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Motor-Neuron Pool Excitability of the Lower Leg Muscles After Acute Lateral Ankle Sprain

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Context: Neuromuscular deficits in leg muscles that are associated with arthrogenic muscle inhibition have been reported in people with chronic ankle instability, yet whether these neuromuscular alterations are present in individuals with acute sprains is unknown.

Objective: To compare the effect of acute lateral ankle sprain on the motor-neuron pool excitability (MNPE) of injured leg muscles with that of uninjured contralateral leg muscles and the leg muscles of healthy controls.

Design: Case-control study.

Setting: Laboratory.

Patients or Other Participants: Ten individuals with acute ankle sprains (6 females, 4 males; age = 19.2 ± 3.8 years, height = 169.4 ± 8.5 cm, mass = 66.3 ± 11.6 kg) and 10 healthy individuals (6 females, 4 males; age = 20.6 ± 4.0 years, height = 169.9 ± 10.6 cm, mass = 66.3 ± 10.2 kg) participated.

Intervention(s): The independent variables were group (acute ankle sprain, healthy) and limb (injured, uninjured). Separate dependent *t* tests were used to determine differences in MNPE between legs.

Main Outcome Measure(s): The MNPE of the soleus, fibularis longus, and tibialis anterior was measured by the maximal Hoffmann reflex (H_{max}) and maximal muscle response (M_{max}) and was then normalized using the H_{max}:M_{max} ratio.

Results: The soleus MNPE in the ankle-sprain group was higher in the injured limb (H_{max}:M_{max} = 0.63; 95% confidence interval [CI], 0.46, 0.80) than in the uninjured limb (H_{max}:M_{max} = 0.47; 95% CI, 0.08, 0.93) (t_6 = 3.62, P = .01). In the acute ankle-sprain group, tibialis anterior MNPE tended to be lower in the injured ankle (H_{max}:M_{max} = 0.06; 95% CI, 0.01, 0.10) than in the uninjured ankle (H_{max}:M_{max} = 0.22; 95% CI, 0.09, 0.35), but this finding was not different (t_9 = –2.01, P = .07). No differences were detected between injured (0.22; 95% CI, 0.14, 0.29) and uninjured (0.25; 95% CI, 0.12, 0.38) ankles for the fibularis longus in the ankle-sprain group (t_9 = –0.739, P = .48). We found no side-to-side differences in any muscle among the healthy group.

Conclusions: Facilitated MNPE was present in the involved soleus muscle of patients with acute ankle sprains, but no differences were found in the fibularis longus or tibialis anterior muscles.

Key Words: arthrogenic muscle response, Hoffmann reflex, fibularis longus, soleus, tibialis anterior

Key Points

- Arthrogenic muscle response seemed to be present in the ipsilateral musculature of patients after acute lateral ankle sprains and manifested as a facilitation of the soleus and an inhibition of the tibialis anterior in between-legs scores.
- The maximal Hoffmann reflex to maximal muscle response ratio was greater in the soleus, was not different in the fibularis longus, and was smaller in the tibialis anterior musculature of the injured limbs of participants with acute ankle sprains compared with their contralateral uninjured limbs and compared with the injury-matched and contralateral limbs of healthy participants.

The ankle is the most commonly injured joint in the lower extremity.[1,2] A history of ankle sprain is the leading risk factor for recurrent ankle sprains.[3] Researchers[4] have reported that up to 30% of patients experiencing an initial ankle sprain develop chronic ankle instability. Chronic ankle instability is associated with increased risk of degenerative osteoarthritis[5] and decreased self-reported function.[6] Currently, the exact factors that contribute to pathogenesis of chronic ankle instability are unclear, yet investigators[7,8] have suggested that neuromuscular factors contribute to ankle instability by possibly disrupting the normal function of the muscles surrounding the injured joint. Understanding the neuromuscular response of the extrinsic ankle muscles to ankle joint injury might provide vital information that leads to improved treatment protocols.

Arthrogenic muscle inhibition is a consequence of joint injury often overlooked by clinicians and is defined as an ongoing reflex inhibition of the uninjured musculature surrounding an injured or distended joint,[9] which might contribute to the dysfunction reported after joint injury.[10] Although arthrogenic muscle inhibition has been hypothesized[11] to be a natural protective mechanism that decreases excessive forces acting on an injured joint, it is a limiting factor in rehabilitation.[9] It has been reported[7] in the soleus and fibularis longus muscles of functionally unstable ankles, whereas facilitated or increased neural excitability has been found[12] in lower leg muscles after an artificial effusion of ankle joints in healthy volunteers.

Altered motor-neuron pool excitability (MNPE) is a hallmark characteristic of the arthrogenic muscle response, which involves a decrease or increase in the number of motor neurons capable of responding to an excitatory stimulus within a given motor-neuron pool.[13,14] The Hoffmann reflex (H-reflex) is used to measure the excitability of α motor neurons located within a targeted motor-neuron pool.[14,15] In addition to the afferent and efferent pathways that contribute to the H-reflex, electric stimulation of the peripheral nerve evokes a purely efferent response along the α motor neuron in the muscle, which is known as the *muscle response* (M-response).[15] The M-response represents the maximal excitability of the motor-neuron pool as measured by the response of the muscle to the stimulated motor neurons.[14]

Although evidence supports the presence of increased reflex MNPE in the leg muscles after ankle-effusion models[12] and inhibited MNPE in participants with functionally unstable ankles,[7] limited evidence is available to confirm the motor-neuron pool response of the soleus, fibularis longus, or tibialis anterior muscle after acute ankle sprains.[16] It is important to determine if acutely sprained ankles exhibit differences in lower extremity MNPE when compared with uninjured contralateral ankles. If differences in MNPE are detected, this information might aid in the immediate treatment provided by athletic trainers.

Therefore, the primary purpose of our study was to determine the effect of acute lateral ankle sprains on MNPE of the soleus, fibularis longus, and tibialis anterior compared with the contralateral uninjured leg and with the legs of healthy participants. Investigating the confounding factors that might contribute to arthrogenic muscle inhibition after an acute ankle sprain also was important, so we secondarily examined the relationships among pain, effusion, and joint damage and alterations in MNPE because they are poorly understood.

METHODS

This case-control study had 2 independent variables: limb (injured, uninjured) and group (acute ankle sprain, healthy). The main outcome measure was MNPE in the soleus, fibularis longus, and tibialis anterior, as measured by normalizing the H-reflex to the M-response. Specifically, maximal H-reflexes (H_{max}) were normalized to maximal M-responses (M_{max}) and expressed as an H_{max}:M_{max} ratio. Subjective pain scores assessed using a visual analog scale, ankle-girth measurements, and self-reported function evaluated with the Foot and Ankle Ability Measure (FAAM)[17] were collected for each participant.

Participants

Twenty people volunteered for this study and were separated into 2 groups (Table 1). The experimental group consisted of patients with acute ankle sprains. The healthy group consisted of participants who were matched for age, sex, mass, height, and activity; who had no history of ankle injury or lower extremity fractures or surgery; and who were not seeking medical attention for any lower extremity injury. The inclusion criterion for the acute ankle-sprain group was operationally defined as all lateral ankle sprains occurring from 24 to 72 hours before the study. Participants were recruited from local public and private high schools, the university student and student-athlete populations, and the general population through referrals and advertisements. All individuals with ankle sprains were included, regardless of the severity of their injuries, and their injured ankles were evaluated by an athletic trainer using a standard ankle-injury evaluation form. The ankle-injury evaluation was used to grade the current condition of each participant's ankle injury. All volunteers were informed of the potential influence of cryotherapy and transcutaneous electric neuromuscular stimulation on MNPE and were instructed to discontinue treatment at least 6 hours before participation in the study. In addition, compression wraps and braces were removed 1 hour before testing. Potential participants with suspected syndesmotic ankle sprains or any previous diagnosis of associated lower extremity fracture, neurologic condition, or cancer were excluded from the study. A single certified athletic trainer (L.W.K.) used our laboratory's standardized grading form to indicate the severity of the ankle sprain, which is a recommended practice.[18] (Ankle sprains included 6 grade I and 4 grade II sprains.) The injury-matched ankle in the healthy group was side matched to his or her injured counterpart. Therefore, if the left ankle of the participant in the ankle-sprain group was sprained, the left ankle of the matched control participant in the healthy group was considered involved or injured. Before testing, written informed consent was obtained for all adults, and minor and parental consent were obtained for all minors (<18 years of age). The study was approved by the institutional review board of the University of Virginia.

Instrumentation

The H-reflex and M-response measurements were collected with disposable, 10-mm, pregelled Ag/AgCl surface electromyography (EMG) electrodes (BIOPAC Systems, Inc, Goleta, CA). The electrodes were positioned 1.75 mm apart over the muscle bellies of the soleus, fibularis longus, and tibialis anterior.[12] Analog-to-digital signal conversion was processed with a 16-bit converter (MP150; BIOPAC Systems, Inc). Acq*Knowl-*

Table 1. Participant Demographics (Mean [95% Confidence Interval])

	Group	
Variable	Acute Ankle Sprain	Healthy
Participants, No.	10	10
Sex, No.		
Female	6	6
Male	4	4
Height, cm	169.4 (164, 174.7)	169.9 (163, 176.5)
Mass, kg	66.3 (59.1, 73.5)	66.3 (59.9, 72.6)
Body mass index	23.2 (21, 25.4)	22.8 (21.5, 24.1)
Age, y	19.2 (16.8, 21.6)	20.6 (18.1, 23.1)
Involved limb		
Right	4	NA
Left	6	NA

Abbreviation: NA, not applicable.

edge software (version 3.7.3; BIOPAC Systems, Inc) was used to visualize the signals and to manipulate the stimuli. The EMG signals were sampled at 2000 Hz, and EMG amplification was set at a gain of 1000 (EMG100C; BIOPAC Systems, Inc). The common-mode rejection ratio of our EMG amplifier was 100 dB, and the input impedance was 2 MΩ. Reflexes were elicited using the stimulator module (STM100A; BIOPAC Systems, Inc) interfaced with a 200-V maximum stimulus isolation adaptor (STMISOC; BIOPAC Systems, Inc); a 2-mm shield disk electrode (EL254S; BIOPAC Systems, Inc); and a 7-cm, carbon-impregnated dispersive pad.

Ankle Evaluation

A licensed certified athletic trainer (L.W.K.) performed an orthopaedic ankle evaluation on all participants. All ankle-sprain data for participants were collected between 24 hours and 72 hours after injury (mean = 50.6 ± 20.9 hours).

Two 100-mm visual analog scales were used to assess ankle pain. The first visual analog scale was used to assess the greatest amount of pain the participant felt in the 24 hours before the study, whereas the second visual analog scale evaluated the participant's current level of ankle pain. The visual analog scale measurements were collected with the participant seated after the standard ankle evaluation.

A flexible tape measure was used to assess ankle circumference for both the injured and uninjured ankles in the experimental group and for the injury-matched and healthy legs in the healthy group. The percentage change in circumference between the injured ankle and the uninjured ankle was used to determine the amount of ankle effusion. The figure-of-8 method was performed with the participant seated, the knee in complete extension, and the ankle in neutral position. The measurement was performed with the "zero" of the tape measure maintained in the middle point between the articular projection of the anterior tibial tendon and the lateral malleolus. The tape measure was guided to the center of the foot along the medial longitudinal arch on the navicular bone, to the lateral malleolus and calcaneal tendon, to the medial malleolus, and to the zero point of the tape measure. The average of 3 measurements was used for data analysis.[19] Effusion was determined by subtracting the girth of the uninjured leg from that of the injured leg.

The FAAM is used to quantify the impairment, activity limitations, and participation restrictions with regard to foot and ankle injury.[17] The form consists of 3 components: activities of daily living (21 items), FAAM sport scale (8 items), and single-assessment numeric evaluation scores for both the FAAM and the FAAM sport scale. These data were collected in all participants for injured and healthy matched legs.

MNPE Measurement

Participants were positioned prone on a table in a quiet, dimly lit room with their knees slightly flexed (15°) and their ankles supported on a foam roller (Figure). We used a commercial platform (Oakworks Inc, New Freedom, PA) to position the participant's head in a neutral position with his or her face down. Participants were instructed to relax and focus on a fixed object on the floor during testing. The recording sites over the soleus, tibialis anterior, and fibularis longus were shaved, debrided, and cleaned with isopropyl alcohol. In addition, a reference electrode was placed on the medial malleolus of the uninjured leg of the experimental participant and of the matched leg of the healthy participant. Two recording electrodes were positioned 1.75 mm apart over the soleus muscle belly 2 to 3 cm distal to the head of the medial gastrocnemius, over the fibularis longus 2 to 3 cm distal to the fibular head, and at the approximate midpoint of the tibialis anterior.[12] We used a strip of hypoallergenic tape to secure the simulating electrode over the superior popliteal fossa proximal to the bifurcation of the tibial and common fibular nerves. The dispersive electrode was positioned on the anterior thigh.

We used previously reported methods[7] to locate the optimal positioning for the stimulating electrode. The stimulating electrode was placed at the fibular head, and a 1-millisecond square-wave pulse with an intensity designed to elicit an M-response and H-reflex in the tibialis anterior and the peroneus longus was administered. The electrode was moved manually in a superomedial direction, periodically administering stimulation to the common fibular nerve. We continued to move the electrode until an M-response could be elicited in all 3 muscles, indicating that the stimulating electrode was over the sciatic nerve before its bifurcation.

A 1-millisecond square-wave stimulus was administered to the sciatic nerve at increasing intensities until H_{max} and M_{max} were found for all 3 muscles. Three H_{max} and M_{max} measurements were taken for each of the muscles. The procedure was performed on both legs. Peak-to-peak values for H_{max} and M_{max} were used to calculate the H_{max}:M_{max} ratio. The investigator (LW.K.) assessing MNPE was not blinded to the group or leg of the participant because placing the electrodes in the appropriate locations without noticing edema or ecchymosis from an injured ankle was nearly impossible.

The H_{max} and M_{max} were processed by a blinded, experienced, independent investigator (B.G.P.) who assessed peak-to-peak amplitudes of both the H_{max} and M_{max} measurements in all 3 muscles. When a peak was observed for both the H_{max} and M_{max}, 3 acceptable measurements were obtained and used for data analysis. During data analysis, waveforms were inspected visually, and any H_{max}:M_{max} ratios greater than 1.0 were removed from the data set and were not used for analysis because this measurement was not physiologically possible and likely represented extraneous measurement error. During this process, we excluded 3 soleus measurements from separate individuals in the ankle-sprain group and 2 soleus measurements from individuals in the healthy group. One fibularis longus measurement was excluded from an individual in the healthy group because tracings could not be interpreted.

Statistical Analysis

Sample size was estimated a priori using means and SDs from a previous study[7] in which the soleus H_{max}:M_{max} ratio was assessed in patients with chronic ankle instability and in healthy people (mean difference = 0.05, pooled SD = 0.2). If a weak effect size was present between legs (Cohen d = 0.36), 9 participants would be needed in both the injured and healthy groups to reach a difference with the α level set at .05 and a 1–β level of .80. Means and SDs were calculated for H_{max} and M_{max}. Two-tailed dependent-samples *t* tests were used to assess differences in H_{max}:M_{max} ratios between injured and uninjured legs in the acute ankle-sprain group and in H_{max}:M_{max} ratios between the injury-matched and healthy legs of the healthy participants for all 3 muscles. Side-to-side differences in the H_{max}:M_{max} ratios for all 3 muscles were calculated in the acute ankle-sprain group (injured versus uninjured ankles) and in the

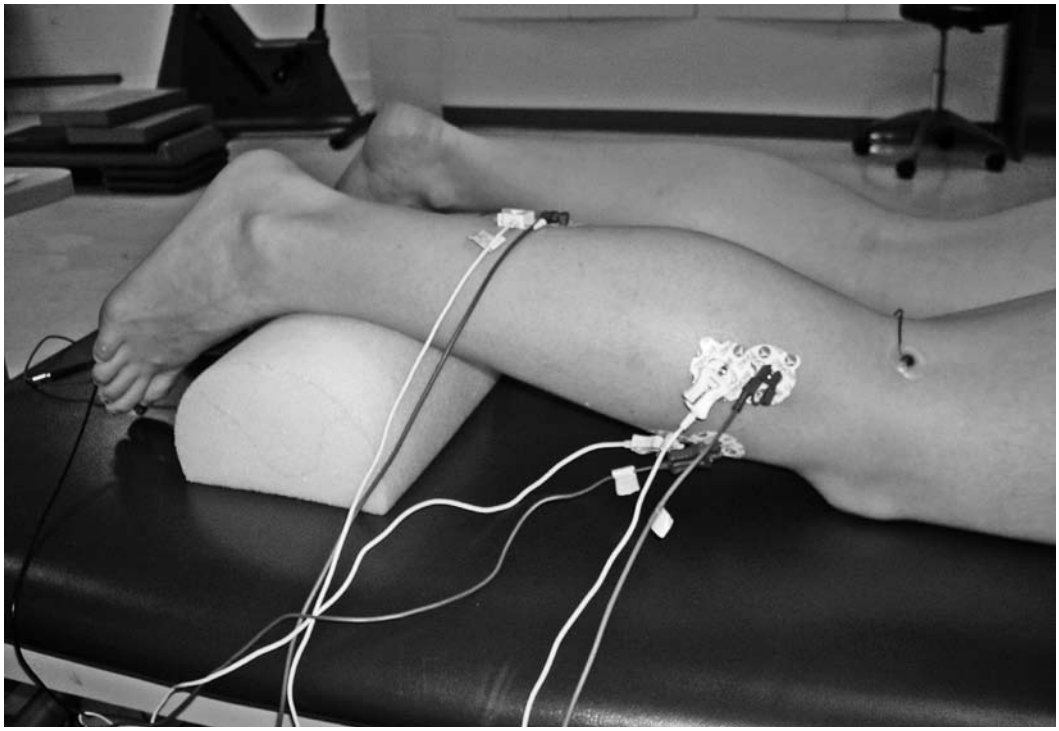

Figure. Placement of the surface electromyography electrodes. The participant was positioned prone with half of a foam roller supporting the dome of the talus in the involved leg. The surface electromyography was applied to standardized placement sites on the soleus, tibialis anterior, and fibularis longus muscles. The stimulating electrode was applied to the superior popliteal fossa proximal to the bifurcation of the tibial and common peroneal nerve, and the dispersive electrode was applied to the anterior quadriceps muscle superior to the patella.

healthy group (injury-matched versus contralateral ankle) by subtracting the injured leg score from the uninjured leg score. Three separate independent-samples *t* tests were used to assess the side-to-side difference scores in H_{max}:M_{max} ratios between the acute ankle-sprain group and the healthy group. In addition, Pearson product moment correlation coefficients were calculated and squared to determine the variance in effusion and pain that was explained by the variance in MNPE. Standardized effect sizes were calculated to assess magnitude of differences in between-legs H_{max}:M_{max} ratios between the acute ankle-sprain group and the healthy group. The effect sizes were calculated by subtracting the between-legs H_{max}:M_{max} ratios of the acute ankle-sprain group from those of the healthy group and dividing by the pooled SD. The strengths of the effect sizes were interpreted using the guidelines described by Cohen,[20] with values less than 0.5 interpreted as *weak*; values from 0.5 to 0.79 interpreted as *moderate*; and values greater than 0.8 interpreted as *strong*. The α level was set a priori at .05. All statistical analyses were performed using SPSS (version 16.0 for Windows; SPSS Inc, Chicago, IL).

RESULTS

MNPE Measurements in Acute Ankle Sprains

The descriptive measures for the MNPE results are shown in Table 2. The soleus H_{max}:M_{max} ratios in the acute ankle-sprain group were larger in the injured limb than in the uninjured limb ($t_6 = 3.62$, $P = .01$). No differences were detected for H_{max}:M_{max} ratios between the injured and uninjured ankles for the fibularis longus ($t_9 = –0.738$, $P = .48$) and tibialis anterior ($t_9 = –2.07$, $P = .07$) MNPE in the acute ankle-sprain group. A moderate effect was found between legs in the soleus (Cohen d = 0.69; 95% confidence interval [CI], –0.39, 1.76; $t_{14} = 0.735$, $P = .01$), indicating increased soleus MNPE in the ankle-sprain group. A strong effect size was found for the tibialis anterior (Cohen d = –1.01; 95% CI, –1.94, –0.08; $t_{18} = –2.19$, $P = .04$), with a negative sign indicating decreased MNPE in the acute ankle-sprain group. A weak effect size was found for the fibularis longus (Cohen d = –0.21; 95% CI, –1.09, 0.67; $t_{14} = –0.38$, $P = .71$), indicating a clinically irrelevant decrease in the fibularis longus MNPE of the individuals with acute ankle sprains compared with the healthy participants. The tibialis anterior was the only muscle with a 95% CI for effect size that did not cross zero, indicating a definitive effect in inhibition was present, but the width of all the CIs was large, indicating wide variability in the reflex measurements.

MNPE Measurements in Healthy Controls

No differences were found between legs for the H_{max}:M_{max} ratios of the soleus ($t_7 = 0.693$, $P = .51$), fibularis longus ($t_8 = –0.235$, $P = .82$), and tibialis anterior ($t_9 = –0.729$, $P = .48$) in the healthy group (Table 2).

MNPE Differences Between Groups

We found no differences when comparing the side-to-side differences in H_{max}:M_{max} ratios for the soleus between the ankle-injury (15.6; 95% CI, 7.2–24.1) and healthy (6.4; 95% CI,

Table 2. Maximal Hoffmann Reflex to Maximal Muscle Pooled Response Ratios Between Groups (Mean ± SD [95% Confidence Interval])

	Group			
	Acute Ankle Sprain		Injury Matched	
Muscle	Injured	Healthy	Uninjured	Healthy
Soleus	0.47 ± 0.24 (0.08–0.93)	0.63 ± 0.23 (0.46–0.80)[a]	0.58 ± 0.24 (0.42–0.76)	0.54 ± 0.25 (0.37–0.72)
Fibularis longus	0.25 ± 0.22 (0.12–0.38)	0.22 ± 0.12 (0.14–0.29)	0.24 ± 0.11 (0.17–0.31)	0.24 ± 0.18 (0.13–0.36)
Tibialis anterior	0.22 ± 0.22 (0.09–0.35)	0.06 ± 0.07 (0.01–0.10)[b]	0.15 ± 0.08 (0.1–0.20)	0.18 ± 0.18 (0.07–0.29)

[a] Indicates greater than the uninjured limb (P = .01).

[b] Indicates less than the uninjured limb (P = .07).

–14 to 26.9) groups (t_{14} = 0.735, P = .48). We also found no differences for the fibularis longus between the ankle-injury (–3.7; 95% CI, –13.5, 6.1) and healthy (–1.4; 95% CI, –7.3, 4.5) groups (t_{17} = –0.378, P = .71). However, we found a difference for the tibialis anterior between the acute ankle-sprain (–16; 95% CI, –31.4, –0.8) and healthy (2.6; 95% CI, –4.5, 9.8) groups (t_{18} = –2.19, P = .04).

Pain

Scores for both the current visual analog scale and visual analog scale in the 24 hours before the study were larger for the injured ankle than for the uninjured ankle in the acute ankle-sprain group (Table 3). The FAAM activities of daily living, FAAM activities of daily living single-assessment numeric evaluation, FAAM sport, and FAAM sport single-assessment numeric evaluation scores were lower in the ankle-sprain group than in the healthy group (Table 4).

Pain, Effusion, and MNPE

The variance in the current visual analog scale pain scores explained a large amount of variance in the tibialis anterior MNPE in the injured legs of the acute ankle-sprain group (r^2 = 0.74, P = .001). However, it did not explain a large amount of variance in the MNPE of the soleus (r^2 = 0.41, P = .76) or fibularis longus (r^2 = 0.27, P = .11) in the injured legs of the acute ankle-sprain group. The amount of effusion in the ankle-sprain group, as measured by ankle girth, did not explain a large amount of variance in the between-legs differences in H_{max}:M_{max} ratios of the soleus (r^2 = 0.266, P = .13), tibialis anterior (r^2 = 0.001, P = .93), or fibularis longus (r^2 = 0.136, P = .29) in the acute ankle-sprain group.

DISCUSSION

We conducted this study to determine the effect of an acute lateral ankle sprain on MNPE of the soleus, fibularis longus, and tibialis anterior compared with the contralateral uninjured ankle and with injury-matched and contralateral ankles in a healthy group. In the ankle-sprain group, soleus MNPE was greater in the injured than in the uninjured ankle. Between-legs change scores did not differ between the acute ankle-sprain and healthy groups. In the ankle-sprain group, tibialis anterior MNPE tended to be lower in the injured than in the uninjured limb, and between-legs change scores were different between the acute ankle-sprain and healthy groups. Interestingly, we found no differences in MNPE for the fibularis longus between injured and uninjured limbs in the acute ankle-sprain group or between the injury-matched and contralateral limbs in the healthy group, and we found no differences when comparing side-to-side differences in MNPE between groups. Our results are unique because MNPE measures have not been reported in leg muscles after acute ankle sprain.

Comprehensive analyses of lower leg MNPE have been conducted in people with functional ankle instability[7] and healthy volunteers with artificially induced ankle joint effusion.[12] Interestingly, these 2 populations produced different results. The artificial-effusion model displayed a marked facilitation of MNPE in the lower leg muscles, which was interpreted as a splinting mechanism at the joint.[12] The lower leg muscles in functionally unstable ankles had decreased MNPE, which is an inhibition hypothesized to contribute to recurrent ankle injury.[7] From these results, we can hypothesize that the amount of effusion might influence the nature of the arthrogenic muscle response, yet our results indicated that no relationship existed between ankle girth and MNPE, indicating that swelling did not contribute to MNPE in people 72 hours after an initial ankle sprain.

Table 3. Visual Analog Scale and Figure-of-8 Measures, Mean ± SD (95% Confidence Interval)

	Group			
	Acute Ankle Sprain		Injury Matched	
Muscle	Injured	Healthy	Uninjured	Healthy
Visual analog scale for pain in 24 h before study, mm	47 ± 22 (32, 60)[a]	0.2 ± 0.6 (–0.19, 0.6)	0.3 ± 0.7 (–0.11, 0.71)	0.1 ± 0.3 (–0.1, 0.3)
Visual analog scale for current pain level, mm	18 ± 15 (–0.11, 0.71)[a]	0.0 ± 0.0 (0, 0)	0.3 ± 0.7 (–0.10, 0.3)	0.1 ± 0.4 (–0.1, 0.3)
Figure-of-8, cm	53 ± 4.0 (50, 55)	51 ± 3.8 (48, 53)	53 ± 3.8 (50.7, 55.3)	53 ± 4.0 (51, 55)

[a] Indicates greater than the uninjured limb (P = .01).

Table 4. Foot and Ankle Ability Measure Measurements (FAAM), Mean ± SD (95% Confidence Interval)

Measure	Group	
	Acute Ankle Sprain	Healthy
FAAM	63.1 ± 17.2 (51, 75)[a]	100 ± 0.0 (100, 100)
FAAM SANE	68.9 ± 19.0 (57, 80)[a]	99 ± 0.6 (99, 100)
FAAM sport scale	35.0 ± 27.5 (17.9, 52)[a]	100 ± 0.0 (100, 100)
FAAM sport SANE	51.0 ± 24.6 (34.9, 67)[a]	99 ± 63 (99, 100)

Abbreviation: SANE, single-assessment numeric evaluation.
[a] Indicates lower than the control group ($P \leq .001$).

The amount of effusion in the people we tested did not correlate directly with the magnitude of the change in MNPE. With previous hypotheses, researchers[11] have suggested that joint mechanoreceptors stimulated by an injured or distended joint capsule might cause these reflexive muscle changes after injury. Authors have confirmed that effusing the ankle[12] and knee[21,22] results in MNPE changes around the effused joint. Conversely, aspirating the joint or removing effusion at the knee also has resulted in changes in MNPE,[10] providing evidence that immediate changes in effusion volume are related to MNPE. We can hypothesize that while immediately altering effusion might cause dramatic changes in MNPE, different mechanisms might drive changes in MNPE at 24 to 72 hours after injury. We can speculate that supraspinal mechanisms might be more influential in modulating MNPE at 24 to 72 hours after acute sprains but that reflexive mechanisms driven by, predominantly, mechanoreceptor stimulation might be responsible for altering MNPE directly after immediate changes in effusion.

A moderate to strong relationship ($r^2 = 0.74$, $P = .001$) was present only between tibialis anterior MNPE and current visual analog pain scores for the tibialis anterior in the injured ankle of the acute ankle-sprain group. The relationship between pain and muscle inhibition often is questioned, but the link between them might not be as strong as is thought intuitively. Although little evidence is available to establish the relationship between pain and changes in MNPE of muscles around the ankle after an acute ankle sprain, some evidence[23] has indicated that pain does not affect muscle activation in the quadriceps after knee injury. We also know from previous experimental joint-effusion models[12,21,22] that changes in MNPE can occur independent of pain. Although suggesting that pain might alter MNPE is still very reasonable, more research is needed to determine the nature of this relationship, as well as which neural pathways and mechanisms are responsible for these changes.

Our results indicated an up-regulation or facilitation of the soleus MNPE coupled with an inhibition of the tibialis anterior. We can interpret this as a possible reflexive response aimed at positioning the injured ankle joint in a plantar-flexed, loose-packed position to increase comfort after trauma. We can speculate that this specific acute arthrogenic muscle response might position the ankle in slight plantar flexion and might contribute to instability at the joint. This reflexive positioning of the ankle into plantar flexion might be linked to previously reported dorsiflexion range-of-motion and posterior talar glide deficits in individuals with chronic ankle instability, which also have been hypothesized[24,25] to contribute to recurrent ankle sprains. Conversely, these results differ from those associated with effusion models, which have indicated facilitation in the soleus, fibularis, and tibialis anterior muscles that has been interpreted as a splinting mechanism.[12]

It is important to not overly generalize these results, which were obtained with participants in a relaxed, nonfunctional position. If testing was performed during weight-bearing activities, results might differ, and involvement of the fibularis longus might be more descriptive. A larger participant population and progressive tracking of the ankle-healing response beyond the acute inflammatory phase also might provide more insight into the progression of healing after ankle sprain.

These data might help the clinician understand the neuromuscular response of the extrinsic ankle muscles to the acutely sprained ankle. Because the soleus is the main plantar flexor of the ankle and because plantar flexion is an inherently more unstable position, avoiding positions of plantar flexion and inversion is better for the ankle. Consequently, increased activation of the soleus might predispose the ankle to injury by situating the joint in an open-packed position, which might be more apt to invert. In addition, the tibialis anterior is responsible for dorsiflexion and eccentric control of plantar flexion. If the tibialis anterior was to be inhibited after an acute ankle injury, this also could place the ankle at greater risk for injury because the tibialis anterior cannot prevent the ankle from moving into an unstable position. Interestingly, fibularis longus MNPE does not seem to be altered immediately after an acute ankle sprain, which, in the presence of a plantar-flexed ankle, might increase the risk of a subsequent inversion ankle sprain. Future study might be needed to determine if a facilitated fibularis longus would be protective immediately after an acute ankle sprain.

Interventions, such as focal joint cooling, transcutaneous electric nerve stimulation, and transcranial magnetic stimulation, have been reported[26,27] to affect MNPE or muscle activation in muscles of the lower extremity after joint injury or effusion. Although investigators[28] have suggested using these modalities to disinhibit motor neurons before or during therapeutic exercise to achieve optimal neuromuscular benefits, little research has been conducted with these modalities at the ankle. We also do not know how facilitated motor-neuron pools would react to a disinhibitory modality, and future research should focus on whether, and when in the healing process, these modalities should be administered after acute ankle sprain.

Our study had limitations. First, the retrospective case-control study design relied on data without preinjury H_{max}:M_{max} ratios for participant normalization measures. Second, patients were tested in a relaxed, nonfunctional, prone position, which provided a stable environment for reflex testing but might have supplied limited information about how reflex excitability is different during activity. Third, progressive tracking of the ankle healing response during recovery was absent. Fourth, although we could not ethically suggest that participants discontinue all treatment, we did attempt to decrease the influence of the effects of interventions known to alter MNPE, such as cryotherapy and transcutaneous electric nerve stimulation,[26,27] by instructing them to discontinue these treatments at least 6 hours before testing. Regardless, we do not know how cumulative interventions applied immediately after the ankle sprain could have altered MNPE assessed hours to days after treatment. The design of future research on acute ankle sprains should take these limitations into consideration.

CONCLUSIONS

From the results of our study, we concluded that arthrogenic muscle response was present in the ipsilateral musculature of patients exhibiting acute lateral ankle sprains. Specifically, this

arthrogenic muscle response manifested as a facilitation of the soleus and an inhibition of the tibialis anterior, which were found with between-legs comparisons. Furthermore, the soleus musculature had an increased H_{max}:M_{max} ratio. We also found no difference in the H_{max}:M_{max} ratio of the fibularis longus and identified a trend toward a decrease in the H_{max}:M_{max} ratio of the tibialis anterior in the injured limbs of patients with acute ankle sprains. Our results are the first to provide insight into the arthrogenic muscle response associated with acute ankle sprain.

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