Juuso Heikkinen

RECOVERY OF CALF MUSCLE ISOKINETIC STRENGTH AFTER ACUTE ACHILLES TENDON RUPTURE
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Abstract

Achilles tendon rupture (ATR) conservative treatment result usually good clinical outcome, but despite the treatment method calf muscle strength deficit persist. Recent evidence suggests that surgery might surpass conservative treatment in restoring strength after ATR, but structural explanations for surgery-related improved strength remain uncertain.

The purposes of this thesis were to compare calf muscle isokinetic strength recovery, calf muscle volume, fatty degeneration and AT elongation after conservative treatment or after open surgical repair of ATR. An additional aim was to assess the role of fascial augmentation in terms of calf muscle isokinetic strength recovery, AT elongation, calf muscle volume atrophy and fatty degeneration, and their relationship with calf muscle isokinetic strength in long-term follow-up after ATR surgery.

Surgery resulted in 10% to 18% greater plantar flexion strength (P = 0.037) compared to conservative treatment. The mean differences between affected and healthy soleus muscle volumes were -18% after surgery and -25% after conservative treatment (P = 0.042). At 18 months, AT were, on average 19 mm longer in patients treated conservatively compared to surgery (P < 0.001). At 18 months, patients with greater (2–3) fatty degeneration had lower soleus muscle volumes and plantar flexion strength in the healthy leg.

In long term, augmentation did not affect any of the strength variables, but the injured side showed 12% to 18% strength deficit compared with the healthy side (P < 0.001). The AT was, on average, 12 mm longer in the affected leg than in the healthy leg (P < 0.001). The mean soleus muscle volume was 13% lower in the affected leg than in the healthy leg (P < 0.001). The mean volumes of the medial- and lateral gastrocnemius muscles were 12% and 11% lower in the affected leg than in the healthy leg, respectively (P < 0.001). AT elongation correlated substantially with plantar strength deficit (ρ = 0.51, P < 0.001) and with both gastrocnemius (ρ = 0.46, P = 0.001) and soleus muscle atrophy (ρ = 0.42, P = 0.002). Calf muscle fatty degeneration was more common in the affected leg compared healthy leg (P ≤ 0.018).

In conclusion, surgery of ATR restored calf muscle isokinetic strength earlier and more completely than conservative treatment. Conservative treatment resulted in greater soleus muscle atrophy and AT elongation compared surgery, which may partly explain the surgery related better strength results. Augmentation provided no long-term benefits compared with simple sutureation, and a 12 to 18% plantar flexion strength deficit compared to the healthy side persisted. AT elongation may explain the smaller calf muscle volumes, greater fatty degeneration, and plantar flexion strength deficit observed in long-term follow-up after surgical repair of ATR.

Keywords: Achilles tendon rupture, compensatory hypertrophy, conservative treatment, early weight bearing, elongation, fatty degeneration, functional rehabilitation, long-term result, muscle volume, operative treatment, tendon length
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Oulun yliopiston tutkijakoulu; Oulun yliopisto, Lääketieteellinen tiedekunta; Medical Research Center Oulu; Oulun yliopistollinen sairaala
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Tiivistelmä
Akillesjännerepeämän (ATR) konservatiivisella ja leikkaushoidolla hoidolla saavutetaan hyvät kliiniset tulokset. Viimeisimmät tutkimukset kuitenkin viittaavat leikkaushoidolla saavutettavan paremmat voimat kuin konservatiivisella hoidolla, mutta rakenteelliset selitykset leikkaushoidon paremmalle pohjelihaksen voimille ovat epäselviä.

Työn tarkoituksena oli verrata pohjelihaksen isokineettisten voimien palautumista, pohjelihastilavuuksia, rasvadegeneraatiota ja akillesjänteen (AT) pidentymistä ATR:n konservatiivisen ja leikkaushoidon jälkeen. Tarkoituksena oli arvioida lihaskalvovahvikkeen merkitystä pohjelihaksen isokineettisten voimien palautumisessa pitkäaikaisseurannassa. Lisäksi tutkimme ATR:n pidentymisen, pohjelihastilavuuksien ja rasvadegeneration seurattua pohjelihaksen isokineettisiin voimiin ATR:n leikkaushoidon jälkeen 14 v seurannassa.

Leikkaushoidolla saavutettiin 10–18 % paremmat pohjelihaksen voimat verrattuna konservatiiviseen hoitoon. Leikkaushoidon jälkeen soleuslihasten tilavuusen puoliero terveen jalan hyväksyi olisi 18 % ja konservatiivisen hoidon jälkeen 25 %. 18 kk kohdalla konservatiivisesti hoidettujen AT oli 19 mm pidempi verrattuna liikkaushoidolla hoidettuihin. 18 kk kohdalla potilaat, joilla vamma jalan soleuslihaksen rasva-degeneraatio oli korkea (2–3), kärsivät suuremmasta soleuslihaksen atrofiasta ja pohjelihaksen voima puolierosta.


Asiasanat: aikainen painonvaraus, akillesjännerepeämä, funktionaalinen kuntoutus, jänteen pituus, jänteen venyminen, kompensatorinen hypertrofia, konservatiivinen hoito, leikkaushoido, lihastilavuus, pitkäaikaistulos, rasvadegeneraatio
To my family
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Oulu, September 2017

Juuso Heikkinen
## Abbreviations

<table>
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<th>Abbreviation</th>
<th>Description</th>
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<tr>
<td>AT</td>
<td>Achilles tendon</td>
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<tr>
<td>ATR</td>
<td>Achilles tendon rupture</td>
</tr>
<tr>
<td>ASPT</td>
<td>Angle specific peak torque</td>
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<tr>
<td>BMI</td>
<td>Body mass index</td>
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<tr>
<td>CT</td>
<td>Computer tomography</td>
</tr>
<tr>
<td>CSA</td>
<td>Cross-sectional area</td>
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<tr>
<td>CI</td>
<td>Confidence interval</td>
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<tr>
<td>EMG</td>
<td>Electromyography</td>
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<tr>
<td>FHL</td>
<td>Flexor hallucis longus</td>
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<tr>
<td>LMM</td>
<td>Linear mixed model</td>
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<tr>
<td>MRI</td>
<td>Magnetic resonance imaging</td>
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<tr>
<td>MTJ</td>
<td>Myotendinous junction</td>
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<tr>
<td>PT</td>
<td>Peak torque</td>
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<tr>
<td>RCT</td>
<td>Randomized controlled trial</td>
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<tr>
<td>ROM</td>
<td>Range-of-motion</td>
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<tr>
<td>RSA</td>
<td>Roentgen stereophotogrammetric analysis</td>
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<td>US</td>
<td>Ultrasound</td>
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1 Introduction

Recent systematic meta-analyses and reviews of high quality randomized controlled trials (RCTs) comparing conservative treatment and surgery for acute Achilles tendon rupture (ATR) have shown that surgery offers no benefits in terms of functional scores and re-rupture rate (Erickson et al. 2015, Holm et al. 2015). Conservative treatment with functional orthosis and early weight bearing has therefore become the method of choice for acute ATR (Hutchison et al. 2015, Lawrence et al. 2017, Sheth et al. 2017). Good clinical outcomes usually result, but surgery and conservative treatment both have the potential to leave the patient with up to 30% smaller calf muscle strength compared to the unaffected side (Keating et al. 2011, Nilsson-Helander et al. 2010, Olsson et al. 2013). Recent RCTs comparing conservative and surgical ATR treatment suggest that surgical treatment may lead to faster and more complete calf muscle strength recovery compared with conservative treatment, but the results are conflicting (Keating et al. 2011, Nilsson-Helander et al. 2010, Olsson et al. 2013).

The reasons that lead to calf muscle strength deficit after ATR are poorly understood. Only two studies have suggested possible structural explanations for plantar flexion strength deficit after conservative and surgical treatment of ATR (Rosso et al. 2013, Schepull et al. 2012). Despite extended debate, Achilles tendon (AT) elongation is one explanation, albeit minimally studied, for calf muscle strength impairments. Previous studies have confirmed a correlation between isokinetic calf muscle strength and heel rise height deficits with AT elongation after surgical ATR treatment (Kangas et al. 2007, Pajala et al. 2009, Silbernagel et al. 2012). As conservative ATR treatment has shifted in a more aggressive direction, concerns have been raised regarding the significant lengthening of the AT during functional conservative treatment (Lawrence et al. 2017, Schepull et al. 2012). Schepull et al. (2012) found no difference in tendon elongation between operative and conservative study groups in their randomized controlled trial with an 18-month follow-up. Rosso et al. (2013) found that, regardless of the treatment method, affected legs exhibited elongated ATs, measured by MRI. A lack of standard rehabilitation protocols is the main weakness of previous comparative trials. The current literature lacks high quality studies of tendon elongation after conservative treatment or surgery of ATR, with subsequent identical modern rehabilitation protocols.

In addition to AT elongation, several muscle related factors, such as muscle volume atrophy, fatty degeneration, and reduction in neural input in calf muscles...
due to immobilization, are proposed to explain calf muscle plantar flexion strength deficits after ATR (Olsson et al. 2013, Rosso et al. 2013, Stevens et al. 2006). These structural changes and their relationship to plantar flexion strength after ATR are poorly investigated, especially when comparing conservative treatment and surgery. Previous studies used computed tomography (CT) to estimate calf muscle atrophy from maximum cross-sectional areas (CSAs) of the muscle. (Hagmark et al. 1979, Leppilahti et al. 2000). Recently, a more accurate method, based on measuring actual muscle volume with magnetic resonance imaging (MRI) was presented (Rosso et al. 2013). This retrospective study found a mean volume deficit of 17% and substantial fatty infiltration in the calf muscle in the affected leg after ATR, but the results did not differ between the conservative and surgical treatments.

Only a few studies have reported long-term outcomes for surgical repair of ATR (Lantto et al. 2015, Rosso et al. 2013). Previously used fascial augmentation of the repair is proposed to lower re-rupture rates and prevent tendon elongation when compared to a simple suture technique. Therefore, augmented repair could result in better calf muscle strength. Calf muscle isokinetic strength improves up to 10 years of follow-up after ATR surgery, but data on long term recovery is limited (Kangas et al. 2007, Lantto et al. 2015, Pajala et al. 2009). Three retrospective studies with 3 to 10 years of follow-up after ATR have previously reported up to a 6 to 18% deficit in isokinetic peak torque (PT) regardless of treatment method (Horstmann et al. 2012, Leppilahti et al. 2000, Rosso et al. 2013). The only long term follow-up study using prospectively collected data reported a 5 to 8% isokinetic strength deficit with only minor improvements from 1 to 11 years for surgically treated ATR patients (Lantto et al. 2015).

The main goal of this thesis was to compare isokinetic calf muscle strength recovery after surgery or conservative treatment for ATRs, and to assess the long-term calf muscle strength recovery of surgically treated patients. Secondary aims were to assess whether AT elongation, calf muscle volume, and fatty degeneration, measured with MRI, could explain the difference in plantar flexion strength between conservative and operative treatment of ATR and the long-term strength deficit after ATR operative treatment.
2 Review of the literature

2.1 Anatomy of Achilles tendon and calf muscles

The calf muscle acts to plantarflex the ankle joint via its conjoint tendon, the AT (Fig. 1). The AT consists of the tendons of the gastrocnemius and soleus muscles, and its purpose is to transmit the power of these muscles to the heel bone and to produce knee flexion, tibiotalar flexion, and subtalar inversion. The lengths of the AT fibers vary from 11–26 cm in the gastrocnemius part and from 3–11 cm in fibers from the soleus (Cummins et al. 1946). The AT is circular in shape at its midpoint, and it is wider at the osteotendinous junction on the heel bone. The tendon fibers turn rotationally, so that the fibers from the posterior gastrocnemius rotate anterolaterally, whereas the anterior soleus tendon fibers run posteromedially (Cummins et al. 1946, Pekala et al. 2017). The AT is not simply straight in its microarchitecture, as the collagen fibers and fibrils of the tendon assume a wavy configuration when the tendon is unstretched. If the tendon is stretched by 2 %, this wavy configuration disappears; more than 4 % stretching causes irreversible changes. Stretching to more than 8 % causes macroscopic failure and tendon rupture (O’Brien 1992). The collagen content of the AT, on a dry weight basis, is about 70 % (Jozsa et al. 1989), and most of the collagen is type I collagen. A small amount of type II collagen is found in the osteotendinous junction, whereas types III, IV, and V are found at the end of the tendon (Kannus & Jozsa 1997). Metabolism can speed up after injury, but immobilization decreases collagen synthesis and increases its degradation, thereby reducing tendon strength (Karpakka et al. 1991). Over the years, the tensile strength of the healthy AT decreases, reaching about 40 % of its value by the age of 70 years when compared to 30 years (Barfred 1973, Thermann et al. 1995).

The calf muscle (triceps surae) consists of the gastrocnemius and soleus muscle (Fig. 1). The soleus lies deep in the gastrocnemius and is a broad, pennate muscle (Moore et al. 1999, Shaw et al. 2008). It is a large flat muscle that was given its name from its resemblance to the sole, a flat fish (Moore et al. 1999). It originates from the oblique line and the middle third of the medial border of the tibia, from a fibrous arch between the fibula and the tibia, and from the posterior surface of the head of the fibula (Cummins et al. 1946, Moore et al. 1999, Standring 2009). It acts only on the ankle joint and can be palpated on either side of the gastrocnemius when the subject stands on tiptoe (Moore et al. 1999). The soleus muscle contains
mainly type I muscle fibers (Elder et al. 1982, Fitts et al. 2010, Maughan et al. 1983), making it a slow muscle that is more efficient at using oxygen for generating continuous, extended muscle contractions over a long time. Type I fibers fire more slowly than fast twitch fibers and can fire for a long time before they fatigue (Lieber 1992, McArdle et al. 1996, Thayer et al. 2000).

The gastrocnemius muscle is a fusiform muscle in which the medial head arises from the popliteal surface of the femur, posterior to the medial supracondylar line and the adductor tubercle. Both heads of the gastrocnemius cross the knee joint. The lateral head is shorter than the medial head. It originates from the posterolateral aspect of the lateral femoral condyle, from a region extending from proximal and posterior to the lateral epicondyle to the most distal aspect of the linea aspera (Standring 2009). The gastrocnemius is therefore the most superficial muscle of the dorsal aspect of the lower leg, and it gives the calf its characteristic bulge (Standring 2009). The gastrocnemius muscle contains mainly type II muscle fibers, which are considered fast twitch fibers. Type II fibers are better at generating short bursts of strength or speed than are slow muscles, but they fatigue more quickly (Lieber 1992, McArdle et al. 1996, Thayer et al. 2000).

In addition to the calf muscles, the flexor hallucis longus (FHL) and the deep flexors (the digitorum longus and tibialis posterior) in the posterior compartment are involved in producing ankle plantar flexion movement and especially when rising on tiptoe (Edama et al. 2016). Based on cross sectional area, ankle plantar flexion resides primarily in the soleus. The medial gastrocnemius contributes half the strength produced by soleus. The rest of the ankle plantar flexion function is produced by the FHL and deep flexors. However the FHL is confirmed as the strongest additional ankle plantar flexor, when compared to the deep flexors (Silver et al. 1985).
Fig. 1. Anatomy of the Achilles tendon and calf muscles according Gray’s anatomy, 2nd ed.

### 2.2 Assessment of calf muscle strength

2.2.1 Functional assessment

Silbernagel et al. (2006) have described a test battery that includes maximal heel rise height, heel rise work, heel rise repetitions, concentric and eccentric maximal power, and two different jump tests to evaluate functional results in patients with AT tendinopathy. This extensive functional test battery has been used in two recent leading RCTs comparing surgical and conservative treatment of ATR (Nillson-Helander 2010, Olsson et al. 2013). This battery seems to have a good resolution power for evaluating lower leg and calf muscle function after ATR. The heel-rise test, as described Silbernagel et al. (2010), measures the height of each heel-rise along with the number of repetitions. The maximum heel-rise height and number of heel-rises are also used when evaluating calf muscle endurance after ATR (Moller et al. 2001, Silbernagel et al. 2006). The definition of the accepted heel-rise has varied between studies and has included either a minimal height required for a successful heel-rise or an adjusted test result based on the heel-rise height of the contralateral side; otherwise, the definition for proper heel rise is not mentioned (Moller et al. 2005, Schepull et al. 2012, Silbernagel et al. 2010). In addition, during the heel-rise test, Silbernagel et al. (2010) described a test that evaluated the height of each heel-rise along with the number of repetitions and forms the heel-rise work to measure calf muscle endurance in a more sensitive manner when compared to bare heel rise repetitions. However, the weakness of the heel rise test battery is that it focuses on more plantar flexion endurance rather than the maximum strength produced at the end range of plantar flexion.

2.2.2 Isometric measurement

During isometric contraction, a muscle generates tension without changing length (Widmaier et al. 2010). The unit for isometric strength is the newton meter (Nm). One Nm is equal to the torque resulting from a force of one newton applied perpendicularly to a moment arm one meter long. The reliability of isometric strength tests is generally high, and the various testing positions for ankle plantar flexion have good test-retest reliability (Alfredson et al. 1998 and 1998). Isometric strength measurement is easy to perform and measure, but a patient performing the isometric plantar flexion strength may be able to use his body strength when producing the maximal effort. In addition, isokinetic strength measurement is considered a more physiologically mimicking normal gait cycle compared with isometric assessment (Mullanney et al. 2006).
2.2.3 *Isokinetic measurement*

During a concentric contraction, the muscle shortens and generates a force at the origin and insertion of the muscle, thereby causing a change in the angle of the joint (Widmaier *et al.* 2010). During isokinetic contraction, the muscle contracts at constant rate of speed. A device called an isokinetic dynamometer is needed to measure isokinetic strength. This device has a lever arm that moves at a constant angular velocity. The subject pushes the lever arm and the computer-guided dynamometer registers the resistance, which equals the muscular forces applied throughout the whole ROM. This method allows the measurement of the muscular forces in dynamic conditions and provides optimal loading of the muscles (Baltzopoulos *et al.* 1989).

The dynamometer produces a torque displacement diagram (Fig. 2). The highest point (y-axis) of the torque displacement diagram, the peak torque (PT), is considered the gold standard of isokinetic strength measurement (Mullanney *et al.* 2006). The PT illustrates the maximal strength without information about the angle in the ROM at which it occurs. Both isokinetic and isometric maximal plantar flexion PT are reached at 0 to 20° dorsiflexion of the ankle for healthy subjects and patients with ATR (Barfod *et al.* 2015, Frasson *et al.* 2007, Hohendorff *et al.* 2008, Mullanney *et al.* 2006, Pajala *et al.* 2009). The lack of information regarding the angle of ROM at which the PT is achieved is a problem since the PT does not reveal weakness in end-range plantar flexion, which may be an important impairment after ATR (Mullaney *et al.* 2006, Nilsson-Helander *et al.* 2010, Nistor *et al.* 1981). The area under the torque-displacement curve is the total average contraction work (in joules = J) for the measured ROM (Fig. 2), and it is considered to illustrate endurance. The weakness of total work measurement is that it tells nothing about the strength deficit locations of the joint ROM or the PT.
The test position for measuring ankle isokinetic plantar flexion strength is usually supine or sitting, and the knee angle may alter from 0° (straight) to 90° flexion (Fugl-Meyer et al. 1979, Leppilahti et al. 2000). When the knee is flexed to 90°, the PT results are 10–20% lower than the results performed with a straight knee, despite the angular velocities (Fugl-Meyer et al. 1979). The hip angle has no impact on the strength results (Fugl-Meyer et al. 1979). This phenomenon is explained by an up to 45% smaller activity of the gastrocnemius muscle when the knee is flexed in a 90° position compared to the knee in a straight position (Fugl-Meyer et al. 1979). In addition, when the knee is extended, the length-tension relationships of all parts of the calf muscle are optimal for the development of maximum force (Herman et al. 1967). Therefore, as straight knee position is suggested as the most appropriate test position when measuring the maximum ankle plantar flexion isokinetic strength.
Several angular velocities, ranging from 30 to 240°/s, can be used to measure the ankle isokinetic strength, and plantar flexion torque production is well known to decrease when the angular velocity is increased (Leppilahti et al. 2000, Moller et al. 2005, Wallace et al. 2004). The highest torque measurements have been obtained for plantar flexion at 30°/s, but higher angular velocities are used in the normal gait cycle, and functional tests that resemble activities of daily life should use higher angular velocity if possible (Boppert et al. 1990, Moller et al. 2005, Oberg et al. 1987). The study of Chester et al. (2003) indicates that ankle isokinetic plantar flexion strength measurements for an angular velocity of 60°/s results in good reliability (Chester et al. 2003).

For assessment of the ankle isokinetic plantar flexion strength over the whole ankle range of motion, Pajala et al. 2009 described the work displacement measurement, which is reformed from torque displacement diagram (Fig. 2) by calculating the work produced for each 10° interval of ankle ROM. The work displacement measurement measures the maximal produced work for each 10° interval for ankle ROM and enables the assessment of the maximal isokinetic plantar flexion strength deficit for the end range of the ankle ROM (Pajala et al. 2009) (Fig. 3 and 4).
Fig. 3. Isokinetic torque displacement diagram using ankle velocity 60°/s. The grey area illustrates the produced work (J) for 10° to 0° interval for ankle ROM as work displacement – measurement.
Another option for assessing strength over the whole ROM is to record PT at specific angles of ROM from repeated measurements (Fig. 5) and compose a new diagram, the angle specific peak torque (ASPT) curve (Fig. 6). Both work-displacement and ASPT curves enable the calculation of a precise percentage strength deficit for each angle or angle area of the ankle ROM; therefore, they are considered more accurate than functional test batteries and bare PT measurements.
Fig. 5. Torque displacement diagram for repeated (5) plantar flexion measurements. Involved side, ankle velocity 60°/s.
Fig. 6. ASPT diagram reformed from the best angle specific peak torque results from 5 repeated measurements for both uninvolved and involved side. Ankle velocity 60°/s.


2.3 The effect of immobilization and tendon injury to muscle strength

The most frequent model used to study disuse atrophy without tendon injury in humans is unilateral limb immobilization, using a brace or cast, and bed rest (Dirks et al. 2016). However, ankle joint immobilization after ankle fractures is the closest situation when evaluating calf muscle regeneration capacity after immobilization or disuse (Christensen et al. 2008, Shaffer et al. 2000, Stevens et al.)
In terms of muscle mass, the observed rate of decline in muscle size (CSA) for each day of immobilization in knee extensors was ~0.40 % and ~0.36 % for plantar flexors during a 42-day follow-up of immobilization without tendon injury (Clark et al. 2006, Hackney and Ploutz-Snyder 2012, Stevens et al. 2004 and 2006).

The biggest loss occurred in the plantar flexors, the gastrocnemius, and the soleus, where the reduction in CSA averaged between 17–24 % during 7 weeks of immobilization (Stevens et al. 2004 and 2006). Ankle immobilization for 7 weeks results in up to a 75 % deficit in isometric and a 50 % deficit in isokinetic PT plantar flexion strength when compared to the healthy leg for patients with surgically treated ankle fractures (Christensen et al. 2008, Shaffer et al. 2000, Stevens et al. 2006). In general, the degree of the rate of disuse muscle atrophy varies depending on the duration and nature of immobilization as well as on the measurement techniques. However, atrophy appears to occur more rapidly in first 3–14 days of unloading, eventually reaching a plateau where further loss of muscle occurs at a slower rate despite continued unloading of the muscle (Bodine 2013).

Shaffer et al. (2000) and Stevens et al. (2006) found that a plantar flexion strength deficit caused by 8 weeks of immobilization because of ankle fracture may fully recover within 10 weeks of supervised physical therapy. Jones et al. 2004 immobilized healthy patients’ lower limbs for 2 weeks and found that atrophied knee extensor muscle mass was recovered over 6 weeks and the observed strength deficit recovered during 4 weeks of rehabilitation (Jones et al. 2004). Stevens et al. (2006) found that, after 7 weeks of ankle cast immobilization because on ankle fracture, both the calf muscle CSA and plantar flexion PT recovered to the value on the contralateral side after 10 weeks of rehabilitation. In contrast to other studies, Stevens et al. (2004) used torque per muscle cross sectional area and found that, after 7 weeks of ankle cast immobilization because of ankle fracture, both the calf muscle CSA and plantar flexion PT failed to recover to the contralateral side values after 10 weeks of rehabilitation.

Immobilization and muscle disuse lead evidently to muscle atrophy; however, the etiology of atrophy is a complex interplay of numerous mechanisms with many unanswered open questions (Atherton et al. 2016, Rudrappa et al. 2016, Dirks et al. 2016). Miokovic et al. (2012) found that atrophy induced by muscle disuse varies within different muscles, such that the fastest atrophy rates occurred in the posterior calf muscles, specifically the gastrocnemius medialis and soleus, when compared to other lower extremity muscles. Explanations for this difference in intramuscular atrophy within different muscles have included differences in muscle size and
muscle fiber type distribution (Elder et al. 1982, Fitts et al. 2010, Maughan et al. 1983). Type I fibers predominate in the soleus, whereas type II fibers predominate in the gastrocnemius; muscle type I fibers are more vulnerable to immobilization than are type II fibers (Booth et al. 1973, Gollnick et al. 1974, Jozsa et al. 1988, Ohira et al. 2006, Thomason and Booth 1990).

The damaged and then healed AT consists of a curved pattern of thin collagen fibers with decreased type I and a greater proportion of weaker type III collagen (Kannus et al. 1991). Changes in tendon structure may also affect its biomechanical properties, which in turn affect its performance and the patient’s coordination in daily life (Beason et al. 2012, Chamberlain et al. 2013, Freedman et al. 2014). The healthy tendon can be extended because of its viscoelastic properties, which are critical for storing elastic energy and producing momentary high forces (Beason et al. 2012, Chamberlain et al. 2013, Wang et al. 2006). Schepull et al. (2015) showed in their studies that a ruptured AT after surgery has inferior viscoelastic properties when compared with the healthy contralateral tendon. An early good elastic modulus correlates with the restoration of the subject’s heel rise index at 18 months after the injury, regardless of the treatment method (Schepull et al. 2012).

Muscle atrophy with tendon injury is well described in the rotator cuff area, where the muscle atrophy is only partly reversible, and where atrophy persists despite successful repair of the ruptured tendon (Jo et al. 2016, Kuenzler et al. 2017). If the tendon remains torn, unloaded, and retracted, macrophages may switch to become profibrotic M2a macrophages and reprogram myogenic precursor cells into the adipogenic pathway. The result is an infiltration of mature adipocytes into the free intermyofibrillar and intramyofibrillar spaces (Frey et al. 2009) in a phenomenon termed fatty infiltration (Barry et al. 2013). Although reloading the dynamic musculotendinous units leads to partial recovery of the atrophy, the fatty infiltration observed before tendon repair seems to remain irreversible (Choo et al. 2014, Gladstone et al. 2007, Killian et al. 2014, Kuenzler et al. 2017). Studies on rotator cuff tendon surgery have suggested that disturbed muscular architecture, muscle atrophy, and fatty infiltration remain irreversible in chronic tendon tears even after successful repair (Kuenzler et al. 2017).

Muscle fatty infiltration and its degree in the rotator cuff muscles play a significant role in determining the functional outcome after cuff repair at both the short and long terms (Gladstone et al. 2007, Laron et al. 2012, Zumstein et al. 2008). The greater deficit in rotator cuff isokinetic strength is related to rotator cuff muscles with a greater degree of fatty degeneration, but a parallel fatty relationship to calf muscle function is unclear (Oh et al. 2010).
Muscle volume atrophy is clearly related to the loss of muscle force, but the reduction in muscle size often lags behind the loss in muscle force by up to 50%; therefore, it cannot entirely account for the decrease in muscle force (Berg et al. 1997, Deschenes et al. 2002). Reduced peripheral neural input in muscles has been proposed to account for much of the remaining force deficits after disuse or immobilization (Deschenes et al. 2002, Desplanches et al. 1997). After immobilization because of ankle fracture, plantar flexion strength mostly recovers during the first 5 weeks, while the muscle neural activation plays a major role in the early phase of strength recovery and the muscle CSA subsequently recovers to the value on its contralateral side (Clark et al. 2006, Stevens et al. 2006).

In conclusion, a healthy musculo-tendinous unit may recover completely after immobilization. However, necessary immobilization of a ruptured tendon, despite surgical repair, often leads to incomplete recovery of muscle atrophy and strength.

2.4 Calf muscle recovery after ATR

Some patients still have gait abnormalities beyond one year after ATR surgery; these can include increased dorsiflexion ROM, co-activation of the lower leg muscles, and decreased step length (Don et al. 2007). The persistence of decreased step length and plantar flexion weakness decreases physical activity and overall quality of life (Olsson et al. 2011). Based on literature to date, the first year post injury seems to be critical for calf muscle strength recovery, and regardless of the ATR treatment method, a calf muscle strength deficit of up to 30 % may persist (Keating et al. 2011, Nilsson-Helander et al. 2010, Olsson et al. 2013). Younger age, kinesiophobia, male gender, a lesser degree of symptoms, and a higher degree of physical activity are related to a better ability to perform a heel rise at 12 weeks from the injury (Olsson et al. 2014). Male gender and younger age are related to better performance in the heel rise index at 12 months post injury (Olsson et al. 2014).

Silbernagel et al. (2010) showed in their study with 78 patients that the heel rise height is affected by ATR, suggesting a weakness at the end range of plantar flexion. The heel-rise test showed that 50 % of patients were unable to perform a single heel-rise at 3 months after the injury (Olsson et al. 2014). At 6 months post injury, patients were able to perform a one leg heel rise, but the mean maximum heel-rise height was as much as 25 to 44 % lower when compared to the healthy side (Nilsson-Helander et al. 2010, Silbernagel et al. 2010 and 2012). At 12 months post injury, the maximum heel-rise height was 21–30 % lower for the affected leg.
than for the healthy leg (Nilsson-Helander et al. 2010, Olsson et al. 2014, Olsson et al. 2013, Silbernagel et al. 2012). At 2 years post injury, as much as a 17% deficit remained in maximal heel rise height in patients with ATR (Olsson et al. 2011). Moller et al. (2002) found that 8–22% of patients were unable to carry out a single heel rise on the injured side at two years after the injury (Moller et al. 2002). Moller et al. (2002) and Olsson et al. 2011 found that, at 2 years after the ATR, a 31–46% endurance deficit persisted when measuring the heel-rise repetitions and comparing the injured leg to the contralateral healthy leg.

Mullanney et al. (2006), in their study with a mean of 1.8-years of follow-up (range 6 months to 9 years), showed that patients who underwent surgical treatment for ATR suffered from isometric plantar flexion weakness on the affected side at 20 degrees and 10 degrees of plantar flexion (34 % and 20 % deficits, respectively), with no torque deficits evident at other angles. In addition, they found that maximum isometric plantar flexion torque occurred at 20 to 10 degrees of dorsiflexion in these patients (Mullanney et al. 2006). Nistor et al. (1981) and Leppilaiti et al. (2000) confirmed these results. Patients after ATR seem to suffer substantially greater isometric strength deficits in the ankle plantar when in the flexed rather than in the neutral or dorsiflexed positions.

The timeline of isokinetic strength recovery seems to center around the first year after injury (Kangas et al. 2003, Moller et al. 2002, Pajala et al. 2009). At 6 months, a 4 to 26% PT strength deficit was observed (Moller et al. 2002). At 1 year post injury, ATR patients suffered from a 2 to 25% PT strength deficit (Keating et al. 2011, Kangas et al. 2003, Moller et al. 2002, Pajala et al. 2009, Willits et al. 2010). At 2 years post injury, patients after ATR suffered from a 10 to 18% isokinetic PT strength deficit (Moller et al. 2002, Willits et al. 2010). Porter et al. (2014), studied 40 operatively treated patients with a mean of 32 months of follow-up time (range 12 to 80) and found a mean 12–18% in PT plantar flexion strength deficits for the affected leg. Leppilaiti et al. (2000) studied 85 surgically treated patients with a mean of 3 years of follow-up and found as much as a 10 % deficit in PT. Nistor et al. (1981) found an isokinetic strength deficit of 11–18 % 2.5 years after ATR. However, Bevoni et al. (2014) treated 66 patients with an identical surgical technique with functional postoperative treatment and found no significant deficit in isokinetic strength for the affected leg after 3 years of follow-up. Rosso et al., in their retrospective study with 7 years of follow-up, found a 13 % lower PT for the affected leg compared to the healthy leg, regardless of the treatment method (Rosso et al. 2015).
Only Pajala et al. (2009) and Barfod et al. (2015) have studied the isokinetic plantar flexion strength for patients with ATR for the whole ROM of the ankle. They found as much as a 50% deficit at 3 months and 40% at 12 months in the end range of plantar flexion. The results of Pajala et al. (2009) and Barfod et al. (2015) suggest that the strength deficit increases while ankle plantar flexion increases. The results of Pajala et al. (2009) and Hohendorff et al. (2008) also suggest that the angle for PT of the affected leg may be more dorsiflexed when compared with the healthy leg. These results, taken together with the earlier isometric ankle ROM strength measurements and the persistent impairment in the heel rise height after ATR, underlines the importance of measuring the calf muscle plantar flexion strength of the ankle for the whole ROM after ATR.

2.4.1 Surgery vs. conservative treatment


Three RCTs, by Nistor et al. (1981), Moller et al. (2002), and Schepull et al. (2012), found no differences in calf muscle isokinetic or isometric strengths or heel rise repetition results between the treatment methods (Table 1). By contrast, five trials, by Nilsson-Helander et al. (2010), Keating et al. (2011), Olsson et al. (2011), Olsson et al. (2013), and Willits et al. (2010), reported faster or better recovery of calf muscle strength and endurance after surgery (Table 1).

From the injury to one year post injury, a tendency is noted whereby the calf muscle strength and endurance of the affected leg recovers slightly faster (with a 7 to 15% smaller deficit) and more completely (with 9 to 17% smaller deficit) in operatively treated patients than in conservatively treated patients. However, regardless of the treatment method, the calf muscle strength does not recover to its contralateral side value during the first year post injury, resulting in as much as a 30% deficit in strength (Keating et al. 2011, Nilsson-Helander et al. 2010, Olsson et al. 2013). Willits et al. (2010) and Olsson et al. (2011) found in their prospective studies that, regardless of conservative or surgical repair of the ATR, the calf muscle strength showed only minor improvement from 1 to 2 years post injury.

Immediate full weight bearing with controlled early motion of the ankle is the gold standard for conservative ATR treatment (Barfod et al. 2015, Ecker et al. 2016, Hutchison et al. 2015). The main weaknesses of previous RCTs are the
variability in the rehabilitation programs between surgical and conservative treatments and the incomplete execution of modern functional rehabilitation programs.

Additionally, previous trials have not provided detailed evaluations of plantar flexion strength for the whole ROM of the ankle. The maximum heel rise height is unlikely to have the best resolution power for the end range plantar flexion strength difference between ATR surgical and conservative treatment.
Table 1. RCT:s comparing ATR conservative and surgical treatments calf muscle strength result.

<table>
<thead>
<tr>
<th>Study</th>
<th>Year</th>
<th>Oper/cons</th>
<th>Identical rehabilitation protocol</th>
<th>Full weight bearing for cons treatment, from the injury</th>
<th>Controlled motion of the ankle</th>
<th>Active ankle ROM for cons treatment</th>
<th>Follow up years</th>
<th>Strength measurement at final follow-up</th>
<th>Strength favor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nistor</td>
<td>1981</td>
<td>45/60</td>
<td>No</td>
<td>4 weeks</td>
<td>4 weeks</td>
<td>no</td>
<td>2.5</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>Moller</td>
<td>2002</td>
<td>59/53</td>
<td>No</td>
<td>8 weeks</td>
<td>4 weeks</td>
<td>no</td>
<td>2</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Willits</td>
<td>2010</td>
<td>72/72</td>
<td>Yes</td>
<td>4 weeks</td>
<td>6 weeks</td>
<td>no</td>
<td>2</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>Nilsson-Helander</td>
<td>2010</td>
<td>49/48</td>
<td>No</td>
<td>6 to 8 weeks</td>
<td>-</td>
<td>2 weeks</td>
<td>1</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Olsson¹</td>
<td>2011</td>
<td>42/39</td>
<td>No</td>
<td>6 to 8 weeks</td>
<td>-</td>
<td>2 weeks</td>
<td>2</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Keating</td>
<td>2011</td>
<td>39/41</td>
<td>No</td>
<td>10 weeks</td>
<td>4 weeks</td>
<td>no</td>
<td>1</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>Schepull</td>
<td>2012</td>
<td>15/15</td>
<td>Yes</td>
<td>3.5 weeks</td>
<td>3.5 weeks</td>
<td>no</td>
<td>1.5</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Olsson</td>
<td>2013</td>
<td>49/51</td>
<td>No</td>
<td>2 weeks</td>
<td>2 weeks</td>
<td>no</td>
<td>1</td>
<td>+</td>
<td>-</td>
</tr>
</tbody>
</table>

¹ The patients were from Nilsson-Helander (2010) RCT.
² After 2 weeks active plantar flexion was added, but dorsiflexion was limited and reduced within intervals of 2 weeks.
2.4.2 Augmentation vs simple repair


Tezeren et al. (2006) randomized prospectively 24 patients to end-to-end suturation (12 patients) and identical suturation with the Lindholm augmentation technique (12 patients). After a mean 33 months’ follow-up (range 24–43 months) no difference in both isokinetic plantar flexion strength and subjective results was detected. Pajala et al. (2009) randomized 60 patients into simple suturation with the Krackow technique (32 patients) or into augmented repair (Silfverskjold) technique (28 patients). At 12 months, Leppilahti score, isokinetic strength and tendon elongation between treatment groups showed no differences (Pajala et al. 2009).

Aktas et al. (2007), in their prospective nonrandomized study, compared a simple Krackow suturation technique (14 patients) and identical suturation with augmentation for the plantaris longus tendon (16 patients). At a mean follow-up of 18 months (range, 4–33 months), no difference was noted in AOFAS scores or in the ability to do heel rises (Aktas et al. 2007). Jessing et al. (1975), in a retrospective study, compared simple tendon suturation (54 patients) and suturation augmented by the Silfverskjold technique (48 patients) and found similar functional results. Nyyssonen et al. (2003) retrospectively compared 98 patients with an acute ATR treated with plantaris tendon augmentation repair (n = 59) and patients with an end-to-end suture (n = 39) with mean follow-up of 44 months (range 22–69) and found similar subjective questionnaire results.

Augmentation of ATR repair provides better tensile strength that is obtained with simple suturation (Woo et al. 2000). The strongest reported techniques are the triple-bundle technique, which fails at 453 N and a 4-strand Krakow repair with epitendinous augmentation, which fails at 323 N (Jaakkola et al. 2000, Lee 2003). However, the data comparing simple suturation and suturation with augmentation in clinical use are equivocal, and no long-term results of prospectively collected patients exist.

2.4.3 Long-term recovery of calf muscle strength

Only a few long-term (≥10 years) follow-up studies after ATR have been conducted (Hohendorff et al. 2008, Horstmann et al. 2012, Lantto et al. 2015).
Hohendorff et al. (2008), in their retrospective study with a mean of 12 years (range 7 to 18) of follow-up, and with 42 ATR patients treated surgically (11) or with fibrin glue (31), found no strength deficit when comparing the affected leg to the healthy leg.

Horstmann et al. (2012) studied 63 ATR patients treated with an augmentation suturation technique and a mean of 10 years (SD 3.4) of follow up. Patients with surgically treated ATR had lower results in heel-raise tests, smaller calf circumference of the affected leg compared to the healthy leg, and a 15 % lower total isokinetic work during plantar flexion in the affected leg compared to the healthy leg.

Lantto et al. (2015) published the only prospectively designed study, in which 37 patients with a mean 11 years of follow-up (minimum 10 years) were treated with augmented suturation (Silfverskjold), and randomized into two different postoperative regimens: early functional treatment vs. cast immobilization in tension. No differences were noted between the two postoperative treatment groups, but the patients with operatively treated ATR showed a mean deficit of 5 % in isokinetic peak torque and a mean deficit of 8 % in average work at 11 years of follow-up.

Previous long-term studies have shown that ATR results in significant impairment of the affected leg for several years after treatment. However, the calf muscle and AT structural changes and their relationship to strength in long-term are unclear.

2.5 Calf muscle volume and fatty degeneration after ATR

Calf circumference is often used to evaluate muscle atrophy after ATR and during the recovery phase, but its correlation with calf muscle strength is equivocal (Moller et al. 2002, Rosso et al. 2015). In addition to calf circumference, calf muscle atrophy and fatty degeneration have been measured with CSA of the calf muscle, using CT (Haggmark et al. 1979, Leppilahti et al. 2000) and MRI (Rosso et al. 2013). MRI enables the measurement of the actual muscle volume, and it is probably a more accurate technique when compared to other methods (Leppilahti et al. 2000, Moller et al. 2002, Rosso et al. 2013). Investigating the structural explanation for calf muscle strength deficit between ATR treatment methods, in addition to AT elongation, muscle volume, and fatty degeneration, are the major objects of interest.
2.5.1 CT based methods

Häggmark et al. (1979) found that 6 weeks of immobilization after ATR surgical repair led to a 23 % atrophy in CSA, as measured by CT. Häggmark et al. (1986) retrospectively compared 15 surgically and 8 conservatively treated patients with different rehabilitation protocols and measured calf muscle CSA with CT at 3–5 years post injury. Surgically treated patients had similar CSA values to those measured for the healthy control calf, whereas conservative treatment resulted in a 13 % CSA deficit. In addition, conservatively treated patients suffered from a 29 % deficit in the heel rise test, but the surgically treated patients showed no such deficit. However, despite the deficit in the CSA and heel rise test observed for the conservatively treated patients, they suffered no isokinetic PT deficit (Häggmark et al. 1986). Leppilahti et al. 2000 retrospectively measured calf muscle CSA with CT 3 years after repair of ATR. Surgery lead to a 15 % CSA and 18 % PT deficit for the affected leg compared to the healthy leg. The correlation coefficient between the CSA and isokinetic PT varied in the range 0.52–0.61, depending the angular velocity of the measurement. Leppilahti et al. found also some evidence of calf muscle fatty degeneration in patients after ATR at 3 years post-surgery (Leppilahti et al. 2000). However, CSA may not be the most accurate method for evaluating muscle volume, since muscles do not atrophy uniformly across the entire length of a single muscle (Miokovic et al. 2012).

2.5.2 MRI based methods

MRI enables the identification of each muscle of the calf in three planes (axial, sagittal and coronal), and it is considered the most accurate method for evaluating the structural changes of muscles (Leppilahti et al. 2000, Rosso et al. 2013). The study by Rosso et al. (2013) is to date the only study to use MRI to evaluate the actual calf muscle volume and fatty degeneration after ATR and to correlate this volume to plantar flexion strength. The conclusion from this retrospective study, with a mean of 91 months of follow up, was that the mean volume deficit of the calf muscle in the affected leg was 17 % and substantial fatty infiltration had also occurred. No difference was found between conservative and surgical treatments in any variables (Rosso et al. 2013).

No prospective RCT has yet compared calf muscle volume and fatty degeneration after surgical and conservative ATR treatments with identical rehabilitation protocols. In addition, no prospective long-term studies have used
MRI to focus on calf muscle volume and fatty degeneration after ATR surgical repair.

2.6 Tendon elongation

If the tendon is injured and then healed as elongated, the muscle sarcomeres are shortened and forced to contract more in an attempt to compensate for the increase in tendon slack during function (Gordon et al. 1966, Magnaris et al. 2001 and 2003, Suydam et al. 2015). Magnaris et al. (2001) found that as little as 0.5 cm differences in fiber length cause a relative decrease in soleus muscle force.

Tendons heal through progressive remodeling of the hematoma between the tendon ends (Lin 2004, Wang 2006). Therefore, the size of the tendon gap will determine the extent of tendon lengthening (Lin 2004, Wang 2006). As tendon gap size gradually increases, the muscle position on the force-length curve will shift to an unfavorable direction, so that muscle strength is initially unaffected in the dorsiflexion to neutral ROM and then becomes suddenly reduced as the lengthening causes the muscle to operate on the end ROM (Lawrence et al. 2017). This model is compatible with recent study findings of a sudden drop off of strength at a gap $\geq$ 10 mm with the ankle in the neutral position, rather than a direct linear correlation between gap size and torque deficit (Lawrence et al. 2017).

AT elongation has been studied previously using metal radiographic markers placed during operation, followed by roentgen stereo photogrammetric analysis (RSA) techniques. However, these techniques are invasive and the healthy leg cannot act as a control (Kangas et al. 2007, Pajala et al. 2009, Schepull et al. 2012). Recently, Silbernagel et al. 2016 and Barfod et al. 2015 introduced new noninvasive methods using ultrasound (US) and MRI to measure the distance from the AT calcaneal insertion to the soleus or medial gastrocnemius myotendinous junction (MTJ). These new techniques allow the comparison of the AT lengths of the affected and unaffected sides; therefore, they are considered more accurate than metallic markers and RSA and are probably more reliable as well in the long term.

Studies using metal radiographic markers confirm that, after surgical repair, the AT elongates during the first 3 months after the injury, when weight bearing is allowed with an immobilizing cast or a walker. At 3 to 12 months, the AT slightly shortens and results in a 5 to 10 mm elongation of the tendon (Kangas et al. 2007, Pajala et al. 2009). Schepull et al. (2012) used RSA to measure elongation and their results were consistent with those of Kangas et al. (2007) and Pajala et al. (2009).
Schepull et al. (2012) compared tendon elongation between surgery and conservative treatment in a randomized controlled trial with an 18-month follow-up. They used the RSA technique for measurement and found a median 7 mm elongation, but no difference between the study groups. They implanted tantalum beads into the tendon during surgery; however, the conservative treatment group had this implantation performed at 3.5 weeks after the injury, which is a weakness of this study (Schepull et al. 2012).

Pajala et al. (2009) found a substantial correlation between isokinetic calf muscle deficits in PT and AT elongation at 12 months. Similarly, Silbernagel et al. (2012) found that the mean AT length after surgical treatment of ATR was 2.9 cm longer at 6 months and 2.6 cm longer at 12 months in the affected leg than in the healthy leg. This elongation was correlated (Spearman correlation coefficient, $r = 0.943$ at 6 months, $r = 0.738$ at 12 months) with a heel rise height deficit. Rosso et al. (2013) found that the AT in the affected leg was, on average, 1.8 cm longer than in the unaffected leg at 91 months after the injury, regardless of the treatment method for ATR. However, they did not assess any correlation with plantar flexion strength. Finni et al. (2006), in their study on patients with operatively treated ATRs, found that early recovery of plantar flexion torque may be a result of compensation by the FHL muscle, in addition to normalization of the calf muscle function.

No prospective RCTs have yet used MRI to compare the AT length between surgical and conservative treatments after ATR. In addition, no long term prospective trials have yet been published that have a special focus on the AT length and its relationship to calf muscle strength and calf muscle volume.
3 Aims of the present study

The specific aims of this thesis were:

1. To compare calf muscle isokinetic strength recovery after conservative treatment and open surgical repair of ATR, followed by identical accelerated rehabilitation programs.
2. To assess calf muscle volume, fatty degeneration, and AT elongation and their relationship with calf muscle isokinetic strength after surgery and conservative treatment of ATR.
3. To compare calf muscle isokinetic strength recovery after augmented and nonaugmented surgical repair of ATR in long-term follow-up.
4. To assess AT elongation, calf muscle volume atrophy, and fatty degeneration and their relationship with calf muscle isokinetic strength in long-term follow-up after surgical repair of ATR.
4 Patients and methods

4.1 Patients

4.1.1 RCT comparing surgery with conservative treatment (I and II)

All patients with acute ATR treated at Oulu University Hospital between April 2009 and November 2013 were screened for trial eligibility. The inclusion criteria were complete acute ATR and age between 18 and 65 years. The exclusion criteria were a delay of ≥ 1 week between the rupture and treatment, local corticosteroid injection(s) around the AT within 6 months before the rupture, previous surgery on the lower leg, previous ATR on the opposite side, open ATR, pregnancy, skin problems over the area of the AT, living outside the area, diabetes mellitus, or a persistent gap between the ruptured tendon ends in passive plantar flexion as assessed by ultrasound (US).

Two investigators examined all eligible patients and assigned them to interventions. Total ATR was diagnosed if a clear gap was palpable in the AT and the Thompson’s test was positive. A radiologist confirmed the diagnosis by US examination of the AT within 2 days after the injury. The radiologist also assessed whether the tendon ends could be reduced in passive plantar flexion of the ankle joint with the knee extended. A total of 258 patients were screened for trial eligibility; 124 patients were excluded and 74 eligible patients refused to participate. Thus, 60 patients (53 men and 7 women), with a mean age of 39.3 years (range 27–60 years), were randomized into surgical or conservative treatments. Table 2 presents the participants’ mean age at the time of injury, body mass index (BMI), and activity level (competitive athlete, recreational athlete, or non-athletic).
Table 2. Baseline variables for patients with ATRs, randomized into surgery or conservative treatment.

<table>
<thead>
<tr>
<th>Baseline characteristic</th>
<th>Surgery (n = 32)</th>
<th>Conservative treatment (n = 28)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Men/women</td>
<td>30/2</td>
<td>25/3</td>
<td>0.56</td>
</tr>
<tr>
<td>Mean age, y (Range)</td>
<td>40 (27 to 57)</td>
<td>39 (28 to 60)</td>
<td>0.55</td>
</tr>
<tr>
<td>Activity level Recreational athlete, n</td>
<td>32</td>
<td>27</td>
<td>&gt; 0.9</td>
</tr>
<tr>
<td>Mean body mass index, kg/m² (SD1)</td>
<td>27 (2.8)</td>
<td>26 (2.6)</td>
<td>0.33</td>
</tr>
<tr>
<td>Height, cm (SD1)</td>
<td>176.5 (7.1)</td>
<td>177.3 (4.7)</td>
<td>0.59</td>
</tr>
</tbody>
</table>

1 Standard deviation.

4.1.2 Long term follow up comparing augmented with non-augmented surgical repair of ATR (III and IV)

This study was a long-term follow-up of a previously published, prospective, RCT comparing augmented with non-augmented surgical repair of ATR (Pajala et al. 2009). The inclusion criteria were complete acute ATR and age of 18–65 years. The exclusion criteria were a delay of ≥ 1 week after the rupture until treatment, local corticosteroid injection(s) around the AT within 6 months pre-rupture, previous ATR on the opposite side, open ATR, skin problems over the AT area, living outside the country, and diabetes mellitus.

Between October 1998 and January 2001, 83 patients were screened for trial eligibility at a university teaching hospital. Twenty-one patients were excluded and two eligible patients refused to participate. The primary study group comprised 60 patients, including 53 men and 7 women, with a mean age of 38 years (range, 22–56 years) were randomized into augmented or non-augmented surgical repair of ATR. Table 3 presents the participants’ mean age at the time of injury, body mass index (BMI), activity level (competitive athlete, recreational athlete, or non-athletic), smoking habits, and previous AT symptoms.

After ≥ 13 years of follow-up, 57 patients from the original study were contacted. Of these patients, 55 (28 non-augmented, 27 augmented) were re-examined between December 2013 and March 2014, after an average of 14 years of follow-up (SD 0.5 years).

For study IV, patients from study III were combined as one group to study long-term structural changes of the muscles and tendon elongation after surgical repair of ATR.
Table 3. Baseline variables for patients for Non-augmented and Augmented-Repair Groups.

<table>
<thead>
<tr>
<th>Baseline variable</th>
<th>Non-augmented (n = 28)</th>
<th>Augmented Repair (n = 27)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Men/women</td>
<td>24/4</td>
<td>24/3</td>
</tr>
<tr>
<td>Mean age (SD(^1))</td>
<td>39 (9)</td>
<td>36 (7)</td>
</tr>
<tr>
<td>Activity level, n</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Competitive athlete</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Recreational athlete</td>
<td>24</td>
<td>24</td>
</tr>
<tr>
<td>Non-athlete</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Mean body mass index (SD(^1))</td>
<td>25 (2.6)</td>
<td>27 (2.6)</td>
</tr>
</tbody>
</table>

\(^1\) standard deviation.

4.2 Interventions

4.2.1 Conservative Treatment (I and II)

Non-surgical treatment started with a non-weight bearing full equinus cast for the first week. After 1 week, the cast was changed to an orthosis (Vacoped®). The ankle was positioned at 30° plantar flexion for weeks 2 to 3, 15° plantar flexion for weeks 4 to 5, and free movement from 0° to 30° plantar flexion for weeks 6 to 7. Full weight bearing was directed and encouraged during the orthosis immobilization. After week 7, a 1-cm heel raise was used for 1 month. Cycling and swimming exercises were recommended at 2 months; jogging began at 3 months. Sports involving sudden acceleration and jumping were allowed after 6 months.

4.2.2 Operative Techniques

Study I and II

Surgery was performed within 7 days of the injury. Four surgeons were involved in the operations. One surgeon operated on 29 patients and three surgeons each operated on one patient. The Krackow locking loop technique (two non-absorbable sutures and smaller apposition sutures) was used in all patients (Krackow 1986). After surgery, the patients received a full equinus cast for 1 week, without weight bearing. The postoperative treatment and rehabilitation were identical to the conservative treatment protocol. All patients were instructed to perform exercises according to a standard rehabilitation program (Appendix 1).
Study III and IV

All operations were performed by the same surgeon. In non-augmented repair (32 patients), the tendon operation involved the use of the Krackow technique with two polydioxanone 0 absorbable sutures (PDS®, Ethicon, Somerville, NJ) and smaller 2–0 apposition sutures with polyglactin (Vicryl®, Ethicon). In the augmented group (28 patients), an end-to-end repair identical to the non-augmented procedure was used. After repair, a 10-mm-wide central gastrocnemius aponeurosis flap was turned down over the suture line and stitched to the AT with 2–0 Vicryl®. In all cases, after repair, titanium marker clips were placed on both sides of the ruptured tendon ends for later measurement of tendon elongation. The ankle was gently placed in a neutral position, and the skin was closed with 4–0 nylon sutures (Ethilon®, Ethicon). Post-operatively, all patients received a temporary below-knee rigid plaster splint, with their ankle in a neutral position.

Postoperative treatment was identical in both groups. On the first postoperative day, all patients received a Soft Cast® individual below-knee dorsal brace for three weeks, which allowed active free plantar flexion of the ankle and restricted dorsiflexion to neutral. The patients were allowed 20 kg of weight bearing for three weeks, half weight bearing for three to six weeks, and full weight bearing thereafter. Patients were instructed to perform exercises according to a standard rehabilitation program (Pajala et al. 2009).

4.3 Follow-up

The patients were followed up in study I and II at 3, 6, and 18 months, and in study III and IV at 3 and 12 months, and 14 years. Clinical examination, strength measurement, radiographs, and MRI were carried out according to the flow charts (Figs. 7–10).
Fig. 7. Study I flow chart.

Assessed for eligibility (n = 258)

- Excluded (n = 198)
  - Not meeting inclusion criteria (n = 124)
  - Declined to participate (n = 74)

Randomized for treatment (n = 60)

Enrollment

- Surgery (n = 32)
  - Isokinetic strength tests
    - n = 27
    - 5 missing; unsuitable follow-up time
  - Leppilahti score
    - n = 29
    - Missing 3; one or more required angular velocity for score was missed

Allocation

- Conservative treatment (n = 28)
  - Isokinetic strength tests
    - n = 25
    - 3 missing; unsuitable follow-up time
  - Isokinetic strength tests and Rand-36
    - n = 28

- Isokinetic strength tests
  - n = 27
  - 5 missing; unsuitable follow-up time

3 months

- Isokinetic strength tests
  - n = 23
  - 5 missing; unsuitable follow-up time

6 months

18 months

- Leppilahti score
  - n = 29
  - Isokinetic strength tests and Rand-36
    - n = 32
Fig. 8. Study II flow chart.

* These outliers emerged because of inadequate MRI field-of-view (FOV) (150 mm x 150 mm) and impractical FOV adjustment.
Fig. 9. Study III flow chart.

Assessed for eligibility (n = 83)
- Excluded (n = 23)
  - Not meeting inclusion criteria (n = 19)
  - Declined to participate (n = 2)
  - Living outside the country (n = 2)

Randomized for operation (n = 60)

Enrollment

Allocation

Simple repair (n = 32)
  - Received allocated intervention (n = 32)

Augmented repair (n = 28)
  - Received allocated intervention (n = 28)

Simple repair (n = 32)

Augmented repair (n = 28)

3-12 months
- Leppilahti score and strength tests
- 3 months follow-up
  - Leppilahti score and strength tests
- 12 months follow-up
  - Leppilahti score and strength tests

Lost to follow-up (n = 4)
- Living abroad (n = 1)
- Declined to participate (n = 1)
- No contact (n = 2)

Analysed (n = 28)
- Leppilahti score
- Tendon elongation (metal radiographs)
- Strength tests

14 year
- Leppilahti score
- Tendon elongation (metal radiographs)
- Strength tests

Analysed (n = 27)
4.4 Outcome Measures

4.4.1 Calf muscle isokinetic strength

An experienced physiotherapist and an exercise physiologist performed all strength tests.

Before testing, patients were informed of the measurement procedure. They performed a 10-minute warm-up period of ergometer cycling and a few submaximal and maximal repetitions of ankle flexion and extension movements at
the isokinetic test velocity. During testing, the patient was in the supine position, with the knee supported in extension (Fig. 11). The extent of ankle motion was from 15° dorsiflexion to 35° plantar flexion (I and II) and from 10° dorsiflexion to 30° plantar flexion (III and IV). The isokinetic plantar flexion strengths of both ankles were measured at speeds of 60, 120, and 180 degrees/s. Five maximal voluntary muscular torque contractions were required. The dynamometer recorded plantar flexion PT automatically for each performance. To evaluate the isokinetic torque results for the entire ROM of the ankle joint ASPT (I and II), and work displacement curves for each 10 degree interval were manually composed (III and IV). Standardized verbal encouragement was provided to ensure maximal patient effort (McNair et al. 1996).
Study I and II

A Con-Trex biomechanical test and training system computer-based isokinetic dynamometer (CMV AG Duebendorf, Switzerland) was used in study I and II.

Peak plantar flexion torque at an angular velocity of 60 degrees/s was recorded for each leg, at 3, 6, and 18 months post injury. Torque results over the ankle joint’s ROM were assessed by recording the best PT values at specific angles (0°, 10°, and 20°) (ASPT), based on five repetitions for both calves.

Study III and IV

The isokinetic strength of both ankles was assessed 3 months, 12 months, and 14 years after surgery. At the 3- and 12-month follow-ups, a physiotherapist performed strength measurements using a Lido Multi-Joint 2 computer-based isokinetic dynamometer (Loredan Biomedical, Inc., West Sacramento, CA). At the 14-year follow-up, the same physiotherapist and an exercise physiologist performed strength measurements using a Con-Trex biomechanical test and training system computer-based isokinetic dynamometer (CMV AG Duebendorf, Switzerland).

Peak plantar flexion torque at an angular velocity of 60 degrees/s was recorded for each leg. From the five maximal performances, the best 10° interval was recorded and work-displacement curves (−10° to 30°) were calculated for both calves to assess percentage work deficits over the ankle joint’s ROM at 14 years post injury.

4.4.2 Calf muscle volume

The MRI examination was performed on the same day, but before the isokinetic strength tests. MRI was performed at the same time on the affected leg and the unaffected contralateral leg, which served as a normal, matched control. Patients were imaged in the supine position, and the ankles were supported in the plantigrade position, with the ankle at a 90° angle and the knee at a 0° angle.

The calf muscle volumes of both healthy and affected legs were measured from the MRI scans acquired at 3 and 18 months (II) after the injury and at 14 years (IV) after the injury. The cross-sectional area of the muscle or muscle compartment was measured from the axial MRI images at 17 mm (II) and 16.2 mm (IV) intervals from the knee to the upper ankle joint.
The outline of each muscle was identified from axial MR- images and CSA of the muscle or muscle compartment was measured (Fig. 12). The muscle volume was calculated with the formula for a frustum of cones:

\[
\text{Muscle volume} = \frac{h}{3} \left( A_1 + \frac{2}{3} \left( A_1 + A_2 \right) \right)
\]

where \( h \) = the height of the cylinder (16.2 or 17 mm), \( A_1, A_2 \) = the cross-sectional areas at the two ends of the cylinder.

This method is a modification of that described by Rosso et al. (2013), in which authors calculated the volumes using a formula for regular cylinders instead of frustums of cones. The final muscle volume was the sum of all cylinders measured in that muscle. Muscles volumes (in cm\(^3\)) were calculated for each compartment, including the soleus, the medial- and lateral gastrocnemius, the FHL, and the deep flexors (the digitorum longus and tibialis posterior) in the posterior compartment.

The detailed imaging protocol is described in Appendix 3.

![Fig. 12. Axial T1-weighted MR-images of both calves, acquired in a patient with an ATR. The cross-sectional area was measured on a clinical workstation by manually drawing the region of interest around a muscle or muscle compartment. DF = deep flexors, FHL = flexor hallucis longus, S = soleus, GM = gastrocnemius medialis, GL = gastrocnemius lateralis.](image)

**4.4.3 Fatty degeneration (II, IV)**

A grading system, described by Bernegeau and Goutallier and adapted for MRI, was used to classify muscle fatty degeneration (Alizai et al. 2012, Goutallier et al. 1994). Fatty degeneration of the soleus, medial gastrocnemius, and lateral
gastrocnemius of both calves was evaluated from the axial MRI slice that showed the greatest cross-sectional area at the calf level. Grades were defined as follows:

- grade 0, no intramuscular fat;
- grade 1, some fatty streaks;
- grade 2, fat was evident but there was less fat than muscle;
- grade 3, equal amounts of fat and muscle tissue and
- grade 4, more fat than muscle tissue.

Subsequently, grades 0 and 1 were considered to reflect normal variations in the Goutallier classification (Hoffmann et al. 2011, Saupe et al. 2006, Syed et al. 2009).

### 4.4.4 Tendon elongation

**Study II**

The length of the AT in the affected leg was measured on axial and sagittal MRIs acquired at 3 and 18 months after the injury. AT length was measured using the method described by Silbernagel et al. (2016) adapted for MRI. Tendon length was measured from the most distal part of the soleus muscle to the calcaneal insertion of the AT, which was defined as the intersection of the axial plane with the most cranial aspect of the tuber calcanei. Measurements were performed as follows: the MTJ of the soleus was identified on axial slices; thereafter, the distance from this level to the calcaneal insertion was measured on sagittal slices (Fig. 13).
Fig. 13. (A and B). AT length measurement. (A) Axial T1-weighted MRI of the AT shows the MTJ (red) of the soleus. (B) Sagittal T1-weighted MRI shows the AT length (yellow arrow), measured from the MTJ (red line; the level of the soleus identified on the axial slice) to the calcaneal insertion.

**Study III and IV**

Radiographic markers placed during operation on both sides of the rupture were used to measure tendon elongation of the affected leg. Standardized radiographs were taken on the first day postoperatively, and at 12 months and 14 years postoperatively. The distance between the metallic markers was measured (to 1 mm) and AT elongation was recorded between follow-ups. AT elongation measurements were analyzed for both groups and correlated with the strength results.

At 14 years, the AT length was also measured using MRI for both the healthy leg and the affected leg. The axial plane intersecting the most cranial aspect of the tuber calcanei defined the most distal end of the AT. The measurement was performed as follows: The measuring cursor was placed at the distal end of the AT on sagittal slice. Keeping the cursor in this exact position, the researcher scrolled to the sagittal slice showing the most distal end of the medial head of the gastrocnemius muscle; this slice was used to measure the distance. The number of sagittal slices between the two measurement points was recorded and multiplied by
the slice thickness (4.8 mm) to observe the lateral distance between the two measurement points in the coronal plane. The estimate of the real length of the tendon was then calculated using Pythagoras, observing both the craniocaudal measurement on the sagittal slice, and the lateralization estimate (Barfod et al. 2015). Elongation was calculated as side-to-side (affected leg minus healthy leg) differences. Relative elongation \( [(\text{affected} – \text{healthy}) / \text{affected}] \times 100 \) was calculated to assess the correlation with isokinetic strength deficits.

### 4.4.5 Leppilahti score

The clinical outcomes for study I and III were assessed using the disease-specific scoring method described by Leppilahti et al. 1998. The scoring system combines subjective factors (pain, stiffness, muscle weakness, and footwear restrictions) and objective factors (active range of ankle motion and isokinetic calf muscle strength) into one number. The maximum number of points achievable is 100 (Appendix 2).

### 4.5 Ethics

The ethical committee of Oulu University Hospital approved all study protocols (I–IV) and re-examination of the patients (III, IV). All patients in study I-IV received oral and written information about the trial and provided informed consent to participate.

### 4.6 Statistical methods

SPSS for windows (IBM Corp., Released 2012. IBM SPSS Statistics for Windows, Version 21.0, Armonk, NY) and SAS for windows (version 9.4, SAS Institute Inc., Cary, NC) were used for analyses.

Summary measurements are presented as mean and standard deviation (SD) or as median with 25th–75th percentiles, unless otherwise stated. The Student’s t-test or analysis of variance (ANOVA) was used for between-group comparisons (continuous variables). Pearson’s \( \chi^2 \) test or Fisher’s exact test were used for categorical data. The Spearman correlation coefficient (\( \rho \)) was calculated to evaluate correlations. Ninety-five percent confidence intervals (95 % CI) are presented with mean differences and difference between means.

Repeated-measures data were analyzed using a linear mixed model (LMM) in which patients were set as random effects and the correlation between
measurements was taken into account by testing different covariance patterns. The best covariance pattern was chosen according to the Akaike information criteria.

P-values reported as the result of LMM analyses were as follows:

- $P_{\text{time}}$, overall change over time;
- $P_{\text{group}}$, average difference between groups and
- $P_{\text{time} \times \text{group}}$.

When comparing isokinetic muscle strengths between different angles, the reported p-values are: $P_{\text{angle}}$, $P_{\text{group}}$, and $P_{\text{time} \times \text{group}}$. Two-tailed p-values and 95 % confidence intervals (CIs) are presented. A p-value < 0.05 was considered statistically significant.

Randomization

In study I and II, a biostatistician generated a random allocation sequence using computer software. A randomly varying block size (4, 6, or 8) was used, each block having an equal number of surgically and conservatively treated patients. The possible confounding effect of age was controlled by performing the randomization separately in two age groups ($\leq 35$ and $> 35$ years) with the ratio of 1/3 ($n = 20$) and 2/3 ($n = 40$), respectively. In study III, mixed sealed envelopes were used for randomization.

Sample size calculation

The sample size (study I and III) was calculated using the Leppilahti score. The clinically significant difference was set at 10 points ($\text{SD} = 10$, $\alpha = 0.05$, and $\beta = 0.10$), resulting in 17 patients in each group. To compensate for an estimated dropout rate of 30 %, a total of 60 patients were to be enrolled in the study.
5 Results

5.1 Isokinetic calf muscle strength recovery after surgery vs. conservative treatment of ATR (I)

At the 3-month follow-up, both groups achieved similar mean PT results, but at 6 months, the advantage for surgically treated patients was 24% (difference between means 14.8 Nm, 95% CI: 2.7 to 29.9, P = 0.017). The difference between the study groups decreased to 14% at 18 months of follow-up; the average PT for the surgical group was 110.3 Nm vs. 96.5 Nm in the conservatively treated group (difference between means 13.6 Nm, 95% CI: 2.0 to 25.1, P = 0.022) (Fig. 14).

Fig. 14. Median plantar flexion PT values (error bars represent the 25th and 75th percentiles) for plantar flexion of the affected and healthy legs measured at 3, 6, and 18 months.

ASPT curves at the 18-month follow-up had an almost similar shape, but surgically treated patients achieved 10% to 18% higher values throughout the ankle ROM (Fig. 15).
At the 18-month follow-up, the mean Leppilahti scores were 79.5 (SD 10.3) and 75.7 (SD 11.2) for surgically and conservatively treated patients, respectively (difference between means 3.8 points, 95 % CI: -1.9 to 9.5, P = 0.19).

5.2 Structural changes of calf muscles and Achilles tendon after surgery vs. conservative treatment of ATR (II)

5.2.1 Calf muscle volume

At 3 months, calf muscle volumes did not differ between the study groups (Table 4). At 18 months, the mean side-to-side differences in soleus muscle volumes were 83.2 cm³ (17.7 %) for operative treatments and 115.5 cm³ (24.8 %) for conservative treatments (difference between means: 33.1 cm³, 95 % CI: 1.3 to 65.0, P = 0.042). The other muscle volumes were not different between study groups.

Between 3 months and 18 months, patients treated conservatively displayed reduced volume in the affected soleus muscle; the mean side-to-side difference increased significantly from 73.4 to 115.5 cm³ (mean change: 42.0 cm³, 95 % CI:
In contrast, patients treated operatively maintained the soleus volume during follow up; the mean side-to-side difference increased insignificantly from 74.3 to 83.2 cm³ (mean change: 6.6 cm³, 95 % CI: -27.9 to 14.6, \( P = 0.53 \)) (Table 4).

The study groups were combined to assess correlation between calf muscle strength deficit and muscle volumes. At 18 months, the entire study population displayed a mean side-to-side difference between the healthy and affected legs of 29.4 Nm (SD 2.9) in PT, and 30.0 Nm (SD 2.7), 31.7 Nm (SD 2.3), and 32.1 Nm (SD 2.1) in ASPT at 0°, 10°, and 20° plantar flexion, respectively. At 18 months, the soleus muscle volume side-to-side atrophy correlated with the peak torque deficit (\( \rho = 0.412, \ P = 0.001 \)). Additionally, the angle-specific peak torque deficit for the entire range of ankle motion correlated significantly with soleus atrophy (\( \rho = 0.449, \ 0.576, \ \text{and} \ 0.611, \ P < 0.001, \) at 0°, 10°, and 20° of plantar flexion, respectively). These results suggested that soleus atrophy had greater effects on torque as the plantar flexion increased. In contrast, at 3 months, side-to-side differences in soleus muscle volume did not correlate with any strength variable.

In patients treated conservatively, the mean side-to-side differences in FHL muscle volume were 5.6 cm³ at 3 months and -9.3 cm³ at 18 months (mean change: 14.9 cm³, 95 % CI: 10.1 to 19.7, \( P < 0.001 \)). In patients treated operatively, the mean side-to-side differences in FHL muscle volume were 1.7 cm³ at 3 months and -8.4 cm³ at 18 months (mean change: 8.9 cm³, 95 % CI: 4.3 to 13.5, \( P = 0.001 \)). These changes in FHL muscle volume indicated that compensatory hypertrophy occurred in the affected legs of patients in both groups (Table 4). Additionally, deep flexor muscle volumes showed similar compensatory hypertrophy in both groups. In the conservative treatment group, the side-to-side differences were 3.4 cm³ at 3 months and -4.8 cm³ at 18 months (mean change: 8.2 cm³, 95 % CI: 1.1 to 15.3, \( P = 0.026 \)). In the operative treatment group, the mean side-to-side differences were 2.6 cm³ at 3 months and -3.2 cm³ at 18 months (mean change 5.8 cm³, 95 % CI: 0.2 to 11.5, \( P = 0.043 \); Table 4).
Table 4. Comparison of muscle volumes between surgical (operative) and conventional (conservative) treatment groups, measured at 3-month and 18-month follow-ups.

<table>
<thead>
<tr>
<th>Treatment group</th>
<th>3 months</th>
<th>18 months</th>
<th>Comparisons</th>
<th>P&lt;sub&gt;time&lt;/sub&gt;</th>
<th>P&lt;sub&gt;group&lt;/sub&gt;</th>
<th>P&lt;sub&gt;time x group&lt;/sub&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Operative (n = 29)</td>
<td>Operative (n = 29)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td>Soleus, affected</td>
<td>386.3</td>
<td>68.9</td>
<td>385.3</td>
<td>70.6</td>
<td>385.7</td>
<td>78.5</td>
</tr>
<tr>
<td>Soleus, healthy</td>
<td>460.6</td>
<td>87.3</td>
<td>458.7</td>
<td>77.9</td>
<td>468.9</td>
<td>91.6</td>
</tr>
<tr>
<td>Side-to-side difference</td>
<td>74.3</td>
<td>65.4</td>
<td>73.4</td>
<td>86.1</td>
<td>83.2</td>
<td>67.0</td>
</tr>
<tr>
<td>FHL, affected</td>
<td>78.0</td>
<td>19.5</td>
<td>78.3</td>
<td>18.8</td>
<td>91.0</td>
<td>23.5</td>
</tr>
<tr>
<td>FHL, healthy</td>
<td>79.7</td>
<td>14.5</td>
<td>83.9</td>
<td>16.2</td>
<td>82.6</td>
<td>17.5</td>
</tr>
<tr>
<td>Side-to-side difference</td>
<td>1.7</td>
<td>14.4</td>
<td>5.6</td>
<td>13.6</td>
<td>-8.4</td>
<td>15.0</td>
</tr>
<tr>
<td>Deep flexors, affected</td>
<td>137.1</td>
<td>29.1</td>
<td>143.3</td>
<td>23.2</td>
<td>145.0</td>
<td>34.5</td>
</tr>
<tr>
<td>Deep flexors, healthy</td>
<td>140.9</td>
<td>27.8</td>
<td>146.8</td>
<td>27.0</td>
<td>141.8</td>
<td>30.3</td>
</tr>
<tr>
<td>Side-to-side difference</td>
<td>3.8</td>
<td>14.3</td>
<td>3.4</td>
<td>17.1</td>
<td>-3.2</td>
<td>16.6</td>
</tr>
<tr>
<td>Medial gastrocnemius, affected</td>
<td>170.2</td>
<td>48.4</td>
<td>175.6</td>
<td>45.9</td>
<td>186.7</td>
<td>50.4</td>
</tr>
<tr>
<td>Medial gastrocnemius, healthy</td>
<td>204.5</td>
<td>55.3</td>
<td>215.3</td>
<td>45.3</td>
<td>220.6</td>
<td>54.9</td>
</tr>
<tr>
<td>Side-to-side difference</td>
<td>34.3</td>
<td>25.1</td>
<td>39.8</td>
<td>26.5</td>
<td>33.9</td>
<td>37.2</td>
</tr>
<tr>
<td>Lateral gastrocnemius, affected</td>
<td>110.2</td>
<td>32.5</td>
<td>112.1</td>
<td>23.1</td>
<td>126.5</td>
<td>32.3</td>
</tr>
<tr>
<td>Lateral gastrocnemius, healthy</td>
<td>127.5</td>
<td>38.2</td>
<td>129.7</td>
<td>23.3</td>
<td>142.5</td>
<td>33.5</td>
</tr>
<tr>
<td>Side-to-side difference</td>
<td>17.2</td>
<td>16.3</td>
<td>17.6</td>
<td>14.0</td>
<td>16.0</td>
<td>22.0</td>
</tr>
</tbody>
</table>

1 healthy – affected
5.2.2 Muscle fatty degeneration

At 3 and 18 months, the MRIs showed no significant difference between study groups in fatty degeneration of the affected leg soleus or gastrocnemius muscles (Tables 5 and 6). However, at 18 months, patients treated conservatively had somewhat greater fatty degeneration (Tables 5 and 6). At 18 months, fatty degeneration was statistically significantly associated with lower plantar flexion PT (Fig. 16).

Table 5. Soleus muscle fatty degeneration, affected leg.

<table>
<thead>
<tr>
<th>Goutallier classification</th>
<th>Operative 3 months</th>
<th>Conservative 3 months</th>
<th>Operative 18 months</th>
<th>Conservative 18 months</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n²</td>
<td>%¹</td>
<td>n²</td>
<td>%¹</td>
</tr>
<tr>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>3.2</td>
</tr>
<tr>
<td>1</td>
<td>8</td>
<td>27.6%</td>
<td>3</td>
<td>10.7%</td>
</tr>
<tr>
<td>2</td>
<td>20</td>
<td>69.0%</td>
<td>22</td>
<td>78.6%</td>
</tr>
<tr>
<td>3</td>
<td>1</td>
<td>3.4%</td>
<td>3</td>
<td>10.7%</td>
</tr>
<tr>
<td>4</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>29</td>
<td>100%</td>
<td>28</td>
<td>100%</td>
</tr>
</tbody>
</table>

¹ percentage of treatment group, ² number of patients used as comparison
Table 6. Gastrocnemius muscle fatty degeneration, affected leg.

<table>
<thead>
<tr>
<th>Goutallier classification</th>
<th>Medial gastrocnemius muscle fatty degeneration, affected leg</th>
<th>Lateral gastrocnemius muscle fatty degeneration, affected leg</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>3 months Operative</td>
<td>18 months Conservative</td>
</tr>
<tr>
<td></td>
<td>n²</td>
<td>%¹</td>
</tr>
<tr>
<td>0</td>
<td>3</td>
<td>10.3 %</td>
</tr>
<tr>
<td>1</td>
<td>15</td>
<td>51.7 %</td>
</tr>
<tr>
<td>2</td>
<td>11</td>
<td>37.9 %</td>
</tr>
<tr>
<td>3</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>4</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>29</td>
<td>100 %</td>
</tr>
</tbody>
</table>

¹ percentage of treatment group, ² number of patients used as comparison

\( P² = 0.60 \)
\( P³ = 0.054 \)
\( P⁴ = 0.094 \)
\( P⁵ = 0.056 \)
Fig. 16. Association between affected leg PT and soleus muscle fatty degeneration. Only one patient had Goutallier grade 0; thus, this patient was omitted from the analysis. ANOVA analysis $P = 0.017$.

5.2.3 Achilles tendon length

At 3 months, the mean AT lengths were 69.5 mm (SD 3.1) for patients treated surgically and 86.4 mm (SD 3.8) for patients treated conservatively (difference between means: 16.6 mm, 95% CI: 6.4 to 26.7, $P < 0.001$). At 18 months, the mean AT lengths were 68.8 mm (SD 3.0) for patients treated surgically and 87.9 mm (SD 4.3) for patients treated conservatively (difference between means: 18.7 mm, 95% CI: 8.5 to 28.8, $P < 0.001$).

From 3 to 18 months, the mean changes in tendon lengths were -0.63 mm (SD 3.9 mm) for patients treated surgically and 1.5 mm (SD 7.3 mm) for patients treated conservatively (difference between mean changes: 2.1 mm, 95% CI: -5.4 to 1.3, $P = 0.21$).
5.3 Long-term isokinetic calf muscle strength recovery after augmented vs nonaugmented repair of ATR (III)

At the 14-year follow-up, the mean plantar flexion PT for the operated leg did not differ between groups; 112.6 Nm for non-augmented vs. 107.3 Nm for augmented repair [difference between means 5.3 (95% CI: -22.2 to 11.6) Nm]. The work–displacement deficit curves for both groups had a similar shape (Fig. 17). The median plantar flexion work deficit for 10° intervals ranged from 6.7 to 20.3 % with non-augmented vs. from 12.8 to 18.0 % for augmented repair (P = 0.30).

![Fig. 17. Median work-displacement %-deficits in plantar flexion measured in 10° intervals between the study groups at the 14-year follow-up. Error bars represent the 25th and 75th percentiles.](image)

In both groups, the median peak torque deficit did not substantially change between 12 months and 14 years: 9.2 % vs. 11.0 % with non-augmented repair (P = 0.8) and 6.6 % vs. 13.4 % with augmented repair (P = 0.66), respectively.

When combining the study groups as one, at the 14-year follow-up, the mean PT of the injured leg (110.0 Nm) remained lower compared to the non-injured leg (119.3 Nm), mean difference 9.3 (95% CI: 4.1 to 14.4) Nm. The work–displacement curve showed similar differences, with the median deficit for 10° intervals ranging from 12.2 % to 18.0 % for the injured side, (P < 0.001) (Fig. 18).
From 12 months to 14 years, isokinetic calf muscle strength deficits for the injured leg did not substantially change at any work-displacement intervals (P > 0.2, in all) (Fig. 18).

Fig. 18. Comparison of the 3-month, 12-month, and 14-year median work displacement deficits in plantar flexion of the operatively treated leg, as compared with the control (healthy) leg, measured at 10° intervals over the ankle ROM. The error bars represent the 25th and 75th percentiles.

5.4 Tendon elongation and calf muscle structural changes vs strength deficit after ATR in long-term follow-up (III, IV)

5.4.1 Achilles tendon elongation

Metal radiographs (III)

The mean AT elongation was 12.7 mm with non-augmented vs. 13.6 mm with augmented repair [difference between means 3.3 (95% CI: -10.8 to 4.2) mm]. From 12 months to 14 years, mean elongation was 2.9 mm (SD 3.0 mm) for non-augmented vs. 2.2 mm (SD 2.5 mm) for augmented repair [difference between means 1.2 (95% CI: -3.0 to 0.6) mm].
The mean AT elongation for whole study population was 13.7 mm (95 % CI: 10.2 to 17.2 mm, P < 0.001).

MRI (IV)

The mean AT length from the calcaneal insertion to the medial gastrocnemius MTJ was 199 mm in the affected leg versus 187 mm in the healthy leg (mean difference of 12 mm, 95 % CI: 8.6 to 15.6, P < 0.001).

5.4.2 Calf muscle volume

The mean soleus muscle volume was 13 % lower (mean difference of 63 cm³, 95 % CI: 43 to 78, P < 0.001) in the affected leg than in the healthy leg. The mean medial and lateral gastrocnemius muscle volumes were 12 % lower (mean difference of 30 cm³, 95 % CI: 19 to 38, P < 0.001) and 11 % lower (mean difference of 16 cm³, 95 % CI: 10 to 21, P < 0.001) in the affected leg than in the healthy leg, respectively (Table 7).

The mean volume of the flexor hallucis longus (FHL) in the affected leg was 5 % higher (mean difference of 4.6 cm³, 95 % CI: 1.6 to 7.3, P = 0.003) than in the unaffected leg, indicating compensatory hypertrophy of the FHL after ATR (Table 7). There was no difference in the volume of the deep flexors between the affected and healthy legs (P = 0.114) (Table 7).

Table 7. Calf Muscle Volume 14 Years After Surgery for ATR.

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Muscle volume, cm³</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Affected leg Mean</td>
<td>SD</td>
</tr>
<tr>
<td>Soleus</td>
<td>429.1</td>
<td>92.8</td>
</tr>
<tr>
<td>Medial gastrocnemius</td>
<td>207.7</td>
<td>51.5</td>
</tr>
<tr>
<td>Lateral gastrocnemius</td>
<td>133.1</td>
<td>36.0</td>
</tr>
<tr>
<td>FHL</td>
<td>101.4</td>
<td>25.6</td>
</tr>
<tr>
<td>Deep flexors</td>
<td>159.8</td>
<td>35.4</td>
</tr>
</tbody>
</table>

¹ healthy – affected

5.4.3 Muscle fatty degeneration

Fatty degeneration in the soleus and gastrocnemius muscles was substantially more common in the affected leg than in the unaffected leg (Table 8). The soleus muscle
of the affected leg showed greater fatty degeneration than did the medial and lateral gastrocnemius muscle (P < 0.001 for all comparisons) (Table 8).

Table 8. Calf Muscle Fatty Degeneration 14 Years After Surgery for ATR

<table>
<thead>
<tr>
<th>Goutallier classification</th>
<th>Soleus</th>
<th>Gastrocnemius Medialis</th>
<th>Gastrocnemius Lateralis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Healthy leg</td>
<td>Affected leg</td>
<td>Healthy leg</td>
</tr>
<tr>
<td>n2</td>
<td>%1</td>
<td>n2</td>
<td>%1</td>
</tr>
<tr>
<td>0</td>
<td>6</td>
<td>11.5 %</td>
<td>1</td>
</tr>
<tr>
<td>1</td>
<td>35</td>
<td>67.3 %</td>
<td>17</td>
</tr>
<tr>
<td>2</td>
<td>11</td>
<td>21.2 %</td>
<td>32</td>
</tr>
<tr>
<td>3</td>
<td>0</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>4</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>52</td>
<td>100 %</td>
<td>52</td>
</tr>
</tbody>
</table>

P2 < 0.001  P3 < 0.001  P4 = 0.018

5.4.4 Correlation with calf muscle strength

AT elongation measured using metal radiographs did not correlate with any isokinetic strength parameter, calf muscle volume, or AT elongation measured using MRI at the 14-year follow-up (Table 9).

Table 9. AT elongation (measured using metal radiographs) with Plantar Flexion Strength, Muscle Volume and AT elongation measured using MRI.

<table>
<thead>
<tr>
<th>Variable</th>
<th>AT elongation (Measured using metal radiographs)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ρ</td>
</tr>
<tr>
<td>AT elongation (Measured using MRI)</td>
<td>-0.01</td>
</tr>
<tr>
<td>Work displacement deficit 10°–0°</td>
<td>0.04</td>
</tr>
<tr>
<td>Work displacement deficit 0°–10°</td>
<td>0.000</td>
</tr>
<tr>
<td>Work displacement deficit 10°–20°</td>
<td>-0.02</td>
</tr>
<tr>
<td>Work displacement deficit 20°–30°</td>
<td>0.08</td>
</tr>
<tr>
<td>Soleus muscle volume, deficit</td>
<td>0.22</td>
</tr>
<tr>
<td>Medial gastrocnemius volume, deficit</td>
<td>0.24</td>
</tr>
<tr>
<td>Lateral gastrocnemius volume, deficit</td>
<td>0.27</td>
</tr>
</tbody>
</table>

However, AT elongation, as measured from the calcaneal insertion to the medial gastrocnemius MTJ, correlated with the ankle plantar flexion strength deficit (ρ = 0.38 to 0.51, P < 0.001 to 0.006) and with the soleus and medial gastrocnemius
muscle volume deficit \( (p = 0.42 \text{ and } 0.46, \ P = 0.002 \text{ and } 0.001, \text{ respectively}) \) (Table 10).

Table 10. AT Elongation Correlation with Plantar Flexion Strength and Muscle Volume.

<table>
<thead>
<tr>
<th>Variable</th>
<th>AT elongation (Medial gastrocnemius MTJ –Calcaneal pitch)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>( \rho )</td>
</tr>
<tr>
<td>Work displacement deficit 10°–0°</td>
<td>0.18</td>
</tr>
<tr>
<td>Work displacement deficit 0°–10°</td>
<td>0.38</td>
</tr>
<tr>
<td>Work displacement deficit 10°–20°</td>
<td>0.51</td>
</tr>
<tr>
<td>Work displacement deficit 20°–30°</td>
<td>0.47</td>
</tr>
<tr>
<td>Soleus muscle volume, deficit</td>
<td>0.42</td>
</tr>
<tr>
<td>Medial gastrocnemius volume, deficit</td>
<td>0.46</td>
</tr>
<tr>
<td>Lateral gastrocnemius volume, deficit</td>
<td>0.24</td>
</tr>
<tr>
<td>FHL volume, deficit</td>
<td>-0.30</td>
</tr>
<tr>
<td>Deep flexors volume, deficit</td>
<td>-0.06</td>
</tr>
</tbody>
</table>

The soleus and medial gastrocnemius muscle volume correlated with the ankle plantar flexion strength deficit \( (p = 0.41 \text{ to } 0.56, \ P < 0.001 \text{ to } 0.003) \) (Table 11). The FHL muscle volume correlated negatively with the plantar flexion strength deficit \( (p = -0.37 \text{ to } -0.38, \ P = 0.006 \text{ to } 0.008) \) and with AT elongation \( (p = -0.3, \ P = 0.031) \), indicating a FHL muscle compensatory hypertrophy to ankle plantar flexion strength deficit and AT elongation (Table 11).

Table 11. Muscle Volume Correlation with Plantar Flexion Strength.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Soleus muscle volume, deficit</th>
<th>Medial gastrocnemius volume, deficit</th>
<th>Lateral gastrocnemius volume, deficit</th>
<th>FHL volume, deficit</th>
<th>Deep flexor volume, deficit</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>( \rho )</td>
<td>( P )</td>
<td>( \rho )</td>
<td>( P )</td>
<td>( \rho )</td>
</tr>
<tr>
<td>Work displacement deficit 10°–0°</td>
<td>0.1</td>
<td>0.53</td>
<td>0.1</td>
<td>0.21</td>
<td>0.14</td>
</tr>
<tr>
<td>Work displacement deficit 0°–10°</td>
<td>0.43</td>
<td>0.002</td>
<td>0.44</td>
<td>0.001</td>
<td>0.49</td>
</tr>
<tr>
<td>Work displacement deficit 10°–20°</td>
<td>0.56</td>
<td>&lt; 0.001</td>
<td>0.52</td>
<td>&lt; 0.001</td>
<td>0.43</td>
</tr>
<tr>
<td>Work displacement deficit 20°–30°</td>
<td>0.52</td>
<td>&lt; 0.001</td>
<td>0.41</td>
<td>0.03</td>
<td>0.27</td>
</tr>
</tbody>
</table>
6 Discussion

6.1 General considerations

The strengths of study I and II included their prospective randomized design, homogenous group of patients, and good coverage in the MRIs. Immobilization was identical in both study groups. One surgeon treated nearly all the patients. One researcher without prior knowledge of the treatment group or strength measurements evaluated all MRI measurements. The strengths of study III and IV included their prospective design, the homogenous group of patients, and the standard rehabilitation protocol. Few patients were lost to follow-up, with 92% of patients attending the latest check-up. All the results were analyzed using the contralateral leg as a control, and two blinded researchers evaluated all MRI measurements.

A limitation of study I is that the clinical observers who performed the isokinetic strength tests were not systematically blinded to the treatment modality. Also, many eligible patients refused to participate in the study. All patients received identical rehabilitation instructions after immobilization, but we could not monitor the compliance of the patients to the exercise program. Our follow-up time was 18 months, and it is possible, but unlikely, that strength might recover with a longer follow-up, because previous studies have shown only minimal changes of calf muscle strength after 18 months of follow-up (Lanto et al. 2015).

A primary limitation of study II was that we could not evaluate the actual tendon length in the healthy leg. Because AT length correlates substantially with patient height (Rosso et al. 2012), and the baseline data showed no difference in patient heights, substantial error is unlikely. Our method for measuring Achilles tendon length, based on the soleus MTJ, has not been validated for MRI but described recently by Silbernagel et al. (2016) for US. Barfod et al. (2015) validated a similar technique in which they measured the distance between the calcaneus and the medial gastrocnemius MTJ. The soleus MTJ is likely also a valid location for measuring AT length. Although soleus muscles classified as Goutallier grades 2–4 were observed substantially more frequently in the conservative compared to the surgical treatment (89% vs. 65%) at 18 months, the difference was not statistically significant. This result suggested that study II may have been underpowered.

One weakness of study III and IV was the long period between follow-up visits. In addition, patients may not have accurately recollected later ankle injuries. The
isokinetic dynamometer changed during the follow-up period, but this probably had minimal effect as we used the healthy side as a reference. Our long term results may not be generalizable to conservative treatment for ATR.

6.2 Isokinetic calf muscle strength recovery after surgery vs. conservative treatment of ATR

Although surgical and nonsurgical treatments of acute ATRs resulted in similar AT performance score, surgery led to faster and better recovery of isokinetic calf muscle strength. Surgically treated patients had up to 24% higher results in both PT and ASPT at 6 months. At the 18-month follow-up, the strength difference still favored surgically treated patients, by as much as 14% in PT and 18% in ASPT. Both techniques failed to restore muscle strength to that of the contralateral side.

The recovery of calf muscle strength after surgical and nonsurgical treatments has been contradictory in previous studies (Keating et al. 2011, Nilsson-Helander 2010). Our results support those of previous studies that showed differences favoring surgical treatment, at least in the short term (Keating et al. 2011, Nilsson-Helander 2010, Olsson 2013). The clinical relevance of strength deficit and patient satisfaction has not been studied in the ankle joint. Mullaney et al. (2006) found that patients with isometric plantarflexion strength deficits of up to 34% were unable to perform a decline heel rise. Even though up to 65% of our study patients had a plantar flexion strength deficit at 18 months, this did not prevent them from performing their recreational activities. None of our patients was a competitive athlete, which probably explains the high satisfaction rate despite the major calf muscle strength impairment. Also, Nilsson-Helander et al. (2010) and Lawrence et al. (2017) found in their studies that objective and subjective results do not always correlate with each other; these results underscore the importance of using objective strength tests for evaluating outcome after ATR.

The optimal rehabilitation protocol in nonsurgical treatment or after the surgical repair of acute ATR remains controversial. Early motion with early weight bearing encourages tendon healing and may promote reorganization of collagen and therefore may be critical for functional rehabilitation (Valkering et al. 2016). Compared with previous studies with early plantar flexion or early weight bearing, we allowed both early motion and weight bearing (Nilsson-Helander 2010, Olsson 2013, Willits 2010). In contrast to our results, these previous studies reported similar calf muscle strength after nonsurgical treatment and surgery. No study has
compared different conservative treatment protocols in terms of calf muscle recovery.

6.3 Structural changes of calf muscles and Achilles tendon after surgery vs. conservative treatment of ATR

The results of this study showed that conservative treatment of acute ATRs led to greater soleus muscle atrophy when compared to surgical treatment with the identical rehabilitation protocol. Soleus muscle atrophy in the affected leg was compensated with hypertrophy of the FHL and deep flexors in both groups, but surgery produced 10–18% more calf muscle isokinetic strength than conservative treatment after 18 months. Atrophy of the soleus muscle correlated substantially with the isokinetic peak torque deficit in the calf muscle. Patients treated conservatively showed somewhat greater fatty degeneration of the muscle than was observed in patients treated surgically, but the difference was not statistically significant between our study groups. However, higher fatty degeneration was associated with a smaller soleus muscle volume and less plantar flexion strength in the affected leg. The average AT length was 19 mm longer after conservative treatment than after operative treatment. This result suggested that the greater tendon elongation observed in conservative treatment was partly responsible for soleus atrophy.

Rosso et al. (2013) performed a retrospective study on 52 patients with unilateral ATR that were treated with open, percutaneous, or conservative treatment. In contrast to our study, they found no difference in calf muscle volumes among the three treatment methods. However, they found a 17% deficit in the calf muscle volume and marked fatty infiltration, compared to the unaffected leg, regardless of the treatment method (Rosso et al. 2013). Like Rosso et al. (2013), we found no substantial difference in fatty degeneration between study groups, but the affected leg calf exhibited substantial fatty infiltration compared to the unaffected side. The mean follow-up of Rosso et al. (2013) was 91 months, significantly longer than the follow-up period in our study. However, it is unlikely that atrophy or fatty degeneration observed at 18 months would recover during a longer follow-up.

Volume atrophy and fatty degeneration of the soleus muscle may have several explanations, including a change in the muscle fiber type distribution, the degree of immobilization, and AT lengthening. Three other animal studies showed that muscle type I fibers were more vulnerable to immobilization than type II fibers.
Type I fibers predominate in the soleus, and type II fibers predominate in the gastrocnemius; these different distributions of muscle type I and II fibers may explain the finding that the soleus muscle exhibited greater atrophy and fatty degeneration than the gastrocnemius muscle. (Booth and Kelso 1973, Gollnick et al. 1974, Joza et al. 1988). Soleus atrophy may also be related to ankle immobilization with the Vacoped® orthosis. When the ankle is immobilized, the soleus does not work at all, but the gastrocnemius, which crosses the knee joint, remains partly active during knee flexion. This total immobilization of the soleus could be one unavoidable factor in atrophy and fatty degeneration. The results of a comparison of AT lengthening and gastrocnemius recession to immobilization in a rabbit model suggested that the calf muscle underwent atrophy and fatty infiltration after AT lengthening and gastrocnemius recession, but immobilization did not contribute to either process (Booth et al. 2009). The etiology of fatty degeneration of the muscles after tendon injury is probably multifactorial. Therefore, in the present study, taking into account that both treatment groups received the identical rehabilitation protocol, tendon lengthening was the likely explanation for differences in muscle volumes and plantar flexion strengths between the treatment methods.

Our results confirmed those of Rosso et al. (2013), who found a substantial correlation between calf muscle volume and peak plantar flexion torque at the ankle joint. Muscle atrophy measurement based on actual muscle volume is probably more accurate than estimating atrophy from muscle cross-sectional area, because the correlation coefficients in our study and those in the study by Rosso et al. (2015) were substantially higher than correlations reported in previous investigations that based muscle atrophy on cross-sectional area (Haggmark et al. 1986, Mandelbaum et al. 1995, Rebeccato et al. 2001). In contrast to Rosso et al. (2015), we found that fatty degeneration was also associated with the peak torque deficit in the affected leg. This discrepancy may have been due to differences in study design between our study and the study by Rosso et al. (2015); that study had a retrospective design, the treatment methods were heterogeneous, and the follow-up was 91 months.

AT elongation has been proposed as an explanation for weakness in end range plantar flexion after ATR surgical repair (Mullanney et al. 2006, Silbernagel et al. 2012) We found that the correlation between soleus volume and angle-specific peak torque was strongest at 20° plantar flexion. This result suggested that soleus muscle volume atrophy affected muscle strength, particularly at the end of the range of plantar flexion. The highest correlation coefficient for soleus muscle volume was
0.6, which indicated that muscle volume atrophy might explain 36% of the peak torque deficit. Clearly, several factors other than the soleus muscle volume must contribute to difference in reduce torque capacity between treatment groups. In addition, earlier studies have shown that the contribution of the calf muscles to the total plantar flexion torque is approximately 65 to 85% (Gregor et al. 1991, van Zandwijk et al. 1998). Finni et al. (2006) studied patients with operatively treated ATRs and found that early recovery of plantar flexion torque may be due to compensation by the FHL muscle in addition to normalization of the calf muscle function. We also found that the FHL and deep flexors (digitorum longus and tibialis posterior) showed clear hypertrophy from 3 to 18 months, regardless of the treatment method. This finding indicated that the calf muscle weakness had been compensated by other flexors.

Previous investigations with MRI-techniques or tantalum beads have suggested that surgery and conservative treatment resulted in similar elongations of the AT (Rosso et al. 2013, Schepull et al. 2012). In our study, within first 3 months post injury, the AT was, on average, 17 mm longer with conservative treatment than with surgery. This discrepancy with previous studies may be due to the different study designs. The study by Rosso et al. (2013) was retrospective, and the study by Schepull et al. (2012) used tantalum beads, which were implanted into the tendon during surgery, but in the conservative treatment, they were implanted at 3.5 weeks after the injury, during the cast change. This delay might explain the similar elongation measurements between groups, because the AT elongates from the start of treatment to 3 months after the injury, when weight bearing is allowed with an immobilizing cast or a walker (Kangas et al. 2007, Pajala et al. 2009). Rosso et al. (2013) and Schepull et al. (2012) also used less aggressive rehabilitation protocols, compared to that used in our study; this could also partly explain the contrasting results.

We excluded patients from the study if US showed a gap between the tendon ends in passive plantar flexion of the ankle joint. Hutchison et al. (2015), on the contrary, allowed up to 1 cm gap between the tendon ends in passive plantar flexion in their study of 273 patients treated conservatively with functional orthoses and early weight bearing. Despite the gap up to 1 cm, subjective and functional results were satisfactory with only 1.1% re-rupture rate at 9 months follow-up (Hutchison et al. 2015). Lawrence et al. (2017) published prospective cohort study aimed to study, whether the gap between tendon ends for conservatively treat ATR patients would have impact on functional outcome with 6-months follow-up. They concluded that patients with a gap ≥10 mm between tendon ends in the neutral
position of the ankle had significantly greater peak torque deficit than those with gaps < 10 mm.

### 6.4 Long-term isokinetic calf muscle strength recovery after augmented vs non-augmented repair of ATR

The present 14-year follow-up study showed that augmented and non-augmented surgical repair of ATR produced similar clinical results and isokinetic strength results. Clinical scores and isokinetic strength did not substantially change between 12 months and 14 years postoperatively. The median plantar flexion strength deficit for the whole range-of-motion ranged from 12.2 % to 18.0 % when compared with the uninjured side, supporting the permanence of strength deficits measured at one year.

Several augmentation techniques for AT repair have been reported (Garabito et al. 2005, Gerdes et al. 1992, Hollawel et al. 2014, Kangas et al. 2003, Lee et al. 2008, Maffulli et al. 2013 and 2005, Peterson et al. 2014, Ponnapula et al. 2010, Silfverskjold 1941, Singh et al. 2014). In cadaver studies, both flap and cross-stitch suture augmentation increase gap resistance up to 29 % compared with simple sutures in AT repair (Lee 2008, Gerdes et al. 1992). Additionally, the increased collagen obtained by augmentation can strengthen the AT, lower re-rupture rates, and enable earlier weight bearing (Bhandari et al. 2002, Nyyssonen et al. 2003, Tezeren et al. 2006, Zell et al. 2000). However, these theoretical advantages of augmentation were not confirmed by our present results or in previous short- to mid-term clinical studies. Augmentation may also have potential disadvantages, especially delayed wound healing due to increased tendon diameter (Maffulli et al. 2005, Zell et al. 2000).

To date, only two randomized clinical studies have compared augmentation and non-augmentation operative techniques (Pajala et al. 2009, Tezeren et al. 2006). Pajala et al. (2009) and Tezeren et al. (2006) reported no differences between augmentation and non-augmentation in terms of AT performance score or isokinetic strengths after 12 to 24 months of follow-up. However, Pajala et al. (2009) found a more marked tendon elongation in the non-augmented group, and tendon elongation was significantly correlated with strength deficits at 12 months postoperatively in the simple repair group. Moreover, one prospective (Aktas et al. 2007) and two retrospective studies have found no differences between augmentation and non-augmentation in short-term follow-up (Jessing et al. 1975, Nyyssonen et al. 2003).
Our present results showed persistent strength deficits even in long-term follow-up and despite good clinical outcome. Similarly, Olsson et al. (2013) showed good clinical outcome despite functional deficits on the injured side two years after ATR, regardless of whether patients were treated surgically or nonsurgically. The etiology of strength deficit is multifactorial, potentially influenced by both anatomical and neuromuscular factors, especially in long term (Suydam et al. 2015).

6.5 Tendon elongation and calf muscle structural changes vs strength deficit after ATR in long-term follow-up

The results of this study showed that the AT elongated after ATR on average by 12 mm in long-term follow-up, despite surgical repair. Additionally, the affected legs had 10 to 13% lower calf muscle volumes compared to the healthy legs. Atrophy of the soleus and gastrocnemius muscles were partly compensated by 6% hypertrophy of the FHL. Fatty degeneration was more common in the affected leg than in the healthy leg and was focused in the soleus muscle. Elongation of the AT, measured using MRI, correlated substantially with plantar strength deficit and both gastrocnemius and soleus muscle atrophy.

Only Rosso et al. (2013) has used similar MRI-techniques to ours to measure AT elongation. In their retrospective study with 91 months’ follow-up they found that the affected AT elongated on average 18 mm. They had mixed treatment methods including conservative treatment and open or percutaneous surgery, and these differences in study design may explain slightly different results to ours. They did not find any correlation between tendon elongation and muscle volumes but they did not measure calf muscle strength (Rosso et al. 2013)

Previous short term studies using metallic markers have shown that AT elongates substantially after surgery during the first 3 months, but the correlation to isokinetic calf muscle strength in the long term is unclear (Kangas et al. 2007, Pajala et al. 2009). Our long-term result using metal radiographic markers supports earlier studies that AT elongation occurs during the first 3 months post injury and suggests that only minimal changes occur after 1 year post surgery. Both metal radiographic marker and MRI-based methods for measuring AT elongation gave similar mean elongation results at 14 years post-surgery, but the metal radiographs did not correlate with elongation measured using MRI, calf muscle isokinetic strength, or calf muscle volume deficit. Measuring AT elongation using radiographic markers was standardized, but it seems uncertain how well the
markers remain in their original positions over the long term. According to our results, the role of tendon elongation is still controversial, and further study is needed to confirm the efficacy of MRI techniques in measuring elongation.

Immediate motion and early weight bearing after surgical repair of ATR is considered to improve early healing response of the tendon and prevent joint stiffness without substantial risk of AT elongation (Valkering et al. 2016). In contrast to this previous finding, we found AT elongation despite delayed weight bearing in our rehabilitation protocol. Valkering et al. (2016) defined the AT elongation by the difference in dorsiflexion of the both ankles at 2 weeks after surgery, which may explain the contrasting results in tendon elongation. Additionally, our rehabilitation protocol allowed immediately greater active free plantar flexion range of the ankle joint compared to that of Valkering et al. (2016), which may have created a predisposition to AT elongation. Although immediate weight bearing with early motion promotes tendon healing, it probably creates a predisposition to subtle AT elongation after surgery.

The retrospective study of Rosso et al. (2013) is the only long-term follow-up to date measuring the actual muscle volumes using MRI after unilateral ATR treated with open, percutaneous, or conservative treatment. They found a 17% deficit in the calf muscle volume and marked fatty infiltration, compared to the unaffected leg, regardless of the treatment method, with mean 91 months follow up (Rosso et al. 2013). Our results are comparable to these results in terms of calf muscle atrophy, but we also found substantial correlation between calf muscle volume and isokinetic plantar flexion strength of the ankle. In addition, we found that soleus muscle fatty degeneration was associated with lower PT and muscle volume for affected leg. Finni et al. (2006) previously suggested that early recovery of plantar flexion torque might be due to compensation by the FHL muscle in addition to normalization of the calf muscle function in short term. Our long term results are consistent with these previous results, since our long-term study showed that plantar flexion strength partly recovers via FHL hypertrophy.

6.6 Clinical implications and future studies

Conservative treatment with functional rehabilitation including early weight bearing is the gold standard in treating majority of ATR patients. However, when treating physically active and demanding patients, surgical ATR treatment may prevent AT elongation and soleus muscle atrophy, resulting in faster and more complete calf muscle isokinetic strength recovery.
The choice of the right treatment for the patient with ATR should be made individually by observing the patient needs and treatment risks. Regardless of the treatment method, major calf muscle strength deficit may persist; therefore, rehabilitation after the conservative or operative treatment may be the most important phase that should be underlined in clinical practice.

The most optimal rehabilitation protocol for conservative treatment or after surgical repair of acute ATR remains controversial. Future research should assess whether more intensive rehabilitation under physiotherapist guidance during the first 12 months might prevent muscle volume loss and ankle plantar flexion strength deficits after conservative treatment without excessive tendon elongation. Additionally, future studies should clarify whether compensatory hypertrophy in the FHL and deep flexors might have any potential long-term effects on the biomechanics and function of the lower extremities.
7 Conclusions

The following conclusion can be drawn based on the results of this thesis:

1. Surgical repair of an ATR restores calf muscle isokinetic strength earlier and more completely than does conservative treatment, with a 10–18 % permanent strength difference favoring surgery.

2. Conservative treatment of ATR results in greater soleus muscle atrophy but similar fatty degeneration when compared to operative treatment with an identical functional rehabilitation protocol. Additionally, the average AT length is 19 mm longer after conservative treatment when compared to operative treatment. These structural changes partly explain the greater calf muscle isokinetic strength observed in patients treated operatively compared to those treated conservatively.

3. Augmentation provides no benefit compared with simple suture repair of an acute ATR. Calf muscle isokinetic strength recovers during the first year post-injury, with only minimal changes thereafter. At the 14-year follow-up examination, the affected side showed the persistence of a 12–18 % plantar flexion isokinetic strength deficit compared to the unaffected side, but the clinical relevance of this small strength deficit remains unknown.

4. AT elongation may explain the smaller calf muscle volumes, fatty degeneration, and persisting plantar flexion isokinetic strength deficit after surgical repair of ATR. The strength deficit is partly compensated by FHL muscle hypertrophy, but a 11–13 % deficit in soleus and gastrocnemius muscle volumes and 12–18 % deficits in plantar flexion isokinetic strengths remain even after long-term follow-up.
References


Silfverskiold N (1941) Über die subkutane totale Achillessehnenruptur und deren Behandlung. 84(Acta Chir Scand): 393.


Appendices

Appendix 1 Rehabilitation program
Appendix 2 Leppilahti Score
Appendix 3 Imaging protocol
## Appendix 1 Rehabilitation program

### Table 1. TRAINING PROGRAM – ACHILLES TENDON RUPTURE

**Week 8-10**

**Exercise program:**  
Full weight bearing is allowed. 10mm heel raise for shoe is used for 1 month. The purpose of the heel raise is to prevent tendon elongation during walking.

Ankles mobility training, calf muscles and Achilles tendon stretching and training are started upbeat. The following exercises should be done 2-3 times/day for the next three weeks. Recommended sports are exercise bicycling, water running and swimming.  
Thresholds should be started when walking is fluent. Any strong plantar flexion is forbidden during the first 6 months.

<table>
<thead>
<tr>
<th>Exercise</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flex and extend ankles peacently with the maximum movement. When knee extended you feel stretching at calf muscle.</td>
<td>Repeat 20 times, 2-3 times/day.</td>
</tr>
<tr>
<td>Knee extended, pull the ankle to full passive flexion to stretch the Achilles tendon and calf muscle. Bend the upper body on the stretched leg to take up the stretching. Hold 20 sec. Repeat 3-5 times per side, 2-3 times/day.</td>
<td></td>
</tr>
<tr>
<td>Put hand on foot and stretch by extending the ankle. Hold the stretching 30 sec. Feel the stretching on foot and front of the leg.</td>
<td>Repeat 3-5 times per side.</td>
</tr>
<tr>
<td>Butt ankle, changing direction.</td>
<td>Repeat 20 times, 2-3 times/day.</td>
</tr>
<tr>
<td>Standing, light support from the wall at the beginning. Lift the healthy leg from the ground; keep the balance steadily on the whole foot. Do not let the pelvis drop down. Standing on one leg 15-30 sec.</td>
<td>Repeat 20 times, 2-3 times/day.</td>
</tr>
<tr>
<td>Stretching leg calf muscle and Achilles tendon. Brace by hand against the wall/chair. Lean the body forward/down until you feel stretching in the extended legs’ calf muscle. Hold the stretch 30 sec and relax.</td>
<td>Repeat 3-5 times per side, 2-3 times/day.</td>
</tr>
</tbody>
</table>

**Week 11 to 1 year**

**Exercise program:**  
Practice daily for 3 weeks, afterwards 3-4 times per week to one year. Main focus on training is calf muscles endurance and power training. Continue doing the stretching program. 10mm exercise is allowed to start upbeat, avoid leg pressure and repet during the first 4 months from the Achilles tendon rupture. Returning to sports upbeat after four months you may start running, skiing and gym exercise without limitations. Ballgames, contact sports, and competitive sports are allowed after six months from the Achilles tendon rupture.

<table>
<thead>
<tr>
<th>Exercise</th>
<th>Description</th>
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<tbody>
<tr>
<td>Get up on toes and get down slowly. Repeat same movement by heels out rotated.</td>
<td>Repeat 15 times both movements.</td>
</tr>
<tr>
<td>Take alternately small steps straight forward, beside and back with the exercising leg. Return back standing foot by foot gently pushing by ball of the foot. Repeat by another leg.</td>
<td>Repeat 20 times.</td>
</tr>
<tr>
<td>Stretch leg back off the extended leg. Bend the stretched leg from the knee and let body go down until stretching feels in Achilles tendon and bottom of the calf muscle. Hold the stretching 30 sec.</td>
<td>Repeat 3-5 times per side.</td>
</tr>
<tr>
<td>Butt stripe around the foot. Rotate the foot outside. Keep tension 2-3 sec and return slowly start position. Keep the knee steady during the exercise.</td>
<td>Repeat 15 times.</td>
</tr>
<tr>
<td>Place the foot on a stand. Push heel on the floor and keep knee extended. Lean the pelvis forward or take a step over the stand. Stretch the calf muscle and Achilles tendon. Hold the stretching 30 sec.</td>
<td>Repeat 3-5 times per side.</td>
</tr>
</tbody>
</table>
Appendix 2 Leppilahti Score

Pain (15 points)

None 15
Mild, no limitations on recreational activities 10
Moderate, limitations on recreational but not daily activities 5
Severe, limitations on recreational and daily activities 0

Stiffness (15 points)

None 15
Mild, occasional, no limitations on recreational activities 10
Moderate, limitations on recreational but not daily activities 5
Severe, limitations on recreational and daily activities 0

Subjective calf muscle weakness (15 points)

None 15
Mild, no limitations on recreational activities 10
Moderate, limitations on recreational but not daily activities 5
Severe, limitations on recreational and daily activities 0

Footwear restrictions (10 points)

None 10
Mild, most shoes tolerated 5
Moderate, unable to tolerate fashionable shoes, modified shoes tolerated 0

Active range-of-motion difference between ankles (15 points)

Normal (5 deg) 15
Mild (6–10 deg) 10
Moderate (11–15 deg) 5
Severe (16 deg) 0
Subjective result (15 points)

Very satisfied 15
Satisfied with minor reservations 10
Satisfied with major reservations 5
Dissatisfied 0

Isokinetic muscle strength score (15 points)

Excellent 15
Good 10
Fair 5
Poor 0

Maximum possible total 100

At least 90 points excellent,
75–89 points good,
60–74 points fair and
60 points poor
**Isokinetic Ankle Strength Scale for Scoring**

Plantar Flexion and Dorsiflexion Peak Torques of the Ankle at Three Test Speeds (60, 120, and 180 deg/s)

**Plantar flexion peak torque 60 deg/s percentage difference (uninjured-injured) (17 points)**

<table>
<thead>
<tr>
<th>Percentage Difference</th>
<th>Points</th>
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<tbody>
<tr>
<td>≤ 2 %</td>
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<td>15</td>
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<td>&gt; 5 ≤ 10 %</td>
<td>13</td>
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<td>&gt; 50 %</td>
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**Dorsiflexion peak torque 60 deg/s percentage difference (17 points)**

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**Plantar flexion peak torque 120 deg/s percentage difference (17 points)**

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Plantar flexion peak torque 180 deg/s percentage difference (17 points)

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Dorsiflexion peak torque 180 deg/s percentage difference (17 points)

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Maximum possible total 102
Appendix 3 Imaging protocol

Study II

Imaging was carried out with a 1.5-T imaging system (Signa HDxt, General Electric, Milwaukee, WI) between 2009 and 2015. The imaging sequences included T1-weighted spin echo sagittal images (TR/TE 500/13 ms, FOV 150x150 mm, matrix 256x192 pixels, slice thickness 3.5 mm, and 0.5 mm interslice gap) and axial images (TR/TE 420/11 ms, FOV 150x150 mm, matrix 416x224 pixels, slice thickness 3.5 mm, and 0.5 mm interslice gap) of the affected AT and T1-weighted axial spin echo images (TR/TE 640/12 ms, FOV 480x480 mm, matrix 416x224 pixels, slice thickness 5 mm, and 12 mm interslice gap) of both calves, from the distal femoral condyles to the upper ankle joint. Patients were imaged in the supine position, and the ankles were supported in the plantigrade position, with the ankle at a 90° angle and the knee at a 0° angle. The MRI examination was performed on the same day, but before the isokinetic strength tests.

Study III

Imaging was carried out with a 1.5-T imaging system (MAGNETOM Aera; Siemens Healthcare, Erlangen, Germany). The imaging sequences included T1-weighted turbo spin echo (TSE) sagittal (TR/TE 600/9.6 ms, FOV 320x380 mm, matrix 432x512 pixels, slice thickness 4 mm and 0.8 mm interslice gap) and axial (TR/TE 710/9 ms, FOV 380x380 mm, matrix 448x448 pixels, slice thickness 4 mm and 1.6 mm interslice gap) images. T1-weighted sagittal images covered both calves from proximal tibia to distal calcaneus and T1-weighted axial images from distal femoral condyles to distal calcaneus. Patients were scanned in supine position and ankles were supported in the plantigrade position, ankle 90° degrees and knee 0° angle.
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1408. Wang, Qin (2017) Epidemiological applications of quantitative serum NMR metabolomics : causal inference from observational studies


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RECOVERY OF CALF MUSCLE ISOKINETIC STRENGTH AFTER ACUTE ACHILLES TENDON RUPTURE