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Abstract

Task Specific Effects of Repeated Unilateral Eccentric and Concentric Exercise on Spinal Excitability of the Contralateral Homologous Plantar Flexors.

By

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April, 2011

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It is well established that resistance training increases the size and strength of the trained muscles. It is also known that unilateral muscle contractions can produce strength gains in the non-exercised contralateral homologous muscle. This effect of training a muscle and having strength gains on the analogous, opposite side muscle is called cross education. Cross education tends to be greater during eccentric contractions, when the muscle is actively lengthening, compared with concentric contractions when the muscle is actively shortening. The mechanisms behind the strength gains of cross education are less clear. It has been suggested that a change in excitability at the spinal level may mediate cross education. The purpose of this study was to compare spinal excitability in the resting right plantarflexors before, during, and after bouts of unilateral eccentric and concentric contractions of the left plantarflexors. We hypothesized that unilateral plantarflexion facilitates spinal excitability in the resting contralateral plantar flexors, and the facilitation will be task-specific according to the type of muscle contraction. This hypothesis is based on the observation that contraction

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of a remote muscle increases reflex excitability produced in a remote muscle, a phenomenon known as Jendrassik maneuver. Instead of a chronic training study, the present experiment used one exercise session of each contraction type but explored in detail the magnitude and time course of responses in the resting, contralateral right plantarflexor muscles. Subjects participated in two exercise treatments in one day, separated by 10 min of rest. Subjects performed eccentric and concentric contractions, at 90% of maximal voluntary concentric contraction. Each treatment consisted of 5 sets of 10 repetitions, with 120 s of rest between sets, followed by 5 contractions with 120 s of rest between each contraction. During the protocol, H-reflexes were evoked during each contraction over the exercise bouts, every 5 s for 120 s in the between-set rest periods, and every 5 s for 120 s during the follow-up after the 5th exercise bout. Against expectations, spinal excitability decreased ~20% relative to baseline during each of the 5 exercise bouts and returned, in each bout, to baseline in about ~30-35 s after each contraction. In addition, this recovery to baseline was extended in the follow-up so that spinal excitability actually became facilitated and increased ~20% relative to baseline. The data seem to suggest that the somatotopic organization of spinal excitability is more complex than previously thought and it may be inhibitory between pairs of the same muscles during contraction. The data thus suggest that spinal mechanism during exercise is probably not a primary mechanism to mediate cross education but it remains to be determined if the facilitatory after-effects are associated with cross education.

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Task Specific Effects of Unilateral Eccentric and Concentric Exercise on Spinal Excitability of the Contralateral Homologous Plantar Flexors.

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Ryan D. Hill

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Chapter 1: Introduction

It is common knowledge that resistance training will increase the size and strength of the muscles involved in the training. In 1894, a researcher found that with unilateral contractions, there were also strength gains in the resting contralateral homologous muscle (Scripture). This effect of training one side of the body and having strength gains on the opposite side is called cross education. Strength benefits of cross education are often seen after strong voluntary contractions (Hortobagyi, Taylor, Petersen, Russell, & Gandevia, 2003), contractions evoked by electrical stimulition (Hortobagyi, Scott, Lambert, Hamilton, & Tracy, 1999), or even mental rehearsal (Yue & Cole, 1992). Although the effects of cross education are thoroughly documented (Shima, Ishida, Katayama., 2002; Hortobagyi et al., 1999; Shaver et al., 1970), the mechanisms behind these strength gains are less clear. A number of researchers have tried to elucidate the mechanism that mediates cross education. Some studies observed that there is a transmedian signaling at the cortical level that is responsible for the cross education (Hortobagyi et al., 2003; Muellbacher, Facchini, Boroojerdi, & Hallett, 2000), while others observed that there is signaling at the subcortical level (Lee & Carroll, 2007; Meyer, 1995; Muellbacher et al., 2000). Cortical and subcortical mediators are not exclusive, and are thought to occur simultaneously to produce the effects of cross education (Lee & Carroll, 2007). More recently, and less extensively, it has been revealed that excitability at the spinal level might also mediate the benefits of ipsilateral training (Hortobagyi et al., 2003).

Hortobagyi et al. (1997) showed that cross education tends to be greater when using eccentric contractions over concentric or isometric contractions. Hortobagyi et al. (1997) found that concentric training increased concentric strength 30% in the resting limb, and increased isometric strength by 22%, but eccentric training increased eccentric strength by 77% and isometric strength by 39%. This indicates that strength benefits of cross education are task specific to type of contraction. There is some evidence that cortical and spinal excitability are changed according to the type of contraction (Gruber, Linnamo, Strojnik, Rantalainen, Avela, 2009). Nordlund et al. (2002) demonstrated that eccentric contractions had significantly more depression of spinal excitability, about 8%, than compared to concentric contractions in plantar flexors (Nordlund, Thorstensson, & Cresswell, 2002). These data suggest that there is high specificity in contraction type on the way spinal excitability is modulated when specific muscles are voluntarily contracted.

In one study, researchers investigated the effects of chronic training on spinal excitability (Lagerquist, Zehr, & Docherty, 2006). The researchers determined that chronic ipsilateral training produced no increase in spinal excitability. It was concluded that the cross education effect on strength training was due to supraspinal rather than spinal mechanisms; spinal excitability was only measured after the training condition and not during contractions and directly after each contraction.

This study investigated the effect of repeated contractions of the left plantar flexors—i.e., every five seconds for fifty contractions—on spinal excitability during and directly after contractions in the contralateral plantar flexors as measured using the H-reflex.

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Hypotheses

A.) During contraction: depression of spinal excitability would occur under eccentric and concentric contractions

B.) Depression of spinal excitability would vary with contraction type immediately after contraction.

C.) During Rest: There will be task-specific recovery between bouts

D.) During the follow up: There will be task specific recovery after the treatment.

Purpose

The purpose of this study was to explore task specific changes of eccentric and concentric contractions on spinal excitability in the resting contralateral leg during an acute bout of repeated ipsilateral contractions.

Delimitations

The study uses healthy college aged individuals, with no neuromuscular diseases. Individuals outside of this population might have different effects, and the results might not be generalized to these individuals.

Limitations

The H-reflex is sensitive to a variety of factors, including: posting on the joint position of the body, contraction of extraneous muscles, and contraction strength of muscle. Control of all of these factors might be different depending on the participants and might result in different outcomes. Inability of the participant to keep the resting leg from contracting might skew the results.

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Assumptions

All information obtained from the participants is accepted to be true. It was assumed that the available equipment can provide an accurate reading of the h reflex. It was also assumed that the controls were sufficient to minimize extraneous input to the reflex.

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Chapter 2: Literature Review

Cross Education

Cross education is the phenomenon of increasing strength of a muscle group by training the homologous muscle group on the opposite side of the body (Lee & Carroll, 2007). Cross education can be found in both upper and lower extremities (Lee & Carroll, 2007), from the wrist muscles (Hortobagyi et al., 2003) to the larger quadriceps and soleus muscles (Hortobagyi et al., 1999; Shima et al., 2002). This phenomenon has been widely observed, but was first seen by Scripture and co-workers (1894). One researcher observed that the traditional methods of progressive resistance weight training would increase static elbow-flexion strength in the exercised limb as well as in the unexercised limb (Shaver, 1970). Korotkiewski et al. (1979) noticed that isokinetic one-legged exercise of five weeks' duration in ten healthy middle-aged women resulted in a significant increase of muscle force in the exercising leg (14-26%) and, a lesser increase in the nonexercising leg (4-13%). The effects of cross education have been found in both genders (Lee & Carrol, 2007). Also, more recently, researchers have documented this increase in strength by cross education in the unexercised limb (Carroll et al, 2006; Munn et al., 2005). On average, the strength gains made by the resting limb were about a 7% increase from its baseline strength (Carroll, Herbert, Munn, Lee, & Gandevia, 2006; Munn, Herbert, & Gandevia, 2004). In a meta-analysis of cross education studies, Munn et al. (2004) found that, on average, a strength improvement of 35.1 % of the trained limb was seen in the resting limb.

Cross education can be induced by electrical stimulation (Hortobagyi et al., 1999), voluntary effort (Hortobagyi et al., 1999; Maffiuletti, Zory, Miotti, Pellegrino,

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Jubeau, 2006), or even mental rehearsal of unilateral contractions (Yue et al., 1992). This phenomenon has been invoked with various types of muscle contractions (i.e., isotonic, isometric, isokinetic), and is specific to the opposite homologous muscle group and type of contraction (Yue et al., 1992; Hortobagyi et al., 1997).

Eccentric and concentric contractions seem to have different, distinctive, characteristics. In one study, researchers found that eccentric contractions can produce greater force production compared to concentric contractions (Hortobagyi et al, 1997). Along with greater force production, eccentric contractions lead to greater strength gains in both the trained limb and the untrained contralateral limb (Hortobagyi, T., Barrier, Beard, Braspennincx, Koens, Devita, et al., 1997; Hortobagyi et al, 1997). Neural control of muscle contraction seems to also be unique during muscle lengthening. Hortobagyi et al. (1997) found that cross education gains tends to be larger when one uses eccentric contractions compared to concentric. Thus, this indicates that the mechanisms of cross education might be task specific based on contraction type. However, these mechanisms behind why and how cross education occurs are still being debated.

Mechanisms

Two possible mechanisms have been shown to facilitate cross education effects in humans. First, unilateral voluntary contractions can cause complex changes in motor pathways mediating the resting limb (Hortobagyi et al., 2003; Lee & Carroll, 2007). Second, supraspinal adaptations that are predominately involved in the control of the trained limb can be accessed by the untrained, resting limb, when induced to produce a maximal force contraction (Lee & Carroll, 2007). It has been suggested that both of

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these mechanisms provide facilitation of cross education effects, and can occur simultaneously in the body (Lee & Carroll, 2007). Both mechanisms use cross-mediating signals in the central nervous system (CNS) to provide training benefits of unilateral contractions. These cross communicating networks can be broken down into either supraspinal (above the spinal cord and vertebral column) or spinal pathways (Carroll et al., 2006). Both sides of the body are connected via different mechanisms that help both sides share information. Various levels of the neural pathway (cortex, subcortex, spinal cord) share information by interneurons, callosal pathways (Iwamura , Taoka , Iriki, 2001), and commissural pathways (Jankowska , Edgley , Krutki , and Hammar , 2005), which act as mediators between the different levels and sides of the bodies neural system and might hold the keys to cross education.

The Neural Pathway

Transcranial magnetic stimulation (TMS)

TMS is a noninvasive method to excite neurons in the brain: weak electric currents are induced in the tissue by rapidly changing magnetic fields. This way, brain activity can be triggered with minimal discomfort, and the functionality of the circuitry and connectivity of the brain can be studied. It can be used to demonstrate the conduction of nerve impulses from the motor cortex to the spinal cord. By stimulating different points of the cerebral cortex and recording responses, e.g., from muscles, one may obtain maps of functional brain areas. TMS is helpful in showing excitability in the motor cortex, and may help discover the mechanisms behind cross education. In many fields of research, TMS is used to stimulate motor evoked potentials (MEP) in the motor cortex. MEP is an electrical potential recorded from the nervous system, more

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specifically the motor cortex, following presentation of a stimulus, as distinct from spontaneous potentials as detected by electromyography (EMG) (George, Lisanby, Sackeim, 1999).

Motor Cortical Pathway

The motor cortex is the section of the cerebral cortex involved in the preparation, control, and implementation of voluntary motor functions. As the motor axons travel down through the cerebral white matter, they move closer together and form part of the posterior limb of the internal capsule. These fibers continue descending down into the brainstem where several of them, after crossing the midline, distribute to the cranial nerve motor nuclei, with a minority of motor fibers synapsing with lower motor neurons on the same side of the brainstem. After crossing over to the medulla oblongata, the axons travel down the spinal cord as the lateral corticospinal tract. Fibers that do not intersect in the brainstem travel down a separate ventral corticospinal tract and most of them cross over to the contralateral side in the spinal cord, curtly before reaching the inferior motor neurons.

Hortobagyi et al. (2003) and Francis, et al. (2009) supported the idea that during ipsilateral contractions the ipsilateral motor cortex, the side controlling the resting limb, has increased excitability. Hortobagyi et al. (2003) showed that with voluntary ipsilateral wrist contractions, there was increased excitability in the ipsilateral motor cortex. This increased excitability in the ipsilateral cortex, which controls the contralateral side, is possibly part of the mechanism behind cross education.

Muellebecher et al. (2000) also looked at motor cortical excitability during voluntary forceful ipsilateral right hand contractions. They stimulated MEPs using TMS

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to investigate if there was any excitability during the ipsilateral contraction in the ipsilateral motor cortex. With forceful voluntary contractions they found facilitation of the right motor cortex, by an increase in ipsilateral MEPs with stronger contraction of the right abductor pollicis. This supports that with a strong voluntary ipsilateral contraction there is an increased excitability in the ipsilateral motor cortex (Muellbacher et al., 2000). These investigators believe that the involvement of the ipsilateral hemisphere might originate from a subcortical network with connections to both primary motor cortices, and that this connection could provide early co-activation of the ipsilateral hemisphere during such forceful muscle contractions.

In a recent study, Hortobágyi et al. (2009) investigated whether there were task specific responses to, lengthening and shortening, ipsilateral exercise on motor cortical excitability. Thirty-one right-handed participants ipsilaterally contracted left wrist flexors, both concentrically and eccentrically, while right wrist flexors remained at rest. TMS protocols were used to evaluate the excitability of the ipsilateral (left) motor cortex at rest and during voluntary contraction. Preliminary data indicated that eccentric contractions produced more excitation in the ipsilateral cortex than shortening contractions. This supports the idea that there are task specific responses to ipsilateral eccentric and concentric contractions on excitability in the ipsilateral motor cortex.

In summary, with a strong contralateral contraction, there is an increased excitability of the ipsilateral motor cortex by many different pathways, which is associated with increased strength on the contralateral side. Additionally, motor cortical excitation seems to be task specific between concentric and eccentric contractions.

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Spinal Pathway

The Hoffman reflex (h-reflex) is an electrically elicited response of a monosynaptic stretch reflex, which provides a noninvasive method of monitoring the integrity and functionality of the central nervous system, particularly information about the monosynaptic pathway (Murphy et al., 2008). The H-reflex bypasses the muscle spindle and, therefore is useful for assessing modulation of monosynaptic reflex activity in the spinal cord. This measurement can be used to assess the response of the nervous system, and can illustrate excitability of alpha motor neuron given that other factors affecting presynaptic inhibition are controlled (Murphy et al., 2008). Differences in the magnitude of the H-reflex in the intended muscle, rested and active, suggest specific mechanisms modulating the spinal and cortical pathway. Inhibition of the contralateral H-reflex on the homologous muscle was observed in a study in which participants forcefully contracted ipsilateral wrist flexors (Hortobagyi et al., 2003). The authors found that this inhibition of the H-reflex on the contralateral side was only depressed at high percentage, 50% to 75%, of a maximal contraction. Interestingly, this inhibition of the H-reflex lasted, on average, about thirty seconds before returning to resting levels. This is a dramatically different than cortical excitability that only lasts, on average, five seconds.

The reasons behind this inhibition and why it lasts so long after the contraction are still unclear, but there are some theories. Hortobagyi et al. (2003) suggested that alpha motor neurons controlling the targeted muscle could be inhibited presynaptically as a result of a strong contralateral contraction. Lee & Carroll (2007) suggested that inhibition of the H-reflex is caused by reciprocal inhibition, or trans-synaptic

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transmissions between 1a afferent fibers and motor neurons through a circuit of interneurons. These interneurons are facilitated by many different inputs including: the brain, contralateral spinal segments, propriospinal pathways and other undiscovered inputs

In short, results seem to indicate that contralateral spinal excitability is depressed during a forceful voluntary ipsilateral contraction. This depression also seems to stay depressed, on average, for 30 seconds after the contraction. There seems to be enough evidence to indicate that spinal pathways are modulated during a single strong contralateral contraction. The task specificity of different contraction types (i.e eccentric and concentric) on spinal excitability has not been investigated and still remains unclear.

Chronic Exercise

Lagerquist et al. (2006) examined modulation of the H-reflex after a 5-week long strength training of contralateral ankle plantarflexors. The researches found that there was no modulation of the H-reflex in the resting limb, although there was an increase in strength in the resting limb. This is the only study that has looked at chronic training on cross education in terms of neural plasticity. The researchers concluded that cross education's affect on strength may be due to supraspinal pathways opposed to spinal mechanisms. The researchers only looked at spinal excitability during rest before and after the longitudinal study, but not during contraction. The present study looked at spinal excitability both during and after a set of contractions, to investigate if there is different modulation control for eccentric and concentric contractions. This study also investigated if there is a task specific recovery pattern after an acute bout of exercise.

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Summary

The review of literature indicates that there is evidence that both cortical and spinal mechanisms play a role in cross education. Research suggests that eccentric and concentric contractions are mediated through different mechanisms both muscular and neural. The data also support the idea that cortical excitability is task specific and is modulated differently between eccentric and concentric contractions and task specific differences for eccentric and concentric contractions on spinal excitability may exist. There has been little research into the task specific affect of acute repeated ipsilateral contractions, concentric and eccentric, on spinal excitability on the contralateral homologous muscle. This is why the present study took a cross sectional look at how a single bout of repeated contractions, lengthening and shortening, effects spinal excitability. This study investigated if both during and after repeated bouts of contractions there are task specific differences on spinal excitability in the resting contralateral leg, and if there is a task specific recovery pattern after the acute bout of exercise.

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Chapter 3: Methods

Subjects

Ten healthy, young college-aged right-footed subjects (mean age of 21 +/- 3 years).

Inclusion Criteria

Right-footed young individuals based upon what foot they use to kick a ball were used. All participants were healthy and with no present or past history of any neuromuscular injury or disorder. Participants did not currently have or have had past history of disorders that might affect nerve conduction. All participants refrained from having caffeine within 12 hours of the study. Participants did not have current or past history of fracture of the upper or lower limbs. Participants were able to provide informed consent. Presence of H Reflex recruitment pattern in the right soleus muscle was mandatory.

Exclusion Criteria

Individuals with past history of fracture in lower limb and any other systemic disorder were excluded from this study. History or onset of any neuromuscular disorder, which is characterized by altered nerve conduction, was excluded from this study. Also, people with pacemakers were excluded from this study. Even if all inclusion criteria are met, participants with an absence of an h reflex in right soleus could not participat

Study Design

This study was a repeated measures design, with all subjects participating in all conditions. The study's design included two contraction types, eccentric and concentric contractions, at 90% of the participant's max voluntary contraction. Each treatment

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consisted of five bouts of exercise, concentric or eccentric contractions, and each set included ten repetitions (trials), with 120 seconds of rest between each set. Five follow bouts consisting of 120 seconds were taken after each treatment (figure 1). Subjects participated in both treatments, eccentric and concentric contractions, and were randomly assigned to which they would participate in first. Both treatments were performed on the same day, with ten minutes of rest between the two treatments. During the protocol, H-reflexes were evoked during the ten trials and at rest every 5 seconds for 120 second in the right soleus. H-reflexes were also taken during the follow up trials. In all conditions, the right leg remained at rest during the entire experiment.

Equipment

A Digitimer stimulator model DSA7 (Digitimer Limited. Welwyn Garden City, UK) was used to stimulate the tibial nerve and evoke h reflex at right soleus. This stimulator used a pulse at 400 V with pulse duration of 1 mS, with a stimulus intensity range of 0 to ~25 mA. A Biopac 100c system, using two standard gold cup electrodes, was used to collect the EMG data from the soleus muscles. A 770 HUMAC Isokinetic Dynamometer (Computer Sports Medicine, Inc. Stoughton, MA.) was used for left plantar flexor movement. Signal version 3 software, by Cambridge Electronics, was used to collect data. Data were converted from analogue to digital using a CED 1401 A/D board (CED limited. Cambridge, England)

Experimental Protocol

All subjects were college students recruited from East Carolina University (ECU). All subjects filled out a self-reported medical history indicating that they have no known physiological or functional conditions that would prohibit them from performing exercise

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for a brief period of time, and had no known, recent, or previous injuries that would prevent them from participating. Subjects were then given, and explained, an informed consent document of the experiment and its inherent risks. Participants were then probed for presence of H Reflex in the right soleus.

Participants reported to the ECU's Biomechanics Lab having at least two-hours of rest from exercise and twelve-hour abstention from alcohol, caffeine, and any medication that affects the central nervous system.

Probing For H-Reflex

Participants were asked to lie down, prone, on the HUMAC dynamometer. Right and left soleus muscles were palpated. Skin was prepared for EMG by using alcohol pads and lemon preparation gel to clean area of dead skin and oils. Electrodes were placed along the belly of the soleus muscle in the direction of the muscle fibers and attached to the Biopac 100c system. Signa Gel electrode gel was used on the electrodes to decrease impendence. The first electrode was placed two centimeters distal to the lateral gastrocnemius and two centimeters lateral to the posterior midline of the leg. The second was placed half the distance between the popliteal fold and the medial malleolus. One ground electrode was placed on each shank to decrease signal noise. A three-cm interelectrode distance was used. The Tibial nerve was stimulated via a Bipolar stimulating electrode attached with the Digitimer Stimulator and delivered over the popliteal fossa. The electrode was placed over the posterior Tibial nerve in the popliteal fossa. Cathode was distal and anode was proximal for the stimulus electrode, and was expected to give the best results (Zehr, 2002). The h reflex was probed before the protocol started, and h-max was then found. When h-max was found, stimulus

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intensity was then decreased until half of h-max is found. This stimulus, used to elicit half the h-max, was recorded and used for the protocol.

Procedure

Participant's left foot was strapped into the dynamometer while lying prone. Participants contracted left plantar flexors, at 15 degrees per second, over a 30-degree range of motion (-15 to 15^{o}). The h reflex was again elicited in the right soleus muscle using the same procedure as used for the probing. The protocols stimulus intensity was set to elicit 50% of the h-max. EMG recordings were also recorded in both the right and left soleus muscles. Right plantar flexors, along with the rest of the body, remained at rest for the entire experiment. Participants were advised, and reminded to contract left plantar flexors while the rest of their body remains at rest.

The max voluntary contraction (MVC) force of the left plantar flexors was recorded for the concentric and eccentric contractions on the HUMAC. These max forces were used to calculate the absolute force of the concentric and eccentric contractions. Throughout the rest of the protocol, the participants contracted at 90% of their concentric or eccentric absolute force. Torque was controlled for by using visual targets that the participants matched on every contraction. Participants then plantar flexed for a set of 10 repeated contractions (trials) with each contraction lasting two seconds and a three second reset back into dorsiflexion. Participants participated in five consecutive bouts, with two minutes (120 seconds) of rest between sets. H reflexes were taken both, during the contraction, and every five seconds during rest (figure 13). This design was repeated for both eccentric and concentric contractions, and subjects were randomly assigned to their initial condition. During the entire experiment, participants were

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reminded to keep their right leg, neck, shoulder, and other muscles relaxed. Both the concentric and eccentric protocols were done in one session.

Background EMG

Voluntary activation of right soleus during contractions in left plantarflexor was measured by background EMG activity. Right soleus EMG was measured as a percentage of maximum EMG produced during left plantarflexor MVC.

Data Analysis

H-reflexes were analyzed for peak-to-peak amplitude with Signal 3.1 software. EMG and force data were extracted into an Excel spreadsheet. Average H-reflex values were calculated for each trial.

Statistical Analysis

Two Intervention contraction (2) by bout (5) by trials (10) ANOVAs with repeated measures on all 3 factors were used to analyze change in H reflex amplitude during contraction and during the inter-bout rest. A separate, contraction (2) by Trial (8) ANOVA with repeated measures on both factors was used to analyze change in h reflex amplitude of the follow-up. Sphericity was adjusted for during the bout and trial measures, but there were no changes in significance. When appropriate, Tukey's post hoc contrast was used to determine the means that are different at $p < 0.05$. Student T-tests were used to analyze the modulation of spinal excitability from baseline during contraction, inter-bout rest, and follow-up periods; paired t-tests were used to analyze the task specificity of the modulation. A contraction (2) by bout (5) ANOVA was used to analyze the background EMG.

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Chapter 4: Results

The main finding of the present study was that ten voluntary contractions of the left plantar flexors produced almost 30% depression of the right soleus' spinal excitability— with no effect across bouts. On average, spinal excitability started recovering after the contractions and stabilized at the control value after almost 20-25 seconds, but continued to facilitate past baseline values; a facilitation of 20% was recorded at the end of the inter-bout rest period. The purpose of this study was to look at task specific changes of eccentric and concentric contractions on spinal excitability in the resting contralateral leg during acute repeated ipsilateral contractions. This chapter is separated into five sections: 1) spinal excitability during contraction, 2) during rest, 3) and during the follow-up, 4) background EMG, and 5) control experiments. Data for all the results discussed here are given in Appendix C in table format.

Right Soleus H Reflex during Left Plantarflexor Contraction

Contraction Main Effect

Figure 1A represents the main effect during contraction averaged across bout and trial. There was significant depression, ~40%, after the concentric contraction (p=.002; CI= 17.99-58.01), and a significant depression after the eccentric contraction (p=001; CI= 24.78-55.83). Figure 1A also shows that there was no significant difference between contractions types (p=.855, CI= -31.72- 22.11).

Bout and Trial Main Effect

Figure 1B shows the bout main effect during contraction collapse across contraction and trial. There was no significant effect across bouts (p=.359, F=1.12)

Figure 2 represents the trial main effect during contraction collapsed across

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contraction type and bout. There was significant trial main effect at p=.001, F=16.7; Tukey's post hoc analysis revealed significant depression after each trial in relation to T1 (p<.050).

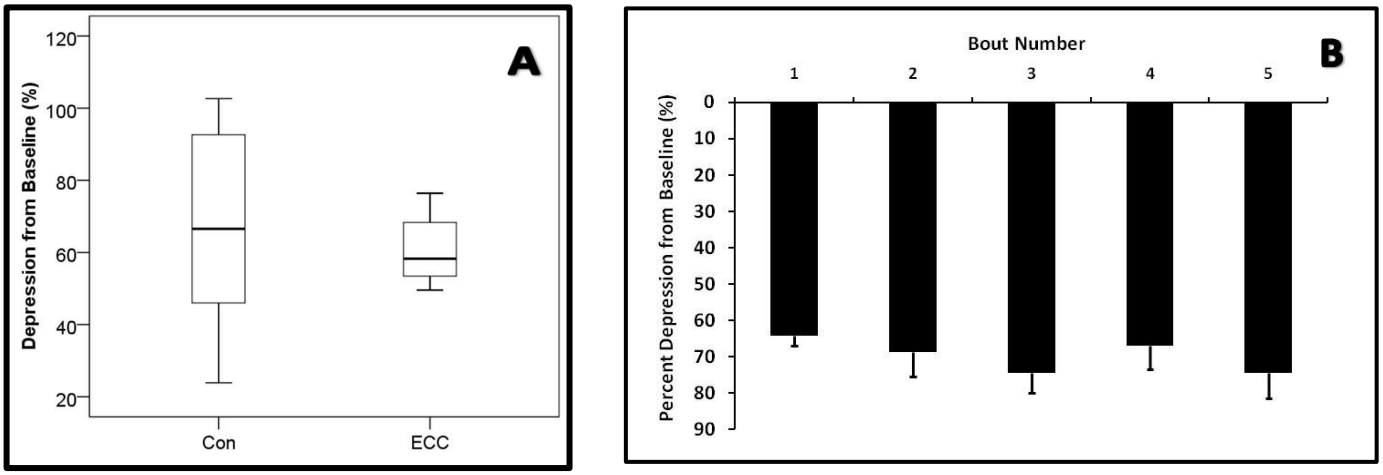

Figure 1: **Contraction (A) and bout (B) main effects during contraction. Box plot was constructed from 10 averages (one for each participant) of depression from baseline during contraction—for both treatments**.

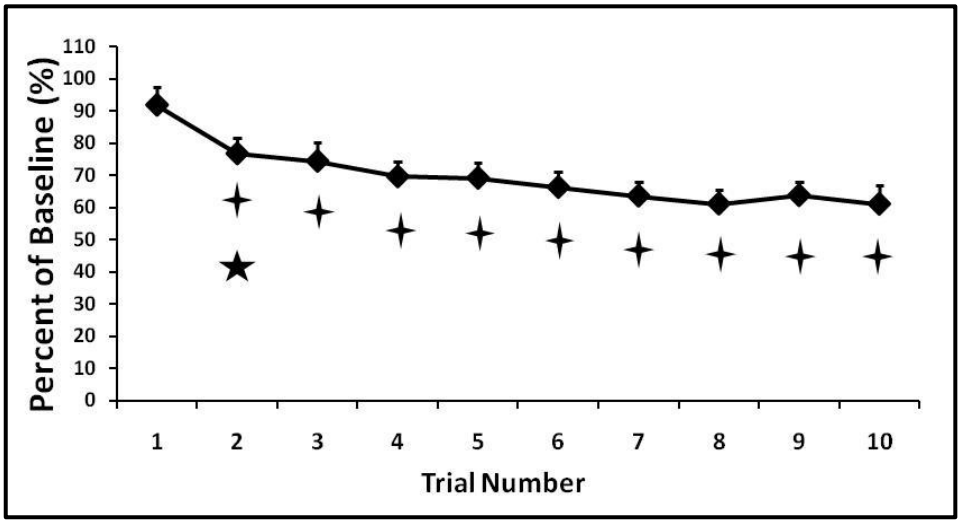

Figure 2: **Trial main effect during contraction. Tukey's post hoc analysis (p<.05) indicated by:**

★ **= significantly different from previous trial**

✦ **= significantly different from T1**

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Right Soleus H Reflex during Inter-Bout Rest

Contraction Main Effect

Figure 3 shows the contraction main effect during inter-bout rest. There was significant difference between contraction types (p=.029, CI=1.64-24.1), with a mean difference of 12.92%. There was a significant facilitation from baseline concentric (P=.037, CI=1.70-42.1)—with and mean facilitation of 6%, while eccentric (P=.033, CI=-7.55-3.) was not significant. These values based on a paired t-test (appendix C).

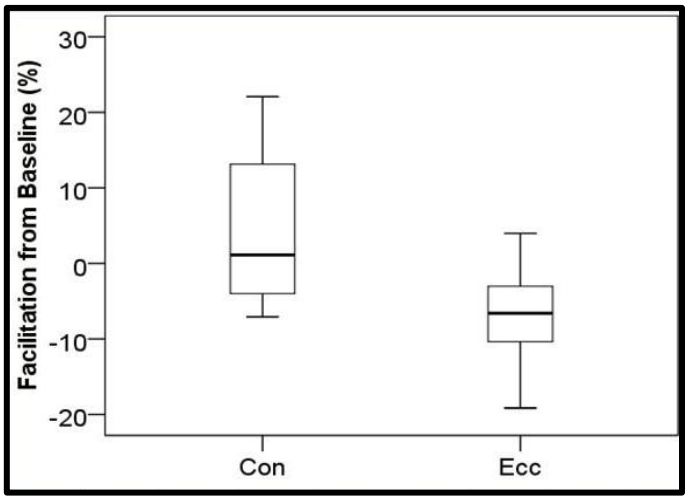

Figure 3: Contraction Main effect during inter-bout rest. Box plot was constructed from 10 averages (one for each participant) of depression from baseline during inter-bout rest—for both treatments.

Bout and Trial Main Effect

Figure 4 shows the bout main effect during inter-bout rest. There was no significant bout main effect or bout by contraction interaction effect during the inter-bout rest (p=.455, F=.953).

v Figure 5 represents the trial main effect during inter-bout rest, collapsed across

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contraction and bout. There was a significant trial main effect at p<.000, F= 24.1; Tukey's post hoc analysis revealed significant depression after each trial in relation to T1 (p<.050).

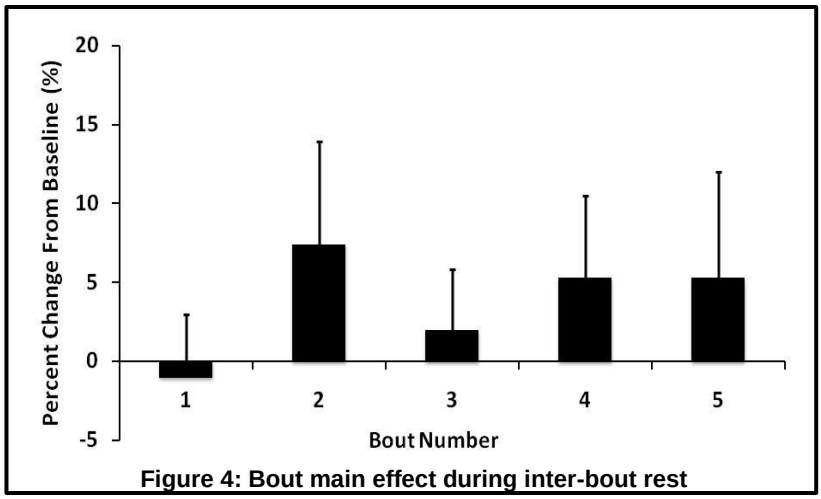

Figure 4: Bout main effect during inter-bout rest

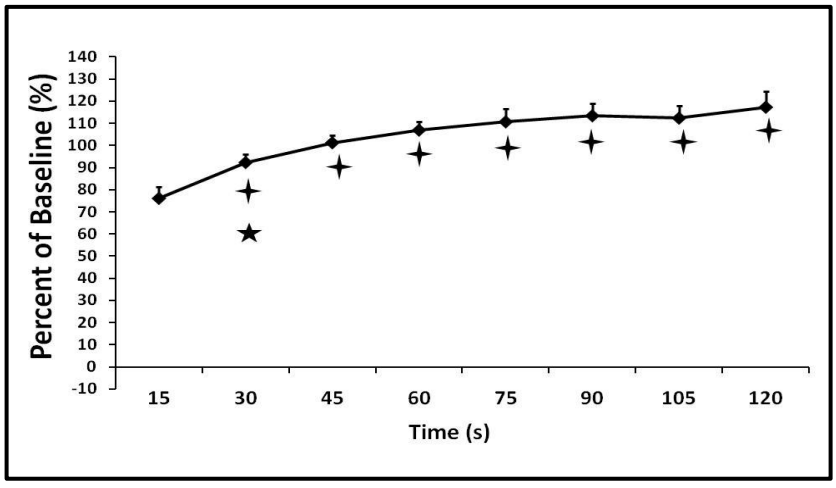

Figure 5: Trial main effect during inter-bout rest. Tukey's post hoc analysis (p<.05) indicated by:

★ **= significantly different from previous trial**

✦ **= significantly different fromT1**

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Right Soleus H Reflex during Follow-Up

Contraction Main Effect

Figure 7 shows the contraction main effect during inter-bout rest collapsed across bout and trial. A difference of 5% between eccentric and concentric contractions was found to be not significant (p=.351, F= .966). There was a significant facilitation from baseline for both concentric (P=.014, CI= 2.87-19.91)—with a mean facilitation of 11.4%, and eccentric (P=.040, CI= -2.04-14.88)—with a mean facilitation of 4.4%.

Bout Main Effect

Figure 8 shows the bout main effect during inter-bout rest, collapse across contraction and trial. There was a significant bout main effect during the inter-bout rest (p=.013, F=3.69). Tukey's post hoc analysis revealed significantly less facilitation after bout 2 in relation to B1 (p<.050).

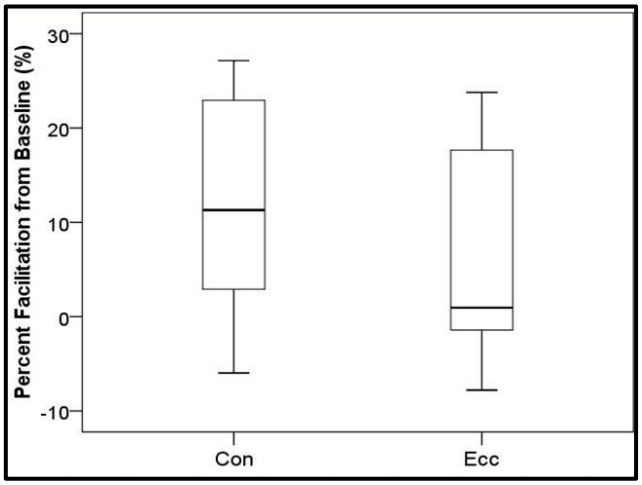

Figure 7:Contraction main effect during follow-up. Box plot was constructed from 10 averages (one for each participant) of depression from baseline during follow-up—for both treatments.

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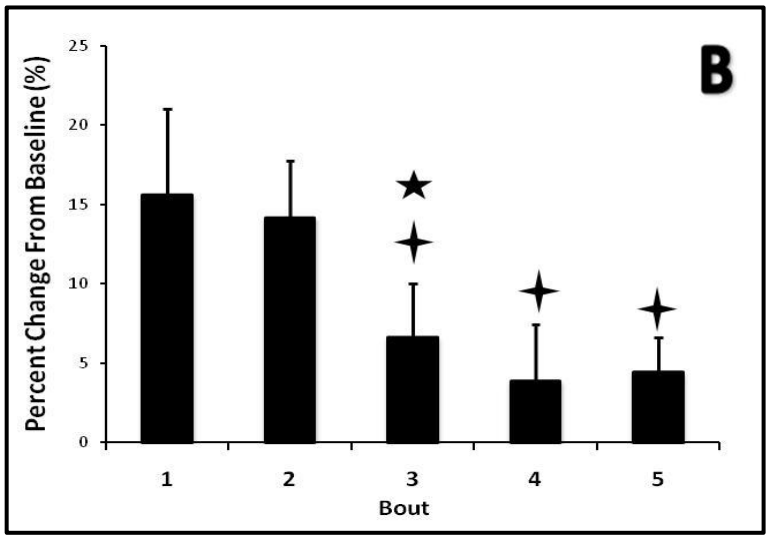

Figure 8: Bout main effect during follow-up; Tukey's post hoc analysis (p<.05) indicated by:

★ = significantly different from previous trial

✦ = significantly different from T1

Background EMG Activity

Left Leg EMG

Contraction Main Effect

Table 1 shows the average EMG (50ms window during contraction) in the left soleus during contraction—collapsed across trial and bout. There was no significant difference between contraction types.

	Eccentric	Concentric	P-value	Difference	CI
EMG (%MVC)	79.6 ± 2.7	80± 2.1	.702	1.56	-11.07— 7.07

Table 1:

Average EMG activity in left (contracting) soleus. Average EMG displayed as a percentage of max EMG during MVC.

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Bout Main Effect:

Figure 10 represents the average EMG activity in left soleus during contraction collapsed across contraction and trial.

There was no significant bout main effect at p=.510, F=.839.

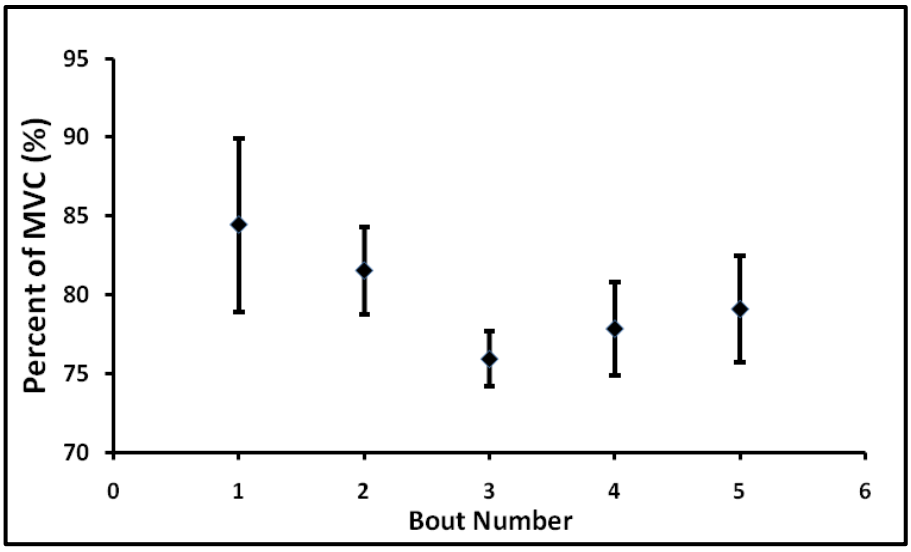

Figure 10: Average EMG activity in left (contracting) soleus. Average EMG displayed as a percentage of max EMG during MVC

.*Right Leg EMG*

Contraction Main Effect:

Table 2 shows the average EMG (50ms window during contraction) in the right (resting) soleus during contraction—collapsed across trial and bout. There was no significant contraction main effect at p=.176, F=2.15.

Bout Main Effect:

Figure 12 represents the average EMG activity in right soleus during contraction collapsed across contraction and trial. There was no significant difference between contraction types.

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	Eccentric	Concentric	P-Value	Difference	CI
EMG (% MVC)	3.2 ± 1.0	5.1 ± 1.2	.176	1.64	-4.4— -.889

Table 2: Average EMG activity in right (resting) soleus. Average EMG displayed as a percentage of max left soleus EMG during MVC.

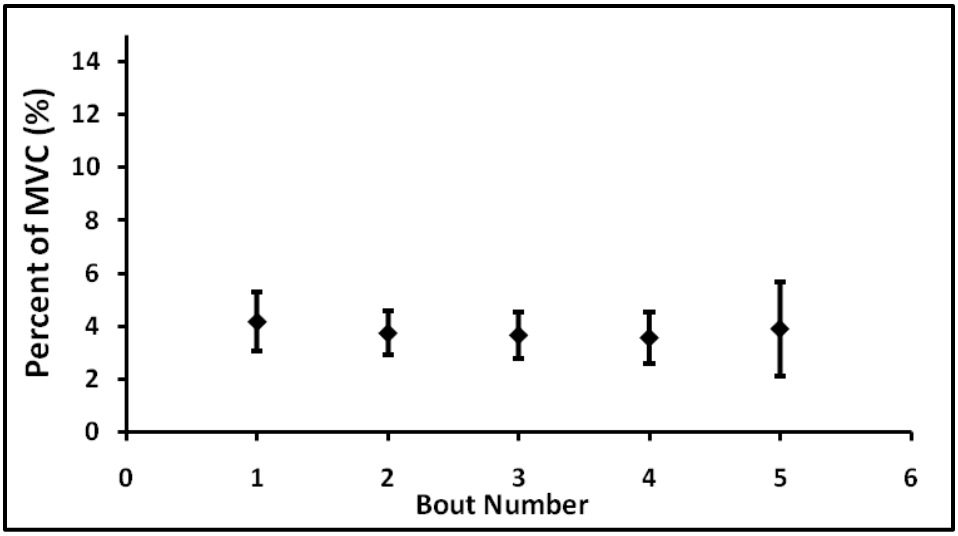

Figure 12: Average EMG activity in right (resting) soleus. Average EMG displayed as a percentage of max left soleus EMG during MVC.

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Chapter 5: Discussion

The purpose of this study was to explore task specific changes of eccentric and concentric contractions on spinal excitability in the resting contralateral leg during an acute bout of repeated ipsilateral contractions. The main finding was that spinal excitability was actually depressed during contralateral homologous muscle contraction. This depression lasted ~45s and recovered to baseline; additionally, this depression, after returning to baseline, reverted to facilitation. This recovery of depression to facilitation seems to be task (contraction) specific, with more facilitation after concentric contractions. Furthermore, this facilitation is long-lasting because it is still present four minutes after the last contraction and more so, again, after concentric contractions.

Spinal Inhibition

Bikmullina et al. (2005) investigated the effects of a single unilateral contraction on spinal excitability in the contralateral plantarflexors, and found similar results. During a single contraction, spinal excitability was depressed by ~80% in the contralateral leg during maximal contraction. A similar result was established during unilateral pedalling movement of the leg (Cheng, Brooke, Misiaszek, & Staines, 1998). In this study, spinal excitability in the stationary, contralateral leg was significantly depressed. Hortobágyi et al. (2003) investigated the effects of unilateral contractions on contralateral spinal excitability, but, in this case, used wrist flexors; researchers found the spinal excitability in the resting wrist flexor was significantly depressed for up to 30 seconds. This might connote that this depression of spinal excitability from unilateral contraction is not specific to plantarflexors, and that dynamic movement, not just isokinetic contractions, initiates this depression.

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The results of this study indicate that, in addition to isolated, individual contractions, repeated isokinetic unilateral contraction also produced significant depression, ~40%, in the contralateral—resting—muscle.

This long-lasting depression has been suggested to be a result of inhibition of 1a afferent motoneuron synapse (Hortobágyi et al., 2003). That is, cortical excitation in addition to various inputs from interneurons might be affecting the excitability of spinal motor neurons controlling the resting limb. The data from this study indicates that these presynaptic inhibitions of the spinal motor neurons are not task specific; that is, this depression is seen regardless of contraction type. This idea is supported by previous data collected in this lab (Motawar., 2010). This data might indicate that strength gain differences, from unilateral contraction, between shortening and lengthening contractions, as seen in past studies (Hortobágyi et al., 1996), might be mediated through other mechanisms.

Spinal Inhibitory Mechanisms

It has been hypothesized that during voluntary contraction, activation of Ia afferents can cause an inhibitory effect on the motor neuron affecting the antagonist muscle—i.e. reciprocal inhibition (Lee & Carroll, 2007). Delwaide et al (1991) hypothesized that there was a cross-mediating system that connected both sides of the body. That is to say, an inhibitory interneuron, which crosses the midline, connects with Ia inhibitory neuron on the other side that synapses with the antagonist muscle group. That is, left (agonist) plantarflexor motor neurons are connected to an inhibitory interneuron which synapses with the motor neuron of the right (antagonist) dorsiflexors. In addition, right dorsiflexors motor neurons are thought to be attached to another Ia

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inhibitory interneuron that suppresses right (agonist) plantarlexor motor neurons. This suggests that activation of contralateral Ia afferents through voluntary movement can modulate ipsilateral spinal processes.

Supraspinal Modulation Mechanisms

There have been several hypotheses that supraspinal mechanism could play a role in the modulation of spinal pathways. In a recent study, researchers found that during strong ipsilateral wrist flexion interhemispheric inhibition, inhibition from right M1 to left M1, was significantly diminished (Howatson et al., 2011). This might indicated an interaction between intracortical and interhemispheric connections that regulates the excitability of the, supposedly inactive, contralateral M1, and, therefore, might influence spinal excitability of the resting limb. Jankowska et al. (2006) argued that there might be networks of neurons interconnecting two sides of the gray matter at the brainstem and spinal levels, as well as intrahemispheric transcallosal connections. Researchers have also used functional MRI's (F-MRI) to elucidate if the contralateral M1 is activated during ipislateral contraction (Francis et al., 2009). Francis and colleagues found that a significant number of voxels were active in the contralateral M1 during ipsilateral ankle dorsiflexion.

In addition, some researchers have studied the descending corticospinal fibers originating in the contralateral motor cortex as a possible mechanism for the modulation of spinal pathways. In an early study by Armand and Kuypers, cats were used to investigate the organization of these descending corticospinal fibers (Armand & Kuypers., 1980). Previously it was thought that 100% of the corticospinal fibers originating in the motor cortex crossed the midline and controlled the contralateral side

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of the body. Armand and Kuypers found that not all of the descending corticospinal fibers crossed the midline—in cats. In a more recent study, Nathan and colleagues studied the descending corticospinal fibers in humans (Smith & Deacon., 1990). Nathan and colleagues found that about 90% of the descending corticospinal tracts travel ipsilaterally to the medulla, where they then cross the midline at the pyramidal decussation; those that cross the midline then travel down the lateral corticospinal tract. Although, about 10% of the descending corticospinal fibers do not cross the midline and add to the lateral corticospinal tract on the same side. These uncrossed corticospinal tracts might be a possible location for the modulation of the excitability of spinal motor neuron controlling resting muscle. In combination with the results from the present study, it might be possible that supraspinal mechanisms might modulate spinal processes.

Post-Contraction Facilitation

After cessation of the contraction, the depression, seen during the contraction, recovered to baseline, on average, in about 45 seconds. This recovery time is in accordance with what Hortobágyi and colleagues saw during their experiments (Hortobágyi et al., 2003). This recovery to baseline was extended in the follow-up so that spinal excitability actually became facilitated and increased ~6% relative to baseline. In addition, the recovery of depression to facilitation seems to be task (contraction) specific, with more facilitation after concentric contractions—9% more facilitation. Because of the small differences between contraction types these findings should be taken carefully, and further research is needed to investigate the task specificity. This super compensation, or facilitatory effect, has not been seen in previous

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research, and is difficult to explain.

Gandevia and colleagues found that fatigue from voluntary contractions can cause changes in cortical and spinal facilitation and inhibition based on EMG recordings, and a decline in supraspinal “drive” based on force recordings (Gandevia et al., 2001). It is unlikely that this process would be contributing to the facilitation seen in our study, because fatigue was specifically controlled for to not influence our results. In addition to the facilitation, it seems that concentric contractions produce greater facilitation than eccentric contractions. We have made several hypotheses to why this super compensatory facilitation might be task specific based on contraction.

There have been numerous studies that have documented the numerous differences between concentric and eccentric contractions. In a previous study, researchers found that concentric contractions are associated with estimates of whole body energy cost (oxgen uptake) that are higher than for eccentric activity at a similar intensity (Asmussen., 1957). Additionally, concentric contractions elicit greater changes in Heart reate, Mean arterical blood pressure, and rate-pressure product during exercise than eccentric contractions (Overend, Versteegn, Thompson, Birmingham, Vandervoort, A 2000). This indicates that concentric contractions induce greater increases in cardiovascular stress than eccentric contractions. There also seems to be differences in hormonal responses from concentric and eccentric contractions. Durand and colleagues established that concentric exercise increases growth hormone concentrations to a much greater extent than eccentric exercise at the same intensity (Durand et al., 2003). These differences in oxygen uptake, cardiovascular stress, and hormonal responses between concentric and eccentric exercise could potentially influence the nervous

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system, and might be a reason for task specificity of this super compensation effect (i.e. facilitation) seen post-exercise.

Conclusions

The purpose of this study was to explore task specific changes of eccentric and concentric contractions on spinal excitability in the resting contralateral leg during an acute bout of repeated ipsilateral contractions. We hypothesized that there would be task specific changes of eccentric and concentric contractions on contralateral spinal excitability during contraction, inter-bout rest, and follow-up. The results indicated that although there were no task specific changes in spinal excitability during the contraction and follow-up; concentric contractions had greater facilitation during the inter-bout rest. The post-exercise facilitation was long lasting, and is still present two minutes after the cessation of exercise. This facilitation needs to be further studied, as well as the task specificity of this facilitation. In general, this supports our hypothesis. As of now the mechanisms behind the depression during contraction, and the facilitation post-exercise is unclear. It seems likely that both cortical and spinal processes are responsible for the modulations seen during unilateral contraction, and the effects of cross education seen in chronic training studies are probably not directly mediated at the spinal level. The data thus suggest that spinal mechanism during exercise is probably not a primary mechanism to mediate cross education but it remains to be determined if the facilitatory after-effects are associated with cross education.

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Appendix A: Consent Form

Consent Form
Interhemispheric Plasticity in Humans – Version 2

Biomechanics Laboratory
Investigator: Tibor Hortobágyi, Ph.D.
Address: 332A Sports Medicine Building, East Carolina University, Greenville, NC 27858
Telephone: (252) 737 - 4564

I am asked to voluntarily participate in this research project conducted by Tibor Hortobágyi. The **purpose** of this study is to determine how muscle strength is increased in the non-exercised limb after strength training of the muscles in the other limb. The study involves different strength training programs of the right calf muscles, including voluntary, imagined, and electrically evoked muscle contractions. It also involves magnetic stimulation of the brain, electrical stimulation of a leg muscle that is associated with some discomfort, or short-term leg immobilization. My involvement will last for about 8 weeks. I will have to be right-leg dominant determined by ball kicking. There will be about 130 subjects in the study over five years. I understand that my written consent is required before I can participate in this project.

Training procedures. I understand that only the training procedures that are circled will apply to me. A specific training procedure will be randomly assigned to me by chance. For each experiment, the name of each treatment group will be written on a separate piece of paper. The principal investigator will then draw one of these marked papers out of a box. The name of the treatment group written on the paper will be my group assignment.

1. Orientation. There will be two, about 60-minute orientation sessions during which I will be familiarized with the laboratory environment and equipment.

2. If I participate in Experiment 1, I may be randomly assigned to one of the following groups. A. Exercising the right calf muscles with 100% effort. B. Exercising the right calf muscles with 50% effort. C. Exercising the right calf muscles with 10% effort. D. Exercising the right calf muscles with maximal effort imagined muscle actions, or E. Exercising the right calf muscles by having the foot moved by a machine while I relax my leg.

3. If I participate in Experiment 2, I may be randomly assigned to one of the following groups. A. Exercising the right calf muscles with medium intensity voluntary effort. B. Exercising the right calf muscles with muscle contractions produced by therapeutical electrical stimulation. C. Exercising the right calf muscles with medium intensity voluntary effort while my right arm muscles are electrically stimulated, or D. I will not exercise but will report to the laboratory 18 times ("Control group").

4. If I participate in Experiment 3, my left ankle will be put in cast and immobilized for 4 weeks. I will walk around on crutches. I may be randomly assigned to one of the following groups. A. I will not exercise but will report to the laboratory 18 times ("Control group"). B. Exercising the right calf muscles with medium-intensity. C. Exercising the right calf muscles with muscle contractions produced by therapeutical electrical stimulation or D. Exercising the right calf muscles with 100% effort.

Testing procedures. I understand that only the testing procedures that are circled will apply to me. These procedures will be done over two days, totaling 6 hours. I will lie on my stomach on an examination bench.

Voluntary strength. The amount of force I can produce with voluntary effort during ankle extension and ankle flexion will be measured on a computerized strength-measuring device. My foot will be strapped to the measuring arm of the device. As a warm-up, I will do several low-intensity practice trials and my scores will be recorded for six maximal efforts, each lasting 1-2 seconds.

Electrical muscle stimulation. With my foot strapped to the strength-measuring device, water-soaked 2 x 2-inch sponges will be placed on my calf. On the top of these sponges electrodes will be placed and the sponges and the electrodes will be fastened to my leg with Velcro. I will have the opportunity over several practice trials to get familiar with the feeling of my muscle being stimulated. I will feel some discomfort but mostly a "buzzing" sensation. The force my muscles can produce at the highest level of stimulation intensity will be determined. Approximately 6 high-intensity trials will be performed on my muscle. The duration of stimulation will be about 1 to 2 seconds. I will hold the stimulator's safety switch in my hand and I can turn off the stimulation at any time.

Nerve stimulation. The nerve on the back of my knee will be stimulated with a very brief (approximately one hundredth of a second long) electrical pulse. I will receive about 30 pulses at 10 to 15 seconds intervals. These pulses are so short that at low intensities I will not feel anything. At high intensities I will feel my muscle contract.

Magnetic brain stimulation. This technique activates areas of the brain with a magnetic pulse that travels through the skull. A wire coil will be placed near the top of my head. A very brief (one hundred thousand of a second) electrical current is passed through the wire coil and this creates the magnetic pulse that activates or stimulates the brain. When this is done I will hear a click and feel a snapping sensation on the skin under the coil. If the coil is placed over an area of the brain that controls muscles, I will feel a twitch in the muscle, which is often large enough to move the limb. In other cases there I may be feeling a movement or a tingling sensation in my foot. My eyes may blink and my face twitch mildly but I should never feel pain associated with the pulse. The electrical activity in the leg muscle will be recorded with electrodes taped to my skin. My scalp may be marked but these markings will be removed at the end of the session. I will receive about 1000 magnetic pulses with at least 10 seconds between two pulses. I will be told how many stimuli to expect. These experiments last 2 hours. I will be allowed to get up and move around or leave the room.

Vibration. This technique gently vibrates my feet. I will be asked to stand on the platform with my knees slightly bent for 30 seconds to 1 minute. The vibration may create tickling sensation.

Exclusion criteria: I may not participate in this project if: I have orthopedic impairments of the lower extremities; I have neurological impairments, including current or past peripheral or central nervous system dysfunction; I am on medications that affect neuronal conduction; I have a pacemaker; I have an implanted medication pump or a metal plate in the skull; I have metal objects in the eye or skull (for example after brain surgery or shrapnel wounds); I am a diabetic, and I consume more than moderate amounts of alcohol or caffeine (more than 4 cups prior to testing). If I am a woman I must use effective means of birth control because the effects of magnetic brain stimulation on embryonic development are unknown and maximal effort contractions are also contraindicated in pregnancy.

Risks: Maximal effort is associated with an increase in heart rate and blood pressure and such changes involve the risk of a heart attack or restriction of blood supply to the heart. Dizziness, overexertion, muscle strain or joint sprain may also occur.

Risks associated with electrical stimulation, such as electrocution or burns, will be avoided by using a so-called isolation unit. This unit isolates me from the main electric line in the wall. Because the duration of the pulse is extremely brief during nerve stimulation, the risks for nerve damage are minimal.

The risks associated with the type of vibration used in this study are minimal. In some individuals it creates tickling sensation and a sensation that the muscles are still being vibrated after the vibration was stopped. It is important to bend the ankle, knee, and hip joints for the vibration so that vibration targets the

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muscles. The vibration used in the study is actually used to improve muscle strength in healthy individuals and clinical populations.

Exposure to magnetic brain stimulation is contraindicated in people who have a pacemaker, an implanted medication pump, a metal plate in the skull, or metal objects inside the eye or skull (for example, shrapnel wounds). Magnetic stimulation may cause slight discomfort lasting less than a second on the scalp near the coil. It may cause some twitching of the face or jaw, which may be unpleasant but not painful. Magnetic brain stimulation has been used on thousands of individuals in the United States and around the world without any serious problems. The risk of a stroke or other permanent injury is minimal. There are no known long-term risks of magnetic brain stimulation. The principal investigator received training at the National Institutes of Health as well as at the Prince of Wales Medical Research Institute, Sydney, Australia to administer magnetic brain stimulation.

Limb immobilization is inconvenient but pain-free. In extremely rare cases immobilization may cause deep vein thrombosis (DVT). The chance of this occurring in a healthy individual is very small. Please notify the principal investigator at once if symptoms of DVT appear, including swelling of the leg, swelling of the toes, pain inside the leg, or any unusual symptoms while wearing the cast. Individuals with a history of varicose veins (i.e., swollen veins), severe calf muscle injury, leg bone fracture, and smoking are at a greater risk to develop DVT.

I will be fitted with a stump sock and felt pads over bony spots to avoid the bruising of the skin in the cast. I will be asked to report to the laboratory the day after the cast was applied to determine that is comfortable (not too tight or loose). Based on this inspection, the cast will be modified if necessary. I am asked to contact the research staff immediately if the cast causes any discomfort. Immobilization reduces muscle strength but the training protocols will reduce this strength loss.

Benefits: The principal investigator or his associate will explain me the results that came from the specific experiment I participated in after the data will become available (1-2 months after my participation ends). These experiments help us better understand how the two sides of the brain work together and control voluntary movement.

Compensation: If I am in Experiment 1, I will be entitled to $300. If I am in Experiment 2, I will be entitled to $300 (voluntary group), $500 (electrical stimulation group), or $150 (control group). If I am in Experiment 3, I will be entitled to $1,000. The payment will be available to me upon the completion of the study or will be prorated in proportion to the extent of participation according to the following schedule. I will receive about 25%, 50%, 75%, or 100% of the payment for about 25%, 50%, 75%, or 100% completion of the specific experiment.

Withdrawal, Injury, Confidentiality: The nature and purpose of the procedures, the known risks involved, and the possibility of complications has been explained to me, and I understand them. No guarantee of assurance has been given by anyone as to the results that may be obtained. I understand that not all risks and side effects of these treatments are foreseeable.

I understand that participation in these experiments is voluntary and refusal to participate will involve no penalty or loss of benefits to which I am otherwise entitled, and I may discontinue participation at any time without penalty. The principal investigator may terminate my participation in case of I manifest an undesirable response to the training or testing protocol. The principal investigator may also end my participation in the study if I am not abiding by the inclusion criteria in the study. The policy of East Carolina University does not provide for compensation or medical treatment for subjects because of physical or other injury resulting from this research activity. However, every effort will be made to make the facilities of the School of Medicine available for treatment in the event of such physical injury.

I understand that my personal data will be held in strict confidence by the investigators. I understand that if any publications result from this study my name or any identifiable codes will not be used.

Contact person. If I have any questions about the research or possible research-related injury, I may contact Dr. Hortobágyi at home ([252] 355 - 7715) or work ([252] 737- 4564). Also, if questions arise about my rights as a research subject, I may contact the Chair of the University and Medical Center Institutional Review Board ([252] 744 - 2914). I have read the above material and it has been explained to me by Dr. Hortobágyi. I have been encouraged to ask questions about the study and all inquiries have been answered to my satisfaction.

_____
Subject's Name (Print)

_____ _____
Subject's Signature Date

_____
Name of Witness (Print)

_____ _____
Signature of Witness Date

Tibor Hortobágyi
Name of PI

_____ _____
Signature of PI Date

Joseph Armen, DO
Name of Physician

_____ _____
Signature of Physician Date

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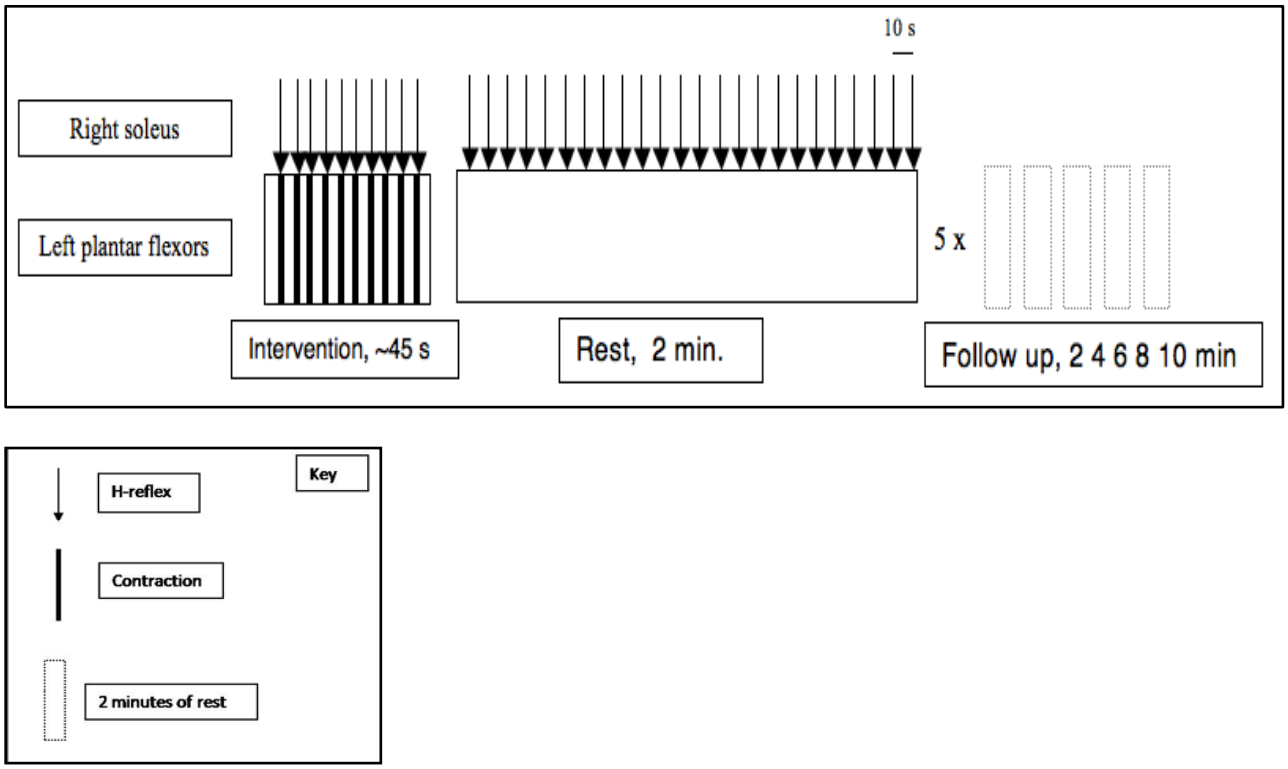
TO 04/27/11

Appendix B

Figure 13: Experimental Design



Appendix C: Supplemental Tables

Table 3: Statistical data for spinal excitability during contraction

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
contraction	Sphericity Assumed	995.792	1	995.792	.036	.855	.004	.036	.053
	Greenhouse-Geisser	995.792	1.000	995.792	.036	.855	.004	.036	.053
	Huynh-Feldt	995.792	1.000	995.792	.036	.855	.004	.036	.053
	Lower-bound	995.792	1.000	995.792	.036	.855	.004	.036	.053
Error(contraction)	Sphericity Assumed	251840.444	9	27982.272					
	Greenhouse-Geisser	251840.444	9.000	27982.272					
	Huynh-Feldt	251840.444	9.000	27982.272					
	Lower-bound	251840.444	9.000	27982.272					
block	Sphericity Assumed	16487.747	4	4121.937	1.128	.359	.111	4.513	.318
	Greenhouse-Geisser	16487.747	2.810	5867.015	1.128	.354	.111	3.171	.260
	Huynh-Feldt	16487.747	4.000	4121.937	1.128	.359	.111	4.513	.318
	Lower-bound	16487.747	1.000	16487.747	1.128	.316	.111	1.128	.159
Error(block)	Sphericity Assumed	131522.668	36	3653.407					
	Greenhouse-Geisser	131522.668	25.292	5200.127					
	Huynh-Feldt	131522.668	36.000	3653.407					
	Lower-bound	131522.668	9.000	14613.630					
trial	Sphericity Assumed	80081.000	9	8897.889	16.749	.000	.650	150.737	1.000
	Greenhouse-Geisser	80081.000	2.468	32452.879	16.749	.000	.650	41.329	1.000
	Huynh-Feldt	80081.000	3.471	23069.257	16.749	.000	.650	58.140	1.000
	Lower-bound	80081.000	1.000	80081.000	16.749	.003	.650	16.749	.952
Error(trial)	Sphericity Assumed	43032.343	81	531.263					
	Greenhouse-Geisser	43032.343	22.208	1937.654					
	Huynh-Feldt	43032.343	31.242	1377.389					
	Lower-bound	43032.343	9.000	4781.371					

Table 4: Statistical Data for Spinal Excitability during Inter-Bout Rest

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
contraction	Sphericity Assumed	33392.988	1	33392.988	6.721	.029	.428	6.721	.637
	Greenhouse-Geisser	33392.988	1.000	33392.988	6.721	.029	.428	6.721	.637
	Huynh-Feldt	33392.988	1.000	33392.988	6.721	.029	.428	6.721	.637
	Lower-bound	33392.988	1.000	33392.988	6.721	.029	.428	6.721	.637
Error(contraction)	Sphericity Assumed	44716.942	9	4968.549					
	Greenhouse-Geisser	44716.942	9.000	4968.549					
	Huynh-Feldt	44716.942	9.000	4968.549					
	Lower-bound	44716.942	9.000	4968.549					
block	Sphericity Assumed	7034.817	4	1758.704	.935	.455	.094	3.738	.266
	Greenhouse-Geisser	7034.817	2.947	2387.475	.935	.436	.094	2.754	.225
	Huynh-Feldt	7034.817	4.000	1758.704	.935	.455	.094	3.738	.266
	Lower-bound	7034.817	1.000	7034.817	.935	.359	.094	.935	.140
Error(block)	Sphericity Assumed	67743.686	36	1881.769					
	Greenhouse-Geisser	67743.686	26.519	2554.537					
	Huynh-Feldt	67743.686	36.000	1881.769					
	Lower-bound	67743.686	9.000	7527.076					
trial	Sphericity Assumed	130394.218	7	18627.745	24.171	.000	.729	169.196	1.000
	Greenhouse-Geisser	130394.218	1.655	78796.102	24.171	.000	.729	39.999	1.000
	Huynh-Feldt	130394.218	1.981	65833.607	24.171	.000	.729	47.874	1.000
	Lower-bound	130394.218	1.000	130394.218	24.171	.001	.729	24.171	.991
Error(trial)	Sphericity Assumed	48552.065	63	770.668					
	Greenhouse-Geisser	48552.065	14.893	3259.955					
	Huynh-Feldt	48552.065	17.826	2723.670					
	Lower-bound	48552.065	9.000	5394.674					

Table 5: Statistical Data for Spinal Excitability during Follow-Up

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
contraction	Sphericity Assumed	4945.492	1	4945.492	.966	.351	.097	.966	.143
	Greenhouse-Geisser	4945.492	1.000	4945.492	.966	.351	.097	.966	.143
	Huynh-Feldt	4945.492	1.000	4945.492	.966	.351	.097	.966	.143
	Lower-bound	4945.492	1.000	4945.492	.966	.351	.097	.966	.143
Error(contraction)	Sphericity Assumed	46056.588	9	5117.399					
	Greenhouse-Geisser	46056.588	9.000	5117.399					
	Huynh-Feldt	46056.588	9.000	5117.399					
	Lower-bound	46056.588	9.000	5117.399					
block	Sphericity Assumed	19771.191	4	4942.798	3.693	.013	.291	14.773	.836
	Greenhouse-Geisser	19771.191	2.442	8095.087	3.693	.034	.291	9.020	.670
	Huynh-Feldt	19771.191	3.419	5781.928	3.693	.018	.291	12.629	.785
	Lower-bound	19771.191	1.000	19771.191	3.693	.047	.291	3.693	.404
Error(block)	Sphericity Assumed	48180.952	36	1338.360					
	Greenhouse-Geisser	48180.952	21.981	2191.904					
	Huynh-Feldt	48180.952	30.775	1565.571					
	Lower-bound	48180.952	9.000	5353.439					
trial	Sphericity Assumed	3031.285	7	433.041	1.705	.124	.159	11.935	.650
	Greenhouse-Geisser	3031.285	2.560	1184.243	1.705	.199	.159	4.364	.359
	Huynh-Feldt	3031.285	3.664	827.333	1.705	.177	.159	6.247	.445
	Lower-bound	3031.285	1.000	3031.285	1.705	.224	.159	1.705	.216
Error(trial)	Sphericity Assumed	16000.832	63	253.981					
	Greenhouse-Geisser	16000.832	23.037	694.567					
	Huynh-Feldt	16000.832	32.975	485.237					
	Lower-bound	16000.832	9.000	1777.870					

Table 6: Statistical Data for EMG in Left Leg

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
Contraction	Sphericity Assumed	67.562	1	67.562	.156	.702	.017	.156	.064
	Greenhouse-Geisser	67.562	1.000	67.562	.156	.702	.017	.156	.064
	Huynh-Feldt	67.562	1.000	67.562	.156	.702	.017	.156	.064
	Lower-bound	67.562	1.000	67.562	.156	.702	.017	.156	.064
Error(Contraction)	Sphericity Assumed	3909.230	9	434.359					
	Greenhouse-Geisser	3909.230	9.000	434.359					
	Huynh-Feldt	3909.230	9.000	434.359					
	Lower-bound	3909.230	9.000	434.359					
Bout	Sphericity Assumed	874.477	4	218.619	.839	.510	.085	3.355	.241
	Greenhouse-Geisser	874.477	1.931	452.901	.839	.445	.085	1.620	.169
	Huynh-Feldt	874.477	2.448	357.159	.839	.467	.085	2.054	.188
	Lower-bound	874.477	1.000	874.477	.839	.384	.085	.839	.130
Error(Bout)	Sphericity Assumed	9383.014	36	260.639					
	Greenhouse-Geisser	9383.014	17.378	539.952					
	Huynh-Feldt	9383.014	22.036	425.807					
	Lower-bound	9383.014	9.000	1042.557					
Contraction * Bout	Sphericity Assumed	366.465	4	91.616	.676	.613	.070	2.705	.199
	Greenhouse-Geisser	366.465	1.716	213.608	.676	.501	.070	1.160	.138
	Huynh-Feldt	366.465	2.081	176.135	.676	.526	.070	1.407	.148
	Lower-bound	366.465	1.000	366.465	.676	.432	.070	.676	.114
Error(Contraction*Bout)	Sphericity Assumed	4876.927	36	135.470					
	Greenhouse-Geisser	4876.927	15.440	315.857					
	Huynh-Feldt	4876.927	18.725	260.445					
	Lower-bound	4876.927	9.000	541.881					

Table 7: Statistical Data for EMG in Right Leg

Measure:MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
Contraction	Sphericity Assumed	68.867	1	68.867	2.154	.176	.193	2.154	.260
	Greenhouse-Geisser	68.867	1.000	68.867	2.154	.176	.193	2.154	.260
	Huynh-Feldt	68.867	1.000	68.867	2.154	.176	.193	2.154	.260
	Lower-bound	68.867	1.000	68.867	2.154	.176	.193	2.154	.260
Error(Contraction)	Sphericity Assumed	287.685	9	31.965					
	Greenhouse-Geisser	287.685	9.000	31.965					
	Huynh-Feldt	287.685	9.000	31.965					
	Lower-bound	287.685	9.000	31.965					
Bout	Sphericity Assumed	4.685	4	1.171	.139	.967	.015	.554	.076
	Greenhouse-Geisser	4.685	1.962	2.388	.139	.868	.015	.272	.068
	Huynh-Feldt	4.685	2.503	1.872	.139	.910	.015	.347	.070
	Lower-bound	4.685	1.000	4.685	.139	.718	.015	.139	.063
Error(Bout)	Sphericity Assumed	304.268	36	8.452					
	Greenhouse-Geisser	304.268	17.655	17.234					
	Huynh-Feldt	304.268	22.527	13.507					
	Lower-bound	304.268	9.000	33.808					
Contraction * Bout	Sphericity Assumed	32.698	4	8.174	.975	.433	.098	3.899	.277
	Greenhouse-Geisser	32.698	2.138	15.292	.975	.400	.098	2.084	.199
	Huynh-Feldt	32.698	2.825	11.576	.975	.416	.098	2.753	.229
	Lower-bound	32.698	1.000	32.698	.975	.349	.098	.975	.143
Error(Contraction*Bout)	Sphericity Assumed	301.915	36	8.387					
	Greenhouse-Geisser	301.915	19.244	15.689					
	Huynh-Feldt	301.915	25.422	11.876					
	Lower-bound	301.915	9.000	33.546					

a. Computed using alpha = .05

One-Sample Test

	Test Value = 0					
	t	df	Sig. (2-tailed)	Mean Difference	95% Confidence Interval of the Difference	
					Lower	Upper
Con	4.297	9	.002	38.000	17.99	58.01

Table 8: Statistical Data for Percent Depression during Concentric Contraction

One-Sample Test

	Test Value = 0					
	t	df	Sig. (2-tailed)	Mean Difference	95% Confidence Interval of the Difference	
					Lower	Upper
ECC	5.872	9	.000	40.31	24.78	55.8

Table 9: Statistical Data for Percent Depression during Eccentric Contraction

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	Con - Ecc	12.927	15.76	4.98	1.64	24.19	2.592	9	.029

Table 10: Statistical Data for Task Specificity during Inter-Bout rest

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	Con – Ecc	4.97	15.99	5.05	-6.470	16.41	.983	9	.351

Table 11: Statistical Data for Task Specificity during Follow-Up

One-Sample Test

	Test Value = 0					
					95% Confidence Interval of the Difference	
	t	df	Sig. (2-tailed)	Mean Difference	Lower	Upper
Con	1.176	7	.048	4.435	-4.4810	13.351

Table 12: Statistical Data for Percent Facilitation during Inter-Bout Rest (Concentric)

One-Sample Test

	Test Value = 0					
					95% Confidence Interval of the Difference	
	t	df	Sig. (2-tailed)	Mean Difference	Lower	Upper
Ecc	-2.833	7	.425	-6.8944	-12.648	1.14042

Table 13: Statistical Data for Percent Facilitation during Inter-Bout rest (Eccentric)

One-Sample Test

	Test Value = 0					
	t	df	Sig. (2-tailed)	Mean Difference	95% Confidence Interval of the Difference	
					Lower	Upper
Con	3.026	9	.014	11.39	2.876	19.913

Table 14: Statistical Data for Percent Facilitation during Follow-Up (concentric)

One-Sample Test

	Test Value = 0					
	t	df	Sig. (2-tailed)	Mean Difference	95% Confidence Interval of the Difference	
					Lower	Upper
Ecc	1.716	9	.040	6.42	-2.041	14.887

Table 15: Statistical Data for Percent Facilitation during Follow-Up (eccentric)

Paired Samples Test

	Paired Differences					t	df	Sig. (2-tailed)
	Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1 Con - ECC	-4.803	32.199	11.3841	-31.72	22.115	-.422	7	.686

Table 10: Statistical Data for Task Specificity during Contraction

Paired Samples Test

	Paired Differences					t	df	Sig. (2-tailed)
	Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1 Con - Ecc	-1.6597	3.575	1.13079	-4.2176	.89821	-1.468	9	.176

Table 11: Statistical data for Task Specificity between EMG in Right Leg.

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	Con - Ecc	-1.6439	13.1812	4.1683	-11.0732	7.7853	-.394	9	.702

Table 11: Statistical data for Task Specificity between EMG in Left Leg.