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Force-sharing within the Triceps Surae: An Achilles Heel in Achilles Tendinopathy

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ABSTRACT

CROUZIER, M., K. TUCKER, L. LACOURPAILLE, V. DOGUET, G. FAYET, M. DAUTY, and F. HUG. Force-sharing within the Triceps Surae: An Achilles Heel in Achilles Tendinopathy. *Med. Sci. Sports Exerc.*, Vol. 52, No. 5, pp. 1076–1087, 2020. **Purpose:** The primary aim of this study was to determine whether the distribution of force between the three heads of the triceps surae differs between people with Achilles tendinopathy and controls. We also aimed to determine the effect of this force distribution on subtendon strain. **Methods:** Data were collected for 21 participants with Achilles tendinopathy and 21, case-wise paired, asymptomatic controls. Ultrasonography was used to measure muscle volume, fascicle length, pennation angle and subtendon length at rest. Muscle activation was estimated using surface electromyography during maximal and submaximal isometric plantarflexion tasks. The product of normalized activation, physiological cross-sectional area, and the cosine of the pennation angle was considered as an index of individual muscle force. Displacement of the distal myotendinous junction of each muscle was measured during the submaximal contractions. **Results:** The contribution of the gastrocnemius lateralis to the overall triceps surae physiological cross-sectional area and activation was 8.5% ($P = 0.047$, $d = 0.75$) and 24.7% lower (main effect group $P = 0.009$, $d = 0.67$) in people with Achilles tendinopathy than in the controls, respectively. Consequently, gastrocnemius lateralis contributed approximately 28% less (main effect group $P = 0.025$, $d = 0.62$) of the triceps surae force in people with Achilles tendinopathy. The contribution of gastrocnemius medialis and soleus was not different between groups. Subtendon strain was not different between groups ($P = 0.835$). **Conclusions:** These results provide evidence for a difference in force-sharing strategy within the triceps surae in people with Achilles tendinopathy compared with the controls. Whether this altered strategy is a cause or a consequence of Achilles tendinopathy should be explored further. **Key Words:** MUSCLE COORDINATION, GASTROCNEMIUS, PHYSIOLOGICAL CROSS-SECTIONAL AREA, ELECTROMYOGRAPHY, MUSCULOSKELETAL DISORDER

Achilles tendinopathy is associated with localized pain during activities that load the tendon. As such, Achilles tendinopathy is associated with lower exercise tolerance, impaired function, and reduced quality of life. The lifetime incidence of Achilles tendinopathy is approximately 1 in every two long-term middle- and long-distance runners (1), and symptoms may last for many years (2). The etiology of this disease is poorly understood. New approaches are required to understand the mechanisms that underlie the development of Achilles tendinopathy, its persistence, and resolution.

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The Achilles tendon is the largest tendon in the body and links the triceps surae (TS) to the calcaneus. Its architecture is complex, as it is composed of three subtendons (or three main fascicle bundles), that each arises from gastrocnemius medialis (GM), gastrocnemius lateralis (GL), and soleus (SOL). Together, the subtendons exhibit a twisted structure, the degree of which varies between individuals (3). Nonuniform displacements within the Achilles tendon have been observed using ultrasound imaging during isometric contractions (4) and passive stretching (5). Nonuniform displacements are consistent with a nonuniform distribution of load within the tendon as suggested by *in vivo* (6) and *in vitro* studies (7). In addition, shear force at the interface of tendon fascicles has been evidenced by measuring Lubricin concentration, a protein whose expression is stimulated by shear force (8). Nonoptimal distribution of load and/or the amount of shear forces between subtendons have been hypothesized to contribute to the development of Achilles tendinopathy (8,9). Using a modeling approach, Handsfield et al. (10) suggested that the nonuniform distribution of load or strain within the Achilles tendon is mainly determined by the distribution of force among the heads of the TS. This link between the distribution of load within the Achilles tendon

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and the distribution of force among individual muscles has also been suggested by indirect *in vivo* measurement (4). Together, these works suggest that the distribution of muscle force might be involved in the development of Achilles tendinopathy; but this remains to be tested.

Muscle coordination relates to the distribution of force among individual muscles (or force-sharing strategy) to produce a motor task (11). As there is no noninvasive experimental technique that can measure the force produced by individual muscles *in vivo*, it is not possible to directly determine the role of muscle coordination strategies in the development of musculoskeletal disorders, including Achilles tendinopathy. Recently, we published a series of studies that considered the product of muscle activation, pennation angle and muscle physiological cross-sectional area (PCSA) as an index of the force produced by individual muscles during isometric contractions (12–14).

The primary aim of this exploratory study was to determine whether the distribution of force between the heads of the TS differs in people with Achilles tendinopathy compared with controls. We hypothesized that people with Achilles tendinopathy would exhibit a different force distribution between the heads of the TS during submaximal isometric tasks. In the case that a difference in force sharing strategy was observed between our groups, we further aimed to determine whether particular force-sharing strategies in people with Achilles tendinopathy were associated with recovery of symptoms over a 6-month follow-up period. Our secondary aim was to determine the effect of this force distribution on subtendons' strain.

MATERIALS AND METHODS

Participants

The study was conducted with 45 volunteers who were recruited by local advertisement (Table 1), that is, 23 participants with Achilles tendinopathy and 22 controls. First, all participants were directed to an online questionnaire to determine if they were likely eligible to participate. Then, the eligible participants with Achilles tendinopathy attended a physical screening conducted by a physiotherapist to verify whether they met the following inclusion criteria: (i) between 18 and 45 yr old, (ii) self-reported pain within the Achilles tendon within the last week, rated at least 3 on an 11-point scale from 0 (no pain) to 10 (unbearable pain), (iii) increased pain when walking or running slowly, and (iv) pain duration of at least 3 wk. Pain on palpation of the tendon was used to confirm the location of sensitivity. The exclusion criteria for both groups were: (i) pain/injury within the lower limbs within the previous 6 months (except in the Achilles tendon for people with Achilles tendinopathy), (ii) corticosteroids injection in the lower limb within the previous 6 months, and (iii) a history of lower limb surgery. To provide validated information about Achilles tendon pain and disability, all participants completed the Victorian Institute of Sport Assessment—Achilles questionnaire (VISA-A) (15). Physical activity level was estimated using the International Physical Activity Questionnaire (IPAQ) [evaluation tool of physical activity (16)] and the type of physical activity was investigated with questions on disciplines that were practiced regularly. Recruitment of control participants was targeted to match between group by type and volume of physical activity, and then by age, height, and body mass (all within ±15%).

Two participants with Achilles tendinopathy and one healthy control were excluded for technical reasons that are explained further below. Therefore, data were reported for 21 participants in each group. Table 1 provides the demographic characteristics of these participants. Within each group, 18 of the 21 participants practiced running 2 h 36 min ± 2 h 18 min·wk^{-1}. Four participants with Achilles tendinopathy had bilateral Achilles tendinopathy, in which case, the side with the highest level of pain just prior to data collection was chosen for data collection. Five of the participants with Achilles tendinopathy had insertional tendinopathy, and 16 had midportion tendinopathy, as determined by the physiotherapist during palpation. The average duration of Achilles tendinopathy symptoms was 3.6 ± 2.7 months. Ten of 21 participants with tendinopathy had their dominant side measured as this was their painful (or more painful) side at the time of testing. The side considered for the control group was chosen to match with dominance of the tested side of their paired-Achilles tendinopathy participant. This resulted in that the proportions of dominant (48%) and nondominant sides tested (52%) were similar in the two groups. All participants were informed of the protocol and methods used before providing written informed consent. The experimental procedures were approved by the local ethics committee (Rennes Ouest V-CPP-MIP-010), and all procedures adhered to the declaration of Helsinki.

Experimental Protocol

Participants attended one laboratory session. First, ultrasound images were taken at rest and used to estimate muscle volume,

TABLE 1. Demographic and injury characteristics.

	Control Group (*n* = 21)	Achilles Tendinopathy Group (*n* = 21)	*P*
Age (yr)	35.1 ± 7.7	36.2 ± 8.3	0.660
Height (cm)	177.5 ± 7.8	175.9 ± 8.0	0.525
Body mass (kg)	71.8 ± 10.5	72.7 ± 8.7	0.763
BMI (kg·m^{-2})	22.7 ± 2.4	23.4 ± 1.5	0.251
Physical activity (MET·min·wk^{-1})	4601 ± 1983	3903 ± 2105	0.275
Gender [females/males]	3/18	3/18	–
VISA-A (24 = severe Achilles tendinopathy; 100 = healthy)	99.8 ± 0.5	65.6 ± 15.2[a]	<0.001

Participants were paired-matched by type and volume of physical activity, age, height, and body mass.
[a]Significant difference between groups.
BMI, body mass index.

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muscle fascicle length, tendon length, and tendon thickness. Second, participants underwent six passive plantarflexion motions during which the displacement of each myotendinous junction was measured using B-mode ultrasound. Third, after a standardized familiarization with the plantarflexion task and a standardized warm-up, the participants performed four maximal isometric voluntary ankle plantarflexion tasks (MVC) for 3 s each, with 120 s rest in between. Then, submaximal isometric tasks involved matching target torques of 20% and 40% of MVC. Six 8-s trials were performed at both intensities, leading to 12 submaximal contractions with at least 20 s of rest in between. Feedback of the target and torque output was provided using visual feedback displayed on a monitor. Both muscle activation and myotendinous junction displacement were measured during these submaximal contractions.

During all of the aforementioned measurements, except the ultrasound estimation of muscle volume (knee at 135° of flexion), the participants were lying prone on an isokinetic ergometer (Con-Trex; CMV AG, Dübendorf, Switzerland) with hip and knee fully extended, and with the ankle angle set at 0° (shank perpendicular to the foot). Their foot was fixed with rigid straps, and an electronic goniometer (Penny and Giles twin-axis, Biometrics Ltd, UK) was placed along the distal fibula and the lateral part of the fifth metatarsal to measure small changes in ankle angle that inevitably occur during plantarflexion. Participants were asked to mention any feeling of pain throughout the experimentation.

Six months after this laboratory session, participants with Achilles tendinopathy were contacted by email, and asked to complete the VISA-A and IPAQ questionnaires, and they were asked whether they had received treatment for their Achilles tendinopathy. Twenty of the 21 included participants responded at 6.8 ± 0.5 months after data collection. The participants were considered to have recovered if their VISA score had improved by at least 20 points (17).

Muscle Architecture

Muscle volume. Volume of GM, GL, and SOL were estimated using freehand 3-dimensional (D) ultrasound. Specifically, multiple 2D ultrasound images of the muscles were combined with 3D motion of the ultrasound probe to reconstruct the muscle in 3D (Stradwin v5.4; Mechanical Engineering, Cambridge University, UK). B-mode ultrasound images (9.5 cm depth) were recorded using a 40-mm linear probe (2–10 MHz; Aixplorer; Supersonic Imagine, Aix-en-Provence, France). Position and orientation of the probe were recorded by using a six-camera optical motion analysis system (Optitrack, Natural Point, USA) to track a rigid cluster of four markers attached to the probe. For this assessment, participants were prone, with their lower leg in a custom-made water bath, with knee and ankle angle at approximately 135° and 0°, respectively (Fig. 1). Four to six parallel sweeps were performed from the knee to the ankle at a low speed leading to approximately 15 s per sweep. A gating threshold of 5 mm was set in Stradwin, which ensured that B-mode images were recorded every time that the ultrasound probe has moved by 5 mm. The B-mode ultrasound images of GM, GL, and SOL were then segmented manually by two examiners. Volume was estimated for each muscle head using the calculation provided by the Stradwin software (Fig. 1).

Muscle fascicle length and pennation angle. The ultrasound scanner coupled with a 50-mm linear probe (4–15 MHz; Aixplorer, Supersonic Imagine, Aix-en-Provence, France) was used in panoramic mode to estimate the fascicle length and the pennation angle of GM and GL, as further detailed elsewhere (14). Three images were recorded for each muscle. As SOL has short muscle fascicles, conventional B-mode ultrasound images were used for this muscle. The ultrasound probe was placed on SOL medially and laterally, below the GM and GL myotendinous junctions, respectively.

Ultrasound images were analyzed using MATLAB (The Mathworks, Natick, MA), where fascicles and aponeuroses were visually identified and tracked. Fascicles were drawn over the path of fascicle fragments or in the same directions of surrounding fragments. We aimed to measure the length and pennation angle of three fascicles per image (one each from the proximal, mid and distal regions), resulting in up to nine fascicles for each muscle. As some fascicles exhibited small curvatures, we used segmented lines. The pennation angle was considered as the average of the acute angles between the fascicle, and both the superficial and deep aponeurosis, respectively. For each muscle, values from the measured fascicles were averaged to get a representative fascicle length and pennation angle of the entire muscle. After visual inspection for image quality, 41 images out of the 504 images (3 trials-4 locations × 42 participants) were excluded from the analysis because the fascicles were not adequately within the plane of the image. This resulted in an average number of 8.6 ± 1.5 (GM), 8.0 ± 1.6 (GL), and 15.9 ± 1.6 (SOL) fascicles per participant, with a minimum of three.

Muscle physiological cross-sectional area. Muscle PCSA was estimated as follows:

$$\mathrm{PCSA} = \frac{\text{muscle volume}}{\text{fascicle length}} \quad [1]$$

with muscle volume in cubic centimeters, fascicle length in centimeters, PCSA in square centimeters.

The ratios of PCSA were calculated as follows:

$$\frac{\text{muscle } (i)}{\text{triceps surae (TS)}}(\%) = \frac{\text{PCSA muscle } (i)}{(\text{PCSA GM} + \text{GL} + \text{SOL})} \times 100 \quad [2]$$

The ratio of GL PCSA over the sum of GL and GM PCSA (GL/Gas) was also considered.

Anatomical Tendon Features

To measure the thickness of the tendon, the linear ultrasound probe (4–15 MHz; Aixplorer; Supersonic Imagine, Aix-en-Provence, France) was aligned with tendon fascicles in the longitudinal plane and positioned such that both the anterior and the posterior boundaries of the Achilles tendon were visible. The probe was placed 1 cm proximally to the calcaneal attachment.

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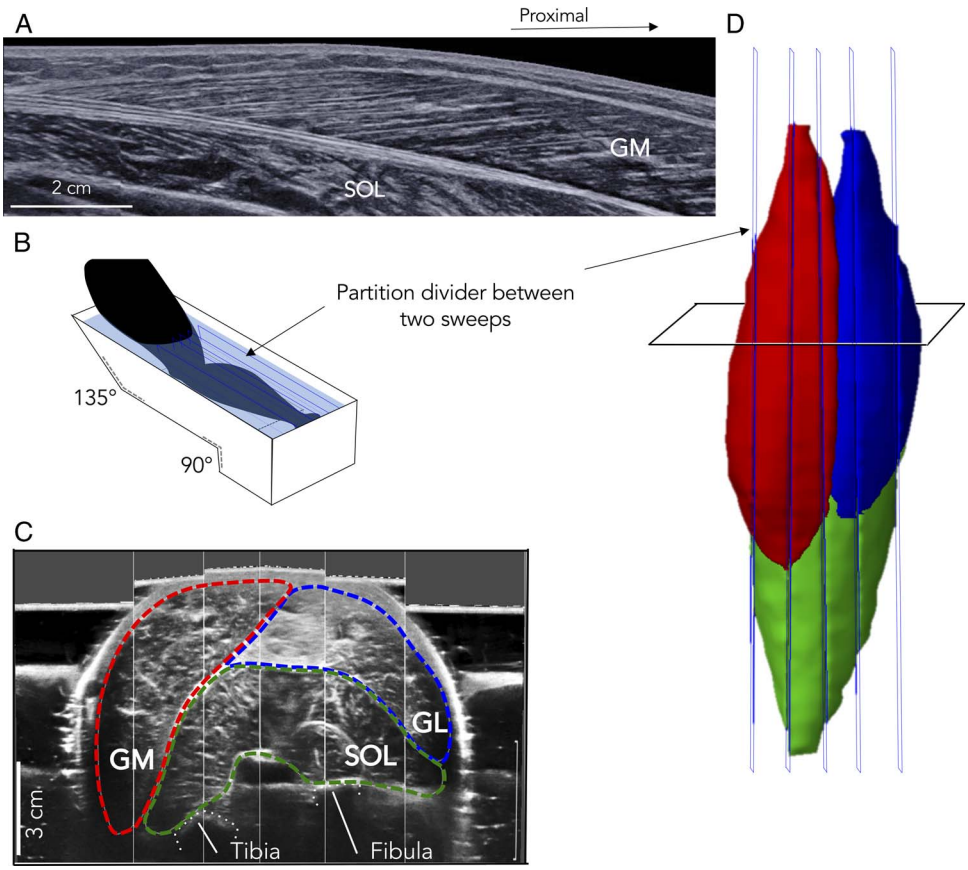

FIGURE 1—Muscle architectural measurements. The fascicle length was assessed using panoramic ultrasound images (panel A). An example of the GM muscle is shown on this figure. Three-dimension reconstruction of TS was realized with the freehand 3D ultrasound technique. Participants were prone, with their lower leg in a custom-made water bath, with knee and ankle angle at approximately 135° and 0°, respectively (panel B). Each muscle was segmented manually from the axial slices (panel C). The axial slice depicted in this figure is from ~33% of the proximodistal part of the leg. The 3D reconstruction of the segmented GM (*red*), GL (*blue*), and SOL (*green*) is shown on panel D.

The thickness of the tendon was calculated as the maximum perpendicular distance between the anterior and the posterior tendon boundary over the free tendon.

The length of each subtendon was estimated by measuring the distance between the insertion of the Achilles tendon on proximal part of the calcaneus and the distal myotendinous junction of each muscle as identified using B-mode ultrasound.

Muscle Activation

Maximal voluntary activation level. It was very important for each participant to produce a true maximal contraction during their MVC trials because the amplitude of the EMG signal recorded during submaximal contractions was normalized to that measured during MVC. To determine if the plantarflexors were activated maximally, a constant current stimulator (DS7AH; Digitimer, UK) delivered a doublet electrical stimulus (interstimulus interval: 10 ms; duration: 1 ms; amplitude: 400 V) to the tibial nerve during two of the four MVCs. For this stimulation, a self-adhesive cathode (50 mm diameter) was placed on the skin that lay directly over the tibial nerve, in the popliteal fossa, and the anode (80 × 130 mm) was placed on the skin over the *anterior tibialis* tuberosity. While the participants were at rest, the output current was increased incrementally (from 10 mA, with incremental steps of 10 mA) until a maximum plantarflexion torque was reached despite an increase in current intensity. We used 120% of this intensity for the rest of the protocol (mean intensity used: 103.4 ± 36.9 mA). The supramaximal doublet stimulus was delivered during the plateau of the MVC, and within 5 s in the subsequent rest period to elicit superimposed and resting twitches, respectively. Participants were informed about the electrical stimulation prior to these contractions.

Torque signals were sampled at 1000 Hz (Labchart V8; ADInstruments, Dunedin, New Zealand) and low-pass filtered at 10 Hz using a second-order Butterworth filter. Then, MVC torque was calculated from the maximal contractions as the maximal torque over a 300-ms time window. Note that for the trials with twitch interpolation, only the torque signal before the superimposed twitch was considered. The percentage of voluntary activation was calculated as previously described by Place et al. (18).

Of the 45 participants who were recruited, one participant with Achilles tendinopathy was unable to reach a maximal voluntary activation level >50%. This participant's data were not considered, and no paired control was recruited. The

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demographic information for this participant is not included in Table 1.

Surface electromyography. Myoelectrical activity was collected via surface EMG from GM, GL, and SOL. First, the skin was shaved and cleaned with alcohol and wireless surface electrodes (Trigno Flex; Delsys, Boston, MA) were attached to the skin with double-sided tape. For each *gastrocnemii*, the electrode was placed on the middle line of the muscle belly at its two third distal. For SOL, two electrodes were placed below GM and GL myotendinous junctions, to record the two portions of the posterior part of SOL. As there was no difference in normalized EMG amplitude between these two regions, data from these two electrodes were averaged. Electrode location was checked with B-mode ultrasound to ensure that they were positioned away from the borders of the neighboring muscles and aligned with the direction of the fascicles. The EMG signals were band-pass filtered (10–850 Hz) and digitized at a sampling rate of 2000 Hz (Trigno; Delsys), and they were recorded on the same acquisition system that was used for the torque signal (Labchart V8; ADInstruments).

Raw EMG signals were inspected for electrical noise and movement artifacts. Because of noise or artifacts, 24 trials (11 MVC and 13 submaximal tasks) among the 672 trials (16 trials [4 MVC +12 submaximal trials] × 42 participants) were excluded. Because all MVC trials of a participant with Achilles tendinopathy had to be excluded, this participant and their matched control were excluded from all analyses. For the MVC trials, the root mean square of the EMG signal (RMS EMG) was calculated over a moving time window of 300 ms with 99% overlap. The resulting highest value over the four contractions was considered as the maximal RMS EMG value for further analysis. For the submaximal isometric force-matched tasks at 20% and 40% of MVC, the RMS EMG was calculated over 5 s at the middle of the force plateau and normalized to that determined during the maximal isometric contractions. As for PCSA, the following ratios of normalized RMS EMG were calculated: GL/Gas, GM/TS, GL/TS, and SOL/TS (equation 2).

Index of Individual Muscle Force

We considered that the difference of force produced by synergist muscles during isometric plantarflexion tasks depends mainly on their difference in activation and PCSA. To consider the component of the force acting in the line of action of the muscle, we also considered the pennation angle of the fascicles. An index of force was, therefore, calculated as follows (14):

$$\text{index of force} = \text{PCSA} \times \cos(\text{pennation angle}) \times \text{normalized RMS EMG} \quad [3]$$

Where normalized RMS EMG is expressed as a percentage of maximal RMS EMG, PCSA in square centimeters and pennation angle in degrees. As was done for EMG and PCSA, the following force ratios were calculated: GL/Gas, GM/TS, GL/TS, and SOL/TS (equation 2).

Myotendinous Junction Displacement

Displacement of the myotendinous junction of each muscle (GM, GL, or SOL) was measured using a 50-mm US linear probe (4–15 MHz) at a frame rate of 53 Hz. The GM and GL myotendinous junctions were defined as the intersection of the superficial and deep aponeurosis, forming an easily identifiable acute angle (Fig. 2). For SOL, the myotendinous junction was defined as the most distal point at which muscle fibers were inserted onto the Achilles tendon (free tendon), as identified within the curved shape of the distal SOL (Fig. 2). To account for possible movement of the ultrasound probe during acquisitions, a thin piece of tape was attached to the skin to represent a fixed reference landmark on the B-mode images (Fig. 2).

The displacements of the myotendinous junction of each muscle were measured during the submaximal isometric contractions at each target torque, that is, 20% and 40% of MVC. Note that a small joint rotation (heel lift) inevitably occurs during isometric plantarflexion tasks, which result in an overestimation of the myotendinous junction displacement, as the muscle–tendon unit logically decreases in length. To account for the effect of this joint rotation, the ankle angle was measured and a correction factor was calculated for every participant. To this end, each participant first underwent passive ankle rotations from 2° of dorsiflexion to 15° of plantarflexion at $1°\cdot s^{-1}$. During these ankle rotations, ankle angle and myotendinous junction displacements were measured using the electrogoniometer and B-mode ultrasound, respectively. Two trials were conducted for each muscle in a randomized order, resulting in six passive trials. EMG was used to check for the absence of muscle activation during the passive plantarflexion motion. In cases in which muscle activity was detected visually, the trials were repeated.

The longitudinal displacement of the myotendinous junction was tracked manually using a custom-made Matlab script on every five frames. First, the relationship between ankle angle and myotendinous displacement assessed during passive motions was fitted with a third-order polynomial fit. When the two ultrasound videos used for tracking a myotendinous junction were judged to be of high quality, they were both kept and results were averaged (73.4% of the cases). If both ultrasound videos were not judged to be of high quality, only the video with the highest quality was kept (22.8% of the cases). For the remaining 3.8% of the cases, both trials were excluded. Therefore, data are reported for 19 pairs (38 participants). To correct for the displacement of the myotendinous junction due to the ankle rotation that occurred during isometric contractions, the displacement associated with this change in ankle angle was first identified from the relationship between ankle angle and myotendinous junction displacement assessed during passive motion, and then subtracted from the raw displacement. Only the corrected values are reported herein. For each subtendon (GM, GL, and SOL) and each level of submaximal contraction (20% and 40% of MVC), tendon strain was calculated by dividing the corrected displacement of the myotendinous junction by the resting length of the subtendon.

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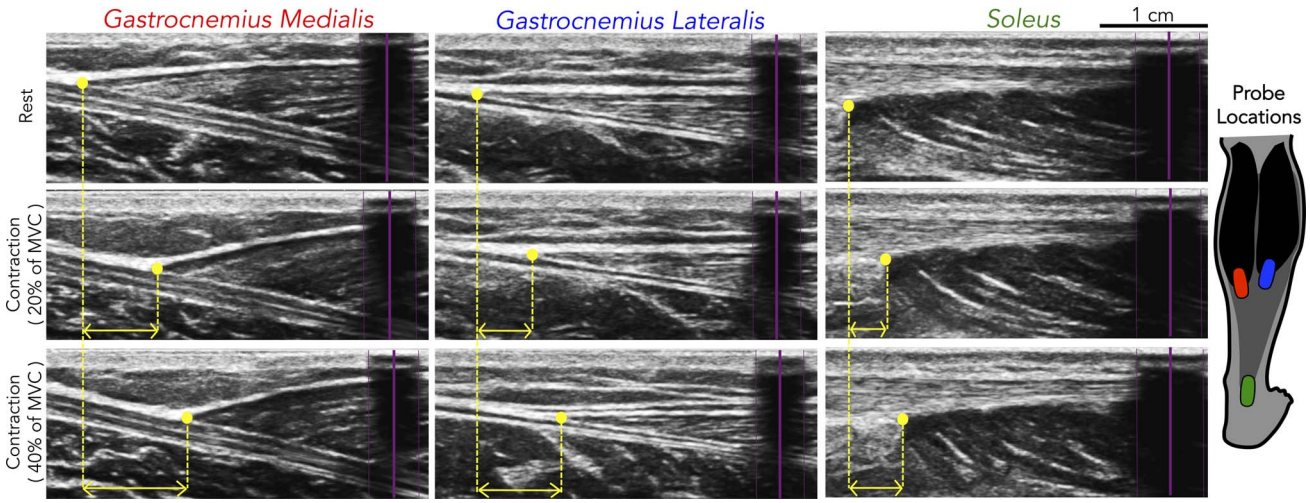

FIGURE 2—Typical example of ultrasound images used for the tracking of the myotendinous junction. Ultrasound images are depicted for the relaxed state (top images) and the two contraction intensities (20% and 40% of MVC). The myotendinous junction is shown by the yellow dot. Each myotendinous junction was tracked manually using a custom-made program. A hypoechoic tape was used as a reference landmark, and it is visible as the *large black strip* (highlighted in purple, within each of the ultrasound images). The *double yellow arrow* represents the displacement of the myotendinous junction of each muscle. The location of the ultrasound probe placement relative to the calf is shown in the right image.

Statistics

A skewness and kurtosis test was used to test for a normal distribution (Stata v12.0; StataCorp LP, Lakeway). If distributions did not pass the normality test, data were transformed as recommended by the software ("ladder" option). Specifically, all of the indices of individual muscle force were transformed using a square root function. The GL/Gas EMG and force ratios and the SOL/TS force ratio were squared. To test the interrater reliability of the volume measurement, muscle segmentation was performed by a second examiner and data were compared using the intraclass correlation coefficient (ICC) and the SEM. All data are reported as mean ± standard deviation.

Statistical analyses were performed in Statistica v7.0 (Statsoft, Tulsa, OK). Tendon thickness, demographic, and injury characteristics were compared between groups using unpaired *t* tests. Subtendon length, muscle volume, muscle fascicle length, pennation angle, and muscle PCSA were compared between groups using a two-way repeated-measures ANOVA (within subject factor: muscle [GM, GL and SOL]; between-subject factor: group [control and Achilles tendinopathy]). Each ratio of PCSA (GL/Gas, GM/TS, GL/TS, and SOL/TS) was compared between groups using unpaired t tests. To test our main hypothesis, each ratio of activation and index of force was compared between groups with a two-way repeated-measures ANOVA (within subject factor: intensity [20% and 40% of MVC]; between-subject factor: group [control and Achilles tendinopathy]). To determine whether people with Achilles tendinopathy exhibited different muscle activation, muscle force and subtendon strain than the controls, we used a three-way repeated-measures ANOVA (within subject factors: muscle or subtendon [GM, GL, and SOL] and intensity [20% and 40% of MVC]; between-subject factor: group [control and Achilles tendinopathy]). When appropriate, *post hoc* analyses were performed using Fisher's LSD test. To test the association between recovery in people with Achilles tendinopathy at 6 months and particular force-sharing strategies, we compared the force ratios between people who recovered and people who did not recover using an unpaired *t*-test. We tested the relationship between ratios, duration of symptoms, and severity of the pathology (VISA-A score) using Pearson's correlation.

The level of significance was set at $P \leq 0.05$. The Cohen's *d* values (the standard deviation of the control group was used as the standardizer) were calculated for the primary outcomes and reported as measures of effect size, with 0.20, 0.50, and 0.80 as small, medium, and large effects, respectively.

RESULTS

Anatomical Tendon Features

The thickness of the Achilles tendon was greater in participants with Achilles tendinopathy (6.7 ± 1.4 mm) than in the controls (5.7 ± 1.2 mm; $P = 0.018$, $d = 0.81$). When considering the length of each subtendon, there was a main effect of muscle ($P < 0.001$, Table 2). Specifically, SOL subtendon (5.5 ± 1.8 cm) was shorter than both GM subtendon (18.9 ± 2.3 cm; $P < 0.001$, $d = 2.08$) and GL subtendon (21.0 ± 1.9 cm; $P < 0.001$, $d = 2.07$). In addition, the GM subtendon was shorter than GL subtendon ($P < 0.001$, $d = 0.80$). On this measure, there was neither a main effect of group ($P = 0.564$) nor an interaction between muscle and group ($P = 0.603$).

Muscle Architecture

The ICC values calculated for GM, GL, and SOL volume were excellent (all ICC > 0.94). The SEM values were lower than 14.7 cm^3 (SOL). For further analysis, only values from the most experienced operator have been considered. There was a main effect of muscle on volume ($P < 0.001$, Table 2), with SOL being larger than both GM ($P < 0.001$, $d = 1.72$) and GL ($P < 0.001$, $d = 1.83$). Gastrocnemius medialis was also

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TABLE 2. Muscle and tendon measurements.

		Controls			Achilles Tendinopathy		
		GM	GL	SOL	GM	GL	SOL
Muscle architecture (n = 21 and 21)							
Volume (cm^3)		244.2 ± 41.9	154.6 ± 32.6	451.0 ± 57.7	232.8 ± 38.5	145.4 ± 28.9	424.9 ± 71.9
Fascicle length (cm)		5.9 ± 0.7	6.7 ± 0.8	4.0 ± 0.7	5.5 ± 0.7	6.6 ± 0.9	3.4 ± 0.5
Pennation angle (°)		17.7 ± 1.6	13.4 ± 2.3	22.6 ± 3.7	18.4 ± 2.1	13.2 ± 2.0	24.8 ± 4.2
PCSA (cm^2)		41.8 ± 6.6	23.2 ± 5.0	115.4 ± 24.1	42.5 ± 5.8	22.1 ± 4.1	124.5 ± 21.4
Muscle activation (n = 21 and 21)							
	% MVC						
RMS EMG (% MVC)	20	13.5 ± 5.8	8.0 ± 4.1	16.6 ± 4.8	16.9 ± 6.1	**6.0 ± 3.3**[a]	17.4 ± 6.3
	40	27.1 ± 5.2	23.3 ± 8.6	25.7 ± 7.3	29.1 ± 6.7	**18.7 ± 8.5**[a]	30.5 ± 12.1
Muscle force (n = 21 and 21)							
Index of force (au)	20	5.4 ± 2.6	1.9 ± 1.3	17.6 ± 6.3	6.9 ± 2.9	1.3 ± 0.8	19.7 ± 8.3
	40	10.7 ± 2.5	5.3 ± 2.5	27.3 ± 10.2	11.8 ± 3.3	4.1 ± 2.2	34.7 ± 15.5[b]
Subtendon characteristics (n = 19 and 19)							
Length (cm)		18.5 ± 2.4	20.7 ± 2.0	5.5 ± 2.1	19.2 ± 2.2	21.3 ± 1.8	5.5 ± 1.6
MTJ displacement (cm)	20	0.54 ± 0.21	0.43 ± 0.12	0.46 ± 0.16	0.56 ± 0.19	0.46 ± 0.18	0.42 ± 0.17
	40	0.92 ± 0.29	0.68 ± 0.26	0.61 ± 0.28	0.90 ± 0.29	0.74 ± 0.23	0.62 ± 0.23
Strain (%)	20	2.9 ± 1.0	2.1 ± 0.7	9.1 ± 3.5	2.9 ± 1.0	2.2 ± 0.8	8.3 ± 4.0
	40	5.0 ± 2.0	3.3 ± 1.3	11.9 ± 5.7	4.7 ± 1.5	3.5 ± 1.1	12.4 ± 6.3

Data are reported for the Control and Achilles tendinopathy groups.
Note that significant interactions are not shown within the table.
[a](bold cells) Significant main effect of group.
[b]Significant difference between groups. Note that significant interactions are not shown within the table.

larger than GL ($P < 0.001$, $d = 1.51$). However, there was neither a significant interaction between muscle and group ($P = 0.353$) nor a main effect of group ($P = 0.230$) on this measure.

A significant main effect of muscle was observed for both fascicle length and pennation angle (both $P < 0.001$; Table 2). Soleus exhibited shorter fascicles and greater pennation angle than both GM (both $P < 0.001$, both $d > 1.52$) and GL (both $P < 0.001$, both $d > 1.88$). Gastrocnemius medialis had shorter fascicle length and greater pennation angle than GL (both $P < 0.001$, both $d > 1.16$). There was no significant main effect of group for fascicle length ($P = 0.059$) or pennation angle ($P = 0.172$). In addition, there was no significant interaction between muscle and group ($P = 0.118$ for fascicle length or $P = 0.051$ for pennation angle). Of note, the P value for the pennation angle was close to being significant.

When considering muscle PCSA (Table 2), there was a main effect of muscle ($P < 0.001$). Specifically, SOL PCSA was larger than both GM ($P < 0.001$, $d = 1.89$) and GL PCSA ($P < 0.001$, $d = 1.96$). The GM PCSA was also larger than GL PCSA ($P < 0.001$, $d = 1.76$). There was neither a main effect of group ($P = 0.352$) nor a significant interaction between muscle and group ($P = 0.120$). When considering the whole TS, that is, the sum of GM, GL, and SOL PCSA, there was no significant difference between groups (180.5 ± 32.2 and 189.1 ± 26.9 cm^2 for the Control and Achilles tendinopathy groups, respectively; $P = 0.352$).

When considering the ratios of PCSA, a between-group difference was observed for GL/TS, which was approximately 8.5% lower in participants with Achilles tendinopathy than controls ($P = 0.047$, $d = 0.75$) (Table 3). No difference was observed between groups for GM/TS PCSA ($P = 0.292$, $d = 0.29$) and SOL/TS PCSA ($P = 0.071$, $d = 0.54$).

Muscle Activation

Voluntary activation level. None of the patients reported pain in the Achilles tendon region during data collection. The maximal isometric plantarflexion torque was not different between groups (controls, 143.5 ± 24.7 N·m and Achilles tendinopathy, 136.1 ± 31.5 N·m; $P = 0.403$, $d = 0.30$). Participants reached a voluntary activation level close to 100%, with no difference between groups (controls, 97.4% ± 6.7% and Achilles tendinopathy, 97.9% ± 4.8%; $P = 0.798$, $d = 0.07$). Thus, we considered that the maximal RMS EMG amplitude measured during the MVC tasks represented the maximal activation for all muscles, in all participants.

Submaximal isometric contractions. When considering the normalized EMG RMS values, we observed a significant main effect of muscle ($P < 0.001$, Table 2), a significant main effect of intensity ($P < 0.001$) and an interaction between muscle and group ($P = 0.049$). However, there was no significant interaction between muscle, intensity, and group ($P = 0.067$). For

TABLE 3. Ratios of PCSA, muscle activation, and indexes of force.

		Controls				Achilles Tendinopathy			
	% MVC	GL/Gas	GM/TS	GL/TS	SOL/TS	GL/Gas	GM/TS	GL/TS	SOL/TS
PCSA (%)		35.6 ± 3.5	23.4 ± 2.9	12.9 ± 1.4	63.7 ± 3.6	34.2 ± 4.4	22.6 ± 2.5	11.8 ± 2.0[b]	65.6 ± 3.2
Activation (%)	20	38.6 ± 17.9	34.9 ± 13.5	20.8 ± 8.6	44.3 ± 13.4	**27.0 ± 14.1**[a]	41.6 ± 13.1	**15.1 ± 8.7**[a]	43.4 ± 14.0
	40	45.2 ± 9.3	36.1 ± 6.7	30.0 ± 7.2	33.8 ± 7.9	**38.0 ± 13.4**[a]	38.4 ± 11.0	**23.4 ± 9.1**[a]	38.2 ± 11.5
Force index (%)	20	27.7 ± 18.6	22.7 ± 10.8	7.5 ± 3.9	69.8 ± 11.7	**17.9 ± 12.5**[a]	26.0 ± 11.6	**5.3 ± 4.0**[a]	68.7 ± 12.8
	40	32.0 ± 9.1	25.9 ± 6.4	12.1 ± 3.9	62.0 ± 8.4	**26.0 ± 12.7**[a]	25.6 ± 10.4	**8.8 ± 5.1**[a]	65.6 ± 12.4

Data are reported for the control and Achilles tendinopathy groups.
Note that significant interactions are not shown within the table.
[a](bold cells) A significant main effect of group.
[b]Significant difference between groups.

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the sake of clarity, we only report below the between-group differences, which are related to the main aims of this study. *Post hoc* showed that regardless the contraction intensity, GL RMS EMG was approximately 22.8% lower in the people with Achilles tendinopathy than controls ($P = 0.043$, $d = 0.33$). There was no between-group difference for GM ($P = 0.094$, $d = 0.31$) and SOL ($P = 0.083$, $d = 0.37$).

Activation ratios were calculated to represent the distribution of activation within TS (Table 3). There was a main effect of intensity on GL/Gas, GL/TS, and SOL/TS EMG ratios (all $P < 0.001$) but this was not the case for GM/TS EMG ratio ($P = 0.585$). There was no significant interaction between intensity and group (all $P > 0.132$); however, we observed a main effect of group for both GL/Gas ($P = 0.011$, $d = 0.76$) and GL/TS EMG ratios ($P = 0.009$, $d = 0.67$). Specifically, GL/Gas and GL/TS EMG ratio were lower in people with Achilles tendinopathy than in the controls (Table 3).

Distribution of Muscle Force

When considering the index of force values, we observed significant main effects of muscle ($P < 0.001$), and intensity ($P < 0.001$), an interaction between muscle and intensity ($P = 0.018$) and between muscle intensity and group ($P = 0.042$). Again, we only report below the between-group differences. At 40% of MVC, SOL index of force was approximately 27.0% higher in people with Achilles tendinopathy than controls ($P = 0.039$, $d = 0.65$). There were no other between-group differences (all P values >0.251).

Force ratios were calculated to represent the distribution of force within TS (Fig. 3; Table 3). There was no interaction between group and intensity for any of the ratios (all $P > 0.095$); however, there was a main effect of group for both GL/Gas ($P = 0.025$, $d = 0.67$) and GL/TS force ratios ($P = 0.023$, $d = 0.62$). Specifically, regardless the intensity, GL/Gas and GL/TS ratios were 11.2% and 28.5% lower, respectively, in people with Achilles tendinopathy compared with the controls. When considering the Achilles tendinopathy group, there were no significant correlations between either GL/Gas or GL/TS force ratios and the duration of symptoms (both r values <0.16, $P > 0.2$). Similarly, no significant correlation was observed between these force ratios and the VISA-A scores (both r values <0.11, $P > 0.2$).

Six months after the collection of the original data, 11 participants reported a clinically significant improvement in their symptoms (VISA-A increased by 33.3 ± 14.9 points) and 9 others did not (VISA-A increase by 4.3 ± 6.2 points). The comparison of the GL/TS force ratio at 20% of MVC of these

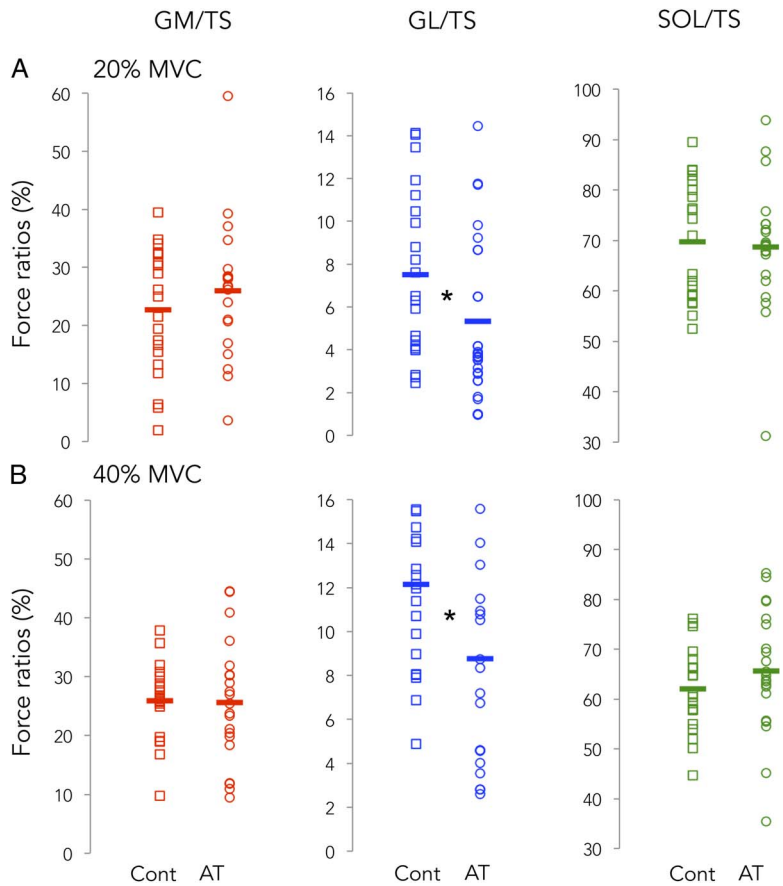

FIGURE 3—Individual and mean data for each muscle force ratio. Data are presented for both the control (*open squares*) and Achilles tendinopathy (*open circles*) groups during 20% of MVC (A) and 40% of MVC (B) contractions. Note the different *y*-axis scales for each panel. Indexes of force are calculated as the product of activation, pennation angle, and physiological cross-sectional area. *Statistical difference between the Control and Achilles tendinopathy group data ($P < 0.05$).

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two subgroups indicated that people who did not recover had a smaller GL/TS force ratio (4.0% ± 2.5%) than people who recovered (7.1% ± 4.6%; $P = 0.087$). Note that the effect size was large ($d = 0.84$), but the difference was not significant. There were no differences for any other force ratio at 20% of MVC with all P values > 0.171. No between-group differences were found when considering the higher contraction intensity, that is, 40% of MVC (all $P > 0.158$).

Myotendinous Junction Displacement and Subtendon Strain

Displacement of each myotendinous junction (GM, GL, and SOL) was measured to estimate the change in each subtendon length during contraction. The ankle joint increased by 2.5° ± 1.5° (i.e., more plantarflexed) and by 5.4° ± 2.0° during the isometric contractions at 20% and 40% of MVC, respectively.

There were main effects of muscle ($P < 0.001$), intensity ($P < 0.001$), and a significant interaction between muscle and intensity ($P < 0.001$) on myotendinous junction displacement (Table 2). Specifically, at both 20% and 40% of MVC, GM myotendinous junction displacement was larger than that of both GL (for both intensities $P < 0.001$; $d = 0.48$ and 0.63 at 20% and 40% of MVC, respectively) and SOL (for both intensities $P < 0.001$; $d = 0.49$ and 0.85 at 20% and 40% of MVC, respectively). Although there was no difference between GL and SOL at 20% of MVC ($P = 0.652$, $d = 0.05$), GL myotendinous displacement was larger than that of SOL at 40% of MVC ($P < 0.001$; $d = 0.30$). However, there was neither a main effect of group ($P = 0.530$) nor an interaction between muscle and group ($P = 0.980$), indicating that the displacement of the myotendinous junctions did not differ between groups.

When considering the myotendinous displacement normalized to resting tendon length (tendon strain), there was a main effect of subtendon ($P < 0.001$), intensity ($P < 0.001$), and a significant interaction between intensity and subtendon ($P < 0.001$). Specifically, at 20% of MVC, the strain of both GM and GL subtendons was smaller than that of SOL subtendon (both $P < 0.001$ and both $d > 1.46$), with no difference between GM and GL ($P = 0.165$, $d = 0.72$). At 40% of MVC, both GM and GL subtendons strain was less than that of SOL subtendon (both $P < 0.001$ and $d > 1.37$), but GM subtendon had a higher strain than GL subtendon ($P < 0.001$; $d = 0.74$). There was neither a main effect of group ($P = 0.835$) nor an interaction between subtendon and group ($P = 0.746$).

DISCUSSION

The aim of this study was to determine whether the force distribution between the heads of the TS (muscle coordination) differs in people with Achilles tendinopathy compared with controls. We observed a different force distribution between the two populations with the GL muscle contributing significantly less to the overall submaximal isometric plantarflexion force in people with Achilles tendinopathy compared with the controls. This difference is explained by both a lower relative GL PCSA and GL activation in people with Achilles tendinopathy. Despite this altered force distribution in people with Achilles tendinopathy, the displacement of the myotendinous junctions did not differ between the populations during the submaximal isometric contractions. This is the first study to identify a difference in force distribution between muscles of the TS in people with Achilles tendinopathy compared with controls, which, moreover, might be associated with the potential for recovery. Additional work is needed to understand the role of this altered coordination strategy in the development or persistence of Achilles tendinopathy.

Muscle force-generating capacities. The maximal plantarflexion torque measured during the maximal contractions was not different between our two groups, which aligns with some (19,20) but not all previous studies (21,22). To the best of our knowledge, the current study is the first to use the twitch interpolation technique to verify that the voluntary activation was maximal in both participants with Achilles tendinopathy and controls. The inability to reach maximal voluntary contraction (as observed in one excluded participant with Achilles tendinopathy) might explain why some studies observed a lower maximal plantarflexion torque in people with Achilles tendinopathy. Another explanation for these discrepancies might be the characteristics of the task with most previous studies reporting a lower overall maximal force in people with Achilles tendinopathy for data collected during dynamic tasks (which contrasts with our isometric plantarflexion tasks). O'Neillet al. (21) reported a lower peak torque in people with Achilles tendinopathy than controls during dynamic maximal tasks performed at various knee positions. As large between-group differences were observed regardless of the position of the knee, they concluded that the monoarticular SOL had the greatest impact on the lower torques observed.

In our study, we considered the difference in PCSA as being representative of the difference in force-generating capacity between muscles. When considering the ratio of PCSA, we observed no between-group differences for GM/TS and SOL/TS PCSA but a significantly 8.5% lower GL/TS PCSA in people with Achilles tendinopathy. To the best of our knowledge, our study is the first to estimate PCSA of each head of the TS in people with Achilles tendinopathy. One study compared muscle volume between the affected and nonaffected limbs in people with Achilles tendinopathy (23) and reported differences, which aligned with our findings. Specifically, they reported a 14% smaller GL volume in the affected limb compared with the nonaffected limb.

Muscle activation. The between-day reliability of the distribution of normalized EMG amplitude among the heads of the TS has been demonstrated in a previous study (24), suggesting that it represents a robust individual strategy. Previous studies have estimated the activation of the TS muscles in people with Achilles tendinopathy during walking (25), running (26), isometric plantarflexion tasks (22), and dynamic plantarflexion tasks (27,28). However, these studies used different normalization procedures and reported different activation parameters

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(e.g., onset/offset timings and EMG amplitude), which makes it difficult to directly compare their results with our results. Using a protocol similar to the protocol used in the present study, Masood and colleagues (22) observed greater EMG amplitude of the SOL in the symptomatic legs of people with Achilles tendinopathy compared with healthy controls. Even though we reported no significant difference in SOL EMG amplitude between the two groups, we observed a 27.0% higher SOL force in people with Achilles tendinopathy than controls at 40% of MVC. This is likely explained by both a larger, albeit nonsignificant, SOL activation and SOL PCSA in participants with Achilles tendinopathy than controls (Table 2). Most notably, we observed both a lower normalized GL EMG amplitude and a lower GL/TS activation ratio providing evidence of a lower GL contribution to the overall activation in people with Achilles tendinopathy. Even though the amplitude of the difference was only moderate (effect size: $d = 0.67$), it is consistent with the smaller relative GL/TS PCSA, which strengthens the likelihood that these outcomes are relevant.

Force-sharing strategies. Even though the distribution of force among the heads of the TS has been regarded as a potential contributor to Achilles tendon problems (9,11,21), this has never been investigated experimentally. This may be because there is no noninvasive technique to measure individual muscle force, leading to indirect approaches that rely on either experimental measures, biomechanical models, or both. As proposed in previous works (12–14), we considered that the difference of force produced between synergist muscles during isometric contractions is mainly due to their difference in muscle activation and PCSA. Therefore, we assumed that the three heads of the TS operated at a similar relative length of their optimal length, which seems reasonable considering the similar anatomy and function of GM and GL and previous works suggesting similar optimal angles for GM (19° of dorsiflexion (29)] and SOL [15° of dorsiflexion (30)]). We also assumed that specific tension would not impact the outcome because tasks were conducted at submaximal intensities, for which mainly slow twitch fibers are likely recruited for the three muscles in both groups.

Using the aforementioned approach, we observed a lower contribution of GL force to overall TS force, regardless the intensity of the contraction. We contend that this provides strong evidence that people with Achilles tendinopathy use a different force-sharing strategy during this simple isometric plantarflexion task. Even though we did not study higher contraction intensities, it is very likely that such differences would be also observed at higher force levels. This is because as intensity increases, the difference between individual muscles relies more on PCSA than activation differences as activation of all the muscles logically converge toward 100% (14).

Impact of altered force sharing strategies on Achilles tendon mechanical behavior. To provide a better understanding of the mechanical effect of the force sharing strategies on the Achilles tendon behavior, we assessed the displacement of each of the three myotendinous junctions using ultrasound imaging. Of note, inevitable ankle rotation was observed during the submaximal isometric plantarflexion tasks. As detailed in Materials and Methods, we applied a correction factor to account for this rotation. As the correction factors were not different between groups, it is unlikely that this ankle rotation influenced our main outcomes.

The tendon strain measured in the control group at 40% of MVC for GM (5.0% ± 2.0%) was either higher [2.2%, Finni et al. (31)] or similar [4.5%, Wolfram (32)] to values reported in previous studies for the same muscle at close contraction intensity. It is noteworthy that only a few studies have investigated both GM and GL, and those that have done so have reported either a similar strain (33,34) or a larger strain for GM than GL (32). Consistent with Wolfram (32), we report a larger strain for GM than GL. This difference observed in both controls and participants with Achilles tendinopathy might be explained by the combination of both a shorter GM tendon length and a larger GM force index compared with GL.

Despite our evidence that the contribution of GL to the overall TS force was lower in participants with Achilles tendinopathy, we did not observe any between-group difference in the myotendinous junction displacement or the tendon strain for any subtendon. Three reasons may explain this result. First, even though a degree of independence between the subtendons has been suggested (35), it is likely that the transmission of lateral force occurs between the subtendons (36). A different degree of force transmission between the populations may obscure the differences in force distribution. Second, the strain experienced by a subtendon depends on both muscle force and its mechanical properties. As tendons remodel over time and adapt to the forces they are subjected to, it is possible that the mechanical properties of the GL subtendon changed as a result of long-term lower contribution of the GL muscle to the TS force in our Achilles tendinopathy group. This proposition is consistent with an emerging body of literature that suggests lower stiffness of the Achilles tendon in people with Achilles tendinopathy (37). However, these studies used elastography, which cannot distinguish between the Achilles subtendons. Therefore, we do not know whether the lower stiffness that has been observed is specific to the GL subtendon or generalized to all of the subtendons. Third, it is possible that the contraction intensities (20% and 40% of MVC) were too low to identify between-group differences. As mentioned above, large force differences between muscles are expected at higher intensities when these differences are closely related to the differences in PCSA. Thus, it is likely that different muscle force distribution between populations would lead to different Achilles tendon behavior at higher levels of contraction. It is consistent with previous studies that reported a larger GM subtendon strain in people with Achilles tendinopathy during maximal contractions (19).

Clinical relevance. The lower contribution of GL to the overall TS force observed in people with Achilles tendinopathy is supported by a lower contribution of both PCSA and activation. As muscle PCSA and activation are estimated using different techniques, we are confident that the observed differences

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are biomechanically relevant. However, our experimental design does not allow us to determine whether altered force-sharing strategies are the cause or the consequence of the pathology. Overall, the clinical relevance of this difference must be further explored.

It is important to note that the effect size of the between-group differences in GL/TS force is only moderate. This can be explained by the interindividual variability that exists, especially among people with Achilles tendinopathy, with the GL/TS force ratio ranging from 2.6% to 20.2% at 40% of MVC (Fig. 3). If we consider altered force-sharing as a cause of Achilles tendinopathy, this high interindividual variability might be explained by the well-known multifactorial etiology of Achilles tendinopathy, with altered force-sharing being present only in a subgroup of individuals. An alternative explanation is that altered force-sharing is a consequence of Achilles tendinopathy, in which case, we may expect a correlation between the duration of the symptom and the GL/TS force ratio. However, such a correlation was not found (r = 0.17 and −0.05 at 20% and 40% of MVC, respectively).

Interestingly, a previous study has provided evidence that eccentric training can change activation ratios within the TS group in healthy people (34). In addition, Masood and coworkers (38) observed a significant increase in GL activation during isometric plantarflexion tasks in people with Achilles tendinopathy after a 12-wk training program. Of note, no change in either GM or SOL activation was observed with training. As their participants had significant relief from their symptoms after the rehabilitation program, this result does provide support for a relationship between the contribution of the GL muscle to plantarflexion force and Achilles tendinopathy symptoms. This potentially echoes the observation we made that people whose symptoms improved over a 6-month period, tended to have higher GL/TS force ratio at the time of the original testing. However, it should be confirmed on a larger sample size. Of note, in our study, an improvement in the symptoms was neither associated with a change in physical activity level (as assessed by comparing IPAQ before the study and 6 months later), nor associated with a healthcare intervention.

Because the estimation of muscle force during a dynamic task relies on indirect information, such as the force length and force velocity relationship, we chose to study a more controlled task. Then, it remains unclear if the lower contribution of the GL force would also be observed during a dynamic task.

CONCLUSIONS

We observed different muscle coordination strategies between the two populations, with the GL muscle in people with Achilles tendinopathy contributing significantly less to the overall submaximal isometric TS force than for controls. Further investigations are needed to unravel the biomechanical consequences of altered muscle coordination strategies on the Achilles tendon and to determine whether altered coordination is the cause or consequence of the pathology. Consideration of muscle coordination during dynamic tasks would provide further insights, but it would require the use of a different approach that combines experimental data with musculoskeletal modeling.

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The results of the study are presented clearly, honestly, and without fabrication, falsification, or inappropriate data manipulation. The results of the present study do not constitute endorsement by ACSM.

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