Delays in Grip Initiation and Termination in Persons With Stroke: Effects of Arm Support and Active Muscle Stretch Exercise

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Seo NJ, Rymer WZ, Kamper DG. Delays in grip initiation and termination in persons with stroke: effects of arm support and active muscle stretch exercise. J Neurophysiol 101: 3108–3115, 2009. First published April 8, 2009; doi:10.1152/jn.91108.2008. Stroke survivors’ difficulty in releasing grasped objects may be attributable not only to impaired finger extension but also to delays in terminating activity in the gripping flexor muscles. This study was undertaken 1) to quantify the time needed to initiate and terminate grip muscular activity following stroke and 2) to examine effects of arm support, grip location, and active muscle stretch on the delays recorded in the paretic hand. Delays in initiation and termination of finger flexor muscle activity in response to an auditory stimulus were measured for both paretic and nonparetic hands of ten stroke survivors with chronic hemiparesis and the dominant hand of five neurologically intact subjects. Additionally, the delays for the paretic hand were recorded while an external arm support was used and after 30 min of active muscle stretch. We found that delays in grip initiation and termination were greatest for the paretic hand (1.9 and 5.0 s), followed by the nonparetic hand (0.5 and 1.6 s), and least for the control hand (0.2 and 0.4 s). Arm support reduced delay in grip initiation 37% for the paretic hand. Repeated active muscle stretch resulted in 24% reduced delay in grip initiation and 32% increased delay in grip termination for the paretic hand. Therapies and interventions reducing these delays may improve the ability to grasp and release objects and thus increase functional independence for stroke survivors.

INTRODUCTION

Stroke is a leading cause of long-term disability among American adults (AHA 2003). Stroke survivors often experience chronic motor deficits, especially in the hand, resulting in diminished capacity to manipulate objects with the hand affected by the stroke (Gray et al. 1990; Nakayama et al. 1994a,b; Parker et al. 1986). These deficits may relate not only to proper creation of grasp posture (e.g., hand opening and closing; Cruz et al. 2005; Kamper and Rymer 2001), but also to the timely release of the object being held. In the typical paretic upper extremity posture (i.e., flexed elbow, pronated forearm, flexed wrist), gravity can assist object release despite limited active finger extension, provided that the finger flexor muscles are relaxed. Thus the observed difficulties in letting go of objects may result from sustained finger flexor muscle activity or inability to terminate finger flexor muscle activity in a timely manner. Delays in grip force generation and termination following stroke can result in inefficient grip force scaling during grip-and-lift tasks (Nowak et al. 2003, 2007), deficits in timing, and coordination of movement (Nowak et al. 2007) and thus decreased motor function (Chae et al. 2002, 2006).

Previous studies have described delays in initiating general motor activities: index finger movement (Howes and Boller 1975) and arm movement (Jones et al. 1989) in response to a “go” signal have been shown to be slower for the nonparetic hand of stroke survivors, compared with neurologically intact individuals. Also, initiation of motor activities in the arm (Jones et al. 1989), wrist (Chae et al. 2002), and lower limb (Chae et al. 2006) have been shown to be slower for the paretic side than for the nonparetic side. Delays in terminating general motor activities have been shown only for the paretic wrist (Chae et al. 2002) and lower limb (Chae et al. 2006), compared with the nonparetic side, but not for the hand.

The sources of these delays, however, and the factors that influence them have not been well described. Sustained finger flexor muscle activity after a voluntary contraction following stroke has been only anecdotally described (Kamper and Rymer 2001; Kamper et al. 2003; Kline et al. 2007). The duration of this activity is unclear, despite its significant implication in hand function. Due to the presence of persistent coupling between muscles (the flexion synergy) in the upper extremity (Brunnstrom 1970), difficulties with terminating finger flexor muscle activity may be exacerbated by activation of more proximal arm muscles, such as occurs when supporting the hand in space. Similarly, grasp initiation and termination may also vary with location in workspace. Past work has shown, for example, that stroke survivors have more difficulty reaching away from the body than close to the body (Kamper and Rymer 2001); the proximal muscle activation necessary for a far reach may affect grasp. Furthermore, stretching has often been prescribed for stroke survivors to reduce muscle hypertonicity (Wu et al. 2006). As such, it may influence sustained muscle activation during release.

Accordingly, the aims of this study were: 1) to examine the time course of grasp by comparing the times needed to initiate and terminate a grip activity for stroke survivors (paretic and nonparetic hands) and neurologically intact individuals and 2) to examine the effects of factors such as external arm support, grip location, and active muscle stretch on these delays. We hypothesized that delay in grip termination for the paretic hand would be shorter when the hand is near the torso compared with when the hand is far from the torso and when the arm is supported than when the arm is unsupported. We also hypothesized that the delays in grip termination for the paretic hand would decrease after upper extremity muscle stretch.

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METHODS

Subjects

Ten chronic hemiparetic stroke subjects (≥ 9 mo poststroke) and five neurologically intact (control) subjects participated in this experiment. The inclusion criteria for stroke subjects were 1) the occurrence of a single stroke ≥ 9 mo prior to the study; 2) no cognitive dysfunction that precludes comprehension of experimental tasks; 3) no history or clinical signs of concurrent medical problems; and 4) Stage 2 to 3 on the Chedoke–McMaster stage-of-hand scale (severe hand impairment level) (Gowland et al. 1995). Stroke subjects were composed of seven males and three females with the mean age of 51 ± 10 yr, with ages ranging from 39 to 68 yr (see Table 1). The mean time since the cerebrovascular incident was 6 ± 6 yr. Of these individuals, seven subjects had involvement of the left side of the body and three subjects had involvement of the right side. Three subjects had impairment of the formerly dominant hand and seven subjects had impairment of the nondominant hand. Control subjects were composed of five males with a mean age of 51 ± 4 yr. All subjects signed the informed consent form approved by the Institutional Review Board of Northwestern University prior to testing.

Procedure

Each subject participated in a single-session experiment to assess delays in grip initiation and termination using an electromyographic (EMG) signal. For stroke survivors, both the contralesional (paretic) hand and the ipsilesional (nonparetic) hand were tested. For the control subjects, only the dominant hand was tested.

Subjects were seated with a back support in front of a vertical cylinder suspended from the ceiling by a string. The cylinder was 6.7 cm in diameter and its surface was finished with smooth polyester. Its vertical position was adjusted to the height of the subject’s forearm when the upper arm was vertical by the trunk and the elbow was flexed at 90°. The paretic hand was placed around the cylinder by the experimenter while the stroke subject was at rest. For the nonparetic and control hands, subjects voluntarily placed their hand around the cylinder. Subjects were instructed to relax the fingers before a trial began. No significant change in limb positions was visually observed during a given trial.

A trial consisted of subjects relaxing the fingers for the first 5 s, gripping maximally for 5 s, and then relaxing the hand for 20 s without moving the location of the hand (see Fig. 1). A computer-generated audible tone was played during the gripping period (5 s) to cue subjects when to start and stop grip exertions. Subjects were instructed to grip maximally, as quickly as possible, in response to the start of the tone, and to relax as quickly as possible as soon as the tone ended. Maximum grip exertions were used as opposed to submaximal exertions. This was done to ensure that 1) subjects recruited muscle fibers as quickly as possible (Kroemer and Marras 1980) and 2) EMG levels during grip exertions were statistically distinguishable from the baseline EMG, especially for the paretic hand that had low EMG levels with high variance.

Bipolar active surface electromyogram electrodes (Delsys, Boston, MA) were placed over the muscle bellies on the forearm about halfway between the medial epicondyle of humerus to the styloid process of the ulna targeting flexor digitorum superficialis (FDS) and about the ¼ point from the lateral epicondyle to the styloid process of the ulna targeting extensor digitorum communis (EDC) (Basmajian 1989). The FDS muscle was targeted because this study focused on the initiation and termination of long finger flexor muscle activity in an isometric condition. The FDS muscle has primary involvement in power grip (Kaufmann et al. 2007) and its superficial location allows recording of EMG signals using noninvasive surface electrodes (Perotto and Delagi 1994). The EDC muscle activity was recorded to monitor the antagonist muscle activity. It is possible that the EMG targeting the FDS was contaminated by activities of adjacent forearm muscles such as flexor carpi radialis, flexor carpi ulnaris, and palmaris longus. Likewise, the EMG targeting the EDC could have had cross talk from wrist extensor muscles, especially from the extensor carpi ulnaris. The EMGs were recorded during the entire 30-s trial at 500 Hz using a Delsys amplifier and data acquisition system (NI PCI-MIO-16XE-10 and LabVIEW, National Instruments, Austin, TX). The EMG signals were initially low-pass filtered at 225 Hz. After the experiment, the EMG signals were rectified and low-pass filtered at 10 Hz using MATLAB (The MathWorks, Natick, MA) to create envelopes that clearly displayed the aggregate muscle activity.

Stroke subjects were asked to repeat this grip-and-relax task with the paretic hand for different grip locations, arm support, and stretching conditions. Three grip location/arm support conditions were tested: 1) a comfortable distance from the torso with no arm support, 2) as far from the torso as possible without an external arm support, and 3) as far from the torso as possible with an external arm support. The three conditions, presented to subjects in a random order, were tested again after active repeated muscle stretching (making a total of six conditions). All six conditions were examined for effects in the paretic hand (for the nonparetic hand and control subjects, only the comfortable grip location with no arm support was tested). Three trials were performed for each condition. A break of ≥ 1 min was given between any successive trials.

Both comfortable and far grip locations were self-determined by the stroke subjects for the paretic hand. The far grip location was limited by the paretic arm’s capability to maintain a static posture during a trial. The horizontal distance from the cylinder center in the hand to the mid torso was recorded using a measuring tape. Once the distance for the comfortable grip was determined for the paretic hand, the same distance was used by the nonparetic hand of the stroke subject. Thus within a given stroke subject, the same target (com-

### Table 1. Stroke subject characteristics

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age</th>
<th>Gender</th>
<th>Months After Stroke</th>
<th>Lesioned Hemisphere</th>
<th>Dominant Hand (Before Stroke)</th>
<th>Chedoke Hand Stage</th>
<th>Grip Strength (Paretic/Nonparetic)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>68</td>
<td>F</td>
<td>223</td>
<td>L</td>
<td>R</td>
<td>3</td>
<td>49%</td>
</tr>
<tr>
<td>2</td>
<td>60</td>
<td>M</td>
<td>62</td>
<td>R</td>
<td>R</td>
<td>3</td>
<td>24%</td>
</tr>
<tr>
<td>3</td>
<td>49</td>
<td>M</td>
<td>16</td>
<td>R</td>
<td>L</td>
<td>2</td>
<td>45%</td>
</tr>
<tr>
<td>4</td>
<td>62</td>
<td>M</td>
<td>161</td>
<td>L</td>
<td>L</td>
<td>2</td>
<td>19%</td>
</tr>
<tr>
<td>5</td>
<td>42</td>
<td>M</td>
<td>118</td>
<td>R</td>
<td>R</td>
<td>3</td>
<td>23%</td>
</tr>
<tr>
<td>6</td>
<td>46</td>
<td>M</td>
<td>45</td>
<td>R</td>
<td>R</td>
<td>3</td>
<td>13%</td>
</tr>
<tr>
<td>7</td>
<td>39</td>
<td>F</td>
<td>23</td>
<td>R</td>
<td>R</td>
<td>3</td>
<td>33%</td>
</tr>
<tr>
<td>8</td>
<td>49</td>
<td>M</td>
<td>30</td>
<td>R</td>
<td>R</td>
<td>3</td>
<td>21%</td>
</tr>
<tr>
<td>9</td>
<td>51</td>
<td>F</td>
<td>47</td>
<td>R</td>
<td>R</td>
<td>3</td>
<td>21%</td>
</tr>
<tr>
<td>10</td>
<td>43</td>
<td>M</td>
<td>15</td>
<td>L</td>
<td>R</td>
<td>3</td>
<td>49%</td>
</tr>
</tbody>
</table>

Grip strength was measured using a Jamar dynamometer.

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fortable grip) distance was used for both the paretic and nonparetic hands. The horizontal distance from the hand to the mid torso was 34 ± 6 cm for the comfortable grip location and 48 ± 6 cm for the far grip location (on average across subjects). The distance for the far grip location was significantly greater than that for the comfortable grip location (paired t-test, \( P < 0.01 \)). The mean comfortable grip distance across all stroke subjects was used for the control subjects (control subjects were tested after all stroke subjects were tested). External support was provided by a gravity-compensating orthosis, the Therapy Assistant Wilmington Robotic Exoskeleton (TA-WREX) (Iwamuro et al. 2008; Rahman et al. 2007). The TA-WREX is a passive orthosis that compensates for the effects of gravity on the arm at any point in the arm workspace.

Stretching consisted of about 30 min of actively performed muscle stretches. Stroke subjects followed a written active muscle stretch protocol with illustrations. The protocol consisted of flexion/extension of the thumb, fingers, wrist, elbow, and shoulder, thumb abduction/adduction, forearm rotation, and shoulder rotation and abduction/adduction across a range of motion. All stretches were performed manually (with no assistive device). Subjects were instructed to use the muscles of the affected side as much as possible and use the nonparetic hand to help as needed. The end position of each stretch was held for \( \approx 5 \) s. Each stretch was repeated 10 times. The subjects were observed during the active stretch to ensure that they correctly followed the instructions and completed all repetitions for each stretch. On completion of the repeated active muscle stretch protocol, the subjects performed the grip-and-relax trials for the differing grip location and arm support conditions with the paretic hand.

### Data analysis

The EMG values for each muscle in each hand were first normalized by the peak value of the processed EMG across all trials for that muscle and hand. Delays in grip initiation and in grip termination were then determined from the forearm flexor EMG: baseline EMG was determined as the lowest value for the EMG averaged across a 0.5-s moving window recorded during the initial resting period at the beginning of each trial prior to the start of the audible tone (referred to as “Baseline” in Fig. 1). The FDS EMG was assumed to have been initiated when all EMG values in a 0.5-s moving window (starting from the onset of the audible tone) surpassed the mean value plus 3SDs of the baseline EMG (Di Fabio 1987). The start of this window was defined as the time of grip initiation. Delay in grip initiation equaled the difference between this time and the beginning of the audible tone (Fig. 1). The FDS EMG was assumed to have terminated when all EMG values in a 0.5-s moving window fell below the mean plus 3SDs of the baseline EMG or its 95% confidence interval overlapped with that of the baseline. The start of this window was used as the time of grip termination. Delay in grip termination equaled the difference between this time and the end of the audible tone (Fig. 1). All analyses were performed using MATLAB.

A repeated-measures MANOVA was first run to determine whether delays in grip initiation and termination varied among the three limb types (paretic, nonparetic, and control hands), as recorded during the sole condition of no intervention and comfortable reach distance. Since the variance in delay was different among the three limb types (with \( P < 0.05 \) in the Levene’s test of equality-of-error variances), the
delay data were transformed into the logarithmic scale (P > 0.05 in the Levene’s test of equality-of-error variances after transformation). The significant limb effect in MANOVA then led to the performance of univariate repeated-measures ANOVA for each of the dependent variables, logarithms of grip initiation, and termination times. Tukey’s post hoc tests were subsequently performed to examine significant differences among the three limb types.

An unbalanced repeated-measures analysis was performed to determine whether delay varied significantly with grip location, arm support, and/or muscle stretch for the paretic hands, for grip initiation, and termination separately. Because differences in the forearm flexor and extensor EMG levels during maximum grip exertion and the forearm extensor EMG levels during grip termination could influence the delay in grip termination, additional analyses were performed. Maximum forearm flexor activation was determined by finding the highest FDS EMG value, averaged across a 0.5-s moving window, during grip exertion. The forearm extensor EMG level was determined as the mean EDC EMG during the period from the end of the audible tone to the termination of the forearm flexor EMG. Unbalanced repeated-measures analysis was performed for the forearm flexor and extensor EMG levels to look for variations across grip location, arm support, or muscle stretch for the paretic hand.

RESULTS

We found that there were systematic differences in delays for grip initiation and termination between the different limb classes studied (paretic, nonparetic, and control; P < 0.05 from MANOVA). Means, ranges, and medians of delays in grip initiation and termination for the stroke subjects’ paretic hand and nonparetic hand and the control subjects’ hands are summarized in Table 2 and Fig. 2. The delays in grip initiation and termination of the paretic hand for each grip location, arm support, and stretch condition are summarized in Table 3. Specific findings for grip initiation and termination are described in the following text. The forearm flexor EMG level during maximum grip exertion and the forearm extensor EMG level during grip termination did not significantly vary with grip location, arm support, or stretch conditions (P > 0.05 for all).

Grip initiation

Delay in grip initiation was significantly different among the three limb types (P < 0.05), when tested at the comfortable grip location with no arm support. Post hoc testing revealed that initiation delay was significantly different for each of the three limb types: paretic, nonparetic, and control (P < 0.05). The mean delay in grip initiation was fourfold greater for the paretic hand (1.922 ± 0.915 s) than that for the nonparetic hand (0.474 ± 0.147 s) and was twofold greater for the stroke subjects’ nonparetic hand than that for the control subjects’ hand (0.237 ± 0.110 s) (see Fig. 2). Delay in grip initiation varied significantly for the repeated active muscle stretch (P < 0.05), whereas grip location and arm support did not significantly affect delay in grip initiation (P > 0.05). After the repeated active muscle stretch protocol, mean delay in grip initiation decreased 32% on average (Δ 0.490 s).

Grip termination

Delay in grip termination was significantly different among the three limb types (P < 0.05). Post hoc tests revealed that the termination delays were distinct for the paretic, nonparetic, and control limbs (P < 0.05). The mean delay in grip termination was approximately threefold greater for the paretic hand (5.046 ± 4.334 s) than for the nonparetic hand (1.574 ± 1.552 s) and fourfold greater for the stroke subjects’ nonparetic hand than for the control subjects’ hand (0.414 ± 0.176 s) for the comfortable grip location with no arm support only (see Fig. 2).

Delay in grip termination varied significantly for the arm support (P < 0.05) and the repeated active muscle stretch (P < 0.05), but not for the grip location (P > 0.05). Use of the arm support TA-WREX resulted in, on average, a 37% reduction in the delay in grip termination (Δ 2.596 s; compared with the comfortable and far grip location conditions without the arm support; stretch conditions pooled). After the repeated active muscle stretch protocol, mean delay in grip termination increased 24% (Δ 1.346 s; grip location and arm support conditions pooled).

**TABLE 2.** Mean ± SD, range (min, max), and median of delays in grip initiation and termination for the control, nonparetic, and paretic hands for the comfortable grip location with no arm support before active repeated muscle stretch

<table>
<thead>
<tr>
<th>Hand</th>
<th>Delay in Initiation, s</th>
<th>Delay in Termination, s</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean ± SD</td>
<td>Min</td>
</tr>
<tr>
<td>Control</td>
<td>0.237 ± 0.110</td>
<td>0.147</td>
</tr>
<tr>
<td>Nonparetic</td>
<td>0.474 ± 0.147</td>
<td>0.232</td>
</tr>
<tr>
<td>Paretic</td>
<td>1.922 ± 0.915</td>
<td>0.783</td>
</tr>
</tbody>
</table>

n = 10 for stroke; n = 5 for control.
As expected from earlier reports, delays in grip initiation and grip termination were significantly greater for the paretic hand than for the nonparetic hand (see Table 2, Fig. 2). Delays for the nonparetic hand were also greater than those for the control hand. The delays in grip termination for the paretic hand could be reduced by using the arm support. Repeated active muscle stretch decreased delay in grip initiation, but it increased delay in grip termination for the paretic hand. Possible mechanisms underlying these results are discussed in the following sections.

Comparison with previous studies

Our study used the same stimulus and testing conditions described in previous studies (Chae et al. 2002, 2006) that measured delays not only in motor initiation but also in termination in the wrist and tibialis anterior (the start and end of an auditory stimulus as cues to initiate and terminate isometric maximal muscular activities, respectively). Mean delays in initiation and termination of isometric maximal paretic muscle activity recorded for the hand with no interventions (comfortable location without arm support before active stretch) in the present study (1.922 and 5.046 s, respectively) are much longer than those reported for wrist and lower limb muscles in stroke survivors (0.4 and 1.6 s, respectively, for wrist muscles; 0.3 and 1.0 s, respectively, for the tibialis anterior) (Chae et al. 2002, 2006). The longer delays seen in this study may be ascribed to the fact that subjects with severe hand impairment participated in the present study, whereas subjects who participated in the earlier wrist study (Chae et al. 2002) were mildly impaired, and subjects with a wide range of motor impairment (from severe to mild) took part in the tibialis anterior study (Chae et al. 2006). The greater delays seen in the present study could also be due to the greater motor impairment typically seen after stroke for the distal upper extremities compared with proximal or lower limbs (Gray et al. 1990; Nakayama et al. 1994a,b; Parker et al. 1986). The greater delay in termination than in initiation for the control subjects in this study (Δ 177 ms) may be due to slower detection of the end of an auditory sensation as opposed to the onset of an auditory sensation (Emmerich et al. 1976; Goldstone 1968; Kemp and Irwin 1979).

Disturbed interhemispheric inhibition following stroke

In addition to slowed sensory information processing following stroke, altered interhemispheric inhibition could potentiate the delay in grip termination of the paretic hand and the delay in grip initiation of the paretic hand, as evidenced by studies using transcranial magnetic stimulation: termination of voluntary hand muscle contraction was found to be preceded by increased excitability of motor cortical inhibitory circuits (Buccolieri et al. 2004). Decreased inhibition from the lesioned to the nonlesioned hemisphere (Butefisch et al. 2008) and increased excitatory activity with reduced inhibitory activity in the nonlesioned motor cortex (Butefisch et al. 2003; Shimizu et al. 2002) could have furthered the delay in grip termination for the nonparetic hand and thus may explain differences regarding delay in grip termination between the nonparetic hand and the control hand (Δ 0.923 s) after accounting for the slowed sensory information processing for stroke. Likewise, increased inhibition from the nonlesioned hemisphere to the lesioned hemisphere (Duque et al. 2005) may explain greater delay in grip initiation for the paretic hand compared with the nonparetic hand (Δ 1.448 s), as previously demonstrated (Murase et al. 2004).

Intrinsic motoneuron properties and the effect of repeated active muscle stretch

In addition to increased inhibition from the nonlesioned to the lesioned hemisphere, fewer functioning motor units (McComas et al. 1973) and slower motor unit firing rates (Gemperline et al. 1995; McComas et al. 1973; Rosenfalck and Andreassen 1980) for the paretic limb may also be responsible for slowed bilateral sensory information processing following stroke

Greater delay in initiation for the nonparetic hand compared with the control hand (Δ 0.237 s) was observed. These delays are in concert with previous studies where simple reaction time was found to be greater for the nonparetic hand than that for a control hand (Howes and Boller 1975; Jones et al. 1989). The increase in delay may be attributable to slower auditory information processing for stroke survivors compared with control subjects because sensory information processing is lateralized on both hemispheres for both visual (Jones et al. 1989) and auditory (Howes and Boller 1975) information. Bilateral activation of sensory cortex following auditory and visual cues in intact humans has been reported (Brouwer et al. 2005; Catalan et al. 1998; Sadato et al. 1996). Therefore disruption of bilateral brain activity following unilateral stroke may have contributed to delays in sensory information processing and subsequent delays in muscle activation and termination of the nonparetic hand.

TABLE 3. Mean ± SD, range (min, max), and median of delays in grip initiation and termination for the paretic hand for each test condition (two grip locations, two arm support conditions, before vs. after an active repeated muscle stretch protocol)

<table>
<thead>
<tr>
<th>Hand</th>
<th>Stretch</th>
<th>Grip Location</th>
<th>Support</th>
<th>Delay in Initiation, s</th>
<th>Delay in Termination, s</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Mean ± SD</td>
<td>Min</td>
</tr>
<tr>
<td>Paretic</td>
<td>Before</td>
<td>Comfortable</td>
<td>No support</td>
<td>1.922 ± 0.915</td>
<td>0.783</td>
</tr>
<tr>
<td></td>
<td>Before</td>
<td>Far</td>
<td>No support</td>
<td>1.209 ± 0.316</td>
<td>0.783</td>
</tr>
<tr>
<td></td>
<td>Before</td>
<td>Far</td>
<td>Arm orthosis</td>
<td>1.491 ± 0.854</td>
<td>0.506</td>
</tr>
<tr>
<td></td>
<td>After</td>
<td>Comfortable</td>
<td>No support</td>
<td>1.018 ± 0.708</td>
<td>0.326</td>
</tr>
<tr>
<td></td>
<td>After</td>
<td>Far</td>
<td>No support</td>
<td>1.080 ± 0.685</td>
<td>0.325</td>
</tr>
<tr>
<td></td>
<td>After</td>
<td>Far</td>
<td>Arm orthosis</td>
<td>1.056 ± 0.749</td>
<td>0.340</td>
</tr>
</tbody>
</table>

Data are from 10 stroke subjects.
for the greater delay in grip initiation for the paretic hand compared with the nonparetic hand. Delay in grip termination for the paretic hand, however, may be related largely to the plateau behavior of spastic motoneurons in which they remain firing even after a synaptic input is removed (Gorassini et al. 2002a; Heckmann et al. 2005). Plateau potentials could also contribute to reduction of motor unit recruitment threshold following voluntary muscle contractions as shown in humans (Gorassini et al. 2002b). The plateau response has been shown to become larger and more prolonged after repeated muscle stretch and voluntary muscle activity in decerebrate cats (Bennett et al. 1998). The characteristics of these intrinsic motoneuron properties coincide with the decreased delay in grip initiation and increased delay in grip termination seen after repeated active muscle stretch in the present study (Table 3). Enhanced plateau behavior has been shown following administration of monoamines including serotonin (Hornby et al. 2002) along with increase in the resting potential of motoneurons (Wang and Dun 1990) in animal models. Thus sustained finger flexor muscle activity may be related to increased influence of monoaminergic brain stem pathways following stroke.

Flexion synergy and the effect of arm support and grip location

Data support the hypothesis that delay in grip termination decreases with use of an external arm support (Table 3). The arm support has been shown to reduce shoulder and elbow muscle activities (Iwamuro et al. 2008; Prange et al. 2009). The decreased delay in grip termination seen with use of the arm support may have coincided with a reduction in flexion synergy, in which active shoulder abduction produces involuntary flexion at the elbow and hand in stroke survivors (Brunnstrom 1970; Dewald et al. 1995, 1999). Indeed, support of the arm has been shown to lead to improved elbow extension and reaching (Beer et al. 2004, 2007), accompanied by a reduction in involuntary elbow flexion (Iwamuro et al. 2008). This flexion synergy may result from reduced cortical inhibition to the reticulospinal tract (Matsuyama et al. 2004) and subsequent facilitation of the reticulospinal pathway following stroke (Kamper et al. 2003; Kline et al. 2007) because the reticulospinal pathway favors flexion in the upper extremity (Davidson and Buford 2004; Mori 1987; Schepens and Drew 2004; Werner et al. 1997).

In contrast, delay in grip termination was not significantly different between the comfortable grip location and the far grip location. It is possible that the far grip location triggered two mechanisms that counteract each other. First, as initially anticipated, increased proximal arm muscle activity for the far grip location would contribute to increased delays via the flexion synergy (Brunnstrom 1970; Dewald et al. 1995, 1999). Second, changes of the upper arm posture for the far grip location (e.g., shoulder flexion/abduction, elbow extension) and subsequent changes in sensory feedback from the proximal arm muscles may have shifted the balance between descending inputs from the vestibulospinal and the reticulospinal systems toward the vestibulospinal system, resulting in reduced excitability of finger flexor muscles in stroke survivors, as evidenced by others (Ellis et al. 2007; Hoffmann 2008). For example, reduction of stretch reflexes of the finger flexors has been demonstrated for flexed shoulder and extended elbow posture compared with neutral shoulder and flexed elbow posture (Hoffmann 2008). These two mechanisms may have canceled each other with respect to the excitability of the finger flexor muscles, resulting in little change in delays.

The torso posture was not constrained in the present study. Thus for the far cylinder location, subjects could have flexed the torso to move the paretic hand ahead rather than extending the affected elbow, as previously reported in reaching (Roby-Brami et al. 2003). Using this compensatory behavior, the subjects may have been able to maintain the similar level of shoulder and arm muscle activities for the two cylinder locations, thereby mitigating the effect of the different grip locations.

Implication in hand function

Mean delay in grip termination for the paretic hand of 5.046 s (for the comfortable grip location) is significantly longer than the relaxation times for the control (0.414 s) or the nonparetic hands (1.574 s). This sustained grip activity may induce serious impairment in hand function during object manipulation. For example, when stroke survivors attempt to relax the hand and let go of a grasped object, sustained finger flexor muscle activity may prevent the object from being released. This may lead to further frustration and disuse of the arm. It should be noted, though, that the delays were recorded for maximal grip exertions in this study. It is possible that for submaximal grip exertions, which are often used in daily activities, delay in grip initiation could be longer and delay in grip termination may be shorter than those reported in the present study because a smaller number of motor units are recruited in submaximal grip.

This study demonstrated that subjects could instantly decrease delay in grip termination by using the arm orthosis, TA-WREX (Table 3; Δ 2.596 s). It conforms to the previous report that stroke survivors could achieve increased hand function by using the TA-WREX (Rahman et al. 2007). Because the basic role of the arm orthosis is to support the weight of the arm, it is conceivable that simply supporting the affected arm’s elbow using the stronger hand or against a table may help shorten the delay in grip termination for the paretic hand, especially when one cannot let go of grasped objects in the hand.

Repeated muscle stretch has previously been shown to improve hand function (Carey 1990). Increased maximum voluntary muscle strength (Selles et al. 2005), reduced spastic muscle tone (Nuyens et al. 2002; Schmit et al. 2000), and decreased delay in grip initiation after stretch (Table 3; Δ 0.490 s) after repeated muscle stretch could contribute to increased hand function. Increased delay in grip termination after 30-min repeated active muscle stretch (Table 3; Δ 1.346 s), however, could adversely affect hand function. Thus the long-term effect of active muscle stretch and exercises on delay in grip termination needs to be investigated. Note that an active muscle stretch protocol was used in this study. Different results may be obtained if passive muscle stretch was used because passive stretch alone may have limited influence on plateau response (Bennett et al. 1998). Last, pharmacological intervention that downgrades the influence of the monoaminergic pathways, the
plateau behavior, or the reticulospinal pathway may help mitigate stroke symptoms and decrease delays in grip termination.

Conclusions

This work examined delay in finger flexor muscle contraction and sustained finger flexor muscle activity after grip following stroke. Slowed bilateral sensory information processing, disturbed intercortical inhibition, and the increased influence of the brain stem pathway and the subsequent change in intrinsic motoneuron properties following stroke may be responsible for the delays in grip initiation and termination for both the paretic and nonparetic hands.

Sustained finger flexor muscle activity (threefold longer than that for the nonparetic hand) can impose serious difficulty in stroke survivors’ ability to let go of a grasped object. Supporting the weight of the arm can be effective in shortening the sustained finger flexor muscle activity for the paretic hand. Repeated active muscle stretch can shorten delay in grip initiation but lengthen delay in grip termination immediately after the stretch, whereas its long-term effect on delays is unknown. Therapies and interventions that reduce the delays in initiating and terminating finger flexor muscle activity may improve an ability to grasp and release objects and thus increase functional independence for stroke survivors.

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