

Alterations in Evertor/Invertor Muscle Activation and Cop Trajectory during a Forward Lunge in Participants with Functional Ankle Instability

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Abstract

Objectives: Participants with ankle instability demonstrate more foot inversion during the stance phase of gait than able-bodied subjects. Invertor excitation, in combination with evertor inhibition may contribute to this potentially injurious position. This alteration may be more pronounced when additional loads are placed on the foot during functional movements. The purpose of this experiment was to examine evertor/invertor muscle activation and foot center of pressure (COP) trajectory during foot loading in participants with functional ankle instability (FI).

Methods: Twelve subjects were identified with FI and matched to healthy controls. Tibialis anterior (TA) and peroneus longus (PL) electromyography (EMG), as well as COP, were recorded during the entire stance portion of a forward lunge. Functional analyses were used to detect differences between FI and control subjects with respect to normalized EMG amplitude and COP trajectory during the stance portion of the forward lunge.

Results: The functional analysis revealed no differences between groups during the stance phase of the lunge for PL EMG or COP. The FI group did exhibit significantly less TA activation at the beginning and end of stance. However, TA activation increased in the FI group relative to controls during the loading portion of stance.

Conclusion: While COP did not deviate during a loading task, the observed motor strategies could contribute to movement/stabilization alterations in a FI population. Treatment of FI should consider interventions focused on forcing normal activation of the invertors/evertors.

Keywords: Peroneuslongus; Plantarpressure; Tibialisanterior

Introduction

Ankle injury is the most common injury in sports [1,2]. Ankle sprains represent a large portion of orthopedic injuries among the general population [3], with an associated cost of approximately \$2 billion annually [4]. Some ankle sprains are treated with limited or no long-term consequence, however as many as 73% of physically active individuals who suffer an ankle sprain experience repeated ankle injury, and 59% report significant long-term disability and residual symptoms [5]. These findings characterize chronic ankle instability (CAI), a firmly established orthopedic problem, which may also play a significant role in ankle osteoarthritis [6].

CAI was characterized by Delahunt et al. [7] as “an encompassing term used to classify a subject with both mechanical and functional instability.” Several factors contribute to the chronic nature of ankle instability. Mechanical factors, such as joint laxity, play a significant role in CAI [8]. Sensorimotor deficits (functional instability: FI) also play a primary role in perpetuating the chronic nature of ankle instability [9]. Reported sensorimotor deficits include muscle weakness and dysfunction [10-13], static and dynamic postural control alterations [14,15], altered integration of sensory information at the CNS [16,17], and altered muscle spindle sensitivity [11,18]. Many of these sensorimotor deficits have been linked to altered mechanics during various functional movements in patients with ankle instability [19,20].

The foot evertors, as the primary resistance to inversion stress, have received considerable attention for their potential contribution to FI [11,13,21,22]. Indeed, peroneal dysfunction has been well documented in subjects with ankle instability [11,13,21,22]. While the evertors likely do not provide a timely protective contraction to prevent injury during unanticipated foot inversion [23,24], they do help control foot position

during functional movement [24]. Invertor/evertor contraction during movement facilitates a neutral position, aids in balance, and controls loads during the stance phase of gait [24,25]. In addition to reported decreased evertor activation during functional movement [11,22], there is limited evidence of increased invertor activation during functional movement in participants with ankle instability [24,26]. Increased invertor activation coupled, with decreased evertor activation, could result in a more supinated foot position during the stance phase of movement in patients with ankle instability. This idea is consistent with reports of laterally-deviated plantar pressure during the stance phase of gait in patients with ankle instability [27,28].

It is unknown whether a more demanding load on the foot during movement would exacerbate this problem. The forward lunge may be considered a more demanding functional task of the lower extremity compared to walking. A single footed landing during a forward lunge would require a greater stabilizing co-contraction of the lower extremity and increased balance demands [29]. This type of movement could magnify neuromechanical deficits in patients with ankle instability.

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The purpose of this study was to evaluate invertor/evertor activation and plantar pressure deviations in participants with FI during a forward lunge. We hypothesized that FI participants would demonstrate increased tibialis anterior (TA), decreased peroneus longus (PL) activation, and laterally deviated center of pressure (COP) trajectories relative to matched controls. This is the second study examining the effects of foot position and leg muscle activation during functional movements from a sample of FI participants [30].

Methods

Subjects (n=12, 5 males and 7 females; Table 1) were identified with FI via the Functional Ankle Ability Measure (FAAM), the Modified Ankle Instability Index (MAII), and a physical examination. FI inclusion criteria were an ADL (activities of daily living) score of 90% or less and a sport score of 80% or less on the FAAM [31] and 2 “yes” answers on questions 4-8 on the MAII confirming a sensation of ankle instability (Table 1) [32]. The physical exam included a talar tilt and anterior drawer performed by an experienced, licensed physical therapist. Potential subjects were excluded if excessive laxity with no distinct end feel during either test was observed or a sulcus sign during the anterior drawer was present. Subjects with a positive physical exam test were excluded in order to examine subjects with sensorimotor deficits without mechanical instability (MI). Controls (n=12, 5 males and 7 females; Table 1) had normal scores (above 90% on ADL and 80% on sport and no “yes” answers) on the FAAM and MAII and no history of ankle injury. All subjects were otherwise healthy and physically active. Controls were matched according to sex, height, and weight. All subjects read and signed an informed consent form approved by the university institutional review board.

A between group (FI, control) comparison was used to evaluate changes in COP trajectory and muscle activation characteristics during the stance portion of a forward lunge (Figure 1). The independent variable was group (FI, control). Three dependent variables were surface electromyography (EMG) amplitude for the TA and PL and medial-lateral position of the COP during the stance phase. Stance was defined as the time from initial foot contact on the force plate to the point where the foot was completely removed or lifted from the force plate.

Subjects reported once to the laboratory for a single data collection session. Subjects completed the informed consent, FAAM, MAII, and physical exam. Qualifying subjects were then prepared for surface EMG and COP measurements. The skin over the TA, PL, and medial malleolus (reference electrode) was shaved, lightly abraded and cleaned with isopropyl alcohol. Electrodes were placed over the TA and PL according to methods described by Basmajian and DeLuca [33]. EMG data were visually inspected during manual muscle testing to confirm proper electrode placement. Subjects were also fit with standard athletic shoes that contained F-scan insoles (Tekscan, Boston, MA, USA).

COP trajectories were calculated from pressure measurements collected using the aforementioned insoles (0.15 mm thick), imbedded with a grid of sensels (3.9 sensels/cm²). Each sensel recorded pressure at a sampling rate of 256 Hz with a measureable pressure up to 862 KPa. The force from each sensel was used in the following formula to calculate the COP for each time point. COP trajectory was described using x and y coordinates throughout stance, where the x coordinate

described the medial-lateral position of the COP. COP data were time normalized to the entire stance phase of each lunge. The lunge was performed to the beat of a metronome set to 40 bpm.

EMG data (2400 Hz; Tel100, Biopac Systems Inc., Goleta, CA, USA) were bandpass-filtered (10-500 Hz), smoothed using a root mean square algorithm (time window=50 ms), and time normalized to 100% of the stance phase. EMG amplitudes were normalized to the EMG signal during quiet standing (2 sec), as quiet stance provided the most stable and consistent reference value.

The involved limb was evaluated in all FI subjects. The limb of the control subject was matched according to leg dominance (leg with which a ball is kicked). In other words, if the involved leg of the FI subject was their dominant leg, then we evaluated the dominant leg of the matched control subject. Subjects completed a general 5 min warm-up period at self-selected speed on a treadmill. After the warm-up, subjects completed 5 repetitions of the lunge in synch with a metronome set at 48 beats/min. The subject moved to the deepest flexion position on one beat (without the contra-lateral knee contacting the floor) and back to standing on the next beat. The length of the lunge was calculated as 100% of leg length (ASIS to medial malleolus), and the floor was marked with the beginning point and the end point of the step. The hands hung free, positioned to the sides of the hips. Synchronized EMG and pressure data were recorded for 5 consecutive stance phases. Start of pressure data measurements triggered the beginning of EMG data collection.

All data (throughout the entire stance phase) from each dependent variable were used in the statistical analysis to determine differences between the FI and control subjects. A functional analysis of variance (FAOV) [34] was used to determine differences between the FI and control subjects with respect to normalized EMG and COP trajectory during lunging ($\alpha=0.05$) over the entire stance phase. A FAOV analysis allows for a comparison of treatment effects as functional effects (polynomial functions) over the entire stance phase, rather than univariate or multivariate (discrete values) effects. In other words, we can detect whether there is a difference between the 2 groups, and where in the stance phase those differences exist. As the means for inference, any difference between groups (effect) that exceeds zero (no effect) at 95% confidence level is deemed statistically and clinically significant. This allows for statistical significance to be determined differentially as a function of the stance phase.

Results

Figures 2-4 summarize the functional analysis results. The zero lines in Figures 2-4 represent a no difference in means between the FI and control groups. Values above the zero lines represent greater FI values, while negative deviations from zero represent greater control group values. The shaded bands provide 95% confidence intervals for the population mean effect size (difference between control and treatment) throughout the stance phase. Therefore, when the zero line is outside of the shaded bands, statistically and clinically significant between-group differences exist. The functional analyses revealed no significant differences between groups during the stance phase of the lunge for PL EMG or COP trajectory ($p<0.05$; Figures 2 and 3). The FI group exhibited significantly less TA activation at the beginning and

Group	Age (yr)	Height (m)	Mass (kg)	FAAM ADL	FAAM Sport Scale	MAII “Yes”
FI	23 ± 4	1.74 ± 0.14	71.6 ± 17.6	83 ± 7.5	63 ± 12.3	3.3 ± 1.2
Control	23 ± 4	1.76 ± 0.17	71.4 ± 16.3	99.8 ± 0.7	99.6 ± 1.9	0.4 ± 0.2

Table 1: Subject demographics. FI inclusion criteria included a Functional Ankle Ability Measure (FAAM) ADL score of 90% or less and a sport score of 80% or less, and 2 “yes” answers on questions 4-8 on the Modified Ankle Instability Index (MAII).

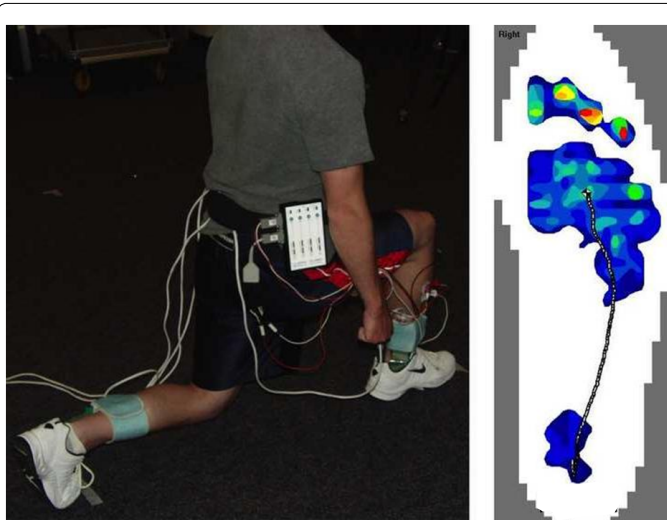


Figure 1: (A) Subject setup and position during the forward lunge. (B) A representative center of pressure (COP) trajectory is pictured summarizing one stance phase during a typical trial; the various colors indicate pressure magnitude at the end of stance (warm colors indicate high pressure, while cool colors represent low pressure).

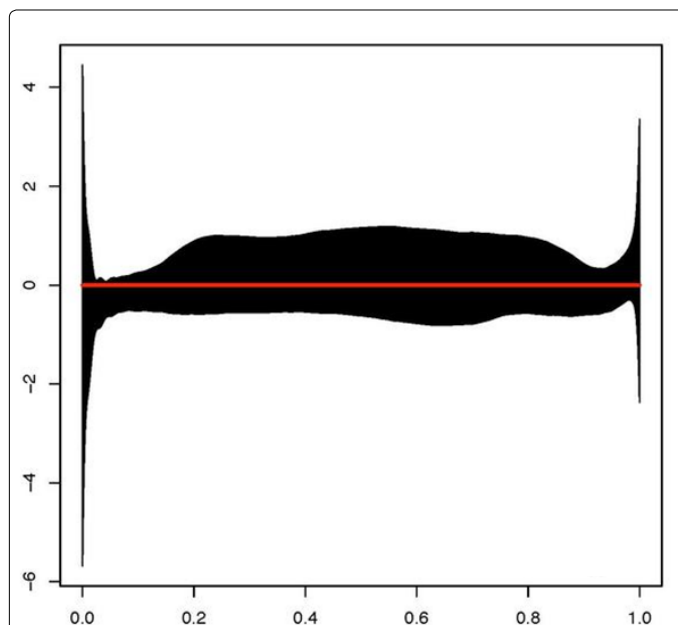


Figure 2: Results from the functional analysis of COP trajectory ($p > 0.05$). The center line (0) defines separation between groups (FI=+, control=-), and the vertical axis represents differences in x coordinate data. Shaded areas represent 95% confidence bands. Significant differences are defined by any area where the edges of the confidence bands are separated from 0.

end of stance, and increased TA activation during the loading portion (25%) of stance ($p < 0.05$; Figure 4).

Discussion

Our data did not support the hypothesis that FI participants would exhibit laterally-deviated COP trajectories during the stance phase of a forward lunge, relative to matched controls. Further, no PL alterations were detected during the stance portion of the lunge. The TA, however, was less active at foot strike and toe off, and more active during the loading phase (25% of stance), relative to matched controls (Figure 4).

Researchers have previously reported that plantar pressure, or foot position, deviates laterally in subjects with ankle instability during the stance phase of gait [19,27,28,30,35]. Indeed, we found a laterally deviated COP during walking in the exact same subject pool [30]. The current data are not consistent with these findings during walking, with the qualification that the forward lunge requires a different neuromechanical strategy than normal gait. The forward lunge is a task that requires a high level of sensorimotor control, integrating and processing sensory information to adapt muscle co-activation of the lower extremity for successful task completion. The task (forward lunge) requires balance and control of body load through a full range of motion. Altered postural control neural pathways, in addition to deficits in motoneuron pools, may enhance variability in COP measurements during the forward lunge. This increased variability could make detection of statistical differences between the groups more difficult. Indeed, static and dynamic postural control deficits have been well documented in those with ankle instability [14,15], and these deficits would equate to increased variability for COP trajectory during the relatively demanding task of a forward lunge. Another potential reason for not detecting COP differences between the groups is that the forward lunge requires a relative “centering” of the COP on the foot in order to complete the task. Otherwise, the subject would not be able to maintain a single-footed position during the stance phase of the lunge. In this respect, COP trajectory during the lunge might inherently differ from COP trajectory during walking. Regardless of the reason for differences between walking and the lunge task studied here, it is a significant finding that FI subjects use a different strategy depending on the task. Perhaps, FI subjects are trying to place the foot in a perceived “safer” position during tasks, like the forward lunge, where they might anticipate a need for greater stiffness.

PL dysfunction, in terms of strength [10], onset [11], and contraction time [22], has been a common finding in patients with ankle instability. Conversely, others have reported no PL deficits in the same population [36,37]. Santilli et al. [22] reported decreased PL contraction time during stance (walking) in participants with CAI. Delahunt and colleagues, [38] reported an increase in PL activation

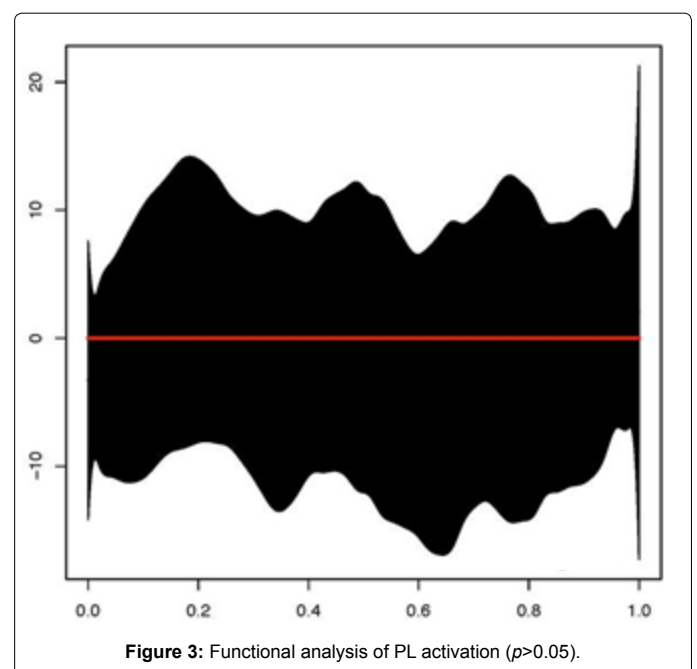
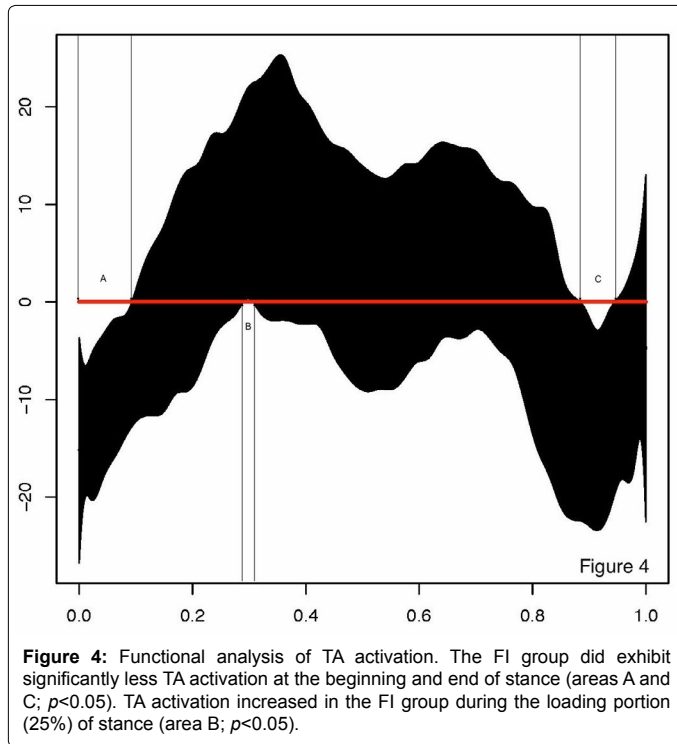


Figure 3: Functional analysis of PL activation ($p > 0.05$).



during the stance phase of gait in the same patient population along with a concentric evertor moment at initial heel contact. Our data are in agreement with previous reports of no evertor alterations between groups [36,37]. However, one should be cautious in their interpretation of these reports. PL activation, as it relates to the positioning of the foot, should be considered along with other muscles that aid in the same position. In this case the invertors should be considered. In the current study, while PL activation did not alter during loading, TA activation did. Without a paired response from the PL, TA alterations could ultimately alter foot kinematics. More data are needed to determine if these altered kinematics could expose the ankle injury and/or be a contributing factor to the disability reported by those with ankle instability. It is also important to note that large variability in PL activation could be a major contributor to the present lack of statistical differences for PL EMG and to the disparity in previous PL activation findings. Several investigators have reported large variability in PL activation during the stance phase of gait in participants with ankle instability and in able-bodied subjects [22,24,38,39]. The forward lunge would likely create a similar environment of high variability in PL EMG measurement.

TA activation produces both dorsiflexion and inversion of the foot during gait, making our findings difficult to interpret. The tibialis posterior (TP) is the primary coupling muscle with the PL for foot positioning [25]. Our initial intention was to measure TP EMG in addition to the data that have been reported. However, difficulty in consistent placement of fine wire electrodes and excessive noise made TP EMG recordings during the forward lunge unreliable. Louwerens et al. [24] reported increased TA activation in participants with ankle instability during the stance phase of gait. Delahunt et al. [26] also reported increased TA activity during a hopping movement. Our data are consistent with these. During the loading portion of the forward lunge (25%), TA activity increased in FI subjects relative to controls. Increased TA activation could be interpreted as a motor strategy in participants with FI, designed to keep the foot in a dorsiflexed, relatively stable position. While limited dorsiflexion range of motion

has been reported in this patient population [40,41], others have reported no change in sagittal plane ankle position during movement [38]. If FI participants are limited in dorsiflexion, then the TA could be contracting to overcome these limitations, whereas the process of loading might play that role in otherwise healthy ankles. It is also possible that the TA is firing as part of a patterned compensatory response to joint injury [42]. Regardless of the underlying reason, the consequence of TA facilitation could be imbalanced inversion torque, creating a poor position of the foot during functional movement or during some type of perturbation (e.g., stepping on a foot or in a hole).

At initial foot contact and prior to the foot leaving the ground we observed a decrease in TA activation in FI subjects relative to matched controls. We speculate that these activation deficits are a voluntary strategy intended to position the foot in a "safer" (more everted) position during initial contact and toe off. Whether this strategy is effective or has unintended consequences is unknown. Another potential reason for our observation of TA activation deficits at the beginning and end of stance is an inability of the TA to activate with appropriate timing. Perhaps the TA simply fires late and quits early in the stance phase of the forward lunge. More data are needed to confirm this idea, and determine what causes this response.

A few factors limit the implications of the present data. First, while we used 2 well-accepted instruments (FAAM and MAII) for inclusion in the FI group, we intentionally precluded patients with mechanical instability (positive talar tilt or anterior drawer). Our intention was to examine a portion of the CAI population that displays functional limitations due to sensorimotor deficits [9]. Another limitation is our small sample. Additionally, high variability was evident in this patient population. As previously mentioned, FI participants likely develop varying motor strategies to help negotiate physical demands that are present following injury. Therefore, while we did identify a motor strategy that could help perpetuate FI, the variability could result in an inability to detect alterations between groups.

Conclusion

Participants with FI exhibit altered TA activation patterns during a relatively demanding task – forward lunge. The TA was less active at foot strike and again at toe off and more active during the dynamic loading phase (25%) of stance relative to matched controls. This altered TA activation pattern during loading, without a coupled increase in PL activation, could produce injurious foot kinematics and increase susceptibility of repeated ankle inversion injury in this patient population.

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