Kinetic and kinematic workspaces of the index finger following stroke

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Summary

The objective of this study was to explore motor impairment of the index finger following stroke. More specifically, the kinetics and kinematics of the index finger were analysed throughout its workspace. Twenty-four stroke survivors with chronic hemiparesis of the hand participated in the trials, along with six age-matched controls. Hand impairment was classified according to the clinical Chedoke–McMaster Stage of Hand scale. Subjects were instructed to generate fingertip force in six orthogonal directions at five different positions within the workspace. Split-plot analysis of variance revealed that clinical impairment level had a significant effect on measured force ($P < 0.001$), with the weakness in stroke survivors being directionally dependent ($P < 0.01$). Electromyographic recordings revealed altered muscle activation patterns in the more impaired subjects. Unlike the control subjects, these subjects exhibited peak muscle excitation of flexor digitorum superficialis, extensor digitorum communis and first dorsal interosseous during the generation of fingertip flexion forces. Subjects also attempted to reach locations scattered throughout the theoretical workspace of the index finger. Quantification of the active kinematic workspace demonstrated a relationship between impairment level and the percentage of the theoretical workspace that could be attained ($P < 0.001$). The stroke survivors exhibited a high correlation between mean force production and active workspace ($R = 0.90$). Thus, our data suggest that altered muscle activation patterns contribute to directionally dependent weakness following stroke. Both the modulation of muscle excitation with force direction and the independence of muscle activation seem to be reduced. These alterations translate into a significantly reduced active range of motion for the fingers.

Keywords: finger workspace; fingertip force; stroke

Abbreviations: DIP = distal interphalangeal; EDC = extensor digitorum communis; EMG = electromyography; FDI = first dorsal interosseous; FDS = flexor digitorum superficialis; MCP = metacarpophalangeal; PIP = proximal interphalangeal


Introduction

Individuals with chronic hemiparesis of the upper extremity following a stroke typically experience difficulty in controlling the hand. In fact, hand dysfunction is the most common impairment after a stroke (Trombly, 1989). The deficits in finger motor control may limit participation in daily activities (Wade, 1994). The nature of these deficits, however, has not been fully characterized. While a few studies have sought to quantify voluntary finger performance in individuals with chronic hemiplegia (Jeannerod, 1984; Kamper and Rymer, 2001; Hermensrofer et al., 2003; Lang and Schieber, 2004), the relationship between the employed measures and functional impairment has not been well established. Tasks for these studies examined only a limited subset of possible finger functions.

Previous studies have reported a strong relationship between weakness and arm impairment scores on clinical evaluations (Heller et al., 1987; Boissy et al., 1999; Mercier and Bourbonnais, 2004). This relationship is thought to be especially strong for the more distal musculature due to greater direct corticospinal excitation (Porter and Lemon, 1993). Indeed, control of distal muscles is often more greatly impaired than that of proximal muscles (Colebatch and Gandevia, 1989; Mercier and Bourbonnais, 2004). Grip strength alone can be predictive of recovery (Sunderland...
et al., 1989). Yet, while multidirectional torque production about the more proximal joints, such as shoulder and elbow, has been quantified after stroke (Dewald and Beer, 2001; Lum et al., 2003), this has not been fully explored for the fingers. This is especially true for force generation at different finger postures, despite the fact that posture can have a profound effect on force deficits after stroke (Beer et al., 2000; Ada et al., 2003; Koo et al., 2003).

Additionally, few studies have examined both strength and muscle activation. These types of inquiries are important for identifying the origins of impairment. Excessive coactivation, in conjunction with limited overall activation, has been shown to contribute to weakness (Kamper et al., 2003b), as observed for torque productions about more proximal joints (Dewald et al., 1995; Levin et al., 2000; Chae et al., 2002). It is not known, however, whether altered muscle activation patterns contribute to impairment. The extent of aberrant activation patterns at more proximal arm muscles is a matter of debate (Dewald et al., 1995; Canning et al., 2000; Lum et al., 2003; Reisman and Scholz, 2003).

Thus, the goal of this study was to fully explore motor capabilities in the hand following stroke. Towards this end, we chose to focus on a single digit to facilitate analysis and minimize possible confounding interactions among digits. We selected the index finger because of both its functional importance, with its involvement in lateral and palmar pinch, and its precise control, which allows the index finger to have the greatest independence of movement with respect to the other fingers (Schieber, 1991).

Subjects were asked to generate maximal fingertip forces in six orthonormal directions at different locations within the theoretical workspace of the finger. The ability of the subjects to move to locations throughout the extent of the workspace was also tested. We sought to determine whether weakness correlates with severity of hand impairment and whether this weakness exhibits a directional specificity, whether altered activation patterns contribute to this weakness and how these factors shape the attainable kinematic workspace.

Methods

Protocol

Subjects, either 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 the kinematic and/or kinetic workspaces of their index fingers. The impaired hand was tested in 24 stroke survivors (10 women, 14 men; ages 39–68; 7 ± 5 years since the cerebrovascular incident). Of these individuals, 6 had primary involvement of the right arm and 18 had primary involvement of the left arm. The stroke survivors were divided into four groups based upon clinical classification of hand impairment using the Chedoke–McMaster Stage of Hand scale (Gowland et al., 1995). The Chedoke–McMaster uses an ordinal scale ranging from 1 (poorest) to 7 (best) to rate hand performance. The subjects for this study were evenly distributed among Chedoke Stages 2–5, with six subjects in each Stage. Chedoke Stage 2 implies little or no voluntary movement; Stage 3 includes some active movement, but mostly in flexion; Stage 4 implies good movement in flexion and some extension; Stage 5 includes movement in flexion, extension and abduction. This evaluation tool has been shown to have high inter- and intra-rater repeatability, as well as strong correlation with the Fugl–Meyer score (Fugl-Meyer et al., 1975; Gowland et al., 1993). The fifth subject group consisted of six control subjects (4 women, 2 men; ages 38–63) in which the dominant hand was examined for both the kinematic and kinetic experiments.

The kinetic experiment consisted of voluntary isometric force generation at the fingertip for different directions and different finger postures. The wrist and forearm were placed within a fibreglass cast secured to a tabletop with a magnetic clamp (Kamper et al., 2003b). The cast maintained neutral wrist flexion/extension and forearm ulnar/radial deviation, while preventing hand displacement. The tip of the index finger was affixed to a six degree-of-freedom load cell (JR3, Inc., Woodland, CA) through a set of four screw bolts embedded in casting material placed around the fingertip (Fig. 1A). The other three fingers were allowed to rest against a post positioned so that contact with the load cell was prevented but a relaxed posture was maintained. A strap was used to hold the thumb in an extended and abducted position, away from the load cell.

For each trial, the subject was instructed to generate maximal force at the fingertip in one of six orthogonal directions with respect to the distal segment of the finger: palmar, dorsal, lateral, medial, proximal and distal (Fig. 1A). Subjects sustained the isometric force for an average of 5 s during the trial and rested for 1 min between trials. The force data measured by the load cell were filtered with a 4th-order low-pass Butterworth at 125 Hz, and then sampled at 500 Hz. Muscle activity was recorded from a representative extrinsic flexor muscle, an extrinsic extensor muscle and an intrinsic muscle. Specifically, surface electrodes were placed over the flexor digitorum superficialis (FDS), extensor digitorum communis (EDC) and first dorsal interosseous (FDI) for electromyographic (EMG) analysis (Delsys Inc., Boston, MA). The data were filtered at 225 Hz, and sampled at 500 Hz.

Forces were measured at five different finger postures (Fig. 1B). Postures were chosen to position the fingertip at locations centred within the theoretical workspace area for the index finger. This workspace was computed according to passive ranges of motion for each of the three joints (metacarpophalangeal, MCP; proximal interphalangeal,PIP; distal interphalangeal, DIP) in the sagittal plane of the finger using forward kinematics, given the dimensions of a representative index finger (Kamper et al., 2003a). The five locations were evenly spaced along a logarithmic spiral fit to the theoretical workspace (Kamper et al., 2003a). The joint angles for each location were chosen from inverse kinematics with the criterion that DIP flexion be minimized. This criterion was necessary to represent physiologically feasible postures of the index finger, as flexion of the DIP joint while maintaining extension of the PIP and MCP joints is difficult to accomplish. The same sets of joint angles were employed for each subject.

Subjects then participated in a second set of experiments examining the kinematic workspace of the index finger. Each subject was fitted with a volar wrist orthotic splint made of Thermoplast that held the wrist in the neutral position and was secured to the table. Beneath the splint, the subject wore a CyberGlove® (Immersion Corp., San Jose, CA) on his/her impaired (stroke subject) or dominant (control subject) hand. The glove recorded the flexion/extension of the DIP, PIP and MCP joints of the index finger. The CyberGlove® was first calibrated for each subject (Kamper et al., 2003a). Due to difficulties
with the fit of the CyberGlove typical resulting from a small hand, three of the stroke survivors from the kinetic experiment were unable to participate in this experiment. Thus, three new subjects were added, resulting in slightly different age and gender distributions for the 24 hemiparetic subjects (9 women, 15 men; ages 33–68). The control group remained the same.

A theoretical workspace for the tip of the index finger was first formulated (Fig. 2). The boundary of the workspace was computed using forward kinematics, given feasible joint ranges for the experimental setup [MCP (0–70°), PIP (0–90°), and DIP (0–70°)] and finger segment lengths (distal, middle and proximal) measured for each subject. This boundary was printed on a transparency placed above the hand and aligned with the MCP joint of the index finger. The subject was instructed to position his/her fingertip at points randomly selected by a computer to lie throughout the theoretical workspace. The subject tried to match the location of a laser pointer, shone through the transparency towards the table by an experimenter to match the position of a corresponding point shown with respect to the theoretical workspace on the computer display. In this fashion, the experimenter performed the transformation of the points from the computer screen to the plane of the finger for the subject. Four trials, each with 10 points shown for 8 s apiece, were performed. During a fifth trial, the subject traced the theoretical workspace to the best of his/her ability. All the glove sensor data were sampled at 100 Hz.

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 filter. For each trial, maximum force was averaged across a 50-ms window centred at the point of maximum force in the attempted direction. This force data was collected for the desired axis, as well as the two axes orthogonal to it. Thus, in addition to the peak component of the force vector in the desired direction, the description of the resultant force vector in spherical coordinates was also obtained. As perceived weakness may result from an inability to properly direct force rather than from an inability to generate force, we examined the magnitude of the resultant vector to test for a correspondence with the magnitude of the desired force component. Additionally, we analysed the yaw and pitch angles of the resultant vector.

The EMG data were rectified and then digitally low-pass filtered at 25 Hz using a 30th-order finite impulse response filter to create envelopes of activity. Each envelope was normalized to the subjects’ peak level of activity during the entire testing session. The EMG envelope for each of the three muscles was averaged over the same 50-ms window as used for the force data.

For the kinematic trials, the data from the CyberGlove were digitally filtered forwards and backwards with a 20th-order finite
impulse response having a cut-off frequency of 10 Hz (Kamper et al., 2003a). Location of the index fingertip was computed from the joint angle data and segment lengths using forward kinematics. The fingertip location was computed with respect to a coordinate system based at the MCP joint of the index finger. All the trials for a given subject were pooled so that fingertip locations for all the trials were contained in one set. Using this set of fingertip locations, the percentage of the theoretical workspace actually traversed was computed for each subject. The theoretical workspace for each individual was divided into three subregions equally spaced along the axial direction of the index finger in order to determine if certain sections of the workspace were more difficult to attain than others (Fig. 2). The boundaries for the extent of the active fingertip motion within each subregion were found from the data. The area encompassed by each boundary was computed numerically and expressed as a percentage of the total area of that theoretical subregion. Summation of the area within each subregion yielded the total area attained for a given subject, which was also expressed as a percentage of the total theoretical workspace.

Statistics

For the four dependent variables of interest for the kinetic data (force, FDS EMG, EDC EMG and FDI EMG), a doubly MANOVA was first performed using SPSS software (SPSS Inc., Chicago, IL) to determine if the independent variables of subject group (Group), force direction (Direction) or finger posture (Posture) impacted the output. Group was the between-subject factor, while Direction and Posture were the within-subject factors. Due to the modest sample size, the results from the averaged multivariate tests were used. For independent variables for which the Wilks’s lambda value showed significance, post hoc analyses were conducted using a univariate split-plot ANOVA for each dependent variable. For the split-plot ANOVA, the Greenhouse–Geisser correction was employed as Mauchly’s test indicated possible violation of the sphericity assumption. Post hoc Tukey tests were performed to determine statistically distinct levels of the main effects. Eta-squared values were computed to estimate the variance explained by each factor.

For the kinematic data, a split-plot ANOVA was run with the between-subject factor Group and the within-subject factor workspace region (Region). Again, post hoc Tukey tests were performed and eta-squared values were determined. To test the correspondence between the kinetic and kinematic workspaces, we computed the Pearson correlation coefficient for the mean force and total workspace area for each of the 27 subjects who completed both the kinetic and kinematic studies. Total workspace was computed by summing the percentage of the workspace attained for each of the three regions. Mean force was obtained by averaging the force produced by a given subject across the four directions planar with the kinematic workspace (proximal, distal, dorsal and palmar) for all positions.

Results

Assessment of the kinetic and kinematic workspaces was conducted for 30 subjects, 24 of whom exhibited chronic hand motor impairment subsequent to stroke. The other 6 subjects had no known neuromuscular deficits.

Table 1 Results of the split-plot ANOVA performed on the force data

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<thead>
<tr>
<th>Independent variables</th>
<th>ANOVA results</th>
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<td>Posture</td>
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<td>Group × Direction</td>
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<tr>
<td>Group × Posture</td>
<td>10.7</td>
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<tr>
<td>Direction × Posture</td>
<td>6.3</td>
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<tr>
<td>Group</td>
<td>4</td>
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DF, degrees of freedom; F, F-statistic; Sig., P-value.

Kinetic workspace

The doubly MANOVA revealed that all the independent variables (Group, Posture, Direction) had a significant impact on the kinetic output (Wilks’s lambda < 0.01). Each of the interaction terms, aside from the three-way interaction Group–Direction–Posture, also had a significant effect (Wilks’s Lambda < 0.05). Thus, split-plot ANOVA was performed for each of the four dependent variables using all three independent variables.

As anticipated, functional impairment had a highly significant impact on force deficits, as attested to by the statistical significance of Group in the split-plot ANOVA for force (P < 0.001, see Table 1). Post hoc Tukey tests revealed two distinct groupings, with the more impaired subjects (Chedoke Stages 2–4) producing less overall force than the less impaired (Chedoke Stage 5) or control subjects (P < 0.01).

This evidence for gross finger weakness was substantiated by the magnitude of the resultant force vector, with the subjects at Chedoke Stages 2–4 producing 6.7, 9.6 and 12.0 N of force, respectively, while the subjects at Stage 5 and the controls generated 25.0 and 26.7 N of force. Across subjects, Direction was significant, as were the interaction terms Group–Direction and Direction–Posture (P < 0.01), and the term Group–Posture (P < 0.05).

Examination of the Group–Direction interaction revealed that stroke subjects exhibited directionally dependent weakness in force production, in comparison with control subjects (Fig. 3). For example, the groups at Chedoke Stages 2 and 3 showed greater relative strength in the palmar rather than dorsal direction (20% and 34% of control values for palmar force, respectively, as compared to 2% and 15% of control values for dorsal force).

Interestingly, for certain desired directions it appeared more difficult for the stroke survivors to properly direct force. For example, during lateral force production, subjects at Chedoke Stage 2 generated a resultant force pointing 68.7° towards the palmar axis and 66.8° from the lateral axis, while for the control subjects these values were 4.8° towards the dorsal axis and 27.8° from the lateral axis (Fig. 4). Proximal force production generated a resultant force vector rotated 66.1° towards the palmar axis while this rotation was only 9.7° for the control subjects. For the other force directions,
however, the resultant vector orientations for these two groups were very similar.

Certain finger postures seem less conducive to force production in a specified direction, as evidenced by the significance of the Direction–Posture interaction (Table 1). In the posture with greatest MCP flexion (Posture 5 in Fig. 1B), subjects generated the least force in the palmar, lateral and distal directions. Subjects generated the least amount of dorsally directed force when the MCP was most extended (Posture 1). Postural effects were also dependent upon Group (eta-squared = 0.248), with subjects at Chedoke Stages 2 and 3 being strongest at Posture 1, unlike the subjects in the control and Chedoke Stage 5 groups.

EMG patterns were also very different for the different subject groups. As the EMG data were normalized to the recording during the maximum voluntary contraction for each subject, there were no significant differences in EMG magnitude among subject groups for any of the three muscles targeted. The interaction term Group–Direction, however, was significant for all muscles, thereby implying an alteration in control (see Table 1 for summary).

For FDS, the factors Posture and Direction, and the interaction terms Group–Direction and Group–Posture, were all significant ($P < 0.01$), but only the latter three explained a large percentage of the variance (eta-squared $\geq 0.300$, see Table 2). Across subjects, FDS activity was greatest for distally directed forces and least for proximally directed forces. The control subjects displayed greater activity during dorsal force production (16% of maximal voluntary contraction) than during palmar force production (9%), while the opposite held true for the more impaired subjects of Chedoke Stages 2 and 3 (7% and 11% for dorsal force as opposed to 22% and 22% for palmar force). This transformation in FDS activity followed a monotonic pattern across subject groups (Fig. 5A). Activity was also greatest for the posture with greatest MCP flexion (19%) and least for the finger posture with greatest MCP extension (15%) across subjects. This was not true for the subjects of Chedoke Stage 2, for whom FDS activity was roughly the same at every posture.

For the EDC activity, Direction and Group–Direction were statistically significant ($P < 0.01$, see Table 3). Across subjects, the EMG signal for EDC was smallest for forces in the

![Fig. 3](image1.png) **Fig. 3** Mean forces in each of the six directions for each Chedoke group. Wings represent 95% confidence intervals. The upper half of the plot represents palmar, lateral and distal directions, with opposing directions (dorsal, medial and proximal) appearing in the lower half.

![Fig. 4](image2.png) **Fig. 4** Differences in direction of the resultant force vector for control subjects (solid vector) and the most impaired (Chedoke Stage 2) stroke survivors (dotted vector). Large differences were apparent for only two directions: (A) lateral and (B) proximal.

### Table 2 Results of the split-plot ANOVA performed on the FDS EMG data

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<th>Independent variables</th>
<th>ANOVA results</th>
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<td>DF</td>
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<td>Direction × Posture</td>
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<td>Group</td>
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DF, degrees of freedom; $F$, $F$-statistic; Sig., $P$-value.
distal direction ($P < 0.01$), and greatest for proximally directed forces (statistically greater than all directions except for dorsal, $P < 0.01$). For the control subjects, EDC activity during dorsal force production was 77% greater than during palmar force production. However, for the subjects in Chedoke Stage 2, EDC activity was actually 87% greater during palmar rather than dorsal force production (Fig. 5B).

For the FDI EMG, the terms Direction, Group–Direction and Direction–Posture were significant ($P < 0.01$, see Table 4). Across subjects, FDI activity was greatest during lateral force production (statistically greater than all other directions except for distal, $P < 0.01$) and least during proximal force production (statistically smaller than for all other directions, $P < 0.01$). For the subjects in Chedoke Stage 2, however, FDI activity was greatest for force in the palmar direction (Fig. 5C). Unlike FDS and EDC, the activity of FDI was influenced by the Direction–Posture interaction term, but this influence was relatively small (eta-squared = 0.118).

### Kinematic workspace

The extent of the kinematic workspace was also determined for each subject. The factor Group was significant ($P < 0.01$, see Table 5). The percentage of the theoretical workspace that could be reached followed the clinical impairment scale (Fig. 6). Tukey tests revealed three distinct groupings in

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**Table 3** Results of the split-plot ANOVA performed on the EDC EMG data

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DF, degrees of freedom; $F$, $F$-statistic; Sig., $P$-value.

**Table 4** Results of the split-plot ANOVA performed on the FDI EMG data

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DF, degrees of freedom; $F$, $F$-statistic; Sig., $P$-value.
which Chedoke Stage 5 and the control group covered a
greater percentage of the workspace than the subjects in
Chedoke Stages 2–4, although Chedoke Stage 4 subjects
had a greater workspace than the Chedoke Stage 2 subjects
(Fig. 7). Across subjects, the largest percentage of the theo-
retical workspace was attained for the uppermost region,
Region 1 (see Fig. 2), and the smallest percentage was
attained for Region 3 (P < 0.05). The interaction term
Group–Region was not significant.

Finally, for the 27 subjects who completed both trials, the
kinematic and kinetic workspaces showed a significant
correlation. The mean force, averaged across position and
the four planar directions, exhibited a high Pearson correla-
tion coefficient (R = 0.90) with the percentage of workspace
attained for the stroke survivors (P < 0.001). This correlation
was reduced when the data from control subjects were
included in the analysis (R = 0.75), because of the poor
correlation between these measures for the control subjects.

**Discussion**

Stroke survivors with chronic hemiparesis of the hand and
age-matched controls participated in experiments to measure

**Table 5 Results of the split-plot ANOVA performed on
the kinematic data**

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DF, degrees of freedom; F, F-statistic; Sig., P-value.

**Fig. 6** Total workspace expressed as a percentage of the
theoretical workspace for each subject group. Error bars denote
95% confidence intervals.

**Fig. 7** Representative workspace traces from a subject within each
distinct grouping: (A) Chedoke 2, (B) Chedoke 4 and (C) Control/
Chedoke 5.
force production and workspace exploration of the index finger. Despite the diversity of subject stature and age within a group, overall finger strength of the stroke survivors was highly dependent upon the level of clinical impairment, as has been described previously (Chae et al., 2002; Mercier and Bourbonnais, 2004). The subjects with greater hand impairment, those rated to be within Stage 2 or 3 of the Chedoke–McMaster scale, were significantly weaker than control subjects, while those at Chedoke Stage 5 did not exhibit any significant weakness.

This weakness had a directional dependence for the more impaired subjects, with flexion being relatively (in relation to control subjects) much stronger than finger extension. For Chedoke Stage 4 and higher, strength deficits were more uniform or non-existent. This directionally dependent weakness has been described for the elbow, but the outcome was reversed such that extensor strength was preserved to a greater extent than flexor strength (Colebatch et al., 1986; Koo et al., 2003). Other studies, however, have not observed this imbalance between antagonist muscle groups (Bohannon and Smith, 1987; Beer et al., 1999; Ada et al., 2000). Part of the difference in results may be explained by the difference in muscle groups examined. The latter studies focused on more proximal muscles activating the elbow and shoulder while this study was focused on the fingers. Also, subjects with a range of impairment levels were grouped together for analysis in these studies. We found the impairment level to be important, as the imbalance was strongest for the more impaired subjects (Stages 2 and 3). Supportive of this finding, another study reported selective weakening of lateral force production for the index finger for some hemiparetic subjects but not for others (Colebatch and Gandevia, 1989).

In the past, finger weakness has been attributed to improper coactivation of agonists and antagonists, as well as an inability to voluntarily excite motoneurons (Kamper et al., coactivation of agonists and antagonists, as well as an inability to voluntarily excite motoneurons, 2003). The current results also suggest a role for improper patterns of muscle activation. Overall, directional specificity of muscle activation was reduced after stroke, especially for subjects at the lower Chedoke Stages, in agreement with other studies (Lang and Schieber, 2004). While control subjects had ratios of 5 : 1 or greater for EMG activity in one force direction versus another for a given muscle, subjects at Stage 2 had ratios in the order of 2 : 1, with a tendency for the muscle to be active independent of direction. Subjects with less clinical impairment (Stages 4 and 5) had activation patterns more closely resembling those of control subjects, although proper activation of the intrinsic FDI muscle was still troublesome for some of these subjects in the Chedoke Stage 4 group.

Intriguingly, the specificity in activation that did remain for the more impaired subjects was altered in terms of force direction. Subjects at Chedoke Stages 2 and 3 tended to maximally activate the FDS muscle during production of a palmar-directed (flexion) force at the fingertip. For control subjects, however, FDS activation was actually greater for force generation in the opposite direction. This is due to the fact that FDS activation tends to extend the DIP joint when the fingertip is fixed in place. For subjects at Stages 2 or 3, FDI and EDC activity were also highest for the palmar-directed forces and lowest for the dorsally directed forces, in contrast with the control subjects. Independence of activation seemed to be compromised for these subjects as FDS, EDC and FDI were similarly activated for a given force direction, possibly exacerbated by diminished reciprocal inhibition (Nakashima et al., 1989; Baykousheva-Mateva and Mandeliev, 1994; Crone et al., 1994). Indeed, stimulation input, which normally produces reciprocal inhibition, may generate facilitation following stroke (Crone et al., 2003). This coactivation was especially apparent during attempts to generate laterally or proximally directed forces, which resulted in significant coupling with flexion.

The flexor bias at the fingers has also been described for spasticity, where FDS exhibited a hyperexcitable reflex response to stretch while EDC did not (Kamper et al., 2003b). Rather, EDC reflex activity occurred only in the presence of FDS reflex excitation during stretch of FDS. The question remains as to why activation would be favoured for fingertip flexion rather than force production in any of the other directions. As we have not found this flexion bias to be prevalent in individuals with clinically complete, high cervical spinal cord lesions (with sparing of motoneurons to finger muscles), we assume a supraspinal origin.

One explanation centres on cortical reorganization following stroke. A number of studies have described cortical changes in the months following a stroke (Weiller et al., 1993; Nudo, 1998; Hallett, 2001; Ward et al., 2003). It is possible that this restructuring favours flexion, especially as extension may require greater cortical activity, even in unimpaired individuals (Yue et al., 2000). Conceivably, axonal sprouting from these cortical pathways could spread to both flexor and extensor motoneurons, especially in the absence of competing extensor pathways, thereby leading to increased coactivation.

Alternatively, this flexor bias may originate from expression of a different area in the brain, such as the brainstem. For example, the rubrospinal tract has been shown to be excitatory for flexor motoneurons and inhibitory for extensor motoneurons of the hand in primates (Kuypers, 1991). Destruction of the rubrospinal tract following bilateral pyramidalotomy in rhesus monkeys resulted in an extended hand posture and difficulty with closing the hand for rhesus monkeys (Lawrence and Kuypers, 1968). Yet, some question the existence of the rubrospinal tract in humans (Nathan and Smith, 1982).

Other areas of brainstem may also promote finger flexion as part of an autonomic ‘fight or flight’ response, mediated by the sympathetic nervous system. Assumption of a fisted hand posture is a common response to stressful stimuli. Following stroke, loss of cortical inhibition could lead to an elevated reaction (such as a fisted posture) to sympathetic
innervation. Furthermore, both hyperhidrosis and hyperthermia, signs of increased sympathetic activity, have been described for the paretic limb relative to the contralateral limb subsequent to stroke (Wanklyn et al., 1994; Koripelainen et al., 1999). Drive to the sympathetic nervous system may also be heightened, possibly arising from the hypothalamus, intimately involved in controlling the sympathetic nervous system. It has been noted that endocinial secretions from the hypothalamus may be elevated following stroke (Franceschini et al., 1994; Qu et al., 1995).

Finger posture also impacted force production, with the interactions of force Direction–Posture and subject Group–Posture being significant. This finding may have implications for clinical assessment. For example, lateral force production, which requires generation of an MCP abduction torque, was smallest across all subjects for the posture with greatest MCP flexion. We have observed that as the DIP, PIP and MCP become more flexed, creation of any abduction torque about MCP becomes more difficult. Testing finger abduction in the flexed posture typical in stroke hands may lead to underestimation of ability. For example, subjects at Chedoke Stage 4 (with limited voluntary abduction) produced a significant amount of lateral force when the index finger was held in an extended posture. This postural dependence seems especially strong in individuals with stroke (Ada et al., 2003; Koo et al., 2003).

As seen previously with reaching (Kamper et al., 2002), the size of the active finger workspace correlated closely with clinical impairment. Subjects in Chedoke Stages 2 and 3 could attain less than 20% of the theoretical workspace. Surprisingly, however, we did not observe a significant dependence of workspace deficits on the region of the workspace. We anticipated that stroke survivors would have the most difficulty reaching the upper region, which required MCP flexion of less than 55°. The lack of a significant effect of the interaction of subject Group–Workspace region may be attributable to the a priori selection of regions. It appears that the region near the workspace boundary was the most difficult to obtain. These boundary areas require asynchronous joint movement, such as MCP extension with PIP and DIP flexion or MCP flexion with PIP and DIP extension, and thus controlled coactivation of extensors, flexors and intrinsics (Darling et al., 1994).

Active workspace was highly correlated with overall strength for the stroke subjects. Hence, strength appears to be a predictor of finger control, with greater strength implying greater active range of motion. This relationship did not, of course, hold for the control subjects, in part because the movement of the finger throughout the workspace required only submaximal force generation for these subjects. It should be noted that weakness cannot entirely explain motor deficits following stroke. The subjects rated Chedoke Stage 5 had nearly normal levels of strength and size of active workspace. Clinically, however, these subjects still exhibited deficits in task performance, particularly those tasks requiring finger individuation.

Conclusions

These findings provide evidence of a directionally dependent weakness in the index finger following stroke. The preferential sparing of flexion forces at the fingertip seems to be supported by heightened muscle activation for this direction. However, this heightened activity is indiscriminate across muscles, in accordance with the observed aberrant activation patterns for all directions. Overall weakness is manifested in an inability to actively explore portions of the finger workspace.

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