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The effects of joint mobilizations on lower leg reflex excitability in people with chronic ankle instability

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The Effects of Joint Mobilizations on Lower Leg Reflex Excitability in People with

Chronic Ankle Instability

by

Matthew S. Harkey, ATC

Submitted to the Graduate Faculty as partial fulfillment of the requirements for the

Master of Science Degree in Exercise Science

Dr. Brian Pietrosimone, Committee Chair

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The University of Toledo
May 2012
An Abstract of

The Effects of Joint Mobilizations on Lower Leg Reflex Excitability in People with Chronic Ankle Instability
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Objective: The objective of this study was to determine the efficacy of a posterior talar joint mobilization at altering both spinal reflexive and corticomotor neural excitability in patients with chronic ankle instability (CAI). Design and Setting: A single blinded, randomized control trial was conducted in a laboratory setting. Subjects: Sixteen patients with CAI were randomly assigned to an experimental group who received a joint mobilization treatment (4 males, 4 females; 22.63±4.21 years; 1.72±5.40 m; 73.01±12.11 kg), or to a control group (2 males, 6 females; 20.63±1.77 years; 164.94±8.20 m; 63.99±12.91 kg). Procedure: Experimental measures were measured pre- and post-intervention period. Patients were positioned in a Biodex System II Pro dynamometer during excitability testing. The Hoffmann reflex (H-reflex) of the tibialis anterior, fibularis longus, and soleus muscles were measured to estimate spinal reflex excitability using a BIOPAC MP150. Maximal H-reflexes were normalized to the maximal muscle response to create an H:M ratio used for analysis. A Magstim Rapid was utilized to administer the transcranial magnetic stimulation to the motor cortex during corticomotor testing. The Biodex dynamometer was used to collect maximal voluntary isometric torques for each muscle. Submaximal contractions at five percent of this maximal
contraction were used to standardize voluntary contraction during corticomotor testing. Active motor threshold (AMT) was established as the lowest intensity required to elicit a motor evoked potential (MEP) of ≥100µV amplitude in at least 5 of 10 trials. Five MEPs were collected at 120% of AMT then normalized to the M-wave for analysis. After the pre-test, a Certified Athletic Trainer administered three 60 second grade IV posterior talar mobilizations to participants in the experimental group. Participants in the control group sat quietly for the same duration needed to perform the joint mobilization. Significance was determined using dependent t-tests between pre and post values (p<0.05). Cohen’s $d$ effect sizes with corresponding 95% confidence intervals were calculated. Results: No significant differences were found for spinal reflex excitability in all muscles and effect sizes were small with wide confidence intervals. Although not significant (p=0.20), soleus AMT decreased following the mobilization producing an effect size of $d=-0.71$. Corticomotor excitability at 120% of AMT for all muscles were not significant and the effects sizes were small. Conclusion: This study provides limited support that a posterior talar joint mobilization has the potential to restore corticomotor excitability of the lower leg musculature in people with chronic ankle instability. Restoration of neural excitability is an imperative step in rehabilitation that will allow for greater rehabilitation gains which may result in quicker return to functional activities.
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Chapter 1

1.1 Introduction

Reestablishing neuromuscular control following lower extremity injury is imperative in the prevention of chronic joint ailments and disability that affects quality of life in a variety of patient populations.¹ A new rehabilitation paradigm that uses specific interventions to target the neurological origins of muscle dysfunction prior to traditional strength training has been reported to enhance rehabilitation outcomes.¹ Unfortunately, only a limited amount of interventions have been reported to have the capability to alter neural excitability and new interventions with these capabilities need to be developed.¹⁻⁴ There is recent data to suggest that lower extremity joint mobilizations may affect joint and muscle function,⁵⁻⁹ yet a comprehensive understanding of how this intervention affects influential neural pathways remains unknown. It is of critical need to determine the implications that joint mobilizations have on influential neural pathways, because this knowledge will be fundamental in the development of safe and cost-effective treatments to target neural dysfunction prior to traditional rehabilitation. We evaluated ankle joint mobilizations on patients with chronic ankle instability, because this group exhibits decreased neural excitability,¹⁰ altered joint function,¹¹ and decreased neuromuscular control.¹² Additionally, neuromuscular phenomena are easily measured at the ankle joint. We expect that findings from this study will be relevant in the development of treatment
for patients suffering from multiple lower extremity joint pathologies that exhibit neuromuscular deficits.

Arthrogenic Muscle Inhibition (AMI) is a common neural impairment of the musculature surrounding an injured joint.\(^1,13\) This inhibition may be manifested by one of two neural pathways: spinal reflexive or corticomotor.\(^13\) However, both of these processes, even though physiologically different, will both exhibit diminished motor output.\(^13\) Spinal reflexive excitability can be objectively measured by Hoffmann reflex testing.\(^10,13-20\) Corticomotor involvement is determined through transcranial magnetic stimulation.\(^13\) The inability to achieve sufficient activation of the motor neuron pool may inhibit proper movement and leave the patient susceptible to further injury.\(^1,10\) A paradigm shift in rehabilitation that includes disinhibitory modalities is needed to improve neural function prior to strength training, thereby creating a more progressive rehab strategy.\(^1,10\)\(^1,10\) Cryotherapy and Transcutaneous electrical nerve stimulation (TENS) have already been reported to have these disinhibitory effects\(^21\), indicated by an increase in quadriceps motoneuron pool excitability. They also improved strength and muscle activation outcomes in pathological populations,\(^2\) yet there are logistical concerns about when and how these modalities can be used in the clinical setting. Joint mobilizations are a simple, cost-effective alternative that may allow clinicians the same neuromuscular benefits without the logistical restrictions of ice and TENS.\(^1-3\) Joint mobilizations are already an established modality at increasing range of motion, decreasing pain, as well as other functional measures.\(^5-9,22\) There is already some early evidence of a novel indication for joint mobilizations because of their disinhibitory properties.\(^23,24\) Our laboratories have demonstrated proficiency, through previously
published results, at measuring neural excitability using the Hoffmann-reflex and transcranial magnetic stimulation.

1.2 Statement of the Purpose

The objective of this study was to determine if joint mobilizations can alter both spinal and corticomotor neural pathways in patients with chronic ankle instability.

1.3 Dependent Variables

1) Spinal Reflexive Excitability measured by the Hoffmann Reflex
   - H-reflex: M-wave (H:M) Ratio of maximal H-reflex amplitude to maximal M-wave amplitude for tibialis anterior, soleus, and fibularis longus

2) Corticomotor Excitability measured by Transcranial Magnetic Stimulation
   - Motor evoked potentials (MEP) at 120% of active motor threshold for tibialis anterior, soleus, and fibularis longus

1.4 Independent Variables

1) Group
   - Experimental = subjects receiving posterior talar joint mobilization
     - Fifteen subjects were randomly assigned to the experimental group
   - Control = subjects receiving no joint mobilization
     - Fifteen subjects were randomly assigned to the control group. Subjects will sit for the same duration of time needed to perform the joint mobilization intervention. This will be done to ensure blinding of the PI.
2) Time

- Pre = measurements taken just prior to the intervention
- Post = measurements taken immediately after the intervention

1.5 Research Questions

Do posterior talar joint mobilizations result in changes of excitability in the spinal reflexive and corticomotor neural pathways of the tibialis anterior, soleus, and fibularis longus musculature in persons with chronic ankle instability?

1.6 Null Hypothesis

Immediately following a posterior talar joint mobilization, there will be NO significant differences in excitability of the spinal reflexive and corticomotor neural pathways of the tibialis anterior, soleus, and fibularis longus.

1.7 Research Hypothesis

Immediately following a posterior talar joint mobilization, there WILL BE significant, alterations in the excitability of both the spinal reflexive and corticomotor neural pathways of the tibialis anterior, soleus, and fibularis longus.

1.8 Specific Aims

The Research Hypothesis will be tested through the examination of two specific aims:
1. To examine the immediate effects of ankle joint mobilizations on the excitability of spinal reflexive neural pathways.
   
   a. This aim was accomplished by testing the spinal reflexive contributions utilizing Hoffmann reflex testing

2. To examine the immediate effects of ankle joint mobilizations on the excitability of corticomotor neural pathways

   a. This aim was accomplished by testing the corticomotor involvement using transcranial magnetic stimulation

1.9 Operational Definitions

- Posterior Talar Joint Mobilization – a manual therapy technique that attempts to direct the talus from an anterior to posterior position.

- CAI – chronic ankle instability. Inclusion criteria for CAI:
  
  o Have a history of at least one acute lateral ankle sprain which resulted in swelling, pain and temporary loss of function (but none in the last 3 months)
  
  o Have a history of multiple episodes (more than 2) of the ankle “giving away” in the past 6 months
  
  o Score <90% on the FADI and <80% on the FADI Sport

- FADI/ FADI Sport – Functional Ankle Disability Index
  
  o Questionnaire used to determine degree of disability in subjects. FADI consists of questions regarding activities of daily living. FADI Sport is comprised of more functional activities primarily used during sports.
• AMI – Arthrogenic muscle inhibition
  o Ongoing inhibition of musculature surrounding a joint due to injury or
distention of joint; due to afferent activity disruption caused by injury
• H-reflex – Hoffmann reflex. Testing method used to determine spinal reflexive
neural contributions in muscle.
• H:M ratio – a method of normalizing spinal reflexive data obtained through H-
reflex testing. Ratio of maximal H-reflex to maximal M-wave amplitudes
• TMS – Transcranial magnetic stimulation. Testing method used to establish
corticomotor contributions to neuromuscular activity.
• MEP – Motor evoked potential. Electromyographical representation used to
quantify the corticomotor contributions to neuromuscular activity during TMS
testing.

1.10 Potential Limitations

Previous literature\textsuperscript{23,24} supports the statements made in each of our aims, however,
there is still a possibility that the joint mobilization intervention will not affect the spinal
and corticomotor neural pathways of the lower extremity. In the case of such an unlikely
event, the use of a Mulligan\textsuperscript{25} weight-bearing mobilization with movement could be
utilized as an alternative to the Maitland mobilization. This more functional manual
therapy technique may be able to provide stronger results. Additionally, we will be
making the assumption that included subjects with chronic ankle instability will present
with an anteriorly translated talus, which is indicated for posterior talar joint
mobilizations.
Another possible limitation may be sample size. This is unlikely as laboratories have completed previous studies with at least 30 subjects and we have an extensive database of individuals with chronic ankle instability living in the greater Toledo metropolitan area.

1.11 Significance

Conventional therapy focuses targeting strength and functional movement deficits without attempting to restore the neurological restraints\(^1,10\). The inability of current rehabilitation techniques to restore normal function in those with lower extremity injury\(^26\) may contribute to the economic strain chronic joint injury puts on the healthcare system.\(^27\) However, the utilization of a therapy to restore the neurological capabilities to the muscle would aid the current standard of care by providing previously unattainable rehabilitation gains.\(^1\) We believe that implementation of a cost-effective joint mobilization will create immediate changes both spinally and cortically. Due to the abundance of patients with chronically unstable ankles, we feel this is the best population to decipher the neural factors affecting this functional disability. Examination of these findings at the ankle will allow us to better understand the neural involvement of manual therapy. We expect that findings from this proposed research could be helpful for clinicians who seek to treat neuromuscular dysfunction at other joints such as knee or spine.
Chapter 2 - Literature Review

2.1 Joint Mobilization

Ankle sprains are considered one of the most ubiquitous musculoskeletal injuries, occurring at an incidence of 23,000 a day in the United States\(^{28}\). Ankle sprains are often considered innocuous due to their overwhelming abundance in our athletic culture. However, many debilitating side effects can accompany ankle sprains including decreased dorsiflexion (DF) that occurs at the talocrural joint. Decreased DF may contribute to a plethora of problems such as: an abnormal axis of rotation occurring at the talocrural joint, compensatory hypermobility of joints surrounding the talocrural joint, and altered neuromuscular function in muscles controlling the ankle\(^{29,7,14}\). All of these will culminate in decreased function of the ankle and a higher prevalence of reinjury. Recurrence rates of 80% have been seen with patients who have suffered ankle sprains\(^{30}\).

Another ailment that is speculated to arise from ankle sprains is an anterior positional fault of the talus in respect to the mortise of the ankle\(^{6,9,29,31}\). Despite the maladaptive changes that a “simple” ankle sprain can generate, research has suggested that a simple joint mobilization technique can help reverse these detrimental side effects\(^{5-9,22,25,32-34}\).

Movement of an extremity is a culmination of two types of motion: osteokinematic and arthrokinematic. Osteokinematic motion is the physiological movement that is produced by muscle contractions: flexion, extension, abduction, adduction, etc. Arthrokinematic motion is the accessory motion occurring inside the joint...
that allows for the full range of osteokinematic movement to be achieved; these consist of spin, roll, or glide\textsuperscript{35}. Spin involves twisting around a stationary longitudinal axis. Rolling occurs when a series of points comes in contact with a series of points on the articular surface. Gliding is when a specific point on one surface makes contact with a series of point on the articulating surface. If any of these arthrokinematic motions are altered it will result in a deficit in the total amount of range of motion at a joint. Dorsiflexion restriction after sustaining an ankle sprain is believed to be due in part by an impairment of one or more of these arthrokinematic motions. Research suggests that this obstruction is due to an anterior positional fault of the talus\textsuperscript{6,9,29,31}. Posterior talar mobilizations are theorized to return the talus to its correct anatomical location, which can restore dorsiflexion ROM and help modulate pain. Maitland has created a series of passive accessory mobilizations used to reduce pain and restore function.

Table 2.1: Maitland’s passive joint mobilization grading system. The grade depends on the amplitude of motion and where the motion is in the joint’s range of motion

<table>
<thead>
<tr>
<th>Grade</th>
<th>Amplitude</th>
<th>Location in ROM</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Small</td>
<td>Beginning</td>
</tr>
<tr>
<td>II</td>
<td>Large</td>
<td>Beginning-Middle</td>
</tr>
<tr>
<td>III</td>
<td>Large</td>
<td>Middle-End</td>
</tr>
<tr>
<td>IV</td>
<td>Small</td>
<td>End</td>
</tr>
</tbody>
</table>

Grades I and II are used mainly for pain reduction, while III and IV are used for treating stiffness\textsuperscript{34,35}. Most studies utilized Grade III or IV mobilizations because their main outcome measure was an increase in ROM\textsuperscript{6,32,33}, however one study used the Grade I technique and produced similar results\textsuperscript{5}. Maitland describes three roles that joint
mobilizations accomplish: 1) restoring the normal arthrokinematics of a joint 2) stretching the joint to increase ROM 3) relieving pain. The specific Maitland mobilization used to increase dorsiflexion is a posterior talar mobilization. Studies using this technique use a variety of variations, but ultimately the talus is mobilized in the posterior direction in regards to the mortise of the ankle.

Overwhelming evidence suggests that the use of a posterior talar joint mobilization will result in significant increases in dorsiflexion at the talocrural joint\textsuperscript{5-9,22,32,33}. These studies performed variants of posterior talar joint mobilizations on the ankle, and all provide evidence that the use of one of these will provide increased dorsiflexion. Due to the use of different variations of dorsiflexion tests, it is difficult to compare the results between the studies.

Green et al\textsuperscript{5} conducted a study that demonstrates RICE (rest, ice, compression, and elevation) combined with posterior talar joint mobilizations resulted in subjects able to reach full pain-free dorsiflexion in less treatments than with RICE alone. The ability to restore dorsiflexion sooner is of clinical importance because a quicker transition into therapeutic rehabilitation may ultimately result in earlier return to unrestricted activity. This study also demonstrates that the posterior talar mobilizations may improve stride speed, especially after the first treatment. This is imperative because of the functionality that stride speed brings to athletes. This correlation between dorsiflexion and stride speed portrays the impact that dorsiflexion may have on functional activities.

Prolonged immobilization of the ankle is sometimes required for more severe ankle sprains. This is not the ideal treatment of ankle injuries because of the detriment that it can have on the ROM and strength at the ankle joint. Landrum et al\textsuperscript{6} provides
evidence that even after prolonged immobilization a single application of a posterior joint mobilization can help counteract deficits in talocrural dorsiflexion.

There is controversy on the exact mechanism that results in increases in dorsiflexion following a posterior talar joint mobilization. Collins et al$^{22}$ conducted a study that again showed that joint mobilization increased dorsiflexion, however, it also used measures of pressure pain threshold (PPT) and thermal pain threshold (TPT) to demonstrate that mechanical effects are the main reason for the technique’s effectiveness. Pain thresholds are taken both pre- and post-intervention. Joint mobilizations were discovered not to produce hypoalgesia, which suggests the likeliness that a mechanical fault (probably the anterior position of the talus) is the reasoning for the effectiveness of posterior talar mobilizations for restoring dorsiflexion and not pain modulation.

One stipulation usually used as a decider of an athlete’s ability to return to play is whether or not they have restored full, pain free dorsiflexion. However, every athletic trainer does not utilize joint mobilizations in his or her everyday practice. So how is this positional fault corrected without the use joint mobilizations? Denegar et al$^{29}$ shows that athletes that return to play do restore their ROM so that it is equal to the contralateral limb, but they have a deficit in arthrokinematics due to an anterior displacement of the talus. A change in the arthrokinematics can result in an altered axis of dorsiflexion or excessive hypermobility of the joints surrounding the talocrural joint. The fact that they have achieved full physiological motion, but are still lacking correct arthrokinematics could be detrimental to the health of their ankle. These explanations potentially demonstrate why there is such a high recurrence rate because it places the ankle in altered position that may leave it susceptible to injury.
Table 2.2: Talocrural Mobilization Studies. The chart below summarizes the different techniques, and results of the studies using joint mobilizations.

<table>
<thead>
<tr>
<th>Study</th>
<th>Joint Mob Technique</th>
<th>DF Technique</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Collins et al</td>
<td>WB MWM (Mulligan)</td>
<td>Lunge technique</td>
<td>Increase DF, no change in mechanical/thermal pn threshold</td>
</tr>
<tr>
<td>de Souza et al</td>
<td>Maitland Grade III/IV</td>
<td>Biplane Goniometer</td>
<td>Increase DF</td>
</tr>
<tr>
<td>Green et al</td>
<td>Maitland Grade I</td>
<td>Hydrogoniometer</td>
<td>Increase DF, improves stride speed</td>
</tr>
<tr>
<td>Landrum et al</td>
<td>Maitland Grade III</td>
<td>Bubble inclinometer</td>
<td>Increase DF, increase joint stiffness, decrease posterior talar glide</td>
</tr>
<tr>
<td>Reid et al</td>
<td>WB MWM (Mulligan)</td>
<td>Lunge technique</td>
<td>Increase DF</td>
</tr>
<tr>
<td>Venturini et al</td>
<td>Maitland Grade III</td>
<td>Goniometer</td>
<td>Increase DF</td>
</tr>
<tr>
<td>Vincenzino et al</td>
<td>WB MWM/ NWB MWM</td>
<td>Lunge technique</td>
<td>Increase DF, increase posterior talar glide</td>
</tr>
</tbody>
</table>

These studies convincingly portray the usefulness that a posterior talar mobilization presents to the clinical community. However, most of these studies were only applied over a single treatment session and results were measured immediately after the intervention. This necessitates more studies using multiple techniques over a longer period of time, in addition to post-testing over time.

2.2 Hoffmann Reflex

Following joint disruption, either by injury or surgery, there is noticeable weakness in the musculature surrounding the joint; even though no direct damage has been bestowed upon the muscles. This decrease in muscle output is due to a
neuromuscular phenomenon termed Arthrogenic Muscle Inhibition (AMI). This response is ongoing neural inhibition of the muscles surrounding a joint, due to a summation of damage or swelling to the joint structures.\textsuperscript{10,15,18} This inhibition is created by a reduction in the motor neuron (MN) pool recruitment, which is caused by altered afferent activity to the spinal cord.\textsuperscript{15} Measurement of the amount of MN pool excitability, and an indirect measure of AMI, is achieved by measurement of the Hoffmann reflex (H-reflex).

### 2.2.1 Identifying the H-Reflex and M-Wave

The H-reflex is simply the electrical analog of the mechanical spinal stretch reflex. This stretch reflex is monosynaptic and is due to stretching of a muscle.\textsuperscript{36} As the muscle lengthens, this triggers activation of the muscle spindle, which stimulates the Ia afferent neurons.\textsuperscript{36} This afferent activity causes excitation of the alpha-motor neurons (aMN), which elicits an efferent response (contraction) of the muscle.\textsuperscript{36} The H-reflex is representative of this process except that it bypasses the muscle spindle and directly stimulates Ia afferent neurons through external electrical stimulation.\textsuperscript{17}

Applying a low-level electrical stimulus to the nerve that innervates the muscle of choice is the first step in generating the H-reflex.\textsuperscript{17,20} A low-intensity stimulus is needed in order to selectively stimulate the larger diameter primary afferent fibers (Ia afferents) that are responsible for eliciting the H-reflex. The stimulation of the Ia afferents creates action potentials sent toward the spinal cord where they will synapse with the MNs.\textsuperscript{17,20} If the stimulus created by the external stimulator is large enough, the primary afferents will depolarize the MNs and send a signal to the muscle that creates a contraction.\textsuperscript{20,36} EMG electrodes are placed on the muscle to record the muscle activity created by the
electrically elicited contraction (H-Reflex). Increasing the intensity of stimulation causes
more afferent fibers to be stimulated, thus stimulating more MNs and creating a larger H-
reflex. However, once the intensity is enough to stimulate the smaller diameter efferent
MNs, the M-wave will begin to appear and suppress the H-reflex until it disappears.\textsuperscript{17,20}

Antidromic transmission of the efferent neurons is responsible for the disappearance of
the H-reflex. As the efferent MNs are stimulated and the action potential is transmitted to
the muscle, there is also an antidromic (reverse) signal sent back toward the spinal cord.
This backwards signal eventually collides with the initial H-reflex activity, and if the
antidromic activity is equal to or larger than the H-reflex it will result in a disappearance
of the H-reflex from the EMG.\textsuperscript{15,17} As the stimulus continues to increase, eventually all of
the efferent fibers will be depolarized. Once no more efferent neurons are able to be
stimulated the M-wave will plateau and should be a stable value.\textsuperscript{15,17}

Latency is the time between the stimulus application and when the EMG signals
appear. The latency for H-reflexes vary depending on which muscle is being
stimulated.\textsuperscript{17} The latency will be shorter for the vastus medialis (VM) when compared to
the tibialis anterior (TA) because of the shorter distance the signal has to travel, due to
the distance that the VM is away from the spinal cord. Also, the M-wave latency is
smaller than the H-reflex, because the M-wave only has to pass down the efferent
neurons, while the H-reflex has to travel up the afferent pathway to the spinal cord then
back down the entire alpha MN.\textsuperscript{17}

\textbf{2.2.2 Implications of the H-reflex and M-wave}
Those were the basic appearances and tendencies of the H-reflex and M-wave; the following explains how they are involved in the neuromuscular excitability of muscles. Since the H-reflex is created by electrical signals propagated through afferent neurons, it can provide an estimate of motorneuron excitability.\textsuperscript{15,17,18} The $H_{\text{max}}$ is deemed the maximal reflex activation, which is the greatest number of MNs in the specific muscle that can be afferently activated.

Once a specific stimulation intensity is administered to the nerve the M-wave will plateau. This leveling off is the $M_{\text{max}}$ value and is interpreted to be the maximum muscle activation, in other words, the maximum MN pool available to the muscle.\textsuperscript{15,17} Creating a ratio $H:M$ ratio (dividing $H_{\text{max}}$ by $M_{\text{max}}$) can be done to normalize the data. This ratio allows us to create a percentage that represents the amount of the total MN pool that can be activated afferently.\textsuperscript{15,17} A decrease in this value is inferred to be the affects of AMI on the muscles surrounding a joint. An increase in the latency of the H-reflex has also been used as an indicator of AMI.\textsuperscript{14} The $H:M$ ratio can be utilized to evaluate and quantify the amount of AMI present following injury and assess the validity of therapeutic interventions.

\textbf{2.2.3 Ankle H-reflex Setup}

We are interested in investigating the effects of AMI of the musculature surrounding the ankle joint. The three muscles of interest are the soleus, fibularis longus (FL), and the tibialis anterior (TA). There is an established protocol that allows elicitation of the H-reflex of all three muscles when using a single external stimulus.\textsuperscript{19}
First, the correct positioning of the EMG electrodes for each muscle needs to be discussed. The soleus EMG electrodes will be placed over the midline of the muscle belly in the distal third portion of the lower leg. The FL electrodes will be placed distal to the fibular head about 2 to 3 cm. Finally, to capture the TA the electrodes will be placed on the midline of the muscle belly.

Once these are laid, the proper placement of the stimulating electrode needs to be found. The theory is to have the electrode placed over the sciatic nerve just before it splits into the tibial and common fibular nerve. To discover this site of the bifurcation the stimulating electrode is first placed at the fibular head in order to elicit activity of the FL and TA. The electrode is then moved in a superior medial direction towards the superior popliteal fossa. Once a response is triggered in the soleus, this is deemed the bifurcation of the sciatic nerve and the electrode will be secured to this position and used for the rest of the H-reflex testing.

### 2.2.4 Studies using H-reflex to measure AMI

McVey et al\(^{10}\) used subjects with functional ankle instability (FAI) to determine if there was AMI present, and deemed responsible for the chronic joint instability. H:M ratios were elicited in soleus, fibularis and TA muscles of both ankles in a FAI group and a control group. They discovered that the H:M ratio in the soleus and fibularis muscles were depressed in the FAI group when comparing injured and non-injured; there was no difference between muscles in the TA group. The lower H:M ratio indicates the presence of AMI in these muscles, but it is not evident if the AMI was a cause or result of the initial ankle injury. An important note is that there were no differences found between
the two limbs of the healthy group, which is evidence that FAI could be due to the neural dysfunction caused by AMI. The authors give the explanation that the reason the FAI develops in this specific population is because they are not able to “cope” with the neuromuscular interference caused by the initial injury, and they are left with the residual instability seen in FAI. To help with these people that are not neurally “coping” with the injury they will need more than just traditional ankle rehabilitation to fully recover.

Hall et al\textsuperscript{14} sought to compare the difference between H-reflex amplitudes and latencies in both the invertor and evertor musculature in subjects who recently sustained an inversion ankle sprain. The subject pool was small with a wide variability in how far post-injury they were (6.5 +/- 3 days). Also, the author’s classification of ankle sprains was very subjective and was probably a source of error for the study. The main result they found from this study was a delay in the latency of the flexor digitorum longus (FDL) (p = 0.002); along with a moderate positive correlation (r = 0.73) found between the FDL latency and ankle girth. They concluded that this reflex delay would help decrease the magnitude and velocity of the inversion moment around the ankle, thus minimizing stress and pain at the ankle. There was a trend that showed an increased latency at the peroneus longus (PL) (p = 0.04), but the authors deemed this insignificant. The H-reflex amplitude for both the FDL and PL did not produce statistically significant differences.

Palmieri et al\textsuperscript{16}, like the McVey study, used patients with FAI to discover if the AMI present in these neurally dysfunctional ankles was responsible for peroneal activation deficits. To test peroneal activation, the authors utilized a custom made walkway that included bilateral trapdoors that when activated would drop and allow the
ankle to invert 30°. The use of special goggles that blocked the subject’s inferior view and randomly dropping trap doors were used to ensure the subjects were not anticipating the ankle perturbation. This study fortifies previous results of the presence of AMI in peroneal muscles of subjects with FAI, seen by their decrease in H:M ratios. They also observed that the healthy subjects reported no side-to-side differences in the peroneal H:M ratio, which also supports the conclusion that AMI exists in FAI sufferers. The FAI group also presents with a decreased peroneal activation. However, even though both decreased neural excitability and muscle activation is present in the FAI subjects there is no relationship between the two. It makes clinical sense that if there is a decrease in the motorneuron pool excitability then there should be a decrease in the muscle activation, but this does not seem to be the case. The authors provide the reasoning that the AMI only inhibits the muscle by 10%, and during a dynamic activation task (like the one in the study) the entire motorneuron pool is not needed to counteract the inversion moment. So in this type of activity the effects of AMI are not clinically relevant. Even though these two variables are seen not to be related in this study, it is of clinical importance that both of these neuromuscular issues be corrected before the athlete returns to play or they may be more susceptible to future injury.

Palmieri et al also produced an earlier study that disputes using the term AMI, but institutes using the term Arthrogenic muscle response (AMR), because they found a facilitation and not inhibition in the musculature surrounding the ankle. The key difference in this study with the previous ones is that the researchers used a healthy subject and injected their ankles with saline to simulate an ankle infusion. This was interesting procedure choice because most of the causes of AMI are due to the disruption
of the joint receptors and how that alters the afferent output to the spinal cord. This simulated ankle effusion created a facilitation in the H-reflexes of the soleus, peroneal, and tibialis anterior muscles. An interesting note is that the M-wave of both the tibialis anterior and the peroneal muscles both increase. This is intriguing because the M-wave is supposed to be a stable measurement and is important because it allows for normalization of the H-reflex measurement.

2.3 Transcranial Magnetic Stimulation

The Hoffmann reflex testing is a measurement of the spinal reflexive contributions to Arthrogenic muscle inhibition (AMI).\textsuperscript{10,13-17,20} However, spinal pathways implicated in AMI encompass only part of the neural dysfunction.\textsuperscript{13} There is also corticomotor involvement that can influence the effects of AMI.\textsuperscript{13} Most AMI research has been predominantly focused on the spinal reflexive aspects, but because of the extensive descending projections on the interneurons and motorneurons involved in AMI, the corticomotor involvement has now become an imperative part of understanding the effects of AMI.\textsuperscript{13} Transcranial magnetic stimulation (TMS) is a safe, reliable way to determine corticomotor excitability involvement in the lower leg.\textsuperscript{37-41} TMS utilizes a magnetic stimuli delivered to the motor cortex to create motor evoked potentials (MEPs) that can be collected by EMG electrodes.

2.3.1 TMS Terms and Measures

When performing TMS, the first step is to determine the “hot spot” where all other data will be collected.\textsuperscript{37,38} The hot spot is defined as the optimal stimulation point of
the cortex.\textsuperscript{37} To find this location, the research begins with the magnetic coil at the vertex of the cranium, and depending on the target muscle, the coil is moved to the position where the lower motor threshold evokes a MEP response.\textsuperscript{37}

Once the hot spot is found, the active motor threshold (AMT) is the first TMS value usually discerned. The AMT is a measure that reflects the corticomotor excitability of a muscle.\textsuperscript{37} Motor threshold is assessed as the lowest intensity required to elicit a MEP in at least 5 of 10 trials. The required amplitude of the MEP differs depending on the study, values range from $25\mu V$\textsuperscript{38} to $50\mu V$\textsuperscript{37} to $100\mu V$.

This value of AMT is then used to create a stimulus response curve (SR curve). This curve takes the stimulus intensity required to evoke the AMT and increases this at intervals of 5\% intensity and the MEPs are recorded until 140\% of the AMT is reached. The SR curve portrays the relationship between the rise in the MEPs with the increase in the TMS intensity, and the slope of this relationship is an indicator of the strength of the cortical projections.\textsuperscript{37,38}
Chapter 3

3.1 Study Design

Single Blinded, Randomized Controlled Trial: A block randomization was used to allocate participants to one of two groups: control or grade IV posterior talar joint mobilization. Investigators conducting outcome measures were blinded to group assignment.

3.2 Subjects

All participants were between the ages of 18 and 35 and exhibit chronic ankle instability, which will be described as we have previously described using percentages from the Functional Ankle Disability Index ($< 90\%$) and the Functional Ankle Disability Index Sport Scale ($< 80\%$).\textsuperscript{42} In addition, all participants had a history of one lateral ankle sprain resulting in swelling, pain and temporary loss of function that lead to a history of multiple lateral ankle sprains (more than 2) in the past 6 months. We excluded all potential participants with a history of a joint hypermobility dysfunction, lower extremity ligament sprain or rupture (other than the ankle), fracture as well as those who have engaged in rehabilitation during the past 6 months. We excluded all those with a history of: concussion or head injury in the past 6 months, history of stroke, cardiac condition, epilepsy, cranial neurosurgery, migraines, cancer in the brain or thigh musculature,
diagnosed psychiatric disorder; or has a cardiac pacemaker, implanted cardiac defibrillator or intracranial metallic clips. All participants provided written informed consent approved by the institutional review board at the University of Toledo prior to performing any of these proposed experiments. All participants were instructed to refrain from ingesting caffeine for 12 hours prior to the study.

Means and standard deviations of Hoffmann reflex change scores were extracted from a related study evaluating the effect of a modalities used to excite the neural system\textsuperscript{43} to estimate sample size. A pooled mean difference of 2.26 and the pooled standard deviation of 1.5 were used to calculate sample size. An alpha of .05 and a 1-beta level of .80 were used in the calculation. Our calculations estimated that 12 patients were needed per group for a total of 24 patients.

Participants were recruited from the University of Toledo community using advertisements and by meeting with groups of students and various classrooms. Our laboratory also possesses a database of people living in the greater Toledo Metropolitan area with chronic ankle instability, which has been generated by researchers in the Athletic Training Research Laboratory and the Joint Injury and Muscle Activation Laboratory. The Institutional Review Board at the University of Toledo approved all recruitment methods.

3.3 Instrumentation

1. The BIOPAC MP150 (BIOPAC Systems Inc., Goleta CA, USA) was used to convert analog electromyography signal (used in spinal reflexive and corticomotor testing) to
digital signal, which was used for data processing. Additionally, this piece of equipment was used to trigger the electrical stimulus needed to perform the spinal reflex testing.

2. A Magstim Rapid (Magstim Company, Wales, UK) was used to elicit motor evoked potentials from the brain to collect corticomotor excitability measurements.

3. Biodex System II Pro dynamometer (Biodex Medical Systems, Shirley, NY) was used to measure force data for the voluntary isometric contraction torque to perform corticomotor excitability measurements.

3.4 Procedures

Subjects reported to the lab where all outcome measures were conducted prior to and immediately after the interventions.

3.4.1 Outcome Measures

**Spinal Reflex Excitability:** Measurements were conducted in 3 muscles around the ankle including the tibialis anterior, fibularis longus and soleus as previously published by the faculty advisor. Participants were positioned in a dynamometer (Biodex System II Pro dynamometer, Biodex Medical Systems, Shirley, NY) with hips flexed to 85°, knees flexed to 10° and the ankle plantar flexed to 10°. The lower limb was supported on a padded supporting arm and the calcaneus was secured in a rubber heel cup mounted on a flat platform (See Figure 3-1). Two 10mm pre-gelled Ag/AgCl EMG (BIOPAC Systems, Inc., Goleta CA, USA) electrodes were positioned 1.75 mm apart over the selected muscle bellies. Electrodes were placed at the midpoint of the muscle belly for the tibialis anterior. For the fibularis longus, the EMG electrodes will be
placed 2 to 3 cm distal to the fibular head. The soleus electrodes was placed on the midline of the muscle belly over the distal third of the lower leg. A ground electrode was placed over the involved medial malleolus. A 2mm shielded disc stimulating electrode (EL254S BIOPAC Systems, Inc., Goleta CA, USA) was positioned over the popliteal space as previously reported in order to stimulate the sciatic nerve prior to its split into the common fibular and tibial nerves. Analog to digital signal conversion was processed with a 16-bit convertor (MP150, BIOPAC Systems Inc., Goleta CA, USA). The Acqknowledge BIOPAC Software (MP150, BIOPAC Systems Inc., Goleta CA, USA) was used to visualize the signals, as well as manipulate the stimuli. EMG signals were sampled at 2000 Hz and EMG amplification was set at a gain of 1000 (EMG100C BIOPAC Systems, Inc., Goleta CA, USA), interfaced with a 200-volt maximum stimulus isolation adaptor (STIMISOC BIOPAC Systems, Inc., Goleta CA, USA). Maximal Hoffmann reflexes were normalized to the maximal muscle responses creating a H:M ratio which were used for analysis.

![Figure 3-1](image.jpg)

Figure 3-1: Subject seated on the dynamometer with hips flexed to 85°, knees flexed to 10° and the ankle plantar flexed to 10°. This position is used for both spinal and corticomotor
Corticmotor Excitability: Participants were positioned in a dynamometer as previously explained during the spinal reflex excitability measurements. Before testing, participants were given a lycra swim cap (Sprint Aquatics, Rothhammer International Inc. San Luis Obispo, CA), used to mark reference lines for the stimulation, and earplugs, used to muffle the sound of the stimulator. Two lines were drawn onto the swim cap, one running from the nose to the occiput (separating the hemispheres sagittally) and one running from the apex of one ear to the other (bisecting the other line). The intersection of these two lines is the vertex of the skull, used as a reference point of the motor cortex. The 10mm pre-gelled Ag/AgCl EMG electrodes were positioned at the same points as previously stated for Hoffmann reflex testing. Participants performed submaximal contractions at five percent of the maximal isometric quadriceps contraction in order to standardized volitional muscle contraction during active motor threshold/MEP testing. A double cone coil (Magstim Company, Wales, UK) was positioned over the vertex of the cranium and a Magstim rapid (Magstim Company, Wales, UK) was used to produce a maximum magnetic stimulus of 1.4 Tesla (See Figure 3-2). With the stimulator over the vertex, the researcher moved the coil anterior to posterior until the largest peak-to-peak motor evoked potential was produced using a constant stimulus. Motor threshold was assessed as the lowest intensity required to elicit a MEP of $\geq 100\mu V$ amplitude in at least 5 of 10 trials in the tibialis anterior, fibularis longus, and soleus. Five MEPs at 100% and 120% of AMT were collected and normalized to the M-wave then used in data analysis.
3.4.2 Interventions

**Grade IV Posterior Talar Joint Mobilization:** A single Certified Athletic Trainer with six years of experience, licensed in the state of Ohio performed the posterior talar mobilization. Group allocation was concealed in an opaque envelope, which was opened by the athletic trainer immediately prior to the intervention and disposed of in her personal office. Participants were seated on a plinth while the therapist stabilized the distal tibia with one hand and made contact with the anterior talus with the opposite hand (See Figure 3). The therapist applied three 60-second grade IV anterior to posterior joint mobilizations of the talus with one-minute rest in between sets. These were low amplitude mobilizations at the end range of the physiological range of motion.

Figure 3-2: After location of largest peak-to-peak motor evoked potential the Magstim coil was secured to prevent movement. Notice the proximity of the treatment table to the dynamometer. The location minimized the amount of patient movement between outcome measure testing and the intervention.
Control: Participants were positioned the same as the experimental intervention yet the therapist did not contact the participant during the session. The participant sat quietly for the same period of time needed to perform the joint mobilization intervention.

3.5 Data Analysis

Maximal Hoffmann reflexes and muscle responses were processed by a blinded experienced independent investigator; who assessed peak-to-peak amplitudes of both the Hoffmann reflexes and muscles response measurements in all three muscles. Once three maximal Hoffmann reflexes and muscle responses were obtained a H:M ratio was calculated from the means of the three measurements. Active motor thresholds were obtained as previously indicated and imputed into a data spreadsheet by a blinded investigator.

3.6 Statistical Analysis

A priori alpha levels were set at P≤ 0.05 for all inferential statistics, which were evaluated using SPSS 17.0 statistical software. Dependent samples t-tests were used to

Figure 3-3: A certified Athletic Trainer performed the posterior talar joint mobilizations on a padded plinth.
evaluate differences within groups before and after treatments in the tibialis anterior, fibularis longus and soleus muscles for both reflex and corticomotor excitability outcome measures. Standardized effect sizes with 95% confidence intervals were calculated to evaluate the magnitude of the changes in reflexive and corticomotor excitability outcomes between pretest and posttest scores for the two intervention conditions.
Chapter Four

Results

Means and standard deviations for all variables at pre-test and post-test time intervals are found in Table 4.1. Significance and Cohen’s $d$ within-group effect sizes with respective 95% confidence intervals are in Table 4.2.

4.1 Spinal Reflex Excitability

The H:M ratios for the tibialis anterior (p=0.40), fibularis longus (p=0.61), and soleus (p=0.25) muscles of the mobilization group were not significantly different before and after joint mobilization (Table 4.1). Small-negative effect sizes with wide 95% confidence intervals (CIs) crossing 0 were found for the tibialis anterior ($d$=-0.11, 95% CI: -1.09, 0.88), fibularis longus ($d$=-0.08, 95% CI: -1.06, 0.90), and soleus ($d$=-0.25, 95% CI: -1.22, 0.75). (Table 4.2)

No differences were detected for the H:M ratios for the tibialis anterior (p=0.51), fibularis longus (p=0.37), and the soleus (p=0.77) following the control intervention (Table 4.1). Weak-positive effect sizes with CIs crossing 0 were found for the tibialis anterior ($d$=0.27, 95% CI: -.80, 1.30) and soleus ($d$=0.11, 95% CI: -0.87, 1.09). A small-negative effect was observed in the fibularis longus ($d$=-0.32, 95% CI: -1.29, 0.69; Table 4.2).
4.2 Corticomotor Excitability

Soleus active motor thresholds (AMT) decreased from 54.1 to 49.6 following joint mobilization. Although not statistically significant (p=0.20), this produced a strong effect size ($d=-0.71$ 95% CI: -1.67, 0.34) implying a potential clinically relevant increase in corticomotor excitability (Table 4.1). However, the control group presented with a similar magnitude in decrease, but due to the variability this produced a small effect size. There were no statistically significant differences observed in the mobilization group’s AMT for the tibialis anterior (p=0.60) or fibularis longus (p=0.80; Table 2). The effect sizes for the tibialis anterior and fibularis longus were weak with expansive 95% CIs crossing 0 (Table 4.2).

The amplitude of the motor evoked potentials (MEPs) at 120% of the subject’s AMT was not significantly different pre to post for any of the three lower leg muscles (TA p=0.44, FL p=0.97, Sol p=0.93). Normalizing the 120% MEPs to the subject’s M-wave did not produce any statistical significance (TA p=0.51, FL p=0.59, Sol p= 0.92). All effect sizes were weak with wide 95% CIs that crossed 0. (Table 4.2)

All corticomotor excitability variables for the control group (AMT, 120 amplitude, and 120 normalized to M-wave) were statistically insignificant for each of muscle. The effects sizes produced were weak with large 95% CIs crossing 0. (Table 4.2)
Table 4.1: Pre and post intervention means and standard deviations for the Joint Mobilization and Control group. AMT measured in % Tesla, MEP 120 measured in µV.

<table>
<thead>
<tr>
<th></th>
<th>Joint Mobilization</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
</tr>
<tr>
<td></td>
<td>Avg</td>
<td>n</td>
</tr>
<tr>
<td>TA</td>
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<tr>
<td>AMT</td>
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<tr>
<td>MEP 120</td>
<td>0.224</td>
<td>8</td>
</tr>
<tr>
<td>MEP 120:M</td>
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<td>8</td>
</tr>
<tr>
<td>FL</td>
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<td></td>
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<tr>
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</tr>
<tr>
<td>MEP 120</td>
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</tr>
<tr>
<td>MEP 120:M</td>
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<td>8</td>
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<tr>
<td>Sol</td>
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<td></td>
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<tr>
<td>H:M Ratio</td>
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<td>8</td>
</tr>
<tr>
<td>AMT</td>
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</tr>
<tr>
<td>MEP 120</td>
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<td>8</td>
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<td>MEP 120:M</td>
<td>0.045</td>
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</table>
Table 4.2: Significance and effect sizes with respective 95% confidence intervals for the joint mobilization and control group.

<table>
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<tr>
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<th>95 % CI</th>
<th></th>
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<tr>
<td></td>
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<td>95 % CI</td>
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<td>Effect Size ($d$)</td>
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<td></td>
<td></td>
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<td></td>
<td>Lower</td>
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<td>AMT</td>
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<td>H:M Ratio FL</td>
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<td>-0.03</td>
<td>-1.01</td>
<td>0.95</td>
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</table>
Chapter 5

Discussion

This pilot study was conducted to investigate the effectiveness of a posterior talar joint mobilization on altering spinal reflexive and corticomotor excitability in subjects with chronic ankle instability (CAI). Due to our small sample size, the current study is significantly underpowered; thus our conclusions are based on effect sizes and 95% confidence intervals rather than inferential statistics. Altered excitability has been reported as an impairment manifested in people with CAI, which also presents a sequela that includes altered functional performance, reduced joint range of motion, and impaired joint arthrokinematics. Since CAI is a leading risk factor of posttraumatic osteoarthritis in the ankle,\textsuperscript{45,46} establishing interventions that alleviate these maladaptive joint changes can potentially reduce joint degeneration.\textsuperscript{31} No statistical significance between any of the variables for both our mobilization and control group, but the joint mobilization decreased the soleus active motor threshold (AMT) enough to produce a strong effect size. However, the control group did produce a similar mean change in soleus AMT, but the variability was very high which lowered the effect size. Reduction of AMT is indicative of an increase in corticomotor excitability because decreased stimulus intensity is required to produce an efferent muscle response. This upregulation of corticomotor excitability will allow the brain to respond to stimuli more efficiently, which will create more specified motor execution at the peripheral joints.
The leading risk factor for suffering an ankle sprain is a previous history of ankle sprain. Due to this, up to 75% of people who sprain their ankle may develop CAI.\textsuperscript{45,46} This may be contributed to a mechanical malady sustained by this population that is never resolved following the initial injury. Mulligan\textsuperscript{25} originally purported that incurring a sprain at the ankle will result in an altered positional fault of the bony structures at the joint. Recent radiographic evidence has solidified this theory, with discovery of possible positional faults at the talus\textsuperscript{31} and fibula\textsuperscript{47} relative to the tibia in patients with CAI. Improper bony alignment disrupts the natural arthrokinematics occurring at the joint. Denegar et al\textsuperscript{29} demonstrated that full osteokinematic dorsiflexion can be obtained without proper restoration of posterior talar glide. This achievement of full ROM without restored arthrokinematics results from excessive stretching of the plantarflexors, excessive motion at the surrounding joints in the ankle, or an abnormal axis of rotation at the talocrural joint.\textsuperscript{29} These mechanical adaptations following injury may help to restore motion at the joint, but create a cascade of neuromuscular co-morbidities that impair functional performance. Alterations in resting length of the musculature surrounding an injured joint as a result of positional faults may change the normal length/tension relationship of the musculature. Disruptions of optimal length/tension relationships may diminish the effectiveness of the musculature to produce contractions needed to provide dynamic stability of the joint.

Bony malalignment at the ankle may also produce altered afferent activity that hinders the efferent response of the surrounding musculature, creating a disruption in the normal mechanics of multiple joints. This arthrogenic manifestation has been established in the ankles of patients with CAI; due to the decrease in spinal reflex excitability.\textsuperscript{10,16}
This investigation demonstrates the potential of joint mobilizations at restoring corticomotor excitability that may be diminished following injury. Additionally, CAI patients have exhibited altered neural excitability of muscles surrounding proximal joints.\textsuperscript{48} Sedory et al\textsuperscript{48} identified an inhibition of the hamstrings with concurrent facilitation in the ipsilateral quadriceps in patients with CAI. These neural alterations could be the culprit for functional consequences seen at the proximal joints; such as decreased peak torque at the knee\textsuperscript{49} and deficits in dynamic postural control.\textsuperscript{50,51} Thus, it is imperative to find an intervention that alleviates the positional fault procured during initial ankle injury in order to prevent multiple suboptimal neural adaptations that may follow.

There is evidence that manual therapy techniques applied to the ankle can target central nervous system dysfunction in subjects with chronic ankle instability.\textsuperscript{52} Grindstaff et al\textsuperscript{52} displayed that a mobilization at the distal tibiofibular joint could increase spinal excitability of the soleus muscle. Despite our small sample size, we showed that there may be a clinically relevant increase in corticomotor excitability in the soleus musculature following a posterior mobilization of the talocrural joint. An inclusion criterion for CAI is the functional sensation of the ankle “giving way” during activity. Restoring corticomotor excitability may be imperative to correcting this conscious instability by allowing this population to unconsciously ameliorate dangerous joint positions before the perceived “giving way” of the ankle. The use of a disinhibiting modality, like a joint mobilization, following joint injury may also augment the gains sustained from rehabilitation.\textsuperscript{1,15} The implementation of modalities to neurally “unlock”
muscles prior to traditional strength training may allow for greater improvements than the current paradigm that does not include disinhibiting modalities.

Currently, this pilot study is substantially under-powered statistically; therefore our claims are based off of effect sizes rather than inferential statistics. Even though we did not have enough subjects to produce statistical significance, our change in soleus active motor threshold did produce a strong effect size. However, the respective 95% confidence intervals (CIs) were wide and cross zero, thus limiting the clinical applicability. All of the other variables were not statistically significant and revealed only small effect sizes. The limited time between the pre and post-testing can be seen as a limitation of the study. Perhaps the nervous system needs an extended period to adapt to the restoration of afferent input to create more robust corticomotor alteration. Also, the neural excitability measures were tested in a non-functional environment, which may reduce our ability to generalize our findings.

CAI is an intricate problem that can be modulated by many functional and mechanical disruptions of the neuromuscular system. Due to this, there is the possibility that joint mobilizations will not be capable of targeting the specific proponents that have created the manifestation of CAI in all cases. Perhaps only the people with detriments in arthrokinematics will be able to reap the neuromuscular benefits of this manual therapy technique. The development of methods to more accurately measure arthrokinematics will allow us to administer the most appropriate intervention for CAI patients. Performing a joint mobilization on a patient without an arthrokinematic deficit is not targeting the source of their instability. Further, the correct mobilization needs to be applied relative to the positional fault that is present. There is evidence of a potential
fault occurring at either the talus or the distal fibula following ankle sprain.\textsuperscript{31,47} Researchers are developing techniques to more reliably measure arthrokinematics of both the talus and the fibula.\textsuperscript{53} Studies utilizing more stringent inclusion criteria and investigating the efficacy of mobilizations on a population with the corresponding positional fault will allow for a more efficient and individually tailored rehabilitation protocol following injury. Future studies should also investigate how a mobilization with movement (MWM) may affect neural dysfunction, since it has shown potent effectiveness with ROM.\textsuperscript{7} This study utilized a Maitland mobilization, but a MWM may produce a more robust alteration. Since the subjects in our study did not walk in between receiving the mobilization and post testing, their motor cortex may have never been able to interpret the new afferent signals sent from the corrected joint positioning. The introduction of a long-term study that allows the subjects to complete functional activities after receiving the mobilization may allow for the neural plastic changes to occur in the brain to allow for a greater change in neural excitability.

From the results of this study, we conclude that a posterior talar joint mobilization can potentially increase corticomotor excitability of the soleus muscle in subjects with chronic ankle instability. The correction of the positional fault of the talus that was suffered following the initial injury is the theorized origin of this alleviating effect. Interventions that produce this disinhibitory effect may reduce functional instability associated with CAI, as well as augmenting rehabilitation gains when coupled with traditional strengthening exercises.
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Considerations and Applications for Use in Sports Medicine and Athletic Training

simulated ankle joint effusion. *British Journal of Sports Medicine.* 10/24/10

measurements arising from the soleus, peroneal, and tibialis anterior musculature.


21. Hopkins JT, Ingersoll CD, Edwards J, Klootwyk TE. Cryotherapy and
transcutaneous electric neuromuscular stimulation decrease arthrogenic muscle


Appendix A: Consent Form

ADULT RESEARCH SUBJECT INFORMATION AND CONSENT FORM

THE EFFECTS OF JOINT MOBILIZATIONS ON LOWER LEG REFLEX EXCITABILITY AND DYNAMIC STABILITY IN PEOPLE WITH CHRONIC ANKLE INSTABILITY

Principal Investigator: Brian Pietrosimone PhD, ATC

Other Staff (Co-Investigator): Phillip Gribble PhD, ATC, Masafumi Terada MS, ATC, Michelle McLenn M.S. PhD, Matthew Harkey ATC, Ashley Wallis ATC

Contact Phone number(s): (419) 530-4467

What you should know about this research study:

- We give you this consent/authorization form so that you may read about the purpose, risks, and benefits of this research study. All information in this form will be communicated to you verbally by the research staff as well.

- Routine clinical care is based upon the best-known treatment and is provided with the main goal of helping the individual patient. The main goal of research studies is to gain knowledge that may help future patients.

- We cannot promise that this research will benefit you. Just like routine care, this research can have side effects that can be serious or minor.

- You have the right to refuse to take part in this research, or agree to take part now and change your mind later.

- If you decide to take part in this research or not, or if you decide to take part now but change your mind later, your decision will not affect your routine care.

- Please review this form carefully. Ask any questions before you make a decision about whether or not you want to take part in this research. If you decide to take part in this research, you may ask any additional questions at any time.

- Your participation in this research is voluntary.

PURPOSE (WHY THIS RESEARCH IS BEING DONE)
You are being asked to take part in a research study looking at the nerve function of leg, balance and self-reported pain, stiffness and stability. The purpose of the study is to determine if people with previous ankle injuries will have changes in reflex and brain activation, balance, range of motion and self reported stiffness, pain and stability following small movements to the ankle joint. You were selected as someone who may want to take part in this study because you have chronic ankle instability. There will be approximately 50 people participating in this study at the University of Toledo.
DESCRIPTION OF THE RESEARCH PROCEDURES AND DURATION OF YOUR INVOLVEMENT

If you decide to take part in this study, you will be asked to report to the Joint Injury and Muscle Activation (JIMA) Laboratory in the Health Science and Human Services building (Room 1409). You will be asked to fill out Ankle Injury Questionnaires about how your ankle feels during different activities. You will also be asked to fill out an Exclusion Criteria Screening Sheet regarding your history of injury and rehabilitation to your lower extremity; joint hypermobility or connective tissue disorders; concussion or head injuries; stroke; heart condition; cranial neurosurgery; epilepsy; migraines; cancer in the brain or thigh musculature; diagnosed psychiatric disorder; cardiac pacemaker placement; implanted cardiac defibrillator; and/or intracranial metallic clips.

After filling out the Ankle Injury Questionnaires and Exclusion Criteria Screening Sheet, we will then test the neural function of both of your legs using Reflex Testing and brain testing and have you perform some range of motion and balance tests. You may then be randomized to have small joint movements applied to your ankle or you may be asked to sit quietly for a short period of time. After you receive the joint mobilizations or sit for a period of time you will again perform the reflex, balance testing and ankle questionnaires. This study will consist of one session lasting approximately 1.5 hours.

Ankle Injury Questionnaires
• You will be asked to provide us information regarding your previous history of your joint injury, current and past level of activity and how your joint injury currently affects you during different activities. You will also let us know how stiff, stable and painful you ankle feels during two points in the study. Your will be excluded from this study if you have any of the following: Injury to the lower extremity other than the ankle in the previous six months, Rehabilitation for a lower extremity injury in the last 6 months, joint hypermobility of connective tissue disorder, history of concussion or head injury in the past 6 months, history of stroke, cardiac condition, epilepsy, cranial neurosurgery, migraines, cancer in the brain or thigh musculature, diagnosed psychiatric disorder; or has a cardiac pacemaker, implanted cardiac defibrillator or intracranial metallic clips.

Reflex Testing
This testing provides an estimate of how well nerves in the lower leg are functioning. You will be instructed to stand on your dominant leg or lie on a table. You will have sticky electrodes placed on your lower legs and thigh. These electrodes are called EMG electrodes which stand for Electromyography which is a recording of the electrical (reflex) activity in skeletal muscle. The sites of the EMG electrodes will be shaved and cleaned with alcohol. An electrode that provides a stimulus will be taped behind your knee and in the front of your hip. Several reflex measurements will be taken while you are balancing or lying down.
• These measurements include a 1-millisecond stimulus.
• The intensity of this stimulus will vary depending on the reflex being elicited.
• The stimuli in this study feel similar to static electricity felt as you touch a door knob after walking across a carpet.
• A series of measurements will be taken on both legs

Brain Testing
This testing provides us important information regarding how your brain is sending messages to muscles in your legs. You will be asked to lie on a table with your hands at your side. We will position a coil over your head and adjust the position of the coil until it is in the correct spot. We will ask you to wear a bathing cap and ear plugs. A brief magnetic stimulus will then be produced which will sound like a “click.” You will not have and associated pain or discomfort in your head, but rather may feel a brief
muscle contraction in the muscles of your leg or thigh. You will be asked to flex certain leg muscles at a small to moderate intensity while we provide a series of brief magnetic stimuli to your head.

**Balance Testing**

A member of the research team will demonstrate the dynamic balance test, called the Star Excursion Balance Test (SEBT). The SEBT requires you to stand on one leg in the middle of a grid on the floor and then try to reach with the other leg to touch a spot on the floor as far as you can along a line on the grid. If you lose your balance, put too much weight on your reaching foot or move the foot of the leg you are standing on, the reaching trial is repeated. After the demonstration, you will practice the SEBT standing on their right leg 6 times and then on their left leg 6 times so that they can become familiar with how to perform the test. Then will be given 5 minutes to rest.

**Range of Motion Testing**

We will test how far you are able to comfortably move your ankle. You will first be asked to point your toes by moving your ankle joint toward your head as far as you feel comfortable. We will measure this in a standing position and while you are lying on your back.

**Joint Mobilizations**

You may be asked will lie down on a padded table while a member of the research team apply mild pressure to the front of the ankle. You will not feel pain in your ankle during this technique, yet you may feel pressure. If you do feel pain you should notify the investigator.

**RISKS AND DISCOMFORTS YOU MAY EXPERIENCE IF YOU TAKE PART IN THIS RESEARCH**

**Likely Risks**
- Mild discomfort for a very brief period during the electrical stimulation.

**Less Likely Risks**
- Mild, transient skin irritation from the sticky electrodes.

**Very Unlikely Risks**
- Mild, transient soreness from the joint mobilizations
- Possible risk that you fail when you are performing the balance testing.
- Mild, transient headache following magnetic stimulation
- In people with a history of seizures there is a slight possibility of causing a seizure with the magnetic stimulation; therefore you must tell us prior to testing if you have ever had a seizure so we can exclude you from the study.

**RISKS TO UNBORN CHILDREN**

It is unknown how the electrical stimulation used in this study would affect an unborn fetus; therefore, if you are pregnant you will not be allowed to participate in this study.

**POSSIBLE BENEFIT TO YOU IF YOU DECIDE TO TAKE PART IN THIS RESEARCH**

Although information that is gained from this research that may be used to assess and treat various ankle injuries, we cannot and do not guarantee or promise that you will receive any benefits from this research.

**COST TO YOU FOR TAKING PART IN THIS STUDY**

You are not directly responsible for making any type of payment to take part in this study. However, you are responsible for providing the means of transportation to the Joint Injury and Muscle Activation Laboratory. You will not be compensated for gas for travel or any other expenses to participate in this study.
PAYMENT OR OTHER COMPENSATION TO YOU FOR TAKING PART IN THIS RESEARCH
You will not be compensated for participating in this study.

ALTERNATIVE(S) TO TAKING PART IN THIS RESEARCH
The only alternative is not to participate in this study.

CONFIDENTIALITY
The researchers will make every effort to prevent anyone who is not on the research team from knowing that you provided this information, or what that information is. The consent forms with signatures will be kept separate from responses, which will not include names and which will be presented to others only when combined with other responses. Although we will make every effort to protect your confidentiality, there is a low risk that this might be breached.

IN THE EVENT OF A RESEARCH-RELATED INJURY
In the event of injury resulting from you taking part in this study, treatment can be obtained at a health care facility of your choice. You should understand that the costs of such treatment will be your responsibility. Financial compensation is not available through The University of Toledo or The University of Toledo Medical Center. By signing this form you are not giving up any of the legal rights of your son/daughter/legal charge as a research subject. In the event of an injury, contact Brian Pietrosimone, PhD, ATC (419) 530-4487

VOLUNTARY PARTICIPATION
Taking part in this study is voluntary. You may refuse to participate or discontinue participation at any time without penalty or a loss of benefits to which you are otherwise entitled. If you decide not to participate or to discontinue participation, your decision will not affect your future relations with the University of Toledo or The University of Toledo Medical Center

NEW FINDINGS
You will be notified of new information that might change your decision to be in this study if any becomes available.

Continued On Next Page
OFFER TO ANSWER QUESTIONS
Before you sign this form, please ask any questions on any aspect of this study that is unclear to you. You may take as much time as necessary to think it over. If you have questions regarding the research at any time before, during or after the study, you may contact: Dr. Brian Pietrosimone- (419) 530-4467. If you have questions beyond those answered by the research team or your rights as a research subject or research-related injuries, please feel free to contact the Chairperson of the University of Toledo Biomedical Institutional Review Board at 419-383-6796.

SIGNATURE SECTION (Please read carefully)
YOU ARE MAKING A DECISION WHETHER OR NOT TO PARTICIPATE IN THIS RESEARCH STUDY. YOUR SIGNATURE INDICATES THAT YOU HAVE READ THE INFORMATION PROVIDED ABOVE, YOU HAVE HAD ALL YOUR QUESTIONS ANSWERED, AND YOU HAVE DECIDED TO TAKE PART IN THIS RESEARCH.

BY SIGNING THIS DOCUMENT YOU AUTHORIZE US TO USE OR DISCLOSE YOUR PROTECTED HEALTH INFORMATION AS DESCRIBED IN THIS FORM.

The date you sign this document to enroll in this study, that is, today’s date, MUST fall between the dates indicated on the approval stamp affixed to the bottom of each page. These dates indicate that this form is valid when you enroll in the study but do not reflect how long you may participate in the study. Each page of this Consent/Authorization Form is stamped to indicate the form’s validity as approved by the UT Biomedical Institutional Review Board (IRB).

Name of Subject (please print)  Signature of Subject or Person Authorized to Consent  Date

Relationship to the Subject (Healthcare Power of Attorney authority or Legal Guardian)  Time

Name of Person Obtaining Consent (please print)  Signature of Person Obtaining Consent  Date

Name of Witness to Consent Process (when required by ICH Guidelines) (please print)  Signature of Witness to Consent Process (when required by ICH Guidelines)  Date

YOU WILL BE GIVEN A SIGNED COPY OF THIS FORM TO KEEP.
Appendix B: FADI/FADI Sport

Foot and Ankle Disability Index (FADI)

Please answer every question with the response that most closely describes your condition over the past week.

If the activity limitation is limited by something other than your foot or ankle treat "not applicable" (NA).

<table>
<thead>
<tr>
<th>Activity</th>
<th>Ability</th>
<th>Speed</th>
<th>Stairs</th>
<th>Situp</th>
<th>Stool</th>
<th>NA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Standing</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
<tr>
<td>Walking on even ground</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
<tr>
<td>Walking on even ground altar</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
<tr>
<td>Walking up hill</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
<tr>
<td>Walking down hill</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
<tr>
<td>Going up stairs</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
<tr>
<td>Going down stairs</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
<tr>
<td>Walking on uneven ground</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
<tr>
<td>Sleeping</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
<tr>
<td>Sitting</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
<tr>
<td>Sleeping</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
<tr>
<td>Coming upon your toes</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
<tr>
<td>Walking, fast</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
<tr>
<td>Walking 5 minutes or less</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
<tr>
<td>Walking approximately 15 minutes</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
<tr>
<td>Walking 15 minutes or greater</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
</tbody>
</table>

Approved by
UNIVERSITY OF TOLEDO IRB
Due to your foot and ankle, how much difficulty do you have with:

<table>
<thead>
<tr>
<th>Activity</th>
<th>No</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
<th>Unce sure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Home responsibilities</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Activities (light work)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Personal care</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Light to moderate work</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heavy work (sweating)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Recreational Activities</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Please rate your pain level as it relates to your foot and ankle:

<table>
<thead>
<tr>
<th>Activity</th>
<th>No</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
<th>Unce sure</th>
</tr>
</thead>
<tbody>
<tr>
<td>General level of pain</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Activity</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>During your normal activity</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Starting or the morning</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

FADI Sports Scale

Because of your foot and ankle, how much difficulty do you have with:

<table>
<thead>
<tr>
<th>Activity</th>
<th>No</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
<th>Unce sure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Running</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Throwing</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Landing</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Starting and stopping</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cutting and jumping</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Use of sport equipment</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ability to perform activity with your usual technique</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ability to participate in your usual sport as long as you exercise less</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Approved by

UNIVERSITY OF TOLEDO IRB
Appendix C: Exclusion Criteria Screening Sheet

Please indicate if you have a history of any of the following conditions, experiences or pathologies. If 'yes' please briefly explain:

<table>
<thead>
<tr>
<th></th>
<th></th>
<th>Explain</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>No</td>
<td>Injury to the lower extremity other than the ankle in the previous 6 months</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Rehabilitation for a lower extremity injury in the last 6 months</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Joint hypermobility of connective tissue disorder</td>
</tr>
<tr>
<td></td>
<td></td>
<td>concussion or head injury in the past 6 months</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Stroke</td>
</tr>
<tr>
<td></td>
<td></td>
<td>A heart condition</td>
</tr>
<tr>
<td></td>
<td></td>
<td>cranial neurosurgery</td>
</tr>
<tr>
<td></td>
<td></td>
<td>epilepsy</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Migraines</td>
</tr>
<tr>
<td></td>
<td></td>
<td>cancer in the brain or thigh musculature</td>
</tr>
<tr>
<td></td>
<td></td>
<td>diagnosed psychiatric disorder</td>
</tr>
<tr>
<td></td>
<td></td>
<td>cardiac pacemaker</td>
</tr>
<tr>
<td></td>
<td></td>
<td>implanted cardiac defibrillator</td>
</tr>
<tr>
<td></td>
<td></td>
<td>intracranial metallic clips</td>
</tr>
</tbody>
</table>