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ACHILLES TENDON MORPHOLOGICAL PROPERTIES IN POSTSTROKE SURVIVORS

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SUMMARY

The force developed by the contractile component can be affected by morphological properties of tendon structures, which can be altered by spasticity in stroke survivors. The purpose of this study was to compare the tendon length of the soleus (S) and medial gastrocnemius (MG) muscles between stroke survivors with ankle spasticity and healthy subjects. The study included 12 stroke survivors and 12 healthy subjects (used as a control group). An isokinetic dynamometer was used to passively position the subjects' ankle at 0°, 20°. Subjects were also asked to perform a maximal dorsiflexor contraction (MDC) to obtain ultrasonography images to determine tendon length. No differences were found at 0° or 20° of passive dorsiflexion between the spastic and non-spastic limbs of stroke survivors and between stroke survivors and the control group for both S and MG. Similarly, no between-groups difference was observed for tendon length at MDC in both muscles. Spasticity does not appear to change S and MG tendon length.

INTRODUCTION

Spasticity has been defined as a velocity-dependent increase in tonic stretch reflexes resulting from hyperexcitability of stretch reflex, and is present in about two thirds of chronic stroke patients [1]. The spastic muscle undergoes significant morphological and structural alterations which contribute to muscle weakness, restricted joint range of motion and increased passive joint stiffness [2], alterations that may affect tendon length. Tendon length may be angle position-dependent, and angle adaptations promote length changes that modify tendon stiffness. Tendon stiffness may be related to the ankle passive stiffness [3], and the power transmission of a tendon decreases or increases accordingly to these changes. Studies have successively applied *in vivo* ultrasonography imaging to investigate muscular properties in persons with neurologic disorders [4]. Thus, the aim of this study was to compare the S and MG tendon length using ultrasonography. Tendon length was passively measured at two different joint angles and during a maximal dorsiflexor contraction (MDC) in stroke survivors with ankle spasticity and was compared to healthy subjects.

METHODS

Twelve stroke survivors with ankle spasticity (4 females and 8 males; 59.6 ± 11.2 years old; 78.6 ± 12.7 kg of body mass; 170 ± 0.06 cm of height) and twelve healthy subjects (4 females and 8 males; 59.8 ± 5.9 years old; 75.1 ± 12.7 kg of body mass; 169 ± 0.08 cm of height) signed an informed consent form to participate in the study, which was approved by the University Ethics Committee in Human Research. Ashworth Scale [5] was used to quantify ankle spasticity (1.5 ± 0.6). Body mass, standing height and lower limb length were measured in all subjects. Subjects were sat on the isokinetic dynamometer chair (Biodex Medical System, Shirley, NY, USA) where the knee joint remained fully extended and the ankle joint was kept neutral (0°). The ankle joint was randomly positioned at 0° and 20° to measure S and MG tendon length. In addition, subjects were asked to perform a MDC to measure maximal tendon length. Ultrasound images were gathered from the muscles using an ultrasound scanner (SSD 4000, 51 Hz, ALOKA Inc., Tokyo, Japan). Tendon length was determined as the distance between the common tendon insertion into the calcaneus notch and the muscles' tendon junction. Subjects were instructed to relax for the two different ankle joint position measurements, and to perform a maximal isometric dorsiflexor contraction (MDC), which was performed during 5-sec effort, to measure maximal tendon length. A synchronization unit (HORITA Video Stop Watch VS-50; HORITA Co. Inc., California, USA) was used to synchronize the isokinetic dynamometer with the ultrasound scanner. Stroke survivors limbs (spastic x non-spastic) were

randomly selected during testing, whereas only the dominant limb was selected for testing in healthy subjects and was used as control. All images were analyzed in ImageJ software (National Institute of Health, USA).

RESULTS AND DISCUSSION

The MDC was obtained at different ankle joint angles between stroke survivors (range = -5° to -10°) and healthy subjects (range = -10° and -20°). There were no significant differences between groups (stroke survivors vs. healthy) for age (p=0.94), body mass (p=0.50) and standing height (p=0.90). No differences were observed for S tendon length when comparing the spastic to non-spastic limb at different joint angles (0°, p=0.09; 20°, p=0.23) in stroke survivors and when comparing the spastic limb to the control group (0°, p=0.56; 20°, p=1.00). Similar results were observed between the non-spastic and the control limbs (0°, p=1.00; 20°, p=0.67). During MDC, maximal S length was similar between spastic x non-spastic (p=0.37) between spastic x control (p=1.00) and between non-spastic and control (p=0.50) limbs, respectively. In MG tendon there were no differences between spastic and non-spastic limbs, spastic and control limbs or non-spastic and control limbs at both joint angles. Similar results were found during MDC with no significant between-groups differences for maximal tendon length.

Table 1. Soleus (S) and medial gastrocnemius (MG) tendon length of spastic, non-spastic and healthy (control) limbs at two different ankle joint angles at rest and during maximal dorsiflexor contraction (MDC) (mean ± SD).

	S	MG
0°		
Spastic	5.2 ± 1.11	20.8 ± 1.59
Non-spastic	6.3 ± 1.04	20.8 ± 1.70
Control	5.4 ± 1.60	20.0 ± 2.78
20°		
Spastic	4.7 ± 0.96	19.8 ± 1.94
Non-spastic	5.4 ± 0.88	20.1 ± 1.72
Control	4.9 ± 1.30	20.2 ± 2.44
MDC		
Spastic	6.2 ± 1.73	21.1 ± 1.68
Non-spastic	7.3 ± 1.70	21.5 ± 1.80
Control	6.1 ± 1.29	21.0 ± 3.13

No significant between-groups differences (p>0.05).

As spasticity is known to change the structure and function of the muscle-tendon unit, we expected to see differences between the spastic and non-spastic tendon length in stroke survivors. The fact that no difference was observed suggested that spasticity did not change both S and MG tendon length. However, as spasticity may also change the contralateral limb, reducing its range of motion and limb usage, a between-limbs similarity could perhaps be observed in stroke survivors. However, the fact that no differences were observed when comparing the control side of the healthy group to both the spastic and non-spastic limbs of the stroke survivors group suggests that spasticity does not change the tendon length in these two muscles, despite the different maximal dorsiflexion range of motion between the two groups.

The no-significant difference at different ankle joint angles between spastic and non-spastic limbs observed in this study was controversial with other studies that observed an increase in S tendon length in the spastic limb [6]. This longer length in the spastic side (6%) was related to the low level of loading which may have developed chronic adaptations and decreased the tendon stiffness. The similarity between the spastic limb and the control limb for S tendon length was observed on a previous study which compared stroke survivors and healthy subjects after a stretching treatment with no between-groups changes before and after intervention [7]. The fact that S and MG have different joint functions (monoarticular x biarticular) might have implications to tendon stiffness. While S tendon length ranged between 3-11 cm, MG tendon length ranged between 11-26 cm [8]. Therefore, different knee positions might increase or decrease tendon stiffness and power transmission to the bone, although our results that spasticity caused no differences in tendon structure suggest that no functional changes should be observed. Spasticity is multifactorial and neural in origin, a condition that occurs to the upper motor neuron located in the central nervous system that affects muscles and tendons in a fairly stereotypical manner depending on the amount and activity type imposed upon them [9]. Although decreased physical activity cause muscle-tendon deleterious adaptations, our results seem to indicate that there is no plantarflexor tendon structural changes in spastic limbs of stroke survivors.

CONCLUSIONS

Stroke survivors have no significant differences in S and MG tendon length determined by spasticity.

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