# Coordination of Hemiparetic Locomotion after Stroke Rehabilitation

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Objectives. *Determine whether a rehabilitation program targeting functional motor recovery of persons with poststroke hemiparesis improved motor coordination.* Methods. *A subgroup of 20 persons with poststroke hemiparesis (*n = *11 in intervention and* n = *9 in control group) was investigated from within a larger randomized controlled single-blind clinical trial of 100 patients. Motor coordination was measured using a pedaling task, and subjects in the intervention group pedaled during an intensive broad-based home exercise program that targeted flexibility, strength, balance, and endurance. Coordination variables based on paretic leg pedal forces and EMG of 4 thigh muscles were measured while pedaling pre- and postintervention.* Results. *Despite extensive pedaling practice, up to 30 half-hour sessions that were progressively more intense, there was no effect (*P > *0.05) of the intervention on percent of total work done by the paretic leg, quantitative measures of EMG, or pedaling speed. However, walking speed was improved and pedaling and walking faster were associated after the intervention.* Conclusions. *There is no evidence of improved locomotor coordination postintervention. The increased walking and pedaling speed was likely achieved by a more proficient use of the same impaired pattern without EMG timing changes, likely because of increased strength and endurance postintervention. A more task-specific intervention may be required to improve coordination, consistent with principles of use-dependent plasticity.*

Key Words: *Stroke—hemiparesis—locomotion—coordination—rehabilitation*

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There has been insufficient establishment of effective therapeutic interventions to rehabilitate motor deficits following stroke, in part because of the historical general acceptance of Cajal's century-old expressions of pessimism about the potential for plasticity in the adult nervous system (see ref. 1 for a review). His belief that there was little potential for changes (plasticity) to synaptic connections in adulthood received few challenges from neuroscientists and general acceptance by clinicians, and led to the consensus that recovery potential was limited following CNS injury in adulthood. However, it is now generally accepted that the adult nervous system is capable of tremendous plasticity.[2,3] Nevertheless, for recovery of complex motor functions such as walking, little evidence exists that documents improvements in locomotor performance resulting from restoring deficits in motor coordination.

Teixeira-Salmela and others[4] conducted a strengthening exercise program and demonstrated that subsequent increases in walking speed were related to changes in motor coordination of the paretic leg (predominantly improved ankle plantarflexor, hip flexor, and hip extensor performance, as judged by increased joint moments and power). Although this study provides a wealth of information about changes in walking performance, they did not collect EMG data and the kinetic changes they observed were consistent with increased muscle force generation without necessarily changing the timing of muscle activity. Thus, specific underlying changes in coordination were unclear. Additional studies have also showed improved postintervention walking velocity, which by itself is not sufficient to demonstrate substantial restoration of motor coordination, because increased strength or endurance could produce increased gait speed (although note that strength and endurance are not completely independent of coordina-

 

tion), as could more proficient compensatory use of the already impaired motor pattern. Although improved compensation may lead to immediate increases in functional status, the rational design of therapeutic interventions requires knowledge of whether a functional effect results from a compensatory strategy or a true restitution of preinjury motor functions.

As a step toward establishing relationships between muscle excitation and motor performance in relatively complex lower extremity motor tasks, Kautz and Brown[5] developed a quantitative model of motor coordination in pedaling by relating EMG timing abnormalities to impaired motor performance. They were able to interpret their results in the context of quantitative definitions of muscle function determined within a biomechanical model-based analytical framework. The timing of EMG in individual paretic limb muscles exhibited 2 distinct types of abnormalities that were significantly correlated with reduced mechanical work production: prolonged excitation in vastus medialis (resulting in increased negative work production) and phase-advanced excitation (both early initiation and early termination that resulted in increased negative work if initiation began during lengthening and decreased positive work if muscle turned off when it was usually producing positive work) in rectus femoris and semimembranosus. The results suggested that muscles were differently affected depending on their function because external power producing muscles (e.g., vastus medialis) showed prolonged excitation and muscles that normally maintain crank progression during limb transitions (e.g., semimembranosus, rectus femoris) showed phase-advanced excitation. Subsequently, this model was successfully used to investigate the relationship between muscle coordination and pedaling performance with increased effort[6] and speed.[7] Although these function-specific impairments of muscle excitation were correlated with reduced motor performance during the pedaling task, they were also correlated with poor motor recovery (lower limb synergies component of the Fugl-Meyer assessment) as assessed irrespective of pedaling performance.

The goal of the present study was to use a similar framework to determine whether a therapeutic exercise program that targets functional motor recovery in persons with poststroke hemiparesis also resulted in improved lower extremity motor coordination. Specifically, we used a model[5] of coordination to test whether motor coordination during a locomotor task was improved after an intensive home exercise program that targeted flexibility, strength, balance, endurance, and overall upper extremity function. The data presented here are the results of a supplemental investigation of a subgroup of a larger study.[8] Duncan and others[8] at the Center on Aging at the University of Kansas performed a randomized controlled single-blind clinical trial of 100 patients. Of particular relevance to this study is that all intervention subjects pedaled an exercise ergometer for aerobic conditioning, with a structured progressive program of up to 30 min of pedaling. Those subjects in the subgroup (which included subjects from both the control and intervention groups of the larger study) also pedaled an instrumented ergometer pre- and postintervention so that coordination variables[5] could be used to test for improved motor coordination.

## METHODS

Twenty subjects with poststroke hemiparesis (Table 1) were tested in a randomized, controlled, single-blind clinical trial ($n$= 11 in intervention and $n$= 9 in control group). The intervention consisted of a structured, progressive, physiologically based, therapist-supervised in-home program of thirty-six 90-min sessions over 12 weeks targeting flexibility, strength, balance, endurance, and upper-extremity function. Subjects who received the intervention performed a large amount of pedaling (more than 13 h of pedaling during up to 30 half-hour sessions), with all subjects progressing in resistance levels and/or intensity from the initial session to the final session. The 20 subjects were a subset of a larger clinical trial, and full details of the inclusion/exclusion criteria, intervention, and assessment in the larger study are described in Duncan and others.[8] Subjects in the intervention group were tested pre- and postintervention (approximately 3 and 6 months poststroke), and those in the control group were tested twice with a similar intervening time period. Assessment variables are identified in Table 1. Note in particular that 10-m walking speed is determined during the assessment. In addition to these assessments, all subjects also performed a pedaling task on a stationary bicycle similar to that used for the cardiovascular stress test and the endurance training within the intervention. The objective of this test was to provide a quantitative assessment of muscle coordination during pedaling to determine whether the intervention program changed muscle coordination patterns.

The subjects pedaled at 3 different cadences (self-selected, 40 rpm, and as fast as comfortably possible) in each of 2 sessions (pre- and posttreatment or 3-month control period) on a stationary bicycle ergometer, and data were collected for 20 s. Data collection always commenced once a steady cadence was established (about 10 s). During the 40-rpm condition, a metronome was provided to establish a steady cadence, then it was turned off and data collection commenced. Subjects were instructed to pedal until told to stop (approxi-

Table 1. Characteristics of Subjects

| Variable | Control ($n$ = 9) | Intervention ($n$ = 11) |
|---|---|---|
| Age (years) | 68.9 (8.3) | 62.6 (6.5) |
| Lower-Extremity Fugl-Meyer (max 34) | 25.1 (2.7) | 24.6 (2.7) |
| Berg (max 56) | 45.3 (6.8) | 48.9 (6.6) |
| Peak VO2 (ml/kg/min) | 12.1 (3.9) ($n$ = 8) | 13.9 (3.6) |
| 10-m gait speed (s) | 0.70 (0.31) | 0.91 (0.27) |
| 6-min walk (m) | 261.8 (101.5) | 327.2 (105.7) |

Note: Numbers in parentheses are standard deviations.

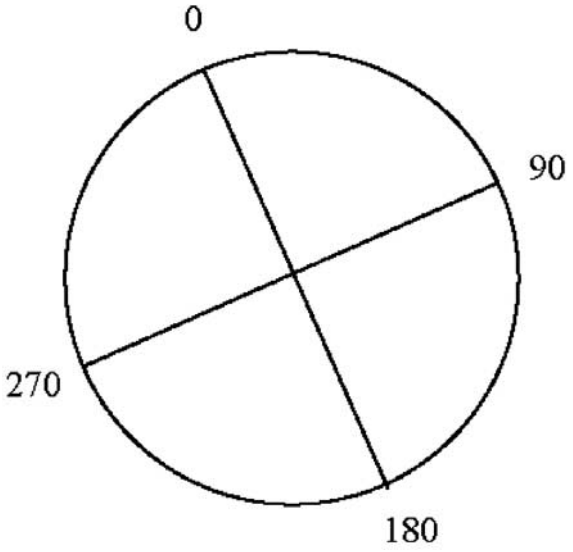


**Figure 1.** Definition of crank angles, with progression of the crank occurring in a clockwise direction. Quantitative measures of EMG calculate percentage of activity between 180 and 270 deg. From 0 to 180 deg represents limb extension, and from 180 to 360 deg represents limb flexion. Thus, the window of interest represents the 1st half of the limb flexion phase.

mately 30 s). Data collection began roughly 2 to 3 s after the metronome was halted. Bilateral pedal forces (shear and normal) were measured with pedal dynamometers,[9] and crank and pedal angles were measured using digital optical encoders mounted to the crank and pedals (the pedal dynamometer and optical encoder system was identical to that used in Kautz and Brown[5]). Surface EMGs were measured from 4 paretic leg muscles that had previously been shown to exhibit timing abnormalities that were related to pedaling performance[5]: vastus medialis (VM), rectus femoris (RF), biceps femoris long head (BF), and semimembranosus (SM). All force, kinematic, and EMG data were sampled at 1000 Hz. The particulars of the measurements were similar to those of recent studies at the VA Palo Alto Laboratory.[10,11]

The raw EMG data were high-pass filtered using a 4th-order, zero-lag digital Butterworth filter with a 20 Hz cutoff to eliminate movement artifacts. A mean value was calculated and subtracted so that the EMG could be rectified by taking the absolute value. Then the average EMG while the crank was between 180 and 270 deg (3rd quadrant in Figure 1) was calculated for each muscle and expressed as a percentage of the total average EMG for that muscle over the entire crank cycle (corresponding variables defined as RF3, VM3, BF3, and SM3). The window between 180 and 270 deg was selected because the resulting values correspond to the previously established measures of impaired EMG timing in hemiparetic pedaling.[5] Note that EMG data were not normalized with respect to amplitude (e.g., not expressed as a percentage of maximum voluntary contraction) and we only attempt to determine whether there were changes in the timing of activity as a result of intervention (because percentage of total activity in target window measures timing and not amplitude). Kinetic data (pedal forces) and kinematic data (pedal and crank angles) were filtered with 4th order, zero-lag low-pass digital Butterworth filters with cutoff frequencies of 20 and 9 Hz, respectively. The tangential crank force, which is that component of the pedal force that is oriented perpendicular to the crank arm and acts to rotate the crank, was calculated from the shear and normal pedal forces and the angular position of the crank and pedal. Mechanical external work performed was also calculated for each leg by integrating the tangential crank force over the entire crank cycle.[5] The percentage of total work performed by the paretic leg was then calculated by dividing the work performed by the paretic leg by the total work performed by both legs.

All kinematic, kinetic, and EMG measures of coordination were calculated for each cycle and then averaged across cycles to produce the value for each trial. In the interest of maintaining comparison of conditions as consistent as possible, the 40-rpm trial was used as the basis for the calculation of the mean values. In 4 instances, there were technological difficulties that compromised the data from the 40-rpm trial, so data from the self-selected speed trial were used after determining that it fell within the expected range of variability for the 40-rpm trial. The variables used to quantify the coordination of the paretic leg were percentage of external mechanical work performed, prolonged excitation of vastus medialis (VM3), and phase-advanced excitation of rectus femoris (RF3) and hamstrings (BF3 and SM3).

Average coordination measures were computed from the initial time period for all subjects in both groups to ensure that this subject population was consistent with previous literature on hemiparetic patients. Then the groups were split into those who did and did not receive the intervention in the parent study, with separate comparisons using Wilcoxon signed-rank tests of pre- and

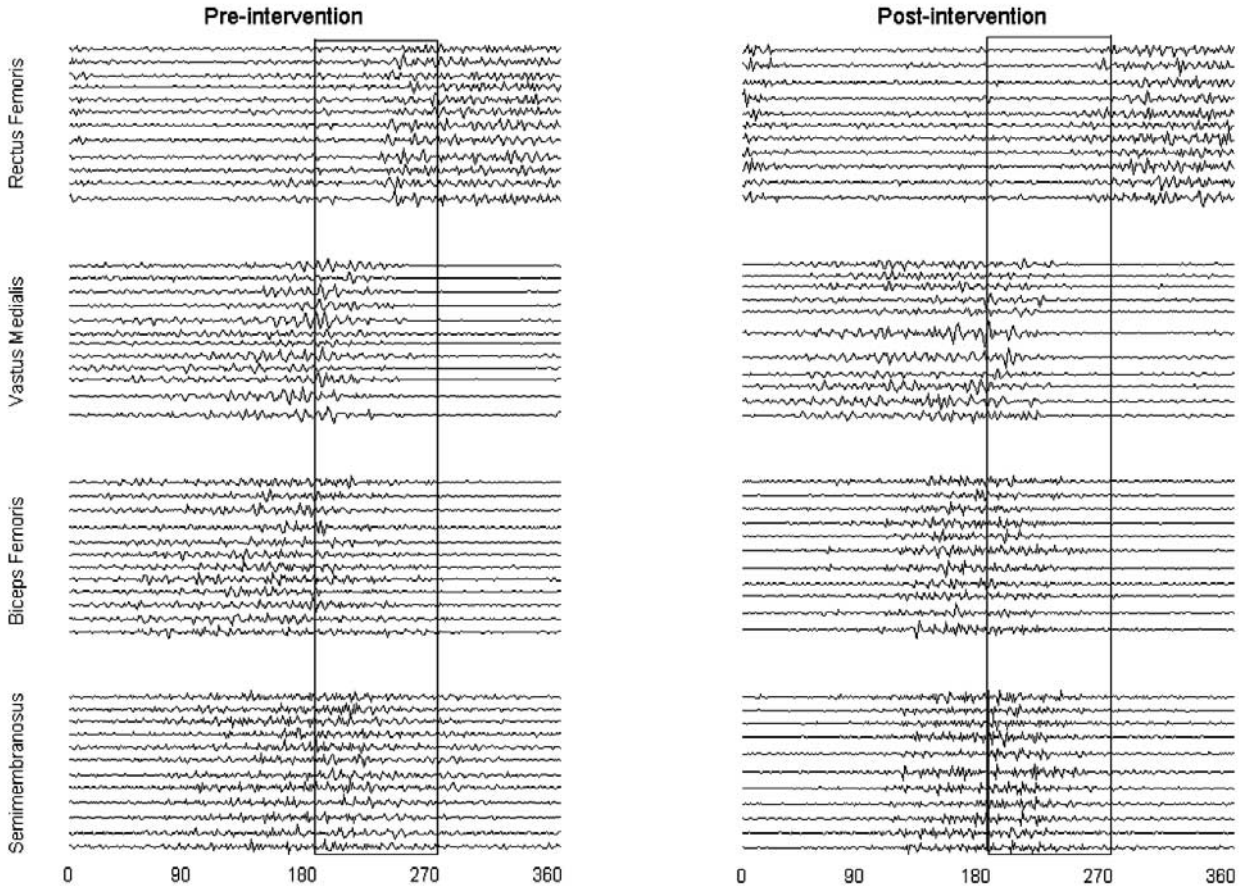


**Figure 2.** Pattern of raw EMG data for a subject in the intervention group (pre- and postintervention) with consecutive revolutions of data stacked vertically. Note the relatively unchanged patterns in the different muscles. The same consecutive revolutions of activity are shown for each muscle in each trial. The boxes represent the region over which the percent of EMG activity is calculated. Note that no comparisons of amplitude should be made either between muscles or between trials for the same muscle, as the scales differ for each comparison.

postintervention pedaling speed, percentage of external mechanical work performed, and RF3, VM3, BF3, and SM3 values for each group. Wilcoxon rank sum test was used for comparisons between the intervention and usual care groups. Spearman rank correlation coefficient was used to quantify the association between pedaling and walking speeds in each treatment group. The significance of the differential associations between pedaling and walking speeds was assessed with a regression model for walking speed, which contained treatment group, pedaling speed, and an interaction term between treatment group and pedaling speed.

## RESULTS

Subjects in this study showed coordination patterns that are characteristic of subjects with hemiparesis. Individual subject EMG data are displayed for 1 representative subject from the intervention group (Figure 2) and individual tangential crank force data for all subjects (Figure 3). The average for all subjects for the coordination measures in the pretreatment session (Paretic Work = 6.8% ± 23.6%, RF3 = 13% ± 8%, VM3 = 14% ± 8%, BF3 = 26% ± 9%, and SM3 = 33% ± 7%) are comparable to those of previous studies.[5–7]

There was no effect of the intervention on motor coordination as assessed by our quantitative measures (Table 2). When the raw data were considered, this effect was supported by a visual similarity between pre- and postintervention data in coordination, with few obvious changes being evident (Figures 2 and 3). Note that although the raw data for the rectus femoris appears to be slightly less phase-advanced for this subject (Figure 2), the group data did not support a change (Table 2 and Figure 4). The tangential force curves (Figure 3) do demonstrate a slight increase in positive work production in the posttest (increased positive area under the curve), but the change was observed for both the control and intervention group (Table 2), so there was no effect of

Table 2. Average Coordination Measures for the Different Groups

| Measure | Control ($n$ = 9) | | Intervention ($n$ = 11) | |
|---|---|---|---|---|
| | Pre | Post | Pre | Post |
| Paretic work | 6.4% (27.7) | 9.6% (33.4) | 7.1% (21.1) | 10.8% (18.0) |
| RF3 | 13% (6) | 12% (6) | 13% (9) | 11% (5) |
| VM3 | 14% (5) | 16% (8) | 14% (10) | 15% (10) |
| BF3 | 28% (9) | 31% (7) | 23% (8) | 25% (13) |
| SM3 | 34% (6) | 32% (6) | 33% (8) | 32% (11) |

Note: Numbers in parentheses are standard deviations. RF = rectus femoris; VM = vastus medialis; BF = biceps femoris; SM = semimembranosus.

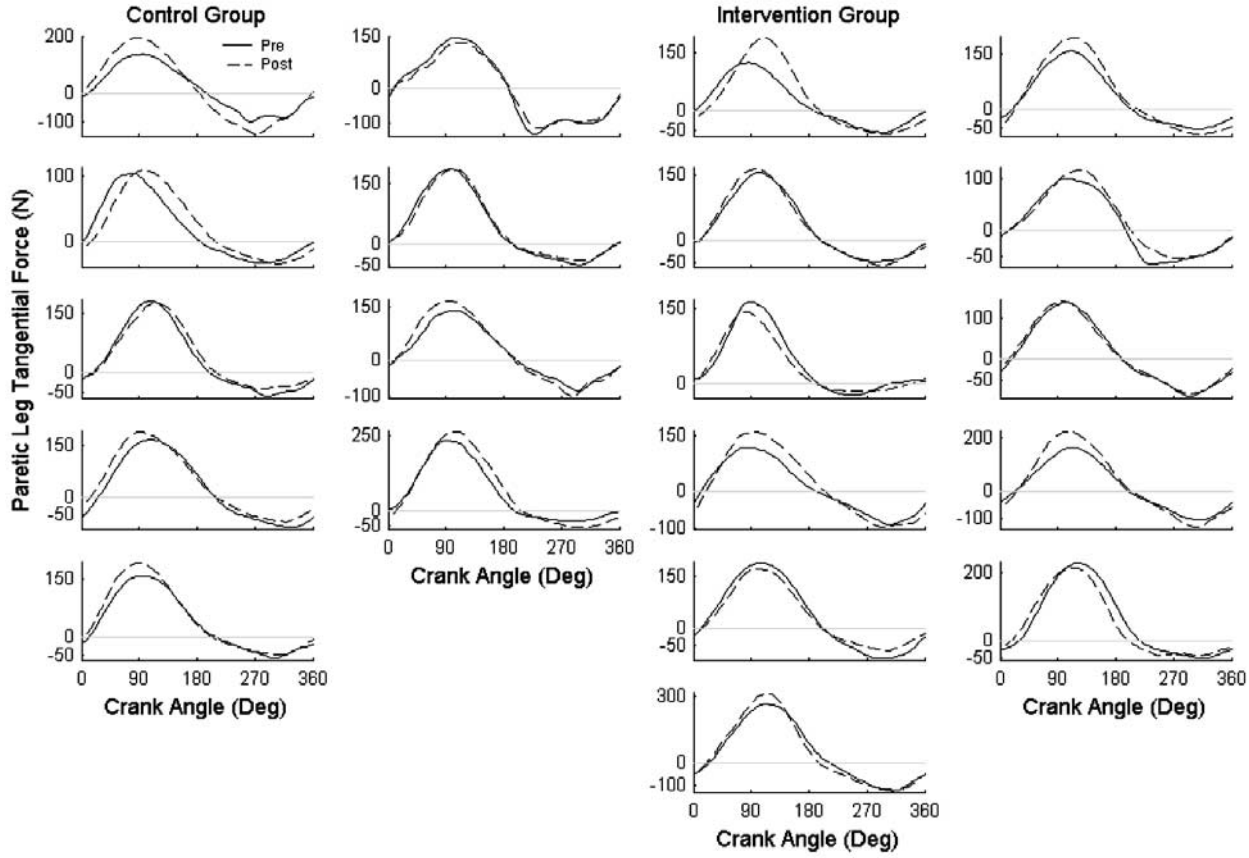


**Figure 3.** Average tangential pedal force data for each subject (pre- and postintervention) were mostly consistent. Subjects tended to have characteristic force trajectories that were relatively unchanged (regardless of their grouping).

the intervention. Group EMG data revealed similar lack of observable differences between pre- and postconditions (Figure 4).

Pedaling speed was not improved by the intervention, although pedaling faster and walking faster were associated after the intervention. The effect of the intervention on pedaling speed gain was not significant ($P$ = 0.3734), because those in the intervention group (median increase = 2.34; $P$ = 0.5195) and the control group (median increase = 2.86; $P$ = 0.1289) had similar nonsignificant increases. Nevertheless, we also tested whether pedaling faster and walking faster were associated after the intervention and found that there was a correlation for subjects in the intervention group ($r$ = 0.59 and $P$ = 0.0556) compared to the control group ($r$ = 0.03 and $P$ = 0.9322 for control group). Although the interaction representing these differential associations in treatment groups was not significant ($P$ = 0.0623) by the strict $\alpha$ = 0.05 criterion, it appears to suggest some evidence that pedaling and walking speed correlation exist only in the intervention group.

As a single group, all subjects successfully pedaled at the same speed and generated similar work pre- (cadence 41.4 ± 3.7 rpm, workload 94.0 ± 11.7 J [joules])

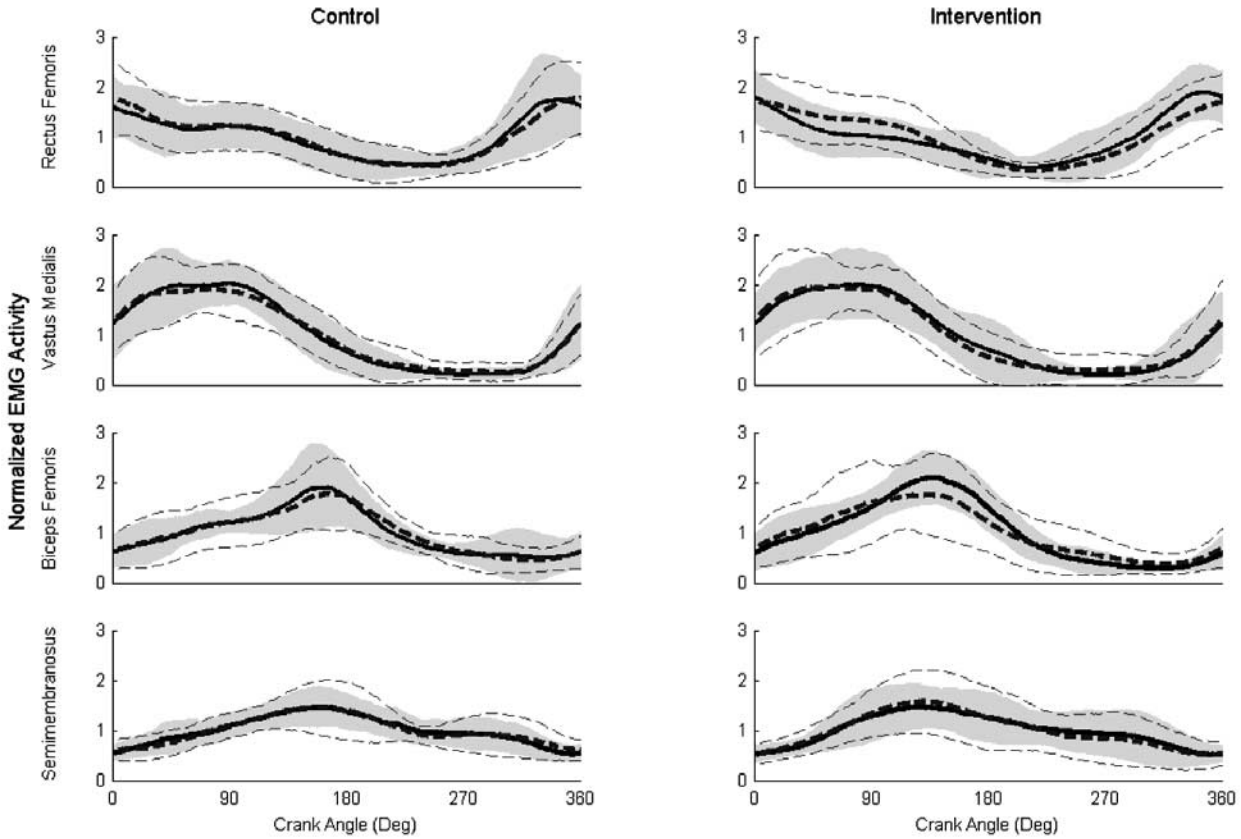


**Figure 4.** Group average EMG data (pre- and postintervention) for the individual muscles are plotted for visual display and demonstrate that there were no dramatic changes in EMG activity in pre- (solid line and shaded region indicate mean ± 1 *SD*) or postintervention (thick dashed line and region described by thin dashed lines indicate mean ± 1 *SD*).

and postintervention (cadence 41.5 ± 3.8 rpm, workload 96.0 ± 14.6 J). Cadence ranged from 32 to 49 rpm, and workload ranged from 66 J to 127 J. Previous studies have demonstrated that these coordination measures for hemiparetic persons change little with cadence and workload variations of this magnitude.[6,7] In the self-selected and fastest comfortable trials, cadences from the initial assessment were 38.4 ± 8.6 rpm and 55.984 ± 12.8 rpm, respectively, indicating that the 40 rpm test condition was relatively comfortable and well below maximum capacity for the subjects in this study.

## DISCUSSION

The goal of the present study was to determine whether motor coordination during a locomotor task was improved after an intensive home exercise program to ascertain whether there was a restitution of motor function, as opposed to adaptive or compensatory changes. This knowledge is crucial for the rational modification of future interventions. The intervention was a structured, progressive, physiologically based therapist-supervised program that targeted impairments in flexibility, strength, balance, endurance, and upper-extremity function. Subjects who received the intervention performed a large amount of pedaling to address their impaired endurance, and they substantially increased their pedaling performance over the course of rehabilitation. They performed more than 13 h of pedaling during up to 30 half-hour sessions, with all subjects progressing in intensity from the initial session to the final session. Despite this substantial pedaling practice, all measures of coordination remained unchanged as a result of the intervention, with very few subjects showing any evidence of changes in EMG timing or mechanical work production after the intervention. Thus, there was no improvement in motor coordination during pedaling as a result of the intervention.

Because reduced motor coordination measures have been correlated with poorer motor recovery[5] as assessed by a subcomponent of the Fugl-Meyer[12] lower limb assessment, they may also serve as a general assessment of motor coordination of locomotor tasks in general, in

addition to being specifically relevant to pedaling. Although the hemiparetic subjects walked faster as a result of the therapeutic exercise program,[8] the improved gait speed did not correlate with changes in motor coordination as measured in the pedaling task. In addition to walking faster as a result of the therapeutic exercise program, subjects also pedaled faster at their maximum comfortable speed (the control group subjects also increased cadence). However, again there was no improvement in the EMG measures of coordination associated with the increased speed. Thus, the increased speed resulted from better ability to use the same impaired pattern without any changes in EMG timing and most likely resulted from the observed improvements in strength and endurance.[8] This finding is consistent with those in previous studies of adaptations to changing task mechanics in hemiparetic pedaling where the subjects were able to adapt their motor pattern to the task mechanics in a manner similar to control subjects, but their pattern still remained similarly impaired.[6,7,13] Furthermore, for subjects in the intervention group, there was a correlation between pedaling faster and walking faster after the intervention. Thus, because postintervention changes in maximum comfortable pedaling speed were correlated with changes in walking speed, and because these changes in pedaling speed were not associated with changes in motor coordination, we believe that it is likely that increased walking speed may have also been gained without improved motor coordination. Several previous studies have shown that motor function can be facilitated poststroke for walking, as evidenced by improvements from locomotor training paradigms using body-weight-supported treadmill training.[14–20] This type of training has been shown to reduce energy expenditure and cardiovascular demands of hemiparetic gait in chronic stroke patients.[15,16] Significant improvements in functional balance, motor recovery, overground walking speed, and endurance were reported in a large group of poststroke patients who had been trained on a treadmill with body weight support.[21] Although there have been several studies indicating that gait speed can be increased,[8,22] it remains to be established to what extent increased gait speed represents an improvement in the central nervous system control of locomotion, as opposed to the result of nonneural factors that have been correlated with gait speed such as increased endurance,[8] increased strength,[23,24] or more efficient use of abnormal compensatory mechanisms.

Although we believe that no changes in motor coordination occurred as a result of the intervention in our study, possible limitations with respect to generalizability, reliability, and specificity need to be addressed. First, it is not clear how generalizable these results are to the whole spectrum of hemiparetic persons, because the sample size was limited ($n$= 20). Thus, it is possible that we were unable to detect an actual change in coordination. Furthermore, the included subjects were relatively high functioning (mean gait speed ~0.8 m/s). Second, because the coordination measures are not widely used, their retest reliability has not been established. We believe that the measures that we used are appropriate for detecting changes, if they had been present, because we have used them to successfully understand coordination during numerous experimental conditions.[5–7,25] We have collected the measures in approximately 100 hemiparetic persons over multiple studies. One strength of our EMG measures is that they measured percent of total activity occurring in a specific region of the pedal cycle, which removes the need for careful normalization of the absolute amplitude of excitation to make comparisons within and across subjects. Thus, many factors that contribute to the variability in amplitude of EMG activity (e.g., skin impedance, exact electrode placement, etc.) are unlikely to have a substantial influence on the coordination measures. Although this is our 1st study using the measures longitudinally, we are confident that they are appropriate because the variability between subjects was much greater than the pre-post variability within a subject. We did not see substantial pre-post variability, so it is not likely that possible changes were masked by high variability. Thus, if any changes were missed, they must be relatively subtle. Finally, the coordination measures were collected during pedaling and lack specificity because we are most interested in coordination of walking. Future development of a similar model of coordination specific to walking will be necessary to directly test this hypothesis. However, the understanding of individual muscle function in walking is only beginning to reach the level such that this goal can be achieved.[26,27] In conclusion, although limitations exist, we believe that our results accurately reflect that the subjects in this study did not improve their motor coordination as a result of the intervention.

We believe that one potential reason that we did not show an improvement in motor coordination is that the rehabilitation intervention may not have been specific enough. Although the intervention included components that targeted strength, balance, and aerobic endurance, and pedaling exercise was performed for the endurance training, the training was not designed to improve the pedaling performance by the paretic leg. Because the 2 legs are simultaneously acting on a single crank in pedaling, it is possible for the nonparetic leg to completely compensate for the paretic leg (e.g., Kautz

and Brown[5]). Thus, in this context, it is not necessary for the paretic leg performance to improve (cf. when pedal force feedback is presented to increase symmetry[28]).

In conclusion, evidence for improved motor coordination of locomotion as a result of stroke rehabilitation remains elusive. Because the finding that motor coordination did not improve after rehabilitation was based on an assessment during pedaling, we cannot rule out the unlikely result that increased gait speed during walking in our patients was the result of improved motor coordination. However, we believe that it is much more likely that the increased gait speed was achieved by a more proficient use of the same impaired pattern, as was the case for the observed increase in pedaling speed. Investigations of promising rehabilitation interventions, such as body-weight-supported treadmill gait-training paradigms that use walking-specific training,[21,22,29–31] may allow the demonstration of improved motor coordination as a result of the intervention. Although gait speed improvement can result in changes in the functional status of hemiparetic individuals[32] even when motor coordination does not change, intervention results are likely to improve if there is a better understanding of the underlying mechanisms of improved functional status.

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