ABSTRACT: The origins of impaired finger and hand function were examined in 10 stroke survivors with chronic spastic hemiparesis, with the intent of assessing whether mechanical restraint or altered neurophysiological control mechanisms are responsible for the well-known impairment of finger extension. Simultaneous extension of all four metacarpophalangeal (MCP) joints of the impaired hand was either externally imposed using a rotary actuator or attempted voluntarily by the subject. Trials were conducted both before and after administration of a local anesthetic, blocking the median and ulnar nerves at the elbow. The anesthetic was administered to reduce the activity of the muscles flexing the MCP joints, in order to distinguish mechanical from neuronal resistance to imposed MCP rotation. We found that the nerve blockade resulted in a reduction in velocity-dependent torque ($P = 0.01$), thereby indicating significant joint impedance due to spasticity. Blockade also produced a posture-dependent reduction in static torque in declaratively relaxed subjects ($P = 0.04$), suggesting some tonic flexor activity for specific hand postures. No change in either extensor isometric ($P = 0.33$) or isokinetic (0.53) torque was apparent, but 3 of the 10 subjects did exhibit substantial ($>10^\circ$) improvement in voluntary MCP extension following the blockade. This improvement seemed largely due to a decrease in inappropriate flexor activity during the movement, rather than an increase in extensor activity. We argue that persistent and inappropriate flexor activation plays a role in limiting voluntary finger extension, and that this activation is potentially a reflection of altered supraspinal control of key spinal pathways. In all cases, this inappropriate activation was compounded by weakness, apparent in both the extensor and flexor muscles.


RELATIVE CONTRIBUTIONS OF NEURAL MECHANISMS VERSUS MUSCLE MECHANICS IN PROMOTING FINGER EXTENSION DEFICITS FOLLOWING STROKE

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Roughly one-third of all individuals who experience a stroke will have some residual impairment of the upper extremity.13,30,34 Finger extension is the motor function most likely to be impaired.42 The mechanisms of this impairment are not well understood, especially the origins of the stereotypical flexion bias following stroke. Motor cortical inputs are excitatory to the α-motoneurons supplying the hand musculature,35 and these inputs are about equally distributed between flexors and extensors.10 The impact on forearm muscles, however, is clearly asymmetric, in that the extensor impairment is more severe than that of the flexors.42 This suggests that brain injury resulting from stroke leads to a number of secondary changes affecting limb motion.

Abbreviations: EDC, extensor digitorum communis; EMG, electromyography; FDI, first dorsal interosseous; FDS, flexor digitorum superficialis; MCP, metacarpophalangeal
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The nature and role of these secondary changes remain a matter of debate. A number of researchers have documented changes resulting in abnormal neuronal activity such as spasticity,\textsuperscript{36,41} altered multi-joint coordination,\textsuperscript{3,6,37} inappropriate coactivation of flexors and extensors,\textsuperscript{8,14} and reduced reciprocal inhibition.\textsuperscript{5,29} Yet, other researchers have proposed that the more relevant changes are peripherally mediated, such as contracture\textsuperscript{35} or muscle atrophy.\textsuperscript{39} Furthermore, some researchers have described a reduction, rather than augmentation, of long-latency reflexes following stroke, further leading them to conclude that mechanical tissue changes are key to producing a flexor bias in the upper extremities.\textsuperscript{9,17} Indeed, an increase in resistance to ankle rotation was determined to result from increases in passive and intrinsic stiffness rather than hyperreflexia in subjects with spasticity related to multiple sclerosis.\textsuperscript{40}

Previously, we described the presence of spasticity during stretch of passive finger flexor muscles\textsuperscript{19} and excessive coactivation during voluntary hand manipulation.\textsuperscript{20} From those studies, however, it was not possible to distinguish the impact of neuronal from mechanical changes on hand function.

Our present study seeks to separate neuronal from passive resistance to finger extension. To do this, the activity of both the flexor and intrinsic muscles was temporarily reduced using local anesthesia. Data recorded during passive and voluntary extension of the metacarpophalangeal (MCP) joints prior to anesthetization were compared to the data recorded following nerve blockade. We anticipated that the nerve blockade would reduce hyperreflexia and improve finger extension and extensor torque, without affecting the relaxed, resting torque.

**MATERIALS AND METHODS**

**Subjects.** Twelve subjects with chronic hemiparesis subsequent to stroke participated in the experiments. All subjects experienced a stroke at least 1 year prior to testing in this study (range 1.5–21 years). Subjects were selected on the basis of hand function (they had a variety of lesion sites), such that they had less than 50% of full finger extension against gravity, but some finger flexion. This corresponds to a clinical assessment of either Stage 2 or Stage 3 on the Stage of Hand section of the Chedoke–McMaster Stroke Assessment.\textsuperscript{12} Hemispheric lesions were widely distributed, being cortical, subcortical, or both. Four subjects had predominantly right arm impairment, whereas eight had predominantly left arm impairment. One control subject also participated to verify that the anesthetization procedure itself did not affect finger extension. All subjects gave informed consent according to the Helsinki Declaration. The Institutional Review Board of Northwestern University approved the experimental protocol.

**Protocol.** The wrist was placed in a cast such that both wrist and forearm were maintained in neutral positions of flexion/extension and pronation/supination. The palm was positioned perpendicular to the tabletop, aligning the MCP joints with the shaft of a servomotor. The fingers were coupled to the shaft through a custom vacuum pillow.\textsuperscript{19–21} Thus, rotation of the motor shaft produced equivalent rotation of the MCP joints, and vice versa, as described in detail elsewhere.\textsuperscript{19} Range of MCP joint rotation was chosen to cover the full comfortable range for each subject, as permitted by the experimental device. Across all subjects, the value of this range was 55.1 ± 10.1°. Fixed extension and flexion limits were employed throughout each experimental session.

MCP torque (torque transducer, TRT-200; Transducer Techniques, Temecula, California), angle (incremental encoder, HA625-2500; DynaTECH, Elm Grove Village, Illinois), and angular velocity (tachometer; PMI Motion Technologies, Radford, Virginia) were measured throughout the trials. Muscle activity was recorded using active surface electromyography (EMG) electrodes (Delsys Inc., Boston, Massachusetts). Differential electrodes were placed over the flexor digitorum superficialis (FDS) to sample the extrinsic flexor muscles, extensor digitorum communis (EDC) to sample the extrinsic extensor muscles, and the first dorsal interosseous (FDI) to sample the intrinsic musculature.

The experimental trials consisted of a series of MCP rotations, with the subject relaxing, generating maximal torque, or producing maximal MCP rotation. These experiments addressed four potential neurological sources of impairment: spasticity, a hyperexcitable velocity-dependent stretch reflex\textsuperscript{25}; tonic muscle activation; coactivation of MCP flexors and extensors; and muscle weakness. The trials in which subjects remained declaratively relaxed were intended to differentiate spasticity and tonic muscle activation from mechanical resistance to stretch. The trials assessing finger movement were meant to gauge the impact of flexor coactivation on finger extension. The trials measuring maximum voluntary torque generation were conducted in order to evaluate muscle weakness.

Accordingly, four different types of trials were conducted: imposed rotation, assisted, isokinetic,
and isometric. Three trials were performed for each combination of parameters described in the following paragraphs.

We first examined the response to imposed extension of the MCP joints, in order to examine the passive and reflex components of resistance to movement. Constant-velocity rotation of the MCP joints was attained by control of the servomotor. Two speeds were employed: a speed slow enough not to produce a stretch reflex (10°/s) and one fast enough to trigger a stretch reflex (300°/s). Rotation was performed from the flexion limit to the extension limit. The MCP joints were held at the extension limit for 2 s, and then returned to the starting position. For the slower speed, further trials were conducted in which the rotational directions were reversed, such that the movement proceeded from extension to flexion. Subjects were instructed to remain relaxed throughout each trial.

To test the effects of neural blockade on finger movement, subjects performed trials of voluntary MCP rotation, with assistance provided by the servomotor when needed. A zero-load was maintained through servo-control of the motor about zero torque. The subject attempted to extend the MCP joints from a position 75% of the way toward the flexion limit to the extension limit. If movement in the flexion ("wrong") direction was detected, or no extension movement was detected over a 2-s interval, the servomotor rotated the MCP joints a net 2° further into extension using position control. If a net flexion torque was detected at the end of the imposed extension, the servomotor extended the MCP joints another 2°. Otherwise, the zero-load condition was resumed (Fig. 1). This algorithm was continued until the extension limit was achieved. The total number of degrees of assistance was recorded. This test was also repeated without assistance in the zero-load condition.

We examined the effect of neural blockade on extensor strength using both isometric and isokinetic trials. Isokinetic extension was performed both eccentrically and concentrically during constant velocity (10°/s) imposed rotations of the MCP joints. Joints were rotated from the limit of extension to the limit of flexion (during which time eccentric extension was performed), maintained at the flexion limit (during which time the subject relaxed), and then rotated from the limit of flexion to the limit of extension (during which time concentric extension was performed).

Both isometric extension and flexion trials were conducted. In order to test for a possible interaction between the nerve blockade and joint posture, the subjects attempted to produce maximal extension torque at three different MCP angles. The servomotor positioned the MCP at the flexion limit, the midpoint between the flexion and extension limits, or an angle halfway between these two postures. Isometric flexion was performed at 20° of MCP flexion.

These trials were conducted before and after neural blockade of the finger flexor muscles, performed by a physician in the following manner. After cleaning the skin area with povidone-iodine solution, the biceps tendon and brachial artery pulsations within the antecubital fossa of the hemiplegic arm were located by palpation. A 26-gauge, 37-mm Teflon-coated MyoJect needle electrode (Oxford Instruments Medical, Hawthorne, New York) was inserted medial to the brachial artery at the antecubital fold. Pulsed electrical stimulation at 1 Hz was delivered through the MyoJect needle tip by a nerve stimulator (DigiStim 3 Plus; Neurotechnology, Houston, Texas) to locate the median nerve. Localization of the nerve was achieved when both wrist and finger flexion were noted with nerve stimulation at less than 1 mA current. If localization could not be achieved, the needle was removed and redirected between the biceps tendon and brachial artery. Once localization was achieved, 3–8 cc of 1% lidocaine was injected until voluntary flexion of the index finger was eliminated.

The ulnar nerve was localized by inserting the MyoJect needle 2 cm proximal to the ulnar groove at the olecranon process of the humerus. The nerve stimulator was used as previously described. When the nerve was localized, 3–8 cc of 1% lidocaine was

![FIGURE 1. Flow chart detailing the control algorithm for the extension assist condition. Assistance was only provided when net movement or torque in the flexion direction was detected. N, no; Y, yes.](image-url)
injected until voluntary flexion of the small finger was absent. The anesthesia remained effective for 1–2 h.

Successful blockade, defined as a 50% or greater reduction in grip strength, was obtained in 10 of the 12 stroke subjects. Only the data from these subjects were included in the study. EMG was monitored during the experiments to ensure that the blockade was sustained.

Seven of the 10 subjects who experienced a successful blockade then returned for another experimental session on a different day in which the contralateral (less impaired) hand was tested, without any anesthesia. Isometric strength and stretch reflexes were assessed in this hand in a manner similar to that just described. Due to limitations in the load borne by the torque cell, maximum isometric flexion was assessed with a hand dynamometer (Jamar; Sammons Preston, Chicago, Illinois).

**Data Analysis.** Torque, velocity, and angle data were low-pass filtered at 250 Hz with a fourth-order Butterworth filter, prior to sampling at 500 Hz with an A/D board (PCI-MIO-16E-4; National Instruments Corporation, Austin, Texas). The data were subsequently digitally low-pass filtered at 10 Hz, using a 30th-order finite impulse response filter.

Raw EMG data were band-pass filtered between 10–250 Hz prior to sampling. The signals were then rectified and low-pass filtered at 10 Hz to generate envelopes for the EMG activity. Envelope amplitudes were normalized by the maximum value recorded during the experimental session.

Flexor spasticity was quantified from the data recorded during imposed rotation of the MCP joints. The peak torque, referenced from the initial resting torque, was found for the period from the start of rotation to the end of the hold phase. The mean peak torque for the slower (10°/s) trials was subtracted from the mean peak torque for the faster (300°/s) trials to obtain the velocity-dependent torque. This velocity-dependent torque does contain a passive component. However, for a given subject, differences in its value before and after neural blockade would signify a neural component as well, which would be attributable to spasticity. Thus, a paired Student’s t-test was performed on the velocity-dependent torque recorded before and after blockade.

Tonic flexor activity was assessed by examining the nominally passive torque. The values of initial torques prior to movement for the relaxed 10°/s trials starting in both flexion and extension were determined. Two paired Student’s t-tests were performed to compare the torque before and after blockade at the extension and flexion limits, respectively. To gauge the absolute magnitude of the passive torque, the magnitude of the MCP torque after blockade was determined for each subject at 0° of MCP extension during a slow rotation from flexion to extension (upper part of hysteresis curves in Fig. 3).

The effect of the nerve blockade on movement was analyzed by examining the motor-assisted trials. The total amount of assistance provided by the servomotor in extending the MCP joints was stored by the computer in units of degrees of MCP rotation for each trial. A paired Student’s t-test was used to compare the amount of assistance before and after blockade. Peak values of the EMG envelopes were determined for assessment of individual activation patterns.

Strength was assessed with the data from the isokinetic and isometric trials. Concentric and eccentric torque generation was estimated separately for the isokinetic trials. Average torque was computed by integrating the torque during either the concentric or eccentric muscle stretch and dividing by the duration of the stretch. Average active torque was computed by subtracting the values obtained during the passive trials, described previously, from those values obtained during trials in which the subject was actively contracting. The effect of the blockade on isokinetic strength was tested with a repeated measures ANOVA, with the factors contraction (eccentric, concentric) and state (pre-anesthesia, post-anesthesia).

For the isometric trials, peak active torque was computed by subtracting peak recorded torque from the resting torque for a given MCP angle. A repeated measures ANOVA was run with the factors joint posture (flexion limit, mid-flexion, midpoint) and state (pre-anesthesia, post-anesthesia). A correlation was performed between peak extension torque at the flexion limit and peak flexion torque to determine whether a relationship existed. As prolongation of flexor activity was of interest, mean torque and flexor EMG envelope values were computed over 1-s intervals at the beginning of the trial, prior to voluntary contraction, and at the end of the trial, at least 2 s after the end of voluntary effort.

Data from the three repeated trials run for each level of the factors were averaged to create a single value for each combination of factors. To obtain an overall significance level of α = 0.05, the significance level for each statistical test was set to 0.01, in accordance with the Bonferroni correction.
RESULTS

Reflex Changes. Finger extension was examined before and after blockade of the nerves to the flexor muscles in 10 stroke subjects and 1 control subject. For imposed rotation in declaratively relaxed stroke subjects, the velocity-dependent torque decreased following nerve blockade (Fig. 2). The decrease was nominally statistically significant \( P = 0.013 \), and was often larger than the magnitude of the residual passive torque. In half of the subjects, the decrease in this torque was more than 0.6 Nm, 33% of the average maximal voluntary flexion across all of the stroke subjects. No such change occurred for the control subject.

Comparison of torque before and after neural blockade revealed (indirectly) the presence of activity of the finger flexors even in static postures when the subject was declaratively relaxed. Across all 10 stroke subjects, the “resting” torque at the extension limit prior to initiation of the passive isokinetic trials decreased after administration of the local anesthetic \( (0.11 \pm 0.14 \text{ Nm, } P = 0.042) \). EMG recordings from FDS confirmed that the greater flexion torque seen before the blockade was caused by tonic flexor activity. Conversely, at the flexion limit, there was no change \( (0.02 \pm 0.09 \text{ Nm, } P = 0.531) \). Thus, this tonic activity, estimated from the torque, was dependent on finger posture. Torque profiles in response to imposed rotation of the MCP joints also demonstrated this postural dependence on the reduction in resistance to MCP rotation after blockade (Fig. 3).

The size of the passive torque was smaller than anticipated. The passive MCP torque after blockade at 0° of MCP extension was generally quite low \( (0.19 \pm 0.16 \text{ Nm for all stroke subjects}) \). For the seven subjects who underwent testing with the contralateral hand, the results were similar \( (0.23 \pm 0.12 \text{ Nm}) \). Up to the extension limits used in this study, passive torques remained within tolerable levels \( (0.35 \pm 0.20 \text{ Nm}) \) that could easily have been overcome by healthy subjects. However, three of the most severely impaired subjects (those unable to produce any finger extension) had the highest passive flexion torque.

In an interesting aside, stretch of the flexors could produce prolonged extensor activity (Fig. 4A), even though rapid stretch of the extensors fails to elicit a stretch reflex (Fig. 4B). After anesthesia of the median and ulnar nerves, however, no extensor activity was observed in conjunction with stretch of the flexors (Fig. 4C).

Movement Changes. With the weight of the fingers supported against gravity, performance of extension prior to blockade varied widely among the subjects, with some requiring little assistance from the motor and others requiring complete assistance. After blockade, there was an overall trend toward reduction in the amount of assistance needed by the stroke survivors to reach the extension limit. Exclusion of one outlier, who inexplicably demonstrated a 40° paradoxical increase in required assistance after blockade, resulted in an average reduction of 5.3 ± 9.0° \( (P = 0.113) \) across the remaining nine subjects.
Improvement was highly dependent upon performance prior to nerve blockade. Only the three subjects who exhibited partial, but not full, extension prior to blockade experienced substantial improvement (>10°) (Fig. 5). As anticipated, the control subject could easily complete the MCP extension both prior to and following the administration of anesthesia.

Results were similar for voluntary extension without motor assistance. In cases where improvement occurred, the improvement seemed to arise from a reduction in flexor coactivation, as peak EMG during movement declined by at least 17% of the maximum value for these three subjects.

This flexor coactivity increased with successive trials of attempted voluntary MCP extension prior to blockade in 3 of the 10 stroke subjects. The delay for onset of flexor activity sometimes decreased, and the amplitude occasionally reached levels sufficient to visibly affect performance (Fig. 6). This augmentation occurred despite a rest period of at least 1 min between trials.

**Strength Changes.** The majority of subjects were unable to reach the extension limit, even after nerve blockade of the flexors, suggesting that extensor weakness was also a contributing factor. Furthermore, neural blockade failed to improve either isokinetic (P = 0.528) or isometric (P = 0.331) MCP extension torque. For the seven subjects who completed trials for both hands, mean peak MCP isometric extension torque in the paretic hand before blockade was 20% of the value for the contralateral hand (1.86 ± 0.53 Nm).

Across all stroke subjects, voluntary isometric MCP flexion torque in the impaired hand was 1.80 ±
1.42 Nm. Flexion and extension strength were neither highly (Pearson’s coefficient $= 0.19$) nor significantly ($P = 0.59$) correlated. For the seven subjects who completed both experiments, grip strength of the paretic hand was on average 33% of that of the contralateral hand.

Excessive flexor activity was manifested during voluntary isometric and isokinetic extension, as well as voluntary movement. Attempts to produce extension prior to blockade often resulted in net flexion (Fig. 7A), so that the maximum recorded flexion torque, averaged across all stroke subjects, was greater than the maximum extension torque during isometric extension.

In addition to the difficulty in modulating flexor activity during the trial, we noted that many of the subjects had trouble in terminating flexor muscle activity. For example, in 5 of 10 subjects, EMG activity in the flexor muscles continued for several seconds after the end of voluntary isometric flexion. The mean flexor EMG envelope (and consequently the torque) was greater 2 s after the end of voluntary contraction, marked by the attainment of a quasi steady-state torque, than at the beginning of the trial (Fig. 7).

**FIGURE 6.** Example of the increase in flexor activity with successive trials of voluntary extension (positive MCP angle) against zero load (subject D). Trials are presented in order of performance (A–C). Peak EMG amplitude (normalized by the maximum value obtained during MVC) increased from 0.2 for the first trial to 0.7 for the last trial.

**FIGURE 7.** Continued flexor activity after attempts to voluntarily produce (A) isometric MCP extension torque (negative MCP torque; subject F) and (B) isometric flexion torque (positive MCP torque; subject J). Note how the “resting” torque at the end of the trial differs from that at the beginning in both cases, and the flexor EMG continues. EMG, gray line represents rectified EMG and black line represents EMG envelope.
DISCUSSION

Mechanical Constraints. Targeted blockade of the median and ulnar nerves innervating the muscles responsible for MCP flexion did reduce the impedance to MCP extension. The reduction was quite large in some cases, greater than the residual, passive impedance. The absolute magnitude of the resistance to imposed rotation after nerve blockade was considerably less than the extension torque capacity of the contralateral hand in the subjects tested.

Certainly, passive resistance to stretch, measured after the nerve blockade, was increased in a few of the subjects with hemiparesis, especially those with the poorest finger extension. This may have resulted from change in passive tissue characteristics. Contracture following stroke was found to be prevalent for the tissues surrounding the elbow.35 Indeed, in animal models, joint fixation in a flexed posture has been shown to lead to loss of sarcomeres within months.16

Muscle shortening may also have a more indirect effect on resistance to joint rotation. By increasing the absolute muscle-fiber length for a given joint rotation, it may increase afferent input, thereby predisposing the corresponding motoneurons to fire during stretch. A strong dependence of spasticity on absolute muscle-fiber length has been noted previously both for the finger flexors and the elbow flexors.21

Neural Constraints. From comparison of results before and after blockade, we observed that anomalous neural activation patterns play a significant role in the impairment of finger extension following stroke. These "positive" features of neural impairment following stroke35 took a variety of forms. A reduction in the velocity-dependent response to stretch after anesthesia, indicative of spasticity, was demonstrable in eight of nine subjects for whom this was measured. A much smaller but still significant response to static stretch of the finger flexors was also present. This finding suggests that group II afferent pathways to muscle spindles may produce tonic activation of flexor motoneurons. Interactions between group Ia and II pathways may further potentiate the spastic response.16

Inappropriate coactivation of the flexors and extensors of the MCP during voluntary MCP extension was also apparent. It was the reduction in this flexor coactivation after blockade that led to the clear improvement in voluntary finger extension for three of the subjects. For other subjects, anesthesia of the flexors served to diminish the net flexion torque or movement produced during attempted voluntary MCP extension, again indicating excessive flexor coactivation prior to blockade.

Difficulty in terminating excitation of FDS and FDI (the clinically described trouble with "letting go" of objects) was also evident, as was a crescendo of excessive flexor activity with repeated trials. Spasticity,41 inappropriate coactivation,20 and sustained activation7 have all been described by researchers following stroke.

The origins of these anomalous activation patterns are currently unclear, but there are several plausible mechanisms worth considering. In particular, we believe these features may originate from a common source. Namely, we surmise that brainstem pathways, such as the vestibulospinal and reticulospinal pathways, released from descending inhibition usually produced by cerebrocerebellar pathways, contribute to the heightened flexor muscle activity. These pathways tend to favor the anti-gravity muscles (flexors in the upper extremities). These brainstem pathways could elevate the resting potential of the motoneurons closer to threshold,36 most likely via interneuronal excitation. This would increase the likelihood of motoneurons firing in response to afferent input, such as group Ia or II afferents from muscle spindles during constant-velocity or static stretch, respectively.26

Monoaminergic pathways conceivably could potentiate the influence of these other brainstem pathways.22 Indeed, the administration of neuromodulators, including serotonin, was shown in vitro to raise resting potential in neonatal rat preparations15 and to enhance plateau behavior in turtles.15 This plateau behavior, in which the neurons are de-recruited at lower levels than they are recruited (and, thus, tend to sustain firing) has also been described in humans.11 Addition of monoamine agonists to cat preparations in vivo led to much greater responses to Ia excitation and stretch reflexes,24,27 and to facilitation of group I and II interneurons in the ventral horn and intermediate zone (though dorsal horn interneurons were depressed).18

Alternatively, cortical reorganization following stroke could produce some of the observed behaviors. Studies with primate models have shown considerable restructuring of the cortex in the weeks following imposed injury.31,32 Following stroke, it appears that changes may occur in a number of different areas of the brain to support recovery.14 This cortical reorganization could lead to heightened coactivation of flexors and extensors if supporting neurons were to assume control of both the finger flexor and extensor pathways. Similarly, pat-
terns of cortical inhibition of certain pathways may be altered, to favor flexion, for example.

This situation would be further exacerbated by a reduction in reciprocal inhibition following stroke, which has been reported by others for the distal upper extremities.\textsuperscript{3,29} Inhibition of flexor activity during extension would then be reduced. We saw an occurrence of the mirror situation in which passive stretch of the flexors triggered extensor activity. This is reminiscent of the “shortening reaction,” first described in the literature in the 19th century.\textsuperscript{4} Conversely, little extensor activity was observed during stretch of the extensors.

Anesthetization of the median and ulnar nerves eradicated the shortening reaction in EDC. This suggests that the reaction is evoked by flexor afferent input rather than homonymous input.\textsuperscript{2} However, another study found no effect of anesthetization of one muscle on the shortening reaction of its antagonist.\textsuperscript{38} We do not know whether the differing results are attributable to differences in subject population (spastic hemiparetic vs. athetoid), to the muscles targeted (finger flexors vs. triceps), or to the application point of the anesthesia (mixed nerve vs. motor point). Others have also suggested that the joint afferents, which may have been affected by the blockade, contribute to the shortening reaction.\textsuperscript{3}

**Muscle Weakness.** Deficits persisted even after the diminishment of some of the positive neural constraints. Isometric and isokinetic testing confirmed that these deficits arose from a profound weakness in the MCP extensor muscles. Overall, both the finger extensors and flexors were much weaker on the paretic side than in the contralateral hand. Greater deficits are observed with extension, in part, because of a threshold-like effect. As absolute levels of MCP extension torque are lower before injury, a similar level of strength reduction would leave residual strength below a threshold level at which passive resistance can be overcome. Conversely, residual flexion strength may still be sufficient to overcome passive resistance. Indeed, we have observed that among individuals with normal hand function, variable responses to the anesthesia. Yet, across subjects, the results suggest that finger extension deficits following stroke have a primarily neural origin, with nonvoluntary flexor activity exacerbating a decreased ability to voluntarily excite extensor motoneurons.

It must be noted that the findings of this study are limited by the small sample size and the heterogeneity of subjects, which seems to have led to quite variable responses to the anesthesia. Yet, across subjects, the results suggest that finger extension deficits following stroke have a primarily neural origin, with nonvoluntary flexor activity exacerbating a decreased ability to voluntarily excite extensor motoneurons.

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