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Investigation of hand muscle atrophy in stroke survivors

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Abstract

Background—Weakness is often profound in the contralesional hand after stroke. Relative contributions of various neural and mechanical mechanisms to this impairment, however, have not been quantified. In this study, the extent of one potential contributor, muscle atrophy, was noninvasively assessed in index finger musculature using ultrasonographic techniques.

Methods—Twenty-five stroke survivors (45–65 years old) with severe hand impairment resulting from a stroke occurring 2–4 years prior participated, along with 10 age-matched control subjects. Muscle cross sectional area and thickness were geometrically measured from ultrasound images on both limbs of participants.

Findings—Muscle size on the paretic limb of stroke survivors was smaller for all 7 hand muscles investigated. An average difference of 15% (SD 4) was seen for muscle cross sectional area and 11% (SD 2) for muscle thickness, while the difference between the dominant and non-dominant limbs for control subjects (6% (SD 2) and 1% (SD 4) for the muscle cross sectional area and muscle thickness, respectively) was not significant.

Interpretation—Although muscle atrophy was detected in the paretic limb following stroke, it is not explanatory of the marked impairment in strength seen in this stroke population. However, other alterations in muscle morphology, such as fatty infiltrations and changes in fiber structure, may contribute to the emergent muscle weakness post-stroke.

Keywords

hand; stroke; muscle; atrophy; ultrasound

Introduction

Weakness in the contralesional limb is a common finding in stroke survivors. Strength deficits in the hands can be especially profound, as evidenced by studies of finger, grip and pinch strength. In fact, studies investigating the index finger indicate losses of greater than 75% (Cruz et al., 2005). While excessive coactivation and an inability to fully activate these muscles contribute to this weakness, muscle atrophy may also play a role.

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The primary characteristic of muscle disuse atrophy is a reduction in muscle fiber size (Evans and Campbell, 1993). It has been well established that muscle physiological cross sectional area is directly proportional to the corresponding muscle's force generating ability (Zajac, 1989). It is therefore a logical inference that as muscle CSA is reduced (such as the case with muscle atrophy) this condition will result in a lowered force production.

The reduction in muscle mass of older adults, or sarcopenia, is very common consequence of the aging process. Strength losses as a result of sarcopenia have been shown to affect the lower extremity muscles to a much greater extent than the upper extremity muscles (Frontera et al., 1991, Lynch et al., 1999) and this phenomenon is believed to result from the selective disuse or modified postural/locomotive duties of these muscle groups with age (Macaluso and De Vito, 2004). For example, as the muscles of the lower extremity weaken, individuals may employ the use of the upper extremity to assist rising from or lowering into a seated position. Additionally, the extent of this atrophy varies with the structure and function of a muscle. While both Type I and Type II muscle fibers can be affected, the stronger fast-twitch Type II muscle fibers appear to be preferentially prone to atrophy in older adults (Jones and Round, 1990).

Muscle atrophy has also been shown to be a consequence of stroke. Indeed, atrophy in the hemiparetic leg has been observed, both in needle biopsy (Dattola et al., 1993) and imaging studies (Klein et al., 2010, Slager et al., 1985). Likewise, similar findings have been reported for paretic muscles of the upper extremity controlling gross movements, such as the biceps brachii (Blijham et al., 2006, Hu et al., 2007, Ploutz-Snyder et al., 2006) and the triceps (Ploutz-Snyder et al., 2006). However, more distal muscles, particularly those controlling the hand, have not been studied in detail. These muscles may be particularly susceptible to changes following stroke due to the heavy cortical innervation of their motoneurons. Additionally, strength deficits in the fingers appear to be non-uniform, with greater loss in extension than in flexion (Cruz et al., 2005, Kamper et al., 2006).

Thus the goal of this study was to quantify the extent of gross muscle atrophy in the hand following stroke. In particular, index finger muscle size was measured *in vivo* by means of ultrasonography and geometrical measurements in the sagittal and transverse planes. The index finger was highlighted due to its functional importance and the prior characterization of its strength deficits after stroke (Cruz et al., 2005). We hypothesized that atrophy of index finger musculature would be substantially greater in stroke survivors than in neurologically intact control subjects. We further hypothesized that within the paretic hand, the intrinsic and long extensor muscles would be atrophied to a greater extent than the long flexor muscles in stroke survivors, in accordance with reported findings of weakness and diminished movement.

Materials and Methods

Subjects

Index finger muscle geometry was measured to assess the extent of muscle atrophy in subjects with chronic hemiparesis subsequent to stroke. Twenty-five subjects with severe hand impairment, classified as Stage 2 or Stage 3 on the Stage of Hand component of the Chedoke-McMaster Stroke Assessment scale (Gowland et al., 1995), participated. All of these subjects (16 male and 9 female) had experienced a stroke between 2–4 years prior to enrollment in the study. Thirteen stroke survivors had primary involvement of the right side of the body and 12 had involvement of the left side. Sixteen subjects had primary impairment of the formerly dominant hand, while 9 subjects had primary impairment of the non-dominant hand. Hand strength deficits in this population were quantitatively assessed through clinical measures of both maximum voluntary power grip force, using a

dynamometer (JAMAR 5030J1 Hand Dynamometer, Clifton, NJ, USA) and finger extension force, using a digital force gauge (Mark-10 Corp. MG200, Copiague, NY, USA) for both limbs.

Ten healthy, age-matched control subjects (7 male and 3 female) were included in this study to gauge the extent to which muscle size varies between the dominant and non-dominant side for the upper extremity. All participants were between 45 and 65 years of age to minimize the possible confounding effects of sarcopenia. Written, informed consent was obtained from all subjects in accordance with the Institutional Review Board of Northwestern University prior to involvement in the study.

Protocol

Ultrasonography (GELogic9, M12L, GE Healthcare Wauwatosa, WI, USA) was used to measure the geometry of the muscles controlling the index finger. Muscle ultrasound has been previously shown to be a reliable method to measure muscle thickness and CSA (Reeves et al., 2004, Reimers et al., 1998, Sanada et al., 2006). The technique has been shown to have a test-retest correlation of 0.98–0.99 (Reeves et al., 2004, Reimers et al., 1996) and 0.99 correlation with MRI (Reeves et al., 2004). Ultrasound has the advantage of real-time feedback, which enabled localization of the proper muscle/muscle compartment through viewing the displacement of muscle fibers in response to isolated muscle contraction or imposed movement of the joint or joints. The musculoskeletal transducer probe was positioned over the muscle belly and then moved to determine the maximal muscle parameter of interest for each of the muscles of the index finger: flexor digitorum superficialis (FDS), flexor digitorum profundus (FDP), extensor digitorum communis (EDC), extensor indicis (EI), first dorsal interosseous (FDI), first palmar interosseous (FPI), and lumbrical (LUM).

To better estimate the extent of muscle atrophy following stroke, two different muscle parameters were evaluated. The first was the muscle cross sectional area (CSA), measured from images taken in the transverse anatomical plane of the forearm and hand. The probe was positioned such that the image plane was perpendicular to the long axis of the muscle. The second parameter was muscle thickness, measured as the muscle depth from sagittal plane images taken along the long axis of the muscle. Both parameters were measured, as while CSA is more traditionally used as a description of atrophy, muscle thickness may offer a simpler and timelier approach for obtaining equally viable information about muscle geometry post-stroke. Once the location for maximal muscle geometry was visually determined, participants were given auditory and visual cues to begin cyclical contraction/relaxation of the muscle of interest. They were instructed to end in the relaxed state, which was maintained for 1–2 sec. During this period of cyclical muscle activation, video clips were recorded in both the sagittal plane, to estimate muscle thickness, and in the transverse plane, to estimate muscle cross sectional area (CSA). If a subject was unable to voluntarily isolate and contract the desired muscle, manual ranging of the digit was conducted to elicit the desired movement. Imaging commenced on the dominant (control group) or the non-paretic (stroke group) side to determine the appropriate settings, such as the location, focus, and depth of recording, for the ultrasound system, thus helping to guide imaging on the opposite limb. Each video clip was at most 5 seconds long, with a period of 1–2 sec of muscle relaxation at the beginning and end of the clip (frame rate = 50 frames/second). This length of time was determined to be sufficient in localizing the desired muscle and distinguishing muscle compartments of the index finger from muscle compartments of the other digits.

To test the precision and reliability of these techniques, data were collected for three control subjects on five separate days prior to the start of this study. The variance in muscle

thickness and CSA over these five visits was calculated for each of the seven muscles examined.

Data Processing

Image files for geometric muscle measurements were extracted off-line from the movie clip data recorded during the experiment. Individual frames of the movie capturing the initial resting state of the muscle were analyzed with a custom graphical user interface (GUI) designed in MATLAB (MathWorks, Natick, MA, USA). The interactive display provided a means to view both the image file and the original movie clip simultaneously, reinforcing accurate muscle distinction and measurement. The measurement scale was calibrated to the recording depth of the movie for each muscle investigated.

For images obtained in the sagittal plane, maximal muscle thickness was measured perpendicular to the grain of the muscle fibers (Fig. 1A). In order to ensure consistent and accurate data, five consecutive measurements were obtained for each muscle. To reduce user error, a standard deviation of the measurements of greater than 5 mm triggered a warning to the analyst and automatically discarded the measurements. The process was repeated until successful measurements were obtained.

A similar procedure was employed for images obtained in the transverse plane to determine the CSA of the muscle. Each muscle was assumed to have an elliptical shape in this plane (Fig. 1B). The major (a) and minor (b) axes of this assumed ellipse were measured, and subsequently used to calculate the estimated elliptical area:

$$\text{Elliptical Area} = \frac{1}{4} ab\pi$$
Equation
1

Again, if the standard deviation of the measurements for either axis was greater than 5mm then the process was repeated until valid results were obtained.

Statistical Analysis

For the dependent variables of interest in this study, (muscle thickness and muscle CSA), a Multivariate ANOVA (MANOVA) was first performed for each group (stroke and control) using SPSS software (SPSS Inc., Chicago, IL, USA) to determine if the independent variables of muscle type (Muscle), or non-dominance/paresis (Hand) impacted the output. Muscle (7 levels) and Hand (2 levels) were the within-subject factors. Subsequently, findings of significance according to the Wilks' lambda value led to running separate repeated measures ANOVAs (RMANOVAs) for each of the two dependent variables. Finally, for a comparison between groups, an RMANOVA was performed using normalized values (paretic normalized to non-paretic side and non-dominant normalized to dominant side) where Muscle (7 levels) was the within-subject factor and Group (2 levels) was the between-subject factor. *Post hoc* Tukey tests were conducted to determine statistically distinct levels of the main effects. A Bonferroni correction for the 3 separate analyses was implemented, such that the significance level was set to 0.0167 ($\alpha/3$) yielding an overall significance level of $\alpha = 0.05$.

Results

To ascertain the extent of muscle atrophy between limbs, estimates of muscle thickness and CSA were obtained for all seven muscles involved in motor control of the index finger in 25 subjects who had sustained a stroke resulting in severe, chronic hand impairment and 10 subjects with no known neuromuscular deficits. The stroke subjects had severe weakness in the paretic hand, as characterized by an 82% deficit in grip strength and an 88% deficit in

finger extension strength in comparison to the non-paretic hand. No significant differences were present in either grip strength or extension force for the non-paretic hand in stroke subjects and the non-dominant and dominant hand in control subjects ($p < 0.001$).

The MANOVA for the stroke group revealed a significant effect on muscle geometry for both independent variables, Hand and Muscle, (Wilks' $\lambda < 0.001$) but not for the interaction term Hand—Muscle (Wilks' $\lambda = 0.522$). Thus, muscle size was reduced on the paretic side, but there was no significant difference in the relative atrophy of the different muscles. The MANOVA for the control group also revealed a significant effect on muscle geometry for the independent variable Muscle (Wilks' $\lambda < 0.001$), but not for Hand (Wilks; $\lambda = 0.411$) or the interaction term Hand-Muscle (Wilks' $\lambda = 1.000$). Hence, for the control subjects, hand dominance did not significantly affect muscle size. Muscle size was not significantly different between the dominant hand in the control subjects and the non-paretic hand in the stroke subjects for muscle thickness ($p = 0.112$) or CSA ($p = 0.639$).

Consequently, RMANOVAs were performed for each of the dependent variables for the independent variables of significance (Table 1). A decrease in muscle CSA was evident in all muscles of the paretic index finger for the Stroke group (Table 2). Surprisingly, those muscles most noticeably impacted were FDS and FDP, which exhibited size reductions of 21% and 19%, respectively, on the paretic as compared to the non-paretic side. The decreases observed for the extrinsic extensor muscles EI (17%) and EDC (12%) and the intrinsic muscles FPI (13%) and LUM (15%) were smaller, although there was no statistical difference. Interestingly, the atrophy observed in the intrinsic FDI muscle (9%) was less than half of that of FDS (Fig. 2). In general, the data showed an average decrease of 15% (SD 4) in muscle CSA of the paretic limb compared to the non-paretic limb for our stroke population whereas the age-matched control group demonstrated only a 6% (SD 2) decrease in CSA of the non-dominant limb compared to the dominant limb. This difference between groups was significant ($p = 0.011$).

Muscle thickness attenuation was detected on the paretic side of stroke subjects in all muscles investigated (Table 3). Thickness of the extrinsic flexors was reduced by 8% in FDS and 12% in FDP, while thickness of the extrinsic extensor muscles was diminished by 11% in EI and 10% in EDC, and thickness of the intrinsic muscles decreased by 6% in FDI, 11% in FPI, and 15% in LUM (Fig 3). Once again, the degree of muscle atrophy was least in FDI. Similarly to CSA, we observed an average decrease of 11% (SD 2) in muscle thickness in the paretic limb compared to the non-paretic limb, while the control group showed less than a 1% (SD 4) decrease in muscle thickness of the non-dominant limb compared to the dominate limb. As with CSA, the difference between groups was significant ($p = 0.001$).

Nearly two thirds of the subjects studied presented with the contralesional (paretic) arm/hand coinciding with his or her formerly dominant side. Muscle thickness was 10% smaller on the paretic side for this group as compared to 13% smaller for individuals in whom the paretic hand was the non-dominant side before the stroke ($p = 0.063$). The relationship between hand dominance and lesion side followed a similar trend for muscle CSA. There was a noticeable difference in muscle CSA observed for subjects with the impaired side corresponding to the formerly dominant side (12%), while a much more substantial difference (22%) was seen for individuals whose impairment primarily impacted the non-dominant side ($p = 0.035$).

Discussion

This study investigated the degree of atrophy following stroke for all muscles involved in motor control of the index finger by quantitatively measuring muscle geometry noninvasively *in vivo* by means of ultrasonography. Maximum thickness and CSA of the muscles were determined for both the paretic and non-paretic limbs of stroke survivors 2–4 years post cerebrovascular incident.

Overall, in accordance with studies of lower extremity muscles (Klein et al., 2010, Blijham et al., 2006, Dattola et al., 1993, Hafer-Macko et al., 2008, Kupa et al., 1995, Slager et al., 1985, Hu et al., 2007), muscle size in the paretic hand was significantly reduced with respect to the muscle size in the non-paretic hand in terms of both muscle thickness and CSA. This 15% deviation in CSA was greater than the 6% difference (not statistically significant) seen between the dominant and non-dominant hands in the age-matched control subjects. There was no significant difference in size between the non-paretic hand muscles in stroke subjects and the dominant hand muscles in neurologically intact subjects; CSA reduction of the impaired hand muscles was 16% with respect to the muscles of the dominant hand in the age-matched control subjects. Thus, size reduction in the paretic limb was 12% beyond what would be expected in a non-dominant limb in neurologically intact subjects. The extent of atrophy in a paretic limb tended to be smaller for a previously dominant than a non-dominant hand (12% and 22% difference in CSA for the formerly dominant paretic and non-paretic sides, respectively with a 10% and 13% difference in muscle thickness).

In disagreement with our other hypothesis, however, there was no significant difference in the amounts of atrophy among the different index finger muscles in the paretic hand. In fact, the extrinsic muscles tended to show greater atrophy than the intrinsic muscles, with the greatest reduction in CSA observed in the extrinsic flexor muscles (FDS and FDP). Absolute muscle size was not related to the percentage of atrophy. Our previous study looking at force production in different directions at the index fingertip in this stroke population reported greater relative sparing in directions for which the flexors dominate (flexion – 73% reduction) than for directions in which the extensors (extension – 92% reduction) or intrinsics (adduction – 84% reduction, abduction – 92% reduction) (Cruz et al., 2005). Thus, muscle atrophy does not appear to contribute to this bias towards relative sparing of flexion in the fingers. In contrast, greater atrophy has been reported in the paretic triceps muscle (25%) as compared to the paretic biceps brachii (5%) (Ploutz-Snyder et al., 2006).

In general, the force deficits directly attributable to muscle atrophy in the hand are small relative to the deficits actually observed. These subjects displayed differences in grip force and finger extension force of 84% and 88%, respectively, between the non-paretic and paretic hands, as compared to the 15% difference in muscle size. Certainly, changes in other muscle properties may contribute to the force deficits. Studies have reported greater relative sparing of muscles comprised predominantly of the less powerful type I muscle fibers in comparison with muscles consisting of predominantly type II fibers (Dattola et al., 1993, Klein et al., 2010, Slager et al., 1985, Hu et al., 2007). Interestingly, a study that investigated the distribution of the fiber types in healthy adult cadaveric specimens (Johnson et al., 1973) found FDI to be composed primarily of type I fibers, while EDC and FDP have a majority of type II fibers. Accordingly, we did observe somewhat less atrophy in FDI than in EDC and FDP, although these differences were not significant. Evidence for fiber hypertrophy has also been reported for type I fibers post-stroke (Blijham et al., 2006, Dattola et al., 1993, Slager et al., 1985). Yet, these changes may be offset by a shift in fiber type from type I to type II following stroke (Hafer-Macko et al., 2008, Landin et al., 1977, Dattola et al., 1993). Other physiological changes such as fatty infiltrations (Klein et al., 2010) and reduced fascicle length (Gao and Zhang, 2008) may also contribute to the deficits. Sarcomere length

has been shown to be profoundly lengthened beyond the optimal fiber length in paretic limb muscles in children with cerebral palsy (Lieber and Friden, 2002). In this study, only muscle size, not muscle quality, was assessed.

Although muscle thickness is not a term conventionally used for describing muscle architecture, it proved to be an easily accessible and viable measure in determining the relative muscle size between limbs of individuals. To test the repeatability of the ultrasonography techniques, repeated measurements were performed on three control subjects over 5 separate days for all 7 muscles. Intrasubject variability across the muscles had a standard deviation of 0.82 mm for muscle thickness and 17.46 mm² for CSA, yielding coefficients of variation of 0.09 and 0.15, respectively. Thus, the muscle thickness measurements yielded similar results to CSA in terms of atrophy, but had less variability. It should be noted that our CSA values for the control and non-paretic hands closely resemble those of previous publications, (Lieber et al., 1990, Lieber et al., 1992, Jacobson et al., 1992), in which more traditional muscle fixation techniques with cadaveric specimens were employed. One anomaly to this trend was the CSA determined for the lumbrical – due to difficulty in aligning the ultrasound probe perpendicular to these muscle fibers while, avoiding signal interference from the metacarpal bones, the true CSA may have been overestimated.

In summary, although muscle atrophy was detected in the paretic limb following stroke, the relatively small percent atrophy observed was not explanatory of the marked impairment in strength seen in this stroke population. Further investigation into possible underlying morphological changes in the muscle tissue is warranted to fully characterize hand muscle alterations and their impact on function after stroke.

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Fig.1. MATLAB Graphical User Interface (GUI) used for the offline measurement of the different muscles/muscle compartments. Left side (A) shows the measurement for the muscle thickness of FDS and the right side (B) shows the measurements for estimating the CSA of FDS.

Fig. 2. Normalized maximum muscle CSA for each of the muscles involved in motor control of the index finger. The values for the paretic limb of the stroke subjects were normalized to his/her non-paretic side. The values for the non-dominant limb of control subjects were normalized to his/her dominant side. Error bars represent the SD of the mean.

Fig. 3. Normalized maximum muscle thickness for each of the muscles involved in motor control of the index finger. The values for the paretic limb of the stroke subjects were normalized to his/her non-paretic side. The values for the non-dominant limb of control subjects were normalized to his/her dominant side. Error bars represent the SD of the mean.

Table 1

Repeated measures ANOVA results obtained for each dependent variable.

	Stroke		Control	
	<i>Raw Muscle Thickness</i>	<i>Raw Muscle CSA</i>	<i>Raw Muscle Thickness</i>	<i>Raw Muscle CSA</i>
Hand	P < 0.001	P < 0.001	-	-
Muscle	P < 0.001	P < 0.001	P < 0.001	P < 0.001

Significance accepted at $p \leq 0.0167$.

Table 2

Values obtained for muscle CSA.

Muscle	Control		Stroke	
	<i>Dominant</i>	<i>Non-Dominant</i>	<i>Non-Paretic</i>	<i>Paretic</i>
FDS	174.14 (51.69)	162.45 (45.82)	189.56 (87.12)	142.08 (55.30)
FDP	229.33 (53.53)	222.78 (68.84)	238.19 (78.54)	186.68 (58.50)
EI	103.23 (20.26)	99.43 (18.56)	94.64 (40.60)	75.74 (30.12)
EDC	102.69 (28.85)	95.53 (21.01)	106.98 (48.51)	91.28 (45.46)
FDI	269.07 (57.15)	254.75 (54.35)	264.21 (68.47)	230.63 (73.69)
FPI	93.91 (23.57)	82.90 (24.77)	115.13 (54.84)	95.47 (41.96)
LUM	74.13 (30.40)	68.93 (25.65)	70.55 (23.51)	57.57 (18.71)

Note: Values are mean (SD) and units are in mm².

Table 3

Values obtained for muscle thickness.

	Control		Stroke	
Muscle	<i>Dominant</i>	<i>Non-Dominant</i>	<i>Non-Paretic</i>	<i>Paretic</i>
FDS	10.35 (2.76)	9.93 (2.23)	10.50 (1.84)	9.68 (2.26)
FDP	18.32 (2.19)	17.38 (2.92)	19.85 (4.44)	17.25 (3.76)
EI	8.90 (1.43)	9.10 (1.57)	8.87 (2.18)	7.82 (1.94)
EDC	8.78 (2.21)	8.65 (2.44)	9.01 (1.96)	7.98 (1.67)
FDI	10.68 (1.56)	10.85 (1.77)	12.02 (1.96)	10.77 (2.22)
FPI	6.68 (1.17)	6.57 (1.10)	9.13 (2.41)	7.80 (2.29)
LUM	5.27 (0.85)	4.91 (0.70)	5.55 (0.90)	4.91 (0.87)

Note: Values are mean (SD) and units are in mm.