DOI: 10.21277/sw.v2i6.259

CALF MUSCLE FUNCTIONAL DEFICIT 1-YEAR AFTER ACHILLES TENDON RUPTURE REPAIR SURGERY

Vaida Aleknavičiūtė-Ablonskė, Albertas Skurvydas, Vilma Juodžbalienė
Lithuanian Sport University, Lithuania
Sigitas Balčiūnas
Šiauliai University, Lithuania

Abstract

The primary muscles responsible for plantar flexion movement are soleus and gastrocnemius which are connected to the calcaneus by the Achilles tendon. Achilles tendon rupture is managed most often with open surgical repair in which the affected limb is immobilized. Understanding the effects of long-term immobilization, how these lead to changes in the physiological properties of the calf muscles changes, may help to improve rehabilitation. Investigating the biomechanical behavior of the calf muscles may provide a better understanding of how the inferior material properties of a scarred Achilles tendon may influence the more global structural properties of the intact muscles.

Keywords: *Isokinetic muscle torque; triceps surae; electromyography.*

Introduction

The Achilles tendon is the thickest and the strongest tendon in the human body (Bressel & McNair, 2001), and it connects the soleus and gastrocnemius muscles to the calcaneus (Kuling & Burnifield, 2008) and serves a basic function in plantar flexion. The Achilles tendon is the most frequently ruptured tendon in the human body (Horstmann, Lukas, Merk, & Mündermann, 2012). Achilles tendon rupture (ATR) disrupts the collagen and elastin components of the tendon and therefore may affect the biomechanical behavior of the muscle-tendon unit (Bressel & McNair, 2001).

ATR is managed most often with open surgical repair (Bressel & McNair, 2001) in which the affected limb is immobilized in an open-toe cast for 4-9 weeks (Horstmann et al., 2012; Naim, Şimşek, Sĭpahĭoĝlu, Esen, & Cakmak, 2005). Prolonged immobilization after ATR results in muscle atrophy and loss of muscle strength (Naim et al., 2005), and can impair lower-limb function (Silbernagel, Helendre-Nilsson, Thomee, Erikson, & Karlson, 2010) in sports and daily life activities. The resultant reduction in dorsiflexion (DF) range of motion between the injured and uninjured legs may remain for 12 months after ATR (Silbernagel, Helendre-Nilsson, Thomee, Erikson, & Karlson, 2009). A deficit in isokinetic calf muscle strength between legs may remain for 6 months to 10 years (Olsson, Silbernagel, Eriksson,

Sansone, Brorsson, Nilsson-Helander, & Karlsson, 2013; Horstman, et al., 2012). The deficit in calf muscle strength and the resultant changes in muscle activity may change the gait pattern after ATR (Horstmann et al., 2012; Finni, Hodgson, Lai, Edgrteo, & Sinha, 2006), and gait abnormalities may be found 24 months after ATR (Silbernagel et al., 2009).

During walking, the angular velocity is ~30°/s in DF and ~220°/s in PF (Kallio, Søgaard, Komi, Selänne, & Linnamo, 2013). Muscles involved in PF are the only human muscles whose fiber length and velocity play an important role in muscle force generation during locomotion (Arnold, Hamner, Seth, Millard, & Delp, 2013). Electromyography (EMG) studies have shown that, during the terminal stance of walking and running, 75% of the muscle activity of DF is performed by triceps surae (Kuling & Burnifield, 2008). The type of muscle contraction is related to different motor unit activation patterns. ATR affects muscle-activation strategies of the soleus (SOL), medial gastrocnemius (GM), and lateral gastrocnemius (GL) muscles (Masood, Kalliokoski, Møller-Bojsen, Magnusson, & Finni, 2014). Dynamic EMG may serve an essential role in differentiating primary gait deficits arising from legs with unilateral impairment after ATR surgery.

Understanding the effects of long-term immobilization, how these lead to changes in the physiological properties of the calf muscles changes, and the best methods to restore muscle physiology and activity after immobilization may help to improve rehabilitation and to prevent Achilles tendon re-rupture. Investigating the biomechanical behavior of the calf muscles may provide a better understanding of how the inferior material properties of a scarred Achilles tendon may influence the more global structural properties of the intact muscle (Bressel & McNair, 2001).

The mechanical changes that occur in PF muscles after ATR surgery and during the healing process have been explored, although only a few studies have examined the long-term effects after ATR repair. Almost all studies have explored these changes for defined periods, such as 6 weeks to 10 years, after ATR surgery. To our knowledge, no previous study has conducted a detailed long-term analysis of isokinetic calf muscle torque and EMG changes after ATR surgery. We sought to understand the relative contributions of the calf muscles to torque during the rehabilitation process after ATR. We hypothesized that, within the first year after unilateral ATR, we would observe significant differences in the neuromechanical outcomes between the injured and uninjured legs during PF and DF.

The aims of our study were: (1) to estimate the isokinetic maximal voluntary contraction (MVC) torque changes during PF and DF and (2) to estimate the EMG parameters for the SOL, GL, GM, and tibialis anterior (TA) during PF and DF movements in injured and uninjured legs at the 1-year follow-up after ATR surgery.

Method and organization

Subjects

Eight male participants who had experienced ATR (age, 36±4 years) volunteered to participate in the study. All participants with ATR had Achilles tendon surgery 6.5-9 weeks previously and underwent not less than 10 rehabilitation procedures. Their ankle range of motion was not less than 15° during PF and DF, and they could walk without pain. All experimental procedures were performed in accordance with the Declaration of Helsinki, and all participants read and signed an informed consent form that had been approved by the Lithuanian Bioethics Committee.

Protocol

The details of the experiment were explained to the participants before testing. All measurements were performed 7 times. The first measurements were performed 6.5-9 weeks after ATR surgery and 10 rehabilitation procedures, and the second after 8 weeks of physiotherapy. The other 5 measurements were performed every 2 months over a 1-year period (see Figure 1). A quantitative survey of the dynamic MVC and EMG of calf muscles was performed using the Biodex medical System PRO 3. PF and DF muscle isokinetic MVC torque was measured in both the injured and uninjured legs. The test started with the uninjured leg. Isokinetic MVC torque during PF and DF was measured in each participant at velocities of 30º/s, 120º/s, and 240º/s. Each participant performed 5 ankle flexion and extension repetitions for each ankle. The rest period between each velocity was 60 seconds.

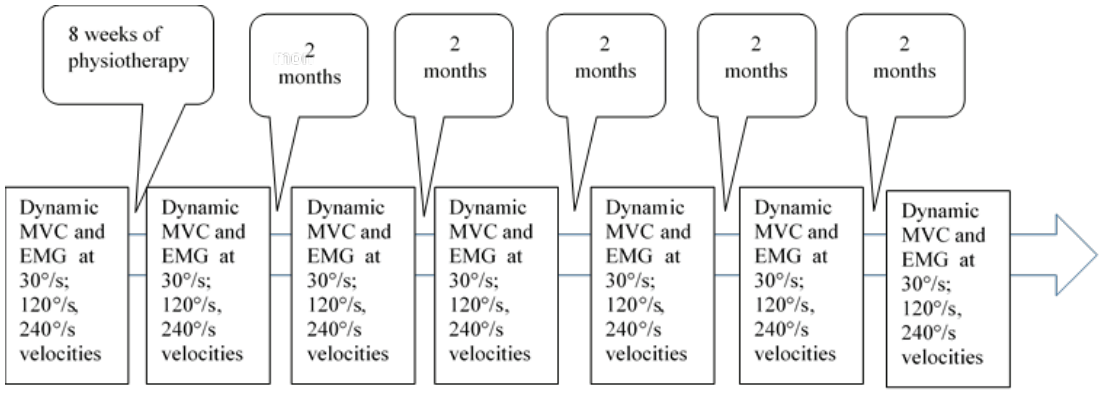

Figure 1. Scheme of protocol

EMG signals of the SOL, GL, GM, and TA were recorded during PF and DF. After careful preparation of the skin by shaving, abrasion, and cleaning with alcohol wipes, bipolar sensor Ag–AgCl surface bar electrodes (DataLog type no. 4X USB, Biometrics Ltd, Gwent, UK) were applied for EMG recording. For the SOL, the electrodes were placed slightly lateral to the middorsal line of the lower leg, about 5 cm distal from the point at which the 2 heads of the gastrocnemius muscle join the Achilles tendon. For the GL and GM, the electrodes were placed longitudinally over the middle of the muscle belly about one-third of the distance proximal to the center of the popliteal crease of the GM and GL heads. For the TA, the electrodes were placed about one-third on the line between the tip of the fibula and the tip of the lateral malleolus. Electrodes were placed on both limbs and in the direction of the muscle fibers. The electrode position for each subject was marked with a pen and its location was measured precisely so that the same recording site was used in each experiment. Muscle activity was recorded at 1000 Hz with a bandwidth of 20 to 460 Hz. EMG files were stored simultaneously on the biometrics memory card and PC hardware, and dedicated software (Biometrics Data Log, Gwent, UK) was used for data processing and analysis. EMG signals were converted to the root mean square (RMS, in mV). All EMG signals were recorded synchronously during the isokinetic MVC.

Statistical analysis

The data were processed using SPSS 22.0 software for mathematical statistical analysis. The data were compared between the uninjured and injured legs during PF and DF. The data

are reported as group mean ± standard deviation (SD) and percentage (relative deficit). The relative deficit in muscle torque was assessed during PF and DF muscle torque (formula 1) also SOL, GL, GM, TA muscles (formula 2) RMS of surface EMG (Lantto, Heikkinen, Flinkkila, Ohtonen, Kangas, Siira, & Leppilahti, 2015).

$$(1)\ \text{Relative deficit} = \frac{UIL - IL}{UIL} \times 100\%;\ (2)\ \text{Relative deficit} = \frac{IL - UIL}{IL} \times 100\%$$

Where UIL is the torque/RMS in the uninjured leg and IL is the torque in the injured leg.

Nonparametric Wilcoxon test was used to identify significant differences between injured and uninjured legs in peak moment during isokinetic flexion and extension at the different velocities (30°/s; 120°/s, and 240°/s). The differences between the injured and uninjured legs at different times (between first and accordingly after 2, 4, 6, 8, and 10 months) were also analyzed. For all tests, the significance level was set at $p<0.05$.

Results

Comparisons of dynamic muscle torque during PF and DF between the uninjured and injured legs are shown in Table 1. During PF and DF, the muscle torque was greater ($p<0.05$) in the uninjured than in the injured leg at all 3 ankle velocities of 30°/s, 120°/s and 240°/s.

Table 1. Changes in MVC of the dynamic plantar flexors and dorsal flexors over the 1-year period after ATR repair

Month	Velocity (°/s)	PF		DF	
		NIL (Nm)	IL (Nm)	NIL (Nm)	IL (Nm)
1	30	64,6± 22,8*	40,4±17,6	31,85±8,09*	20,70±4,72
	120	40,28±6,51*	24,73±7,62	23.13±0.56*	13.58±3
	240	31,85±8,09*	20,70±4,72	19.33±4.7	12.68±4.72
2	30	76,5±9,6*	47,1±17,2	34,70±4,92	23.48±1.76
	120	51,23±9,17*	32,00±7,19	22.28±1.29*	13.2±3.94
	240	34,70±4,92	27,65±5,42	19.35±5.36	14.08±8.24
4	30	74,9±7,7*	55,1±12,9	36.03±3.39	24.13±1.45
	120	55,23±4,54*	35,00±7,05	22.53±2.52*	15.7±4.35
	240	39,30±6,73	30,85±3,68	19.03±0.97*	14.78±4.34
6	30	79,3±2,6*	62,6±9,6	34.03±1.77*	23.58±2.39
	120	54,05±4,57*	40,13±7,74	22.78±3.65*	22.78±3.65
	240	45,73±4,24*	30,63±4,25	20.25±3.94*	15.25±3.92
8	30	84,7±4,7*	65,3±3,6	32.9±5.59*	23.2±2.87
	120	56,88±3,09*	41,43±7,18	21.38±2.22*	16.28±1.35
	240	43,38±5,71*	30,45±3,02	19.95±2.74*	15.18±3.92
10	30	83,5±3,6*	64,9±5,4	34.2±3.1*	24.25±2.85
	120	60,45±10,09*	41,98±6,49	22.8±2.94*	16.36±2.61
	240	48,05±4,96*	33,33±6,19	19.58±5.59*	14.7±3.51
12	30	82,8±4,5*	60,3±12,7	34.1±2.27*	24.55±1.66
	120	57,58±7,88	45,48±5,82	22±2.75*	16.7±4.76
	240	49,70±3,93**	31,53±2,87	21.18±1.49*	16.15±2.92

Data are presented as mean ± SD. *$p<0.05$; **$p<0.001$ compared between the uninjured and injured legs at ankle velocities of 30°/s, 120°/s, and 240°/s.

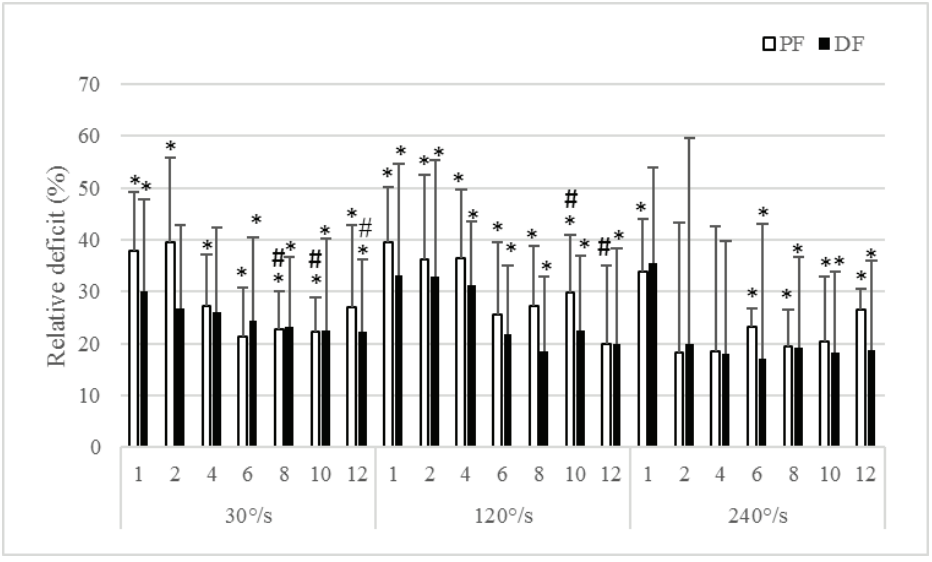

Figure 2. Relative MVC deficits during dynamic PF and DF, expressed as a percentage, between the uninjured and injured legs over the 1-year period after ATR repair

*p<0.05 compared between the uninjured and injured legs. #p<0.05 compared over time.

After 1 year, the relative torque (see Figure 2) deficit (%) remains greater (p<0.05) in UIL than IL leg during PF movement at ankle velocities of 30°/s, and 240°/s and DF at ankle velocities of 30°/s, 120°/s, and 240°/s. During the 1-year period, the relative deficit between the legs during PF decreased (p<0.05) at ankle velocities of 30°/s and 120°/s.

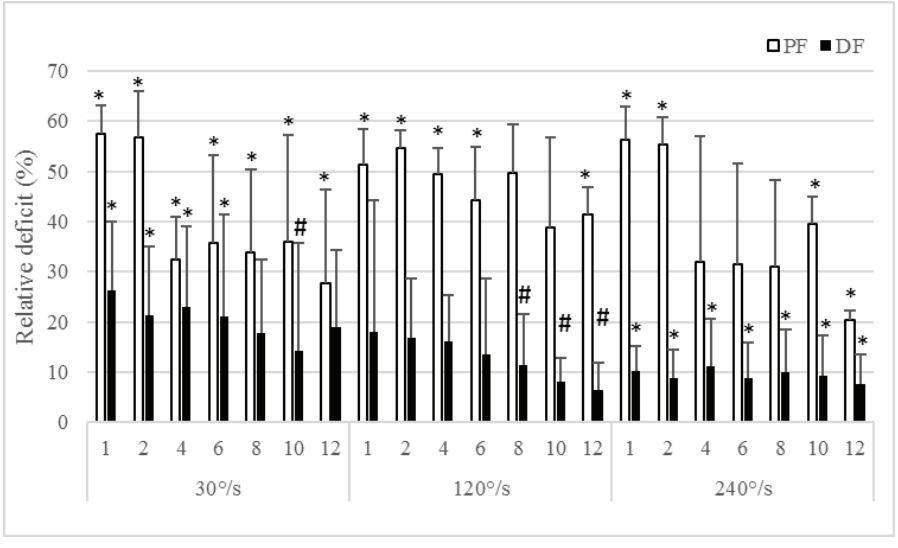

Figure 3. Changes in the relative deficit (%) in the RMS of surface EMG in SOL between the uninjured and injured legs during PF and DF at ankle velocities of 30°/s, 120°/s, and 240°/s over the 1-year period after ATR repair

*p<0.05 compared between the uninjured and injured legs. #p<0.05 compared over time.

After 1 year, the relative deficit (%) in the RMS for the SOL (see Figure 3) remain greater (p<0.05) in IL than UIL leg in PF at an ankle velocity of 30°/s, 120°/s, 240°/s, but

in DF movement at 240°/s velocity. During the 1-year period, the relative deficit decreased ($p<0.05$) at an ankle velocity of 30°/s and 120°/s velocities during DF.

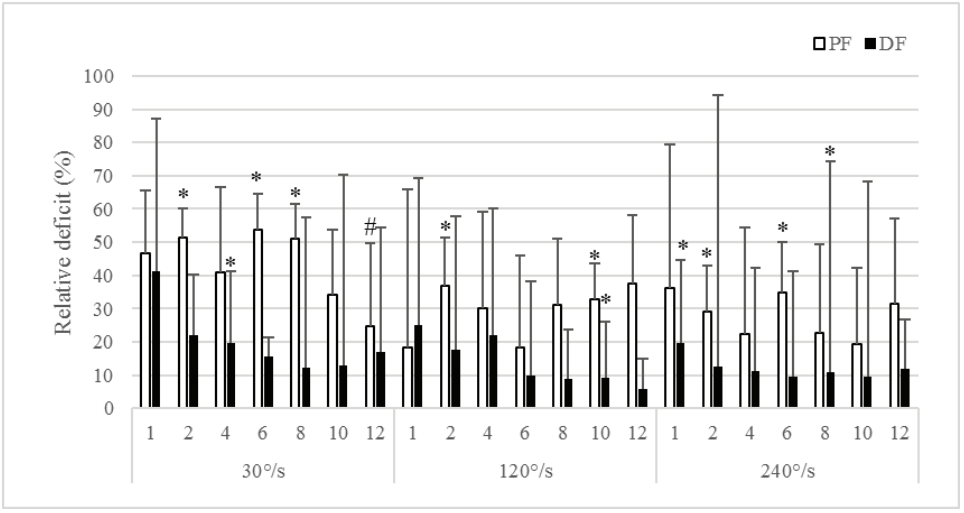

Figure 4. Changes in the relative deficit (%) in the RMS of surface EMG in GL between the uninjured and injured legs during PF and DF at ankle velocities of 30°/s, 120°/s, and 240°/s over the 1-year period after ATR repair

*$p<0.05$ compared between the uninjured and injured legs. #$p<0.05$ compared over time.

After 1 year, the relative deficit (%) in the RMS for GL (see Figure 4) did not differ significantly between the IL and UIL legs. Over the 1 year, the relative deficit decreased ($p<0.05$) at an ankle velocity of 30°/s during PF.

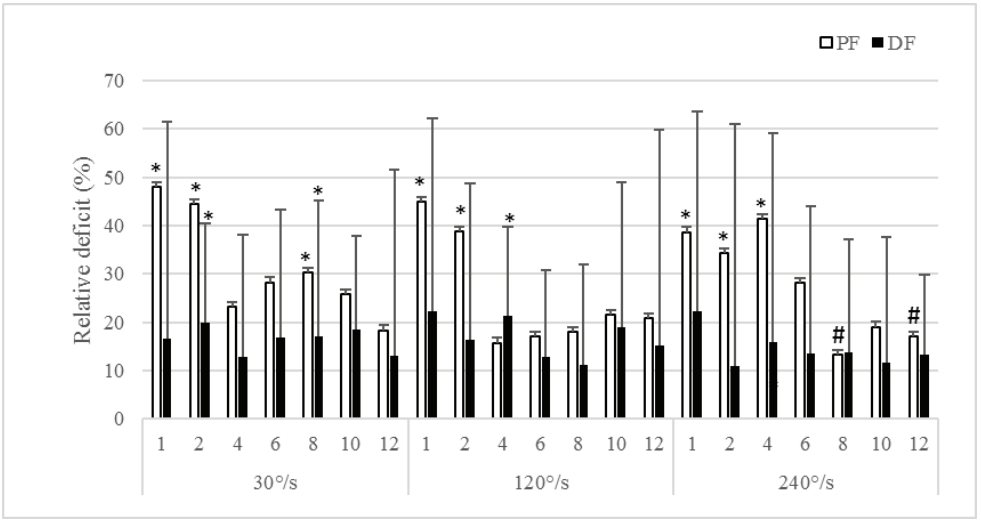

Figure 5. Changes in the relative deficit (%) in the RMS of surface EMG in GM between the uninjured and injured legs during PF and DF at ankle velocities of 30°/s, 120°/s, and 240°/s over the 1-year period after ATR repair

*$p<0.05$ compared between uninjured and injured legs. #$p<0.05$ compared over time

After 1 year, the relative deficit (%) in the RMS for GM (see Figure 5) did not differ significantly between the IL and UIL legs. Over the 1 year, the relative deficit decreased ($p<0.05$) at an ankle velocity of 240°/s during PF.

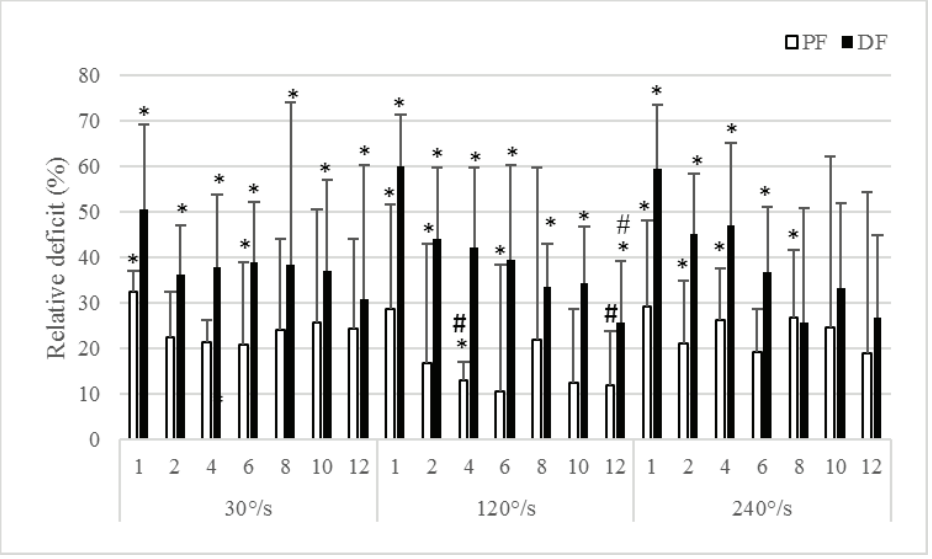

Figure 6. Changes in the relative deficit (%) in the RMS of surface EMG in TA between the uninjured and injured legs during PF and DF at ankle velocities of 30°/s, 120°/s, and 240°/s over the 1-year period after ATR repair

*$p<0.05$ compared between the uninjured and injured legs. #$p<0.05$ compared over time.

After 1 year, the relative deficit (%) in the RMS for TA (see Figure 6) remained greater ($p<0.05$) in IL than UIL leg during DF movement at 30°/s and 120°/s. Over the 1 year, the relative deficit decreased ($p<0.05$) at an ankle velocity of 120°/s during PF and increased ($p<0.05$) at 120°/s during DF.

Discussion

This study was conducted to assess whether, during PF and DF, muscle torque in SOL, GL, GM, and TA, and the RMS of surface EMG differ between the injured and uninjured legs after ATR surgical repair. After 1 year, we found that, during PF and DF, there were differences between the injured and uninjured legs in isokinetic torque and RMS of surface EMG in SOL, GL, GM, and TA.

Calf muscle isokinetic MVC torque

We found that at ankle velocities of 30°/s, 120°/s, and 240°/s during PF and DF (Table 1) muscle torque was greater ($p<0.05$) in the uninjured leg than in the injured leg. It is well known that limited physical activity and prolonged bed rest can cause skeletal muscle atrophy and decrease muscle strength (Horstamann et al., 2012; Kawakami, Akima, Kubo, Muraoka, Hasegawa, Kouzaki, Imai, Suzuki, Gunji, Kanehisa, & Fukunaga, 2001). The decreases after inactivity or bed rest can be greater for strength than for muscle size (Kawakami et al., 2001). Calf muscle atrophy after ATR surgery and prolonged immobilization (Lantto et al., 2015; Horstamann et al., 2012; Akizuki, Gartman, Nisonson, Ben-Avi, & McHugh, 2001) causes

selective atrophy of type I muscle fibers (Horstamann et al., 2012; Langsberg, Ellingsgaard, Madsen, Jansson, Magnusson, Aagaard, & Kjær, 2007; Kawakami et al., 2001). The difference in calf muscle size can be ranged from 4.3 mm to 8 mm (Naim et al., 2006).

Lantto et al. (2015) assessed strength recovery in plantar flexors at 3, 6, and 14 months, and 11 years after ATR. They have found that isokinetic PF muscles strength deficit between injured and uninjured leg at 120°/s velocity after 3 and 6 months in cast group was 19.3% and 19.7% as well as in early motion group was 6.3% and 9.0%. Our results are controversial to the authors' report, we established that the difference between injured and uninjured legs at 120°/s velocity after 4 months, was 36.4% and after 6 months, was 25.5% in PF muscles, although the muscle torque generated during DF tasks differ between IL and NIL legs. Chillemi, Gigante, Verdenelli, Marinelli, Ulisse, Morgantini, & De Palma, (2002) measured isokinetic strength during DF after ATR repair surgery and found 1 year after surgery no significant differences in muscle strength between the injured and uninjured legs during DF at ankle velocities of 60°/s and 120°/s. Our results differ from those by Chillemi et al. (2002) because we observed a significant difference between legs at an ankle velocity of 120°/s.

There are several possible explanations for the lower calf muscle torque in the injured leg compared with the uninjured leg after ATR repair. Kawakami et al. (2001) observed that 20 days of prolonged bed resting affect knee extensor muscle strength, activation, and physiological cross-sectional area. In their study, prolonged bed rest caused significant muscle atrophy and decreased muscle strength by 13-21% and mean physiological cross-sectional area by 4-10%. We propose that the differences in calf muscle torque might be explained by the reduction in physical activity because the leg is immobilized in a cast and because of the pain after surgery. Hu and Nowell (2011) described a load-force sharing strategy between limbs. They manipulated the effector asymmetry of fingers during a bimanual force-matching task by determining the different coefficients of each hand finger that produced force. They found that the finger with greater mechanical efficiency produced more actual force.

There is evidence suggesting that during force tasks in people with lower-limb pain, reduced force in the painful leg might be compensated by increased force produced in the uninjured leg (Hug, Hodges, Carroll, De Martino, Maqnard, &Tucker, 2016). Load transfer from the injured leg to the uninjured leg is hypothesized to be a purposeful strategy for reducing the load within the painful region to protect from further pain. We presume that the differences between the injured and uninjured legs might indicate greater mechanical efficiency in the uninjured leg. Some authors have suggested that differences in muscle strength between the injured leg and the uninjured leg might occur because of the reduced muscle cross-sectional area in the injured leg (Kawakami et al., 2001). It might be assumed that the repaired triceps surae muscle will have a smaller cross-sectional area and lower capacity to produce great force.

Another possible explanation is that PF might by produced by flexor hallucis longus (Masood et al., 2014; Finni et al., 2006) if the Achilles tendon and/or triceps surae is damaged. Some studies have shown that Achilles tendon surgical repair can cause a 50% decrease in triceps surae muscle strength (Manal, Gravare-Silbernagel, & Buchanan, 2011; Naim et al., 2005) and that part of the torque during PF can be compensated by flexor hallucis longus. However, in the longer healing period after ATR repair surgery, the contribution of flexor hallucis longus to total PF torque decreases slightly (Finni et al., 2006). In addition, the contribution of flexor hallucis longus to the overall PF effort is highly individual (Masood et al., 2014). In our study, we did not evaluate the contributions of individual muscles to the total PF torque.

Tendons have elastic mechanical properties: they can stretch in proportion to the force applied and can dissipate a small amount of energy when the force is removed (Lichtwark, Cresswell, & Newsham-West, 2013). Tendon stiffness is determined by the ability of tendon elongation over a given force range (Wang, Chiang, Chen, Shih, Huang, & Jiang, 2013). Tendon stiffness is associated with muscle strength (Muraoka, Muramatsu, Fukunaga, & Kanehisa, 2005). Reduced Achilles tendon stiffness has been observed after ATR repair surgery (Wang et al., 2013; Schepull, Kvist, Anderson, & Aspenberg, 2007). This difference in tendon stiffness may also contribute to the difference in torque between the injured and uninjured legs. However, we did not evaluate viscoelastic properties in the injured and uninjured legs.

Our study shows increased isokinetic torque during PF and DF movements in the injured leg. All of these patients participated in an 8-week physiotherapy program after ATR repair surgery. The physiotherapy program included eccentric muscle strengthening exercises. Six weeks of eccentric muscle-strengthening exercises increased both concentric and eccentric muscle strength (Kaminski, Wabbersen, & Murphy, 1998; Holtermann, Roeleveld, Vereijken, & Ettema, 2007). Four to six weeks of muscle eccentric strengthening exercise can cause muscle structural changes (Holtermann et al., 2007), and 8 weeks of eccentric exercise may cause muscle hypertrophy (Duclay, Martin, Duclay, Cometti, & Pousson, 2009; Farthing & Chilibeck, 2003) and increased muscle strength (Farthing & Chilibeck, 2003).

Calf muscle EMG

We found that during PF and DF (Figures 3-6) at ankle velocities of 30°/s; 120°/s, and 240°/s the RMS of surface EMG in the SOL, GL, GM, and TA was greater ($p<0.05$) in the injured leg than in the uninjured leg. In humans, the activation level of muscle fibers influences the relationship between muscle size and force. As mentioned above, prolonged immobilization increases muscle atrophy and reduces muscle strength. In one study, 5 weeks of leg immobilization caused a 29% decrease in the number of functioning motor units (Kawakami et al., 2001). During ankle immobilization, a greater degree of PF increases the extent of calf muscle atrophy. One study reported that immobilization at 10° of ankle PF after surgery reduced muscle EMG activity by 57% during walking (Akizuki et al., 2001). Recently studies have shown greater triceps surae muscle EMG activity in the injured leg compared with the uninjured leg (Masood et al., 2014; Reid, McNair, Johnson, Potts, Witvrouw, & Mahieu, 2011) in subjects with Achilles tendinopathy. The authors proposed that this could reflect lower MVC and smaller cross-sectional area in the injured leg. In our study, we found greater SOL, GL, GM, and TA muscle activity and lower isokinetic muscle torque during PF and DF in the injured leg. However, we did not measure calf muscle cross-sectional area.

Muscle atrophy is caused by a lack of tension in the muscle (Arnold et al., 2013). However, the atrophied muscle "needs" to contract more because the muscle is contracting without tension during the initial phase of movement. The limited transmission of force through the injured Achilles tendon reduces the effectiveness of the required force generation in muscle after ATR repair surgery. The calf muscle atrophy seen in subjects with ATR can influence the greater calf muscles EMG. After ATR surgery, the Achilles tendon becomes longer, and this longer tendon increases and PF muscles EMG activity. Suydam, Thomas, Buchanan, Manal, & Silbernagel (2015) evaluated the relationship between the lengthened of the Achilles tendon and muscle activation patterns during walking. They found that 6 and 12 months after ATR repair surgery, the Achilles tendon was significantly longer and EMG activity greater in the injured leg than in the uninjured leg. Our study results are similar. We found increased SOL,

GL, GM, and TA muscle activity in the injured leg compared with the uninjured leg. After injury and repair, this longer tendon may require a stronger muscle contraction during foot motion to create force at the ankle joint. In response to the initial increased contraction during the preparation, the force produced at peak contraction may be weaker.

This study has some strengths and limitations. One of the strengths is that, to our knowledge, this is the first longitudinal study of changes in calf muscle isokinetic torque and EMG properties after ATR repair. The main finding of our study is that after 1 year, there remained differences in EMG activity in the calf muscles between the injured and uninjured legs, which may impair joint stability. Our study has some limitations. It included a small sample size, and we did not measure calf muscle cross-sectional area or tendon viscoelastic properties and length.

Conclusions

The relative deficits measured by EMG during PF and DF between the uninjured and injured legs remained 1-year after ATR repair. Muscle torque during PF and DF remained greater in the uninjured leg than in the injured leg; however, there was a tendency for greater EMG activity during PF and DF in the calf muscles of the injured leg than of the uninjured leg.

References

Akizuki, K. H., Gartman, E. J., Nisonson, B., Ben-Avi, S., & McHugh, M. P. (2001). The relative stress on the Achilles tendon during ambulation in an ankle immobiliser: implications for rehabilitation after Achilles tendon repair. *About British Journal of Sports Medicine, 35,* 329-334.

Arnold, E. M., Hamner, S. R., Seth, A., Millard, M., & Delp, S. L. (2013). How muscle fiber length and velocities affects force generation as humans walk and run at different speeds. *The journal of Experimental Biology, 216,* 2150-2160.

Bressel, E., & McNair, P. (2001). Biomechanical behaviour of the plantar flexor muscle-tendon unit an Achilles tendon rupture. *The American Journal of sports medicine*, *29* (3), 321-325.

Chillemi, C., Gigante, A., Verdenelli, A., Marinelli, M., Ulisse, S., Morgantini, A., & De Palma, L. (2002). Percutaneous repair of Achilles tendon rupture: ultrasonographical and isokinetic evaluation. *Foot and Ankle Surgery, 8* (4), 267-276.

Duclay, J., Martin, A., Duclay, A., Cometti, G., & Pousson, M. (2009). Behavior of fascicles and the myotendinous junction of human medial gastrocnemius following eccentric strength training. *Muscle & Nerve, 39* (6), 819-827.

Farthing, J. P., & Chilibeck, P. D. (2003). The effects of eccentric and concentric training at different velocities on muscle hypertrophy. *European Journal of Applied Physiology, 89* (6), 578-586.

Finni, T., Hodgson, J. A., Lai, A. M., Edgrteo, V. R., & Sinha, S. (2006). Muscle synergism during isometric plantar flexion in Achilles tendon rupture patients and in normal subjects revealed by velocity-encoded cine phase-contrast MRI. *Clinical Biomechanics*, *21,* 67-74.

Holtermann, A., Roeleveld, K., Vereijken, B., & Ettema, G. (2007). The effect of rate of force development on maximal force production: acute and training-related aspects. *European Journal of Applied Physiology*, *99,* 605-613.

Horstmann, T., Lukas, C., Merk, J., & Mündermann, R. (2012). Deficits 10-year after Achilles tendon repair. *Journal of Sport Medicine*, *33*, 474-479.

Hu, X., & Newell, K. M. (2011). Aging, visual information, and adaptation to task asymmetry in bimanual force coordination**.** *Journal of applied Physiology, 111,* 1671-1680.

Hug, F., Hodges, P. W., Carroll, T. J., De Martino, E., Maqnard, J., & Tucker, K. (2016). Motor adaptation to pain during a bilateral plantarflexion task: Does the cost of using the non-painful limb mater? *PLoS ONE, 11* (4), 1-17.

Kallio, J., Søgaard, K., Komi, P. A., Selänne, H., & Linnamo, V. (2013). Motor unit firing behaviour of soleus muscle in isometric and dynamic contractions. *PLoS One*, *8* (2), 1-7.

Kaminski, T. W., Wabbersen, C. V., & Murphy, R. M. (1998). Concentric versus enhanced eccentric hamstring strength training: clinical implications. *Journal of Athletic Training, 33*(3), 216-221.

Kawakami, Y., Akima, H., Kubo, K., Muraoka, Y., Hasegawa, H., Kouzaki, M., Imai, M., Suzuki, Y., Gunji, A., Kanehisa, H., & Fukunaga, T. (2001). Changes in muscle size, architecture, and neural activation after 20 days of bed rest with and without resistance exercise. *European Journal of Applied Physiology*, *84,* 7-12.

Kawakami, Y., Akima, H., Kubo, K., Muraoka, Y., Hasegawa, H., Kouzaki, M., Imai, M., Suzuki, Y., Gunji, A., Kanehisa, H., & Fukunaga, T. (2001). Changes in muscle size, architecture, and neural activation after 20 days of bed rest with and without resistance exercise. *European Journal of Applied Physiology*, *84,* 7-12.

Kuling, K., & Burnfield, J. M. (2008). The role of biomechanics in orthopaedic and neurological rehabilitation. *Acta of Bioengineering and Biomechanics*, *10* (2), 3-14.

Langsberg, H., Ellingsgaard, H., Madsen, T., Jansson, J., Magnusson, S. P., Aagaard, P., & Kjær, M. (2007). Eccentric rehabilitation exercise increases peritendinous type I collagen synthesis in humans with Achilles tendinosis. *Scandinavian journal of Medicine and Science in Sports, 17*, 61-66.

Lantto, I., Heikkinen, J., Flinkkila, T., Ohtonen, P., Kangas, J., Siira, P., & Leppilahti, J. (2015). Early functional treatment versus cast immobilization in tension after Achilles rupture repair. Results of a prospective randomized trial with 10 or more Years of follow-up. *The American Journal of Sports Medicine, 43* (9), 2302-2309.

Lichtwark, G. A., Cresswell, A. G., & Newsham-West, R. J. (2013). Effect of running on humans Achilles tendon length-tension properties in the free and gastrocnemius components. *Journal of Experimental Biology, 216,* 4388-4394.

Manal, K., Gravare-Silbernagel, K., & Buchanan, T. S. (2012). A real-time EMG-driven musculoskeletal model of the ankle. *Multibody System Dynamics, 28*, 169-180.

Maquirriain, J. (2011). Achilles tendon rupture: avoid tendon lengthening during surgical repair and rehabilitation. *Yale journal of biology and medicine, 84*, 289-300.

Masood, T., Kalliokoski, K., Møller-Bojsen, J., Magnusson, S. P., & Finni, T. (2014). Plantarflexor muscle function in healthy and chronic Achilles tendon pain subjects evaluated by the use of EMG and PET imaging. *Clinical Biomechanics, 29,* 564-570.

Muraoka, T., Muramatsu, T., Fukunaga, T., & Kanehisa, H. (2005). Elastic properties of human Achilles tendon are correlated to muscle strength. *Journal of Applied Physiology*, *99*, 665–669.

Naim, F., Şimşek, A., Sĭpahĭoğlu, S., Esen, A., & Cakmak, G. (2005). Evaluation of the surgical result of Achilles tendon ruptures by gait analysis and isokinetic muscle strength measurements. *Acta orthopedica et traumatologica Turcica*, *39*(1), 1-6.

Olsson, N., Silbernagel, K. G., Eriksson, B. I., Sansone, M., Brorsson, A., Nilsson-Helander, K., & Karlsson, J. (2013). Stable surgical repair with accelerated rehabilitation versus nonsurgical treatment acute Achilles tendon ruptures: a randomized controlled study. *The American Journal of Sports Medicine. 41* (12), 2867-2876.

Reid, D., McNair, P. J., Johnson, S., Potts, G., Witvrouw, E., & Mahieu, N. (2012). Electromyographic analysis of an eccentric calf muscle exercise in persons with and without Achilles tendinopathy. *Physical Therapy in Sport, 13,* 150-155.

Schepull, T., Kvist, J., Anderson, Ch., & Aspenberg, P. (2007). Mechanical properties during healing of Achilles tendon ruptures to predict final outcome: pilot Roentgen stereophotogrammetric analysis in 10 patients. *Musculoskeletal disorders*, *8* (116), 472-483.

Silbernagel, K. G., Helendre-Nilsson, K., Thomee, R., Erikson, B. I., & Karlson, A. (2009). A new measurement of heel-rise endurance with the ability to detect functional deficits in patients with Achilles tendon rupture. *Knee Surgery, Sports Traumatology*, *Arthroscopy, 8* (2), 258-264.

Suydam, S. M., Thomas, S., Buchanan, T. B., Manal, K., & Silbernagel, K. G. (2015). Compensatory muscle activation caused by tendon lengthening post Achilles tendon rupture. *Knee Surgery, Sports Traumatology, Arthroscopy, 23*(3), 868-874.

Wang, H. K., Chiang, H., Chen, W. S., Shih, T. F., Huang, Y. C., & Jiang, C. C. (2013). Early neuromechanical outcomes of the triceps surae muscle-tendon following an Achilles tendon repair. *Archives of Physical Medicine and Rehabilitation, 94* (8), 1590-1598.

CALF MUSCLE FUNCTIONAL DEFICIT 1-YEAR AFTER ACHILLES TENDON RUPTURE REPAIR SURGERY

Summary

Vaida Aleknavičiūtė-Ablonskė, Albertas Skurvydas, Vilma Juodžbalienė,
Lithuanian Sport University, Lithuania
Sigitas Balčiūnas, Šiauliai University, Lithuania

The Achilles tendon is the thickest and the strongest tendon in the human body, and it connects the soleus and gastrocnemius muscles to the calcaneus. Achilles tendon rupture (ATR) disrupts the collagen and elastin components of the tendon and therefore may affect the biomechanical behavior of the muscle-tendon unit. ATR is managed most often with open surgical repair and it is followed by prolonged immobilization as well as decreased muscle strength. We performed a longitudinal study of changes in calf muscle isokinetic torque and electromyography after ATR repair. We sought to understand the relative contributions of the calf muscles torque during the rehabilitation process after ATR surgery. **The aims** of our study were: (1) to estimate the isokinetic maximal voluntary contraction torque changes during plantar flexion and dorsiflexion and (2) to estimate the EMG parameters for the soleus, lateral gastrocnemius, medial gastrocnemius and tibialis anterior during plantar flexion and dorsiflexion movements in the injured and uninjured legs at the 1-year follow-up after ATR surgery.

Methods: Eight men who underwent ATR surgery and not less than 10 rehabilitation sessions were included. The subjects performed isokinetic plantar flexion and dorsiflexion movements at 30°/s, 120°/s, and 240°/s, and the root mean square (RMS) of surface EMG was recorded for the soleus, lateral gastrocnemius, medial gastrocnemius, and tibialis anterior. Each participant performed 5 ankle flexion and extension repetitions. Isokinetic maximum voluntary contraction torque and RMS were measured during plantar flexion and dorsiflexion 7 times over a 1-year period.

Results: After 1 year isokinetic MVC torque during PF and DF remained greater ($p<0.05$) in the uninjured leg than in the injured leg. The RMS of EMG in soleus, lateral gastrocnemius, medial gastrocnemius, and tibialis anterior remained greater ($p<0.05$) in the injured leg than in the uninjured leg. The relative deficit between the injured and uninjured leg during plantar flexion decreased with time ($p<0.05$) in muscle torque (30°/s velocity), RMS of lateral and medial gastrocnemius (accordingly 30°/s and 240°/s velocities) and tibialis anterior (120°/s velocity) muscles. The RMS during dorsiflexion decreased (both $p<0.05$) in soleus (30°/s, 120°/s velocities),) and tibialis anterior (120°/s velocity) muscles.

Conclusions: The relative deficits measured by EMG during PF and DF between the uninjured and injured legs remained 1-year after ATR repair. Muscle torque during PF and DF remained greater in the uninjured leg than in the injured leg; however, there was a tendency for greater EMG activity during PF and DF in the calf muscles of the injured leg than of the uninjured leg.