Reduced Muscle Selectivity During Individuated Finger Movements in Humans After Damage to the Motor Cortex or Corticospinal Tract

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INTRODUCTION

Precise temporal activations of multiple muscles are required to move a finger by itself (Schieber 1995) or to move several fingers together, as in pinching (Maier and Hepp-Reymond 1995). The motor cortex and the corticospinal tract are crucial for the control of such fine finger movements (Porter and Lemon 1993). The convergence and divergence of motor cortical cells onto the spinal motor- and interneurons affords the ability to selectively activate groups of muscles to perform an enormous repertoire of hand and finger movements.

In monkeys with lesions to the motor cortex or corticospinal tract, muscle activation patterns for the hand and forearm muscles have been studied only occasionally (Hepp-Reymond and Wiesedanger 1972; Hoffman and Strick 1995). Lesions of the motor cortex resulted in an inability to selectively activate arm muscles, such that muscle activity was disordered by abnormal timing of the agonist and synergist muscles, and by the failure to deactivate antagonist muscles (Hoffman and Strick 1995). Although the effect of central lesions on control of more proximal arm muscles has been studied in humans (see DISCUSSION), studies of muscle activation in forearm and hand muscles in people with damage relatively restricted to the motor cortex or corticospinal tract have not been performed.

To better understand the effect of lesions of the corticospinal system, we have recently begun quantitative investigations of finger control in people with damage relatively restricted to the motor cortex or corticospinal tract by studying people with pure motor hemiparesis (Lang and Schieber 2003). Pure motor hemiparesis is a relatively homogeneous clinical syndrome characterized by paresis on one side of the body without sensory, cognitive, or language disturbance (Fisher 1979, 1982; Fisher and Curry 1964). This syndrome most frequently results from relatively small ischemic lesions affecting the corticospinal tract unilaterally in the basis pontis or in the posterior limb of the internal capsule.

The purpose of the current study was to investigate how damage to the motor cortex or corticospinal tract affected the selective activation of finger muscles. Our previous study of finger movements in people with pure motor hemiparesis found differential impairments in finger independence during individuated flexion/extension movements (Lang and Schieber 2003). Because flexion/extension movements are produced primarily by contractions of the multitendoned, extrinsic finger muscles (e.g., flexor digitorum profundus, flexor digitorum superficialis, extensor digitorum communis) and because the degree of neuromuscular compartmentalization of these muscles is unclear at present (Keen 2002; Kilbreath and Gandevia 1994; Kilbreath et al. 2002; Reilly and Schieber 2002, 2003; Segal et al. 2002), it is difficult to use electromyographic studies of these muscles to examine how damage to the motor cortex or corticospinal tract may have altered their selective activation. We chose therefore to examine selectivity of muscle activation during individuated abduction/adduction finger movements in the plane of the palm, during which we could study activation patterns of the single-tendoned, intrinsic finger muscles, each of which acts on only one finger. We first tested whether individuation of abduction/adduction movements was affected to the same degree as individuation of flexion/extension movements. We then studied the activation of 3 intrinsic finger muscles: the abductor pollicis brevis (APB), the first dorsal interosseus (FDI), and the abductor digiti quinti (ADQ),...
each of which acts as an agonist for only one individuated abduction/adduction movements and might therefore be expected to be active in a relatively selective manner. We hypothesized that people with pure motor hemiparesis would not activate finger muscles as selectively as normal subjects during an individuated abduction/adduction finger movement task.

METHODS

Subjects

Seven subjects with pure motor hemiparesis (age range 50–76 yr, see Table 1) and 8 neurologically intact, control subjects (age range 19–44 yr) participated in this study. The study protocol was approved by the Research Subjects Review Board of the University of Rochester Medical Center, Rochester, NY. Informed consent was obtained from all subjects before participation.

Figure 1 shows the magnetic resonance image (MRI) that best illustrates the lesion that produced pure motor hemiparesis in each subject. The lesions were located in the precentral gyrus (2 subjects), in the corona radiata (1 subject), in the internal capsule (4 subjects), or in the basis pontis (1 subject). The MRI for H-06 showed signal changes consistent with old, asymptomatic lacunes in both the left and right putamina in addition to the internal capsule lesion responsible for the hemiparesis. Another subject, H-07, had bilateral internal capsule lesions that occurred 17 mo apart. The lesion on the left side was in the middle portion of the posterior limb of the internal capsule. The lesion on the right side was in the genu of the internal capsule. The MRI for H-09 showed signal changes consistent with an old lesion in the anterior frontal lobe in the right hemisphere, in addition to the precentral gyrus lesion in the left hemisphere responsible for the right-sided paresis.

Hemiparetic subjects underwent a clinical neurological examination to rule out sensory impairments and involvement of other motor system structures. Potential subjects with sensory or cognitive loss or movement disorders suggesting involvement of other motor structures were excluded from participation. None of the affected hands had joint contractions that limited their ability to perform the tasks. At the time of testing, the hemiparetic subjects reported substantial recovery as compared with the severity of their initial symptoms. Mild residual paresis persisted in most subjects, as indicated in Table 1.

Functionally, all hemiparetic subjects could walk short distances with an assistive device and could use a precision grip to hold a pen with the affected hand. For 6 of 8 hands evaluated, hand function was impaired as measured by the Jebsen Test of Hand Function (Jebesen et al. 1969), a standardized clinical test measuring timed performance on: 1) writing a sentence, 2) turning index cards, 3) picking up small objects, 4) using a spoon, 5) stacking checkers, 6) lifting light food cans, and 7) lifting heavy food cans. All of the hemiparetic subjects could complete all the items on the Jebsen test. The mean Jebsen Z score was 1.0 ± 0.3 (mean ± SE) for the control group, 1.7 ± 0.7 for the unaffected hands of the hemiparetic group, and 8.1 ± 3.3 for the affected hands of the hemiparetic group.

In addition, hemiparetic subjects were evaluated with the upper extremity Fugl-Meyer scale (Fugl-Meyer et al. 1975). The Fugl-Meyer is a clinical rating scale that assesses voluntary motor control using test items derived from Brunnstrom’s stages of motor recovery after stroke (Brunnstrom 1970). Possible scores range from 0 to 66, where a score of 66 means there was no observable deficit. The mean Fugl-Meyer score for the affected group was 55 ± 4. Individual Fugl-Meyer scores are provided in Table 1. Arm function as measured by the Fugl-Meyer test was highly correlated with hand function as measured by the Jebsen Test (Pearson correlation = −0.979, P < 0.0001). For subsequent analyses examining relationships between function and finger independence, we used the Jebsen Test because it focuses on the typical use of the hand.

Experimental procedure

Subjects were studied performing individuated flexion/extension finger movements and individuated abduction/adduction finger movements. During the individuated abduction/adduction movements, surface electromyography (EMG) was recorded from 3 intrinsic finger muscles. If possible, subjects with pure motor hemiparesis were studied on both the affected side, contralateral to lesion, and on the unaffected side, ipsilateral to lesion. Two hemiparetic subjects were not studied on the unaffected side, H-06 and H-09, because of MRI evidence of old lesions in the opposite hemisphere. For subject H-07 (bilateral internal capsule lesions), both sides were studied and both sets of data were included in the affected group. Control subjects were tested on one side only. In sum, we evaluated 8 affected hands (5 right, 3 left), 4 unaffected hands (2 right, 2 left), and 8 control hands (4 right, 4 left). All of our control subjects and 6 of 7 hemiparetic subjects reported being right-handed. Given that the ability to individuate finger movements or finger forces is not different in the dominant versus nondominant hand (Hagar-Ross and Schieber 2000; Reilly and Hammond 2000), data from right and left hands were pooled for all analyses.

Table 1. Characteristics of subjects with pure motor hemiparesis

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age</th>
<th>Gender</th>
<th>Lesion Location</th>
<th>Time Since Stroke</th>
<th>Initial Hemiparesis in:</th>
<th>Residual Hemiparesis in:</th>
<th>Jebsen Test Z Score</th>
<th>Fugl-Meyer Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>H-01</td>
<td>71</td>
<td>M</td>
<td>L precentral gyrus</td>
<td>42 mo</td>
<td>f, a, h</td>
<td>h</td>
<td>0.3 ± 0.4</td>
<td>64</td>
</tr>
<tr>
<td>H-05</td>
<td>71</td>
<td>M</td>
<td>R basis pontis</td>
<td>27 mo</td>
<td>f,a,h,l</td>
<td>a,h</td>
<td>3.2 ± 1.0</td>
<td>58</td>
</tr>
<tr>
<td>H-06</td>
<td>65</td>
<td>M</td>
<td>L internal capsule, posterior limb</td>
<td>31 mo</td>
<td>f,a,h,l</td>
<td>a,h,l</td>
<td>20.4 ± 6.4</td>
<td>45</td>
</tr>
<tr>
<td>H-07L</td>
<td>50</td>
<td>F</td>
<td>L internal capsule, posterior limb</td>
<td>8 mo</td>
<td>f,a,h,l</td>
<td>a,h,l</td>
<td>24.6 ± 7.0</td>
<td>35</td>
</tr>
<tr>
<td>H-07R</td>
<td>50</td>
<td>F</td>
<td>R internal capsule, posterior limb</td>
<td>25 mo</td>
<td>f,a,h,l</td>
<td>a,h</td>
<td>3.4 ± 1.0</td>
<td>NE*</td>
</tr>
<tr>
<td>H-08</td>
<td>50</td>
<td>M</td>
<td>L internal capsule, posterior limb</td>
<td>3 mo</td>
<td>f,a,h,l</td>
<td>h</td>
<td>1.5 ± 0.5</td>
<td>65</td>
</tr>
<tr>
<td>H-09</td>
<td>72</td>
<td>M</td>
<td>L precentral gyrus</td>
<td>2 mo</td>
<td>f, a, h</td>
<td>a</td>
<td>7.3 ± 2.0</td>
<td>56</td>
</tr>
<tr>
<td>H-10</td>
<td>76</td>
<td>M</td>
<td>R corona radiata descending fibers</td>
<td>2 mo</td>
<td>f,a,h,l</td>
<td>h</td>
<td>3.7 ± 0.9</td>
<td>62</td>
</tr>
</tbody>
</table>

L, left; R, right; f, face; a, arm; h, hand; l, leg; NE, not evaluated. *Subjects are identified with the same numbers as in Lang and Schieber (2003). See Fig. 1 for magnetic resonance images for each subject. Z score compared to age-, gender-, and hand dominant–appropriate means on the Jebsen Test of Hand Function; higher scores reflect increased time to complete test items; see text for details. Fugl-Meyer Upper Extremity scale; normal arm function = 66. *Extends into white matter beneath. †Extends up into pontine tegmentum. ‡Extends into white matter above and into the posterior of the putamen. H-07R was not evaluated with the Fugl-Meyer because of arm and shoulder pain.
Sensors except the little Glove sensor output was linearly related to joint position for all glove sensors for each tested hand (Hager-Ross and Schieber 2000). Measurements were obtained in standard positions to calibrate the fingers. Before testing, glove sensor output and goniometric joint right- or left-handed instrumented glove (CyberGlove, Virtual Tech-nologies, Palo Alto, CA) was used to measure joint angles of the fingers. Before testing, glove sensor output and goniometric joint measurements were obtained in standard positions to calibrate the glove sensors for each tested hand (Hager-Ross and Schieber 2000). Glove sensor output was linearly related to joint position for all sensors except the little finger distal interphalangeal (DIP) sensor.

Surface EMG was recorded from 3 intrinsic finger muscles, APB, FDI, and ADQ. These 3 muscles were chosen because each one acts as an agonist for only one of the individuated abduction/adduction movements studied here: APB is active during abduction of the thumb in the plane of the palm (although APB’s prime action is abduction of the thumb in the plane orthogonal to the palm, i.e., anatomical thumb abduction), FDI is active during abduction of the index finger, and ADQ is active during abduction of the little finger. Electrodes, 10 mm in diameter (VerMed, Bellows Falls, VT), were placed in a bipolar configuration (7–12 mm apart depending on the size of the subject’s hand) over each muscle, such that signal from the target muscle was optimized. Because of the proximity of the thenar muscles to one another, electrodes on APB may have also transduced signals from opponens pollicis and flexor pollicis brevis. If these muscles were active during the present movements, however, we would expect them to be as selective for thumb movements as APB. Likewise, electrodes on ADQ may also have transduced signals from opponens digiti minimi and flexor digiti minimi because of the close proximity of the hypothenar muscles to one another. Again, however, we would expect that the other hypothenar muscles would be as selective for little finger movements as ADQ. EMG activity was amplified by a factor of 5,000–20,000 to produce a signal that fell within a ±5-V range. Spike2 software and a Micro 1401 interface (Cambridge Electronic Design, Cambridge, UK) were used to collect glove data and surface EMG simultaneously. Each glove sensor was sampled at the maximum rate of 78 Hz and EMG was sampled at 1,000 Hz.

When testing flexion/extension movements, the forearm was positioned in neutral pronation/supination and the wrist was positioned in an approximately 15° extension and stabilized with a vacuum cast (Vershock, Summons Preston, Bolingbrook, IL). The thumbs and fingers were free to move without contacting the vacuum cast or the surface of the table. When testing abduction/adduction movements, the forearm was stabilized on the table in full pronation and the wrist was neutral, such that the palm of the hand rested on the table. During abduction/adduction movements, the smooth material of the glove minimized friction between the fingers and the table surface.

Flexion/extension movements were tested in one block of trials and abduction/adduction movements were tested in another block. The order of the 2 blocks was randomly varied across subjects. For each block, subjects were asked to make cyclic movements of one finger at a time. We instructed them to move one finger, through a “comfortable range of motion,” and to “keep the other fingers still.” For flexion/extension movements, no specific instructions were provided to flex and extend particular joints on each finger, although most subjects tended to primarily flex and extend the metacarpophalangeal (MCP) and proximal interphalangeal (PIP) joints. For abduction/adduction movements, subjects were instructed to keep their fingers loosely spread apart so they could abduct/adduct one finger without the instructed movement being obstructed by other fingers. The movements were paced by a metronome (40 bpm, 0.67 Hz) to maintain the same frequency of movement for each instructed finger during each block. Subjects in all groups were able to maintain this frequency for the duration of a trial (as confirmed by measurements of movement frequency). During movement of an instructed finger, the noninstructed fingers generally remained in a relaxed, flexed posture during flexion/extension movements and in a relaxed, extended, and abducted position during abduction/adduction movements. The hand was in full view of the subject at all times so he/she was able to see the movements of the instructed and noninstructed fingers. Subjects practiced each movement before recording trials to be sure that they understood the task and the instructions. All subjects reported an awareness of movement in the noninstructed fingers when it occurred. After practicing an instructed movement, a 10-s trial was recorded. We recorded 2 consecutive trials for each instructed finger for each block. The order of instructed fingers was held constant across blocks for a given subject, but was varied between subjects.

**Kinematic analysis**

Off-line, glove data were low-pass filtered at 6 Hz. For flexion/extension movements, we used data from 14 of the 22 glove sensors: MCP, PIP, and DIP sensors for the index, middle, and ring fingers (9 sensors); MCP and PIP for the little finger (2 sensors); and MCP, PIP,
and opposition sensors for the thumb (3 sensors). The thumb opposition sensor best captured the movement about the carpometacarpal (CMC) joint in the flexion/extension plane of the thumb phalanges. For abduction/adduction movements, we used data from 4 sensors that detected abduction of the thumb at the CMC joint (1 sensor) and relative abduction between the index–middle, middle–ring, and ring–little finger pairs at the MCP joints (3 sensors). Pilot testing showed that, after manufacturer-suggested corrections, data from the 3 relative abduction sensors could be used to accurately measure abduction/adduction movements of the index, ring, and little fingers but not of the middle finger. Therefore all subsequent analyses of both flexion/extension and abduction/adduction movements were done without using data from instructed middle finger movements or from movement of the middle finger during uninstructed movements.

Sensor data were transformed into joint angles, using the offset and gain values for each sensor for each subject as derived from calibration procedures described above. For flexion/extension movements, we defined finger joint angles such that 0° at each joint was a straight finger, positive numbers indicated flexion (curled finger), and negative numbers indicated hyperextension. For abduction/adduction movements, we defined joint angles such the 0° was complete abduction (fingers held closely together) and positive numbers indicated abduction (fingers spread apart). Finger segment lengths (measured from hand axis and joint angles) were used to calculate fingertip position in the flexion/extension plane (x and y axes) and the abduction/adduction plane of each finger (z and y axes). Fingertip position was defined such that the origin of the axes was at the center of the MCP joint for each of the 4 fingers and at the CMC joint for the thumb.

To determine the relative motion of instructed versus uninstructed fingers, we used the normalized path distance traveled by the fingertip (Lang and Schieber 2003) because path distance was the kinematic measure that best reflected our instructions to the subjects and the subjects’ perception of movement that occurred. We defined the average path distance as the total distance a fingertip traveled during the 10-s trial divided by the number of completed cycles, where one cycle was considered flexion and extension or abduction and adduction of the finger. The average path distance was calculated for the thumb, index, ring, and little fingertips during each 10-s trial. The average path distance values were normalized by dividing the values by the average path distance of that finger when it was the instructed finger. Thus the normalized path distance equals 1 when a finger is the instructed finger, and is usually <1 when it is a noninstructed finger. We then used the normalized path distances to derive individuation indices for each finger to quantify finger independence (Lang and Schieber 2003; Schieber 1991) and thus compare finger independence between flexion/extension and abduction/adduction movements.

The individuation index is a measure of how well a finger is able to move independently (i.e., without the other fingers moving). The individuation index was calculated as 1 minus the average normalized path distances of the noninstructed fingers, or

$$H_i = 1 - \frac{\sum_{j=1}^{n} |D_{ij}| - 1}{n - 1}$$

where $H_i$ is the individuation index of the $i$th finger, $D_{ij}$ is the normalized path distance of the $i$th finger during the $j$th instructed movement, and $n$ is the number of fingers (here $n = 4$). One is subtracted from the sum of the normalized path distances in the numerator and from $n$ in the denominator to remove the normalized path distance of the instructed finger itself. The individuation index will be close to 1 for an ideally individuated movement in which the instructed finger moves with no movement of noninstructed fingers and will be closer to 0 if the more noninstructed finger movement occurs simultaneously with instructed finger movement. Because the individuation index does not depend on the range of motion through which the fingers move, it is a useful means of comparing finger independence between flexion/extension and abduction/adduction movements, 2 types of movements where the available ranges of active motion are quite different.

Individuation indices were calculated separately for each set of instructed movements, where a set equals one trial each of instructed thumb, index, ring, and little finger movement, and the 2 individuation index values were averaged to produce a single value for each instructed finger for each type of movement. We compared individuation indices by first using an overall repeated-measures ANOVA design with one-between-group factor (group: control, affected, unaffected) and 2 within-group factors (finger: thumb, index, ring, little; and movement: flexion/extension, abduction/adduction). All statistical tests were performed using Statistica (Statsoft, Tulsa OK) and significance levels were set at $P < 0.05$. When significant effects were found, $t$-tests with Bonferroni corrections for multiple comparisons were used to identify the specific differences.

Additionally, to test whether finger independence could be explained by differences in range of movement between fingers or groups, we calculated and compared maximum joint excursions for each finger during its instructed movement. Comparisons between the 3 groups and the 4 fingers were performed for each movement using repeated-measures ANOVAs.

**Electromyographic analyses**

Off-line, EMG data were high-pass filtered at 10 Hz to remove movement artifact, rectified, and then low-pass filtered at 30 Hz to produce a smooth signal for subsequent integration. EMG data from each muscle were normalized to the highest maximum value recorded from that muscle within the testing session. To determine the average amount of muscle activity for a given muscle for a given instructed movement, we integrated the EMG data over each 10-s trial and divided this value by the number of movement cycles in that trial. Integrating and dividing by the number of movement cycles allowed us to determine average activation without defining onsets and offsets of EMG activity. Average activation values were then used to determine the selectivity of each muscle.

We quantified the selectivity of each muscle using an index of selective activation (ISA) that was conceptually similar to the individuation index. A muscle that is completely selective will be active for only one individuated movement and will have an index of selective activation equal to 1, whereas a muscle that is completely unselective will be equally active for all 4 of the individuated movements and will have an index of selective activation equal to 0. The index of selective activation for a given muscle was calculated as one minus the mean of the relative activation during the movements when that muscle did not act as the agonist, or

$$ISA = 1 - \frac{1}{n} \sum_{i=1}^{n} A_{ag}$$

where $A_{ag}$ is the average activation during the instructed movement where that muscle was the agonist, and $A_{1,2,3}$ are the average activations during the 3 instructed movements when that muscle was not the agonist (note: the $A_{ag}$ represents the relative activation for a given muscle). For example, for APB, $A_{ag}$ was the average activation during instructed thumb movement, and $A_{1,2,3}$ were the average activation values during instructed index, ring, and little finger movements, respectively. For ADQ, $A_{ag}$ was assigned to whichever value was greater, the average activation during instructed little finger movement or the average activation during instructed ring finger movement. This was done because ADQ was most activate during instructed ring finger movement in about 50% of the control subjects, presumably as a stabilizer of the little finger (see sample EMG traces in Fig. 5A).

Indices of selective activation were calculated separately for the 2 sets of instructed abduction/adduction movements, and the 2 indices were averaged to produce a single value for each muscle. Indices of selective activation were compared using an overall repeated-meas-
RESULTS

We tested 8 affected hands, 4 unaffected hands, and 8 control hands. MRIs (Fig. 1) illustrate the lesions of the motor cortex or corticospinal tract that caused paresis in the affected hands. We first present kinematic results from flexion/extension movements versus abduction/adduction individuated finger movements, followed by electromyographic results from abduction/adduction individuated finger movements.

Independence of flexion/extension versus abduction/adduction finger movements

Fingers of the affected hands of hemiparetic subjects moved less independently than fingers of control hands and of unaffected hands, particularly during abduction/adduction movements. Figure 2 shows plots of fingertip position and the corresponding normalized path distance for a control hand (Fig. 2, A and B, CNT-07) and for the affected hand of a hemiparetic subject (Fig. 2, C and D, H-05) during single trials of instructed index finger movement. The control hand showed little movement of the noninstructed fingers during flexion/extension movements (Fig. 2A) or during abduction/adduction movements (Fig. 2B). In comparison, the affected hand showed slightly more movement of the noninstructed fingers during the flexion/extension trial (Fig. 2C), but substantially more during the abduction/adduction trial (Fig. 2D). This can be seen in both the traces of fingertip position and in the normalized path distance plots.

An ideally independent finger would move without any accompanying motion of the other fingers. We used the individuation index to quantify the degree to which the noninstructed fingers moved when they were supposed to be still (see METHODS). For the control data shown in Fig. 2, the individuation index was 0.93 for instructed flexion/extension of the index finger (Fig. 2A) and 0.94 for instructed abduction/adduction of the index finger (Fig. 2B). For the affected data shown in Fig. 2, however, the individuation index was 0.88 for instructed flexion/extension of the index finger (Fig. 2C) and 0.65 for instructed abduction/adduction of the ring finger.

The control and unaffected groups (Fig. 3, A and B) had a similar degree of finger independence during flexion/extension movements as during abduction/adduction movements. In comparison, the affected group (Fig. 3C) had less independent finger movements, with the greatest impairment generally seen during abduction/adduction movements. Using an overall repeated-measures ANOVA, we found a main effect of group ($P = 0.013$), where the individuation indices for the affected group were lower than the individuation indices for the control and unaffected groups. We found a main effect of finger ($P < 0.001$) and a finger by group interaction ($P = 0.004$), where the individuation indices for the affected ring and little fingers were lower than the individuation indices for the affected thumb and index fingers, and were lower than those for all 4 fingers in the control and unaffected groups. We also found a main effect of movement ($P = 0.018$), where abduction/adduction individuation indices were lower than flexion/extension.

FIG. 2. Single trials of fingertip position and normalized path distance for instructed index finger movement during flexion/extension movements (left) and abduction/adduction movements (right). A and B: control subject, CNT-07. C and D: affected hand of hemiparetic subject, H-05. Fingertip position is shown on the left and normalized path distance is shown on the right for each type of movement. Fingers are color-coded like a rainbow, such that red = thumb, orange = index, green = ring, and blue = little. Note that the fingertip position panels are shown on a larger scale in A and C (flexion/extension) compared with B and D (abduction/adduction), and that the origin of the axes (0, 0) at the metacarpophalangeal (MCP) joint of the fingers and the carpometacarpal (CMC) joint of the thumb is located outside the panels in B and D. Horizontal bars in each plot of normalized path distance are ordered: thumb (top), index, ring, little (bottom).
maximum joint excursion of each instructed finger. During flexion/extension movements, the most excursion typically occurred at the PIP joint of each finger (~80°), followed by the MCP joint (~50°), and then the DIP joint (~20°). The thumb had similar excursions occurring at the MCP and IP joints (~55°), and a smaller excursion occurring at the CMC joint (~30°). These joint excursions were not significantly different across groups (P > 0.05) and were similar to values reported previously (Lang and Schieber 2003; Fig. 6). During abduction/adduction movements, motion occurs at the MCP joint in the index, ring and little fingers, and in the CMC joint of the thumb. Abduction/adduction movements showed the greatest angular range at the thumb (~40°), followed by the index finger (~20°), ring (~18°), and little fingers (~18°). Anatomically, the thumb has about 50° of motion during abduction/adduction in the plane of the palm, twice that of each of the other fingers (Palmer and Eppler 1990). Abduction/adduction joint excursions also were not significantly different between groups (P > 0.05). Thus the range through which fingers joint rotated did not explain the reduced finger independence seen in the affected group.

Muscle selectivity

Muscle activations during individuated abduction/adduction movements were less selective in the affected hands of hemiparetic subjects than in unaffected or control hands. We present individual examples of recordings from FDI, a highly selective muscle in control subjects (Fig. 4) and from ADQ, a muscle that was less selective in control subjects (Fig. 5).

In Fig. 4, the FDI of the control subject (Fig. 4A) was activated only during instructed index finger movement (2nd trace), as expected. In contrast, the FDI of the affected hand (Fig. 4B) was activated during instructed thumb, index, and ring finger movement (1st, 2nd, and 3rd traces). The relative activations of the control FDI during thumb, ring, and little finger movements (Fig. 4C) were much smaller than the relative activations of the affected FDI. In Fig. 5, the ADQ of the control subject (Fig. 5A) was activated phasically during instructed little finger movement (4th trace) as the agonist for abduction, and was activated more tonically during instructed ring finger movement (3rd trace), presumably to stabilize the little finger. The ADQ of the affected hand (Fig. 5B), however, was activated relatively phasically for all 4 instructed movements. The relative activations of the affected ADQ during thumb and index finger movements (Fig. 5D) were larger than the relative activations of the control ADQ (Fig. 5C).

We quantified the selectivity of each muscle using the index of selective activation (see METHODS). The indices of selective activation for the individual subject data shown in Figs. 4 and 5 are shown in Fig. 6, A and B, respectively. Note that for the control subjects, indices of selective activation for ADQ were lower than indices of selective activation for APB and FDI. This was a consistent finding across control subjects and reflects the activation of ADQ during both instructed ring and instructed little finger movements. The hemiparetic subject in Fig. 6A had a Jebsen score within normal limits (Z = 1.5) and more selective muscle activation than the hemiparetic subject in Fig. 6B, who had

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**FIG. 3.** Individuation indices for each finger for the control (A), unaffected (B), and affected (C) groups. Individuation indices are represented with open circles for flexion/extension movements and with filled triangles for abduction/adduction movements. Each data point is the group mean ± SE.

**Range of movement**

To exclude the possibility that range of joint movement affected our measure of finger independence, we compared...
a Jebsen score reflecting marked impairment in hand function ($Z = 7.3$). In both of these subjects, however, indices of selective activation for the affected hands were lower than the indices of selective activation for the control hands for all 3 muscles.

In the affected group as a whole, muscle selectivity was substantially lower than that in the control or unaffected groups (Fig. 6C), indicating that damage to the motor cortex or corticospinal tract results in relatively unselective activation of hand muscles. Using an overall repeated-measures ANOVA, we found a main effect of group ($P < 0.001$), where the indices of selective activation were lower in the affected group than in the control and unaffected groups. Although the control and unaffected group indices of selective activation for ADQ were lower than for APB and FDI, we did not find a significant group by muscle interaction ($P = 0.092$). Likewise, the affected APB had a lower average index of selective activation than that of the affected FDI or ADQ, but this difference did not reach statistical significance.

Relationships between muscle selectivity, finger independence, and hand function

Subjects with greater functional deficits were generally the same subjects that activated their muscles less selectively and moved their fingers less independently. Nonparametric Spearman rank-order correlations were used to look for relationships across all subjects between muscle selectivity (measured by the index of selective activation), finger independence (measured by the individuation index), and hand function (measured by the Jebsen Test of Hand Function). Significant correlations between the index of selective activation for each
muscle and the individuation index for each finger during abduction/adduction movements were found for 10 of 12 possible combinations (3 muscles × 4 fingers), with the r values ranging from 0.455 to 0.655 (different from 0, P < 0.05), suggesting that muscle selectivity and finger independence are moderately related. Correlations coefficients between the index of selective activation for each muscle and the Jebsen Z score were −0.532 for APB (P = 0.028), −0.493 for FDI (P = 0.032), and −0.599 for ADQ (P = 0.007), suggesting that muscle selectively is moderately related to hand function. (Note that the indices of selective activation of the 3 muscles were significantly correlated with each other at the P < 0.05 level.) Last, correlations between the individuation index for each finger and the Jebsen Z score ranged from −0.417 to −0.795 but only 3 of 8 possible combinations (4 fingers × 2 movements) were significantly different from zero, suggesting that finger independence is only loosely related to hand function.

**DISCUSSION**

Motor cortex or corticospinal tract damage in people with pure motor hemiparesis reduced the selectivity of finger muscle activation during individuated abduction/adduction finger movements, resulting in reduced independence of these movements. Abduction/adduction movements showed a trend toward being less independent than flexion/extension movements in the affected hands of hemiparetic subjects. We find it likely that our 3-way ANOVA lacked the power to detect a difference between these 2 types of movements in the affected group because of the relatively small number of patients studied. Additionally, selective activation of finger muscles and finger
produced selectivity of arm muscle activity in people with hemiparesis, although previous reports studied heterogeneous patients with relatively nonspecific lesions, and with a range of motor and nonmotor abnormalities. Hemiparesis consistently results in a diminished capacity to recruit the desired agonist muscles (Gemperline et al. 1995; Hammond et al. 1995), and contraction of agonist muscles frequently is accompanied by co-contraction of antagonist muscles (Bourbonnais et al. 1989; DeWald et al. 1995; Kamper and Rymer 2001; but see Fellows et al. 1994a,b; Gowland et al. 1992). Agonist–antagonist coactivation may be viewed as a reduced ability to selectively activate upper arm and forearm muscles, and thus would be consistent with our findings of reduced selectivity of intrinsic finger muscle activation in subjects with pure motor hemiparesis. The present findings show that loss of selectivity goes beyond movements for which the muscle would be either an agonist or an antagonist. In our subjects with residual hemiparesis, for example, ADQ was activated phasically during movements of the thumb and index finger, fingers on which ADQ exerts no force and hence cannot be considered either an agonist or an antagonist.

Compensation by spared components of the neuromotor system

Although our hemiparetic subjects moved their affected finger joints through the same excursions and at the same frequencies as did control and unaffected subjects, they were not able to activate their finger muscles as selectively. This deficit presumably reflects corticospinal function for which spared components of the neuromotor system could not compensate. We now consider how, after damage to the motor cortex or the corticospinal tract, 1) the spared components of the system might provide compensatory control over the finger muscles, and 2) how this compensatory control might have produced the trend toward different degrees of independence seen during flexion/extension versus abduction/adduction movements.

In the cases where the lesion occurred in the primary motor cortex hand territory (2 cases), both spared primary motor cortex territory and nonprimary cortical motor areas might have provided compensatory, but less-selective control of the hand muscles. Upper extremity territory of the primary motor cortex can reorganize after direct partial lesions (Nudo and Milliken 1996) and after direct partial lesions with subsequent rehabilitation (Nudo et al. 1996). Plastic reorganization of the spared upper extremity territory of the primary motor cortex therefore might have provided some compensatory, though less selective, control of hand muscles in these 2 subjects.

Nonprimary cortical motor areas were spared in our subjects and could also have exerted compensatory control over the hand muscles. Premotor, supplementary, and cingulate motor areas contain representations of the hand and forearm, are interconnected with the primary motor cortex, and send corticofugal projections to subcortical centers, including the spinal cord (for review, see Passingham 1997; Picard and Strick 2001; Rizzolatti et al. 1998). Imaging studies indicate that premotor and supplementary motor areas may be more active during finger movements in hemiparetic subjects than in nor-
nal control subjects (Seitz et al. 1998; Ward et al. 2003; Weiller et al. 1992, 1993). Compensatory control by these areas might have provided less-selective activation of hand muscles compared with the control normally provided by the primary motor cortex.

In cases where the lesion occurred in the corticospinal tract (6 cases), the reduced muscle selectivity may reflect the loss of corticomotoneuronal connections to hand muscles combined with the compensatory capacity of alternative descending pathways. In humans with hemiparesis attributed to corticospinal tract lesions, primary motor cortex activation is preserved and the magnitude of activation is similar to healthy control subjects (Cramer et al. 2002). With damage to the crossed corticospinal tract, the intact and active motor cortex may exert some control over spinal motor neurons by alternate descending pathways.

The uncrossed corticospinal tract, the rubrospinal tract, and the reticulospinal tract all could provide alternate routes for motor cortex output to reach the contralateral spinal cord (Belhaj-Saif and Cheney 2000; Cao et al. 1998; Fisher 1992; Fries et al. 1991, 1993; Kuypers 1993, 1998; Lawrence and Kuypers 1968a,b; Woolsey et al. 1972). The motor cortex sends uncrossed, ipsilateral corticospinal projections to the spinal cord that have a greater influence on motor neurons controlling proximal rather than distal muscles (Colebatch and Gandevia 1989; Kuypers and Brinkman 1970; Nirkko et al. 2001). Because the human rubrospinal tract may not descend all the way through the brachial enlargement of the cervical spinal cord (Nathan and Smith 1982), rubrospinal axons in humans may be more likely to connect with more rostral cervical segments, in which spinal motoneurons innervating proximal muscles are found. The reticulospinal tract appears to descend at least to the thoracic spinal cord in humans (Nathan et al. 1996) and recently has been shown in primates to have relatively direct connections to motoneurons innervating proximal musculature (Davidson and Buford 2002). If these alternate descending pathways provide compensatory control after lesions of the corticospinal tract or motor cortex, this control likely would be mediated by more rostral cervical segments by propriospinal neurons, resulting in less-selective activation of hand muscles, like that we observed in our hemiparetic subjects.

If the above spared components of the nervous system are providing compensatory control over the finger muscles, why did we see this trend toward abduction/adduction individuated movements tending to be more impaired than flexion/extension movements in the affected hands of hemiparetic subjects? The potentially spared primary motor cortex upper extremity territory (as in the 2 hands affected by lesions to the precentral gyrus) and each potential, alternate descending pathway (as in the 6 hands affected by lesions to the corticospinal tract) all have their strongest inputs to the slightly more rostral cervical segments. Because motoneurons innervating the extrinsic finger muscles are found somewhat more rostrally in the brachial enlargement than motoneurons innervating the intrinsic hand muscles (Jenny and Inukai 1983), finger independence during abduction/adduction movements might remain more impaired than finger independence during flexion/extension movements.

Another factor that may contribute to this trend is that spinal motoneurons of the most distal forelimb muscles receive more input from the primary motor cortex, by the corticospinal tract, than do motoneurons of the proximal forelimb muscles (Kuypers 1987; Porter and Lemon 1993). An important proportion of this input makes monosynaptic connections from the primary motor cortex to the motoneurons. In primates, there is strong evidence for a proximal-to-distal gradient in the distribution of corticomotoneuronal inputs to the spinal cord, with the distal motor neuron pools receiving the greatest proportion of these corticomotoneuronal inputs (Clough et al. 1968; Dum and Strick 1996; Fetz and Cheney 1980; McKiernan et al. 1998; Palmer and Ashby 1992; Porter and Lemon 1993; but see Colebatch et al. 1990). The trend in finger independence that we observed during flexion/extension movements versus abduction/adduction movements might reflect the distribution of corticomotoneuronal connections increasing from the extrinsic to the intrinsic finger muscles. Although the lesions in our hemiparetic subjects must have disrupted both corticomotoneuronal and noncorticomotoneuronal axons from the motor cortex, the ability to independently control intrinsic finger muscles may depend on monosynaptic connections to a greater degree than the ability to independently control extrinsic finger muscles. In a similar manner, the fact that a fewer number of muscles can produce abduction/adduction movements compared with flexion/extension movements might contribute to the trend toward a greater deficit in independence during the abduction/adduction movements. For example, only ADQ is available to abduct the little finger, whereas flexor digitorum profundus, flexor digitorum superficialis, and flexor digitii quinti may contribute to flexion of the same finger. After damage to the descending motor pathways, fewer muscle combinations are available for use in recovery of abduction than for flexion movements.

In sum, motor cortex or corticospinal tract damage in people with pure motor hemiparesis reduced the selectivity of finger muscle activation during individuated abduction/adduction finger movements, resulting in reduced independence of these movements. Abduction/adduction movements showed a trend toward being less independent than flexion/extension movements in the affected hands of hemiparetic subjects. These changes in the selectivity of muscle activation and the consequent decrease in individuation of movement were correlated with decreased hand function. Our findings imply that, in humans, spared cerebral motor areas and descending pathways that remain may activate finger muscles, but cannot fully compensate for the highly selective control provided by the primary motor cortex and the crossed corticospinal system.

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