ULTRASONOGRAPHIC INVESTIGATION OF HAND MUSCLE ATROPHY IN STROKE SURVIVORS

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INTRODUCTION

Contralesional limb weakness is a common finding in stroke survivors. Strength deficits in the hands can be especially profound, with losses of greater than 75% [1,2]. While excessive coactivation and an inability to fully activate these muscles contribute to this weakness, muscle atrophy may also play a role. Indeed, atrophy in the hemiparetic leg has been described (initially via needle biopsy [3] or cadaveric specimens and more recently using imaging techniques [4]). However, paretic muscles of the upper extremity, 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, deficits appear to be non-uniform, with greater loss in finger extension than flexion. Thus the goal of this study was to quantify the extent of muscle atrophy in index finger musculature in vivo by means of ultrasonography. It was hypothesized that finger intrinsic and extensor muscle would atrophy to a greater extent than flexor muscle based on clinically observed reductions in fine motor control, individuation and extension force.

METHODS

Twenty-five subjects with severe hand impairment, classified as Stage 2 or 3 on the Stage of Hand component of the Chedoke-McMaster Stroke Assessment [5], participated. All stroke survivors had chronic hemiparesis resulting from a stroke incurred 2-4 years prior. Ten healthy, age-matched controls 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.

Muscle component definition of index finger muscles such as flexor digitorum superficialis (FDs), flexor digitorum profundus (FDP), and extensor digitorum communis (EDC) with imaging modalities with poor time resolution, such as magnetic resonance imaging, is very difficult. Thus, ultrasonography was employed to non-invasively measure the geometry of the muscles controlling the index finger. Utilizing the advantageous real-time feedback, localization of the proper muscle/muscle compartment was conducted by viewing the displacement of muscle fibers in response to isolated muscle contraction or imposed movement of the joint(s). The musculoskeletal transducer probe was positioned over the muscle belly and adjusted to determine the maximal muscle parameter of interest for each of the muscles of the index finger: first compartments of FDS, FDP, and EDC, extensor indicis (EI), first dorsal interosseous (FDI), first palmar interosseous (FPI), and lumbrical (LUM).

Two different muscle parameters were evaluated: peak cross-sectional area (CSA) and thickness, in the anatomical transverse and sagittal planes, respectively. CSA was estimated assuming an elliptical configuration for each muscle. While CSA provides a more complete description of atrophy, muscle thickness was often more accurately measured due to enhanced image quality. Ultrasound images were saved as movie segments and analyzed offline in MATLAB (Fig. 1).

Figure 1: MATLAB GUI for offline measurements/estimation of muscle thickness (left) and CSA (right) of the first compartment of FDS.

For each measured parameter and group, a MANOVA was performed (SPSS Inc., Chicago, IL)
to explore the impact muscle type and non-dominance/paresis had on the output. If significance resulted, additional post-hoc RM ANOVA analyses were performed. A Bonferroni correction for multiple comparisons was implemented ($\alpha = 0.05$).

**RESULTS AND DISCUSSION**

As expected, paretic muscle size was significantly reduced (Wilks’ lambda $< 0.001$) for the stroke group. For the control group, hand dominance did not significantly affect muscle size (Wilks’ lambda $= 0.411$) and the muscle size was not significantly different between the dominant hand (Control) and the non-paretic hand (Stroke) for muscle thickness ($p = 0.112$) or CSA ($p = 0.639$). Overall, the data showed an average decrease of $15 \pm 4\%$ in muscle CSA of the paretic limb compared to the non-paretic limb for this stroke population whereas the age-matched control group demonstrated only a non-significant $6 \pm 2\%$ decrease in CSA of the non-dominant limb compared to the dominant limb. Thus, the true deficit in the stroke group was only $10\%$. In comparison, measured flexion force deficits were found to be over $83\%$ and extension force deficits were $88\%$ in this stroke population.

Decreased muscle CSA was evident in all muscles of the paretic index finger for the stroke group, but surprisingly, there was no significant difference in the relative atrophy of the different muscles. The muscles most noticeably impacted, in fact, 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 not significantly. Interestingly, the atrophy observed in the intrinsic FDI muscle (9%) was less than half of that of FDS (Fig. 2). Hence, muscle atrophy could not account for the relatively greater deficits in force generation in certain directions.

Paretic muscle thickness attenuation was also detected in all muscles investigated of the Stroke group. 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 that of the intrinsic muscles by 6% in FDI, 11% in FPI, and 15% in LUM. Once again, the degree of muscle atrophy in FDI was least. Similarly to CSA, we observed an average decrease of $11 \pm 2\%$ in muscle thickness in the paretic limb compared to the non-paretic limb, with the control group presenting less than a $1 \pm 4\%$ decrease in muscle thickness of the non-dominant limb compared to the dominate limb. Thus, muscle thickness appears to be a reasonable indicator of muscle atrophy, and is fairly easily measured with ultrasonography.

**CONCLUSIONS**

Although muscle atrophy was detected in the paretic limb following stroke, it is not explanatory of the marked impairment seen in this stroke population. Admittedly, other alterations in muscle morphology may contribute to the emergent muscle weakness post-stroke. Fatty infiltrations and changes in fiber structure have been identified in lower limb musculature following stroke. These potential alterations may warrant future examination in upper extremity musculature.

**REFERENCES**


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