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MUSCLE VOLUME AS A PREDICTOR OF MAXIMUM FORCE GENERATING ABILITY IN THE PLANTAR FLEXORS POST-STROKE

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Abstract

Introduction—Post-stroke muscle weakness is commonly thought to be the result of a combination of decreased voluntary activation and decreased maximum force generating ability (MFGA). We assessed the ability of muscle volumes obtained using magnetic resonance imaging (MRI) to estimate the MFGA of the plantar flexor muscle group in individuals post-stroke.

Methods—MRI was used to measure muscle volume for the plantar flexor muscle group for 17 individuals with post-stroke hemiparesis. A modified burst superimposition test was used to measure MVC and predict the MFGA of the plantar flexors.

Results—While muscle volume obtained via MRI provided information on the overall size of muscle, it overestimated the force generating ability of the paretic plantar flexors.

Discussion—Results suggest that MRI-derived muscle volume underestimates the functional impairment in individuals post-stroke. Interestingly, the central activation ratio had a strong relationship with the maximum volitional force of contraction.

Keywords

Magnetic Resonance Imaging; Stroke; Muscle Strength; Plantar Flexors; Electrical Stimulation

Introduction

Muscle weakness is a common impairment for chronic stroke survivors; it is a limiting factor in post-stroke gait ^{1,2} and is often characterized by lower forces during maximal volitional contractions on the affected side ^{3,4}. Weakness in the plantar flexor muscle group, in particular, is associated with decreased walking speed in stroke subjects ⁵. Post-stroke muscle weakness is commonly thought to be the result of a combination of decreased voluntary activation of the muscle due to nervous system impairment and decreased

maximum force generating ability (MFGA) due to structural and morphological changes within the muscle. Discrimination of the contributions to weakness by muscle atrophy versus activation failure is important for determining subject-specific rehabilitation protocols^{6,7}. In particular, individuals with considerable activation deficits may benefit more from an intervention designed to increase volitional control of muscles, such as biofeedback or EMG-triggered electrical stimulation⁸. In contrast, a subject with significant peripheral muscle weakness and minimal central activation impairment may benefit more from rehabilitation focused on strength training⁹ using volitional or stimulation-assisted strengthening.

Various techniques have been used to estimate the volitional activation and MFGA of a muscle or muscle group^{6,10–12}. In particular, the burst superimposition technique has been used at maximal^{13–15} and submaximal volitional effort^{6,7,16} in the quadriceps of healthy^{6,7}, ACL injured,¹³ and elderly populations^{7,10}, but it can overestimate muscle activation at submaximal activation due to incomplete activation of muscle by the stimulation during the test¹¹. Flynn and colleagues demonstrated that when an adjustment equation is applied to the burst superimposition technique to account for submaximal activation, this method has the greatest accuracy and reliability in healthy subjects compared to other commonly used techniques (e.g., twitch and doublet interpolation)¹¹.

In addition to tests that directly measure the force a muscle can produce, such as burst superimposition, imaging techniques have also been used to relate the size of muscle to its force generating ability^{17,18}. In the past, techniques such as dual-energy X-ray absorptiometry (DXA)^{19,20} and computed tomography (CT) have been used to describe muscle atrophy in the post-stroke population^{21,22}. These methods, however, often do not report individual muscles and may be undesirable, as they expose the patient to radiation²³. Previous studies have also used magnetic resonance imaging (MRI) as a method for measuring muscle size.^{24–26} MRI-derived muscle volume has been shown to be preferable to anatomical cross-sectional area for evaluating the force-size relationship of muscle¹⁷, but the ability of muscle volume alone to explain variations in force is diminished with age¹⁸. Recent evidence has suggested that MRI-derived muscle volume relates poorly to volitional strength in the plantar flexors of a post-stroke population,³ but only a few studies have investigated the use of MRI to evaluate the force-producing capability in bilateral ankle plantar flexors of individuals post-stroke.^{3,27}

To date, no study has evaluated the relationship between MRI muscle volume and the MFGA of a muscle or muscle group in a post-stroke population. The objective of this study, therefore, is to assess the ability of MRI to estimate the MFGA in a post-stroke population. We hypothesize that, while MRI will show volume deficits in the plantar flexor muscles of the paretic limb, it will not provide an accurate estimation of the relative MFGA between limbs compared to a more direct measurement method.

Methods

Seventeen subjects post-stroke (15 men, 60.7 ± 8.9 yrs) were recruited to participate in this study. All subjects were at least 6 months post-stroke and signed informed consent forms approved by the Human Subjects Review Board at the University of Delaware.

MR images were collected and analyzed using previously described methods²⁷. Axial MR Images were acquired for both legs using a 1.5 T Signa LX scanner (GE Medical, Milwaukee, WI). During scanning, subjects lay supine with their feet taped together at the toes to limit any movement during the scan period and maintain neutral hip rotation. Two overlapping images were taken for the lower leg using a repetition time of 450 ms, echo time of 10 ms, slice thickness of 10 mm, and a space between slices of 11.5 mm. The 2 consecutive regions were determined to overlap when the image coordinates (representing distance along the limb) overlapped. IMOD software (University of Colorado, Boulder, CO) was used to manually trace the boundaries of the soleus (SOL), medial gastrocnemius (MG), and lateral gastrocnemius (LG) muscles over the entire muscle length (see Ramsay et al., 2011 for details).²⁷ Cross-sectional areas were calculated using a trapezoidal integration algorithm.²⁷ After adjusting the pixel threshold for fat suppression^{28–30}, volume was calculated by summing the cross-sectional areas multiplied by the slice thickness over the length of the muscle.

Muscle strength was tested using the burst superimposition test.¹¹ Subjects lay supine on a KIN-COM III dynamometer (Chattecx Corp, Chattanooga, Tennessee) with the knee in extension and ankle at neutral. Velcro straps were used to hold the foot and shank in position. Restraints were placed on the shoulders of the subject to ensure that all forces were directed into the transducer and not lost to body displacement. An electrical stimulation burst (600 μ s pulse duration, 100 ms train duration, 135 V, 100 Hz train) (Grass Technologies, Warwick, RI) was delivered while subjects produced maximum volitional force. Predicted maximum force generating ability (MFGA) using the burst superimposition test was calculated using the following equations:

$$F_{total} = MVC + F_{stim} \quad (1)$$

$$MFGA = F_{total} \times C \quad \text{where} \quad C = \frac{1}{\left(1 + \frac{\%Error}{100}\right)} \text{ and} \quad (2)$$

$$\%Error = 105 \left(\frac{MVC}{F_{total}} \right)^3 - 157 \left(\frac{MVC}{F_{total}} \right)^2 + 103 \left(\frac{MVC}{F_{total}} \right) - 52.0752$$

where MVC is the volitional force produced by the subject and F_{stim} is the additional force produced by stimulation. The error adjustment¹¹, C , is similar to methods previously used for the quadriceps^{6,7} and was applied to the MFGA prediction to account for incomplete activation of the muscle with the burst.

Data Analysis

The central activation ratio (CAR) is a measure of the level of volitional muscle activation at a given volitional effort, and is defined as the ratio of volitional force (MVC) to the maximum force generating ability of a muscle or muscle group (MFGA) (Eq. 3).

$$CAR = \frac{MVC}{MFGA} \quad (3)$$

It is accepted traditionally that the physiological cross-sectional area of a muscle (PCSA) is related to its maximum force producing capabilities through specific tension³¹. Thus,

$$Force = PCSA \times ST \quad (4)$$

Where *Force* [N] is the maximum force a muscle can produce, *PCSA* [cm²] is the physiological cross-sectional area of the muscle, and *ST* [N/cm²] is the specific tension of a muscle. If we substitute the relationship between muscle volume and PCSA into this equation we get:

$$Force = \frac{volume \times \cos\Phi}{l_{fiber}} \times ST \quad (5)$$

where *Volume* [cm³] is the volume of a muscle, Φ [°] is the pennation angle of the muscle, and *l_{fiber}* [cm] is the muscle optimal fiber length. If we then consider that for a group of muscles, the maximum torque of the muscle group becomes the sum of each individual muscle's maximum force multiplied by the respective moment arms we arrive at:

$$\Sigma MFGA_{torque} = \Sigma \frac{Volume \times \cos\Phi}{l_{fiber}} \times ST \times MA - F_{coactivation} \quad (6)$$

where *MFGA_{torque}* [N·cm] is the maximum torque generated at a joint by a group of muscles, *MA* [cm] is the moment arm for each muscle, and *F_{coactivation}* is the reduction in net torque output due to co-activation of the antagonist muscles. Finally, by taking the ratio of paretic to non-paretic *MFGA_{torque}* and assuming that the optimal fiber length, pennation angle³², specific tension, and moment arm of each muscle is the same between limbs, we are able to eliminate muscle-specific parameters that we are unable to measure through our MRI or burst superimposition techniques. Additionally, we assume that co-activation of antagonist muscles does not significantly change the ratio of paretic to non-paretic torque³ to compute:

$$\frac{\Sigma MFGA_P}{\Sigma MFGA_{NP}} = \frac{\Sigma Volume_P}{\Sigma Volume_{NP}} \quad (7)$$

where the subscripts *P* and *NP* denote the paretic and non-paretic limbs. Thus, from the equation above, the ratio of MFGA between limbs is equal to the ratio of the total muscle volume³³ (regardless of individual muscle volumes), given similar muscle parameters between limbs of an individual. Assuming that the CAR measured through the adjusted

burst superimposition test represents the CAR for the entire muscle group tested, we can substitute muscle volume measured through MRI (*Volume*) for *MFGA* into Eq. 3 to obtain an alternate equation for calculating the ratio of MVCs between limbs:

$$\frac{MVC_P}{MVC_{NP}} = \frac{CAR_P}{CAR_{NP}} \times \frac{\Sigma Volume_P}{\Sigma Volume_{NP}} \quad (8)$$

MVC, MFGA, CAR, and MRI-derived muscle volume measures were analyzed. Furthermore, the paretic to non-paretic ratio was calculated for each of these variables. The relationship of paretic to non-paretic central activation ratio versus paretic to non-paretic MVC ratio and paretic to non-paretic MRI-derived muscle volume versus paretic to non-paretic MVC ratio were examined.

Statistical Analysis

Normality of the data was shown using the Lilliefors test, and parametric statistics were used. Paretic versus non-paretic comparisons were evaluated using a paired *t*-test. Additionally, comparisons between outcome variable ratios were also evaluated using a paired *t*-test. A Pearson product-moment correlation and linear regression were used to evaluate the relationship of paretic to non-paretic CAR and Volume ratios with paretic to non-paretic MVC. Error bars represent standard error of the data.

Results

Maximum volitional force of contraction, maximum force generation using the burst superimposition test, and central activation ratio were compared for paretic versus non-paretic limbs (Figure 1). Average paretic limb MVC plantar flexion torque was approximately 41% of the non-paretic MVC ($p < 0.001$) (Figure 1). Average paretic limb MFGA was 65% of the non-paretic MFGA ($p < 0.001$). The average CAR of the paretic limb was 61% of the non-paretic limb ($p < 0.001$).

Total plantar flexor volume (i.e., the sum of the SOL, MG and LG) was determined from MRI for the paretic and non-paretic sides (Figure 1). The volumes on the paretic side were observed to be $80 \pm 10\%$ of those on the non-paretic side ($P < 0.001$). When muscle volume was substituted for MFGA (cf. Eq. 3), the estimated paretic to non-paretic MVC was 1.3 ± 0.3 times greater ($P = 0.036$) than the experimentally measured paretic to non-paretic MVC.

Paretic to non-paretic CAR was significantly correlated with MVC of the plantar flexor muscles ($P < 0.001$, $R^2 = 0.91$) (Figure 2). No correlation was found between paretic to non-paretic muscle volume and MVC between limbs ($P > 0.1$).

Discussion

The objective of this study was to assess the ability of MRI to estimate the MFGA in a post-stroke population. Paretic to non-paretic MRI-derived volume was found to be significantly different from the paretic to non-paretic MFGA. Additionally, paretic to non-paretic CAR

was found to be highly correlated with paretic to non-paretic MVC ($P < 0.001$) while paretic to non-paretic volume had no correlation with paretic to non-paretic MVC.

The 0.41 ratio of paretic to non-paretic MVC values found in this study are consistent with previous studies that reported paretic plantar flexor muscle strength was 30-38% of the non-paretic side.^{3,4} The paretic side plantar flexor CAR was 0.39, similar to previously reported data of 0.49.³ In contrast, our results showed greater activation impairment on the non-paretic side (0.66 CAR) than previously reported (0.97 CAR).³ We believe that lower non-paretic side CAR in our study can be attributed partially to the use of the adjusted burst superimposition method as opposed to the twitch interpolation method used by Klein and colleagues³. The burst superimposition method has been shown to be more sensitive to activation impairments than either twitch or doublet interpolation, particularly at higher levels of activation.^{10,12} Furthermore, the adjustment applied to the burst superimposition test used in our study has been shown to provide even more sensitivity to impairment than a standard burst superimposition test in healthy subjects¹¹ and to be more reliable than a standard burst superimposition test in post-stroke populations³⁴.

The muscle volume results from this study are also consistent with previous data.³ Total plantar flexor volume was found to be greater in the non-paretic limb, though it is unknown if this difference is due to atrophy in the paretic limb, hypertrophy in the non-paretic limb, or a combination of both. In our study, only the medial and lateral gastrocnemius and soleus muscles were used in the plantar flexor volume calculations. We believe that exclusion of the deep plantar flexors and fibularis muscles is justified, as no difference was found previously between paretic and non-paretic limbs in the volume of the deep plantar flexors and fibularis muscles³. Based on this finding, inclusion of the additional calf muscles would have caused the volume of the paretic and non-paretic limbs to be more similar to each other than reported in our study and would have served to exacerbate differences between MRI and CAR or MVC found in this study.

We hypothesized that if the force-generating ability of a muscle is directly proportional to its volume, then the ratio of the paretic to non-paretic MFGA of a muscle group should be equal its paretic to non-paretic volume. In reality, the use of MRI-derived volume as a substitute for MFGA resulted in a significant difference between estimated and measured paretic to non-paretic MVC. This suggests that either the adjusted burst superimposition test overestimated the CAR of the paretic limb compared to the non-paretic limb, or the MRI muscle volumes overestimated the force producing capabilities of the paretic limb relative to the non-paretic limb. To determine if the adjusted burst superimposition test overestimated the CAR of the paretic limb or if the MRI muscle volumes overestimated the force producing capabilities of the paretic limb, we hypothesized that individuals with greater paretic to non-paretic atrophy and activation deficits would have a lower paretic to non-paretic MVC. In other words, individuals with lower paretic to non-paretic MRI-derived volume would have lower paretic to non-paretic MVC. This theory is supported partially by the predicted CAR, as calculated from the estimate of MFGA obtained using the burst superimposition test (MFGA). A strong positive correlation between paretic to non-paretic CAR and paretic to non-paretic MVC suggests that subjects who have lower volitional activation in their paretic limb compared to their non-paretic limb also have lower volitional

force in their paretic limb relative to their non-paretic limb. The lack of significant correlation between paretic to non-paretic volume and paretic to non-paretic volitional force, however, suggests that relative atrophy assessed through MRI muscle volumes is a poor predictor of the relative volitional force a subject post-stroke can produce, as has been suggested previously.³

While PCSA is a major determinant of the force generating ability of a muscle, several factors can influence muscle force, such as muscle quality, architecture changes, and specific tension. To eliminate the influence of non-contractile tissue on muscle volume calculations, a pixel-threshold method was used in this study to distinguish between adipose tissue and lean skeletal muscle.^{27,28} Beyond fatty infiltration, changes in the contractile tissue itself, as well as differences in architecture, such as reduced muscle fascicle length and changes in pennation angle³⁵, can affect the specific tension of a muscle. Indeed, it has been shown previously that changes occur in passive mechanical properties of the gastrocnemius muscle post-stroke³⁵. These factors were not detectable using our MRI technique and perhaps merit investigation in future studies. While some have argued that specific tension differs for muscle groups³⁴, it is reasonable to consider it to be consistent between limbs. Unfortunately, the effects of paresis on specific tension are currently unknown.

Previous studies have demonstrated a relationship between muscle volume and force generation in healthy young and elderly populations^{17,33}, however, it has been shown that this relationship is much weaker in older adults¹⁸. Our results showed no relationship between MVC or MFGA with total plantar flexor volume for individuals post-stroke. This is interesting to note, as a previous study¹⁷ has shown a relationship between muscle volume and MVC in elbow flexors, with R-squared values of .32 (men) and .47 (women) in elderly populations. This suggests that muscle-specific parameters which determine force producing ability of muscle (i.e. specific tension) may vary more greatly between individuals post-stroke and, ultimately, that the measurement of muscle volumes via MRI may not accurately describe either the volitional force or MFGA of a muscle or muscle group for post-stroke populations. While studies have examined the relationship of MRI muscle volume with volitional force in individuals post-stroke, this study examines the relationship between MFGA and muscle volume. Clinically, if a subject lacks the strength to perform a desired task, clinicians must decide how much of the weakness is central or peripheral and design rehabilitation treatments appropriately. For example, individuals with considerable activation deficits may benefit more from interventions such as biofeedback or EMG-triggered electrical stimulation⁸, which are designed to increase volitional control of muscles, whereas a subject with significant deficits in MFGA and minimal activation impairment may benefit more from strength training³⁴. Interestingly, the CAR calculated using the burst superimposition technique described in this study had a strong relationship with maximum volitional contraction force, and may be a more accurate, useful, and cost effective technique than MRI for measuring muscle weakness and MFGA.

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Abbreviations

MFGA	maximum force generating ability, represents the force the muscle is capable of generating with full muscle activation
MVC	force of the volitional contraction
CAR	central activation ratio of a muscle or muscle group
MFGA_{adj}	maximum force generating ability calculated using the adjusted burst superimposition technique
MFGA_{burst}	maximum force generating ability calculated using the burst superimposition technique
PSCA [cm²]	physiological cross-sectional area of the muscle
ST [N/cm²]	specific tension of a muscle
φ [°]	pennation angle of the muscle
<i>l_{fiber}</i> [cm]	muscle fiber length

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Figure 1.

(a) Maximum voluntary (MVC), maximum predicted torque (MFGA), (b) Volitional activation (CAR) and (c) muscle volumes (MG – Medial Gastrocnemius, LG – Lateral Gastrocnemius, SOL – Soleus) for the paretic and non-paretic plantar flexor muscles in seventeen subjects post-stroke. * indicates significance ($P < 0.001$), # indicates significance ($P < 0.05$)

Figure 2.
Paretic to non-paretic MVC ratio versus paretic to non-paretic volitional activation (CAR) and muscle volume (Volume) outcome ratios for 17 subjects post-stroke