THE EFFECTS OF ACUTE PASSIVE STATIC STRETCHING ON CRAMP THRESHOLD FREQUENCY

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ABSTRACT

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by Gino Panza

Stretching is the intervention of choice for immediate treatment of exercise associated muscle cramps (EAMC). Previous research has been purported to reduce the incidence of EAMCs. This study was designed to investigate the relationship between stretching and susceptibility to cramping. A randomly assigned, repeated measures design with paired T-Tests comparing the Pre to Post measures in each level and mean differences between levels. The independent variable is stretch vs nonstretch condition as the dependent variable is the participant's cramp threshold frequency (CTF). Stretching would increase the participants' CTF. This study showed a significant difference between the Pre and Post values in both the control ($T=2.38(16)=0.016$, $p\leq0.05$) and Stretch Pre vs Post ($T=3.56(16)=0.025$, $p\leq0.05$). No significant difference was found when comparing the perturbations ($T=0(16)=0.5$, $p\geq0.05$). This study suggests that acute passive static stretching does not have a significant effect on increasing CTF.

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Introduction

Exercise associated muscle cramp (EAMC) is a painful, spasmodic, involuntary contraction of skeletal muscle that occurs during or immediately post exercise and is one of the most common clinical problems presented in athletes (1). EAMC affects both recreational athletes and elite competitive athletes. There is evidence showing a high prevalence in triathletes 67% and 30-50% of marathon runners (2). Even with this high prevalence the etiology of EAMC is still not completely elucidated.

EAMC has traditionally been thought to be caused by electrolyte depletion and dehydration (1). These first reports of muscle cramping were primarily anecdotal, and involved physically demanding jobs in hot, humid environments. This led to the development of the term "heat cramps" or "exertional heat cramps". These two terms are often used interchangeably with EAMC (1). Nonetheless, exercise in moderate to cool temperatures and exposure to extreme cold has also been associated with EAMC in swimmers. Additionally, passive heating alone has not caused cramps, nor has cooling relieved the muscle cramps (3, 4).

Consequently there is a debate about EAMC originating from dehydration and electrolyte imbalances. Other possibilities proposed have included neuromuscular junction deformation, nutritional and training status, hyperexcitiability of motor neurons induced by afferent input, central or peripheral neural origins, sarcolemma deformation (5), an imbalance between afferents and efferent's in regards to muscle spindles and Golgi Tendon Organs (GTO), (6, 7, 8 , 9, 1, 3, 10). There has been much disagreement about which of these possible etiologies is the basis of EAMCs. Recent studies direct attention to a neuromuscular origin (5). Currently, the most common treatment for cramping is immediate acute stretching (6, 8), fluid ingestion, and ingestion of a high sodium drink, commonly pickle juice (11, 15).

Typically, when investigating the electrolyte imbalance hypothesis of muscle cramping, physiologist have gathered serum electrolyte levels pre and post exercise to evaluate biochemical markers attempting to identify any deficiencies or imbalances within the body (6, 3, 12, 13). Alternatively, in a laboratory setting, physiologists have used Electrically Induced Muscle Cramps (EIMC) (14, 8, 4, 9). These EIMC are used to initiate muscle cramping by stimulating muscles via muscle belly (9) or specific nerve tract locations (15, 8, 4) in order to cramp the targeted muscle. EIMC have shown virtually identical EMG recordings as actual cramps (14).

Another common exercise modality, static stretching, has also been identified as effecting neuromuscular control and subsequent function. Static stretching is widely accepted as a modality of increasing joint range of motion (flexibility), injury prevention (16) and was previously thought to increase athletic performance (17). Static stretching has been shown to have optimal length effects at 15-30 seconds under tension (18). The acute response of muscle to stretching has been shown to have both mechanical and neural components. These particular components have been shown to have their effects at different times.

Pre-exercise static stretching has recently been suggested to reduce musculotendinous unit (MTU) tension thus reducing force production through contractile components and neural modifications. With stretching, the contractile components do not transmit their force directly to bone (19). This delay in force transmission is termed electromechanical delay (EMD) (20). Wilson, Murphy, and Pryor (1994), as cited by Kokkonen (1998), proposed increasing the stiffness of the musculotendinous system will increase the tension transmitted to bone consequently increasing strength performance.

The reduction in MTU stiffness and the proceeding EMD can reduce force output from the stretched muscle (5, 21). The change in the MTU has one response, labeled as plastic

deformation, which addresses the change in soft supporting tissue of the muscles (19). Prolonged passive static stretching has also shown to induce changes in the passive length-tension relationship (in animals) caused from changes in the MTU's viscoelastic properties (16). This association makes it plausible that static stretching will reduce strength performance via both a mechanical and neural mechanisms. These two mechanisms' difference is the time course of the elicited effects (16, 22).

As for the neural decrease in performance, static stretching has shown a decrease in performance in high intense motor activities (21). Additionally, neural machinery is related to an alteration of sensory mechanisms (proprioception) that affect neural activation (5, 23, 21). The Hoffman reflex (H reflex), which is a measurement of available alpha motor neurons, has shown to be depressed during, immediately after, and up to more than 60 minutes post static stretch (16, 19, 22, 23) and is proportional to the intensity of stretching (22). The H-reflex has been labeled as spindle support (23). Spindle support is the muscle spindles agonistic nature to muscular contraction. This deficit is seen with an increase in compliance of the MTU which may have an alteration in neural activation feedback responses from a reduced mechanical sensitivity to muscle spindles (19), decrease in spinal reflex excitability (24), as the 1a-reflex pathway has been shown to not adapt to a stretching program (22), and not be active during stretch as the H-reflex stays decreased (24). The H-Reflex is shown to decrease with no effect on the M-wave, thus showing a reduced H:M ratio. This decline shows there is no failure at the neuromuscular junction, but impairment of the excitation of the alpha motor neuron pool (16) indicating some neural modification is present after static stretching (19). The muscle spindles are sensitive to the immediate history of the tissue, thus stretching could desensitize the muscle spindle, reducing the reflex arc (24). This shows direct neural involvement during the stretching maneuver.

An acute bout of passive stretching on triceps surae provided a significant 7.8% decrease in jump height with a 2.8% decrease in muscle stiffness and no significant change in IEMG (integrated EMG) during the concentric phase of a countermovement jump. Assuming the MTU is a perfect spring, only 2.8% o the total 7.8% deficit in jump height can be attributed to a loss in muscle stiffness. In the static jump protocol, there was no change in jump height following stretching with decreased IEMG. This result suggests that the experiment may not have been sensitive enough to measure changes in jump height or that there was a possible alteration in motor unit recruitment pattern that preserved static jump height (25).

Costa (2010) indicated significant ($p<0.05$) stretching-induced changes in EMD, peak twitch force (PTF), and rate of force development (RFD) by 28%, 13%, and 9% respectively, following 20 min of passive stretching of the plantar flexor muscles in a pre to post test protocol. No significant changes in p-p M-wave amplitude from SOL and MG. Significant ($p<0.05$) negative, linear relationship between EMD and PTF, pre to post stretch and with EMD and RFD. With no changes in M-wave amplitude, the present findings suggested that stretching-induced deficits measured by evoke twitches may be related to transient decreases in musculotendinous stiffness assuming full activation of the alpha motor neuron pool. Weir (2005) found a significant reduction in MVC in the absence of any significant change in the H: M ratio. Although there was no change in the ratio, Weirs' study showed a total reduction of H and M waves. This change can be caused from the change in histochemistry (24), viscoelastic properties, or a change in neural activation patterns (5).

The attenuation of force through the increased muscle length (length-tension and/or plastic deformation) was evident 30 and 60 minutes post stretch. This change may independently facilitate the influence of neural activation patterns via decreasing the resting discharge of

muscle spindles (21). Immediately following a stretching protocol Fowles (2000) found a significant reduction (16%) in interpolated twitch which shows a decrease in nerve activation immediately after. The same result was found 5 minutes post stretch (13% reduction). The reduction in neural patterns directly leads to a reduction in force generation. Acute static stretching has shown to decrease muscle stiffness (increase in MTU compliance) up to one hour after stretching. This timeframe is matched by the neural modifications that seem to return to normal after an hour. During this hour Fowles (2000) contributed the 25% decrease in MVC partly from reduction in activation and partly mechanical. 60% of the 25% reduction in MVC was from neurological factors and the remaining 40% pertained to mechanical factors.

Regarding a stretching regimen, Guissard (2003) and Hayes (2012) have shown that the increases in flexibility have neural as well as mechanical mechanisms. The immediate, and within 5 minutes, changes are contributed to neural changes. Also, during the stretching maneuver there is a large inhibiting neural contribution (H max decrease) from the GTO (22).

The reduction in H reflex after stretching could be interpreted as increasing the compliance of the muscle which would cause a reduction in the mechanical response from the muscle spindles through a reduced activation of 1a afferents (16), caused by the increase in EMD and altered proprioception (5). This reduction could lead to disfacilitation of the alpha motor neuron pool (19, 22). The changes in MTU vary from muscle group to muscle group. This variability can possibly be explained by changes in training and stretching durations and intensities (16) and gender (22). A sample size with even males and females and larger numbers has shown a larger percentage increase in ROM with less alpha motor neuron pool inhibition(22).

In summary, both mechanical and neural mechanisms of stretching are important to

increase flexibility. The immediate stretch and increase in tension on the muscle has a significant decrease on H max, and rapid decay in passive torque revealing a large inhibitory input from the GTO (5, 22). This shows that both mechanical and neural mechanisms are involved in the increase in ROM of a specific joint.

Immediate treatment for EAMC is an acute bout of passive static stretching of the affected muscles (3, 8, 27, 13). The passive static stretching stimulates the Golgi Tendon Organ afferents producing a volley of orthodromic inhibitory action potentials to the spinal cord, thus reducing the spinal reflex excitability (27, 28, 10, 29). Using electrical stimulation at the muscle motor point does not alter neuromuscular junction or the electrical properties of the sarcolemma (28, 30) thus keeping the attention focused on the perception of muscle length and its correlation with cross-bridge alignment. However, other treatments are purposed and are unsupported by experimental research. Some of these theories are consumption of pickle juice, mustard, other high sodium drinks, cryotherapy, and massage among a host of others (8, 3, 13).

In order to test EAMC, Stone (2003) tested and validated the use of an electrical stimulus to replicate EAMCs. This protocol was developed in order to recreate the occurrence of cramps in a reproducible and manageable scenario. Stone (2003) also showed the high intrasession and intersession reliability of these procedures (15).

The unclear etiology of muscle cramping and reports of better stretching habits reducing the risk of EAMCs (1), and lack of scientific literature regarding stretching and CTF lead us to questioning the effects of acute static stretching on CTF in the currently accepted and validated EIMC model. Therefore the purpose of this study was to determine if acute passive static stretching will alter a participants' CTF. The expected outcome was that a bout of acute static stretching would increase the participants' cramp threshold frequency thereby reducing the

propensity to cramp.

Methods

The current study used a randomly assigned repeated measures design which consists of a randomized cross-sectional approach with one independent variable and one dependent variable. The independent variable is non-stretch verse static stretch applications, and the dependent variable consists of the participant's Cramp Threshold Frequency (CTF) of the Flexor Hallicus Brevis (FHB). Cramp induction via electrically induced muscle cramp (EIMC) of FHB was validated by Marcus Stone in January of 2003. The reproducibility of the protocol shows how the varying factor is the frequency that elicits a cramp. Miller (2007) increased the voltage and followed the same protocol with no adverse or confounding factors associated with EIMC and CTF. CTF was within statistical value of Hz regardless of voltage. The 80V used in the current protocol has resulted in no more pain than other accepted laboratory techniques. This same article validated the need for familiarization session (14). This approach was used to ensure both groups that were tested were represented equally.

Participants

A convenience sample of 17 college students, 13 males and 4 females, the majority from Central Michigan University's College of Health Professions, were recruited. The participants' mean age was 23.0±3.3 years, height of 1.7±0.1 m, weight 80.67±13.1 kg, and BMI 26.45±2.9 (See Table 1). Of the 17 participants, 4 were self reported crampers. Exclusion data included self-reported previous injury to the dominant limb within 6 months of the study, metabolic, neuromuscular, neurological diseases/disorders, or pregnancy. Participants were asked to abstain from any strenuous activities 24 hours prior to the scheduled testing. This project was reviewed and approved my Central Michigan University's IRB on January 24, 2013.

Table 1. Summary of the participants' descriptive characteristics (mean ±SD).

Demographics	
Age (Years)	23.0 ±3.3
Body Mass (kg)	80.7±13.1
Stature (m)	1.7±0.1
BMI	26.5±2.9
Males	13
Females	4
N=17	

Instruments

Electromyography of the FHB was recorded with a MP 150 analog-to-digital wireless bionomadic (Biopac systems) using EL504 1 Inch Solid Gel surface electrode from Biopac systems. The stimulus was applied using a Grass Stimulator S88 REV K which was routed through a Grass Stimulation Unit Model SIU8T (W. Warwick, RI, USA). Stimulation electrodes were Biopac systems EL 258S Shielded 8mm Ag-AgCl TP. A female to male adapter from the EL 285S to the stimulation unit was used. An 8cm square dispersive was used on the lateral malleolus of the stimulated foot. Biometric data was collected using Seca 217 Portable Stadiometer accompanied with a Seca 869 Medical Platform Scale (Chino, CA, USA). The BMI formula utilized was kg/m^2. An informed consent and adult consent form were also used to facilitate the delimitations of the study boundaries.

Procedures

Participants were asked to report to the lab 4 separate times within a span of at least 21 days. Participants were asked to not participate in lower body strenuous exercise within 24 hours of lab time. Participants were randomly placed into one of two groups; Group one: Static Stretch (SS), No Stretch (NS), and group two: NS, SS. On day 1, informed consents were issued as well

as a randomized serial numbers for identification purposes. A detailed explanation of the research was reviewed with each participant. The first day of testing also included collecting height (m) and weight (kg), and assessed identification of dominant leg. Dominant limb was determined by asking the participant to pretend to kick an imaginary ball, a method used in previous research (32). Upon completion of the consent forms and collection of descriptive data the participants were then accompanied on a short walk through the Health Professions building. This walk was to establish a common baseline muscle activity prior to the electrical stimulation procedure. Day one was concluded with eliciting a cramp using the following protocol.

Participants were instructed to lay supine with their dominant ankle towards the researcher. The participant's ankle and knee were slightly elevated off the work table with a series of number labeled foam pads. Each participant always received the same foam pads. Participants were instructed to look at the ceiling, and were given headphones and access to music to eliminate noise and/or distracting stimuli (31). To prepare the participant, standard EMG preparatory procedures were performed (14). Two surface electrodes were then placed over the muscle belly of the FHB, centers separated by 2 cm. EMG was grounded by a single ground electrode placed over the superior aspect of the medial malleolus. Inducing the cramp consisted of placing the 8-mm Ag-AgCl stimulating electrode slightly inferior and posterior to the medial malleolus, as the 8cm dispersive pad was placed over the lateral malleolus. The tibial nerve was then stimulated with an 80V 1-millisecond electrical stimulation until a high degree of Hallicus flexion was observed. Once a proper tibial nerve location was identified it was marked with a permanent marker. The electrode and the dispersive pad were secured using a tight fitting ankle brace. Stimulating electrode placement was marked with a permanent marker and participants were asked to reapply the indicator if they noticed fading.

To determine Cramp Threshold Frequency (CTF) the following procedure was followed. The Grass Stimulator S88 REV K was set at 2 second trains, 80V, and 6 pulses per second (pps or Hz). The SIU8T stimulation unit was dialed in at 16 and the switch was set to low. This indicated, at the start of stimulation, there would be a 1 second pause and then a 1 second stimulation period with 6 pulses of 80V administered. If no cramp was elicited at 6Hz then 2 Hz was added and stimulation was reapplied after a 1 minute break. The process of adding 2 Hz and a one minute break in-between stimuli was continued until a cramp was elicited. The lowest value at which a cramp was elicited was termed the Cramp Threshold Frequency (32).

The previous EMG preparation and electrical stimulation protocol was used in every lab session. The standardized walking distance was also utilized prior to all visits into the EIMC lab room. After the two familiarization periods the randomized cross-over application of the study was administered. Depending on group assignment, one of the two perturbations was administered (Stretch and No Stretch). On the participants day to be stretched they underwent the previous protocol to establish that day's baseline CTF. Once their baseline CTF was established a 10minute break ensued. Once the 10 minute break had ended the participants underwent an acute passive static stretching protocol.

The protocol consisted of 3 bouts of 30 second passive static stretch, administered by the tester, and a 30 second rest. The 3 bouts of time under stretch totaled 90 seconds, as the entire procedure lasted 3 minutes. Range of stretch was determined by the participant and what they perceived as mildly uncomfortable (18) also known as the point of discomfort (POD). After the stretching protocol, the cramp induction protocol was followed to elicit a second cramp for that lab testing day. This procedure gave the daily baseline CTF before stretching (Pre Static Stretch CTF), and a CTF reading immediately after stretching (post static stretching CTF).

The second protocol that was used was the control (no stretch) protocol. Upon arrival to the laboratory the participant would walk the predetermined route with tester, then received the normal EMG preparatory protocol, then undergo the cramp induction protocol. This gave the daily baseline CTF for the non-stretching day. Similar to the stretching day, a ten minute break was administered. Since this was the non-stretch protocol an additional 3 minute time period was administered totaling 13 minutes between EIMCs. This ensured the same amount of time between cramp protocols in both conditions. After the 13 minute break a second cramp was elicited. Once the second cramp was elicited the Pre CTF and Post CTF for the control day was obtained. Every participant received each protocol; 2 familiarization sessions, a static stretching session with two cramps administered that day, and a control day where two cramps were also administered. This gave a CTF for each familiarization day, as well as a Pre and Post CTF for both the static stretching condition and the control condition. Participants received a total of 6 cramp induction protocols throughout their 4 lab visits.

Statistical Analysis

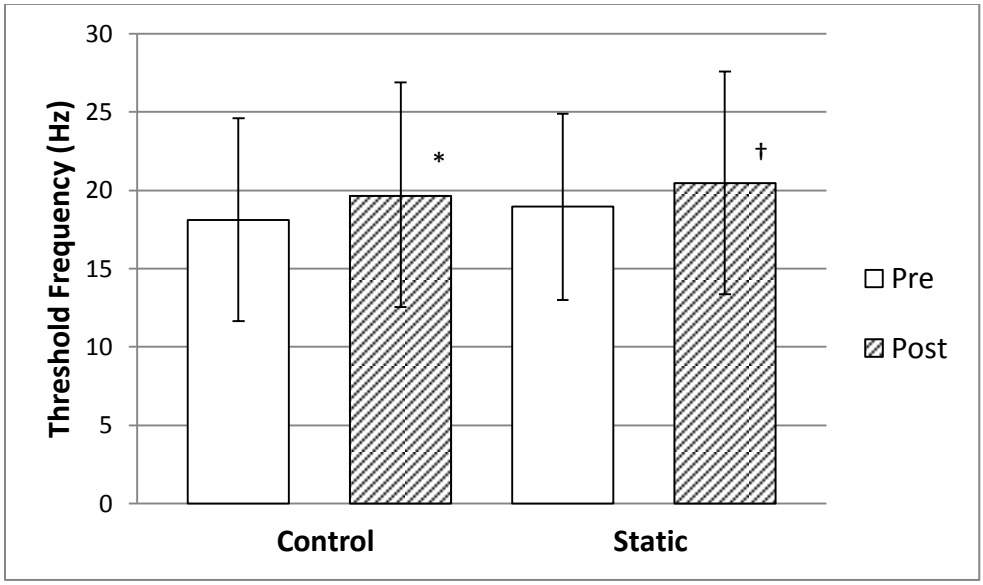
A paired samples t-test was performed to determine the effectiveness of acute passive static stretching on CTF. The within participants t-tests were conducted pre and post in both conditions. The paired t-test was also used to compare the mean differences between control and static stretch post CTF values to determine the treatment effects. An independent T-Test was then used to compare the Pre control to the Pre Static stretch CTF's. Statistical significance was set to $P \leq 0.05$ for all tests.

Results

Of the original 21 participants recruited 4 were excluded from statistical analysis due to

not meeting cramping criteria. Participants either did not cramp in both familiarization sessions, or did not cramp during one of the testing days. There were significant differences within participants' Pre CTF and Post CTF in both the control condition and stretching condition with $T=2.38(16)=0.016$, $p\leq 0.05$ and $T=3.56(16)= 0.025$, $p\leq 0.05$, respectively. However, no significance was found when comparing the mean differences between conditions $T=0(16)=0.5$, $p>0.05$ (Figure 1).

Figure 1. The mean differences in cramp threshold frequency from pre- to post-test in both the control and static stretching condition groups. Control group pre-test CTF (18.1±6.5), post-test CTF (19.7±7.3). Static stretching group pre-test CTF (18.9±6.0), post-test CTF (20.5±7.1). Control Pre CTF (18.1) compared to the static stretching Pre CTF (18.9) was not significant. * Significant difference between pre-test to post-test within the control group ($p\leq 0.05$). †Significant difference between pre-test to post-test within the static stretching group ($p\leq 0.05$). No significant difference was found between treatments.



Discussion

The purpose of this study was to determine if acute passive static stretching would increase cramp threshold frequency. This experiment revealed acute passive static stretching did not have the effect of raising the participant's CTF, when compared to control. Both laboratory

days yielded a significant change in Pre and Post CTF. The significant difference in the Control Pre to Post CTF values is at odds with previously published data (33, 15) that uses the same protocol. It is however in line with data provided by a different protocol of EIMC (34). Furthermore, this study did not have decrease in CTF from day 1 of testing to day two (33, 15). A difference between the control and condition Pre CTFs was noted. The average CTF did rise by 0.82(Hz) from control Pre to Condition Pre. This shift was not statistically significant $T=-2.77(16)=0.32$, $p>0.05$. The differences in this study could be explained by the convenience sample, gender differences, or BMI levels. No differences were observed when comparing the mean differences between the two post CTF values. The protocol that was used utilized has been established and validated by Stone (2003).

An increase in CTF is thought to be indicative of an increase in resistance to cramping. Individuals prone to cramping have been found to have lower CTFs (15, 33). The results of this study indicate that acute static stretching does not increase the resistance of an individual to experience an electrically induced muscle cramp to the level of statistical significance using the methodology described.

This is likely the result of acute static stretching on EIMCs and CTF with the current modalities. Our study did further validate that acute stretching did stop EIMCs, however it did not help retard a future cramping episode. From further evaluation of the literature review evidence stemming from the peripheral mechanisms which could yield explanations of the results found. Emiliano (2008) tested mechanomyography in conjunction with electromyography to test the mechanical and electrical contributions of skeletal muscle after passive static stretching. In this study they stimulated the muscle motor point. Stimulating the muscle motor point achieved a certain level of circumventing the central nervous system which

in turn exploits the peripheral mechanical mechanisms. The study did reveal a decrease in force production post stretch with no change in EMG parameters, neuromuscular synaptic efficiency and sarcolemmal action potential propagation while accompanied by a decrease in MMG (28). With all electrical properties unchanged there was still a decrease in force production revealing an immediate effect of stretching from the peripheral and mechanical mechanisms.

Esposito (2009) continued to show the same information but in regards to a previously fatigued muscle. Stretching a previously fatigued muscle exacerbated the force production decrease with no change in EMG parameters. Moreover, All EMG values post stretch returned to their pre fatigue values suggesting that the electrical modifications were not affected by the stretch and recovered without being hindered. These two studies show a significant impairment to the stiffness of the MTU and intramuscular changes due to stretching at the point of discomfort. The intramuscular changes in MTU and other connective properties could affect the neural feedback patterns (19), proprioception (5), autogenic inhibition (23), and velocity conditions (17). Considering the EMG parameters all returned to normal baseline values post-stretch then the mechanical evidence described may not have had enough influence on the spinal cord to elicit an adequate level of central drive to override inhibitory action potentials, or did not cause sufficient disruption in the neural control from a peripheral trigger to the spinal cord or from the motor cortex to the alpha motor neurons to cause an imbalance of neural communication.

Considering the neural communication was not disrupted from a peripheral mechanical mechanism, or recovered prior to the second elicited cramp the results are similar to a previous study by Minetto (2008). Minetto (2008) also used a different EIMC eliciting protocol, however the study used the same stimulation method as Esposito (2009) and Emiliano (2008). His

consisted of stimulating the muscle motor point and used the abductor Hallicus showing that CTF will raise with successive EIMCs. Miller (2007) also showed an increase in CTF from day 1 of testing compared to day's 3-5, and significant difference in day 2 compared to day 4-5 with no difference between days 3-5. Miller (2007) uses a similar protocol to the one utilized for this study. The changes in participants' CTF from one day of testing to an additional day match previous studies (31), as well as the significant difference from pre to post CTF values in another (34).

In attempts to offer explanations why there was no mean differences between the control and condition post CTFs values, possible limitations will be addressed. A type II error could have been reached from a mechanical and neural standpoint. The error could be caused by the location of the FHB. The metatarsalphalangeal joint may have limited the ROM of the stretch. The design was intended to stretch to the POD (point of discomfort) for 30 seconds. This has been found to suffice in achieving the effects of static stretching in a previous study (18). The effects of static stretching have been shown to desensitize the muscle spindles, and change the compliance of the MTU. In the current study, stretching the FHB to a POD was difficult to achieve because of the articulation site.

If the muscle is not stretched to point of discomfort then the effects of desensitizing the muscle spindles (21, 19, 22) may not be reached. Accompanying the lack of muscle spindle desensitizing there may not be a large increase in the MTU's compliance. Desensitizing the muscle spindles through stretch is the main mechanism that was being tested, as Baldissera (1994) cited Denny-Brown and Foley (1948) as being the first to demonstrate that a cramp is elicited through a major neuronal discharge.

The change in musculotendinous unit is thought to be from an altered fascicular length (23, 5), tolerance to stretch, viscoelastic relaxation (18), and accompanied by velocity conditions (17). The velocity conditions are related to the electromechanical delay (EMD) (20). Viscoelastic relaxation is the non-linear decrease in tension when a muscle is undergoing a stretch (18). While a stretch is being maintained the GTO ceases to fire. GTOs seem to only fire at the onset of a high magnitude of tension development causing autogenic inhibition or the sudden relaxation of a muscle upon rapid tension development (23). The change in EMD could possibly cause a change in proprioception (5, 23), in neural feedback patterns (19) and in autogenic inhibition (17). Hayes (2012) found that the tonic 1a-Reflex pathway is not active during the stretch. When the muscle is under tension from stretch the decrease in the tonic 1a-reflex-pathway may reduce the available alpha motorneurons (decreased H-Max). The reduction would stop the volley of orthodromic excitatory efferent action potentials, thus helping alleviate the cramp. Moreover, the sudden increase in tension on the MTU will stimulate the GTO causing a burst of inhibitory action potentials to the spinal cord. In summary, this shows a reduction in excitatory drive accompanied with an increase in inhibitory action potentials during (or at the onset of a) stretch. These results help facilitate the theory of muscle cramping being a problem with muscle relaxation not contraction via a neurological imbalance.

This may indicate that once the endomysium and fascicle elongate with stretching (18) out of their genetic and biomechanical set length (23), the muscle spindles do not adapt to the new elongations, thus the muscle spindles are still responding to length changes from the original muscle length. Weir (2005) verified a change in plantar flexor muscle length by testing the max strength at an absolute angle then statically stretching the muscles for ten minutes. After ten minutes the joint was placed back to the same absolute angle and MVC was tested again,

showing a 7.1% decrease in MVC. This supports the theories of increased fascicle length and an alteration in length-tension (5). This alteration was supported by the torque curve. When the muscle was returned to original optimal length the force generated plotted in a different location along the curve (23).

Baldissera (1994) demonstrated that cramps, in a pathological state, could be elicited using a tendon tap protocol. This tendon tap protocol stimulates a large amount of muscle spindles, thus showing the relationship of increasing excitatory input and cramping from a monosynaptic reflex. Using the tendon tap protocol, an incremental increase in EMG with every tap was witnessed, until a cramp was elicited. This increase in EMG with each successive tap (35) in combination with the monosynaptic activity elucidates evidence towards muscle spindles facilitating hyperexcitability of the motor neuron pool.

Using his electrical stimulation protocol, which is similar to ours as it too stimulates a nerve branch, his experiment showed with each successive stimulus the H-reflex continued to grow. Since stretching has been shown to desensitize the muscle spindle this would help alleviate the EPSPs on the dendrites of the alpha motor neuron. In combination with the increase in excitatory action potentials from the muscle spindles a decrease in inhibiting APs from the GTOs would also be present, exacerbating the neural imbalance. When stretching to the POD, we expected to see an increase in the compliance of the MTU. Therefore, while being dynamically active the increase in MTU compliance will cause less inhibitory action potentials. This decrease in inhibitory action potentials stems from the decrease in the amount of tension on the MTU at a given ROM. Theoretically, this decrease in inhibitory action potentials would require the body to produce less excitatory action potentials to override the volley of inhibitory action potentials from group III and IV afferents (35, 33) and the GTOs. Decreasing the amount

of excitatory graded potentials on the dendrites of the alpha motor neuron could help avoid reaching the second level of bistability. This further provides evidence for the hyperexcitiability theory behind cramping and that it is a problem with muscle relaxation not contraction.

Accompanying the increase in compliance and reduction in muscle spindle sensitivity, alterations in neural activation feedback responses (19) and a decrease in spinal reflex excitability (24) was expected. These results found a decrease in the H:M ratio, which provided evidence that no impairment at the neuromuscular junction (NMJ) had occurred (28), but did show an impairment in excitation of the alpha motor neuron pool indicating neural modification after stretching (19).

The dampening effects of stretching on the neural output in vivo were thought to decrease the orthodromic efferent action potentials thus reducing the neural drive to the muscles. While the muscle spindles are firing the GTOs are turned down (1) as a result causing more excitatory efferent activity to the muscle and less inhibitory activity, leading to an imbalance of neural communication. Additionally, alpha motor neuron bistability has been shown in man (35). Alpha motor neuron bistability explains that alpha motor neurons have two threshold potentials. One is the resting membrane potential and a second threshold above RMP of the motor nerve and is stimulated at a higher voltage. If this second threshold point is achieved then the RMP is established above threshold. Once this has occurred the alpha motor neuron will not drop below threshold, thus after hyperpolarizatoin the nerve is still at or above threshold. This leads to oscillating firings without external stimuli. This has been shown extensively in cat models, as well as in a pathogenic state in humans (35). This evidence shows an increase in excitatory action potentials.

If the central drive is to overcome the additional inhibitory action potentials from GTOs and group III and IV afferents the increase in excitatory drive may reach the second threshold. Reaching the second threshold could cause a burst of rhythmic efferent drive from the alpha motor neurons, which turns down the GTO's afferent inhibitory action potentials. This positive feedback cycle provides more evidence in the direction of hyperexcitiability and EAMCs being caused by problems with relaxation via a "turn down" mechanism.

The application of stretching is to cause a burst of inhibitory action potentials to the spinal cord in attempts to rebalance the inhibitory and excitatory graded potentials on the dendrites of the alpha motorneuron pool. This increase in inhibitory action potentials could help reset the motor neuron pool to the normal RMP. This also provides evidence to why a cramp is not always immediately alleviated with stretching. If the single static stretch does not send enough inhibitory action potentials to the spinal cord, then the membrane voltage may not be re-established to normal RMP. Considering GTOs cease to fire during the duration of stretch (23) the rhythmic firing from the high level of bistability could keep the alpha motor neuron pool activated. Since the nerves show different amplitudes with the excitatory monosynaptic potentials and differing levels of membrane potentials from nerve to nerve (35), the positive feedback from the maintained contraction could re-excite the alpha motor neuron pool, maintaining the muscle in a cramped state. This large communication within the spinal cord shows evidence involving neural feedback pathways (19) and proprioception (5) and their effects on the dendrites of the alpha motorneuron.

Moreover, with the decrease in H-Max from stretching there is a reduction in excitatory drive to the muscles, with this reduction in excitatory action potentials the GTOs may be able to continue the proper inhibitory responsibilities and help maintain homeostasis. To further show

the effects of group III and IV afferents on the motor neuron pool, Baldissera (1994) stimulated cutaneous receptors. A single stimulus was shown to partially reduce EMG activity, as continued stimulation of these receptors progressively decreased the EMG until silence was obtained. In the same experiment, prolonged stimulation of the cutaneous receptors seemed to prevent cramps. This was true when the H-reflex was attempted to be elicited multiple times (35). It seems that the inhibitory afferent drive from these receptors were sufficient enough to ward off a muscle cramp. It seems, with an external application of inhibitory action potentials, that the body is not able to turn down or over-come the inhibitory action, thus not allowing the motor nerves to oscillate. Considering risk factors for EAMC include a longer history of running, higher intensity running (race, hills and long duration) (1) it is plausible an unbalanced neural communication could be established. The increasing inhibitory propagation from fatigue, pain, and possible GTO activity on the end ROM during running to the alpha motor neuron pool, there would have to be an increase in excitatory drive to continue the activity. This would produce more excitatory graded potentials then inhibitory graded potentials in the spinal cord. At this point the total neural communication could be magnified as a positive feed-back and "turn-down" mechanism could be developed. The GTO seems to be the likely inhibitory mechanism that is turned down. At this point the sudden decrease in inhibitory GPs on the spinal cord may not maintain the correct level of hyperpolarization, which facilitates maintenance of normal RMP. This decrease in the ability to maintain normal RMP could cause the nerve to obtain the upper level of bistability. This would lead to oscillations of the alpha motor neuron pool.

This type of communication (bistability) has been shown in a pathogenic state, as the three patients tested had experienced severe cramping after a career of intensive parachuting (patient 1), heavy drinking which induced rhabdomyolysis that led to neurogenic damage

(patient 2), and polio (patient 3). In all three different pathogenic states cramps where elicited via electrical stimulation and mechanical stimulation and they were eliminated by synaptic inhibition (antiorthodromic activity) and cutaneous skin stimulation which is the same in EAMCs (35).

Other risk factors for EAMC have included higher BMI (see future studies), irregular stretching habits and decreased stretching time (1). The hypothesis was that stretching would cause an increase in CTF; the dampening of the muscle spindles (excitatory drive) was the primary mechanism that led to the hypothesis.

This study had certain limitations the readers should be aware of. The use of a convenient sample could potentially limit the results found. If a different sample was obtained then the possibilities of seeing an effect could have been achieved. Another possible factor was the uneven amount of females recruited when compared to males. The difference of CTF in males and females is not known. Additionally, it was noted that our convenient sample had rather high BMI. BMI was recorded, but not intended for additional use. Schwellnus (1997) stated, from an epidemiological study that high BMI is a risk factor for EAMC, which appears to be at odds with our findings. To our knowledge BMI and CTF have not been studied. It should be noted that Schwellnus' (1997) study was done on marathon runners, which likely have a lower BMI.

Future research should be conducted on CTF in relation to a chronic passive stretching program. There should also be investigation into producing a similar reliable and reproducible cramping protocol for a more applicable muscle for intervention application. Utilizing a different muscle could allow physiologist to more appropriately apply different interventions. Multiple joint angles and CTF should also be established, since participants were to lie supine, the knee

and hip joints should be close to 0°, changing joint angle, and in conjunction with the change in joint angle, the tension on the nerve could be changed. This change could cause an unknown shift in CTF. Furthermore, future studies should investigate the relationship between BMI and CTF, investigation into CTF and gender differences should also be investigated. Additionally, research into eliciting cramps via tendon taps with electromagnetic hammers in health subjects should be done.

Conclusion

In conclusion the present study provides evidence that static stretching of the FHB does not have a significant effect on CTF. It is possible a type II error may have been obtained. This conclusion stems from the difficulty in reaching a POD with the stretching protocol. The body of literature regarding stretching to the point of discomfort and a decrease in the H:M ratio or H-Max was the basis of the formulation of the hypothesis, one cannot be certain that these particular neural effects contribute to the development of EAMCs from an EIMC model in this study. However, the results are validated by the similar repetitive same day EIMC protocol differences (34), and day to day changes in participants' CTF (31) in studies that do not apply an intervention to CTF. With no intervention our shifts in CTF match previous studies (31,34) thus providing validation that static stretching had no changes in CTF. Additionally, this provides further proof that inhibitory mechanisms do lead to the cessation of a cramp. The likely mechanism is the burst of inhibitory action potentials from the GTOs derived from the sudden onset of tension from stretching. This does raise questions regarding muscle spindles and GTO's in their role in muscle cramping and relaxation. Within the limits of this protocol static stretching did not significantly raise the participants' CTF. To our knowledge this is the first study completed examining CTF and acute passive static stretchin

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APPENDICES

APPENDIX A

THE PROBLEM

Research Question

Is the Cramp Threshold Frequency (CTF) changed after an acute static stretch to the Flexor Hallicus Brevis (FHB) in an Electrically Induced Muscle Cramp (EIMC)?

Experimental hypothesis

1. Acute static stretching prior to an EIMC will cause an increase in CTF, reducing the susceptibility of EAMC
2. Control Post Test CTF will be lower than intervention post CTF
3. Differences in post CTF means will differ; Control post CTF will be lower than stretch post CTF

Assumptions

1. Participants answered questionnaires honestly
2. Participants did not participate in strenuous lower body activity 24 hours prior to laboratory visits
3. TF for EIMC is a true measurement of propensity to cramp

Delimitations

1. All participants were between the ages of 18-28 at the time of recruitment
2. All participants were free of neurological and metabolic pathologies
3. All participants were free of injury to the dominant leg within the past 6 months
4. All participants completed the EIMC protocol
5. Cramp protocol was administered immediately following stretching regimen or proper time restraint

Optional Definitions and Abbreviations

1. CTF- Cramp Threshold Frequency : The lowest amount of frequency (Hz) needed to elicit a muscle cramp
2. EIMC- Electrically Induced Muscle Cramp: A muscle cramp that is induced by a train of electrical pulses that results in a maintained contraction and an increase in baseline EMG after cessation of electrical pulses.
3. EMD- Electromechanical Delay: The delay in force transmission from muscle to bone (Costa 2012)
4. EMG- Electromyography: Recording of compound action potentials that reach the skin.
5. EAMC- Exercise Associated Muscle Cramp: is a painful, spasmodic, involuntary contraction of skeletal muscle that occurs during or immediately post exercise (Schwellnus 1997)
6. FHB- Flexor Hallicus Brevis: An intrinsic muscle of the foot. The FHB has an origin on the cuboid and lateral cuneiform bone with an insertion on the medial base side of the 1st proximal phalanx.
7. GTO- Golgi Tendon Organ: Tension receptor found near the tendon which is sensitive to rapid increases in force generation.
8. Hz- Hertz: A unit of frequency per cycle. In this instance it is the number of electrical stimulations over a 1 second period of time.
9. MTU- Musculotendinous Unit or Muscle-Tendon Unit: Is the junction point of the end of the muscle and the beginning of the tendon.
10. Plastic Deformation: Change in soft supporting tissue of the muscles. Re-orientation of supporting tissues (22, 16)

11. Threshold Frequency (TF) analogous to Cramp Threshold Frequency (CTF): Minimum amount of electrical stimulation needed to elicit a muscle cramp in an electrically induced muscle cramp.
12. Viscoelastic properties: non-linear decrease in tension when the muscle is undergoing a stretch (18).

Limitations

1. Cramps were elicited via electrical stimulation rather than exercise.
2. The use of a convenient sample
3. Small Sample Size (17)

Significance of the study

Exercise Associated Muscle Cramps (EAMC) are defined as an involuntary, painful, sudden contractions of skeletal muscle and can be recognized clinically by visible bulging and firmness of the muscle. EAMC's remain a problem for athletes, and the recreational public, as they can interfere with physical activity. In a laboratory setting, it is difficult to test EAMCs do to their unpredictable nature. This causes difficulty in controlling the experiments and other factors. This has led to the development of a reliable and reproducible model of EIMC that allows for proper controlled experiments to be conducted.

One hypothesis for EAMC has been an imbalance between GTO's and muscle spindles resulting in a sustained excitation of the alpha motor neuron pool. Stretching is used as the immediate treatment for EAMC. It has also been shown to decrease the excitatory action potentials in the spine. Thus, if stretching is the immediate treatment for cramping, accompanied with its inhibitory actions at the spinal cord should theoretically reduce the susceptibility of an EAMC. This hypothesis has not been tested in a laboratory setting. Determining the effects of static stretching could help devise a strategy for prevention and treatment techniques.

APPENDIX B

ADDITIONAL METHODS

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Adult Consent Form 1

HEALTH HISTORY QUESTIONNAIRE			
Study Title**: Effects of Acute Passive Static Stretching on Cramp Threshold Frequency**			
Please answer the following questions to the best of your knowledge:			
1.	Do you regularly exercise 20-30 minutes at least twice a week?	Yes	No
2.	Are you currently under a doctor's care?	Yes	No
3.	**Women only:** Are you pregnant or do you think you might be?	Yes	No
4.	Do you have a pacemaker?	Yes	No
5.	Do you have, or suspect that you have, any circulatory problems, conditions, disorders, or diseases?	Yes	No
6.	Has your doctor ever said that you have a heart condition and that you should only do physical activity recommended by a doctor?	Yes	No
7.	Do you have, or suspect that you have, any rheumatoid (joint) or muscular conditions, disorders or diseases?	Yes	No
8.	Do you experience numbness, tingling, or decreased sensation in extremities, or have other neurological problems, conditions, disorders, or diseases?	Yes	No
9.	Do you have any problems, conditions, disorders or diseases that affect your ability to	Yes	No

	keep your balance?		
10.	Have you taken any prescription medication in the last 14 days (Coumadin, Heparin, antidepressants, prescription sinus medicine, etc.)?	Yes	No
11.	If answered Yes to #10. Could you please list them.		
11.	Have you ever experienced electrical stimulation?	Yes	No
12.	If you answered "yes" in question #10, did you have an adverse reaction to electrical stimulation?	Yes	No
13.	Do you have a history of experiencing fainting episodes?	Yes	No
14.	Have you ever, or currently, experienced a muscular injury to the leg and/or foot of the leg you would use to kick a ball?	Yes	No
15.	Do you know of any other reasons why you should not undergo physical activity? This might include severe asthma, diabetes, a recent sports injury, or serious illness.	Yes	No

I (Print Full Name) _____ declare that the above information is correct at the time of completing this questionnaire Date ___/___/___

Please Note: If your health changes so that you can then answer YES to any of the above questions, tell the experimenter/laboratory supervisor. Consult with your doctor regarding the level of physical activity you can conduct.

- If you have answered **YES** to one or more questions:

Talk with your doctor in person discussing with him/her those questions you answered yes. Ask your doctor if you are able to conduct the physical activity requirements.

Doctor's signature ……	Date ……/……/…….
Signature of Experimenter……	Date ……/……/…….

Informed Consent Form

Title of study: *Effects of Passive Static Stretching on Cramp Threshold Frequency*

- I have read and understood the subject information sheet.
- I understand what the project is about, and what the results will be used for.
- I have completed the pre-test questionnaire.
- I am fully aware of all of the procedures involving myself, and of any risks and benefits associated with the study.
- I know that my participation is voluntary and that I can withdraw from the project at any stage without giving any reason.
- I am aware that my results will be kept confidential.

Volunteer's name _____

Volunteer's signature _____

Witness' signature _____

Date _____

Experimenter's signature _____

CMU
CENTRAL MICHIGAN UNIVERSITY

Adult Consent Form 2

Information Sheet

Title: Effects of Acute Passive Static Stretching on Cramp Threshold Frequency

What is this study about?

Exercise-associated muscle cramps (a subcategory of muscle cramp) are a common problem for physically active individuals. Due to muscle cramps unpredictability, they are difficult to study. Since we want to better understand their origin, we have developed a model to electrically cause a muscle cramp. If we can learn more about theories to explain how exercise associated muscle cramps develop, prevention and treatment interventions can be created. To asses weather a single bout of static stretching will have any impact on cramp frequency threshold of the flexor hallucis brevis of the foot.

What will I have to do?

You will be asked to report to the Health Professions Building in the Exercise Physiology Research lab. We ask that you come to the laboratory four times, once for familiarization and consent forms. The other times for complete testing. Each session will last approximately 1 hour and will occur every other day, to compare your response in cramp threshold to acute stretching and no stretching. Your total time commitment will be approximately 3 hours. You will need to wear shorts for all test sessions. Your foot will be stretched according to correct protocol; we will then cramp your big toe using an electrical pulse that lasts two seconds each time (every 1 minute until cramp occurs).

What are the benefits?

Although there are no direct benefits to the participant in this study, the results may determine the neuromuscular mechanisms associated with muscle cramps. These findings may provide insight to the development of prevention protocols for individuals who suffer from exercise-associated muscle cramps.

What are the risks?

Risks are minimal, but as most stimulation treatments you may feel some muscle soreness and uncomfortable sensations in the foot that is being electrically stimulated. There is also a possibility of pain or discomfort at the site of stimulation, which has been shown to dissipate after 30 minutes.

What if I change my mind and no longer want to take part in the study?

If at any time you wish to discontinue being a participant in the study, you are encouraged to withdraw immediately without consequences of any kind. You may also refuse to answer any questions that you do not want to answer. This will remain confidential, as you have the option of pulling out without the risk of information being released.

What happens to the information?

The information retrieved from this study will be dealt with and handled in complete confidence whereby results of the participants, as well as their confidentiality, are the first priority of the researchers carrying out the experiment. After the completion of the study data will be kept

electronically on the principal investigator's password-protected computer. All information collected will be kept for 3 year period in accordance to IRB regulations.

Who else is taking part?

The sample size is estimated to be approximately 30 Central Michigan University students from the college of Health professionals.

What if something goes wrong?

In the unlikely event that something goes wrong all safety procedures will be followed in accordance to rules recommend by the IRB. If a medical emergency does occur at any time throughout the protocol the proper authorities will be contacted for immediate medical attention. The event will be reported to the project supervisor immediately, as well as the submission of the adverse/reportable event form to the IRB.

What if I have more questions or do not understand something?

If you do not understand any aspect of the experiment we would urge you to come forward to myself the researcher, or indeed the principal investigator, contact information is listed below. It is important that the participant feels completely at ease throughout the experiment.

Identification of Investigators

If you have any questions or concerns about this research, please contact:
Primary Investigator: Gino Panza
Home phone: 7343442159
Email: panza1gs@cmich.edu

RIGHTS OF RESEARCH SUBJECTS

You are free to refuse to participate in this research project or to withdraw your consent and discontinue participation in the project at any time without penalty or loss of benefits to which you are otherwise entitled. Your participation will not affect your relationship with the institution(s) involved in this research project.

If you are not satisfied with the manner in which this study is being conducted, you may report (anonymously if you so choose) any complaints to the Institutional Review Board by calling 989-774-6777, or addressing a letter to the Institutional Review Board, 251 Foust Hall Central Michigan University, Mt. Pleasant, MI 48859.

My signature below indicates that all my questions have been answered. I agree to participate in the project as described above.

_____ _____
Signature of Subject Date Signed

A copy of this form has been given to me. _____ Subject's Initials

_____ _____
Signature of Responsible Investigator_____ Date Signed

Specific Testing Protocol
Note sheets in the lab. IE, what is on the GRASS and on the wall, stretch protocol if sore
Other tables that augment Methods

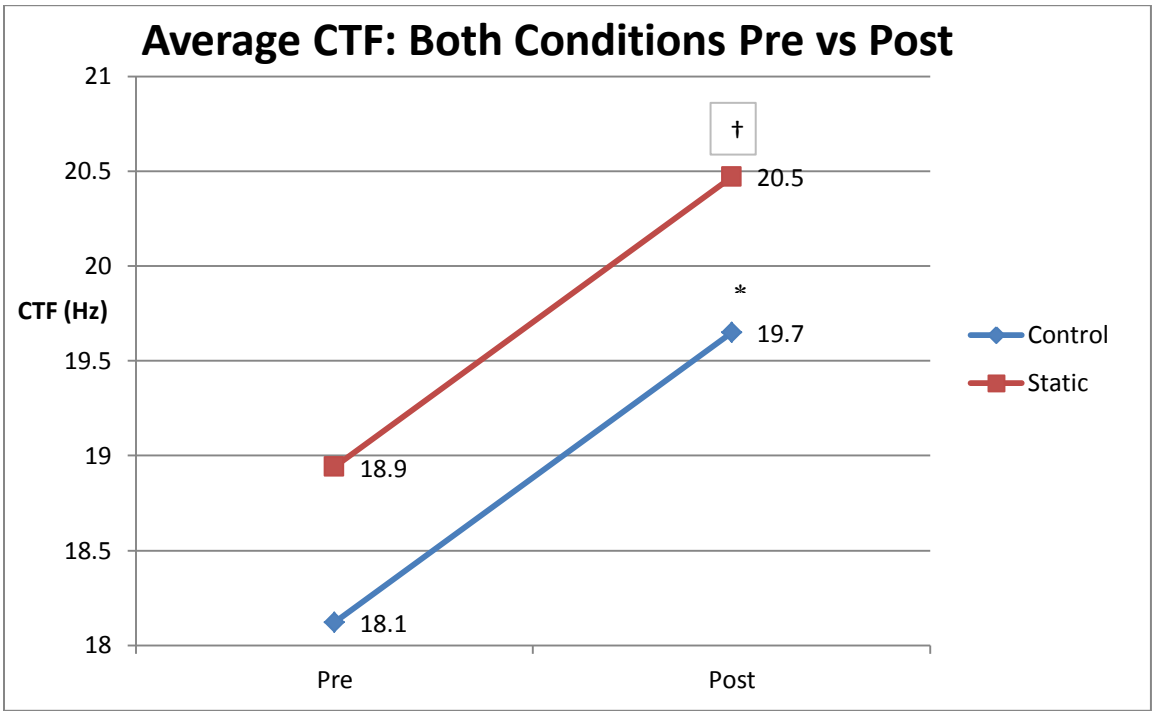
APPENDIX C

ADDITIONAL RESULTS

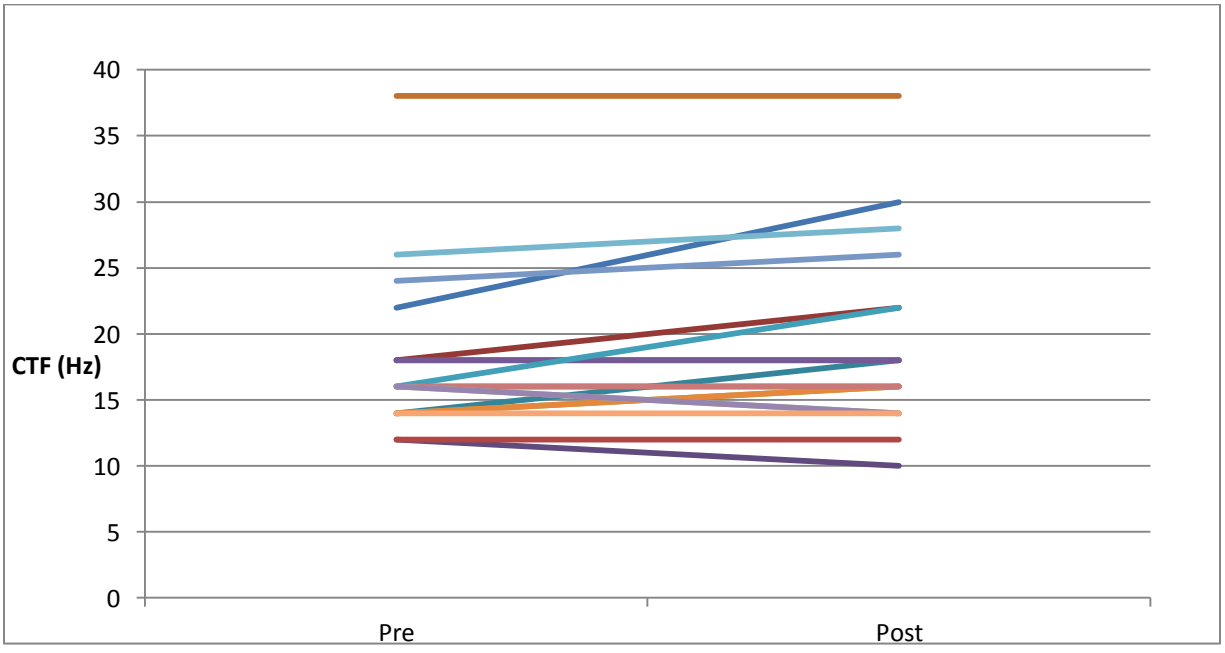
Raw Data of all CTFs of each included participant

	Familiarization 1	Familiarization 2	Control		Static Stretch	
Subject	CTF (Hz)	CTF (Hz)	Pre (Hz)	Post (Hz)	Pre (Hz)	Post (Hz)
85418	12	14	18	18	12	14
14982	16	20	18	22	22	32
34819	20	24	16	16	28	30
96936	12	8	12	10	14	12
52466	8	16	14	18	18	18
73991	24	22	38	38	18	18
17191	20	18	22	30	22	24
19461	12	8	12	12	6	6
53124	16	14	14	16	18	20
49789	NC	34	18	18	20	16
62981	NC	16	16	22	20	22
86112	18	18	14	16	18	22
38643	20	18	24	26	32	32
85357	24	22	16	16	18	18
25517	NC	14	16	14	18	20
96613	22	NC	26	28	24	28
12468	20	14	14	14	14	16
Mean±SD			18.1±6.5	19.7±7.3	18.9±6.0	20.5±7.1

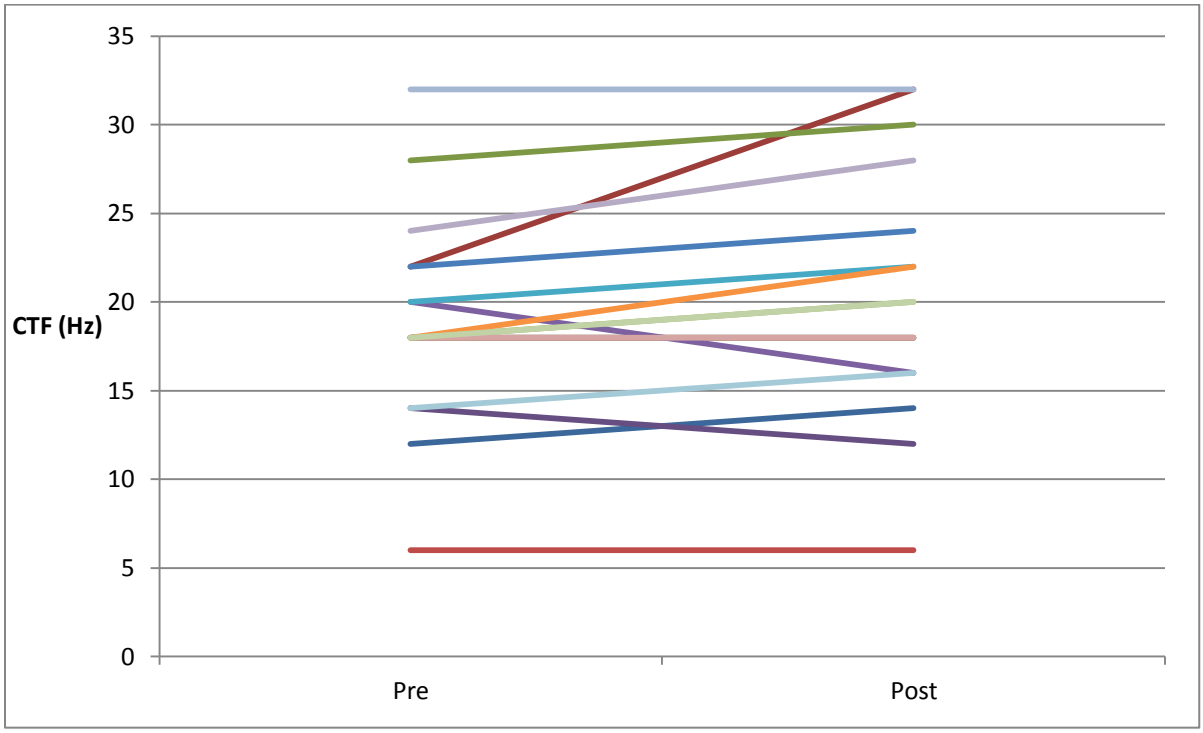
The mean of differences in CTF from pre- to post-test in both the control and static condition groups. * Significant difference between pre-test to post-test within the control group ($p<.05$). †Significant difference between pre-test to post-test within the static stretching group ($p<.05$). No significant difference was found between treatments.



Control Condition CTF Changes Pre vs Post



Static Stretching Condition CTF changes Pre to Post



Data Collection Sheets.

Study: Effects of Acute Passive Static Stretching on Cramp Threshold Frequency

Group 1

Subject ID #	Last Name	First Name	Age (Yrs)	Birthday	Gender (M/F)	Height (cm)	Weight (kgs)	BMI	Dominant Limb	Self-Reporting Cramp	Family History of Cramp	Foam Pads	Fam. 1 Ankle°	Fam. 1 Temp.	Fam. 1 CTF	Fam. 2 Ankle°	Fam. 2 Temp.	Fam. 2 CTF	Static Ankle°	Static Temp.	Pre-Static CTF	Post-Static CTF	No Stretch Ankle°	No-Stretch Temp.	Pre No-Stretch CTF	Post No-Stretch CTF

Group 2

Group 2

Subject ID #	Last Name	First Name	Age (Yrs)	Birthday	Gender (M/F)	Height (cm)	Weight (kgs)	BMI	Dominant Limb	Self-Reporting Cramp	Family History of Cramp	Foam Pads	Fam. 1 Ankle°	Fam. 1 Temp.	Fam. 1 CTF	Fam. 2 Ankle°	Fam. 2 Temp.	Fam. 2 CTF	No Stretch Ankle°	No-Stretch Temp.	Pre No-Stretch CTF	Post No-Stretch CTF	Static Ankle°	Static Temp.	Pre-Static CTF	Post-Static CTF

APPENDIX D

RECOMMENDATIONS FOR FUTURE RESEARCH

1. Chronic passive stretching program and fluctuations in Cramp Threshold Frequency (CTF)
2. The reliability and validity of an EIMC protocol for a more applicable muscle for intervention application.
3. Multiple joint angles and CTF
4. BMI in correlation with “high” or “low” TFs.
5. What is considered “high” or “low” TFs
6. CTF and gender differences
7. Electromagnetic hammer stimulation to elicit cramps in healthy participants

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