Gait Initiation in Multiple Sclerosis

Jebb G. Remelius, Joseph Hamill, Jane Kent-Braun, and Richard E.A. Van Emmerik

Individuals with multiple sclerosis (MS) often have poor balance control that is especially apparent during dynamic tasks such as gait initiation (GI). The purpose of this study was to investigate how balance symptoms due to MS alter spatiotemporal variables, coordination, and temporal margins within the stability boundary during gait initiation. Twelve women with MS (Expanded Disability Status Scale [EDSS] mean = 4.0, SD = 1.4) and 12 women without MS (control group) initiated gait at their preferred speed. MS participants attained a slower anterior velocity because of smaller anterior center of mass displacements and took longer to complete the initiation of gait than the control group. MS participants exhibited a smaller posterior shift in center of pressure during GI and stepped with a longer dual support time than the control group. However, these changes may be due to differences in initiation velocity. Relative timing analysis showed invariance in postural and locomotor phases of gait initiation between groups. The MS group showed different coordination between anterior-posterior and medio-lateral center of pressure components while increasing temporal margins to the posterior and lateral stability boundaries in comparison with the control group. Overall, during gait initiation at their preferred speed the MS participants adopted a functional strategy that produces lower speed and reduced proximity to the stability boundaries prior to stepping.

Keywords: coordination, vector coding, time to contact, stability boundary, balance control

Multiple sclerosis (MS) is an autoimmune disease of the central nervous system characterized by scar tissue (sclerosis) resulting from the repair of damage to the myelin sheath that surrounds neurons. MS affects approximately 2.5 million people worldwide. Symptoms of MS are unpredictable but may include fatigue, vision problems, loss of balance and coordination (ataxia), or depression.

For those with neurological disorders like MS, standing balance and gait often remain functional under moderate symptomatic states, despite the fact that self-reported balance problems are common (Noseworthy, Lucchinetti, Rodriguez, & Weinshenker, 2000). During quiet upright stance, changes in balance from MS are often difficult to determine since stance can appear unaffected even at severe levels of disability (Corradini, Fioretti, Leo, & Piperno, 1997). In contrast, dynamic tasks clearly elicit differences in balance even at low levels of disability due to

The authors are with the Dept. of Kinesiology, University of Massachusetts, Amherst, MA 01003.
MS, as when performing a lean or reach (Frzovic, Morris, & Vowels, 2000; Karst, Venema, Roehrs, & Tyler, 2005) or during gait (Benedetti et al., 1999; Martin et al., 2006). Dynamic tasks involving the upper extremity also tend to elicit symptoms of ataxia, or lack of coordination, in MS (Behbehani, Kondraske, Tintner, Tindall, & Imrhan, 1990; Erasmus et al., 2001; Verheyden et al., 2006). Little is known, however, about the influence of ataxia on dynamic balance and mobility during gait. Dynamic postural tasks typically bring the center of pressure (CoP) and center of mass (CoM) nearer to the stability boundaries (defined as the perimeter of the feet) than quiet stance (Van Wegen, Van Emmerik, Wagenaar, & Ellis, 2001). The stability boundary is sometimes referred to as the base of support. To some extent balance problems can be mitigated by adopting a safer movement strategy such as moving more slowly (Halliday, Winter, Frank, Patla, & Prince, 1998). Slower movements may facilitate postural stability by minimizing stability boundary approaches.

A dynamic task in which the relative lack of coordination in people with MS may have direct consequences for postural stability is the initiation of gait. We define gait initiation (GI) as beginning at the onset of movement (after the initiation signal) and ending at stance foot toe-off. GI can be divided into a postural phase and a locomotor phase at swing foot toe-off. The postural phase of GI is typically referred to as an anticipatory postural adjustment (APA) and occurs prior to gross segmental movement (Brenière & Do, 1986) and stability-boundary changes of the first step. To consider functional balance strategies in the APA of GI, we adopted a simple postural model in which shifts of the CoP and movement of the CoM are considered to be the result of coordination between two mechanisms: an inverted pendulum operating as an “ankle strategy” in the anterior-posterior (A/P) direction, dominated by ankle muscles, and bilateral limb loading creating a “hip strategy” in the medio-lateral (M/L) direction, dominated by hip abductor/adductor muscles (Winter, 1995). Coordinated actions of these two postural mechanisms shift the CoP during the APA in a systematic manner: first a translation of the CoP in lateral and posterior directions together toward the swing foot heel (APA1), followed by a predominantly lateral CoP shift toward the stance foot (APA2) (Halliday et al., 1998). The CoM moves very little during the first part of the APA but begins a ballistic-like displacement in the anterior direction and toward the stance foot during the latter part of the APA. CoM velocity during GI is strongly correlated with posterior CoP displacements during the APA (Brenière, Do, & Bouisset, 1987) in healthy populations.

The dynamic CoP shifts within the stability boundary during the APA exemplify synergistic behavior. Synergies are present when multiple degrees of freedom are constrained relative to anatomical boundaries to form structured movements or integrated formations that imply the presence of “motor-coordination” (Bernstein, 1967). By defining coordination in this way, the contribution of separate elements to a common action is considered. Coordination between separate postural mechanisms must go beyond description of variables in isolation and without the context of anatomical boundaries, such as studying A/P or M/L behavior independently. To examine coordination between A/P and M/L postural mechanisms that shift the CoP during the APA, we computed a local coupling angle (LCA) based on a modified vector coding technique. Vector coding techniques (Sparrow, Donovan, Van Emmerik, & Barry, 1987; Tepavac & Field-Fote, 2001) have previously been used to quantify lower extremity joint coordination (Heiderscheit, Hamill, & Van
Emmerik, 2002). In these prior studies, the vector coding quantified changes in angle-angle diagrams pertaining to the lower extremity. Here we will use the technique to assess the relative motion between A/P and M/L components of CoP shifts during each portion of the APA.

To study dynamic posture in the context of the stability boundary, we computed time to contact (TtC) within the stability boundary for the CoP and CoM during the APA. TtC is an emerging measure of postural stability that incorporates temporal and spatial aspects of behavior in the context of the stability boundary. This technique has been applied to quiet upright stance in healthy adults (Haddad, Gagnon, Hasson, Van Emmerik, & Hamill, 2006), aging populations (Slobounov, Moss, Slobounova, & Newell, 1998), and people with Parkinson’s disease (Van Wegen et al., 2001). However, TtC information may also be important during the initiation of gait when persons voluntarily approach and cross the anterior stability boundary, while simultaneously avoiding posterior and lateral stability boundaries. Adaptive movement strategies chosen to mitigate balance problems in people with MS may be revealed by assessing these boundary-relevant stability measures during GI.

The purpose of this study was to characterize the effect of MS on spatiotemporal patterns across the gait initiation sequence, as well as to examine coordination and temporal margins within the stability boundary during the APA of GI. We hypothesized that the MS participants initiate gait with a slower CoM velocity than the controls. We predicted that decreases in posterior CoP displacement during the APA associate with slower GI velocity (Brenière et al., 1987; Halliday et al., 1998). We further hypothesized that increases in temporal margins within the stability boundary are associated with movement strategies of the MS group during GI.

Methods

Participants

Twelve women with MS and moderate to relatively severe impairment (Expanded Disability Status Scale [EDSS] ranging from two to six; mean = 4.0, SD = 1.4) volunteered for the study (mean = 54.9 years, SD = 8.5; mean = 163.9 cm, SD = 8.6; mean = 71.2 kg, SD = 14.4). EDSS scores were determined by a neurologist’s evaluation and can range from 0 (least impaired) to 10 (death due to MS). Scores for being fully ambulatory extend to 4.5 (Noseworthy et al., 2000). All MS participants self-reported some symptomatic balance problems. None of the MS women reported exacerbations of symptoms in the previous six months. Twelve age-matched women without MS served as a control group (mean = 52.9 years, SD = 9.3; mean = 160.9 cm, SD = 6.0; mean = 70.8 kg, SD = 10.0). No participants reported any lower limb pathologies or history of lower limb injuries. All participants gave informed consent, as approved by The Institutional Review Board from the University of Massachusetts, before testing.

Apparatus

We used the Qualisys track manager software to synchronize six ProReflex cameras and two 464 mm × 508 mm strain gauge force plates at 100 Hz (Qualisys Medical AB, Gothenburg Sweden; Advanced Mechanical Technology Inc., Newton MA).
The two force plates were mounted side by side to create a force collection area of 936 mm × 508 mm. We built a 13 segment 3D biomechanical model from the kinematic data based on established anthropometric data (Clauser, McConville, & Young, 1969). We tracked segment kinematics with thirty-five 2.54 cm retro-reflective polyethylene markers grouped in triads. Ten calibration markers provided references to joint centers. Markers on the shoulder (acromion process), elbow (medial/lateral humerus epicondyle), and wrist (ulna styloid process) tracked the upper limb segments, while triads referenced to calibration markers on the hip (greater trochanter), knee (medial/lateral femoral epicondyle), and ankle (tibial medial malleolus/fibular lateral malleolus) tracked the lower limbs. A triad fastened to the back tracked the torso, and a triad positioned over the bilateral posterior superior iliac crest and sacrum tracked the pelvis. Bilateral calibration markers on the shoulder and hip provided reference to the torso and pelvis segments. A rigid triad worn on the crown of the head tracked the head segment, and bilateral heel, great toe, and fifth metatarsal markers referenced to the ankle calibration markers tracked the foot segments. We estimated the stability boundary from bilateral heel, fibular lateral malleolus, fifth metatarsal, and great toe markers. A light emitting diode served as the initiation signal.

Protocol

Participants stood with their feet parallel and at a self-selected comfortable width apart. Each participant completed four GI trials. The experimental setup allowed participants to take a minimum of four steps. Walking began at a “normal” (i.e., preferred) unhurried pace upon seeing the initiation signal with instructions to initiate gait as when waiting for a crosswalk signal. Participants walked in their own low-heel street shoes and wore a T-shirt and spandex shorts. Each participant also did a timed 7.62 m walk test, performed without kinematics or kinetics measurements. Timed walk instructions were to proceed from quiet standing at a normal pace at the end of a three-second verbal countdown.

Data Analysis

To calculate CoP and CoM position time series, we used Visual3D (C-Motion, Rockville, MD) to process and filter (4 Hz with a second order zero lag Butterworth filter) kinematic and kinetic data. We computed CoP and CoM velocity and acceleration time series with finite difference algorithms in Visual3D.

Velocity, Amplitude, and Timing Measures of GI. We selected peak anterior CoM velocity during GI as an overall measure of GI performance, in which approximately 90% of steady state velocity is attained in healthy volunteers (Jian, Winter, Ishac, & Gilchrist, 1993). Gait speed was computed from the timed walk test data. We extracted stance width during the APA and step lengths from the kinematic data. To examine the nature of CoM displacement during GI, we calculated the maximal lateral displacement of the CoM toward the stance foot from movement onset during GI, as well as the anterior displacement of the CoM at swing foot heel-strike.

For analysis of timing in GI, we divided GI into a postural phase and a locomotor phase (Figure 1). The postural phase of GI is a two-part anticipatory postural
adjustment: APA₁ begins at the onset of movement (start of weight shift toward the swing foot; Figure 1: MO) and ends at the release of swing foot vertical loading (R); APA₂ begins at swing foot release and ends at the swing foot toe-off. Release of swing foot vertical loading corresponds to the maximal lateral departure of the CoP toward the swing foot following MO (Figure 2). The locomotor phase of GI also contains two parts (Figure 1): the unipedal phase (LOCₜ) that begins at swing foot toe-off and ends at swing foot heel-strike (extracted from kinematics); and the double support phase (LOC₉) that begins at swing foot heel-strike and ends at stance foot toe-off. Preceding GI is a preparatory phase that begins at the initiation signal and ends at MO. We identified temporal events of GI by examining bilateral vertical force traces (Nissan & Whittle, 1990), which matched estimations based on CoP (Halliday et al., 1998) and kinematic time series. The timing of events in gait initiation were reported in absolute (seconds) and relative formats. The relative timing measure was a percentage of total GI duration (MO – stance foot toe-off).

Figure 1 — Phases of GI identified from bilateral vertical force traces (% body mass) and CoM velocity (cm/s) from one trial by a control participant. MO: movement onset following initiation signal. Postural phases of GI: APA₁: anticipatory postural adjustment phase one (from MO to release of swing foot vertical loading [R]); APA₂: anticipatory postural adjustment phase two (from R to swing foot toe-off). Locomotor phases of GI: LOCₜ: unipedal phase (from swing foot toe-off to swing foot heel-strike); LOC₉: dual support phase (from swing foot heel-strike to stance foot toe-off).
Coordination in the APA. To assess coordination between separate postural mechanisms during CoP shifts of the APA, we computed a vector-coded (Sparrow et al., 1987; Heiderscheit et al., 2002) local coupling angle (LC_A) to describe the CoP displacement. First, we computed a polar angle (vector) for each consecutive coordinate pair of CoP data (α; Figure 2). Then, we conditioned the resulting angle by a repeated subtraction process (−π, −π/2, −π/4) to fold the results into one-half of a quadrant (45°). The LC_A is the mean of this folded angle for each of the two portions of the postural phase of GI (APA_1 and APA_2). We condensed the vector-coded angle into a 45° space to describe the average vector orientation as either primarily orthogonal (0°) or synchronized (45°). We characterized an orthogonal CoP shift as the action of a single postural mechanism and a synchronized CoP shift as the concerted action of M/L and A/P postural mechanisms, required to move the CoP along a diagonal.

Time to Contact Within the Stability Boundary in the APA. We computed the mean TtC within the stability boundary as a measure of postural stability for each of the two portions of the postural phase of GI (APA_1 and APA_2), using the Slobounov method (Slobounov, Slobounova, & Newell, 1997). TtC, which is typically applied to quiet stance postural control, measures the theoretical time it may take the CoP or CoM, given its instantaneous position, velocity, and acceleration, to contact the stability boundary (defined by the perimeter of the feet; bold dashed lines, Figure 2) (Haddad et al., 2006). The CoP and CoM remain within a stability boundary roughly analogous to quiet stance during the APA. The stability boundary configuration may be changing just prior to the end of APA_2 due to swing foot heel-lift; but the predominant motions of the CoP and CoM near this time are away from the swing foot and in the anterior direction; and, as such, the boundaries of
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interest are lateral stance foot and anterior boundaries. Therefore, we assume here that during the APA of GI, any stability boundary changes generally do not affect the TtC computation.

Statistical Analysis. We conducted a repeated measures analysis of variance (ANOVA) on all dependent variables with group and trial as the independent variables. A criterion alpha level of .05 was set for identifying significant statistical differences (SAS Institute Inc., Cary, NC).

Results

Velocity, Amplitude, and Timing

Lower velocities were observed in the MS participants during GI trials \( (p < .001) \) and the timed walk \( (p = .001; \text{Table 1}) \) in comparison with the control group. In the MS group, peak GI velocity represented a lesser percentage of the average timed walk speed in comparison with the control group \( (p < .001; \text{Table 1}) \). Both MS and control groups showed an increase in speed across trials, but these trial effects showed no interactions by group. Since we observed no interaction effects of group and trial for all the other GI variables reported, we omitted further results on trial effects from this report.

Each participant in this study employed characteristic CoP traces during the APA phase; these include an initial shift toward the swing-foot heel followed by a lateral shift toward the stance foot (Figure 2). The CoP displacements of the APA phase were generally similar between groups, with the primary difference being a smaller posterior CoP shift made during APA by MS participants than by the control group \( (p = .01; \text{Table 2}) \). First step length was significantly shorter in the MS participants \( (p < .001); \) and while the second step of gait occurs after GI, this was also shorter in the MS group \( (p < .001) \) in comparison with the control group (Table 2). The MS participants adopted a wider stance than the control group before the initiation signal \( (p = .02; \text{Table 2}) \), although hip width was not significantly different between groups, \( F(1, 22) = 0.26, p = .61 \). Maximum lateral displacement of

<table>
<thead>
<tr>
<th>Velocity (cm/s)</th>
<th>GI</th>
<th>Walk</th>
<th>GI/Walk Ratio (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>MS</td>
<td>46 (± 12)</td>
<td>89 (± 20)</td>
<td>52.5 (± 14.1)</td>
</tr>
<tr>
<td>Control</td>
<td>81 (± 14)</td>
<td>113 (± 11)</td>
<td>71.5 (± 13)</td>
</tr>
<tr>
<td>( F(1, 22) )</td>
<td>40.39</td>
<td>13.71</td>
<td>19.10</td>
</tr>
<tr>
<td>( p )</td>
<td>.0001</td>
<td>.001</td>
<td>.0002</td>
</tr>
</tbody>
</table>

Abbreviations: MS, multiple sclerosis; CoM, center of mass; GI, gait initiation.
the CoM toward the stance foot between MO and the end of GI was not significantly different between groups \( (p = .14; \text{Table 2}) \). By heel-strike of the swing foot, the CoM had traveled on average 10 mm anterior of the stance foot in the MS group, but significantly farther \( (M = 112 \text{ mm}) \) beyond the stance foot in the control group \( (p < .001; \text{Table 2; Figure 2}) \).

The GI duration \( (p = .01) \) was longer in the MS participants as was time from the initiation signal to movement onset \( (p = .04) \) in comparison with the control group (Table 3). The longer GI durations in the MS participants stemmed from longer APA\(_2\) \( (p = .02) \) and double support (LOC\(_D\); \( p = .01 \)) phases. The APA\(_1\) \( (p = .74) \) and the unipedal phase (LOC\(_U\); \( p = .86 \)) were not different between groups. Relative timing between groups showed equal postural \( (p = .74) \) and locomotor \( (p = .74) \) proportions and equal duration prior to movement onset \( (p = .86) \). However, within the postural and locomotor phases, the MS participants displayed shifts in relative timing. The MS participants had a relatively shorter APA\(_1\) \( (p = .02) \) and a trend toward a relatively longer APA\(_2\) \( (p = .07) \) in the postural phase, and a relatively shorter LOC\(_U\) \( (p = .02) \) and a relatively longer LOC\(_D\) \( (p = .02) \) in the locomotor phase compared with controls.

**Coordination in the APA**

The local coupling measure showed that the MS participants coordinated CoP patterns differently from the control group during the APA (Table 4). The MS participants used a more orthogonal CoP displacement in APA\(_1\) (smaller coupling angle; \( p = .042 \)) and a more synchronized CoP displacement in APA\(_2\) (larger coupling angle; \( p = .028 \)) than the control group.

**Time to Contact Within the Stability Boundary in the APA**

In APA\(_1\), there was a trend toward longer TtC in the MS participants for CoP, \( F(1, 22) = 3.42, p = .07 \), and CoM, \( F(1, 22) = 3.73, p = .06 \), but these failed to reach a significant difference from the control group. In APA\(_2\), TtC was longer in the MS participants for both CoP, \( F(1, 22) = 6.88, p = .01 \), and CoM, \( F(1, 22) = 15.33, p < .001 \), (Figure 3) than in the control group.

**Discussion**

The purpose of this study was to characterize the effect of Multiple Sclerosis on spatiotemporal patterns across the gait initiation sequence as well as to examine coordination and temporal margins within the stability boundary during the APA portion of gait initiation. We hypothesized that people with MS initiate gait with a slower CoM velocity and a smaller posterior CoP shift during the APA than controls. We further hypothesized that increased temporal margins within the stability boundary are associated with the MS group initiation patterns during the APA phase.

Based on our results, the women with MS initiated gait with a slower velocity and a smaller posterior CoP shift during the APA than did controls. Lower CoM velocities allowed the MS group to minimize CoM displacements beyond the anterior stability boundary during the unipedal phase of GI. Postponing the
Table 2  Displacement (mm) Mean (SD) for MS and Control Groups

<table>
<thead>
<tr>
<th>Distance (mm)</th>
<th>CoP APA₁</th>
<th>CoP APA₂</th>
<th>Step length</th>
<th>CoM</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M/L</td>
<td>A/P</td>
<td>M/L</td>
<td>A/P</td>
</tr>
<tr>
<td>MS</td>
<td>26 (± 14)</td>
<td>-10 (± 8)</td>
<td>-129 (± 28)</td>
<td>0 (± 23)</td>
</tr>
<tr>
<td>Control</td>
<td>30 (± 11)</td>
<td>-22 (± 15)</td>
<td>-125 (± 19)</td>
<td>-3 (± 17)</td>
</tr>
<tr>
<td>F(1, 22)</td>
<td>0.84</td>
<td>8.49</td>
<td>0.43</td>
<td>0.17</td>
</tr>
<tr>
<td>p</td>
<td>.37</td>
<td>.01</td>
<td>.52</td>
<td>.68</td>
</tr>
</tbody>
</table>

Note. Positive M/L motion is toward swing (right) foot, positive A/P motion is anterior. APA₁: anticipatory postural adjustment phase one (from movement onset or first bilateral fluctuations of vertical force [MO] to release of swing foot vertical loading [R]); APA₂: anticipatory postural adjustment phase two (from R to swing foot toe-off). CoM M/L max: maximum lateral displacement of CoM from MO toward the stance foot during GI. CoM A/P at HS: anterior displacement of CoM beyond the base of support at the end of unipedal phase of GI (at swing foot heel-strike).
Table 3  Timing Mean (SD) for MS and Control Groups in Absolute (ms) and Relative (Percent of GI) Measures

<table>
<thead>
<tr>
<th>Absolute timing</th>
<th>Preparatory phase (ms)</th>
<th>Postural phase (ms)</th>
<th>Locomotor phase (ms)</th>
<th>Postural phase (%)</th>
<th>Locomotor phase (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>APA₁</td>
<td>APA₂</td>
<td>Total</td>
<td>LOCᵤ</td>
</tr>
<tr>
<td>MS</td>
<td>420 (± 80)</td>
<td>244 (± 59)</td>
<td>531 (± 176)</td>
<td>775 (± 270)</td>
<td>477 (± 85)</td>
</tr>
<tr>
<td>Control</td>
<td>349 (± 70)</td>
<td>251 (± 45)</td>
<td>379 (± 113)</td>
<td>630 (± 140)</td>
<td>471 (± 70)</td>
</tr>
<tr>
<td>F(1, 22)</td>
<td>4.81</td>
<td>0.11</td>
<td>6.34</td>
<td>5.74</td>
<td>0.03</td>
</tr>
<tr>
<td>p</td>
<td>.04</td>
<td>.74</td>
<td>.02</td>
<td>.03</td>
<td>.86</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Relative timing</th>
<th>Preparatory phase (%)</th>
<th>Postural phase (%)</th>
<th>Locomotor phase (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>APA₁</td>
<td>APA₂</td>
</tr>
<tr>
<td>MS</td>
<td>25.1 (±7.3)</td>
<td>14.6 (±3.4)</td>
<td>31.8 (±5.4)</td>
</tr>
<tr>
<td>Control</td>
<td>25.3 (±7.4)</td>
<td>18.2 (±3.5)</td>
<td>27.4 (±4.7)</td>
</tr>
<tr>
<td>F(1, 22)</td>
<td>0.02</td>
<td>6.07</td>
<td>3.54</td>
</tr>
<tr>
<td>p</td>
<td>.86</td>
<td>.02</td>
<td>.07</td>
</tr>
</tbody>
</table>

Note. Preparatory phase (from initiation signal to first bilateral fluctuations of vertical force [MO]). Postural phases of GI: APA₁: anticipatory postural adjustment phase one (from MO to release of swing foot vertical loading [R]); APA₂: anticipatory postural adjustment phase two (from R to swing foot toe-off). Locomotor phases of GI: LOCᵤ: unipedal phase (from swing foot toe-off to swing foot heel-strike); LOC₉: dual support phase (from swing foot heel-strike to stance foot toe-off). GI total: duration of entire postural and locomotor phases or GI sequence.
displacement of the CoM beyond the anterior boundary of the base of support may improve stability in the MS group during the locomotor phase of GI. During the postural phase of GI, the longer temporal margins within the stability boundary may also enhance balance in the MS group. Furthermore, a more synchronized shift of CoP (LCₐ) during APA₂ may signify a control strategy that avoids a direct approach to the lateral stability boundary.

**Invariance in Duration of GI**

Brenière et al. (1987) reported that gait initiation duration remains invariant within a group, regardless of the speed at which participants are instructed to begin walking (slow, normal, and fast). They further noted that APA duration decreases as GI velocity decreases. A study comparing healthy adults to people with unilateral

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**Table 4** Local Coupling Coordination Angle (deg) Mean (SD) of CoP Shifts During the APA for MS and Control Groups

<table>
<thead>
<tr>
<th>Local coupling angle (deg)</th>
<th>CoP APA₁</th>
<th>CoP APA₂</th>
</tr>
</thead>
<tbody>
<tr>
<td>MS</td>
<td>22.52 (± 4.81)</td>
<td>20.00 (± 4.13)</td>
</tr>
<tr>
<td>Control</td>
<td>28.16 (± 7.51)</td>
<td>16.32 (± 4.32)</td>
</tr>
<tr>
<td>F(1, 22)</td>
<td>4.38</td>
<td>5.53</td>
</tr>
<tr>
<td>p</td>
<td>.042</td>
<td>.028</td>
</tr>
</tbody>
</table>

*Note.* Postural phases of GI: APA₁: anticipatory postural adjustment phase one (from movement onset [MO] to release of swing foot vertical loading [R]); APA₂: anticipatory postural adjustment phase two (from R to swing foot toe-off).

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**Figure 3** — Box plots of Time to Contact with the stability boundary (s) in APA₁ and APA₂ (anticipatory postural adjustment phases one and two) of GI: CoP (left) and CoM (right) of MS (shaded) and the control group.
transtibial limb loss (Miff, Childress, Gard, Meier, & Hansen, 2005) also observed invariance in GI duration across gait speeds in both populations but noted an invariance in APA duration across GI speeds as well. In contrast, when we compare our MS group with our control group, the MS participants increased both their total GI and APA durations while initiating gait at lower speeds compared with controls, which may be evidence of some reorganization of the GI sequence because of MS. Although some temporal patterns of GI may have changed in these MS participants, the unipedal locomotor time (LOCU) remained the same between groups. In healthy adults, faster GI speeds sustain shorter swing times (Brenière et al., 1987). Based on this, we expected our MS group to have a longer unipedal phase compared with controls but, this was not observed. Our MS group may have been limiting the unipedal phase and lengthening the dual support phase to create a safer stepping strategy. This trend was also observed during gait in people with MS (Benedetti et al., 1999).

To ensure that group comparisons between our MS and control groups are generalizable, we compared our control group data with data from previous work on healthy adults. Our control group of middle-aged women initiated gait slower (mean = 0.81 m/s) than participants of other studies. Results from GI experiments at normal speeds in healthy younger men and women (ages 18–35 years) reported velocities of 1.35 m/s (Brenière, Do, & Sanchez, 1981), 1.68 m/s (Halliday et al., 1998), and 1.20 m/s (Brunt et al., 1991). Our control group of middle-aged women also took longer to complete GI (mean = 1.38 s) than most other reports, although definitions of the GI sequence vary. Younger healthy adults completed GI in 1.15 s with GI similarly defined (Nissan & Whittle, 1990), 1.23 s with GI defined as initiation signal to stance foot toe-off (Brunt et al., 1991), and 1.07 s (Brenière et al., 1981) and 1.61 s (Miff et al., 2005) with GI defined as from movement onset to first zero crossing of CoM acceleration. This zero crossing typically occurs late in the dual support phase of GI.

While differences in absolute speed or time may vary due to group characteristics, laboratory conditions, or experimental instructions, relative timing of events within an initiation sequence may yield movement invariances not otherwise apparent. Relative timing in our experiment shows that both MS and control groups spent about 46% of GI in the postural phase (APA) and 54% of GI in the locomotor phase. This result points at invariant temporal properties regardless of stepping strategy. A within group relative timing invariance between postural and locomotor phases while initiating gait at different speeds was found by Brunt et al. (1991); across gait speeds, the APA duration was approximately 45% of total GI time. In contrast to the relative timing invariance of total APA, our MS group showed a relatively shorter APA, compared with the control group. Additionally, the MS group showed a relatively shorter unipedal phase and a longer dual support phase in comparison with controls. The MS group may be adapting GI proportions within postural and locomotor phases to ensure safe stepping.

**Dynamic Postural Stability and Gait Initiation**

While the lower GI speed in our MS participants correlates with their smaller posterior CoP shifts during the APA, their lower speed may be a choice that allows a
safer movement strategy. One way to avoid confounding speed and disease issues is to use a measure based on dynamics such as time to contact within the stability boundary, which allows for comparisons that do not require the removal of velocity effects. During APA of GI, MS participants increased the temporal margin within the stability boundary while shifting the CoP in a less direct manner (more synchronous movement between A/P and M/L directions), which may be minimizing potential threats to balance or compensating for postural problems. Smaller and slower shifts in the CoP that approach stability boundaries less directly lowers CoM acceleration and displacement, and this may minimize threats to balance.

MS participants may be minimizing postural threats by keeping both the CoP and CoM from rapidly approaching undesirable stability boundaries (posterior, lateral) and postponing voluntary stability-boundary crossings (anterior). Avoiding undesirable boundaries may be an important factor in maintaining postural stability, especially during CoP shifts in the posterior direction. During maximal lean tasks it has been found that approaches to the posterior stability boundary induce greater levels of postural variability than anterior or lateral approaches (Van Emmerik & Van Wegen, 2002).

An explanation for previously observed increases of postural variability during posterior boundary approaches includes a lack of direct visual feedback information and the anatomical limitation of foot length. If the MS participants, with the additional challenge of peripheral somatosensory changes, produce rapid or large posterior CoP displacements during the APA, the outcome may be even less predictable than comparable movements made by the control group. Somatosensory information in the MS population may be distorted or delayed, which is an important contribution to ataxia and the ability to scale the magnitude of a postural response appropriately (Horak, 2001). However, differences in GI speed may also relate to changes in strength or power of the lower limb musculature. It remains unclear how changes in strength and power of the lower limbs may affect the initiation of gait and locomotion in MS in general.

Clinical Implications

The results from this study on people with MS have much in common with investigations of GI in children, older individuals, and people with Parkinson’s disease and contrast with studies on people with transtibial limb loss. A group of 4- to 6-year-old children (Malouin & Richards, 2000) initiated gait with slower velocities and smaller posterior CoP shifts in the APA than adults, in a similar pattern as employed by our MS group. Parkinson’s patients showed the slowest anterior CoM velocity and the smallest posterior CoP shift in the APA, followed by healthy elders, when compared with young adults (Halliday et al., 1998), but with similar APA durations. In the Parkinsonian group, however, GI time was significantly longer than the young and elderly groups. It appears that our MS group shared strategies with the Parkinson’s group to slow CoM velocity, reduce posterior CoP displacement in the APA, and lengthen GI durations. In contrast, a population with no neurological disorder but with a transtibial limb loss had similar GI times as controls across a range of gait speeds. This result suggests that neurological disorders may influence GI parameters more than physical disability (Miff et al., 2005) or aging.
There may be several reasons why certain populations scale gait speed, posterior CoP shifts in the APA, and GI duration, since less pronounced body perturbations might ensure a safer transition to locomotion. Additionally, if there is inexperience (as in children) or a neurologic disorder with symptoms like ataxia that produces suboptimal postural control, a smaller shift that keeps the CoP away from the undesirable stability boundaries (posterior, lateral) may also add safety to dynamic tasks like gait initiation. Understanding how different populations adapt movement strategies to maintain functionality may lead to clinical measures that can gauge functional disability.

**Conclusions**

When initiating gait at their preferred speed, the MS participants avoided unstable postural states during the postural (APA) and locomotor phases by adopting a functional gait initiation strategy. This strategy featured slower speed, a smaller posterior CoP displacement in the APA, and a reduced anterior excursion of the CoM beyond the base of support during the unipedal stance phase. Further changes in GI strategy that may be less directly related to velocity include reduced temporal margins within the stability boundary associated with coordination changes during the APA, longer initiation times, and a shorter relative unipedal stance time. The expanded use of stability boundary relevant postural stability measures (TtC) can help to understand how movement strategies change to maintain stability during dynamic tasks in populations experiencing balance problems. Compensating for balance problems by adopting a functional strategy is generally effective when people have the option to move in their preferred manner. It remains unclear how balance problems affect dynamic stability in people with MS when larger, faster, or unexpected perturbations occur.

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**References**


