Altered contractile properties of the gastrocnemius muscle poststroke

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Submitted 21 July 2008; accepted in final form 20 October 2008

Gao F, Zhang L-Q. Altered contractile properties of the gastrocnemius muscle post stroke. J Appl Physiol 105: 1802–1808, 2008.—Spasticity, contracture and muscle weakness often occur together poststroke and cause considerable motor impairments to stroke survivors. The underlying changes in contractile properties of muscle fascicles are still not clear. The purpose of this study was to investigate the contractile property changes of the medial gastrocnemius muscle fascicles poststroke. Ten stroke survivors and ten healthy subjects participated in the study. The medial gastrocnemius fascicular length was measured at various combinations of ankle and knee positions using ultrasonography, with the muscle activated selectively using electrical stimulation. The stimulation intensity was kept constant across different ankle and knee positions to establish the active force-length relationship of the muscle fascicles. It was found that stroke survivors showed a shift of the force-length curve with a significantly shorter optimal fascicle length (33.2 ± 3.2 mm) compared with that of healthy controls (47.4 ± 2.7 mm) with \( P < 0.001 \). Furthermore, the width span of the fascicular force-length curve of stroke survivors was significantly narrower with steeper slopes than that of controls (\( P \leq 0.001 \)), suggesting reduced number of sarcomeres along the fascicles and/or reduced sarcomere length poststroke. Regression analysis showed that the medial gastrocnemius fascicular length of stroke survivors varied significantly less with ankle and knee flexions (\( P \leq 0.001 \)) than that of controls, suggesting shorter and stiffer muscle fascicles poststroke, which might be attributed to muscle architectural adaptation. This study showed that there are considerable changes in the contractile properties of muscle fascicles poststroke, which may contribute directly to the joint-level changes of decreased range of motion, increased stiffness, muscle weakness, and impaired motor functions in stroke survivors.

METHODS

Subjects

Ten male stroke survivors (age, 56.8 ± 6.8 yr; body mass, 85.4 ± 16.0 kg; height, 177.6 ± 5.2 cm; stroke history of 88.0 ± 52.6 mo with the right side impaired) and ten male healthy subjects (age, 45.1 ± 17.7 yr; body mass, 73.8 ± 12.7 kg; height, 175.0 ± 5.2 cm) without any neurological disorder participated in the study. The modified Ashworth score (2) at the ankle of the stroke survivors in this study was 2.6 ± 0.6. All subjects gave informed consent before participating in the study.

Experimental Procedure

An adjustable leg-foot linkage with six-axis force/torque sensors (JR3, Woodland, CA) mounted at both the knee and ankle joints was used to constrain the shank and foot (Fig. 1). The knee and ankle joint axes were aligned with the Z-axis (normal to the mounting surface of the sensor) of the corresponding JR3 sensors. Only the flexion moment at each joint was used in this study. The knee and ankle joint positions were adjusted systematically throughout the ranges of motion during the experiment. For both groups bipolar stimulation was applied at the motor point to activate the MG muscle selectively, with the motor point determined...
traction torque could be reached (peak torques ranging from 0.48 to
0.70 Nm). A Compex electrical stimulator was used to generate
stimulation delivered through small electrodes was used in this study
to reach selective activation of the MG muscle. Still, similar torque-
angle relationships were observed across the different levels of acti-
vations. The pattern of the torque-angle relationship obtained in this
study was consistent with those obtained under higher levels of
activation, as reported in the literature (30, 42). The duration of each
electrical pulse train was 2 s, and the interval between the pulse trains
was 3 s to minimize fatigue (Fig. 2). The stimulation amplitude was
adjusted for each individual subject to achieve a proper level of ankle
plantar flexion torque, strong enough for reliable measurement and
tolerated by the subject. The targeted MG muscle was activated
selectively to a steady level of contraction during each of the 2-s
stimulation.

The subject was seated upright on a custom chair with the thigh
secured using Velcro straps. Four knee positions, starting from full
extension with an increment of 30° flexion, were tested. At each knee
flexion, the ankle flexion was systematically varied between 20°
dorsiflexion and 45° plantar flexion, with an increment of 10° in
dorsiflexion and 15° in plantar flexion. Because stroke survivors had
reduced ankle range of motion, the tested ankle positions for stroke
survivors might be smaller than the range described above and were
adjusted individually if needed. The extreme ankle positions that
could be reached for the stroke survivors were 43 ± 6.1° in plantar
flexion and 9.9 ± 8.0° in dorsiflexion.

Ultrasound images were collected using a B-mode ultrasound
scanner with 12 MHz high-resolution linear array probe (M12L
matrix probe, GE LOGIQ-9). Working in the LOGIQView mode, the
probe was placed perpendicular to the skin and moved smoothly and
distally along the middle line of the MG. During the movement, the
probe was kept perpendicular to the skin under moderate pressure to
maintain proper contact between the probe and skin without deforming
the muscle significantly.

Each stimulation trial lasted 50 s, and there were about 10 stimu-
lation-induced contractions per trial. Longitudinal scan of the MG
using LOGIQView was conducted as the muscle reached a steady
level of contraction. Out of the 10 stimulation-induced contractions in
each trial, 3 ultrasound scans were performed randomly along the
same paths and the averaged values were used in further analysis.

Data Analysis

Joint torque signal processing. The torque signals were digitally
filtered with a fourth-order Butterworth low-pass filter (5-Hz cutoff
frequency). Torque change from immediately before the contraction
to the average of the middle 75% of the steady contraction window
were taken as the torque induced by the stimulation. Notice that
although the stimulation intensity was kept constant, the induced
torque might still fluctuate due to variations such as changes in the
baseline torque and in the muscle response to the stimulation. Outliers
of the peak torque signal defined as those out of the 75 percentile were
excluded from further analysis (36, 54).

Ultrasound images analysis. Ultrasound images were saved as
image files and analyzed in MATLAB (The MathWorks, Natick,
MA). As shown in Fig. 3, connective tissues surrounding the muscles
and fascicles were echogenic. The posterior and anterior muscle
aponeuroses met each other to form the musculotendon junction. The
fascicle length has been shown to be fairly consistent with small
variations along the length of the muscle in different regions (27). In
the present study, the length of fascicles at a single location (5 cm
proximal to the musculotendon junction) was measured (Fig. 3) (19).
The posterior and anterior pennation angles were measured as the
angle between the fascicle and the posterior and anterior aponeuroses,
respectively.

Active force-length relationship. The MG fascicular force $F$ was
determined across various ankle and knee positions as follows:

$$F = \frac{\tau}{MA \times \cos(\alpha)} \quad (1)$$

where $\tau$ is the ankle joint torque generated by selective activation of
the MG muscle, $MA$ is the moment arm determined using SIMM
(MusculoGraphics, Santa Rosa, CA), and $\alpha$ is the anterior pennation
angle. For each subject, the force $F$ was normalized to its peak value.
across the various fascicle lengths obtained at the various ankle and knee positions.

The relation between the normalized fascicular force (F) and the corresponding fascicle length (L) measured through ultrasonography was established by curve-fitting the scattered data points for each subject with the model proposed by Kaufman et al. (Eq. 2 in Ref. 17, \( \omega \) was a parameter that controls width), after evaluating a number of models for the force-length relation (3, 35, 50).

\[
F(L) = \exp\left(-\left(\frac{L^\omega - 1}{\omega}\right)^2\right)
\] (2)

For both groups, the peak of the fitted force-length curve and the corresponding optimal fascicle length were determined from the model. The shape of the fitted curve was characterized by the spanning width and slope. The spanning width was measured at specified tension levels, over the range corresponding to 70–90% peak force with an increment of 10% (Fig. 4). In addition, slopes were also determined at specified tension levels ranging from 70 to 90% peak force for both the ascending and descending limbs (Fig. 4). Group average was calculated across the tension levels ranging from 70 to 90%.

Multilinear regression was calculated with the knee (\( \theta_{\text{knee}} \)) and ankle (\( \theta_{\text{ankle}} \)) positions as the independent variables and the muscle fascicle length (\( l_{\text{mf}} \)) as the dependent variable for individual subjects:

\[
l_{\text{mf}} = a \times \theta_{\text{ankle}} + b \times \theta_{\text{knee}} + c
\] (3)

where \( a, b, \) and \( c \) represented the coefficients for the ankle angle, knee angle, and offset, respectively.

Statistical Analysis

ANOVA with repeated measures was performed on the dependent variables with the knee and ankle joint positions as the factors. Student’s t-test was used for comparison of the dependent variables between the stroke and control groups. The dependent variables include the optimal fascicle length of the force-length curve, the spanning width and slope of the force-length curve at a specified tension, the muscle fascicle length, and the coefficients of the multilinear regression. Statistical significance was set as \( P < 0.05 \). All statistics was done in SPSS (SPSS, Chicago, IL).

RESULTS

Compared with healthy controls, stroke survivors showed a left-shifted force-length curve with significantly shorter optimal fascicle length (Fig. 5; \( P \leq 0.001 \)), suggesting reduced number of sarcomeres along the fascicles. The optimal fascicle length was 47.4 ± 2.7 and 33.2 ± 3.2 mm (mean ± SD) for the normal controls and the stroke survivors, respectively (Fig. 5).
Compared with healthy controls, stroke survivors showed a significantly narrower spanning width of the active force-length relationship across the different levels of normalized tension ($P \leq 0.001$). With the bell-shaped pattern, the spanning width decreases with the increment of the normalized tension. Compared with healthy control, stroke survivors had a more gradual decrease of the spanning width with increasing normalized tension (Fig. 6; $P \leq 0.001$), reflecting the smaller fascicle length change.

Across various tension levels of the force-length curve, stroke survivors showed significantly steeper slopes for both the ascending and descending limbs compared with their counterparts of the control group ($P \leq 0.005$; Fig. 7), indicating faster rate of tension change with the fascicle length change and reduced number of sarcomeres along the fascicles.

At comparable joint positions, stroke survivors showed shorter muscle fascicle lengths compared with healthy controls (Fig. 8). For instance, with the knee at full extension and ankle at $0^\circ$ dorsiflexion, stroke survivors showed significantly shorter muscle fascicle length ($37.7 \pm 4.2$ mm) than that of the healthy controls ($55.6 \pm 8.2$ mm; $P < 0.001$). Across the ankle and knee positions, the muscle fascicle length ranged from $25.4 \pm 3.7$ to $68 \pm 5.7$ mm and from $18.4 \pm 6.8$ to $45.4 \pm 6.4$ mm for the healthy controls and stroke survivors, respectively (Fig. 8). However, the surface plot was generated based on extrapolated data, and one should be cautious interpreting the data points beyond the physical range.

The coefficients of multilinear regression in Eq. 3 showed altered dependence of the muscle fascicle length on the ankle and knee flexion positions post stroke, compared with that of healthy controls. The MG fascicle length of stroke survivors varied with ankle flexion significantly less than that of healthy control, as shown by the smaller ankle coefficient of the stroke survivors ($a = 0.24 \pm 0.06$ (mean $\pm$ SD)) compared with that of healthy control ($a = 0.37 \pm 0.08$), with $P = 0.001$. Similarly, the fascicle length of stroke survivors varied significantly less with knee flexion angle than that of the control (the knee coefficient $b$ was $-0.21 \pm 0.05$ and $-0.07 \pm 0.05$ for the control and stroke groups, respectively; $P < 0.001$). With full knee extension and $0^\circ$ dorsiflexion, the corresponding muscle fascicle length, or coefficient $c$ (intercept of the linear regression equation), was significantly lower in stroke ($37.4 \pm 4.2$
DISCUSSION

In vivo ultrasonic and biomechanical measurements were done systematically at various ankle and knee positions, combined with constant electrical stimulation across various ankle and knee positions, to evaluate changes in MG contractile properties characterized as force-length relationship post-stroke. Compared with healthy control subjects, patients post-stroke showed significant changes in the active force-length relation, including shorter muscle fascicles, narrower range of fascicle length change, and steeper slopes of the MG fascicle force-length relation.

The MG fascicle length of healthy subjects in this study falls in the range of MG fascicle length reported in the literature (1, 13, 16, 18, 21, 26, 32). For instance, Arampatzis et al. (1) reported a range from 37 to 67 mm across six ankle and knee angle combinations. Compared with the previous studies, a larger range of combined ankle-knee positions were used in the present study, which resulted in a larger range of change in muscle fascicle length for healthy control, ranging from 25.4 ± 3.7 to 68 ± 5.7 mm. Although Maganaris (26) reported a significantly smaller range of fascicular length change for the MG, ranging from 24 ± 4 to 39 ± 6 mm, it was measured under maximal voluntary contraction, which could shorten the MG fascicular length by 49% and reduced from 45 ± 2.3 mm to 23.4 ± 1.9 mm (27).

In this study, the force-length relation was characterized in the range of the whole ascending limb with an extension to 70% of the peak force in the descending limb. In the literature, Cutts et al. (7) showed that the gastrocnemius muscle operated across the range of the tension-length curve with the sarcomere length ranging from 1.0 μm (at 125° knee flexion and 43° plantar flexion) to 4.4 μm (at 13° knee flexion and 18° dorsiflexion) (7). On the other hand, Maganaris (26) evaluated the force-length characteristic of the MG muscle in vivo and showed a linear instead of a bell-shaped relationship between the fascicular length and force. The linear relationship might be associated with the limited range of muscle fascicle length change involved in the study and the assumption of constant load sharing between the lateral and medial heads across different test conditions (26). Sale et al. (42) evaluated the contractile properties of the whole group of the triceps surae muscles by stimulating the gastrocnemius and soleus muscles using large surface electrodes (cathode of 20 × 15 cm over the gastrocnemius and soleus and anode of 18 × 12 cm encircling the ankle). The contraction torque and joint angle relationship crossed the whole ascending limb and part of the descending limb with the optimal angle for ankle torque production being at 15° dorsiflexion (42). In the present study, the MG muscle was activated selectively at the motor point using small surface electrodes, and the range of the force-length curve covered the operational range of the MG fascicles during functional activities.

The present study showed that compared with normal controls, stroke survivors showed significant changes in the active force-length relation toward the shorter fascicle length, narrower range of the force-length relationship, and steeper slopes at both the ascending and descending limbs, which indicated reduced number of sarcomeres in series along the muscle fascicles. Considering that the optimal fascicle length of healthy control was 47.4 ± 2.7 mm as obtained in this study and the number of sarcomeres in series was about 17,600 for the normal human MG muscle (15), the corresponding sarcomere length was −2.7 μm (black thin line in Fig. 9), which was consistent with the range of optimal sarcomere length of 2.6–3 μm reported in the literature (26, 49). In contrast, assuming the same 17,600 sarcomeres in series, the corresponding sarcomere length of stroke survivors would be 1.9 μm (dashed and dash-dotted curves in Fig. 9), considerably shorter than that of healthy control. However, the assumption that the spastic muscle fascicles consist of the same number of sarcomeres in series as that in normal fibers may not hold, considering that the number of sarcomeres is highly adaptable to changes in muscle loading (14, 46, 51). Shah et al. (43) reported that a significant decrease in sarcomere number was observed in shortened soleus in rats with immobilization, ranging from 12 to 26% (43). Tabary et al. (45) reported a 40% reduction of number of sarcomeres due to immobilization at shortened muscle length. Some studies have compared the resting ankle positions between these two groups with zero-resistance torque, and stroke survivors have demonstrated a slightly plantar flexed resting ankle position compared with healthy control (Gao F, Grant TH, Roth EJ, Zhang LQ, unpublished observations). The shift of the fascicle force-length relation, including the optimal fascicle length toward the shorter length as reported in the present study indicated a reduction of the number of sarcomeres in series. Potentially, a reduction in the number of sarcomeres would bring sarcomere length back to near the optimal sarcomere length (51) and match the left-shifted fascicular force-length curve observed in stroke survivors in this study. Hypothetically, a 30% reduction in the number of sarcomeres in series would make the length of sarcomere near the optimal length (the dashed curve in Fig. 9).
The potential reduction in the number of sarcomeres in series may be tangled with sarcomere length change post stroke. A better understanding of the sarcomeric adaptation in spastic muscles poststroke has direct clinical relevance, such as in understanding the mechanisms underlying potentially increased fiber tension and stiffness in spastic muscles and in evaluating tendon lengthening operations (8). It may also provide guidance to rehabilitation treatment such as stretching a joint with spasticity/contracture, which may affect the sarcomere operating point on the force-length curve. Further studies at the muscle fiber and sarcomere levels need to be carried out to examine the relationship between the muscle architectural and biomechanical changes at the joint and fiber/sarcomere levels (53).

By using a multilinear regression, we have shown that for both stroke and control groups both knee and ankle positions affect the fascicular length of the biarticular MG muscle. The significantly smaller coefficients (in absolute value) for stroke survivors indicates that they have smaller change in muscle fascicle length than that in control, given the same amount of change of knee/ankle flexion angles. The significant smaller coefficients (in absolute value) for stroke survivors agree with the shorter fascicle length and narrow span of the force-length curve reported above and also indicate higher stiffness of the spastic muscle fascicles. This agrees with the reported increased ankle joint stiffness poststroke (6) and with the reported stiffer and shorter spastic muscle cell compared with control (9). The stiffness increase may be attributed to structural changes. For instance, in a study on the spastic control (9). The stiffness increase may be attributed to ultrastructural changes. For instance, in a study on the spastic control (9).

There are limitations with the present study. First, LOGIQView was good for recording relatively larger field of view but care needs to be taken to obtain the images with well-controlled linear movement. Second, the intensity of electrical stimulation depended on the skin condition and on locating the muscle motor point accurately. Skin movement across the different joint positions might affect the stimulation intensity to the motor point accurately. Skin movement across the different joint positions might affect the stimulation intensity to the muscle and change the fitted force-length curve. Third, in determining the MG muscle force, we assumed that the force was completely transmitted through tendon. However, the myofascial linkages might also affect the force transmission from muscle fascicles to bone (25). Fourth, the intensity of electrical stimulation applied on individual subjects varied from person to person to elicit plantar flexion torque within the subject’s comfort limit, which might cause inconsistent contractions across the subjects. However, the muscle activation level in this study was much lower than maximal voluntary contraction with the typical peak torque at \( \sim 0.4 \text{--} 0.8 \text{ N} \cdot \text{m} \), which was similar across the subjects. Under the low levels of activation, the change in fascicle length from the resting length was rather small [around 1\text{--}2\% assuming the maximum torque generated by MG is 50 N\cdot m and the shortening of fascicle length is 49\% under MVC (26)], and the difference in fascicle length between the two groups due to the different activation levels would be negligible. Lastly, the data and results were obtained from the MG muscle only, which may not be true for other muscles.

In summary, this study showed that there were considerable changes in muscle contractile and architectural properties post-stroke, including shorter muscle fascicles, smaller range of fascicle length variation, and steeper slopes in the active force-length relation. Quantitative evaluations of the muscle contractile properties may help us gain insight into the mechanisms underlying motor impairments post stroke and other neurological disorders and develop more effective rehabilitation treatment and outcome evaluations.

**REFERENCES**


