QUANTIFYING MUSCLE MATERIAL PROPERTIES OF PASSIVE AND ACTIVE STROKE-IMPAIRED BICEPS BRACHII

Sabrina S.M. Lee, Sam. Speer, and William Z. Rymer

1 Northwestern University, Chicago, IL, USA
2 The Rehabilitation Institute of Chicago, Chicago, IL, USA
email: s-lee@northwestern.edu, web: http://www.feinberg.northwestern.edu/sites/pthms

INTRODUCTION

Individuals who have had a stroke routinely experience long-term motor and sensory impairments. Decreased strength and range of motion contribute greatly to decreased mobility. In conjunction with neurological changes, muscle properties, including architecture and material properties such as stiffness, may also progressively change. Prior descriptions of muscle material properties in stroke survivors have observed increased muscle stiffness [1]. However, these previous measures are estimates derived from individual joint stiffness or whole muscle groups. In addition, current standard clinical examinations cannot readily distinguish between active reflex and passive tissue components [2]. Thus, the purpose of this study was to compare material properties, by measuring shear wave velocity (SWV) which is related to shear modulus, in spastic paretic muscles at rest and at different activation levels with contralateral muscles in stroke survivors and in age-matched control subjects.

METHODS

Fourteen stroke survivors participated in this study (age: 58.9±7.4yrs; height: 1.68±0.10m; body mass: 85.5±18.2kg; time post-stroke: 11.4±11.6yrs.; Fugl-Meyer: 4-48, 19±15). We tested nine subjects who were gender and age-matched to the stroke survivors (age: 57.9±7.1yrs; height: 1.64±0.10m; mass: 74.4±11.2kg;).

Subjects were seated upright with their upper arm resting on a plastic support, the forearm secured in a fiberglass cast, and the wrist and forearm held in a neutral position in a ring-mount interface mounted to the table. The shoulder was positioned so that the humerus was abducted 45 degrees and the elbow positioned at 90 degrees. Subjects performed a series of isometric elbow flexion contractions at different activation levels (0, 10, 25, 50, 75, 100% maximum voluntary contraction (MVC)) while force and torque were measured at the wrist (load cell). During each trial, muscle activity (electromyography), and ultrasound images (Aixplorer, SuperSonic Imagine) of the biceps brachii were captured. Mean SW velocity was calculated from a 12mm by 12mm region of the ultrasound images. Here we use shear wave ultrasound elastography which measures the velocity at which shear waves propagate through the tissue [3]. Shear waves will propagate faster in a tissue that has a higher elastic shear modulus:

$$\mu = \rho v^2$$

where $\mu$ is the elastic shear modulus, $\rho$ is the muscle mass density ($\rho \approx 1000$ kg m$^{-3}$), and $v$ is the SW velocity. We calculated the slope and $r^2$ of the linear fit between SWV and % MVC until 50%MVC as the SWV at 75% and 100%MVC reached the maximum the ultrasound system is capable of measuring (16m/s). An Analysis of Variance was used to compare the SWV at the different % MVC levels, slope, and $r^2$ of the non-paretic and paretic muscles, and those of the gender, age-matched controls.

RESULTS AND DISCUSSION

Our main findings show that at rest, the SWV was on average 40% greater in the paretic muscle compared to either the contralateral non-paretic muscle or the muscle of control subjects (Fig.1, $p=0.003$, $p=0.002$), and no significant difference in SWV between the non-paretic muscle and control muscle. In active muscle (10, 25, 50%), there was no significant difference in SWS between the non-paretic, paretic, and control muscles (Fig.1). The
slopes of SWV versus %MVC (up to 50%MVC) and R^2 values was 10% and 15% greater, respectively, in the non-paretic muscles compared to that of the paretic muscle, but was not significant (Table 1, p = 0.06).

![Graph showing shear wave velocity (SWV) at rest and at 50%MVC for non-paretic and paretic muscles with brackets indicating the same control subject.]

**CONCLUSIONS**

We demonstrate that SW velocity is faster in the biceps brachii of the paretic limb than in the contralateral non-paretic limb of stroke survivors and age-matched individuals with no neurological impairments when the muscle is at rest. This suggests that paretic muscles have potentially altered muscle material properties, specifically stiffness in both passive and active muscles, but primarily in passive states. This can be a result of increased collagen in the perimysium and abnormal accumulation of the extracellular matrix[4].

Interestingly, this difference in SWV between three muscle groups disappears once the muscle was active. One source of active stiffness is short-range stiffness. Thus, it is possible that the non-contractile elements contribute minimally to stiffness in active muscle and the active short-range stiffness is not altered in stroke-impaired muscle.

These results offer us insight and a first look into active and passive stiffness of stroke muscle. Quantitative measurements of muscle material properties, in particular stiffness, in individual muscles is important for accurately evaluating muscle function and for understanding the fundamental mechanisms of impairment that occur after a stroke. This knowledge has the potential for aiding clinical decisions, and for guiding rehabilitation interventions that may specifically target altered passive and active stiffness.

**REFERENCES**


**ACKNOWLEDGEMENTS**

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**Table 1**: Mean shear wave velocity at rest and at 50%MVC, slope of SW velocity and % MVC, and R^2. Mean (SD).

<table>
<thead>
<tr>
<th>Muscle</th>
<th>SW velocity at rest</th>
<th>SWV at 50%MVC</th>
<th>Slope (ms^{-1}%MVC^{-1})</th>
<th>R^2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paretic</td>
<td>3.65 (1.31)</td>
<td>10.33 (2.03)</td>
<td>0.12 (0.04)</td>
<td>0.73 (0.12)</td>
</tr>
<tr>
<td>Non-paretic</td>
<td>2.30 (0.38)</td>
<td>9.79 (2.36)</td>
<td>0.15 (0.05)</td>
<td>0.81 (0.12)</td>
</tr>
<tr>
<td>Control</td>
<td>2.29 (0.18)</td>
<td>10.42 (2.28)</td>
<td>0.13 (0.05)</td>
<td>0.89 (0.16)</td>
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