



Effects of stroke injury on the shear modulus of the lower leg muscle during passive dorsiflexion

Guillaume Le Sant, Antoine Nordez, François Hug, Ricardo Andrade, Thomas Lecharte, Peter J McNair, Raphaël Gross

► To cite this version:

Guillaume Le Sant, Antoine Nordez, François Hug, Ricardo Andrade, Thomas Lecharte, et al.. Effects of stroke injury on the shear modulus of the lower leg muscle during passive dorsiflexion. *Journal of Applied Physiology*, 2018, 126 (1), pp.11-22. 10.1152/jappphysiol.00968.2017 . hal-04191405

HAL Id: hal-04191405

<https://hal.science/hal-04191405>

Submitted on 10 Jan 2024

HAL is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.

RESEARCH ARTICLE

Effects of stroke injury on the shear modulus of the lower leg muscle during passive dorsiflexion

Guillaume Le Sant,^{1,2} Antoine Nordez,^{1,3}  François Hug,^{1,4,5} Ricardo Andrade,^{1,6} Thomas Lecharte,² Peter J. McNair,³ and Raphaël Gross^{1,7}

¹University of Nantes, Laboratory “Movement, Interactions, Performance” (EA 4334), Faculty of Sport Sciences, Nantes, France; ²School of Physiotherapy, Institut Régional de Formation aux Métiers de Rééducation et de Réadaptation Pays de la Loire, Nantes, France; ³Health and Rehabilitation Research Institute, Faculty of Health and Environmental Sciences, Auckland University of Technology, Auckland, New Zealand; ⁴The University of Queensland, Centre for Clinical Research Excellence in Spinal Pain, Injury and Health, School of Health and Rehabilitation Sciences, Brisbane, Australia; ⁵Institut Universitaire de France, Paris, France; ⁶Universidade de Lisboa, Faculdade de Motricidade Humana, Centro Interdisciplinar de Performance Humana, Lisbon, Portugal; and ⁷Gait Analysis Laboratory, Physical and Rehabilitation Medicine Department, University Hospital of Nantes, Nantes, France

Le Sant G, Nordez A, Hug F, Andrade R, Lecharte T, McNair PJ, Gross R. Effects of stroke injury on the shear modulus of the lower leg muscle during passive dorsiflexion.—Contractures are common complications of a stroke. The spatial location of the increased stiffness among plantar flexors and its variability among survivors remain unknown. This study assessed the mechanical properties of the lower leg muscles in stroke survivors during passive dorsiflexions. Stiffness was estimated through the measurement of the shear modulus. Two experiments were independently conducted, in which participants lay supine: with the knee extended (*experiment 1*, $n = 13$ stroke survivors and $n = 13$ controls), or with the knee flexed at 90° (*experiment 2*, $n = 14$ stroke survivors and $n = 14$ controls). The shear modulus of plantar flexors [gastrocnemius medialis (three locations), gastrocnemius lateralis (three locations), soleus (two locations), flexor digitorum longus, flexor hallucis longus), peroneus longus] and dorsiflexors (tibialis anterior and extensor digitorum longus) was measured using ultrasound shear wave elastography during passive dorsiflexions ($2^\circ/\text{s}$). At the same ankle angle, stroke survivors displayed higher shear modulus than controls for gastrocnemius medialis and gastrocnemius lateralis (knee extended) and soleus (knee flexed). Very low shear modulus was found for the other muscles. The adjustment for muscle slack angle suggested that the increased shear modulus was arising from consequences of contractures. The stiffness distribution between muscles was consistent across participants with the highest shear modulus reported for the most distal regions of gastrocnemius medialis (knee extended) and soleus (knee flexed). These results provide a better appreciation of stiffness locations among plantar flexors of stroke survivors and can provide evidence for the implementation of clinical trials to evaluate targeted interventions applied on these specific muscle regions.

NEW & NOTEWORTHY The shear modulus of 13 muscle regions was assessed in stroke patients using elastography. When compared with controls, shear modulus was increased in the gastrocnemius muscle (GM) when the knee was extended and in the soleus (SOL)

when the knee was flexed. The distal regions of GM and SOL were the most affected. These changes were consistent in all the stroke patients, suggesting that the regions are a potential source of the increase in joint stiffness.

contracture; elastography; muscle; shear modulus; stroke

INTRODUCTION

Stroke injury is a world-leading cause of premature mortality and long-term disabilities (42). For most survivors of a stroke, adaptations in soft tissues arising from nonneurological and neurological pathological processes (20, 21, 38) lead to muscle contracture defined as an increased passive muscle stiffness and a reduction in joint mobility (22, 31). Consequently, contractures potentially influence motor function. For instance, when affecting plantar flexors at the ankle joint, they prevent an appropriate foot position at initial contact during gait. Walking with this abnormal posture (hindfoot to forefoot contact) results in a limited rollover and a restricted ability of the plantar flexor muscles to generate forces at longer muscle lengths during the stance phase of the gait cycle (4).

Clinical and instrumented assessments, including passive joint torque measurements (30, 59), report increased levels of stiffness among stroke survivors. However, such findings cannot provide individual quantification of the stiffness of the numerous muscles that contribute to the passive torque (61). Consequently, the muscle locations affected by increased stiffness remain unknown. Therefore, the clinician still does not know whether some muscles require individualized treatment (28). For instance, it is possible to enhance the effect of stretching on the gastrocnemius medialis muscle by specific positioning of the ankle and subtalar joints (18). Another scenario might be when patients do not respond to conservative interventions, surgical release is an option, but ideally, it would be performed only on a selected part of an aponeurosis that was most affected. Currently, procedures are more “global” (e.g., Vulpis, Strayer, Baumann, Baker, and Green techniques) and may not be targeting the most affected structures/locations.

Address for reprint requests and other correspondence: A. Nordez, Laboratory “Movement, Interactions, Performance” (EA 4334), Faculty of Sport Sciences, Univ. of Nantes, 25 bis, Boulevard Guy Mollet, BP 72206, 44322 Nantes Cedex 3, France (e-mail: antoine.nordez@univ-nantes.fr).

Being more precise during such surgical procedures would potentially limit weakness and reduce the likelihood of complications, such as wound infection (1).

Ultrasound shear wave elastography (SWE) provides a unique opportunity to estimate the mechanical properties of a muscle region (5). Through the estimation of shear wave velocity propagating in a muscle (or through the calculation of a shear modulus, see MATERIALS AND METHODS), SWE has been reported to provide a reliable local quantification of an individual muscle stiffness (16, 46) and the estimation of its slack angle (the angle of the onset of passive tension) (36, 37). Consequently, SWE is a promising tool for the characterization of muscle mechanical properties among stroke survivors.

This study aimed to measure the shear modulus among lower leg muscles in stroke survivors compared with matched controls. For that purpose, passive ankle rotations were performed with the knee extended (*experiment 1*) and with the knee flexed (*experiment 2*). We hypothesized that stroke survivors would exhibit higher muscle stiffness than controls.

MATERIALS AND METHODS

Participants

Twenty-seven patients who had suffered a stroke and 27 age- and sex-matched control subjects volunteered to participate in *experiment 1* (knee extended; $n = 13$) and *experiment 2* (knee flexed; $n = 14$). Patients were recruited through the neurological rehabilitation department of the university hospital (in-patients and out-patients), and controls were recruited through e-mail advertising via a university network. Volunteers were included if they 1) were over 18 yr old and 2) had suffered a cortical or subcortical stroke that affected their ankle mobility. Their demographic information is presented in Table 1. Potential participants were excluded if they had cognitive or speech impediments that affected their ability to follow instructions associated with the protocol; or moderate motor disability quantified as a score <4 on Modified Rankin Handicap Scale (58). Clinically, the spasticity of the triceps surae was assessed by the modified Ashworth Scale (7). The isometric strength in plantar- and dorsiflexion of stroke participants was $\sim 50\%$ of that recorded in the control group (see Table 1), which is similar to the levels reported in the literature (14). The

protocol was approved by the local Institutional Ethics Committee. Participants were informed of the nature of the study before providing written informed consent. The procedures conformed to the Declaration of Helsinki.

Instrumentation

Dynamometer. An isokinetic dynamometer (Biodex 3 Medical, Shirley, New York) was used to passively rotate the ankle of participants, with the knee fully extended (hip at 0° , *experiment 1*) and the knee flexed at 90° [hip at 90° , *experiment 2*] [see Fig. 1 of Le Sant et al. (43)]. The ergometer axis was aligned with the estimated ankle center of rotation (8). The neutral position (i.e., the sole of the foot at right angle to the tibia) was defined as 0° . Ankle angle and joint torque signals were transmitted from the dynamometer to an external 16-bit analog/digital converter (1 kHz; PowerLab ADInstruments, Colorado Springs, CO) and visualized and stored on a computer for later analyses.

Surface electromyography. Surface electromyography (sEMG) signals of gastrocnemius medialis and gastrocnemius lateralis (GM and GL, respectively), soleus (SOL), and tibialis anterior (TA) were simultaneously recorded (1,000 Hz, ME 6000; MEGA Electronics, Kuopio, Finland) using hydrogel adhesive surface electrodes (Kendall 100 foam-series; Covidien, Mansfield, MA). Electrodes were placed according to the SENIAM guidelines (35). Ultrasound imaging ensured that electrodes were placed over the considered muscle. Both sEMG activities and the ankle torque were visualized in real time to ensure that there were no increases in muscle activity during the stretching procedures. If so, the trial was not accepted and repeated. Signals were stored for later off-line analyses.

Elastography. The technique used to measure the shear modulus has been previously described in detail (5, 26). Briefly, SWE relies on the measurement of shear waves velocity that result from mechanical perturbations applied to the tissue. The shear modulus (μ) is directly related to the shear wave velocity (V_s): $\mu = \rho V_s^2$, where μ is the shear modulus of the tissue, and ρ is the density of the tissue (1,000 kg/m³ for muscle).

Shear modulus has been shown to display a strong linear relationship with Young's modulus (R^2 between 0.916 and 0.988), as shown with conventional material testing procedures (16, 40). Thus, the shear wave velocity is directly related to the shear modulus, that is, the stiffer the tissue, the faster the shear wave propagation.

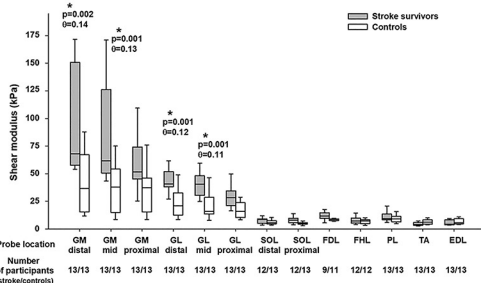
Table 1. Characteristics of stroke survivors and control participants

Characteristics	Experiment 1 (Knee Fully Extended)		Experiment 2 (Knee Flexed)	
	Stroke Survivors ($n = 13$)	Controls ($n = 13$)	Stroke Survivors ($n = 14$)	Controls ($n = 14$)
Sex (male:female)	8:5	8:5	10:4	10:4
Age, yr*	54.3 (12.6)	54.7 (11.9)	58.4 (14.7)	57.1 (14.2)
Height, cm*	170.0 (6.5)	170.7 (8.8)	171.0 (6.1)	173.6 (8.9)
Weight, kg*	69.8 (10.4)	68.8 (10.3)	74.6 (12.5)	73.3 (11.6)
Years after stroke*	1.1 (1.3)	NA	3.4 (3.4)	NA
Ankle dorsiflexion ROM (maximal angle)†	16.6 (8.2)	27.8 (7.2)‡	23.2 (5.4)	37.5 (8.3)‡
Affected side (L:R)	7:6	NA	8:6	NA
Spasticity (yes:no)	13:0	NA	13:1	NA
MAS§ (dorsiflexion, knee extended)	1:4	NA	1:1	NA
	1+:1		1+:2	
	2:2		2:7	
	3:6		3:3	
MAS§ (dorsiflexion, knee flexed)	1:5	NA	1:5	NA
	1+:2		1+:2	
	2:3		2:4	
	3:3		3:2	
Isometric strength in plantarflexion, N·m*	33.4 (21.9)	63.4 (12.0)	26.8 (14.0)	66.0 (32.2)
Isometric strength in dorsiflexion, N·m*	11.2 (11.5)	27.9 (7.7)	13.0 (8.4)	36.8 (12.6)

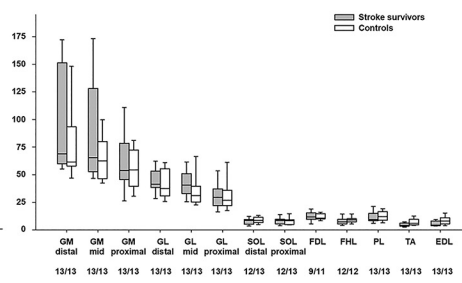
MAS, modified Ashworth scale; ROM, range of motion. *Mean (SD); †median (interquartile range); ‡between-group differences ($P < 0.05$). §1, 1+, 2 and 3 scores refer to the grades of MAS. NA, not applicable.

Experiment 1 (knee fully extended)

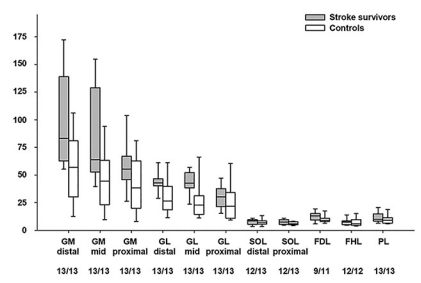
A Maximal common angle in dorsiflexion



B 80% of maximal ROM in dorsiflexion

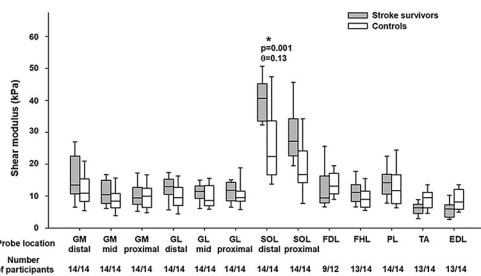


C Maximal common angle in dorsiflexion, from slack angle

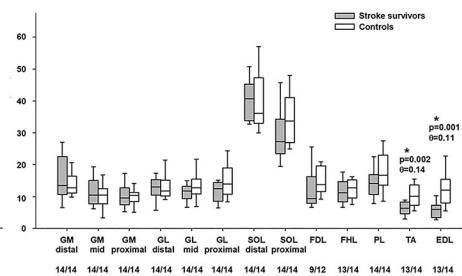


Experiment 2 (knee flexed at 90°)

D Maximal common angle in dorsiflexion



E 80% of maximal ROM in dorsiflexion



F Maximal common angle in dorsiflexion, from slack angle

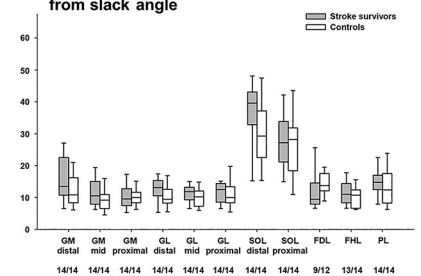


Fig. 1. Box plot of shear modulus values for each transducer location among stroke survivors vs. controls, in both experiments (A to C: knee fully extended; D to F: knee flexed at 90°). Three situations were examined: at the maximal common angle in dorsiflexion displayed among each pair (A and D); at 80% of maximal range of motion (ROM) in dorsiflexion (B and E); and at the maximal common angle in dorsiflexion displayed among each pair accounting from the slack angle (C and F). * $P < 0.05$ [adjusted by Bonferroni correction, 0.05/13 (A, B, D, and E), and 0.05/11 (C and F)]. θ General Mann-Whitney measure of effect size. Box plot legend: median (midline), box (25th and 75th percentiles). distal, distal-leg transducer location; EDL, extensor digitorum longus; FDL, flexor digitorum longus; FHL, flexor hallucis longus; GL, gastrocnemius lateralis; GM, gastrocnemius medialis; mid, mid-leg transducer location; PL, peroneus longus; proximal, proximal-leg transducer location; SOL, soleus; TA, tibialis anterior.

An Aixplorer ultrasound scanner (Supersonic Imagine, v. 6.1, Aix-en-Provence, France) was coupled with one linear transducer (2–10 MHz, SL10-2 or 4–15 MHz, SL15-4; Supersonic Imagine, Aix-en-Provence, France). A transistor-transistor logic pulse was sent by the ultrasound scanner at each shear modulus measurement (i.e., each second) to synchronize shear modulus measurements with the ankle angle, passive torque, and sEMG signals. The transducer was aligned along the longitudinal axis of the leg (i.e., corresponding to physiological plane of lower leg muscle shortening/lengthening direction) and perpendicular to the skin, so that the image plane intersects perpendicularly to the muscle aponeurosis. Thus, the stiffness measurement was always performed in the estimated muscle shortening/lengthening direction, as done with conventional material testing (16, 26). The main plantar flexors [GM, GL, SOL, flexor digitorum longus (FDL), flexor hallucis longus (FHL), peroneus longus (PL)] and dorsiflexors [TA, and extensor digitorum longus (EDL)] were scanned. Three proximo-distal regions were determined for the gastrocnemii (distal, middle, and proximal regions on the muscle), and two for the soleus (distal and proximal). These regions were chosen on the basis of anatomical guidelines used for botulinum toxin injections to treat muscle over activity in neurological conditions (55, 63). SWE has very good reliability for measuring shear modulus of superficial and deep muscles, especially during stretching (15, 43, 51). A previous study showed good interday reliability for shear wave measurements at all the locations used in this study (43).

Protocol

First, for both experiments, the maximal angle in dorsiflexion was measured as the dynamometer moved the ankle joint during a slow

passive stretch (2°/s). When participants felt “maximal tolerable stretch” in the calf (i.e., onset of pain), they pushed a button that stopped the motion. During this motion, subjects were blindfolded. Participants undertook three trials, and the maximum angle recorded was used in the subsequent calculation of 80% of range of dorsiflexion. Second, five ankle rotations from 40° of plantarflexion to 80% of the predetermined maximal ankle dorsiflexion angle were performed for muscle-conditioning purposes (54). Third, one shear modulus measurement was performed for each location in a randomized order during ankle dorsiflexion at a velocity of 2°/s, from 40° of plantarflexion to 80% of the maximal dorsiflexion. This range was used as passive motion; beyond this point often invokes unwanted muscle activation (48). Between each measurement, a 1-min of rest was observed. At the end of the procedure, participants were asked to perform three voluntary maximal isometric contractions (MVC) in plantarflexion and dorsiflexion and the root mean square (sEMG-RMS) of the associated sEMG signals were used to normalize activity recorded during passive motion to 80% dorsiflexion range of motion (ROM).

Data Analysis and Statistics

Data were processed using MATLAB scripts (MathWorks, Natick, MA). Ultrasound videos exported from Aixplorer’s software were sequenced in “jpeg” images. Then, each pixel of the color map was converted into a shear modulus value established from an image-processing algorithm (26). Shear modulus values were averaged over the largest region of interest (ROI) that avoided aponeurosis and artifacts. The mean area of the ROIs ranged between 60 mm² (FDL, stroke survivors) and 180 mm² (TA, controls).

The following analyses were performed for each muscle region (and are depicted in the APPENDIX). First, shear modulus values were compared at two points: 1) at the maximal common angle in dorsiflexion that was attained by patients and controls, and 2) at 80% of maximal dorsiflexion. Second, to provide information about muscle contracture, the “slack angle” was visually determined as the onset of increase in shear modulus during the passive dorsiflexion. This was performed for each muscle region and for each participant by an experienced examiner blinded to the muscle region and the participant. The visual approach for determining the slack angle has been shown to be reliable in previous studies, including those of our research group (36, 37, 43). Third, to account for the potential change in slack angle, the shear modulus values corresponding to the maximal common dorsiflexed position from the slack angle were compared between patients and controls. This latter analysis is known to give insights into the mechanical behavior of muscle tissue *in vivo* (34). Since the maximal ankle dorsiflexion was highly variable among stroke survivors (Table 1), these analyses were performed for each patient and his or her matched control subject to determine the maximal common angle for each pair of patient/control.

The sEMG-RMS were calculated over a 300-ms window centered on each shear modulus measurement (1 Hz) and normalized to the maximal values reached during the MVCs. The sEMG over the stretch ROM and the value reached at 80% of the maximal ROM were assessed in the statistical analyses.

Since distributions failed to pass the Shapiro-Wilk test for normality, Mann-Whitney *U*-tests were conducted to analyze between-group differences in shear modulus, slack angle, and muscle activity. The statistical significance was set at $P < 0.05$. The 0.05 value was adjusted for multiple tests using a Bonferroni correction (shear modulus: 0.05/13, slack length: 0.05/11, EMG: 0.05/4). When significant, between-group differences (stroke vs. controls) and bootstrapped confidence intervals (95% CI) ($n = 1,000$ samples) (19) were computed on shear modulus, slack angle, and muscle activity. Effect sizes were estimated using the general Mann-Whitney measure (θ) ($\theta = U/nm$, where U is the Mann-Whitney statistic, n and m are the sample sizes of both groups, respectively). A value of $\theta = 0.5$ indicates a perfect concordance (i.e., equal distribution of the population data), while for $\theta = 0$ or $\theta = 1$, there is no overlap between the group distributions (52). In addition, data were displayed pictorially by the decreasing order of shear modulus values recorded for all muscle regions among individuals, to qualitatively appreciate the location of the stiffest muscle regions among leg muscles. The muscle region displaying the highest shear modulus was presented in black, while a pale pink color corresponded to the muscle region where the lowest shear modulus was recorded. Data are presented descriptively for these analyses.

RESULTS

All subjects completed the protocol of *experiment 1* or *experiment 2*. However, a low recording quality was observed for 3% of the elastography measurements (artifacts or void areas within the ROI: 10 out of 338 videos from *experiment 1* and 10 out of 364 videos from *experiment 2*; details are provided in Fig. 1). These data were excluded from the analyses to reduce a potential risk of bias.

Experiment 1 (Knee Extended)

Shear modulus-ankle angle relationships are provided for each transducer location of *experiment 1* in Fig. 2. Between-group differences only appeared significant at the maximal common angle in dorsiflexion (Fig. 1A), where the shear modulus was higher for stroke survivors than for controls for gastrocnemii at the distal and middle muscle regions (all P

values < 0.002 ; θ between 0.11 and 0.14) and was indicative of higher muscle-tendon unit stiffness. The between-group difference (stroke-controls) of shear modulus was 31.2 kPa (CI 95% 29.1, 96.2 kPa) for GM distal, 26.7 kPa (CI 95% 18.1, 86.0 kPa) for GM mid, 20.42 kPa (CI 95% 17.0, 25.2 kPa) for GL distal, and 24.6 kPa (CI 95% 18.9, 30.4 kPa) for GL mid, respectively. The slack angle (Table 2 and Fig. 2) occurred at a more plantar muscle-flexed angle in stroke survivors within GM mid ($P = 0.045$, $\theta = 0.17$) and GL (all P values < 0.019 , θ ranges from 0.12 to 0.14). The between-group difference of slack angle was -5.6° (CI 95% -8.8 , -1.5°) for GM mid, -6.16° (CI 95% -7.7 , -3.1°) for GL distal, -5.7° (CI 95% -7.5 , -1.9°) for GL mid, and -5.6° (CI 95% -8.6 , -1.2°) for GL proximal, respectively.

The highest shear modulus was measured on GM for each group (Fig. 3A), and consistently found at the most distal site (85% of stroke survivors).

Experiment 2 (Knee Flexed)

Shear modulus-ankle angle relationships are provided for each transducer location of *experiment 2* in Fig. 4. At the maximal common angle in dorsiflexion (Fig. 1C), shear modulus was higher for stroke survivors for SOL distal ($P = 0.001$, $\theta = 0.13$) with a between-group difference of 18.2 kPa (CI 95% 9.1, 24.6 kPa). At 80% of maximal ROM (Fig. 1E), a lower shear modulus was found among stroke survivors on dorsiflexors [TA: $P = 0.002$; $\theta = 0.14$, between-group difference -3.8 kPa (CI 95% -6.6 , -1.4 kPa); EDL: $P = 0.001$; $\theta = 0.11$, between-group difference -6.0 kPa (CI 95% -9.7 , -4.2 kPa)].

The slack angle (Table 2 and Fig. 4) was measured at a more plantar muscle-flexed angle in stroke survivors in SOL [distal, $P < 0.001$, $\theta = 0.10$, between-group difference -6.4° (CI 95% -10.1 , -2.7°); proximal $P < 0.001$, $\theta = 0.02$, between-group difference -9.1° (CI 95% -11.7 , -6.2°)].

The highest shear modulus values were measured for SOL for each group (Fig. 3B) and were consistently found at the most distal site (86% of stroke survivors).

Muscle Activity

During both experiments, activity of GM, GL, SOL, and TA remained below 5% of maximal activation (Table 3). Despite the subjects being asked to stay relaxed, stroke survivors displayed higher averaged sEMG amplitudes than controls [all P values < 0.05 , θ between 0.08 and 0.19 (exp. 1) and 0.10–0.21 (exp. 2)]. At 80% of maximal ROM in dorsiflexion, between-group sEMG differed for plantar flexor muscles [GM, GL, and SOL: all P values < 0.05 , θ between 0.07 and 0.14 (exp. 1) and GL: $P = 0.009$, $\theta = 0.21$ (exp. 2)]. Between-groups differences and CI95% are reported in Table 3.

DISCUSSION

The present study compared the shear modulus between stroke survivors and healthy controls in several locations of the lower leg muscles. In several muscle regions, the shear modulus values reached at a given ankle angle were higher for the stroke group compared with the control group, but not at 80% of maximal dorsiflexion. Accounting for the slack angle, there were no between-group differences in the shear modulus.

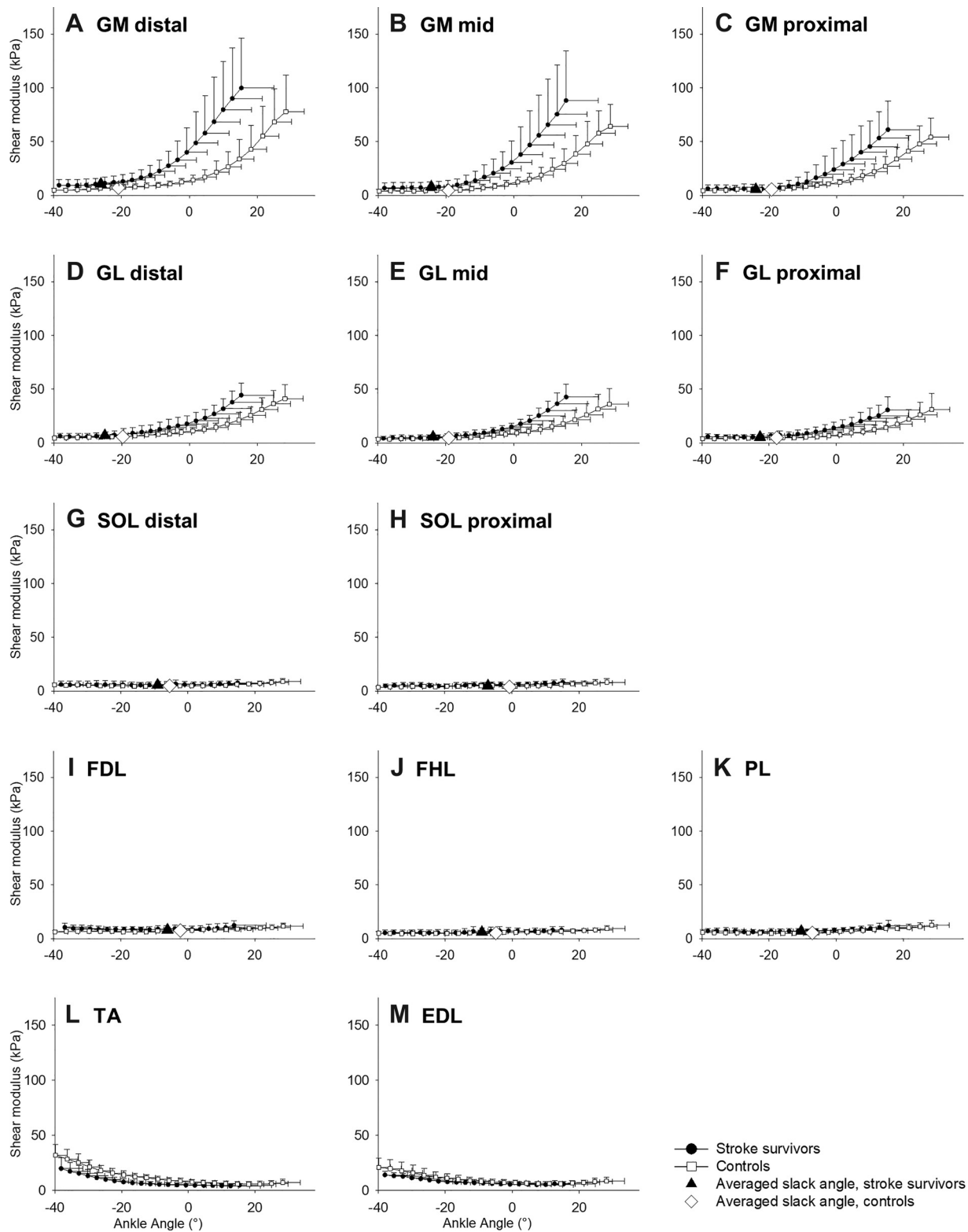


Fig. 2. Averaged (standard deviation, SD) shear modulus–ankle angle relationships during the passive dorsiflexion of the ankle, in *experiment 1* (knee fully extended) for each muscle region. Standard deviation bars were omitted for clarity. The averaged slack angle is provided and depicted on each relationship for each group with bigger symbols (● for stroke participants and □ for healthy controls) for each muscle region (except for dorsiflexors): gastrocnemius medialis (GM) distal (A), GM middle (B), GM, proximal (C), gastrocnemius lateralis (GL) distal (D), GL middle (E), GL proximal (F), soleus (SOL) distal (G), SOL proximal (H), flexor digitorum longus (FDL) (I), flexor hallucis longus (FHL) (J), peroneus longus (PL) (K), tibialis anterior (TA) (L), and extensor digitorum longus (EDL) (M). distal, distal-leg transducer location; mid, mid-leg transducer location; proximal, proximal-leg transducer location.

Table 2. Slack angle of the plantar flexors measured during dorsiflexion, with the knee fully extended (*experiment 1*) and with the knee flexed (*experiment 2*) for each group (stroke survivors and controls)

Transducer Location	Experiment 1 (Knee Fully Extended)		Experiment 2 (Knee Flexed)	
	Stroke Survivors	Controls	Stroke Survivors	Controls
GM distal	-27.1 (-29.0, -23.8)	-19.8 (-23.0, -19.1)	-11.6 (-13.3, -8.1)	-11.8 (-14.6, -8.9)
GM mid	-25.2 (-27.2, -21.9)	-19.6 (-20.2, -17.3)*	-10.7 (-12.6, -6.3)	-8.7 (-12.2, -5.9)
GM proximal	-25.4 (-27.1, -21.0)	-19.5 (-21.7, -18.0)	-9.0 (-13.2, -5.8)	-8.5 (-10.9, -6.6)
GL distal	-25.3 (-27.0, -23.0)	-19.1 (-21.2, -17.9)*	-10.5 (-14.2, -7.2)	-10.6 (-12.9, -7.5)
GL mid	-24.3 (-25.0, -23.3)	-18.6 (-19.7, -17.7)*	-10.9 (-12.6, -8.5)	-9.6 (-11.7, -7.4)
GL proximal	-23.2 (-24.8, -22.2)	-17.6 (-19.2, -16.2)*	-10.6 (-12.9, -8.0)	-7.9 (-11.0, -6.6)
SOL distal	-10.0 (-11.3, -6.5)	-5.4 (-9.3, -1.6)	-12.7 (-14.1, -11.4)	-6.4 (-9.7, -3.4)*
SOL proximal	-6.6 (-7.9, -3.8)	-0.5 (-5.2, -1.3)	-11.4 (-14.5, -7.8)	-2.3 (-4.1, 0.4)*
FDL	-7.5 (-11.2, -2.9)	-4.9 (-7.7, -0.1)	-8.8 (-9.5, -7.7)	-8.1 (-8.6, -5.9)
FHL	-8.9 (-11.1, -8.1)	-5.6 (-7.8, -2.0)	-11.0 (-12.5, -7.9)	-5.5 (-8.2, -3.3)
PL	-8.5 (-12.9, -5.9)	-9.1 (-10.1, -5.5)	-10.7 (-13.7, -7.4)	-7.9 (-10.0, -6.9)

Data are shown as median (quartile 1, quartile 3). * $P < 0.004$ (value adjusted by Bonferroni correction, 0.05/11).distal, distal-leg transducer location; FDL, flexor digitorum longus; FHL, flexor hallucis longus; GL, gastrocnemius lateralis; GM, gastrocnemius medialis; mid, mid-leg transducer location; PL, peroneus longus; proximal, proximal-leg transducer location; SOL, soleus.

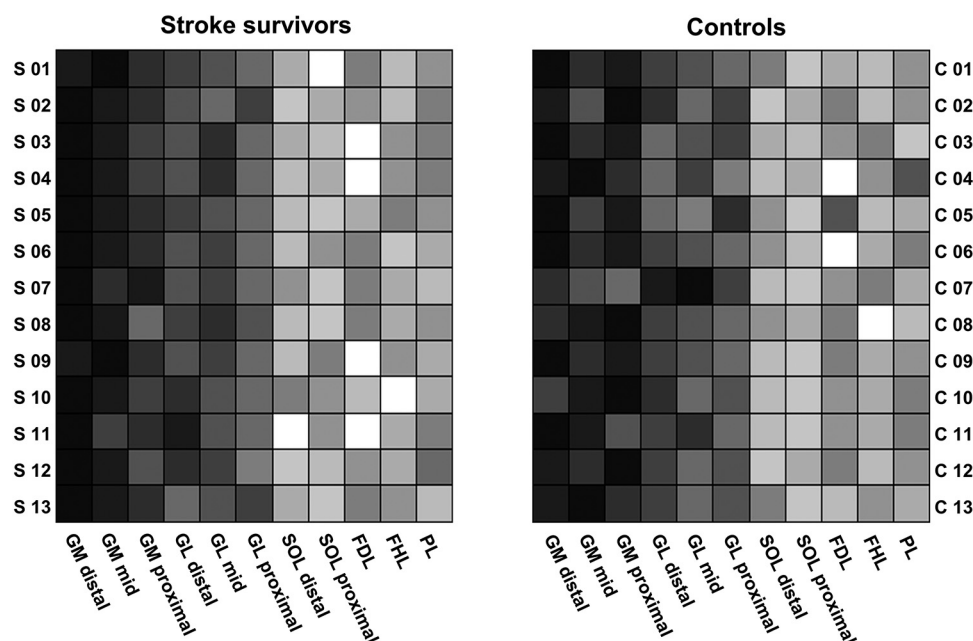
These results conform with studies conducted on healthy participants, also reporting a higher shear modulus for GM (knee extended) (10, 36, 43) and SOL (knee flexed) (43) during passive dorsiflexions. Because the muscle force will be influenced by both elasticity and size, the between-muscle differences in shear modulus should not be interpreted directly as difference in muscle force. For instance, because of differences in cross-sectional area (CSA), if two muscles exhibit the same change in shear modulus during stretching, the bigger muscle will exhibit the larger change in passive force. The ~2 times larger CSA of GM compared with GL (23), combined with higher shear modulus values for the GM, clearly demonstrate an imbalance of passive force in favor of GM when the ankle is dorsiflexed. In the same way, considering their small CSA (23) and low shear modulus values, the passive muscle force of the small plantar flexors (FDL, FHL, and PL) could be considered as negligible. It is more challenging to interpret the balance of passive force between gastrocnemii and SOL because of the volume of SOL is much larger than GM ($\times 2.4$) and GL ($\times 5.2$) (3), while shear modulus values are higher for both gastrocnemii with the knee extended. Further studies combining measurements of muscle size and muscle mechanical properties should be performed to better understand how contracture could influence the individual muscle contributions to the passive torque.

The velocity used (2°/s) and the ROM (80% of the maximal ROM) were set to limit the reflex responses to stretching. However, between-group differences in the amount of sEMG activity were found (Table 3). The values reported remained below 5% of sEMG during MVC. As such, it seems unlikely that muscle activation might influence our measurements, but the actual effect of this factor on our measurements remains unknown. If it did play a notable role, it would lead to an increase in muscle force developed in resistance to stretching at a short muscle length (61). Thus, it would induce a change in muscle slack angle (Table 3) and an increase in shear modulus for stroke survivors compared with controls (Fig. 2). However, some points need to be considered. First, it should be noted that different thresholds are often used to consider “passive conditions.” Regarding the literature, the reader finds notable variability across studies. For instance 1% (48), 2% (50), 5% (24), or 10% (29). In the absence of a consensus on the threshold

that should be used, we firmly encouraged participants to relax, as recommended during clinical examination (28) and in most research studies undertaken in this area. We also carefully checked that there were no increases in sEMG signals during each trial. If an increase in muscle activation was noticeable, the trial was not used for analyses and repeated. Second, the reflex-mediated increase found among the muscle activity of survivors [through an increased excitability of the α -motor neuron at the spinal cord level (53)] has been shown to reach its maximum between 1 and 3 mo after stroke (6). Studies also reported that the contributions of neural contributors to stiffness during stretching may decrease over time in the stroke populations (11, 59). At more chronic stages, such as for the sample recruited in the present study, the response observed during slow passive stretching may primarily be due to the passive intrinsic mechanical properties for slow stretching (49, 59). Thus, we believe that the results of the present study are marginally influenced by the neural contributions. This belief remains to be validated in further experiments. One possibility would be to test the effects of a transient blocking of motor nerve to eliminate muscle activity during stretching (9). A better understanding of the influence of slight EMG activation on passive mechanical responses (i.e., passive torque, shear modulus, and fascicle length) might be then elucidated.

Furthermore, we also provide a pictorial of individual responses through a pictorial mapping of the stiffness levels in multiple muscle regions (Fig. 4). This is novel, since the few studies using SWE in stroke subjects were focused on one muscle thought to be representative of the studied muscle group (17, 39, 44, 47, 62). Our pictorial analyses highlight that stroke survivors’ response is commonly observed at the same muscle regions within plantar flexors: the highest values for GM followed by GL (Fig. 3A, *experiment 1*) and for SOL (Fig. 3B, *experiment 2*). Of particular relevance, the studies of Mathevon et al. (47) and Jakubowski et al. (39) focused on GM and reported higher shear modulus on the affected side of stroke survivors. Although we measured the shear modulus during standardized dynamic stretches, these studies positioned the ankle in the targeted angle first, before scanning the GM with the transducer. These static measurements could induce a stress relaxation effect (45), which may explain their lower values compared with those of the present study.

A Experiment 1 (knee fully extended)



B Experiment 2 (knee flexed at 90°)

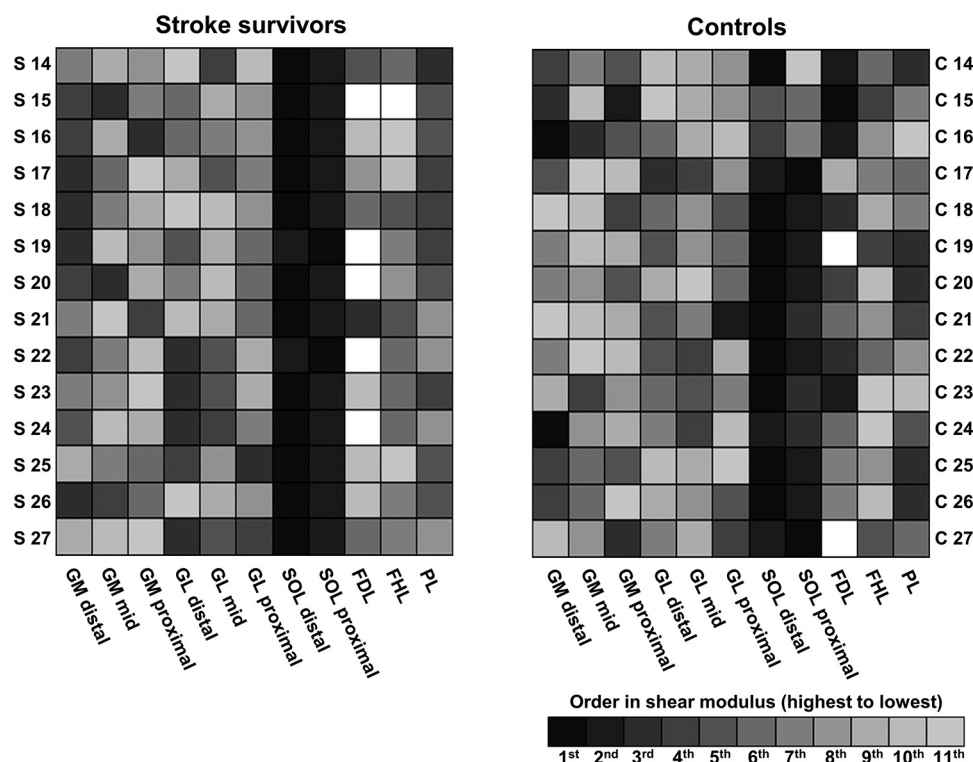


Fig. 3. Individual stiffness distribution at the same ankle angle (maximal common angle) during *experiment 1* (A) and *experiment 2* (B) (knee fully extended and knee flexed at 90°) for each transducer location among plantar flexor muscles, in stroke survivors and matched controls (e.g., S01 corresponding to stroke survivor participant no. 1, and C01 to control participant no. 1). White cells represent missing values. FDL, flexor digitorum longus; FHL, flexor hallucis longus; GL, gastrocnemius lateralis; GM, gastrocnemius medialis; SOL, soleus. distal: distal-leg transducer location; mid: mid-leg transducer location; proximal: proximal-leg transducer location.

Very low shear modulus values were recorded for the other plantar flexors (Fig. 1), supporting the concept that triceps surae muscles might be preferentially affected during passive dorsiflexion after a stroke. This is close to what is also observed among controls (Fig. 3), and elsewhere on healthy participants (43).

Finally, higher shear modulus values were found for TA and EDL in controls (*experiment 2*, see Fig. 4 plots in *L* and *M*). A

number of researchers (2, 27, 56) have commented that the resting position of the foot in a sitting and lying position in stroke survivors is often that of greater plantarflexion than those individuals without stroke. Consequently, a decrease in shear modulus of the dorsiflexors in the more plantar muscle-flexed angles could be observed.

The results of the present study are important because they provide evidence of the spatial locations that are the most

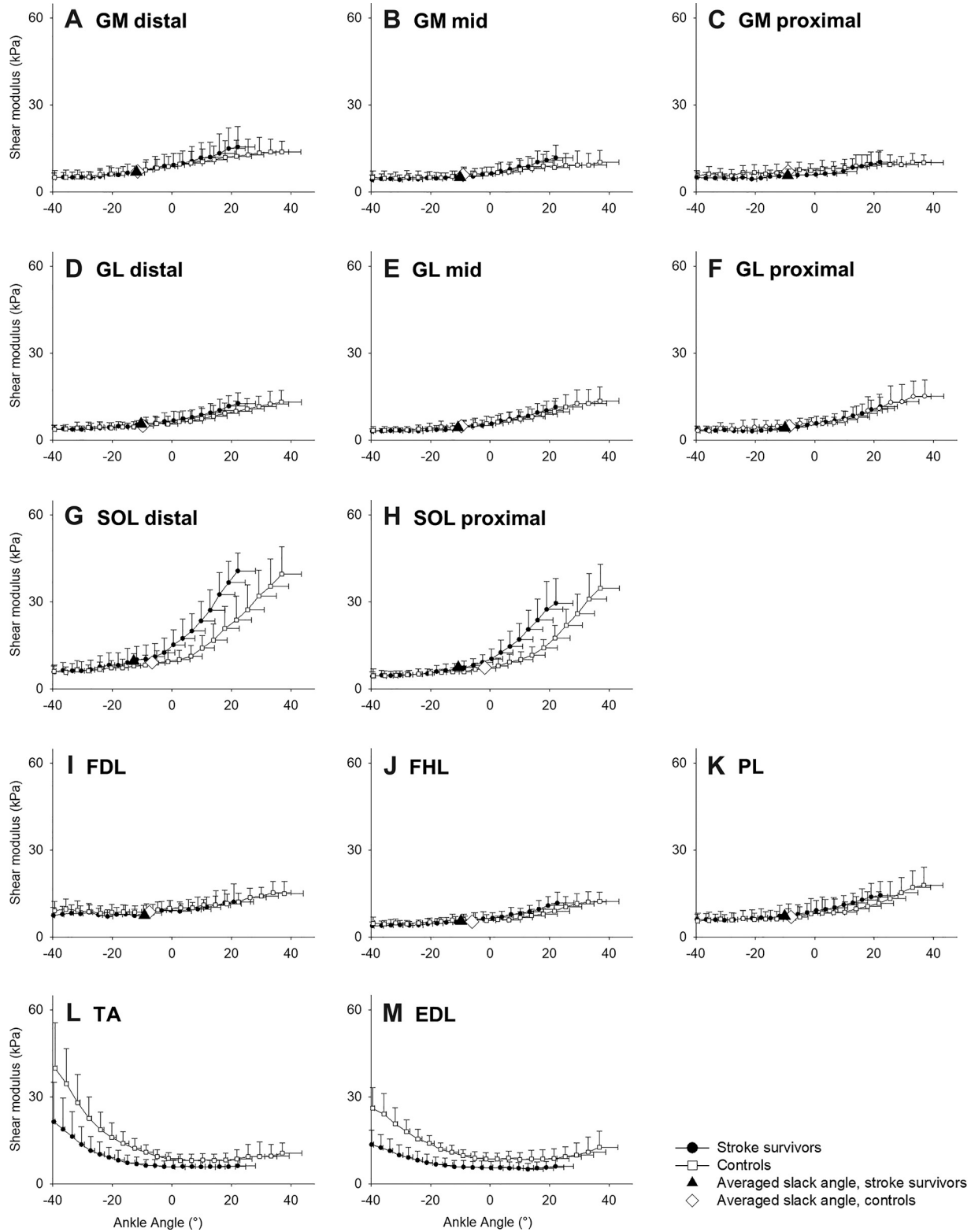


Fig. 4. Averaged (standard deviation) shear modulus–ankle angle relationships during the passive dorsiflexion of the ankle, in *experiment 2* (knee flexed at 90°) for each muscle region. The averaged slack angle is provided and depicted on each relationship for each group with bigger symbols (black triangle for stroke participants, and white diamond for healthy controls) for each muscle region (except for dorsiflexors): gastrocnemius medialis (GM) distal (A), GM middle (B), GM, proximal (C), gastrocnemius lateralis (GL) distal (D), GL middle (E), GL proximal (F), soleus (SOL) distal (G), SOL proximal (H), flexor digitorum longus (FDL) (I), flexor hallucis longus (FHL) (J), peroneus longus (PL) (K), tibialis anterior (TA) (L), and extensor digitorum longus (EDL) (M). distal, distal-leg transducer location; mid, mid-leg transducer location; proximal, proximal-leg transducer location.

Table 3. sEMG amplitude measured during passive dorsiflexion and normalized to that measured during MVC

Muscle	Experiment 1 (Knee Fully Extended)			Experiment 2 (Knee Flexed)		
	Stroke Survivors	Controls	Between-Group Differences (CI 95%)	Stroke Survivors	Controls	Between-Group Differences (CI 95%)
Averaged sEMG amplitude, %						
GM	3.5 (1.2, 4.4)	0.5 (0.2, 0.8)*	2.9 (0.7, 3.9)	4.1 (1.4, 5.4)	1.0 (0.4, 1.6)*	3.1 (0.7, 3.9)
GL	1.8 (0.9, 3.2)	0.5 (0.3, 1.2)*	1.2 (0.2, 2.0)	3.1 (1.5, 6.1)	0.8 (0.4, 1.3)*	1.7 (0.2, 2.0)
SOL	2.2 (1.2, 3.4)	0.7 (0.4, 1.1)*	1.5 (1.0, 1.9)	3.1 (1.7, 4.4)	1.0 (0.5, 1.1)*	2.0 (1.0, 1.9)
TA	0.7 (0.5, 1.2)	0.1 (0.1, 0.4)*	0.5 (0.1, 1.0)	1.1 (0.6, 1.8)	0.3 (0.1, 0.7)*	0.6 (0.1, 1.0)
sEMG amplitude at 80% of maximum dorsiflexion, %						
GM	3.7 (1.5, 5.9)	0.3 (0.1, 0.7)*	3.5 (1.4, 5.6)	4.2 (2.1, 5.8)	1.5 (0.4, 2.6)	ns
GL	2.4 (1.0, 4.4)	0.5 (0.3, 1.0)*	1.9 (0.5, 4.1)	4.4 (1.7, 6.1)	1.0 (0.4, 2.6)*	ns
SOL	2.8 (1.9, 4.9)	1.0 (0.4, 1.3)*	1.8 (1.3, 2.9)	4.5 (2.8, 6.7)	1.3 (1.0, 4.3)	3.1 (1.3, 4.1)
TA	0.8 (0.7, 1.1)	0.4 (0.1, 0.7)	ns	1.0 (0.6, 1.4)	1.9 (0.2, 2.2)	ns

Data are shown as median (quartile 1, quartile 3). * $P < 0.013$ (value adjusted by Bonferroni correction, 0.05/4). CI 95%, confidence interval (95%) about between-group differences in medians from bootstrapping. GL, gastrocnemius lateralis; GM, gastrocnemius medialis; MVC, maximum voluntary contraction; ns, not significant; sEMG, surface electromyography; SOL, soleus; TA, tibialis anterior.

affected after a stroke among the plantar flexor muscle group. This opens perspectives for future studies to evaluate the efficacy of interventions aiming to restore mobility of the ankle joint. While classical stretching exercises do not have significant effects on muscle contracture (31), it is possible to design more intensive stretching programs targeting GM (and/or SOL). For instance, as mentioned previously, a dorsiflexion position at the ankle coupled with an inverted subtalar position (knee extended) can enhance the effect of the maneuver on GM compared with other plantar flexors (18). In addition, when severe contractures are noticeable, release surgeries are offered to survivors to restore ankle joint dorsiflexion (13). However, secondary mobility issues related to such surgery have been observed, including overlengthening of the muscles (1). Relatedly, there is literature in support of the use of isolated gastrocnemius recession techniques, but the precise location of the stiffest regions prevents the surgeon from being sure of the location to incise (12). In lieu of our findings that the shear modulus values were the highest at the distal muscle regions of GM and SOL, it would be interesting to investigate the efficacy of minimally invasive isolated recessions close to the distal myotendinous junction on the aponeuroses of GM and/or SOL muscle (60), and compare results to those from more generalized incisions.

Limitations

The present study was designed to measure shear modulus in several locations of the plantar flexors during passive ankle dorsiflexions. Fascicle length measurements might have been an alternative method to investigate muscle mechanical properties (32, 33). However, with the data collected in the present study, it was only possible to measure the fascicle length of the GM, and hence, this was not pursued. Future studies might compare fascicle length and shear modulus measurements. The shorter slack angle reported in the present study for stroke survivors is in accordance with Gao et al. (25), who reported shorter fascicle length of GM for stroke survivors at 40° of PF. However, the findings are different from those of Kwah et al. (41), who did not report between-group differences in fascicle slack length. Differences in study methodologies (dorsiflexions performed in various knee angle configurations) or participants (sample size, clinical characteristics of participants) might have con-

tributed to contrasting findings. For instance, the between-group ranges in ankle range of motion were higher in the present study (see Table 1) compared with Kwah et al. (41). A more in-depth comparison of the methods used in the present study and those of Kwah et al. (41) is required to better understand the differences. Because it involves the detection of subtle changes, the measurement of the slack length remains challenging, and the most appropriate method still remains to be established.

Finally, although our results show that SWE is relevant to detect the effects of the stroke injury in multiple muscle locations, they cannot be used to infer the cause of the increased shear modulus. It is fundamental to better understand these mechanisms to improve therapeutic decisions (57). Potential mechanisms include changes in connective tissue or extracellular matrix. Active force generation may also be involved through impairment of calcium signaling of the muscle cell. Such a potential change in active force during the stretching cannot be detected with EMG.

Conclusion

Our findings suggest that the gastrocnemii and soleus muscles are most responsible for the increase in stiffness observed in plantar flexors muscles of stroke survivors. Within these muscles, the distal regions of GM and SOL were the most affected. No between-group differences were found when accounting for slack angle, suggesting that the increased level of muscle stiffness can be explained by a decrease in muscle length. In addition, our interindividual analysis revealed that the most affected locations were similar among stroke survivors. These new results provide a better understanding of soft tissue responses after a stroke that affects dorsiflexion of the ankle, a movement critical to the performance of efficient and safe walking.

APPENDIX

Averaged shear modulus–ankle angle (standard deviation bars omitted for clarity) and shear modulus–ankle range of motion in dorsiflexion adjusted from slack angle relationships have been shown for both populations (stroke survivors and controls) during the passive dorsiflexion of the ankle, in *experiment 1* (knee fully extended) for gastrocnemius medialis distal muscle region.

The averaged slack angle value is provided and depicted on each relationship for each group with bigger symbols (● for stroke participants, and □ for healthy controls) (Fig. 5).

Analyses

- 1 at the same ankle angle
- 2 at 80% of maximal ROM in dorsiflexion
- 3 at the same angle, accounting from slack angle

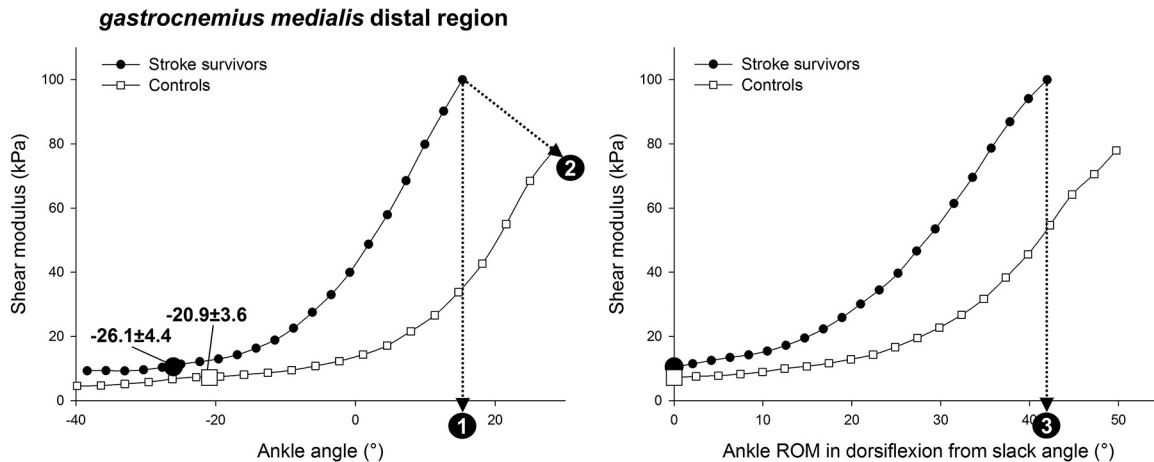


Fig. 5. Averaged shear modulus–ankle angle (standard deviation bars omitted for clarity) and shear modulus– ankle range of motion in dorsiflexion adjusted from slack angle relationships for both populations (stroke survivors and controls) during the passive dorsiflexion of the ankle, in *experiment 1* (knee fully extended) for gastrocnemius medialis distal muscle region. The averaged slack angle value is provided and depicted on each relationship for each group with bigger symbols (● for stroke participants and □ for healthy controls).

As described in MATERIALS AND METHODS, three comparisons of shear modulus values were performed: 1) at the same ankle angle, 2) at 80% of the maximal ROM, and 3) at the same angle accounting from slack angle, for each muscle region, to investigate the between-group differences of muscle shear modulus.

ACKNOWLEDGMENTS

The authors are grateful to all participants involved in the study, as well as to Aurélie Sarcher (Centre Hospitalier Universitaire Nantes/Institut National de la Santé et de la Recherche Médicale UMR 131 Laboratory of Medical Information Processing Brest) for assistance with statistical analysis.

GRANTS

The studies were supported by a grant from the University of Nantes (interdisciplinary program), and the Région des Pays de la Loire (QUETE Project). The funders had no role in design, data collection and analysis, manuscript redaction, and publication.

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS

G.L.S., F.H., R.A., A.N., and R.G. conceived and designed the research; G.L.S., R.A., T.L., and R.G. performed experiments; G.L.S., A.N., F.H., P.M.N. and R.G. analyzed and interpreted data; G.L.S., A.N., F.H., R.A., T.L., P.M.N., and R.G. edited manuscript. G.L.S., A.N., F.H., R.A., T.L., P.M.N., and R.G. approved final version of the manuscript.

REFERENCES

1. Abdulmassih S, Phisitkul P, Femino JE, Amendola A. Triceps surae contracture: implications for foot and ankle surgery. *J Am Acad Orthop Surg* 21: 398–407, 2013. doi:10.5435/JAAOS-21-07-398.
2. Ada L, Canning C. Changing the way we view the contribution of motor impairments to physical disability after stroke. In: *Science-Based Rehabilitation: Theories into Practice*, edited by Refshauge K, Ada L, and Ellis E. New York: Elsevier, 2005, p. 87–206.
3. Albracht K, Arampatzis A, Baltzopoulos V. Assessment of muscle volume and physiological cross-sectional area of the human triceps surae muscle in vivo. *J Biomech* 41: 2211–2218, 2008. doi:10.1016/j.jbiomech.2008.04.020.
4. Baker R, Esquenazi A, Benedetti MG, Desloovere K. Gait analysis: clinical facts. *Eur J Phys Rehabil Med* 52: 560–574, 2016.
5. Bercoff J, Tanter M, Fink M. Supersonic shear imaging: a new technique for soft tissue elasticity mapping. *IEEE Trans Ultrason Ferroelectr Freq Control* 51: 396–409, 2004. doi:10.1109/TUFFC.2004.1295425.
6. Bhakta BB. Management of spasticity in stroke. *Br Med Bull* 56: 476–485, 2000. doi:10.1258/0007142001903111.
7. Bohannon RW, Smith MB. Interrater reliability of a modified Ashworth scale of muscle spasticity. *Phys Ther* 67: 206–207, 1987. doi:10.1093/ptj/67.2.206.
8. Bruening DA, Crewe AN, Buczek FL. A simple, anatomically based correction to the conventional ankle joint center. *Clin Biomech (Bristol, Avon)* 23: 1299–1302, 2008. doi:10.1016/j.clinbiomech.2008.08.005.
9. Buffenoir K, Decq P, Lefaucheur JP. Interest of peripheral anesthetic blocks as a diagnosis and prognosis tool in patients with spastic equinus foot: a clinical and electrophysiological study of the effects of block of nerve branches to the triceps surae muscle. *Clin Neurophysiol* 116: 1596–1600, 2005. doi:10.1016/j.clinph.2004.11.024.
10. Chino K, Kato E, Takahashi H. Regional difference in gastrocnemius tension during passive stretching as assessed with shear wave elastography. *Med Sci Sports Exerc* 48: 898, 2016. doi:10.1249/01.mss.0000487689.26304.ed.
11. Chung SG, Van Rey E, Bai Z, Roth EJ, Zhang LQ. Biomechanical changes in passive properties of hemiplegic ankles with spastic hypertonia. *Arch Phys Med Rehabil* 85: 1638–1646, 2004. doi:10.1016/j.apmr.2003.11.041.
12. Cyhosh CC, Phisitkul P, Belatti DA, Glazebrook MA, DiGiovanni CW. Gastrocnemius recession for foot and ankle conditions in adults: Evidence-based recommendations. *Foot Ankle Surg* 21: 77–85, 2015. doi:10.1016/j.fas.2015.02.001.
13. DeHeer PA. Equinus and lengthening techniques. *Clin Podiatr Med Surg* 34: 207–227, 2017. doi:10.1016/j.cpm.2016.10.008.
14. Dorsch S, Ada L, Canning CG. Lower limb strength is significantly impaired in all muscle groups in ambulatory people with chronic stroke: a cross-sectional study. *Arch Phys Med Rehabil* 97: 522–527, 2016. doi:10.1016/j.apmr.2015.10.106.
15. Dubois G, Kheirredine W, Vergari C, Bonneau D, Thoreux P, Rouch P, Tanter M, Gennisson JL, Skalli W. Reliable protocol for shear wave elastography of lower limb muscles at rest and during passive stretching.

- Ultrasound Med Biol* 41: 2284–2291, 2015. doi:10.1016/j.ultrasmedbio.2015.04.020.
16. Eby SF, Song P, Chen S, Chen Q, Greenleaf JF, An KN. Validation of shear wave elastography in skeletal muscle. *J Biomech* 46: 2381–2387, 2013. doi:10.1016/j.jbiomech.2013.07.033.
17. Eby S, Zhao H, Song P, Vareberg BJ, Kinnick R, Greenleaf JF, An KN, Chen S, Brown AW. Quantitative evaluation of passive muscle stiffness in chronic stroke. *Am J Phys Med Rehabil* 95: 899–910, 2016. doi:10.1097/PHM.0000000000000516.
18. Edama M, Onishi H, Kumaki K, Kageyama I, Watanabe H, Nashimoto S. Effective and selective stretching of the medial head of the gastrocnemius. *Scand J Med Sci Sports* 25: 242–250, 2015. doi:10.1111/sms.12203.
19. Efron B, Tibshirani R. *An Introduction to the Bootstrap*. New York: Chapman & Hall, 1994.
20. English C, McLennan H, Thoires K, Coates A, Bernhardt J. Loss of skeletal muscle mass after stroke: a systematic review. *Int J Stroke* 5: 395–402, 2010. doi:10.1111/j.1747-4949.2010.00467.x.
21. English C, Thoires K, Coates A, Ryan A, Bernhardt J. Changes in fat mass in stroke survivors: a systematic review. *Int J Stroke* 7: 491–498, 2012. doi:10.1111/j.1747-4949.2012.00824.x.
22. Fergusson D, Hutton B, Drodge A. The epidemiology of major joint contractures: a systematic review of the literature. *Clin Orthop Relat Res* 456: 22–29, 2007. doi:10.1097/BLO.0b013e3180308456.
23. Fukunaga T, Roy RR, Shellock FG, Hodgson JA, Day MK, Lee PL, Kwong-Fu H, Edgerton VR. Physiological cross-sectional area of human leg muscles based on magnetic resonance imaging. *J Orthop Res* 10: 926–934, 1992. doi:10.1002/jor.1100106623.
24. Gajdosik RL, Vander Linden DW, McNair PJ, Riggins TJ, Albertson JS, Mattick DJ, Wegley JC. Viscoelastic properties of short calf muscle-tendon units of older women: effects of slow and fast passive dorsiflexion stretches in vivo. *Eur J Appl Physiol* 95: 131–139, 2005. doi:10.1007/s00421-005-1394-4.
25. Gao F, Grant TH, Roth EJ, Zhang LQ. Changes in passive mechanical properties of the gastrocnemius muscle at the muscle fascicle and joint levels in stroke survivors. *Arch Phys Med Rehabil* 90: 819–826, 2009. doi:10.1016/j.apmr.2008.11.004.
26. Gennissou JL, Deffieux T, Fink M, Tanter M. Ultrasound elastography: principles and techniques. *Diagn Interv Imaging* 94: 487–495, 2013. doi:10.1016/j.diii.2013.01.022.
27. Gracies JM. Pathophysiology of spastic paresis. I. Paresis and soft tissue changes. *Muscle Nerve* 31: 535–551, 2005. doi:10.1002/mus.20284.
28. Gracies JM, Bayle N, Vinti M, Alkandari S, Vu P, Loche CM, Colas C. Five-step clinical assessment in spastic paresis. *Eur J Phys Rehabil Med* 46: 411–421, 2010.
29. Halbertsma JP, Mulder I, Göeken LN, Eisma WH. Repeated passive stretching: acute effect on the passive muscle moment and extensibility of short hamstrings. *Arch Phys Med Rehabil* 80: 407–414, 1999. doi:10.1016/S0003-9993(99)90277-0.
30. Harlaar J, Becher JG, Snijders CJ, Lankhorst GJ. Passive stiffness characteristics of ankle plantar flexors in hemiplegia. *Clin Biomech (Bristol, Avon)* 15: 261–270, 2000. doi:10.1016/S0268-0033(99)00069-8.
31. Harvey LA, Katalinic OM, Herbert RD, Moseley AM, Lannin NA, Schurr K. Stretch for the treatment and prevention of contractures. *Cochrane Database Syst Rev* 1: CD007455, 2017. doi:10.1002/14651858.CD007455.pub3.
32. Herbert RD, Clarke J, Kwah LK, Diong J, Martin J, Clarke EC, Bilston LE, Gandevia SC. In vivo passive mechanical behaviour of muscle fascicles and tendons in human gastrocnemius muscle-tendon units. *J Physiol* 589: 5257–5267, 2011. doi:10.1113/jphysiol.2011.212175.
33. Herbert RD, Héroux ME, Diong J, Bilston LE, Gandevia SC, Litchwark GA. Changes in the length and three-dimensional orientation of muscle fascicles and aponeuroses with passive length changes in human gastrocnemius muscles. *J Physiol* 593: 441–455, 2015. doi:10.1113/jphysiol.2014.279166.
34. Herbert RD, Moseley AM, Butler JE, Gandevia SC. Change in length of relaxed muscle fascicles and tendons with knee and ankle movement in humans. *J Physiol* 539: 637–645, 2002. doi:10.1113/jphysiol.2001.012756.
35. Hermens HJ, Freriks B, Disselhorst-Klug C, Rau G. Development of recommendations for SEMG sensors and sensor placement procedures. *J Electromyogr Kinesiol* 10: 361–374, 2000. doi:10.1016/S1050-6411(00)00027-4.
36. Hirata K, Kanehisa H, Miyamoto-Mikami E, Miyamoto N. Evidence for intermuscle difference in slack angle in human triceps surae. *J Biomech* 48: 1210–1213, 2015. doi:10.1016/j.jbiomech.2015.01.039.
37. Hug F, Lacourpaille L, Maisetti O, Nordez A. Slack length of gastrocnemius medialis and Achilles tendon occurs at different ankle angles. *J Biomech* 46: 2534–2538, 2013. doi:10.1016/j.jbiomech.2013.07.015.
38. Hunnicutt JL, Gregory CM. Skeletal muscle changes following stroke: a systematic review and comparison to healthy individuals. *Top Stroke Rehabil* 24: 463–471, 2017. doi:10.1080/10749357.2017.1292720.
39. Jakubowski KL, Terman A, Santana RVC, Lee SSM. Passive material properties of stroke-impaired plantarflexor and dorsiflexor muscles. *Clin Biomech (Bristol, Avon)* 49: 48–55, 2017. doi:10.1016/j.clinbiomech.2017.08.009.
40. Koo TK, Guo JY, Cohen JH, Parker KJ. Relationship between shear elastic modulus and passive muscle force: an ex-vivo study. *J Biomech* 46: 2053–2059, 2013. doi:10.1016/j.jbiomech.2013.05.016.
41. Kwah LK, Herbert RD, Harvey LA, Diong J, Clarke JL, Martin JH, Clarke EC, Hoang PD, Bilston LE, Gandevia SC. Passive mechanical properties of gastrocnemius muscles of people with ankle contracture after stroke. *Arch Phys Med Rehabil* 93: 1185–1190, 2012. doi:10.1016/j.apmr.2012.02.009.
42. Langhorne P, Bernhardt J, Kwakkel G. Stroke rehabilitation. *Lancet* 377: 1693–1702, 2011. doi:10.1016/S0140-6736(11)60325-5.
43. Le Sant G, Nordez A, Andrade R, Hug F, Freitas S, Gross R. Stiffness mapping of lower leg muscles during passive dorsiflexion. *J Anat* 230: 639–650, 2017. doi:10.1111/joa.12589.
44. Lee SS, Spear S, Rymer WZ. Quantifying changes in material properties of stroke-impaired muscle. *Clin Biomech (Bristol, Avon)* 30: 269–275, 2015. doi:10.1016/j.clinbiomech.2015.01.004.
45. Magnusson SP. Passive properties of human skeletal muscle during stretch maneuvers. A review. *Scand J Med Sci Sports* 8: 65–77, 1998. doi:10.1111/j.1600-0838.1998.tb00171.x.
46. Maisetti O, Hug F, Bouillard K, Nordez A. Characterization of passive elastic properties of the human medial gastrocnemius muscle belly using supersonic shear imaging. *J Biomech* 45: 978–984, 2012. doi:10.1016/j.jbiomech.2012.01.009.
47. Mathevon LD, Michel FD, Aubry SD, Testa RD, Lapole TD, Arnaudeau LF, Fernandez BD, Parratte BP, Calmels PP. Two-dimensional and shear wave elastography ultrasound: A reliable method to analyse spastic muscles? *Muscle Nerve* 57: 222–228, 2018. doi:10.1002/mus.25716.
48. McNair PJ, Dombroski EW, Hewson DJ, Stanley SN. Stretching at the ankle joint: viscoelastic responses to holds and continuous passive motion. *Med Sci Sports Exerc* 33: 354–358, 2001. doi:10.1097/00005768-200103000-00003.
49. McNair PJ, Hewson DJ, Dombroski E, Stanley SN. Stiffness and passive peak force changes at the ankle joint: the effect of different joint angular velocities. *Clin Biomech (Bristol, Avon)* 17: 536–540, 2002. doi:10.1016/S0268-0033(02)00062-1.
50. Nakamura M, Ikezoe T, Takeno Y, Ichihashi N. Acute and prolonged effect of static stretching on the passive stiffness of the human gastrocnemius muscle tendon unit in vivo. *J Orthop Res* 29: 1759–1763, 2011. doi:10.1002/jor.21445.
51. Nakamura M, Ikezoe T, Umegaki H, Kobayashi T, Nishisita S, Ichihashi N. Shear elastic modulus is a reproducible index reflecting the passive mechanical properties of medial gastrocnemius muscle belly. *Acta Radiol Open* 5: 2058460115604009, 2016. doi:10.1177/2058460115604009.
52. Newcombe RG. Confidence intervals for an effect size measure based on the Mann-Whitney statistic. Part 1: general issues and tail-area-based methods. *Stat Med* 25: 543–557, 2006. doi:10.1002/sim.2323.
53. Nielsen JB, Crone C, Hultborn H. The spinal pathophysiology of spasticity—from a basic science point of view. *Acta Physiol (Oxf)* 189: 171–180, 2007. doi:10.1111/j.1748-1716.2006.01652.x.
54. Nordez A, Cornu C, McNair P. Acute effects of static stretching on passive stiffness of the hamstring muscles calculated using different mathematical models. *Clin Biomech (Bristol, Avon)* 21: 755–760, 2006. doi:10.1016/j.clinbiomech.2006.03.005.
55. Parratte B, Tatu L, Vuillier F, Diop M, Monnier G. Intramuscular distribution of nerves in the human triceps surae muscle: anatomical bases for treatment of spastic drop foot with botulinum toxin. *Surg Radiol Anat* 24: 91–96, 2002. doi:10.1007/s00276-002-0022-9.

56. **Perry J.** Determinants of muscle function in the spastic lower extremity. *Clin Orthop Relat Res* (288): 10–26, 1993.
57. **Pingel J, Bartels EM, Nielsen JB.** New perspectives on the development of muscle contractures following central motor lesions. *J Physiol* 595: 1027–1038, 2017. doi:[10.1113/JP272767](https://doi.org/10.1113/JP272767).
58. **van Swieten JC, Koudstaal PJ, Visser MC, Schouten HJ, van Gijn J.** Interobserver agreement for the assessment of handicap in stroke patients. *Stroke* 19: 604–607, 1988. doi:[10.1161/01.STR.19.5.604](https://doi.org/10.1161/01.STR.19.5.604).
59. **Vattanasilp W, Ada L, Crosbie J.** Contribution of thixotropy, spasticity, and contracture to ankle stiffness after stroke. *J Neurol Neurosurg Psychiatry* 69: 34–39, 2000. doi:[10.1136/jnnp.69.1.34](https://doi.org/10.1136/jnnp.69.1.34).
60. **Villanueva M, Iborra Á, Rodríguez G, Sanz-Ruiz P.** Ultrasound-guided gastrocnemius recession: a new ultra-minimally invasive surgical technique. *BMC Musculoskelet Disord* 17: 409, 2016. doi:[10.1186/s12891-016-1265-7](https://doi.org/10.1186/s12891-016-1265-7).
61. **Weppler CH, Magnusson SP.** Increasing muscle extensibility: a matter of increasing length or modifying sensation? *Phys Ther* 90: 438–449, 2010. doi:[10.2522/ptj.20090012](https://doi.org/10.2522/ptj.20090012).
62. **Wu CH, Ho YC, Hsiao MY, Chen WS, Wang TG.** Evaluation of post-stroke spastic muscle stiffness using shear wave ultrasound elastography. *Ultrasound Med Biol* 43: 1105–1111, 2017. doi:[10.1016/j.ultrasmedbio.2016.12.008](https://doi.org/10.1016/j.ultrasmedbio.2016.12.008).
63. **Yi KH, Rha DW, Lee SC, Cong L, Lee HJ, Lee YW, Kim HJ, Hu KS.** Intramuscular nerve distribution pattern of ankle invertor muscles in human cadaver using sihler stain. *Muscle Nerve* 53: 742–747, 2016. doi:[10.1002/mus.24939](https://doi.org/10.1002/mus.24939).