Recovery of Thumb and Finger Extension and Its Relation to Grasp Performance After Stroke

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Lang CE, DeJong SL, Beebe JA. Recovery of thumb and finger extension and its relation to grasp performance after stroke. J Neurophysiol 102: 451–459, 2009. First published May 20, 2009; doi:10.1152/jn.91310.2008. This study investigated how the ability to extend the fingers and thumb recovers early after stroke and how the ability to extend all of the digits affects grasping performance. We studied 24 hemiparetic patients at 3 and 13 wk post stroke. At each visit, we tested the subjects’ ability to actively extend all five digits of their contralesional, affected hand against gravity and to perform a grasp movement with the same hand. Three-dimensional motion analysis captured: 1) maximal voluntary extension excursion of each digit and 2) grasp performance variables of movement time, peak aperture, peak aperture rate, and aperture path ratio. We found that finger and thumb extension improved from 3 to 13 wk, with average improvements ranging from 12 to 19° across the five digits. Grasp performance improved on two of the four variables measured. Peak apertures and peak aperture rates improved from 3 to 13 wk, but self-selected movement time and aperture path ratio did not. Stepwise multiple regression models showed that the majority of variance in grasp performance at 13 wk could be predicted by the ability to extend the index or middle finger at 3 wk, plus the change in the ability to extend the index finger from 3 to 13 wk. R2 values ranged from 0.55 to 0.89. Our data indicate that the amount of recovery in finger and thumb extension and grasping is small from 3 to 13 wk post stroke. In people with relatively pure motor hemiparesis, one important factor underlying deficits in hand shaping during grasping is the inability to extend the fingers and thumb. Without sufficient volitional control of finger and thumb extension, successful grasping of objects will not occur.

INTRODUCTION

Loss of the ability to extend the fingers and thumb after stroke is a critical impairment observed by clinicians and is a common complaint from patients. The inability to extend the digits is primarily due to a limited ability to activate the finger and thumb extensor muscles (Kamper and Rymer 2001; Kamper et al. 2003, 2006). Historically, there has been little success in improving finger and thumb extension capabilities with targeted rehabilitation techniques (Trombly et al. 1986). The goal of improving digit extension is highly valued because the ability to extend the fingers and thumb post stroke is a key criterion for participation in (Boake et al. 2007; Page et al. 2004; van der Lee et al. 1999; Wolf et al. 2006) and a predictor of outcomes in constraint-induced movement therapy (Fritz et al. 2005). Despite the clear importance of the problem of digit extension in stroke rehabilitation, we could find no quantitative information about how the ability to extend the fingers and thumb recovers in the first few weeks and months after stroke. This is the time period when the majority of recovery is expected to occur (Duncan et al. 2000; Jorgensen et al. 1995; Nakayama et al. 1994) and the time when the majority of rehabilitation monies are spent (Caro et al. 2000; Spieler et al. 2004).

A limited ability to extend the digits disrupts daily life because people need to be able to adequately and appropriately extend the digits to grasp and release objects when interacting with their environment. Emerging models of computational motor control have aided our understanding of the complexity of grasping (Flanagan et al. 2006; Ghez et al. 1991; Kawato 1999; Shadmehr and Krakauer 2008; Valero-Cuevas 2005). Grasping presents a computationally challenging problem of how to transform visual information about an object’s properties into the appropriate muscle actions to grasp the object (Castiello 2005). This involves planning both arm trajectory and hand shaping during the transport phase and force control during contact with the object (Flanagan et al. 2006; Jeannerod 1984).

We have largely viewed deficits in grasping post stroke as being a motor execution problem, i.e., a reduced ability to activate spinal motoneurons and not a motor planning problem, i.e., a disruption in the visuomotor transformation process (Lang et al. 2005, 2006). Recent data showing that grasping with the affected, contralesional hand can be improved after first grasping with the unaffected, ipsilesional hand suggest, however, that critical processes during the visuomotor transformation may be affected, even in those people with subcortical strokes (Raghavan et al. 2006a). Here, we test our previous hypothesis, by investigating how performance on the finger extension task relates to performance on a grasping task after stroke. The first task does not require a visuomotor transformation and deficits during this task have been shown to be due to a motor execution problem (Kamper et al. 2006). In contrast, on the second task, the nervous system must transform visual information about the object into motor command such that specific muscles of the arm and hand are activated at the appropriate times and to the appropriate magnitude.

The purposes of this study were to 1) quantify the amount of finger and thumb extension recovery that occurs between 3 and 13 wk after stroke, 2) examine relationships between the ability to extend the digits and the ability to grasp at each time point, and 3) determine whether the initial ability and the subsequent finger and thumb extension recovery could predict grasping performance at 13 wk post stroke. When studying digit extension, subjects were instructed to extend all five digits together
to avoid potentially confounding deficits in fractionation of digit movements (Lang and Schieber 2003, 2004; Raghavan et al. 2006b). When studying grasping, the task required only a minimal amount of hand translation (≤100 mm) to minimize any potentially confounding effects of reaching deficits on grasping performance (Lang et al. 2005). Analysis of grasping performance was restricted to hand shaping during the transport phase because this characterizes the planning associated with the required visuomotor transformations.

METHODS

Subjects

Twenty-four subjects with hemiparesis due to stroke participated in this study. Subjects were recruited from the Cognitive Rehabilitation Research Group Stroke Registry based on the presence of hemiparesis. Subjects were included if they 1) had a recent diagnosis of ischemic or hemorrhagic stroke by a stroke neurologist, 2) had CT or MRI imaging data consistent with clinical presentation, 3) had persistent hemiparesis as indicated by a score of 1 to 4 on the Motor Arm item of the National Institutes of Health Stroke Scale (NIHSS), 4) had evidence of preserved cognition as indicated by a score of 0 or 1 on the Consciousness and Communication item of the NIHSS, and 5) had the ability to follow 2-step commands. Patients were excluded from the study if they 1) had orthopedic or other medical conditions that limited the affected upper extremity prior to the stroke, 2) had a prior history of hemiparesis or stroke, 3) had hemispatial neglect as evidenced by a score of 2 on the Extinction and Inattention item of the NIHSS, 4) had severe aphasia as evidenced by a score of 2 or 3 on the Language item of the NIHSS, 5) had hemianopsia as evidenced by a score of 2 or 3 on the Visual item of the NIHSS, or if 6) subjects were unable to give informed consent. Characteristics of the group are provided in Table 1. This study was approved by the Washington University Human Research Protection Office and all subjects provided informed consent prior to participation.

All of our subjects participated in standard stroke rehabilitation, typically a 2- to 3-wk stay in an inpatient rehabilitation hospital, followed by outpatient therapy on discharge, between the two time points. Both inpatient and outpatient rehabilitation at our facilities are typical in their daily life. For both digit extension and palmar grasping, subjects were instructed to move at a self-selected pace, as would be typical in daily life. They were instructed to grasp a cylindrical target object (long axis = 110 mm; diameter = 38 mm) where the long axis was parallel to the ground. This object and orientation were chosen because the required grasp replicates the natural action of grasping a handle. The object was positioned above mid thigh on the affected side such that minimal hand translation (≤100 mm) was required to complete the grasp. Subjects were instructed to move at a self-selected pace, as would be typical in their daily life. For both digit extension and palmar grasping, three trials of each movement were recorded. Due to upgrades in the data collection system during the study, data were collected at either 60 or 100 Hz and stored off-line for subsequent analyses.

Additional testing

Additional clinical tests were conducted at each visit to provide a more thorough description of the sample (Table 1). Maximum grip

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mean (SD)</th>
<th>Range/Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grip strength (% of unaffected hand)</td>
<td>32 (29)</td>
<td>44 (32)</td>
</tr>
<tr>
<td>Index finger joint position sense</td>
<td>19 normal</td>
<td>21 normal</td>
</tr>
<tr>
<td>5 impaired</td>
<td>3 impaired</td>
<td></td>
</tr>
<tr>
<td>Modified Ashworth Scale</td>
<td>0.3 (0.7)</td>
<td>0.7 (1.0)</td>
</tr>
<tr>
<td>Fingers</td>
<td>1.1 (1.3)</td>
<td></td>
</tr>
<tr>
<td>Wrist</td>
<td>44 (32)</td>
<td></td>
</tr>
<tr>
<td>Stroke Impact Scale</td>
<td>17 (25)</td>
<td></td>
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</tbody>
</table>

* In all, 18 subjects had a definitive acute lesion as seen on clinical CT or MR scan. Six subjects were without a definitive acute lesion. These 6 subjects had clinical CT scans upon admission to the acute neurology service only, making it unlikely that an acute ischemic lesion could have been detected. b For multiple lesions, diameter of largest lesion reported. Scale range is 0–4, where 0 is the normal tone. c Scale range is 0–100, where 100 is the normal hand function.
strength was measured with a dynamometer during a five-finger grip of the affected and unaffected sides (Schmidt and Toews 1970). Values were measured in kilograms and expressed as a percentage of the unaffected side. In healthy individuals, the percentage of strength between the two sides of the body is typically between 85 and 115% (Mathiowetz et al. 1985). Joint position sense was evaluated on both sides at the index finger using standard clinical technique where normal equals correct on at least three of five trials. Spasticity was evaluated on the affected side using the Modified Ashworth Scale at the fingers and the wrist. Scale scores on this measure range from 0 to 4, where 0 equals normal tone. Self-perceived hand function was evaluated using the Stroke Impact Scale Hand Function subscale (Duncan et al. 1999, 2001). For this subscale, subjects answer questions about their ability to use their affected hand in five daily tasks. The score for this scale ranges from 0 to 100, where 100 indicates normal hand function.

**Analyses**

Kinematic data were low-pass filtered at 6 Hz using a second-order Butterworth filter. Motion Monitor software (Innovative Sports Training Chicago, IL) was used to extract position, velocity, and angle data from the sensor data using standard rigid body methodology (Wu et al. 2005). Custom-written software in MATLAB (The MathWorks, Natick, MA) was used to do subsequent analyses. For each trial of the digit extension task, the variables of interest were the excursion of each finger, i.e., the range through which the thumb, index, middle, ring, and little fingers could be extended. Finger and thumb extension excursions were calculated as the difference between the start position and the maxima in the digit angle data. Digit angles were Euler angle calculations of the angles between the markers on the fingernails and the marker on the back of the hand and represented the rotation at all three joints of each digit (Lang and Beebe 2007; Theverapperuma et al. 2006). Start positions were not different across the two time points ($P = 0.54$), so a change in extension excursions represented a change in how far the digit could be extended against gravity.

For each trial of the palmar grasping movement, the variables of interest were movement time, peak aperture, peak aperture rate, and aperture path ratio. These variables were chosen to quantify performance because they capture important characteristics needed for hand shaping during grasping. Movement time was the duration from onset of movement to steady grasp of the object. For each trial, movement onset was systematically identified as the time when the three-dimensional resultant velocity of the hand first exceeded 5% of its peak value. Grasp of the object (end of movement) was the time when, with the hand on the object, the thumb–index finger aperture was held at a minimum for $>300$ ms after already attaining its peak value. Peak aperture—or how far the fingers and thumb opened during the movement—was the maximum three-dimensional distance between sensors on the tip of the thumb and the tip of the index finger (Jeannerod 1984). During palmar grasping of a cylindrical object, the opening of the middle, ring, and little fingers paralleled the opening of the index finger, making it possible to quantify aperture using data from the thumb and index fingers only. Peak aperture rate—or how quickly the fingers were opened/closed—was the maximum absolute aperture rate attained during the movement. Between the first peak of the aperture trace and the end of movement, we determined the path length that the aperture curve actually traveled, as well as the length of an ideal straight-line path (Lang et al. 2005, 2006). Aperture path ratio, a measure of how directly the thumb and index fingers opened and closed, was calculated by dividing the actual aperture path length by the length of the straight line. An aperture path ratio of 1 represents a single, smooth opening and closing of the thumb and index fingers (ideal) and an aperture path ratio of $>1$ represents abnormal opening and closing of the fingers, typically seen when subjects made multiple attempts to open and close the fingers on the target. Poorer grasp performance would be indicated by slower movement times, smaller peak apertures, smaller peak aperture rates, and/or higher aperture path ratios.

**Statistical analyses**

Variables were calculated separately for each trial and then the average of the three trials for each subject was used to represent the digit extension excursion or grasping performance variable for that individual. SPSS version 13 was used for all statistical analyses and the criterion for statistical significance was set at $P < 0.05$. Distributions of variables were examined for normality using the Kolmogorov—
Smirnov test. Aperture path ratio values were not normally distributed and were transformed for further statistical analyses. Values were transformed using the percentile ranks and all subsequent analyses with this variable were done with the transformed data.

Changes over time in the ability to extend the digits were evaluated using a repeated-measures ANOVA with two within-subject factors: fingers and time. Relationships between the ability to extend the five digits at each time point were evaluated using Pearson Product Moment correlations. Changes over time in the ability to grasp were evaluated using paired t-test.

Relationships between the ability to extend the digits and the ability to grasp were evaluated within each time point using Pearson Product Moment correlations. Stepwise linear regression was used to determine the most parsimonious model of how the initial ability to extend the digits (at 3 wk) and the recovery in the ability to extend the digits (change from 3 to 13 wk) could predict grasping performance at 13 wk post stroke. Separate models were generated with each of the grasping performance variables at the 13-wk time point used as dependent variables. The independent variables were the finger and thumb excursion variables at the 3-wk time point and the changes in finger and thumb excursion from the 3- to the 13-wk time points. Additional models were examined to evaluate whether the clinical variables of grip strength, joint position sense, and/or spasticity made any further predictive contributions to grasp performance.

RESULTS

Twenty-four subjects with acute hemiparesis were included in this study (Table 1). Average age was 55 yr old and the sample was 62% male. Average time since stroke was 19 days to the 3-wk visit and 98 days to the 13-wk visit. A radiologist inspected clinical CT or MR images for each subject and determined that 1) lesion location within the motor system was highly variable; this is consistent with our recruitment based on the presence of hemiparesis versus recruitment based on lesion location, 2) lesions varied in size, and 3) all but one lesion was ischemic. Most subjects were affected on their right side (88%), but this equated to only 50% of the sample being affected on their dominant side. Average grip strength in the affected hand was 10.5 to only 50% of the sample being affected on their dominant side.

Five subjects were unable to initiate a grasp at the 3-wk time point (open squares). Group averages for finger and thumb extension excursions (Fig. 3) were limited to 22 and 44° across the five digits at the 3-wk time point (filled squares). At the 13-wk time point (open squares), average finger and thumb extension excursions had increased <20° at each digit (averages: thumb change = 19°, index change = 15°, middle change = 14°, ring change = 12°, little change = 15°). A repeated-measures ANOVA found a main effect of time (P = 0.0002, F = 14.0, df = 1), indicating that finger and thumb extension excursion improved between 3 and 13 wk post stroke. A main effect of digit was also found (F = 0.006, F = 3.74, df = 4), such that the index, middle, and ring fingers had larger excursions than those of the thumb and little finger (post hoc t-test, P values <0.005). No interactions between time and digit were found (P = 0.986, F = 0.1, df = 4), indicating that recovery of extension was similar across the five digits. Finger and thumb extension excursions were correlated across the five digits within each time point, where the range of correlation coefficients was r = 0.73–0.91 at the 3-wk time point and r = 0.59–0.93 at the 13-wk time point (all P values <0.05).

Grasp performance and changes in grasp performance

Five subjects were unable to initiate a grasp at the 3-wk time point and are therefore not included in the quantification and
analyses of grasp performance. Of these five, three subjects regained some ability to initiate a grasp at the 13-wk time point and two did not.

For the remaining 19 subjects, the ability to grasp a cylindrical object post stroke was examined with kinematic analyses. Figure 2B shows aperture traces during grasping from a single subject (same subject as in Fig. 2A) at both time points. At the 3-wk time point (solid line), lifting the hand against gravity caused the digits to close a little at first (from time 0 to ~800 ms). This was followed by an initial opening of the digits, another closing, and opening again, and then eventually closing on the object around 2,300 ms (marked by the arrow). At the 13-wk time point (dashed line), the initial digit opening occurred much sooner. A smaller adjustment of the aperture followed with quicker closing on the object around 1,300 ms (arrow). In this example, digit opening and digit closing during grasping are better at the 13-wk time point than that at the 3-wk time point, although it remains distinctly different from direct opening and direct closing of the digits during grasping in healthy, neurologically intact subjects (e.g., see Fig. 3A of Lang et al. 2006).

These features of grasp performance were quantified in each subject using four variables: movement time, peak aperture, peak aperture rate, and aperture path ratio (Fig. 4). Movement time (Fig. 4A), a measure of self-selected time to complete the grasp, was well within the broad range of normal when compared with previously published values in a similar reach-to-grasp task (Lang et al. 2005, 2006). Movement time did not improve from the 3- to the 13-wk time point ($P = 0.86$). This was not unexpected given that subjects were instructed to move at their self-selected speed and were not instructed to move as fast as possible. Peak aperture (Fig. 4B), a measure of how wide the fingers and thumb were opened during the grasp, was 79 mm initially and increased by 20 mm from the 3- to the 13-wk time point ($P = 0.001$). As a reference, neurologically intact control subjects of similar age have an average peak aperture of 125 mm on this task (Lang et al. 2006). Peak aperture rate (Fig. 4C), a measure of how quickly the fingers and thumb opened during the grasp, increased by 175 mm/s from the 3- to the 13-wk time points ($P = 0.02$). Aperture path ratio (Fig. 4D), a measure of how smoothly/efficiently the fingers and thumb opened and closed on the object, was poor initially (normal values for healthy controls are 1.0; see METHODS). Aperture path ratio did not improve from the 3- to the 13-wk time point ($P = 0.51$). Thus at the 13-wk time point, the largest deficit in grasp performance was seen in the aperture path ratio, which remained on average 2.5-fold greater than normal.
Relationships between finger extension and grasp performance

The five people who could not initiate grasping at the 3-wk time point all had $<10^\circ$ of extension excursion at each digit. The two people who did not regain any ability to grasp by the 13-wk time point continued to have $<10^\circ$ of extension excursion at each digit. Correlation coefficients were used to examine relationships between digit extension excursion and grasp performance within each time point in the other 19 subjects (Table 2, top). Based on our sample size, correlations $\geq 0.44$ were statistically significant at the $P = 0.05$ level (Cohen and Cohen 1983). Finger and thumb extension excursions had minimal relationships with movement time at 3 and 13 wk post stroke. Generally, finger and thumb extension excursions had the strongest relationships with peak aperture at either time point. At the 13-wk time point, finger and thumb extension excursions became more strongly related to aperture path ratio across the index, middle, ring, and little fingers. Extension excursion of the thumb was related to only one of four grasping variables at the two time points.

The relationship between the ability to extend the digits early after stroke, the recovery of digit extension, and the ability to grasp later after stroke was examined with regression models, using data from the same 19 subjects. Linear regression models using stepwise entry of independent variables (3-wk digit extension excursions and change in digit extension excursions from the 3- to 13-wk time points) were generated to find the most parsimonious predictors of eventual grasp performance at the 13-wk time point. Three models were generated, using peak aperture, peak aperture rate, and aperture path ratios as the dependent variables (Table 2, bottom). Movement time was not evaluated as an independent variable because there were no significant zero-order, bivariate correlations between it and digit extension. The overall amount of variance in eventual grasp performance that could be predicted by the ability to extend the digits ranged from 55 to 89% ($P$ values $<0.05$) across the models. Each model had only two predictors that entered. The first predictor was either index finger or middle finger extension excursion at the 3-wk time point, whereas the second predictor was always the change in index finger excursion from the 3- to 13-wk time point. These models suggest that the eventual ability to grasp after stroke is strongly related to how far the digits can be actively extended against gravity. Across the five digits, extension excursion values were highly intercorrelated ($r = 0.73–0.91$). Similarly, recovery of extension excursion was not different across the five fingers (ANOVA, nonsignificant finger $\times$ time interaction effect). These findings imply that the index and middle finger values were only slightly better predictors than the values for the other digits.

Additional exploratory regression models were generated to determine whether other variables might be further predictive of grasping ability. Grip strength, index finger joint position sense, and the spasticity of the fingers and wrist at the 3-wk time point were tested in the models. Of the new models examined for the four grasping variables, spasticity of the wrist was the only variable to add unique variance to predict aperture path ratio at the 13-wk time point. Having spasticity of the wrist in the model produced a total $R^2$ of 0.73 ($P < 0.05$) when it was paired with middle finger extension excursion as predictors of aperture path ratio.

**DISCUSSION**

We investigated how the ability to extend the fingers and thumb recovers early after stroke and how the ability to extend the fingers and thumb affects grasping performance in a sample of people with relatively pure motor hemiparesis post stroke. Our results show that the amount of digit extension recovery from 3 to 13 wk was $<20^\circ$ on average and was similar across the five digits. Grasp performance was impaired initially, as expected. From 3 to 13 wk post stroke, we found improvements in two of four grasp performance variables. The ability to extend the five digits was moderately related to some of the grasp performance variables within each time point. The initial ability to extend the fingers and thumb and recovery of the ability to extend the fingers and thumb predicted the majority of variance in the grasp performance variables.

**Limited recovery of digit extension**

The inability to extend the digits post stroke is a major problem for functional use of the hand. Previous work in people with chronic hemiparesis post stroke has shown that the primary mechanism underlying the finger extension impairment is the limited ability to activate the finger extensor muscles (Kamper and Rymer 2001; Kamper et al. 2006). Secondary mechanisms include the inability to turn off the
finger flexor muscles and increased resting flexor muscle tone (Kamper et al. 2003). Our findings build on this earlier work by quantifying the amount of finger and thumb extension recovery after stroke. We consider the changes in digit extension from the 3- to the 13-wk time point to be small, particularly since the change was across three joints on each digit (composite rotation) and not at each joint. We would not anticipate much change beyond the 13-wk time point because this is when the most recovery in reach–grasp movements (Lang et al. 2006) and general motor recovery has been shown to occur post stroke (Duncan et al. 2000; Jorgensen et al. 1995; Nakayama et al. 1994).

The amount of change in digit extension during this time period was due to some combination of natural recovery and rehabilitation care. All of our subjects participated in standard stroke rehabilitation between the two time points (see METHODS). Because all subjects received rehabilitation, we were not able to determine whether the changes in digit extension would happen naturally or were a result of rehabilitation. Regardless of the reason, the amount of digit extension recovery was <20° on average, indicating that additional digit extension is still desirable.

Contributions of digit extension ability to grasping

We had previously operated under the hypothesis that deficits in grasping post stroke were due to a motor execution problem, i.e., a reduced ability to activate spinal motoneurons and not a motor planning problem, i.e., a disruption in visuomotor transformation process (Lang et al. 2005, 2006). Here, we found that grasping performance at 13 wk could be largely predicted by the ability to extend the digits at 3 wk and the recovery of digit extension between 3 and 13 wk post stroke ($R^2$ values 0.55–0.89). These data partially support our previous assumption. Our results are the first to show that an important factor underlying hand shaping deficits during grasping may be the inability to extend the digits.

Our sample of people with stroke had relatively pure motor hemiparesis. Like the majority of motor control studies in people post stroke, we excluded people who had significant aphasia, hemi-neglect, and/or cognitive dysfunction. Thus our results may be biased away from those individuals with the most obvious motor planning deficits such as apraxia (Jax et al. 2006). Our subjects were recruited based on the clinical presence of hemiparesis and not based on specific lesion locations. The presence of hemiparesis in each patient suggests that a common element in all of the lesions was some degree of damage to the corticospinal system, i.e., the motor cortical areas and/or the corticospinal tract. The degree of motor deficits post stroke is determined by the amount of damage to the corticospinal system (Pineiro et al. 2000). Our finding that the majority of deficits in grasp performance post stroke may be due to an execution problem is consistent with the understanding that corticospinal system structures constitute the neural substrate for skilled muscle action (Clough et al. 1968; Dum and Strick 1996, 2002; Fetz and Cheney 1980; Palmer and Ashby 1992; Porter and Lemon 1993).

Our previous hypothesis was only partially, not fully, supported. Some portions of variance in grasp performance were unexplained by the ability to extend the digits and the recovery of digit extension in our sample. Other possible explanations for the unexplained variance are activation (execution) deficits in other muscles used during grasping, motor planning deficits (disruption in the visuomotor transformation process), and inadequately measured somatosensory deficits.

The inability to activate and modulate motoneurons post stroke (Dietz et al. 1986; Frontera et al. 1997; Gemperline et al. 1995; Jakobsson et al. 1992; Rosenfalck and Andreassen 1980; Young and Mayer 1982) affects all muscles, not just the finger extensors (Bard and Hirschberg 1965; Beebe and Lang 2008, 2009). Thus other muscles used in grasping may also not have been sufficiently activated or selectively activated (Dewald et al. 1995; Lang and Schieber 2004) during grasping. The relationship between the ability to extend the digits and the ability to grasp is probably not strictly linear. Figure 5 shows our revised working hypothesis of how these two motor actions may be related. Without some baseline level of finger extensor activation, there is no ability to initiate a grasp, as demonstrated by our five subjects who had <10° of digit extension and no ability to grasp at the first time point. When the baseline is exceeded, increasing levels of digit extensor activation result in increasing grasp performance, as demonstrated by the portions of grasping variance explained in our regression models. At some upper threshold, additional digit extensor activation likely no longer leads to improved grasping. This upper threshold may be a moving target that is a function of the size and shape of the object to be grasped and the size of the hand.

We speculate that it is above this upper threshold—when there is sufficient activation of hand and finger muscles—that abnormal grasping performance may be due to deficits in motor planning and/or somatosensation (Blennerhassett et al. 2007; Hemsdorfer et al. 2003; Nowak et al. 2003; Raghavan et al. 2006a). Raghavan and colleagues found that people with stroke could improve force production during grasping with the affected hand if they had previously grasped the object with the unaffected hand. Their sample of 8 subjects with well-defined subcortical lesions was more chronic and more mildly affected (on average) than our sample of 24 subjects. Their finding indicates that some key parameters regarding the appropriate scaling of grasp forces must have transferred to the affected hand from the unaffected hand. Their study examined only...
force production (kinetics) while the hand was already on the target, whereas our study examined only the kinematics as the subject grasped the target. With respect to somatosensation, the appropriate scaling of grasp forces post stroke was somewhat related ($r = 0.34, R^2 = 0.12$) to the ability to discriminate differences in surface friction but not related to the ability to discriminate differences in weight (Blennnerhassett et al. 2007). Interestingly, this sample had mild motor deficits post stroke as well. Looking across data from these studies and ours, it is possible that deficits in motor planning and somatosensation contribute most to grasping control in those with more mild motor deficits post stroke and contribute less when motor deficits are greater or more varied. To gain a better understanding of how motor planning, somatosensation, and motor execution deficits contribute together to grasping deficits, future studies need to: 1) examine both the kinematics and kinetics of grasping during the transport, grasp, and release phases; 2) use more sensitive laboratory measures of somatosensation; and 3) have well-developed probes to determine which planning aspects of grasping are impaired and in which subjects.

Limitations

Four limitations need to be emphasized when interpreting our data. One, we studied a sample of people with relatively pure motor hemiparesis post stroke. This means that our results may not generalize to all individuals with stroke. Second, we studied only the movement to and initial grasp of the object. Clearly, the forces and movements once the object is grasped are equally important to everyday life. Additional studies are under way in our laboratory to capture these parameters. Third, we did not have access to research-level lesion data. We are therefore able to make conclusions based on clinical presentation of motor deficits, but can only speculate about the specific lesion locations that caused the motor deficits. This is similar to what happens in the clinical realm where rehabilitation clinicians providing therapy services assess the clinical deficits but often do not have access to specific lesion information. Finally, we have investigated only one mechanism underlying grasp performance and have not investigated others, such as the accuracy of the visuomotor transformation itself. Further work clearly needs to be done.

Conclusions

Our results show that the amount of recovery in finger and thumb extension was <20° on average for each digit from 3 to 13 wk post stroke. Grasp performance improved on two of four grasping variables. The initial ability to extend the fingers and thumb and the recovery of finger and thumb extension predicted the majority of variance in grasping performance at 13 wk post stroke. In people with relatively pure motor hemiparesis, an important factor underlying deficits in hand shaping during grasping may be the inability to extend the digits.

The majority of poor grasping performance in this common patient population was related to their inability to activate the finger and thumb extensor muscles. Without adequate digit extension, successful grasping of objects will not occur. Although digit extension can be partially accomplished through passive placement or tenodesis (i.e., flexion of the wrist resulting in biomechanical extension of the fingers), our results indicate that functional grasping requires a substantial degree of volitional digit extension control. Further work is needed to determine whether the importance of this supersedes problems that may occur through disturbances in motor planning or somatosensation. Appreciating which aspects of grasp control are disrupted in people with functional grasping deficits provides a more comprehensive view of the neural control of grasping.

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