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Short communication

# Functional deficits may be explained by plantarflexor remodeling following Achilles tendon rupture repair: Preliminary findings

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ABSTRACT

Achilles tendon ruptures are common injuries that often lead to long-term functional deficits. Despite the prevalence of these injuries, the mechanism responsible for limited function has not yet been established. Therefore, the purpose of this study was to present preliminary findings that support a hypothesis that skeletal muscle remodeling is the driving factor of poor outcomes in some patients. Biomechanical and ultrasonography assessments were performed on a patient that presented with poor functional outcomes 2.5 years after a surgically-repaired acute Achilles tendon rupture. Single-leg heel raise height was decreased by 75% in the affected limb (3.0 cm compared to 11.9 cm) while walking mechanics showed no deficits. Ultrasonography revealed that the affected medial gastrocnemius muscle was less thick and had shorter, more pennate fascicles compared to the unaffected limb. A simple computational model of a maximal-effort plantarflexion contraction was employed to test the implications of changes in muscle architecture on single-leg heel raise function. Subject-specific measurements of fascicle length and pennation were input into the model, which supported these architectural parameters as being drivers of heel raise function. These preliminary findings support the hypothesis that an Achilles tendon rupture elicits changes in skeletal muscle architecture, which reduces the amount of work and power the joint can generate. This multidisciplinary framework of biomechanical, imaging, and computational modeling provides a unique platform for studying the complex interactions between structure and function in patients recovering from Achilles tendon injuries.



## 1. Introduction

Achilles tendon ruptures often lead to long-term functional deficits (Lantto et al., 2015a; Leppilahti et al., 2000; Silbernagel et al., 2012; Willy et al., 2017) despite improved treatment and rehabilitation protocols (Kangas et al., 2003; Mandelbaum et al., 1995; Willits et al., 2010). An elongated tendon following injury is considered to be a predictor of functional deficits in a single-leg heel raise during the first year following surgical repair (Silbernagel et al., 2012). While tendon elongation is an important clinical indicator of patient function, such as single-leg heel raise height (Silbernagel et al., 2012), the underlying musculoskeletal mechanisms that dictate plantarflexor function in patients treated for Achilles tendon ruptures remain unclear.

The purpose of this study was to present preliminary findings utilizing a multidisciplinary framework for linking plantarflexor remodeling with functional deficits in patients who have suffered Achilles tendon ruptures. To accomplish this, we quantified plantarflexor morphology and patient function using an integrated ultrasonography and motion capture approach (Fukunaga et al., 2001). Further, we simulated a maximal-effort plantarflexion contraction using a computational model (Baxter et al., 2012) to test the effects of muscle remodeling on plantarflexor function. Using this framework, we began to build support for future testing of our hypothesis that long-term functional deficits are associated with decreased muscle fascicle length and increased pennation.

## 2. Methodology

A 27-year old male (1.83 m and 84 kg) with a poor clinical outcome 2.5 years following an acute Achilles tendon rupture participated in this IRB approved study. The ruptured Achilles tendon on the right leg was surgically repaired by another provider using an open-reduction within 1 week of the initial injury. The patient described inability to participate in recreational activities due to loss of ankle strength and reported experiencing no pain. Poor clinical outcomes were confirmed using a clinical outcome score

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(Achilles Tendon Total Rupture Score: 49/100, (Nilsson-Helander et al., 2007)), an evaluation by a fellowship-trained foot and ankle surgeon, and the inability to perform a single-leg heel raise of at least 50% of the unaffected side.

Morphological measurements of the medial gastrocnemius were made on images acquired using ultrasonography (Telemed SmartUs, Vilnius, Lithuania) on the affected and unaffected sides. Muscle fascicle pennation, length, and muscle belly thickness (Fig. 1) were acquired using an 8 MHz linear ultrasound probe (Telemed LV8-5L60N-2, Vilnius, Lithuania) (Baxter and Piazza, 2014). These measurements of medial gastrocnemius morphology were acquired with the patient seated in an isokinetic dynomameter (System 4, Biodex Medical Systems, Shirley, NY) with an extended knee and ankle held in neutral position. During image acquisition, the medial gastrocnemius of the affected limb was in a retracted state that prevented us from imaging the muscle midbelly, which resulted in aponeuroses that were not parallel. To control for this transducer position, we decided to image the contralateral muscle belly at the same position proximal of the muscle-tendon junction, which also resulted in non-parallel aponeuroses. A single investigator identified the deep and superficial aponeuroses as well as a single fascicle using a custom-written script (MATLAB, The Mathworks, Natick, MA). Lines were manually fit to each of these structures and extrapolated off of the image until the fascicle intersected both deep and superficial aponeuroses to define fascicle length ($l_f$) (Zhou et al., 2015). The angle between the deep aponeurosis and fascicle was calculated to define muscle pennation ($\theta_f$) (Fukunaga et al., 1997). Muscle belly thickness ($t_f$) was calculated as the distance between the superficial and deep aponeuroses in the mid-section of the image. Repeated measures of uninjured plantarflexors in another cohort found these measurements to be reliable (Appendix). Because the aponeuroses were non-parallel, we were not able to calculate the change in tendon length based on measurements of muscle architecture (Fukunaga et al., 2001). Physiological cross sectional area (PCSA) of the medial gastrocnemius muscle was estimated by first calculating muscle volume as a cylinder (Eq. (1) and normalizing this volume by the fascicle length (Morse et al., 2005).

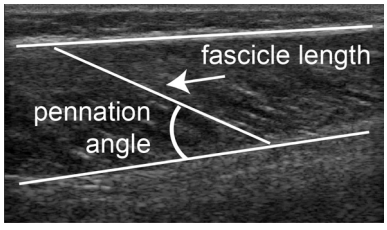


**Fig. 1.** Ultrasound images of the medial gastrocnemius muscle were analyzed to quantify the fascicle length, pennation angle, and thickness (not shown for clarity).

Supplementary data associated with this article can be found, in the online version, at https://doi.org/10.1016/j.jbiomech.2018.08.016.

$$PCSA = (\pi * (0.5 * t_f)^2 * (l_f \cos\theta_f))/l_f \quad (1)$$

Plantarflexor function was assessed through a battery of tests that consisted of isometric strength testing, walking, and single-leg heel raises. Isometric plantarflexion strength were tested in an isokinetic dynamometer with the patient seated with a fully extended knee and neutrally-aligned ankle (Baxter and Piazza, 2014). Lower extremity biomechanics were quantified during comfortable speed walking and single-leg heel raises using a 12-camera motion capture system (Raptor series, Motion Analysis Corporation, Santa Rosa, CA) and 3 force plates (BP600900, Advanced Mechanical Technology, Inc., Watertown, MA). Fascicle shortening was synchronously acquired (Appendix) with an 8 MHz ultrasound probe during this battery of functional tests (Lichtwark and Wilson, 2005). Plantarflexion kinematics, torque, power, and fascicle shortening dynamics were calculated to establish the link between muscular and patient function. Peak ankle plantarflexion, torque, and power were calculated using open-source musculoskeletal software (Delp et al., 2007) (Opensim v3.3, Stanford University), and fascicle shortening dynamics were quantified using a custom-written tracking routine.

The effects of fascicle length and pennation on single-leg heel raise height were tested using a simple computational model (Fig. 2A). To simulate a single-leg heel raise, maximal plantarflexion contractions of a foot pushing against a moving wall were simulated (Baxter et al., 2012). The wall retreated at a constant speed of 0.15 m/s, which was determined based on the average vertical velocity of the patient's pelvis when performing single-leg heel raises on the unaffected limb. A single hill-type muscle model in series with an elastic element inserted into the foot and leg segments. Fascicle lengths and pennation angles were varied within the ranges of the patients' affected and unaffected limbs (Table 1) to isolate the implications of these musculoskeletal parameters on plantarflexor function. Specifically, fascicle length and pennation angles were varied from 4 to 11 cm (1 cm increments) and 10 to 40 degrees (10 degree increments), respectively. Tendon slack lengths were determined geometrically by subtracting the medial

**Table 1**
Medial gastrocnemius morphology.

| | Unaffected | Affected | %Change |
|---|---|---|---|
| Fascicle length | 11.2 cm | 4.6 cm | −59% |
| Pennation | 13 degrees | 34 degrees | 162% |
| Thickness | 2.9 cm | 2.2 cm | −24% |
| PCSA | 6.4 $cm^2$ | 3.2 $cm^2$ | −51% |

PCSA – physiologic cross sectional area.

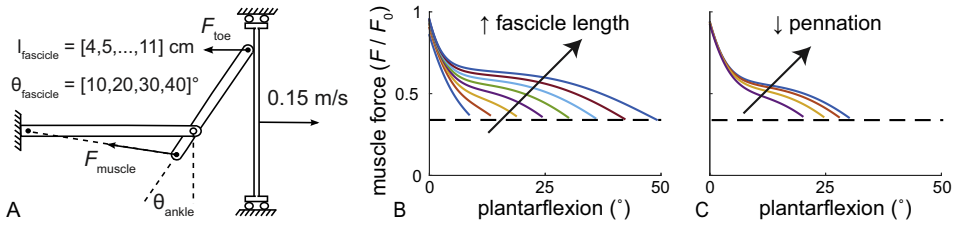


**Fig. 2.** (A) A single-leg calf raise was simulated using a computational model of a foot maximally plantarflexing against a retreating wall. (B) Increasing fascicle lengths and (C) decreasing pennation increases the amount of muscle force that can be generated throughout the simulated single-leg calf raise. (B) pennation and (C) fascicle lengths were held constant at 10 degrees and 5 cm, respectively, to characterize the effects of changing a single muscular parameter. Muscle forces are presented as a ratio of the peak isometric force of the muscle ($F/F_0$). Simulations were stopped when the muscle was unable to generate ground reaction forces that exceeded the scaled body weight of the subject (horizontal dashed line) or when the ankle reached 50 degrees of plantarflexion.

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