Deficits in Peroneal Latency and Electromechanical Delay in Patients with Functional Ankle Instability

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ABSTRACT: The purpose of this study was to compare alterations in peroneal latency and electromechanical delay (EMD) following an inversion perturbation during walking in patients with functional ankle instability (FAI) and with a matched control group. Peroneal latency and EMD were measured from 21 patients with unilateral FAI and 21 controls. Latencies were collected during a random inversion perturbation while walking. EMD measures were collected during stance using a percutaneous stimulus. Two-way ANOVAs were used to detect differences between leg (affected, unaffected) and group (FAI, Control). Functionally unstable ankles displayed delayed peroneus longus (PL) latencies and EMD when compared to the unaffected leg and a matched control group. Peroneal latency and EMD deficits could contribute to recurrence of ankle injury in FAI subjects. How these deficits are associated with the chronic symptoms associated with FAI remains unclear, but gamma activation and subsequent muscle spindle sensitivity likely play a role. © 2009 Orthopaedic Research Society. Published by Wiley Periodicals, Inc. J Orthop Res 27:1541–1546, 2009

Keywords: dynamic joint stability; muscle spindle sensitivity

Acute lateral ankle injury is one of the most common injuries in the physically active.[1–4] Of these acute injuries, it is estimated that approximately 40%–80% of these patients may suffer repeated episodes and/or chronic instability of the ankle.[4–6] Chronic ankle instability may be influenced by several factors including mechanical and functional deficits.[7] While mechanical instability is focused on issues of laxity surrounding tissue damage, functional ankle instability (FAI) focuses on sensorimotor deficits that affect stability during functional movement. The complexity of the ankle functional anatomy and the nature of the loads placed on the ankle during sports and performance make definitive understanding of FAI difficult. However, if we are to develop effective prevention, treatment, and rehabilitation strategies, then we must continue to develop a better understanding of the factors involved.

Several sensorimotor deficits have been proposed to contribute to FAI, including impaired proprioception,[8–11] postural control,[12–14] strength,[15–17] and neuromuscular firing.[18–21] Neuromuscular firing considers the magnitude and timing of the muscles that activate, altering dynamic stability of the ankle. The short latency response is the first component of the neuromuscular response during any injury movement or position. The short latency response is largely mediated by the muscle spindle. Alterations in muscle spindle sensitivity would affect the timing of the neuromuscular firing as the primary spindle afferents would fire at varying thresholds. Therefore, alterations to the muscle spindle could be a primary contributor to short latency responses to ankle inversion injury and ankle instability. When considering ankle instability, the peroneal muscles are of particular interest. While the peroneals serve as the primary evertors of the foot, perhaps more important is their role to maintain foot position during movement and functional activity. Inadequate firing of the peroneals could result in uncontrolled rearfoot supination,[22] which would be consistent with reports of "giving way" in patients with FAI.

Several authors have reported deficits in peroneal latency in FAI patients following inversion perturbation.[18–21] However, the generalizability of these data is limited as latency was measured in a standing, static position. Neuromuscular firing has been reported to vary significantly when perturbation during functional movement is compared to a standing, static model.[23,24] Muscle spindle sensitivity changes throughout the gate cycle as body mass is transferred from one leg to another.[25,26] Therefore it seems necessary to consider the changeable state of the sensorimotor system during functional movement when determining the timing of peroneal contraction in patients with FAI.

While the latency of motor responses provides an estimate of the neurological contribution to motor responses, the mechanical contribution or electromechanical delay (EMD) is equally important. EMD provides an estimate of the time required to develop protective tension from the motor response. EMD is a measure of the time necessary for activation of a muscle to remove slack in the musculotendinous unit and provide tension on the bony insertions. Latency plus EMD provides a comprehensive estimate of the timing component of neuromuscular firing in response to an injury mechanism. Like a short latency response, EMD is very sensitive to changes in muscle spindle sensitivity as the level of muscle preactivation is related to gamma motoneuron activity.[23,24]

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In order to better understand whether neuromuscular firing deficits exist in persons with FAI, we set out to compare alterations in peroneal short latency responses and EMD following inversion perturbation during walking in patients with FAI and with a matched control group. We hypothesized that patients with FAI would exhibit deficiencies in peroneal latency and EMD compared to a matched control group following inversion perturbation during walking.

METHODS

This investigation employed a cross-sectional study design. The independent variables were group (FAI and control) and limb (affected and unaffected). The dependent variables were the peroneal latency and electromechanical delay (EMD).

Subjects

Twenty-one (18F, 3M) subjects with unilateral FAI (age $= 21 \pm 2$ years, height $= 171 \pm 7$ cm, weight $= 65 \pm 9$ kg) and 21 (18F, 3M) uninjured, matched controls (age $= 21 \pm 3$ years, height $= 169 \pm 9$ cm, weight $= 64 \pm 10$ kg) volunteered to participate in this study. Participants assigned to the FAI group met criteria set forth by both the Functional Ankle Instability Questionnaire[27] and the Ankle Instability Instrument.[28] For inclusion in the FAI group, subjects answered yes to question 1 along with "yes" responses to four total questions on the Ankle Instability Instrument. Further, subjects answered yes to questions 3, 5, 6, 7, and 9, and no to questions 4, 8, and 10 on the Functional Ankle Instability Questionnaire. Exclusion criteria included: any subject who presented with a history of surgery or fracture to the lower leg, presence of a neuromuscular disorder, mechanical instability in either ankle as measured on the Ankle Instability Instrument, or acute symptoms of any lower extremity pathology. All volunteers were recreationally active (Tegner score 5 or 6) and signed an informed consent prior to study commencement. Prior to testing, subjects were also asked to identify a dominant limb, which was described as the leg which he or she would use to kick a ball.

Instrumentation

A custom-built 8.5-m runway consisting of seven 1.22-m segments was utilized for this study.[29] Four of the seven segments were constructed with a bilateral trapdoor mechanism, which freely fell to 30° of inversion when triggered (Fig. 1). An adhesive, nonslip material was placed on the walkway to create a footpath and to prevent slipping when the trapdoors were dropped. An electric switch regulated a solenoid that when triggered resulted in the release of one of the trapdoors. Once released, the trapdoor was supported by a spring-ball plunger that held it in place until the foot made contact with the door. The fall of the trapdoor was denoted by the release of an electromagnetic switch. The switch was normally open, meaning the switch was closed when the doors were aligned and, when the trap fell, breaking the switch, a voltage was supplied.

Surface electromyography (EMG) measurements were collected using a commercial data acquisition system (MP150, Biopac Systems, Goleta, CA). Signals were amplified (TEL100C, Biopac Systems) from disposable, 10-mm pre-gelled Ag-AgCL electrodes (EL503, Biopac Systems). The input impedance of the amplifier was 2 megaohms, with a common mode rejection ratio of 110 dB, and a signal-to-noise ratio of
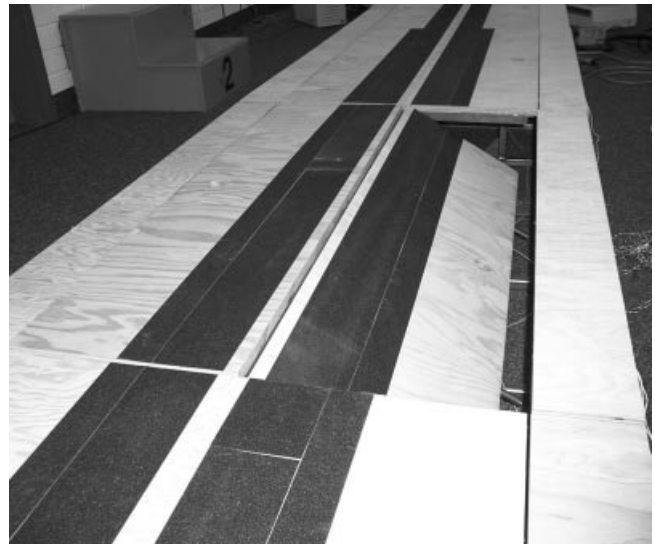

Figure 1. Runway used during inversion perturbation while walking. Eight traps were built into the runway, and each was triggered (12 times) following a randomly generated pattern over 25 trials.

0.75dB. EMG signals were band-pass filtered at 20 and 500 Hz and sampled at 2,000 Hz using the Acknowledge 3.7.3 software package (Biopac Systems).

A AMTI force plate (OR6-7, Watertown, MA) was used to measure the mechanical component of the EMD measurement. Force was sampled at 2,000 Hz.

Testing Procedures

Prior to testing, each subject was shaved (if necessary), debrided, and cleaned with isopropyl alcohol for reception of the EMG electrodes. The electrodes were placed bilaterally, 2 cm center-to-center, and in line with the longitudinal axis of the peroneus longus, approximately 4 cm distal to the fibular head. A ground electrode was placed over the tibial tuberosity.

EMD Procedures

To assess EMD, we used a supramaximal percutaneous electrical square wave (1.0 ms pulse width) stimulus (STMISOC, Biopac Systems) of the common peroneal nerve.[23] The stimulation electrode was placed over the common peroneal nerve as it passes behind the fibular head. Subjects maintained a double leg stance with the test leg on a marked spot on the force plate and the non-test leg off the force plate over a marked spot on the floor. Subjects maintained body posture by placing their hands on a support, positioned at the level of their navel. Subjects were instructed to hold this position, looking straight ahead. The maximal muscle response elicited with a supramaximal stimulus (Mmax) was identified through increases in stimulus intensity until the maximum motor response was identified by EMG inspection. This stimulus intensity was used to elicit contractions for all measurements. Measurements were recorded with a 15-s rest period between stimuli. Lateral ground reaction force (Y) represented the mechanical contribution induced by stimulation.[23,24] The onset times of the Mmax response and lateral ground force were defined as the point where the signal was 3 SD higher than the mean resting activity (from the 150 ms prior to stimulation). The EMD was defined as the time interval between the onset of the peroneus longus EMG activity and the
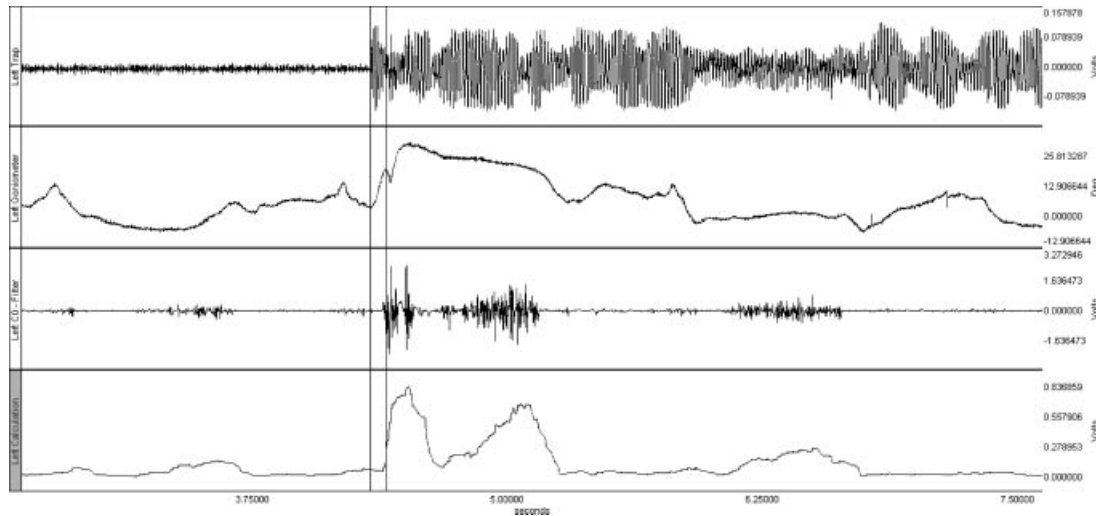
Figure 2. Sample tracing from one of the runway trials showing, switch from the trap (channel 1), data from the electrogoniometer (channel 2), raw PL EMG data (channel 3), and processed PL EMG (channel 4). Onset of perturbation is marked by the first vertical line (left), and onset of PL EMG is marked by the second vertical line (right).

onset of lateral ground reaction force deviation.[23,24] Custom software was used to calculate all onset points and EMD. Six trials were collected for each leg. All EMD measurements were collected prior to all walkway measurements.

Walkway Procedures
A custom walkway was used to collect short latency responses following inversion perturbation. In preparation for the walkway trials, subject were fitted with goggles that impeded their inferior field of view, and with headphones equipped with a built-in metronome set at a cadence of 110 beats/min. Subjects were instructed to walk to the beat of the metronome, to walk along the footpath outlined by the grip tape, and to stop once they reached a sign at the end of the walkway.[29] Data were collected for 25 walking trials, in which 12 of the 25 trials were randomly perturbed by dropping the trapdoors (6 trials for each the right and left side). Trials where subjects' feet were not completely on the footpath during the perturbation, or trials where subjects anticipated the dropping of the trapdoor, were discarded and repeated. Approximately 30 s of rest was allotted between trials.

Peroneal EMG data were processed utilizing custom software. For each trial, data were transformed into linear envelopes (zeroed to baseline, rectified, and bandpass filtered at 10 and 500 Hz). Muscle onset was then defined as the EMG level 3 SD above the mean calculated from the 150 ms prior to the trapdoor falling. Peroneal latency was defined as the time from the drop of the trapdoor to the onset of peroneal EMG activation (Fig. 2).

Statistical Analysis
Peroneal latency and eversion EMD were analyzed by way of mixed-effects ANOVAs. Each ANOVA had two independent factors, limb (affected or unaffected) and group (FAI or control). Limb was modeled as a within-subject factor, while group was modeled as a between-subjects factor. In the control group, the non-dominant leg was analyzed as the "affected" leg, while the dominant leg was analyzed as the "unaffected" leg. Univariate F tests and Sidak's t multiple comparison procedures were used to make post hoc comparisons. The experiment-wise type I error rate for all tests was set at $p \leq 0.05$.

RESULTS
Summary data are presented in Table 1. The mixed effects ANOVAs revealed group × limb interactions for peroneal latency ($F_{1,40} = 16.98$; $p < 0.0001$) and EMD ($F_{1,40} = 28.30$; $p < 0.0001$). Post hoc testing noted significant limb differences for peroneal latency ($p = 0.0001$) and EMD ($p = 0.0002$) in the FAI group, but no between-limb differences for either variable in the control group (Latency, $p = 0.470$; EMD, $p = 0.218$). The affected limb in the FAI group had significantly slower peroneal latency and EMD when compared to the unaffected limb. Further, the affected limb of the FAI group had delayed peroneal latency ($p = 0.045$) and EMD ($p = 0.001$) compared to the matched controls.

Table 1. Summary Data (Mean ± SD) for PL Latency and EMD[a]

Group	PL Latency (ms)		PL EMD (ms)	
	Affected	Unaffected	Affected	Unaffected
FAI	106.6 ± 27.6*,**	74.3 ± 23.3	33.7 ± 14.3*,**	16.6 ± 4.0
Control	84.6 ± 18.6	83.3 ± 15.3	19.5 ± 10.1	21.2 ± 14.0

[a]In the control group, the non-dominant leg was analyzed as the "affected" leg, while the dominant leg was analyzed as the "unaffected" leg.
*Different than the unaffected leg; $p < 0.05$.
**Different than the same leg of the control group; $p < 0.05$.

DISCUSSION

The findings of this study show delayed peroneus longus (PL) latency and EMD when compared to the unaffected leg and a matched control group. Our findings also support the idea that these deficiencies exist during a functional movement (walking), wherein the sensorimotor system changes with various positions and movements. While other investigators have used a different perturbation model, our findings support their reports[18–21] of PL latency deficits in patients with ankle instability. We measured a 26% PL latency deficit in patients with FAI, which falls well within the range of 16%–33% reported by other investigators.[19–21] Our data do not agree with other authors who reported no significant difference in PL latency between healthy and unstable ankles.[30,31] Our EMD data support those of Mora et al.[24] who found deficits in patients with FAI, while Vaes et al.[31] found no alterations in EMD among patients with ankle instability. While there is obvious support for and disparity between our data and those reported in the literature, it should be noted that direct comparisons are cumbersome. Each of these studies varies in their definition of ankle instability, their methods of measurement, and how the data are reduced for analysis. However, there appears to be compelling evidence from this study, as well as others, that FAI is closely related to neuromuscular firing deficits, and these deficits persist during movement.

Given the latencies measured in this and other studies,[19–21] immediate muscle activation was most likely mediated through a short reflex pathway. This reflex could be triggered by joint mechanoreceptors[19,32] as well as muscle spindle fibers in the PL. The delayed PL latencies measured in this study could be due to a number of factors. Freeman[33] argued that patients suffer partial deafferentation following joint injury, reducing reflexive activity that would be initiated by joint mechanoreceptors. Konradsen and Ravn[19] suggested that slower conducting nerve fibers (type II) may substitute for the faster conducting (type I) fibers, reducing reflexive activity from Ia interneurons. While these ideas may play some role in altering short latency responses, the key is likely gamma motoneuron activity and muscle spindle sensitivity. Gamma activation plays a large role in neuromuscular firing following injury and a primary role in short latency responses.[7] A lack of proprioceptive information from partial deafferentation and replacement sensorimotor strategies could chronically suppress gamma activation and desensitize the muscle spindle.[34] Khin-Myo-Hla et al.[34] suggested that mechanoreceptor and/or nociceptor irritability at the injury site may also suppress gamma activation and spindle sensitivity. Therefore, Freeman's[33] hypothesis that suppressed joint mechanoreceptor activation and/or deafferentation is responsible for delayed reflex activation is only partially supported. Altered joint mechanoreceptor activation likely plays a role in changing gamma motoneuron activity, and therefore muscle spindle sensitivity. Therefore, the key to short-term stability seems to be centered in the sensitivity of the muscle spindle.

EMD is an important measurement that accounts for the time necessary to develop tension following activation of the muscle. This delay may be attributed to propagation of the action potential along the excitable muscle membranes, $Ca++$ release into the sarcoplasm and subsequent binding to active sites, the formation of cross-bridges, and tension to the series elastic component (SEC).[35] Increases or decreases in EMD may be attributed to changes in the excitation-contraction mechanism as well as the SEC.[24,35] Mora et al.[24] argued that a change in EMD is an indirect indication of muscle stiffness and tone, and therefore an important factor in assessing joint stability. Like latencies previously discussed, EMD would be affected by changes in muscle spindle sensitivity, as decreased sensitivity and the resultant stiffness change would increase the amount of slack taken up by cross-bridging and, therefore, increase the amount of time necessary to produce force or tension.[36] Our latency and EMD data are consistent with the idea that subjects with FAI may have gamma activation deficiencies, reducing muscle spindle sensitivity and resting muscle stiffness. This factor could play a significant role in the recurrence of ankle injury in patients with chronic ankle instability.

This study differs from several others that have measured peroneal latencies following sudden inversion perturbation. An inversion platform has frequently been used to model the kinematics of ankle injury.[19,30,37–41] This model typically utilizes a small platform with trapdoors that fall to a restricted position (approximately 30°). The participant stands quietly on the platform until one of the trapdoors is released from under a foot. This model permits the evaluation of muscle activity at the ankle and lower leg in response to the sudden ankle inversion. However, the validity and generalizability of this model is limited since it is restricted to sudden inversion of an even and constant load in a static stance position, with the joint primarily relying on stability from alignment of articular surfaces.[42] The standing model also does not consider the functional anterior/posterior or medial/lateral motion of the patient, nor does it consider ankle joint muscle activation during movement. During gait, many of the ankle muscles are activated prior to and during the early stance phase in order to help stabilize the ankle and foot.[43] Nakazawa et al.[43] reported that spindle sensitivity is increased during this phase of gait. These changes to the nervous system during functional movement should be considered as part of the overall picture when generalizing inversion perturbation data to a physically active injured population. Considering these points, it is necessary to note that our measures of EMD were recorded while the subject was in a static, standing position. A standing position will consider sensory information that is available from loaded joints. However, as stated above, functional changes during movement will not be considered due to a static position.

The consequences of PL latency and EMD deficits in patients with FAI are not yet completely understood. While delayed latencies and EMD certainly leave the joint susceptible to injury for a longer period of time, many injurious loads to joints occur prior to a protective response.[44] However, ankle inversion loads that occur during many activities of daily living (walking, stair climbing, etc.) may allow for normal neuromuscular responses to protect the joint. In these cases, the slower latencies and EMD may place the joint at an increased risk of reinjury. However, these data may be indicative of a larger problem. If gamma activation is suppressed in patients with ankle instability, then lack of activation or tone in many or all of the muscles surrounding the injured joint may lead to a decrease in joint stiffness, potentially contributing to instability. The joint, in this circumstance, would be more susceptible to injury without appropriate levels of dynamic joint stability through muscle contraction. As suggested by Hertel,[45] the key to treating FAI may be to find an intervention strategy that will enhance gamma activation and subsequent spindle activation. Future work should focus on common rehabilitation and other intervention and their effects on peroneal deficits associated with ankle instability.

FAI subjects in this study were classified as such through use of the Functional Ankle Instability Questionnaire[27] and the Ankle Instability Instrument.[28] While these instruments have been widely used to identify those with ankle instability, they do not provide specific qualitative information on function. Future work with this population should consider instruments such as the Foot and Ankle Disability Index[46] and the Foot and Ankle Activity Measure.[47] Further, it should be mentioned that subjects with mechanical instability were excluded from the current study. While our intent was to examine functional deficiencies in subjects with ankle instability, several authors[48–50] have suggested that ankle instability is a multifactorial problem, consisting of mechanical and functional deficits. Future work should consider how stability might be affected by mechanical deficiencies as well as functional.

In conclusion, patients with FAI display delayed PL latency and EMD following inversion perturbation during walking. Peroneal latency and EMD deficits could contribute to recurrence of ankle injury in FAI subjects. How these deficits are associated with the chronic symptoms associated with FAI remains unclear, but gamma activation and subsequent muscle spindle sensitivity likely play a role.

ACKNOWLEDGMENTS

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