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Challenging Standing Balance Reduces the Asymmetry of Motor Control of Postural Sway Poststroke

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ABSTRACT

Background: Ankle plantarflexor muscle impairment contributes to asymmetrical postural control poststroke. Objective: This study examines the relationship of plantarflexor electromyography (EMG) with anterior–posterior center of pressure (APCOP) in people poststroke during progressive challenges to standing balance. Methods: Ten people poststroke and 10 controls participated in this study. Anteriorly directed loads of 1% body mass (BM) were applied to the pelvis every 25–40 s until 5%BM was reached. Cross-correlation values between plantarflexor EMG and APCOP (EMG:APCOP) position and velocity were compared. Results: EMG:APCOP velocity correlations were stronger than EMG: APCOP position across all muscles ($p < .01$), and correlations were predominately stronger in the nonparetic compared with the paretic leg ($p < .05$). Increasing challenge to standing balance reduced asymmetry of EMG:APCOP relationships. Conclusions: These data suggest that sensory information reflected in APCOP velocity interacts more strongly with plantarflexor activity in people poststroke and controls than APCOP position. Furthermore, increasing challenge to standing balance reduces postural control asymmetry between legs poststroke.

Keywords: paresis, plantarflexors, postural control, stroke

INTRODUCTION

During quiet stance, maintaining the body's center of mass within the base of support (BOS) is largely achieved by modulation of muscle activity about the ankle (Di Giulio, Maganaris, Baltzopoulos, & Loram, 2009; Horak & Nashner, 1986; Winter, Palta, Ishac, & Gage, 2003). Using cross-correlation analysis between electromyography (EMG) signals of the ankle plantarflexor muscles and force platform recordings of postural sway, it has been shown that, in the sagittal plane, modulation of the ankle plantarflexor muscle activity is moderately correlated with, and precedes, postural sway position to maintain postural control in healthy controls (Gatev, Thomas, Kepple, & Hallett, 1999; Masani, Popovic, Nakazawa, Kouzaki, & Nozaki, 2003; Masani, Vette, Abe, Nakazawa, & Popovic, 2011). Based on these findings, it has been suggested that the central nervous system modulates plantarflexor muscle activity in anticipation of body sways (Gatev et al., 1999; Masani et al., 2003, 2011). In healthy individuals, postural sway velocity has been shown to demonstrate a stronger relationship than postural sway position with plantarflexor EMG modulation (Masani et al., 2003; Portela, Rodrigues, & de Sa Ferreira, 2014). Postural sway velocity is suggested to contain greater afferent

information (direction and speed of position change) than sway position, hence facilitating more effective motor commands to control standing balance (Masani et al., 2003; Portela et al., 2014).

Poor balance following stroke is associated with a loss of independent mobility and an increased risk of falls, which could lead to injury and further disability (Garland, Gray, & Knorr, 2009). Early research aimed at improvement in balance following stroke focused on the presence of asymmetrical weight-bearing and restoring symmetrical weight-bearing to improve standing balance. However, outcomes using this approach were only weakly linked to functional improvement (Geurts, de Haart, van Nes, & Duysens, 2005). This is likely due to measurement of weight-bearing failing to reflect the complexity of maintaining postural control when standing balance is challenged. More recently, greater emphasis has been placed on understanding the impairment of postural control following stroke using advanced measures (de Haart, Geurts, Huidekoper, Fasotti, & van Limbeek, 2004; Marigold & Eng, 2006; Roerdink, Geurts, de Haart, & Beek, 2009; van Asseldonk et al., 2006). Standing balance impairment following stroke is suggested to be strongly related to decreased motor coordination about the ankle (Dickstein & Abulaffio, 2000; Hocherman, Dickstein, Hirschbiene, & Pillar, 1988).

Following stroke, ankle plantarflexor muscle weakness is common across the spectrum of severity (Fimland et al., 2011), and the paretic gastrocnemius muscles of people poststroke demonstrate greater muscle atrophy than the soleus muscle (Ramsay, Barrance, Buchanan, & Higginson, 2011). Therefore, understanding of postural control about the ankle poststroke requires the investigation of the role of each plantarflexor muscle in the control of postural sway.

Interestingly, despite the asymmetry of postural control following stroke, during maintenance of standing balance under external loading, a moderate level of common drive to the medial gastrocnemius motor units bilaterally remains in people poststroke, suggesting at least partial preservation of a common command between paretic and nonparetic plantarflexor muscles during standing (Garland, Pollock, & Ivanova, 2014). However, how the plantarflexor muscles of the paretic and nonparetic legs of people poststroke each modulate with postural sway to control standing balance is not known and may provide further insight into the asymmetrical postural control strategy of people poststroke. Rehabilitation of postural control poststroke aims to engage both the paretic and the nonparetic legs in maintenance of postural

control when standing balance is challenged. Therefore, exploring how the strength of the relationship between the center of pressure and plantarflexor muscle activity modulation is affected in both the paretic and nonparetic legs by increasing levels of challenge to standing balance and associated increased levels of muscle activation, external ankle torque, and measures of postural control performance, may further the understanding of the response of both paretic and nonparetic muscle to standing balance challenges. This study examines the relationship of the ankle plantarflexor muscles (medial and lateral gastrocnemius and soleus) with postural sway position and velocity in people poststroke compared with age-matched controls in response to an increasing anteriorly directed challenge to standing balance.

METHODS

Ten people with chronic stroke (>3 months poststroke) and 10 age-matched controls provided written informed consent to participate in this study. Individuals poststroke were included if they were ambulatory with or without a walking aid and could stand independently for a minimum of 5 min. Individuals were excluded due to health conditions that impacted mobility (e.g., severe

osteoarthritis). Controls were free from neurological or musculoskeletal impairment, which resulted in mobility restrictions and/or balance deficits. The study conformed to the standards set by the latest revision of the Declaration of Helsinki and was approved by the University of British Columbia Clinical Research Ethics Board (H12-00723).

Motor impairment severity following stroke was measured at the foot and ankle using the Chedoke-McMaster Stroke Assessment (CMSA; Gowland et al., 1993). The CMSA describes seven stages of motor recovery; 0/7 refers to flaccid paralysis and 7/7 refers to movement equated to a “normal” sensory-perceptualmotor system (Gowland et al., 1995). Both participants poststroke and controls were assessed for ambulatory balance using the Community Balance and Mobility Scale (CB&M; 96, higher scores reflecting a higher level of walking balance; Howe, Inness, Venturini, Williams, & Verrier, 2006; Knorr, Brouwer, & Garland, 2010).

Experimental Protocol

Participants stood barefoot, with their feet shoulder-width apart, with each foot on a separate force platform (AMTI OR6-6; Advanced Mechanical Technology Inc., Watertown, MA). A postural control challenge paradigm was employed, which has been shown to

increase external torque applied to the ankle joint (Pollock, Ivanova, Hunt, & Garland, 2014). A belt was secured around the pelvis of each participant and was attached to a horizontal cable in front of the participant. External loads of 1 percentage body mass (%BM) were applied by a cable-pulley system attached to the front of the belt. The loads were dropped into a basket from 40 cm above every 25–40 s (random timing), and each load remained in the basket until 5%BM was maintained. Application of the load was detected (deflection from baseline) from the signal of a force transducer in-line with the cable.

Kinetic and Kinematic Data

Kinetic data were collected using two floor-mounted force platforms (detailed previously). Anterior–posterior center of pressure (APCOP) position and the vertical ground reaction force were measured for each foot. The percentage weight-bearing through the paretic leg of participants poststroke and the right leg of controls was calculated from the vertical component of the ground reaction force of the limb divided by the total vertical ground reaction force of both limbs of the participant. APCOP velocity was calculated as the derivative of the APCOP position signal. Passive reflective markers were affixed to the posterior aspect of the calcaneus (heel),

head of the first metatarsal, and lateral malleolus to capture the anterior and posterior limits of the foot and the ankle joint center. Eight high-speed digital cameras (Raptor-E; Motion Analysis Corp., Santa Rosa, CA) sampled movement of the markers at 100 Hz. Kinematic data were analyzed using a custom-written program (Mathworks Inc., Natick, MA, USA). To compare the displacement of center of pressure among participants, APCOP position was also converted to a percentage of the length of the foot or base of support (%BOS) within each foot. BOS was calculated from the kinematic markers of the heel (0%) and the head of the first metatarsal (100%). External torque about the ankle was calculated bilaterally as the product of the perpendicular distance between the ankle center and the line of action of the vertical component of the ground reaction force.

Electromyography

The use of high-density surface electromyography provides sampling from a broad area of each muscle reflecting the global activity of each muscle. High-density surface electromyography was collected from the soleus (24 electrode grids, 2-cm interelectrode distance) and the medial and lateral gastrocnemius (20 electrode grids each, 1.5-cm interelectrode distance) bilaterally

(OT Bioelectronica, Turin, Italy), sampledat 2,048Hz.High-densitysurfaceelectromyography signals wereanalyzed in bipolar configurations resulting in 18 EMG signals from the soleus muscle and 16 from each of the medial and lateral gastrocnemius muscles. EMG signals were bandpass filtered (20–400 Hz) and a notch filter at 60 Hz was applied. Root mean square (RMS-EMG) amplitude was calculated for each EMG signal from each muscle and normalized to the RMS-EMG amplitude during quiet standing measured at baseline before load application.

Outcome Measures

To capture the control of postural sway under conditions of increased level of anteriorly directed challenge, parameters were measured for 15 s epochs, while participants maintained each load level. Each epoch was measured beginning approximately 5 s post application of each load to ensure the initial response to load application was not included. This epoch size and timing was chosen to ensure that the center of pressure behavior was more reflective of postural control under sustained anteriorly directed challenge to standing balance rather than to abrupt external forces acting on the body. Measures calculated for each leg included: APCOP position, APCOP velocity, external ankle torque and EMG

amplitude of the medial and lateral gastrocnemius, and soleus muscles. The APCOP position signals were visually inspected, and epochs were selected that did not include any intentional shift of position beyond sway. (Sometimes participants poststroke would adjust their body position posteriorly as the postural challenge increased.) In addition, the mean APCOP position as a percentage of the BOS and the percentage weight-bearing on the paretic leg of participants poststroke and the right leg of controls were calculated during each epoch to determine the absolute position within the BOS and symmetry of stance at each load level. The SD of APCOP position and velocity were calculated to explore the variability of postural sway of each leg at each load level as these measures have been shown to demonstrate changes in postural control performance associated with increased challenge in postural tasks (Carpenter, Frank, Silcher, & Peysar, 2001).

Cross-Correlation

The EMG signals were full-wave rectified. Both EMG envelopes and APCOP data were low-pass filtered using a fourth-order, zero-phase lag Butterworth filter with 4 Hz cutoff (Masani et al., 2003). Using a custom-written program (Mathworks Inc.), cross-correlation was applied to each detrended EMG envelope from each muscle

(16 for medial and lateral gastrocnemius muscles and 18 for soleus muscle) with the APCOP position and velocity across the 15-s epoch at each load level. Peak correlation coefficients within the epoch at each load level and the corresponding timing of the peak cross-correlation function were calculated for each signal within each grid and the median value of each muscle was calculated.

Statistical Analysis

Participant characteristics (age and CB&M score) were compared between groups using student t tests (age) and Mann–Whitney U test (CB&M scores).

Two-way repeated measures analyses of variance with Bonferroni adjustment for multiple comparisons were employed in each group separately (participants poststroke and controls) to explore the effect of load (1–5%BM) and leg (paretic and nonparetic of participants poststroke; right and left legs of controls) for the following parameters: APCOP position (%BOS), external ankle torque, SDs of APCOP position and velocity, and percentage weight-bearing (paretic leg and control right leg for this parameter). Planned paired comparisons within group were used to explore the effect of leg at each load level. This analysis explored the kinetic and kinematic response to the perturbation paradigm in each group.

To compare the relationships between EMG and APCOP position and velocity, the correlation coefficients were transformed using a Z transformation.

Separate two-way repeated measures analyses of variance with planned comparisons with Bonferroni adjustment were performed in each group (stroke and controls) and each muscle (medial, lateral gastrocnemius, and soleus muscles) to explore effect of load (1–5%BM) and leg (paretic and nonparetic of participants poststroke; right and left legs of controls) differences in the correlation Z scores, timing of the peak correlation, and EMG amplitude. Planned paired comparisons were employed to compare each of these parameters between legs of participants poststroke (paretic and nonparetic) and controls (right and left) at each load level to specifically explore the effect of increased load on between leg differences.

A complementary partial correlation analysis, controlling for load, was performed separately in the paretic and nonparetic muscles to explore the relationships between the correlation Z scores and the EMG amplitude, external ankle torque and the SD of the APCOP position and velocity. This analysis examined if modulation of the EMG amplitude, external ankle torque, and the

variability of APCOP measures during postural sway were related to change in the level of EMG:APCOP correlations.

The level of significance was $p = .05$. Data are presented as mean and SD unless otherwise noted. Peak correlation coefficients, rather than Z scores, are presented later to assist the reader's interpretation of results.

RESULTS

Table 1 shows the characteristics of participants poststroke and controls. Age was not significantly different between groups ($p = .65$). Participants were in the chronic stage poststroke and were able to ambulate in the community with mild to moderate impairments in walking balance and motor control of the foot and ankle. Participants poststroke scored significantly lower than controls in ambulatory balance as measured by the CB&M scale ($p < .01$).

The motor impairment scores of the foot and ankle of participants showed an interquartile range of 3–6/7. A CMSA score of 3/7 reflects a motor control impairment, which can be described as marked spasticity present, some voluntary movement, and

synergistic patterns with inability to move quickly between plantarflexion and dorsiflexion. A motor control impairment scored as CMSA 6/7 can be described as spasticity no longer present, a large variety of patterns of movement are now possible, abnormal patterns of movement with faulty timing emerge when rapid or complex actions are requested (Gowland et al., 1995).

Kinematic and Kinetic Parameters of Postural Sway

The APCOP position and external ankle torque are shown in Figures 1a and 1b, respectively. The APCOP position (%BOS) showed a significant anterior progression with the addition of loads ($p < .01$) with no significant difference between legs of participants poststroke ($p = .60$) or controls ($p = .68$). Greater ankle torque is necessary to sustain a greater load applied as shown in Figure 1b ($p < .01$). The higher levels of external ankle torque noted on the nonparetic side compared with the paretic side did not reach significance ($p = .14$), and there was no significant difference between the legs of controls ($p = .22$). In summary, APCOP position progressed significantly forward in each foot and external ankle torque increased in each ankle.

In participants poststroke, there was no effect of load on the SD of the APCOP position (Figure 1c, $p = .31$) or velocity (Figure 1d, p

= .27). When collapsed across loads, the nonparetic leg showed a tendency for greater SDs of APCOP position (p = .06) and APCOP velocity (p = .07) than the paretic leg, suggesting greater variability of postural sway parameters of the nonparetic leg; however, this finding did not reach significance. In controls, there was no significant effect of load the SD of APCOP position (Figure 1c, p = .48) or APCOP velocity (Figure 1c, p = .89). There was also no difference between the right and left legs of controls in the SDs of the APCOP position (p = .27) or APCOP velocity (p = .17).

There was no significant effect of load on the percentage weight-bearing on the right leg of controls or the paretic leg of participants poststroke (p = .20). Also, there was no significant difference between groups (stroke and controls, p = .32) for the mean percentage of weight-bearing across loads; paretic leg was 47.6% ± 7.0% and control right leg was 50.3% ± 5.2%.

Plantarflexor Muscle EMG

The EMG amplitude of each muscle is shown in Figure 2a. For each plantarflexor muscle of participants poststroke and controls there was a significant effect of load on EMG amplitude (p < .01). Between leg differences in the EMG amplitude were noted only in the lateral gastrocnemius muscles of participants poststroke with

the nonparetic side showing greater EMG amplitude compared with the paretic side at load Levels 3 and 4%BM ($p < .05$).

Peak Correlation Coefficients Between EMG and APCOP Position (EMG:APCOP Position)

There was no significant difference between the legs or a significant effect of load on the timing of the peak correlation of EMG:APCOP position in participants poststroke or controls (Table 2, $p > .05$).

A representative figure of the cross-correlations is shown in Figure 3. Moderate positive correlations were found between the medial and lateral gastrocnemius muscles EMG:APCOP position and weak to moderate positive correlations were found in soleus EMG:APCOP position across load levels in both participants poststroke and controls. There were no significant differences between legs and no effect of load in the ankle plantarflexor muscles of controls (Figure 2b, $p > .05$).

In participants poststroke, the correlation coefficients showed stronger relationships on the nonparetic than the paretic side for EMG:APCOP position in the medial gastrocnemius muscle at load Levels 1–3%BM ($p < .05$) and in the lateral gastrocnemius muscle at load Levels 1 and 2%BM ($p < .05$). The between leg difference in the EMG:APCOP position correlation coefficients of the paretic

and nonparetic soleus muscles at 1 and 2%BM did not reach statistical significance (Figure 2b, 1%BM $p = .11$, 2%BM $p = .06$).

Peak Correlation Coefficients Between EMG and APCOP Velocity (EMG:APCOP Velocity)

Each plantarflexor muscle activity correlated more strongly with APCOP velocity than APCOP position. The correlation coefficient for soleus muscle EMG:APCOP velocity showed a stronger relationship on the left leg than the right leg of controls at load Levels 1 and 3%BM ($p < .05$).

In participants poststroke, the correlation coefficients for medial and lateral gastrocnemius muscle EMG:APCOP velocity showed stronger relationships on the nonparetic than the paretic side at load Levels 1–5%BM (Figure 2c, $p < .05$, except at 5%BM, $p = .08$ for medial gastrocnemius). The soleus muscle had stronger correlation coefficients for EMG:APCOP velocity on the nonparetic than the paretic leg, but only at load Levels 1 and 2%BM (Figure 2c, $p < .05$).

Association of Performance Parameters With EMG:APCOP Correlation Coefficients

In the plantarflexor muscles of participants poststroke, the narrowing of the difference between paretic and nonparetic peak

correlation coefficients for EMG: APCOP position cannot be entirely explained by an effect of load. Parameters of postural sway, external ankle torque, EMG amplitude, and the correlation Z scores were examined with partial correlations, controlling for load (Table 3). There were significant correlations between the EMG amplitude and the Z scores in all muscles. The Z scores of all nonparetic plantarflexor muscles, but not paretic muscles, demonstrated a significant relationship with external ankle torque. Parameters of postural sway (SD of APCOP position and velocity) demonstrated significant relationships with the Z scores for paretic and nonparetic plantarflexor muscles.

DISCUSSION

During anteriorly directed progressive challenges to maintaining standing balance, the plantarflexor muscle activity of participants poststroke and age-matched controls was positively correlated with APCOP position and APCOP velocity. The timing in which the modulation of each muscle preceded postural sway position was similar between participants poststroke and controls. In participants poststroke, the relationships of ankle plantarflexor EMG:APCOP velocity were stronger in the nonparetic leg than in the paretic leg

at each load level (aside from the soleus muscle at 3–5%BM and the medial gastrocnemius at 5%BM). This suggests that, following stroke, motor control asymmetry may be, in part, due to the ability to sufficiently make use of the complexity of the afferent information pertaining to postural sway velocity on the nonparetic as opposed to the paretic side. The relationships of ankle plantarflexor muscles EMG:APCOP position were significantly stronger in the nonparetic leg than in the paretic leg only at the lower two to three load levels in the gastrocnemius muscles and was lost at the higher (4–5%BM) load levels, revealing an improvement in the symmetrical motor control strategy between legs with increased levels of postural challenge.

Fundamental Motor Control of Standing Balance Following Stroke

The peak correlation coefficients as a measure of the relationship between the modulations of ankle plantarflexor muscle activity and postural sway in this study are in agreement with correlation coefficients during quiet stance in healthy subjects (Gatev et al., 1999; Masani et al., 2003). The control of the ankle plantarflexors, described by Gatev et al. (1999), suggests that the CNS maintains standing balance using the constant input of afferent information to

anticipate the position of the center of mass in the next instance and actively controls this position with activation of the ankle plantarflexor muscles to maintain balance (Gatev et al., 1999; Masani et al., 2003).

The timing between the EMG and postural sway position signals is proposed to be composed of the time associated with afferent feedback and motor command and therefore is suggested to be representative of the central integrative command (Gatev et al., 1999; Masani et al., 2003). The lack of difference in the timing of the peak correlation of EMG:APCOP position in participants poststroke and age-matched controls suggests that this anticipatory mechanism of postural control is maintained in the plantarflexor muscles in participants poststroke. Masani et al. (2011) investigated the timing of the peak correlation between soleus EMG activity and postural sway in young and older adults and found no effect of age. Our findings suggest that the timing between the EMG activity and the resultant postural sway is also similar in people poststroke. That is, a time delay associated with central integrative command does not appear to contribute to the motor control impairment noted in these participants with chronic stroke.

The relationship of plantarflexor muscle activation with APCOP velocity demonstrated stronger relationships and more extensive differentiation between the paretic and nonparetic legs of participants poststroke across load levels than the EMG:APCOP position relationship. This supports the importance of feedback of postural sway velocity to inform the CNS of direction and speed of displacement, facilitating the prediction of imminent displacement and signaling the necessity of postural adjustments to maintain standing balance (Masani et al., 2003; Portela et al., 2014).

At the level of muscle, the paretic gastrocnemii muscles (but not soleus) of participants poststroke demonstrated stronger relationships with postural sway as EMG amplitude increased. The roles of the plantarflexor muscles differ in quiet stance; the soleus muscle demonstrates tonic activation, whereas the gastrocnemii muscles have more phasic activity in response to anterior displacement of the center of pressure with respect to the ankle joint (Loram, Gollee, Lakie, & Gawthrop, 2011; Vieira, Loram, Muceli, Merletti, & Farina, 2012). Human gastrocnemius muscles have been described as being composed of approximately 50% fast-twitch fibers, whereas the soleus muscle is composed of 70–100% slowtwitch fibers (Johnson, Polgar, Weightman, & Appleton, 1973).

Paresis following stroke has been shown to lead to greater atrophy in the gastrocnemius muscles than the soleus muscle (Ramsay et al., 2011). These patterns of atrophy may be related to the greater proportion of Type II motor unit loss and remodeling of Type I motor units (innervation of orphaned Type II muscle fibers resulting in larger Type I motor units) reported to occur in paretic muscle following stroke (Lukacs, Vecsei, & Beniczky, 2008). The findings of this study are somewhat suggestive of a greater impact of stroke on the role of the paretic gastrocnemii compared with the soleus muscle under conditions of a challenging postural task. This may be a reflection of how the remodeling of paretic muscle that occurs poststroke impacts the function of the gastrocnemius muscle more than the soleus muscle.

Clinical Relevance

The asymmetry of postural control favoring the nonparetic leg following stroke is well established (Roerdink et al., 2009; van Asseldonk et al., 2006). The tendency for larger variance of postural sway displacement and velocity in this study on the nonparetic side (Figure 1) suggests compensatory postural control provided by the nonparetic leg and the exploratory nature of postural sway (Carpenter, Murnaghan, & Inglis, 2010; Gatev et al., 1999). As

postural sway has been found to include anticipatory control mechanisms, it has been proposed that active postural sway may serve to continually gather afferent input regarding position of the body relative to the BOS, allowing for anticipation of the body's position in the next movement instance and signaling of motor commands to maintain balance (Carpenter et al., 2010; Gatev et al., 1999). Our results suggest that sufficient challenges to standing balance may lead to a more active role of the paretic leg in the exploratory aspect of postural sway position specifically because the EMG: APCOP position correlations increase on loads 4–5%BM.

Larger EMG amplitude of the paretic medial and lateral gastrocnemius muscles was associated with a stronger EMG:APCOP position relationship. Conversely, the EMG:APCOP position relationship was negatively associated with amplitude of the nonparetic ankle plantarflexor muscles and with external ankle torque. That is, the strength of the nonparetic EMG:APCOP position relationship shows a slight decrease with increased level of challenge (Figure 2). Altogether, these relationships suggest that the challenge of this task may involve an interlimb postural control strategy such that the paretic plantarflexor muscles must increasingly participate in the active control of sway position

specifically to maintain standing balance, thereby requiring less from the nonparetic. Clinically, this further suggests the importance of the fundamental components of postural control when establishing therapeutic levels of challenge to standing balance in rehabilitation interventions.

The response of the paretic and nonparetic plantarflexors to progressive challenges to standing balance as seen in this study, supports previous clinical research which suggested a positive role of standing reaching tasks during rehabilitation poststroke (Lin, Wu, Chen, Chern, & Hong, 2007; McCombe & Prettyman, 2012) and further suggests physiological rationale for the improved bilateral postural control noted. Furthermore, this study suggests that future research into rehabilitation strategies involving more progressive challenges to standing balance and the physiological effects on postural control between the paretic and nonparetic leg is warranted. Determining the level of challenge required to achieve physiological gains is an important next step.

Limitations

There are important limitations to consider in the interpretation of this data. The study is limited by a small sample size which may have, in some cases, resulted in false retention of the null

hypothesis. In addition, the kinematic measurements represent only sagittal joint excursion, and therefore, movement in the frontal and transverse planes, which may reflect important components of the postural strategy used by people poststroke and controls, were not explored. Finally, the withingroup analysis used in this study somewhat limits the conclusions that can be drawn about the similarities or differences between groups.

CONCLUSIONS

The results of this study suggest that, similar to age-matched controls, moderate relationships exist between the paretic and nonparetic ankle plantarflexor muscles, and postural sway position and velocity in participants poststroke. The timing of the EMG activity in the ankle plantarflexor muscles preceding postural sway as reflected by the timing of peak correlation appears to be maintained poststroke. An increased level of challenge to standing balance has limited effects on controls; however, results in a reduction in the postural control asymmetry of the EMG: APCOP relationship in participants poststroke across all plantarflexor muscles. These findings suggest a possible benefit of examining the fundamental components of postural control of standing

balance when establishing the level of challenge required in the rehabilitation of standing balance and the inclusion of progressive sustained challenges to standing balance in the rehabilitation of balance poststroke.

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TABLES

Table 1: Participant Characteristics

	Age (years)	Sex (male/ female)	Post Onset (years)	Paretic Side (R/L)	CMSA (0–7)	CB&M (0–96)
Stroke	66.2 ± 9.2	8/2	6.6 ± 3.6	5/5	3 [3–6]	23.0 [16.8–49.0]
Control	68.0 ± 8.2	7/3	n/a	n/a	n/a	83.0 [79.8–84.3]

Chedoke-McMaster Stroke Assessment (CMSA) scores and Community Balance and Mobility (CB&M) scores are reported as median and interquartile range (IQR). R/L = right and left.

Table 2: EMG Timing Delay (ms) at the Point of the Peak Correlation of EMG:APCOP Position

	MG	LG	SOL
Paretic	−198.5 (5.4)	−205.9 (5.5)	−207.7 (5.5)
Nonparetic	−193.0 (4.7)	−198.1 (4.0)	−204.0 (4.5)
Left	−193.8 (5.4)	−183.8 (4.9)	−197.8 (5.8)
Right	−190.4 (4.7)	−200.7 (6.2)	−201.4 (5.7)

Mean (SE), collapsed across loads. EMG = electromyography; APCOP = anterior–posterior center of pressure; MG = medial gastrocnemius muscle; LG = lateral gastrocnemius muscle; SOL = soleus muscle.

Table 3 Partial Correlation Controlling for Load, Between the Z Scores of the Peak EMG:APCOP Position (a) or EMG:APCOP Velocity (b) Correlation Coefficients for Paretic and Nonparetic MG, LG, and SOL Muscles and External Ankle Torque, Mean RMS-EMG Amplitude, SD of the APCOP Position and Velocity

Muscle	External Ankle Torque	RMS-EMG Amplitude	SD of APCOP Position	SD of APCOP Velocity
(a) EMG:APCOP position Paretic MG	−0.08	0.39**	0.69**	0.68**
Paretic LG	−0.03	0.52**	0.63**	0.39**
Paretic SOL	−0.13	−0.26***	0.70**	0.64**
Nonparetic MG	−0.38**	−0.17	0.43**	0.50**
Nonparetic LG	−0.26***	−0.15	0.53**	0.53**
Nonparetic SOL	−0.29***	−0.48**	0.46**	0.52**
(b) EMG:APCOP velocity Paretic MG	0.11	0.57**	0.36*	0.61**
Paretic LG	0.12	0.59**	0.29***	−0.18
Paretic SOL	−0.03	−0.08	0.49**	0.65**
Nonparetic MG	−0.19	−0.26***	0.05	0.36*
Nonparetic LG	0.32***	−0.02	0.18	0.48**
Nonparetic SOL	−0.04	−0.34*	0.18	0.50**

EMG = electromyography; APCOP = anterior–posterior center of pressure; MG = medial gastrocnemius muscle; LG = lateral gastrocnemius muscle; SOL = soleus muscle; RMS = root mean square. *p ≤ .05. **p ≤ .01. ***p ≤ .1.

FIGURES

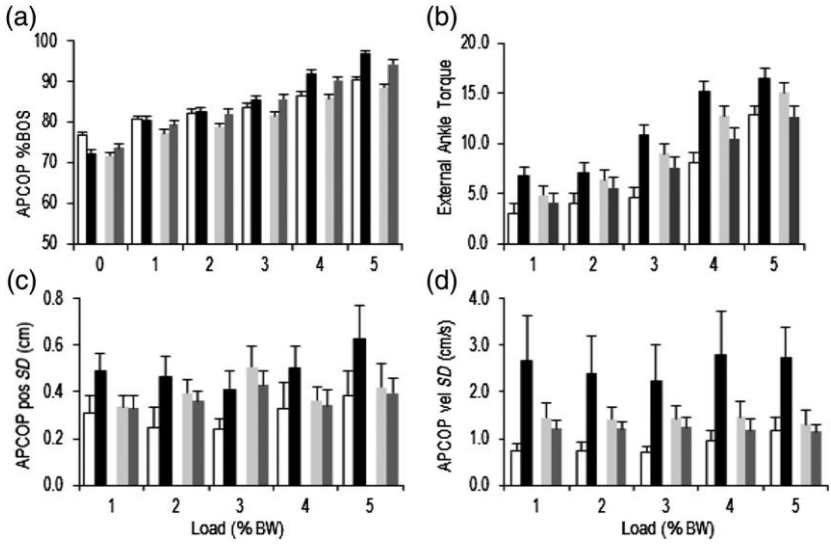

Figure 1: Kinematic and kinetic parameters of postural sway for participants poststroke: paretic leg (white bars) and nonparetic leg (black bars), and controls: left leg (light gray) and right leg (dark gray). (a) APCOP forward progression in BOS (%BOS—0% represents the heel and 100% represents the marker placed at the base of the metatarsal head of the great toe). (b) Increase in external ankle torque from quiet stance (N·m). (c) SD of APCOP position (cm). (d) SD of APCOP velocity (cm/s). Data are mean ± SE. BOS = base of support; APCOP = anterior–posterior center of pressure.

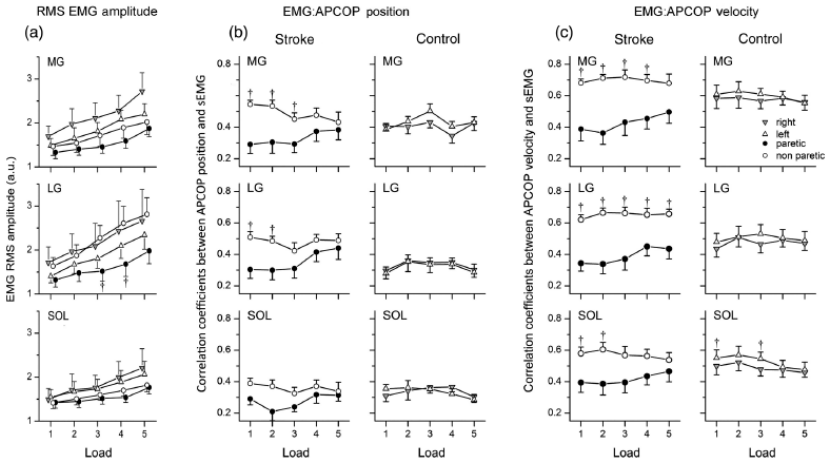

Figure 2 — Plantarflexor EMG amplitude and relationships between EMG:APCOP position and velocity during the maintenance of anteriorly directed loads in standing. Presented for participants poststroke (paretic: black circles and nonparetic: open circles) and controls (right: gray triangles and left: open triangles).MGand LG and SOL muscles. (a) RMS-EMG amplitude in response to maintenance of increasing load, normalized to quiet stance. (b) Cross-correlation coefficients for EMG:APCOP position. (c) Cross-correlation coefficients for EMG:APCOP velocity. *p < .05. Data are mean ± SE. APCOP = anterior–posterior center of pressure; MG= medial gastrocnemius; LG = lateral gastrocnemius; SOL = soleus; EMG = electromyography; RMS = root mean square; sEMG = surface electromyography.

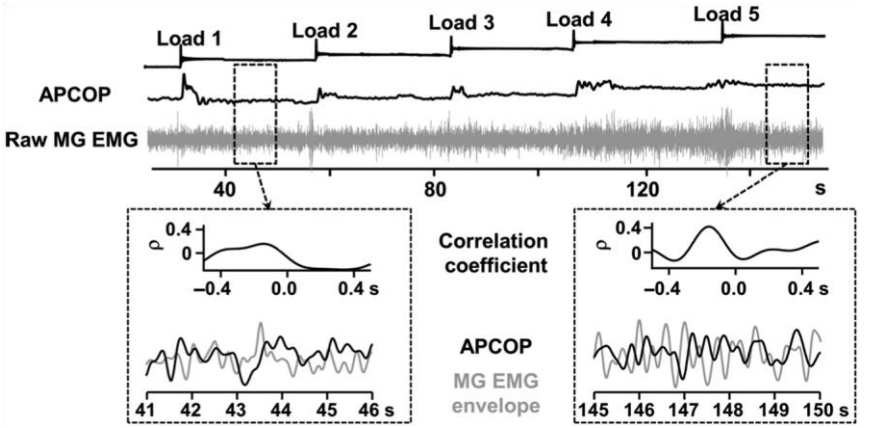

Figure 3: Representative example of cross-correlation between modulations of paretic MG muscle activity (EMG) and APCOP in a participant poststroke. Tracings from top: force transducer signal showing application of perturbation (load), APCOP position, and raw EMG trace from paretic medial gastrocnemius muscles. Inset boxes (bottom) show signal variance for one channel of paretic MG EMG envelope, while maintaining 1%BM (left) and 5%BM (right). Cross-correlation functions between the EMG envelope and APCOP signals are at the top of the inset. APCOP = anterior–posterior center of pressure; MG = medial gastrocnemius; EMG = electromyography; %BM = percentage body mass.