IMPAIRMENT OF VOLUNTARY CONTROL OF FINGER MOTION FOLLOWING STROKE: ROLE OF INAPPROPRIATE MUSCLE COACTIVATION

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Accepted 20 December 2000

Residual chronic hemiparesis occurs in over 40% of those individuals with an acute hemiparesis following stroke. Chronic deficits are especially prevalent in the distal upper extremities, and finger extension is the motor function most likely to be impaired. Although the effects of therapy on finger extension following stroke have been studied, classification and quantification of voluntary deficits have not been thoroughly examined. Furthermore, the origins of the deficits in finger extension have not been well established. Although increased passive resistance to extension of the metacarpophalangeal (MCP) and interphalangeal (IP) joints impedes voluntary finger extension in some cases, it is absent in others. Thus, increased passive resistance is neither a universal nor sole cause of impaired finger extension. Indeed, spontaneous finger extension—seemingly triggered by a yawn or stretching of the finger flexors—has been observed, and anecdotal evidence of spontaneous finger extension has been reported by patients.

Increased active resistance to imposed finger extension, resulting from a spastic response to stretch of the long finger flexors, has been described. However, the true role of spasticity in degrading voluntary movement is not clear. Difficulty in producing extension torque occurs even in isometric situations, whereas spasticity is dependent on change in muscle length and is described as a velocity-dependent phenomenon.

Diminished ability to voluntarily activate extensor muscles undoubtedly plays a large role in finger extension deficits. Yet preliminary data led us to surmise that another factor may contribute significantly to such deficits. We hypothesized that motor dysfunction in the fingers involves improper coactivation of the finger flexors and extensors. This phenomenon has been shown to occur in the wrist muscles following stroke, likely contributing to impairment. It has also been recorded in the biceps and triceps, although many researchers have not found significant cocontraction of these muscles.

Thus, we sought to assess the extent of coactiva-

Abbreviations: EMG, electromyography; FDI, first dorsal interosseous; IP, interphalangeal; MCP, metacarpophalangeal

Key words: coactivation; fingers; reciprocal inhibition; stroke; voluntary control

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tion during voluntary extension of the MCP joints in individuals with chronic hemiplegia. Subjects attempted to voluntarily extend the fingers about the MCP joints while either opposed or assisted by a servocontrolled motor under isometric, isokinetic, and free-range conditions. The extent of coactivation was assessed through analysis of torque, position, and electromyographic (EMG) signals.

MATERIALS AND METHODS

Subjects. Eleven stroke subjects (five women and six men) with chronic unilateral motor deficits participated in the original study, which focused exclusively on isometric contractions. Subjects averaged 59 years of age (range, 51–74 years), and their duration of deficit ranged from 2 to 20 years. Lesion sites were varied, but each resulted in significant involvement of the contralateral hand. The unilateral lesion was frontal in 2 (subjects A and I), frontoparietal in 2 (subjects C and K), frontotemporal in 1 (subject D), basal ganglionic in 3 (subjects B, G, and J), capsular in 2 (subjects B and E), diffuse subcortical areas in 1 (subject F), and paraventricular in 1 (subject H). Subjects were selected for the presence of an involuntary bias toward a flexed finger posture. Every subject was rated a minimum of 2 out of 4 on the 5-point Ashworth scale for resistance to imposed extension of the fingers \(^2\) (0 indicates normal resistance, whereas 4 indicates a rigid limb), and exhibited a hyperreflexive response to tendon tap of the fingers while extending from the shaft. For each trial, the servomotor moved the MCP joints to an angle randomly selected from the set of six angles and maintained this position. Two trials were performed at each position for each subject. A fiberglass cast, placed around the wrist and secured to a table with clamps, kept the wrist in a neutral posture with respect to the forearm and prevented pronation/supination.

The second set of experiments examined finger extension under isokinetic and free-range, no-load conditions. The isokinetic trials began with the fingers positioned such that the MCP joints were at the testing-range limit of extension. The servomotor rotated the fingers into maximum flexion at a constant rate, typically 2°/s, held that position for 2 s, and then returned to the original position. The rate of rotational velocity was kept below the threshold level necessary for eliciting a stretch reflex.\(^20\) Subjects were instructed to contract their extensor muscles eccentrically as their fingers were rotated from extension to flexion and concentrically as their fingers were rotated from flexion to extension.

For the free-range trials, the subjects were instructed to actively extend the MCP joints as far as possible from an initial posture removed from the testing extension limit by 75% of the range. The motor controller maintained zero torque during these trials so that no external loads impeded free rotation of the MCP joints.

Angular position, rotational velocity, and torque were measured throughout the trials with a position encoder (PMI Motion Technologies, #138647), tachometer (PMI Motion Technologies), and torque transducer (Transducer Techniques, Temecula, California; TRT-200). Surface EMG recordings were made from surface electrodes (Con Med Corporation, Utica, New York, or DelSys Inc., Boston, Massachusetts) positioned above the extrinsic MCP flexors (flexor digitorum superficialis/profundus) and extensor (extensor digitorum communis). The activity of the largest intrinsic MCP flexor, the first dorsal interosseous (FDI), was additionally measured during these trials so that no external loads impeded free rotation of the MCP joints.

Protocol. The first set of experiments examined the ability of the subjects to generate flexion and extension torques about the MCP joints of all four fingers under isometric conditions. Peak active torques were recorded at six different positions, evenly spaced across the testing range for the MCP joints. The testing range consisted of the middle 75% of the available passive range of motion for the MCP joints. Metacarpophalangeal angle was controlled through coupling of the fingers to the shaft of a servomotor (PMI Motion Technologies, Comack, New York; 1.4 HP), as previously described.\(^20\) The fingers were positioned such that the MCP joints were aligned roughly along a vertical line extending from the shaft. For each trial, the servomotor moved the MCP joints to an angle randomly selected from the set of six angles and maintained this position. Two trials were performed at each position for each subject. A fiberglass cast, placed around the wrist and secured to a table with clamps, kept the wrist in a neutral posture with respect to the forearm and prevented pronation/supination.

For the isometric experiments, peak active
torque in the desired direction was determined for each trial from the signal obtained by subtracting the initial, passive torque from the peak torque recorded. The maximum EMG envelope value across all trials was found for each subject for both the extensor and flexor EMG. Envelope values were subsequently normalized for comparison between subjects by division by the appropriate maximum. The normalized envelope was integrated for ±100 ms, centered about the peak extension torque for trials in which extension torque was to be generated or peak flexion torque for trials in which flexion torque was to be generated. Division of the integral value by its 200-ms range yielded the average EMG during the time of peak torque. Because attempts by stroke subjects to create an extension torque sometimes resulted in a net flexion torque, average EMG for the extension trials was computed for the period of largest absolute torque as well as for peak extension torque. Paired t-tests were used to compare EMG activity in attempted flexion and extension in the flexors and extensors. Comparisons of muscle activity between stroke and control subjects were made through t-tests. A repeated-measures analysis of variance (ANOVA) was utilized to evaluate the effect of static MCP angle, relative to the testing range, on the degree of coactivation.

For the isokinetic experiments, an average active torque was computed for both the eccentric and concentric movements. Active torque was calculated by subtracting estimated passive torque from the recorded torque. Passive torque was assumed to vary linearly with MCP angle, in accordance with prior findings. The slope of the line was determined by dividing the difference between the passive torque at the end of the rest portion of the trial and that at the beginning of the stretch by the elapsed time. The negative of this slope was employed for the concentric portion of the trial (Fig. 1). Mean active torque was computed by integrating the active torque during the stretch and dividing by the duration of the stretch. Paired t-tests were used to compare active torques during the eccentric and concentric contractions across all stroke subjects and all control subjects.

Average EMG activity was computed for the extensors, flexors, and FDI by integrating the normalized EMG envelopes with respect to time during movement and by dividing this value by the elapsed time of the movement. Muscle activity was compared through an ANOVA, with the factors muscle group (extensor, flexor, or FDI), movement direction (concentric or eccentric), and the interaction term. T-tests were utilized to examine possible differences in EMG activity between stroke and control subjects.

For the free-range trials, peak MCP displacement in both the flexion and extension directions were found for each trial. The amounts of flexion and extension were measured with respect to the neutral position, the posture where the proximal phalanges were aligned with the metacarpal bones. Peak values for the flexor, extensor, and FDI envelopes were also determined. Single-factor ANOVAs were employed with post hoc Tukey tests to compare the peak EMG envelopes across stroke subjects and across control subjects. Relative EMG activity in the two groups was evaluated with a t-test.

As surface electrodes were used to measure EMG, the possibility existed that volume conduction of the EMG signals may have led to cross-talk between the electrodes, thereby confounding the assessment of coactivation. To evaluate this hypothesis, cross-correlations were performed on the raw EMG signals for each of the trials with the control subjects. Amplitude of the cross-correlation was normalized with respect to the value of the autocorrelation at zero time shift. Peak correlation amplitude was determined for each trial.

RESULTS
Isometric Muscle Activation. In the isometric extension protocol, only two of the 11 stroke subjects were able to generate a net extension torque greater than 0.21 N-m, a value less than 16% of the mean level produced by the control subjects. Conversely, control of voluntary flexion torque was seen in all subjects, as each could generate at least 0.75 N-m of

![FIGURE 1. Estimation of active and passive torque from the torque values recorded during the isokinetic trials (data from control subject C-B).](image-url)
torque about the MCP joints. Attempts to generate significant voluntary isometric extension torque frequently resulted in the inappropriate production of flexion torque instead (Fig. 2). This occurred despite the presence of measurable extensor EMG activity (Fig. 2 and Table 1).

As the amount of flexor and extensor EMG activity did not significantly vary with MCP angle, data for the different positions were grouped together in subsequent EMG analyses. Interestingly, one such analysis revealed that extensor activity was typically greater during isometric voluntary flexion than during voluntary extension (Fig. 2 and Table 1). T-tests revealed that for six of the stroke subjects, average extensor EMG activity measured around the time of the peak flexion torque during maximum voluntary isometric flexion was significantly greater than that for peak extension torque during attempted maximum voluntary isometric extension ($P < 0.001$). However, the normalized flexor EMG activity was still significantly greater during flexion than extension ($P < 0.001$). For the normal control subjects, extensor activity was greater during voluntary extension ($P < 0.001$) and flexor activity was greater during voluntary flexion ($P < 0.001$).

Comparisons of EMG activity between stroke and control groups revealed excessive coactivation in the stroke subjects. Using the data for EMG activity at peak torque regardless of direction, we found that average flexor activity during attempted isometric extension was significantly greater in stroke (0.199) than in control (0.081) subjects ($P < 0.001$). Normalized extensor activity was smaller for stroke (0.266) than for control (0.347) subjects ($P = 0.001$). For voluntary flexion, relative extensor activity in the stroke subjects (0.346) was much greater ($P < 0.001$) than in the controls (0.056). Yet the normalized flexor activity was statistically indistinguishable (stroke, 0.349; controls, 0.365).

**Isokinetic Finger Movement.** Isokinetic trials of voluntary finger extension revealed an even greater propensity for inappropriate finger flexion during attempted finger extension (Fig. 3). This occurred more commonly during concentric than eccentric contractions and, correspondingly, extension torque values were significantly smaller ($−0.537 \pm 0.407$ versus $0.002 \pm 0.382$; $P < 0.001$). In fact, none of the stroke subjects generated a net extension torque during the concentric portion of the extensor movement, even though net extension could be produced during eccentric contractions (Fig. 4A).

The diminution of MCP extension torque was not caused by a large increase in flexor activity as, for the stroke subjects as a group, flexor EMG activity increased by 22% ($\pm 48\%$), whereas FDI activity decreased by 10% ($\pm 47\%$) during the concentric contractions. Rather, it seemed to result from a change in extensor activity, which dropped by 48% ($\pm 67\%$) during concentric as opposed to eccentric movements. The decrease in extensor activity with the change in direction was significant in the ANOVA ($P < 0.001$). Normalized extensor, flexor, and FDI activity were not significantly different from each other.

The control subjects were always able to generate net extension torques, both eccentrically and concentrically, although eccentric torques were greater

![FIGURE 2: Example of attempted isometric voluntary extension (A) and flexion (B) for subject J. Efforts to create an extension torque resulted in the development of a flexion torque instead. Rectified EMG signals (lighter) are shown along with corresponding envelopes (darker). Note that both flexor and extensor EMG activity are much greater during voluntary flexion.](image)
Average normalized extensor EMG activity was almost identical for the two directions (0.319 versus 0.311). Extensor EMG activity was significantly greater than flexor (0.047 and 0.036) and FDI (0.021 and 0.019) activity.

Comparisons of average EMG activity between groups again revealed excessive coactivation in the stroke subjects. Similar to the isometric condition during voluntary extension, the stroke subjects had less relative extensor activity and greater flexor and FDI activity for both eccentric and concentric contractions (overall \( P < 0.001 \)).

**Free-Range Movements.** Even with no load, it was difficult for stroke subjects to generate finger extension. Again, attempts to generate MCP extension often resulted in inappropriate MCP flexion instead (Fig. 5 and Table 2). Only two of the seven stroke subjects who participated in these trials were able to produce significant MCP extension. However, even these two subjects were not able to attain the testing range limit of MCP extension (maximum possible extension of 32.1° for subject D and 48.3° for subject K). In contrast, all the control subjects easily reached their testing range limits of extension.

The two stroke subjects who were able to generate some MCP extension exhibited greater normalized extensor EMG activity than that for the flexors and FDI. The other stroke subjects did not. Overall, ANOVA results for the stroke subjects revealed that peak normalized EMG envelopes were not distinct for extensors, flexors, or FDI. The control subjects had much greater extensor than flexor or FDI activity (\( P < 0.001 \) for Tukey tests; Table 2).

The presence of improper coactivation was affirmed by EMG measurement. Across stroke subjects, normalized peak extensor activity was significantly smaller than for control subjects (stroke, 

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**Table 1. Voluntary isometric contractions.**

<table>
<thead>
<tr>
<th>Subject</th>
<th>Ashworth score, Finger Flexors</th>
<th>Peak Active Torque</th>
<th>Mean Extensor EMG</th>
<th>Mean Flexor EMG</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Ext</td>
<td>Fix</td>
<td>Ext</td>
</tr>
<tr>
<td>A</td>
<td>3+</td>
<td>0.006</td>
<td>0.759</td>
<td>0.161</td>
</tr>
<tr>
<td>B</td>
<td>3</td>
<td>0.016</td>
<td>3.002</td>
<td>0.057</td>
</tr>
<tr>
<td>C</td>
<td>3</td>
<td>0.017</td>
<td>3.424</td>
<td>0.170</td>
</tr>
<tr>
<td>D</td>
<td>2</td>
<td>0.674</td>
<td>3.056</td>
<td>0.253</td>
</tr>
<tr>
<td>E</td>
<td>2</td>
<td>0.047</td>
<td>2.644</td>
<td>0.174</td>
</tr>
<tr>
<td>F</td>
<td>3+</td>
<td>0.203</td>
<td>0.912</td>
<td>0.254</td>
</tr>
<tr>
<td>G</td>
<td>3+</td>
<td>0.024</td>
<td>2.100</td>
<td>0.227</td>
</tr>
<tr>
<td>H</td>
<td>2</td>
<td>0.064</td>
<td>2.625</td>
<td>0.169</td>
</tr>
<tr>
<td>I</td>
<td>2</td>
<td>0.110</td>
<td>2.349</td>
<td>0.178</td>
</tr>
<tr>
<td>J</td>
<td>2</td>
<td>0.014</td>
<td>2.978</td>
<td>0.013</td>
</tr>
<tr>
<td>K</td>
<td>2+</td>
<td>0.390</td>
<td>1.695</td>
<td>0.452</td>
</tr>
<tr>
<td>C-A</td>
<td>0</td>
<td>1.650</td>
<td>2.350</td>
<td>0.377</td>
</tr>
<tr>
<td>C-B</td>
<td>0</td>
<td>2.283</td>
<td>3.855</td>
<td>0.376</td>
</tr>
<tr>
<td>C-C</td>
<td>0</td>
<td>1.915</td>
<td>4.892</td>
<td>0.425</td>
</tr>
<tr>
<td>C-D</td>
<td>0</td>
<td>1.332</td>
<td>5.393</td>
<td>0.293</td>
</tr>
<tr>
<td>C-E</td>
<td>0</td>
<td>1.729</td>
<td>5.402</td>
<td>0.381</td>
</tr>
<tr>
<td>C-F</td>
<td>0</td>
<td>2.246</td>
<td>4.887</td>
<td>0.245</td>
</tr>
</tbody>
</table>

Abbreviations: Ext, attempted isometric extension; Ext*, maximum absolute torque during attempted isometric extension; Fix, attempted isometric flexion.

*C-* indicates control subject.
0.283; control, 0.583; P < 0.001), whereas flexor (stroke, 0.235; control, 0.066; P < 0.001) and FDI (stroke, 0.222; control, 0.040; P < 0.001) activity were much greater.

**Estimation of EMG Cross-Talk.** Cross-correlation analyses supported the supposition that the coactivity recorded by the flexor and extensor electrodes did not merely result from cross-talk among the electrodes due to volume conduction of the signal. Peak, normalized correlation values for all of the control subjects were less than 0.1 for all subjects (Table 3).

**DISCUSSION**

Our experiments confirmed the presence of chronic deficits in motor control of finger extension in the involved hands of hemiplegic stroke patients across all three conditions tested: isometric, isokinetic, and free movement. The subjects were not selected a priori for impairment in voluntary finger extension but rather for an involuntary flexion posture bias and heightened resistance to imposed finger extension. In fact, Ashworth score was not significantly correlated with performance. With the isometric protocol, only two of the 11 stroke subjects could generate substantive MCP joint extension torque. During the isokinetic trials, all the stroke subjects failed to produce measurable extension torque during concentric contractions. For the free movements, only two of seven stroke subjects could ex-

![FIGURE 4.](image)

![FIGURE 5.](image)

**Table 2.** Attempted free extension.

<table>
<thead>
<tr>
<th>Subject</th>
<th>Free Movement (°)</th>
<th>Peak EMG</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Extension</td>
<td>Flexion</td>
</tr>
<tr>
<td>C</td>
<td>0.03</td>
<td>24.29</td>
</tr>
<tr>
<td>D</td>
<td>18.82</td>
<td>2.16</td>
</tr>
<tr>
<td>G</td>
<td>0.03</td>
<td>6.23</td>
</tr>
<tr>
<td>H</td>
<td>0.02</td>
<td>18.87</td>
</tr>
<tr>
<td>I</td>
<td>1.38</td>
<td>14.23</td>
</tr>
<tr>
<td>J</td>
<td>0.02</td>
<td>19.60</td>
</tr>
<tr>
<td>K</td>
<td>12.39</td>
<td>12.51</td>
</tr>
<tr>
<td>C-A</td>
<td>38.83</td>
<td>0.09</td>
</tr>
<tr>
<td>C-B</td>
<td>44.77</td>
<td>5.29</td>
</tr>
<tr>
<td>C-C</td>
<td>48.38</td>
<td>3.03</td>
</tr>
<tr>
<td>C-D</td>
<td>58.30</td>
<td>0.04</td>
</tr>
<tr>
<td>C-E</td>
<td>47.78</td>
<td>0.02</td>
</tr>
<tr>
<td>C-F</td>
<td>47.05</td>
<td>1.25</td>
</tr>
</tbody>
</table>

Values displayed are the means across all trials. *C-* indicates control subject.
Alvarez16 observed similar problems of excessive an-
tometric flexion in stroke subjects. Hallett and
tensor EMG activity recorded during voluntary iso-
duced by the significantly greater normalized ex-
regardless of the intended torque direction, as evi-
shown not to be an artifact of surface EMG, occurred
for stroke subjects than for controls. Coactivation,
during voluntary extension was significantly greater
Across conditions, normalized flexor and FDI activity
to voluntary extensor activity may share a com-
tate extensor excitability, as extensor EMG activity
indicated an inability to voluntarily excite these mo-
ments.11,14,17 The reduced extensor EMG activity
coactivation, shown not to be an artifact of surface EMG, occurred
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denced by the significantly greater normalized ex-
tensor EMG activity recorded during voluntary iso-
motor flexion in stroke subjects. Hallett and
Alvarez16 observed similar problems of excessive an-
tagonist activity and coactivation in athetoid pa-
tients, assumed to have lesions in basal ganglia, as
did some of our subjects.

The consequences of this coactivation were com-
pounded by the reduction in normalized finger ex-
tensor muscle activity during attempted extension. This
decline in extensor activity in stroke subjects has
been also been reported in the wrist and elbow
muscles.11,14,17 The reduced extensor EMG activity
indicated an inability to voluntarily excite these mo-
tor units at the desired time. Interestingly, in a num-er of cases, flexor activity actually seemed to facili-
tate extensor excitability, as extensor EMG activity
was greater during voluntary flexion than voluntary
extension. In contrast, the level of normalized flexor
activity during isometric flexion was indistinguish-
able between the stroke and control groups.

The origins of these anomalous EMG patterns
are unknown, but several possibilities bear consider-
ation. Indeed, inappropriate coactivation and re-
duced voluntary extensor activity may share a com-
mon origin. Potential causes include loss of
reciprocal inhibition of the long flexors, preferential
activation of cortical areas responsible for coactiva-
tion of limb muscles, and cortical or spinal segmen-
tal reorganization.

Loss of descending input to spinal interneurons
and alpha motoneurons may alter the patterns of
reciprocal inhibition.8 Indeed, the normal disynap-
tic inhibition of antagonist motoneurons and presyn-
aptic inhibition of antagonist Ia afferents by agonist
afferents5 is reduced for the ankle flexor afferents in
multiple sclerosis patients9 and the forearm extensor
afferents in stroke subjects.26 In our study, we ob-
erved that the normalized flexor activity in stroke
subjects was significantly higher during voluntary
MCP extension than in controls. This may indicate a
reduction in the inhibition of the finger flexors by
extensor afferents. Conversely, during the isokinetic
trials, extensor EMG activity dropped by almost 50%
from the eccentric portion of the trial to the con-
centric portion, the period during which flexors
were being stretched. Excitation of the flexor stretch
receptors may aggravate flexor coactivation15 and
thereby lead to extensor inhibition. Alterations in
reciprocal inhibition between flexors and extensors
have been observed after spinal cord injury7 and
stroke,3 leading to coactivation of muscle groups
with subsequent degradation in movement.

Increased coactivation may also derive from se-
lective damage of neurons in the brain. Humphrey19
proposed that separate classes of motor cortical cells
exist: the first class activates synergistic muscles,
whereas the second class coactivates antagonistic sets
of muscles.19 Relative sparing of the coactivation
zone with respect to the synergistic zone may lead to
the favoring of finger flexor–extensor coactivation
over independent activation. Similarly, Purkinje cell
activity in the cerebellar cortex has been found to
decrease in monkeys when coactivation rather than
reciprocal activation of the flexors and extensors is
performed.15 it is therefore conceivable that reduc-
tions in Purkinje cell excitability, mediated by loss of
corticopontine projections damaged in stroke, may
also promote coactivation.

Central reorganization following stroke may con-
tribute to the coactivation, as well. In adult monkeys,
areas in premotor cortex become involved in control
of the hand, thereby enabling partial return of func-
tion, following ablation of the motor cortex associ-
ated with the hand.24 In a rat model, axonal sprout-
ing of corticospinal neurons occurred into areas of
the spinal cord left denervated by unilateral transec-
tion of the corticospinal tract.4 Magnetic stimulation
studies have revealed both an increase in size and a
shift in location of the cortical area capable of excit-
ing the abductor pollicis brevis after therapy in hu-

<table>
<thead>
<tr>
<th>Subject</th>
<th>Peak flexor vs extensor, mean (SD)</th>
<th>Peak FDI vs extensor, mean (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>C-A</td>
<td>0.020 (.016)</td>
<td>0.021 (.034)</td>
</tr>
<tr>
<td>C-B</td>
<td>0.009 (.007)</td>
<td>0.008 (.013)</td>
</tr>
<tr>
<td>C-C</td>
<td>0.013 (.014)</td>
<td>0.016 (.027)</td>
</tr>
<tr>
<td>C-D</td>
<td>0.046 (.037)</td>
<td>0.022 (.016)</td>
</tr>
<tr>
<td>C-E</td>
<td>0.042 (.015)</td>
<td>0.023 (.027)</td>
</tr>
<tr>
<td>C-F</td>
<td>0.013 (.009)</td>
<td>0.011 (.009)</td>
</tr>
</tbody>
</table>
mans with hemiplegia resulting from stroke. This cortical reorganization may lead to a more unfocused descending input, less suitable for the required fine-scale coordination of finger flexors and extensors and possibly favoring the flexors.

Neural reorganization can also occur at the segmental level in the spinal cord. Axonal sprouting may take place to fill some of the gaps left behind the degeneration of descending axons. This sprouting would alter the organization of segmental pathways, possibly including those coordinating activity between the agonist and antagonist. However, evidence of segmental reorganization following stroke is scarce.

The findings of this study suggest that improper coactivation combined with reduced voluntary extensor excitation plays a fundamental role in the measured reduction in control of finger extension in chronic hemiplegia. When coupled with mechanical attributes favoring flexion, such as the greater physiological cross-sectional area and equivalent or greater moment arms of the flexors, these neural phenomena provide an explanation for the net flexion that typically results during intended extension.

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