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Lower Extremity Muscle Activation in Patients With or Without Chronic Ankle Instability During Walking

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Context: Ankle sprains are among the most common musculoskeletal injuries, and many individuals with ankle sprains develop chronic ankle instability (CAI). Individuals with CAI exhibit proprioceptive and postural-control deficits, as well as altered osteokinematics, during gait. Neuromuscular activity is theorized to play a pivotal role in CAI, but deficits during walking are unclear.

Objective: To compare motor-recruitment patterns as demonstrated by surface electromyography amplitudes between participants with CAI and healthy control participants during walking.

Design: Descriptive laboratory study.

Setting: Laboratory.

Patients or Other Participants: Fifteen adults with CAI (5 men, 10 women; age = 23 ± 4.2 years, height = 173 ± 10.8 cm, mass = 72.4 ± 14 kg) and 15 matched healthy control adults (5 men, 10 women; age = 22.9 ± 3.4 years, height = 173 ± 9.4 cm, mass = 70.8 ± 18 kg).

Intervention(s): Participants walked shod on a treadmill while surface electromyography signals were recorded from the anterior tibialis, peroneus longus, lateral gastrocnemius, rectus femoris, biceps femoris, and gluteus medius muscles.

Main Outcome Measure(s): Preinitial contact amplitude, postinitial contact amplitude, time of activation relative to initial contact, and percentage of activation time across the stride cycle were calculated for each muscle.

Results: Time of activation for all muscles tested occurred earlier in the CAI group than in the control group. The peroneus longus was activated for a longer duration across the entire stride cycle in the CAI group (36.0% ± 10.3%) than the control group (23.3% ± 22.2%; $P = .05$). No differences were noted between groups for measures of electromyographic amplitude at either preinitial or postinitial contact ($P > .05$).

Conclusions: We identified differences between the CAI and control groups in the timing of muscle activation relative to heel strike in multiple lower extremity muscles and in the percentage of activation time across the entire stride cycle in the peroneus longus muscle. Individuals with CAI demonstrated neuromuscular-activation strategies throughout the lower extremity that were different from those of healthy control participants. Targeted therapeutic interventions for CAI may need to be focused on restoring normal neuromuscular function during gait.

Key Words: ankle sprains, gait, peroneus longus muscle, neuromuscular control

Key Points

- During walking, activation of lower extremity muscles acting on the ankle, knee, and hip occurred earlier for the chronic ankle instability group than for the control group.
- Across the entire stride cycle, the percentage of activation time of the peroneus longus muscle was greater in the chronic ankle instability group than in the control group.
- In patients with chronic ankle instability, preactivation may allow normal ambulation but may not provide adequate dynamic stability to the lateral ankle to prevent injury.

Ankle sprains are among the most common musculoskeletal injuries and are estimated to account for 15% of all sport-related injuries.[1] Researchers[2–4] have estimated that more than 325 000 ankle sprains occur each year in high school athletes alone, and nearly half (49.3%) of all ankle sprains occur during athletic activities. After an initial ankle sprain, up to 40% of individuals develop *chronic ankle instability* (CAI).[5,6] This condition is characterized by mechanical and functional instability of the ankle, as well as residual symptoms of "giving way" or ankle instability for at least 1 year after an initial sprain.[7] *Mechanical instability* refers to excessive inversion laxity of the rearfoot or excessive anterior laxity of the talocrural joint that results in a physiologic or accessory range of motion that is beyond what is expected for the ankle joint.[7] *Functional instability* is characterized by self-reported episodes of giving way and feelings of ankle-joint instability.[7]

Hertel[8,9] first identified the overlapping nature of mechanical and functional instability and later noted that individuals with CAI have a continuum of deficits associated with the use of afferent stimuli, reflexive responses, and efferent motor control. Investigators have identified specific deficits in proprioception,[10,11] peroneal reaction time,[12,13] and postural control[14,15] in individuals with CAI. Alterations in lower extremity kinematics during gait[16–19] and jump landings[20,21] also are evident in individuals with CAI, whereas strength deficits are controversial.[22–26] It is unclear whether these deficits and alterations occur in individuals before or after an initial ankle sprain.

Neuromuscular function related to CAI is of great concern; unfortunately, most reported neuromuscular

Table 1. Participant Demographics, Mean ± SD

Characteristic	Group	
	Chronic Ankle Instability	Healthy
Age, y	23 ± 4.2	22.9 ± 3.4
Height, cm	173 ± 10.8	173 ± 9.4
Mass, kg	72.4 ± 14	70.8 ± 18
Previous sprains, n	4.5 ± 3.2	NA
Time since last sprain, mo	15.2 ± 9.3	NA
Godin Leisure Time Exercise Questionnaire	94 ± 47	84 ± 40
Foot and Ankle Ability Measure Activities of Daily Living score	87.2 ± 7.1	100 ± 0
Foot and Ankle Ability Measure Sport	68.5 ± 5.7	100 ± 0

Abbreviation: NA, not applicable.

deficits are related to peroneal activation in response to experimentally induced inversion perturbation.[12,13,27–30] In these studies,[12,13,27] researchers typically have measured muscle activity after a perturbation via an "unanticipated" trap door. Perturbation studies are an important piece of the CAI literature, but analyzing muscle activation during more functional tasks, such as walking, would be beneficial. Dynamic stabilization of the ankle is attributed to preparatory and responsive muscle activity of the extrinsic foot muscles.[31] Santello[31] reviewed the role of preparatory muscle activation (open loop) as a means of increasing muscle stiffness to control rapid joint rotations that occur after ground contact. Preparatory muscle activity is a protective mechanism that occurs in a predictable pattern during learned tasks and scales proportionally with the expected stimulus on ground contact.[31] Consequently, postcontact muscle activity and dynamic joint stability likely are extensions of preparatory muscle activity combined with reflexive responses.[31] Indirect measures of muscle activation can be determined noninvasively with surface electromyography (sEMG) amplitudes that are used to approximate electrical muscle activity.[32]

The decreased ability to use afferent input, deficits in reflexive responses, and reduced efferent motor control may result in alterations in the normal preparatory and responsive muscle activity before and after ground contact, even in repetitive tasks such as walking. To this point, individuals with CAI have demonstrated a more inverted foot position before and after heel strike,[17,19] as well as an increased rate of change in inversion during the same point in the gait cycle.[17] Therefore, a concentric evertor moment may be present in individuals with CAI, whereas an eccentric invertor moment is present in their healthy counterparts.[17] This has been supported by increased neuromuscular activity of the peroneus longus in individuals with CAI during the first 80 milliseconds after heel strike.[19] However, Santilli et al[33] identified a decrease in peroneal muscle activation in the injured limb compared with the uninjured limb. We believe that examining the neuromuscular control of the entire gait cycle and the activity of muscles acting on the ankle, knee, and hip may be beneficial. A more comprehensive view of preparatory and responsive muscle activity during gait may provide insight into compensatory motor-control patterns in relation to the aforementioned deficits. Therefore, the purpose of our study was to compare the sEMG activity of the anterior tibialis, peroneus longus, lateral gastrocnemius, rectus femoris, biceps femoris, and gluteus medius during shod treadmill walking to determine if individuals with CAI exhibited altered neuromuscular-firing patterns in time of onset and motor-recruitment patterns compared with healthy control individuals. We hypothesized that individuals with CAI would have delayed muscle activation relative to initial contact, decreased sEMG amplitude at preinitial and postinitial contact, and decreased percentage of activation time when compared with healthy control individuals.

METHODS

Design

We performed a descriptive laboratory study. The independent variable was group (CAI, healthy control). The dependent variables were sEMG amplitudes at preinitial and postinitial contact, timing of muscle onset relative to initial contact, and percentage of activation time across the stride cycle in the anterior tibialis, peroneus longus, lateral gastrocnemius, rectus femoris, biceps femoris, and gluteus medius muscles.

Participants

Fifteen participants with CAI (5 men, 10 women; age = 23 ± 4.2 years, height = 173 ± 10.8 cm, mass = 72.4 ± 14 kg) and 15 matched healthy control participants (5 men, 10 women; age = 22.9 ± 3.4 years, height = 173 ± 9.4 cm, mass = 70.8 ± 18 kg) volunteered (Table 1). The control group was matched by sex and test limb and was self-reported to be healthy and have no history of ankle sprain. The inclusion criteria for the CAI group was a history of more than 1 ankle sprain, with the initial sprain occurring more than 1 year before the study, and self-reported functional deficits at the time of the study due to ankle symptoms that were qualified by a score of less than 85% on the Foot and Ankle Ability Measure (FAAM) Sport scale. Given the neuromuscular nature of our outcomes measures, we did not use mechanical instability as an inclusion criterion. All participants reported they were *physically active*, which was defined as being involved in at least 20 minutes of exercise per day for at least 3 days per week; had no history of lower extremity injury, including ankle sprains, within the 6 weeks before the study; and had no history of lower extremity surgery, balance disorders, neuropathies, diabetes, or other conditions known to affect balance (eg, lumbosacral radiculopathy, Marfan syndrome, vestibular conditions, or other musculoskeletal conditions that could affect outcomes). On the first day of testing, participants were screened and completed a general medical history form, the Godin Leisure Time Exercise Questionnaire,[34] the FAAM Activities of Daily Living scale, and the FAAM Sport scale.[35,36] All participants provided informed consent, and the study was approved by the university's Institutional Review Board for Health Sciences Research.

Participants were allocated to groups based on their ankle health status (CAI or healthy). The test limb of participants with CAI was the involved limb. In the case of bilateral CAI, the test limb was the limb that each participant perceived as worse. The test limb of the healthy participants was assigned as a side-matched control to the participants with CAI, and

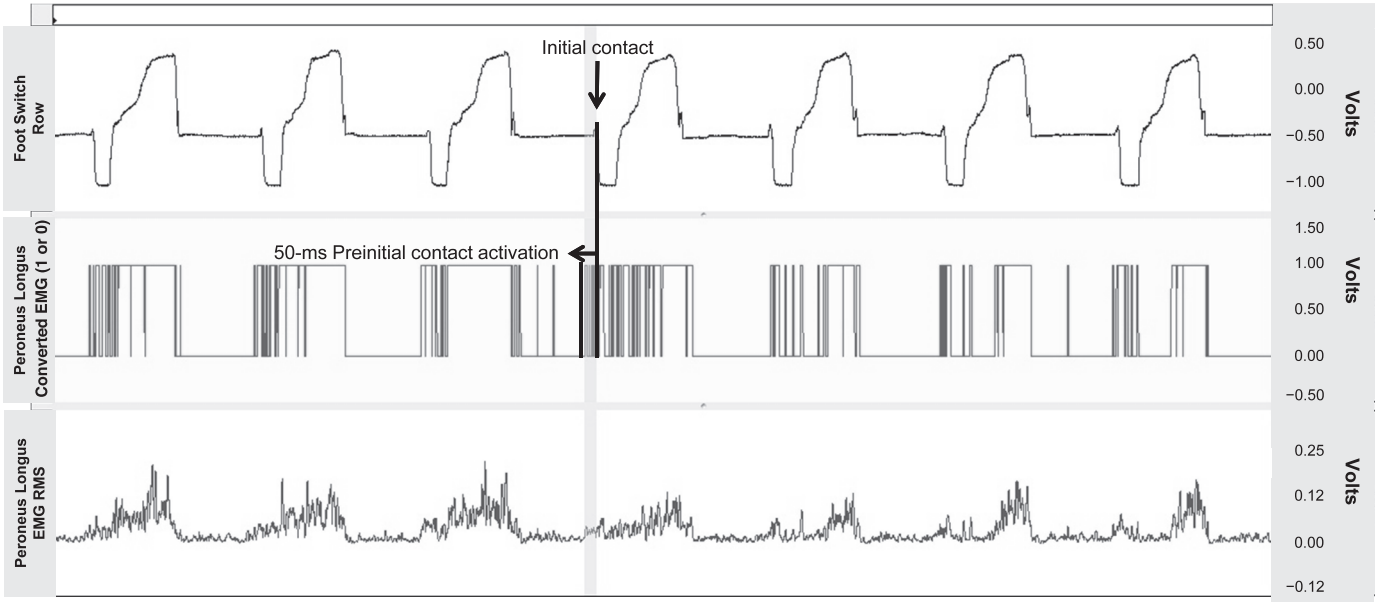

Figure 1. Representative profile for the time of activation relative to the initial contact of the peroneus longus muscle in a participant with chronic ankle instability. Abbreviations: EMG, electromyography; RMS, root mean square.

limb dominance was not controlled during matching. The investigators were not blinded to group membership.

Instruments

Surface EMG signals were collected from disposable, pregelled, 10-mm, round Ag/AgCl electrodes; amplified with a high-gain, differential-input, biopotential amplifier with a gain of 1000; and digitized with a 16-bit data-acquisition system (model MP 150; BIOPAC Systems, Inc, Goleta, CA) at 2000 Hz with a common-mode rejection ratio of 110 dB, an input impedance of 1.0 MΩ, and a noise voltage of 0.2 mV. Acqknowledge software (version 4.0; BIOPAC Systems, Inc) was used for data collection and processing of EMG signals. The EMG data were collected using real-time processing with a 10- to 500-Hz bandpass filter and a 10-sample moving average root mean square (RMS) algorithm. Participants performed walking trials on a treadmill (Gait Trainer 3; Biodex Medical Systems, Shirley, NY). We used a foot switch (BIOPAC Systems, Inc) to identify the onset and termination of stance during walking. All participants wore a standard athletic shoe (model X755WB; New Balance Athletic Shoe, Inc, Boston, MA).

Testing Procedures

Screening was performed on day 1 as described. Participants returned to the laboratory on day 2 of testing (within 7 days of the screening visit). Surface electrodes were placed 2 cm apart and oriented parallel to the muscle-fiber orientation over the midline of the muscle belly, as determined via manual palpation during a voluntary contraction. To minimize skin impedance, the skin was shaved, abraded, and cleansed with isopropyl alcohol. Electrodes were placed over the anterior tibialis, peroneus longus, lateral gastrocnemius, biceps femoris, rectus femoris, and gluteus medius. Before testing, participants performed a 5-minute walking warm-up at a self-selected pace.

Quiet Standing. Surface EMG data were recorded for reference with participants in a quiet standing position. They stood with their feet shoulder-width apart and their hands on their hips. Data were recorded for 15 seconds.

Walking. Participants performed walking trials on a treadmill at a 3.0-mi (4.8-km)-per-hour pace at a 0% incline. They increased speed at a comfortable rate until reaching 3.0 mi (4.8 km) per hour. Data were collected for 30 seconds after they reported a normal gait pattern. Initial contact and termination of the stance phase were identified and synchronized during continuous sEMG data collection.

Data Processing

Quiet Standing. We selected a 500-ms epoch during quiet standing. The mean RMS value and SD were calculated and used to normalize activation measures during gait.[37]

Time of Activation Relative to Initial Contact. We used a total of 9 ground contacts for data processing of time of activation. Three consecutive ground contacts during the first, middle, and last 10 seconds of the 30-second trial were analyzed. *Time of activation* was defined as the point when the magnitude of the RMS value exceeded 10 SDs above the signal recorded during quiet standing. If the muscle was activated at initial contact, time was measured from the initial contact back until the muscle initially activated during the swing phase and was recorded as a negative value, indicating *preactivation* (Figure 1). If the muscle was not activated at initial contact, the time was measured as a positive value until the muscle activated, indicating *postactivation* (Figure 2). The average time of activation for the 9 trials was calculated and recorded.

Percentage of Activation Time. We analyzed 5 consecutive stride cycles during the middle 15 seconds of the trial. Activation was normalized again to any RMS value exceeding 10 SDs of quiet-standing signal for each muscle.

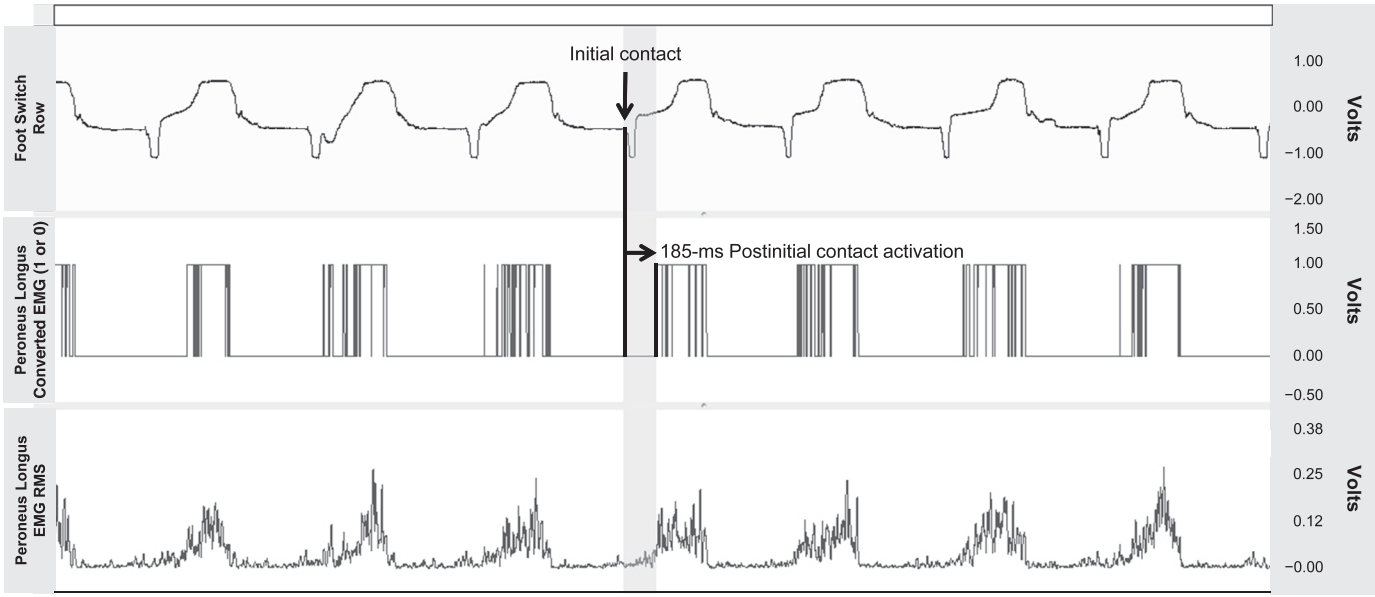

Figure 2. Representative profile for the time of activation relative to the initial contact of the peroneus longus muscle in a healthy participant. Abbreviations: EMG, electromyography; RMS, root mean square.

The RMS values during the 5-stride cycles that exceeded the 10-SD threshold were assigned a value of 1, and any RMS value that did not exceed the threshold was assigned a value of 0. Next, we calculated the mean to determine the percentage of time each muscle exceeded the threshold value during the 5 stride cycles as an indication of percentage of activation time. The mean value represented the total duration of time that a given muscle exceeded the set threshold divided by the total duration of the stride cycle.

Preinitial and Postinitial Contact Amplitude. We used a 100-millisecond epoch immediately before heel strike (preinitial contact) and a 200-millisecond epoch immediately after heel strike (postinitial contact) to calculate the area under the RMS curve.

Statistical Analysis

We performed an a priori sample-size estimate based on published data[38] showing a minimal change of 6% in lateral gastrocnemius activation during normal walking. In addition, the largest variability was a 5% SD in peroneus longus activation in the healthy participants.[38] We estimated that 15 participants per group would be sufficient to find differences at an α level (type I error) of .05 and power ($1-\beta$) of 0.8. We performed an independent t test for each dependent variable to compare groups. The level at which findings were considered different was set a priori at $P \leq .05$ for all analyses. Consistent with contemporary biostatistical recommendations, we did not correct for multiple comparisons.[39] We also calculated the Cohen d effect size and associated 95% confidence intervals. Data were analyzed using IBM SPSS Statistics for Windows (version 20.0; IBM Corp, Armonk, NY).

RESULTS

Time of Activation Relative to Initial Contact

We found a significant difference in time of activation between groups for the peroneus longus ($P < .001$) and the rectus femoris ($P = .03$) during walking. Time of activation was earlier in the CAI group than in the control group for all muscles tested (Table 2).

Percentage of Activation Time

The peroneus longus was activated for a longer duration throughout the stride cycle in the CAI group (36.0% ± 10.3%) than in the control group (23.3% ± 22.2%; $P = .05$). No other differences were identified between groups for percentage of activation time during walking (Table 3).

Preinitial and Postinitial Contact Walking Amplitude

No differences were noted between groups for amplitude measures at preinitial or postinitial contact (Table 4).

Table 2. Independent *t* Test Results, Effect Sizes, and 95% Confidence Intervals for Time of Activation Relative to Initial Contact[a]

Muscle	Group, s (Mean ± SD)		*P* Value	Effect Size (95% Confidence Interval)
	Chronic Ankle Instability	Healthy		
Anterior tibialis	−0.36 ± 0.16	−0.29 ± 0.18	.24	−0.41 (−1.13, 0.31)
Peroneus longus	−0.07 ± 0.10	0.16 ± 0.17	<.001	−1.36 (−2.15, −0.56)
Lateral gastrocnemius	0.18 ± 0.14	0.25 ± 0.13	.17	−0.51 (−1.24, 0.22)
Rectus femoris	−0.14 ± 0.15	−0.04 ± 0.11	.03	−0.97 (−1.73, −0.21)
Biceps femoris	−0.23 ± 0.10	−0.18 ± 0.08	.18	−0.56 (−1.29, 0.17)
Gluteus medius	−0.08 ± 0.10	−0.06 ± 0.12	.63	−0.16 (−0.88, 0.55)

[a] Negative values indicate preactivation relative to initial contact, and positive values indicate postactivation relative to initial contact.

Table 3. Independent *t* Test Results, Effect Sizes, and 95% Confidence Intervals for Percentage of Activation Time per Stride Cycle

Muscle	Group, % of Stride Cycle (Mean ± SD)		*P* Value	Effect Size (95% Confidence Interval)
	Chronic Ankle Instability	Healthy		
Anterior tibialis	53.1 ± 26.7	52.7 ± 25.0	.96	0.02 (−0.70, 0.73)
Peroneus longus	36.0 ± 10.3	23.3 ± 22.2	.05	0.57 (−0.16, 1.30)
Lateral gastrocnemius	21.1 ± 12.2	18.6 ± 11.6	.58	0.22 (−0.50, 0.93)
Rectus femoris	36.9 ± 24.5	26.6 ± 22.0	.24	0.47 (−0.26, 1.19)
Biceps femoris	29.4 ± 19.6	27.8 ± 17.6	.81	0.09 (−0.63, 0.81)
Gluteus medius	27.2 ± 25.2	22.8 ± 22.4	.62	0.20 (−0.52, 0.91)

DISCUSSION

We identified differences between the CAI and control groups in the timing of muscle activation relative to initial contact in multiple lower extremity muscles and in the percentage of activation time across the entire stride cycle in the peroneus longus muscle. However, we did not identify differences in sEMG amplitude between groups at preinitial or postinitial contact. Our results identified an earlier, but not always significantly different, onset of activity for muscles acting on the ankle, knee, and hip, including the anterior tibialis, peroneus longus, rectus femoris, biceps femoris, and gluteus medius in the CAI group compared with the healthy control group during walking.

Delahunt et al[19] suggested that individuals with ankle instability develop an altered "feed-forward" motor-control pattern in preparation for initial contact. The feed-forward mechanism has been believed to be an important mechanism in injury prevention, specifically in individuals with CAI, to explain how peroneus longus preactivation can help protect against an inversion moment.[19] We posit, however, that the shift to a feed-forward pattern of motor control for the peroneus longus during gait may also have negative consequences. Our findings indicated that healthy individuals did not activate the peroneus longus until approximately midstance in the gait cycle, which is supported in gait-analysis studies.[40] In addition, the biomechanical function of the peroneus longus should be considered for its contribution to lateral dynamic stability of the ankle in weight bearing because of its role in propulsion during gait but not for its ability to actively evert the ankle. As the peroneus longus exerts a plantar-flexion force at the ankle and pulls down the first ray, assisting in pronation and subsequently stabilizing the first ray as a rigid lever for propulsion, the increased muscle tone provides tremendous dynamic stability to the lateral ankle.[41] Therefore, the activation time associated with the peroneus longus in a healthy population during midstance is correlated with the biomechanical function of the foot during normal weight bearing and indicates that peroneus longus preactivation during gait may not be the ideal protective mechanism for individuals with CAI. During a single-legged drop landing, Delahunt et al[20] demonstrated an equivalent but opposite abnormal peroneal preactivation pattern in participants with functional ankle instability. Participants had decreased levels of peroneus longus preactivation during a single-legged drop landing compared with healthy controls. This task, albeit functionally different from walking, may represent the eccentric equivalent to the peroneus longus function described. Delahunt et al[19] found results during gait that were similar to ours, noting a trend toward increased peroneus longus amplitude in individuals with functional instability in the 200-millisecond epoch preinitial contact; however, perhaps the activation time of 70 milliseconds in our study needs to be a shorter epoch to capture differences in amplitude.

Researchers[12,13,28–30,42] have extensively studied the protective mechanism of the peroneus longus regarding its ability to respond to inversion perturbations. Strong emphasis has been placed on the peroneus longus for its reflexive ability due to the reflex contraction initiated by intrafusal muscle fibers against inversion moments. Normal function of the muscle spindles allows for a protective response to sudden changes in muscle length with a reflex-corrective contraction.[43] During concentric muscle contractions, intrafusal muscle fibers may shorten and, thus, are unable to signal further changes in muscle length.[35] Healthy individuals demonstrate a pattern known as α-γ *coactivation*, in which the simultaneous intrafusal muscle contraction via the γ motor-neuron system keeps the central region of the muscle spindle loaded and capable of sensing small changes in muscle length even during concentric α motor-

Table 4. Independent *t* Test Results, Effect Sizes, and 95% Confidence Intervals for Preinitial and Postinitial Contact Walking Amplitudes

Muscle	Group, Normalized Surface Electromyography Amplitude (Mean ± SD)				Preinitial Contact[a]		Postinitial Contact[b]	
	Chronic Ankle Instability		Healthy					
	Preinitial Contact	Postinitial Contact	Preinitial Contact	Postinitial Contact	*P* Value	Effect Size (95% Confidence Interval)	*P* Value	Effect Size (95% Confidence Interval)
Anterior tibialis	2.66 ± 2.33	3.59 ± 3.27	2.94 ± 2.3	4.47 ± 4.12	.74	–0.12 (−0.84, 0.59)	.51	–0.21 (−0.93, 0.50)
Peroneus longus	0.46 ± 0.23	1.22 ± 0.64	0.32 ± 0.34	1.12 ± 1.6	.18	0.41 (−0.31, 1.13)	.82	0.06 (−0.65, 0.78)
Lateral gastrocnemius	0.21 ± 0.11	0.55 ± 0.34	0.19 ± 0.17	0.57 ± 0.49	.69	0.12 (−0.60, 0.83)	.94	–0.04 (−0.76, 0.67)
Rectus femoris	0.75 ± 0.44	0.75 ± 0.44	0.48 ± 0.46	1.32 ± 1.25	.11	0.59 (−0.14, 1.32)	.11	0.67 (−0.06, 1.41)
Biceps femoris	1.42 ± 1.09	1.21 ± 0.96	1.95 ± 1.87	1.24 ± 1.02	.34	–0.31 (−1.03, 0.41)	.93	–0.03 (−0.75, 0.69)
Gluteus medius	0.53 ± 0.49	1.93 ± 1.9	0.40 ± 0.37	1.59 ± 1.76	.35	0.35 (−0.38, 1.09)	.35	0.19 (−0.54, 0.92)

[a] 100-ms epoch preinitial contact.
[b] 200-ms epoch postinitial contact.

neuron contractions.[43] This α-γ coactivation is an autonomic process that maintains the muscle-spindle sensitivity needed for dynamic stability. Regarding neuromuscular dysfunction in individuals with CAI, researchers[44–46] have explored dynamic stability by measuring α motor-neuron–pool excitability.

The Hoffman reflex has been used extensively to assess α motor-neuron–pool excitability in healthy individuals and individuals with CAI.[44–46] Investigators[46] have demonstrated that individuals with CAI have a decreased Hoffman reflex while lying prone and standing in bipedal and unipedal stances compared with healthy counterparts. Furthermore, motor-neuron–pool excitability is decreased to a greater extent as the difficulty of the postural-control task increases.[47] Riemann and Lephart[48] described reflexive neural activation as crucial for developing extrinsic joint stiffness. They also characterized reflexive neural activation as relying on the excitability of the motor-neuron pool, which subsequently is substantially dependent on muscle-spindle sensitivity. Although it may be beyond the scope of our study, the centrally modulated progressive decrease in motor-neuron–pool excitability conceivably could be related to a concurrent decrease in the sensitivity of the muscle spindles to perturbations during active concentric muscle contractions. If this is the case, the peroneus longus preactivation demonstrated in our study would be detrimental to the dynamic stability of the lateral ankle in individuals with CAI, as the slackened muscle spindles before heel strike would be incapable of detecting and initiating a response to slight changes in muscle length at ground contact, when ankle sprains occur.

We also found that the rectus femoris was activated earlier relative to heel contact in the CAI group. In a healthy population, the rectus femoris is activated for weight acceptance directly at initial contact and in the early stance phase.[40] In our study, the CAI group activated the rectus femoris an average of 108 milliseconds earlier than the control group. This earlier preactivation of the rectus femoris may be due to the decreased pronation in participants with CAI.[49] Primary purposes of pronation are to absorb shock during gait and provide a greater base of support during midstance. In individuals with CAI, the supinated gait patterns of the foot and ankle cause additional shock absorption and weight acceptance to occur at more proximal joints. We believe additional loads may be dissipated through the eccentric action of the rectus femoris, leading to a "bracing-for-impact" coping strategy and excessive preactivation in this muscle.

Our results indicated that not only did the peroneus longus and rectus femoris fire earlier in individuals with CAI, but the percentage of the gait cycle during which the peroneus longus was activated was approximately 13% greater in the CAI than in the healthy control group. The ability to selectively inhibit and maintain normal asynchronous firing of motor units is an essential aspect of muscle function.[50] Asynchronous firing of motor units and selective inhibition are important factors in preventing fatigue and maintaining maximal motor-neuron availability for injury prevention. Whereas we did not analyze the extent of activation for the entire time the muscle was activated, we believe that activating the peroneus longus for a longer time may increase fatigability and that preactivation before initial contact may decrease the available motor units capable of protecting against inversion moments. Collectively, the coping strategy that the participants with CAI demonstrated may be physiologically inefficient compared with that of their healthy counterparts.

Researchers have debated the latency associated with peroneal reaction time, specifically during inversion perturbations. Santilli et al[33] compared the latency of the peroneus longus during the stance phase of gait in the injured and the uninjured limbs as a short-term consequence of lateral ankle sprains in a population with functional ankle instability. They noted an immediate neuromuscular deficit between limbs within participants. Our findings demonstrate a possible progression from a functional deficit after an ankle sprain to a chronic compensatory pattern in individuals with CAI. The neuromuscular compensatory pattern is supported by Hopkins et al,[51] who observed that individuals with functional instability displayed a laterally deviated center-of-pressure (COP) trajectory during walking compared with matched control individuals. They speculated that the lateral COP trajectory in participants with functional instability was due to alterations in anterior tibialis and peroneus longus neuromuscular function.[51] Schmidt et al[49] found that individuals with CAI also exhibited slower loading responses of the lateral and medial rearfoot and the medial midfoot during jogging. When comparing individuals with CAI and individuals who sustained isolated ankle sprains but had no subsequent feelings of instability during running, Morrison et al[16] showed that individuals with CAI presented with a more lateral COP of the rearfoot at foot strike and a more lateral COP trajectory during loading. Both the lateral COP and slower loading responses could suggest that a neuromuscular response occurs in individuals with CAI to maintain a functional "close-packed" supinated position of the subtalar and midfoot joints. In addition, the preactivation of the peroneus longus that we noted could help explain the lateral COP exhibited in individuals with CAI. Recruiting the peroneus longus as an open chain evertor in the presence of excessive inversion[17,19,20] preinitial contact may improve foot position at initial contact. However, altering normal muscle function to compensate for abnormal kinematics may substantially decrease the ability of the peroneus longus to aid in pronation and stabilize the first ray while weight bearing, contributing to the lateral COP during both the loading and propulsion phases of gait. The earlier onset and preactivation of not only the peroneus longus but also the anterior tibialis, lateral gastrocnemius, rectus femoris, biceps femoris, and gluteus medius is a compensatory (coping) gait pattern that may allow individuals to increase limb stability or functionally "brace" for ground contact.

A limitation of our study was that we were unable to collect concurrent biomechanical data on ground reaction forces or kinematic data on limb velocity during the gait cycle. Investigators should examine associated muscle activity during concurrent biomechanical and kinematic analysis to determine if preactivation has a specific role in increasing or decreasing impact forces and altering joint position or limb velocity throughout the gait cycle. Similarly, examining muscle-offset times, in addition to onset times, would provide a more comprehensive characterization of muscle activation throughout the gait

cycle and could lead to improved understanding of the sensorimotor adaptations associated with CAI.

CONCLUSIONS

We identified an earlier onset of activity for lower extremity muscles acting on the ankle, knee, and hip during walking and a greater percentage of activation time of the peroneus longus muscle across the entire stride cycle in the CAI than in the healthy control group. Preactivation may be a coping strategy that is effective for normal ambulation and allows individuals with CAI to complete functional tasks, but it may be ineffective in providing adequate dynamic stability to the lateral ankle to prevent injury.

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