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Ankle Bracing, Plantar-Flexion Angle, and Ankle Muscle Latencies During Inversion Stress in Healthy Participants

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Context: Ankle braces may enhance ankle joint proprioception, which in turn may affect reflexive ankle muscle activity during a perturbation. Despite the common occurrence of plantar-flexion inversion ankle injuries, authors of previous studies of ankle muscle latencies have focused on inversion stresses only.

Objective: To examine the latency of the peroneus longus (PL), peroneus brevis (PB), and tibialis anterior (TA) muscles in response to various degrees of combined plantar-flexion and inversion stresses in braced and unbraced asymptomatic ankles.

Design: Repeated measures.

Setting: University biomechanics laboratory.

Patients or Other Participants: Twenty-eight healthy females and 12 healthy males (n = 40: mean age = 23.63 years, range = 19 to 30 years; height = 172.75 ± 7.96 cm; mass = 65.53 ± 12.0 kg).

Intervention(s): Participants were tested under 2 conditions: wearing and not wearing an Active Ankle T1 brace while dropping from a custom-made platform into 10°, 20°, and 30° of plantar flexion and 30° of inversion.

Main Outcome Measure(s): The time between platform drop and the onset of PL, PB, and TA electromyographic activity was measured to determine latencies. We calculated a series of 2-way analyses of variance to determine if latencies were different between the conditions (braced and unbraced) and among the plantar-flexion angles ($\alpha = .05$).

Results: No interaction was found between condition and plantar-flexion angle. No significant main effects were found for condition or plantar-flexion angle. Overall means for braced and unbraced conditions were not significantly different for each muscle tested. Overall means for angle for the PL, PB, and TA were not significantly different.

Conclusions: Reflexive activity of the PL, PB, or TA was unaffected by the amount of plantar flexion or by wearing an Active Ankle T1 brace during an unanticipated plantar-flexion inversion perturbation.

Key Words: ankle injuries, lower extremity, biomechanics, orthoses, reaction time

Key Points

- In this study of participants with healthy ankles, latencies of the peroneus longus, peroneus brevis, and tibialis anterior muscles were not affected by the amount of plantar flexion during an unanticipated plantar-flexion and inversion perturbation.
- The Active Ankle T1 brace did not have an excitatory or inhibitory effect on peroneus longus, peroneus brevis, or tibialis anterior muscle latencies during an unanticipated plantar-flexion and inversion perturbation.

During a joint perturbation, reflexive muscle activity occurs in response to stimulation of mechanoreceptors within ligaments and muscles,[1–3] presumably to reduce the magnitude of joint movement.[4] During an ankle plantar-flexion and inversion perturbation, for instance, the ankle dorsiflexor and evertor muscles may be reflexively activated to decelerate the plantar-flexion and inversion movements.[4–6] The time between a perturbation and reflexive muscle activation is known as the latency period,[4–6] which is essentially the duration of a muscle's stretch reflex. In addition to the latency period is the electromechanical delay (EMD), the delay between muscle activation and the production of tension at the muscle's skeletal attachments. This lag occurs because time is required for the action potential's propagation along the sarcolemma, the excitation-contraction coupling process, and the removal of slack in the elastic elements.[7,8] If the combined muscle latency and EMD are shorter than the time it takes for the ankle joint to reach its physiologic motion limits, the muscles may help to decelerate ankle joint movement and reduce ligamentous strain.

Ankle muscle latencies are commonly measured in laboratory experiments by having participants drop unexpectedly on a tilting platform. Muscle latencies are determined by measuring the interval between the platform release and the onset (or marked increase) of ankle muscle electromyographic (EMG) activity.[9–14] The tilting platforms used by most previous authors[9,10,12,15–17] allowed participants to drop primarily into inversion, replicating the frontal-plane motion that commonly causes lateral ligament sprains[4–6] but not the sagittal-plane plantar-flexion motion that also occurs during most lateral ligament sprains.[4–6] One notable exception is a study by Lynch et al,[4] in which latencies were measured as participants

dropped into 30° of inversion and either 0° or 20° of plantar flexion. Lynch et al reported that peroneus longus (PL) and peroneus brevis (PB) latencies were longer at 20° of plantar flexion, whereas tibialis anterior (TA) latencies were unchanged. The authors speculated that peroneal muscle latencies were longer at 20° of plantar flexion because of greater approximation of the peroneal muscle attachments in the more plantar-flexed position.

Ankle braces have been used in some investigations of ankle muscle latencies[15,16,18,19] to determine if bracing alters reflexive muscle activity during perturbations. Ankle braces control joint position[12,20–22] and enhance ankle joint proprioception by stimulating cutaneous mechanoreceptors.[23] These findings have led to speculation that braces may have a positive effect on reflexive ankle muscle activity, including muscular latencies.[24] In previous work by Shima et al[16] and Papadopoulos et al,[17] however, PL latencies were longer during inversion perturbations when healthy participants wore ankle braces, decreasing the likelihood of protective muscular reaction. In contrast, Cordova et al[25] and Nishikawa and Grabiner[24] reported no differences in PL latencies between braced and unbraced healthy ankles during inversion stress.

Given the lack of consensus on the effects of bracing on ankle muscle latencies and the report by Lynch et al[4] of longer peroneal latencies with increasing plantar flexion, we conducted this study to investigate the combined effects of ankle bracing and various plantar-flexion angles on PL, PB, and TA muscle latencies during a sudden inversion perturbation. Including plantar-flexion movement with an inversion perturbation more closely replicates the mechanics of the ankle during a lateral ligament injury.[4–6] Based on the work of Lynch et al,[4] we anticipated that peroneal latencies would increase as plantar flexion increased. The lack of consensus in the literature makes it difficult for us to speculate on how bracing will affect muscle latencies in our study. Nonetheless, we believe it is important to determine if bracing has an inhibitory or excitatory effect on the protective muscular response to a multidirectional perturbation, because either effect may influence ankle brace usage.

METHODS

Participants

A convenience sample of 40 uninjured university students (age = 23.63 ± 2.06 years, range = 19 to 30 years) was recruited for this study (see Table 1 for demographics). Twenty-eight participants were female and 12 were male (pooled height = 172.75 ± 7.96 cm, pooled mass = 65.53 ± 12.00 kg). Participants were excluded from the study if they had experienced an ankle injury (defined as any injury to the ankle that limited typical activity for more than 48 hours[26,27]) to either ankle within 6 months of testing. Participants were also excluded from the study if they had a history of ankle or foot surgery or a lower extremity fracture. Before testing began, each participant signed a statement of informed consent. The Institutional Review Board for the Protection of Human Subjects approved the study protocol. Each volunteer participated in a single 45-minute testing session.

Instruments

A specially constructed tilting platform was built, which allowed each foot to drop into combined plantar flexion and inversion from a standing neutral position (Figure 1). The onset of the platform tilt was recorded by a universal self-aligning electrogoniometer (model ULGN-67; Therapeutics Unlimited, Iowa City, IA) attached to the back of the right side of the platform. The electrogoniometer consisted of a rotational potentiometer with a scale factor of 5.55 mV/°. The electrogoniometer was interfaced to an amplifier (Therapeutics Unlimited). Electrogoniometer output was connected to a 16-bit analog-to-digital (A/D) converter (Run Technologies, Laguna Hills, CA) within a personal computer. The computer simultaneously digitized the analog information and the analog muscle activity at 1000 Hz in the Data Pac 2000 software (Run Technologies).

Muscle activity data were collected with a multichannel EMG amplifier/processor unit (model 67; Therapeutics Unlimited), using bipolar Ag/AgCl disc surface electrodes (19-mm fixed interelectrode distance) interfaced with a personal computer. Each electrode had an on-site, solid-state differential amplifier (gain = 35 ± 10%) embedded in a plastic enclosure. The common mode rejection ratio was 87 dB at 60 Hz, and the input impedance was greater than 15 mΩ at 100 Hz. The amplifier/processor unit was capable of acquiring 40- to 4000-Hz raw EMG signals and had additional gain settings of 500, 1000, 2000, 5000, and 10 000. Data were digitally acquired at 10 000 Hz with the use of a 16-bit analog-to-digital converter (Run Technologies, Inc) enclosed in a personal computer. We thought this high sampling rate was necessary to acquire representative data during the high-velocity inertial exercises. All

Table 1. Subject Demographics

	Mean ± SD	Range
Females (n = 28)		
Age, y	23.25 ± 2.05	19–29
Height, cm	170.32 ± 6.99	157–182
Mass, kg	60.64 ± 9.09	45–90
Males (n = 12)		
Age, y	24.50 ± 1.88	23–30
Height, cm	178.42 ± 7.39	169–197
Mass, kg	76.91 ± 10.29	62–100
Females and males combined (n = 40)		
Age, y	23.63 ± 2.06	19–30
Height, cm	172.75 ± 7.96	157–197
Mass, kg	65.53 ± 12.00	45–100

Figure 1. Plantar-flexion and inversion platform.

raw EMG data from the unit were collected and stored on a personal computer using custom Data Pac software.

A universal, self-aligning electrogoniometer (model ULGN 67; Therapeutics Unlimited) was used to measure knee range of motion during the 8 seated quadriceps exercises. The electrogoniometer consisted of a rotational potentiometer with a scale factor of 5.55 mV/° rotation and resistive load of ±1.0%. Two flexible plastic arms extended from the potentiometer. Electrogoniometer data were collected simultaneously with the EMG data.

Procedures

We prepared 3 muscle sites on the lower extremity for surface electrode placement by shaving, abrading, and cleaning the areas with alcohol. Gel was placed on each electrode after attaching an adhesive collar. The electrodes were then centered over the most prominent portions of the muscle bellies using the landmarks suggested by Lynch et al.[4] For the PL, the electrode was placed at the junction of the proximal and middle thirds of the fibula over the palpable lateral compartment. The electrode for the PB muscle was placed three quarters of the distance between the fibular head and the lateral malleolus, immediately anterior to the PL tendon. For the TA, the electrode was placed at the junction of the proximal and middle thirds of the tibia, over the largest portion of the muscle belly. Electrode positioning was verified by observing the EMG signal on a computer monitor during maximal voluntary ankle eversion for the PL and PB and during maximal voluntary ankle dorsiflexion for the TA. The same experimenter (M.M.) completed the procedures for each participant to control for differences in preparation and placement techniques.

Each participant was tested under 2 conditions: braced and unbraced. The Active Ankle T1 model (Active Ankle Systems, Inc, Louisville, KY) used during the braced condition testing is a semirigid brace consisting of a neoprene padding system and hinged, molded thermoplastic strips along the medial and lateral aspects of the lower leg. The order of testing (braced or unbraced) was randomized for each participant by a coin flip. We controlled for variations in footwear by having each participant wear running shoes (model 625; New Balance, Inc, Boston, MA) fitted according to the manufacturer's guidelines. Before the braced condition, participants were instructed to tighten the brace as if they were to begin participating in a running or jumping sport. Perturbations were performed on the titling platform, with each participant's feet shoulder-width apart and body weight equally distributed over both feet (Figure 2). Equal distribution of weightbearing was subjectively determined. Angled cuts in the platform allowed it to tilt to 10°, 20°, or 30° of plantar flexion with a constant 30° inversion angle. Mechanical stops were integrated to enable each of these 3 positions to be assessed. We selected a tilt combining plantar flexion and inversion because ankle sprains often occur in this position.[4–6] The 30° inversion angle was chosen because inversion angles ≥40° are purported to cause untoward stress and potential injury to the lateral ligaments.[5,28–30]

Two hook-and-loop straps, secured over the dorsum of the feet, held the ankles in the desired starting position. A sheet was tied around the participant's waist and draped to prevent views of the platform or the platform release. The platform was released manually for all participants by the same experimenter (M.M.) when baseline EMG activity was observed on the monitor. The interval between draping and platform release

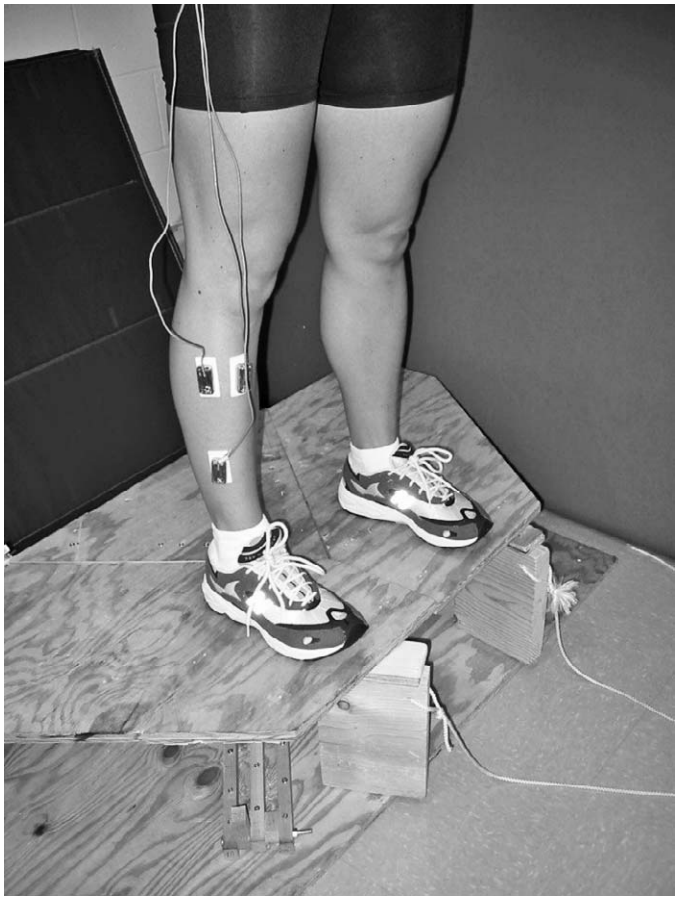

Figure 2. Experimental setup and electrode placement for the peroneus longus, peroneus brevis, and tibialis anterior muscles.

was randomized to avoid anticipatory muscle activation. Participants listened to music through headphones to muffle the sound of the platform release. A trial perturbation was administered to each ankle to allow the participants to become familiar with the experimental procedure.

The side of the tilt and the amount of combined plantar flexion and inversion were also randomized among trials. We only collected data on the right ankle because previous investigators[6,29–31] reported no difference in peroneal muscle latency between the right and left ankles. However, left ankle perturbations were also performed to prevent participants from anticipating which side would be perturbed and to promote equal weightbearing on both feet. Data were collected during 3 trials at each tilt angle (10°, 20°, and 30° of plantar flexion plus 30° inversion) during both conditions (braced and unbraced). The rest interval between trials was approximately 30 to 45 seconds to allow the experimenter to reposition the platform and the participant to regain equal weight distribution over both feet. The onset of platform tilt (as determined by the electrogoniometer channel) signaled the onset of the perturbation. As in previous investigations of muscle latency,[6,32] muscle activation 10 SDs beyond the baseline resting EMG activity signaled the onset of muscle activation. Latency was calculated from the onset of platform tilt (as determined by the electrogoniometer channel) until the time of muscle activation (see Figure 3 for a detailed description of this procedure). The data collection protocol was identical for the braced and unbraced conditions.

The Data Pac software was used to calculate the latency

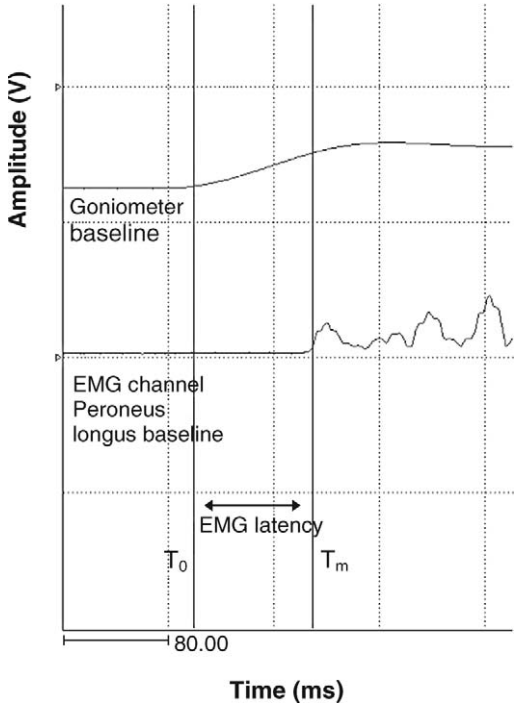

Figure 3. A typical electromyographic (EMG) latency trial is depicted. Only 2 analog channels (goniometer and processed EMG for the peroneus longus muscle) are shown for clarity. Latency values were measured from the change in amplitude related to 2 events (T_0 and T_m). T_0 was determined from the goniometer measurement to be time when the goniometer deflected beyond 2 SDs from baseline, indicating initiation of platform drop. Muscle activation onset (T_m) was determined separately from each of the processed EMG channels, where the root mean squared activation level was beyond 10 SDs of baseline muscle activation.[6,32] Latency was the time difference between T_m and T_0 in milliseconds (ms). T_0 was the same for all muscles, whereas T_m was based on the baseline EMG activation of each muscle assessed: peroneus longus, peroneus brevis, and tibialis anterior.

times based on these 2 events for each EMG channel. The mean muscle latency was calculated from the 3 trials at each tilt angle (10°, 20°, or 30° of plantar flexion plus 30° of inversion) during both conditions (braced and unbraced) for the right ankle only.

Statistical Analysis

We used a within-subjects design with 2 independent variables: condition (braced or unbraced) and plantar-flexion angle (10°, 20°, or 30°). A series of 3 × 4 fully repeated-measures analyses of variance were calculated to investigate the effects of ankle brace condition and plantar-flexion angle on PL, PB, and TA muscle latency. The dependent measure was muscle latency time in milliseconds. Data from male and female participants were pooled, and no comparisons were made between the sexes because ankle muscle latencies are similar in men and women.[33] This sex-pooled, case-controlled model has been used successfully in similar previous experiments.[10,12] The measurement reliability of 3 trials for each angle and condition were calculated using an intraclass correlation coefficient (2,1). Data were analyzed using SPSS computer software (version 10.1 for Windows; SPSS Inc, Chicago, IL). Alpha was set a priori at .05.

Table 2. Muscle Latency by Brace Condition and Plantar-Flexion Angle for the Peroneus Longus, Peroneus Brevis, and Tibialis Anterior (Mean ± SD, ms)

		Plantar Flexion		
Muscle	Condition	10°	20°	30°
Peroneus longus	Braced	52.6 ± 10.1	51.9 ± 9.9	52.7 ± 9.8
	Unbraced	50.3 ± 9.9	53.8 ± 8.8	53.1 ± 7.9
Peroneus brevis	Braced	52.9 ± 10.4	51.7 ± 9.2	53.1 ± 10.3
	Unbraced	51.2 ± 9.2	53.9 ± 8.4	53.4 ± 9.1
Tibialis anterior	Braced	51.3 ± 9.7	51.7 ± 9.4	51.2 ± 9.9
	Unbraced	51.2 ± 10.9	53.4 ± 9.7	52.3 ± 9.9

RESULTS

No significant interaction was reported between braced condition and angle for the PL ($F_{2,38} = 2.87$, $P = .08$), PB ($F_{2,38} = 1.74$, $P = .18$), or TA ($F_{2,38} = .34$, $P = .73$). For the PL, the observed power was .48 with an effect size of 0.06; for the PB, power was .12 with an effect size of 0.01; and for the TA for the brace and angle interaction, power was .09 with an effect size of 0.009. Means and SDs for the average muscle latency by condition and plantar-flexion angle for each muscle are presented in Table 2.

No difference was noted among the 3 plantar-flexion and inversion tilt angles for the PL ($F_{2,38} = 1.61$, $P = .22$), PB ($F_{2,38} = .95$, $P = .38$), or TA ($F_{2,38} = .65$, $P = .54$), For the PL, the observed power was .18 with an effect size of 0.19 and a standard error of 1.21; for the PB, power was .33 with an effect size of 0.05 and a standard error of 1.22; and for the TA, power was .08 with an effect size of 0.33 and a standard error of 1.11.

No difference was found between the braced and unbraced condition for the PL ($F_{1,39} = .01$, $P = .90$), PB ($F_{1,39} = .31$, $P = .61$), or TA ($F_{1,39} = 1.58$, $P = .23$). For the PL, the observed power was .04 with an effect size of 0.00; for the PB, power was .05 with an effect size of 0.00; and for the TA, power was .22 with an effect size of 0.23. Mean differences ranged from 1% to 3% depending on the muscle tested and the plantar-flexion angle.

Intraclass correlation coefficients were calculated separately for each of the 3 trials at each angle and for each condition. The coefficients ranged between .75 and .86, and the standard error of measurement (SEM) ranged between 1.1 and 2.1 milliseconds. Based on the SEM values, 95% of the participants demonstrated muscle latencies between 45.8 and 58.2 milliseconds across all angles and conditions. Average time for participants to reach the 30° of plantar flexion and 30° of inversion position was 91 ± 1.98 milliseconds.

DISCUSSION

To our knowledge, this is the first study on the combined effects of ankle bracing and various degrees of plantar flexion on muscle latencies in participants with healthy ankles during a sudden inversion perturbation. Our primary objective was to see if the PL, PB, or TA muscles responded any slower or faster if the ankle joint underwent multidirectional displacement when an ankle brace was worn. Previous authors[4–6] have focused on inversion movement alone, but we chose to include the plantar-flexion component because lateral ankle sprains are usually the result of excessive plantar-flexion and inversion displacement.

Based on the report by Lynch et al[4] of longer peroneal latencies at 20° of plantar flexion versus 0°, we anticipated that peroneal latencies would be longer with increasing plantar flexion. As an extension of their work, we measured latencies at 10°, 20°, and 30° of plantar flexion to further analyze the relationship between latency and plantar-flexion angle. In contrast to Lynch et al,[4] we did not find a significant difference in latencies among the 3 plantar-flexion angles for the PL, PB, or TA. Thus, in our study, the latency of each of these muscles was seemingly unaffected by the amplitude of the perturbation. This finding suggests, for example, that the PL (as well as the PB and TA) became active at the same time after each platform release and perturbation, regardless of the ending plantar-flexion angle. One plausible explanation for this finding is that the latencies we measured were ''short-loop'' (spinal-level, monosynaptic) reflexes, which are thought to occur within the first 50 milliseconds after passive muscle lengthening.[34,35] Our latency values seem to support this explanation: 95% of the participants in our study had latencies between 45.8 and 58.2 milliseconds across all angles and conditions. This finding is consistent with the 50- to 60-millisecond peroneal latency values reported by Ebig et al,[5] Rosenbaum et al,[36] and Konradsen and Ravn[28,37] in their studies of participants with healthy ankles. If we were indeed measuring this short-loop reflex, then muscle lengthening that occurred after the initial spindle stimulation (ie, with increasing plantar flexion or inversion) would not be expected to affect measured latencies. This possibility could account for our finding that latencies were independent of the amount of perturbation.

Lynch et al[4] speculated that increased peroneal muscle latency with increased plantar flexion was due to approximation of the peroneal muscle's attachments in the more plantar-flexed position. It is not clear, however, how this length change during the perturbation would affect the initial spindle excitation. The platform used by Lynch et al[4] was hydraulically controlled to maintain a constant angular velocity of either 50°/s or 200°/s when released. In our experiment, the angular velocity of platform movement was not controlled. By design, we would expect our platform to accelerate after release until it reaches the preselected stopping angle. We were unable to directly measure the angular acceleration of the platform with the attached goniometer due to the 3-dimensional nature of the platform drop during the trials. Nonetheless, differences in the platform behavior between our study and that of Lynch et al[4] may have contributed to differences in our latency findings and theirs. Despite these differences, our finding that peroneal muscle latency measures were reliable during these perturbation tests concur with those of Lynch et al,[4] Hopper et al,[38] and Benesch et al.[9]

Similar to Cordova et al[25] and Nishikawa and Grabiner,[24] we found no differences in latencies between the braced and unbraced conditions. The lack of difference may be because the Active Ankle brace did not impair initial muscle lengthening (ie, the short-loop reflex) during each perturbation. That is, muscle activation may have been initiated at the same time after each platform release and perturbation, regardless of brace use. Shima et al[16] and Papadopoulos et al,[17] who reported longer PL latencies when participants were braced, may have used braces that constrained the ankle more, resulting in a slowing of muscle lengthening immediately after platform release. Thus, the interval between platform release and the onset of the short-loop reflex may have been lengthened and resulted in longer measured latencies. Therefore, differences among studies on brace use and muscle latencies could be either partially or solely attributable to differences in the mechanical performance of the braces used.

We determined that it took an average of 91 ± 1.98 milliseconds for participants to reach the 30° of plantar-flexion and 30° of inversion position. This suggests that the peroneal muscles or TA would need to generate active tension to reduce the rate or magnitude (or both) of ankle movement in roughly 90 milliseconds or less after this perturbation. Based on our muscle latency values, it takes 50 to 54 milliseconds (or roughly 46 to 58 milliseconds when SEM is factored in) for muscle activation to occur after the perturbation. Therefore, the EMD would need to be roughly 36 milliseconds or less for the peroneals and/or TA to produce an active decelerating torque to counteract the motion. Although we did not measure EMD, Konradsen and Ravn[37] reported that the EMD of the peroneus longus was roughly 72 milliseconds for the unloaded ankle. Based on our muscle latency measurements and the EMD reported by Konradsen and Ravn,[37] it is unlikely that the PL has adequate time to generate an active eversion countertorque during an unexpected inversion. This is likely true for the TA also.

Despite the uncertainty of whether the ankle muscles are activated before ligamentous injury occurs, how much countertorque these muscles produce during plantar-flexion and inversion ankle perturbations is not known. Cordova and Ingersoll[19] reported that bracing enhanced peroneal EMG amplitude during an inversion perturbation, indicating higher levels of muscle activation. Still, whether these muscles can generate a sufficient countertorque to modulate injury severity, even with the increased level of peroneal muscle activity from wearing a brace, is unclear. Thus, aside from the issue of whether or not the latency and EMD are brief enough to sufficiently activate the ankle muscles, we need to determine if the ankle muscles can generate enough torque to prevent or even reduce ankle injury.

LIMITATIONS AND SUGGESTIONS FOR FUTURE RESEARCH

We only used 1 type of semirigid ankle brace; thus, our results can only be applied to questions of muscle latencies with the Active Ankle T1 brace. Further research is needed to determine if different semirigid and lace-up braces somehow constrain the initial muscle lengthening that may ultimately influence muscle latency. Consistent with past studies of this nature, the electrogoniometer in our study was applied to the platform, not the participant. Thus, the ''latency'' measured was actually the interval between the onset of platform movement and the onset of EMG activity and not necessarily the interval between the onset of ankle movement and the onset of EMG activity. Motion sensors or another electrogoniometer could be applied to the participant's leg, for instance, to register the onset of ankle movement and subsequently to see how this relates to the onset of platform movement. This approach would test the hypothesis that braces that better constrain the ankle and do not impair initial muscle lengthening after a perturbation do not affect latencies.

Our study was conducted on participants with healthy ankles only. Establishing whether latencies are different between braced and unbraced conditions or at different plantar-flexion and inversion tilt angles in injured or unstable ankles is necessary. Evidence is mixed on whether muscle latencies are

different after an inversion stress in injured or unstable ankles relative to healthy ankles. Although some authors found no difference in peroneal muscle latency between patients with functional ankle instability and healthy participants,[5,6,31,32,39–41] other authors found longer latencies in patients with ankle instability.[13,14,37,42]

In our study, we could not accurately measure the rate of plantar-flexion or inversion motion individually with the electrogoniometer due to its alignment on an oblique axis, resulting from the angled cuts of our perturbation platform. These angled cuts were necessary to allow both plantar flexion and inversion to occur. Lynch et al,[4] using a hydraulically controlled platform, reported that peroneal latencies were shorter during inversion moments at 200°/s versus 50°/s.[4] Perhaps if we too had examined muscle latencies at different angular velocities (50°/s and 200°/s) as Lynch et al[4] did, we might have reported shorter peroneal muscle latency with greater tilt velocities.

We did not measure EMD in our investigation. Previous investigators[8,43–49] have reported EMDs ranging from 7 to 122.9 milliseconds. There is a need to determine EMD along with ankle muscle latencies at various angles of plantar flexion and inversion to see if the combined latency period and EMD is brief enough to enable the ankle muscles to generate active tension during a perturbation.

CLINICAL IMPLICATIONS

Ankle braces may provide a prophylactic benefit by constraining ankle motion[12, 20–22] and enhancing ankle joint proprioception through stimulation of cutaneous mechanoreceptors.[23] Our results suggest, however, that the hinged brace used may not alter protective activation of the PL, PB, or TA muscles during unanticipated plantar-flexion and inversion stresses in participants with healthy ankles. This finding could be interpreted positively in that the brace we used did not appear to have an inhibitory influence on the muscles studied and, therefore, result in greater latencies. Thus, our results suggest that sports medicine practitioners may be able to employ this brace without impairing an athlete's reflexive ankle muscle response(s) to sudden plantar-flexion and inversion perturbations. Our findings also support the hypothesis that the prophylactic benefit of ankle braces may depend on their ability to mechanically constrain ankle motion[12] and not on their ability to enhance ankle muscle latencies.

CONCLUSIONS

In our study of healthy ankles, latencies of the PL, PB, and TA muscles in response to unanticipated plantar-flexion and inversion perturbations were not affected by use of an Active Ankle T1 brace or by the amount of plantar flexion during the perturbation. Our findings also suggest that the Active Ankle TI brace did not have an excitatory or inhibitory effect on reflexive ankle muscle activity. Our results support the contention that ankle braces may prevent ankle injuries by constraining frontal-plane ankle motion and not by affecting ankle muscle latencies.

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