Objective: The main purposes of this study were to examine, in subjects with chronic hemiparesis following a stroke: (i) the correlations between tests of muscle tone, stiffness, spasticity, paresis and co-contraction, and (ii) the correlations of these tests and measurements of impairment to upper extremity motor performance.

Design: Prospective, cross-sectional, correlation matrix using sample of convenience.

Subjects: Thirteen subjects with chronic hemiparesis secondary to a cerebrovascular accident (stroke) were tested.

Methods: Subjects were assessed using the Fugl-Meyer Upper Extremity Motor Assessment, modified Ashworth scale, deep tendon reflexes, and muscle characteristics that included quantification of muscle stiffness, paresis and co-contraction during a voluntary reaching task and during passive movements. Surface electromyographic and myotonometric muscle stiffness data were obtained during movement trials.

Results: Biceps and triceps brachii muscle paresis and excess biceps brachii co-contraction during voluntary reaching had the highest correlations to decreased motor performance. Muscle tone measurements did not have significant correlations to upper extremity performance.

Conclusion: Paresis of elbow flexors and extensors and excess biceps brachii co-contraction during voluntary reaching appear to be most predictive of upper extremity motor performance. Results are discussed in relation to the specific challenges these findings pose for spastic paresis clinical management.

Key words: stroke, myotonometer, paresis, co-contraction, spasticity.


Correspondence address: Charles T. Leonard, School of Physical Therapy and Rehabilitation Sciences, The University of Montana, Missoula, MT 59803 USA. E-mail: charles.leonard@umontana.edu

Submitted April 11, 2005; accepted January 26, 2006

INTRODUCTION

The development of upper extremity spastic paresis is common following stroke and other neurological disorders. Spastic paresis is comprised of positive and negative symptoms that occur to varying degrees in each patient. Positive symptoms include spasticity (velocity dependent resistance to passive stretch), hypertonia (resistance to passive stretch), increased muscle stiffness (tissue displacement per unit of applied force) and excessive co-contraction between agonist (shortening) and antagonist (lengthening) muscles. Negative symptoms include muscle paresis (decreased muscle activation) and discoordination. It has long been of interest to discern the relationships of these positive and negative symptoms and impairments to each other and to motor performance.

The few studies that have attempted to explore the relationships among neurological tests, impairments and motor performance have been equivocal. There are conflicting reports regarding the degree to which altered stretch reflexes (1, 2), resistance to passive stretch (3, 4), excessive antagonist muscle co-contraction (5–7), and musculo-tendonous stiffness (2, 8) affect motor performance. It is likely that, in part, equivocal results can be attributed to study specifics. For instance, past assessments of antagonist muscle co-contraction of subjects post-stroke were typically measured during maximal voluntary contractions of the agonist muscle (5, 7). Other studies assessed voluntary submaximal efforts (6). It remains unknown whether co-contractions observed during maximal efforts of subjects relate to their muscle activation levels during submaximal voluntary reaching tasks. This was one question addressed in the present study.

The study had 3 specific aims. The first was to examine, in subjects with chronic spastic hemiparesis following a stroke, the relationships among impairments and upper extremity motor performance. The second was to examine the relationships between passive neurological tests (DTRs, MAS), muscle characteristic data (stiffness, paresis, co-contraction) and motor performance. The third aim was to determine the degree of correlation between myotonometric biceps brachii muscle stiffness measurements obtained from a passively moved arm to measurements obtained during a voluntary reaching task.
We hypothesized that triceps brachii muscle paresis, increased biceps brachii stiffness, and MAS would have the highest correlation to motor performance as measured by the Fugl-Meyer Upper Extremity Motor Function test. Secondly, we hypothesized that biceps brachii DTRs, muscle stiffness, and MAS would correlate to each other. Thirdly, it was hypothesized that passive and voluntary movement myotonometer measurements of biceps brachii stiffness would be significantly different but correlated.

MATERIAL AND METHODS

This study received approval from The University of Montana's Institutional Review Board and was conducted in accordance with the Declaration of Helsinki. Testing of subjects took place, following informed consent, in The Motor Control Research Laboratory at The University of Montana.

Subjects

Thirteen subjects, with a history of stroke, with a mean age of 62.8 (SD 9.5) years, participated in this study. Table I summarizes subject characteristics. Subjects were screened using the following inclusion/exclusion criteria. To be included in the study, subjects had to have: (i) a diagnosis of spastic-type hemiparesis involving the upper extremity of at least 10 months’ duration with accentuated upper extremity DTRs and a modified Ashworth scale (MAS) score of >1, (ii) an ability to follow simple commands, and (iii) an ability to initiate a reaching movement involving shoulder horizontal adduction and elbow extension. Exclusion criteria included: (i) signs of extra-pyramidal involvement, such as resting or active tremors or dystonic postures, (ii) orthopedic involvement of the upper extremity (e.g. acute sprains, history of surgeries, joint replacements), (iii) pain during active or passive upper extremity movement and (iv) current use of any tone-altering medications.

Study design

The study was a prospective, cross-sectional research design in which all data were collected during a single, 90-minute experimental session. Subject histories, DTRs (scale = 0–5), MAS (9), and Fugl-Meyer Upper Extremity Motor Function (10) data were obtained first. The DTRs, MAS and Fugl-Meyer Upper Extremity Motor Function data were acquired with the subject in a sitting position using standardized clinical procedures. Subjects were then positioned in an upper extremity armature for muscle stiffness, strength and co-contraction data collection during biceps and triceps brachii maximal voluntary contraction (MVC) trials, voluntary reaching to a target and during passive movements that mimicked the speed and trajectory of the subjects’ voluntary movements. Muscle stiffness data were collected with a Myotonometer. All other muscle characteristic data were collected using surface electromyography (sEMG).

Instrumentation

Upper extremity armature testing apparatus. An upper extremity armature device was constructed and used for the study. The device was used so that subjects did not have to support the arm against gravity, to ensure identical planar movements among all trials, to isolate subject movements solely to the elbow and shoulder joints, and to permit measurements of accelerations and joint velocities during movements. The armature supported the upper extremity and permitted only flexion and extension movements of the elbow and shoulder horizontal abduction and adduction. The armature was mounted to a table and consisted of a proximal plexiglass support that suspended the arm from the axilla to the elbow. The distal plexiglass cuff supported the forearm from the elbow to the metacarpals. There was an articulation at the elbow and the shoulder joints that used needle bearings and thrust plates to approximate frictionless movement in the horizontal plane at the shoulder and the elbow. The armature was equipped with an accelerometer (Biopac model TSD109) and 2 electrogoniometers (Biopac model TSD130B). The accelerometer was secured at the distal end of the forearm support and the electrogoniometers were secured at the armatures’ elbow and shoulder joints. The armature was also equipped with a plastic dowel that extended from the wrist plate to slightly beyond the subjects’ fingers. This dowel acted as the pointing device that subjects used to point to the intended target. The target was a 14 cm circle with concentrically smaller circles decreasing by 2 cm in diameter, similar to a rifle target. The armature and target were mounted to a table that represented the subject workspace.

Electromyography. sEMG data were acquired and analyzed using the BIOPAC Systems MP150 and Acknowledge 3.7.3 software. Electrodes (Ag-AgCl, 2 cm between active sites, onsite pre-amplification; Therapeutics Unlimited) were placed over the muscle belly following appropriate skin preparation. Signals were sampled at 2 kHz, high pass filtered at 20 Hz and amplified 2–5 K as needed. A reference electrode was placed on the opposite forearm just proximal to the volar aspect of the wrist. sEMG data were used to measure biceps brachii, triceps brachii and posterior deltoid muscle activity during MVCs, and during voluntary and passive reaching tasks.

Myotonometer®. The Myotonometer (Neurogenic Technologies, Inc.) was used to quantify biceps brachii muscle stiffness. The Myotonometer is a patented, FDA approved, computerized electronic tissue compliance device that quantifies the amount of tissue displacement per unit force applied by a probe as it is pressed perpendicularly onto the skin overlying a muscle. The Myotonometer is reliable (11, 12) and valid for use with individuals with neurological involvement (3, 13).

The location for the Myotonometer probe placement was marked over the biceps brachii muscle approximately 2 cm distal to the biceps brachii EMG electrode. Measurements were taken at 8 force increments (0.25, 0.50, 0.75, 1.0, 1.25, 1.50, 1.75 and 2.00 kg). Computational software created force-displacement curves and calculated area under the curve (AUC) of these curves based on the data obtained. These data provided a measure of muscle tone (stiffness) (13). For the present experiments, myotonometer measurements of biceps brachii muscle stiffness were acquired at rest, during MVC, during voluntary reaching and during passive movement of the subject’s arm.

Data acquisition procedures

Following DTR, MAS and Fugl-Meyer data acquisition, subjects were positioned in the upper extremity armature. Chair height was adjusted so that the subject’s test shoulder was held in 90° of shoulder abduction. The forearm was placed in a pronated position within the armature and secured at the midpoint of the forearm and the upper arm by Velcro straps. The subject’s trunk was stabilized with adjustable straps, one around the waist and one around the chest.

The subjects’ uninvolved upper extremity was tested first, followed by the involved arm. Resting Myotonometric readings were taken once the subject was positioned in the upper extremity testing apparatus. The subject’s arm was placed in a maximally lengthened position of shoulder horizontal abduction and elbow extension (Fig. 1). Two sets of Myotonometer measurements of biceps brachii muscle stiffness were recorded from this position.

Resting stiffness trials were followed by MVCs of the biceps and triceps brachii muscles with the elbow joint positioned at 75° of elbow flexion and a self-selected position of shoulder horizontal adduction. Subjects were instructed to build slowly into a maximal effort over a 5-second period. Five seconds of maximal effort sEMG data were recorded for future root mean square (RMS) analysis. Maximal effort was determined post hoc by finding the peak RMS activity (Biopac signal analysis software) during the 5-second trial and computing the RMS of ±1 second on either side of this peak. Myotonometer measurements of the biceps brachii muscle were taken during the maximal effort of the contraction.

Following MVC data collection, subjects were placed in the starting position for reaching trials. This consisted of 90° of elbow flexion and 90° of shoulder horizontal adduction (Fig. 1). The target was placed at the maximum distance the subject could actively reach using elbow extension and shoulder horizontal abduction.

With each subject’s arm aligned in the start position, the subject was instructed to look at the target and reach towards it at a self-selected speed as if they were “reaching for a glass of water.” This movement
required the subject to extend the elbow and horizontally abduct the shoulder. This type of movement is often referred to as a movement “out of synergy.” sEMG recordings were obtained prior to and during reaching. Myotonometric measurements of the biceps brachii muscle coincided with target acquisition. Five reaching trials were performed.

Following the voluntary reaching task, sEMG and myotonometric data were obtained during a passive reaching movement. For the passive reaching movement, subjects remained relaxed while the armature was moved manually with the same velocity and time to target as recorded during voluntary reach. A computerized clock was used for this determination and practice trials were used to ensure consistency prior to data collection trials. Five trials of passive movement were obtained. In order to be considered a valid trial for data analysis, each passive trial was within $\pm 10\%$ of time to target as during voluntary reaching trials.

Data analysis procedures

Biceps brachii muscle stiffness measurements. Myotonometric measurements were obtained from the involved and uninvolved biceps brachii muscles. The percentage difference in stiffness between the 2 extremities was computed for each subject and grouped data used for subsequent correlational statistical analysis as presented in Table II. Differences in biceps brachii stiffness were calculated during voluntary movements (Table II: column 2, row 2) and during passive movements of each extremity (Table II: column 1, row 1).

Biceps and triceps brachii paresis. The presence and amount of biceps and triceps brachii muscle paresis were determined by comparing the RMS of each muscle during MVC trials. The percent difference between the involved and uninvolved extremities was calculated (e.g. biceps RMS during MVC of involved extremity was compared to biceps RMS of uninvolved extremity. Grouped means were used for correlational analyzes (Table II: columns/rows 6 and 7).

Muscle co-contraction. Muscle co-contraction, between the biceps and triceps brachii muscles, were obtained during the following conditions: (i) biceps brachii MVC trials, (ii) triceps brachii MVC trials, and (iii) during voluntary reaching task trials.

As previously described for MVC trials, 2-seconds of sEMG data representing the subject’s maximal elbow flexion or extension efforts were used for analysis. The triceps and biceps RMS was computed. For elbow extension trials, the triceps RMS value set as 100% and any biceps brachii activation that occurred during this time was computed as a percentage of this value. If the biceps brachii was not active during triceps contraction it received a value of zero. If the biceps brachii amplitude was one-half that of the triceps it received a value of 50%, and so on. Grouped means were used for further correlational analyzes. These data are presented in Table II (% co-contraction involved triceps MVC). Similar procedures were followed for elbow flexion trials, but with RMS values for the biceps brachii set as 100%.

Table I. Characteristics of the 13 Patients with chronic hemiplegia secondary to a cerebrovascular accident (CVA).

<table>
<thead>
<tr>
<th>Age (years)/Sex</th>
<th>Months after CVA</th>
<th>Involved side</th>
<th>MRI scan data</th>
<th>Elbow flexor MAS (0–4)</th>
<th>Biceps brachii DTR (1–5)</th>
<th>Fugl-Meyer (0–50)</th>
</tr>
</thead>
<tbody>
<tr>
<td>62/M</td>
<td>161</td>
<td>Left</td>
<td>Occlusion of right carotid artery</td>
<td>1.5</td>
<td>3.5</td>
<td>32</td>
</tr>
<tr>
<td>55/M</td>
<td>227</td>
<td>Right</td>
<td>Left temporal-parietal region, occlusive infarction</td>
<td>2.7</td>
<td>3.5</td>
<td>27</td>
</tr>
<tr>
<td>55/M</td>
<td>52</td>
<td>Right</td>
<td>Left putamal subcortical white matter</td>
<td>1.5</td>
<td>3.5</td>
<td>16</td>
</tr>
<tr>
<td>70/M</td>
<td>21</td>
<td>Right</td>
<td>No scan data, but “posterior aspect of brain” reported</td>
<td>1.5</td>
<td>3.5</td>
<td>94</td>
</tr>
<tr>
<td>63/M</td>
<td>116</td>
<td>Left</td>
<td>Embolus to left inferior frontal gyrus</td>
<td>1.5</td>
<td>3.5</td>
<td>39</td>
</tr>
<tr>
<td>56/M</td>
<td>41</td>
<td>Left</td>
<td>Middle-sagittal artery distribution infarct</td>
<td>1.5</td>
<td>3.5</td>
<td>14</td>
</tr>
<tr>
<td>57/M</td>
<td>10</td>
<td>Left</td>
<td>Occlusion of right carotid artery</td>
<td>1.5</td>
<td>3.5</td>
<td>22</td>
</tr>
<tr>
<td>75/F</td>
<td>9</td>
<td>Left</td>
<td>Middle cerebral artery distribution infarct</td>
<td>1.5</td>
<td>3.5</td>
<td>77</td>
</tr>
<tr>
<td>118/M</td>
<td>32</td>
<td>Right</td>
<td>No scan data</td>
<td>1.5</td>
<td>3.5</td>
<td>25</td>
</tr>
<tr>
<td>56/M</td>
<td>45</td>
<td>Right</td>
<td>Embolus to left middle cerebral artery</td>
<td>1.5</td>
<td>3.5</td>
<td>12</td>
</tr>
<tr>
<td>75/F</td>
<td>9</td>
<td>Left</td>
<td>Middle cerebral artery distribution infarct</td>
<td>1.5</td>
<td>3.5</td>
<td>11</td>
</tr>
</tbody>
</table>

MRI = magnetic resonance imaging; MAS = modified Ashworth scale; DTR = deep tendon reflexes.

Fig. 1. Start (top) and end (bottom) positions (target acquisition) for voluntary and passive movement reaching tasks. The small vertical rectangle represents the target position during testing.
Table II. Impairment and motor performance correlation matrix table. Within each cell, the top number is the r-value, the bottom number is the p-value. Bold numbers indicate significance. Muscle stiffness comparisons are represented within the first 2 columns and rows.

<table>
<thead>
<tr>
<th>% Difference between involved &amp; uninvolved passive</th>
<th>Elbow flexor MAS</th>
<th>Biceps brachii DTR</th>
<th>Fugl-Meyer</th>
<th>Biceps paresis</th>
<th>Triceps paresis</th>
<th>% Co-contraction mV of involved</th>
<th>% Co-contraction mV of uninvolved</th>
<th>% Co-contraction MVC</th>
<th>% Co-contraction MVC</th>
</tr>
</thead>
<tbody>
<tr>
<td>% Difference between involved &amp; uninvolved active</td>
<td>Correlation</td>
<td>sig.</td>
<td>(2-tailed)</td>
<td>Correlation</td>
<td>sig.</td>
<td>(2-tailed)</td>
<td>Correlation</td>
<td>sig.</td>
<td>(2-tailed)</td>
</tr>
<tr>
<td>% Difference between involved &amp; uninvolved passive</td>
<td>Correlation</td>
<td>sig.</td>
<td>(2-tailed)</td>
<td>0.797</td>
<td>0.002</td>
<td>0.146</td>
<td>0.975</td>
<td>0.003</td>
<td>0.126</td>
</tr>
<tr>
<td>% Difference between involved &amp; uninvolved active</td>
<td>Correlation</td>
<td>sig.</td>
<td>(2-tailed)</td>
<td>-0.091</td>
<td>-0.150</td>
<td>-0.012</td>
<td>-0.898</td>
<td>0.004</td>
<td>0.140</td>
</tr>
<tr>
<td>% Difference between involved &amp; uninvolved passive</td>
<td>Correlation</td>
<td>sig.</td>
<td>(2-tailed)</td>
<td>0.778</td>
<td>0.642</td>
<td>0.100</td>
<td>0.921</td>
<td>0.005</td>
<td>0.134</td>
</tr>
<tr>
<td>% Difference between involved &amp; uninvolved active</td>
<td>Correlation</td>
<td>sig.</td>
<td>(2-tailed)</td>
<td>-0.453</td>
<td>-0.366</td>
<td>-0.161</td>
<td>-0.255</td>
<td>0.007</td>
<td>0.217</td>
</tr>
<tr>
<td>% Difference between involved &amp; uninvolved passive</td>
<td>Correlation</td>
<td>sig.</td>
<td>(2-tailed)</td>
<td>0.139</td>
<td>0.241</td>
<td>0.600</td>
<td>0.485</td>
<td>0.005</td>
<td>0.165</td>
</tr>
<tr>
<td>% Difference between involved &amp; uninvolved active</td>
<td>Correlation</td>
<td>sig.</td>
<td>(2-tailed)</td>
<td>0.455</td>
<td>0.241</td>
<td>0.600</td>
<td>0.110</td>
<td>0.241</td>
<td>0.600</td>
</tr>
<tr>
<td>% Difference between involved &amp; uninvolved passive</td>
<td>Correlation</td>
<td>sig.</td>
<td>(2-tailed)</td>
<td>0.239</td>
<td>-0.422</td>
<td>0.114</td>
<td>0.114</td>
<td>0.239</td>
<td>-0.422</td>
</tr>
<tr>
<td>% Difference between involved &amp; uninvolved active</td>
<td>Correlation</td>
<td>sig.</td>
<td>(2-tailed)</td>
<td>-0.453</td>
<td>-0.366</td>
<td>-0.161</td>
<td>0.110</td>
<td>0.241</td>
<td>0.600</td>
</tr>
<tr>
<td>% Difference between involved &amp; uninvolved passive</td>
<td>Correlation</td>
<td>sig.</td>
<td>(2-tailed)</td>
<td>-0.160</td>
<td>-0.269</td>
<td>0.119</td>
<td>0.119</td>
<td>0.619</td>
<td>0.398</td>
</tr>
<tr>
<td>% Difference between involved &amp; uninvolved active</td>
<td>Correlation</td>
<td>sig.</td>
<td>(2-tailed)</td>
<td>0.013</td>
<td>0.013</td>
<td>0.013</td>
<td>0.545</td>
<td>0.171</td>
<td>0.712</td>
</tr>
<tr>
<td>% Difference between involved &amp; uninvolved passive</td>
<td>Correlation</td>
<td>sig.</td>
<td>(2-tailed)</td>
<td>-0.156</td>
<td>0.137</td>
<td>-0.500</td>
<td>0.137</td>
<td>0.500</td>
<td>0.137</td>
</tr>
<tr>
<td>% Difference between involved &amp; uninvolved active</td>
<td>Correlation</td>
<td>sig.</td>
<td>(2-tailed)</td>
<td>0.032</td>
<td>0.099</td>
<td>0.510</td>
<td>0.032</td>
<td>0.099</td>
<td>0.510</td>
</tr>
<tr>
<td>% Difference between involved &amp; uninvolved passive</td>
<td>Correlation</td>
<td>sig.</td>
<td>(2-tailed)</td>
<td>0.015</td>
<td>0.015</td>
<td>0.015</td>
<td>0.331</td>
<td>0.331</td>
<td>0.331</td>
</tr>
<tr>
<td>% Difference between involved &amp; uninvolved active</td>
<td>Correlation</td>
<td>sig.</td>
<td>(2-tailed)</td>
<td>0.309</td>
<td>0.317</td>
<td>0.803</td>
<td>0.309</td>
<td>0.317</td>
<td>0.803</td>
</tr>
</tbody>
</table>

% Difference between involved and uninvolved passive = Myotonometric muscle stiffness measurements of the differences between extremities during passive movements; % Difference between involved and uninvolved active = Myotonometric muscle stiffness measurements indicating differences between extremities during voluntary movements; Triceps/biceps muscle co-contraction values are represented during voluntary reaching tasks (% co-contraction mV) and also during biceps and triceps MVC trials (% co-contraction MVC). MVC = maximum voluntary contraction; DTR = deep tendon reflexes; MAS = modified Ashworth scale; mV = millivolts.
Co-contraction was also determined during voluntary reaching tasks. The onset and duration of triceps brachii EMG activity was determined (visual displacement from baseline; Biopac Software Systems). Root mean square values were calculated for the duration of the triceps EMG activity. This same time period was used to analyze any concomitant biceps brachii EMG activity. Biceps brachii EMG amplitudes were determined using the same RMS analyses and compared to triceps brachii RMS values. A percentage of biceps brachii co-contraction was derived using the triceps brachii RMS value as 100%.

**Statistical analysis**

Dependent variables consisted of Fugl-Meyer assessment, the modified Ashworth scale, DTRs, biceps/triceps RMS during MVC (paresis), myotonometric measurements of biceps brachii muscle stiffness at rest and during voluntary and passive reaching tasks and biceps and triceps brachii percent co-contraction (RMS) during voluntary and passive reaching. A correlation matrix was generated to assess the degree of correlation among the variables. Pearson’s product-moment coefficient of correlation was used for parametric data (myotonometric, sEMG and Fugl-Meyer data). A Spearman’s rank correlation coefficient was used for non-parametric data (MAS and DTRs). Not all of the ranges of the MAS or DTR data were represented in our subject population. Correlational analysis is not possible with empty bins. Modified Ashworth and DTR data, therefore, were condensed into the following groupings: the MAS scores (range 1.0–3.0) were assigned to 1 of 4 groups: score of 1 = group 1; score of 1.5 = group 2; score of 2 = group 3; score of 3 = group 4. The DTR data (range 2–4) were also assigned to 1 of 3 groups: scores 2–3 = group 1; scores of 3.5 = group 2; scores of 4 = group 3. The following scale was used for interpretation of correlation: 1.00–0.90 = very high; 0.89–0.70 = high; 0.69–0.50 = moderate; 0.49–0.26 = low; and less than 0.25 = poor (14). Paired t-test comparisons were used to analyze within subject, between limb differences for myotonometric and sEMG data. Statistical significance was set at \( p \leq 0.05 \).

**RESULTS**

**Fugl-Meyer Upper Extremity Motor Function Test correlations**

Of the 14 parameters examined for correlation to the Fugl-Meyer Upper Extremity Motor Function Test, only 3 had significant associations. These were biceps paresis \( (r = -0.667; p = 0.013) \), triceps paresis \( (r = -0.752; p = 0.003) \) and co-contraction (the amplitude of biceps brachii activation during the voluntary reaching task) \( (r = -0.762; p = 0.006) \).

**Biceps and triceps brachii paresis correlations**

Triceps and biceps brachii paresis of the involved upper extremity were highly correlated \( (r = 0.789; p = 0.001) \). DTRs paresis was also significantly correlated to excessive biceps brachii co-contraction (increased biceps brachii amplitudes) that occurred during voluntary reaching at self-selected speeds \( (r = 0.756; p = 0.007) \) and the excess biceps brachii co-contraction that also occurred during triceps brachii MVC trials \( (r = 0.645; p = 0.032) \). Biceps brachii paresis was significantly correlated to its level of activation (co-contraction) during the voluntary reaching task \( (r = 0.683; p = 0.021) \).

**Co-contraction correlations**

As reported previously, biceps brachii EMG amplitudes (a measure of co-contraction) during the voluntary reaching task, correlated with biceps \( (r = 0.683; p = 0.021) \) and triceps \( (r = 0.756; p = 0.007) \) brachii paresis and Fugl-Meyer testing \( (r = -0.762; p = 0.006) \). The amount of biceps brachii co-contraction that occurred during voluntary reaching also had a moderate but non-significant correlation to the co-contraction that occurred during triceps brachii MVC testing \( (r = 0.551; p = 0.099) \).

The only significant correlation, with regard to co-contraction, that occurred during MVC testing of either the biceps or triceps brachii was the relationship between biceps co-contraction during triceps brachii MVC testing and paresis of the triceps brachii \( (r = 0.645; p = 0.032) \).

Comparisons between involved and uninvolved biceps brachii EMG amplitudes during triceps brachii MVC trials, using paired t-tests, showed significantly higher amplitudes of the involved biceps brachii muscle. The mean difference was 14.17% (SD 17.43), \( p = 0.030 \) indicating more co-contraction of the involved biceps brachii muscle.

**Myotonometric muscle stiffness correlations**

The relationship between muscle stiffness measurements obtained during voluntary reaching and those obtained during passive movements were highly correlated for both tested extremities (uninvolved biceps brachii, \( (r = 0.882; p = 0.050) \); involved biceps brachii, \( (r = 0.853; p = 0.068) \)). Biceps brachii stiffness of the involved upper extremity during voluntary reaching (AUC = 19.20) and uninvolved stiffness measurements (AUC = 18.21) were not significantly different \( (p = 0.712) \). Similar findings were acquired during passive movement testing \( (p = 0.562) \). Differences between involved and uninvolved biceps brachii stiffness obtained passively and differences obtained during voluntary reaching were correlated \( (r = 0.797; p = 0.002) \). These data indicate that absolute biceps brachii stiffness measurements obtained during voluntary elbow extension at a self-selected speed and passive elbow extension are not significantly different and are highly correlated. Biceps brachii sEMG (RMS during duration of activation) of the involved upper extremity and that of the uninvolved upper extremity were not significantly different (3.37 (SD 2.7) mV; 2.66 (SD 2.96) mV, respectively; \( p = 0.319 \)). The stiffness measurements obtained during voluntary or passive movements did not correlate significantly to the MAS, DTRs or Fugl-Meyer testing (Table II).

**DISCUSSION**

Paresis of the biceps and triceps brachii and co-contraction of the biceps brachii during voluntary reaching were the impairments most significantly correlated to motor performance, as measured by Fugl-Meyer Upper Extremity motor testing. Paresis following stroke has been a general finding (1, 5, 15). Biceps and triceps brachii paresis both correlated to increased levels of biceps brachii co-contraction during voluntary reaching (see Table II).
Excessive co-contraction of a lengthening muscle post-stroke does not appear to be as general of a finding as paresis. Studies that examined co-contraction during MVCs of individuals post-stroke, reported excess co-contraction of antagonist muscles (5, 7). This was not the case in studies that used either self-selected speed or force of movements (6, 16). Increased force or speed requirements appear to increase the amount of co-contraction. The present study used self-selected speed of movement and no increase in biceps brachii co-contraction or stiffness was noted. In summary, the present study, and others, indicate that lengthening muscle co-contraction increases in response to the force generation required of the shortening muscles (5, 7, 17), direction (6, 16, 18), speed of movement (15), and whether the shortening muscle is a flexor or extensor (1, 15).

In the present study, there was a strong correlation between biceps brachii co-contraction and Fugl-Meyer Upper Extremity direction (6, 16), speed of movement (15), and whether the lengthening muscle stiffness increases in response to the velocity that did not elicit reflex-induced increases in lengthening muscle stiffness.

Spatistic muscles typically demonstrate a velocity-dependent resistance to stretch (26). During self-selected movement speeds, however, stretch reflexes of a lengthening muscle do not appear to impede movement in any way (1, 27, 28). It would appear that although increased passive muscle stiffness and decreased reflex thresholds are indeed present in individuals with chronic hemiparesis post stroke, these impairments do not appear to be the primary limitations during voluntary, unperturbed movement to a predicted target.

Present findings clearly indicate that paresis and co-contraction of a lengthening muscle contribute to upper extremity dysfunction post-stroke. Additionally, triceps brachii paresis and biceps brachii co-contraction are strongly correlated. These findings pose considerable challenges for clinicians because interventions that decrease excess tone and co-contraction, such as various pharmacological and injection protocols, tend to cause muscle paresis. Myotonometric measurements might be useful in monitoring these treatment effects since the device quantifies muscle stiffness (tone) and measurements taken during isometric muscle contraction quantify muscle strength (29, 30). The study adds to a body of literature (20, 23, 31) that does not support the continued use of the MAS as a clinical or research tool because of validity/reliability issues and because it does not correlate to stretch reflexes, muscle stiffness, or upper extremity motor performance. The present study emphasized correlations among impairments and motor performance. Intervention studies will greatly assist in determining the causality of the relationships among impairment, motor performance and function.

ACKNOWLEDGEMENTS

We thank Steven C. Fehrer, PT, PhD for his review. This work was partially funded by a M. J. Murdock Charitable Trust Foundation grant to Dr C. T. Leonard. Dr C. T. Leonard is co-patent holder of the Myotonometer, a device used in the study, and is a principal in Neurogenic Technologies, Inc., the manufacturer and distributor of the device.

REFERENCES


