Lack of Hypertonia in Thumb Muscles After Stroke

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Towles JD, Kamper DG, Rymer WZ. Lack of hypertonia in thumb muscles after stroke. J Neurophysiol 104: 2139–2146, 2010. First published June 28, 2010; doi:10.1152/jn.00423.2009. Despite the importance of the thumb to hand function, little is known about the origins of thumb impairment poststroke. Accordingly, the primary purpose of this study was to assess whether thumb flexors have heightened stretch reflexes (SRs) following stroke-induced hand impairment. The secondary purpose was to compare SR characteristics of thumb flexors in relation to those of finger flexors since it is unclear whether SR properties of both muscle groups are similarly affected poststroke. Stretch reflexes in thumb and finger flexors were assessed at rest on the paretic side in each of 12 individuals with chronic, severe, stroke-induced hand impairment and in the dominant thumb in each of eight control subjects also at rest. Muscle activity and passive joint flexion torques were measured during imposed slow (SS) and fast stretches (FS) of the flexors that span the metacarpophalangeal joints. Putative spasticity was then quantified in terms of the peak difference between FS and SS joint torques and electromyographic changes. For both the hemiparetic and control groups, the mean normalized peak torque differences (PTDs) measured in thumb flexors were statistically indistinguishable (P = 0.57). In both groups, flexor muscles were primarily unresponsive to rapid stretching. For 10 of 12 hemiparetic subjects, PTDs in thumb flexors were less than those in finger flexors (P = 0.03). Paretic finger flexor muscle reflex activity was consistently elicited during rapid stretching. These results may reflect an important difference between thumb and finger flexors relating to properties of the involved muscle afferents and spinal motoneurons.

INTRODUCTION

The stroke-impaired hand is typically characterized by a diminished ability to extend and abduct/adduct the digits from their flexed resting postures (Woodson 1995). Consequently, stroke survivors will often employ compensatory grasp strategies when using the impaired hand to interact with the environment. Such strategies largely exploit the passive joint range of motion of paretic finger and thumb joints and the closed posture of the paretic hand to trap an object between the fingers, between the fingers and palm, or between the fingers and thumb. Occasionally effective, such compensatory strategies are ill-suited for accomplishing a variety of tasks that are important to daily life requiring controlled movement and force production between the thumb and fingers.

Despite the benefits of current rehabilitation approaches to hand function following a cerebrovascular accident (CVA) (Kraft et al. 1992; Suputtithada et al. 2004), rehabilitation outcomes can likely be improved. The potential for improvement exists in part because the origin of hand impairment following stroke is not fully understood, at least from the standpoint of the involvement of the spinal cord. Little is known whether alterations in spinal circuitry serve, in part, as a basis for stroke-induced thumb impairment. Given that the thumb plays critical roles in grasping and manipulating objects, this omission could have a significant impact on our understanding of hand impairment and the design of increasingly effective rehabilitation strategies for the stroke population.

There have been few quantitative studies of the thumb after stroke (Lang and Schieber 2003; Raghavan et al. 2006) and limited investigations of the reflexes of the thumb (Marsden et al. 1973, 1976, 1977a,b). The stretch reflex properties of most thumb flexors have not been characterized. Enhanced stretch reflexes (SRs), an indicator of muscle hypertonia (Lance 1980) and a common secondary occurrence in stroke (O’Dwyer et al. 1996), could contribute to thumb impairment. Although abnormal resistance to passive stretch of thumb flexor muscles (both intrinsic and extrinsic) has been observed clinically (Tafti 2008; Waters 1978), we were unaware of any studies that objectively and quantitatively determined whether this resistance is neurally mediated, i.e., due to hypertonia as defined by Lance (1980).

Thus we sought to evaluate SR responses in thumb flexor muscles of persons with chronic, severe hand impairment. We also explored whether SR responses in the thumb and fingers were similarly altered (e.g., elevated) in the same hand following stroke. There is sufficient interdigit neuroanatomical difference (Schieber 1999; Volkmann et al. 1998) to preclude simply assuming that the paretic thumb and fingers behave similarly. We hypothesized, however, that SR responses of the flexor muscles of both the thumb and fingers would be abnormally elevated following hemiparetic stroke, given that the resting postures of the fingers and thumb are typically abnormally hyperflexed.

Our results unexpectedly show that SR responses of the paretic thumb flexors, in contrast to the flexors of the paretic finger, were not consistently elevated and thus metacarpophalangeal (MP) joint flexion torques and muscle activity, following rapid stretching, more closely resembled findings in the control group. These results may reflect an important difference between finger and thumb flexors in relation to the properties of the involved muscle afferents and spinal motoneurons.

METHODS

Subjects

Twelve stroke survivors (subjects 1–12 in Table 1) with chronic and severe hand impairment, as evaluated by the Chedoke–McMaster
TABLE 1. Subject characteristics

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age, yr</th>
<th>Gender</th>
<th>Paretic Side (1–12)</th>
<th>Elapsed Time Since Stroke, mo</th>
<th>CMSA Score (normal = 7)</th>
<th>Maximum Thumb Flexion Torque, Nm</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>64</td>
<td>M</td>
<td>L</td>
<td>85</td>
<td>3</td>
<td>0.48</td>
</tr>
<tr>
<td>2</td>
<td>85</td>
<td>F</td>
<td>R</td>
<td>64</td>
<td>4</td>
<td>1.33</td>
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<tr>
<td>3</td>
<td>50</td>
<td>F</td>
<td>L</td>
<td>29</td>
<td>2</td>
<td>0.94</td>
</tr>
<tr>
<td>4</td>
<td>32</td>
<td>M</td>
<td>R</td>
<td>32</td>
<td>3</td>
<td>1.20</td>
</tr>
<tr>
<td>5</td>
<td>49</td>
<td>M</td>
<td>L</td>
<td>38</td>
<td>2</td>
<td>0.66</td>
</tr>
<tr>
<td>6</td>
<td>87</td>
<td>M</td>
<td>L</td>
<td>17</td>
<td>2</td>
<td>0.16</td>
</tr>
<tr>
<td>7</td>
<td>57</td>
<td>F</td>
<td>L</td>
<td>27</td>
<td>2</td>
<td>0.31</td>
</tr>
<tr>
<td>8</td>
<td>56</td>
<td>F</td>
<td>R</td>
<td>126</td>
<td>2</td>
<td>0.33</td>
</tr>
<tr>
<td>9</td>
<td>59</td>
<td>M</td>
<td>R</td>
<td>24</td>
<td>3</td>
<td>0.11</td>
</tr>
<tr>
<td>10</td>
<td>60</td>
<td>M</td>
<td>L</td>
<td>59</td>
<td>3</td>
<td>0.25</td>
</tr>
<tr>
<td>11</td>
<td>64</td>
<td>M</td>
<td>L</td>
<td>132</td>
<td>2</td>
<td>0.24</td>
</tr>
<tr>
<td>12</td>
<td>68</td>
<td>F</td>
<td>R</td>
<td>228</td>
<td>3</td>
<td>0.29</td>
</tr>
<tr>
<td>Mean (range)</td>
<td>61 (32–87)</td>
<td></td>
<td></td>
<td>72 (17–228)</td>
<td>0.52 (0.11–1.33)</td>
<td></td>
</tr>
</tbody>
</table>

Chedoke–McMaster Stroke Assessment (CMSA) test was used to evaluate each stroke subject’s level of hand impairment on the most affected side using an ordinal scale ranging from 1 (most impaired) to 7 (normal). Note that CMSA 2–3 is severe impairment, CMSA 4–5 is moderate impairment. Subjects 1–12 were hemiparetic; subjects 13–20 were control.

Stroke Assessment (CMSA) test (Gowland et al. 1993), and eight control individuals with no evidence of neurologic dysfunction affecting the dominant hand (subjects 13–20 in Table 1) participated in the study. All subjects were recruited from the Rehabilitation Institute of Chicago and/or the Chicago area. All subjects gave informed consent to permit testing of either their most affected (stroke) or dominant (control) side according to the Helsinki Declaration and the Institutional Review Board of Northwestern University approved the experimental protocol.

Protocol

The following experimental approach, adapted from a previous study (Kamper and Rymer 2000), was designed to examine SR responses in the thumb and finger flexor muscles that cross the MP joints. The MP joint was chosen because it is experimentally tractable and, taken together, the MP joints are spanned by nearly all of the hand muscles (Table 2).

At the start of the experiment, each stroke subject’s level of hand impairment on the paretic side was evaluated by a therapist using the Stage of Hand Section of the CMSA (Gowland et al. 1993). Each subject was seated with the shoulder abducted nearly 30°, the elbow flexed about 90°, the forearm fully pronated, and the wrist slightly extended (~20°), where possible. The thumb was coupled to the shaft of a servo-motor (1.4 HP; PMI Motion Technologies, Kollmorgen, Radford, VA), such that the MP joint was positioned over the motor’s rotation axis and the trapeziometacarpal (TMC) joint was extended about 30° and neutrally abducted (Fig. 1A). Casting material and a fixation device were used to maintain the postures of the forearm, wrist, and the TMC joint. A splint was used to stabilize the interphalangeal (IP) joint in neutral flexion. The MP joint range of motion was acquired for each subject. While the subject was at rest, the motor rotated the MP joint throughout the middle 75% of its joint range using a trapezoidal angular velocity profile to stretch the flexor muscles that cross the joint. The velocity profile reached a plateau at either a slow (10 deg·s⁻¹) or a fast (290 deg·s⁻¹) speed and maintained this speed over the middle 80% of the joint range traversed. These speeds represented conditions under which a stretch reflex response would be likely (290 deg·s⁻¹) or unlikely (10 deg·s⁻¹) to occur in the hands of individuals with poststroke hemiparesis (Kamper and Rymer 2000).

For each trial, the motor rotated the thumb MP joint from a flexed posture to an extended posture at either the slow or fast speed, held the joint in the extended posture for 2 s, and then returned the joint to the starting flexed posture (i.e., a ramp-and-hold perturbation paradigm). The joint angle difference between the flexed and extended postures represented 75% of the MP joint range. Three trials were randomly performed at each speed. In 9 of 12 hemiparetic and 3 control subjects, muscle activity was recorded from the flexor pollicis longus (FPL), the intrinsic thumb muscles (INT: adductor pollicis [ADP], ulnar/radial head of flexor pollicis brevis [FPBu/r], abductor pollicis brevis [APB]), extensor pollicis longus (EPL), and extensor pollicis brevis (EIP).

TABLE 2. Muscles that cross the metacarpophalangeal (MP) joints

<table>
<thead>
<tr>
<th>Flexors</th>
<th>Thumb</th>
<th>Extensors</th>
<th>Flexors</th>
<th>Fingers</th>
</tr>
</thead>
<tbody>
<tr>
<td>FPL (2.08)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>INT</td>
<td>ADP + FPBu (1.94)</td>
<td>EPL</td>
<td>DI (4.7)</td>
<td>EDC</td>
</tr>
<tr>
<td></td>
<td>APB (0.68)</td>
<td></td>
<td>FDP (7.92)</td>
<td>EIP</td>
</tr>
<tr>
<td></td>
<td>FPBr (0.66)</td>
<td></td>
<td>FDS (6.25)</td>
<td>LUM (0.33)</td>
</tr>
</tbody>
</table>

FPL, flexor pollicis longus; INT, intrinsic thumb muscles [ADP, adductor pollicis; APB, abductor pollicis brevis; FPBr, flexor pollicis brevis (radial head); FPBu, flexor pollicis brevis (ulnar head)]; EPL, extensor pollicis brevis; EPL, extensor pollicis longus; DI, dorsal interosseus; FDP, flexor digitorum profundus; FDS, flexor digitorum superficialis; LUM, lumbrical; PI, palmar interosseus; EDC, extensor digitorum communis; EIP, extensor indicis proprius. All listed muscles potentially contribute to MP joint torques, but only activity in the underlined muscles or muscle group was recorded. Activity was recorded in just the first dorsal interosseus (DI). For this study, DI is considered an intrinsic muscle of the fingers, even though it also inserts in the thumb. DI does not cross the MP joint of the thumb and thus is not stretched when the thumb’s MP joint is rotated. Physiologic cross-sectional area values (cm²) are listed in parentheses.
A cast-clamp system was used to fix the position of the forearm, wrist, and trapeziometacarpal joint (thumb’s base joint). The interphalangeal (IP) joint (end joint) was stabilized in neutral with a splint. The thumb was attached to a metal frame that, in turn, was attached to the shaft of a motor. The motor extended and flexed the metacarpophalangeal (MP) joint (middle joint) as desired.

Following the muscle stretch trials, subjects performed three trials of maximal voluntary contractions (MVCs) in the extension and flexion directions while the MP joint was in the neutral posture. During data collection, joint torque, angular position, and angular velocity were measured, in addition to muscle activity, with a torque transducer (TRT-200; Transducer Techniques, Temecula, CA), a position encoder (#138647; PMI Motion Technologies), and a tachometer (PMI Motion Technologies), respectively, connected to the motor (Fig. 1).

Measured quantities were low-pass filtered at 225 Hz (Delsys) and sampled at 500 Hz. Data were collected using LabVIEW (National Instruments, Austin, TX).

A similar protocol was followed in the assessment of the SRs in the paretic finger flexor muscles. Activity in the flexor digitorum superficialis (FDS), extensor digitorum communis (EDC), and the first dorsal interosseus (DI) muscles (Table 2) was measured with surface electrodes and all four fingers were simultaneously extended and flexed at the MP joints (Fig. 1).

Surface electrodes were sufficient to record reflex activity in initially inactive, stretched muscles without signal contamination from nontargeted muscles. Only stretched (or shortened) muscles had the potential to be activated. During voluntary muscle activation, however, surface recordings of the deep (FPL, EPL) and small (EPB) thumb muscles were most susceptible to signal contamination by the activity of other muscles.

**Data analysis**

Data were analyzed using MATLAB (The MathWorks, Natick, MA) and SPSS (Chicago, IL). The three trials collected for each stretch condition (i.e., each muscle–stretch and speed–digit combination) for each subject were averaged together. Muscle activity data were rectified and digitally low-pass filtered at 10 Hz (second-order Butterworth filter).

Reflex activity in initially inactive muscle was characterized by a burst in electromyographic (EMG) activity that exceeded the mean baseline activity by 5%. The elapsed time (ET) from the initiation of the stretch to the start of the reflex was quantified for each initial reflex response. If the response occurred 150 ms or later (Day et al. 1983, 1989; Moses et al. 2007) following stretch initiation, the response was considered a possible mix of reflex and voluntary activity. Mixed responses, each representing an upper bound on an actual reflex response, and nonmixed responses were pooled together. Elapsed times were compared across finger muscles and across thumb muscles separately (one-way ANOVA, two-tail, $\alpha = 0.05$).

To facilitate comparison of reflex activity (i.e., mixed, nonmixed, and those indistinguishable from background muscle recordings) between the thumb and fingers, peak differences (PDs) between fast-stretch (FS) and slow-stretch (SS) joint torques were computed for each subject and normalized by an aggregate physiologic cross-sectional area (PCSA) index for either the thumb or the fingers. The PCSA index, which enabled comparisons between the thumb and four fingers, was defined as the sum of the PCSAs of the flexor muscles being stretched (Jacobson et al. 1992; Lieber et al. 1992) (Table 2). Normalized peak torque differences (PTDs) associated with stretches of the paretic thumb muscles were compared with those associated with stretches of the paretic finger muscles (Student’s $t$-test, pairwise, two-tail, $\alpha = 0.05$) and those associated with stretches of the nonparetic thumb muscles (Student’s $t$-test, two-tail, $\alpha = 0.05$). Previous studies (Kamper and Rymer 2000; Powers et al. 1988) have used PTDs to quantify spastic responses in muscle.

For a given muscle in which activity was recorded, integrated electromyographic activity (IEMG), computed over the constant velocity plateau and with respect to mean baseline EMG, was quantified. Integrated EMG for a given muscle was normalized by a maximal contraction (MC) measurement—i.e., MVC or peak reflex EMG—and the time window over which the integration took place. Finally, maximum MP flexion torques produced by the thumb were compared between groups (Student’s $t$-test, two-tail, unequal variance assumption, $\alpha = 0.05$).

**RESULTS**

**Reflex activity**

**Magnitude.** We recorded joint torques and muscle activity elicited by sequential interleaved slow and rapid stretches of thumb and finger flexor muscles in hemiparetic and control subjects (e.g., Fig. 2). For the 12 subjects in the hemiparetic group, the mean joint torque responses in thumb flexors were comparable to those observed in the control group. Mean PTDs and associated SDs [i.e., mean (SD)] for both groups [hemi-
paretic: $0.0142$ (0.0061) Nm·cm$^2$/H11002 control: $0.0150$ (0.0060) Nm·cm$^2$/H11002 were statistically indistinguishable ($P = 0.57$) from one another. Peak differences for both groups ranged from $0.0017$ Nm·cm$^2$ (stroke, subject 9) to $0.0205$ Nm·cm$^2$ (stroke, subject 7) and from $0.0069$ Nm·cm$^2$ (control, subject 20) to $0.0244$ Nm·cm$^2$ (control, subject 16), respectively (Fig. 3). During rapid stretches of the paretic thumb flexors, reflex activity was elicited in INT in 4 of 12 subjects [5.2 (2.2)% of MVC] and in EPL in 1 of 12 subjects (6.7% of MVC). In the control group, reflex activity was elicited in INT in 1 of 8 subjects (0.1% MVC). Reflex responses for FPL and EPB, in either group, were indistinguishable from background muscle activity in resting subjects. Overall, reflex activity of the intrinsic thumb flexors (INT: APB, FPBr, FPBu, ADP) could not be consistently elicited during fast stretches (e.g., Fig. 2B).

For 10 of 12 subjects in the hemiparetic group, normalized PTDs in thumb flexors were less than those in the finger flexors ($P = 0.03$; Fig. 4). Specifically, thumb flexor muscle PTDs were $0.32$ (0.44) Nm·cm$^{-2}$ less. In general, reflex activity occurred inconsistently in paretic thumb flexor muscles as described earlier, but consistently in the paretic finger flexors. In 10 of 12 hemiparetic subjects, finger flexor muscle activity was elicited most frequently in FDS (9 subjects), second most frequently in DI (8 subjects), and sometimes in EDC (6 subjects). In FDS, the mean magnitude of the (initial) reflex response was $25$ (30)% of MVC; in DI, $10$ (7)% of MVC; and in EDC, $14$ (16)% of MVC. In numerous instances, there were multiple responses and, in some cases, responses that continued until the fingers were returned to their starting position (Fig. 2A). In general, this was not the case in the thumb.

**Voluntary activity**

Finally, the hemiparetic group was generally weaker in the thumb than the control group (Table 1). The hemiparetic group produced maximal thumb flexion torques at the MP joint that
ranged between 0.11 and 1.33 Nm (mean: 0.52 Nm) compared with a much larger range and greater mean (P/H11005 0.006) for the control group, 2.77 to 12.24 Nm (mean: 5.62 Nm).

**DISCUSSION**

The goal of this study was to assess SR responses in paretic thumb and finger flexor muscles with a view toward identifying a potential source of thumb impairment and toward determining whether SRs are similarly altered in the thumb and fingers after hemiparetic stroke. Contrary to what was hypothesized, the SR responses of the paretic thumb flexors, in contrast to the flexors of the paretic finger, were not consistently elevated and therefore normalized PTDs (P/H11005 0.57) and muscle activity more closely resembled findings in the control group. Changes in thumb flexor muscle activity due to rapid muscle stretching were largely absent in the control group, a finding consistent with previous studies of healthy finger flexors (Kamper and Rymer 2000). Normalized PTDs in the paretic thumb flexors were statistically less (P = 0.03) than those measured in the paretic finger flexors. That result likely suggests that overall flexor muscle activity was less in the paretic thumb than in the paretic fingers, assuming similar passive properties between the fingers and thumb.

**Reflex activity in thumb muscles**

Responses in thumb muscles were modest, both in terms of magnitude and frequency of occurrence of the activity. Muscle responses were generally substantially smaller than flexor muscle MVC activity (<7% MVC), occurred inconsistently in various muscles and usually unaccompanied by additional responses, and occurred in a small percentage of the cases as in the control group. Further, when activity was elicited, it occurred 182 ms (mean across all muscles in which there was a burst of activity during the stretch) after initiation of the stretch, which was 131 ms later than the mean elapsed time for the first responses in finger muscles. Thus most muscle responses measured were considered a possible mix of reflex and voluntary activity.

Differences in the SR response between the thumb and fingers were unexpected, especially since digital cortical representation is dispersed, overlapping (Rathelot and Strick 2006; Schieber 1999; Schieber and Hibbard 1993), and repetitive (Pappas and Strick 1981; Strick and Preston 1982), although plausible explanations exist relating to the possible consequences of hand-area cortical damage on the thumb. As it relates to voluntary muscle control, it has been argued (Lang and Schieber 2003) that a partial lesion in M1’s hand area could lead to (preferential) sparing of neurons responsible for controlling the thumb. For example, if a lesion affects one part of the hand area and assuming that the thumb has a greater representation in the hand area than the fingers (Penfield and Boldrey 1937; Penfield and Rasmussen 1950; Woolsey et al. 1952), more neurons controlling the thumb could be spared. Although, from our data, we have no way of knowing to what extent these neurons were spared [e.g., the profound kinetic weakness we measured during maximum torque production (Table 1) could be due to abnormal muscle cocontractions] and, although it is unclear how sparing in M1 affects reflex behavior in the thumb, we cannot rule out its possible influence, given that previous findings have implicated the cortex’s involvement in the reflex circuit of the long thumb flexor (Marsden et al. 1972, 1973, 1976).

Thumb muscles are generally thought to have greater cortical input than those of the fingers (Penfield and Boldrey 1937; Penfield and Rasmussen 1950; Woolsey et al. 1952) and thus...
may be affected more directly by sensory and/or motor cortical damage. Marsden et al. (1973) provided evidence that the human stretch reflex likely receives input from the cortex. It was shown in several investigations (Marsden et al. 1972, 1973, 1976), for example, that the time in which FPL’s reflex occurred was longer than the time expected for a reflex involving only the spinal cord [e.g., 45 ms (FPL) vs. 23 ms (finger tendon reflex); Marsden et al. 1973]. Results from earlier studies (Marsden et al. 1973, 1977a,b) strongly support the idea that damage to the cortex (sensory and motor) and/or supporting pathways can reduce, if not abolish, reflex responses in the long thumb flexor. In particular, this also includes damage to the sensory cortex because many of the brain-injured patients that Marsden and colleagues tested (also with depressed SRs) had impaired joint position sense, vibration sense, and two-point discrimination of the thumb. These findings may help explain the lack of exaggerated reflexes in paretic thumb flexor muscles in this study.

**Reflex activity in finger muscles**

The observed exaggerated SR responses in the flexor muscles of the fingers following hemiparetic stroke were expected, in agreement with earlier findings. Elevated SRs are a typical sequel of an upper motor neuron (UMN) lesion. As we previously noted, in earlier studies (Kamper and Rymer 2000; Kamper et al. 2003) we have illustrated that, following chronic hemiparetic stroke, finger flexor muscles have elevated SR responses without muscle preactivation when flexor muscles are rapidly stretched. These initial responses, which usually begin during the ramp/dynamic stretch phase of the perturbation, are usually accompanied by additional responses. These additional responses often extend beyond the threshold for voluntary activity (~150 ms) and the start of the hold/static stretch phase and, in some subjects, persist until the end of the ramp-and-hold stretch perturbation (Fig. 2A). The source of the additional responses is unclear partly due to the time-varying, continuous nature of the ramp-and-hold stretch perturbation, partly because muscle spindles are known to respond to both dynamic (type Ia) and static (type Ib, II) muscle stretching (Cordo et al. 2002), and partly since volitional activity becomes a possible factor relatively early in the stretch compared with the length of the stretch.

Notwithstanding, the causes for the elevated initial responses are uncertain. In primates, it is known that the reticulospinal tract provides monosynaptic and disynaptic input to forearm muscles (Davidson and Buford 2004, 2006)—which could include extrinsic finger muscles—and intrinsic hand muscles (Riddle et al. 2009), although evidence suggests the latter could be limited to intrinsic thumb muscles. Therefore one possible argument for exaggerated SRs in the finger flexors involves disinhibition of the reticulospinal pathway (Kamper et al. 2003). Disinhibition could depolarize the membrane potential of motor neurons (MNs) controlling finger flexor muscles, which in turn could increase motor neuronal excitability to flexor muscle-spindle input (e.g., primary afferent input).

**Comparison of reflex characteristics between thumb and finger muscles**

Results in the study may reflect an important difference between finger and thumb flexors in relation to the properties of the involved muscle afferents and spinal motoneurons. Specifically, spinal motoneurons of the finger flexors may have had a lower activation threshold (Lee et al. 1987; Powers et al. 1988) or were associated with a higher reflex loop gain (Gotlieb et al. 1978; Rack et al. 1984). Perhaps, muscle spindle signaling properties or activation machinery in the paretic thumb and finger flexors differed, although we could not find support in the literature for either view. Finally, perhaps finger and thumb flexors had different “offsets” for responding spastically to rapid muscle stretching. We have shown in previous work (Kamper et al. 2001) that the timing of reflex responses in spastic muscle can be affected by the starting length of muscle. Specifically, longer lengths gave rise to earlier responses. Perhaps in this study, paretic thumb flexors would have responded consistently with elevated SR responses if the starting posture of the thumb were more extended so as to increase the initial muscle length.

**Weakness as a factor of thumb impairment**

Although abnormal SRs in flexor muscles did not appear to be a factor of thumb impairment (as evaluated by the CMSA test), reduced muscle activation (Dietz et al. 1991; Fellows et al. 1994; Kamper et al. 2006), abnormal muscle activation (Kamper and Rymer 2001), or muscle atrophy (O’Dwyer et al. 1996) may have been a factor. The hemiparetic group produced only 9% of normal thumb flexion strength (Table 1). Although it is likely that both reduced muscle activation and abnormal muscle coordination are important factors, we do not have supporting data. Thumb muscle activity was quantified using surface electrodes, which was useful for evaluating SR responses but limiting for evaluating voluntary muscle activity of the deep thumb muscles.

Few studies of the thumb following hemiparetic stroke exist to which to compare our findings. One study (Lang and Schieber 2003) actually reported substantial sparing of the thumb following pure motor hemiparesis. The study found that participants could produce normal independent flexion–extension movements on the contralateral side, execute a precision grip, and complete all seven tasks of the Jebsen–Taylor Hand Function test (Jebsen et al. 1969), a timed clinical examination that tests a user’s ability to perform a range of everyday tasks. By comparison, the digits of the participants in this study were substantially impaired and exhibited little spared ability. In almost all cases, voluntary movement was limited to partial wrist extension, partial finger or wrist flexion, and/or lateral pinch grip (CMSA score of ≤3). However, it is unclear from our data whether the fingers or the thumb had the greatest involvement.

Another study (Marsden et al. 1977a) reported that the long-latency reflex response of FPL was abnormally depressed following stroke. Measurement of depressed reflex responses was beyond the scope of this study because our participants, in contrast to the participants in the study by Marsden and colleagues (1977b), remained at rest throughout the stretch trials. Latency calculations, for the purpose of determining
reflex latency type, were also beyond the scope of this study. Often, a reflex latency is computed following a quasi-instantaneous input to tendon (e.g., that from a tendon tap). By design, the ramp-and-hold perturbation paradigm provides a time-varying, continuous input to muscles/tendons. As such, it makes less sense (than if the perturbation were a tendon tap) to talk about short and long latencies. Further, one of our earlier studies (Kamper et al. 2001) showed that muscle length influences the magnitude and timing of a reflex response. Thus characteristics of the ramp could affect whether and when a muscle reflex response occurs. In addition, it is unclear what effect the “hold” or a static muscle stretch has on the reflex characteristics of muscle, although it seems from our data that there was a correlation between the hold phase and additional responses observed. In a few instances, additional responses persisted until the fingers were returned to their starting posture (e.g., Fig. 2A). Perhaps, this is further evidence that designations such as M1 or M2 may not apply when ramp-and-hold perturbations are applied.

The results of this study may be limited to the specific stroke population tested and limited by sample size despite the relatively homogeneous subject populations; most in the hemiparetic group had severe hand impairment (i.e., CMSA 2 or 3, Table 1) and subjects in the control group were approximately age-matched with those in the hemiparetic group. The results of the study may also be limited by the decision to pool nonmixed (reflex) and mixed (reflex plus voluntary) thumb muscle responses together because those responses were compared with the reflex activity of finger muscles. However, since there was such a large discrepancy between thumb muscle responses and those of the finger muscles, we believe overall findings were unaffected. Although we believe the experimental design of the study did not allow proper analysis of the additional finger muscle responses, it would be useful to determine the cause of the additional responses because they could relate to important stroke-induced nervous system changes. To the best of our knowledge, this is the first study of the flexor muscles of both the thumb and fingers in the same paradigm. Overall findings indicate that, whereas parietic finger flexor motoneurons were hyperexcitable, parietic thumb flexor motoneuronal activity resembled that of the control group. On the rare occasion when thumb flexor muscle activity was recorded during the stretch, the timing of the activity was very much delayed compared with that of the finger flexors. These findings intimate one or more differences in hand impairment mechanisms following chronic, severe, hemiparetic stroke.

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DISCLOSURES

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