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Neuromechanical adaptations in the gastrocnemius muscle after Achilles tendon rupture during walking

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ABSTRACT

Although some Achilles tendon rupture (ATR) patients regain function in low-force levels activities, it is not yet well known how neuromuscular and structural alterations after ATR manifest during everyday-locomotion. This study assessed medial gastrocnemius (MG) fascicle shortening during walking 1-year after ATR. Additionally, we explored neuromuscular alterations in lateral gastrocnemius (LG), soleus and flexor hallucis longus (FHL) muscles.

We observed 3.1 pp (95 %CI 0.8–5.4 pp) higher average and 14.5 pp (95 %CI 0.5–28.5 pp) higher peak LG surface electromyography amplitude in the injured compared to the un-injured during walking, but no differences were observed in soleus or FHL. The injured limb fascicles were 12.9 mm shorter while standing compared to the un-injured limb. In absolute terms, MG shortening in the injured limb was 2.8 mm (95 %CI 0.96–4.6 mm) smaller compared to the un-injured limb. However, when normalized to standing fascicle length, the amount of shortening was not different between the limbs.

Our results showed that 1-year after ATR, MG muscle had remodelled, which manifested as shorter fascicle length during standing. During walking, injured and un-injured MG fascicles showed similar shortening relative to the standing fascicle length, suggesting that MG could function effectively at the new mechanical settings during everyday locomotion.

1. Introduction

The Achilles tendon (AT) remains one of the most frequently injured tendons in the human body with increasing incidence in the recent decades (Holm et al., 2015; Lantto et al., 2015). AT rupture (ATR) requires a long recovery period and leads to permanent morphological changes, such as tendon elongation and shorter fascicles in the medial gastrocnemius (MG) muscle (Baxter et al., 2018; Peng et al., 2017; Baxter et al., 2018; Peng et al., 2017; Silbernagel et al., 2012; Svensson et al., 2019; van Dijk et al., 2023; Svensson et al., 2019; van Dijk et al., 2023). After ATR, patients often experience long-term functional deficits characterized by limited heel raise capability and weakness at the end range of plantar flexion (Mullaney et al., 2006; Mullaney et al., 2006; Silbernagel et al., 2012). Although some patients regain capacity to perform in slow motion activities that require low force levels, functional impairments are observed in high demand tasks such as jogging or hopping (Jandacka et al., 2017; Willy et al., 2017). It is not yet well known how neuromuscular and structural adaptations in the triceps surae (TS) muscle–tendon unit after ATR manifest during everyday locomotion.

Muscle atrophy and shorter fascicles are observed in the MG after ATR due to the increased length of the tendon and prolonged ankle immobilization in a shortened position (Baxter et al., 2018; Khair et al., 2022). Tendon elongation shifts the operating length leftwards on the ascending limb of the force–length relationship (Hoeffner et al., 2023; Stäudle et al., 2020), limiting force production capacity and reducing the excursion of the MG muscle. The muscle adjusts to the extra slack of the tendon by reducing sarcomeres in series to maintain optimum actin-myosin overlap comparable to pre-injury settings (Hoeffner et al., 2023). This mechanism might ameliorate the loss of force production capability, facilitating force generation within a smaller operating range (Stäudle et al., 2020). Stäudle et al., (2022) reported that MG contractile behaviour was similar between the ruptured and contralateral leg during walking at a speed of 1.5 $m \cdot s^{-1}$ 2 years after ATR. Due to relatively fast adaptation of muscles, it is reasonable to assume that already 1-year

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after ATR, MG muscle has regained sufficient plantar flexion force required for locomotion activities such as walking.

Regardless of the treatment strategy, studies have shown increased surface electromyography (EMG) normalized amplitude of the TS muscles during plantar flexion (Khair et al., 2023; Wenning et al., 2021) and higher mean frequency (McHugh et al., 2019) after rupture. Wenning et al., (2021) found higher normalised EMG activity of the gastrocnemii muscles during the push-off phase of gait but no differences were observed in the soleus muscle (Wenning et al., 2021). Similarly, Suydam et al., (2015) observed higher normalized EMG during gait at 6 months in the lateral gastrocnemius (LG), and MG at 12 months after rupture (Suydam et al., 2015). Neuromuscular control is further altered so that deep flexors such as flexor hallucis longus (FHL) are recruited to potentially alleviate tensile forces transmitted through the AT (Masood et al., 2016). This mechanism is thought to start early in the recovery stage and persist due to the shortened position of the TS (Finni et al., 2006; Heikkinen et al., 2017; Khair et al., 2023). Few studies have investigated MG dynamic behaviour after Achilles tendon rupture (Stäudle et al., 2022; van Dijk et al., 2023), while no studies have explored the neuromuscular alteration and functional contribution of FHL in patients with ATR during walking.

Due to the high prevalence of ATR and long-term functional impairment associated with the injury; it is imperative to determine how TS muscle–tendon unit remodelling manifests in everyday locomotion. Therefore, the objective of this study was to investigate the fascicle shortening of the MG during walking 1-year after non-surgically treated ATR. Additionally, we examined the EMG activity of LG, soleus and FHL. It was hypothesized that the injured limb MG would have a comparable normalised fascicle shortening to the un-injured limb during walking. Moreover, we expected the relative EMG activity of LG, soleus and FHL to be higher in the injured limb compared to the contralateral limb.

2. Materials and methods

Twenty-four participants (21 males, 3 females) with unilateral ATR were recruited for this cross-sectional study within a clinical cohort study "Non-operative treatment of Achilles tendon Rupture in Central Finland: a prospective cohort study – NoARC, trial registration: NCT03704532". Rupture was diagnosed in accordance with the American Academy of Orthopaedic Surgeons (AAOS) based on a minimum of 2 of the following 4 criteria: a positive Thompson test, decreased plantarflexion strength, presence of a palpable gap, and increased passive ankle dorsiflexion with gentle manipulation (Surgeons, 2009). All participants (means ± SD age: 40.7 ± 10.6 years, height: 177.2 ± 8.3 cm, mass: 85.2 ± 15.2 kg) were treated non-surgically with early mobilization (Reito et al., 2017). Data from one year are presented, with participants tested (mean ± SD) 13 ± 1 months after rupture.

2.1. Lab protocol

Upon arrival at the laboratory, participants were prepared for surface electromyography (EMG) measurements. The skin was shaved and abraded with alcohol pads to reduce impedance. Disposable dual surface silver-silver electrode Ambu BlueSensor N electrodes (Ambu A/S, Ballerup, Denmark) were placed on the TS muscles with an interelectrode distance of 22 mm according to SENIAM recommendations (Stegeman and Hermens, 2007). For FHL, electrodes were placed between the soleus insertion and the FHL muscle–tendon junction where only the FHL muscle belly lies (Péter et al., 2015), with an interelectrode distance of 16 mm. Ultrasound imaging of isolated big toe flexion was performed to confirm the FHL electrode placement. EMG signals were collected at 1500 Hz using a Noraxon wireless EMG system (Noraxon Inc., Scottsdale, AZ, USA) via a 16-bit A/D board (Power 1401, Cambridge Electronic Design, Cambridge, UK) connected to the computer. Signals were recorded using Spike2 software (Cambridge Electronic Design, Cambridge, UK).

Participants were then seated in a custom-made ankle dynamometer (University of Jyväskylä, Finland) with hip at 120°, knee fully extended 0°, ankle at 90°, and first metatarsophalangeal joints at 0°. The foot and thigh were securely fixed with a strap to prevent any heel lift or postural changes. Once participant position was secured, a series of submaximal contractions were performed to precondition the participants and familiarize them with the equipment. Starting with the un-injured limb, participants were asked to perform at least three isometric maximal voluntary contractions (MVC) with an overall contraction and relaxation time of 6 s, and the highest MVC torque was used for subsequent EMG normalization. Torque was sampled at 1 KHz via a transducer in the foot pedal of the ankle dynamometer.

Following the MVC's, participants were asked to walk with a speed ranging from 1.46 – 1.76 $m \cdot s^{-1}$, which corresponds to the average walking speed of young adults (Stenroth et al., 2017) across a 6-meter walkway with photocells at both ends. A 6-cm linear ultrasound probe (96-element −7 MHz – EchoBlaster 128; Telemed, Vilnius, Lithuania or LV8-5 N60-A2; 5–8 MHz – ARTUS EXT-1H, Telemed, UAB) was placed first on the mid-belly of the MG of the injured limb to measure the MG fascicle behaviour during walking (Lichtwark et al., 2007). After the participants had performed a familiarisation trial to get acquainted with the walking path, 2–3 walking trials was performed and saved for offline analysis. Then, the ultrasound probe was attached to the MG of the un-injured limb and the walking trials were repeated.

2.2. Ultrasound

MG fascicle length changes during gait cycles were analysed using a validated semi-automated tracking algorithm (Cronin et al., 2011) in MATLAB R2022b, combined with manual key frame corrections to reduce drift. Briefly, the muscle region of interest was selected to encompass the entire muscular tissue, then a fascicle representing the average fascicle orientation in the mid-region of the muscle belly was selected. In cases where the fascicle extended outside the field of view, extrapolation was used to estimate the location of the fascicle endpoints. Fascicle length changes were tracked using a Lucas-Kanade optical flow algorithm with affine optic flow extension (Cronin et al., 2011). After checking the quality, we excluded ultrasound data from six participants where fascicles were not clearly visible or moved out of the ultrasound imaging plane.

2.3. EMG

EMG data were filtered using a fourth order Butterworth filter between 30 – 350 Hz with MATLAB R2022b. Higher corner frequency was used to augment artifacts but accepting that filtering reduces EMG signal (De Luca et al., 2010). Root mean square (RMS) envelopes were computed using a moving 50 ms window and normalized to the RMS of maximal EMG amplitude. Maximal EMG amplitude was estimated from a 1-sec window around peak torque during MVC. All EMG signals were visually inspected to detect movement artifacts or noise. Upon quality assessment, soleus data from one participant and LG data from two participants were excluded from analysis. Furthermore, data from four FHL gait cycles were discarded due to excessive artifacts. Additionally, in two participants the insertion of soleus muscle was exceptionally low, hence, those cases were excluded to avoid potential cross-talk between FHL and soleus muscles (Péter et al., 2015). Only artifact-free cycles were processed (Fig. 1). Processed EMG data were interpolated from each gait cycle to 100 data points then averaged to cycles within a trial. The walking EMG data was normalised to MVC and expressed as a percentage (%) of the maximal EMG amplitude. Peak and average EMG amplitudes were calculated from the chosen cycles for further analysis. Comparisons between limbs are expressed as percentage points (pp).

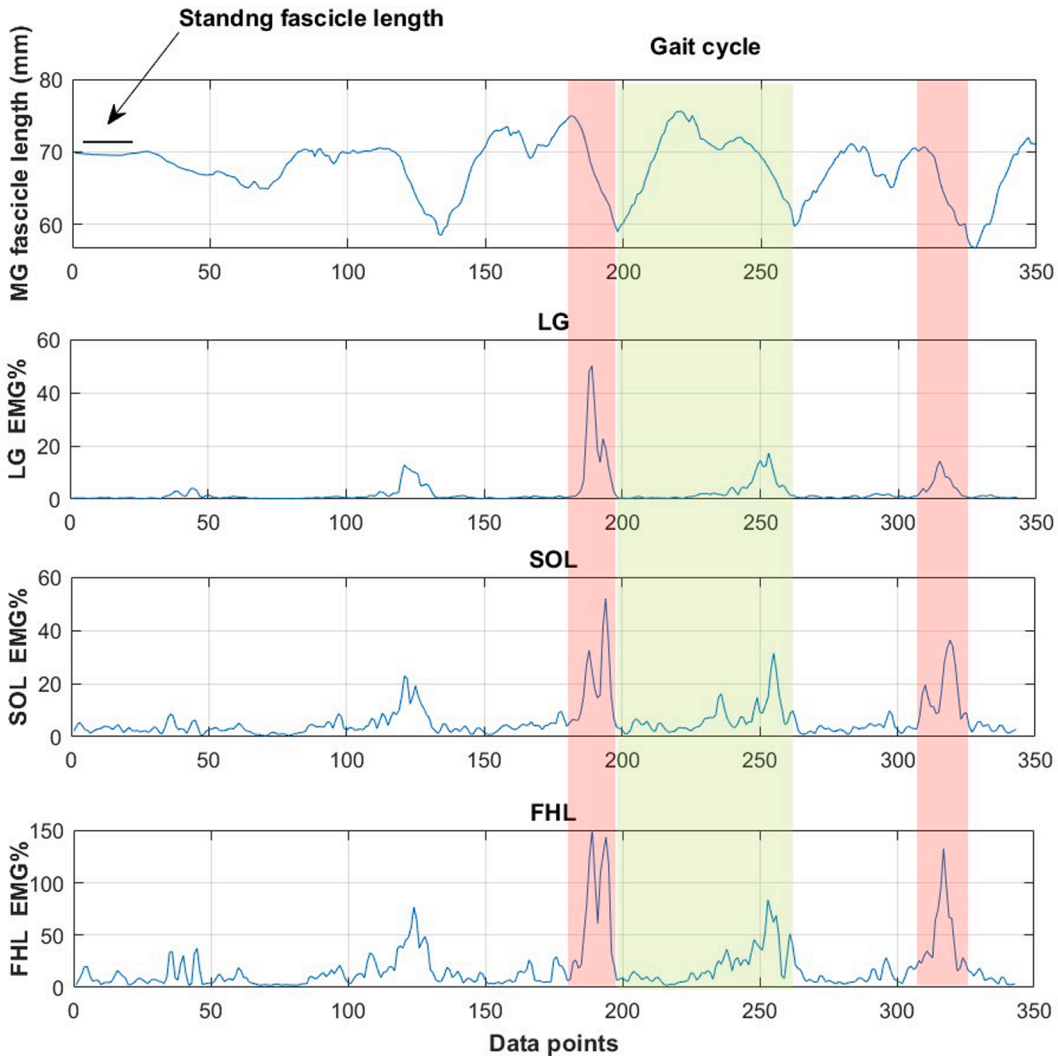


Fig. 1. Illustration of the method used to identify accepted stance phases (green) and exclude steps where artifacts (red) were detected based on fascicle length and/or EMG data. Acceptable cycles were determined to begin once the EMG activity of the LG muscle ceased, marking the approximate end of the stance phase. All EMG signals were visually inspected to detect movement artifacts or noise, and cycles that were artifact-free were included. In this example, after excluding the first step that included acceleration, the cycles in the middle were inspected for noises and artifacts and the clean one (green) was accepted for further analysis.

2.4. Gait cycle detection

Walking trials included the acceleration and deceleration phases, hence the first and last cycles were excluded. From ultrasound recordings, data from 2 to 3 cycles in the middle of the trial were manually identified and interpolated to 100 data points. Cycles were determined based on EMG. Cessation of the normalized LG muscle EMG activity below 2 % was used to mark the approximate end of the stance phase (Fig. 1) (Islam et al., 2020). Artifact free cycles were averaged and fascicle shortening was determined as maximal fascicle shortening by subtracting minimal fascicle length from maximal fascicle length during the gait cycle (Fig. 2). Reference fascicle length was estimated as the length of the fascicles in standing position before initiation of walking (van Dijk et al., 2023). Normalized fascicle shortening was expressed as percentage (%) of fascicle length change during walking relative to the reference standing fascicle length. Comparisons between limbs are expressed as percentage points (pp).

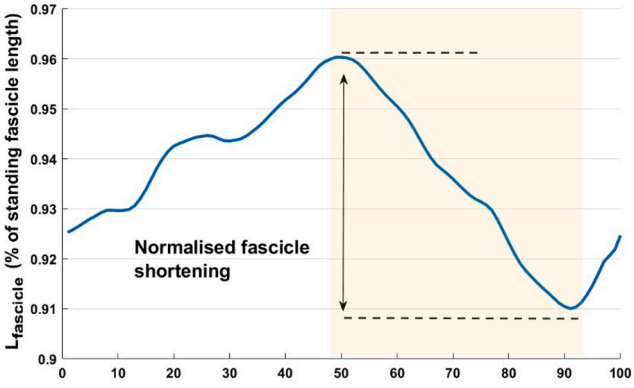


Fig. 2. Representation of the calculation of fascicle shortening. Shortening length was calculated as the difference between maximal and minimal fascicle length (dotted horizontal lines) during the gait cycle normalized to standing fascicle length ($L_{fascicle}$). The cycle in the figure starts with the swing phase followed by the stance phase.

2.5. Statistical analysis

Pairwise T-test was used to compare maximum MVC torque, standing fascicle length, MG fascicle shortening and neuromuscular properties

between the un-injured and injured limbs after checking kurtosis and skewness of the data. If skewness of the data was detected, Wilcoxon signed rank test was used. Statistical analysis was performed using JASP (JASP, Amsterdam, Netherlands). The level of significance was set at $p < 0.05$ and descriptive data are presented as mean ± standard deviation.

3. Results

Of the 24 participants 6 were excluded due to data quality and the analysis were done on the following sample: 18 participants for MG fascicle shortening during walking, 16 for LG EMG, 17 for soleus EMG, and 12 for FHL EMG.

Maximal torque was lower in the injured limb (mean ± SD: 171.7 ± 51.5 Nm) compared to the un-injured limb (226.4 ± 65.2 Nm), with a mean difference (95 %CI) of 54.7 Nm (33.6 to 75.8 Nm). Participants walked at the same speed when the un-injured (mean ± SD 1.73 ± 0.55 $m{\cdot}s^{-1}$) and injured limb (1.74 ± 0.57 $m{\cdot}s^{-1}$) was assessed for MG fascicle shortening. The un-injured limb showed longer fascicles during standing with a mean difference (95 % CI) of 12.9 mm ([7.7 to 18.3 mm], $df = 17$, $t = 5.2$; $p < 0.001$) compared to the injured limb (Fig. 3). In absolute terms, the average shortening of the MG in the injured leg was smaller with a mean difference of 2.8 mm ([95 % CI 0.96 to 4.6], $df = 17$, $t = 3.2$; $p = 0.004$) compared to the un-injured limb. However, when normalized to standing fascicle length, the amount of shortening did not differ ($df = 17$, $t = 0.7$; $p = 0.436$) between the injured (mean ± SD 12.0 ± 5.1 %) and un-injured limbs (13.3 ± 4.8 %).

3.1. EMG activity

During walking, LG showed higher EMG peak amplitude in the injured limb with a mean difference of 14.5 pp ([95 %CI 0.5 to 28.5 pp], $df = 15$, $t = -2.87$; $p = 0.011$) and higher average amplitude with a mean difference of 3.1 pp ([95 %CI 0.8 to 5.4 pp], $df = 15$, $t = -2.19$; $p = 0.043$) compared to the un-injured limb. No statistical differences were observed in soleus or FHL (Fig. 4).

4. Discussion

This study aimed to understand neuromuscular function of the calf muscles after ATR. There was a higher EMG amplitude in the injured LG, but the EMG amplitude was not different in soleus or FHL during the gait cycle. The MG fascicle shortening during walking differed between limbs in absolute terms but not when related to the standing fascicle length. This indicates that re-modelling of the MG muscle after ATR – with potential removal of sarcomeres in series – might be sufficient to enable adequate force production in low-force tasks such as walking.

In this study, MG fascicle length during standing was on average 13 mm shorter in the injured limb. This is likely due to an increase in tendon length after rupture. Since the TS muscle–tendon complex remains the same length, the extra tendon slack is accompanied by re-modelling of the MG muscle to shorter fascicles (Baxter et al., 2018; Hoeffner et al., 2023; Hullfish et al., 2019; Peng et al., 2017). This fascicle adaptation is linked with changes in sarcomere lengths influencing force production capacity (Hoeffner et al., 2023). In long term, increase in tendon stiffness along with MG muscle adaptations would allow muscle to produce more force in the new operating length (Stäudle et al., 2020).

Absolute MG fascicle shortening was lower in the injured limb during walking. Nevertheless, when normalised to standing fascicle length, MG shortening was comparable between limbs. This could indicate that the injured MG was operating within a similar region of the force–length curve at a sarcomere level to the un-injured limb during walking. Stäudle et al., (2022) investigated walking at 1.5 $m{\cdot}s^{-1}$ on a treadmill and found that contractile behaviour of the MG was similar between limbs approximately 4-years post ATR in surgically treated patients. It should be noted that the operating length in Stäudle et al., (2022) was normalised to the optimal sarcomere length at the plateau region of the human sarcomere force–length relation (Herzog et al., 1991). While we normalized to fascicle length during standing, our findings are consistent with those of Stäudle et al., (2022), as we observed similar normalized MG shortening during walking in non-surgically treated patients 1-year after ATR. Jandacka and colleagues reported functional deficits in activities that have higher force requirements such as running, but did not find any asymmetry during overground walking (Jandacka et al., 2018). It is plausible that MG re-modelling via the reduction of sarcomeres in series maintains optimum actin-myosin overlap comparable to pre-injury settings (Hoeffner et al., 2023), preserving low force production in everyday activities such as walking. However, limitations may persist in high demand tasks such as heel raises (van Dijk et al., 2023), jogging or hopping (Jandacka et al., 2017; Trofa et al., 2017; Willy et al., 2017). These limitations possibly result from a reduction in the range of motion at which force can be produced (Stäudle et al., 2020) (Fig. 5), and decreased work capacity of the plantar-flexor muscles of the injured limb (Baxter et al., 2019).

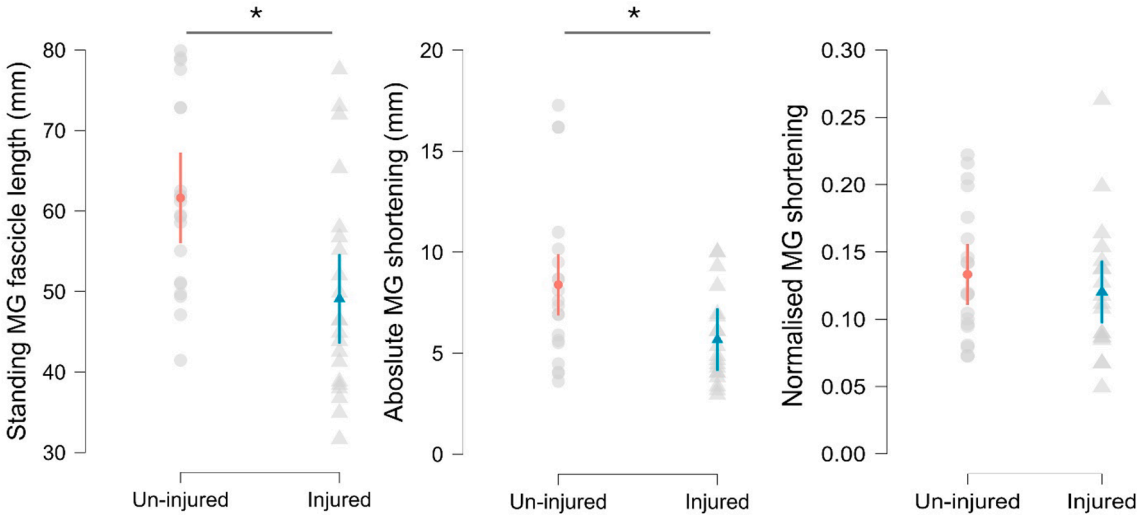


Fig. 3. MG fascicle length during standing (left), absolute MG shortening during walking (middle), and MG fascicle shortening during walking normalized to standing fascicle length (right). Grey dots represent each individual (n = 18) and coloured dots mean with line lengths indicating SD. *Significant difference between limbs ($P < 0.05$).

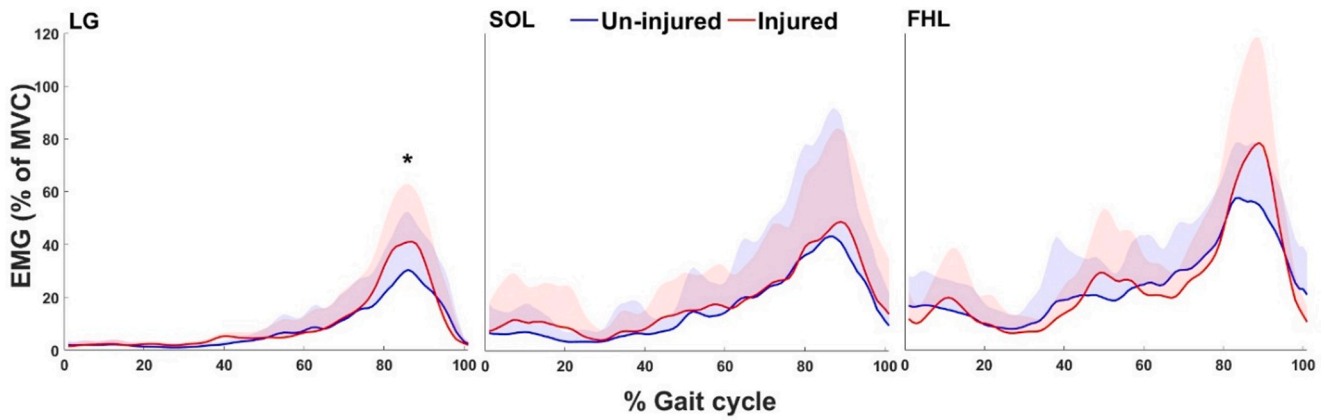


Fig. 4. Mean EMG amplitude data during walking (solid lines) and standard deviation (shaded areas) of all participants (LG n = 16, soleus n = 17, and FHL n = 12). *Significant difference between limbs ($P < 0.05$).

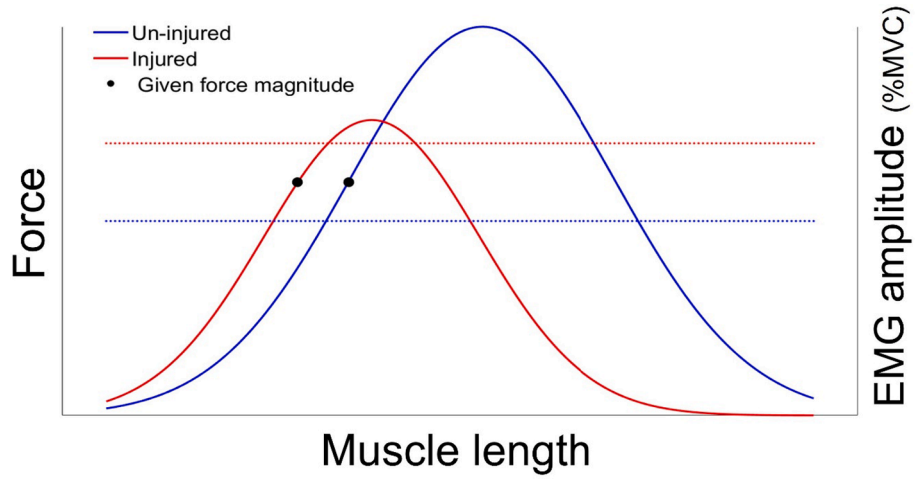


Fig. 5. Schematic representation of the TS muscle force–length curve in the un-injured and injured limb after re-modelling to shorter muscle length. Force curves were estimated based on Hill's equation (Gordon et al., 1966; Lieber and Fridén, 2000) with smaller width and 24% less maximal force generation capacity determined from this cohort. Dotted lines show required EMG activation at a given force magnitude, in injured (red) and un-injured limbs (blue) (Suydam et al., 2015). The black dot illustrates that the injured limb operates at shorter length where greater activity is needed to achieve a given absolute torque as the contralateral limb.

During walking, mean and peak LG EMG amplitudes were higher in the injured limb compared to the contralateral limb, but the EMG amplitude of soleus and FHL did not differ between limbs. Concordance with these results, previous studies reported higher activity of LG and MG but not in soleus during gait (Suydam et al., 2015; Wenning et al., 2021). Wenning et al., (2021) reported higher activity of these muscles during the push-off phase of gait. The increased length of the tendon after rupture might necessitate a stronger muscle contraction during the push-off phase of gait to create adequate force at the ankle joint, since the TS muscle is probably acting outside the optimum region on the force–length curve (Fig. 5), where greater activity is needed to achieve the same absolute torque as the contralateral limb (Nourbakhsh and Kukulka, 2004). Regarding soleus muscle, its physiological cross sectional area is almost 5 times greater than that of LG (Ward et al., 2009), so it is plausible that soleus is still capable of sufficient contribution during walking despite potential atrophy following ATR (Aufwerber et al., 2020; Nicholson et al., 2020). It must also be noted that soleus is a large and complex muscle with multiple compartments, and due to the inherent nature of surface EMG, the electrodes only measure activity from a specific area. It is unknown, for example, whether deep compartments compensate for the altered force production caused by the increased length of the tendon. Furthermore, soleus contains more slow-type muscle fibers which may adapt more slowly than in the faster LG muscle (Lieber and Fridén, 2000; Ward et al., 2009).

Contrary to the initial hypothesis, FHL EMG amplitude in the injured limb was not different compared to the other limb during waking or heel raise. Although higher EMG amplitude in the FHL muscle could be observed in Fig. 4 relative to the contralateral limb, there was large interindividual variability in FHL EMG amplitudes, with no clear distinction between groups of FHL users and non-users. Based on previous studies looking at isometric contractions (Finni et al., 2006; Khair et al., 2023; Masood et al., 2016), FHL was expected to show compensatory activity in the ruptured limb. However, it may be that the increased CSA of the FHL muscle in patients with ATR (Heikkinen et al., 2017), may enhance FHL force generating capacity, hence reducing the need to increased EMG activation in order to achieve a given force amplitude. Furthermore, the FHL compensatory mechanism may not be operative in all patients with ATR and data would need to be looked at individually. According to previous findings (Khair et al., 2023), it could be hypothesized that patients with excessive AT lengthening after rupture have higher FHL EMG activity than patients with lower inter-limb difference in AT length.

4.1. Limitations

This study is not without limitations. First, tracking of the fascicle behaviour from a two-dimensional field of view might not fully capture the complex three-dimensional non-uniform behaviour of the MG muscle (Rana and Wakeling, 2011). Manual frame correction was used to minimize errors and limit drift. Secondly, due to male-dominance of Achilles tendon rupture incidence (Čretnik and Frank, 2004; Houshian et al., 1998; Leppilahti and Orava, 1998), we were unable to have adequate representation of both sexes but inter-limb analysis within individuals should not affect the integrity of the analysis, since no difference between sexes is expected after ATR in non-surgically treated patients (Grävare Silbernagel et al., 2015). Additionally, EMG and ultrasound measurements were performed simultaneously during the walking trials, and it is challenging to maintain the ultrasound probe in an appropriate location and reduce undesired EMG artifacts during walking (Vigotsky et al., 2018). In a few cases, excessive motion of EMG wires during walking generated artifacts. Hence, all data were visually inspected and trials that did not meet the quality check were discarded. This led to a smaller sample size, which reduced the statistical power of the comparison between limbs, especially in the case of FHL. Lastly, using isometric plantarflexion to normalize EMG may not fully activate all examined muscles, which is an inherent problem with EMG studies. In spite of these limitations, our findings are consistent with previous studies that investigated MG contractile behaviour (Stäudle et al., 2022) and EMG activity in the TS muscles after ATR (Suydam et al., 2015; Wenning et al., 2021).

5. Conclusion

MG function during gait is relatively preserved 1-year after ATR in non-surgically treated patients. This suggests that re-modelling of the MG muscle to shorter fascicles compensates for the well-documented MG atrophy and tendon elongation after ATR and might be sufficient to enable adequate force production in daily activities such as walking, where TS muscles operate at sub-maximal force levels.

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CRediT authorship contribution statement

Raad M. Khair: Writing – review & editing, Writing – original draft, Visualization, Investigation, Data curation. **Jadyn Watt:** Writing – review & editing, Data curation. **Maria Sukanen:** Writing – review & editing, Investigation. **Neil J Cronin:** Writing – review & editing, Supervision, Software, Conceptualization. **Taija Finni:** Writing – review & editing, Supervision, Resources, Funding acquisition.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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