EVIDENCE FOR STRENGTH IMBALANCES AS A SIGNIFICANT CONTRIBUTOR TO ABNORMAL SYNERGIES IN HEMIPARETIC SUBJECTS

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Following stroke, the first voluntary movements that return are stereotyped mass patterns called flexor and extensor synergies.8,26 In the upper limb, the flexor synergy incorporates elbow flexion, shoulder abduction, and external rotation, whereas the extensor synergy involves elbow extension, shoulder adduction, and internal rotation.8 Patients who continue to recover develop the ability to produce movements outside of these synergy patterns, and finally to perform isolated movements. This suggests that abnormal synergies are a significant impairment that should be addressed with rehabilitation.

Methods that precisely quantify these synergies would aid in understanding their origins, which in turn could aid in the development of rehabilitation treatments. It is presumed that these synergies are predominantly due to abnormal neural coupling between motor neuron pools during voluntary effort. However, another possible cause of synergies is strength imbalances, which occur when much greater reduction in maximal voluntary torque occurs with a particular joint action than with the opposite joint action. For example, during attempts to abduct the shoulder, a coactivation strategy at the elbow would increase stiffness and stabilize the elbow joint in normal subjects, but result in significant flexion torque in stroke subjects unless central mechanisms compensate for a strength imbalance toward flexion at the elbow. Thus, an imbalance toward elbow flexion manifests itself as synergistic coupling between abduction and elbow flexion during attempts to abduct the shoulder. A possible mechanism that can explain strength imbalances is greater activation impairment in certain muscle groups compared to their antagonists after stroke.9,10 A second possible mechanism is the loss of

Abbreviations: ANOVA, analysis of variance; DOF, degree-of-freedom; ECG, electrocardiogram; EMG, electromyogram; FM, Fugi-Meyer assessment; MVC, maximal voluntary contraction; NDT, neurodevelopmental treatment approach; RMS, root-mean-square

Key words: electromyography (EMG); joint torque; maximal voluntary contraction (MVC); neural coupling; synergies

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control options after stroke. For example, the inability to selectively activate an agonist muscle without cocontracting the antagonist represents a loss of control options that would result in strength imbalances and synergistic activity.

One approach to quantifying synergies is to measure electromyographic (EMG) signals from several muscle groups simultaneously. In an early study, EMG was recorded from elbow muscles during force generation in several directions. Compared to the less-impaired limb, elbow flexors and extensors in the paretic limb had systematic shifts in the direction of force generation that elicited the largest EMG amplitude. In a more recent study, it was found that the shoulder muscles had similar shifts, and the elbow muscles were active over an increased range of force directions. There was also greater coactivation in elbow flexors–extensors, elbow flexors–shoulder abductors, and elbow extensors–shoulder adductors. Although EMG methods are useful, they provide a limited assessment of the contribution of synergies to functional impairment. EMG can quantify the activation level during a particular task relative to maximal activation; however, EMG alone cannot be used to assess absolute muscle force. Thus, a muscle with severe activation impairment can have an EMG pattern that appears normal even though the force from that muscle is abnormally low. Furthermore, it is difficult to noninvasively record EMG from all of the muscles of the upper limb. Nevertheless, when coupled with joint torque measurements, patterns of EMG activity are useful for investigating the origins of abnormal synergies.

Joint torques summarize the effects of all relevant muscles, and so offer a description of synergies that has more relevance to functional impairments than EMG. This involves simultaneously measuring torques at several degrees-of-freedom (DOFs). Previous studies have focused on the elbow flexion/extension, internal/external rotation, abduction/adduction, and shoulder flexion/extension. Boissy et al. used a custom apparatus to measure torques due to involuntary associated movements in the more-affected upper limb during voluntary grasp in the opposite hand. In severely impaired subjects, there was abnormally increased shoulder flexion, internal rotation, and elbow flexion torque in the more-affected limb. Increased EMG amplitude in several muscles was consistent with these abnormal torques. More recently, Dewald et al. measured joint torques at the shoulder and elbow during maximal voluntary contractions (MVCs). They defined the joint torque to be maximized as the “primary” torque and the torques at other DOFs were defined as “secondary” torques. They found coupling between primary and secondary torques that paralleled clinical descriptions of the flexor and extensor synergies.

In this study, we extended this line of investigation by quantifying synergies during MVC with both joint torque and EMG methods. Our goal was to gain insight into the respective contributions of strength imbalances and neural coupling to abnormal secondary torques. Neural coupling would be evidenced by abnormal coactivation between agonist muscles for the MVC and muscles that contribute torque at secondary DOFs. Evidence for strength imbalances would be the presence of abnormal torques in the direction of strength imbalances at secondary DOFs, and significant correlations between strength imbalance and secondary torque magnitudes.

MATERIALS AND METHODS

Subject Characteristics. Twenty-seven subjects with hemiparesis participated in the study. Subjects were included in the study if they had a diagnosis of a single stroke and were more than 6 months post-stroke. Subjects were excluded if they exhibited any upper extremity joint pain or range-of-motion limitations that would limit their ability to complete the protocols. No subject had unstable cardiovascular, orthopedic, or neurological conditions. Subjects were excluded if they were unable to cooperate with the study tasks, or if attention or comprehension scores on the Cognistat were in the severely impaired range. Eight neurologically normal control subjects also participated in the study. All protocols were approved by the local institutional review committee and informed consent was obtained from all subjects.

Clinical Evaluations. An occupational therapist tested all stroke subjects with the upper limb portion of the Fugl-Meyer assessment (FM). The sensory portion of the FM assesses light touch and proprioception at the arm, hand, and wrist, whereas the motor portion of the FM examines motor impairments in the arm and hand. The validity and reliability of the FM have been established. The portion of the motor FM covering proximal movement (shoulder and elbow) was used to segregate subjects into three impairment groups. Subjects in the severe impairment group had scores below 10 (out of a maximum of 36), whereas the moderate impairment group had scores from 10 through 19. The mild impairment group had scores of 20 and
above. The choice of these grouping criteria was based on our subject pool. Because the actual scores in our subject pool ranged from 1 to 30, we divided this range of scores into even thirds in order to achieve three distinct groups.

**Experimental Arrangement and Test Protocols.** A customized apparatus was used to measure the MVCs. Subjects were seated in a high-back wheelchair in front of a height-adjustable table. To reduce movement of the wheelchair during the testing, we replaced the cloth backing with a solid back, and the cloth seat with a solid cushion. If flexing of the wheelchair was apparent during the testing, one of the experimenters manually stabilized the chair by grasping the hand holds on each of the side bars. Straps and the contoured seat limited torso movement. The forearm was strapped to a reinforced, custom-made splint that restricted wrist and hand movement. This splint was attached to the table through a six-axis force/torque sensor with 0.25-N resolution (Delta 330-30, ATI Industrial Automation, Inc., Apex, NC). The elbow was flexed to 90°, and the shoulder was placed in 30° abduction, neutral flexion, and neutral rotation. Length measurements of the upper limb segments were used to estimate the location of the elbow and glenohumeral joint centers relative to the sensor. A simple algorithm based upon these measurements converted the sensor data to torque at the following four DOFs: elbow flexion/extension; shoulder flexion/extension; shoulder abduction/adduction; and shoulder internal/external rotation. Data from the sensor were collected at 1000 Hz and stored on computer for later analysis.

Disposable pediatric electrocardiographic (ECG) electrodes (2.2-cm diameter, 1-cm spacing; Model P-4, Lead-Lok, Inc., Sandpoint, ID) were used to record surface EMG over several muscles during the MVCs. Biceps, triceps (long head), pectoralis major, anterior deltoid, middle deltoid, posterior deltoid, and infraspinatus were all monitored. Electrode placement was in accordance with recommendations by Perotto et al. An eight-channel EMG system (Viking Ile, Nicolet Biomedical, Inc., Madison, WI) was used to amplify and view the EMG traces during the testing. EMG data were band-pass filtered between 20 and 500 Hz and collected at 1000 Hz.

In each test session, two MVCs were performed for eight joint actions in the following order: elbow flexion; elbow extension; external rotation; internal rotation; abduction; adduction; shoulder flexion; and shoulder extension. The experimenter first demonstrated the required torque by manually resisting the less-affected limb and instructing the subject to “push against my hands.” The hands were placed in standard positions and the experimenter applied force in directions to encourage the activation of the target muscle groups. Then the subject was instructed to perform the same action with the more-affected limb, and to hold their maximum for 2 s. Verbal encouragement was given during the effort, but no additional instructions were given until the trial was over. During the MVC, the primary joint torque was instantaneously displayed to the subject on the computer screen in the form of a bar graph. The test session was performed with the more-affected limb of the stroke subjects on two occasions within the same week. Controls performed the test with their nondominant limb on one occasion.

**Data Reduction.** For each trial, the shoulder and elbow torques were calculated as described earlier. The primary torque refers to the joint action the subject was instructed to maximize, and the secondary torques are the torques at the other DOFs. Gravity and passive tissue effects were eliminated by biasing all data with resting torque levels. A 0.5-s moving average window was passed across all the torque time traces. After this time-averaging, the largest torque value achieved over the two MVCs for a joint action defined the peak primary torque. The secondary torques were the average torque values in the secondary DOFs over the 0.5-s window associated with the peak primary torque. The EMG amplitudes during the peak primary torque were calculated as the root-mean-square (RMS) values over this same 0.5-s window. For each joint action, the 0.5-s window that had the largest average torque defined the peak within-session torque. For each muscle, the 0.5-s window that had the largest RMS EMG value defined the peak within-session EMG amplitude.

A special procedure was used to eliminate ECG artifact in the EMG traces. For each 0.5-s window of interest, the pectoralis major EMG trace was inspected to determine the time periods when ECG artifact appeared. Inspection was aided by viewing the 0.5-s window alongside another trace from that muscle that had ECG artifact, but no EMG activity. This provided visual information of the characteristic shape of the artifact. It was not possible to identify contaminated periods if the EMG amplitude was greater than the amplitude of the artifact. The contaminated time periods (usually 40–60 ms) were marked and the subsequent RMS calculations ignored these periods. There was never more than one contaminated period within each 0.5-s window.
Table 1. Group characteristics.

<table>
<thead>
<tr>
<th>Impairment</th>
<th>Severe (n = 9)</th>
<th>Moderate (n = 9)</th>
<th>Mild (n = 9)</th>
<th>Controls (n = 8)</th>
<th>P-value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>60.9 ± 4.0</td>
<td>64.0 ± 2.5</td>
<td>68.1 ± 3.9</td>
<td>63.5 ± 3.3</td>
<td>&gt;0.52</td>
</tr>
<tr>
<td>Male/female</td>
<td>5/4</td>
<td>8/1</td>
<td>6/3</td>
<td>4/4</td>
<td>&gt;0.32</td>
</tr>
<tr>
<td>Months post-stroke</td>
<td>22.0 ± 4.6</td>
<td>39.8 ± 8.7</td>
<td>26.1 ± 7.2</td>
<td>N/A</td>
<td>&gt;0.19</td>
</tr>
<tr>
<td>Right/left stroke</td>
<td>8/1</td>
<td>6/3</td>
<td>N/A</td>
<td>&lt;0.031</td>
<td></td>
</tr>
<tr>
<td>Sensory FM (max = 12)</td>
<td>7.2 ± 1.3</td>
<td>7.9 ± 1.6</td>
<td>11.7 ± 0.2</td>
<td>N/A</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Motor FM (max = 66)</td>
<td>9.6 ± 1.3</td>
<td>15.3 ± 0.9</td>
<td>44.2 ± 3.3</td>
<td>N/A</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Proximal motor FM (max = 36)</td>
<td>5.1 ± 0.9</td>
<td>12.2 ± 0.7</td>
<td>25.6 ± 1.4</td>
<td>N/A</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

Data expressed as mean ± standard error of the mean.
FM, upper-limb Fugl–Meyer assessment.
*Continuous data tested with analysis of variance (ANOVA); categorical data tested with chi-square test.

Once these contaminated periods were identified in the pectoralis major EMG trace, the synchronous periods in the other muscles were inspected and ignored in the RMS calculation if there was ECG artifact.

In order to compare residual strength levels in subjects of vastly different body type, the percentage residual strength was obtained by normalizing each subject’s peak within-session torque with normative strength values. Estimates of normative strength for each subject were obtained from regressions on age, gender, and weight reported in the literature.2,18 We used normative data from the literature instead of measuring strength in the contralateral limb due to the possibility of strength deficits ipsilateral to the CVA.1 To quantify strength imbalances at each DOF, the peak within-session torque in one direction was subtracted from that in the opposite direction. This difference was then normalized by the torque range, defined as the sum of the peak torques in the two directions.

To evaluate abnormal synergies independent of strength deficits, joint torques and EMG amplitudes were normalized by peak within-session values. For the stroke subjects, metrics from the two sessions were averaged before group comparisons. Abnormalities in the stroke groups were defined as significant differences compared to the control group.

Statistical Analysis. The stroke groups were compared to the neurologically normal (control) group. Group differences in subject characteristics were evaluated with analysis of variance (ANOVA, continuous and ordinal data) and chi-square tests (categorical data). Group differences in performance metrics were tested with ANOVA. When all possible intergroup comparisons were of interest, post hoc testing was done with Tukey’s test. When comparison to the control group was the primary focus, Dunnett’s test was used.

Potential cross-talk between the three heads of the deltoid was evaluated with a cross-correlation method.28 This method calculates the percentage of common signal between any two EMG traces recorded simultaneously, and is usually performed over a period when the muscle contractions are fairly constant. For each of the 0.5-s periods used to calculate the EMG amplitudes, the cross-correlation technique was applied to two of the three heads of the deltoids in turn, and in all possible combinations.

RESULTS

An analysis of group characteristics is presented in Table 1. Sensation was more impaired in the severe group than in the mild group (P < 0.037). In terms of the upper limb motor FM, the severe and moderate groups were no different (P > 0.15), but both were different from the mild group (P < 0.0001). All three stroke groups were distinguishable by their proximal motor FM (P < 0.0001).

Residual strength in the stroke groups is presented in Figure 1. All stroke groups had significant strength deficits compared to controls in all joint actions (P < 0.001). The severe and moderate groups were not distinguishable in terms of any joint action. Compared to the mild group, the severe and moderate groups had larger deficits in several joint actions (P < 0.025).

Strength imbalances were calculated at the four DOFs (Fig. 2). In general, the stronger joint actions were elbow flexion, internal rotation, adduction, and shoulder flexion. Compared to the control group, there were significant imbalances toward these joint actions, particularly in the severe group.
The secondary torques during MVCs are presented in Figure 3. There were significant abnormalities in all stroke groups. In 16 of 19 cases, the abnormalities occurred during MVCs toward the stronger joint actions, and involved increased torques in the stronger directions at secondary DOFs (summarized in Table 2). Therefore, torque coupling was predominantly within the set consisting of elbow flexion, internal rotation, adduction, shoulder flexion. These abnormalities were most prevalent in the severe impairment group, and least prevalent in the mild impairment group.

There was evidence of abnormal neural coupling in the EMG data (Fig. 4). In the severe and moderate groups, there was a tendency for some muscles to have higher EMG amplitude during MVC toward a joint action that was not the primary biomechanical action of the muscle. For example, triceps EMG amplitude in the severe group was higher during internal rotation than elbow extension. All of the EMG abnormalities in muscles at secondary DOFs occurred during MVCs toward the stronger joint actions (Table 3). EMG amplitudes were abnormally increased in muscles that contribute torque to the stronger joint actions (pectoralis major, biceps, and anterior deltoid), and these were generally consistent with torque abnormalities. However, during MVC involving shoulder flexion and elbow flexion, there were six cases of increased internal rotation and adduction torque that were inconsistent with the EMG recordings. These increased secondary torques were not caused by increased EMG amplitude of pectoralis major nor by reductions in EMG amplitude in external rotators and abductors (infraspinatus, posterior deltoid, middle deltoid).

When acting as an antagonist to the primary torque direction, there was increased cocontraction in triceps, infraspinatus, middle deltoid, and posterior deltoid. At secondary DOFs, there was increased cocontraction in biceps–triceps in two cases, and in anterior-posterior deltoid in three cases. However, the strength imbalances were not due to increased cocontraction in antagonist muscles. Strength imbal-
ances were in the direction of elbow flexion, internal rotation, adduction, and shoulder flexion. If cocontraction produced these imbalances, increased EMG activity of biceps would be expected during elbow extension MVC, increased EMG activity of pectoralis major during external rotation or abduction MVC, and increased EMG activity of anterior deltoid or pectoralis major during shoulder extension MVC. None of these abnormalities was present in the EMG data (Fig. 4).

Subjects with larger strength imbalances tended to generate larger secondary torques. Correlations were calculated between strength imbalances and average secondary torque during MVCs for the stronger joint actions. Significant correlations were found for all DOFs (P < 0.006). Pearson correlation coefficients ranged from 0.52 to 0.76. Correlations were also calculated between residual strength and average secondary torque (Fig. 5). For all DOFs, average secondary torque was not significantly correlated with residual strength in the stronger joint action (P > 0.1), but was so in the weaker joint action (P < 0.002). For example, stroke subjects with the largest strength deficit in elbow extension also

FIGURE 3. Joint torques in the severe (black bars), moderate (gray bars), mild (white bars), and control (dotted bars) groups during MVC testing. Each column represents the primary and secondary torques during a particular MVC condition. Each row represents the relative joint torque levels at a specific DOF (i.e., elbow flexion/extension) across all eight MVC conditions. Post hoc comparisons detected differences between the stroke groups and the control group: * P < 0.05; * P < 0.10. Error bars are standard error of the mean.
generated the largest elbow flexion secondary torques. In contrast, there was no correlation between elbow flexion strength and elbow flexion secondary torques. This pattern was repeated in the other DOFs.

We calculated the cross-talk levels between the three heads of the deltoid. When averaged across all subjects and all cases, anterior–middle deltoid cross-talk was 11.1%, anterior–posterior deltoid cross-talk was 11.6%, and middle–posterior deltoid cross-talk was 17.5%. Because these cross-talk values were higher than expected from purely random events, we reevaluated our significant results after neglecting subjects with greater than 10% cross-talk in the deltoids. In all cases, the statistical results were unchanged.

**DISCUSSION**

We found evidence that both strength imbalances and neural coupling contribute to joint torque synergies in the paretic upper limb following stroke. In some cases, increased secondary torques were consistent with increased EMG amplitudes in biceps, pectoralis major, and anterior deltoid. In other instances, increased secondary torques were better explained by strength imbalances. Due to large strength imbalances, similar muscle activation patterns produced larger secondary torques in the stroke subjects than controls. This explanation is supported by the finding that subjects with larger strength imbalances tended to generate larger secondary torques.

Increased adduction and internal rotation torques during MVC involving shoulder flexion was the most prevalent abnormality that was not consistent with EMG data. Other studies have reported this synergy during attempts to perform movements involving shoulder flexion in one case, this was attributed to increased reliance on pectoralis major. Because our study used a maximum-effort task and pectoralis major is a shoulder flexor, it was not surprising that all groups had nearly maximal activation of pectoralis major during MVC of shoulder flexion (Fig. 4). Pectoralis major also produces significant adduction and internal rotation torque in this posture. It is therefore possible that, in the stroke groups, there was a lack of counteracting external rotation and abduction torques that would normally be produced to stabilize the joint. This explanation is consistent with the finding that the largest internal rotation and adduction secondary torques were produced by subjects with the largest deficits in external rotation and abduction strength. An alternative explanation is that activation was abnormally increased in muscles that were not monitored, such as subscapularis, teres major, and lattissimus dorsi.

In several instances, EMG amplitudes were abnormally increased in muscles associated with the more weakened joint actions (e.g., posterior deltoid, middle deltoid, infraspinatus, triceps), but these abnormalities were not consistent with the torque data (Table 3). For example, in the group with severe impairment, middle-deltoid EMG amplitude was increased during MVC of shoulder flexion, but adduction torque was increased. This illustrates the difficulty in interpreting EMG data in weak muscles. Despite increased normalized EMG amplitude, these weak muscles were not able to produce sufficient force to cause abnormalities in joint torque.

**Comparison to Clinical Observations.**

Some of the joint torque synergies we observed are consistent with clinical descriptions. Internal rotation–adduction and external rotation–abduction joint torque coupling are consistent with extensor and flexor synergies. However, these couplings were not classified as abnormal because they were also present in controls. This is not surprising, because, from biomechanical considerations, these synergies...
are required to perform many functional daily tasks that involve force generation at the hand.

Most of the abnormal shoulder–elbow synergies we observed involve coupling between elbow flexion and adduction, internal rotation, and shoulder flexion. These are not consistent with clinical descriptions of the complete flexor and extensor synergies.

However, closer examination of Brunnstrom’s description of these synergies reveals that the strongest component of the flexor synergy (elbow flexion) and the strongest components of the extensor synergy (shoulder adduction and internal rotation) are commonly expressed together.25 The elbow flexion–shoulder flexion synergy we observed is also consis-
tent with a previous study that reported abnormal coupling between biceps and anterior deltoid.7

**Comparison to Previous Studies.** A common mechanism may underlie the expression of synergies during MVCs, involuntary associated movements, and the flexion reflex. The most prevalent synergy we observed was coupling within the set consisting of elbow flexion, internal rotation, adduction, shoulder flexion. A similar synergy was observed in a study of associated movements in the paretic limb during voluntary grasp in the opposite limb.6 The associated movements consisted of simultaneous elbow flexion, shoulder flexion, and internal rotation torque. This synergy was also observed in a study of flexion reflexes elicited by electrical stimulation.12 The flexion reflex consisted of elbow flexion in all eight subjects, and shoulder flexion in seven, adduction in five, and internal rotation in five subjects.

Our experimental protocol is similar to that of Dewald et al.11 Both studies found abnormally increased elbow flexion, internal rotation, and adduction torque during shoulder flexion MVC. However, Dewald et al. reported abnormal coupling between elbow flexion and abduction that we did not classify as abnormal, although trends in our data were consistent with this coupling. During abduction MVC, the mean level of elbow flexion torque was nearly

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Elbow flexion</th>
<th>Internal rotation</th>
<th>Shoulder adduction</th>
<th>Shoulder flexion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Biceps</td>
<td>x*</td>
<td>x</td>
<td>x*</td>
<td>x*</td>
</tr>
<tr>
<td>Triceps</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x*</td>
</tr>
<tr>
<td>Infraspinatus</td>
<td>x*</td>
<td>x</td>
<td>x</td>
<td>x†</td>
</tr>
<tr>
<td>Middle deltoid</td>
<td>x*</td>
<td>x†</td>
<td>x</td>
<td></td>
</tr>
<tr>
<td>Anterior deltoid</td>
<td>x†</td>
<td>x</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Posterior deltoid</td>
<td>x</td>
<td></td>
<td></td>
<td>x‡</td>
</tr>
</tbody>
</table>

MVC, maximal voluntary contraction; x, abnormal.
*Consistent between torque and EMG.
†P < 0.1 significance level (P < 0.05 for all other entries).

**FIGURE 5.** Correlation analysis between average secondary torques and residual strength. For each chart, the x-axis represents the mean secondary torque during MVCs for the stronger joint actions. The positive y-axis represents residual strength in the stronger joint action (circles), and the negative y-axis represents residual strength in the opposite direction (diamonds). Each stroke subject is represented in each chart by one circle and one diamond that both have the same x-axis value. *Significant correlation, P < 0.002.
two times larger in the moderately impaired group than controls, and significant abduction torque also occurred during MVC of elbow flexion. These secondary torques were not found to be abnormal because there was considerable variability, and controls performed similarly. Consequently, in our study, the presence of an abnormal elbow flexion–abduction synergy may have been masked by the performance of the control group. The control groups performed differently in the two studies, and this might be explained by differences in limb posture that may have imposed different stability requirements at the shoulder, prompting different secondary torques.

Limb posture can also directly influence abnormal synergies in hemiparetic subjects. Many of our more severely impaired subjects required passive constraint to position the resting limb into the test posture. This passive constraint could have stretched certain muscle groups (especially internal rotators) and may have contributed to their overactivity. There is evidence that holding an antagonist muscle in a stretched position may result in increased cocontraction during isometric testing. Furthermore, a test position placing the limb in shoulder flexion may have resulted in less activity in shoulder flexors and increased activity in shoulder extensors compared to our results. In future studies, the postural dependence of synergies should be evaluated.

Mechanisms and Implications for Treatment. Possible mechanisms for strength imbalances include differential loss of descending drive to antagonistic muscle groups, or differential alteration of excitability of motor neuron pools in the spinal cord. Another possibility is inability to selectively activate an agonist muscle without cocontracting the antagonist muscles, which represents a loss of control options. Abnormal neural coupling could be due to altered descending commands from the cortex, or a greater reliance on brainstem pathways with their more diffuse connectivity, spanning several motor neuron pools. Alternatively, neural coupling could be due to a spinal mechanism, caused by a change in the tonic firing level of interneurons, which increases the excitability of selected motor neuron pools. The severely impaired group had significantly more sensory impairment than the mildly impaired group, and also had the largest abnormal synergies. Compromised sensation may impede recovery from abnormal synergies. Many stroke subjects develop abnormal synergies, but only some of these subjects progress to recover isolated movement. Feedback from muscle spindles, Golgi tendon organs, and tactile sensors may be critical to producing the focused contractions observed in the mildly impaired group. This sensory feedback may also be critical to the cortical reorganization that is believed to drive the recovery process.

Our results have implications for the rehabilitative treatment of motor dysfunction following stroke. According to the neurodevelopmental treatment approach (NDT), normalizing synergistic movement at secondary DOFs is a primary focus. This is often accomplished by guiding subjects to inhibit activation of the muscles producing the synergistic movements. This technique would be appropriate when abnormal neural coupling is the cause of synergistic movements. Because attempts to control secondary torques may lead to concomitant loss of primary torque strength, training that emphasizes simultaneous control of muscles at both primary and secondary DOFs may be needed to limit abnormal neural coupling. However, our data also suggest that some synergistic activity is due to an abnormal lack of neural drive to weak muscles at secondary DOFs. In such cases, methods to increase neural drive to weaker muscle groups would be more effective than attempts to inhibit stronger muscles.

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