

Effect of Peripheral Afferent Alteration of the Lateral Ankle Ligaments on Dynamic Stability*

Joseph B. Myers,†‡ PhD, ATC, Bryan L. Riemann,§ PhD, ATC, Ji-Hye Hwang,|| MD, PhD, Freddie H. Fu,† MD, and Scott M. Lephart,† PhD, ATC

From the †Neuromuscular Research Laboratory, Department of Orthopaedic Surgery, University of Pittsburgh, Pittsburgh, Pennsylvania, the §Graduate Athletic Training Program, Georgia Southern University, Statesboro, Georgia, and the ||Department of Physical Medicine and Rehabilitation, Samsung Medical Center, College of Medicine, Sungkyunkwan University, Seoul, Korea

Background: The sensorimotor influence of the lateral ankle ligaments in muscle activation is unclear.

Hypothesis: The lateral ankle ligaments have significant sensorimotor influence on muscle activation.

Study Design: Controlled laboratory study.

Methods: Muscle-firing characteristics in response to a high-speed inversion perturbation and during gait were assessed in 13 normal subjects. Solutions (1.5% lidocaine or a placebo of saline) were injected bilaterally into the anterior talofibular and calcaneofibular ligaments (1.5 ml per ligament) to alter peripheral afferent influence. Subjects were again tested with the same protocol.

Results: The protective response of the anterior tibialis and peroneal muscles during inversion perturbation and mean muscle activation amplitude decreased during running after both injections. After injection, no significant differences were seen for muscle reflex latencies, maximum amplitude, time to maximum amplitude during inversion perturbation, or mean amplitude during walking.

Conclusion: The lateral ankle ligaments have a sensorimotor influence on muscle activation.

Clinical Relevance: Induced edema from the injected solutions may have altered the sensorimotor influence of the lateral ankle ligaments, thereby inhibiting the dynamic ankle stabilizers. This finding suggests that dynamic stability may be compromised because of swelling after joint injury.

© 2003 American Orthopaedic Society for Sports Medicine

Much has been written about the mechanical role of the ligaments that surround the ankle joint in providing ankle joint stability.^{22,30,34,35,56} The anterior talofibular ligament limits talar tilt throughout sagittal plane motion, especially with the joint in a position of plantar flexion.^{30,56} The calcaneofibular ligament appears to limit talar tilting in dorsiflexion as well as in talocalcaneal adduction.^{22,34,35}

In addition, embedded within these ligaments are mechanoreceptors believed to be responsible for providing a proprioceptive role in maintaining ankle joint stability.¹⁵ Proprioception, which results from the afferent neural input originating from mechanoreceptors about the joint, contributes to dynamic joint stability mechanisms and coordinated motor patterns.⁴² Michelson and Hutchins⁵⁰ demonstrated histologically that Ruffini-type, Pacinian corpuscle type, and Golgi tendon organ receptors are dispersed throughout the ankle ligaments, with Pacinian and Golgi tendon-like organs making up a majority of the mechanoreceptors present. Johansson and colleagues²⁷ described how ligamentous mechanoreceptors like these play a role in providing joint stability that is equally as important as the purely mechanical role of the ligaments. By use of traction forces well below those associated with

* Presented at the interim meeting of the AOSSM, Dallas, Texas, February 2002.

‡ Address correspondence and reprint requests to Joseph B. Myers, PhD, ATC, Neuromuscular Research Laboratory, UPMC Center for Sports Medicine, 3200 South Water Street, Pittsburgh, PA 15213.

No author or related institution has received any financial benefit from research in this study.

tissue damage and nociception, stimulation of the ligamentous mechanoreceptors has been demonstrated to have potent effects on sensitivity and activation of the gamma motor neurons associated with the muscle spindle.^{27,28,51,57,63} These potent effects provide significant contributions to muscle stiffness, motor control, and reflexive characteristics.^{28,29} Except for temporal assessment of reflex latencies,^{8,13,24,31,33,37-39,44,47} no research has specifically measured the contribution of ankle ligament mechanoreceptors to muscle-firing characteristics associated with joint stability. It is feasible that the influence of these mechanoreceptors on muscle-firing characteristics may have nontemporal implications, such as amplitude of muscle activation, which may be overlooked in temporal (latency) assessments.

The peripheral afferent contribution of ligamentous mechanoreceptors on neuromuscular mechanisms, such as muscle-firing characteristics, has been measured by using anesthesia to block them. Feuerbach et al.¹⁴ injected lateral ankle ligaments with an anesthetic to block peripheral afferent influence, thereby determining the role that ligaments play in supplying joint proprioception. Both Hertel et al.²⁰ and DeCarlo and Talbot⁹ injected an anesthetic into the lateral ankle ligaments to ascertain the role of peripheral afferent information from these ligaments in postural control tasks. Thus far, two groups have examined muscle reflex latencies of dynamic restraints after injection into lateral ankle ligaments; both failed to reveal significant latency changes in healthy subjects.^{33,39} However, because of the influence of ligamentous mechanoreceptors on gamma reflex loops,^{27,28,51,57,63} it is plausible that they mediate other characteristics of the response; to date, no research has considered this possibility. Furthermore, we are unaware of any research that has examined the influence of the lateral ankle ligaments on muscle-firing characteristics during dynamic tasks such as walking and running. The purpose of this study was to evaluate the role of peripheral afferent information from the lateral ankle ligaments in spatial and temporal muscle activation characteristics during an inversion perturbation and during dynamic gait tasks such as walking and running.

METHODS

Thirteen subjects (seven men, 24.7 ± 4.2 years of age with a height of 180.1 ± 6.1 cm and mass of 78.8 ± 6.8 kg; and six women, 22.3 ± 0.95 years of age with a height of 162.6 ± 6.8 cm and mass of 62.3 ± 5.2 kg) participated in this study. All subjects were right-leg dominant (operationally defined as the foot the subject would use to kick a ball) and had no significant history of lower extremity injury. Informed consent was obtained before participation, in accordance with the requirements of the University of Pittsburgh Institutional Review Board. Each subject attended two testing sessions at least 48 hours apart. A pretest-posttest research design was used during each session.

Instrumentation

The ankle inversion perturbation device is a specially designed platform that allows the ankle joint to drop from a neutral position into 30° of inversion while the subject is standing (Fig. 1). Both right and left plates of the platform are connected to two tension springs that pull each plate down into 30° of inversion at an average angular velocity of 440 deg/sec . The plates are held level by spring plungers connected to two independent levels that release each plate independently when triggered, inducing the inversion perturbation. Each plate is fitted with a 2g accelerometer (Newh Ghant Technologies, LaGrangeville, New York) to signal onset of platform drop. The inversion plates are positioned over a Bertec forceplate (Model #K80801, Type 4060-10, Bertec Corporation, Columbus, Ohio) to ensure equal weight distribution between limbs at the time of perturbation. Pilot testing of the device to establish reliability for measurement of muscle-firing characteristics was performed on 15 subjects and yielded intraclass correlation coefficients (2,1) ranging from 0.71 to 0.76, with standard error of measurement of 6.1 to 8.2 msec calculated according to Denegar and Bell¹¹ and Shrout and Fleiss.⁶⁰

Electromyographic Instrumentation and Preparation

Electromyographic data were collected with the Noraxon Telemyo (Noraxon, Scottsdale, Arizona) EMG system, a

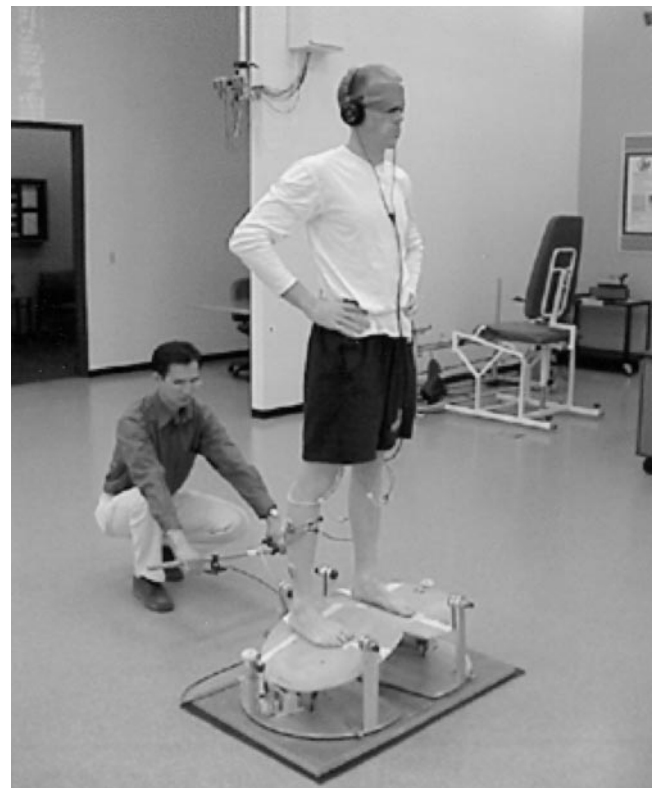


Figure 1. A subject positioned on the ankle inversion perturbation device.

frequency modulated (FM) telemetry system. Electromyographic signals collected from the electrodes were passed through a single-ended amplifier (gain 500) to an eight-channel FM transmitter. A receiver unit obtained the telemetry signals from the transmitter, where the receiver amplified (gain 500) and filtered (15 Hz low-pass, 500 Hz high-pass Butterworth filter, common mode rejection ratio of 130 db) the signals. Signals from the EMG receiver, force plate, and acceleration were converted from analog to digital data via a PCM16S/12 (16-channel, 12-bit) analog-to-digital board (ComputerBoards, Inc., Middleboro, Massachusetts) at a rate of 1000 Hz. The digital data were collected and stored with Myoresearch 2.02 (Noraxon) on a personal computer for later data reduction.

Silver-silver chloride surface electrodes (Medicotest, Inc., Rolling Meadows, Illinois) were used for measurement of muscle activity. Skin preparation to lower impedance included shaving any hair present, mild abrasion with a low-abrasive emery board, and wiping the area with 70% isopropyl alcohol. A ground electrode was placed just distal to the tibial tuberosity, and two surface electrodes were placed side by side and perpendicular to the orientation of the anterior tibialis, peroneus longus, and peroneus brevis muscle fibers, with 2 cm separating their centers.^{6,10} The same investigator applied electrodes on all subjects during all sessions to control for intertester variability. The peroneal muscles were chosen because of their evertor function, reported activity during the stance phase of gait when the ankle is at risk of injury, and for the role they play in dynamic balance during gait tasks.⁴⁶ The anterior tibialis muscle was chosen because of its function as an antagonist to the peroneal muscles.⁴⁶ Correct position of all electrodes was confirmed through isolated manual muscle tests of each muscle.⁶ Komi and Buskirk³⁶ established the reliability of surface electrode EMG as 0.88 to 0.91 intraclass correlation coefficient within sessions and 0.64 to 0.73 intraclass correlation coefficient between testing sessions.

Stance and swing phases of the walking and running tasks were identified with the NorSwitch (Noraxon) foot switch system, which uses pressure-sensitive resistors that output consistent voltage to signal gait events. The pressure-sensitive foot switches were secured to the calcaneus and to the first ray on the plantar surface of the foot.

Testing Procedures

Once informed consent and EMG preparation was completed, each subject was oriented to the testing procedures. For inversion perturbation testing, all subjects were asked to stand barefoot on the platform with the second ray of both feet aligned with the axis of rotation of the plates and with their hands placed on their hips. Subjects were asked to wear a blindfold and a headphone set emitting white noise to negate visual and auditory cues. Before the start of the inversion trials, the subjects were asked to maintain a quiet stance on the platform for a period of 12 seconds. During that time, center of pressure and EMG activity were assessed. Immediately after

the quiet stance, 12 randomized pretest inversion trials (6 trials per limb) were performed. Subjects were unaware of which platform would drop for each trial. These pretest trials served as control trials for comparison of muscle activity before and after injection of the solution.

During the dynamic gait activities, subjects were asked to walk and run barefoot on a treadmill (Biodex Treadmill, Biodex Medical, Shirley, New York) (Fig. 2). Treadmill speeds were standardized at 3.2 km/hr and 7.6 km/hr for walking and running, respectively. Subjects were instructed to walk and run using their normal gait mechanics. The subjects were not allowed to touch the handrails of the treadmill to eliminate the influence that external support has on muscle-firing patterns during gait.⁴⁶ Subjects were given ample time for familiarization with the treadmill and speed of movement before trial recording. Once the standardized speed was obtained and the subject indicated that a fluid gait pattern was achieved, EMG and foot switch recording was initiated. Recording of the EMG and foot switch signals continued until at least 20 strides were completed, allowing for analysis of 12 strides for all trials. A review of the literature indicated that at least six strides are needed to obtain a representative EMG profile during gait activity.^{4,59,73}

Immediately after pretesting, a physician injected either 1% preservative-free lidocaine HCl (Astra USA, Inc., Westborough, Massachusetts) or a placebo solution (preservative-free 0.9% sodium chloride, Abbott Laboratories, North Chicago, Illinois) with a 25-gauge 5/8-inch syringe into both the anterior talofibular and calcaneofibular ligament regions (1.5 ml for each site) of each ankle (Fig. 3).^{14,20} Subjects, who were blinded as to which solution was being injected, were asked to remain inactive for 20 minutes after injection to allow for induction of the anesthetic effects. Subjects were again tested with identical inversion perturbation and treadmill protocols. Testing protocols were alternated within testing sessions. Subjects returned for a second session at least 48 hours after the initial session and were tested with identical procedures but were injected with the other solution.

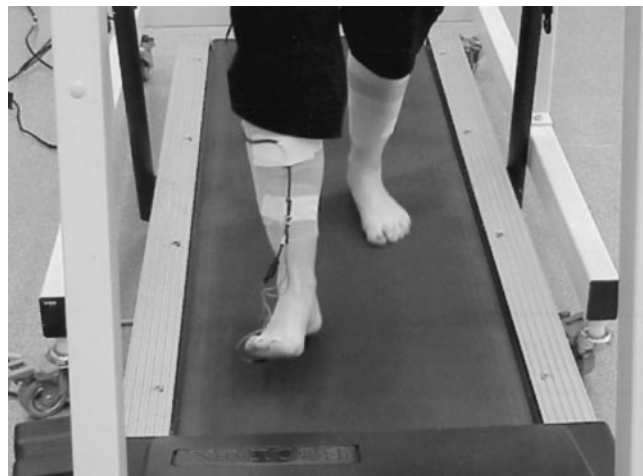


Figure 2. Assessment of muscle activity during dynamic activities on the treadmill.

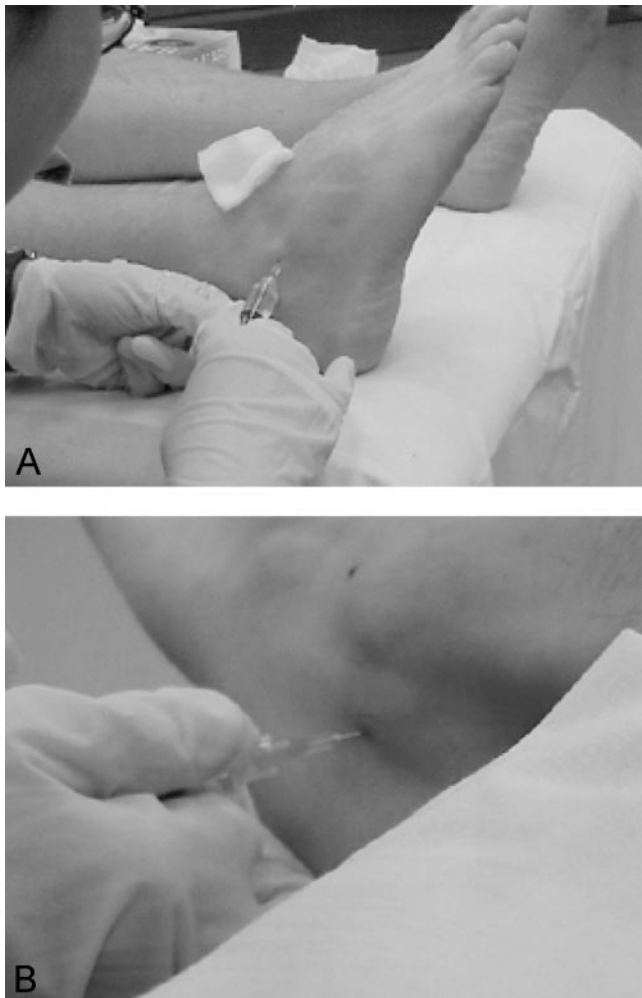


Figure 3. Injection into the anterior talofibular (A) and calcaneofibular (B) ligaments.

Data Reduction

Inversion Perturbation. Because limb dominance has been demonstrated to cause differences between limbs, only the dominant limb was analyzed.^{13,24} This decision was justified because the purpose of the investigation was not to measure bilateral differences but to measure changes after peripheral afferent alteration, which can be accomplished by making comparisons within the same limb. Subjects were unaware that only the data from the dominant limb would be considered. Accelerometer, EMG, and force plate data were exported and conditioned with LabVIEW 5.1 customized software (National Instruments, Austin, Texas). Force and moment data obtained by the force plate and accelerometer were filtered with a 10-Hz cutoff, fourth-order zero-phase lag Butterworth filter.⁷⁰ Trial EMG data were rectified, filtered (20 to 500 Hz bandpass fourth-order zero-phase lag Butterworth filter), and normalized to the mean amplitude of a rectified filtered linear envelope for each respective muscle during the quiet stance trial.⁶⁷

Perturbation onset and muscle-firing characteristics were calculated with customized software written in Visual Basic for Applications (Microsoft, Redmond, Washington). Perturbation onset was determined by using the acceleration data. Muscle activation onset was ascertained by using a similar algorithm of five standard deviations above baseline activity 150 msec before perturbation.²⁴ This method of determining onset has been proven reliable.²⁴ The customized software allowed user interaction so that a reviewer could subjectively accept or reject data that contained nonphysiologic activity, similar to the method described by Di Fabio¹² and Lynch et al.⁴⁷

The customized software calculated the *maximum amplitude*, defined as the maximum amplitude of EMG activity within 100 msec after muscle onset. *Time to maximum amplitude* was defined as the time from perturbation onset to maximum amplitude. The *protective response*, the mean amplitude of the window from muscle activity onset to 100 msec after onset, was calculated.⁴⁴ Figure 4 illustrates the measured muscle-firing characteristics.

Center-of-pressure standard deviation in the mediolateral direction was calculated from the force and moment data and compared with the center of pressure for both the quiet stance and the window from 150 μ sec before perturbation to perturbation onset to avoid unequal limb loading due to subject anticipation. Any deviation in the center of pressure three times that during quiet stance indicated that unequal weight distribution occurred, invalidating that trial. Invalid trials were discarded.

Muscle Activity during Gait. Raw EMG signals for all muscles were both rectified and second-order low-pass Butterworth filtered (6 Hz cutoff).⁷¹ Stance and swing phases of the walking and running tasks were visually identified by the foot switch signals within the Myoresearch software. Twelve complete gait cycles were estab-

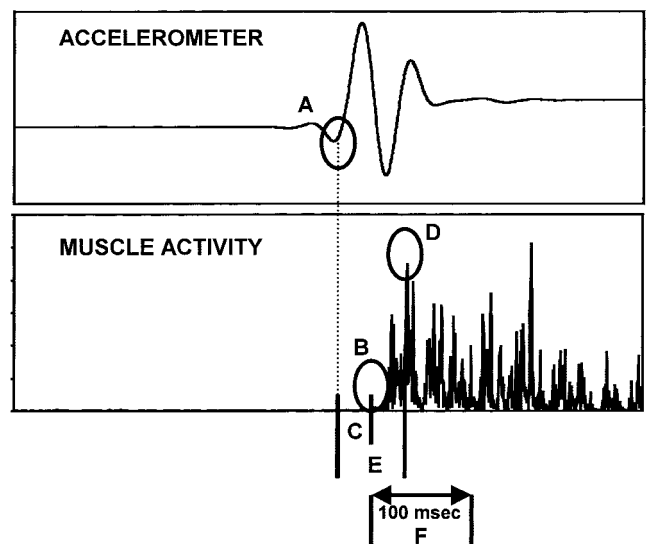


Figure 4. Muscle reflex characteristics. A, ankle perturbation onset; B, muscle reflex onset; C, muscle reflex latency; D, maximum amplitude; E, time to maximum amplitude; and F, muscle protective response.

lished for analysis and were normalized to both time and mean amplitude for the gait cycle for establishment of a linear envelope.⁷³ The mean amplitude for both stance and swing phases was used for statistical analysis.

Statistical Analysis

Statistical analysis for each dependent variable consisted of separate (two within and one between) repeated-measures analysis of variance models. The within factors were test (pretest versus posttest) and muscle (anterior tibialis, peroneus longus, and peroneus brevis), and the between factor was treatment (placebo versus anesthetic). Although each subject received both anesthesia and placebo treatments, the weak reliability of between-session comparisons for surface EMG results demonstrated in the literature warranted analyzing the two treatments as a between factor rather than a within factor in the analysis of variance model.³⁶ A Tukey post hoc analysis was used for pairwise comparisons of significant results ($P < 0.05$)

RESULTS

Inversion Perturbation Reflex Characteristics

The descriptive statistics for muscle latency, maximum amplitude, time to maximum amplitude, and protective muscle response appear in Tables 1 to 4, respectively. Statistical analysis revealed no significant difference within or between factors for muscle latency, maximum amplitude, and time to maximum amplitude. A test main effect ($F[1,24] = 11.10$, $P = 0.03$) did manifest for the protective muscle response. These results indicated suppressed muscle activity after injection of either solution into the lateral ankle ligaments (Fig. 5).

Dynamic Activity Muscle-Firing Characteristics

The descriptive statistics for mean amplitudes of each muscle during both the stance and swing phase while walking and running appear in Tables 5 and 6. For analysis of the gait data, separate repeated-measures analysis of variance models demonstrated decreased mean amplitude of the per-

TABLE 1
Muscle Reflex Latency in Milliseconds (Mean \pm SD)

Muscle	Anesthesia		Placebo	
	Pretest	Posttest	Pretest	Posttest
Anterior tibialis	87.92 \pm 15.21	94.26 \pm 15.71	98.20 \pm 19.66	91.59 \pm 15.05
Peroneus longus	88.92 \pm 9.95	89.94 \pm 7.38	89.61 \pm 15.40	90.04 \pm 15.43
Peroneus brevis	81.38 \pm 8.79	83.39 \pm 10.06	84.78 \pm 14.20	85.36 \pm 12.09

TABLE 2
Maximum Muscle Amplitude^a (Mean \pm SD)

Muscle	Anesthesia		Placebo	
	Pretest	Posttest	Pretest	Posttest
Anterior tibialis	49.29 \pm 43.59	34.54 \pm 31.55	48.04 \pm 46.17	45.54 \pm 34.26
Peroneus longus	36.00 \pm 23.47	41.53 \pm 19.22	49.66 \pm 37.04	47.18 \pm 34.28
Peroneus brevis	43.19 \pm 21.70	43.77 \pm 17.37	41.62 \pm 15.29	41.73 \pm 18.09

^a Muscle amplitudes are normalized to quiet stance and listed as a percentage of quiet stance EMG amplitude.

TABLE 3
Time to Maximum Muscle Amplitude in Milliseconds (Mean \pm SD)

Muscle	Anesthesia		Placebo	
	Pretest	Posttest	Pretest	Posttest
Anterior tibialis	118.94 \pm 22.7	117.05 \pm 23.3	126.39 \pm 24.2	118.71 \pm 19.7
Peroneus longus	105.21 \pm 14.5	107.02 \pm 14.0	109.34 \pm 20.1	107.41 \pm 15.3
Peroneus brevis	111.37 \pm 14.9	113.97 \pm 19.5	115.10 \pm 22.7	113.08 \pm 16.2

TABLE 4
Normalized Muscle Protective Response^a (Mean \pm SD)

Muscle	Anesthesia		Placebo	
	Pretest	Posttest	Pretest	Posttest
Anterior tibialis	31.99 \pm 18.31	25.64 \pm 20.36	31.16 \pm 22.53	28.83 \pm 21.72
Peroneus longus	29.56 \pm 17.60	25.24 \pm 13.85	24.87 \pm 13.15	21.49 \pm 11.67
Peroneus brevis	28.80 \pm 17.13	26.14 \pm 17.62	24.14 \pm 13.39	23.18 \pm 13.96

^a Muscle amplitudes are normalized to quiet stance and listed as a percentage of quiet stance EMG amplitude.

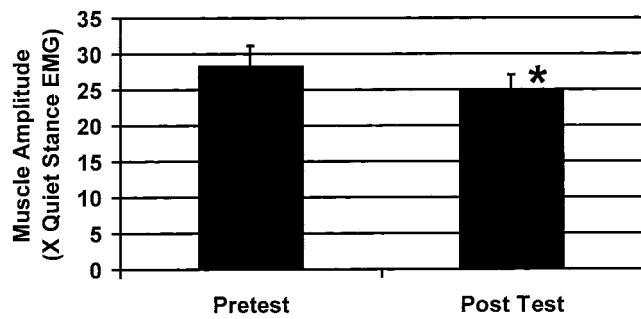


Figure 5. Muscle reflex protective response. *, significantly different from pretest mean amplitude.

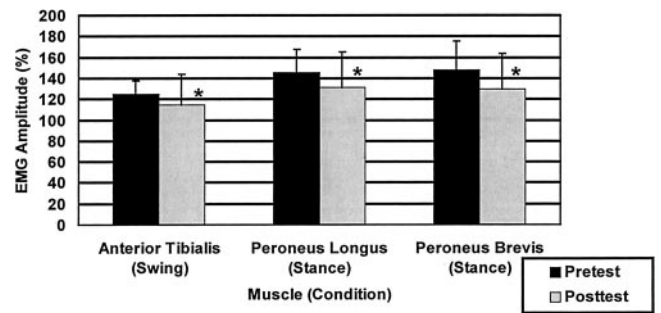


Figure 6. Mean EMG amplitude during running. *, significantly different from pretest amplitudes.

TABLE 5
Muscle Amplitude^a during Treadmill Walking (Mean ± SD)

Muscle	Anesthesia		Placebo	
	Pretest	Posttest	Pretest	Posttest
Stance phase				
Anterior tibialis	79.66 ± 10.42	76.67 ± 21.19	70.81 ± 11.15	72.70 ± 18.90
Peroneus longus	115.77 ± 10.42	103.65 ± 23.00	115.29 ± 15.20	115.02 ± 42.04
Peroneus brevis	109.57 ± 10.95	100.11 ± 17.99	114.52 ± 12.60	107.40 ± 35.16
Swing phase				
Anterior tibialis	148.15 ± 20.94	135.46 ± 31.43	154.69 ± 18.51	154.94 ± 34.01
Peroneus longus	59.78 ± 26.31	53.95 ± 33.23	72.54 ± 26.40	62.80 ± 31.39
Peroneus brevis	61.01 ± 18.00	82.02 ± 49.48	72.58 ± 22.85	68.93 ± 24.86

^a Values expressed as percentage of the mean activity over the entire gait cycle.

TABLE 6
Muscle Amplitude^a during Treadmill Running (Mean ± SD)

Muscle	Anesthesia		Placebo	
	Pretest	Posttest	Pretest	Posttest
Stance phase				
Anterior tibialis	78.79 ± 10.64	67.97 ± 19.48	67.41 ± 15.66	71.85 ± 23.44
Peroneus longus	150.81 ± 20.47	129.01 ± 27.83	139.65 ± 24.30	132.93 ± 40.73
Peroneus brevis	142.33 ± 22.92	127.45 ± 42.80	153.89 ± 31.65	131.34 ± 24.59
Swing phase				
Anterior tibialis	120.30 ± 10.61	108.29 ± 32.71	128.84 ± 13.54	121.06 ± 24.10
Peroneus longus	51.92 ± 16.41	48.70 ± 23.57	63.69 ± 22.06	53.20 ± 18.72
Peroneus brevis	61.01 ± 18.00	57.84 ± 28.86	55.96 ± 17.28	53.44 ± 16.80

^a Values expressed as percentage of the mean activity over the entire gait cycle.

oneus longus muscle during the swing phase of walking ($F[1,24] = 6.10, P = 0.021$), the anterior tibialis muscle during the swing phase of running ($F[1,24] = 5.33, P = 0.03$), and the peroneus longus ($F[1,24] = 8.07, P = 0.009$) and peroneus brevis muscles ($F[1,24] = 10.62, P = 0.003$) during the stance phase of running between pretest and posttest (Fig. 6). No significant differences existed between the lidocaine and placebo solution conditions.

DISCUSSION

The mechanical role of the anterior talofibular and calcaneofibular ligaments on ankle joint stability has been established.^{22,30,34,35,56} It is believed that lateral ankle ligament injury results in not only mechanical instability^{40,58} but also ligamentous deafferentation, contributing to instability.^{15,38,66} Unfortunately, the sensorimotor influence of the lateral ankle ligaments is not completely

understood. The purpose of this study was to gain a better understanding of their sensorimotor role through alteration of the peripheral afferent influence that these ligaments may have on muscle-firing characteristics by using a previously described lidocaine model.^{9,14,20,33,39} Activities that resemble the mechanism of lateral ankle ligament injuries (inversion perturbation) and the type of activities that are performed when these injuries are sustained (dynamic activities like running) were chosen to assess these sensorimotor influences.

Inversion Perturbation Muscle Reflex Characteristics

It was predicted that peripheral afferent alteration of the lateral ankle ligaments with lidocaine would influence the muscle reflex characteristics of the dynamic stabilizers of the ankle joint. Results from this study indicated that this was not the case. Similar to previous reports,^{33,39} periph-

eral afferent alteration with a lidocaine model had no significant effect on muscle reflex latencies. Unlike these previous investigations, however, this study also included measurement of reflex amplitude characteristics (maximum amplitude and protective mechanism) after peripheral afferent alteration. Again, however, lidocaine injection had no significantly different effect compared with that of the placebo solution.

A test main effect was present for the protective mechanism measured. This result demonstrated that injection of a solution (independent of which solution was injected) caused decreased muscle-firing amplitude over a 100-msec period after muscle onset (Fig. 5). Because the results were independent of which solution was injected, a mechanism other than anesthesia was most likely responsible. Injection of solution into the ligament region possibly induced artificial swelling similar to that seen with injury. Unfortunately, we are unaware of any published data assessing the effects of edema and joint effusion on muscle-firing characteristics at the ankle joint. The effects of joint effusion, whether through joint injury or injection of saline solution, on muscle-firing characteristics have been reported at the knee, however. Knee joint effusion has been shown to have potent inhibitory effects on motoneuron pool recruitment, strength, and muscle activity of the quadriceps muscle.^{23,25,32,64,65} As with reported results in the knee, injection of fluid into the ankle joint may inhibit the musculature that surrounds the ankle.

The question exists as to why both the lidocaine and the placebo saline solution would have an inhibitory effect on muscle-firing characteristics. Spencer et al.⁶⁴ demonstrated that injection of saline solution into the knee joint had inhibitory effects on the quadriceps muscle, but the addition of lidocaine to the knee joint curbed the quadriceps muscle inhibitory effects. In contrast, Agostinucci¹ reported no difference in soleus muscle motoneuron excitability between anesthetic and placebo conditions. Any suspected differences between the lidocaine and placebo conditions, like those in the knee, may be less pronounced in the ankle because type I Ruffini receptors are most likely responsible for the inhibitory response associated with joint pressure,⁶⁴ and there is a lack of Ruffini receptors compared with type II and type III receptors in the ankle ligaments and joint capsule.^{50,72} Another causative factor for suppressed muscle activity for both conditions might have been the insertion of the needle, but data presented by Jensen and Graf²⁶ demonstrated that the insertion of a catheter had no effect on quadriceps muscle inhibition, as measured by isokinetic strength. More research is needed to examine the influence of edema on reflex muscle-firing characteristics about the ankle joint as well as the role that anesthesia plays in affecting these characteristics.

Dynamic Gait Muscle-Firing Characteristics

Similar results were seen from the analysis of muscle activity during the walking and running tasks. No differences between solutions existed within muscles before and after injection of solution during the stance or swing

phases of both walking and running activities. Test main effects were also present, indicating decreased muscle amplitude after injection of solution (independent of which solution was injected). During running trials, the mean amplitude of peroneal muscles decreased during the stance phase of running and the anterior tibialis muscle mean amplitude was depressed during the swing phase (Fig. 6). Peroneus longus muscle activity also decreased during the swing phase of walking.

Comparison of the dynamic activity data was difficult, given the lack of published data examining the influential role that lateral ankle ligaments play on muscle-firing characteristics during dynamic gait activities. Again, some references can be made to the research about the knee. The results indicate decreased muscle activity during gait after injection of solution. The existence of some induced edema may have influenced the muscle activity. Torry et al.⁶⁵ demonstrated quadriceps muscle activity from artificially induced knee joint effusion during gait activities that contributed to a quadriceps muscle avoidance gait pattern. Similar to the suppressed muscle activity at the knee resulting from induced joint effusion, ankle muscle-firing characteristics may have been altered with the induction of solution into the lateral ankle ligament, inducing edema accumulation similar to that with injury.

Clinical Significance

Interpretation of these results suggests that the injection of solution (independent of which solution was injected) resulted in suppressed muscle activity both during inversion perturbation and dynamic activities. The accumulation of fluid within the lateral ankle, similar to the edema formation that exists with ankle injury, would suggest that not only is mechanical stability compromised because of the ligamentous trauma, but dynamic stability mechanisms may also be affected. The suppressed activity may compromise dynamic stability during the weightbearing position during dynamic activities when the ankle is vulnerable to injury.¹⁶

The consequence of this suppressed activity by the peroneal muscles is the decreased intrinsic stiffness that exists at the ankle joint. Intrinsic stiffness is obtained from mechanical properties of both muscle and passive components about the joint.^{17,53} Intrinsic stiffness is strongly influenced by the level of muscle contraction present.⁶⁹ As muscle contraction increases, stiffness also increases.^{7,45,48,52,54,68,74} Muscle contraction creates stable crossbridges that resist stretch¹⁸ while assisting with the storage of muscle energy.⁵ Intrinsic stiffness provides the first line of defense for joint stability when force is applied to the joint.^{2,3,7,49,61,62} Intrinsic stiffness provides an immediate and substantial response to perturbation.⁴³ Therefore, when some type of injury inversion mechanism occurs, the dynamic restraints system will be less likely to prevent the perturbing force from injuring the ankle joint when muscle activity is suppressed.

The suppressed activity of the ankle dynamic stabilizers during inversion perturbation is also problematic. When a destabilizing event like an inversion mechanism does oc-

cur, the magnitude of the muscular response, as measured by the 100-msec protective response,⁴⁴ to protect the ankle may also compromise joint stability. Some authors suggest that joint injury, such as that associated with an inversion mechanism, happens too fast for the reflex activity and accompanying force production to stiffen the joint to prevent the injurious mechanisms.^{19,41,49,55,61,62} This hypothesis suggests that reflexive activity plays less of a role than does the intrinsic stiffness associated with joint stability. More research is needed on the role that reflexive activity plays in providing joint stability.

Because the injection of either solution elicited a change in muscle activity, rather than lidocaine as compared with placebo, as hypothesized, more research is needed to determine whether the resulting edema had an effect on muscle-firing characteristics during both the dynamic gait and inversion perturbation activities. In addition, in vivo models are needed in which the proprioceptive role of ankle ligaments in joint stability can be examined, given that joint deafferentation is often described as a contributor to functional instability and "giving way" episodes.^{15,21,38,66} It may be that the proprioceptive influence of the ankle ligaments contributes more to joint position awareness and postural control capabilities rather than to muscle-firing patterns.

CONCLUSIONS

The results of this study failed to demonstrate differences in muscle-firing characteristic between lidocaine and placebo treatment for both dynamic gait activities and during inversion perturbation. The results do indicate that the lateral ankle ligaments play a sensorimotor role as evidenced by the muscle suppression that manifested after the solution injection. Suppressed muscle activity was demonstrated by decreased muscle-firing amplitude during the stance and swing phases of gait activities and the reflexive response of the ankle dynamic stabilizers. Clinically, this result suggests that dynamic stability may be compromised due to swelling after joint injury or injection of fluid.

REFERENCES

- Agostinucci J: The effects of topical anesthetics on skin sensation and soleus motoneuron reflex excitability. *Arch Phys Med Rehabil* 75: 1233-1240, 1994
- Akazawa K, Aldridge JW, Steeves JD, et al: Modulation of stretch reflexes during locomotion in the mesencephalic cat. *J Physiol* 329: 553-567, 1982
- Akazawa K, Milner TE, Stein RB: Modulation of reflex EMG and stiffness in response to stretch of human finger muscle. *J Neurophysiol* 49: 16-27, 1983
- Arsenault AB, Winter DA, Marteniuk RG: Is there a "normal" profile of EMG activity in gait? *Med Biol Eng Comput* 24: 337-343, 1986
- Asmussen S, Bonde-Petersen F: Storage of elastic energy in skeletal muscles in man. *Acta Physiol Scand* 91: 385-392, 1974
- Basmajian JV, Blumenstein R: Electrode placement in electromyographic biofeedback, in Basmajian JV (ed): *Biofeedback: Principles and Practice for Clinicians*. Third edition. Baltimore, Williams & Wilkins, 1989, pp 369-382
- Blanpied P, Smidt GL: Human plantarflexor stiffness to multiple single-stretch trials. *J Biomech* 25: 29-39, 1992
- Brunt D, Andersen JC, Huntsman B, et al: Postural responses to lateral perturbation in healthy subjects and ankle sprain patients. *Med Sci Sports Exerc* 24: 171-176, 1992
- DeCarlo MS, Talbot RW: Evaluation of ankle proprioception following injection of the anterior talofibular ligament. *J Orthop Sports Phys Ther* 8: 70-76, 1986
- DeLuca CJ: The use of surface electromyography in biomechanics. *J Appl Biomech* 13: 135-163, 1997
- Denegar CR, Bell DW: Assessing reliability and precision of measurement: An introduction to intraclass correlation and standard error of measurement. *J Sport Rehabil* 2: 35-42, 1993
- Di Fabio RP: Reliability of computerized surface electromyography for determining the onset of muscle activity. *Phys Ther* 67: 43-48, 1987
- Fernandes N, Allison GT, Hopper D: Peroneal latency in normal and injured ankles at varying angles of perturbation. *Clin Orthop* 375: 193-201, 2000
- Feuerbach JW, Grabiner MD, Koh TJ, et al: Effect of an ankle orthosis and ankle ligament anesthesia on ankle joint proprioception. *Am J Sports Med* 22: 223-229, 1994
- Freeman MA: Instability of the foot after injuries to the lateral ligament of the ankle. *J Bone Joint Surg* 47B: 669-677, 1965
- Garrick JG: The frequency of injury, mechanism of injury, and the epidemiology of ankle sprains. *Am J Sports Med* 5: 241-242, 1977
- Gottlieb GL, Agarwal GC: Dependence of human ankle compliance on joint angle. *J Biomech* 11: 177-181, 1978
- Gregory JE, Morgan DL, Proske U: Changes in size of the stretch reflex of cat and man attributed to aftereffects in muscle spindles. *J Neurophysiol* 58: 628-640, 1987
- Grillner S: The role of muscle stiffness in meeting the changing postural and locomotor requirements for force development by the ankle extensors. *Acta Physiol Scand* 86: 92-108, 1972
- Hertel J, Guskiewicz KM, Kahler DM, et al: Effect of lateral ankle joint anesthesia on center of balance, postural sway, and joint position sense. *J Sport Rehabil* 5: 111-119, 1996
- Hintermann B: Biomechanics of the unstable ankle and clinical implications. *Med Sci Sports Exer* 31(Suppl 7): S459-S469, 1999
- Hollis JM, Blasler RD, Flahiff CM: Simulated lateral ankle ligamentous injury: Changes in ankle stability. *Am J Sports Med* 23: 672-677, 1995
- Hopkins JT, Ingersoll CD, Krause BA, et al: Effect of knee joint effusion on quadriceps and soleus motoneuron pool excitability. *Med Sci Sports Exerc* 33: 123-126, 2001
- Hopper D, Allison G, Fernandes N, et al: Reliability of the peroneal latency in normal ankles. *Clin Orthop* 350: 159-165, 1998
- Iles JF, Stokes M, Young A: Reflex actions of knee joint afferents during contraction of the human quadriceps. *Clin Physiol* 10: 489-500, 1990
- Jensen K, Graf BK: The effects of knee effusion on quadriceps strength and knee intraarticular pressure. *Arthroscopy* 9: 52-56, 1993
- Johansson H, Sjolander P, Sojka P: A sensory role for the cruciate ligaments. *Clin Orthop* 268: 161-178, 1991
- Johansson H, Sjolander P, Sojka P: Reflex actions on the gamma muscle spindle systems of muscle activity at the knee joint elicited by stretch of the posterior cruciate ligament. *Neuro-Orthop* 8: 9-21, 1989
- Johansson H, Sojka P: Action on gamma-motoneurons elicited by electrical stimulation of cutaneous afferent fibres in the hind limb of the cat. *J Physiol* 366: 343-363, 1985
- Johnson EE, Markolf KL: The contribution of the anterior talofibular ligament to ankle laxity. *J Bone Joint Surg* 65A: 81-88, 1983
- Johnson MB, Johnson CL: Electromyographic response of peroneal muscles in surgical and nonsurgical injured ankles during sudden inversion. *J Orthop Sports Phys Ther* 18: 497-501, 1993
- Kennedy JC, Alexander IJ, Hayes KC: Nerve supply of the human knee and its functional importance. *Am J Sports Med* 10: 329-335, 1982
- Khin-Myo-Hla, Ishii T, Sakane M, et al: Effect of anesthesia of the sinus tarsi on peroneal reaction time in patients with functional instability of the ankle. *Foot Ankle Int* 20: 554-559, 1999
- Kjaergaard-Andersen P, Wetheland JO, Helmig P, et al: Effect of the calcaneofibular ligament on hindfoot rotation in amputation specimens. *Acta Orthop Scand* 58: 135-138, 1987
- Kjaergaard-Andersen PL, Wetheland JO, Nielsen S: Lateral talocalcaneal instability following section of the calcaneofibular ligament: A kinematic study. *Foot Ankle* 7: 355-361, 1987
- Komi PV, Buskirk ER: Reproducibility of electromyographic measurements with inserted wire electrodes and surface electrodes. *Electromyography* 10: 357-367, 1970
- Konradsen L, Ravn JB: Prolonged peroneal reaction time in ankle instability. *Int J Sports Med* 12: 290-292, 1991
- Konradsen L, Ravn JB: Ankle instability caused by prolonged peroneal reaction time. *Acta Orthop Scand* 61: 388-390, 1990
- Konradsen L, Ravn JB, Sorensen AI: Proprioception at the ankle: The effect of anaesthetic blockade of ligament receptors. *J Bone Joint Surg* 75B: 433-436, 1993
- Larsen E: Experimental instability of the ankle. A radiographic investigation. *Clin Orthop* 204: 193-200, 1986

41. Latimer HA, Tibone JE, Pink MM, et al: Shoulder reaction time and muscle-firing patterns in response to an anterior translation force. *J Shoulder Elbow Surg* 7: 610–615, 1998
42. Lephart SM, Riemann BL, Fu FH: Introduction to the sensorimotor system, in Lephart SM, Fu FH (eds): *Proprioception and Neuromuscular Control in Joint Stability*. Champaign, IL, Human Kinetics, 2000, pp xvii–xxiv
43. Loeb GE, Brown IE, Cheng EJ: A hierarchical foundation for models of sensorimotor control. *Exp Brain Res* 126(1): 1–18, 1999
44. Lohrer H, Alt W, Gollhofer A: Neuromuscular properties and functional aspects of taped ankles. *Am J Sports Med* 27: 69–75, 1999
45. Louie J, Mote CD Jr: Contribution of the musculature to rotatory laxity and torsional stiffness at the knee. *J Biomech* 20: 281–300, 1987
46. Louwerens JWK, van Linge B, de Klerk LW, et al: Peroneus longus and tibialis anterior muscle activity in the stance phase. A quantified electromyographic study of 10 controls and 25 patients with chronic ankle instability. *Acta Orthop Scand* 66: 517–523, 1995
47. Lynch SA, Eklund U, Gottlieb D, et al: Electromyographic latency changes in the ankle musculature during inversion moments. *Am J Sports Med* 24: 362–369, 1996
48. Ma SP, Zahalak GI: The mechanical response of the active human triceps brachii to very rapid stretch and shortening. *J Biomech* 18: 585–598, 1985
49. McNair PJ, Wood GA, Marshall RN: Stiffness of the hamstring muscles and its relationship to function in anterior cruciate deficient individuals. *Clin Biomech* 7: 131–137, 1992
50. Michelson JD, Hutchins C: Mechanoreceptors in human ankle ligaments. *J Bone Joint Surg* 77B: 219–224, 1995
51. Miyatsu M, Atsuda Y, Watakabe M: The physiology of mechanoreceptors in the anterior cruciate ligament. An experimental study in decerebrate-spinalized animals. *J Bone Joint Surg* 75B: 653–657, 1993
52. Morgan DL: Separation of active and passive components of short-range stiffness of muscle. *Am J Physiol* 232: C45–C49, 1977
53. Mussa-Ivaldi FA, Hogan N, Bizzi E: Neural, mechanical, and geometric factors subserving arm posture in humans. *J Neurosci* 5: 2732–2743, 1985
54. Olmstead TG, Wevers HW, Bryant JT, et al: Effect of muscle activity of valgus/varus laxity and stiffness of the knee. *J Biomech* 19: 565–577, 1986
55. Pope MH, Johnson RJ, Brown DW, et al: The role of musculature in injuries to the medial collateral ligament. *J Bone Joint Surg* 61A: 398–402, 1979
56. Rasmussen O, Tovborg-Jensen I: Mobility of the ankle joint: Recording of rotatory movements in the talocrural joint in vitro with and without the lateral collateral ligaments of the ankle. *Acta Orthop Scand* 53: 155–160, 1982
57. Raunest J, Sager M, Burgener E: Proprioceptive mechanisms in the cruciate ligaments: An electromyographic study on reflex activity in the thigh muscles. *J Trauma* 41: 488–493, 1996
58. Renstrom P, Wertz M, Incavo S, et al: Strain in the lateral ligaments of the ankle. *Foot Ankle* 9: 59–63, 1988
59. Shiavi R, Frigo C, Pedotti A: Electromyographic signals during gait: Criteria for envelope filtering and number of strides. *Med Biol Eng Comput* 36: 171–178, 1998
60. Shrout PE, Fleiss JL: Intraclass correlations: Uses in assessing rater reliability. *Psychol Bull* 86: 420–428, 1979
61. Sinkjaer T, Hayashi R: Regulation of wrist stiffness by the stretch reflex. *J Biomech* 22: 1133–1140, 1989
62. Sinkjaer T, Toft E, Andreassen S, et al: Muscle stiffness in human ankle dorsiflexors: Intrinsic and reflex components. *J Neurophysiol* 60: 1110–1121, 1988
63. Sojka P, Johansson H, Sjoland P, et al: Fusimotor neurones can be reflexively influenced by activity in receptor afferents from the posterior cruciate ligament. *Brain Res* 483: 177–183, 1989
64. Spencer JD, Hayes KC, Alexander IJ: Knee joint effusion and quadriceps reflex inhibition in man. *Arch Phys Med Rehabil* 65: 171–177, 1984
65. Torry MR, Decker MJ, Viola RW, et al: Intra-articular knee joint effusion induces quadriceps avoidance gait patterns. *Clin Biomech* 15: 147–159, 2000
66. Tropp H, Ekstrand J, Gillquist J: Stabilometry in functional instability of the ankle and its value in predicting injury. *Med Sci Sports Exerc* 16: 64–66, 1984
67. Viitasalo JT, Salo A, Lahtinen J: Neuromuscular functioning of athletes and non-athletes in the drop jump. *Eur J Appl Physiol Occup Physiol* 78: 432–440, 1998
68. Weiss PL, Hunter IW, Kearney RE: Human ankle joint stiffness over the full range of muscle activation levels. *J Biomech* 21: 539–544, 1988
69. Wilkie DR: The relation between force and velocity in human muscle. *J Physiol (London)* 110: 249–280, 1950
70. Winter DA: *Biomechanics and Motor Control of Human Movement*. Second edition. New York, John Wiley & Sons, Inc, 1990
71. Winter DA: The locomotion laboratory as a clinical assessment system. *Med Prog Technol* 4: 95–106, 1976
72. Wyke B: Articular neurology—a review. *Physiotherapy* 58: 94–99, 1972
73. Yang JF, Winter DA: Electromyographic amplitude normalization methods: Improving their sensitivity as diagnostic tools in gait analysis. *Arch Phys Med Rehabil* 65: 517–521, 1984
74. Zhang LQ, Portland GH, Wang G, et al: Stiffness, viscosity, and upper limb inertia about the glenohumeral abduction axis. *J Orthop Res* 18: 94–100, 2000