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# Short and Medium Latency Responses in Participants With Chronic Ankle Instability

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***Context:*** The determinant role of medium latency responses (MLRs) during compensatory postural adjustments in postural stability and the lack of clarity about the mechanisms behind chronic ankle instability (CAI) sustain the hypothesis that these postural responses are impaired in this condition. However, to the best of our knowledge, no authors have assessed MLRs in patients with CAI; most of the research regarding compensatory postural adjustments has been directed at the timing of short latency responses (SLRs).

***Objective:*** To evaluate bilateral compensatory postural responses, including SLRs and MLRs, in response to a unilateral simulated ankle-sprain mechanism in participants with CAI.

***Design:*** Case-control study.

***Setting:*** Laboratory.

***Patients or Other Participants:*** Twenty-four participants with CAI and 20 controls.

***Intervention(s):*** Bilateral electromyography of the peroneus longus (PL), peroneus brevis, tibialis anterior (TA), and soleus (SOL) muscles was collected during a unilateral sudden-inversion perturbation (30°).

***Main Outcome Measure(s):*** Muscle-onset activations and magnitudes of SLRs and MLRs in the support and perturbed positions.

***Results:*** Participants with CAI showed (1) later-onset activation of the TA and SOL in the uninjured limb and bilateral decreases in the magnitude of the TA MLR in the support position, (2) increased magnitude of the PL MLR in the uninjured limb in the support and perturbed positions, and (3) increased magnitude of the PL SLR and MLR in the injured limb in the perturbed position.

***Conclusions:*** Chronic ankle instability was associated with later TA and SOL activation in the uninjured limb and bilaterally decreased magnitude of the TA MLR in the support position.

***Key Words:*** postural control, muscle-onset timing

**Key Points**

- Participants with chronic ankle instability displayed bilateral impairment in postural adjustments while in a support position.
- Both short and medium latency responses were impaired in patients with chronic ankle instability.
- After unilateral ankle sprain, both limbs should addressed in rehabilitation.

The maintenance of upright quiet stance is a challenging task for the central nervous system, which has the goal of controlling an intrinsically unstable biomechanical system under the effect of gravity. Sensory systems, such as the vestibular, visual, and somatosensory systems, play significant roles in the aforementioned motor task. In fact, a disorder in any of these systems, such as chronic ankle instability (CAI), may lead to postural instability.[1] In addition to affecting 40%–80% of patients who sustained the most common injury related to physical activity—acute lateral ankle injury[2]—CAI has been associated with changes in the ankle muscles that provide critical proprioceptive input for maintaining a stable upright stance.[3]

Chronic ankle instability may be influenced by several factors, including mechanical and functional deficits.[4] Mechanical ankle instability can be attributed to pathologic laxity, arthrokinematic changes, and synovial and degenerative changes.[5] Changes in talar mobility lead to abnormal movement of the instantaneous axis of joint rotation,[6] resulting in altered proprioceptive input and altered motor-control programs.[7] *Functional ankle instability* describes recurrent ankle instability and the sensation of joint instability due to sensorimotor deficits.[5] Several sensorimotor deficits have been proposed to contribute to functional ankle instability, including impaired proprioception,[8] strength,[9] magnitude, and timing of muscle activation during short-latency compensatory postural responses.[10]

Evoked compensatory postural responses are produced in response to instability and are composed of a primary burst with an onset latency of approximately 40–50 milliseconds (attributed to a monosynaptic excitation of spinal motoneurons from the group Ia afferent fibers[11] and from joint mechanoreceptors[12]) and a secondary burst with an onset latency of approximately 70 milliseconds (medium latency response [MLR]) mediated by group II afferents through an oligosynaptic spinal pathway[11] and possibly via group Ib afferents.[13] Freeman[14] argued that patients experience partial deafferentation after ankle sprain, reducing the reflexive activity that would normally be initiated by the joint mechanoreceptors. A lack of proprioceptive information resulting

**Table 1. Characteristics of the Control and Chronic Ankle Instability Groups**

| Variable | Mean ± SD | | |
|---|---|---|---|
| | Control (n = 20) | Chronic Ankle Instability (n = 24) | *P* Value |
| Age, y | 21.8 ± 2.21 | 20.6 ± 2.52 | .098 |
| Height, m | 1.78 ± 0.09 | 1.76 ± 0.09 | .800 |
| Body weight, kg | 73.8 ± 11.5 | 70.0 ± 11.3 | .650 |
| No. of previous ankle sprains | NA | 3.1 ± 1.53 | NA |
| Time since last sprain, mo | NA | 9.0 ± 2.90 | NA |
| | | No. | |
| Classification of chronic ankle instability | | | |
| Functional | | 14 | |
| Mechanical | | 10 | |
| Frequency of giving way | | | |
| Rarely | | 8 | |
| Frequently | | 10 | |
| Often | | 6 | |
| Severity of ankle sprain | | | |
| Severe | | 1 | |
| Moderate | | 22 | |
| Mild | | 1 | |

Abbreviation: NA, not applicable.

from partial deafferentation and altered sensorimotor strategies could chronically suppress γ activation and desensitize the muscle spindle.[15] This could explain both the impairment in short latency responses (SLRs) previously demonstrated in patients with CAI[10] and the possible impairment in MLRs. Growing support for the crucial role of MLRs in the control of perturbations has emerged.[16] Indeed, evidence suggests that muscle-spindle type II fibers play a more relevant role than group Ia fibers in the control of bipedal stance[17] because only MLRs have a stabilizing effect during perturbations of stance and these fibers are more influenced by the "postural set."[16] The hypothesis that MLRs are impaired in participants with CAI can also explain bilateral involvement after unilateral ankle sprain as MLRs occur bilaterally, even when the muscles are stretched unilaterally.[18]

Despite the known importance of MLRs in postural stability, to the best of our knowledge, no study has evaluated MLRs in participants with CAI. Therefore, the purpose of this investigation was to evaluate bilateral compensatory postural responses, including SLRs and MLRs, to a simulated unilateral ankle-sprain mechanism in participants with CAI. Although it has been suggested that MLRs may substitute for SLRs in participants with CAI, reducing reflexive activity from group Ia interneurons,[12] we hypothesized that those with CAI would also present with bilateral impairment of MLRs.

## METHODS

### Participants

Twenty-four athletes (6 women, 18 men) with unilateral CAI and 20 uninjured athletes (3 women, 17 men) participated in this study (Table 1). Participants assigned to the CAI group met the criteria set by the International Ankle Consortium.[19] For inclusion in the CAI group, participants had to meet the following criteria: (1) history of at least 1 significant unilateral ankle sprain, (2) the initial sprain must have occurred at least 12 months before enrollment in the study, (3) at least 1 ankle sprain was associated with inflammatory symptoms, (4) at least 1 ankle sprain resulted in at least 1 interrupted day of desired physical activity, (5) the most recent injury must have occurred more than 3 months before enrollment in the study, and (6) history of the previously injured ankle joint "giving way" or recurrent sprain or experiencing "feelings of instability." To meet this last criterion, individuals must have answered *yes* to question 1 ("Have you ever sprained an ankle?") along with *yes* to at least 4 questions related to perceived ankle instability and giving-way episodes: (2) "Does your ankle ever feel unstable while walking on a flat surface?" (3) "Does your ankle ever feel unstable while walking on uneven ground?" (4) "Does your ankle ever feel unstable during recreational or sport activity?" (5) "Does your ankle ever feel unstable while going up stairs?" (6) "Does your ankle ever feel unstable while going down stairs?" The CAI group consisted of participants with mechanical ankle instability, functional ankle instability, or both. Participants were considered to have *mechanical ankle instability* if they had 1 or both of the following conditions: (1) pain or talocrural-joint mobility greater than 3 mm on the anterior drawer manual stress test (assessed using a triaxial accelerometer [Biosignals Research, Lisbon, Portugal]), compared with the uninjured side, and (2) talar tilt greater than 7° together with a difference of more than 0° in relation to the contralateral (uninjured) ankle (assessed using an electrogoniometer [Biometrics Ltd, Newport, United Kingdom]). The exclusion criteria for the CAI group set by the International Ankle Consortium[19] were (1) a history of previous surgery to the musculoskeletal structures in either lower extremity, (2) a history of fracture in either lower extremity requiring realignment, (3) acute injury to the musculoskeletal structures of other joints of the lower extremity in the previous 3 months that affected joint integrity and function and resulted in at least 1 day of interrupted desired physical activity, or (4) a history of bilateral ankle sprain.

The exclusion criteria were similarly applied to healthy control participants, who were also excluded if they had a history of ankle sprain. All volunteers were athletes involved in sports with a high risk of ankle sprain, including basketball, handball soccer, and volleyball. Before testing, participants were asked to identify the *dominant limb*, which was described as the leg they would use to kick a ball. No differences were observed between the dominant and nondominant limbs of healthy individuals in a previous study[10] that used a similar protocol, so we selected the nondominant limb in the healthy control group for evaluation. In the CAI group, both limbs were evaluated.

The study was approved by the local ethics committee and was implemented according to the Declaration of Helsinki. All recruits gave their written informed consent to participate.

### Instruments

The activity of the agonist muscles in active ankle stability—tibialis anterior (TA), soleus (SOL), peroneus longus (PL), and peroneus brevis (PB)—was assessed by electromyography. The electromyographic signal of these muscles was monitored using a wireless signal-acquisition system (Biosignals Research). The signals were collected at a sampling frequency of 1000 Hz and were preamplified in each electrode and then fed into a differential amplifier with an adjustable gain setting (25–500 Hz; common-mode rejection ratio = 110 dB at 50 Hz, input impedance = 100 MΩ, and gain = 1000). Self-adhesive silver chloride electromyographic electrodes were used in a bipolar configuration with an interelectrode distance of 20 mm. Skin impedance was measured with an electrode impedance checker. The electromyography and force-platform signals were analyzed using Acqknowledge software (version 3.9; Biopac Systems, Inc, Goleta, CA).

The Ankle Instability Instrument was designed to classify patients with functional ankle instability and has been shown to be a reliable and valid tool.[20] The instrument also has high test-retest reliability (intraclass correlation coefficient [ICC] = 0.95). Internal consistency reliability estimates (α coefficients) for each factor and the total measure ranged from 0.74 to 0.83.

A tilt platform was used to produce 30° of subtalar joint inversion. The platform consisted of 2 movable plates (trapdoors) so that either foot could be tilted independently, thus eliminating the possibility of anticipatory effects. A triaxial accelerometer (model ACC 18012018; Biosignals Research) connected to the signal-acquisition system was placed in each movable plate to detect the onset of the tilt mechanism (first deflection of the accelerometer signal). For safety reasons, the tilt platform was surrounded by a handrail at the front and both sides of the participant. Also, an adhesive, nonslip material was placed to create a footpath and to prevent slipping when the trapdoor was opened.

### Procedures

The skin surface of the selected muscles midbelly and of the patella was prepared (shaved and dead skin cells and nonconductor elements were removed with alcohol and an abrasive pad) to reduce the electrical resistance to less than 5000 Ω. This electrode placement was based on recommendations in the literature.[21] For the TA, the electrode was placed at one-third of the line between the tip of the fibular head and the tip of the medial malleolus. For the SOL, the electrode was placed 2 cm distal to the lower border of the medial gastrocnemius muscle belly and 2 cm medial to the posterior midline of the leg. For the PB, the electrode was placed at one-fourth of the line between the tip of the fibular head and the tip of the lateral malleolus. For the PL, the electrode was placed anterior to the PL tendon at one-fourth of the line from the tip of the lateral malleolus to the fibular head.

All individuals were asked to stand quietly with the support base aligned at shoulder width with 1 foot on each trapdoor, keeping their arms by their sides, and to focus for 30 seconds on a target 2 m away and at eye level. In addition, they were instructed to ensure that their weight was equally distributed between the limbs. One limb at a time was randomly exposed to the simulated unilateral ankle sprain and was identified during the analysis of each trial as the perturbed limb. The unexposed limb was the support limb. Each limb was exposed to the simulated ankle sprain 3 times in random order. In each trial, we randomly released a trapdoor by pushing a foot switch that was not visible to the participant. The participant did not know the side or the timing of the perturbation in advance. In the CAI group, the electromyographic signal was collected from both limbs (injured and uninjured), and both were evaluated as support and perturbed limbs. In the control group, only the nondominant limb was monitored as the support and perturbed limb. Upon release, the platform fell down through an arc of 30°, which was predetermined by a mechanical stop leading to ankle subtalar inversion. During 60-second rest periods between trials, the participants sat down while maintaining their foot position.

The electromyographic signals were filtered using a zero-lag, second-order Butterworth filter with an effective bandpass of 20 to 450 Hz, and the root mean square was calculated. Muscle latency was detected in a time window from −200 to +200 milliseconds in relation to the first deflection of the accelerometer signal ($T_0$). The *latency* of the PL, PB, TA, and SOL muscles of each limb (support and perturbed positions) was defined as the 50 milliseconds (or more) when its electromyographic amplitude was higher than the mean of its baseline value plus 3 standard deviations (SDs), measured from −500 to −450 milliseconds, using a combination of computational algorithms and visual inspection. The magnitude of overall compensatory response was evaluated over a 50-millisecond window starting at the latency of each muscle. To examine the SLRs and MLRs, we defined two 20-millisecond windows. The first window started at the onset of the SLRs (muscle latency), and the second window started 30 milliseconds later. The 10-millisecond division between the windows ensured a clear separation. The magnitude of the electromyographic signal in each interval was normalized to the baseline value to assess the degree of magnitude modulation of each muscle during the compensatory responses in relation to upright standing posture. We normalized the electromyographic signal to the signal obtained in upright standing posture, which represented a submaximal voluntary contraction normalization method.[22] The average of the values obtained in the 3 perturbation trials was used for analysis.

### Data Analysis

We analyzed the data using SPSS (version 20; IBM Corp, Armonk, NY). Means, SDs, and 95% confidence intervals were calculated for descriptive analysis. The minimal detectable difference (MDD) was determined using the equation $1.96 \times \sqrt{2} \times \text{SEM}$. The SEM was calculated using the equation $(\text{SEM} = \text{SD}\sqrt{(1 - \text{ICC})}$, where the ICC reflected the 3 trials performed in each position.

The independent-samples $t$ test was used to compare muscle latencies and the magnitudes of the SLRs and MLRs between the control and CAI groups. We conducted the paired-samples $t$ test to compare muscle latencies and the magnitude of SLRs and MLRs between the injured and uninjured limbs of the CAI group. The Shapiro-Wilk test and the histogram analysis method indicated that the data

**Table 2. Muscle-Onset Activation in the Control and Chronic Ankle Instability (CAI) Groups in the Support and Perturbed Positions[a]**

| Group | Position | Muscle | Uninjured Limb, ms | | | Between-Subjects Comparisons | | Injured Limb, ms | | | Between-Subjects Comparisons | | Within-Subject Comparisons | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Mean ± SD | 95% CI | MDD | *t* Value | *P* Value | Mean ± SD | 95% CI | MDD | *t* Value | *P* Value | *t* Value | *P* Value |
| Control | Support | Tibialis anterior | 51.2 ± 23.49 | 49.5, 69.6 | 32.55 | −2.697 | **.012** | NA | | | −0.842 | .406 | NA | |
| CAI | | | 68.3 ± 12.16 | 59.7, 75.5 | 17.40 | | | 58.1 ± 24.38 | 48.9, 76.7 | 23.00 | | | 0.783 | .450 |
| Control | Perturbed | | 46.1 ± 21.81 | 40.8, 63.9 | 32.43 | −0.717 | .479 | NA | | | −0.580 | .566 | NA | |
| CAI | | | 51.2 ± 15.74 | 40.6, 61.5 | 18.19 | | | 50.1 ± 17.97 | 45.0, 65.6 | 29.75 | | | −0.519 | .613 |
| Control | Support | Soleus | 62.1 ± 25.49 | 63.5, 80.8 | 35.66 | −2.143 | **.04** | NA | | | 0.343 | .734 | NA | |
| CAI | | | 80.6 ± 21.60 | 65.0, 91.5 | 37.03 | | | 59.2 ± 21.62 | 48.4, 75.4 | 15.96 | | | 3.694 | **.004** |
| Control | Perturbed | | 59.0 ± 32.02 | 55.7, 83.9 | 56.06 | −0.718 | .478 | NA | | | −0.494 | .624 | NA | |
| CAI | | | 67.0 ± 29.84 | 47.0, 86.6 | 37.16 | | | 63.3 ± 14.86 | 56.3, 75.3 | 19.17 | | | 0.051 | .960 |
| Control | Support | Peroneus longus | 67.5 ± 19.12 | 56.9, 78.1 | 27.52 | −0.919 | .367 | NA | | | −0.164 | .871 | NA | |
| CAI | | | 77.3 ± 35.97 | 53.9, 101.6 | 51.77 | | | 60.0 ± 20.72 | 48.5, 75.5 | 30.10 | | | 1.026 | .327 |
| Control | Perturbed | | 51.1 ± 24.34 | 47.8, 71.7 | 35.36 | −0.571 | .572 | NA | | | −0.411 | .684 | NA | |
| CAI | | | 55.7 ± 20.91 | 41.9, 69.7 | 28.96 | | | 54.2 ± 21.00 | 44.0, 72.0 | 50.04 | | | −0.284 | .781 |
| Control | Support | Peroneus brevis | 65.5 ± 31.17 | 63.7, 89.6 | 63.56 | −1.547 | .133 | NA | | | 0.332 | .742 | NA | |
| CAI | | | 78.3 ± 14.68 | 68.7, 88.2 | 35.84 | | | 62.4 ± 21.39 | 48.9, 77.1 | 20.03 | | | 2.513 | **.029** |
| Control | Perturbed | | 61.7 ± 36.05 | 48.9, 9.1 | 93.30 | 0.399 | .693 | NA | | | 0.637 | .528 | NA | |
| CAI | | | 57.1 ± 25.45 | 40.0, 73.7 | 64.20 | | | 55.4 ± 17.98 | 45.7, 70.2 | 33.19 | | | −0.064 | .950 |

Abbreviations: CI, confidence interval; MDD, minimal detectable difference; NA, not applicable.

[a] Bold values represent *P* values < .05.

were normally distributed. A .05 significance level was used for inferential analysis.

## RESULTS

Globally, a tendency to later onset of ankle-muscle activation was observed in the CAI group compared with the control group (Tables 2 through 4). Activation of the TA and SOL muscles was faster in the control group than in the uninjured limb of the CAI group in the support position (Table 2). When the injured and uninjured limbs of the CAI group were compared, a tendency to later-onset activation was observed in the uninjured limb. Later-onset activation of the SOL and PB muscles was noted in the support position.

Differences between groups were also observed in the magnitude of the SLRs and MLRs (Tables 3 and 4). Specifically, the magnitudes of the uninjured SOL SLR in the support position and the injured PL SLR in the perturbed position were increased in the CAI group (Table 3). The injured limb also displayed decreased values of PL SLR in the support position when compared with the uninjured limb of the same group (Table 3). As to the magnitude of the MLRs, the CAI group presented bilateral decreased values of TA MLR and bilateral increased values of PL MLR in the support and perturbed positions, respectively (Table 4). An increased PL MLR was observed only in the uninjured limb of the CAI group in the support position (Table 4). In this position, decreased values of PL MLR were noted in the injured limb versus the uninjured limb of the CAI group (Table 4).

## DISCUSSION

Despite extensive investigation, the mechanism behind CAI remains unclear. We found no differences in the peroneus muscle latencies of participants with CAI versus the healthy control group, which indicates that this component of compensatory postural responses is not a determining factor in CAI. Our findings are supported by a meta-analysis from Munn et al,[23] who pooled studies with a broad definitional criterion for ankle instability and showed that the peroneal reaction time was not impaired in CAI. We observed no differences in the peroneus muscle latencies of the CAI group compared with the control group in either the perturbed or support position. Although few authors[10,24,25] who have studied participants with CAI have evaluated postural adjustments in the support position with perturbations applied to the contralateral limb, none demonstrated differences in peroneal latency.

The major differences in muscle latencies between groups were observed in the TA and SOL muscles of the uninjured limb in the support position. These differences were accompanied by decreased magnitudes of TA MLR in both the uninjured and injured limbs in the support position and increased SOL SLR in the uninjured limb in both the perturbed and support positions. Considering the role of spindle group II fibers in postural-control adjustments,[17] changes in the magnitude modulation of the TA MLR observed in the CAI group could be a key factor in CAI. In closed kinetic chain activities, the TA and SOL muscles have important roles in regulating the projection of the center of pressure on the base of support.[26] The later-onset activation of the TA and the SOL and the decreased

**Table 3. Relative Magnitude of Short Latency Responses in Control and Chronic Ankle Instability (CAI) Groups in the Support and Perturbed Positions[a,b]**

| | | | Uninjured Limb | | | Between-Subjects Comparisons | | Injured Limb | | | Between-Subjects Comparisons | | Within-Subject Comparisons | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Group | Position | Muscle | Mean ± SD | 95% CI | MDD | *t* Value | *P* Value | Mean ± SD | 95% CI | MDD | *t* Value | *P* Value | *t* Value | *P* Value |
| Control | Support | Tibialis anterior | 4.3 ± 3.25 | 2.3, 5.6 | 7.1 | 1.828 | .077 | NA | | | 1.433 | .162 | NA | |
| CAI | | | 2.8 ± 1.87 | 1.2, 5.3 | 3.8 | | | 3.1 ± 1.88 | 1.1, 4.7 | 4.2 | | | −0.565 | .579 |
| Control | Perturbed | | 4.6 ± 8.32 | 1.4, 3.9 | 20.4 | 0.288 | .775 | NA | | | 1.069 | .291 | NA | |
| CAI | | | 4.0 ± 2.73 | 2.4, 6.4 | 1.4 | | | 2.7 ± 1.48 | 1.6, 4.1 | 3.4 | | | −0.638 | .532 |
| Control | Support | Soleus | 0.7 ± 0.26 | 0.6, 0.9 | 0.5 | −2.063 | **.047** | NA | | | −1.292 | .204 | NA | |
| CAI | | | 1.0 ± 0.41 | 0.6, 1.0 | 0.8 | | | 1.0 ± 0.46 | 0.4, 1.1 | 0.6 | | | 1.312 | .207 |
| Control | Perturbed | | 0.9 ± 0.31 | 0.7, 1.0 | 0.4 | −1.692 | .100 | NA | | | −1.087 | .284 | NA | |
| CAI | | | 1.1 ± 0.47 | 0.7, 1.2 | 0.9 | | | 1.0 ± 0.37 | 0.7, 1.0 | 0.6 | | | −0.647 | .526 |
| Control | Support | Peroneus longus | 2.0 ± 1.11 | 1.3, 2.7 | 1.3 | −0.199 | .844 | NA | | | 0.986 | .330 | NA | |
| CAI | | | 2.1 ± 1.57 | 0.8, 3.2 | 3.1 | | | 1.5 ± 0.78 | 0.9, 1.7 | 1.2 | | | 2.379 | **.028** |
| Control | Perturbed | | 1.8 ± 1.36 | 1.2, 2.6 | 1.9 | −1.899 | .065 | NA | | | −2.251 | **.030** | NA | |
| CAI | | | 2.7 ± 1.63 | 1.6, 4.0 | 3.6 | | | 2.8 ± 1.55 | 1.8, 2.9 | 2.8 | | | 0.500 | .626 |
| Control | Support | Peroneus brevis | 1.9 ± 1.03 | 1.3, 2.5 | 2.4 | 0.349 | .729 | NA | | | −0.377 | .708 | NA | |
| CAI | | | 1.8 ± 0.88 | 1.2, 2.9 | 1.6 | | | 2.1 ± 1.46 | 1.4, 2.7 | 3.5 | | | 0.130 | .898 |
| Control | Perturbed | | 2.6 ± 2.46 | 1.2, 4.4 | 3.3 | −0.387 | .701 | NA | | | −0.783 | .438 | NA | |
| CAI | | | 2.9 ± 1.94 | 1.4, 4.2 | 0.8 | | | 3.2 ± 1.92 | 2.0, 4.6 | 3.6 | | | −0.849 | .407 |

Abbreviations: CI, confidence interval; MDD, minimal detectable difference; NA, not applicable.

[a] The relative magnitude represents the ratio between the electromyographic magnitudes demonstrated in the short latency responses and during upright standing.

[b] Bold values represent *P* values $< .05$.

**Table 4. Normalized Relative Magnitude of Medium Latency Responses in Control and Chronic Ankle Instability (CAI) Groups in the Support and Perturbed Positions[a,b]**

| Group | Position | Muscle | Uninjured Limb | | | Between-Subjects Comparisons | | Injured Limb | | | Between-Subjects Comparisons | | Within-Subject Comparisons | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Mean ± SD | 95% CI | MDD | *t* Value | *P* Value | Mean ± SD | 95% CI | MDD | *t* Value | *P* Value | *t* Value | *P* Value |
| Control | Support | Tibialis anterior | 23.1 ± 9.18 | 13.6, 27.1 | 8.8 | 2.969 | **.010** | NA | | | 2.512 | **.016** | NA | |
| CAI | | | 12.4 ± 4.78 | 0.2, 11.4 | 10.7 | | | 13.6 ± 8.89 | 0.9, 21.0 | 7.5 | | | −0.621 | .542 |
| Control | Perturbed | | 16.2 ± 10.60 | 8.7, 21.9 | 11.1 | −0.179 | .859 | NA | | | 1.450 | .159 | NA | |
| CAI | | | 16.9 ± 13.70 | 7.4, 16.4 | 13.9 | | | 11.3 ± 6.02 | 3.9, 25.3 | 6.6 | | | 1.739 | .098 |
| Control | Support | Soleus | 2.2 ± 1.33 | 1.2, 2.8 | 2.1 | 0.336 | .739 | NA | | | −0.796 | .430 | NA | |
| CAI | | | 2.1 ± 1.50 | 0.3, 1.9 | 0.9 | | | 2.9 ± 2.46 | 0.1, 3.6 | 5.7 | | | −1.265 | .227 |
| Control | Perturbed | | 1.8 ± 0.83 | 1.2, 2.2 | 1.6 | −0.648 | .522 | NA | | | −1.306 | .201 | NA | |
| CAI | | | 2.0 ± 1.00 | 1.3, 2.7 | 2.6 | | | 2.2 ± 1.46 | 1.4, 2.6 | 2.8 | | | −1.084 | .300 |
| Control | Support | Peroneus longus | 5.6 ± 3.22 | 4.2, 9.2 | 6.8 | −2.293 | **.028** | NA | | | 0.939 | .354 | NA | |
| CAI | | | 8.6 ± 4.63 | 2.8, 9.1 | 11.3 | | | 5.3 ± 2.82 | 1.6, 9.8 | 5.2 | | | 2.842 | **.012** |
| Control | Perturbed | | 6.0 ± 4.48 | 5.0, 9.8 | 4.6 | −2.294 | **.028** | NA | | | −3.020 | **.004** | NA | |
| CAI | | | 9.4 ± 4.23 | 7.7, 14.9 | 6.2 | | | 10.6 ± 5.40 | 4.6, 12.8 | 12.5 | | | −0.791 | .441 |
| Control | Support | Peroneus brevis | 7.9 ± 4.65 | 4.8, 11.5 | 7.5 | 0.295 | .770 | NA | | | 0.250 | .803 | NA | |
| CAI | | | 7.4 ± 3.98 | −0.1, 13.4 | 4.1 | | | 7.5 ± 5.39 | 7.4, 16.4 | 14.5 | | | −0.424 | .677 |
| Control | Perturbed | | 10.6 ± 7.03 | 7.8, 19.2 | 9.3 | −0.693 | .493 | NA | | | −1.117 | .271 | NA | |
| CAI | | | 12.2 ± 7.68 | 1.3, 19.6 | 8.5 | | | 13.8 ± 8.13 | 8.7, 18.7 | 15.1 | | | −1.101 | .285 |

Abbreviations: CI, confidence interval; MDD, minimal detectable difference; NA, not available.

[a] Relative magnitude represents the ratio between the electromyographic magnitudes demonstrated in the medium latency responses and that obtained during upright standing.

[b] Bold values represent *P* values $< .05$.

magnitude of the TA MLR could lead to decreased capacity when accelerating the center of pressure in the direction of the support limb to dampen the contralateral ankle-sprain mechanism.[10] Consequently, impaired postural responses of the TA and the SOL in the uninjured limb in the support position could compromise the stability of the contralateral injured ankle in the presence of CAI, whereas impaired TA postural responses in the injured limb in the support position could compromise the stability of the contralateral uninjured ankle. Future studies involving center-of-pressure displacement variables as a measure of stability are required to confirm this hypothesis. These bilateral deficits after unilateral injury are explained by the coupled neural circuits that control each leg.[27] An experiment[28] in animals demonstrated the existence of a group of interneurons that received supraspinal input from the vestibulospinal and reticulospinal pathways and pyramidal tract and bilateral peripheral input from group II fibers. In this sense, the peripheral input provided by these afferents may be critically involved in sustaining coordination between limbs and can explain the negative influence of unilateral impairment of the contralateral limb's postural-control responses. The MDD values presented in the "Results" section estimate the change for each variable that would need to occur within each group for us to be 95% sure that the change was not associated with instrument error. The MDD values indicate that only differences between sessions greater than 32.6 and 35.7 milliseconds, for the activation of the uninjured-limb TA and SOL muscles, respectively, in the support position and greater than 10.7 and 7.5 for the magnitude of the TA MLR in the uninjured and injured limbs, respectively, in the support position can reveal potential improvements or deficits in athletes with CAI.

Our findings in the CAI group reinforce the idea of an interlimb connection, as later-onset activation of the PB and SOL muscles was seen in the uninjured limb compared with the injured limb in the support position. In fact, the later activation of the PB and SOL muscles in the uninjured limb in the support position was probably related to decreased proprioceptive information from the injured limb in the perturbed position. Participants with CAI presented with increased errors in inversion movement detection and evertor force sense in the injured limb.[29]

The peroneal muscles have been described as the primary muscles protecting against lateral ankle injury via their eccentric action in providing lateral stabilization of the ankle.[30] The participants in our CAI group presented with increased magnitude of SLRs and MLRs of the PL muscle in the perturbed position in the injured limb and of MLR in the uninjured limb versus the control group. The increased PL compensatory responses in the CAI group appeared to be related to the reduced TA MLR observed in both limbs in the support position. This compensatory strategy seemed to occur not only in the injured perturbed limb but also in the uninjured limb in the support position. These findings reinforce the idea that impaired peroneal postural responses are not key to understanding CAI and corroborate the concept that the peroneal response, which was widely seen as the most prominent defense against ankle inversion, is in fact part of a generalized postural-equilibrium response that involves synergy between both limbs. However, this possibility should be explored in future studies evaluating interlimb coordina-

tion in participants with CAI as they perform functional tasks, such as gait initiation and the double-support phase of gait, when interlimb coordination is highlighted.[31–33]

### Limitations

For SLRs, we evaluated both activation and relative magnitude in a predefined epoch. However, for MLRs, we evaluated only the relative amplitude in a predefined epoch. Future researchers should assess the muscle-activation timing of the MLRs.

The lack of evaluation of other biomechanical variables is the major limitation of the present study. Center-of-pressure displacement must be examined to evaluate the effects of the postural-control impairments of the CAI group and the compensatory postural-control strategies on global postural-control indices to more accurately assess their relation to the risk of injury. Not having determined the level of disability of the CAI group also limits the comparisons of our results with those of previous authors.

## CONCLUSIONS

While assuming a support position, participants with CAI displayed bilateral postural-control deregulation as expressed through bilateral deficits in amplitude modulation of the TA MLR and later-onset activation of the TA and the SOL of the uninjured limb. In the perturbed position, the uninjured limb demonstrated increased magnitude of the PL MLR, whereas the injured limb presented increased magnitude of the PL SLR and MLR. These findings show that CAI seems to be characterized by deregulation of bilateral postural control, expressed mostly in the support position. Therefore, rehabilitation specialists should include both lower limbs in rehabilitation protocols for individuals with unilateral ankle sprains to restore proper motor control.

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