UNIVERSITY OF
GLOUCESTERSHIRE

This is a peer-reviewed, post-print (final draft post-refereeing) version of the following published document, This is an Accepted Manuscript of an article published online by Taylor & Francis in The Physician and Sportsmedicine on 7th November 2019, available online: https://doi.org/10.1080/00913847.2019.1685364 and is licensed under All Rights Reserved license:

Nicholson, Gareth, Walker, Josh, Bissas, Athanassios ORCID logoORCID: https://orcid.org/0000-0002-7858-9623 and Harris, Nick (2020) Morphological and functional outcomes of operatively treated Achilles tendon ruptures. Physician and Sportsmedicine, 48 (3). pp. 290-297. doi:10.1080/00913847.2019.1685364

Official URL: https://doi.org/10.1080/00913847.2019.1685364
DOI: http://dx.doi.org/10.1080/00913847.2019.1685364
EPrint URI: https://eprints.glos.ac.uk/id/eprint/7906

Disclaimer

The University of Gloucestershire has obtained warranties from all depositors as to their title in the material deposited and as to their right to deposit such material.

The University of Gloucestershire makes no representation or warranties of commercial utility, title, or fitness for a particular purpose or any other warranty, express or implied in respect of any material deposited.

The University of Gloucestershire makes no representation that the use of the materials will not infringe any patent, copyright, trademark or other property or proprietary rights.

The University of Gloucestershire accepts no liability for any infringement of intellectual property rights in any material deposited but will remove such material from public view pending investigation in the event of an allegation of any such infringement.

PLEASE SCROLL DOWN FOR TEXT.

Morphological and functional outcomes of operatively treated Achilles tendon ruptures

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

ABSTRACT

Background: Achilles' tendon (AT) rupture leads to functional impairments and these may be underpinned by morphological changes in the muscle-tendon unit.

Hypothesis: Functional performance of the injured limb will be impaired regardless of time since surgery and these impairments occur alongside changes in muscle-tendon morphology.

Study Design: Descriptive laboratory study.

Methods: Following operative treatment of AT rupture (mean, 4.4 years postoperative; range, 6-36 months) and short term immobilisation, 12 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 from isokinetic (30 °/s, 60 °/s) plantar flexion strength, jumping performance and walking-running were collected. Differences were expressed relative to the non-injured limb.

Results: The repaired limb showed a shorter muscle fascicle length (12.1-19.6% diff), increased fascicle pennation (18.0% diff) and reduced muscle thickness (9.1-20.1% diff) in the Gastrocnemius and/or Soleus along with greater tendon cross-sectional area (46.7% diff). Functionally, impairments were found for countermovement jump height (-12.6% diff) and drop jump contact times (+5.5% diff). Also, the repaired limb showed reduced hip internal-external ROM (6.3% diff) but no differences existed between limbs for plantar flexion ROM and strength or gait characteristics. Good ATRS outcomes were reported (mean 87.9) 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 AT 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.

Clinical Relevance: Morphological changes will have functional implications in high-velocity movements and these may persist many years post-surgery.

Key Terms: isokinetic, ATRS, muscle morphology, gait

What is known about the subject: AT rupture is known to result in long-term reductions in ROM, strength and joint function in the injured leg.

What this study adds to existing knowledge: Despite morphologically remodelling of the triceps surae complex following AT rupture, these compensatory mechanisms may be insufficient to prevent long-term impairments in high-velocity functional performance.

INTRODUCTION

The Achilles Tendon (AT) is the strongest tendon in the human body with a crucial role in transmitting mechanical energy generated by the triceps surae complex. Incidence of AT rupture has been estimated at between 6-21.5 per 100,000 (Maffulli et al. 1999; Lantto et al. 2015) with notable increases being reported in recent decades (Aujla et al. 2018). Although non-operative treatment of AT 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 (Cetti et al. 1993; Calder et al. 2005; Wilkins & Bisson, 2012).

Impaired functional outcomes shown for individuals with a history of AT rupture include reduced ankle proprioception, decreased plantar flexor strength and asymmetric performance in activities requiring propulsion (Mortensen et al. 1999; Don et al 2007; Wang et al. 2013). Despite the prevalence of AT ruptures, research into post-operative deficits has generally focussed on relatively short follow-up periods of 6-36 months (Naim et al., 2005; Willits et al., 2010). Recent research has provided evidence of long-term deficits (>10 years) post-surgery (Horstmann et al., 2012; Barfod et al., 2017) which include reduced ROM, strength and joint function 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 (De la Fuente et al., 2016; Valeo et al. 2017).

Apart from functional deficits, AT rupture results in alterations of the neuromechanical properties of the muscle-tendon unit including tendon elongation, triceps surae (TS) atrophy and compensatory changes in muscle activation (Horstmann et al. 2012; Heikkinen et al. 2017; Peng, 2017). Manegold and co-workers (2018) showed that AT elongation and TS 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 (Peng et al. 2017; Baxter et al. 2018) have suggested medial gastrocnemius remodelling following AT rupture as a compensatory mechanism to restore resting tendon tension. However, such changes (e.g. larger pennation, reduced volume are known to be related to impairments in activities requiring force transmission and muscle excursion (e.g. heel raise; Peng et al. 2017). 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 investigated post-surgery periods longer than 2.5 years.

Therefore, despite the wealth of research investigating the outcomes of operatively treated AT 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 AT ruptures.

METHODS

Twelve patients participated in the current study (ten men, two women; 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 AT rupture and had undergone operative treatment, the time since surgery of the cohort was 4.4 ± 2.6 years. Following a medical screening assessment, participants provided informed consent and completed the Achilles Tendon Rupture Score (ATRS) to assess patient reported outcomes (Nilsson-Helander et al., 2007). The study received approval from the university research ethical committee and was in accordance with the Declaration of Helsinki.

Operative treatment

All participants underwent identical operative treatment 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.

Testing protocol

Jumping performance

Participants performed unilateral countermovement jumps (CMJ) and bilateral 30 cm drop jumps (DJ) 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 (DJ only) were calculated. Jump height was calculated from flight time ($d = v_i t * ½ at^2$) whilst and RSI by dividing the jump height in the DJ by the contact time; for the CMJ, contact time was replaced by time to take-off in order to calculate RSImod (Ebben and Petushek, 2010).

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 $m \cdot s^{-1}$ for several minutes before 30 s of data collection whilst - the same protocol was replicated for running at 2.22 $m \cdot s^{-1}$. 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.

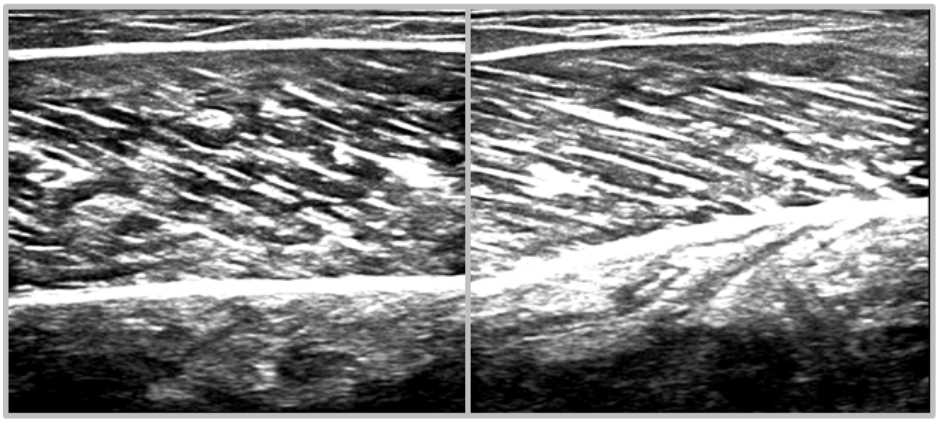
Range of motion assessments

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 (MTC)

Static ultrasound measurements (figure 1) of the Triceps surae MTC 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 (GM), *m.* Gastrocnemius lateralis (GL) and *m.* Soleus (Sol) 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 AT for the measurement of the cross-sectional area (CSA) were taken 40 mm proximal to the calcaneal insertion, while longitudinal images of the AT 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

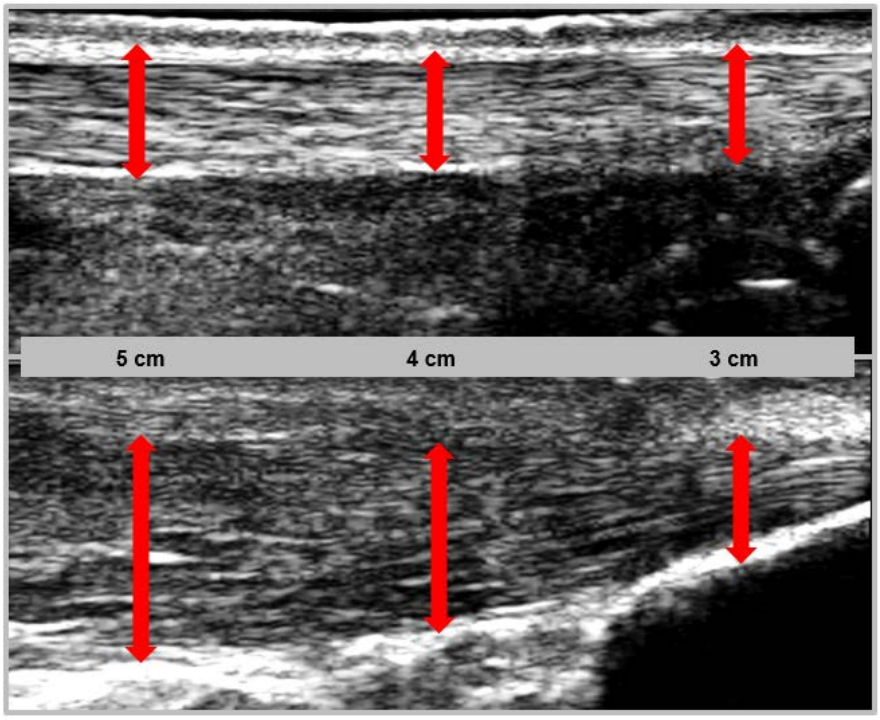


were chosen since the region 20-60 mm proximal to the calcaneal insertion is the most commonly ruptured region (Hess, 2010).

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

Ultrasound images were analysed using ImageJ (Java, National institution of health). AT thickness was measured from the superficial to deep edges of the tendon with muscle thickness measured from the deep to superficial aponeuroses. 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. Fascicle length was established through trigonometry using the pennation angle and muscle thickness.

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



Strength assessment

Participants underwent strength assessments of the TS 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 concentric torque was measured for the injured and non-injured leg at two angular velocities (30 and 60°·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

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.

ATRS Outcomes	Score	*r*
1	8.67 (2.06)	0.76
2	9.17 (0.83)	0.87
3	7.50 (1.98)	0.74
4	9.42 (1.00)	0.58
5	9.42 (1.51)	0.58
6	9.33 (1.61)	0.61
7	9.42 (1.38)	0.62
8	8.42 (2.84)	0.70
9	8.17 (2.92)	0.76
10	8.42 (2.57)	0.59
Total	87.92 (16.22)	0.79

For the responses to each ATRS item, there was a lower mean score for item 3 with the highest mean scores being reported for items 4, 5 and 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.

Strength Outcomes

Despite a mean difference in PT (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 PT limb symmetry values showed an improvement when the test was performed with the knee flexed.

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.

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

Functional Outcomes

Regarding functional performance, CMJ 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 DJ 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%).

Table 3 Performance variables for the CMJ and DJ tests for the injured and non-injured limbs. *r* values depict the relationship between jump outcomes and months since surgery. *significant difference from non-injured side.

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

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

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.

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

Morphological Outcomes

The injured limb demonstrated morphological differences in the AT (figures 2 and 3) and lower-limb muscles (table 6) compared to the non-injured limb. Specifically, the AT 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 CSA (46.7 ± 34.5, LSI: 146.9 ± 34.5%, p=0.001) than on the non-injured side.

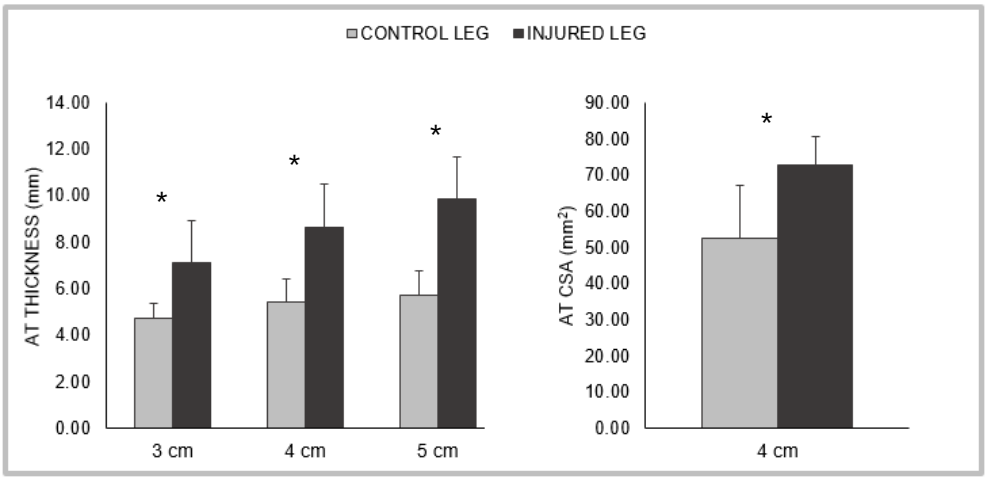


Figure 3. Achilles tendon thickness and CSA at the different measured sites. * significant difference between injured and control leg.

The GM (LSI: 90.9 ± 9.4%, p=0.01) and SOL (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 GM (LSI: 80.4 ± 18.0%, p=0.00) and GL (LSI: 87.9 ± 18.9%, p=0.03) and SOL (LSI: 80.4 ± 15.3%, p=0.00). Only the fascicles of the GM showed a significantly greater pennation angle (LSI: 118.0 ± 22.1%, p=0.02) on the injured side.

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.

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

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 5). 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 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.

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

DISCUSSION

This study examined the morphological and functional outcomes of operatively treated AT 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 AT structure following surgical repair, the muscle fibres in the TS 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 (Horstmann et al., 2012; Heikkinen et al. 2017) 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 (Riemann et al. 2011; Vieria et al. 2013) 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 (Franchi et al., 2018). Whilst chronic atrophy of TS complex theoretically indicates a reduced capacity for force generation, the significant increases in AT thickness and CSA more likely reflect the healing process rather than improved mechanical properties of the tendon (Freedman et al. 2014). Although increased AT thickness has been consistently observed post rupture (Eliasson, 2018), 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 (Burkholder & Lieber, 1998; Koh & Herzog, 1998) and whilst changes in plantar flexor architecture have been recently observed in humans following AT rupture (Peng, 2017; Baxter et al. 2018), 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 (Kearns et al. 2000) and tendon tension whilst decreases in fascicle length may have served to restore resting tension caused by tendon elongation (Peng, 2017). 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 AT length and fibre length asymmetries are supportive of such outcomes (Kangas et al., 2007; Eliasson, 2018).

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 (Khan et al. 2003), 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 (Trezise et al., 2016). 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 LSI values of below 85% years after surgery. Indeed, previous investigations (Horstmann et al. 2012; Bäcker et al., 2019) have proved controversial when attempting to link morphological and strength changes following AT 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 AT rupture (Mullaney et al. 2006).

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 every day activities. Baxter et al. (2018) 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 (Fukunaga et al., 2001). 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 (Horstmann et al., 2012).

Although recent research (Peng et al., 2017; Baxter et al., 2018) 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 (Kawakami et al. 1998) and reductions in peak power at higher angular velocities due to shorter length of fibres (Lichwark & Wilson, 2008). The significant reductions in jump height, increased take-off times and reduced RSImod scores in the unilateral CMJ are indicative of impaired concentric propulsion capabilities of the plantar flexors (Hubley & Wells, 1983). Moreover, it would appear that patients placed a greater reliance on the non-injured limb during the bilateral DJ 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 (So & Pollard, 1997; Franchi et al. 2016).

The reduced hip external and internal rotation range of motion of the injured limb was a novel finding of the present investigation. That being said, previous investigations into running mechanics following AT ruptures have observed increased knee joint loads (Willy et al., 2017) and internal knee abduction moments (Jandacka et al. 2018) in the injured limb. It is not possible to identify if impairments in the range of motion in the current study are the result of post-operation movement management or they existed prior to the AT rupture.

However, as previous research has linked femoral mechanics with lower limb compensatory mechanisms and loading of AT, there may be a need to consider hip function in clinical assessments aimed at preventing AT rupture or re-rupture (So & Pollard, 1997; Schepsis et al. 2002; Hess, 2010).

Long lasting differences between the injured and non-injured limbs have been observed previously (Horstmann et al., 2012; Barfod et al., 2017) in individuals who have undergone surgical repair of an AT 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 (Olsson et al. 2011). 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 AT 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.

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 confirmed our hypothesis that the TS complex undergoes architectural and structural changes following surgical repair of AT 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.

REFERENCES

1. Aujla R, Sapare S, Bhatia M. Acute Achilles tendon rupture treatment: Where are we now? Journal of arthroscopy and joint surgery. 2018 Sep 1;5(3):139-44.
2. Bäcker HC, Yenchak AJ, Trofa DP, Vosseller JT. Strength Measurement After Achilles Tendon Repair. Foot & ankle specialist. 2018 Dec 17:1938640018819779.
3. 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 May 1;56(3):440-4.
4. 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 Oct 5;79:238-42.
5. Burkholder T, Lieber RL. Sarcomere number adaptation after retinaculum transection in adult mice. Journal of Experimental Biology. 1998 Feb 1;201(3):309-16.
6. Calder JD, Saxby TS. Early, active rehabilitation following mini-open repair of Achilles tendon rupture: a prospective study. British journal of sports medicine. 2005 Nov 1;39(11):857-9.
7. 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 Nov;21(6):791-9.
8. 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 Dec;51(12):1013-21.
9. 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 Feb 1;22(2):211-20.
10. Ebben WP, Petushek EJ. Using the reactive strength index modified to evaluate plyometric performance. The Journal of Strength & Conditioning Research. 2010 Aug 1;24(8):1983-7.

11. 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 Aug;46(10):2492-502.
12. 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 Dec;5(1):94.
13. 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 Mar;28(3):846-53.
14. Freedman BR, Gordon JA, Soslowsky LJ. The Achilles tendon: fundamental properties and mechanisms governing healing. Muscles, ligaments and tendons journal. 2014 Apr;4(2):245.
15. 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 Feb 7;268(1464):229-33.
16. 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 Sep 20;99(18):1509-15.
17. Hess GW. Achilles tendon rupture: a review of etiology, population, anatomy, risk factors, and injury prevention. Foot & ankle specialist. 2010 Feb;3(1):29-32.
18. Horstmann T, Lukas C, Merk J, Brauner T, Mündermann A. Deficits 10-years after Achilles tendon repair. International journal of sports medicine. 2012 Jun;33(06):474-9.
19. 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 Jan 1;50(2):247-54.
20. 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 Jun 20;6(6).
21. 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.
22. Kawakami Y, Ichinose Y, Fukunaga T. Architectural and functional features of human triceps surae muscles during contraction. Journal of applied physiology. 1998 Aug 1;85(2):398-404.
23. Kearns CF, Abe T, Brechue WF. Muscle enlargement in sumo wrestlers includes increased muscle fascicle length. European Journal of Applied Physiology. 2000 Nov 1;83(4-5):289-96.

24. 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.
25. 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 Jul 1;31(7):593-9.
26. 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.
27. 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 Jun 21;252(4):662-73.
28. Maffulli N. Rupture of the Achilles tendon. JBJS. 1999 Jul 1;81(7):1019-36.
29. 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
30. 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 Jul 1;81(7):983-90.
31. 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 Jul;34(7):1120-5.
32. 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.
33. 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 Mar;35(3):421-6.
34. 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 Aug 1;19(8):1385-93.
35. 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 Mar 1;43:50-7.
36. 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 Mar 1;25(3):634-9.

37. Schepsis AA, Jones H, Haas AL. Achilles tendon disorders in athletes. The American journal of sports medicine. 2002 Mar;30(2):287-305.
38. So V, Pollard H. Management of Achilles tendon disorders: a case review. Australasian Chiropractic & Osteopathy. 1997 Jul;6(2):58.
39. 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 Jun 1;116(6):1159-77.
40. 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.
41. 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 Aug 1;32(4):753-67.
42. 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 Aug 1;94(8):1590-8.
43. 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 Sep;40(9):2154-60.
44. 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 Dec 1;92(17):2767
45. 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 Apr;45(5):1124-33.