ORIGINAL ARTICLE

Arthrogenic muscle response to a simulated ankle joint effusion

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Background: Arthrogenic muscle inhibition (AMI) is a continuing reflex reaction of the musculature surrounding a joint after distension or damage to the structures of that joint. This phenomenon has been well documented after knee joint injury and has been generalised to occur at other joints of the human body, yet minimal research has been conducted in this regard. The response of the muscles crossing the ankle/foot complex after ankle injury and effusion is not well understood. AMI may occur after an ankle sprain contributing to residual dysfunction.

Objective: To determine if AMI is present in the soleus, peroneus longus, and tibialis anterior musculature after a simulated ankle joint effusion.

Methods: Eight neurologically sound volunteers (mean (SD) age 23 (4) years, height 171 (6) cm, mass 73 (10) kg) participated. Maximum H-reflex and maximum M-wave measurements were collected using surface electromyography after delivery of a percutaneous stimulus to the sciatic nerve before its bifurcation into the common peroneal and posterior tibial nerves.

Results: The H-reflex and M-wave measurements in all muscles increased ($p \leq 0.05$) after the simulated ankle joint effusion.

Conclusions: Simulated ankle joint effusion results in facilitation of the soleus, peroneus longus, and tibialis anterior motoneurone pools. This may occur to stabilise the foot/ankle complex in order to maintain posture and/or locomotion.

Ankle sprains are common during athletic competition and recreational activity.^{1–3} Acute ankle sprains comprise 12% of all injuries seen in emergency rooms and 15% of all athletic injuries.^{1–2} Functional ankle instability (FAI) or a subjective report of “giving way” at the ankle may be prevalent in as many as 40% of patients after an acute sprain of the lateral ligament complex.^{4–5} A definitive cause of FAI continues to elude clinicians. The absence of a pathophysiological mechanism for this chronic instability precludes the development of an effective treatment protocol.

Peroneal muscle weakness may exist after an ankle sprain precipitating FAI. Weakness of the evtor musculature has been extensively examined, but its existence is still questioned because of conflicting results.^{6–9} The possible weakness of the evtor musculature has been suggested to occur as a result of peroneal nerve injury,⁷ inadequate rehabilitation,⁹ and/or inhibition because of pain or oedema.¹⁰ After ankle injury it is difficult to determine which of the above mechanisms lead to the evtor weakness as all of those proposed may result. The use of an ankle effusion model is an effective method to determine whether inhibition is associated with swelling within the ankle joint. Neuromuscular inhibition of the evtor musculature may exist in concurrence with inhibition or facilitation of the other muscles that stabilise the ankle complex. This inhibition may occur after ankle joint injury and is a possible cause of FAI, therefore further examination is warranted.

Neuromuscular inhibition has been widely reported in the quadriceps as a result of knee joint injury^{11–13} or simulated joint effusion.^{14–16} This phenomenon has been termed arthrogenic muscle inhibition (AMI) and is a continuing reflex inhibition of the musculature surrounding a joint after distension or damage to structures of that joint.¹⁷ The quadriceps musculature sustains no direct damage, but severe weakness and wasting occurs, creating disability and

prolonging the rehabilitation process. AMI has been generalised to occur at all joints in the musculoskeletal system,^{18–19} yet minimal research^{20–22} has been conducted to confirm this hypothesis. A simulated effusion at the ankle joint has been shown to result in facilitation of the soleus H-reflex,²³ but it is not known how the other muscles that stabilise the ankle/foot complex react to joint trauma. Petrik *et al*²² hypothesised that facilitation of the soleus occurs in addition to inhibition of the tibialis anterior and peroneus longus musculature, preventing dorsiflexion of the ankle, thereby decreasing joint volume. Establishing the relation between the plantarflexors/dorsiflexors and invertors/evertors would be useful in understanding the interaction of the muscles after trauma. The purpose of this investigation was to compare changes in the soleus, tibialis anterior, and peroneus longus H-reflex measurements resulting from ankle joint effusion over a one hour time period.

METHODS

Subjects

Eight healthy neurologically sound college students (four women and four men, mean (SD) age 23 (4) years, height 171 (6) cm, mass 73 (10) kg), with no history of lower extremity surgery and no lower extremity injury for 12 months before the study, volunteered. Subjects were excluded if they reported allergies to lidocaine and/or latex. Each subject was instructed to refrain from ingesting any stimulating or depressing substances for 24 hours before data collection. In addition, participants were asked not to exercise before data collection. Each subject provided written consent

Abbreviations: AMI, arthrogenic muscle inhibition; EMG, electromyography; FAI, functional ankle instability

after the purpose of the study had been explained. The protocol was approved by the School of Health and Human Performance human subjects review committee.

Instrumentation

H-reflex and muscle response (M-wave) measurements were collected using surface electromyography (EMG) (MP100; BIOPAC Systems Inc, Santa Barbara, California, USA).

Signals were amplified (DA100B; BIOPAC Systems Inc.; gain 1000) from disposable, 10 mm pre-gelled Ag/AgCl electrodes (BIOPAC Systems Inc). The EMG signal was band pass filtered from 10 to 500 Hz and sampled at 2000 Hz with a common mode rejection ratio of 50 dB. The BIOPAC stimulator module (STM100A; BIOPAC Systems Inc) was used with a 200 V (maximum) stimulus isolation adapter (STMISOC; BIOPAC Systems Inc) and a shielded disc electrode (EL254S; BIOPAC Systems Inc).

Subject preparation

Four locations were shaved, abraded with fine sandpaper, and cleaned with isopropyl alcohol for the application of the EMG electrodes. Surface EMG electrodes were placed 2 cm apart centre to centre on the peroneus longus, tibialis anterior, and soleus musculature to capture the maximum peak to peak amplitude of the H-reflex and M-wave for each muscle. The peroneus longus surface electrodes were positioned 2–3 cm distal to the head of the fibula. Recording electrodes for the tibialis anterior were placed at the approximate midpoint of the muscle belly. EMG electrodes for the soleus were positioned 2–3 cm distal to the medial head of the gastrocnemius. A ground electrode was placed on the ipsilateral lateral malleolus.

A stimulating electrode was placed in the superior portion of the popliteal fossa in order to access the sciatic nerve before its bifurcation into the common peroneal and tibial nerves. The corresponding anode was placed superior to the patella. To find the sciatic nerve bifurcation, the stimulating electrode was placed at the fibular head. A 1 millisecond square wave pulse was then delivered using a stimulus intensity eliciting a motor response in the peroneus longus and tibialis anterior. The stimulating electrode was then advanced in a superior medial direction toward the midpoint of the popliteal fossa. Once a response was seen in all three muscle groups, we determined that the sciatic nerve bifurcation had been located. An adhesive collar was applied to the stimulating electrode to maintain its position for the duration of data collection.

H-reflex and M-wave procedures

Subjects were positioned supine, with the involved knee flexed to approximately 15° and the heel resting in a secure pad, designed to keep the foot stable and in a neutral position throughout data collection. All participants placed their hands to their sides with their palms open. They were asked to keep their head forward, and were required to listen to ocean wave sounds through headphones during testing.

H/M recruitment curves²³ were mapped for all muscles simultaneously by increasing the intensity of the stimulus in 0.2 V increments (10 second rest intervals were given between stimuli) until the maximum M-wave was obtained for each muscle. The peak to peak amplitudes of the H-reflexes and M-waves were recorded for all of the test stimulations. A single peak to peak amplitude of the H-reflex and M-wave was recorded for all of the test stimulations. The maximum values of the H-reflex, M-wave, and H/M ratios for the peroneus longus, tibialis anterior, and soleus musculature were examined and used for data analysis. This protocol has been shown to be reliable for all three muscle groups.²⁴

Joint effusion procedure

An area on the anteromedial portion of the ankle was cleaned with alcohol and betadine. Each participant was then initially injected superficial to the ankle capsule with 3 ml lidocaine; care was taken not to anaesthetise the joint. After the skin had been anaesthetised, the ankle joint proper was injected with 10 ml sterile saline through a medial approach (fig 1). After the effusion, subjects were asked not to move the ankle to minimise the loss of saline from the joint capsule. A new pair of sterile disposable gloves was worn for each participant. All materials were disposed of in the proper sharps and biohazard containers according to Occupational Safety and Health Administration guidelines.

Testing procedures

Stimulating and recording electrode placement sites were prepared as previously described. Baseline recruitment curves (before injection) for all three muscles were recorded for each subject (time 1). Once the recruitment curves were collected, the ankle joint effusion was induced. Recruitment curves were again recorded immediately after the effusion (time 2). Additional measurements were taken every five minutes after the injection for one hour (times 3–14).

Statistical analysis

A one way repeated measures analysis of variance was performed to determine if time differed on maximum H-reflex, maximum M-wave, and the H/M ratio for each muscle. Bonferroni multiple comparison tests were used post hoc to locate specific group differences. The α level was set a priori at $p \leq 0.05$.

Results

An overall difference was detected in the H-reflex ($F_{7,13} = 6.61$; $p = 0.0001$; $\text{Eta}^2 = 0.452$; table 1), M-wave ($F_{7,13} = 5.28$; $p = 0.0001$; $\text{Eta}^2 = 0.421$; table 2), and H/M ratio ($F_{7,13} = 2.219$; $p = 0.013$; $\text{Eta}^2 = 0.217$; fig 2) for the soleus. The H-reflex and H/M ratios were lower before the injection than at all time intervals after (Bonferroni $p < 0.05$). The M-wave had also increased immediately after injection as well as at 5, 10, and 15 minutes after (Bonferroni $p < 0.05$). The M-wave immediately after the injection was greater than at 35, 50, 55, and 60 minutes after (Bonferroni $p < 0.05$).

An overall increase was also seen in the H-reflex ($F_{7,13} = 4.11$; $p = 0.0001$; $\text{Eta}^2 = 0.593$; table 1) and M-wave ($F_{7,13} = 6.12$; $p = 0.0001$; $\text{Eta}^2 = 0.504$; table 2) for the peroneus longus muscles. The H-reflex before the injection was lower than at other time intervals after: immediately after, 5, 10, 20, 25, 35, and 40 minutes after (Bonferroni $p < 0.05$). The M-wave of the peroneus longus before injection was smaller than at all time intervals after (Bonferroni $p < 0.05$). No difference was detected in the H/M ratio

Figure 1 Ankle after injection of saline. Note the swelling around the lateral malleolus.

Table 1 Peak to peak amplitude (mV) of the maximum H-reflex for all muscle groups at each time interval

Time	Soleus	Peroneus	Tibialis anterior
1	9.82 (2.39)	3.07 (1.29)	1.10 (0.49)
2	11.08 (2.57)	3.98 (1.28)	1.40 (0.48)
3	11.15 (2.83)	3.96 (1.15)	1.39 (0.47)
4	10.94 (2.62)	3.85 (1.00)	1.33 (0.42)
5	11.00 (2.73)	3.86 (1.01)	1.34 (0.47)
6	10.98 (2.73)	3.85 (1.05)	1.35 (0.47)
7	10.96 (2.67)	3.80 (1.01)	1.32 (0.45)
8	10.73 (2.85)	3.79 (1.00)	1.31 (0.44)
9	10.95 (2.56)	3.80 (1.05)	1.31 (0.45)
10	11.01 (2.69)	3.79 (1.03)	1.33 (0.48)
11	10.88 (2.58)	3.74 (1.04)	1.28 (0.43)
12	10.85 (2.58)	3.77 (1.09)	1.28 (0.43)
13	10.82 (2.63)	3.77 (1.05)	1.28 (0.44)
14	10.80 (2.66)	3.76 (1.06)	1.27 (0.45)

Values are mean (SD).

Table 2 Peak to peak amplitude of the maximum M-wave (mV) for all muscle groups at each time interval

Time	Soleus	Peroneus	Tibialis anterior
1	15.24 (2.07)	10.34 (1.58)	4.42 (1.30)
2	16.44 (2.52)	11.95 (1.72)	5.53 (1.34)
3	16.32 (2.55)	11.90 (1.63)	5.79 (1.49)
4	16.31 (2.48)	11.97 (1.72)	5.75 (1.66)
5	16.17 (2.48)	12.07 (1.83)	5.69 (1.69)
6	15.99 (2.87)	11.95 (1.66)	5.69 (1.72)
7	15.91 (2.80)	11.96 (1.79)	5.66 (1.69)
8	15.85 (2.62)	11.89 (1.77)	5.69 (1.68)
9	15.83 (2.76)	11.92 (1.81)	5.64 (1.68)
10	15.84 (2.69)	11.86 (1.81)	5.58 (1.62)
11	15.85 (2.74)	11.88 (1.77)	5.55 (1.59)
12	15.81 (2.76)	11.85 (1.83)	5.53 (1.59)
13	15.79 (2.74)	11.72 (1.73)	5.52 (1.53)
14	15.80 (2.75)	11.64 (1.69)	5.54 (1.58)

Values are mean (SD).

($F_{7,13} = 1.78$; $p = 0.056$; $\text{Eta}^2 = 0.182$; $1-\beta = 0.872$; fig 2) in the peroneus longus.

An overall increase was also found for the tibialis anterior H-reflex ($F_{7,13} = 1.90$; $p = 0.04$; $\text{Eta}^2 = 0.464$; table 1) and M-wave ($F_{7,13} = 13.67$; $p = 0.0001$; $\text{Eta}^2 = 0.639$; table 2). The H-reflex before the injection was lower than the time intervals after: immediately, 5, 15, and 20 minutes after (Bonferroni $p < 0.05$). The M-wave before injection was smaller than all other time intervals (Bonferroni $p < 0.05$). No difference was detected in the H/M ratio ($F_{7,13} = 0.536$; $p = 0.897$; $\text{Eta}^2 = 0.071$; $1-\beta = 0.296$) in the tibialis anterior (fig 2).

DISCUSSION

This study was carried out to examine if a simulated ankle joint effusion affects motoneurone pool excitability of the soleus, peroneus longus, and tibialis anterior musculature. After the injection of 10 ml sterile saline into the ankle joint capsule, an increase in the H-reflex of all three muscles was observed. This suggests that, in the presence of an acute, non-inflammatory ankle joint effusion, motoneurone pool excitability was facilitated, and remained facilitated for up to one hour.

Freeman *et al*¹⁴ postulated that FAI results from a neuromuscular deficit. Many investigators continue to suggest neuromuscular dysfunction as a possible cause of FAI. However, there have been few studies²² of the neuromuscular pathways from which this deficit would result. We here used H-reflex measurements to compare the availability of motoneurons within the soleus, peroneus longus, and tibialis anterior motoneurone pools before effusion and for one hour after the effusion. A change in excitability of the motoneurone pool may be manifested as a change in H-reflex amplitude, as more or fewer motoneurons are excited.¹⁵

The H-reflex amplitude has previously been used to show that a simulated knee joint effusion results in inhibition of the quadriceps musculature.¹⁴⁻¹⁶ AMI is a natural response designed to protect the joint from further damage.¹⁷ Our results suggest that AMI is not present in the soleus, peroneus longus, and tibialis anterior musculature during an ankle joint effusion. The induced effusion did produce an arthrogenic response. However, this reaction was manifested as a facilitation of the joint musculature. Therefore, we believe that arthrogenic muscle response is a more appropriate term for the reaction that occurs in the joint musculature after injury.

Our findings agree with a study that examined the effects of ankle joint effusion on the soleus H-reflex.²² The authors found an increase in the maximum H-reflex immediately after injection of saline into the joint capsule. They concluded

that the facilitation was a protective mechanism to prevent the joint from moving into dorsiflexion. Further, they felt that this would result from a reciprocal inhibition of the tibialis anterior. Our findings show that the tibialis anterior was not inhibited but facilitated, negating the idea of a reciprocal muscular shut down. However, we do feel that the lower leg musculature is facilitated as a result of reciprocal innervation.

During normal voluntary movement, antagonist musculature opposing a contraction is reflexively relaxed to allow speed and efficiency of movement about a joint.²⁵ This is known as reciprocal inhibition and is mediated through Ia interneurons. Ia interneurons allow information from supraspinal centres to execute coordinated movement between opposing muscles through a single command.²⁵ The Ia interneurone also receives input through Renshaw cells, which inhibit the Ia interneurons.²⁶ In situations in which precision and joint stabilisation are crucial, it is advantageous to "shut off" reciprocal inhibition and simultaneously activate (co-contraction) agonist and antagonist muscle groups. Co-contraction is also mediated through the Ia interneurons. For co-contraction to occur, the Renshaw cell needs to disinhibit the Ia interneurone, thus decreasing reciprocal inhibition and allowing activation of the antagonist muscle group(s).²⁵ Activity in segmental afferents may have influenced the Renshaw cells in this study, causing the disinhibition of the Ia interneurons resulting in the muscle facilitation. Co-contraction may have occurred after the ankle effusion in order to immobilise the ankle/foot complex in

Figure 2 H/M ratios for the three muscle groups at each time interval.

neutral, thereby minimising stress to ligamentous/joint structures, which may also help to diminish pain.

The above hypothesis deviates from the neural pathway typically thought to mediate AMI. AMI is thought to result from excitation of slowly adapting Ruffini endings within the knee joint capsule and stimulation of the Ib inhibitory interneurone.^{14 17 27} Ib interneurone inhibition can originate from golgi tendon-like receptors, as well as joint and cutaneous afferents, and results in widespread inhibition of homonymous and synergistic motoneurons and excitation of other motoneurons in the limb—that is, inhibition of the quadriceps and facilitation of the soleus.²⁸ Iles *et al*²⁷ have examined this pathway after knee joint effusion in humans using a spatial facilitation technique. They concluded that joint effusion inhibits quadriceps motoneurons and in part act through the Ib inhibitory pathway. Although the Ib interneurone may be critical in muscle inhibition, many other spinal mechanisms—for example, presynaptic inhibition, recurrent inhibition, tonic descending inhibition, Ia inhibition—probably contribute to AMI/arthrogenic muscle response but have yet to be investigated.

Preliminary results²⁰ on the effects of grade I and grade II inversion ankle sprains on neuromuscular excitability suggest that motoneurone pool excitability of the flexor digitorum longus and peroneus longus is unaltered, which is contradictory to our results. A likely reason for the differing results is the use of the effusion model instead of an actual ankle injury. We caution readers to examine the design and methods of this study,²⁰ as the authors fail to provide an adequate description of the methods used to obtain the H-reflex and M-wave. In addition, the results may have been different if measurements were obtained before injury rather than comparing the uninjured limb with the injured limb. A prospective investigation of the existence of an arthrogenic muscle response in musculature surrounding the ankle/foot complex is warranted to better understand if the state of the motoneurone pool is altered after an ankle sprain.

Our results show a significant facilitation in the M-wave of all muscles after the effusion. The increase in the M-wave has been previously described with joint effusion.²² Changes in the M-wave are often thought to reflect changes in recording conditions. We are confident that the increase in the M-wave seen in this study was not due to altered recording conditions. Subjects maintained the same position throughout testing, the stimulating electrode was secured, and the recording electrodes were maintained in the same position throughout data collection. A study from our laboratory has confirmed that this protocol produces reliable H-reflex and M-wave measurements.²⁴ As the methods used were stable over time and no change in muscle geometry took place, we are unclear how to interpret the facilitation in the M-wave. The maximum M-wave is usually a stable value, as it is thought to represent activation of all motor axons arising from the motoneurone pool of interest. Our results appear to contradict the idea that the maximum M-wave represents the entire motoneurone pool. With the above said, we can only speculate on the physiological alteration that resulted in the facilitated M-wave. The maximum M-wave has been shown to increase with an altered muscular environment.²⁹ If the neuromuscular system perceived the effusion as injury and began to guard the ankle joint through co-contraction, it is possible that the involved musculature was altered and may account for some of the increase in the maximum H-reflex and M-wave. Another factor that may account for the initial changes in M-wave is a change in temperature. The saline we introduced was stored at room temperature and when injected was cooler than body temperature. Although initially the temperature would have been different, it probably quickly warmed to body temperature and would not account

Take home message

The results suggest that a neuromuscular change occurs when ankle swelling is present after injury. Rehabilitation protocols after ankle sprains should focus on neuromuscular as well as mechanical alterations that occur as the result of ankle injury.

for the facilitation of the maximum M-wave throughout the entire hour. We have also considered the possibility that the H-reflex and M-wave both changed by the same mechanism. However, the H-reflex can be affected by many central pathways (Renshaw cells, presynaptic inhibition, Ib interneurons, Ia interneurons, tonic descending activity) whereas the M-response is not susceptible to these factors. We therefore feel that it was unlikely that the responses changed by a similar mechanism. Although a sympathetic reaction may have resulted from the introduction of the needle, the catecholamines released during this response would have been absorbed within minutes and cannot account for the facilitated H-reflex and M-wave for the entire hour. Further investigation of the facilitation of the M-wave is necessary to understand the physiological cause of the increase.

The H/M ratios were not altered in the peroneus longus and tibialis anterior after the joint effusion. This typically suggests that facilitation did not occur in these motoneurone pools. We chose to interpret the original H-reflex and M-waves rather than the ratios caused by the facilitation of the maximum M-wave. The H-reflex itself is representative of motoneurone excitability. Our aim was to see how the state of the motoneurone pool was affected after effusion. We feel this is best achieved by studying the H-reflex despite the fact of the facilitated M-wave.

The results of this study were obtained with the subjects in a relaxed, non-functional position. If testing were to be performed during a functional task (walking, running), tonic descending activity may override the neuromuscular facilitation seen. Investigations are being conducted to determine if results similar to this study are obtained if the subjects are tested when conducting a functional task.

When examining our results, it must be considered that a model was used to simulate ankle oedema. If testing were to be performed after an actual ankle sprain, different results may be expected. With an ankle injury, ligamentous damage would result, and inhibition of the surrounding musculature may occur. In addition, a true effusion would not occur after an ankle injury because of the ligamentous/capsular damage. The oedema present would leak into the interstitial space and would not be contained solely within the capsule.

CONCLUSIONS

From the results of this study, we conclude that motoneurone excitability is facilitated in the peroneal, soleus, and tibialis anterior muscle groups after an ankle joint effusion. We believe that this facilitation is a reaction necessary to stabilise the ankle/foot complex in order to maintain postural control and locomotion.

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