loads, i.e. physical inactivity) is also believed to induce degenerative abnormalities [34]. The response to load becomes especially complex on an individual level as it varies depending on factors such as (epi)genetics, adiposity, age, systemic disease, medication, recovery time, biomechanics etc.) [47, 95, 100, 128, 141]. Ageing, for example, alters tendon structure by resulting in a decreased undulating pattern of the collagen fibrils and a decrease in elasticity [75]. This interplay between load, degeneration and individual factors is complicated even more by the limited regenerative capacity of the Achilles tendon [53, 54]. Data using Carbon-14 tracing showed the Achilles tendon is limited in regeneration, especially at the core [53, 54]. It is hypothesized that as a result of (years of) accumulating (micro)traumata and cellular changes combined with the interplay between degeneration and lack of regeneration, the tendon eventually fails, resulting in ATR [45].

After injury, ATR results in increased stiffness and histological degeneration consisting of extracellular matrix disorganization and redistribution of cellular alignment [102]. Repair is characterized by changes in tendon composition and morphology, with thickening and restructuring of elastic properties and increased and altered metabolic activity and vascularization [2, 42, 132]. The early increase in metabolic activity and vascularization is thought to be related to long-term patient-reported and functional outcomes [123, 145]. Throughout the recovery phase, new tendinous tissue is formed and the continuum of severed ends are restored [148]. Nevertheless, the tendon remains thickened for years after injury with persisting changes in the dorsal paratenon and disorganized internal structure [79] and musculoteninous fatty degeneration [52].

**Ruptures of the Achilles tendon: diagnosis and management**

**History and clinical examination**

Patients who sustain an ATR present with sudden pain and/or functional impairment, a feeling of being hit in the calf, an audible snap and tenderness [21]. Because several other tendons (metatarsal flexors, tibialis posterior, peronei, plantaris) are usually still intact, active
plantar movement of the ankle is still possible and patients can still walk [21]. Despite the
typical patient population and clear history and clinical presentation, the diagnosis of ATR is
often missed resulting in delayed treatment and high negligence claims [129].

In 1962 the American orthopedic surgeon Theodore Campbell Thompson described a
test for diagnosis of ATR [149]. The examiner squeezes the calf muscle of a patient lying
prone or kneeling upon a stool or chair. An intact tendon (and soleus muscle) should
produce plantarflexion. If no anatomical motion of the foot is seen, the test is positive
and usually indicative of an ATR (96% sensitive and 93% specific) [91, 149]. Several
years earlier in 1957, the British surgeon Franklin Adin Simmonds described a similar test
[140], combining the squeezing maneuver with the degree of equinus of the injured foot.
To this day, the Simmonds-Thompson test is the standard clinical test used to diagnose
ATR. This test is not 100% specific, as several other muscles (plantaris, flexor halluces
longus, flexor digitorium longus and the tibialis posterior) can plantarflex the foot, even
in the absence of Achilles tendon continuity with the tibialis surae [143]. Several other
diagnostic tests can therefore be applied for diagnostic confirmation. The Matles test
[98] introduced in 1975 observes the foot position with the knee in 90 degree flexion.
In patients without an ATR the foot is held in plantarflexion, and any degree of falling
into neutral or dorsiflexion is considered indicative of an ATR. Lastly, Maffulli described
the method of palpation of the tendon gap along the course of the tendon [91]. Several
other tests, such as the Copeland test which requires a sphygmamometer cuff, and the
O’Brien test which involves inserting a 21-gauge needle, have been described [35, 118].
These tests are generally not applied due to the invasive character [91]. In addition to
proper history-taking and physical examination, it is therefore recommended to use
the Simmonds-Thompson test, the Matles test and palpation of the tendon gap. Each
individual test has adequate diagnostic accuracy and the sensitivity/specificity figures
approach 100% when all three tests are combined [91], perhaps explaining the limited
use of imaging in the diagnostic phase.

Imaging
Both ultrasound and magnetic resonance imaging (MRI) can be used for the diagnosis
of ATR. The Achilles tendon is easy to visualize sonographically and, next to structural
damage to the tendon, even small effusions can be identified [107, 127]. The main
disadvantage of ultrasound, however, is the degree of operator dependency in applying
transducer positioning and tilt [127]. MRI is proven to be useful for accurately depicting
the anatomy of the Achilles tendon and showing pathological changes within the tendon,
as a result of trauma or overuse [121]. Compared to ultrasound however, MRI is less
dynamic, more time-consuming, less readily available and more expensive. Despite the
proven ability of imaging to show Achilles tendon pathology its role in the diagnosis of
ATR is inconclusive and diagnostic accuracy figures are lacking [29]. In addition to the
diagnostic application, both ultrasound and MRI can be used in the recovery phase and
show long-term tendon pathology [4, 64, 71, 104], but the role in predicting outcome or
monitoring healing is unknown.
Several advanced, novel, ultrasonographic imaging techniques to assess the Achilles tendon, such as ultrasound tissue characterization (UTC) have been introduced recently [133]. UTC uses a probe that is fit to a construct that moves automatically over the Achilles tendon collecting images at 0.2 millimeter fixed intervals [133]. This method standardizes operator dependent variables such as transducer tilt, angle and depth and provides an objective method of ultrasound visualization. A 3-D tendon view is created and the quality of the tendon structure is quantified into four echo-types (I-IV) from fibrillar (I) to amorphous (IV) [133]. UTC is a potentially objective imaging method but its use in assessing the Achilles tendon after ATR is unknown and lacks standardized methodology for administration.

Treatment
The aims of treatment of ATR are to restore tendon structure and function and to facilitate (prompt) return to pre-injury levels of activity participation. Treatment can be broadly divided into non-surgical and surgical. The basis for non-surgical treatment was set by Alexander Monro (1697-1767) who constructed a strap that allows for the adjustment of the degree of plantarflexion in order to oppose tendon ends [105]. To this day, non-surgical treatment often consists of initial immobilization in a non-weight bearing cast in 45 degree plantarflexion (equinus) for the initial 2-4 weeks [29, 88], although numerous protocols have been described and no consensus exists. Currently, plantarflexion is gradually regressed to neutral position using either weight-bearing or non-weight bearing (plaster cast) orthoses.

Open surgical repair was popularized in 1888 by French surgeon Gustave Polaillon [26]. With this procedure tendon ends are sutured through a direct posteromedial incision exposing the rupture site and stumps. A modern alternative technique is to suture the tendons together via small stab incisions through the skin (“percutaneous technique”) without directly exposing the site of rupture. This technique was first introduced by Ma and Griffith in 1977 as a response to wound complications following open repair [90]. More recently, minimally-invasive techniques have received increased attention, with the introduction of several suture guiding devices to assist in the percutaneous suturing [6, 16].

The discussion about the best treatment modality, surgical or non-surgical, is still vivid. Quenu and Stoianovich presented the first major case-series in 1929, comparing surgical and non-surgical treatment of ATRs [125]. Since then, an abundance of observational and randomized controlled studies comparing surgical and non-surgical treatment of ATRs followed, resulting in the publication of numerous systematic reviews and meta-analyses [36, 43, 56, 74, 114, 142, 155, 161]. Most studies comparing or observing ATR treatment focused on re-rupture rate as the primary outcome, which is a measure that expresses the biomedical aspects of injury but misrepresents structure, function, patient-reported deficits and consequences for return to participation. The most recent meta-analysis showed surgical treatment is associated with a slightly lower risk of re-ruptures (1.6%), but a slightly higher risk of other complications (infection, nerve injury, etc.) [114]. These complications are dependent on the technique of primary treatment. Traditionally, non-
surgical treatment was chosen for older patients or those with lower functional demands. However, with the introduction of newer weight-bearing orthoses there is increasing evidence for first-choice non-surgical treatment in the ATR population [62, 84]. This has resulted in a decline in the number of surgically treated ATRs. Moreover, non-surgical treatment has even become the primary treatment method of ATR patients in Scandinavia and Canada [49, 137]. Despite this trend, the popularity of non-surgical treatment among Dutch clinicians treating ATRs is unknown. Additionally, the methods of rehabilitation and monitoring recovery after treatment of ATRs in the Netherlands are unknown.

Because of the minimal difference in outcome between surgical and non-surgical treatment, increasing emphasis is placed on the rehabilitation phase [25, 97]. Especially functional rehabilitation is gaining popularity. This technique uses braces where braces that gradually decrease the degree of plantarflexion are used; thus allowing for early weight-bearing. Functional rehabilitation shows promising results and might have a greater contribution to successful recovery than primary (surgical/non-surgical) treatment [36, 97, 161]. Early weight-bearing is associated with an earlier return to work/sport and higher patient satisfaction, without increasing the complication rate [74, 97].

**Functional recovery after ATR**

“At the beginning of the disease we must foretell that it will never be so cured, and that some relics may remain...” [8] – Ambroise Paré 1575

*Musculotendinous function after ATR*

ATR results in major functional deficits such as reduced range of motion, soleus and gastrocnemius atrophy, reduced strength and endurance and altered gait and biomechanics [41, 44, 51, 52, 101, 106, 131]. Although improving gradually throughout rehabilitation, the improvement of musculotendinous function seems to come to a halt around two years post-injury [23, 115] and several limitations persist for up to 10 years after ATR [23, 58, 79, 115]. Early after injury, there is significant tendon elongation [66], which continues up to six months post-injury [41]; this is especially significant in non-surgically treated patients [31, 51, 52, 106]. Lengthening of the tendon reflects increased dorsiflexion [31], and decreased end-range plantarflexion [24, 106, 130, 157] and is related to unfavorable muscle strength, biomechanics [39, 51, 139] and patient-reported symptoms [27].

Tendon elongation and several of the functional deficits are thought to be the result of sub-optimal rehabilitation [58], and an important aspect of restoring function is individualizing both short– and long-term rehabilitation based on the individual patient's capacity and goals [23, 25]. Given the early elongation and decline of improvement in the second year after injury, it is thought that rehabilitation should be maximized early on, preferably within the first year after injury [115, 156]. This is challenging given the large variation in individual outcomes, inconclusiveness concerning predicting factors [20, 37, 116] and lack of knowledge on the recovery after ATR [58, 106].
Activities and participation after ATR

“Why is it that the patient is more often right than the doctor?” [140] – Franklin Adin Simmonds, 1957

Patient-reported outcome

ATR patients report subjective deficits and reduced quality of life throughout the entire recovery phase, persisting up to seven years after injury [18, 44, 109]. One year after initial injury, almost a quarter of patients report problems in domains other than function, such as pain and discomfort, anxiousness, mobility and self-care [44]. Nevertheless, longitudinal data concerning patient-reported outcomes and deficits after ATR are scarce. For the assessment of patient-reported deficits and functional limitations, only one valid and reliable disease-specific patient-reported outcome measure exists: the Achilles tendon Total Rupture Score (ATRS) [110]. This questionnaire has been proven valid and reliable in several languages, including Dutch (ATRS-NL) [117]. Despite proven validity and reliability in assessing outcome after treatment, data on the ability of the ATRS(-NL) in monitoring change throughout recovery (responsiveness) are lacking, limiting its applicability in clinical follow-up and longitudinal (recovery-related) research.

Additionally, despite known deficits in widespread health-related domains, the association between ATR and psychological factors such as fear of movement and re-injury and decreased tendon satisfaction is unknown. The patient-reported psychological consequences of injury have been extensively studied in other sport-related injuries such as anterior cruciate ligament ruptures [60], but data after ATR is lacking.

Return to participation in sports and work

Recovery and return to sporting activities after ATR is a tedious process. Literature shows the time to return to sport (RTS) is unpredictable and often takes more than six months [29, 162]. For yet unknown reasons, approximately 20% of all (recreational) athletes do not RTS at all after ATR [162]. In professional athletes, this rate is even poorer, 30% retire and those that do return play significantly less matches/games and show inferior performance in the first two years after injury [150]. Of all (surgically treated) sports injuries, ATR has the worst RTS rate [101]. Perhaps this is due to the psychological consequences of the injury. Research on anterior cruciate ligament ruptures show that, despite acceptable functional outcomes, patients are burdened with fear of movement (kinesiophobia) and fear of re-injury, and they dread undergoing a repeated long rehabilitation [11–13]. This loss of physical activity may have extensive consequences, such as a predisposition for an unhealthy lifestyle, potentially resulting in an increased risk of chronic disease [19]. Additionally, although this has not been previously assessed, it is assumed that failed RTS negatively influences other outcomes such as quality of life, especially given that ATR is an injury mostly sustained by the recreationally active population. Besides compromising physical activity and RTS, ATR also greatly influences return to work [44]. After initial injury patients are burdened with sick leave of on average 3-15 weeks [97, 114]. Return to work data, however, is limited as only a few studies report on it, and those that do report
insufficient information [114]. Especially data on work productivity and performance is lacking; it can be hypothesized that, similar to tendinopathic injuries of the lower extremity (patellar and Achilles tendinopathy) [57, 153, 160], work performance is reduced after ATR, especially in those performing physically demanding jobs. The latter is complicated by the fact that physical work itself is a risk factor for tendinopathic abnormalities, further exacerbating the effect of ATR on work performance on a population level [154, 160].

In general, most studies evaluating ATR treatment and outcome focus on re-rupture and complication (rate) [36, 43, 56, 74, 114, 161] and disregard the effect of injury on structure, functioning and activities and participation. Additionally, the interaction between these various domains has not been described, and is essential in counseling patients and drafting treatment protocols.

**Aims of the thesis**

The general aim of this thesis is to gain insight into clinical practice and outcome of ATR. The first aim was to evaluate current ATR management and to determine the role of imaging. The second aim was to investigate the outcome after ATR considering the different components of the ICF model, i.e. tendon structure, body functions and activities and participation.

**Outline of the thesis**

This thesis is divided in two parts.

**Part I: Achilles tendon rupture: imaging and clinical practice**

The management of ATRs is addressed in chapters 2-4. In chapter 2 a nationwide survey study on the management of ATRs by surgeons in the Netherlands is described. In chapter 3 a systematic review on the role of imaging in diagnosis and treatment monitoring of ATRs is presented. Chapter 4 expands on the role of imaging and provides a systematic review on the role of a novel ultrasonographic imaging technique, UTC, for assessing tendon structure and pathology.

**Part II: Outcome after Achilles tendon rupture**

The outcome after ATR is addressed in chapters 5-9. Chapter 5 encompasses the protocol of a multicenter prospective cohort study aiming to gain insight into the recovery after ATR using the ICF framework. Chapter 6 assesses the responsiveness (ability to detect change over time) of the only valid and reliable disease-specific patient-reported outcome measure used after ATR, the ATRS-NL. Functional and patient-reported outcomes in terms of physical functioning and participation in sport are evaluated in chapter 7. Chapter 8 provides insight into the psychological factors during the rehabilitation process after ATR. Chapter 9 presents the results of novel imaging (UTC) in assessing Achilles tendon structure after ATR. Chapter 10 provides the general discussion, in which the results of the studies in this thesis are examined and theoretical and practical implications and recommendations for future research are presented.
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Part I

Achilles tendon rupture: imaging and clinical practice