The effects of cryotherapy on quadriceps corticospinal excitability in patients with patellofemoral pain

Christopher D. Johnston

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The Effects of Cryotherapy on Quadriceps Corticospinal Excitability in Patients with Patellofemoral Pain

by

Christopher D. Johnston, ATC

Submitted to the Graduate Faculty as partial fulfillment of the requirements for the Master of Science Degree in Exercise Science

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An Abstract of

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Objective: The purpose of this study was to determine if cryotherapy can alter quadriceps corticospinal excitability in patients with patellofemoral pain. Design and Setting: A blinded, crossover study was conducted in a laboratory setting. Subjects: Eight patients (1 male, 7 females; 19.6±1.11 years; 163.37±10.27 cm; 59.53±10.11 kg) with patellofemoral pain received a control and cryotherapy intervention. The order in which the patients received the interventions was randomly assigned between two sessions. Procedure: Outcome measures were administered before the intervention and at 10, 20, 35, and 50 minutes after the start of the intervention for both sessions. Patients were positioned in a Biodex System III Pro dynamometer during excitability testing. Corticospinal excitability was estimated with transcranial magnetic stimulation by collecting five motor evoked potentials (MEPs) at active motor threshold (AMT), 120% of AMT, and at 120% of baseline AMT. MEP’s were normalized to maximum muscle response. During the cryotherapy condition, a Certified Athletic Trainer applied two, 1.5 liter ice bags, to the anterior and posterior aspect of the knee for 20 minutes. Patients sat quietly in the same position for 20 minutes during the control session. Separate 2x4 ANOVA’s with repeated measures on time were conducted to determine differences
between conditions over time for all three dependent variable percent change scores. Paired t-tests were conducted in the occurrence of a significant interaction. Cohen’s $d$ effect sizes with 95% confidence intervals were calculated for pre to posttest change scores for the cryotherapy and control conditions. **Results:** No significant main effects were found for condition or time for all variables. There was a significant interaction for time by condition for changes in AMT ($F_{1,1.951}=4.483, p=.021$). Post-hoc paired t-tests indicated no significant changes in AMT from baseline for all time points. A moderate effect ($d=-0.41$, 95% confidence interval -1.40, 0.64) occurred at 50 minutes into the cryotherapy condition for changes from baseline, but the confidence interval crosses zero. **Conclusion:** From the results of this study, we can conclude that cryotherapy may not have an effect on corticospinal excitability of the quadriceps in patients with patellofemoral pain. Although it does not seem that cryotherapy affects corticospinal excitability, it is imperative to continue researching possible solutions to enhance corticospinal excitability following joint injury.
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Chapter 1

Introduction

1.1 Introduction

Following lower extremity joint injury, reestablishing optimal neuromuscular control is vital in preventing long term disabilities. Arthrogenic muscle inhibition (AMI) is a common condition following joint injury that inhibits uninjured musculature surrounding an injured joint, which affects the muscles ability to activate motor neurons for muscle contraction. AMI has been demonstrated to manifest in two different neural pathways, including spinal reflexive and corticospinal.

Following knee injury numerous studies have reported activation deficits and decreases in α-motor neuron excitability in the quadriceps. Patients with anterior knee pain (AKP) are shown to have inhibition of the quadriceps. Hart et al. demonstrated that quadriceps activation is significantly reduced in patients presenting with AKP. Patients with AKP averaged lower levels of quadriceps activation than patients with ligamentous injuries as well. Inhibition of the quadriceps can result in improper kinematics and decreased strength that may lead to long term degradation of the knee joint. Rehabilitation paradigms focusing on certain therapeutic interventions that target neurological impairments which cause muscle dysfunction, along with traditional strength training may enhance patient rehabilitation outcomes.
Cryotherapy is a modality that has been studied and shown to have disinhibitory properties, as well as increasing strength and muscle activation in pathological patients.\textsuperscript{1,2,8,13,14} Cryotherapy demonstrated an increase spinal reflexive excitability through the measurement of the H-reflex.\textsuperscript{13} Cryotherapy shows increased excitability immediately following treatment and effects are shown to last for up to an hour.\textsuperscript{13} In a knee effusion study, cryotherapy not only increased excitability back to baseline measures, but facilitated excitability above baseline measures for spinal reflex excitability.\textsuperscript{13} As well, cryotherapy increases the central activation ratio (CAR) in the quadriceps.\textsuperscript{8,14} In a conventional rehabilitation program, cryotherapy is typically used as a way to moderate pain. More research on this modality may provide indications for use of cryotherapy before traditional rehabilitation in order to recover more efficiently.

Though research shows that cryotherapy increases spinal reflex excitability, no studies have been conducted that show the effects of cryotherapy on the excitability of the $\alpha$-motor neuron pool through the cortical neural pathway. Hopkins et al.\textsuperscript{15} suggested that because cryotherapy facilitated quadriceps activation above baseline measures, manipulation of supraspinal pathways are likely involved. Patients with patellofemoral pain should exhibit decreased quadriceps activation and provide a good patient model for these phenomenon. Therefore, we will study the effects of cryotherapy on corticospinal excitability in patients with patellofemoral pain to help further the development of rehabilitation protocols that incorporate the reestablishment of neuromuscular control following joint injury. We will also continue to look at the effects of cryotherapy on spinal reflexive excitability as well as pain.
1.2 Statement of Purpose

The purpose of this study is to determine if cryotherapy can alter quadriceps corticospinal excitability in patients with patellofemoral pain. As a secondary aim, we will further investigate its effects on spinal reflexive excitability and pain.

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 the vastus medialis

2) Cortical Excitability measured by Transcranial Magnetic Stimulation

   - Motor evoked potentials (MEP) at 120% of active motor threshold for vastus medialis

3) Visual Analog Pain Scale

   - Pain will be measured on a lined scale measured 0-10 with 10 being the worst pain imaginable and 0 meaning no pain. The participant will mark where on the scale their pain level is.

1.4 Independent Variables

1) Condition

   - Experimental= the session the participants receive the cryotherapy intervention.
2. Control = testing session the participants do not receive the cryotherapy intervention.

2) Time

- Pre = measurements made before clinical intervention or control session.
- 10 minutes = measurements taken at 10 minutes after the initial application of ice.
- 20 minutes = measurements taken 20 minutes after the initial application of ice. (Ice removed after 20 minutes).
- 35 minutes = measurements taken 35 minutes after initial application of ice.
- 50 minutes = measurements taken 50 minutes after initial application of ice.

1.5 Research Question

Does cryotherapy result in changes in excitability in the cortical pathway that corresponds to the vastus medialis musculature in patients with patellofemoral pain?

1.6 Null Hypothesis

Following 20 minutes of cryotherapy of the knee joint, there will be NO significant changes in excitability of the cortical neural pathway in the vastus medialis.

1.7 Research Hypothesis

Following 20 minutes of cryotherapy of the knee joint, there WILL BE a significant increase in excitability of the cortical neural pathway in the vastus medialis as
measured by active motor threshold and motor evoked potentials at 120% of the active motor threshold.

1.8 Specific Aims

1) To examine the effects of cryotherapy on the excitability of the cortical neural pathway of the vastus medialis.
   a. This aim will be accomplished by testing the cortical involvement using transcranial magnetic stimulation.
2) To examine the effects of cryotherapy on the excitability of spinal reflexive neural pathways of the vastus medialis.
   a. This aim will be accomplished by testing the spinal reflexive contributions utilizing Hoffmann reflex testing.
3) To examine the effect of cryotherapy on perceived pain.
   a. This aim will be accomplished by providing a visual analog pain scale.

1.9 Operational Definitions

- AMI – Arthrogenic muscle inhibition
  - Ongoing inhibition of musculature surrounding a joint due to injury or distention of joint; due to afferent activity disruption caused by injury
- 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 cortical contributions to neuromuscular activity.
- MEP – Motor evoked potential. Electromyographical representation used to quantify the cortical contributions to neuromuscular activity during TMS testing
- TENS- Transcutaneous electrical nerve stimulation. Electrical stimulation given to a desired area of the body that targets the afferent nerve receptors.

1.10 Significance

Traditional rehabilitation techniques, which involve only targeting strength and ROM deficits, may not restore proper neuromuscular control. Failing to restore neuromuscular control leads to further joint damage as well as functional constraints that affect the daily lives of injured individuals and these deficiencies may last through one’s lifetime if left untreated properly.\textsuperscript{2,3,9} Implementing therapeutic modalities that increase neural excitability can enhance an individual’s rehabilitation and reduce long term consequences of joint injury.\textsuperscript{1} We feel that cryotherapy will provide an increase in excitability of the cortical neural pathway. Combining these expected results with previous literature that states cryotherapy increases excitability in spinal reflexive neural pathways, could be helpful to clinicians who treat other joint injuries that lead to neuromuscular dysfunctions. With the disinhibitory effects of cryotherapy combined with therapeutic exercise, patients may see an increase in recovery time and efficiency due to increases in excitability from both spinal reflexive and corticospinal neural pathways.
Chapter 2

Literature Review

2.1 Introduction

The purpose of this literature review is to summarize neuromuscular consequences of knee injury. Understanding neural dysfunction associated with knee injury can allow for the development of new rehabilitation paradigms to enhance patient outcomes and prevent long-term disability.

2.2 Arthrogenic Muscle Inhibition

Following joint injury, a common neuromuscular response is arthrogenic muscle inhibition (AMI). 1-8 AMI presents as a decreased ability to voluntarily contract muscle following joint injury, although no damage has occurred to either the muscle or the nerve innervating the muscle. 2-6,10,12,16 Literature shows that AMI is closely associated with muscle weakness and atrophy following injury. 2,5 Following an acute injury, AMI may contribute to long term joint degradation. 2,3,9 It is important for clinicians to understand the neural mechanisms of AMI and incorporate this into the rehabilitation process after a joint injury.

Following knee injury, one of the reasons quadriceps activation diminishes is AMI. 7 To better understanding why this happens, it is important to know how the knee is
innervated. Sensory receptors at the knee can be broken down into two classes, group II afferents, and group III and IV afferents. Group II afferents are classified as large myelinated nerves, with the majority of the nerve endings being Ruffini endings, Paciniform corpuscles, or Golgi tendon organ-like endings. This group of afferents is highly sensitive to mechanical stimuli such as stretching and pressure. The majority of the sensory receptors in the knee are group III and IV afferents. This group is classified as being lightly myelinated or unmyelinated and having a high firing threshold. These nerves act as nociceptors and therefore are linked to signaling that damage has occurred, or is about to occur at the knee joint. Several factors have been shown to disrupt normal afferent firing and therefore contribute to AMI. These factors include swelling, inflammation, joint laxity, and damage to the actual articular sensory receptor caused by structural damage.

Changes in afferent firing can in turn disrupt spinal reflex excitability pathways. Afferent sensory receptors are the endings of sensory nerves. When a receptor is stimulated, it creates an action potential that is sent to the cell body and along the axon until it reaches the spinal cord. In the spinal cord, the sensory nerve synapses with the interneuron. The interneuron can inhibit the α motor neuron pool. The α motor neuron pool is important for the contraction of muscle because the muscle spindle synapses directly to the α motor neuron pool causing it to become excited. If the inhibitory interneuron is signaled from sensory afferent receptors, the muscle spindle will synapse with the inhibitory interneuron causing inhibition of the α motor neuron pool. This decreases the excitability of the MN pool of the quadriceps resulting in muscle activation deficits. Decreased excitability can occur in three different pathways: Group I
non-reciprocal (Ib) inhibitory pathway, Flexion reflex, and Gamma (γ)-loop. Disruption of each pathway may lead to AMI of the quadriceps muscle. The excitability of the α-motor neuron pool also decreases due to decreased excitability from the cortical motor pathway.  

AMI in the knee has been closely related to anterior knee pain (AKP), ACL deficient knees (ALC-d), ACL reconstructed knees (ACL-r), and knee osteoarthritis (OA). Evidence suggests that not only do patients with ACL injured knees exhibit AMI in the short term but in the long term as well. Interestingly, AMI has been demonstrated in both the injured and contralateral limb. Bilateral decreases in the central activation ratio (CAR) of the quadriceps in patients who are ACL-d, ACL-r, and with AKP shows that AMI persists in the uninjured limb. CAR is assessed by having the patient maximally contract the quadriceps while delivering an electrical stimulus to the muscle to increase the activation of the muscle MN pool beyond how much the patient can activate voluntarily. The CAR measure is the ratio of voluntary MN pool activation over the electrically induced MN pool activation. Reported as a percentage, it is used to determine how much of the available MN pool an individual can volitionally activate. Quadriceps inhibition in both limbs is one possible indication that supraspinal, or cortical, mechanisms may be responsible for the decreased activation.

Decreased muscle strength is one of the many consequences that can result from a deficit in muscle activation. Following injury, the quadriceps muscle may experience inhibition, therefore leading to a decrease in force output. Atrophy is linked with decreases in quadriceps activation. Because motor units are not being fired, the muscle fibers begin to decrease in size. One of the primary quadriceps muscles
associated with weakness and atrophy is the vastus medialis.\textsuperscript{4,7} The vastus medialis is an important dynamic stabilizer of the knee joint so compromise of this muscle’s function can predispose a patient to future injury. A decrease in quadriceps activation can also be linked with altered walking and jogging biomechanics as a result of diminished range of motion.\textsuperscript{4,12} Muscle weakness alters the dissipation of forces across the knee joint and therefore changes the loading and shock absorption capabilities of the knee joint. This can lead to joint degradation and consequently OA.\textsuperscript{2-6,9,11} AMI is directly correlated with muscle weakness; therefore it can be an explanation for why patients with a history of a knee injury develop OA over time.

2.3 Hoffmann Reflex

As mentioned previously, AMI is directly associated with decreased excitability of the $\alpha$-motor neuron pool, resulting from altered afferent activity in the spinal reflexive pathway. Measuring $\alpha$-motor neuron pool excitability is one way to detect the magnitude of AMI.\textsuperscript{5,16} Measuring the Hoffmann reflex (H-reflex) has shown to be an effective way of measuring $\alpha$-motor neuron pool excitability.\textsuperscript{16}

The H-reflex is an electrical measurement that represents the spinal stretch reflex typically seen from triggering the muscle spindle.\textsuperscript{16} When a muscle lengthens, the muscle spindle is activated causing stimulation of the afferent motor neurons, signaling interneurons in the spinal cord causing excitation of the motor neuron leading to a muscle contraction. The H-reflex is a representation of this because through electrical stimulation, it bypasses the muscle spindle to directly stimulate the $\alpha$-motor neuron.\textsuperscript{16}
To elicit the H-reflex, a low level electrical stimulus is applied over the nerve that innervates the desired muscle. A low level stimulus is used because the primary nerve fibers associated with H-reflex are large diameter afferent motor neurons. The most effective stimulation duration is 1-millisecond. Electrically stimulating afferent fibers creates action potentials that travel down the afferent neurons until they reach the interneuron where they synapse on the α-motor neuron. The efferent response to the electrical stimulation, if enough of a stimulus is applied, is a muscle contraction due to the depolarization of the α-motor neuron. The contraction, measured electromyographically (EMG), is referred to as the H-reflex. Increasing the electrical stimuli causes additional afferent fibers to reach threshold, therefore, resulting in increased α-motor neuron activation and a larger H-reflex. Increasing the electrical stimulus increases the H-reflex amplitude. With increased stimulus intensity, small diameter motor axons may also be depolarized, resulting in the appearance of the muscle response, or M-wave and an eventual decrease in the H-reflex amplitude. The decrease in H-reflex is due to antidromic collision. Antidromic collision occurs when electrical signals collide as they travel along the motor axon. The H-reflex is reduced because as the electrical signal from the spinal cord travels back to the corresponding muscle, it collides with electricity traveling in the opposite direction. If the antidromic signal is larger than the reflexive afferent signal, then the collision causes the signal to stop and therefore, no H-reflex will occur. The increase in M-wave directly correlates with a decrease in the H-reflex. As the stimulus continues to increase, so will the M-wave until all efferent fibers are depolarized and the H-reflex will decrease until it is completely
The M-wave continues to increase with an increase in stimulus until the entire α-motor neuron pool is activated. At this point, the M-wave plateaus.

The maximal reflex activation ($H_{max}$) is determined by peak-to-peak H-reflex amplitude as visualized by EMG while increasing the electrical stimulus. The $H_{max}$ may be used as an estimation of the amount of α-motor neuron’s available at a given point in time. The maximal M-wave ($M_{max}$) is represented by the EMG amplitude at which the M-wave plateaus. Two primary methods have been used to normalize H-reflex in order to show accurate results in quantifying AMI. The first is representing H-reflex as a percentage of $M_{max}$. This can be beneficial because once $M_{max}$ occurs, in theory it remains stable. Therefore, any changes in H-reflex can be seen as a change in wave amplitude compared to the $M_{max}$. Another normalization process is the H:M ratio which is simply the $H_{max}$ divided by the $M_{max}$. The $H_{max}$ represents the afferent excitability while the $M_{max}$ represents the total MN pool activation. Changes in afferent activation can thus be used to quantify AMI following a joint injury.

Unipolar setup calls for the cathode to be placed over the femoral nerve while the anode is placed on the opposite side of the limb on the hamstrings. Initial stimulation intensity is low, followed by increases until maximal H-reflex amplitudes are elicited. The duration of stimulation for the best results is 1 millisecond. The frequency should be no less than every 10 seconds to reduce post-activation depression.

### 2.4 Transcranial Magnetic Stimulation

Corticospinal pathways are another neurological pathway that may contribute to AMI. Descending neural pathways in the corticospinal tract interact with different MNs
than those of the spinal reflexive pathway.\textsuperscript{2,5,17-20} Cortical neurons carry information downstream of the motor cortex to MNs and synapse, causing either a facilitating or inhibitory response.\textsuperscript{5} Transcranial magnetic stimulation (TMS) has been shown to be a reliable method in determining the amount of cortical excitability in muscles of the lower leg and the knee.\textsuperscript{17-20} TMS is the use of a magnetic stimuli over the motor cortex that induces a motor evoked potential (MEP) in a desired area of the body. The MEP resulting from TMS is recorded through surface EMG of the target muscle.\textsuperscript{17,18}

TMS is conducted by first finding the optimal stimulation point, the “hot spot,” on the motor cortex of the brain.\textsuperscript{18} Finding this spot consists of using magnetic coils used to direct magnetic energy into the motor cortex and elicit the desired MEP response.\textsuperscript{18} To find the hot spot, the coil is initially placed at the vertex of the head on the contralateral side of the target muscle to elicit MEP’s.\textsuperscript{18} The coil is moved until the stimulus evokes a MEP.\textsuperscript{18} Once the “hot spot” is found, testing can take place. The motor threshold is represented by the lowest intensity required to elicit an MEP of $\geq 100\mu V$ over a series of 5 out of 10.\textsuperscript{18} After finding the motor threshold a stimulus response (SR) curve can be created. This is done by taking the motor threshold and increasing the intensity by a percentage of the motor threshold intensity, up until a desired point. The SR curve is useful in that the curve of the line shows the correlation between MEPs and TMS intensity, where the slope indicates the level of cortical excitability.\textsuperscript{17,18}

Heroux et al.\textsuperscript{17} conducted a study on cortical excitability following unilateral knee dysfunction secondary to ACL injury. TMS intensity was increased in intervals of 5% from the motor threshold intensity until no further changes in MEP could be elicited in two consecutive intervals.\textsuperscript{17} This study demonstrated that TMS is reliable in measuring
corticospinal excitability in the knee. \cite{17} Resting motor thresholds and SR curves relative to quadriceps activation were decreased in patients with knee dysfunction when compared to healthy patients. Based on this, the assumption is that TMS is reliable in measuring cortical excitability and can be useful in measuring AMI caused by dysfunction in the corticospinal tract. \cite{17} Cacchio et al. conducted a study that determined TMS to be reliable in measuring cortical involvement in the tibialis anterior muscle with 95\% confidence intervals in patients who are healthy. \cite{18} Evidence suggests that using TMS to measure changes in cortical excitability can allow for researchers to test different interventions that may evoke changes in the corticospinal tract.

### 2.5 Disinhibitory Modalities

Traditional rehabilitation methods for muscle weakness following joint injury consist primarily of muscle strengthening exercises. Based on the relationship of muscle weakness with AMI, rehabilitation should also focus on disinhibiting the affected musculature. \cite{4} Addressing muscle inhibition prior to traditional strengthening may allow for a more optimal recovery following joint injury. Inadequately treating underlying neural inhibition may result in persistent strength deficits, decreased performance, and increased susceptibility to further injury as well as degenerative changes such as early onset osteoarthritis (OA). \cite{4}

Neuromuscular electrical stimulation (NMES) has previously been thought of as an intervention to combat AMI; however, NMES stimulates the muscle directly, bypassing the inhibited MN pool. NMES may be effective in decreasing atrophy and increasing strength when combined with strengthening exercises, although the likelihood
of changes in AMI are minimal.\textsuperscript{2,5} Injecting patients with pharmacological agents has also been studied as a way to treat muscle inhibition. Lidocaine decreases muscle inhibition but also blocks all perceived pain, interfering with sensory feedback.\textsuperscript{5} This can lead to further joint damage because all kinesthesia properties are affected, therefore, injection may not be a practical or ethical means of treating AMI.\textsuperscript{5} Oral medications have also been investigated and eliminated as a potential treatment against AMI. Though NSAIDs were previously thought of as at possibility to reduce AMI, the negative consequences can lead to increased progression of OA due to decreased pain and increased joint loading.\textsuperscript{5} As well, NSAID’s show no increase in the excitability of $\alpha$ motor neuron pool. AMI still persists even in the absence of pain so treatment of only pain can be detrimental to the long term outcome of a patient, therefore NSAID’s are not treatment of AMI.\textsuperscript{2}

Transcutaneous electrical nerve stimulation (TENS) and cryotherapy are two therapeutic modalities that have been shown to have disinhibitory properties.\textsuperscript{4,8,13,14} TENS uses an electrical current to stimulate the sensory nerves in a desired area. This has long been used as a treatment to modulate pain following injury and has recently been shown to decrease the effects of AMI. Pietrosimone et al.\textsuperscript{8} showed changes in the central activation ratio (CAR) of the quadriceps with the use of TENS. CAR measures at 20, 30, and 45 minutes from baseline with TENS application showed increases in quadriceps activation. The same measures were repeated with cryotherapy. Ice was applied to the anterior and posterior aspects of the knee for 20 minutes followed by measures of CAR at 20, 30, and 45 minutes following the removal of ice. Quadriceps activation in this group also increased with the highest activation occurring at the 45
minute interval. This study shows that both TENS and cryotherapy are useful in increasing voluntary quadriceps activation deficits, a known effect of AMI.

Hopkins et al. performed a similar study in healthy subjects following an artificial knee effusion. This study used H-reflex as a way of assessing quadriceps activation. H-reflex measures were recorded prior to saline injection. Participants were either assigned to a TENS group, cryotherapy group, or a control group that received no intervention. H-reflex amplitudes were recorded in 15 minute increments for 1 hour. H-reflexes amplitudes were conducted by delivering a 0.3 millisecond, 100 to 200 volt, electrical stimulation to the femoral nerve in 20 second intervals. In the study, both the cryotherapy and TENS groups received the intervention for 30 minutes. Recordings at 15 minutes occurred while ice was still secured to the knee or while TENS was taking place. Measurements at 30 minutes were conducted immediately following the removal of the modality. TENS increased H-reflex amplitudes compared to the pre injection measures, but only during and immediately following application. At 45-60 minutes after treatment, there is a decrease in the amount of disinhibiting taking place meaning TENS is useful while treatment is applied, but does not have long-term effects. Additionally, TENS only returned activation levels back to pre-injection level. Cryotherapy showed an increase in quadriceps activation beyond baseline measures following knee effusion, indicating that cryotherapy has facilitative properties. To observe changes in excitability from pre-injection to post-injection trials, percent change scores between pre-injection H-reflex amplitude and post injection times. Cryotherapy showed an increase in the percentage of H-reflex amplitude compared to baseline for all trials, including following cryotherapy application, while TENS showed an increase only during
the time the treatment was applied. These studies confirm that cryotherapy and TENS are immediately effective in increasing quadriceps muscle activation.

The exact neural mechanism through which TENS and cryotherapy affect AMI is not completely understood. Further research is needed to determine if TENS and cryotherapy affect not only the spinal reflex pathway, but also corticospinal excitability. The purpose of this research will be to investigate the effects of cryotherapy on corticospinal excitability of the quadriceps. The study design will be similar to that of Pietrosimone et al. and Hopkins et al. in terms of time and intervention. Research findings from this experiment may lead to future research of other modalities, specifically TENS, on their effects on corticospinal excitability. As well, if the desired results occur, more research can be conducted on incorporating these modalities into rehabilitation protocols and seeing the effects on neuromuscular control.
Chapter 3

Methods

3.1 Study Design.

Blinded Crossover study: Participants reported for two testing sessions where they were given the intervention or served as their own control. The order for the condition was randomized. Testing sessions were conducted 3-14 days apart. Outcome measures were recorded before intervention and at 10, 20, 35, and 50 minutes after the initial application of the ice. The investigator was blinded from the intervention during testing sessions to ensure that the investigator did not know which session the participant received the cryotherapy intervention.

3.2 Participants

All patients presented with Patellofemoral pain and were between the age range of 18 to 45 years old. Subjects were recruited from local orthopedic and sports medicine clinics and well as the student population at the University of Toledo. In order to qualify for the study, participants met one of the three inclusion criteria:1) diagnosed with PFP by a physician, athletic trainer or physical therapist, 2) presented with diffuse anterior knee pain experienced for at least eight weeks, 3) their knee pain increased while
running in at least one of the following activities: going up or down stairs, walking, running, and squatting, after sitting for a prolonged period of time.

Participants were disqualified for the study if they had a previous history of lower extremity injury other than PFP, had surgical procedures that would have caused major structural changes to the knee joint, or were currently receiving rehabilitation or had rehabilitation within the past year. Participants were also excluded from the study if they had 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 had a cardiac pacemaker, implanted cardiac defibrillator or intracranial metallic clips or are currently pregnant/breastfeeding. Participants were also told not to consume caffeine for at least 12 hours prior to testing.

3.3 Power Analysis

Means and standard deviations of Hoffmann reflex change scores were taken from a related study dealing with the effects of disinhibitory modalities on spinal reflexive excitability in order to establish a powerful sample size. A mean difference of 2.26 with a standard deviation of 1.5 with an alpha level of .05 and 1-beta level of .8 was used to calculate an ideal sample size of 12. Each participant was tested twice, one for each condition, so a total of 24 testing session total is recommended. We aimed to test at least 15 participants, 30 total testing sessions, in the chance that some participants were disqualified from the study.

All participants provided written informed consent approved by the institutional review board at the University of Toledo prior to performing any of these proposed
3.4 Evaluation of Spinal Reflex Excitability

Participants were positioned in a dynamometer (Biodex Inc, Biodex System II) with the knee flexed to 70°. The ankle was securely attached using an ankle cuff to allow for accurate force readings. Participants were reclined with hips flexed to 40 degrees (Figure 1). A pillow was provided for low back support while seated in the dynamometer (Biodex Inc, Biodex System II) (See Figure 1). The hair over the collection sites was shaved and the skin over the recording electrode site was debrided and cleaned with alcohol. Two 10mm, pre-gelled Ag-AgCl (EL503, BIOPAC Systems Inc) surface electromyography electrodes were positioned 2cm apart over the vastus medialis oblique.\textsuperscript{15} Analog to digital signal conversion was processed with a 16-bit convertor (MP150, BIOPAC Systems Inc., Goleta CA, USA). EMG signals were sampled at 2000Hz with EMG amplification set at a gain of 1000. A 2mm shielded disc stimulating electrode (EL2524S, BIOPAC Systems Inc) was positioned over the femoral nerve and secured with hypoallergenic tape and a 7x13cm self-adhesive electrode was positioned over the hamstring and used as a dispersive electrode. A 1ms square wave stimulus was produced with a BIOPAC stimulator module (STM100A, BIOPAC Systems, Inc) and a 200 volt maximum stimulus adaptor (STMISOC, BIOPAC Systems Inc) and delivered to the femoral nerve.

During testing participants were instructed to maintain a constant head, eye, and hand position by focusing on a point on the ceiling. The stimulus was increased in .2 volt
increments until a maximum Hoffmann reflex is elicited, and then 3 maximal Hoffman reflexes were collected at that voltage. The stimulus was increased until a maximal muscle response is elicited, in which 3 maximal muscle responses were elicited.

Figure 3-1: The participant was seating in the dynamometer with hips flexed to 40° and knees flex at 70°. This position will be used for both spinal reflexive and corticospinal excitability testing.

3.5 Evaluation of Cortical Motor Evoked Potentials (MEPs)

Participants were positioned in a dynamometer as previously described for spinal reflex excitability testing. Participants wore a lycra swim cap which allowed the investigator to optimally position the magnetic coil and make marks if necessary. Additionally formable disposable earplugs were inserted to protect the participant’s ears, as an audible noise is heard during magnetic stimulation. Two 10mm, pre-gelled Ag-
AgCl surface electromyography electrodes are positioned the same as previously mentioned for spinal reflexive excitability testing.

Participants were instructed to perform a maximal volitional isometric muscle contraction in the position of 70° of knee flexion which was measured using a dynamometer. Five percent of the maximal isometric quadriceps contraction was used as a standardized volitional muscle contraction during active motor threshold/MEP testing. To elicit an MEP on the contralateral limb, a double cone coil (Magstim Company, Wales, UK) was positioned over the vertex of the cranium and Magstim rapid (Magstim Company, Wales, UK) was used to produce a maximum magnetic stimulus of 1.4 Tesla. The coil was moved approximately 1cm in an anterior-to-posterior direction over the vertex until a MEP response was found and marked on the swim cap by the investigator.

Motor threshold refers to the lowest TMS intensity necessary to evoke a MEP in the contralateral target muscle in response to a single pulse of stimuli applied over the motor cortex. Motor threshold was assessed as the lowest intensity required to elicit a MEP of ≥100µV amplitude in at least 5 out of 10 trials in the vastus medialis. A maximum MEP response was obtained by increasing the stimulus intensity to 20% greater than the resting motor threshold. The maximum MEP was recorded and used for determining MEP amplitude.

Participants were instructed to lay still and contract their quadriceps to 5% of the maximal voluntary quadriceps contraction. Visual feedback was provided on a computer in front of them to the participant so that they reach 5% of the maximal voluntary quadriceps contraction, which they were asked to hold until the MEP is elicited.
3.6 Intervention

Cryotherapy: The intervention was conducted immediately following the pretest recordings of spinal reflexive and cortical reflex excitability. Participants received 2, 1.5 L ice bags filled with crushed ice secured to the anterior and posterior aspect of the knee by an experienced research assistant, blinded to the investigator, using elastic bandage, avoiding contact with the vastus medialis as much as possible for 20 minutes.\textsuperscript{8,14} Participants stayed seated in the dynamometer and instructed to refrain from moving their leg.

Control: During the control testing session, participants were instructed to sit quietly for 20 minutes and seated in the same position as previously mention for the intervention.

3.7 Data Analysis

Maximal Hoffmann reflexes and muscle responses were processed by the investigator; who will assess peak-to-peak amplitudes of both the Hoffmann reflexes and muscles response measurements in the quadriceps. 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, MEP’s at 100% and 120% of the active motor threshold, and 120% of baseline active motor threshold were obtained as previously indicated and imputed into a data spreadsheet by a blinded investigator. Percent change scores were calculated for each outcome measure between baseline and each posttest time point \(((\text{posttest} – \text{baseline})/ \text{baseline} \times 100)\).
3.8 Statistical Analysis

A priori alpha levels were set at $P<.05$ for all inferential statistics. All statistics were evaluated using SPSS 17.0 statistical software. Separate 2x4 ANOVA’s with repeated measures on time were conducted to determine differences between conditions over time for all three dependent variable percent change scores. Paired t-tests were conducted in the occurrence of a significant interaction. Standard effect sizes with 95% confidence intervals were calculated for pre to posttest change scores for the cryotherapy and control conditions. Means were imputed in the case of missing data due to participant attenuation. Outliers were removed and means were not imputed if individual scores were more than three two SD greater than the group mean.
Chapter 4

Results

A total of 8 patients consented to participate in this study (Table 1.). One patient failed to report for the control test session and a second patient was unable to complete the 35 and 50 minute time points for the cryotherapy session for reasons unrelated to this study. Means were imputed for missing data for these two patients. One outlier was discovered in the 120% of baseline AMT and was removed from the analysis.

Table 4.1. Patient Demographics

<table>
<thead>
<tr>
<th>Cryotherapy Condition</th>
<th>Participants</th>
<th>Age (Yrs.)</th>
<th>Mass (Kg)</th>
<th>Height (cm)</th>
<th>Sex</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n=8</td>
<td>19.6±1.11</td>
<td>59.53±10.11</td>
<td>163.37±10.27</td>
<td>Male: 1</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Female:7</td>
</tr>
<tr>
<td>Control Condition</td>
<td>n=7</td>
<td>19.3±0.7</td>
<td>56.38±5.11</td>
<td>160.22±5.51</td>
<td>Male: 0</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Female:7</td>
</tr>
</tbody>
</table>

Yrs.: Age in Years, Kg: Kilograms, cm: centimeters, n: number of participants

Means and standard deviations for corticospinal excitability outcomes, at all time points, for each condition are reported in Table 2. There were no main effects for time for changes in AMT ($F_{1,1.951}= .504$, $p=.605$), 100% AMT ($F_{1,3}=.577$, $p=.633$), 120% AMT, ($F_{1,3}= 1.428$, $p=.248$), or 120% baseline AMT ($F_{1,1.459}= .749$, $p=.446$) (Table 3.). There were also no main effects for condition for changes in AMT ($F_{1,14}= .005$, $p=.945$), 100%
AMT (F_{1,14}=0.045, p=0.835), 120% AMT (F_{1,14}=3.197, p=0.095), or 120% of baseline AMT (F_{1,13}=0.675, p=0.426). No time by condition interactions were observed for changes in, 100% AMT (F_{1,3}=1.810, p=0.160), 120% AMT (F_{1,3}= 2.464, p=0.076), or 120% of baseline AMT (F_{1,459}=1.108, p=0.332). There was a significant interaction for time by condition for changes in AMT (F_{1,1.951}=4.483, p = 0.021) (Table 3.). Post-hoc paired t-tests indicated no significant changes in AMT from baseline to 10 minutes (t= -1.288, p=.239), 20 minutes (t= .390, p=.708), 35 minutes (t=.442, p = .672), or 50 minutes (t= .158, p = .879).

The control condition yielded no moderate or strong effect sizes for AMT, MEP amplitude at 100% AMT, 120% AMT, or at 120% AMT from baseline at any time point (Table 4.). A moderate effect (d= -0.41, 95% confidence interval -1.40, 0.64) occurred at 50 minutes for a change in AMT from baseline during the cryotherapy condition but, the confidence interval crosses 0 (Table 4.)
Table 4.2. Means with standard deviations for both cryotherapy and control sessions at all time points.

<table>
<thead>
<tr>
<th></th>
<th>Baseline (n=7)</th>
<th>10 (n=7)</th>
<th>20 (n=7)</th>
<th>35 (n=7)</th>
<th>50 (n=7)</th>
<th>Baseline (n=8)</th>
<th>10 (n=8)</th>
<th>20 (n=8)</th>
<th>35 (n=8)</th>
<th>50 (n=8)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>AMT</strong></td>
<td>43.63 ± 8.18</td>
<td>43±8.518</td>
<td>40.63 ±9.87</td>
<td>40.5 ±9.21</td>
<td>39.86 ±10.46</td>
<td>41.71 ± 19.62</td>
<td>38.43 ± 18.79</td>
<td>40.57 ± 20.00</td>
<td>40.5 ± 9.21</td>
<td>39.57 ±19.29</td>
</tr>
<tr>
<td><strong>MEP100</strong></td>
<td>0.0103± 0.0037</td>
<td>0.0103±0.0027</td>
<td>0.011 ±0.0032</td>
<td>0.012 ±0.0028</td>
<td>0.011 ±0.0021</td>
<td>0.012 ± 0.0052</td>
<td>0.012 ± 0.0057</td>
<td>0.012 ± 0.0060</td>
<td>0.013 ± 0.0045</td>
<td>0.011 ± 0.0057</td>
</tr>
<tr>
<td><strong>MEP120</strong></td>
<td>0.0306 ± 0.021</td>
<td>0.025 ±0.013</td>
<td>0.028 ±0.015</td>
<td>0.027 ±0.017</td>
<td>0.026 ±0.013</td>
<td>0.020 ±0.015</td>
<td>0.018 ±0.013</td>
<td>0.022 ±0.021</td>
<td>0.024 ± 0.018</td>
<td>0.020 ± 0.014</td>
</tr>
<tr>
<td><strong>MEP120 Baseline</strong></td>
<td>0.031 ± 0.021</td>
<td>0.025 ±0.015</td>
<td>0.036 ±0.029</td>
<td>0.031 ±0.025</td>
<td>0.042 ±0.042</td>
<td>0.020 ±0.015</td>
<td>0.0024 ± 0.017</td>
<td>0.025 ± 0.23</td>
<td>0.027 ± 0.022</td>
<td>0.0227± 0.0167</td>
</tr>
</tbody>
</table>

AMT’s are measured in % Tesla. MEP, MEP 120, and MEP 120 baseline are measured in µV.

Table 4.3. Changes from baseline at each time point

<table>
<thead>
<tr>
<th></th>
<th>10 minute</th>
<th>20 minute</th>
<th>35 minute</th>
<th>50 minute</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cryotherapy</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>AMT</strong></td>
<td>-1.57</td>
<td>8.00</td>
<td>8.10</td>
<td>-7.49</td>
</tr>
<tr>
<td><strong>100 MEP:M</strong></td>
<td>3.54</td>
<td>8.00</td>
<td>23.86</td>
<td>0.90</td>
</tr>
<tr>
<td><strong>120 MEP:M</strong></td>
<td>-9.89</td>
<td>8.00</td>
<td>23.14</td>
<td>-10.00</td>
</tr>
<tr>
<td><strong>Baseline 120 MEP:M</strong></td>
<td>-6.66</td>
<td>8.00</td>
<td>36.99</td>
<td>12.50</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>10 minute</th>
<th>20 minute</th>
<th>35 minute</th>
<th>50 minute</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Control</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>AMT</strong></td>
<td>-8.60</td>
<td>8.00</td>
<td>11.08</td>
<td>-3.85</td>
</tr>
<tr>
<td><strong>100 MEP:M</strong></td>
<td>0.16</td>
<td>8.00</td>
<td>16.88</td>
<td>2.20</td>
</tr>
<tr>
<td><strong>120 MEP:M</strong></td>
<td>-7.96</td>
<td>8.00</td>
<td>22.51</td>
<td>6.84</td>
</tr>
<tr>
<td><strong>Baseline 120 MEP:M</strong></td>
<td>24.13</td>
<td>7.00</td>
<td>21.11</td>
<td>-6.33</td>
</tr>
</tbody>
</table>

AMT: Active motor threshold; SD: standard deviation
Table 4.4. Significance and effect sizes with respective 95% confidence intervals cryotherapy condition.

<table>
<thead>
<tr>
<th>Cryotherapy Condition</th>
<th>10 minutes</th>
<th>20 minutes</th>
<th>35 minutes</th>
<th>50 minutes</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Effect Size (d)</td>
<td>95 % CI</td>
<td>Effect Size (d)</td>
<td>95 % CI</td>
</tr>
<tr>
<td>AMT</td>
<td>-0.07</td>
<td>-1.05</td>
<td>0.91</td>
<td>-0.33</td>
</tr>
<tr>
<td>100% AMT</td>
<td>-0.02</td>
<td>-0.99</td>
<td>0.97</td>
<td>0.04</td>
</tr>
<tr>
<td>120% AMT</td>
<td>-0.37</td>
<td>-1.34</td>
<td>0.64</td>
<td>-0.13</td>
</tr>
<tr>
<td>120% Baseline AMT</td>
<td>-0.29</td>
<td>-1.25</td>
<td>0.71</td>
<td>0.21</td>
</tr>
</tbody>
</table>

Control Condition

<table>
<thead>
<tr>
<th></th>
<th>10 minutes</th>
<th>20 minutes</th>
<th>35 minutes</th>
<th>50 minutes</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Effect Size (d)</td>
<td>95 % CI</td>
<td>Effect Size (d)</td>
<td>95 % CI</td>
</tr>
<tr>
<td>AMT</td>
<td>-0.17</td>
<td>-1.21</td>
<td>0.89</td>
<td>-0.06</td>
</tr>
<tr>
<td>100% AMT</td>
<td>0.01</td>
<td>-1.04</td>
<td>1.06</td>
<td>0.04</td>
</tr>
<tr>
<td>120% AMT</td>
<td>-0.15</td>
<td>-1.19</td>
<td>0.91</td>
<td>0.11</td>
</tr>
<tr>
<td>120% Baseline AMT</td>
<td>0.11</td>
<td>-0.95</td>
<td>1.15</td>
<td>0.03</td>
</tr>
</tbody>
</table>

*d*=Cohen's d  effect sizes, CI=confidence interval, AMT: Active motor threshold
Chapter 5

Discussion

This study was conducted to investigate the effect of a cryotherapy intervention on quadriceps $\alpha$-motor neuron pool excitability in corticospinal pathways in patients with patellofemoral pain. We hypothesized that the application of an ice bag surrounding the knee joint would increase corticospinal excitability over time in patients with patellofemoral pain compared to no treatment. However, there were no main effects for time or condition for changes in AMT, MEP amplitude at 100% AMT, 120% of AMT, or 120% of baseline AMT. The only significant interaction for condition by time occurred for change for AMT. There were no other changes for all other outcome variables. Although there was a significant condition by time interaction for AMT no statistically significant changes were found between conditions at baseline, 10, 20, 35, and 50 minutes following the cryotherapy application. This is likely due to large standard deviations in the means. No moderate or strong effects were observed during the control condition. For the cryotherapy condition, a moderate effect ($d = -0.41$, 95% CI: -1.40, .64) was demonstrated at the 50 minute time point for change in AMT. Although a moderate effect occurred, the clinically applicability is limited due to a wide 95% confidence interval that crossed zero.

The corticospinal tract is an important neural pathway for the generation of movement. $^{1,2}$ Patients with patellofemoral knee pain or anterior knee pain often exhibit
quadriceps muscle inhibition, which contribute to deficits in quadriceps strength
neuromuscular control, and joint kinematics that in the long-term can negatively alter the
integrity of the knee joint. A combination of neuromuscular alterations at the knee joint
may lead to tibiofemoral osteoarthritis. It is important to understand where this
inhibition originates, and target this potential site with an intervention. The corticospinal
tract primarily involves voluntary movements of a desired body part, so a dysfunction
involving this pathway can hinder how a person voluntarily activates musculature to
move their body. Cryotherapy has previously been demonstrated to increase spinal
reflex excitability of the quadriceps for both Hoffmann reflex amplitudes and central
activation ratio of the quadriceps following twenty minutes of icing. Central
activation ratio is used to measure voluntary activation of the motor neuron pool while
the Hoffmann reflex is used to measure available spinal reflexive excitability. MEPs, are
used to measure electrical potentials in a given muscle caused by a stimulus to the motor
cortex in the brain. This may explain why we found no significant changes in
corticospinal excitability. Cryotherapy may have a greater effect on the spinal reflexive
pathway compared to corticospinal excitability. Age may also contribute to why we did
not see any significant changes in cortical excitability. The age range was eighteen to
twenty-two. Currently, there is no research to indicating that age may be a factor in
corticospinal excitability deficits in this patient population so it is important to continue
to investigate its effects.

Based on our results, there is currently limited indication that 20 minutes of ice
can increase corticospinal excitability in patients with patellofemoral pain. Although the
results do not show any increase in corticospinal excitability, cryotherapy can still be a
useful tool for the treatment of knee pain. Cryotherapy is clinically used to minimize swelling and reduce pain following joint injury. Cryokinetics, a combination of ice and exercise, has also previously yielded positive outcomes for restoring range of motion and quicker return to play from joint injuries.¹

There were several limitations associated with our study. The sample size in the study was small, contributing to underpowered results. Increasing the sample size may lead to a more powerful study and possibly different results. One participant failed to report back for the second test session, which was randomized to be the control session. Another participant quit the study during the cryotherapy session at the thirty five minute mark due to time constraints and was unable to be rescheduled. Additionally, cryotherapy alone is rarely used for rehabilitation. Generally, cryotherapy is used in conjunction with exercise as part of the rehabilitation paradigm, which may have an effect on corticospinal excitability while cryotherapy itself has no effect. Our PFP patient population also limits the generalizability of the study to other types of joint injury. The age range was eighteen to twenty two; therefore, we cannot generalize these results to an older population. Furthermore, only one male participated in the study compared to seven females and failed to complete the entire study. With the majority of the participants being female, we cannot generalize these results to the male population.

Our current study did not show any positive benefit of using cryotherapy to enhance corticospinal excitability in participants with PFP, but more research is needed to confirm our results. Other disinhibitory modalities such as transcutaneous electrical nerve stimulation (TENS), may have an effect on cortical excitability and should be investigated. In previous studies, TENS, like cryotherapy, increased spinal reflex
excitability and therefore may benefit patients with decreased cortical excitability. More research is needed to determine if cryotherapy can alter cortical excitability in patients with various knee pathologies. Different patient populations may respond differently to cryotherapy than others. TENS, joint mobilization, and biofeedback rehabilitation interventions are alternative therapies that may affect corticospinal excitability and need to be researched. Continuing to investigate modalities and rehabilitation techniques is essential to help combat arthrogenic muscle inhibition and prevent long term joint damage such as osteoarthritis.

Our study did not determine if our PFP patient population had deficits in corticospinal excitability before the intervention. Cryotherapy did not increase corticospinal excitability in these patients because there may have not been a deficit to begin with. Continuing to investigate how individual patients respond to cryotherapy will help target what patient population can be treated effectively with this therapy.

From the results of this study, we conclude that cryotherapy may not have an effect on corticospinal excitability of the quadriceps in patients with patellofemoral pain. Although this study did not find a statistically significant effect, it is imperative to continue researching possible solutions to enhance corticospinal excitability following joint injury.
References


9. Pietrosimone BG, Hertel J, Ingersoll CD, Hart JM, Saliba SA. Voluntary Quadriceps Activation Deficits in Patients with Tibiofemoral Osteoarthritis: A


