ORIGINAL ARTICLE

Relationships Between Muscle Activity and Anteroposterior Ground Reaction Forces in Hemiparetic Walking

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ABSTRACT. Turns LJ, Neptune RR, Kautz SA. Relationships between muscle activity and anteroposterior ground reaction forces in hemiparetic walking. Arch Phys Med Rehabil 2007;88:1127-35.

Objective: To determine relationships between muscle activity and propulsive impulse in hemiparetic walking.

Design: Cross-sectional.

Setting: Gait analysis laboratory.

Participants: Forty-nine poststroke patients with chronic hemiparesis, stratified into hemiparetic severity subgroups based on Brunnstrom stages of motor recovery, walking at their self-selected speed.

Interventions: Not applicable.

Main Outcome Measures: Percent of muscle activity in the paretic and nonparetic legs and net anteroposterior (AP) ground reaction force impulse (ie, the time integral of the AP ground reaction force) within 4 regions of the stance phase (first double support, first and second halves of single support, and second double support).

Results: Medial gastrocnemius and soleus muscle activity correlated positively with paretic propulsion in the second half of single support and double support across all subjects and subjects grouped by hemiparetic severity. Tibialis anterior correlated negatively with paretic propulsion during preswing across all subjects and for subjects with moderate and severe hemiparesis. Rectus femoris activity also correlated negatively with preswing propulsion for the severe group. Uniarticular knee extensor activity correlated only with increased paretic braking in the first double-support phase for the severe hemiparesis group. Nonparetic leg muscle activity correlated with propulsive impulses across all subjects, but not within the severe group exclusively.

Conclusions: Paretic propulsion is strongly associated with increased plantarflexor activity and also negatively associated with increased leg flexor activity, especially in the severe hemiparesis group. These results suggest that exaggerated flexor muscle activity may counteract the effects of the plantarflexors by offloading the leg and interfering with the limb's ability to generate appropriate AP ground reaction forces. There is also evidence for specific relationships between paretic braking and nonparetic propulsive forces and changes in timing of muscle activation.

Key Words: Electromyography; Hemiparesis; Rehabilitation; Walking.

STROKE IS THE LEADING cause of long-term disability, with less than 50% of surviving stroke patients walking in the community.[1] Rehabilitation strategies often focus on improving walking,[2,3] with emphasis on achieving faster speeds because walking speed is strongly correlated with functional status[3,4] and whether a patient will be homebound or functional in the community.[1] Generating the appropriate anteroposterior (AP) ground reaction forces is essential for achieving a given walking speed because they are responsible for advancing the body center of mass (COM).

The body COM is accelerated by the propulsive (positive, anteriorly directed) AP ground reaction force and decelerated by the braking (negative, posteriorly directed) AP ground reaction force. During steady-state walking, the net braking and propulsive impulses (ie, the time integral of the negative and positive AP ground reaction forces, respectively) must be approximately equal in order to maintain a given walking speed (ie, no net acceleration because the external resistance provided by air is practically negligible). The AP ground reaction force patterns of healthy walkers are bilaterally symmetric, with a reversal from braking to propulsion near midstance, while hemiparetic ground reaction forces demonstrate substantial asymmetries between the 2 limbs with markedly different propulsion and braking patterns.[5] In general, paretic leg braking is increased with an accompanying decrease in propulsion. Thus, to maintain a given walking speed, the nonparetic leg must compensate and generate a greater propulsive impulse.[6,7] Bowden et al[5] determined that subjects with severe hemiparesis had the most profound deficits in generating paretic propulsion; some of their subjects, however, achieved community-walking status even with these deficits. This could be accomplished by compensatory generation of propulsion by the nonparetic leg or by increased hip flexor activity in the paretic leg to help advance the leg in pre- and early swing. Conversely, the subjects with mild hemiparesis in Bowden's study showed little asymmetry in propulsion generation. Subjects with moderate hemiparesis generated varying levels of propulsion with their paretic leg. Our goal in this study was to relate specific changes in muscle activity to altered bilateral ground reaction forces.

Previous modeling and simulation work suggests that altered muscle activity will have a predictable effect on ground reaction forces. Such work has shown that the plantarflexors are the primary muscles contributing to the propulsive impulse in the last half of stance during normal, unimpaired walking.[8,9] Therefore a reduction in paretic propulsion could be the result of decreased force production of these muscles because of inadequate muscle activation.[10-12] Additionally, flexor muscles, such as the rectus femoris, reduce ground reaction force during late stance.[8] Thus, flexor activity could negatively correlate with paretic propulsion in late stance, as increased leg flexor activity would act to offload the leg and decrease propulsion.

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In addition to the decreases in propulsion, altered muscle activity could also directly contribute to an increased braking impulse in early stance. Poststroke patients who take longer paretic steps relative to the nonparetic leg demonstrate increased paretic braking during early stance after paretic foot-strike.[13] The uniarticular knee extensor muscles (vasti) are among the primary contributors to the braking impulse in early stance in normal walking.[8,9] Therefore, excessive or prolonged force generation of these muscles during early stance could substantially increase paretic braking. In addition, premature activation of muscles not normally active during early stance, such as the plantarflexors,[14-16] may also generate increased braking.

To further understand and delineate the possible neuromotor mechanisms responsible for the disrupted ground reaction force patterns in hemiparetic populations, we analyzed the relationships between muscle electromyographic and ground reaction force data collected from the same 49 subjects with poststroke hemiparesis reported by Bowden et al.[5] Because deficits in paretic propulsion vary across hemiparetic severity and self-selected walking speed,[5] we examined these relationships across the entire population of subjects, as well as within subgroups based on hemiparetic severity. We hypothesized that: (1) plantarflexor activity would positively correlate with paretic propulsion in late stance; (2) uniarticular knee extensor activity would positively correlate with paretic braking in early stance; (3) flexor activity would negatively correlate with paretic propulsion in late stance; and (4) there would be relationships between altered muscle activity and propulsive impulses in the nonparetic leg resulting from compensatory patterns.

METHODS

Forty-nine subjects with chronic hemiparesis (42 men, 7 women; age, 62.7±10.2y; time since stroke, 4.25±3.67y; affected side: left, 25; right, 24) were recruited at the Rehabilitation Research and Development Center at the Palo Alto Department of Veterans Affairs Medical Center. The data presented here were collected (but not reported) as part of a study that investigated the relationships between gait characteristics and bone density in chronic stroke survivors.[17] Inclusion criteria were: ability to walk 10m in 50 seconds or less without contact assistance, at least 12 months poststroke, and unilateral weakness. Subjects were excluded if they had more than 1 previous cerebrovascular incident, had any orthopedic or neurologic conditions in addition to the stroke, or if they were unable to provide informed consent. Written informed consent was obtained from all participants and the Stanford University Administrative Panel on Human Subjects in Medical Research approved the protocol.

We used Brunnstrom's stages of recovery[18] to determine hemiparetic severity for each subject because the study participants differed in ability to perform movements outside of extensor and flexor synergy patterns. Subjects were classified as severely impaired with a Brunnstrom stage 3 score (n=19), moderately impaired with a Brunnstrom stage 4 or 5 score (n=20), and mildly impaired with a Brunnstrom stage 6 score (n=10). While the reliability of Brunnstrom stages has not been established independently, Brunnstrom stage determination closely follows the scoring of the valid and reliable lower-extremity Fugl-Meyer Assessment (FMA),[19] specifically the items that measure the ability to move in and out of synergy. We categorized subjects based on Brunnstrom's stages because it was strongly related to the AP propulsive force generating ability reported in our previous study.[5] Nevertheless, we also measured lower-extremity FMA. Subjects were permitted to use their usual mobility aids (canes or ankle-foot orthoses) during testing. Self-selected walking speed and spatiotemporal parameters were measured as subjects walked on a 4.3-m long GAITRite portable walkway system.[a] Additionally, electromyographic and ground reaction force (GRF) data were acquired as subjects walked at their self-selected speed along a 10-m walkway. Surface electromyographic activity was recorded from the tibialis anterior, medial gastrocnemius, soleus, rectus femoris, vastus lateralis, biceps femoris, semimembranosus, and gluteus medius from both legs. Electromyographic data were sampled at 2000Hz. Three-dimensional ground reaction forces were collected from both legs using 3 embedded force platforms[b,c] and were sampled at 200Hz.

We collected a minimum of 4 and a maximum of 15 trials for each subject to ensure adequate foot contact on the forceplate. Self-selected walking speeds, step lengths, electromyographic activity, and ground reaction forces were determined by averaging multiple trials. We computed a step-length ratio (SLR) to assess the relative asymmetry between the paretic and nonparetic leg by dividing the paretic step length by the nonparetic step length.

Electromyographic Data Collection Protocol

The subject's skin was cleaned with alcohol before the electrodes were applied and, when necessary, hair was shaved to ensure good contact. Electromyography electrodes[d] were placed over the muscle belly so that the electrode nodes were in parallel with the muscle fibers. Silver-silver chloride electrodes (interelectrode distance, 22mm; diameter, 8mm) were secured with prewrap and tape. The electrodes provided 35 times preamplification and their input impedance was greater than 15MΩ at 100Hz. The common mode rejection ratio was 87dB at 60Hz. Amplifier gain was selectable from 500 to 10,000 times, with a bandwidth of 20 to 4000Hz.

Forceplate and Electromyographic Data Processing

Electromyographic and force data were processed using customized Matlab programs.[e] The force data were filtered with a fourth-order zero-phase shift low-pass Butterworth filter with a cutoff frequency of 20Hz, and then normalized by bodyweight. Electromyographic data were filtered with a fourth-order high pass Butterworth filter at 40Hz. The data were debiased, rectified, and smoothed with a fourth-order zero-phase shift low-pass Butterworth filter with a cutoff frequency of 20Hz.

Data for the stance phase were analyzed as average values within 4 bins defined by stance phase events (determined from the average spatiotemporal data when contralateral forceplate measures were unavailable). The stance phase (defined as heel-strike to toe-off) was subdivided into bins for each leg to roughly correspond with braking (bin 1, bin 2) and propulsive (bin 3, bin 4) phases that occur in normal, unimpaired walking (fig 1). Bin 1 corresponded to double-limb support following foot strike, bin 2 corresponded to the first 50% of single-limb stance, bin 3 corresponded to the second 50% of single-limb stance, and bin 4 corresponded to double-limb support preswing (see fig 1).

The horizontal component of the impulse was calculated to quantify the amount of braking and propulsion produced by each leg. The net impulse was determined for each bin by computing the time integral of the AP ground reaction force within that bin. Negative bin impulses indicated net braking for the corresponding bin and positive bin impulses indicated net propulsion for the corresponding bin. The rectified electromyographic signal was summed within each bin (ie, the integrated electromyographic activity within that bin), and then the sum in each bin was divided by the total sum of all bins (ie, the total integrated electromyographic activity for that muscle over the gait cycle) to give the percentage of the rectified electromyographic signal that occurred within

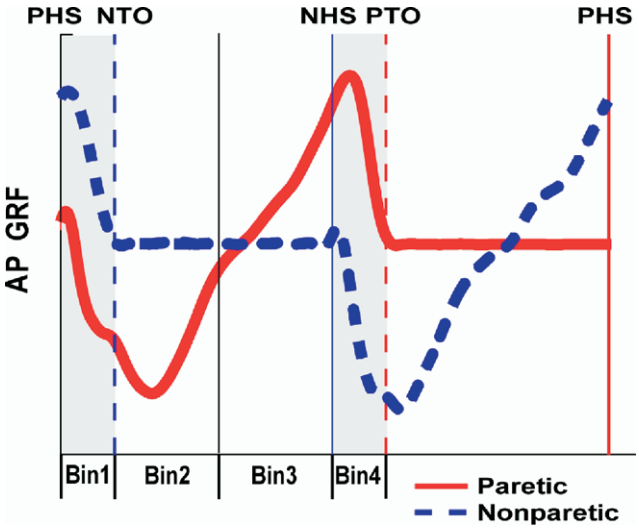

Fig 1. Bins associated with the AP ground reaction forces of the paretic leg are illustrated (solid line). The gray shaded regions represent double-support phases. Abbreviations: NHS, nonparetic heel-strike; NTO, nonparetic toe-off; PHS, paretic heel-strike; PTO, paretic toe-off.

each bin. Thus, this is a hybrid measure that includes information related to both the magnitude and timing. Because this measure does not actually measure absolute magnitude or absolute timing, we refer to the measure as reflecting "relative timing."

To present the horizontal ground reaction force data, we averaged them across trials by fitting the data points with a cubic spline and interpolating the resulting equations at even percent increments of the gait cycle (defined as heel-strike to the following heel-strike for each leg). These average curves are presented in figures 2–4, but they were not used for analysis.

Statistical Analysis

We performed correlation analyses to identify relationships between the electromyographic activity and net AP ground reaction force impulses within each bin by computing Pearson correlation coefficients between the variables. Analyses were performed for the paretic and nonparetic legs across the entire group, then for subgroups based on hemiparetic severity (ie, those classified with severe, moderate, and mild hemiparesis). We used Minitab[f] and SPSS[g] for statistical analysis and set statistical significance at P less than .05. We did not correct for multiple comparisons because not all measures were independent (see Discussion).

RESULTS

Average lower-extremity FMA scores for the groups with severe, moderate, and mild hemiparesis, respectively, were 18 of 34, 23 of 34, and 27 of 34 of the total possible points (note that complete lower-extremity FMA scores were not available for 6 subjects). Fifteen subjects used their usual unilateral mobility aid(s) (cane and/or ankle-foot orthosis) during testing.

We analyzed a total of 659 AP ground reaction force records. There was an associated contralateral forceplate record for 436 records (66%). To perform the bin analysis, 2 contralateral events must be identified for each step (ie, contralateral toe-off defines the end on bin 1 and contralateral heel-strike defines the beginning of bin 4). Thus, 436 (33%) of the 1318 contralateral events required for bin analysis were defined from the forceplate data. When a contralateral event was not available from the forceplate data, we used the average percentage of the stance phase in which that event occurred during the GAITRite trials to define that event. As a check on this methodology, we compared the spatiotemporally derived assumed value with the actual forceplate-determined value for the 218 events in which the contralateral events were available. The average error in the bin definition was 2.4%±1.9% of the paretic gait cycle.

Walking speeds for the hemiparetic subjects ranged from 0.11 to 1.34m/s. Based on their Brunnstrom stage, 10 subjects were classified as mild, 20 were classified as moderate, and 19 were classified as severely hemiparetic. Analysis of the SLR showed that 22 subjects walked nearly symmetrically (SLR range, 0.9−1.1), while 23 took longer paretic step lengths (SLR>1.1) and 4 took longer nonparetic step lengths (SLR<0.9). Ratios between 0.9 and 1.1 were considered nearly symmetrical because these values indicate an SLR that is ±10% of perfect symmetry. Healthy adults walk with an SLR of 1.0 ±5%, so bounds of ±10% were considered to be conservative. Outside of these bounds subjects were considered clearly asymmetric.

Figures 2, 3, and 4 illustrate the AP ground reaction force, net bin impulse, and net bin electromyographic data for representative subjects classified as mild, moderate, and severe, respectively, who generated different amounts of paretic and nonparetic leg propulsion and braking. Mildly impaired subjects usually generated fairly symmetrical AP ground reaction force patterns with equivalent paretic and nonparetic braking and propulsive impulses (see fig 2) (compare AP Imp between nonparetic and paretic bins). Compared with less impaired subjects, however, more impaired subjects primarily generated braking with the paretic leg, which was accompanied by a decrease in braking and an increase in propulsion in the nonparetic leg (see figs 3, 4) (compare AP Imp between paretic and nonparetic bins). Also, some subjects had appropriate paretic plantarflexor activity duration and timing (see fig 2) (paretic column, medial gastrocnemius, and soleus, bin 2 and bin 3), while others experienced prolonged (see fig 3) (paretic column, medial gastrocnemius, and soleus bins 1−3) or premature (see fig 4) (paretic column, medial gastrocnemius, and soleus bin 1) activity in these muscles.

The results in the individual data were confirmed in the group analyses (tables 1, 2). In the results below, we present all correlations related to our 4 stated hypotheses.

Correlations Between Plantarflexor Activity and Paretic Propulsion in Late Stance

When all subjects were included in the analysis, there were significant positive correlations for the paretic medial gastrocnemius (P=.000) and soleus (P=.001) with the bin 3 impulse (see table 1). Not all correlations were found, however, when subjects were analyzed by hemiparetic severity. Soleus activity correlated positively with the net bin 4 impulse (P=.007) in subjects with mild hemiparesis, while medial gastrocnemius activity correlated positively with the bin 3 impulse (P=.024) in subjects with moderate hemiparesis (see table 2). In subjects with severe hemiparesis, the net bin 3 impulse correlated positively with the medial gastrocnemius (P=.029) and soleus (P=.014) activity, while the net bin 4 impulse correlated positively with medial gastrocnemius activity (P=.038). Thus, our hypothesis that

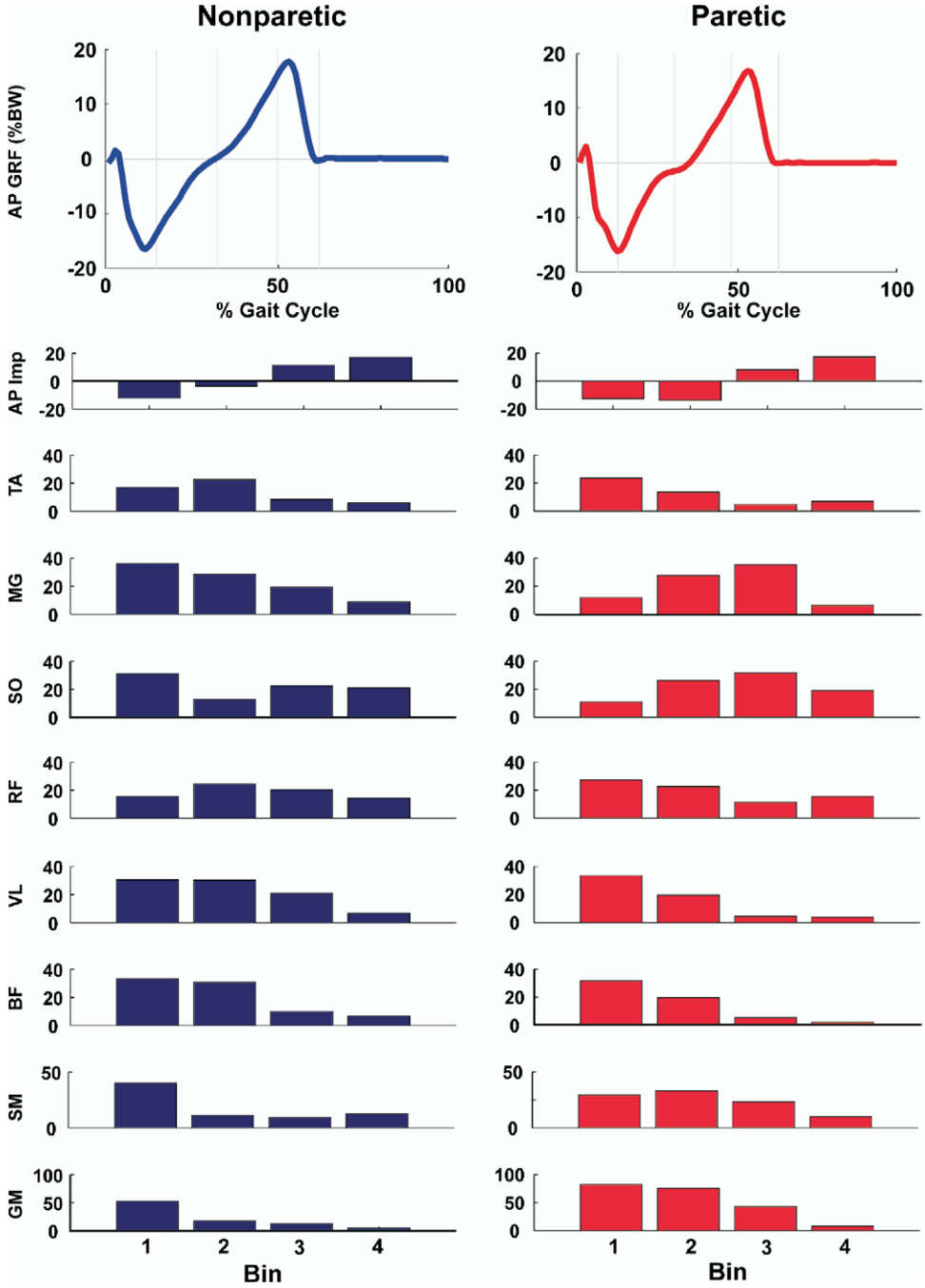

Fig 2. AP ground reaction force, impulse, and bin electromyographic data for a representative subject classified as mildly hemiparetic with a self-selected walking speed of 1.26m/s. Net bin impulses are similar, as is the duration of stance spent on each leg. Vertical lines in the ground reaction force plot indicate bin boundaries. Note that bins 1 through 4 correspond to the stance phase only. The units for the AP impulse (AP Imp) and bin electromyography are percentage of body weight (BW) × seconds and percentage of total integrated electromyography, respectively. Abbreviations: BF, biceps femoris; GM, gluteus medius; MG, medial gastrocnemius; RF, rectus femoris; SM, semimembranosus; SO, soleus; TA, tibialis anterior; VL, vastus lateralis.

plantarflexor activity would positively correlate with paretic propulsion in late stance was supported.

Correlations Between Uniarticular Knee Extensor Activity and Paretic Braking in Early Stance

As a group, there was no correlation between vastus lateralis activity and paretic braking in bins 1 or 2, indicating the hypothesis that uniarticular knee extensor activity would positively correlate with paretic braking in early stance was not supported. There was, however, a significant, negative correlation between the vastus lateralis and the paretic net bin 1 impulse ($P=.048$) for the severe group (see table 2), indicating vastus lateralis activity increased braking. There were no correlations found in the mild or moderate groups.

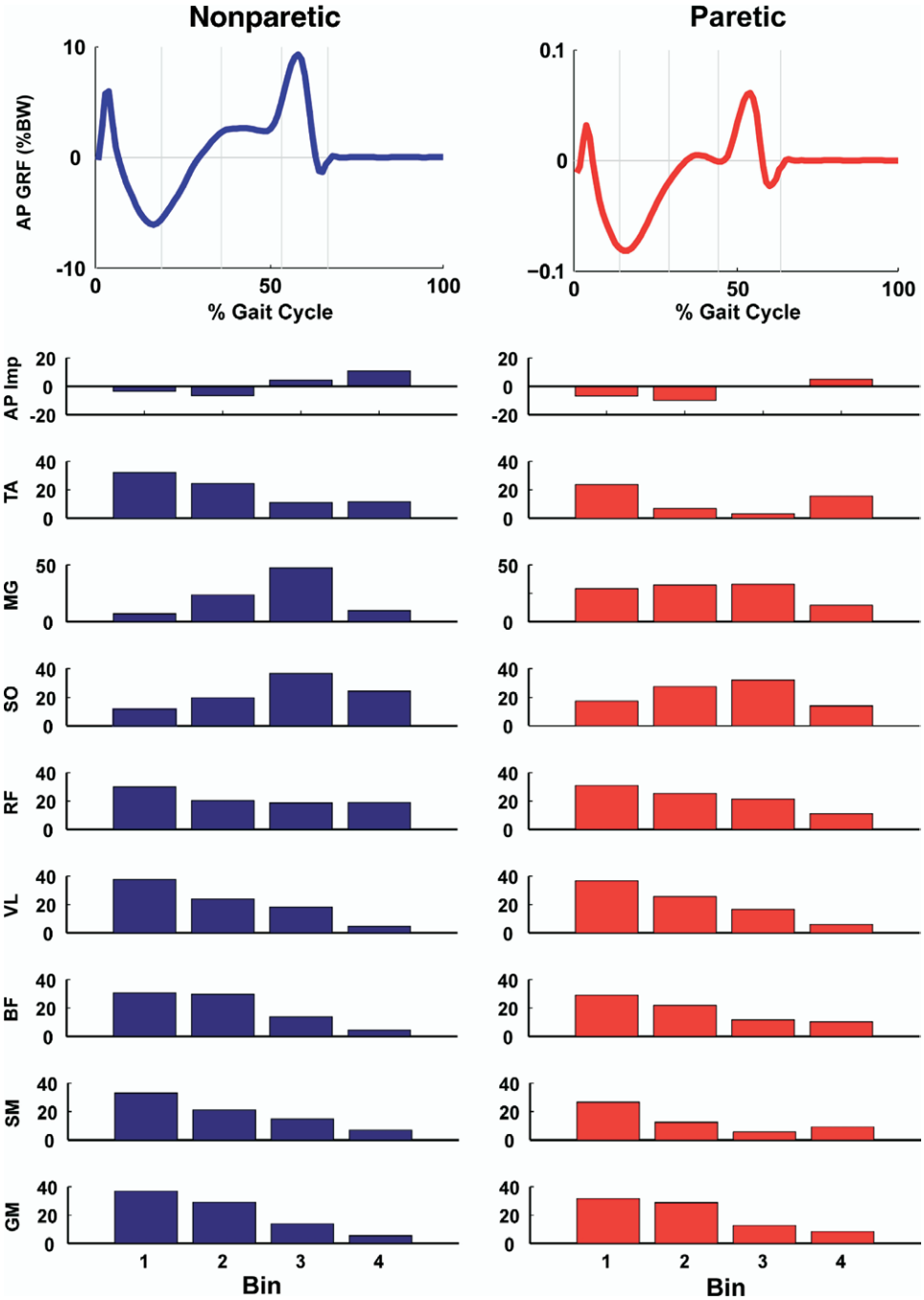

Fig 3. AP ground reaction force, bin impulse, and bin electromyographic data for a representative subject classified as moderately hemiparetic with a self-selected walking speed of .73m/s. Net bin impulses are asymmetrical, however this subject generated some paretic propulsion in bin 4. Vertical lines in the ground reaction force plot indicate bin boundaries. Note that bins 1 through 4 correspond to the stance phase only. The units for the AP impulse (AP Imp) and bin electromyography are percentage of body weight × seconds and percentage of total integrated electromyography, respectively. Abbreviations: see fig 2 legend.

Correlations Between Flexor Activity and Paretic Propulsion in Late Stance

As a group, tibialis anterior activity correlated negatively with the bin 4 impulse (P=.000) (see table 1). There was no correlation with rectus femoris or semimembranosus activity. In subjects with mild hemiparesis, tibialis anterior activity correlated positively with the net bin 3 impulse (P=.034), while for those subjects with moderate hemiparesis, a significant, negative correlation was found for the bin 4 impulse with tibialis anterior activity (P=.028) (see table 2). For subjects with severe hemiparesis, significant, negative correlations were found for the net bin 4 impulse with tibialis anterior (P=.001) and rectus femoris (P=.006) activity. Thus, the hypothesis that flexor activity would

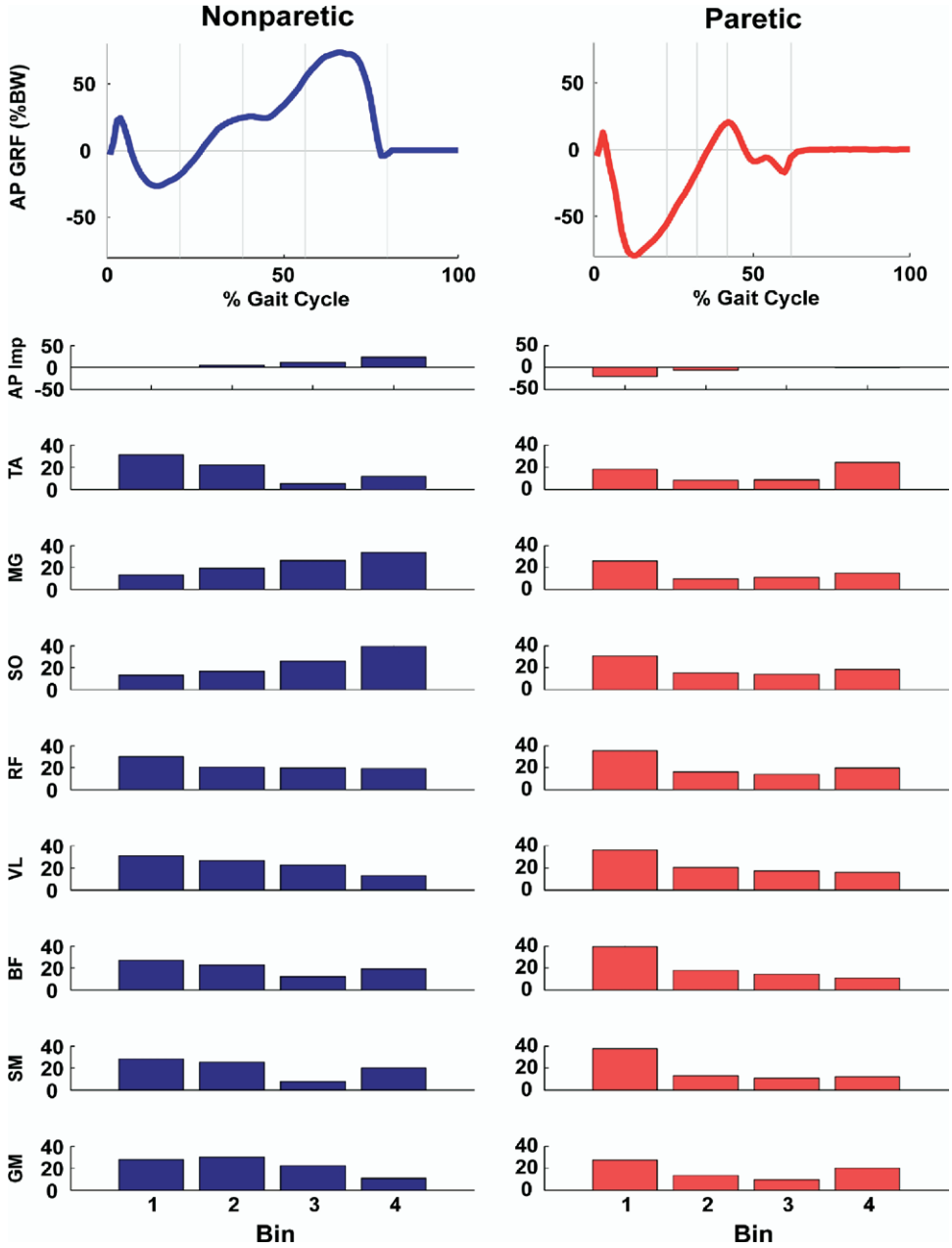

Fig 4. AP ground reaction force, bin impulse, and bin electromyographic data for a subject classified as severely hemiparetic with a self-selected walking speed of .44m/s. Net bin impulses are asymmetrical, with the paretic leg primarily generating braking and the nonparetic leg primarily generating propulsion. Vertical lines in the ground reaction force plot indicate bin boundaries. Note that bins 1 through 4 correspond to the stance phase only. The units for the AP impulse (AP Imp) and bin electromyography are percentage of body weight × seconds and percentage of total integrated electromyography, respectively. Abbreviations: see fig 2 legend.

correlate negatively with paretic propulsion in late stance was partially supported, most strongly in the severe hemiparesis group.

Correlations Between Nonparetic Muscle Activity and Propulsion

As a group, nonparetic leg medial gastrocnemius ($P=.002$) and soleus ($P=.007$) activity correlated positively with the bin 1 impulse, while semimembranosus activity correlated negatively with the bin 2 impulse ($P=.034$) (see table 1). In the nonparetic leg, gluteus medius ($P=.015$) and tibialis anterior ($P=.040$) activity correlated positively with the bin 3 impulse in subjects with mild hemiparesis, while net bin 4 impulse correlated positively with vastus lateralis ($P=.005$) and gluteus medius ($P=.043$) activity (see table 2). For the moderate group, net bin 4 impulse correlated negatively with medial gastrocnemius activity ($P=.041$), while there were no significant correlations for the nonparetic leg in the severe group. Thus, there is support for the hypothesis that there would be relationships between altered muscle activity and propulsive impulses in the nonparetic leg as a result of compensatory patterns.

Table 1: Correlations of Braking and Propulsive Ground Reaction Force Impulses With Muscle Activity for All Hemiparetic Subjects

Bin Impulse	Muscle	*r*	*P*
Paretic bin 2 impulse	SM	−.391	.007
Paretic bin 3 impulse	MG	.514	.000
Paretic bin 3 impulse	SO	.451	.001
Paretic bin 3 impulse	GM	.355	.017
Paretic bin 4 impulse	TA	−.626	.000
Nonparetic bin 1 impulse	MG	.446	.002
Nonparetic bin 1 impulse	SO	.386	.007
Nonparetic bin 2 impulse	SM	−.310	.034

NOTE. Braking impulses are negative and usually dominate bins 1 and 2 while propulsive impulses are positive and usually dominate bins 3 and 4. Thus, a negative correlation in bins 1 and 2 tended to indicate that as muscle activity increased, braking increased, whereas a negative correlation in bins 3 and 4 tended to indicate that as muscle activity increased, propulsion decreased. Similarly, a negative correlation in bins 1 and 2 occurred when muscle activity decreased while braking decreased, whereas a negative correlation in bins 3 and 4 indicated that as muscle activity decreased, propulsion increased.
Abbreviations: GM, gluteus medius; MG, medial gastrocnemius; SM, semimembranosus; SO, soleus; TA, tibialis anterior.

DISCUSSION

Our overall aim in this study was to further our understanding of the neuromotor mechanisms responsible for disrupted ground reaction force patterns in hemiparetic walking. Specifically, we sought to identify relationships between muscle activity and AP ground reaction forces, inasmuch as reduced paretic propulsion is directly related to achieving a functional walking speed for this population.[5] Analysis of the subjects as 1 group and then across groups separated by hemiparetic severity revealed that activity of the plantarflexor muscles was consistently associated with the generation of propulsion by the paretic leg. Conversely, activity of flexor muscles during preswing appeared to counteract plantarflexor activity in subjects with severe hemiparesis, thus contributing to a reduced paretic propulsive impulse. Additionally, inappropriate nonparetic (eg, soleus, medial gastrocnemius, semimembranosus) and paretic (eg, tibialis anterior) muscle activity might indirectly contribute to increased paretic braking in early stance and reduced paretic propulsion in preswing, respectively, by altering gait mechanics. Because all of the muscles, as well as the particulars of the walking mechanics, influence the impulse, it should not be expected that any single correlation would be particularly high. Thus, we did not correct for multiple comparisons in our analyses, which has the potential for increased type I errors. We believe that this was appropriate because our data analysis resulted in several significant correlations that were nearly all consistent with our a priori hypotheses based on our previous work that impaired propulsion is related to both decreased plantarflexor and increased limb flexor activations. Thus, the data are sufficient to meet our purpose in this study, which was to provide evidence that changes in muscle timing can be related to the propulsive force generation.

Based on previous analyses of normal unimpaired walking,[8,9] we predicted that plantarflexor activity would correlate positively with paretic propulsion in late stance. There was strong evidence that paretic propulsion was associated with increased plantarflexor activity in late single-limb stance (bin 3) and less evidence in preswing (bin 4), because bin 3 medial gastrocnemius activity correlated positively with paretic propulsion when the analysis was performed across all subjects (see table 1) and within groups of subjects with moderate and severe hemiparesis (see table 2). Similarly, bin 3 soleus activity correlated positively with paretic propulsion when the analysis was performed across all subjects (see table 1) and within the group with severe hemiparesis (see table 2). The only significant correlations in bin 4 were a positive correlation with paretic propulsion for soleus activity in subjects with mild hemiparesis and a negative correlation between propulsion and medial gastrocnemius activity in the most severe group. The negative correlation for the medial gastrocnemius likely relates to subjects with very prolonged paretic preswing phases[13] that usually indicate poor propulsion. In that case, medial gastrocnemius activity may be either weak, ineffective at overcoming additional flexor activity, or perhaps is abnormally associated with increased limb flexor recruitment. These results agree with those of previous studies showing that plantarflexor activity is important for attaining faster walking speeds in poststroke hemiparetic populations.[10,20]

Reduced paretic propulsion during preswing may result from several factors, including decreased neural drive, muscle atrophy, and/or the position of the leg (eg, if the nonparetic leg takes a short step, the plantarflexors may be put at a biomechanic disadvantage). The reduced paretic propulsion may also be the result of exaggerated flexion at the ankle, knee, or hip joint acting to offload the leg. Flexor activity at this time would counteract the effects of the plantarflexors, thus interfering with the limb's ability to generate appropriate ground reaction forces. Our results, particularly those from the group with severe hemiparesis, support this suggestion. Negative correlations for the paretic tibialis anterior with the bin 4 impulse were

Table 2: Correlations of Braking and Propulsive Ground Reaction Force Impulses With Muscle Activity for Subjects Grouped by Hemiparetic Severity

Functional Status	Bin Impulse	Muscle	*r*	*P*
Mild	Paretic bin 2 impulse	TA	−.708	.022
	Paretic bin 2 impulse	SO	.737	.015
	Paretic bin 3 impulse	TA	.670	.034
	Paretic bin 4 impulse	SO	.786	.007
	Nonparetic bin 3 impulse	TA	.654	.040
	Nonparetic bin 3 impulse	GM	.738	.015
	Nonparetic bin 4 impulse	VL	.802	.005
	Nonparetic bin 4 impulse	GM	.648	.043
Moderate	Paretic bin 2 impulse	SM	−.563	.012
	Paretic bin 3 impulse	MG	.516	.024
	Paretic bin 4 impulse	TA	−.502	.028
	Nonparetic bin 4 impulse	MG	−.473	.041
Severe	Paretic bin 1 impulse	VL	−.472	.048
	Paretic bin 1 impulse	BF	−.497	.036
	Paretic bin 3 impulse	MG	.514	.029
	Paretic bin 3 impulse	SO	.57	.014
	Paretic bin 4 impulse	TA	−.722	.001
	Paretic bin 4 impulse	MG	−.491	.038
	Paretic bin 4 impulse	RF	−.616	.006
	Paretic bin 4 impulse	GM	−.608	.01

NOTE. Braking impulses are negative and usually dominate bins 1 and 2, whereas propulsive impulses are positive and usually dominate bins 3 and 4. Thus, a negative correlation in bins 1 and 2 tended to indicate that as muscle activity increased, braking increased, whereas a negative correlation in bins 3 and 4 tended to indicate that as muscle activity increased, propulsion decreased. Similarly, a negative correlation in bins 1 and 2 occurred when muscle activity decreased while braking decreased, whereas a negative correlation in bins 3 and 4 indicated that as muscle activity decreased, propulsion increased.
Abbreviations: see fig 2.

found across all subjects and for subjects with moderate and severe hemiparesis. These results suggest that tibialis anterior activity is associated with decreased paretic propulsion in general and particularly with more impaired subjects. Interestingly, mildly impaired subjects do not exhibit this trend, which is consistent with patterns experienced by healthy walkers. Furthermore, there were negative correlations for the rectus femoris and tibialis anterior with paretic impulse during preswing in the severe group, indicating these subjects may be experiencing inappropriate flexor synergies[18] in the paretic leg as the nonparetic leg begins its extension phase. The paretic leg has been shown to be strongly influenced by the sensorimotor state on the nonparetic leg during a pedaling paradigm.[21] This confounding affect of hip and ankle flexion may offload the leg such that inadequate hip extension leads to insufficient posterior foot placement during push-off. These findings agree with de Quervain et al[22] who found that patients who walked slower were unable to perform movements outside of a mass synergy pattern, such as dorsiflexion of the ankle with extension of the hip.

We hypothesized that prolonged or increased uniarticular knee (vastus lateralis) extensor activity may contribute to increased paretic braking in early stance because these muscles are the primary contributors to the braking impulse during normal unimpaired walking.[9,23] These studies have also shown that the uniarticular hip extensors also contribute significantly to braking in early stance. We were, however, only able to collect electromyography from the vastus lateralis in this study. As a group, subjects did not exhibit abnormal vastus lateralis activity in early stance, suggesting additional factors may be responsible for increased paretic braking (although the severe group did demonstrate this relationship for vastus lateralis—see below). Altered gait mechanics associated with increased paretic step length, rather than active paretic leg muscle force generation, may contribute to excessive braking experienced by the paretic leg during early stance. In general, although step length asymmetry is very heterogeneous between subjects, many hemiparetic subjects take longer paretic step lengths relative to the nonparetic leg and spend more time in nonparetic stance.[24-26] The increased paretic step results in increased paretic braking,[13] most likely the result of a larger braking vector associated with exaggerated anterior placement of the paretic foot relative to the COM at foot-strike.

Analysis of the subjects grouped by hemiparetic severity revealed relationships unique to the severe group. We found evidence that abnormal muscle activity in early stance contributed to excessive paretic braking, whereas decreased paretic propulsion in preswing may result from altered mechanics resulting from exaggerated hip flexion. The vastus lateralis and biceps femoris correlated positively with paretic braking in bin 1, suggesting that increased activity of these muscles was associated with increased braking in early stance. The vastus lateralis extends the knee[8,27] while the biceps femoris acts to extend the hip and knee at this time,[27] therefore inappropriate force production or prolonged activation of these muscles[28] would cause the paretic leg to exert a greater braking force. With regard to decreased paretic propulsion, the gluteus medius, which contributes to hip extension in late stance,[27] correlated negatively with paretic propulsion during preswing, suggesting that reduced propulsion is related to an inability to achieve adequate hip extension[5] during push-off.

Study Limitations

A potential limitation of this study is that the method we used does not consider the mechanics of foot placement with respect to the pelvis. In normal walking, the pelvis crosses over the foot near midstance, with the pelvis spending an equivalent amount of time posterior (braking) and anterior (propulsion) relative to the foot. Nonparetic medial gastrocnemius and soleus activity correlated negatively with nonparetic braking in bin 1, which suggests that the medial gastrocnemius and soleus are associated with increased nonparetic propulsion during the increased paretic preswing phase. In general, nonparetic steps are shorter, and the nonparetic leg may spend more time posterior to the pelvis rather than anterior to it, which would explain the increased nonparetic propulsive impulse. Unfortunately, the method we used does not account for pelvis-foot interactions to elucidate how mechanics play a role. Future work will be directed toward investigating the relationship between foot-pelvis mechanics with the generation of propulsion and braking.

Another potential limitation of this study is the number of muscles for which electromyographic data were collected. While most major muscle groups were analyzed, a significant omission was collection of data for the uniarticular hip flexors. This was because of the inaccessibility of these muscles with surface electrodes. In healthy subjects, hip flexors provide the second largest amount of work in walking,[29] and in poststroke populations slow gait velocity has been linked to an inability to achieve adequate hip extension,[22] which could be the result of inappropriate hip flexor activity. Although there was rectus femoris activity, it is not known whether it was synergistic with the uniarticular hip flexors or adductor longus.

An additional limitation is that, in many instances, (eg, whenever consecutive foot strikes were not captured on individual forceplates) some of the bins were defined based on average values determined in separate trials collected immediately prior to forceplate data collection. Identifying the 4 bins required us to determine the timing of 4 events (ipsilateral and contralateral heel-strike and toe-off). The force records always determined the 2 ipsilateral events. As reported in the results, forceplate records determined 33% of all contralateral events. Thus, the timing of bins could be defined directly from the data for 1754 (67%) of the 2636 events that were determined for the 659 force records. In addition, we believe that the error introduced by using the spatiotemporal data was minimal because our check revealed that the average change in the bin boundary by using the spatiotemporally defined bins was only 2.4% of the gait cycle. The use of spatiotemporally defined bins would have been substantially more problematic if we had failed to find significant correlations between electromyographic data and propulsive impulses. Because we were able to find support for most of our a priori hypotheses within the data, however, we believe that we have shown persuasively that impaired propulsion is related to both decreased plantarflexor and increased limb flexor activity.

CONCLUSIONS

This study presents a first step toward understanding the neuromotor mechanisms responsible for the disrupted AP ground reaction forces in hemiparetic walking. Future studies will incorporate kinematic variables and spatiotemporal parameters so that the complex relationships between the pelvis, foot placement, and muscle function can be further elucidated. Even so, electromyography can only provide limited insight into specific muscle force deficits resulting from the stroke. Future work will also incorporate modeling and simulation techniques to analyze individual muscle force contributions to the disrupted AP ground reaction force patterns. This will provide additional insight into motor impairments and muscle coordination deficits that limit walking performance and for designing effective therapy for people with poststroke hemiparesis.

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