Motor unit rate coding is severely impaired during forceful and fast muscular contractions in individuals post stroke

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Chou LW, Palmer JA, Binder-Macleod S, Knight CA. Motor unit rate coding is severely impaired during forceful and fast muscular contractions in individuals post stroke. J Neurophysiol 109: 2947–2954, 2013. First published April 3, 2013; doi:10.1152/jn.00615.2012.—Information regarding how motor units are controlled to produce forces in individuals with stroke and the mechanisms behind muscle weakness and movement slowness can potentially inform rehabilitation strategies. The purpose of this study was to describe the rate coding mechanism in individuals poststroke during both constant (n = 8) and rapid (n = 4) force production tasks. Isometric ankle dorsiflexion force, motor unit action potentials, and surface electromyography were recorded from the paretic and nonparetic tibialis anterior. In the paretic limb, strength was 38% less and the rate of force development was 63% slower. Linear regression was used to describe and compare the relationships between motor unit and electromyogram (EMG) measures and force. During constant force contractions up to 80% maximal voluntary contraction (MVC), rate coding was compressed and discharge rates were lower in the paretic limb. During rapid muscle contractions up to 90% MVC, the first interspike interval was prolonged and the rate of EMG rise was less in the paretic limb. Future rehabilitation strategies for individuals with stroke could focus on regaining these specific aspects of motor unit rate coding and neuromuscular activation.

motor control; CVA; muscle weakness and slowness

STROKE IS THE NUMBER ONE CAUSE of adult disability worldwide (Dobkin 2005). Stroke-induced damage to motor areas in the brain causes physical weakness and slowness, spasticity, and impaired coordination (Chae et al. 2002; Garland et al. 2009; McCrea et al. 2003; Mottram et al. 2010). Weakness affects the ability to perform routine daily tasks such as grasping an object (Chae et al. 2002) or walking (Gerrits et al. 2009; Kim and Eng 2003). Impaired performance of rapid contractions in stroke is a less studied motor deficit (Canning et al. 1999; Clark et al. 2006). The inability to produce quick movements likely limits the ability to function independently by impairing the ability to react rapidly to alterations in postural stability. This may potentially lead to an increased risk of falls (Moroz et al. 2004; Salter et al. 2007). To date, limited research has investigated these impairments at the level of the motor unit.

During voluntary contractions, the recruitment of motor units and the modulation of their discharge rates (rate coding) are the primary central nervous system mechanisms used to control muscular force (Freund 1983; Person and Kudina 1972). These motor unit control mechanisms are determined by descending commands from the brain, spinal level influences, and intrinsic motoneuron properties, each of which may be directly or indirectly affected by stroke. In addition to cortical neuron cell death, a cerebral vascular accident can cause secondary degeneration of the corticospinal tract (Kinoshita et al. 2002), and spinal motor unit loss can become evident as early as 2 wk following an upper motor neuron lesion (Hara et al. 2004). Furthermore, the symptom of spasticity appears to be due to the abnormal occurrence of low-level synaptic input to spinal motoneurons (Mottram et al. 2010).

A decrease in the estimated number of motor units in upper extremity muscles of the paretic limb has been observed compared with the nonparetic limb and controls (Arasaki et al. 2006; Li et al. 2011). There is also evidence of increases in the electrophysiological size of the motor unit in the paretic limb, perhaps due to reinnervation (Kallenberg and Hermens 2011; Li et al. 2011) and with moderate correlations to the Fugl-Myer clinical test (Kallenberg and Hermens 2011).

In the biceps brachii of the paretic limb, the modulation of motor unit firing rates is impaired and lower motor unit firing rates were observed in 3/6 subjects (Gemperline et al. 1995). In tibialis anterior, low-threshold paretic motor units produced firing rates within the lower end of the normal range, and high-threshold units discharged below their normal range or were not recruited (Frontera et al. 1997). Surface-based motor unit recording methods in the first dorsal interosseous muscle also support compression of the rate coding mechanism in stroke (Suresh et al. 2011).

To our knowledge, no previous studies have investigated deficiencies in the rate coding mechanism during rapid muscle contractions of the lower extremity in patients with stroke. In these types of contractions, motor units in healthy young and older adults typically discharge at greater rates than during ramp contractions to maximal force (Klass et al. 2008; van Cutsem et al. 1998). For example, in tibialis anterior, the median initial discharge rates were 72 and 59 pulses per second (pps) for young and elderly adults, respectively (Klass et al. 2008). It has also been reported that during contractions in tibialis anterior, approximately three times more motor units are recruited to produce a quick force pulse compared with a slow ramp contraction with the same amplitude (Desmedt and Godaux 1977).

Considering the variety of possible stroke-related changes that affect input to the spinal motor neuron pool as well as those that affect the output from motor neuron to the muscle...
fibers, the aim of the present study was to describe the motor unit rate coding mechanism in patients with stroke during both forceful and fast isometric muscular contractions. In addition to motor unit recordings, surface electromyograms (EMG) provide gross measures of muscle activation. It was hypothesized that 1) motor unit discharge rates would be lower and the range of motor unit discharge modulation would be less in stroke, and 2) surface EMG would demonstrate reduced neural activation of muscle. A more complete understanding of impaired motor unit rate coding in stroke could elucidate the motor consequences of stroke and inform us of rehabilitation strategies intended to improve strength and speed of movement.

METHODS

Subjects. Nine hemiplegic stroke survivors [1 female; 62 yr old (SD 10.8); 3.9 yr poststroke (SD 3.3)] participated in this study. The inclusion criteria were chronic stroke involving cerebral cortical regions, >6 mo poststroke, ability to produce ankle dorsiflexion on the paretic side, and no history of other neurological disorders. The exclusion criteria were diabetes, dementia (Mini-Mental State Exam score <22), and an inability to understand instructions or communicate with investigators. The subjects signed informed consent documents, which were approved by the University of Delaware Human Subjects Review Board.

Clinical testing was conducted during a separate test session. Self-selected gait speed was determined by a 6-m walk test (Evans et al. 1997), and the average of three trials was taken. Timed-up-and-go (TUG) tests (Flansbjer et al. 2005) were also performed, and the average of three trials was taken. All clinical testing was performed by a licensed physical therapist.

Fig. 1. Top: example recording of a stepwise constant force isometric contraction in which the subject maintained forces approximating 20, 40, and 60% maximal voluntary contraction (%MVC) in the paretic limb. From top to bottom are isometric force, the surface electromyogram (EMG), and 3 channels of motor unit recordings (MU Ch1–Ch3). Fluctuations in force were more common in the paretic limb due to impaired control. For analysis of firing rate and root-mean-square EMG (rmsEMG), data were selected from the most stable regions at each force level. The consequence of electrode instability and the strength of the multichannel approach are evident as the amplitude of the motor unit action potentials changes over time. Although difficult to see among the originally recruited motor unit (due to the greater spike density), a new motor unit was recruited as force increased from 40 to 60%MVC near 22 s. Bottom: higher resolution excerpt of the same recording near 28 s. In these 3 plots for MU Ch1–Ch3, 2 different motor units are identified (mu1 and mu2). Differences in shape and amplitude characteristics across the 3 channels facilitate accurate spike sorting.

Experimental setup. Subjects were seated comfortably on a chair with the knee flexed at 90° and hip flexed at 110°. Seat belts and hook-and-loop fasteners were used to stabilize the subject’s trunk and thigh. The foot and ankle were stabilized at neutral position on a custom force-measuring device that included a strain gauge force transducer (SM-100; Interface, Scottsdale, AZ). Dorsiflexion forces, EMG, and motor unit action potentials were digitized at 51.2 kHz (PowerDAQ II; United Electronic Industries, Canton, MA). Data acquisition and biofeedback were controlled using DasyLab Software (Measurement Computing, Norton, MA).

Maximal voluntary contraction (MVC) forces in ankle dorsiflexion were determined as the greatest force from three maximal effort trials. Subjects were instructed to increase force from rest to their maximum for a 3-s period and received verbal encouragement and real-time visual feedback of their force on a computer monitor. After the MVC force was determined, biofeedback of force amplitude was then prescribed as a percentage of maximal force (%MVC) from within the same limb. Force was displayed to the subject as a vertical bar graph. All subsequent analyses of force were normalized to the ipsilateral MVC.

Surface EMG were recorded from the tibialis anterior muscle using disposable Ag-AgCl pregelled electrodes (1-cm diameter, 5-cm spacing, A10033; Vermed, Bellows Falls, VT). The recording sites were shaved and cleansed with ethyl alcohol. EMG were bandpass filtered from 30 Hz to 3 kHz, and gains were adjusted to optimize resolution within a 10-V range.

Motor unit action potentials were recorded from the tibialis anterior using a custom stainless steel needle electrode containing four 50-μm stainless steel wires epoxied in a 200-μm square array at a side port 3 mm from the tip of the cannula. Differential amplification of three pairs of wires provided three channels of motor unit recordings (Fig. 1). To facilitate spike sorting, each channel presented the same motor unit action potential with different shape and amplitude characteristics due to the
motor unit representation across the full range of force levels. At each recording site the constant force conditions were performed before the rapid force pulses.

**Rapid isometric force pulses.** Subjects were instructed to produce force pulses as quickly as possible to approximate amplitudes of 20, 40, 60, 80, and 100%MVC, with immediate relaxation thereafter (Bellumori et al. 2011; Freund and Budingen 1978). Subjects were told that accuracy was not important. The resulting variability resulted in representative data across the force continuum (see Fig. 4). There were ~3–5 s of rest between rapid force pulses, and each recording of this condition contained between 15 and 20 contractions (Figs. 2 and 3). At least 1 min of rest followed each recording. Based on subjective assessment of the recordings in real time, the investigator sought data from representative force levels while keeping the total number of contractions minimal. After multiple recordings were obtained at a recording site, the electrode was repositioned to sample from other locations.

**Rate of force development scaling factor.** From the rapid contractions, a rate of force development curve was computed as the derivative of the force-time curve over overlapping 0.1-s segments (Fig. 2). By using numerous pulses within each limb, the relationship between the peak rate of force development and peak force was then analyzed with linear regression (see Fig. 5). The slope of this relationship, the rate of force development scaling factor, describes the ability of the subjects to scale their rates of force development with the amplitude

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Fig. 2. Sample results from the paretic limb in both conditions after the spike sorting process. **Top:** instantaneous discharge rate data (circles) are available for 5 motor units, including a newly recruited unit above 90%MVC. The stability of the recording of the first 4 motor units supports the identity of mu6 as a newly recruited unit rather than electrode movement toward an already active unit. Also note the de-recruitment and re-recruitment of mu2 as force decreased near 3–5 s. Within each row of discharge rate data [0–15 pulse/s (pps) scale], firing rates remained low (~8–10 pps) despite large increases in force. **Bottom:** in the rapid isometric force pulse condition, discharge rates of 2 motor units (also within 0–15 pps scale) were surprisingly low, and there was no evidence of upward modulation with increases in pulse amplitude. The new recruitment of mu3 for the >60%MVC pulses demonstrates one means to increase neuromuscular drive in the absence of initial motor unit discharge rates.

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Fig. 3. Example recording of a rapid isometric force pulse from the paretic limb. From top to bottom are the rate of force development (RFD), isometric force pulse, rectified surface EMG, and 3 channels of motor unit action potentials. Stars mark 4 action potentials from the same motor unit, and the waveforms from all 3 channels are overlaid as they would be during manual spike-sorting procedures.
of force pulses across the continuum of submaximal-to-maximal force levels (Freund and Budingen 1978). Note that the rate of force development scaling factor is only a measure of the scaling of quickness with the size of a contraction and not a measure of quickness alone. The rate of force development scaling factor is known to have good day-day reliability (Bellumori et al. 2011) and is determined partly by initial motor unit discharge rates (Klass et al. 2008; van Cutsem et al. 1998).

Neuromuscular activation. Analysis of surface EMG was performed using Spike2 software (version 5.21; CED, Cambridge, UK). For the constant force conditions, the dependent measure of interest was the root-mean-square amplitude of EMG (rmsEMG) at each force level of interest. The rmsEMG was measured at segments of the EMG corresponding to stable force production and where corresponding observations of motor unit discharge rates were available. For the rapid isometric contractions, the dependent measure of interest was the rate of rise in the EMG. Rate of EMG rise was computed as the peak slope (0.1-s window) of the rectified and smoothed (0.05-s moving average window) EMG burst.

Motor unit analysis. The original motor unit recording contains motor unit action potentials (spikes) from one or more motor units within the recording volume of the needle. Spikes were sorted into series belonging to individual motor units using custom automatic and manual template-matching algorithms that use amplitude, shape, and probability criteria (Figs. 1 and 2). For each identified motor unit, time histories of discharge were then used to compute measures of discharge behavior. At each force level in the constant force condition, mean discharge rates were calculated from five consecutive action potentials. For rapid isometric pulses, the first interspike interval (SI1) was selected as the dependent measure of interest.

Statistical analysis. The relationships between EMG or motor unit firing behavior and force level were compared between limbs through comparisons of linear regression equations (see Fig. 4). Before statistical tests were applied to regression models from aggregated data, individual data were visually inspected to ensure that similar force levels were represented in both limbs. Cases were excluded from analysis as necessary to promote balance. In addition to the comparisons of linear regression equations from aggregate data, regression equations were computed for each limb within individuals (see Tables 2 and 3). The intent was to characterize the main effects of stroke and also present individual variability. The threshold for statistical significance is \( \alpha = 0.05 \).

RESULTS

Among nine subjects, one experienced spasms in the lower extremity and testing was terminated before quick force pulses could be recorded. Motor unit data were obtained from both limbs in eight subjects in the constant force condition and four subjects in the rapid force pulses condition. Approximately 16 recordings were obtained from each subject, distributed across two limbs and two conditions as follows. There were 8.4 (SD 1.7) recordings from the nonparetic limb, with an average of 2.3 (SD 0.5) recordings from the rapid force pulse condition. There were 8.1 (SD 3.3) recordings from the paretic limb, with an average of 2.5 (SD 0.6) recordings in the rapid force pulse condition. In the rapid force pulse condition, each subject performed between 37 and 50 isometric force pulses per limb. Maximal effort dorsiflexion contractions were 38% weaker [MVC: paretic, 117 N (SD 69); nonparetic, 197 N (SD 45); \( t = 4.2, P = 0.004 \)] and 63% slower [rate of force development: paretic, 279 N/s (SD 149); nonparetic, 662 N/s (SD 199); \( t = 6.35, P = 0.001 \)] in the paretic limb.

Motor unit sample. In either condition, it was not possible to confirm the uniqueness of motor units across different recordings or different needle locations. Between one and nine unique motor units were identified in each recording, with a median of four unique motor units per recording (e.g., 2 unique units in Fig. 1, 5 in Fig. 2). In the rapid force pulse condition, there was less electrode stability and motor units could not always be reliably tracked across force pulses. Between one and five unique motor units were identified in each force pulse, with a median of two unique motor units (e.g., 2 motor units in Fig. 2).

Constant force condition. The slope of the relationship between rmsEMG and force was significantly greater than zero in both limbs (Fig. 4A, nonparetic: \( t = 7.8, P < 0.001 \); paretic: \( t = 5.6, P < 0.001 \)). The slope of this relationship was less in the paretic limb (\( t = 3.7, P < 0.001 \)), with the greatest differences at higher force levels. The slope of the relationship between motor unit discharge rate and force was significantly greater than zero in both limbs (Fig. 4C, nonparetic: \( t = 14.9, P < 0.001 \); paretic: \( t = 12.5, P < 0.001 \)). Similar to the results of rmsEMG, the slope of this relationship was significantly less in the paretic limb (\( t = 5.6, P < 0.001 \)), with the greatest difference in firing rates at the higher force levels. The predicted firing rate at 80% MVC was 34% less in the paretic limb (11.8 pps) than in the nonparetic limb (17.8 pps). There were few exceptions to the overall regression results (see Table 2).

Rapid force pulses. The positive scaling of rate of force development with peak force was markedly less in paretic than in nonparetic limbs (Fig. 5). Rate of force development scaling factors computed for individual limbs were significantly lower in nonparetic limbs (Fig. 5). In the rapid force pulse condition, there was less strength across the continuum of submaximal-to-maximal force levels (Freund and Budingen 1978). Note that the rate of force development scaling factor is only a measure of the scaling of quickness with the size of a contraction and not a measure of quickness alone. The rate of force development scaling factor is known to have good day-day reliability (Bellumori et al. 2011) and is determined partly by initial motor unit discharge rates (Klass et al. 2008; van Cutsem et al. 1998).

DISCUSSION

Despite the challenges of obtaining motor unit data at numerous force levels from both limbs and in two conditions, only one person withdrew due to discomfort. The effects of
stroke were large, based on a 38% reduction in strength and a 63% reduction in rate of force development in the paretic dorsiflexors. In the constant force condition, differences in firing rates became more pronounced as force levels increased (Fig. 4C). In the rapid force pulse condition, the rates of force development scaled with pulse amplitude, albeit with less gain (Fig. 5). The first interspike interval was substantially prolonged (Fig. 4D) in the paretic limb, and both surface EMG measures (Fig. 4, A and B) demonstrated differences between limbs that agree with the motor unit results.

Although the results offer a logical extension of the current knowledge of motor unit discharge behavior in stroke, there are important limitations to consider. Even though the sample was small and highly variable, based on subject characteristics and function (Table 1), the individual results in Tables 1, 2, and 3 demonstrate that the overall regression models were indeed representative of the majority. For all subjects and in both conditions, the mean firing rates were lower and mean ISI1s were all prolonged in the paretic limb. Another limitation was the inability to track motor units across force levels due to electrode movement at higher forces and in rapid contractions. This limits the interpretation of the results to a description of observed firing rates at different force levels rather than a description of the modulation of firing rates within motor units. Also, despite an efficient experimental design, the test sessions lasted between 60 and 90 min, and the performance of several contractions in both limbs could have caused fatigue.

The main differences between limbs summarized by the regression models were large enough to achieve statistical significance. Nevertheless, interesting variability in the regression plots warrants further consideration. Some individuals presented data indicating high rmsEMG (Fig. 4A) and brief ISI1s (Fig. 4D) in the paretic limb. This is evidence that some aspects of neuromuscular activation can operate within normal ranges despite pronounced functional limitations. One subject (subject 613) had similar dorsiflexion strength in both limbs. Although this could be interpreted as the absence of impairment, this strength ratio is paired with similar firing rates in...
both limbs, and these particular rates were among the lower values observed in the paretic limbs of the entire sample. Thus this subject’s stroke may have affected neuromuscular activation in both of his limbs, and/or his poststroke reductions in physical activity may have resulted in bilateral maladaptation.

In the rapid force pulse condition, there were several ISIs that were <20 ms in duration. The majority (10/12) of these ISIs were from subject 616, but this individual also provided 15 prolonged ISIs (>50 ms) that were representative of the paretic limb.

**Constant force.** In the paretic limb during the constant force condition, motor unit discharge rates were lower and increased less with increases in volitional drive. This is consistent with what others have described as a compressed motor unit rate coding mechanism (Barry et al. 2007; Christie and Kamen 2006; Connelly et al. 1999; Patten and Kamen 2000). In both limbs, the range of motor unit discharge rates was lower than that reported for healthy elderly adults (Kamen et al. 1995). In addition, three of their six subjects showed significant reduction in mean motor unit discharge rates in the paretic muscle compared with the contralateral side. In the present results (Fig. 4C, Table 2), the majority of subjects demonstrated this firing rate reduction. Similarly, Frontera et al. (1997) found that low-threshold paretic motor units discharged within the lower end of their normal range, and high-threshold units discharged below their normal range or were not recruited.

**Rapid isometric force.** During the performance of rapid isometric force pulses, the paretic limb exhibited less of an increase in rates of force development as pulse amplitude increased (Fig. 5). This reduced scaling is in the same direction as that observed in older adults (Klass et al. 2008) and opposite in direction to the effects of power training (van Cutsem et al. 1998). A favorable scaling of rate of force development with peak force is associated with brief interspike intervals at the onset of contraction and a greater rate of EMG rise (van Cutsem et al. 1998). In the present sample, ISI1 was severely prolonged in the paretic limb. Predicted ISIs for 50%MVC pulses were 27 ms in the nonparetic limb and 89 ms in the paretic limb, a 2.3-fold difference. Also in tibialis anterior, Klass et al. (2008) found that ISIs were 13.9 and 17.1 ms for young and older adults, respectively. Therefore, not only is ISI1 prolonged in the nonparetic limb compared with normal patients with stroke. Gemperline et al. (1995) compared motor unit discharge behavior in the paretic and contralateral biceps brachii and found that all six subjects demonstrated impaired discharge rate modulation of motor units in the paretic muscle.

### Table 1. Subject characteristics, functional tests, mean firing rate, and interspike interval results

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age, yr</th>
<th>Time Since Stroke, mo</th>
<th>Ratio of MVC</th>
<th>Ratio of RFD</th>
<th>6-Meter Walk, m/s</th>
<th>Timed Up and Go, s</th>
<th>Firing Rate (NP, P), pps</th>
<th>ISI1 (NP, P), ms</th>
</tr>
</thead>
<tbody>
<tr>
<td>607</td>
<td>74</td>
<td>59</td>
<td>0.92</td>
<td>0.68</td>
<td>ND</td>
<td>12.66</td>
<td>16.8, 12.7</td>
<td>ND</td>
</tr>
<tr>
<td>608</td>
<td>59</td>
<td>11</td>
<td>0.74</td>
<td>0.56</td>
<td>0.83</td>
<td>8.16</td>
<td>14.1, 9.3</td>
<td>ND</td>
</tr>
<tr>
<td>613</td>
<td>78</td>
<td>38</td>
<td>1.03</td>
<td>0.60</td>
<td>1.15</td>
<td>11.15</td>
<td>8.0, 7.2</td>
<td>ND</td>
</tr>
<tr>
<td>616</td>
<td>58</td>
<td>16</td>
<td>0.66</td>
<td>0.34</td>
<td>0.73</td>
<td>14.25</td>
<td>13.3, 8.2</td>
<td>36, 66</td>
</tr>
<tr>
<td>617</td>
<td>70</td>
<td>10</td>
<td>0.33</td>
<td>ND</td>
<td>0.39</td>
<td>31.44</td>
<td>10.1, 6.1</td>
<td>ND</td>
</tr>
<tr>
<td>618</td>
<td>52</td>
<td>122</td>
<td>0.43</td>
<td>0.28</td>
<td>1.29</td>
<td>8.87</td>
<td>12.2, 7.2</td>
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<td>620</td>
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<td>88</td>
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<td>0.24</td>
<td>1.44</td>
<td>10.87</td>
<td>12.8, 7.9</td>
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<td>621</td>
<td>62</td>
<td>77</td>
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<td>0.22</td>
<td>1.31</td>
<td>11.28</td>
<td>17.8, 8.9</td>
<td>27, 70</td>
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<tr>
<td>Average</td>
<td>63.5</td>
<td>52.6</td>
<td>0.61</td>
<td>0.42</td>
<td>1.02</td>
<td>13.59</td>
<td>13.1, 8.4</td>
<td>27, 89</td>
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<td>SD</td>
<td>9.4</td>
<td>41.1</td>
<td>0.28</td>
<td>0.19</td>
<td>0.38</td>
<td>7.47</td>
<td>3.2, 2.0</td>
<td>19, 56</td>
</tr>
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</table>

Ratios of maximal voluntary strength (MVC) and rate of force development (RFD) are expressed for the paretic limb relative to the nonparetic limb. ISI1, first interspike interval; NP, nonparetic limb; P, paretic limb; ND, no data.

### Table 2. Individual linear regression results for the constant force condition

<table>
<thead>
<tr>
<th>Subject</th>
<th>Motor Unit Firing Rate vs. Force</th>
<th>rmsEMG vs. Force</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Nonparetic</td>
<td>Paretic</td>
</tr>
<tr>
<td></td>
<td>R²</td>
<td>Slope</td>
</tr>
<tr>
<td>607</td>
<td>0.057</td>
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<tr>
<td>608</td>
<td>0.070</td>
<td>0.18</td>
</tr>
<tr>
<td>613</td>
<td>0.66</td>
<td>0.14</td>
</tr>
<tr>
<td>616</td>
<td>0.58</td>
<td>0.16</td>
</tr>
<tr>
<td>617</td>
<td>0.64</td>
<td>0.10</td>
</tr>
<tr>
<td>618</td>
<td>0.74</td>
<td>0.21</td>
</tr>
<tr>
<td>620</td>
<td>0.13</td>
<td>0.06</td>
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<tr>
<td>621</td>
<td>0.69</td>
<td>0.17</td>
</tr>
<tr>
<td>Average</td>
<td>0.52</td>
<td>0.13</td>
</tr>
<tr>
<td>SD</td>
<td>0.25</td>
<td>0.06</td>
</tr>
</tbody>
</table>

rmsEMG, root-mean-square amplitude of electromyogram; Y-int, y-intercept.
aging, but the magnitude of the difference between limbs in stroke survivors is immense compared with the difference attributed to the aging process.

Although one could suggest that an increase in initial discharge rate in the nonparetic side might occur due to greater dependence on this limb in activities of daily living, the nonparetic limb cannot be assumed to be unaffected by stroke, and there is also a potential disuse effect due to general reductions in physical activity after stroke. Given the advantage of high motor unit discharge rates on augmenting the rate of force development (Binder-Macleod and Barrish 1992; Christie and Kamen 2006; Garland and Griffin 1999), the inability to discharge closely spaced action potentials at the beginning of quick force pulse is a major factor causing slowed force production and movement. In the absence of high initial discharge rates, motor units delivered more action potentials to the muscle fibers to reach the intended pulse amplitude.

Surface EMG. Although one must consider contributions from motor unit recruitment to the amplitude of the surface EMG, it is not surprising that the EMG-to-force relationships were generally consistent with those between firing rate and force. The present findings add to the existing support for clinical applications of surface EMG where neuromuscular activation is deficient. However, the data also indicate that sensitivity to between-limb differences during constant contractions might be lost at lower force levels (Fig. 4A).

Compared with the effect size (approximated by t-statistics) of the rmsEMG measure in constant force conditions, the difference between limbs in the rate of EMG rise was considerable. Even with the relatively large dispersion in the data from the nonparetic limb (Fig. 4B), there was very little overlap with the rate of EMG rise of the paretic limb. Others have shown that although the rate of EMG rise measure may not differentiate between middle-aged and older healthy adults, it is quite capable of differentiating between older adults with and without mobility limitations (Clark et al. 2011). The present findings provide further support for the utility of the rate of EMG rise measure in rehabilitation. Furthermore, the corresponding deficiencies in ISI1 and rate of EMG rise in the paretic limb further strengthen the link between motor unit behavior and this more accessible, surface-based measure (van Cutsem et al. 1998). Other potential contributors to the reduced rates of force development include loss of large motor units (Lukacs et al. 2008), altered motor unit recruitment (Suresh et al. 2011), and greater muscle cocontraction (Chae et al. 2002).

Interestingly, the mechanical slowing occurs despite a shift toward fast myosin heavy chain isoforms in the paretic limb of stroke survivors (De Deyne et al. 2004).

Mechanisms. The deficient motor unit rate coding mechanism has possible explanations, including decreased descending excitation to the lower motor neuron pool (McComas et al. 1971) and changes in the dynamic sensitivity with which spinal motor neurons enhance neural activation of muscle (Baldissera et al. 1998). It is also possible that commands to motor units from the affected hemisphere are interrupted before reaching the final common pathway, possibly by excessive inhibition from the unaffected hemisphere (Murase et al. 2004; Takeuchi et al. 2005). Larger, higher threshold motor units are more severely affected following stroke (Lukacs et al. 2008), and such units are usually associated with higher firing rates (Kudina and Churikova 1990) and greater dynamic sensitivity (Baldissera et al. 1998). Thus, with increasing time since stroke and reduced levels of physical activity, the motor neuron pool can undergo biased loss of the neurons that contribute most to rapid movement. One must also consider the potential role of muscle atrophy in this particular model. Based on measures from magnetic resonance imaging poststroke, there is relatively little atrophy in the tibialis anterior muscle (4%) compared with that seen in the medial gastrocnemius (38%) (Ramsey et al. 2011).

Conclusion. Together with existing literature, the current results make it clear that substantial reductions in motor unit firing rates accompany the pronounced reductions in physical strength and quickness of the paretic lower extremity of patients with stroke. Although the sample size for the rapid contractions is small, the results show relatively large differences in neural factors that ultimately contribute to speed of movement. Although the current data do not explain the extent to which the reduced discharge rates are caused by deficiencies in descending drive or motor neuron function, rehabilitation researchers should strongly consider the gains that may be possible through strategies to improve neural substrates for quickness. Fortunately, it is already known that maximal motor unit firing rates (Kamen and Knight 2004) and initial interspike intervals (van Cutsem et al. 1998) are both adaptable with standard resistance training practices. The effect of such training on motor unit behavior in patients with stroke remains unknown.

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DISCLOSURES

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AUTHOR CONTRIBUTIONS


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