ABSTRACT: Despite its potential importance in hand dysfunction, spasticity in the finger muscles following stroke has not been well described. To explore this area, we assessed the role of finger flexor spasticity, along with that of passive mechanical forces, in resisting finger movement in 13 chronic stroke subjects. Subjects were tested with a device that stretched the extrinsic finger muscles through imposed rotation of the metacarpophalangeal (MCP) joints. Both maintained and constant-velocity stretches were imposed. For the constant-velocity stretches, eight of the 13 stroke subjects exhibited strong stretch reflexes, as determined by electromyography and net work. The net work of this reflex response, calculated from the integral of the torque-angle plots, increased proportionally with increasing velocity, indicating a contribution from flexor muscle spasticity. Conversely, nine of the 13 stroke subjects did not possess distinctly greater passive, mechanical resistance to MCP rotation than control subjects. While extensor spasticity was not observed, stretch of the extrinsic finger flexors also produced some reflex activity in the finger extensors concomitant with reflex excitation of the flexors. These findings suggest that resistance to muscle stretching following stroke is mediated primarily by neurological rather than biomechanical disturbances, although changes in muscle fiber length may exaggerate the resistance.


QUANTITATIVE FEATURES OF THE STRETCH RESPONSE OF EXTRINSIC FINGER MUSCLES IN HEMIPARETIC STROKE

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Accepted 1 March 2000

Common clinical practice assumes that the muscles which flex the fingers become spastic, defined by a heightened velocity-dependent reflex response to stretch, following hemispheric stroke. This spasticity, in conjunction with mechanical changes such as contracture, is believed to be the underlying cause of the difficulty in extending the fingers after stroke. Injections with botulinum toxin and electrical stimulation have both been used in efforts to reduce spasticity and, thus, improve finger function. Tendon releases and transfers have also been tried to mitigate the effects of muscle shortening and contracture.

However, to date, relatively few studies examining the pathophysiology of abnormal finger tone and impaired movement following stroke have been performed. Changes in passive stiffness opposing metacarpophalangeal (MCP) rotation have not been reported with any frequency, and spasticity of the finger flexors has not been quantified. Although spasticity has been documented in the muscles controlling elbow rotation, the direct nature of the excitatory cortical control of hand muscles suggests that potential differences might exist between the pathophysiology of finger muscle control and that of the elbow muscles in stroke.

Although numerous studies have examined hand stretch reflexes through mechanical perturbation of the fingers, these studies routinely utilized preactivation of the muscles being stretched, limiting their applicability to observations made in passive limbs. One study actually observed a reduction or dissipation of the long-latency stretch reflex in the long flexor of the thumb in individuals with cortical lesions.

In light of these deficits in our knowledge of the sources of finger dysfunction following hemiparetic
stroke, the purpose of this study was to quantify passive resistance and reflex response to stretch of the extrinsic muscles of the fingers. This effort was undertaken with the intent to improve our understanding of the underlying disturbance in control of the hand, with the view toward guiding more effective hand rehabilitation. To accomplish this task, the extrinsic finger muscles were stretched by servo-controlled rotation of the MCP joints. Both static and constant-velocity stretches were employed.

**MATERIALS AND METHODS**

**Experimental Configuration.** Stretch of the extrinsic finger muscles was accomplished though simultaneous rotation of the MCP joints of the four fingers. The flexor digitorum superficialis (FDS) and profundus (FDP) and the extensor digitorum communis (EDC) were the muscles of interest. Although other muscles such as the lumbricals and interossei are involved in MCP rotation, the targeted three extrinsic muscles are the major contributors due to their greater stretch during MCP rotation and their larger moment arms and cross-sectional areas.1,3–5

The MCP joints were aligned roughly along a vertical line extending from the shaft of a perturbing motor. The fingers were coupled directly to the servomotor (1.4 HP, PMI Motion Technologies, Kollmorgen Corporation, Radford, Virginia) such that rotation of the shaft produced an equivalent angular rotation of the MCP joints (Fig. 1).

A fiberglass cast was placed on the wrist to maintain the wrist in a neutral position with respect to the forearm. The cast also served to keep the thumb extended and abducted from the palm. The forearm was clamped to a table so as to vertically align the MCP joints of the four fingers.

The motor shaft extended vertically above the table. An aluminum channel was connected to the shaft and to a U-shaped piece into which the fingers were placed. A bead-filled bladder surrounding the fingers was attached to the inner surface of the U-piece. Evacuation of the bladder with a vacuum pump provided a rigid coupling between the fingers and the U-piece and, thus, the motor shaft. Use of the beads provided the flexibility to enable coupling with fingers that often proved difficult to position. The bladder contact was confined to the proximal phalanges of the fingers, thereby allowing the proximal and distal interphalangeal (PIP and DIP) joints to flex naturally.

**Subjects.** Thirteen subjects who had experienced an hemispheric stroke participated in the study. All subjects had chronic unilateral motor deficits and were at least 2 years post-incident (range, 2–20 years). The group was comprised of five women and eight men ranging in age from 52 to 74 years (mean, 61.5 years). Eight of the subjects had a right hemiparesis and five had a left hemiparesis. Lesion sites were diverse (Table 1). Two control subjects from the laboratory also participated in the study. Subjects gave informed consent according to the Helsinki Declaration. The Institutional Review Board of Northwestern University approved the experimental protocol.

**Protocol.** A brief physical examination was performed, in which the examiner quantified resistance to rotation of the elbow, wrist, and fingers using the 5-point Ashworth scale (ranging from 0–4).6

The subject’s arm was then mounted in the experimental device and the subject instructed to relax. The feasible range of motion for the MCP was found by manually rotating the motor shaft slowly. The limit of extension was determined from the onset of mild discomfort, as reported by the subject. The limit of flexion was defined by contact of the supporting frame with the palm. The range of MCP rotation for all testing was set equal to the middle 75% of this span.

Static passive torque data were recorded at six evenly spaced angular positions across the testing range. The servomotor slowly moved the MCP joints to an angle randomly selected from the set of six angles and held this position for several seconds, at the end of which data were collected for 2 s.

For the dynamic trials, constant-velocity (ramp-and-hold) stretches of the extrinsic finger flexors were imposed by rotating the MCP joints from the limit of flexion to the limit of extension. The MCP
joints were maintained in an extended position for 2 s, and then returned to the limit of flexion. Accel- eration to a constant velocity occurred over the first 10% of the range, with deceleration occurring over the last 10%. Five trials were performed at each of seven different constant velocities. The constant ve- locity was randomly chosen for each trial. In most cases, 6, 50, 100, 150, 200, 250, and 300°/s were the velocities used.

Angular position, rotational velocity, and torque were measured throughout the trials with a position encoder (#138647, PMI Motion Technologies), tachometer (PMI Motion Technologies), and torque transducer (TRT-200, Transducer Techniques, Temecula, California). Surface electromyography (EMG) recordings were taken from pairs of surface electrodes positioned above the FDS, FDP, and EDC.

In control subjects, stretch reflexes were elicited by having the subject apply an initial flexion torque prior to the start of the stretch, and then maintain that level of voluntary activation throughout the trial (the "do not intervene" protocol13).

Analysis. The position, torque, velocity, and EMG signals were low-pass filtered at 250 Hz and then sampled at 500 Hz. EMG signals were then notch filtered at 60 Hz and rectified. Envelopes were derived for the EMG signals through additional low-pass filtering of the rectified EMG, with a 10-Hz corner frequency.

For the static trials, resistance to stretch was calculated by averaging the recorded torque over a 2-s interval. Linear regressions were performed to relate the average torques to MCP rotation angle. Post-hoc regressions were conducted to analyze potential statistical differences between the slopes for two different subjects.

For the dynamic trials, the reflex response was quantified in terms of both the reflex threshold26 and the net work needed to stretch and release the finger flexors.18 The reflex threshold represents the angular range traversed before initiation of the stretch reflex response. Pragmatically, this threshold is estimated from the change in joint stiffness, which increases sharply with muscle activation. Joint stiffness was computed for each trial from the derivative of the reflex torque during the middle 80% of the stretch, the period of constant velocity. The reflex torque was estimated by subtracting the passive torque, fitted to the data from the trials run at a very slow velocity eliciting no reflex (6°/s), from the total torque measured. Differentiation of the reflex torque with respect to joint angle by using a five-point formula yielded the stiffness. Reflex threshold was defined as the angular displacement at which stiffness attained 20% of its maximum value for that trial.

The net work (W) represents the amount of work needed to stretch the fingers and then return them to their initial positions. This parameter provides a measure of reflex response since passive elastic forces do not contribute to it, as long as the elastic forces are roughly linear. The effects of force changes resulting from small misalignments of the MCP joints with the motor shaft are minimized as well. Net work was determined by computing through integration the area inside the hysteresis loop, as demarcated by the shaded region in Figure

### Table 1. Clinical tone and static stiffness.

<table>
<thead>
<tr>
<th>Subject</th>
<th>Lesion site</th>
<th>Ashworth score finger flexors</th>
<th>Limit of MCP extension (deg)</th>
<th>Slope of regression line (N-m/deg) × 10²</th>
<th>Intercept MCP angle (deg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>L frontal lobe</td>
<td>3+</td>
<td>19.4</td>
<td>1.18*</td>
<td>-48.0</td>
</tr>
<tr>
<td>B</td>
<td>NA</td>
<td>2+</td>
<td>4.1</td>
<td>0.58</td>
<td>-41.6</td>
</tr>
<tr>
<td>C</td>
<td>L paraventricular area</td>
<td>2</td>
<td>25.0</td>
<td>0.67</td>
<td>-11.2</td>
</tr>
<tr>
<td>D</td>
<td>L frontal lobe</td>
<td>2</td>
<td>43.5</td>
<td>0.39</td>
<td>-25.1</td>
</tr>
<tr>
<td>E</td>
<td>R subcortical areas</td>
<td>3+</td>
<td>10.1</td>
<td>1.06*</td>
<td>-53.5</td>
</tr>
<tr>
<td>F</td>
<td>R frontal and parietal lobes</td>
<td>3</td>
<td>43.0</td>
<td>0.46</td>
<td>-29.5</td>
</tr>
<tr>
<td>G</td>
<td>NA</td>
<td>1−</td>
<td>48.0</td>
<td>0.51</td>
<td>-17.1</td>
</tr>
<tr>
<td>H</td>
<td>L frontal and parietal lobes</td>
<td>2+</td>
<td>18.1</td>
<td>0.63</td>
<td>-31.2</td>
</tr>
<tr>
<td>I</td>
<td>R basal ganglia</td>
<td>2</td>
<td>27.2</td>
<td>0.64</td>
<td>-32.4</td>
</tr>
<tr>
<td>J</td>
<td>L frontotemporal lobe</td>
<td>2</td>
<td>17.9</td>
<td>0.96</td>
<td>-13.7</td>
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<tr>
<td>K</td>
<td>L basal ganglia</td>
<td>3+</td>
<td>15.2</td>
<td>1.54*</td>
<td>-39.0</td>
</tr>
<tr>
<td>L</td>
<td>R basal ganglia and internal capsule</td>
<td>3</td>
<td>35.0</td>
<td>0.50</td>
<td>-28.8</td>
</tr>
<tr>
<td>M</td>
<td>R internal capsule</td>
<td>2</td>
<td>25.0</td>
<td>1.52*</td>
<td>-18.8</td>
</tr>
<tr>
<td>AA†</td>
<td>—</td>
<td>0</td>
<td>40.0</td>
<td>0.65</td>
<td>-23.9</td>
</tr>
<tr>
<td>BB†</td>
<td>—</td>
<td>0</td>
<td>37.0</td>
<td>0.66</td>
<td>-23.4</td>
</tr>
</tbody>
</table>

†Control subject.

* Slope statistically different from that for controls (P < 0.05).

L, left; NA, Not available; R, right.
2, for the plot of torque vs. MCP angle. Torque was integrated with respect to MCP angle over the range of the MCP rotation used in the stretch. The value obtained from the portion of the trial returning from extension to flexion was subtracted from the value calculated during the stretch from flexion to extension to yield the net work. Net work was normalized through division by the range of the MCP rotation, thereby yielding the term WN. Normalization of the work term permitted comparisons among subjects. As spasticity is a velocity-dependent phenomenon, linear regressions were performed to characterize the relationship between WN and stretch velocity. Post-hoc regressions were used to compare slopes between subjects.

RESULTS

Passive Response to Stretch. Attempts to manually range the supposedly relaxed fingers encountered strong resistance in 12 of the 13 stroke subjects. Only one stroke subject was deemed to have tone in the finger flexors of magnitude less than 2/4 on the Ashworth scale (Table 1). Resistance to wrist extension was also consistently high (average 2+), with slightly less resistance to extension at the elbow.

Quantification of static passive stiffness through use of the servomotor revealed an almost linear relationship between MCP rotation angle and passive torque for the joint range utilized. Across all 15 subjects, the average coefficient of determination ($R^2$) for the linear regression was equal to 0.88 ($\pm0.09$). The slopes of the regression lines for just four of the stroke subjects were statistically greater than the slopes for the controls for $P < 0.05$ (Table 1). The MCP intercept, the joint angle at which the net passive torque about this joint is zero, was among the largest in magnitude for three of these four subjects, indicating a shift in the location of the neutral position toward flexion.

The limits of passive MCP extension were significantly correlated with both the regression slope and intercept ($P < 0.05$), with a smaller extension limit associated with a larger regression slope and a larger flexion intercept angle. However, the magnitudes of the Pearson correlation coefficients were not large (coefficient values of −0.52 and 0.53, respectively). Flexor and extensor EMG activities recorded at the static positions were negligible.

Dynamic trials employing ramp-and-hold stretches revealed the passive viscoelastic characteristics of the involved tissues. The peak torque values measured during the stretch increased modestly as a function of the velocity of the MCP rotation, even in the absence of any reflex activity (Fig. 3). The passive torque increase was 25% of the weakest reflex torque increase for which the reflex response was still significant. Relaxation resulting in hysteresis between the stretch and the return to the starting position was also observed (Fig. 3). The amount of hysteresis increased with the stretch velocity, thus denoting an increase in energy dissipation.

Reflexive Response to Stretch. Significant reflex responses to MCP joint extension were elicited in eight of the 13 stroke subjects, as determined from analysis of torque and EMG signals. These reflexes were obtained in an initially passive muscle; the start-
ing torque values ranged between zero and a slight extension torque (Fig. 4). Reflex activity in extensors associated with imposed flexion of the fingers was very rare, seen in only a couple of trials.

No stretch reflexes were observed in the control subjects when their muscles were initially passive. Stretch reflexes could be evoked, however, when the flexors were voluntarily activated prior to stretch.

For the eight subjects with a strong reflex response, stiffness curves were computed for each trial. On average, maximum stiffness values occurred at 77% of the angular range for the rotations of the MCP joints. The average reflex threshold angle across all subjects was attained at less than 15% of the range. Indeed reflex threshold had often been reached prior to completion of the first 10% of the angular deflection, the period contaminated by inertial effects, thereby limiting the usefulness of the threshold in quantifying spasticity in these subjects. Figure 5 illustrates this absence of change in angular reflex threshold with increasing velocity for one subject. The curves rise sharply at the beginning of the stretch due to inertial effects and then settle immediately into almost constant slopes. As slope represents stiffness, one can easily discern that stiffness threshold is reached almost immediately.

The other spasticity parameter, normalized net work $W_N$ (computed from the total recorded torque), did exhibit significant dependence on velocity. $W_N$ increased fairly linearly with respect to stretch velocity across all subjects, as verified by the average coefficient of determination of the linear regressions (mean $R^2 = 0.75 \pm 0.12$), thereby suggesting that a linear fit was adequate for purposes of comparison. The slopes of $W_N$ vs. velocity derived from the regression equations were significantly greater than those for the controls in nine of the 13 stroke subjects (Fig. 6). Furthermore, the eight highest slopes belonged to the aforementioned eight subjects with significant stretch-evoked EMG activity. The ninth subject displayed stretch reflexes only inconsistently.

**Reflexive Extensor Activity.** Curiously, activation of stretch reflexes in the MCP flexor muscles in the stroke subjects was often accompanied by an anomalous increase in extensor muscle activity (Fig. 4). In one subject, the applied stretch induced oscillations in flexor and extensor activity which produced a mild clonus that was apparent in the torque signal as well as visually. In fact, stretch of the flexors some-
times triggered visible extension of the PIP and DIP joints (Fig. 7). Four of the subjects displayed some degree of spontaneous finger extension during stretch of the finger flexors beyond what they could perform voluntarily.

**DISCUSSION**

In our study, spasticity was typically present in the extrinsic finger flexors following stroke, but not in the finger extensors. Passive mechanical resistance to stretch was not distinct from that of control subjects. Curiously, stretch of the flexors often elicited extensor activity, sometimes of a greater magnitude than the flexor response.

**Reflex Contributions to Resistance to Extension.**

The majority of the stroke subjects exhibited significant stretch reflex responses to constant-velocity rotation of the MCP joints. Reflexes were elicited without any preactivation and were asymmetrical, with imposed finger extension but not finger flexion eliciting a response; the flexor muscle reflex thresholds could be reached readily by stretch but those for the MCP extensors could not. Supporting this view, the magnitude of the net work required to stretch and release the MCP flexor muscles increased with increasing velocity of MCP joint extension, again indicating the existence of spasticity in flexor muscles.

This increase in reflex response of the long finger flexors following stroke contrasts with the reported reduction of the stretch reflex in the long thumb flexors in subjects with cortical damage in the sensorimotor region. However, a number of subjects in that study did exhibit exaggerated short-latency reflexes, thought to be associated with spinal neural circuitry. Lesion location may explain some of the disparity in response as a number of the subjects in this study had infarcts in subcortical brain structures as opposed to the predominance of lesions in sensorimotor cortex in the thumb study. Increased stretch reflex responses have been recorded at the elbow following stroke.

Interestingly, unlike the situation at the elbow, a clear and discernible angular reflex threshold was often absent at the MCP joints, in that the reflex response in the flexors began virtually at the onset of the stretch. This lack of a distinct threshold may be due, in part, to the fact that the finger flexors lie at a rather long initial fiber length. With the wrist positioned at zero degrees of flexion, the muscle fibers for FDP and FDS are reported to be close to their optimal lengths. If the muscle spindles follow suit, which is likely, then the motoneurons may be more excitable because of sustained tonic afferent excitatory input from these muscles. Spasticity induced by MCP rotation could perhaps be reduced by flexion of the wrist.

**Passive Mechanical Characteristics of Finger Rotation.**

For the majority of our stroke subjects, the static MCP joint stiffness was no greater than that of the control subjects. This lack of a significant difference in passive stiffness is similar to that observed at the elbow. In both sites, consistent physical therapeutic manipulation may have aided in maintaining passive joint range of motion. However, it differs from the increased passive stiffness recorded at the ankle. Although it is true that, in our present study, the PIP and DIP joints were allowed to flex, thereby slightly reducing forced extension of the tissues, FDS and FDP muscle fibers still were stretched to at least nine-tenths of their optimal length. Thus, resistance to MCP rotation does not result entirely from alterations in mechanical tissue properties.

Four of the 13 stroke subjects did exhibit statistically greater static passive stiffness opposing MCP joint rotation. The regression intercepts for three of these four subjects were among those with the greatest degree of flexion, suggesting possible muscle shortening shifting the neutral position, at which torque about the MCP joints was zero, toward MCP flexion. The mechanisms of this shortening in human muscles are unclear. In mice, muscle immobilization in a shortened position has been shown to lead to the reduction in the number of sarcomeres in series in the soleus muscle, resulting in an increase in the resistance to passive stretch. Immobi-
lization has also produced an increase in the passive stiffness of the soleus in rabbits.16

However, the possibility that intrinsic hand muscles actively contributed to the resistance cannot be entirely dismissed. Although EMG of the extrinsic finger muscles confirmed their passivity, EMG activity of the intrinsic muscles was not recorded. Activity in these muscles would be surprising, though, as finger abduction and adduction, the primary finger movements produced by the intrinsic muscles, are typically two of the most difficult motions for individuals to produce following stroke.

Rotation of the MCP joints at different velocities revealed that passive muscles and joint tissues displayed viscoelastic behavior. Specifically, both the magnitude of the peak torque and the area of the hysteresis loop in the torque-angle curve increased as a function of velocity, even in the absence of any reflex response, thus supporting the existence of viscous elements. The origins of this viscosity are unclear, since neither passive muscle nor tendon exhibits such behavior in isolation. Potentially, the friction between tendons and pulley mechanisms, such as the flexor retinaculum,2 or ligaments constraining tendon motion in the fingers may contribute to this velocity dependence.

**Anomalous Extensor Shortening Reflexes.** Paradoxically, stretch of the finger flexors often evoked unanticipated extensor activity. This contrasts with responses observed at the ankle and the elbow, where the antagonist remained quiet during flexor muscle stretch. However, similar patterns of reflex activation during shortening have been reported previously in a number of other limb movement or perturbation studies. For example, coexcitation has previously in a number of other limb movement or activation during shortening have been reported muscle stretch. However, similar patterns of reflex responses observed at the ankle and the elbow, unanticipated extensor activity. This contrasts with doxically, stretch of the finger flexors often evoked utue to this velocity dependence.

The finding that was especially intriguing in our study was that the stretching of the flexors actually produced spontaneous finger extension in a number of subjects, and this extension sometimes appeared even greater than that which could be generated voluntarily. Thus, finger extension could be triggered by MCP extension, but not necessarily controlled.

Preliminary studies we have conducted examining voluntary finger torque generation following stroke have also displayed a significant degree of co-contraction between flexors and extensors during attempts at both voluntary flexion and extension. Reciprocal inhibition between the flexors and extensors, found to play a significant role in the control of forearm muscles,9,12 has been shown to be compromised following stroke.7

The requirements for proper coordination of flexor and extensor motoneurons for the finger muscles are assuredly even greater than those for the forearm due to the complex firing patterns required for finger manipulation. Disruption of this coordination may be one of the primary causes of hand deformity following stroke. This area warrants further examination in future studies.

The authors thank Dr. Brian Schmitt for his help in preparing this manuscript. The work was supported by NIH grant T32 No. I F32 NS10679 (D.K.) and NS19931 (W.Z.R.).

**REFERENCES**

16. Herbert RD, Balhove RJ. The effect of position of immobili-


