Relationships between timing of muscle excitation and impaired motor performance during cyclical lower extremity movement in post-stroke hemiplegia

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Summary
We used an ergometer pedalling paradigm to relate abnormalities in the timing of muscle excitation to the impaired ability to perform mechanical work in the plegic lower limbs of persons with hemiplegia. The EMGs of seven leg muscles and pedal forces were measured bilaterally during pedalling for 15 persons with hemiplegia and 12 neurologically intact age-matched control subjects. Subjects were asked to pedal at a moderate workload (135 J) and cadence (40 r.p.m.). While intersubject variability was high, the external mechanical work output of the plegic leg was significantly less (from 79.6% to –28.9% of the work produced by average leg of control subjects) as a result of less positive work and more negative work being done. The timing of EMG in individual plegic limb muscles exhibited two distinct types of abnormalities that were significantly correlated with this lesser work production: prolonged excitation in the vastus medialis and phase-advanced excitation (both early initiation and early termination) in the rectus femoris and semimembranosus. These results suggest that muscles were differently affected depending on their function, external power-producing muscles (e.g., vastus medialis) showing prolonged excitation and muscles that normally maintain crank progression during limb transitions (e.g., semimembranosus) showing phase-advanced excitation.

Keywords: hemiplegia; stroke; EMG; biomechanics; locomotion

Abbreviations: BF = biceps femoris; IEMG = integrated EMG; MG = medial gastrocnemius; RF = rectus femoris; SM = semimembranosus; SO = soleus; TA = tibialis anterior; VM = vastus medialis; \( W_C \) = net external mechanical work done by each limb of control subject; \( W_P \) = net external mechanical work done by the plegic limb

Introduction
One of the problems persons with post-stroke hemiplegia have is an inability to appropriately time the excitation of the muscles of the lower extremity during cyclical locomotor movements, including walking. However, establishing the functional consequences of inappropriate muscle excitation has proved difficult due to the complexity of multijoint movements in general and gait in particular. Knutsson and Richards (1979) were able to describe several types of disturbed motor control in hemiplegic gait through analysis of EMG patterns and kinematic data (joint angles and muscle lengths). However, they did not quantify the EMG abnormalities or the gait deficits. While quantitative indices of EMG abnormalities have been calculated in persons with symmetrical spasticity in pedalling (Benecke et al., 1983), gait (Conrad et al., 1985) and partial body weight-supported gait (Fung and Barbeau, 1989; Dietz et al., 1995), these studies did not quantify motor performance. In fact, no studies have quantitatively related the extent of EMG timing abnormalities to the level of motor performance (e.g., lack of weight-bearing on plegic side) in a functional lower extremity movement in subjects with hemiplegia.

It has been difficult to relate measures of EMG timing abnormality to measures of impaired performance in gait for several reasons, including the biomechanical complexity of gait, the possible superposition of ‘protective’ mechanisms related to maintaining balance (Conrad et al., 1983) and the
existence of large interlimb and interindividual differences in limb kinematics (for review, see Olney and Richards, 1996). Computer simulations of gait that include the musculoskeletal system could elucidate the causal relationships between alterations in muscle excitation and the functional consequences of the movement (Yamaguchi et al., 1991). Unfortunately, such simulations are not yet tractable for the full gait cycle.

In order to understand the relationship between the timing of muscle excitation and impaired motor performance during a functional locomotive task, we chose to study pedalling. As a consequence of its mechanics, pedalling has three immediate advantages over gait as a bilateral, cyclical, lower extremity locomotor movement for studying the mechanical consequences of the neural control. First, the mechanical coupling between the two legs allows even non-ambulatory subjects to pedal cyclically so that a steady-state excitation pattern can emerge in the plegic limb (Brown and DeBacher, 1987). Secondly, the kinematics of pedalling is mostly determined by the setup of the ergometer (Kautz and Hull, 1995), so that successful pedalling necessitates that essentially the same trajectory be followed by plegic, non-plegic and neurologically normal legs. Thirdly, postural support can be provided to remove the subject’s need to employ confounding ‘protective’ mechanisms for maintaining balance (Brown et al., 1996a).

Pedalling also provides a rich theoretical framework for investigating intermuscular co-ordination. The mechanics of pedalling is well understood (Kautz and Hull, 1993; Fregly and Zajac, 1996; Raasch et al., 1997), and pedalling performance can be biomechanically quantified (e.g. in terms of the total mechanical work and net negative work done by the plegic limb). Furthermore, because of the constrained kinematics, computer simulations of pedalling are more tractable than those of gait. Such simulations (Raasch et al., 1997) have suggested that normal pedalling requires muscular activity to perform four different functions: (i) to produce external power during extension; (ii) to aid limb recovery during flexion; (iii) to assist in the transition between extension and flexion; and (iv) to assist in the transition between flexion and extension. Further simulations show that muscular excitations during limb transition are necessary for continuous smooth progression of the crank, and that performance is severely impaired when muscles are excited in a strictly alternating flexion/extension synergy pattern (Raasch, 1995) based on the clinical observation of abnormal extensor/flexor synergy patterns in hemiplegia (e.g. Brunnstrom, 1970).

The goal of this experiment was to determine to what extent the impaired ability of persons with hemiplegia to perform mechanical work with their plegic leg during pedalling was related to abnormalities in the timing of muscle excitation. We felt that a suitable understanding of muscle function (in terms of ‘external power production’ and ‘transition’) and mechanics during pedalling would allow us to relate abnormalities in the timing of muscle excitation to specific functional consequences in the impaired movement. Establishing these relationships will lead to quantitative measures for assessing disturbed motor control and will provide insights into the mechanisms underlying specific motor performance deficits in persons with post-stroke hemiplegia.

### Methods

#### Subjects

Twelve healthy elderly subjects and 15 subjects with post-stroke hemiplegia of >6 months onset were tested (see Table 1). Subjects with hemiplegia were recruited from the surrounding community and were selected if they had sustained a single, unilateral cerebrovascular accident with residual lower limb plegia and had no severe perceptual, cognitive or sensory deficits, no significant lower limb contractures and no significant cardiovascular impairments contraindicative to pedalling, and could tolerate sitting on a bicycle seat for ~1 h. All subjects gave informed consent, as approved by the internal review board of the Stanford University School of Medicine and consistent with the Declaration of Helsinki. Patients underwent the lower limb part of the Fugl-Meyer Assessment (Fugl-Meyer et al., 1975) for assessment of global motor function. The healthy elderly subjects showed no signs or symptoms of neurological disease or lower limb orthopaedic impairment.

The hemiplegic subjects in this study ranged in their walking ability from non-ambulatory to mildly impaired. In addition, they also varied in their ability to perform movements outside of extensor/flexor synergy patterns (e.g. Brunnstrom, 1970), as assessed clinically by a part of the modified Fugl-Meyer assessment (see Synergy performance in Table 1). Subjects scoring ≤14 were able to move only within the synergy patterns (n = 2) (e.g. only basic limb flexion or extension synergies were performed voluntarily and were sufficiently developed to show definite joint movements), those scoring 15–18 were additionally able to combine elements of the synergy patterns (n = 6) (e.g. some movement combinations that deviate from basic limb synergies become available), and the best-performing subjects (scoring >18) were able to at least partially perform a movement independent of the synergy patterns.

#### Protocol

A standard ergometer with a frictionally loaded flywheel was modified by including a backboard seating mechanism with shoulder and lap harnesses to stabilize the subject and remove the need to control balance (see Fig. 1). The feet were firmly attached to instrumented pedals which allowed subjects to create shear and vertical forces (which were measured). Angular rotation of the crank and pedals was measured by optical encoders. Further details concerning the apparatus are presented elsewhere (Brown et al., 1996a).
Table 1 Subject population characteristics

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age</th>
<th>Sex</th>
<th>Post-stroke interval (months)</th>
<th>Synergy performance (max. = 22)</th>
<th>Total modified Fugl-Meyer (%)</th>
<th>Plegic side</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>58</td>
<td>M</td>
<td>7</td>
<td>21</td>
<td>97</td>
<td>L</td>
</tr>
<tr>
<td>B</td>
<td>63</td>
<td>M</td>
<td>9</td>
<td>16</td>
<td>90</td>
<td>L</td>
</tr>
<tr>
<td>C</td>
<td>64</td>
<td>F</td>
<td>17</td>
<td>21</td>
<td>95</td>
<td>R</td>
</tr>
<tr>
<td>D</td>
<td>77</td>
<td>F</td>
<td>90</td>
<td>17</td>
<td>83</td>
<td>L</td>
</tr>
<tr>
<td>E</td>
<td>60</td>
<td>M</td>
<td>12</td>
<td>22</td>
<td>100</td>
<td>L</td>
</tr>
<tr>
<td>F</td>
<td>60</td>
<td>M</td>
<td>87</td>
<td>18</td>
<td>92</td>
<td>R</td>
</tr>
<tr>
<td>G</td>
<td>64</td>
<td>M</td>
<td>38</td>
<td>9</td>
<td>72</td>
<td>R</td>
</tr>
<tr>
<td>H</td>
<td>69</td>
<td>M</td>
<td>32</td>
<td>16</td>
<td>88</td>
<td>L</td>
</tr>
<tr>
<td>I</td>
<td>74</td>
<td>F</td>
<td>46</td>
<td>7</td>
<td>71</td>
<td>L</td>
</tr>
<tr>
<td>J</td>
<td>64</td>
<td>M</td>
<td>119</td>
<td>21</td>
<td>93</td>
<td>L</td>
</tr>
<tr>
<td>K</td>
<td>65</td>
<td>M</td>
<td>132</td>
<td>18</td>
<td>88</td>
<td>R</td>
</tr>
<tr>
<td>L</td>
<td>63</td>
<td>M</td>
<td>15</td>
<td>19</td>
<td>86</td>
<td>L</td>
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<tr>
<td>M</td>
<td>69</td>
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<td>8</td>
<td>20</td>
<td>91</td>
<td>R</td>
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<tr>
<td>N</td>
<td>72</td>
<td>M</td>
<td>15</td>
<td>16</td>
<td>85</td>
<td>L</td>
</tr>
<tr>
<td>O</td>
<td>57</td>
<td>M</td>
<td>8</td>
<td>NA</td>
<td>NA</td>
<td>L</td>
</tr>
<tr>
<td>Plegic</td>
<td></td>
<td></td>
<td>65.3 ± 5.8</td>
<td>42.3 ± 43.1</td>
<td>0–14, n = 2</td>
<td>87.9 ± 8.4</td>
</tr>
<tr>
<td></td>
<td>(n = 15)</td>
<td></td>
<td></td>
<td></td>
<td>15–18, n = 6</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>19–22, n = 6</td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td></td>
<td></td>
<td>69.5 ± 8.4</td>
<td>7 M</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td></td>
<td>(n = 12)</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

Note: Synergy performance reflects the ability to move within (0–14), to combine (15–18) or move outside (19–22) of extensor/flexor synergy patterns as measured in a portion of the modified Fugl-Meyer assessment. Total modified Fugl-Meyer is combined percentage on lower extremity and balance subsections (100% = 96 points).

The experimental protocol, conducted in an hour, consisted of measuring pedal forces, pedal and crank kinematics and EMG during pedalling at 12 randomly ordered workload and cadence combinations. This report is based on the data from a moderate workload and cadence combination (workload of 135 J and target cadence of 40 r.p.m.). Data from the additional conditions form the basis of a study of the effects of exertion (workload and cadence) on hemiplegic motor performance that will be reported elsewhere. Analysis of these data revealed that subjects did not exhibit significant changes in EMG timing with changes in workload and cadence (presented in abstract form in Brown et al., 1996b).

Therefore, the moderate workload and moderate cadence selected for this study resulted in a typical excitation pattern for each subject. Subjects were given visual feedback and instructed to maintain a steady cadence (average cadence of 40 r.p.m.). Once a steady cadence had been achieved, 15 s of EMG, pedal force and encoder data were collected (1200 samples per second).

Surface EMG was recorded from the tibialis anterior (TA), soleus (SO), medial gastrocnemius (MG), rectus femoris (RF), vastus medialis (VM), biceps femoris (BF) and semimembranosus (SM) muscles of the right leg in healthy subjects, and of both legs in subjects with hemiplegia. EMG electrodes (Therapeutics Unlimited, Iowa City, IA, USA) were positioned over the distal half of the muscle belly such that contact surfaces were aligned with the longitudinal axis of the muscle fibres. Electrode sites were prepared by cleaning the skin with isopropyl alcohol and shaving the hair, when necessary, to ensure good contact. Silver–silver chloride electrodes (interelectrode distance = 22 mm, diameter = 8 mm) were attached using adhesive pads and electrode gel. Electrodes provided preamplification with a gain of ×35. A common reference electrode was placed on the distal end of the right tibia. Amplifier gain was selectable from ×500 to ×10 000 with a bandwidth of 20–4000 Hz. The common mode rejection ratio was 87 dB at 60 Hz and the input impedance was >15 MΩ at 100 Hz.

Data processing and analysis

The net external mechanical work done by each limb was calculated from the kinematic and kinetic data and was used as a measure of motor performance. First, a third-order Butterworth low-pass filter was used to filter the pedal forces (20 Hz cutoff) and the crank and pedal angles (8 Hz cutoff). The force component oriented tangentially with respect to the crank arm created a torque about the crank centre (referred to as the crank torque) which contributed to the angular acceleration or deceleration of the crank. The crank torque was plotted against crank angle, and the area under the resulting curve yielded the net external work done by that leg. The positive and negative areas were computed separately in order to measure both the propulsive (positive area) and retarding (negative area) work done by the limb.

EMG was quantified in terms of the percentage of the total excitation that was present during four phases in the pedalling cycle in order to relate potentially "inappropriate" excitation to decreased limb performance. Four equal phases (90°) were defined by axes parallel and perpendicular to the

EMG and impaired hemiplegic pedalling

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Fig. 1 Experimental setup for studying cyclical lower extremity movements on a modified bicycle ergometer. The inset defines position of phases used in subsequent EMG analysis. Note that contiguous phases I and II represent limb extension, II and IV represent limb flexion, II and III represent posteriorly directed movement that encompasses the transition from extension to flexion, and IV and I represent anteriorly directed movement that encompasses the transition from flexion to extension.

座管（图1）和可能与特定的肢体功能相关。Phase I 相当于肢体的伸展（脚离开骨盆）和前部肢体的运动（与躯干/骨盆轴相比），Phase II 相当于肢体的伸展和后部运动，Phase III 相当于肢体的屈曲（脚逐渐靠近骨盆）和后部运动，以及 Phase IV 相当于肢体的屈曲和后部运动。对于每一个阶段，方波 EMG 被积分（IEMG）并以总 IEMG 的百分比来表示。这个我们之前使用过的（Brown et al., 1997）量度，被用于取代传统的开-关 EMG 基地的定义，因为感兴趣的是那些导致功能活动改变的时机，这种活动的中功能影响可以在生物力学肌肉功能的背景下解释。另外，这个量度还有一个优点，它很容易计算，并且可以提供一个定量的测量，即使在特定肌肉中无法通过单个脉冲的开-关时间来表征 EMG 时

我们可视化地检查了每一项，非平均化的踏板运动学、动力学和 EMG 活动，以生成趋势。然后我们计算了每一项的量度（每一转的总正向，总负向，和净外力机械工作）和 EMG（阶段 IEMG 的百分比）并计算每个量度的平均值。控制组和半肢瘫痪组的平均值之间的差异被使用了。
EMG and impaired hemiplegic pedalling

Table 2  Kinetic measures of motor performance

<table>
<thead>
<tr>
<th>Subject</th>
<th>Total work (J)</th>
<th>Average cadence (r.p.m.)</th>
<th>Work done by plegic limb</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Net (J)</td>
</tr>
<tr>
<td>A</td>
<td>131.3</td>
<td>42.3</td>
<td>48.6</td>
</tr>
<tr>
<td>B</td>
<td>135.3</td>
<td>42.9</td>
<td>0.1</td>
</tr>
<tr>
<td>C</td>
<td>129.4</td>
<td>40.7</td>
<td>42.7</td>
</tr>
<tr>
<td>D</td>
<td>139.0</td>
<td>30.3</td>
<td>28.9</td>
</tr>
<tr>
<td>E</td>
<td>135.4</td>
<td>41.9</td>
<td>11.5</td>
</tr>
<tr>
<td>F</td>
<td>127.4</td>
<td>44.9</td>
<td>30.4</td>
</tr>
<tr>
<td>G</td>
<td>136.7</td>
<td>28.0</td>
<td>−1.4</td>
</tr>
<tr>
<td>H</td>
<td>129.8</td>
<td>41.6</td>
<td>29.7</td>
</tr>
<tr>
<td>I</td>
<td>83.6*</td>
<td>33.4</td>
<td>9.0</td>
</tr>
<tr>
<td>J</td>
<td>129.2</td>
<td>37.7</td>
<td>32.3</td>
</tr>
<tr>
<td>K</td>
<td>141.1</td>
<td>40.3</td>
<td>3.1</td>
</tr>
<tr>
<td>L</td>
<td>126.5</td>
<td>39.0</td>
<td>54.3</td>
</tr>
<tr>
<td>M</td>
<td>140.4</td>
<td>39.5</td>
<td>27.2</td>
</tr>
<tr>
<td>N</td>
<td>88.2*</td>
<td>37.5</td>
<td>−2.4</td>
</tr>
<tr>
<td>O</td>
<td>125.2</td>
<td>40.5</td>
<td>−19.8</td>
</tr>
<tr>
<td>Plegic (n = 13*)</td>
<td>132.8 ± 5.4</td>
<td>39.2 ± 4.8</td>
<td>22.1 ± 21.8</td>
</tr>
<tr>
<td>Control (n = 12)</td>
<td>136.6 ± 5.5</td>
<td>43.0 ± 2.9</td>
<td>68.3 ± 8.0</td>
</tr>
</tbody>
</table>

Results

Thirteen of the 15 hemiplegic subjects successfully pedalled at the test condition (cadence = 39.2 ± 4.8 r.p.m., workload = 132.8 ± 5.4 J). The other two subjects (I and M) successfully pedalled at a lower workload (83.6 and 88.2 J, respectively). Actual cadence ranged from 28.0 to 44.9 r.p.m. (see Table 2). The control subjects pedalled at 136.6 ± 5.5 J and 43.0 ± 2.9 r.p.m. Since subjects I and M did not pedal against the same workload, their results were not included in the hemiplegic group data for statistical analyses or the regression analyses based on these data, and are presented separately.

Kinetic measures of motor performance

The net external mechanical work done by the plegic limb (Wp) in persons with hemiplegia was greatly reduced compared with the net external mechanical work done by each limb of the elderly control subjects (WC) (average of both legs = 22.1 ± 21.8 versus 68.3 ± 8.0 J, P < 0.0001) (see Table 2). While Wp was always less than WC, there was great variability in work production by the hemiplegic subjects as Wp ranged from 54.3 to −19.8 J (negative net work, meaning that the plegic leg provided a net resistance to crank progression). The tangential crank torques for a representative hemiplegic (plegic leg) and control subject (right leg) show that the hemiplegic subject’s lower net work production was due to a combination of reduced downstroke propulsion and increased upstroke resistance (see Fig. 2). Overall, hemiplegic subjects produced less positive external work (positive area under the curve, mostly during phases I and II: 48.8 ± 13.3 versus 70.0 ± 4.8 J, P < 0.0001) and more negative external work (negative area, mostly during

Fig. 2  Crank torque versus crank angle for plegic limb of subject E and the control leg of a representative control subject. Positive mechanical work (total positive area under curve) is reduced and negative mechanical work (total negative area) is increased for E. Therefore, net mechanical work was decreased.
phases III and IV: \(-26.7 \pm 14.0\) versus \(-6.3 \pm 4.6\) J, \(P < 0.0001\).

**EMG data**

For nearly all the hemiplegic subjects, the EMG timing of all the muscles in the non-plegic leg was similar to that of the corresponding muscles of the control subjects. The only significant differences \((P < 0.05)\) identified between percentage IEMG in a phase in the non-plegic leg and control subjects were in phases with substantial IEMG \([i.e.\ increases\ in\ phase\ I\ in\ the\ non-plegic\ TA\ (32.5 \pm 13.1\ versus\ 23.1 \pm 7.3\%)\]\ and VM \((64.3 \pm 9.1\ versus\ 57.9 \pm 5.0\%)\), and decreases in phase III in the non-plegic BF \((37.5 \pm 8.1\ versus\ 45.9 \pm 10.9\%)\) and SM \((36.5 \pm 9.7\ versus\ 48.1 \pm 13.6\%)\). Thus, the EMG from the non-plegic leg reveals no change in timing. The remaining report of results focuses on the plegic leg.

Substantial differences in EMG timing are evident when the rectified EMG from all seven muscles for the plegic leg of a control subject and the plegic leg of subject E are compared (see Fig. 3). Many of the other hemiplegic subjects showed similar differences from the timing of the control subjects, as evidenced by a substantial percentage of the total plegic IEMG occurring in phases in which the same muscle was not excited in control subjects (see Fig. 4) \(\text{the specific differences \(P < 0.05\) identified for individual muscles are detailed in the following paragraphs)}\). For the purpose of describing the results for individual muscles below, the EMG activity in a phase was qualitatively defined as ‘not excited’ when the percentage IEMG in that phase was \(<10\%\) \(i.e.\ the\ muscle\ was\ ‘off’\) and ‘excited’ when the percentage IEMG was \(>20\%\) \(i.e.\ the\ muscle\ was\ ‘on’\) or neither clearly ‘on’ nor ‘off’\).

Excitation of the plegic quadriceps muscles (VM and RF) showed substantial differences in timing with respect to control subjects. When compared with a representative control subject, the rectified EMG from subject E (see Fig. 3) shows prolonged VM excitation and ‘phase-advanced’ RF excitation \(\text{‘phase-advanced’ is defined as excitation in which both the onset and offset occur earlier in the crank cycle than normal)}\). The hemiplegic group data revealed that the plegic RF and VM were typically excited during phase III \(\text{see Fig. 4)}\), whereas RF and VM were never excited in that phase in the control subjects. Plotting the EMG of individual hemiplegic subjects and the group average from the control subjects reveals that two different timing changes were responsible for the increased relative excitation seen in phase III (see Fig. 3).
The plegic VM excitation in phase III typically resulted from prolonged excitation, while the plegic RF excitation in phase III resulted from prolonged excitation in some subjects (A, C, F, J and L) and phase-advanced excitation initiated in phase III (earlier in limb flexion) and terminated in phase I (early in limb extension) in other subjects (B, E, G, I, N and O) (see Fig. 4). The plegic hamstrings (BF and SM) also exhibited phase-advanced excitation. The rectified EMG from subject E (see Fig. 3) showed phase-advanced excitation in both BF and SM. Similar trends can be seen in the hemiplegic group data (see Figs 4 and 5), in which the percentage IEMG in nearly all instances was above the mean for the control subjects for phase II and below the mean for phase III.

Fig. 4 IEMG in phases for individual muscles, showing individual subjects compared with control subjects. Data symbol representing percentage IEMG in a phase is the subject’s identification letter (see Tables 1 and 2). The control subjects’ percentage IEMG in each phase is represented by the mean activity (dotted line) ± 2 SD (shaded region).
The timing of excitation of the muscles crossing the ankle (TA, SO and MG) was typically less severely affected than that of other muscles. The rectified EMG from subject E (see Fig. 3) shows prolonged SO excitation, phase-advanced MG excitation and no change in TA excitation. The group data showed that the plegic SO was typically prolonged into phase III (see Fig. 4), whereas excitation of the plegic MG was both initiated earlier (in phase I instead of phase II) and...

**Fig. 5** Average RF and VM EMGs for all control subjects (light shading, top row) and for the individual hemiplegic subjects arranged in descending order of total mechanical work done by the plegic limb. EMGs were smoothed by averaging over a 67 ms window for purposes of display. All complete revolutions were averaged, and individual subjects’ EMGs were normalized to the maximum value in the cycle. Increased percentage EMG in the sum of phases III + IV in RF (phase-advanced excitation) and in phase III in VM (prolonged excitation) were both correlated with decreased external work, while decreased percentage EMG in phase III in SM (phase-advanced excitation resulted in decreased SM excitation in phase III) was correlated with decreased external work. RF and VM of subject K were excluded from regression analyses since the total IEMG of each was 2 SD less than the mean of the control subjects.
terminated earlier (typically in late phase II or early phase III instead of late phase III) than in control subjects (see Fig. 4).

**Relationship between EMG timing and limb movement**

The type of EMG timing abnormality observed in a plegic muscle was related to whether that muscle was normally excited only during limb extension (external power-producing muscle) or whether that muscle was normally excited during a period which included both limb extension and flexion (transition muscle). Prolonged excitation was observed in the muscles that were normally excited only during extension (VM and SO), as well as in the RF of five hemiplegic subjects (subjects A, C, F, J and L), in whom RF was substantially excited throughout extension. On the other hand, phase-advanced excitation was observed in muscles that were normally excited during both limb extension and flexion (subjects BF, SM, MG and RF). Furthermore, this phase advance substantially affected the balance of excitation, as the phase (extension or flexion) that included a majority of the excitation was reversed when each plegic muscle was compared with the corresponding muscle in the control subjects. Specifically, the phase-advanced RF excitation seen in six hemiplegic subjects (B, E, G, I, N and O) resulted in substantially increased relative IEMG of the RF during flexion (sum of IEMG in phases III and IV) when compared with that seen in control subjects (68.7 ± 5.2 versus 23.7 ± 6.9%, P < 0.0001). In contrast to the RF, the phase-advanced excitation of the other plegic muscles resulted in the opposite effect as relative excitation was increased during limb extension in the plegic SM (57.0 ± 7.5 versus 31.9 ± 8.9%, P < 0.0001), BF (61.4 ± 12.2 versus 40.0 ± 10.7%, P < 0.0001) and MG (61.9 ± 8.5 versus 47.0 ± 14.9%, P = 0.003) when compared with the corresponding muscle in the control subjects.

**Relationship between EMG timing and kinetic measures of motor performance**

The EMG abnormalities in plegic VM, RF and SM were associated with impaired pedalling performance. When the subjects are ranked with respect to the net mechanical work done by the plegic limb (Wp) it is apparent that the prolonged excitation of VM and the phase-advanced excitation of RF and SM were progressively more pronounced in the subjects who produced less Wp (see Fig. 5). Regression analysis verified these qualitative observations in that both the prolonged excitation of the plegic VM (i.e. percentage IEMG in phase III) and the phase-advanced excitation of the plegic RF (i.e. sum of percentage IEMG in phases III and IV) were inversely related to Wp (r = −0.875, P = 0.0002 for VM and r = −0.896, P = 0.0001 for RF). The phase-advanced excitation of the plegic SM (i.e. sum of percentage IEMG in phases I and II) was weakly inversely related to Wp (r = −0.546, P = 0.053), although statistical significance was not reached. However, one aspect of phase-advanced excitation, the early termination of SM (i.e. percentage IEMG in phase III), was strongly inversely related to Wp (r = −0.744, P = 0.003). In contrast to VM, RF and SM, neither the prolonged excitation of SO nor the phase-advanced excitation of MG and BF was significantly associated with Wp. Taken together, the regression analyses demonstrate that prolonged excitation of VM and phase-advanced excitation of RF and SM are strongly associated with impaired pedalling performance.

Note that while a low level of excitation by a plegic muscle is probably functionally significant, it was felt that percentage IEMG in a phase was not an appropriate measure of excitation when the total IEMG was very small. Therefore, trials in which muscles had IEMG values less than two standard deviations below the mean for control subjects were excluded from the regression analyses (only four instances: RF of subject K, VM of K, and BF of J and N).

**Relationship between post-stroke recovery and kinetic measures of motor performance**

The level of motor recovery, as measured by the Fugl-Meyer assessment (Fugl-Meyer et al., 1975) of the lower limb, was not significantly associated with Wp (r = 0.42, P = 0.15). However, the part of the Fugl-Meyer assessment that assesses the ability to move within, to combine, or move out of ‘extensor/flexor synergy patterns’ (Brunnstrom, 1970) was significantly correlated with Wp (r = 0.56, P = 0.046). Furthermore, subjects able to move outside of the synergy patterns (those with scores of 19–22; see Table 1) averaged 36 J for Wp (n = 6, range = 11.5–48.6), those only able to mix elements of the synergies (scores of 15–18; see Table 1) averaged 15.0 J (n = 6, range = −2.4 to 30.4), and those only able to move within the synergies (scores of 0–14; see Table 1) averaged 4 J (n = 2, values = −1.4 and 9.0).

**Discussion**

We achieved our goal of determining to what extent the impaired ability of persons with hemiplegia to perform mechanical work with their plegic leg during a locomotor task was related to abnormalities in the timing of muscle excitation. By using a pedalling paradigm and a biomechanical model of muscle function we were able to both theoretically and experimentally relate identifiable and quantifiable changes in the timing of excitation of individual muscles to a quantified measure of impaired motor performance, Wp (external mechanical work done by the plegic limb). Furthermore, these changes were found to be dependent on a muscle’s function, in that transition muscles showed phase-advanced excitation while external power-producing muscles showed prolonged excitation.
**Muscle function in pedalling**

In order to provide the necessary framework for interpreting how observed abnormalities in EMG timing of plegic muscles impaired performance, a biomechanical model of muscle function during pedalling was developed. Simulation studies have shown that muscle excitation is required during limb extension (combined phases I + II) to overcome the external load (generate external power) and lift the contralateral limb, and also during limb transitions between extension and flexion to assure continued crank progression (either combined phases II + III or IV + I) (Raasch et al., 1997). Accordingly, we will refer to muscles normally excited throughout limb extension (VM and SO) as ‘external power-producing’ muscles and those normally excited during both extension and flexion (SM, BF, MG and TA) as ‘transition’ muscles. The RF was found to be an exception in that it performed both external power-producing and transition functions, as evidenced by >75% of normal RF excitation occurring during external power production and simulation studies showing that the observed excitation in phase IV is crucial for continued crank progression during the transition from limb flexion to extension (Raasch et al., 1997).

Our EMG data suggest that the chief determinant of a muscle’s function during normal pedalling is its anatomical architecture. The kinematics of the combined muscle–tendon complexes of each muscle is similar between subjects because it is almost entirely determined by the constrained kinematics of pedalling. Previous pedalling research has shown that some muscles (primarily uniarticular muscles such as VM) shorten only during limb extension, while others (primarily biarticular ones such as RF, BF and SM) shorten during a limb transition period that includes both extension and flexion (Hull and Hawkins, 1990). While muscle length was not calculated in our study, it is possible to infer with relative confidence from their study that, for VM, RF, BF and SM (in our control subjects, the period of excitation corresponded to the period of shortening. Thus, in normal pedalling muscles appear to be excited when they can do positive mechanical work, those that shorten during limb extension functioning as external power-producing muscles and those that shorten during transitions functioning as transition muscles.

**Relationship between EMG abnormalities and motor performance**

The net external mechanical work done by the plegic limb \( W_P \) in all subjects was less than the net work done by the average limb of the neurologically normal control subjects \( W_C \), and this diminished work output was associated with altered timing of muscle excitation. Two different EMG timing abnormalities were associated with the abnormal \( W_P \): prolonged excitation of external power-producing muscles and phase-advanced excitation of transition muscles.

Prolonged excitation was seen in VM and SO in most hemiplegic subjects, and in RF in the five subjects who substantially excited RF during the external power-producing phase (L, A, C, J and F; see Fig. 5). In VM and RF excitation was prolonged into phase III, which is the beginning of limb flexion and during which these two muscles were beginning to lengthen (Hull and Hawkins, 1990). The muscles were therefore being excited while they were lengthening, and so produced negative work. Inappropriate excitation during lengthening is consistent with the significant inverse relationship between the degree of prolongation in a particular subject (i.e. the percentage of VM IEMG in phase III) and \( W_P \). Although often prolonged, there was no relationship between altered SO excitation and performance. This is understandable given the primary role of the SO of transferring rather than producing power (Fregly and Zajac, 1996). Furthermore, since the kinematics of the ankle joint can vary substantially between subjects, it is not clear to what extent the prolonged excitation of SO actually produced negative work in any given subject.

The transition muscles in the plegic limbs (RF, BF, SM and MG) exhibited phase-advanced excitation (i.e. early initiation and/or early termination). A phase advance can impair motor performance in two ways: (i) early initiation can result in the muscle doing negative work while it is lengthening, and (ii) early termination can reduce the amount of positive work done by the muscle while it is shortening. When not exhibiting prolonged excitation, RF typically exhibited both early initiation and early termination (see Fig. 5) and so contributed to lowering \( W_P \) by both these mechanisms. Indeed, there was a significant inverse relationship between the degree of RF phase advance (i.e. the percentage IEMG in phases III and IV) and the subject’s motor performance (i.e. \( W_P \)). In SM the percentage IEMG in phase III (but not phase I) was significantly inversely correlated with \( W_P \) suggesting that early termination (not early initiation) was the primary contributor to reduced \( W_P \) (see Fig. 5). For BF and MG, the degree of phase advance was not correlated with \( W_P \). In the case of MG this may be because, like SO, it is primarily a power-transferring muscle rather than a power-producing muscle.

The clinical model of extension/flexion synergies (Twitchell, 1951; Brunnstrom, 1970; Perry, 1993) has certain similarities with the phase-advanced excitation of the transition muscles. Specifically, excitation during pedalling was advanced towards extension in those muscles that typically show increased activity during the stance phase [i.e. the muscles belonging to the extensor synergy, namely MG, BF and SM (Perry, 1993)] and advanced towards flexion in RF, which typically shows increased activity during the swing phase [flexor synergy (Perry, 1993)]. Furthermore, there was a significant inverse relationship between the clinical assessment of the level of motor recovery with respect to extensor/flexor synergy patterns (see Table 1) and motor performance \( (W_P) \). However, we did not observe a strict synergistic pattern of mass extensor activation alternating with mass flexor activation (except perhaps in subject E; see Fig. 3). Rather, there seems to be an impairment in the ability...
to fine-tune the coordination of the muscles which results in degradation of timing.

**Disturbed motor control in hemiplegia**

Although previous studies have quantified EMG timing abnormalities in the lower extremity of neurologically impaired subjects (usually with spastic paraparesis), no studies in the hemiplegic gait literature have demonstrated that the extent of an EMG timing abnormality can be quantitatively related to the level of impaired performance during the task. Previous studies have sought to objectively assess spasticity during a dynamic lower extremity task (Benecke et al., 1983; Conrad et al., 1985; Fung and Barbeau, 1989), with the ratio of a muscle’s IEMG during its normal ‘on’ and ‘off’ periods as the measure of disordered muscle excitation. However, these studies did not have a biomechanical model of muscle function during the investigated movement and they did not quantify motor performance. Our study is the first to demonstrate that the extent of an EMG timing abnormality can be quantitatively related to the level of impaired performance during a functional task.

Similarities between prolonged excitation of power-producing muscles and phase-advanced excitation of transition muscles and some of the EMG timing changes reported for the same muscles in the hemiplegic gait literature suggest that pedalling may reproduce the characteristics of abnormal muscle timing in hemiplegic gait while providing a much more controlled environment for future investigations. Specifically, many subjects have shown increased relative excitation (percentage of total EMG) during the stance phase in MG, BF and SM (Knutsson and Richards, 1978; Perry, 1993) and increased relative excitation during the swing phase in RF (Pinzur, 1987; Perry, 1993). These changes are somewhat analogous to phase advances of the normal excitation during gait and occur in muscles that we found to be phase-advanced in pedalling. Prolonged excitation of VM throughout the stance phase has also been shown (Hirschberg and Nathanson, 1952; Shiavi et al., 1987; Perry, 1993), although it did not usually continue into the swing phase, as did VM excitation during pedalling. Since our study determined that the degree of ankle muscle timing dysfunction was not associated with reduced performance in pedalling, we have accordingly focused on thigh muscles.

Timing changes in individual muscles should probably be an expected consequence of a central lesion, some changes being the direct consequence of the lesion and others being compensatory adaptations to secondary changes such as those in muscle fibre and connective tissue properties. At least for ankle muscles, it is likely that the impaired timing is related to secondary changes in muscle fibre and connective tissue properties following stroke (Dietz et al., 1981, 1986; Thilmann et al., 1991). A major contribution of this study is that we were able to show, regardless of the initial cause, obvious biomechanical consequences of EMG timing changes consistent with decreased performance as the muscles whose timing changes were most strongly correlated with decreased performance either produced negative work by being active while lengthening (VM, RF) or produced less positive work by not being active when normally shortening (SM). This establishes a theoretical basis for the correlation between increased abnormality of muscle timing and decreased pedalling performance. Thus, if muscle timing abnormalities were related in part to compensatory adaptations to changes in muscle fibre and connective tissue properties following stroke, the changes were not effective at equalizing mechanical work production by the plegic and non-plegic limbs.

We have proposed that the plegic muscles are differentially affected depending on their function, which probably reflects a varying level of influence by different spinal circuitry and/or descending pathways on the motor neuron pools of the external power-producing muscles when compared with the transition muscles. For example, the circuitry involved in relayingafferent information related to the transition from extension to flexion (e.g. hip position, load receptors, etc.) seemed to inhibit transition muscles (BF, SM and MG) early in limb flexion, to the point that they could be de-recruited, while the prolonged external power-producing muscles were simultaneously showing a net excitation. The much more controlled environment provided by pedalling when compared with gait potentially facilitates exploration of the underlying mechanisms of these EMG timing abnormalities. Furthermore, since the EMG timing abnormalities were so strongly correlated with net mechanical work during pedalling, future work could also determine whether pedalling performance offers a quantitative measure of the underlying motor control disturbance, which would provide an outcome measure for assessing, and ultimately improving, movement deficits in persons with hemiplegia.

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