Citation:
Nicholson, G and Walker, J and Dawson, Z and Bissas, A and harris, N (2019) Morphological and functional outcomes of operatively treated Achilles tendon ruptures. Physician and Sportsmedicine. ISSN 0091-3847 DOI: https://doi.org/10.1080/00913847.2019.1685364

Morphological and functional outcomes of operatively treated Achilles tendon ruptures

Authors: Nicholson, G; Walker, J; Dawson, Z, Bissas, A; Harris, N

Abstract

Objectives: Achilles tendon rupture leads to functional impairments and these may be underpinned by morphological changes in the muscle-tendon unit. Functional performance of the injured limb will be impaired regardless of time since surgery and these impairments occur alongside changes in muscle-tendon morphology.

Methods: Following operative treatment of Achilles tendon rupture and short term immobilisation, 12 patients completed a battery of tests during a single visit to the laboratory (performed an average of 4.4 ± 2.6 years post-surgery). Patients completed the Achilles' tendon rupture score (ATRS), tests of ankle and hip range of motion (ROM) and ultrasound measurements of muscle-tendon architecture. Data on isokinetic (30 °/s, 60 °/s) plantar flexion strength, jumping performance and walking-running were also collected on the same visit. Percentage deficits were expressed relative to the non-injured limb and determined for statistical significance ($p < 0.05$). Relationships between outcomes measures and time since surgery were tested using Pearson's correlation coefficients ($p < 0.05$).

Results: The repaired limb showed a shorter muscle fascicle length (12.1-19.6%), increased fascicle pennation (18.0±22.14%) and reduced muscle thickness (9.1-20.1%) in the gastrocnemius and/or soleus along with greater tendon cross-sectional area (46.7±34.47%). Functionally, the repaired limb displayed lower countermovement jump height (-12.6±15.68%) and longer drop jump contact times (5.5±5.7%). Also, the repaired limb showed reduced hip internal-external ROM (6.3±8.2%) but no differences existed between limbs for plantar flexion ROM and strength or gait characteristics. Good ATRS outcomes were reported (mean: 87.9±16.2, range: 43-100) which related to time since surgery ($r=0.79$) but individual ATRS items did not correlate with corresponding objective measures.

Conclusion: Plantar flexor atrophy following surgically treated Achilles tendon rupture is partially compensated for by remodelling of the fascicles however, impairments may still persist many years into the postoperative period although these may be more pronounced in high-velocity activities.

Keywords: isokinetic, ATRS, muscle morphology, gait

Introduction

The Achilles tendon is the strongest tendon in the human body with a crucial role in transmitting mechanical energy generated by the triceps surae complex. Incidence of Achilles tendon rupture has been estimated at between 6-21.5 per 100,000 [1,2] with notable increases being reported in recent decades [3]. Although non-operative treatment of Achilles tendon ruptures avoids many of the complications associated with surgery (e.g. infection), decreased rates of re-rupture, reduced tendon lengthening and less atrophy have been reported with operative management [4-6].

Impaired functional outcomes shown for individuals with a history of Achilles tendon rupture include reduced ankle proprioception, decreased plantar flexor strength and asymmetric performance in activities requiring propulsion [7-9]. Despite the prevalence of Achilles tendon ruptures, research into post-operative deficits has generally focussed on relatively short follow-up periods of 6-36 months [10,11]. Recent research has provided evidence of long-term deficits (>10 years) post-surgery [12,13] which include reduced range of motion (ROM), lower strength and impaired functional performance (i.e. heel raise) in the injured leg. Whilst information on the long-term outcomes is important, it is unclear whether the patients underwent homogenous surgical treatment and postoperative rehabilitation strategies, which are known to impact on patient outcomes [14,15].

Apart from functional deficits, Achilles tendon rupture results in alterations of the neuromechanical properties of the muscle-tendon unit including tendon elongation, triceps surae atrophy and compensatory changes in muscle activation [12, 13, 16]. Manegold and co-workers [17] showed that Achilles tendon elongation and tricep surae atrophy are related to the deficits in strength and ROM that are often seen in the ankle joint. This remains one of few studies that has sought to understand the mechanisms underpinning deficits in functional outcomes. In terms of muscle morphology, recent studies [16,18] have suggested medial gastrocnemius remodelling following Achilles tendon rupture (larger pennation, reduced thickness, reduced aponeurosis stiffness and shorter fascicles) as a compensatory mechanism to restore resting tendon tension. However, the same remodelling changes have been found to be connected to impairments in the triceps surae function as it was measured through heel raise tests [16,18,19]. Although these studies provide important information about morphological remodelling, they have not integrated a wide range of functional tasks (e.g. jumping) alongside the morphological measures nor have these studies investigated post-surgery periods longer than 2.5 years.

Therefore, despite the wealth of research investigating the outcomes of operatively treated Achilles tendon ruptures, there is a need to investigate morphological and biomechanical outcomes alongside a broad range of functional activities over a long follow-up period whilst standardising confounding factors relating to the surgical treatment and rehabilitation strategies. This will permit a clearer understanding of the morphological factors underpinning deficits in functional tasks which impact on daily living allowing rehabilitation strategies to be optimised. Consequently, this study aimed to retrospectively examine the morphological and functional outcomes of operatively treated Achilles tendon ruptures.

Materials and Methods

Twelve patients (ten men, two women) participated in the current study (characteristics upon testing; age [mean ± SD]: 43.3 ± 13.6 years; stature: 1.74 ± 0.09 m; body mass: 80.2 ± 10.5 kg). They had all previously suffered unilateral Achilles tendon rupture and had undergone operative treatment, the time since surgery of the cohort was 4.4 ± 2.6 years (range: 0.7-9.4 years). Following a medical screening assessment, participants provided informed consent and completed the Achilles Tendon Rupture Score (ATRS) to assess patient reported outcomes [20]. The study received approval from the university research ethical committee and was in accordance with the Declaration of Helsinki.

Operative treatment

All participants underwent an identical operative procedure performed by the same surgeon. Each patient was positioned prone and a 5cm posteromedial curvilinear incision was made centred over the Achilles tendon rupture. The frayed ruptured ends were debrided and Bunnell type sutures were inserted into each end of the ruptured tendon ends using number 1 absorbable Vicryl plus sutures. The foot was placed in maximum plantar flexion and the ends of the ruptured tendon approximated and tensioned with 2 knots medially and laterally. The leg was initially immobilised in full plantar flexion in plaster for 2 weeks, following suture removal the patients were placed in a semi-equine weight bearing cast for a further 2 weeks. At 4 weeks the plaster was removed and patients were placed in a walker boot in a plantigrade position for another 4 weeks during which time fully weight bearing was permitted along with range of motion exercises. At 8 weeks post-surgery patients started to mobilise out of the boot and commenced formal departmental physiotherapy for a 6 month period.

Testing protocol

Jumping performance

Participants performed unilateral countermovement jumps and bilateral 30 cm drop jumps onto two synchronised force platforms (Kistler; Switzerland) sampling at 2,000 Hz. Following familiarisation, participants performed five maximal jumps per condition with hands akimbo and they were urged to keep contact time as minimum as possible. The average values for jump height, contact time, reactive strength index (RSI) and for peak landing force (drop jump only) were calculated. Jump height was calculated from flight time ($d = v_i t * ½ at^2$) whilst RSI by dividing the jump height in the drop jump by the contact time; for the countermovement jump, contact time was replaced by time to take-off in order to calculate RSImod [21].

Gait assessment

The gait assessment was carried out using a treadmill (h/p/cosmos, Gaitway; Germany) instrumented with two embedded force platforms. To replicate normal and running gait, participants walked at 1.11 $m·s^{-1}$ for several minutes before 30 s of data collection whilst the same protocol was replicated for running at 2.22 $m·s^{-1}$. Step length and contact time were obtained for both limbs during both conditions. Weight acceptance and push off rates were assessed to quantify the efficacy of the plantar flexor complex in accepting and producing force during locomotion.

Range of motion assessments

A knee-to-wall test was carried out by progressively moving the foot away from a wall and moving the knee forward until it made contact with the wall. The test was then terminated when the knee could no longer reach the wall, with the best successful attempt being taken as the measurement. This measurement was taken at the toe and heel (to compensate for potential differences in foot length). Maximal ROM of internal and external rotation at the hip joint was taken in prone, supine and seated positions with a goniometer (E-Z Read; Greendale, USA). Each measurement was repeated three times to ensure repeatability.

Morphological measurements of the muscle-tendon complex

Static ultrasound measurements (figure 1) of the triceps surae muscle-tendon complex were taken with the participants laying prone on a physiotherapy bench and the ankle joint in a neutral position. Longitudinal images were taken of the muscle belly of *m.* Gastrocnemius medialis, *m.* Gastrocnemius lateralis and *m.* Soleus in both legs using B-mode ultrasound with a 50 mm linear array probe (5 to 12 mHz; Acuson P300, Siemens; Munich). Bilateral transverse plane images of the Achilles tendon for the measurement of the cross-sectional area were taken 40 mm proximal to the calcaneal insertion, while longitudinal images of the Achilles tendon were taken 30, 40 and 50 mm proximal to the calcaneal insertion using a 40 mm linear array probe (6 to 18 mHz). These sites were chosen since the region 20-60 mm proximal to the calcaneal insertion is the most commonly ruptured region [22].

FIGURE 1 HERE

Ultrasound images were analysed using ImageJ (Java, National institution of health). Achilles tendon thickness was measured from the superficial to deep edges of the tendon with muscle thickness measured from the deep to superficial aponeuroses [23]. An average of three images was used for all variables. The angle between the deep aponeuroses and a fascicle that met the deep aponeuroses was defined as the pennation angle [18]. Fascicle length was established through trigonometry using the pennation angle and muscle thickness [16].

FIGURE 2 HERE

Strength assessment

Participants underwent strength assessments of the triceps surae using an isokinetic dynamometer (System 4 Pro, Biodex Medical Systems; NY, USA) in two testing positions: a) seated with a hip angle of approximately 65° and a fully extended knee joint, b) same hip angle with the knee in 50° of flexion. Following familiarisation, the peak concentric torque was measured for the injured and non-injured leg at two angular velocities (30 and $60°\cdot s^{-1}$) over three maximal trials.

Data analysis

Statistical analysis was carried out using the Statistical Package for the Social Sciences (SPSS, version 24.0). Between-limb differences (injured vs. non-injured limb) were analysed using a

Paired-samples t-test. Percentage differences were calculated between the two limbs as well as the limb symmetry index ([LSI] injured limb value / non-injured limb vale x 100). Potential relationships between variables and time since surgery were tested using Pearson's correlation coefficient. For all statistical tests, homogeneity of data was confirmed and significance levels were set to $p < 0.05$.

Results

ATRS Outcomes

For the responses to each ATRS item, there was a lower mean score for perceived stiffness (item 3) with the highest mean scores being reported for perceived pain (item 4), limitations during daily living (item 5) and limitations in stair climbing (item 7) (table 1). Strong positive correlations (r = 0.70-0.87) were observed between a number of ATRS outcomes and the months since surgery with the strongest correlation being observed for ATRS item 2 (tiredness at the ankle).

TABLE 1 HERE

Strength Outcomes

Despite a mean difference in peak torque (straight leg) at 30°/s and 60°/s for the two limbs, ankle plantar flexion PT was not statistically different between the two sides at either testing speed (table 2) mainly due to the variability of scores. The peak torque limb symmetry values showed an improvement when the test was performed with the knee flexed. No significant correlations were observed between the strength limb symmetry outcomes and time since surgery.

TABLE 2 HERE

Functional Outcomes

Regarding functional performance, countermovement jump height was significantly lower (p=0.02) on the injured compared to the non-injured limb (LSI: 87.4 ± 15.7%) with significant differences (p=0.01) also being observed in RSImod between the two limbs (LSI: 85.8 ±

17.6%) (table 3). Similar functional deficits were observed in the drop jump with a significantly longer contact time (p=0.01) for the injured side (LSI: 94.5 ± 5.7%) but there were no significant differences in landing forces (LSI: 88.4 ± 19.7%). No significant correlations were observed between the jump limb symmetry outcomes and time since surgery.

TABLE 3 HERE

In contrast to the functional deficits observed during jump activities, there were no significant differences between limbs for any gait variable measured (table 4). Furthermore, LSI for numerous gait outcomes appeared unrelated to the time since surgery.

TABLE 4 HERE

Morphological Outcomes

The injured limb demonstrated morphological differences in the Achilles tendon (figures 2 and 3) and lower-limb muscles (table 5) compared to the non-injured limb. Specifically, the Achilles tendon on the injured limb displayed significantly increased thickness at 3cm (LSI: 149.7 ± 29.5%), 4cm (161.3 ± 32.2%) and 5cm (177.6 ± 41.6%) and greater cross-sectional area (46.7 ± 34.5, LSI: 146.9 ± 34.5%, p=0.001) than on the non-injured side.

FIGURE 3

The medial gastrocnemius (LSI: 90.9 ± 9.4%, p=0.01) and soleus (LSI: 79.9 ± 19.0, p=0.01) displayed significantly greater thickness on the non-injured side as well as a significantly longer fibres for the medial gastrocnemius (LSI: 80.4 ± 18.0%, p=0.00) and lateral gastrocnemius (LSI: 87.9 ± 18.9%, p=0.03) and soleus (LSI: 80.4 ± 15.3%, p=0.00). Only the fascicles of the medial gastrocnemius showed a significantly greater pennation angle (LSI: 118.0 ± 22.1%, p=0.02) on the injured side. No significant correlations were observed between the morphological limb symmetry outcomes and time since surgery.

TABLE 5 HERE

Range of Motion Outcomes

No significant differences were observed between the two limbs for ankle ROM, furthermore the LSI for ankle ROM showed no significant relationships with time since surgery (table 6). For the hip ROM in seated position, there was markedly less internal (LSI: 89.92 ± 15.07%) and total (LSI: 93.75 ± 8.16%) ROM in the injured limb with the total ROM reaching statistical significance (p=0.02).

TABLE 6 HERE

Discussion

This study examined the morphological and functional outcomes of operatively treated Achilles tendon ruptures. Significant differences were found between injured and non-injured limbs many years after surgery with individual variation in deficits despite standardised treatment and rehabilitation. In addition to the changes in Achilles tendon structure following surgical repair, the muscle fibres in the triceps surae complex display architectural changes resulting in superior muscle quality in the non-injured limb. Importantly, these alterations occur alongside deficits in functional performance, with propulsion and landing during jumping activities being impacted to a greater extent than gait activities. Finally, it was noted that patient reported symptoms do not always reflect objective measures of functional deficits despite being widely utilised.

The significant reductions in muscle thickness in the medial gastrocnemius and soleus agree with previous studies but the current findings highlight the fact that plantar flexor atrophy may be chronic in nature [12,13] and therefore represents an ongoing challenge during rehabilitation. Interestingly the lateral gastrocnemius did not demonstrate the same magnitude of atrophy as the other plantar flexors. It is plausible the differing responses of the plantar flexors reflect their contrasting contributions to different activities [24,25] or that regional differences in muscle atrophy were not detected by the single region uniplanar ultrasonography despite ultrasound corresponding well with gold-standard imaging techniques [26]. Whilst chronic atrophy of triceps surae complex theoretically indicates a reduced capacity for force generation, the significant increases in Achilles tendon thickness and cross-sectional area more likely reflect the healing process rather than improved mechanical properties of the tendon [27]. Although increased Achilles tendon thickness has been consistently observed post rupture

[28], the findings underline that asymmetries in thickness may be chronic in nature and are more pronounced at more proximal regions.

Although the changes in muscle and tendon thickness post rupture were somewhat expected, the significant increases in fascicle pennation and decreases in fascicle length in the injured limb demonstrate the multi-faceted nature of muscle-tendon function and highlight the need to consider tissue quality as well as tissue quantity. Skeletal muscle has been shown to remodel is response to alterations in surrounding structures in animal models [29,30] and whilst changes in plantar flexor architecture have been recently observed in humans following Achilles tendon rupture [16,18,19], this study highlights the long-lasting nature of these changes. It seems likely that the changes in fascicle pennation and length may have occurred as one of many compensatory mechanisms to the loss in muscle size. Specifically, the observed increases in fibre pennation may have facilitated increased fibre density [31] and tendon tension whilst decreases in fascicle length may have served to restore resting tension caused by tendon elongation [16,18]. In terms of fascicle length, Svensson et al. [19] showed significant reductions (18%) in fascicle length of the medial gastrocnemius 2 years after Achilles tendon rupture with the authors highlighting the importance of this change with respect to the capacity to perform dynamic movements effectively. Although the consequences of greater pennation cannot be fully appreciated through two-dimensional ultrasonography, the trends towards tendon elongation in the injured limb and associations between Achilles tendon length and fibre length asymmetries are supportive of such outcomes [32,28].

Functionally, the chronic atrophy and changes in tendon characteristics did not affect ankle plantar flexion isokinetic strength suggesting that compensatory remodelling of the triceps surae is successful at limiting reductions in ankle joint function. Imaging asymmetries have been observed to exist without impaired functional performance [33], something that necessitates the inclusion of function assessment during rehabilitation. Obviously, the success of the compensatory remodelling mechanisms presumes a given level of neural activation during the strength assessment, so increased neural activation could be another mechanism for the recovering leg as the effect of enhanced neural drive on maximum contractions is well established [34]. Whilst the isokinetic assessments in the current study were strengthened by the use of differing knee positions to discern between gastrocnemius and soleus contribution, variation was evident in the magnitude of strength asymmetries with some individuals displaying limb symmetry values of below 85% years after surgery. Indeed, previous investigations [12,35] have proved controversial when attempting to link morphological and strength changes following Achilles tendon ruptures and although isokinetic testing provides a reliable means of isolating the joint over a range of motion, time-dependent variables such as angular work may be more sensitive to changes that take place following Achilles tendon rupture [36].

The fact that no strength impairments were observed in the injured limb was also consistent with the absence of gait asymmetries despite some individuals reporting perceived impairments in locomotion during everyday activities. Baxter et al. [18] suggested that gait analysis is a poor test of patient function following an AT rupture since stereotypical gait patterns have been previously observed in patients with severely compromised plantar flexors [37]. Whilst the lack of differences in gait variables may be population sample related, it is also plausible that mechanisms such as elastic energy transfer and/or optimisation of muscle activation have compensated over time for the gait asymmetries [12].

Although recent research [16,18,19] correlated architectural remodelling with reductions in functional daily activities (i.e. heel raise measurements), the present findings go one step further by highlighting the architectural remodelling that occurs alongside the high-velocity functional activities that are performed in many sports. Whilst the aforementioned architectural changes would appear to minimise impairments in the injured limb, it is likely that these changes may also result in decreases in muscle force transmission due to fascicle angulation [38] and reductions in peak power at higher angular velocities due to shorter length of fibres [39]. The significant reductions in jump height, increased take-off times and reduced RSImod scores in the unilateral countermovement jump are indicative of impaired concentric propulsion capabilities of the plantar flexors [40]. Moreover, it would appear that patients placed a greater reliance on the non-injured limb during the bilateral drop jump as suggested by the significantly longer ground contact times. Whilst individuals may adopt an altered landing strategy to protect the injured side during propulsive and landing phases of jumping, sustained chronic loading of the non-injured side may itself pose a greater risk of further injury. Therefore, there is a strong rationale to develop rehabilitation programmes which integrate functional jumping/landing and targeted resistance training aimed at restoring fascicle morphology [41,42]. However, the challenging decision is the determination of the onset of such rehabilitation programmes along with their intensity and load characteristics. Studies by Eliasson et al. [28,43] have shown clearly that the tendon undergoes a healing process many months following repair with full function regained at least 1 year after surgery. Therefore, it is reasonable to speculate that the nature and size of the muscular adaptions discussed above may be driven by the tendon recovery process, as different muscle adaptations will result from a tendon exposed to excessive loading during healing compared to a tendon undergoing a moderate load recovery process.

The reduced hip external and internal rotation ROM of the injured limb was a novel finding of the present investigation. That being said, previous investigations into running mechanics following Achilles tendon ruptures have observed increased knee joint loads [44] and internal knee abduction moments [45] in the injured limb. It is not possible to identify if impairments in the ROM in the current study are the result of post-operation movement management or they existed prior to the Achilles tendon rupture. However, as previous research has linked femoral mechanics with lower limb compensatory mechanisms and loading of Achilles tendon, there may be a need to consider hip function in clinical assessments aimed at preventing Achilles tendon rupture or re-rupture [41,46,23].

Long lasting differences between the injured and non-injured limbs have been observed previously [12,13] in individuals who have undergone surgical repair of an Achilles tendon rupture. Whilst some individuals showed limited or no impairments in the injured limb, notable (i.e. LSI <85%) long-lasting differences manifested themselves for a number of individuals in the functional, morphological and self-reported measures. This further supports the previous suggestion that there are limited meaningful improvements as the time since surgery increases [47]. Only the self-reported patient outcomes showed significant correlations with time since surgery indicating reduced perceived impairments as time goes by. The lack of association between objective patient outcomes and time since surgery may be indicative of the variation seen between individuals in the magnitude of asymmetries. This variation was evident despite patients having followed the same rehabilitation pathway after undergoing identical surgical procedures. This further highlights the multi-faceted nature of Achilles tendon rupture outcomes, a factor which healthcare practitioners should consider when managing patient expectations. The lack of association between objective outcomes and time since surgery should be interpreted with caution given the small sample size of the current study. Indeed, a number of individuals displayed marked impairments in the injured limb a number of years following surgery, which may highlight the higher sensitivity of biomechanical markers over subjective markers for monitoring the time-course of recovery. This is supported by the fact that the perceived impairments scored on ATRS items 1, 3, 8 and 9 did not closely reflect objective assessments of strength, stiffness, running and jumping.

In addition to the long-lasting impairments observed in many patients, individual variation was evident in the magnitude of inter-limb asymmetries even in patients matched by time since surgery. Furthermore, this individual variation was evident despite this being one of few studies to examine surgical outcomes of Achilles tendon ruptures in individuals that have followed the same post-operative limb loading protocol and the same 6-month physiotherapy follow-up after undergoing an identical surgical protocol by the same surgeon. Research [14,15,28,43] has shown the aforementioned factors to be important factors in influencing patient outcomes and the reporting of these aspects should be encouraged within the scientific literature. It must be acknowledged that there are still multiple factors capable of impacting on patient outcomes which were not controlled in the current study including the degree of tendon separation prior to surgery [48], the magnitude of tendon elongation post-surgery [32] and an individual's level of engagement in the rehabilitation programme. As such, healthcare practitioners should be mindful of the individual nature of recovery when managing patient expectations.

This study was affected by several limitations. The study is retrospective, meaning pre-injury levels of asymmetry are not known. The sample size was restricted and heterogeneous for age and time post-surgery. Whilst this may have resulted in some variability, the standardisation of surgical methods, surgeon and rehabilitation pathway permitted the control of important confounding variables. As such, the study provides novel information on self-reported, morphological and functional outcomes following AT ruptures treated in a homogenous manner.

Conclusion

This study highlighted that the triceps surae complex undergoes architectural and structural changes following surgical repair of Achilles tendon rupture. These chronic changes appear to partially compensate for plantar flexor atrophy and tendon elongation in the postoperative period. Nevertheless, functional impairments in the repaired limb may persist which may be more pronounced in high-velocity activities. Further work is required to optimise morphological rehabilitation strategies in the postoperative period with the aim of minimising long-term changes in the structure and architecture of the muscle-tendon unit. From a monitoring perspective, it would appear that patient reported symptoms do not always closely reflect objective measures of functional outcomes. Finally, individual variation in patient outcomes exists despite standardised treatment and rehabilitation routes.

Funding

There are no sources of funding to declare.

Declaration of interest

The authors have no other relevant affiliations or financial involvement with any organization or entity with a financial interest in or financial conflict with the subject matter or materials discussed in the manuscript.

References

1. Maffulli N. Rupture of the Achilles tendon. JBJS. 1999;81(7):1019-36.
2. Lantto I, Heikkinen J, Flinkkilä T, Ohtonen P, and Leppilahti J. Epidemiology of Achilles tendon ruptures: Increasing incidence over a 33-year period. Scandinavian journal of medicine & science in sports. 2015;*25*(1), e133-e138.
3. Aujla R, Sapare S, Bhatia M. Acute Achilles tendon rupture treatment: Where are we now? Journal of arthroscopy and joint surgery. 2018;5(3):139-44.
4. Cetti R, Christensen SE, Ejsted R, Jensen NM, Jorgensen U. Operative versus nonoperative treatment of Achilles tendon rupture: a prospective randomized study and review of the literature. The American Journal of Sports Medicine. 1993;21(6):791-9.
5. Calder JD, Saxby TS. Early, active rehabilitation following mini-open repair of Achilles tendon rupture: a prospective study. British journal of sports medicine. 2005;39(11):857-9.
6. Wilkins R, Bisson LJ. Operative versus nonoperative management of acute Achilles tendon ruptures: a quantitative systematic review of randomized controlled trials. The American Journal of Sports Medicine. 2012;40(9):2154-60.
7. Mortensen NH, Skov O, Jensen PE. Early motion of the ankle after operative treatment of a rupture of the Achilles tendon: a prospective, randomized clinical and radiographic study. JBJS. 1999;81(7):983-90.
8. Don R, Ranavolo A, Cacchio A, et al. Relationship between recovery of calf-muscle biomechanical properties and gait pattern following surgery for Achilles tendon rupture. Clinical biomechanics. 2007;22(2):211-20.

9. Wang HK, Chiang H, Chen WS, Shih TT, Huang YC, Jiang CC. Early neuromechanical outcomes of the triceps surae muscle-tendon after an Achilles' tendon repair. Archives of physical medicine and rehabilitation. 2013;94(8):1590-8.
10. Naim F, Simşek A, Sipahioğlu S, Esen E, Cakmak G. Evaluation of the surgical results of Achilles tendon ruptures by gait analysis and isokinetic muscle strength measurements. Acta orthopaedica et traumatologica turcica. 2005;39(1):1-6.
11. Willits K, Amendola A, Bryant D, Mohtadi NG, Giffin JR, Fowler P, Kean CO, Kirkley A. Operative versus nonoperative treatment of acute Achilles tendon ruptures: a multicenter randomized trial using accelerated functional rehabilitation. JBJS. 2010;92(17):2767
12. Horstmann T, Lukas C, Merk J, Brauner T, Mündermann A. Deficits 10-years after Achilles tendon repair. International journal of sports medicine. 2012;33(06):474-9.
13. Heikkinen J, Lantto I, Piilonen J, et al. Tendon length, calf muscle atrophy, and strength deficit after acute Achilles tendon rupture: long-term follow-up of patients in a previous study. JBJS. 2017;99(18):1509-15.
14. De la Fuente CI, Lillo RP, Ramirez-Campillo R, et al. Medial gastrocnemius myotendinous junction displacement and plantar-flexion strength in patients treated with immediate rehabilitation after Achilles tendon repair. Journal of athletic training. 2016;51(12):1013-21.
15. Valeo M, Gurzì M, Alviti F, Di Giorgio L, Di Martino L. Achilles Tendons Total Rupture, Open Surgical Treatment with PRF Augmentation: Clinical, Morphological and Functional Evaluation. Clin Res Foot Ankle. 2017;5(236):2.
16. Peng WC, Chang YP, Chao YH, Fu SN, Rolf C, Shih TT, Su SC, Wang HK. Morphomechanical alterations in the medial gastrocnemius muscle in patients with a repaired Achilles tendon: Associations with outcome measures. Clinical Biomechanics. 2017;43:50-7.
17. Manegold S, Tsitsilonis S, Gehlen T, Kopf S, Duda GN, and Agres AN. Alterations in structure of the muscle-tendon unit and gait pattern after percutaneous repair of Achilles tendon rupture with the Dresden instrument. Foot and Ankle Surgery. 2018;25:529-533.
18. Baxter JR, Hullfish TJ, Chao W. Functional deficits may be explained by plantarflexor remodeling following Achilles tendon rupture repair: Preliminary findings. Journal of biomechanics. 2018;79:238-42.
19. Svensson RB, Couppé C, Agergaard AS, Ohrhammar Josefsen C, Jensen MH, Barfod KW, Nybing JD, Hansen P, Krogsgaard M, Magnusson SP. Persistent functional loss following ruptured Achilles tendon is associated with reduced gastrocnemius muscle fascicle length,

elongated gastrocnemius and soleus tendon, and reduced muscle cross-sectional area. Translational Sports Medicine. 2019;00:1-9.

20. Nilsson-Helander K, Thomeé R, Grävare-Silbernagel K, et al. The Achilles tendon total rupture score (ATRS) development and validation. The American journal of sports medicine. 2007;35(3):421-6.
21. Ebben WP, Petushek EJ. Using the reactive strength index modified to evaluate plyometric performance. The Journal of Strength & Conditioning Research. 2010;24(8):1983-7.
22. Hess GW. Achilles tendon rupture: a review of etiology, population, anatomy, risk factors, and injury prevention. Foot & ankle specialist. 2010;3(1):29-32.
23. Barfod KW, Sveen TM, Ganestam A, Ebskov LB, Troelsen A. Severe functional debilitations after complications associated with acute Achilles tendon rupture with 9 years of follow-up. The Journal of Foot and Ankle Surgery. 2017;56(3):440-4.
24. Riemann BL, Limbaugh GK, Eitner JD, LeFavi RG. Medial and lateral gastrocnemius activation differences during heel-raise exercise with three different foot positions. The Journal of Strength & Conditioning Research. 2011;25(3):634-9.
25. Vieira TM, Minetto MA, Hodson-Tole EF, Botter A. How much does the human medial gastrocnemius muscle contribute to ankle torques outside the sagittal plane?. Human movement science. 2013;32(4):753-67.
26. Franchi MV, Longo S, Mallinson J, Quinlan JI, Taylor T, Greenhaff PL, Narici MV. Muscle thickness correlates to muscle cross-sectional area in the assessment of strength training-induced hypertrophy. Scandinavian journal of medicine & science in sports. 2018 ;28(3):846-53.
27. Freedman BR, Gordon JA, Soslowsky LJ. The Achilles tendon: fundamental properties and mechanisms governing healing. Muscles, ligaments and tendons journal. 2014;4(2):245.
28. Eliasson P, Agergaard AS, Couppe C, et al. The ruptured Achilles tendon elongates for 6 months after surgical repair regardless of early or late weightbearing in combination with ankle mobilization: a randomized clinical trial. The American journal of sports medicine. 2018;46(10):2492-502.
29. Burkholder T, Lieber RL. Sarcomere number adaptation after retinaculum transection in adult mice. Journal of Experimental Biology. 1998;201(3):309-16.
30. Koh TJ, Herzog W. Increasing the moment arm of the tibialis anterior induces structural and functional adaptation: implications for tendon transfer. Journal of biomechanics. 1998;31(7):593-9.

31. Kearns CF, Abe T, Brechue WF. Muscle enlargement in sumo wrestlers includes increased muscle fascicle length. European Journal of Applied Physiology. 2000;83(4-5):289-96.
32. Kangas J, Pajala A, Ohtonen P, Leppilahti J. Achilles tendon elongation after rupture repair: a randomized comparison of 2 postoperative regimens. Am J Sports Med. 2007;35(1):59-64.
33. Khan KM, Forster BB, Robinson J, et al. Are ultrasound and magnetic resonance imaging of value in assessment of Achilles tendon disorders? A two year prospective study. British Journal Sports Medicine. 2003;37(2):149–153.
34. Trezise J, Collier N, Blazevich AJ. Anatomical and neuromuscular variables strongly predict maximum knee extension torque in healthy men. European journal of applied physiology. 2016;116(6):1159-77.
35. Bäcker HC, Yenchak AJ, Trofa DP, Vosseller JT. Strength Measurement After Achilles Tendon Repair. Foot & ankle specialist. 2018:1938640018819779.
36. Mullaney MJ, McHugh MP, Tyler TF, Nicholas SJ, Lee SJ. Weakness in end-range plantar flexion after Achilles tendon repair. The American journal of sports medicine. 2006;34(7):1120-5.
37. Fukunaga T, Kubo K, Kawakami Y, Fukashiro S, Kanehisa H, Maganaris CN. In vivo behaviour of human muscle tendon during walking. Proceedings of the Royal Society of London. Series B: Biological Sciences. 2001;268(1464):229-33.
38. Kawakami Y, Ichinose Y, Fukunaga T. Architectural and functional features of human triceps surae muscles during contraction. Journal of applied physiology. 1998;85(2):398-404.
39. Lichtwark GA, Wilson AM. Optimal muscle fascicle length and tendon stiffness for maximising gastrocnemius efficiency during human walking and running. Journal of theoretical biology. 2008;252(4):662-73.
40. Hubley CL, Wells RP. A work-energy approach to determine individual joint contributions to vertical jump performance. European Journal of Applied Physiology and Occupational Physiology. 1983;50(2):247-54.
41. So V, Pollard H. Management of Achilles tendon disorders: a case review. Australasian Chiropractic & Osteopathy. 1997;6(2):58.
42. Franchi MV, Atherton PJ, Maganaris CN, Narici MV. Fascicle length does increase in response to longitudinal resistance training and in a contraction-mode specific manner. Springerplus. 2016;5(1):94.

43. Eliasson P, Couppé C, Lonsdale M, Svensson RB, Neergaard C, Kjær M, Friberg L, Magnusson SP. Ruptured human Achilles tendon has elevated metabolic activity up to 1 year after repair. European journal of nuclear medicine and molecular imaging. 2016;43(10):1868-77.
44. Willy RW, Brorsson A, Powell HC, Willson JD, Tranberg R, Grävare Silbernagel K. Elevated knee joint kinetics and reduced ankle kinetics are present during jogging and hopping after Achilles tendon ruptures. The American journal of sports medicine. 2017;45(5):1124-33.
45. Jandacka D, Plesek J, Skypala J, Uchytil J, Silvernail JF, Hamill J. Knee Joint Kinematics and Kinetics During Walking and Running After Surgical Achilles Tendon Repair. Orthopaedic journal of sports medicine. 2018;6(6).
46. Schepsis AA, Jones H, Haas AL. Achilles tendon disorders in athletes. The American journal of sports medicine. 2002;30(2):287-305.
47. Olsson N, Nilsson-Helander K, Karlsson J, Eriksson BI, Thomée R, Faxén E, Silbernagel KG. Major functional deficits persist 2 years after acute Achilles tendon rupture. Knee Surgery, Sports Traumatology, Arthroscopy. 2011;19(8):1385-93.
48. Westin O, Svensson M, Helander KN, Samuelsson K, Silbernagel KG, Olsson N, Karlsson J, Olofsson EH. Cost-effectiveness analysis of surgical versus non-surgical management of acute Achilles tendon ruptures. Knee Surgery, Sports Traumatology, Arthroscopy. 2018;26(10):3074-82.

Figures

Figure 1. Example ultrasound image of the Medial Gastrocnemius in a non-injured (left) and repaired (right).

Figure 2. Typical example of Achilles tendon thickness measurements in a non-injured (top) and repaired (bottom).

Figure 3. Achilles tendon thickness and cross-sectional area at the different measured sites.

Tables

Table 1. Descriptive statistics (mean [standard deviation]) for the 10 items in the ATRS along with the total score. *r* values depict the relationship between each ATRS item and months since surgery. * significant at the $p < 0.05$ level ** significant at the $p < 0.01$ level

Item Number	Nature of Limitation	Score	*r*
1	Strength	8.67 (2.06)	0.76**
2	Tiredness	9.17 (0.83)	0.87**
3	Stiffness	7.50 (1.98)	0.74**
4	Pain	9.42 (1.00)	0.58*
5	Daily Activities	9.42 (1.51)	0.58*
6	Uneven walking	9.33 (1.61)	0.61*
7	Walking up hill/stairs	9.42 (1.38)	0.62*
8	Running	8.42 (2.84)	0.70*
9	Jumping	8.17 (2.92)	0.76**
10	Heavy Physical Work	8.42 (2.57)	0.59*
	Total	87.92 (16.22)	0.79

Table 2. Peak torque (N m) for the ankle plantar flexor muscles for the injured and non-injured limb. *r* values depict the relationship between strength outcomes and months since surgery. * significant at the $p < 0.05$ level ** significant at the $p < 0.01$ level

Isokinetic Outcomes	Injured side	Non-injured side	% diff	*r*
Peak Flexor Torque (knee straight)				
30 ^{o}/s	82.67 (33.89)	91.67 (27.70)	-11.9 (16.0)	-0.07
60 ^{o}/s	68.43 (26.22)	75.78 (21.51)	-11.7 (24.6)	0.06
Peak Flexor Torque (knee flexed)				
30 ^{o}/s	86.80 (33.84)	90.46 (35.87)	-1.3 (34.4)	0.05
60 ^{o}/s	75.27 (30.14)	79.53 (26.11)	-7.5 (26.9)	0.19

Table 3. Performance variables for the countermovement jump and drop jump tests for the injured and non-injured limbs. *r* values depict the relationship between jump outcomes and months since surgery. * significant at the $p < 0.05$ level ** significant at the $p < 0.01$ level

Jump Outcomes	**Injured side**	**Non-injured side**	**% diff**	***r***
Countermovement Jump				
Jump Height (cm)	0.10 (0.03)*	0.12 (0.04)	-12.6 (15.7)	-0.12
RSImod	0.12 (0.04)*	0.15 (0.05)	-14.2 (17.6)	-0.14
Drop Jump				
Peak Landing Force (N)	1597 (474)	1874.05 (599)	-11.6 (19.7)	0.14
Contact Time (s)	0.34 (0.08)*	0.36 (0.09)	-5.5 (5.7)	0.05

Table 4. Gait variables collected during running and walking for the injured and non-injured limbs. *r* values depict the relationship between gait outcomes and months since surgery. * significant at the $p < 0.05$ level ** significant at the $p < 0.01$ level

Gait Outcomes	**Injured side**	**Non-injured side**	**% diff**	***r***
Weight Acceptance Rate				
Walking (N/s)	6233.40 (2260.94)	6991.95 (2558.59)	-9.1 (18.1)	0.23
Running (N/s)	17956.88 (6971.58)	17501.48 (6256.44)	2.9 (18.5)	-0.09
Push Off Rate				
Walking (N/s)	5874.15 (1265.73)	5809.76 (1252.62)	1.4 (6.2)	0.46
Running (N/s)	13155.06 (4791.33)	12283.11 (4898.21)	8.9 (16.4)	-0.23
Step Length				
Walking (cm)	70.26 (19.17)	70.30 (19.17)	-0.3 (6.3)	0.18
Running (cm)	96.52 (32.50)	96.61 (31.91)	-0.2 (5.4)	-0.23
Contact Time				
Running (s)	0.306 (0.054)	0.312 (0.055)	-1.7 (3.5)	0.23

Table 5. Morphological characteristics of the lower-limb muscles of the injured and non-injured limbs. *r* values depict the relationship between morphological outcomes and months since surgery. * significant at the $p < 0.05$ level ** significant at the $p < 0.01$ level

Morphological Outcomes	**Injured side**	**Non-injured side**	**% diff**	***r***
Muscle Thickness				
Medial Gastrocnemius (mm)	17.18 (2.35)*	19.03 (2.99)	-9.12 (9.39)	0.23
Lateral Gastrocnemius (mm)	13.51 (3.04)	14.39 (2.53)	-6.32 (14.16)	0.14
Soleus (mm)	11.62 (2.78)*	15.00 (3.92)	-20.14 (19.00)	0.16
Fibre Pennation				
Medial Gastrocnemius (^{o})	25.31 (4.72)*	21.82 (3.84)	17.99 (22.14)	0.26
Lateral Gastrocnemius (^{o})	15.99 (3.14)	14.79 (2.43)	9.99 (23.75)	-0.30
Soleus (^{o})	24.40 (3.74)	24.44 (6.16)	4.46 (25.03)	0.34
Fibre Length				
Medial Gastrocnemius (mm)	40.97 (6.37)**	52.32 (9.49)	-19.56 (17.99)	-0.11
Lateral Gastrocnemius (mm)	49.76 (11.46)*	57.32 (12.06)	-12.11 (18.89)	0.51
Soleus (mm)	29.34 (5.71)**	37.13 (7.03)	-19.57 (15.33)	-0.10

Table 6. Ankle and hip range of motion (internal, external and combined) for the injured and non-injured side. *r* values depict the relationship between range of motion outcomes and months since surgery. * significant at the $p < 0.05$ level ** significant at the $p < 0.01$ level

ROM Outcomes	**Injured side**	**Non-injured side**	**% diff**	***r***
Ankle ROM				
Knee-to-Wall (cm)	7.96 (4.41)	8.83 (4.90)	-6.4 (39.6)	0.31
Knee-to-Wall Mod (cm)	33.41 (5.58)	34.54 (5.42)	-3.5 (10.7)	0.35
Hip External ROM				
Prone	44.11 (15.63)	44.17 (17.97)	3.7 (18.2)	0.29
Supine	37.47 (14.60)	36.64 (17.78)	8.6 (28.2)	-0.33
Seated	41.81 (14.72)	42.89 (18.30)	1.4 (19.0)	0.02
Hip Internal ROM				
Prone	46.19 (20.38)	47.72 (16.10)	-4.1 (20.6)	-0.07
Supine	42.83 (20.55)	45.44 (15.44)	-4.0 (35.0)	0.30
Seated	44.81 (17.71)	49.64 (15.34)	-10.1 (15.1)	-0.07
Hip Total ROM				
Prone	90.31 (34.56)	91.89 (32.12)	-1.6 (13.5)	0.16
Supine	80.31 (33.91)	82.08 (30.78)	-2.0 (18.1)	0.07
Seated	86.61 (30.44)*	92.53 (30.66)	-6.3 (8.2)	-0.07