Gait Termination Control Strategies Are Altered in Chronic Ankle Instability Subjects

ERIK A. WIKSTROM1,2, MARK D. BISHOP3, AMRUTA D. INAMDAR4, and CHRIS J. HASS4

1Biodynamics Research Laboratory, Department of Kinesiology, University of North Carolina at Charlotte, Charlotte, NC; 2Center for Biomedical Engineering Systems, University of North Carolina at Charlotte, Charlotte, NC; and 3Department of Physical Therapy, University of Florida, Gainesville, FL; and 4Center for Exercise Science, Department of Applied Physiology and Kinesiology, University of Florida, Gainesville, FL

ABSTRACT

WIKSTROM, E. A., M. D. BISHOP, A. D. INAMDAR, and C. J. HASS. Gait Termination Control Strategies Are Altered in Chronic Ankle Instability Subjects. Med. Sci. Sports Exerc., Vol. 42, No. 1, pp. 197–205, 2010. Despite the high incidence of chronic ankle instability (CAI), the underlying neurophysiologic mechanism is unknown. Evidence suggests that both feed-forward and feedback mechanisms may play a role. However, no investigation has examined both control mechanisms during the same movement task in the same cohort of CAI patients. Purpose: To determine the neuromuscular and biomechanical control alterations present in CAI patients during planned (feed-forward) and unplanned (feedback) gait termination. Methods: Twenty subjects with CAI and 20 uninjured controls completed planned and unplanned gait termination protocols. Both tasks began with subjects walking at a self-selected speed across a 12-m walkway. Unplanned gait termination required subjects to stop during randomly selected trials on two adjacent force plates when cued. Planned gait termination required purposeful stopping on the force plates. Propulsive and braking force magnitude and the dynamic postural stability index were calculated from the resulting ground reaction forces. In addition, muscle activity from the soleus, tibialis anterior, and gluteus medius was collected bilaterally. Results: Both maximum propulsive (CAI = 99.8 ± 40.8 N, control = 88.6 ± 33.6 N) and braking (CAI = 207.1 ± 80.9 N, control = 161.6 ± 62.2 N) forces were significantly higher in the CAI group. The dynamic postural stability index revealed higher scores in the CAI group (0.24 ± 0.03) compared with the control group (0.22 ± 0.03). Muscle activation of the soleus and tibialis anterior differed during unplanned and planned gait termination between groups (P < 0.05) and between the limbs of the CAI group (P < 0.05). Conclusions: Altered biomechanical strategies during both planned and unplanned gait termination indicate that patients with CAI have alterations in feed-forward neuromuscular control and suggest the presence of feedback neuromuscular control deficits. Key Words: EMG, KINETICS, STOPPING, DYNAMIC POSTURAL CONTROL

Address for correspondence: Erik A. Wikstrom, Ph.D., ATC, Department of Kinesiology, University of North Carolina at Charlotte, 9201 University City Blvd, Charlotte, NC 28223; E-mail: ewikstrom@uncc.edu. Submitted for publication February 2009. Accepted for publication May 2009.

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the lower extremity (2–4,23). Furthermore, planned and unplanned gait termination have characteristically different kinetic and muscle recruitment patterns (29), thus experiments can be designed to challenge both feed-forward neuromuscular control (i.e., planned stopping) and the motor response to an external stimuli (i.e., unplanned stopping), which is heavily influence by feedback neuromuscular control mechanisms.

Previously, EMG studies have demonstrated altered evertor muscle activity during uninterrupted gait (9) and altered evertor, dorsiflexor, and plantarflexor muscle activity during jump landings (10,13) in CAI patients. However, Hase and Stein (15) have indicated that about the ankle, only the tibialis anterior (TA) and soleus (SOL) play a major role in gait termination. In addition, the dynamic postural stability index (DPSI) (36,37) has illustrated balance deficits in CAI patients as they transitioned from a dynamic to a static state during jump landing investigations. However, neither EMG nor the DPSI has examined neuromuscular control in subjects with CAI during gait termination.

Therefore, the purpose of this investigation was to determine neuromuscular and biomechanical control alterations present in CAI patients during planned and unplanned gait termination. We hypothesized that during planned and unplanned gait termination, CAI patients would demonstrate a limited repertoire of available stopping strategies as indicated by increased reliance on a single-limb braking force strategy rather than concurrently modulating both propulsive and braking forces as normal healthy controls will. In addition, we anticipated increased DPSI scores in patients with CAI when these subjects are required to stabilize using their injured extremity.

METHODS

Experimental Design and Subjects

This investigation was a single-session mixed-model design that examined the neuromuscular control strategies in 20 subjects with CAI (20.5 ± 1.0 yr, 169.8 ± 9.8 cm, 74.2 ± 20.2 kg) and 20 uninjured controls (20.85 ± 1.6 yr, 164.3 ± 7.9 cm, 64.2 ± 10.62 kg) during planned and unplanned gait termination. All participants read and signed the Institutional Review Board (IRB)-approved informed consent form before participation. All participants were recreationally active, which was operationally defined as performing three aerobic exercise sessions per week for a total of 90 min.

Those with CAI met specific criteria including 1) a history of at least one unilateral lateral ankle sprain that required immobilization for at least 3 d (mean ± SD: 11.5 ± 9.2 d); 2) at least one episode of giving way within the past year (8.5 ± 6.7); 3) at least one recurrent ankle sprain between 3 and 6 months before study participation (2.9 ± 1.8); 4) a report of pain, instability, and/or weakness in the involved ankle; 5) attributing these signs to their initial ankle injury; 6) failure to resume all preinjury level of activities; 7) no previous ankle fractures; 8) no previous head and acute lower extremity injury within the past 3 months; and 9) no formal rehabilitation of the involved ankle (37). The requirement of a re-injury between 6 and 3 months before participation was to ensure that subjects still had physical manifestations of CAI but that acute symptoms would have been resolved. Uninjured controls were excluded if they were not free from acute lower extremity and head injuries for the previous 3 months and did not suffer from any equilibrium disorders or chronic lower extremity pathologies. Unpublished data from our laboratory illustrate strong significant negative correlations (−0.379 to −0.694) between our CAI inclusionary criteria and several validated questionnaires of self-reported function. Therefore, questionnaires of self-reported function were not used to categorize CAI subjects in the current investigation.

Protocol

Testing was completed in the Applied Neuromechanics Laboratory at the University of Florida. All trials of gait and gait termination were performed along a 12-m walkway surrounded by an eight-camera optical motion capture system (120 Hz; Vicon Peak, Lake Forrest, CA). Ground reaction forces were collected at 1200 Hz using two force platforms (Bertec, Corp, Columbus, OH) mounted flush with the surrounding floor. Before data collection, demographic and anthropometric measurements were taken. Subjects were tested barefoot in form fitting clothing. Thirty-six passive retroreflective markers were then placed over anatomical landmarks according to the Plug-in-Gait marker system. The 36 markers were used to construct a simple 15-segment model. Estimates of segment COM were based on Dempster’s anthropometric data, and the location of whole-body COM was calculated using Vicon software.

Bipolar 1 × 10-mm² Ag/AgCl surface electrodes with an interelectrode distance of 1.5 cm were then placed over six lower extremity muscles according to previously established criteria (25). Before the electrode application, the skin was shaved and cleaned with alcohol to reduce skin impedance. Electrode placement was confirmed with manual muscle testing and checked for cross-talk with real-time oscilloscope displays. EMG data were collected bilaterally from the TA, SOL, and gluteus medius (GM) using a Konigsberg T-42AL-8T telemetric EMG system (Konigsberg Instruments, Inc, Pasadena, CA) because previous research has identified these muscles as playing a major role in gait termination (15). EMG data were time-synchronized to the collection of force plate and kinematic data and were used to confirm the kinetic results of gait and gait termination trials. Once instrumented, subjects were instructed on and familiarized with the first testing protocol, unplanned gait termination.
Unplanned gait termination. During this experimental protocol, participants completed two tasks: normal gait and unplanned gait termination. The sequence of completing these tasks was randomly generated before data collection, accounted for multiple failed trials, and was unique for each subject. Both tasks began with subjects walking at a self-selected speed across a 12-m walkway with their head up looking at a target placed at eye level at the far end of the walkway. According to the task sequence, subjects were cued to stop during randomly selected trials (2,3) using an audio cue signal. These randomly selected trials represented the unplanned gait termination task, during which the gait velocity of the CAI (1.25 ± 0.11 m s⁻¹) and controls (1.24 ± 0.11 m s⁻¹) did not differ (P > 0.05). The audio cue was manually triggered at heel strike (mean ± SD: 6.1 ± 0.1 ms after heel strike) of the lead limb, which allowed subjects to stop within the next foot strike after the onset of the audio cue (24). The delivery timing of the audio cue was equal between the CAI (0.05 ± 0.08 ms) and the control (0.07 ± 0.07 ms) groups (P > 0.05). Because CAI subjects only had a unilateral pathology, each limb (right, left) served as the lead limb in five unplanned gait termination trials (3). Normal gait trials (catch) were integrated into this protocol to prevent counting or anticipating of the audio cue. A total of five catch trials were collected, but the total number of catch trials conducted equaled at least 50% of all trials conducted in this experimental protocol per subject. Gait velocity during catch trials did not differ between the CAI group (1.24 ± 0.11 m s⁻¹) and the control (1.22 ± 0.10 m s⁻¹) group (P > 0.05). More importantly, gait velocity did not differ between catch (1.23 ± 0.11 m s⁻¹) and unplanned (1.25 ± 0.11 m s⁻¹) gait termination trials (P > 0.05). Failed catch or unplanned gait termination trials, defined as a subject missing the force plates in the either the sagittal or the frontal plane, forgetting to stop on an audio cue, or failing to stop in a single step after the audio cue, were discarded and repeated in accordance with the task sequence assigned to the respective subject. There was no apparent difference in the number of failed trials between groups.

Planned gait termination. Before data collection, subjects were allowed to practice this protocol so that their natural footfall would occur on the force plates. The protocol also began with subjects walking at a self-selected speed, which did not differ between the CAI (1.20 ± 0.11 m s⁻¹) and the control (1.20 ± 0.12 m s⁻¹) groups (P > 0.05), across a 12-m walkway with their head up, but subjects stopped on the second force plate. A total of 10 trials were recorded with each limb serving as the lead limb in five trials. Failed trials were similarly discarded and repeated.

Data Reduction

All kinematic and kinetic data were reduced using Vicon Nexus 1.311 software. Gait velocity was calculated by determining the velocity of the whole-body COM during the stride before the stop cue during unplanned trials and the same stride (relative to position in the room) during catch and planned trials. Ground reaction forces were exported and reduced using Microsoft Excel (Microsoft, Corp, Redman, WA). The magnitudes of the propulsive and braking force data were recorded during foot contact with the first and second force plates, respectively, during gait termination and catch trials.

EMG. Raw EMG data were band-pass–filtered from 20 to 4000 Hz and then smoothed using a sliding average with a window frame of 25 ms. The smoothed integrated EMG data were used to calculate muscle activity during multiple phases of gait. Specifically, six phases of gait were identified on the basis of identifiable vertical ground reaction force landmarks: four during the stance phase of the lead limb (force plate no. 1) and two for the swing leg (force plate no. 2). Whereas the subphases of the lead limb had been previously established (2,3), we were also interested in capturing the muscle activity of the swing limb as the subject prepared to and then applied a braking force. Therefore, two subphases were created: subphase no. 5, from second peak loading of the lead limb (identified by ground reaction forces on force plate no. 1) to heel strike of the swing limb on force plate no. 2; and subphase no. 6, heel strike of the swing limb to peak loading of the swing limb. After determining muscle activity for each leg during normal gait, an identical reduction technique was completed for both gait termination protocols. The muscle activity of each limb during each subphase of gait termination was then normalized to the corresponding limb’s subphase muscle activity during normal gait (3).

Dynamic postural stability. Dynamic postural stability was measured from ground reaction force data during both planned and unplanned gait termination trials. Dynamic postural stability was calculated within a 1-s interval starting from the time of heel strike on the second force plate (swing limb). The initiation of data collection and temporal parameters were chosen to capture the transition from dynamic to a static state while also transitioning from a double- to single-limb stance phase of gait termination. The collection window is shorter than those previously reported (36,37) because pilot data demonstrated a more rapid decay of the ground reaction forces during gait termination relative to jump landings, most likely because gait termination is a less ballistic task. Subjects were instructed to stabilize and remain motionless as quickly as possible after they had stopped. Trials were reduced using a custom LabVIEW program (National Instruments, Austin, TX) that calculated a modified DPSI and modified directional indices as described by Wikstrom et al. (37). Specifically, each data point in all directions was divided by the subject’s mass, thus producing normalized scores that facilitated individual comparisons. These indices represent the SD fluctuations around a zero point, so that higher scores indicate greater variability. The directional indices...
(Mediolateral Stability index (MLSI), Anterioposterior Stability index (APSI), and Vertical Stability index (VSI)) correspond with the frontal (Y), sagittal (X), and transverse (Z) axes of the force plate, respectively. The DPSI is a composite score and is thus sensitive to changes in each direction.

Statistical Analysis

A three-way MANOVA (2 (group) × 2 (limb) × 3 (condition)) with repeated measures on the last factor was computed for propulsive and braking force using Wilks’ λ for interpretation. Bonferroni post hoc analysis was performed when necessary. EMG data were analyzed using separate three-way ANOVA for the stance limb (2 (group) × 4 (phase) × 2 (limb)) and swing limb (2 (group) × 2 (phase) × 2 (limb)). Our specific comparisons of interest were interactions between group and phase and main effects for group. Two-way ANOVA were performed to specifically compare the activation of the injured limb to the uninjured limb in subjects with CAI. Separate comparisons were performed for each task. A three-way MANOVA (2 (group) × 2 (limb) × 2 (condition)) was computed for the dynamic postural control variables. A traditional level of significance (α = 0.05) was used on all statistical tests, and a Bonferroni correction for multiple comparisons was conducted when necessary. In addition, effect sizes were calculated for force and DPSI differences between uninjured controls and those with CAI and interpreted (small = 0.10–0.24, medium = 0.25–0.39, large > 0.40) according to Cohen (6).

RESULTS

Propulsive and braking forces. The three-way MANOVA revealed significant group \( F(2,189) = 12.25, P < 0.01 \) and condition \( F(4,378) = 67.57, P < 0.01 \) main effects. Specifically, both maximum propulsive and braking forces were significantly greater in the CAI group compared with the control group (Fig. 1). In addition, post hoc testing revealed that: 1) the maximum propulsive force generated during normal walking (catch trials) was significantly greater than that produced during planned and unplanned gait termination trials and 2) maximum braking forces were

![Figure 1](http://www.acsm-msse.org)

**FIGURE 1**—Group \( \times \) condition means and SD for the maximum propulsive and braking forces. The maximum braking force during catch trials represents the maximum impulse. *Statistically different \( (P < 0.05) \) from planned gait termination maximum propulsive forces. †Statistically different \( (P < 0.05) \) from unplanned gait termination maximum propulsive forces. ‡Statistically different \( (P < 0.05) \) from unplanned gait termination maximum braking forces. §Statistically different \( (P < 0.05) \) from the control group for both maximum propulsive and braking forces.

![Figure 2](http://www.acsm-msse.org)

**FIGURE 2**—Lead limb muscle activation during planned gait termination. Phases are based on the corresponding ground reaction forces as indicated in the top center of the figure. Phase no. 1: Heel strike to peak loading. Phase no. 2: Peak loading to midstance. Phase no. 3: Midstance to the second peak loading. Phase no. 4: Second peak loading to toe off.
significantly different among all three conditions. However, no significant two-way interactions were detected: condition × group \( [F(4,380) = 0.94, P = 0.441] \), condition × limb \( [F(2,189) = 0.14, P = 0.873] \), or group × limb \( [F(2,189) = 0.95, P = 0.953] \). Similarly, no significant condition × group × limb \( [F(2,189) = 0.67, P = 0.935] \) interaction or limb main effect \( [F(2,189) = 0.022, P = 0.978] \) was noted.

**Planned gait termination EMG.** Distinct muscle activation patterns were observed for all subjects consistent with previously reported stereotypical activation patterns associated with gait termination. Lead limb, TA \( [F(1,36) = 6.8, P = 0.001] \) and GM \( [F(1,36) = 6.2, P = 0.017] \) activation increased during phase 4, whereas SOL activity \( [F(1,36) = 5.3, P = 0.027] \) decreased during midstance, phase 3 (Fig. 2). For the swing limb, all muscles were

![FIGURE 3](image-url) Swing limb muscle activation during planned gait termination. Phases are based on the corresponding ground reaction forces as indicated in the top right corner of the figure. Phase no. 5: Second peak loading of the lead limb to heel strike of the swing limb. Phase no. 6: Heel strike of the swing limb to peak loading of the swing limb.

![FIGURE 4](image-url) Lead limb muscle activation during unplanned gait termination. Phases are based on the corresponding ground reaction forces as indicated in the top center of the figure. Phase no. 1: Heel strike to peak loading. Phase no. 2: Peak loading to midstance. Phase no. 3: Midstance to the second peak loading. Phase no. 4: Second peak loading to toe off.
activated to a greater degree than during comparable phase in normal walking (Fig. 3).

Group differences were evident for TA \( F(1,36) = 5.3, P = 0.027 \), SOL \( F(1,36) = 4.9, P = 0.033 \), and GM \( F(1,36) = 3.8, P = 0.030 \) on the lead limb. In each case, subjects in the control group activated these muscles to greater extent than members of the CAI group. No such main effects were observed for the swing limb. For the CAI group, significant interactions were noted between limb and phase for TA \( F(1,17) = 5.4, P = 0.032 \) and SOL \( F(1,17) = 4.7, P = 0.040 \). Specifically, TA activation of the injured limb was lower than the uninjured during phase 4, and SOL activation of the injured limb was greater than the uninjured throughout. Potentially, this scenario indicates that inhibition of the SOL did not occur when the injured limb was the lead limb during planned stopping. In the swing limb, SOL activation was much greater on the uninjured limb \( F(1,17) = 4.3, P = 0.050 \). No other effects were noted.

**Unplanned gait termination EMG.** Similar to planned stopping, the expected phase effects were noted with increases in TA \( F(1,36) = 19.2, P < 0.001 \) and GM \( F(1,36) = 9.1, P = 0.005 \) activation during late stance (phase 4) and inhibition of SOL \( F(1,36) = 6.5, P = 0.016 \) during phase 3. Likewise, lead limb activation of the extensor muscles (SOL and GM) was greater than the equivalent phases of normal gait (Fig. 4). In contrast to planned stopping, however, an interaction between phase and group occurred for TA \( F(1,36) = 6.5, P = 0.015 \) whereby control subjects demonstrated steady increases in activation of the TA throughout stance phase, whereas subjects with CAI increased TA activity very late in stance. No other interactions were noted.

When activation was compared between the injured and uninjured limbs of subjects with CAI, no limb × phase interactions were noted for any of the muscles tested on the lead limb. For the swing limb, there was a limb effect for TA \( F(1,17) = 4.7, P = 0.038 \). Specifically, when the injured limb was the swing limb, the preparatory TA activity (phase 5—before initial contact) was 150% of the uninjured side (Fig. 5). No other effects were noted.

**Dynamic postural stability.** A three-way MANOVA revealed significant main effects for group \( F(4,149) = 4.68, P < 0.001 \) and condition \( F(4,149) = 8.39, P < 0.001 \). Subsequent post hoc comparisons indicated that the CAI group had significantly higher APSI \( (0.15 \pm 0.03) \) and DPSI \( (0.24 \pm 0.03) \) scores relative to the control group (APSI: \( 0.13 \pm 0.02 \), DPSI: \( 0.22 \pm 0.03 \)). No group differences were detected for the MLSI (CAI: \( 0.03 \pm 0.01 \), control: \( 0.03 \pm 0.02 \)) or VSI (CAI: \( 0.18 \pm 0.04 \), control: \( 0.17 \pm 0.03 \)). Similarly, post hoc comparisons revealed that unplanned gait termination produced greater APSI \( (0.15 \pm 0.02) \) and DPSI \( (0.23 \pm 0.03) \) scores relative to planned gait termination (APSI: \( 0.13 \pm 0.02 \), DPSI: \( 0.22 \pm 0.03 \)). However, no significant limb main effect \( F(4,149) = 1.82, P = 0.12 \) or condition × group \( F(4,149) = 0.77, P = 0.54 \), condition × limb \( F(4,149) = 0.27, P = 0.89 \), group × limb \( F(4,149) = 0.89, P = 0.46 \), or condition × group × limb \( F(4,149) = 0.87, P = 0.48 \) interactions occurred.

**DISCUSSION**

The principal finding of this investigation was that patients with CAI had neuromuscular and biomechanical
control alterations during both planned and unplanned gait termination indicated by a diminished ability to reduce propulsive forces and thus increased reliance on braking forces of the swing limb. Furthermore, APSI and DPSI scores were increased in subjects with CAI during both planned and unplanned gait termination. Thus, the results support the hypothesis that development of CAI leads to altered gait termination strategies.

Biomechanical strategies. Findings from experimental studies indicate that subjects without neurological impairment modulate force production bilaterally such that the limb behind the COM (lead limb) reduces propulsive forces as the limb in front of the COM (swing limb) concurrently increases braking forces. The current investigation demonstrated that subjects with CAI had significantly higher propulsive forces relative to uninjured controls during both planned ($d = 0.43$) and unplanned ($d = 0.28$) gait termination. This finding suggests a reduced ability to appropriately alter muscular activity to reduce the velocity of the COM while the lead limb is in contact with the ground. As a result, CAI subjects had to produce significantly higher braking forces in their swing limb, injured or uninjured, to stop within one step during both planned ($d = 0.69$) and unplanned ($d = 0.67$) gait termination. This suggests that the demands of both planned to unplanned stopping lead to bilateral (feed-forward) alterations in neuromuscular control in CAI subjects.

Although speculative, we believe that a delayed delivery or activation of the centrally mediated and stereotypical TA activation and SOL inhibition after the audio cue may explain the increased propulsive forces in the CAI group during unplanned gait termination. Delayed SOL inhibition and TA activation may be caused by alterations in the gamma motoneuron system, making both the TA and SOL muscle spindles less sensitive to changes in muscle length. Thus, SOL force generation was likely greater during a longer period in CAI subjects. Interestingly, during the late stance phase of planned stopping, lead limb TA activity was decreased in CAI subjects relative to controls. Furthermore, TA activity was decreased, whereas SOL activity was increased in the injured limb relative to the uninjured limb of CAI subjects. Potentially, these findings represent a reduction in the available strategies used to terminate gait (planned or unplanned) such that patients with CAI predominantly relied on their swing limb to stop their forward momentum. Similar patterns in both planned and unplanned stopping not only provide strong evidence of feed-forward neuromuscular control deficits in CAI subjects but also suggest that feedback deficits may also exist.

Previous empirical data have revealed deficits in proprioception and muscle latency in subjects with CAI relative to controls, which suggest that subjects with CAI have some level of feedback impairment but the literature is controversial at best. Unplanned stopping is modulated in part by feedback neuromuscular control because subjects are reacting to an external stimulus. The primary external stimulus, in this case, was an auditory cue. However, peripheral feedback would also occur regarding velocity (and, therefore, the amount of force required to stop) and contribute to the estimate of the magnitude of activation required to achieve the desired outcome. Late TA activation suggests the presence of longer muscle latencies in CAI subjects, and this longer latency (and potentially delayed response) may be responsible for the increased propulsive and subsequent braking forces during unplanned gait termination. However, anticipation of the audio cue, despite randomization and the large number of catch trials may also have affected the results. Therefore, feedback deficits may be present in subjects with CAI, but current sensorimotor measure limitations prevent the determination of local versus central alterations.

Even if we assume that feedback deficits are present, feedback deficits alone do not explain the similar kinetic pattern seen during planned gait termination. Indeed, group differences in planned gait termination may have more clinical relevance given the larger effect sizes and can only be explained by alterations in feed-forward neuromuscular control. Previous research supports this finding and has demonstrated that the development of CAI leads to altered movement patterns relative to uninjured controls during both gait and jump landings. Furthermore, examination of muscular activity within the swing limb during planned gait termination suggests that the uninjured limb acts similarly to the limbs of the uninjured control subjects. However, when the ankle with CAI is acting as the swing limb, it seems that the SOL and TA are cocontracted before and at heel strike, which may improve ankle stability. Previous reports indicate that CAI subjects land in a more dorsiflexed position but have less dorsiflexion at heel strike while jogging. It is possible that the CAI subjects in these investigations were also cocontracted, but EMG data were not reported. Similarly, we were unable to determine the amount of dorsiflexion present at heel strike in our current CAI sample.

Whereas alterations in feed-forward neuromuscular control are likely responsible for the differences between subjects with CAI and healthy controls, the causal mechanism for these feed-forward alterations is less understood. It is possible that feedback deficits incurred in the period after the initial injury lead to alterations in the control of movement. Thus, the failure or inability to control a specific body part (i.e., limb) would force the patient to reorganize movement patterns to compensate. However, it is also possible that if feedback deficits do exist, then the impairments are mutually exclusive. Therefore, the bilateral impairments during unplanned gait termination could be a cumulative effect of altered feed-forward and feedback mechanisms. In this scenario, CAI patients who have altered gait and gait termination motor programs may be overloaded by the sudden “stop” command. When the proposed feedback deficits were added to the already
altered motor control program, the cumulative effect may have been to activate the “stop” motor program simultaneously on both limbs.

**Dynamic postural stability.** The current investigation revealed that patients with CAI had significantly higher APSI and DPSI scores regardless of the gait termination protocol they completed, suggesting that they were less stable. That these impairments were found in both planned and unplanned gait termination supports the contention that patients with CAI have altered feed-forward neuromuscular control and suggests the presence of feedback impairments. Increased APSI and DPSI scores are consistent with previous investigations that have used a jump landing model (36,37). However, the current group means are lower than previously reported, but this should be expected because of inherent tasks differences (36,37). Despite lower group means, the current calculated effect sizes for the APSI ($d = 0.38$) and the DPSI ($d = 0.45$) indicate clinically meaningful differences in neuromuscular control between subjects with CAI and uninjured controls.

**Clinical implications.** It has been well documented that the ankle joint is more susceptible to injury when there is a sudden shift or deceleration of whole-body COM. Because gait termination involves a rapid deceleration of the body’s forward momentum and requires a complex interaction of the neuromuscular system, it is possible to challenge both feed-forward neuromuscular control and the reactive motor response to an external stimuli. As a result, we were able to ascertain that patients with CAI have impairments in feed-forward neuromuscular control. Our results also imply deficits in feedback neuromuscular control, but limitations in our sensorimotor measures impair our ability to draw absolute conclusions.

Despite suffering recurrent episodes of giving way and recurrent injury within the past year, current CAI subjects successfully completed both gait termination protocols, albeit under highly constrained conditions. The translation of the current results to a less constrained environment is not well understood (29), but it is assumed that the magnitude of the identified group differences would increase as the demand on interlimb coordination of force modulation increases (4). In previous gait termination work that studied subjects with parkinsonism (3), increased reliance on a single stopping strategy resulted in an increase in the time and number of steps required to control the COM during an unplanned stopping maneuver. The young adults tested in our current study may not have the same risk of falling during an unplanned stop as the subjects with parkinsonism, but they did rely on a single stopping strategy during a highly constrained task and environment. We therefore speculate that, if not addressed, the reinforcement of a single strategy now may become problematic with increasing task demands and/or as these subjects age. For this reason, we recommended global coordination training for both the injured and the uninjured limbs (1,20,28,34) as part of a compressive prophylactic and/or rehabilitative plan.

### CONCLUSIONS

Patients with CAI have altered biomechanical strategies relative to uninjured controls during both planned and unplanned gait termination as evidenced by kinetic and muscle activity alterations. Furthermore, CAI subjects have dynamic postural control impairments as evidenced by increased APSI and DPSI scores during gait termination. These results indicate that patients with CAI have alterations in feed-forward neuromuscular control.

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