7-2006

CORRELATION BETWEEN IMPAIRMENT AND MOTOR PERFORMANCE DURING REACHING TASKS IN SUBJECTS WITH SPASTIC HEMIPARESIS

Charles Leonard
University of Montana - Missoula, charles.leonard@umontana.edu

Kelsey A. Gardipee
University of Montana - Missoula

Jennifer R. Koontz
University of Montana - Missoula

John-Henry Anderson
University of Montana - Missoula

Scott A. Wilkins
University of Montana - Missoula

J Rehabil Med 2006; 38: 243–249

Taylor & Francis
Taylor & Francis Group

CORRELATION BETWEEN IMPAIRMENT AND MOTOR PERFORMANCE DURING REACHING TASKS IN SUBJECTS WITH SPASTIC HEMIPARESIS

Charles T. Leonard, PhD, PT[1,2], Kelsey A. Gardipee, DPT[1], Jennifer R. Koontz, DPT[1], John-Henry Anderson, DPT[1] and Scott A. Wilkins, DPT[1]

From the [1]Motor Control Research Laboratory, School of Physical Therapy and Rehabilitation Science, The University of Montana, Missoula and [2]Neurogenic Technologies, Inc., Missoula, USA

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 co-contraction of the biceps brachii 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.

J Rehabil Med 2006; 38: 243–249

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.

Passive tests (i.e. tests performed on a non-voluntarily moving arm), such as deep tendon reflexes (DTRs), and modified Ashworth scores (MAS) are typically used to characterize muscle tone changes. Newer methods, such as myotonometric muscle stiffness measurements, are also typically done on a resting or passively moved limb. It remains unclear whether or not these passive tests are representative of tone changes that occur during voluntary movements or have a relationship 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.

 ISSN 1650-1977
DOI: 10.1080/16501970600609808

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 myotonometric 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 abduction 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 armature's 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 provide a measure of muscle tone (stiffness) (13). For the present experiments, myotonometric 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 Myotonometer 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. Myotonometric 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

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

Age (years)/Sex	Months after CVA	Involved side	MRI scan data	Elbow flexor MAS (0–4)	Biceps brachii DTR (1–5)	Fugl-Meyer (0–50)
62/M	161	Left	Occlusion of right carotid artery	1.5	3.5	32
55/M	227	Right	Left temporal-parietal region, occlusive infarction	2	3.5	27
55/M	52	Right	Internal capsule extending to left globus pallidus and thalamus	1.5	3.5	16
70/M	21	Right	No scan data	2	3.5	9
43/M	11	Right	Left mid cerebral artery	2	3	11
68/M	49	Right	Left parietal subcortical white matter	3	3.5	26
64/F	135	Left	No scan data	1.5	3	14
58/F	19	Left	No scan data, but "posterior aspect of brain" reported	2	4	7
77/M	324	Right	No scan data	1	3.5	22
56/M	41	Right	No scan data	3	3.5	2
75/F	9	Left	Middle cerebral artery distribution infarct	1	2	2
71/M	156	Left	No scan data	1.5	3.5	39
63/M	59	Right	Embolus to left inferior frontal gyrus	1	3	41
Mean (SD)						
62.85 (9.49)	97.23 (97.19)			1.77 (0.67)	3.31 (0.48)	19.08 (13.21)

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.

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 represented 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 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*

		% Difference between uninvolved and involved passive	% Difference between uninvolved and involved active	Elbow flexor MAS	Biceps brachii DTR	Fugl-Meyer	Biceps paresis	Triceps paresis	% Co-contraction mV of involved	% Co-contraction mV of uninvolved	% Co-contraction involved during triceps MVC	% Co-contraction involved during biceps MVC
% Difference between uninvolved & involved passive	Correlation sig. (2-tailed)											
% Difference between uninvolved & involved active	Correlation sig. (2-tailed)	**0.797** **0.002**										
Elbow flexor MAS	Correlation sig. (2-tailed)	−0.091 0.778	−0.150 0.642									
Biceps brachii DTR	Correlation sig. (2-tailed)	−0.453 0.139	−0.366 0.241	−0.161 0.600								
Fugl-Meyer	Correlation sig. (2-tailed)	0.485 0.110	0.428 0.165	−0.155 0.612	−0.081 0.792							
Biceps paresis	Correlation sig. (2-tailed)	−0.239 0.455	−0.422 0.171	0.114 0.712	0.149 0.626	**−0.667** **0.013**						
Triceps paresis	Correlation sig. (2-tailed)	−0.160 0.619	−0.269 0.398	0.119 0.699	0.153 0.619	**−0.752** **0.003**	**0.789** **0.001**					
% Co-contraction mV of involved	Correlation sig. (2-tailed)	−0.164 0.652	−0.394 0.259	0.292 0.384	−0.181 0.594	**−0.762** **0.006**	**0.683** **0.021**	**0.756** **0.007**				
% Co-contraction mV of uninvolved	Correlation sig. (2-tailed)	−0.124 0.702	0.053 0.871	0.208 0.495	−0.331 0.270	−0.309 0.305	−0.015 0.961	0.158 0.606	0.563 0.071			
% Co-contraction involved triceps MVC	Correlation sig. (2-tailed)	−0.207 0.542	−0.474 0.141	−0.156 0.647	0.137 0.688	−0.500 0.118	0.475 0.140	**0.645** **0.032**	0.551 0.099	−0.223 0.510		
% Co-contraction involved biceps MVC	Correlation sig. (2-tailed)	0.359 0.309	0.353 0.317	−0.091 0.803	−0.586 0.075	−0.351 0.320	0.535 0.111	0.508 0.134	0.312 0.380	−0.166 0.646	0.473 0.167	

% Difference between uninvolved and involved passive = Myotonometric muscle stiffness measurements of the differences between extremities during passive movements; % Difference between uninvolved and involved 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+ = group 2; score of 2 = group 3; score of 3 = group 4. The DTR data (range 2–4) were 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 ($r = 0.789$; $p = 0.001$). Triceps 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 motor testing ($r = -0.76$; $p = 0.006$). However, when data from all subjects were considered ($n = 13$), excess co-contraction did not appear to increase during voluntary movements. One interpretation of these findings would be that individuals post-stroke, in the absence of force requirements, move at speeds that do not elicit co-contraction. However, those with severe upper extremity limitations (lowest Fugl-Meyer scores) co-contract regardless of agonist force and speed requirements.

The second aim of the study was to assess the relationship of neurological testing procedures to muscle characteristic data and motor performance. The MAS did not correlate with deep tendon reflexes, muscle stiffness or upper extremity motor performance (Table II). Others have reported a lack of a relationship between the MAS and stretch reflexes or muscle stiffness (4, 19, 20). It would appear that the MAS is an inappropriate test to assess muscle tone or the lack of physiological correlations are secondary to statistical limitations of the measurement. For example, poor MAS reliability (21) increase variance, and clustering of scores, secondary to poor MAS discriminative ability (22), will both negatively affect correlation. Despite these limitations, the MAS has moderate correlations with self-rated spasticity scores (23).

The third aim of the study was to assess the correlation between passive measurements of biceps brachii muscle stiffness and those obtained during voluntary reaching. Clinically, myotonometric measurements of muscle stiffness are typically obtained from a muscle at rest (3, 12, 13). It was of interest to determine if these passive measurements were indicative of stiffness changes of a lengthening muscle that might occur during functional tasks such as voluntary reaching. Results indicated strong correlations among the measurements. Measurements obtained from a resting muscle, therefore, provide a clinical prediction of the amount of lengthening muscle stiffness that can be anticipated during voluntary reaching tasks. Myotonometric data, however, did not indicate significantly increased biceps brachii stiffness of the involved upper extremity during reaching tasks. Previous work, using high velocity or large amplitude torque motor induced stretches, reported increased stiffness of the lengthening muscle of subjects with chronic hemiparesis (3, 24). Furthermore, spastic muscle biopsies have shown the muscles to be atrophic, shorter and stiffer than normal muscle (25). The current study assessed stiffness of the lengthening muscle during voluntary movement at a self-selected speed and only through a range that the subject could obtain using volitional effort. It is possible that agonist muscle paresis limited the available range before opposing muscle stiffness increases became measurable or that self-selected speeds were of a velocity that did not elicit reflex-induced increases in lengthening muscle stiffness.

Spastic 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

1. Levin MF, Hui-Chan C. Ankle spasticity is inversely correlated with antagonist voluntary contraction in hemiparetic subjects. Electromyogr Clin Neurophysiol 1994; 34: 415–425.

2. Pandyan AD, Barnes CI, Johnson GR. A biomechanical investigation into the validity of the modified Ashworth Scale as a measure of elbow spasticity. Clin Rehabil 2004; 17: 290–293.
3. Rydahl SJ, Brouwer BJ. Ankle stiffness and tissue compliance in stroke survivors: a validation of Myotonometer measurements. Arch Phys Med Rehabil 2004; 85: 1631–1637.
4. Sommerfeld D, Eek U, Svensson A, Holmqvist L, von Arbin M. Spasticity after stroke: its occurrence and association with motor impairments and activity limitations. Stroke 2004; 35: 134–139.
5. Chae J, Yang G, Kyu Park B, Labatia I. Muscle weakness and cocontraction in upper limb hemiparesis: Relationship to motor impairment and physical disability. Neurorehabil Neural Repair 2002; 16: 241–248.
6. Gowland C, deBruin H, Basmajian JV, Plews N, Burcea I. Agonist and antagonist activity during voluntary upper limb movement in patients with stroke. Phys Ther 1992; 72: 624–633.
7. Hammond MC, Fitts SS, Kraft GH. Co-contraction in the hemiparetic forearm: Quantitative EMG evaluation. Arch Phys Med Rehabil 1988; 69: 348–351.
8. Damiano D, Quinlivan J, Owen B, Shaffrey M, Abel M. Spasticity versus strength in cerebral palsy: relationships among involuntary resistance, voluntary torque and motor function. Eur J Neurol 2001; 8: 40–49.
9. Bohannon RW, Smith MB. Interrater reliability of a modified Ashworth scale of muscle spasticity. Phys Ther 1987; 67: 206–207.
10. Fugl-Meyer AR, Jaasko L, Leyman I, Olsson S, Steglind S. The post-stroke hemiplegic patient. I. A method for evaluation of physical performance. Scand J Rehab Med 1975; 7: 13–31.
11. Aarrestad D, Williams M, Fehrer S, Mikhailenok E, Leonard C. Intra- and inter-rater reliabilities of the Myotonometer when assessing the spastic condition of children with cerebral palsy. J Child Neurol 2004; 19: 894–902.
12. Leonard C, Deshner W, Romo J, Suoja E, Fehrer S, Mikhailenok E. Myotonometer intra- and inter-rater reliabilities. Arch Phys Med Rehabil 2003; 84: 928–932.
13. Leonard C, Stephens J, Stroppel S. Assessing the spastic condition of individuals with upper motoneuron involvement: Validity of the Myotonometer. Arch Phys Med Rehabil 2001; 82: 1416–1420.
14. Domholdt E. Physical therapy research: principles and applications. Philadelphia: WB Saunders Co., 1993.
15. Knutsson E, Martensson A, Gransberg L. Influences of muscle stretch reflexes on voluntary, velocity-controlled movements in spastic paraparesis. Brain 1997; 120: 1621–1633.
16. Tang A, Rymer WZ. Abnomal force: EMG relations in paretic limbs of Hemiparetic human subjects. J Neurol Neurosurg Psychiatry 1981; 44: 690–698.
17. Kamper DG, Rymer WZ. Impairment of voluntary control of finger motion following stroke: role of inappropriate muscle coactivation. Muscle Nerve 2001; 24: 673–681.
18. Dewald JP, Pope PS, Given JD, Buchanan TS, Rymer WZ. Abnormal muscle coactivation patterns during isometric torque generation at the elbow and shoulder in hemiparetic subjects. Brain 1995; 118: 495–510.
19. Fellows SJ, Kaus C, Ross HF, Thilmann AF. Disturbances of voluntary arm movement in human spasticity: the relative importance of paresis and muscle hypertonia. In: Thilmann AF, ed. Spasticity: mechanisms and management. Berlin: Springer-Verlag, 1993, p. 139–149.
20. Vattanasilp W, Ada L. The relationship between clinical and laboratory measures of spasticity. Aust J Physiother 1999; 45: 135–139.
21. Pandyan AD, Johnson GR, Price CI, Curless RH, Barnes MP, Rodgers H. A review of the properties and limitations of the Ashworth and modified Ashworth scales as measures of spacticity. Clin Rehabil 1999; 13: 373–383.
22. Katz RT, Rymer WZ. Spastic hypertonia: mechanisms and measurement. Arch Phys Med Rehabil 1989; 70: 144–155.
23. Platz T, Eickhof C, Nuyens G, Vuadens P. Clinical scales for the assessment of spasticity, associated phenomena, and function: a systematic review of the literature. Disabil Rehabil 2005; 27: 7–18.
24. Dewald JP, Schmit BD, Dhaher Y, Rymer WZ. Reflex torque response to movement of the spastic elbow: theoretical analyses and implications for quantification of spasticity. Ann Biomed Eng 1999; 27: 815–829.
25. Friden J, Lieber R. Spastic muscle cells are shorter and stiffer than normal cells. Muscle Nerve 2003; 27: 157–164.
26. Sanger TD, Delgado MR, Gaebler-Spira D, Hallett M, Mink JW, Task Force on Childhood Motor Disorders. Classification and definition of disorders causing hypertonia in childhood. Pediatrics 2003; 111: 89–97.
27. Lee WA, Boughton A, Rymer WZ. Absence of stretch reflex gain enhancement in voluntarily activated spastic muscle. Exp Neurol 1987; 98: 317–335.
28. Fellows SJ, Ross HF, Thilmann AF. The limitations of the tendon jerk as a marker of pathological stretch reflex activity in human spasticity. J Neurol Neurosurg Psychiatry 1993; 56: 531–537.
29. Bizzini M, Mannion A. Reliability of a new, hand-held device for assessing skeletal muscle stiffness. Clin Biomech 2003; 18: 459–461.
30. Leonard C, Brown J, Price T. Comparison of surface electromyography and myotonometric measurements during isometric contractions. J Electromyogr Kinesiol 2004; 14: 709–714.
31. Pomeroy VM, Dean D, Sykes L, Faragher EB, Yates M, Tyrrell PJ, et al. The unreliability of clinical measures of muscle tone: implications for stroke therapy. Age Ageing 2000; 29: 229–233.