ABSTRACT: We sought to establish whether spastic hypertonia results from changes in intrinsic muscle properties or from altered stretch reflex properties. We hypothesized that finger flexor spastic hypertonia is primarily of neural origin, and that the dynamics of spastic muscle responses to stretch should therefore reflect the dynamics of muscle spindle receptor responses. In 12 stroke survivors, we recorded torque and electromyographic (EMG) responses of extrinsic finger flexors to constant-velocity rotation of the metacarpophalangeal (MCP) joints of the affected hand, over a range of initial muscle lengths. Stretch velocity was set to 6°, 50°, 150°, or 300° per second. Muscle length changes were imposed by changing wrist angle between 0°, 25°, and 50° of flexion. We found that reflex torque and EMG responses exhibited both velocity and length dependence, and there were significant interactions between velocity and length, replicating known characteristics of muscle spindle receptors. Our results support the hypothesis that finger flexor hypertonia is primarily of neural origin, and that it accurately reflects spindle receptor firing properties.

EFFECTS OF CHANGING WRIST POSITIONS ON FINGER FLEXOR HYPERTONIA IN STROKE SURVIVORS

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Chronic deficits after stroke are especially prevalent in the hand and fingers. A stereotypical pattern of hand impairment often results after stroke, including flexed resting postures of the wrist and fingers, muscle atrophy and contractures, muscle weakness, and an inability to voluntarily extend the fingers. The impairment could be of neural origin, originating in spastic hypertonia,11,12 or a result of changes in intrinsic properties of spastic muscles.5,23 In addition, these two potential sources could work synergistically to increase overall impairment.22

Velocity-dependent spastic hypertonia14 may itself result from changes in intrinsic muscle properties or from altered neuronal reflex properties.12 After assessing the relative contributions of neural and muscle mechanics using ulnar and median nerve blockade at the elbow, Kamper et al.11 found that finger flexor hypertonia was predominantly of neural origin. The altered reflex properties associated with spastic hypertonia may be due to increased motoneuronal excitability, increased stretch-evoked synaptic excitation to motoneurons, or altered supraspinal inputs. These neural mechanisms could, in turn, induce decreases in reflex threshold, which increase motoneuronal excitability, or increases in reflex gain (amplification of stretch-evoked excitation).12

A series of experiments performed on spastic elbow muscles24–26 provided evidence that the exaggerated stretch reflexes in spastic hypertonia result primarily from a decrease in reflex threshold; the motoneurons seem to reside in a sustained state of supraspinally mediated depolarization.12 In support of this view, a preliminary study10 showed that the absolute fiber length has a profound effect on the stretch reflex in terms of both torque and electromyographic (EMG) responses in stroke survivors, even after accounting for length-dependent changes in biomechanical parameters. By altering the wrist angle, and thereby changing the fiber length of the extrinsic finger flexor muscles, the responses to imposed
rotation at the metacarpophalangeal (MCP) joints were drastically altered.

The effect of change of the joint angle, and thus absolute muscle length, on the exaggerated reflex response has been reported in recent studies.\(^{18,20,27,28}\) This effect has been thought to be mediated by heteronymous group II pathways in subjects with spastic hemiplegia.\(^{18}\) Selective inhibition of this interneuron-mediated group II afferent pathway could reduce spasticity and bring it close to a normal level.\(^{27}\)

This positional effect in the upper-limb muscles is rarely addressed, however, especially in stroke survivors. In the study cited previously,\(^{10}\) only one velocity level was tested while the length of finger flexor was systematically changed. Accordingly, we planned a more extensive investigation, including systematic changes in both stretch velocity and muscle length. We hypothesized that the dynamics of spastic muscle activation reflects the properties of muscle stretch receptors, in that there would be clear effects of changes in absolute muscle length and velocity, analogous to those elicited in muscle spindle receptors.\(^{7}\)

**METHODS**

**Subjects.** Twelve (9 men, 3 women, ranging from 40 to 74 years old) subjects with hemiparesis subsequent to stroke that had occurred between 2 and 22 years earlier (mean ± SD: 7.8 ± 5.4 years) participated in the experiment. The stroke affected the premorbid dominant hand in 7 instances (5 right, 2 left); in the other 5 patients, the nondominant left hand was affected. MCP muscle tone was rated as 2 in 5 cases and 3 in the 7 other subjects according to the Ashworth scale.\(^{1}\) Subjects were screened and referred by a research therapist according to the following inclusion/exclusion criteria.

Inclusion criteria included: (1) a history of stroke at least 1 year earlier (at this stage, recovery of hand function has stabilized)\(^{19}\); (2) an acceptable range of motion at the wrist joint, such that the impairment level, rated by a research therapist, was at stage 3 of the hand impairment inventory on the Chedoke-McMaster Stroke Assessment Scale, which yields reliable and valid results and a strong correlation with the Fugl-Meyer test.\(^{6}\) Exclusion criteria were: (1) previous history of stroke or radiological evidence of multiple vascular lesions; (2) major muscle contracture limiting the range of motion in the impaired hand and fingers; (3) hemispatial neglect, apraxia, or major sensory or proprioceptive loss, extensive cardiovascular disease, or visual deficits; (4) significant cognitive impairment precluding the ability to provide informed consent; or (5) any other severe concurrent medical problems.

All subjects gave informed consent according to the Helsinki Declaration, and the institutional review board of Northwestern University approved the experimental protocol.

**Procedures.** The experimental setting (Fig. 1) was similar to those used previously in our laboratory.\(^{9,10}\) The seated subject was positioned in an adjustable chair such that the shoulder was abducted approximately 50° and the elbow flexed at around 90°. The forearm was placed in a fiberglass cast and secured in the neutral position to a clamp attached to the table. This arrangement prevented translation and rotation of the arm. A strap kept the thumb extended and abducted from the palm so it did not interfere with finger flexion. A fiberglass cast secured the forearm without involvement of the wrist. The wrist was stabilized by a universal U-piece positioned just distal to the wrist (see Fig. 1). Wrist angle was controlled by the orientation of this wrist clamp, which had a magnetic base that afforded easy manipulation.

The MCP joints were aligned along a vertical line extending from the shaft of a perturbing motor. The four fingers, kept straight by attaching them to a small plastic plate, were coupled directly to the servomotor (1.4 hp; PMI Motion Technologies, Radford, Virginia) such that rotation of the shaft pro-
duced an equivalent angular rotation of the MCP joints. Although the axes of the different MCP joints may not have been perfectly aligned, we were confident that the effects of misalignment were small in relation to the torques of interest. Furthermore, any misalignment would result in pushing or pulling forces at the interface of the phalanges and device, and would not contribute to the torque measured about the axis of the servo shaft. The motor shaft extended vertically above a table to which the forearm was clamped. An aluminum channel was connected to the shaft and to a second 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 U-piece and, thus, the motor shaft.

The muscle length of the extrinsic finger flexors is influenced by the position of the wrist, MCP, distal interphalangeal (DIP), and proximal interphalangeal (PIP) joints. Since the DIP and PIP joints were kept in the neutral extended positions, changes in muscle length resulted only from changes in wrist and MCP angles. The wrist angle was monitored and measured with a plastic goniometer. Potential variation in measuring the wrist angle across subjects was negligible in relation to the size of the angular displacements between the three wrist positions. The chosen wrist postures, maintained during each set of constant-velocity MCP rotations, were: WR0 (neutral position); WR25 (25° of wrist flexion); and WR50 (50° of wrist flexion). For each trial, a ramp–hold–ramp stretch was imposed on the MCP joints (Fig. 2). The MCP joints were passively rotated from 30° of MCP flexion to the neutral position during the first ramp and rotated back to the initial position after a 2-second pause. By changing the wrist angle, the range of absolute muscle fiber lengths produced by the given MCP rotation was varied. The sequence of the wrist positions was randomized for each subject.

Four different constant velocities, 6°/s, 50°/s, 150°/s, and 300°/s, were imposed on the MCP joints at each wrist position. Five trials were performed for each velocity, with the velocity for a given trial randomly selected from the specified set.

Only the affected side was tested. Reflex responses are not routinely elicited from resting finger flexors in normal subjects using the current protocol. Although reflex responses can be evoked in voluntarily preactivated muscles of the contralateral limb in hemiparetic stroke patients, maintaining the same degree of voluntary preactivation across different wrist angles and different stretch velocities presents a formidable challenge. This is because maintenance of constant voluntary muscle activation cannot be monitored, as recorded torque and EMG signals reflect the combination of both voluntary and reflex activity.

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, Temple, California), respectively. Surface EMG recordings were made to ensure that reflex activity occurred. Differential surface electrodes (DelSys, Boston, Massachusetts) were positioned above the flexor digitorum superficialis (FDS), the extensor digitorum communis (EDC), and the first dorsal interosseous (FDI). We felt confident in using surface electrodes to indicate reflex activity of these muscles, as in previous studies we observed little evidence of motion artifact (only body segments distal to the electrodes are moving) or cross-talk between electrodes.

Data Analysis. For all experiments, angular position and velocity, torque, and EMG data were low-pass filtered at 225 Hz (fourth-order Butterworth filters) and sampled at 500 Hz. Flexor spasticity was quantified from the data recorded during imposed extension rotation of the MCP joints. Peak torque, referenced to the initial resting torque, was found for the period between the start and end of rotation. The resting torque (TRQref) was estimated as the mean value over the first 100 ms of a trial. Torque measured at 6°/s was taken to be the passive, mechanical resistance to the imposed MCP rotation. After subtracting this passive torque, recorded at each wrist position, the residual torque was equated with the velocity-dependent reflex torque, attributable to flexor spasticity. The residual torque does contain a passive component, but it is small and negligible in size. The peak reflex torque (TRQref) was determined for each trial. TRQref was normalized as a percentage of the maximum peak torque, chosen from a trial with the maximum torque during 300°/s stretch at WR0 for each subject.

Only EMG signals from the FDS were used to quantify reflex responses. Sampled EMG signals were rectified and the resulting signals were low-pass filtered forward and backward with a 30th-order FIR filter at 10 Hz to obtain envelopes of muscle activity. EMG envelopes were first examined to verify that a reflex response had occurred.
The criterion for a reflex EMG burst was the signal magnitude surpassing a threshold equal to three times the standard deviation of the baseline. The baseline EMG (EMGBG) was estimated from the first 200 ms of the trial, prior to initiation of the stretch, and the reflex EMG activity was estimated from a 100-ms window centered on the peak EMG for the period between the start and the end of rotation. Analogous to the torque analysis, the EMG value was normalized to the maximal EMG activity, which was recorded during the trial and demonstrated the maximum torque for each subject.

**Statistics.** For the dependent variables of interest (torque and EMG), a doubly multivariate analysis of variance (MANOVA) was first performed to determine whether the independent variables of wrist position and velocity impacted the output (total torque and EMG values). In cases where the Wilk’s lambda value showed significance, separate univariate analyses of variance (ANOVAs) were then performed to assess the impact of velocity and position on reflex responses (reflex torque and EMG values). The within-subject dependent variables were position (three levels) and velocity (three levels). ANOVAs were also performed to examine the effect of changing wrist position (three levels) on background EMG and passive torque. Tukey’s post hoc tests were utilized whenever necessary. The level of significance was set at $P < 0.05$.

**Figure 2.** Example of velocity dependence of the spastic stretch reflex at a specified wrist position. Total torque and EMG responses are plotted from a set of single trials from one subject during external rotations imposed on the metacarpophalangeal (MCP) in the neutral position. EMG signals were from the flexor digitorum superficialis. A “ramp–hold–ramp” stretch protocol was used for all trials. Note that both torque and EMG responses are velocity dependent. Negative torque and position indicate extension.
RESULTS

No significant length dependence of resting EMG activity was found. When averaged across all subjects, mean rectified background EMG was 0.013 V (±0.008 V), 0.013 V (±0.007 V), and 0.013 V (±0.007 V) for WR0, WR25, and WR50, respectively. Background torque at each wrist position was also not significantly different with regard to changes of initial muscle length. TRQBG was 0.19 Nm for WR0, 0.16 Nm for WR25, and 0.19 Nm for WR50.

Based on the lack of significant EMG response to muscle stretch, the torque responses derived from 6°/s stretches were believed to be essentially passive torques. Peak passive torque was 0.32 Nm for WR0, 0.26 Nm for WR25, and 0.28 Nm for WR50.

Reflex responses, by contrast, exhibited a profound dependence on absolute muscle length and the rate of stretch. MANOVA revealed that both muscle length (Wilk’s lambda[4,8] = 0.193, P = 0.006) and stretch velocity (Wilk’s lambda[6,6] = 0.170, P = 0.037) had significant effects on torque and reflex EMG activities. For a given wrist position (e.g., WR0 in Fig. 2), both torque response and reflex EMG activity showed velocity dependence. Varying muscle length by changing wrist position also induced systematic changes in joint torque and EMG responses to stretch, but did not alter background muscle activity. Torque responses at a given stretch velocity (e.g., at 300°/s in Figs. 3 and 4) also demonstrated position-dependent behavior.

According to the univariate ANOVA, the TRQREF showed dependence on stretch velocity, initial wrist position, and their interactions (Fig. 5). TRQREF increased with stretch velocity ($F_{(2,22)} = 40.20$, $P < 0.001$). It was significantly larger at 300°/s (22.3%) than at 150°/s (15.0%). Both were significantly larger than that at 50°/s (7.7%). TRQREF was also position-dependent ($F_{(2,22)} = 21.87$, $P < 0.001$). It was significantly larger at WR0 (23.9%) than at WR25 (13.0%) and WR50 (8.1%). The latter two were not significantly different.

Post hoc tests for a significant interaction of position $\times$ velocity ($F_{(4,44)} = 3.38$, $P = 0.017$) revealed that a slower stretch at a wrist position with longer flexor muscle lengths generated a larger reflexive torque than a faster stretch imposed at a more flexed wrist position with shorter muscle lengths. For example, TRQREF generated at WR0 by 150°/s stretch (23.8%) was significantly larger than that at WR50 with 300°/s (11.3%; $P < 0.002$) (see Fig. 5).

The velocity–position interaction effect on reflex torque was accompanied by parallel changes in reflex EMG activities (Fig. 5). To compare across subjects, the normalized EMG with respect to the EMG response from the same trial with the maximum torque was used. According to a 3 $\times$ 3 two-way ANOVA (position $\times$ velocity), the normalized EMG showed a pattern similar to that in TRQREF; that is, a velocity-dependent ($F_{(2,22)} = 71.53$, $P < 0.001$), position-dependent ($F_{(2,22)} = 12.34$, $P < 0.001$), and significant interaction ($F_{(4,44)} = 4.99$, $P = 0.002$).
DISCUSSION

The primary finding was that finger flexor hypertonia after stroke demonstrated velocity dependence, length dependence (postural effect) and a significant interaction between velocity and length dependence. Specifically, velocity dependence was more evident at a longer muscle length, whereas length dependence was significant during faster stretches. These results confirmed and expanded previous results describing the effects of changing muscle length on finger flexor muscle hypertonia after stroke.

These findings mirror the established dependence of spindle dynamic responses on absolute muscle length and velocity. During externally imposed stretch, afferent input to motoneurons includes excitation from velocity-sensitive primary spindle afferent inputs (group Ia) from monosynaptic and disynaptic secondary endings (group II) sensitive to the actual muscle length, and polysynaptic inputs from cutaneous mechanoreceptors, joint afferents, and tendon afferents. In the decerebrate cat soleus muscle, Houk et al. demonstrated that the dynamic response from spindle afferent discharge of both groups (Ia and II) (Fig. 6), showing the position and velocity sensitivity of the afferent firing, and the interaction between the two (see also Lennerstrand and Thoden). Based on these findings, a model of spindle firing was proposed in which the overall spindle discharge was related to muscle length, but only proportional to a fractional power

![Figure 4](image_url)

**FIGURE 4.** Velocity dependence and position dependence of total torque responses. The torque–angle relations are plotted from a set of single trials from one subject. The torque measured at the neutral wrist position (WR0) increases with stretch velocity (A). In (B), the torque increases when the wrist joint extends (length increases). Positive angles on the x-axis indicate metacarpophalangeal (MCP) joint flexion. WR0, WR25, and WR50: wrist in neutral, 25°, and 50° flexion, respectively.

![Figure 5](image_url)

**FIGURE 5.** Interactions of velocity and length dependence of reflex responses. Averaged across trials and subjects, the normalized reflex torque is both velocity and position dependent, and a significant interaction occurs between velocity and position (A). Reflex torques are accompanied by parallel changes in EMG activities (B). Standard errors are shown by error bars.
of the velocity of lengthening. Therefore, the combined effect of spindle discharge resulting from an external stretch could be achieved by a variety of combinations of velocity and absolute muscle length, thus giving rise to a significant interaction between velocity and length. Recently, position sensitivity of muscle spindles was further confirmed in human wrist and finger extensors and in lower-extremity muscles in patients with various neurological disorders. The present study has demonstrated dependence of finger flexor spasticity after stroke on muscle length and velocity similar to spindle dynamic responses seen in cat muscles.

According to the findings from a recent study showing that varying muscle length could change reflex threshold for spastic finger flexors, one plausible explanation is that the observed pattern of reflex responses most likely resulted from a reduced reflex threshold, without overt change in motoneuron properties. Theoretically, with enhanced motoneuronal excitability, a small amount of additional synaptic (sensory) input is sufficient to surpass the reflex threshold in the spastic muscles, thereby generating exaggerated stretch reflexes in these muscles. It follows that the attainment of a spindle discharge rate sufficient to trigger the spastic reflex responses could be achieved by a variety of combinations of velocity (rate of stretch) and position (absolute muscle length) based on the known dynamic responses of muscle spindles, resulting in the significant positional effect on the reflex response and its interaction with velocity dependence in finger flexor hypertonia observed in the present study. Alternatively, increased spindle gain may be present following stroke. The combined effects of length and velocity on spindle firing would thereby be amplified, resulting in motoneuronal firing and the observed results.

The present results demonstrate that the spastic stretch reflex varies in a manner akin to anticipated neuronal sensory input. This association, in conjunction with the lack of any effect of wrist angle on passive MCP joint torque, emphasizes the importance of the neural contribution to spasticity and perceived tone.

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REFERENCES