Motor impairment of the thumb following stroke

1 Diminished capacity to modulate motor activation patterns according to task
2 contributes to thumb deficits following stroke

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Summary

The objective of this study was to explore motor impairment of the thumb following stroke. More specifically, we quantitatively examined kinetic deficits of the thumb. We anticipated that force deficits would be non-uniformly distributed across the kinetic workspace due in part to varying levels of difficulty in altering the motor activation pattern to meet the task.

Eighteen stroke survivors with chronic hemiparesis participated in the trials, along with nine age-matched controls. Of the stroke survivor group, 9 subjects had moderate hand impairment and the other 9 subjects had severe hand impairment. Subjects were instructed to generate maximal isometric thumb tip force, as measured with a load cell, in each of six orthogonal directions with respect to the thumb tip. Activity of three representative thumb muscles was monitored through intramuscular and surface electrodes. Univariate split-plot analysis of variance revealed that clinical impairment level had a significant effect on measured force ($P < 0.001$), with the severely impaired group producing only 13% of the control forces and the moderately impaired group generating 32% of control forces, on average. Weakness in the moderately impaired group exhibited a dependence on force direction ($P = 0.015$), with the least relative weakness in the medial direction. Electromyographic recordings revealed that stroke survivors exhibited limited modulation of thumb muscle activity with intended force direction. The difference in activation presented by the control group for a given muscle was equal to 40% of its full activation range across force directions, whereas this difference was only 26% for the moderately impaired group and 15% for the severely impaired group. This diminished ability to modify voluntary activation patterns, which we previously observed in index finger muscles as well, appears to be a primary factor in hand impairment following stroke.

**Keywords:** hand, weakness, cerebrovascular disorders, and EMG
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**Abbreviations:** APB = abductor pollicis brevis, CMC = carpometacarpal, DOF = degrees-of-freedom, EMG = electromyogram, EPB = extensor pollicis brevis, FIR = finite impulse response, FPL = flexor pollicis longus, IP = interphalangeal, MCP = metacarpophalangeal
Introduction

Stroke survivors often experience severe hand impairment following stroke. This hand impairment greatly affects quality of life, as many activities of daily living rely on hand function for their successful implementation. Diminished control of the thumb could especially impact hand use due to the importance of the human thumb to function (American Medical Association 1990; Lang et al. 2009). The thumb has the greatest independence of movement of any of the digits (Hager-Ross and Schieber 2000), more precise proprioception than other digits (Gandevia et al. 2002), and disproportionately large cortical representation, both in motor cortex (Penfield and Rasmussen 1950) and sensory cortex (Nakamura et al. 1998).

Yet, identification of thumb impairment mechanisms has been fairly limited relative to the thumb’s functional importance. Deficits in voluntary thumb extension (Lang et al. 2009), grasp timing and production (Nowak and Hermsdorfer 2003; Raghavan et al. 2006a), thumb grasp force direction (Seo et al. 2010), and thumb individuation (Raghavan et al. 2006b) have been reported, but the exact causes of these impairments remain unclear. A number of studies have examined impairment mechanisms in the fingers (Cruz et al. 2005; Kamper and Rymer 2001), but the issues for the thumb may differ. For example, while we have found spastic stretch reflexes to be prevalent in the finger flexors of stroke survivors with severe to moderate hand impairment (Kamper et al. 2006b; Kamper and Rymer 2000), spasticity was largely absent in the thumb muscles in subjects with similar levels of hand impairment (Towles et al. 2010).

Additionally, greater deficits in control of the thumb compared to those previously reported for the workspace of the index finger (Cruz et al. 2005) may occur due to the thumb’s relatively greater reliance on intrinsic muscles for dexterity and force production (Milner and Dhaliwal 2002). The intrinsic hand muscles receive substantial input from corticospinal
Motor impairment of the thumb following stroke pathways (Porter and Lemon 1993) and thus may be especially likely to be compromised following stroke. Weakness has been reported for torque generation about the knee (Lomaglio and Eng 2008), shoulder (Andrews and Bohannon 1989), elbow (Canning et al. 2004) and finger (Kamper and Rymer 2001) joints following stroke. While these deficits may not contribute directly to the extent of impairment (few everyday tasks require maximal force production), the nature of these deficits may speak to the underlying impairment. For example, it has been shown that the strength of elbow extension torque in stroke survivors varies with the amount of required shoulder adduction torque to support the weight of the arm (Dewald et al. 1995). This suggests an abnormal neurological coupling between the shoulder abductors and the elbow flexors which inhibits voluntary elbow extension.

Force deficits in the thumb, however, remained largely unexplored. Thus, we sought to examine motor control of the thumb during isometric force generation in individuals with chronic hand impairment subsequent to stroke. We hypothesized that thumb deficits would be non-uniform across the kinetic workspace. Specifically, in accordance with findings in the stroke finger, we anticipated that thumb extension deficits would be relatively greater than those for thumb flexion. Additionally, we anticipated that abnormal motor activations would be present, such that modulation of activation patterns would be limited.

A total of 27 subjects, 9 with severe hand impairment, 9 with moderate hand impairment, and 9 with no neuromuscular injury, participated in the study. They were asked to demonstrate their control of force production across the kinetic workspace by producing maximal thumb tip forces in each of six specified directions with respect to the thumb tip. Three-dimensional thumb tip forces and the activity from representative muscles were evaluated and compared across groups. While an inability to fully activate the motoneuron pool certainly
Motor impairment of the thumb following stroke contributes to these substantial thumb force deficits, our findings suggest that a diminished capacity to modulate thumb muscle activation with force direction in the hemiparetic hand also plays a key role post-stroke.
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Methods

Subject Population

Subjects with chronic hemiparesis subsequent to stroke (at least 9 months post-injury), or with no history of neuromuscular impairment (controls), were asked to perform tasks for the assessment of thumb kinetics and muscle activation patterns. Written informed consent was obtained from all subjects in accordance to the Declaration of Helsinki, and all procedures were approved by the Institutional Review Boards of Northwestern University and the Edward Hines, Jr. VA Hospital. The affected hand was tested in 18 stroke survivors (1 woman, 17 men; 40-80 years of age; 7 ± 5 (mean ± standard deviation) years since the cerebrovascular incident).

Eleven stroke subjects had left-sided hemiparesis while the other seven subjects had primary involvement of the right side. The stroke survivors were evenly divided into two groups based upon clinical classification of hand impairment according to the Stage of Hand section of the Chedoke-McMaster Stroke Assessment (Gowland et al. 1995). The Chedoke-McMaster Assessment uses an ordinal scale to characterize the stage of recovery, ranging from Stage 1 (minimal function) to Stage 7 (normal function). Nine subjects for this study were classified as having severe hand impairment (Stage 2 or 3) and another 9 were classified as having moderate hand impairment (Stage 4 or 5). The dominant hand was tested in 9 control subjects (9 men, 40-80 years of age). Table 1 lists subject characteristics classified by group.

<table>
<thead>
<tr>
<th>Group</th>
<th>Age (years)</th>
<th>Years Post Stroke</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>51 ± 6.8</td>
<td>NA</td>
</tr>
<tr>
<td>Moderate Impairment</td>
<td>65 ± 10.9</td>
<td>7.6 ± 4.3</td>
</tr>
<tr>
<td>Severe Impairment</td>
<td>56 ± 12.3</td>
<td>6.3 ± 5.7</td>
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</table>

Values are mean ± SD.
Experimental Protocol

The experiment consisted of voluntary generation of isometric force at the distal segment of the thumb. The thumb was secured to a six degree-of-freedom load cell (model # 20E12A-I25, JR3, Inc., Woodland, CA) through a set of four set screws embedded in casting material placed around the thumb segment. Thumb posture was maintained throughout the experiment, with the carpometacarpophalangeal (CMC) joint extended approximately 50°, the metacarpophalangeal (MCP) joint flexed 20° and the interphalangeal (IP) joint flexed 30°. The thumb was abducted at the CMC joint such that the longitudinal axis of the distal phalanx was rotated approximately 30° lateral to the longitudinal axis of the forearm. The wrist and forearm were kept in neutral wrist flexion/extension and ulnar/radial deviation and neutral forearm pronation/supination by forming a fiberglass cast around them (Kamper et al. 2003). This cast was clamped to a table to prevent hand displacement during the experiment.

Metacarpophalangeal joint flexion/extension of the fingers was maintained in a neutral posture (Fig 1). The subject was seated comfortably in a Biodex II chair (Biodex Medical Systems, Shirley, NY), which was used to help control posture of the proximal arm and trunk. Chair position, height and rotation were adjusted to ensure 30° of shoulder abduction, 20° of shoulder flexion and 110° of elbow extension.

In order to examine motor control throughout the kinetic workspace, subjects were instructed to generate maximal force with the distal thumb segment in one of six orthogonal directions with respect to the thumb tip: flexion, extension, medial, lateral, proximal, or distal (Fig 2). Subjects performed three trials in each direction for a total of 18 trials. For each trial, subjects sustained the isometric force for an average of three seconds. A one-minute rest break
Motor impairment of the thumb following stroke was instituted between trials. To eliminate the possible confounding factors resulting from the passive stretching required for subject positioning, the relative change in the force and/or EMG activity between the resting state in the predefined posture and the exertion state during the maximal force production as used for all data representation in this study.

Insert Fig 2.

Three-dimensional force data were measured throughout each trial by the load cell. Muscle activity was recorded from a representative extrinsic flexor muscle (flexor pollicis longus (FPL)), an extrinsic extensor muscle (extensor pollicis brevis (EPB)), and an intrinsic muscle (abductor pollicis brevis (APB)). Intramuscular electrodes were used to collect electromyogram (EMG) data for the two extrinsic muscles, FPL and EPB. These muscles were first located using tungsten needle microelectrodes (60mm/5KΩ, Frederick Haer & Co., Inc., Bowdoinham, ME), in accordance with published procedures (Burgar et al. 1997; Keen and Fuglevand 2004a; b; Perotto 1994). Then, two 55-μm stainless steel intramuscular bipolar electrodes were inserted into each muscle through a hypodermic needle. In all cases electrode insertion was guided by audible feedback from muscle contraction. Proper placement was confirmed by appropriate thumb motion in response to imposed electrical stimulation (Digitimer Constant Current Nerve Stimulator model DS7A, Digitimer, LTD., England). We have successfully used these techniques in the past to locate and stimulate (Kamper et al. 2006a) as well as record (Qiu et al. 2009) activity from the muscles of the index finger. A surface electrode (Delsys Inc., Boston, MA) was placed over the thenar eminence to target APB. Force and EMG data were low pass filtered at 125 Hz and 450 Hz, respectively, with a 4th-order Butterworth filter prior to sampling at 1 kHz.
Data processing

The force data from the kinetic study were digitally low-pass filtered forwards and backwards at 10 Hz using a 30th-order finite impulse response (FIR) filter. For each trial, maximum force was averaged over a 200-ms window centered at the point of maximum force in the intended direction. These force data were collected for the desired axis as well as the two other orthogonal axes. As perceived weakness may result from an inability to properly direct force (Seo et al. 2010), the magnitude of the resultant vector was examined to test for a correspondence with the magnitude of the desired force component for the thumb. Thumb force data for the stroke subjects were normalized by the averages of the peak forces attained by the control subjects, and thus represented as a percentage of the mean control peak force.

The EMG data were rectified and then digitally low-pass filtered at 10 Hz using a 30th-order FIR filter to create envelopes of activity. Each envelope was normalized to the subject’s peak level of voluntary activity during the entire experimental session. The EMG envelope for each of the three muscles was averaged across a 200-ms window, beginning 50 ms prior to the occurrence of the window for peak force production in the intended direction. These values were used to compare the activation patterns among subject groups. The minimum and maximum values across all force directions were then determined for each subject to compute the extent to which she modulated muscle activation with force direction.

Statistical analysis

For the four dependent variables of interest in this study, (intended force, APB EMG, EPB EMG and FPL EMG), a MANOVA was first performed using SPSS software (SPSS Inc., Chicago, IL) to determine if the independent variables of subject group (Group), or intended force direction (Direction) impacted the output. Group (3 levels) was the between-subject factor while Direction (3 levels) was the within-subject factor. A second MANOVA was performed to
examine the modulation of EMG activation patterns across the different force productions. Subsequently, findings of significance according to the Wilks’ lambda value led to running separate univariate split-plot ANOVAs for each of the dependent variables. *Post hoc* Tukey tests were conducted to determine statistically distinct levels of the main effects. A Bonferroni correction for the 2 separate analyses was implemented, such that the significance level was set to 0.025 ($\alpha/2$) yielding an overall significance level of $\alpha = 0.05$. 
Results

Assessment of the kinetic workspace of the most affected thumb in 18 stroke survivors with chronic hand motor impairment (9 moderate and 9 severe) and the dominant thumb in 9 intact subjects was conducted. Both maximum thumb force production and muscle activation patterns varied across subject group. The MANOVA revealed that both independent variables (Group and Direction) as well as the interaction term Group—Direction had a significant effect on the kinetic output (Wilks’ lambda < 0.001). Consequently, univariate split-plot ANOVAs were performed on each of the four dependent variables.

<table>
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<th>APB EMG</th>
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<td>Group</td>
<td>$P &lt; 0.001$</td>
<td>$P = 0.574$</td>
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<td>Direction</td>
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Significance accepted at $P < 0.05$.

Ability to generate voluntary forces in the intended direction was distinct between groups with differing degrees of functional impairment (Table 2). The absolute force in the intended direction averaged across all directions was 38.9 ± 21.0 N for the control group, 12.5 ± 7.5 N for the moderately impaired group and only 5.0 ± 3.3 N for the severely impaired group. Thus, the thumb force for the group with severe impairment was only 13% of that of the control group, while the moderately impaired group produced 32% of normal force levels (Fig. 3a). While misdirection of the applied force may have partly contributed to the observed thumb force deficits, the magnitude of this contribution was relatively minor. Weakness in the resultant forces for each direction was very similar to the values recorded for the intended direction only; 15% and 35% of the control forces for the severely and moderately impaired subjects, respectively (Fig 3b).
Interestingly, while the maximum thumb force produced changed substantially with intended direction ($P < 0.001$), this pattern differed across subject groups (Direction—Group: $P < 0.001$). Control subjects produced maximum force in the distal direction, for example, but moderately impaired subjects generated maximal force in the medial direction and severely impaired subjects produced maximal force in the proximal direction (Fig 3).

To better examine the dependence of force deficits on direction within the kinetic workspace, force values for the stroke subjects were normalized by the mean values for the control subjects (Fig 4). No significant directional dependence was seen for the force deficits for the severely impaired subjects ($P = 0.149$). Indeed, the percentage of control force produced ranged only from $6\% \pm 10\%$ (extension) to $20\% \pm 20\%$ (proximal). The moderately impaired subjects did exhibit a directional dependence for force deficits ($P = 0.015$). Normalized force values ranged from $21\% \pm 15\%$ (distal) to $54\% \pm 35\%$ (medial).

EMG patterns across the different directions for each of the subject groups were also quite distinct (Fig 5), as confirmed by the significance of the Direction—Group interaction term for all three muscles: APB, EPB, and FPL ($P < 0.001$, Table 2). The lack of modulation of thumb muscle activity with intended direction was the most striking feature of the stroke activation patterns. MANOVA results revealed a significant impact of subject group on the range of EMG values ($P < 0.001$). The variation in FPL activity across force directions was significantly reduced for both the Severe ($P < 0.001$) and Moderate ($P < 0.02$) groups in comparison to the Control group. FPL values ranged from $12\%$ to $23\%$ for the Severe group, $11\%$ to $24\%$ for the Moderate group and $3\%$ (during extension force production) to $44\%$ (during
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flexion force production) for the Control group (Fig. 5). Similarly, EPB modulation was significantly reduced for both the Severe \((P < 0.001)\) and Moderate \((P < 0.02)\) groups in comparison to the Control group, with ranges from 33\% to 37\% for the Severe group, 11\% to 21\% for the Moderate group, and 5\% to 43\% for the Control group. APB variation was also reduced for the Severe group \((P < 0.001)\); APB activity ranged from 15\% to 30\% for the Severe group, 4\% to 30\% for the Moderate group, and 3\% to 42\% for the Control group.

Thus, these results show substantial thumb force deficits but with an interesting relationship to intended force direction. This relationship seems to result from a diminished capacity to modulate thumb muscle activation with force direction.

Insert Fig. 5
Discussion

Stroke subjects with chronic paresis of the hand, and age-matched controls, participated in experiments to examine thumb motor control during isometric tasks. Overall, substantial thumb impairment was observed, as evidenced by the large force deficits recorded. The level of weakness was associated with the degree of impairment: subjects with severe functional hand impairment (rated Stage 2 or 3 on the Chedoke-McMaster scale) were substantially weaker across directions than subjects with moderate impairment (rated Stage 4 or 5) who in turn were substantially weaker than control subjects. The maximum values for the Control subjects were similar to those reported in a prior study (Valero-Cuevas et al. 2003). The relationship between hand impairment and thumb weakness is in accordance with previous findings pertaining to the index finger (Cruz et al. 2005). Across all force directions, the severely impaired subjects were able to generate only 12% of the forces that the control subjects generated, while moderately impaired subjects produced 32% of normal force values. Although difficulty in directing thumb forces has been reported (Seo et al. 2010), the force deficits we observed did not result from misdirection, as attested to by the similar deficit patterns for both force in the intended direction and resultant force for the stroke subjects. These force deficits were as great or greater than those we observed in the index fingers of stroke survivors with similar levels of impairment in a previous study (Cruz et al. 2005), despite the fact that another study reported greater sparing of control of the thumb relative to other digits (Lang and Schieber 2003). One explanation for these discrepancies is that our subjects most likely had more extensive lesions, possibly affecting both cortical and sub-cortical structures.

Surprisingly, while deficits were not uniform across the kinetic workspace, the observed relationships between weakness and region of the workspace were unexpected. We had
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anticipated that force deficits would be smallest for thumb flexion, as we saw in our previous
study examining index finger forces (Cruz et al. 2005). Instead, thumb flexion deficits were on
par with those for other directions within the kinetic workspace. For the severely impaired
subjects, there were no statistically significant differences in relative force limitations across the
six directions. The moderately impaired subjects exhibited greatest relative sparing in the medial
force direction, which typically requires primary activation of intrinsic muscles (Valero-Cuevas
et al. 2003). We had thought these directions needing substantial contributions from the
intrinsic would be preferentially impacted by stroke, but this was not the case. Of course, it
should be noted that creation of thumb tip forces in any one of the specified directions used in
this study requires the coordination of 3 or more of the 8 thumb muscles.

While atrophy (Carin-Levy et al. 2006; Ryan et al. 2002) and increased passive stiffness
(Chung et al. 2004; Mirbagheri et al. 2008) might contribute to weakness, our data suggest that
the deficits are namely neurological in origin, as we observed previously with the fingers
(Kamper et al. 2003). Essentially, the isometric results serve to illustrate the extent to which
motor control of the thumb is compromised following stroke.

Indeed, the most striking feature of the data from the stroke survivors was the reduced
range of muscle activation with intended force direction. While the control group exhibited a
difference in activation of a given muscle equal to 40% of its full activation range across the six
force directions, this difference was at most 26% for the moderately impaired group and 15% for
the severely impaired group. EPB especially showed little variation with direction, especially for
severely impaired subjects (4%) but also for moderately impaired subjects (10%). This lack of
modulation with direction is especially poignant as all EMG signals for a given subject were
normalized to the values obtained during maximum voluntary contractions by that subject.
Undoubtedly, there is a diminished capacity to voluntarily excite these paretic muscles (Clark et al. 2006), so the true ranges of muscle excitation in relation to muscle capacity are even smaller for the stroke subjects than these numbers suggest. For the severely impaired subjects, in fact, there was no significant change in EPB activation with direction, although APB and FPL did show significant, albeit reduced, modulation. Similar results were shown for the finger muscles (Cruz et al. 2005). Interestingly, there was not only a limitation on the maximum excitation in the preferred direction but also a limitation on the minimum excitation generated in a non-preferred direction. In severely impaired subjects, for example, EPB activation was approximately 35% regardless of intended force direction.

Reduced reciprocal inhibition (Crone et al. 2000), abnormal synergies (Dewald et al. 1995), or even impaired motor planning (Daly et al. 2006) may contribute to degrading these activation patterns. Reduction of corticospinal input may also lead to increased influence of brainstem pathways, such as the reticulospinal pathway, which are thought to provide less flexible control of individual muscles (Riddle et al. 2009). Alternatively, the results may be indicative of alterations in the motor units following stroke. Spontaneous discharge and co-modulation of motor units have been shown to increase following stroke (Mottram et al. 2009) and the firing rate of a given motor unit becomes more compressed (Gemperline et al. 1995). As noted, in more proximal muscles, a limited capacity to activate the muscle has been shown (Patten et al. 2004), thereby suggesting a reduced pool of motor units available for activation. With fewer units, and with those units having a reduced range of firing rates and greater likelihood of being co-modulated, precise control of muscle activation would be compromised, leading to diminished directional specificity.
Clinical Implications

The deficits detected in thumb flexion may be especially limiting. For pinch or cylindrical grasp, the force provided normal to the object by the thumb must oppose that of the other digits. Weakness in generating these normal forces subsequently limits the magnitude of the frictional shear force that can be applied to the object to prevent it from slipping while being held or transferred.
Acknowledgements

The authors would like to thank Megan Conrad, Ph.D. and Gilles Hoffmann, Ph.D. for their assistance with statistical analysis.

Grants

This work was supported by a merit grant from the Department of Veteran Affairs [B4358R]; and the Coleman Foundation.
References


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Figure Legends

Fig. 1: Experimental setup. Wrist was casted and clamped to tabletop. Fingers were held in position by an air bladder. The thumb was maximally abducted, casted and held in place by the fixture on the load cell.

Fig. 2: Orientation of thumb tip secured to the load cell. The vectors shown depict the direction of force, with respect to the thumb, that each subject was instructed to generate.

Fig. 3: (a) Maximum force in the intended direction and (b) resultant force for all three subject groups. Control subjects represented by the Xs, moderate stroke subjects by open circles and severe stroke subjects by inverted open triangles. The mean value of each group representing each of the intended directions is depicted by the filled squares (Black = Control, Grey = Moderate, White = Severe with error bar showing the corresponding standard deviation of the data.

Fig. 4: Normalized force in the intended direction for the Moderate and Severe stroke groups. Absolute force values were normalized by the max values produced by the control subjects. Error bar indicates standard deviation.

Fig. 5: Activation patterns for each muscle for each intended force direction a) Control group; b) Moderately impaired group; c) Severely impaired group. Error bar indicates standard deviation.
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**Tables**

Table 1: Subject Characteristics

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Values are mean ± SD.

Table 2: Univariate split-plot ANOVA results for each dependant variable.

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</tr>
</tbody>
</table>

Significance accepted at *P < 0.05*.
Normalized Force

Intended Direction

- Flexion
- Extension
- Lateral
- Medial
- Proximal
- Distal

Moderate
Severe