Neuromuscular Mechanisms Underlying Changes in Force Production During an Attentional Focus Task:

By

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Abstract

The objective of this thesis was to examine changes in maximal voluntary force output of the elbow flexors with attentional focus feedback cues and possible underlying physiological mechanisms for these changes. Eleven recreationally active males participated in two randomized experimental sessions (Day 1: n=11, Day 2: n=10); 1) Stimulation session where corticospinal excitability was measured and 2) No stimulation session where only electromyography and elbow flexor force was measured. In both sessions, four randomized blocks of three maximal voluntary contractions (MVC) were performed. Each block consisted of either externally or internally attentional focus cues given before each MVC. During the stimulation session transcranial magnetic, transmastoid and Erb's point stimulations were used to induce motor evoked potentials (MEPs), cervicomedullary MEP (CMEPs) and maximal muscle action potential (M_{max}). All MEPs and CMEPs were normalized to M_{max}. Results showed participants could produce greater MVC force without stimulation and given an external focus cue before the MVC compared to an internal cue. Muscle co-activation data (expressed as % triceps/biceps rmsEMG) during the no stimulation session was greater with internally cued compared to externally cued contractions. There was no difference in corticospinal excitability shown between external and internal focus cues in the stimulation session. In conclusion, maximal voluntary force production of the elbow flexors was greater when an external focus feedback cue was provided. This appeared to be due to less coactivation of the triceps and biceps brachii. Secondly, stimulating the corticospinal pathway seemed to have some confounding effect on attentional focus. The distressing stimulations distracted participants from attentional focus cued feedback or stimulating the corticospinal pathway may have disrupted areas of the cortex responsible for attention and focus.

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List of Symbols, Nomenclature or Abbreviations

MVC	Maximum voluntary contraction
TMES	Transmastoid electrical stimulation
CMEP	Cervicomedullary-evoked potential
TMS	Transcranial magnetic stimulation
MEP	Motor-evoked potential
CSE	Corticospinal excitability
TES	Transcranial electrical stimulation
Mmax	Maximal muscle compound potential
EMG	Electromyography
EF	External Focus
IF	Internal Focus

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Chapter 1: Review of Literature

1.1: Introduction

In sport and motor learning, feedback is often provided to athletes. Feedback is provided to aid acquisition and development of motor skills. It is likely that providing feedback augments the athlete's perception of their performance. It can be suggested that feedback will have different effects on athletic motor performance and learning depending on how and what forms of feedback are provided, whether it be visual, audio based, or tactile. If it does, what are potential underlying mechanisms of these effects? The following literature review will delve into this question examining different forms of augmented feedback, how they influence motor performance and learning, and different mechanisms that may account for these influences.

1.2: Augmented Feedback

The term augmented feedback in the field of motor learning refers to information provided by an external source, such as an instructor, a coach, or a video (1, 2). There is a large amount of evidence demonstrating that different types of feedback have a considerable impact on motor task performance. These effects have been seen to impact performance across a variety of activities that require skills such as strength and power (5, 9), movement efficiency (6, 15), and balance (32, 17). In addition, the effect of feedback is seen in many populations from children (16), to young and older adults (10), athletes, and clinical populations (17, 18). Thus, it is well known that feedback influences motor performance. There have been three types of feedback that has been examined (1,3,4). These include the type of attentional focus induced (external versus internal

focus), the extent to which they support the performer's autonomy (controlling versus supportive), and the promoted performance expectancies (Positive vs. Negative) (1). Each type of feedback has been studied extensively and shown to affect motor task performance.

1.2.1 Attentional Focus

Attentional focus has been defined as instructions that direct one's attention (1). Instructions that direct one's attention towards their body is known as internal focus. Internal focus has been consistently found to hinder motor task performance (1, 26). Meanwhile, external feedback is directing one's attention towards the movement outcome, or to an external object relative to the task. Unlike internal, external focus tends to enhance performance (1, 26). To illustrate, asking a participant to focus on contracting their biceps and/or bending their elbow during a maximum voluntary contraction (MVC) is an example of internal feedback, while asking that participant to focus on pulling the strap/bar (an external object) is external focus. Out of the three types of feedback, attentional focus has been the most researched form of feedback examined with an abundance of studies conducted on it to this date (1, 3).

An external focus of attention has been shown to enhance force, speed, power and balance within resistance exercises or activities while internal focus has shown to decrease these three (7, 19, 20). For instance, Halperin et al. (26) found that when given an external focus cue during an isometric mid-thigh pull, trained athletes applied 9% more force compared to those that received an internal cue, and 5% more force than control. Greater force performances were also seen in single-joint and dynamic movements with external versus internal feedback. Marchant et al. (19) found that during concentric elbow flexion completed at a set speed, subjects produced a 7% greater net joint torque with their elbow flexors when an external focus cue was used. Performance in activities that require power and speed has also been shown to improve with external feedback

and diminish with internal feedback. When compared with internal feedback and control, external focus lead to improved performance in punching force and velocity and sprint speeds (1, 21, 27). Specifically, Halperin et al. (27) found that athletes punching with an external focus cue were 4% faster and 5% more forceful than those given an internal focus cue, and were 2% faster and 3% more forceful than control. External attentional focus cues have also been shown to reduce fatigue. Lohse and Sherwood (33) found that athlete's given an external focus cue had increased time to failure and reduced perceived exertion during a fatiguing task. A person's balance has also been shown to benefit from external focus feedback. Rotem et al. (17) showed that participants utilizing an external focus of attention improved significantly in three stability indices compared to those using an internal focus of attention. External focus feedback overall tends to improve performance through increased force, speed, and power and increased whereas internal focus cues appear to hinder each.

While external instructions have been shown to be advantageous for resistance exercises and balance activities, many of the studies conducted have been on untrained and recreational subjects. In general, the effect of external instructions is inconclusive with trained individuals. For instance, studies with trained swimmers (8) and sprinters (22) showed that speeds improved in control conditions but not in external focus conditions. As well, a study with trained tennis players suggested agility performance to be unaffected by attentional focus feedback. (23). On the contrary, Halperin et al. (27 and 28) reported the punching performances of intermediate and expert competitive boxers and kickboxers to improve with external feedback compared to the other two conditions. More research pertaining to trained individuals is warranted to make a conclusion to the effects of attentional feedback on performance.

Overall, the literature generally indicates, that regardless of the activity, external focus leads to better performance compared to internal focus which tends to decrease performance in the areas of strength, speed, power or balance. Whether or not training status influences the effects of attentional focus feedback is still not clear due to the inconsistent results in studies with trained athletes.

The attentional focus findings in which external instructions lead to a better performance output than internal and control conditions can be explained by the *constrained action hypothesis* proposed by Wulf et al. (1, 24). The hypothesis states that internal focus leads participants to be conscious of their movement which disrupts their natural automatized movements (1, 24). The *constrained action hypothesis* has been supported by studies that showed increased EMG activity when individuals were given an internal focus cue compared to control and/or an external cue (6, 19, 25). The increased EMG activity represented more neuromuscular activity which may suggest poorer motor control. The *constrained action hypothesis* was also supported through studies that investigated reaction times. When performing a motor activity, participants who receive an external cue had faster reaction times than those who received an internal cue suggesting greater automatic control due to less conscious interference. The use of EMG appears to be the only technique used to explain the physiological mechanisms behind the effect of attentional focus on motor task performance. Therefore, while there is an abundance of research supporting the attentional focus effect on motor performance, there is a grey area in the literature on the basic physiological mechanisms behind it.

1.2.2 Positive and Negative Feedback

Another form of augmented feedback is known as feedback valance. Feedback valence is feedback that describes a performance in a positive or negative way (12). An example of positive

feedback would be informing a participant that their MVC force output on trial 2 was 10% better than their previous trial and/or they ranked in the top percentile in force output compared to other participants. In contrast, for negative feedback the participant would be told that their second attempt was worse than their first and/or their performance was one of the worse of all the participants in the study. Compared to attentional focus feedback, not as much research has been done on positive and negative feedback.

Feedback valance has been shown to influence motor performance in tasks involving strength, endurance, and balance (3, 15, 34). For instance, positive feedback compared to negative and/or neutral feedback has been shown to enhance motor performance (12-14). When examining muscular strength and endurance performance, Hutchinson et al. (12) found that participants had an increased time to exhaustion during a submaximal handgrip endurance test when given false-positive feedback but had a decreased time to exhaustion when given false-negative feedback compared to control. Balance performance has also been shown to improve with positive feedback. Lewthwaite and Wulf (34) investigated the effect of social comparative feedback on a balance task. False feedback was given about the average score of other participants performance in a trial. Those who were told they were performing better than the average (Positive feedback) had better performance scores than those who were told they were doing worse than the average (Negative). Studies examining feedback valence appears limited and therefore more are required to thoroughly examine how positive and negative feedback influences a wide range of motor tasks.

The effect of training status on valence feedback effectiveness is not completely known. Participants in the examined studies were generally untrained and there does not appear to be many studies that examined trained athletes. Stoate et al. (15) however examined whether providing experienced runners with positive feedback would improve running efficiency. They found that

compared to control, experienced runners given positive feedback had decreased oxygen consumption and reduced fatigue. Future studies should examine whether trained or untrained participants are influenced more by feedback valence and if so, to what extent.

The effects of feedback on performance are suggested to be due to the interaction between perception and motivation (12, 15). This appears to be partly the case as these studies have shown that participants given positive feedback, compared to negative, had decreases in the rate of perceived exertion, increased enjoyment of the activity, and improved self-efficacy all indicative of changes in perception and motivation. (12, 15). However, to the best of my knowledge, no current research has examined the basic physiological mechanisms behind feedback valance and motor task performance. There were studies that examined fatigue (12, 15) but none of them looked at central and peripheral measures of neuromuscular fatigue. Therefore, as with attentional focus feedback, a grey area in the literature to be examined are the basic physiological mechanisms behind why motor task performance is improved with positive feedback.

1.2.3 Autonomy

Giving people choice, even small choices, in regard to practice and exercise can have positive outcomes on their performance and motivation in that activity when compared to no choice. (3, 4, 36). These positive effects of choice have been demonstrated across various motor tasks requiring balance (35) accuracy (37) as well as motivation to exercise with greater intensity (38). To study these effects participants are assigned to either a choice group or no choice group. Participants in the choice group can decide the training variables of their program. This would include the amount of trials to complete, how long the session will be, and how demanding these trials are. The no choice group participants are then matched to group participants are required to complete the same session the choice group participants completed (39, 40, 42).

Therefore, if a participant from the choice group chooses to complete 15 repetitions of a motor task, then a participant from the no-choice group will complete 15 repetitions as well. This participant is not given the opportunity to choose how many repetitions to complete. Instead they have to do the amount of repetitions the instructor asks. Providing choice has been shown to influence many aspects of motor performance. Accuracy, as measured with ball tossing tasks, golf putting and basketball shooting is enhanced when participants receive choices in the practice conditions (39, 43, 44). For example, participants provided with a choice of when to stop the practice session involving dart throwing with the non-dominant hand improved their accuracy to a greater extent than participants from the no-choice group which threw a comparable amount of repetitions (43).

Similarly, participants who could choose when to receive external feedback about their throwing accuracy in a beanbag toss outperformed those from the no-choice group. As well, receiving the choice to receive feedback outperformed control groups in which participants received no feedback at all. Balance is another training outcome that has been shown to improve when given choices. When given the choice to use the assistance of a support pole during balance tasks during practice, participants improved their balance to a greater extent compared with those from the no-choice group (45, 46). Remarkably, the effects of the self-controlled practice have been shown to persist even when the choices were unrelated to the completed tasks. For instance, Lewthwaite et al. (39) have shown that something as simple as choosing the color of golf balls improved golf putting accuracy compared to those given a golf ball group.

The effects of providing persons with a choice were recently shown to influence exercise behavior (38). In a study by Wulf et al. (38) subjects chose the order of five calisthenics exercises to be performed (choice group), or were told they would complete the

exercises in a specified order (no-choice group). Subsequently, subjects in the two groups were asked to decide on the number of sets and repetitions they would like to complete in each of the five exercises (38). While subjects in both groups had similar levels of fitness, those who were allowed to choose the order of exercises were able to complete 60% more repetitions overall. Thus, having a choice appeared to increase an individuals' motivation to exercise. However, to date, the effects of choices on performance is appeared to be limited to accuracy and balance tasks, and to the best of my knowledge no study has directly investigated the effects of choices of strength and power measures in trained athletes. The effects of choices on performance can be explained by psychological and biological pathways. According to the self-determination theory, the ability to make choices (autonomy) is considered a fundamental psychological need (47, 48). Others proposed that making choices is even a biological necessity (25, 49), as both humans (50) and animals (51) prefer having choices over not having them. It seems as if having control is inherently rewarding. The act of making choices has been associated with activation in a brain region (anterior insula) associated with a sense of agency, a state associated with dopamine release (52). The positive effects of choices on motor learning and performance have been reported for a range of populations, including children (41), young (43) and older (10) adults, as well as participants with motor impairments (54). However, an unexplored question is whether the benefits of providing choices is also seen in well-trained athletes or individuals who are familiar with the motor task. This is because, among other reasons, trained athletes respond to training differently than non-trained athletes (53) due to their familiarity with training. Most of the studies on choices had participants who were unfamiliar with the motor task to allow researchers to study the learning acquisition of these skills. Nonetheless, there is also a need to investigate if the choices lead to greater performance in tasks which the participants have experience with, and even with

tasks that they have reached a level of mastery at. Therefore, there is a need to examine the if providing choices also enhance performance of other more complex athletic tasks and tasks that require greater force and power outputs.

1.3: Motor Cortex Output

Human motor output depends on the motor commands from motor areas in the cerebral cortex. Cortical motor commands descend through the corticobulbar and corticospinal tracts. Corticobulbar fibers control the motor nuclei in the facial muscles, whereas the corticospinal fibers control the spinal motoneurones that innervate the trunk and limb muscles. Corticospinal fibers terminate directly onto spinal motoneurones or indirectly via interneurones of the spinal cord, which then project to spinal motoneurones. These connections contribute to the organization of single and multi-jointed movements, such as reaching or walking (57). Thus, the assessment of the corticospinal tract role in voluntary contraction is essential in understanding movement of the human body.

1.3.1 Assessing Corticospinal Tract Excitability

Changes in Corticospinal Excitability (CSE) can occur at a supraspinal and/or spinal level (55). Non-invasive magnetic and electrical stimulation techniques of the brain and spinal cord are used to evaluate corticospinal, spinal and supraspinal excitability in non-healthy and healthy individuals (56). This section will focus on the various central nervous system levels underlying corticospinal excitability and the stimulation techniques used to measure it.

Corticospinal Excitability

The corticospinal tract output can be altered by multiple variables, such as exercise, injury, disuse and disease. The use of transcranial magnetic stimulation (TMS) has been used over the years to investigate corticospinal excitability due to its ease and safety (58). The magnetic field stimulation passes un-attenuated and painless through the scalp and skull making it applicable for most individuals. When the motor cortex is stimulated by TMS, it produces a motor evoked potential (MEP) in a muscle when the stimulus intensity is above the motor threshold (i.e. supra-threshold) required to induce a MEP. By using surface electromyography (EMG) recording electrodes a MEP can be recorded in a desired muscle following a supra-threshold TMS pulse delivered to the motor cortex. It has been shown that there are multiple components of the MEP (59). By using epidural or single motor unit recordings, short latency direct waves (D-waves) followed by several longer latency indirect waves (I-waves) can be found. The D-wave is best activated by using high intensity TMS or transcranial electrical stimulation (TES) and is thought to be caused by direct depolarization of the initial axon segment of the corticospinal neurone. Approximately 1.5ms following the D-wave, I-waves will occur, showing the delay required for the synaptic firing. The first I-wave is thought to be caused monosynaptically by the depolarization of an axon synapsing directly onto a corticospinal neurone. By using low TMS intensities the I-waves that follow may require local polysynaptic circuits (60). The likely cause for preferential recruitment of I-waves using TMS is the current flowing parallel to the surface of the brain. To stimulate the biceps brachii muscle for example, in the primary motor cortex the biceps brachii area is thought to be in the center of the central sulcus. However, it is probable that the area continues to some degree along the surface of the precentral gyrus (61). The pyramidal neurones that are in the area of stimulation will participate in the threshold responses; this is because they

are nearest to the surface of the scalp. If the stimulation intensity is increased then deeper-laying pyramidal neurones, which are parallel orientated to the brain, in the anterior bank of the central sulcus may be recruited (62).

Motor threshold, MEP amplitude, area, latency and silent period, and recruitment curves are the most common measurements to examine changes in corticospinal excitability using TMS. Motor threshold is defined as the lowest TMS intensity or magnetic stimulator output (MSO) that can evoke a MEP in the muscle of interest at rest or during a contraction. It is usually lower at rest and in distal muscles compared to an active state (i.e. muscle contraction) and in proximal muscles (62, 63). Motor threshold is determined by increasing the intensity of the stimulator by small increments until a MEP is elicited reliably. Motor threshold is defined as the stimulation that elicits a MEP with the peak-to-peak amplitude greater than 50μV in 50% of the stimulation trials (i.e. 5 out of 10 trials). However, this is only applicable in a resting state. In an active state, motor threshold is defined as a MEP that is discernible from the background EMG (64) of the muscle of interest. Changes in resting threshold can result from a multitude of reasons such as: the structure and number of excitatory projections onto the primary motor cortex, the neurone membrane, axonal electrical properties, or upregulation of receptors of this region (65). Therefore, motor threshold at rest represents a global assessment of the excitability of inactive pyramidal neurones (65,66). Meanwhile, in an active state it is thought that the magnitude of voluntary drive to the corticomuscular pathway results in a significant reduction of motor threshold compared to resting conditions (67) because pyramidal neurones are now active or in a state of subliminal fringe.

Another outcome measure of excitability is MEP amplitude. When TMS is utilized over the motor cortex at an intensity higher than motor threshold I-waves are elicited in the corticospinal tract (68). These I-waves are modulated by multiple mechanisms such as: activity-dependent

changes (i.e. voluntary contraction) (69), interneurones contacted by corticospinal tract cells, neurotransmitters (i.e., glutamate, GABA), and modulators of neurotransmission (i.e., acetylcholine, norepinephrine, and dopamine) (70). Evidently, all these factors can also influence the MEP amplitude. However, MEP amplitude can be altered at either the cortical or the spinal level making it difficult to locate where within the corticospinal tract change has occurred. A reduction or increase in MEP amplitude can be an indicator of alterations within the neuromuscular system (64). In addition, another usage of MEP amplitude to assess CSE is through the development of a recruitment curve. A recruitment curve or an input-output curve illustrates the increase in MEP amplitude with increasing TMS intensity. The recruitment curve enables an assessment of neurones that are intrinsically less excitable or further away from the central activation of the TMS (71). The slope of the input-output curve is a measurement of the excitability of the cortical motor areas (72). A steeper curve is found in muscles with a lower motor threshold, which could be related to the strength of the corticospinal projections (73) . Plateau levels are the level at which the sigmoidal curve approaches Y*max* (maximal response that may be elicited). Slope and plateau levels show motor unit recruitment efficiency and overall summation of inhibitory and excitatory drive from the corticospinal tract(74).

The silent period is defined as the period of interruption in voluntary activation after TMS has been delivered. The silence in the EMG can last upwards of 200 to 300 msec, but mainly it depends on the stimulus intensity. The physiological basis behind the silent period is still not fully understood, however it includes inhibition at both the spinal cord and at the motor cortex. The first part of the silent period (50-60ms) is attributed to the spinal cord (activation of Renshaw cells), whereas the later section is attributed to the cortex (y-aminobutyric acid (GABA) type B receptor mediated inhibition) (75, 76). Although useful, the silent period is difficult to interpret because if

alterations are found it cannot be determined whether the change is due to cortical or spinal components or both.

Variations in the size of the MEP amplitude during different conditions are used to infer changes in the central nervous system. It is important to have a method that activates the corticospinal output at a subcortical level to allowing a better interpretation of responses evoked at the cortex (77, 78, 79, 80). This is because a variation in any of the corticospinal excitability measurements may be caused by changes at the cortex, spinal cord or at the muscle.

Spinal Excitability

Motoneurones are the final common pathway to muscle contraction. Understanding how motoneurones respond to synaptic input and their subsequent output is essential to motor control. However, in humans it is difficult to test motoneurones in a controlled manner (81). Like previously stated TMS directly and/or indirectly activates corticospinal neurones leading to the activation of motoneurones, which results in a response in the muscle. However, the response in the muscle depends on the excitability of both cortical neurones and spinal motoneurones. Thus, TMS alone cannot determine the specific central nervous system site where modulation in excitability has occurred. Stimulation techniques that are used to determine changes in spinal/motoneurone excitability include: 1) TMES, which activates corticospinal axons of the spinal cord and directly activates motoneurones resulting in a response in the muscle (82), 2) nerve stimulation that activates Ia afferents (which are primary muscle spindle afferents) to induce an H-reflex in the muscle, and 3) nerve stimulation to induce an F-wave, which is the result of antidromic activation of a motoneurone. Each of these stimulation techniques are used to describe motoneurone excitability but all have limitations when testing motoneurone excitability.

In 1991, Ugawa et al.(83) developed a method to stimulate the descending axons at a subcortical level in order to test the excitability of the spinal excitability (i.e. motoneurones). This method involved passing an electrical stimulus between the mastoid processes, creating a single descending volley. This single volley contrasts with that of TMS because TMS evokes multiple descending volleys that stimulates corticospinal motoneurones multiple times. TMES evokes a muscle response that is termed a cervicomedullary MEP (CMEP), which can be utilized as a measure of motoneurone excitability (81, 84, 85). A fixed latency of the response shows activation of fast descending axons at the level of the pyramidal decussation at the cervicomedullary junction (86). The stimulation is made possible due to the bending of axons at the decussation, however stimulation at this site is uncomfortable. What makes TMES the most direct motoneurone measurement is that the descending tracts are not subject to conventional presynaptic inhibition due to primary afferent depolarization (88). One major issue with TMES is the possibility of activating ventral roots in addition to stimulating the descending axons in the spinal cord (89). The ventral root bends along the spinal canal exit, thus enabling it to be a susceptible site for activation. If the ventral root is stimulated, which may occur with an increase in stimulation intensity or improper positioning of the electrodes, the latency of the recorded response will decrease by ~2ms (83, 90). If a decrease in latency occurs, then some peripheral axons have been activated and the final response will reflect a mix of both pre-synaptic activation of the motoneurone (i.e. cortical spinal tract) and postsynaptic motoneurone activation (i.e. antidromic activation of the motoneurone via the ventral root). If stimulation intensity is too high, then the CMEP response will become partially occluded. One possible solution to this limitation is to place the anode on the same side as the muscle in which the CMEP is being recorded from, due to depolarization of the peripheral nerve occurring closer to the cathode (83).

Another way to stimulate the axons of the spinal cord and subsequently motoneurones is by magnetic stimulation with a double-cone magnetic coil evoking motor responses with the same latencies as TMES (91). However, magnetic stimulation induced-responses at rest tend to be very small compared to the TMES. The benefit of the magnetic stimulation is that it is far less painful. However, the downfall is that positioning of the coil on the back of the head makes it relatively easy to stimulate the lower threshold nerve roots, thus careful positioning of the coil is needed to avoid their activation (92).

If TMES is to be compared to TMS then it is important to know whether both stimulate the same corticospinal axons. When the two stimulations are delivered at appropriate interstimulus intervals in the biceps brachii, the antidromic volley of the CMEP (from TMES) collides and almost fully (>95%) obstructs the MEP (from TMS) (82). In addition, if a longer interstimulus is used a facilitative effect will occur due to interactions at the motoneurones (81 ,82, 83). Therefore, it can be said that for the hand and elbow flexors the volley evoked by TMES travels in many of the same axons that are evoked during TMS. The interaction between the two stimulations, however are complex due to the multiple descending volleys by the TMS. Despite this the two measurements are a novel means to test motoneurone responsiveness during muscle activity or fatigue.

The Hoffman Reflex (H-reflex) can be measured from a muscle when electrical stimulation of large-diameter axons of a primary muscle spindle afferents (located in the peripheral nerve) activates motoneurone(s). Increasing the stimulation intensity during a series of stimulations will create a recruitment curve for the H-reflex and the muscle compound action potential (M_{max}). Once the H-reflex reaches its maximum it is known as the H_{max}. Comparing the size of the H-reflex with the size of M_{max} one can estimate the segmental spinal excitability (including the motoneurone)

(93). One major mechanism that affects the size of the H-reflex is presynaptic inhibition that acts on the Ia terminals through other afferent and descending pathways (94). Another mechanism that has been shown to affect the Ia terminal is homosynaptic post-activation. This is caused by the release of transmitter from the terminal resulting in a decrease in efficacy of the action potentials (95). Finally, the last mechanism is repetitive firing of the Ia afferents, which will diminish the axons excitability to electrical stimulation. Therefore, stimulating with the same intensity will no longer elicit the same response (96). The main limitation of H-reflex testing is the difficultly in evoking a response in several muscles, particularly at rest, thus reducing its strength as a technique.

The F-wave is a late response from a stimulation of the peripheral nerve. It reflects the retrograde transmission of a small number of motoneurones that are reactivated by antidromic impulses following supramaximal stimulation (97). F-waves are small and inconsistent in both size and shape, therefore many responses must be recorded and an average calculated in order to interpret the results (98). It is believed that the excitability of the axon initial segment is responsible for the production of the F-wave from the motoneurone (97). The F-wave is a test that activates a small portion of the motoneurone pool and could exclude the smaller, slower motoneurones (99). However, it is problematic when testing proximal muscles as the larger M-wave's orthodromic response overlaps the small F-wave.

Corticospinal and spinal excitability can be influenced by the periphery. The peripheral nerve, neuromuscular junction and muscle, are all outside of the CNS and can be factors that influence peripheral excitability. These properties can be modulated by a number of factors, such as voluntary contraction (100), fatigue (101), pain (102) and limb position (103). When understanding where the corticospinal excitability changes are by analyzing MEPs and CMEPs it is important to eliminate the changes occurred at the peripheral level. Thus, MEP and CMEP

amplitudes can be normalized to the M_{max} to account for any alterations in the periphery. To elicit a M_{max}, a maximal stimulation is applied to the nerve of the muscle of interest, which creates a response in the muscle (104). By normalizing the MEP and CMEP to the M_{max} it allows the investigator to eliminate any potential differences in peripheral excitability and determine where changes occurred along the corticospinal pathway.

In conclusion, MEPs are based on the excitability of the cortical and spinal levels. With the CMEP not being influenced by the cortical level, it offers a possible way to help detect where the change has occurred. To put this in perspective, if MEP amplitude increases in size after an intervention with no significant increase or decrease in CMEP amplitude, then the change can potentially be located at the cortical level. Although the CMEP travels through many of the same axons as the MEP to recruit motoneurones it still has some limitations. The fact that the CMEP is a single volley it may lead to a different motoneuronal responses compared to the MEP due to its multiple descending volleys. (92). With an understanding of how the techniques are used to measure CSE in humans, the way variations in upper limb posture affect CSE can be discussed. While H-reflex and F-waves do test the excitability of the motoneurone and gives useful information, the limitations for each measurement must be considered.

Supraspinal Excitability

Paired-pulse techniques of the TMS allow the study of mechanisms of cortical inhibition and facilitation. Kujirai et al. (105) created the classic method where evoking a suprathreshold MEP test stimulus is preceded by a variable interstimulus interval (ISI) of a conditioning subthreshold stimulus. The test MEPs size is expressed as the percentage of the MEP elicited by the unconditioned stimulus. If the ISI is 7msec or longer the MEP is facilitated, if the ISI is 2 to 4 ms the MEP is depressed. These interactions originate in the cortex from different neuronal

populations and are known as intracortical facilitation (ICF) and short-interval intracortical facilitation (SICI). The difference between the first two techniques and long-interval intracortical inhibition (LICI) is that the conditioning pulse is suprathreshold instead of subthreshold and the ISIs are longer. The test MEPS are facilitated at 20-40ms ISIs and inhibited at ISIs <200ms. This inhibition has also been related with reduced motor cortex excitability (69, 106)

A MEP/CMEP ratio has been used by researchers (69) to show a global assessment of the corticospinal pathway. Since the response from TMS stimulation can be affected by spinal excitability, we can use responses by TMES to explain the spinal excitability. Therefore, by expressing a ratio one can better understand where the changes in CSE has occurred.

Overall, a combination of these stimulation techniques can be used to determine how CSE is altered due to exercise, disease, pain, fatigue or by providing augmented feedback to the participant.

1.4 Conclusion

The literature review has examined different variations of augmented feedback and how they contribute to motor performance. The literature shows the motor performance is improved when 1) external attentional focus feedback, 2) positive feedback and 3) autonomy is provided to an individual. In contrast, motor performance is impaired when 1) internal focus feedback, 2) negative feedback and 3) no autonomy is provided. Areas of motor performance examined include but are not limited to 1) power and force output, balance, accuracy, and speed. These changes have been consistently seen with trained and untrained, healthy and unhealthy, young and old, and male

and female populations. Out of these three variations of augmented feedback, the effects of attentional focus on motor performance seem to be the most documented.

Although the effects of attentional focus feedback on motor performance is well-known, we do not yet know the mechanisms underlying these changes. A couple studies suggest enhanced neuromuscular coordination as one of the mechanisms but the evidence supporting this hypothesis is limiting. The literature review has examined corticospinal excitability and has discussed techniques utilized to measure CSE. Changes in corticospinal excitability is modulated by several factors and could be a contributing mechanism to changes in motor performance seen with attentional focus feedback.

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Chapter 2: Co-authorship Statement

My contributions to this thesis are outlined below:

1. I recruited all participants and analyzed all data collected for this thesis, with the help of my peer Mr. Nick Snow
2. With the assistance of Mr. Behzad Lahouti (masters' student), I collected the experimental data for this thesis.
3. I prepared the manuscript and thesis with the help and guidance of my supervisor, Dr. Duane Button.
4. Dr. Duane Button provided constructive feedback on the manuscript and thesis.

Chapter 3: Neuromuscular Mechanisms Underlying Changes in Force Production During an Attentional Focus Task

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3.1: Abstract

The objective of this thesis was to examine changes in maximal voluntary force output of the elbow flexors with attentional focus feedback cues and possible underlying physiological mechanisms for these changes. Eleven recreationally active males participated in two randomized experimental sessions (Day 1: n=11, Day 2: n=10); 1) Stimulation session where corticospinal excitability was measured 2) No stimulation session where only electromyography and elbow flexor force was measured. In both sessions, four randomized blocks of three maximal voluntary contractions were performed. The blocks consisted of two externally and two internally attentional focus cued blocks. During the stimulation session transcranial magnetic, transmastoid and Erb's point stimulations were used to induce motor evoked potentials (MEPs), cervicomedullary MEP (CMEPs) and maximal muscle action potential (M_{max}), respectively in the biceps brachii. Results showed that force was significantly less (p = .024) under the internal contraction condition (282.4 ± 60.3 N) versus the external contraction condition (310.7 ± 11.3 N). force measurements were significantly smaller (p = .033) during the stimulation session (279.0 ± 47.1 N) than the no-stimulation session (314.1 ± 57.5 N). Muscle co-activation was significantly greater (p = .016) under the internal contraction (26.3 ± 11.5%) versus external contraction condition (21.5 ± 9.4%). There were no significant changes in corticospinal excitability measures between conditions. In conclusion, maximal voluntary force production of the elbow flexors is greater when an external focus feedback cue is provided. This appears to be due to less coactivation of the triceps and biceps brachii. Secondly, stimulating the corticospinal pathway seems to have some confounding effect on attentional focus. The distressing stimulations could distract participants from attentional focus cued feedback and/or stimulating the corticospinal pathway could disrupt areas of the cortex responsible for attention and focus.

3.2: Key Words:

Transcranial magnetic stimulation, transmastoid electrical stimulation, motor evoked potential, cervicomedullary evoked potential, electromyography

3.3: Introduction

The effects of attentional focus instructions on motor learning and performance have been extensively studied in the past 20 years. Specifically, two types of instructions have been compared and contrasted: those that elicit an internal focus (IF) and external focus (EF) of attention (1, 2). EF leads one to focus on the intended effects of movements on the environment. For example, focusing on the bulls eye during a dart throwing task. Conversely, IF leads one to focus on a body part or muscle group. For example, focusing on wrist movement during a dart throwing task. The vast majority of studies report that EF enhances motor learning and physical performance compared to IF (1, 3, 6, 10, 11). This includes tasks that require accuracy, balance, strength and speed. The effects are consisted across children, adults, older adults, and those suffering from mental disease (8, 19, 20). These effects are – arguably – some of the most established ones identified in human movement science.

Despite the impressive number of studies comparing attentional focus strategies across tasks and populations, little is known about the underpinning mechanisms. Few studies directly and thoroughly investigated the pathways that can explain the observed effects. A handful of studies examined if attentional focus strategies lead to different brain activation patterns using Electroencephalography (EEG) and fMRI (22, 23, 24, 25). EEG alpha power is generally lower during EF and associated with more ideal alpha frequencies. An fMRI study observed higher activation in the motor cortex during EF compared to IF. Thus, some evidence shows that the differences between EF and IF occurs in the central nervous system. The most commonly used

tool to shed light on the mechanistic pathway explaining the superiority of EF is surface electromyography (EMG). A repeated—although not consistent—pattern is that IF leads to larger muscle EMG activity from both the agonist and antagonist muscle groups involved in the task execution (4, 5, 12) This is commonly explained by enhanced neuromuscular coordination associated with EF, which promotes effective and efficient movement patterns (1). However, EMG alone cannot pinpoint the pathways leading to the enhanced movement patterns associated with EF. Indeed, EF can promote superior motor performance by eliciting greater nervous system excitation, less inhibition, or a combination of both, possibilities that EMG cannot capture. Hence, there is a need to combine a number of tools to deepen our understanding of the central pathways accounting for the consistent difference in motor learning and performance between EF and IF.

Nervous system excitation and inhibition can be examined through measuring corticospinal excitability via transcranial magnetic stimulation (TMS), and transmastoid electrical stimulation (TMES) (14, 15). TMS elicits a motor evoked potential (MEP) in a muscle of interest, while TMES elicits a cervicomedullary MEP. TMS-evoked MEP amplitudes are used to quantify CSE (16). Alterations in CSE could occur anywhere along the corticospinal pathway (i.e., from cortex to motoneuron). The combined use of the mentioned techniques is used to determine whether the modulation of CSE is predominantly supraspinal or spinal (14). The corticospinal tract is examined due to its importance in the organization of single and multi-jointed movements. The corticospinal fibers control the spinal motoneurons that innervate the muscles of the trunk and limbs (21). Many modulators have been shown to influence CSE from Caffeine indigestion (17) to arousal imagery (18). It is possible that EF may increase corticospinal excitability, decrease corticospinal inhibition, or a combination of both which would account, in part, for the increase in motor performance seen. This would further our understanding of the pathways and underlying

mechanisms to address the changes in motor performance and learning seen with attentional focus feedback.

It is well documented that EF improves performance and IF impairs performance. While there is some EMG evidence to suggest that these changes are due to enhanced neuromuscular coordination associated with EF, more research is required to further support this. To date, few studies have examined co-activation patterns using EMG in relation to EF and IF feedback and force output. As well, changes in CSE with EF and IF feedback has yet to be studied. Examining changes in CSE will deepen our understanding of the magnitude and location of changes in the nervous system to account for the differences in force output between EF and IF feedback. Therefore, the aim of this study was to; 1) compare co-activation patterns of the biceps brachii and triceps brachii between EF and IF cued maximal voluntary contractions of the elbow flexors and 2) compare CSE to the biceps brachii between EF and IF cued maximal voluntary contractions of the elbow flexors. We hypothesized that; 1) co-activation would be greater with an IF cued contraction and 2) CSE would be modulated differently between EF and IF cued contractions.

3.4 Materials and Methods

Twelve university aged resistance-trained males (177 ± 2.83 cm, 84.32 ± 3.22 kg, 23.8 ± 2.36.) were recruited for the experimental study. Resistance-trained status was determined as meeting the Canadian Society of Exercise Physiology guidelines of two hours a week of resistance training for at least a year. We chose to recruit only resistance-trained males because corticospinal excitability is training dependent (36, 37, 38, 39). Participants completed a magnetic stimulation safety checklist prior to participation in order to screen for potential contraindications with magnetic stimulation procedures (35). Participants were told about the procedures to be used during the experiment and if accepted they gave their informed written consent. The study was

approved by The Memorial University of Newfoundland Interdisciplinary Committee on Ethics in Human Research and was in accordance with the Tri-Council guidelines in Canada with full disclosure of potential risks to participants.

3.4.1 Elbow Flexor Force

Participants were seated in a custom-built chair (Technical Services, Memorial University of Newfoundland) in an upright position, with chest and head strapped in place to minimize movement, with hips and knees flexed at 90˚. The forearm was held horizontal, positioned in supination with the shoulders resting against the back of the chair, and placed in a custom-made orthosis that was connected to a load cell. The load cell detected force output, which was amplified (x1000) (CED 1902, Cambridge Electronic Design Ltd., Cambridge, UK) and displayed on a computer screen. Data was sampled at 5000 Hz. Participants were instructed to maintain an upright position with their head in a neutral position during contractions. Visual feedback was given to all participants during each contraction as a line on a computer screen in front of them showing when to begin and end contraction. Information about force production and participants were only able to view their Biceps Brachii EMG activity.

3.4.2 Electromyography

Electromyography (EMG) activity was recorded by using surface EMG recording electrodes (MediTrace Ag-AgCl pellet electrodes, disc shaped and 10 mm in diameter, Graphic Controls Ltd., Buffalo, N.Y., USA) from the dominant arms biceps brachii and triceps brachii. Electrodes were placed 2 cm apart (center to center) over the midpoint of the muscle belly of the participant's biceps brachii and triceps brachii lateral head. A ground electrode was placed over the lateral epicondyle of the dominant knee. Skin preparation for all recording electrodes included

shaving to remove excess hair and cleaning with an isopropyl alcohol swab to remove dry epithelial cells. An inter-electrode impedance of <5 kΩ was obtained prior to recording to ensure an adequate signal-to-noise ratio. EMG signals were amplified (×1000) (CED 1902) and filtered using a 3-pole Butterworth filter with cut-off frequencies of 10–1000 Hz. All signals were analog-digitally converted at a sampling rate of 5 kHz using a CED 1401 (Cambridge Electronic Design Ltd., Cambridge, UK) interface.

3.4.3 Stimulation conditions

Motor Responses from the bicep brachii were elicited via 1) transcranial magnetic stimulation (TMS), 2) transmastoid electrical stimulation (TMES) and 3) brachial plexus electrical stimulation at Erb's point. Stimulation intensities used for TMS and TMES were adjusted similar to that of Pearcy et Al (2014) so that the evoked potentials produced by each, MEPs, and CMEPs, respectively, were of similar amplitude and normalized to a maximal M-wave (Mmax). Stimulation intensities were then set during an isometric elbow flexion contraction equal to 5% of MVC.

Transcranial magnetic stimulation (TMS)

TMS-evoked motor evoked potentials (MEPs) were used to measure corticospinal excitability. A TMS (Magstim 200, maximal output 2.0 Tesla) circular coil (13 cm outside diameter) was placed directly over the vertex of the head to induce MEPs in the active (5% maximal voluntary contraction (MVC)) biceps brachii muscle. The vertex was located by marking the measured halfway points between the nasion and inion and tragus to tragus. The coil was flipped to ensure the induced current flow was anterior to posterior in the target motor cortex (A side up for right side, B side up for left) to activate the dominant biceps brachii. Stimulation

intensity was set to elicit a MEP 10-20% of Mmax taken as an average of eight trials in the biceps brachii during a 5% MVC.

Transmastoid electrical stimulation (TMES)

Stimulation was applied via surface electrodes placed over the mastoid processes and current was passed between them (200μs duration, 80-200 mA); model DS7AH, Digitimer Ltd, Welwyn Garden City, UK). Stimulation intensity was adjusted to prevent ventral root activation by closely monitoring CMEP responses for any decrease in onset latency (~2ms), which shows cervical ventral root activation (Taylor et al. 2006). Stimulation intensity was adjusted to elicit a response that matched the size of MEP amplitude, taken as an average of eight trials, in the biceps brachii during a 5% MVC.

Brachial plexus stimulation

Stimulation of the brachial plexus was used to measure maximal compound muscle action potential (M_{max}). Erb's point was electrically stimulated via a cathode on the skin in the supraclavicular fossa and an anode on the acromion process. Current pulses were delivered as a singlet (200 μs duration, 90-185 mA). The electrical current was gradually increased until M_{max} of the biceps brachii at a 5% MVC was observed.

3.4.4 Experimental Protocol

Participants completed a familiarization session and two experimental sessions that were randomized. Each session took place on separate days.

Familiarization session

Participants performed two 5 second MVCs of the dominant elbow flexors, with 2 minutes of rest between contractions. If the difference between the two MVCs was greater than 5%, a third

MVC was performed. Following completion of the MVCs, participants practiced holding the 5% MVC contraction for 10 seconds at each position. Participants then received the three different types of stimulations at various intensities to ensure that they were comfortable to endure the stimulation paradigm involved in each experimental session.

Stimulation Session

Upon arrival, the participants were prepared for EMG and asked to perform two elbow flexor MVCs. A 10-minute rest period was then issued to ensure no effect of the MVC on the CSE measurements (38). Following the rest period, the experimental procedures began and the stimulation intensities for the M_{max}, MEP, and CMEP of the biceps brachii during 5% MVC were determined. Participants then moved on to perform a semi-randomized protocol where they completed four blocks of 3 MVCs of the elbow flexors with 3 minutes of rest between MVCs. Five minutes of rest was given between each block of contractions. A total of 12 MVCs were performed. Participants were verbally directed with the same attentional focus cue provided immediately before each contraction in each block of contractions. Participants were either asked to “focus on pulling up on the handle as hard and as quickly as you possibly can” (external cue) or to “focus on contracting your biceps as hard and as quickly as you possibly can” (internal cue). In total participants were EF cued six times or IF cued six times. These cues were countered balanced between sets. During each contraction participants received counter-balanced TMSs and TMESs at 1.5 and 3 seconds and an M-wave was given at the 4.5 second mark. See Figure 1 for experimental set-up.

Non-Stimulation Session

The Non-Stimulation session was completed 48 hours from the first. This session was identical to the first except no stimulations were used. This session was included in the study to

examine if stimulations impact a participant's ability to perform a maximal voluntary contraction and their ability to focus on the attentional focus cues.

3.5 Data and Statistical Analysis

3.5.1 Data Analysis

Maximum voluntary isometric contractions (MVCs). Force, EMG, and CSE data were measured offline using Signal 4.0 software (Cambridge Electronic Design Ltd., Cambridge, UK). All offline computations were conducted using Microsoft Office Excel 2016 (Microsoft Corporation, Redmond, WA, USA).

Peak force. Peak elbow flexor force measurements (in Newtons, N) were obtained from all six MVCs under each condition (external cued contraction and internal cued contraction) during both no-stimulation and stimulation sessions. MVC force output was measured as the peak-to-peak amplitude from no force to maximum force.

Electromyography (EMG). Root mean square EMG (rmsEMG) was calculated during the 1 s to 2s interval of each MVC trial from the biceps brachii and triceps brachii muscles under each condition (external cued contraction and internal cued contraction) and during each session (no-stimulation and stimulation). Additionally, muscle co-activation was quantified by computing the percentage of triceps brachii rmsEMG/biceps brachii rmsEMG (Cadigan et al., 2017). To examine the relationship between force production and muscle activation, the percentage ratio of muscle co-activation per Newton of force was calculated for MVCs from both the external and internal cued contraction conditions during both no-stimulation and stimulation sessions.

Corticospinal excitability (CSE). During the stimulation session only, six trials each of elbow flexor MEP, CMEP, and M_{max} peak-to-peak amplitudes (mV) were extracted during all six MVCs under each condition (external cued contraction and internal cued contraction). Since

amplitudes and areas give similar results, we used MEP, CMEP, and M_{max} amplitudes for comparisons (32). MEP and CMEP peak-to-peak amplitudes were normalized to matched M_{max} amplitudes (%M_{max}), given M_{max} is a stable measure of muscle activity during maximal muscle fibre recruitment (27). As well, ratios of matched normalized MEP/CMEP amplitude were calculated (30).

Prior to statistical analyses all data underwent quality control checks in Microsoft Office Excel 2016 (Microsoft Corporation, Redmond, WA, USA) for missing data points and outliers. In terms of missing data, one participant was unable to complete the stimulation session (P09). This participant was not included in CSE analyses; however, their MVC peak force and rmsEMG data (trial 1 to trial 6) for both the external contraction and internal contraction conditions (12 trials) were subsequently imputed for the stimulation session to enable groupwise comparisons across sessions. Additionally, two participants (P10, P11) were missing force data for one MVC trial each (trial 4), under both the internal and external contraction conditions, for the stimulation session alone (four trials). In total, 16 datapoints were missing for MVC peak force (6.1%) and 12 datapoints each were missing for rmsEMG of both biceps brachii (4.5%) and triceps brachii (4.5%). Missing data were imputed by determining the series average for the entire sample, including both conditions (external contraction, internal contraction), at their respective timepoints and sessions using the Missing Values Analysis and Transform functions in SPSS (V26.0, IBM Corporation, Armonk, NY, USA). Outliers were considered datapoints that exceeded the sample mean by ± three standard deviations (SD). No outliers were identified.

3.5.2 Statistical Analysis

Statistical analyses were completed using SPSS (V26.0, IBM Corporation, Armonk, NY, USA). Assumptions of normality (Shapiro-Wilk test), sphericity (Mauchly's test), and

homogeneity of variances (Levene's test) were tested for all outcome measures where appropriate. For the Shapiro-Wilk test, statistical significance was set at $p < .001$ (34) .

All data were normally distributed (MVC: $W_{(11)} = 0.821\text{-}0.966$, $p = .018\text{-}.848$; rmsEMG: $W_{(11)} = 0.699\text{-}0.982$, $p = .001\text{-}.976$; CSE: $W_{(10)} = 0.684\text{-}0.934$, $p = .001\text{-}.490$), with the exception of MEP/CMEP ratio values under the internal contraction condition of the stimulation session ($W_{(10)} = 0.628$, $p = .0001$) and muscle co-activation (% triceps/biceps brachii rmsEMG) under the internal contraction condition of the stimulation session ($W_{(11)} = 0.639\text{-}0.858$, $p = .0001\text{-}.054$). Thus, all MEP/CMEP ratio and muscle co-activation values were square root transformed using the Transform function in SPSS (V26.0, IBM Corporation, Armonk, NY, USA), resulting in normal distributions (MEP/CMEP: $W_{(10)} = 0.740\text{-}0.879$, $p = .003\text{-}.126$; co-activation: $W_{(11)} = 0.745\text{-}0.956$, $p = .002\text{-}.715$). In the event of a violation of the assumption of sphericity, *p*-values were adjusted using the Greenhouse-Geisser correction. If the assumption of homogeneity of variances was violated, *p*-values were adjusted (equal variances not assumed).

To rule out whether measures of MVC peak force, rmsEMG, or CSE changed over subsequent trials (trial 1 to trial 6), separate one-way repeated-measures analyses of variance (ANOVAs) with the factor TRIAL (6 levels) were conducted on all data independently for internal contraction and external contraction conditions, as well as stimulation and no-stimulation sessions. This test was used to guide subsequent analyses in terms of whether trials were pooled or tested separately. For MVC peak force, the main effect of TRIAL was statistically significant in all cases ($F_{(5, 50)} \geq 3.982$, $p \leq .022$). Similarly, with reference to rmsEMG data, the main effect of TRIAL was statistically significant in most cases ($F_{(5, 50)} \leq 6.690$, $p \geq 0001$). However, regarding CSE, the main effect of TRIAL was not statistically significant in any case ($F_{(5, 45)} = \leq 2.137$, $p \geq .150$).

Consequently, in main statistical tests, TRIAL was considered a separate factor for MVC peak force and rmsEMG data, whereas all levels of the factor TRIAL were pooled for CSE.

For main statistical tests, repeated-measures ANOVAs and paired-samples *t*-tests were used, with designs depending on the result of the above one-way repeated-measures ANOVAs. Peak force measurements from MVCs (in Newtons, N) were compared across trials (trial 1 to trial 6), conditions (external cued contraction and internal cued contraction), and sessions (no-stimulation and stimulation) using a 6 × 2 × 2 three-way repeated-measures ANOVA with the factors TRIAL, CONDITION, and SESSION, respectively. Raw rmsEMG values for biceps brachii and triceps brachii were examined separately for each session (no-stimulation and stimulation) across trials (trial 1 to trial 6) and conditions (external cued contraction and internal cued contraction) using 2 × 2 two-way repeated measures ANOVAs with the factors TRIAL and CONDITION, respectively, given they were not normalized (31). Because triceps brachii/biceps brachii co-activation values were normalized, they were compared as square root transformed values across trials (trial 1 to trial 6), conditions (external cued contraction and internal cued contraction), and sessions (no-stimulation and stimulation) using separate 6 × 2 × 2 three-way repeated measures ANOVAs with the factors TRIAL, CONDITION, and SESSION, respectively (31). For CSE, average values across all trials (trial 1 to trial 6) for M_{max} amplitude (mV), as well as MEP/M_{max}, CMEP/M_{max}, and square root transformed CMEP/MEP ratios, were compared across conditions (external cued contraction and internal cued contraction) using separate paired-samples *t*-tests. Finally, to investigate the relationship between changes in peak force and co-activation across stimulation conditions, two analyses were performed. First, a 2 × 2 two-way repeated-measures ANOVA with the factors CONDITION and SESSION was conducted on the percentage ratios of muscle co-activation per Newton of force calculated from MVCs from both

the external and internal contraction conditions during both no-stimulation and stimulation sessions. Last, simple bivariate correlations (Pearson's *r*) were calculated between changes in MVC peak force and triceps brachii/biceps brachii co-activation from external to internal cued contractions in the no-stimulation and stimulation sessions separately. Strength of the correlation coefficients (*r*) was interpreted as < 0.3 (negligible), 0.3-0.5 (weak), 0.5-0.7 (moderate), 0.7-0.9 (strong), and > 0.9 (very strong) (33).

Statistical significance for main tests was set at $p \leq .05$. In the event of a statistically significant ANOVA outcome, pairwise comparisons were completed *post hoc* using the Bonferroni-correction. Effect sizes were estimated using Cohen's d (28) and were calculated as ($[M_1 - M_2]/[\sigma_{pooled}]$) using Microsoft Office Excel 2016 (Microsoft Corporation, Redmond, WA, USA), where $[M_1 - M_2]$ is the mean difference between two measurements and $[\sigma_{pooled}]$ is the pooled standard deviation of those two means. Effect sizes were interpreted as < 0.2 (trivial), 0.2-0.5 (small), 0.5-0.8 (medium), > 0.8 (large) (28). In the text results are reported as mean ± SD; in tables, data are shown as mean ± SD and range; in figures, individual raw data and mean ± SD are displayed.

3.6 Results

3.6.1 Peak Force

MVC peak force are shown in **Figure 2**, **Tables 1-2**. The three-way repeated-measures ANOVA on peak force measurements from elbow flexor MVCs revealed three statistically significant main effects. First, a statistically significant main effect of CONDITION ($F_{(1, 10)}$ = 7.033, p = .024, d = 1.68, large effect) showed that force was significantly less under the internal contraction condition (282.4 ± 60.3 N) versus the external contraction condition (310.7 ± 11.3 N;

p = .024, d = 0.56, medium effect) (**Figure 2A**). Next, a statistically significant main effect of SESSION ($F_{(1, 10)}$ = 6.076, p = .033, d = 1.56, large effect) demonstrated that force measurements were significantly smaller during the stimulation session (279.0 ± 47.1 N) than the no-stimulation session (314.1 ± 57.5 N; p = .033, d = 0.67, medium effect) (**Figure 2B**). Finally, there was a statistically significant main effect of TRIAL ($F_{(5, 50)}$ = 14.262, p = .00001, d = 2.47, large effect) (see **Table 2** for multiple comparisons). Neither the TRIAL × CONDITION ($F_{(5, 50)}$ = 1.701, p = .152, d = 0.82, large effect), TRIAL × SESSION ($F_{(5, 50)}$ = 0.211, p = .891, d = 0.29, small effect), CONDITION × SESSION ($F_{(5, 50)}$ = 1.365, p = .270, d = 0.74, medium effect), nor TRIAL × CONDITION × SESSION interactions ($F_{(5, 50)}$ = 1.344, p = .281, d = 0.74, medium effect) were statistically significant.

3.6.2 Electromyography (EMG)

Biceps brachii and triceps brachii rmsEMG data are displayed in **Tables 1-2**.

Biceps brachii.

No-stimulation session. For biceps brachii rmsEMG during the no-stimulation session there was a statistically significant main effect of TRIAL ($F_{(5, 50)}$ = 7.341, p = .001, d = 1.71, large effect) (see **Table 2** for multiple comparisons). The main effect of CONDITION trended towards significance ($F_{(1, 10)}$ = 3.958, p = .075, d = 1.26, large effect) and indicated that biceps brachii rmsEMG tended to be greater under the external contraction condition (0.73 ± 0.51) compared to internal contraction (0.60 ± 0.38). The TRIAL × CONDITION interaction effect was not statistically significant ($F_{(5, 50)}$ = 1.83, p = .133, d = 0.84, large effect).

Stimulation session. During the stimulation session, the main effect of TRIAL trended towards significance ($F_{(5, 50)}$ = 3.317, p = .068, d = 1.15, large effect) and suggested that rmsEMG tended to be greater under trial 3 (0.58 ± 0.33) versus trial 4 (0.53 ± 0.33) (see **Table 2** for multiple

comparisons). Otherwise, there was neither a statistically significant main effect of CONDITION ($F_{(1, 10)} = 2.407$, $p = .152$, $d = 0.98$, large effect) nor TRIAL × CONDITION interaction effect ($F_{(5, 50)} = 0.506$, $p = .565$, $d = 0.45$, small effect).

Triceps brachii.

No-stimulation session. With reference to triceps brachii rmsEMG throughout the no-stimulation session, there were no statistically significant main effects of TRIAL ($F_{(5, 50)} = 1.722$, $p = .210$, $d = 0.83$, large effect) or CONDITION ($F_{(1, 10)} = 2.178$, $p = .171$, $d = 0.93$, large effect), nor a two-way TRIAL × CONDITION interaction effect ($F_{(5, 50)} = 0.510$, $p = .528$, $d = 0.45$, small effect).

Stimulation session. In the stimulation session, there were no statistically significant effects of TRIAL ($F_{(5, 50)} = 1.443$, $p = .226$, $d = 0.76$, medium effect), CONDITION ($F_{(1, 10)} = 0.141$, $p = .716$, $d = 0.24$, trivial effect), or TRIAL × CONDITION ($F_{(5, 50)} = 0.642$, $p = .583$, $d = 0.51$, medium effect), for triceps brachii rmsEMG.

Co-activation. Muscle co-activation data (expressed as % triceps/biceps rmsEMG) are shown in **Figure 3A** and **Tables 1-2**. The three-way repeated-measures ANOVA on percentage values of co-activation demonstrated a statistically significant main effect of CONDITION ($F_{(1, 10)} = 8.438$, $p = .016$, $d = 1.84$, large effect), whereby muscle co-activation was significantly greater under the internal contraction (26.3 ± 11.5%) versus external contraction condition (21.5 ± 9.4%; $p = .016$, $d = 1.84$, large effect) (**Figure 3A**).

The main effects of TRIAL ($F_{(5, 50)} = 2.123$, $p = .136$, $d = 0.92$, large effect) and SESSION ($F_{(1, 10)} = 0.029$, $p = .869$, $d = 0.11$, trivial effect) were not statistically significant. Likewise, neither the TRIAL × CONDITION ($F_{(5, 50)} = 0.175$, $p = .971$, $d = 0.26$, trivial effect), TRIAL × SESSION ($F_{(5, 50)} = 0.419$, $p = .833$, $d = 0.41$, small effect), CONDITION × SESSION ($F_{(5, 50)} = 1.969$, $p =$

.191, d = 0.89, large effect), nor TRIAL × CONDITION × SESSION ($F_{(5, 50)}$ = 2.072, p = .144, d = 0.91, large effect) interaction effects were statistically significant.

3.6.3 Corticospinal Excitability (CSE)

CSE data are presented for each condition (external contraction, internal contraction), collapsed across trials (trial 1 to trial 6) in **Table 3**.

There was no statistically significant difference ($t_{(9)}$ = -0.508, p = .624, d = 0.06, trivial effect; $t_{(9)}$ = 0.598, p = .565, d = 0.17, trivial effect; $t_{(9)}$ = 0.340, p = .742, d = 0.08, trivial effect; and $t_{(9)}$ = -1.215, p = .255, d = 0.26, small effect) in $M_{max,}$ MEP, or CMEP amplitudes or MEP/CMEP ratios, respectively across external cued contraction and internal cued contraction conditions.

3.6.4 Relationship between Change in Peak Force and Co-activation

Values of percent muscle coactivation per Newton of force production in MVCs, and correlations between changes in MVC peak force and triceps brachii/biceps brachii co-activation, are shown in **Figure 3B-D** and **Tables 1-2**.

Co-activation/MVC peak force. There was a statistically significant main effect of CONDITION for ratios of co-activation/Newton force produced in MVCs ($F_{(1, 10)}$ = 11.307, p = .007, d = 2.13, large effect), which indicated that under the external contraction condition (0.08 ± 0.04%) less muscle co-activation occurred per Newton of force production compared to the internal contraction condition (0.11 ± 0.05%; p = .007, d = 2.13, large effect) (**Figure 3B**). Neither the main effect of SESSION ($F_{(1, 10)}$ = 0.131, p = .725, d = 0.23, trivial effect) nor the CONDITION × SESSION two-way interaction effect ($F_{(5, 50)}$ = 1.333, p = 0.275, d = 0.73, medium effect) reached statistical significance.

Correlations.

No-stimulation session. During the no-stimulation session there was a statistically significant negative correlation between changes in MVC peak force (38.8 ± 48.6 N) and triceps brachii/biceps brachii co-activation (-9.2 ± 13.9%) across external and internal contraction conditions ($r_{(9)}$ = -0.623, p = .041, moderate correlation), suggesting increased co-activation was related to reduced MVC force production in the internal contraction condition (**Figure 3C**).

Stimulation session. In the stimulation session the relationship between changes across external and internal contraction conditions in MVC peak force and triceps brachii/biceps brachii co-activation was not present ($r_{(9)}$ = -0.312, p = .350, weak correlation) (**Figure 3D**).

3.7 Discussion

Overall, our results show that force production is lower when an internal focus cue is provided relative to an external focus cue. More specifically, when a participant was tasked with completing a maximal voluntary elbow flexion contraction, they produced less force when instructed with internal attentional focus cues compared to when they were instructed with external focus cues. Our results show that co-contraction (measured as rmsEMG Triceps Brachii/rmsEMG Biceps Brachii) between the biceps and triceps brachii is greater during an internal focus cued contraction relative to an external focus cued contraction indicating a different neuromuscular strategy that leads to reduced force output. However, a change in neuromuscular strategy did not coincide with a change in corticospinal excitability. Our results support an interaction between the stimulation techniques for measuring CSE and attentional focus which negate the effect of an external focused cue on enhanced force production compared to an internal focused cue. This interaction is supported by the between session analysis which showed that 1) force production

was higher during the non-stimulation sessions and 2) force was greater with an external focus cue during the non-stimulation session but not during the stimulation session

Maximal elbow flexor force is affected by the type of attentional focus cue.

In the current study, participants were able to produce more force when provided an external focus cue (condition 310.7 ± 11.3 N) compared to internal (282.4 ± 60.3 N) prior to contraction during the non-stimulation session. This is consistent with previous research which showed enhanced force production when given an external cue over no cue and internal focus cues. Specifically, Marchant et al. (10) found that during concentric elbow flexion completed at a set speed, an external cue exhibited a significantly higher peak net torque (102.10 ± 2.42%MVC) than the internal condition (95.33 ± 2.08%MVC). Halperin et al. (13) reiterated these results showing that when given an external focus cue during an isometric mid-thigh pull, trained athletes applied 9% more force compared to those that received an internal cue, and 5% more force than control. This supports that external focus cues enhance force output while internal cues impair performance.

While there was an observed difference in force production between conditions in the non-stimulation session, there were no significant changes in force production between conditions during the stimulation session. This finding is not consistent with previous research as it is well documented that attentional focus alters force production (1, 6, 10, 11, 13). Thus, other possible factors were involved. One possible factor to consider is that the stimulation distracted the participants from focusing on the cue provided. The stimulation techniques used were novel to the participants and tend to be intimidating and a cause of discomfort. Participants could have possibly been more focused on the incoming randomized stimulations than the attentional focus cues which would confound any effects these cues had on maximal force production. A second possible factor is that the use of the stimulation techniques disrupted areas of the cortex responsible for attention.

It is known that transcranial magnetic stimulation can disrupt cortical function and there have been a couple studies examining its effect on tasks requiring attention. A study by Ashbridge et al. (40) suggested that transcranial magnetic stimulation disrupts an area in the front parietal lobe responsible for the focal attention necessary for feature binding in a conjunction search task. Another study showed that repetitive TMS of the intraparietal sulcus and the frontal eye fields during an auditory spatial attention task impaired visually cued auditory attention (42). With each stimulation pulse it is possible that more than just the cortical area of interest was being stimulated (41) and therefore it is likely that cortical areas involved in attention were unintentionally disrupted. Either of the mentioned factors, or a combination thereof would confound the effects of external attentional focus on force production and explain the differences seen in force production between sessions.

In the current study, we also showed an effect of stimulation on maximal voluntary force production. Force produced in the stimulation session (279.0 ± 47.1 N) was shown to be significantly less than force produced during the non-stimulation session (314.1 ± 57.5 N). This finding would suggest that the stimulation techniques used impaired voluntary force production. Button and Behm (45) previously showed that the expectation of an interpolated twitch stimulation reduced voluntary force production by 9.5%. However, to date there appears to be a lack of research showing how stimulation of the nervous system using TMS and TMES influences force production. This finding should be further replicated and expanded upon to future studies as it would suggest the use of stimulations could be a confounding variable in program design for studies examining force production.

Mechanisms underlying changes in elbow flexor maximal force with attentional focus cues.

Electromyography

In the current study, we analysed co-activation of the triceps and biceps brachii muscles. Our results showed greater co-activation with an internal focus cue compared to an external cue. This is consistent with a previous study by Lohse et al. (44), who showed greater co-contraction between the lateral aspect of the soleus and the tibialis anterior with an internal focus cue during a submaximal plantar flexion task where participants were instructed to contract at 30% of MVC. Both of these findings further support Wulf et al's (1) "Constrained action hypothesis" that internal cues impair neuromuscular coordination. Greater co-activation of the agonist and antagonist musculature is another mechanism underpinning why maximal force production was less during internal than external focused cues. Based on the current EMG findings, it appears that force production is impaired with an internal cue due to disruption of natural automatized movement as supported by increases in co-activation compared to the external focused cue and that EMG was enhanced with an external cue due to greater motor unit recruitment and/or rate coding.

To date, there has only been a handful of studies that examined neuromuscular activation changes with attentional focus feedback using electromyography. These studies have shown increased EMG activity when given an internal focus cue during a contraction compared to control and external focus cues (4, 10, 12). It has been proposed through the *constrained action hypothesis* by Wulf et al. (1, 43) that internal focus leads participants to be conscious of their movement which disrupts their natural automatized movements (1, 43). This increase in EMG activity seen in these previous studies indicated more neuromuscular activity which may suggest poorer motor control. Unlike the previous studies we did not find a significant difference in EMG activity between focus cues. However, a strong effect a trend ($p = 0.075$) for greater neuromuscular activity of the biceps

brachii was found with an external focus cue. While internal cues impair performance through disrupting natural automatized movement as proposed by Wulf et al (1), external focus cues may improve performance through other means such as increased motor unit recruitment and rate coding typically measured through increases in EMG activity (14).

In the current study, there were no significant changes in EMG activity or co-activation measurements observed in the stimulation session. This is to be expected as there was no effect of attentional focus on force production during that session.

Corticospinal Excitability

In our study, measures of corticospinal excitability were used during one of the two sessions (stimulation session). This allowed us to determine whether or not the increase in maximal elbow flexor force with an external focus cue was due, in part, to enhanced corticospinal excitability of the biceps brachii. While one of the goals of this study was to examine the influence of attentional focus on corticospinal excitability, we were unable to due to the confounding effect of stimulation on attentional focus as discussed earlier. During the stimulation session there were no significant changes in force production between attentional focus cues and as such there were no significant changes in corticospinal excitability between these cues as well. We expected to see an increase in corticospinal excitability of the biceps brachii with an external focus cue as increased central drive is a well-known mechanism underlying increases in force production (26, 38, 39). However, we were unable to support this possibility with the current study and to date this appears to be the only study examining corticospinal excitability and attentional focus feedback.

Methodological considerations

Measures of central drive remains a possible mechanism underlying differences in elbow flexors MVC force between external and internal focus cues. However, the confounding effect of the stimulation techniques used to examine corticospinal excitability on attentional focus makes this difficult to achieve. Future studies should examine how stimulations affects attention exactly and then adapt the protocol to address the proposed issue. As discussed earlier, it is possible that the discomfort of the stimulations may distract participants from the attentional focus cues. It is possible that this may be only for participants who are not accustomed to being stimulated. Further research could examine differences in responses to stimulations between participants who are and are not accustomed to stimulations and if accustomed participants are distracted less by stimulations. This would open up the possibility of using accustomed participants to study changes in corticospinal excitability with attentional focus cues. A second possibility mentioned was that the stimulations may disrupt areas of the cortex involved with attention. Further studies should aim to confirm this and then locate and utilize more accurate and precise stimulation techniques to ensure that only the area of interest of the motor cortex is being stimulated.

3.8 Conclusion

In conclusion, force production during a maximally voluntary contraction of the elbow flexors is impaired by an internal attentional focus cue. Greater co-activation of the triceps brachii and biceps brachii appears to be an underlying mechanism for this impairment. Additionally, the use of stimulation techniques impairs attention during an attentional focus task. This makes it difficult to examine the influence of central drive as a possible mechanism for the impairments in force seen with internal focus cues. Finally, stimulations confound the ability to produce force during a maximal force production task.

3.9 References

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3.11 Tables

Table 1. Maximum voluntary isometric contraction (MVC) force and electromyographic (EMG) data, collapsed across trials (trial 1 to trial 6), presented for conditions (external contraction, internal contraction) during both no-stimulation and stimulation sessions.
Data presented as mean (M), standard deviation (SD), and range. N, Newton; rmsEMG, root mean square of EMG signal.

Session	**No-stimulation**						**Stimulation**					
Condition	**External Contraction**			**Internal Contraction**			**External Contraction**			**Internal Contraction**		
	M	SD	Range	M	SD	Range	M	SD	Range	M	SD	Range
MVC Peak Force (N)	333.5	43.7	242.6-375.5	294.8	76.7	184.2-398.6	287.9	38.7	245.3-376.0	270.1	62.4	124.3-367.1
Biceps Brachii rmsEMG	0.73	0.51	0.27-1.88	0.60	0.38	0.16-1.47	0.59	0.33	0.26-1.41	0.53	0.25	0.21-1.04
Triceps Brachii rmsEMG	0.12	0.03	0.07-0.18	0.15	0.09	0.05-0.34	0.11	0.04	0.05-0.17	0.12	0.04	0.05-0.17
Co-activation (% Triceps/Biceps rmsEMG)	22.2	13.8	8.6-49.6	31.5	19.5	12.1-77.2	24.3	15.7	11.3-63.0	26.8	17.5	12.7-74.4
% Co-activation per Newton Force	0.06	0.04	0.03-0.14	0.12	0.08	0.03-0.24	0.09	0.06	0.03-0.23	0.11	0.08	0.03-0.29

Table 2. Maximum voluntary isometric contraction (MVC) force and electromyographic (EMG) data, collapsed across sessions (no-stimulation, stimulation) and conditions (external contraction, internal contraction), for MVC trial 1 to trial 6.
Data presented as mean (M), standard deviation (SD), and range. N, Newton; rmsEMG, root mean square of EMG signal. [a], statistically significant difference versus trial 1, $p < .05$. [b], statistically significant difference versus trial 2, $p < .05$. [c], statistically significant difference versus trial 3, $p < .05$. [d], statistically significant difference versus trial 4, $p < .05$. [e], statistically significant difference versus trial 5, $p < .05$.

MVC Trial #	**1**			**2**			**3**			**4**			**5**			**6**		
	M	SD	Range	M	SD	Range	M	SD	Range	M	SD	Range	M	SD	Range	M	SD	Range
MVC Peak Force (N)	313.8 *d, e*	45.8	229.9-373.8	304.7 *d, e*	42.1	224.4-372.2	308.1 *d, e*	36.2	258.9-377.7	276.0 *a, b, c*	34.1	227.5-336.1	284.0 *a, b, c*	30.4	23.4-320.9	288.6	34.8	219.4-335.6
Biceps Brachii rmsEMG																		
Stimulation	0.72	0.46	0.25-1.83	0.70 *e*	0.46	0.22-1.76	0.69 *d*	0.45	0.21-1.71	0.64 *c*	0.43	0.21-1.59	0.64 *b*	0.45	0.23-1.67	0.61	0.40	0.17-1.50
No Stimulation	0.63	0.28	0.31-1.10	0.57	0.31	0.18-1.27	0.58	0.33	0.20-1.38	0.53	0.33	0.18-1.35	0.52	0.26	0.21-1.14	0.53	0.27	0.20-1.15
Triceps Brachii rmsEMG																		
Stimulation	0.14	0.05	0.07-0.23	0.15	0.06	0.07-0.26	0.14	0.06	0.06-0.25	0.14	0.08	0.06-0.35	0.13	0.06	0.06-0.25	0.12	0.05	0.06-0.20
No Stimulation	0.12	0.04	0.05-0.16	0.11	0.04	0.05-0.17	0.12	0.04	0.06-0.17	0.11	0.04	0.05-0.18	0.12	0.05	0.05-0.19	0.11	0.04	0.05-0.16
Co-activation (%Triceps/Biceps rmsEMG)	23.9	9.8	12.8-40.5	27.0	13.4	13.1-53.9	25.5	12.1	12.3-48.7	27.6	12.6	12.1-55.8	26.8	12.9	12.5-54.5	26.3	14.2	11.8-55.9
%Coactivation per Newton Force	0.08	0.03	0.03-0.13	0.09	0.04	0.04-0.16	0.08	0.04	0.03-0.15	0.10	0.04	0.04-0.18	0.09	0.04	0.04-0.17	0.09	0.05	0.04-0.18

Table 3. Corticospinal excitability (CSE) data collapsed across trials (trial 1 to trial 6), for external and internal contraction conditions. Data presented as mean (M), standard deviation (SD), and range. M_{max}, maximal compound motor unit action potential; MEP, motor evoked potential; CMEP, cervicomedullary MEP.

Condition	**External Cued Contraction**			**Internal Cued Contraction**		
	M	SD	Range	M	SD	Range
M_{max} mplitude (mV)	7.16	4.97	1.88-18.53	7.43	4.75	1.63-17.47
MEP Amplitude (Ratio of M_{max})	1.49	1.08	0.49-3.49	1.32	0.92	0.48-2.95
CMEP Amplitude (Ratio of M_{max})	1.07	0.79	0.35-2.73	1.00	0.98	0.36-3.42
MEP/CMEP Ratio	1.64	0.84	0.92-3.64	1.97	1.65	0.82-6.44

3.12 Figures Legend

Figure 1. Participants were positioned up right in an elevated chair with shoulders at 0 degrees and elbows at 90 degrees. Each participant completed two experimental sessions which were randomized. Within each session, participants completed two blocks of three externally cued contractions and two blocks of internally cued contractions which were also randomized. Maximal voluntary contractions were held for 5 seconds, beginning and ending at 2 and 7 seconds respectively, and during the stimulation session a TMS and TMES pulse was randomly delivered at 3.5 and 5.0 second marks with an M-Wave delivered each time at the 6.5 second mark.

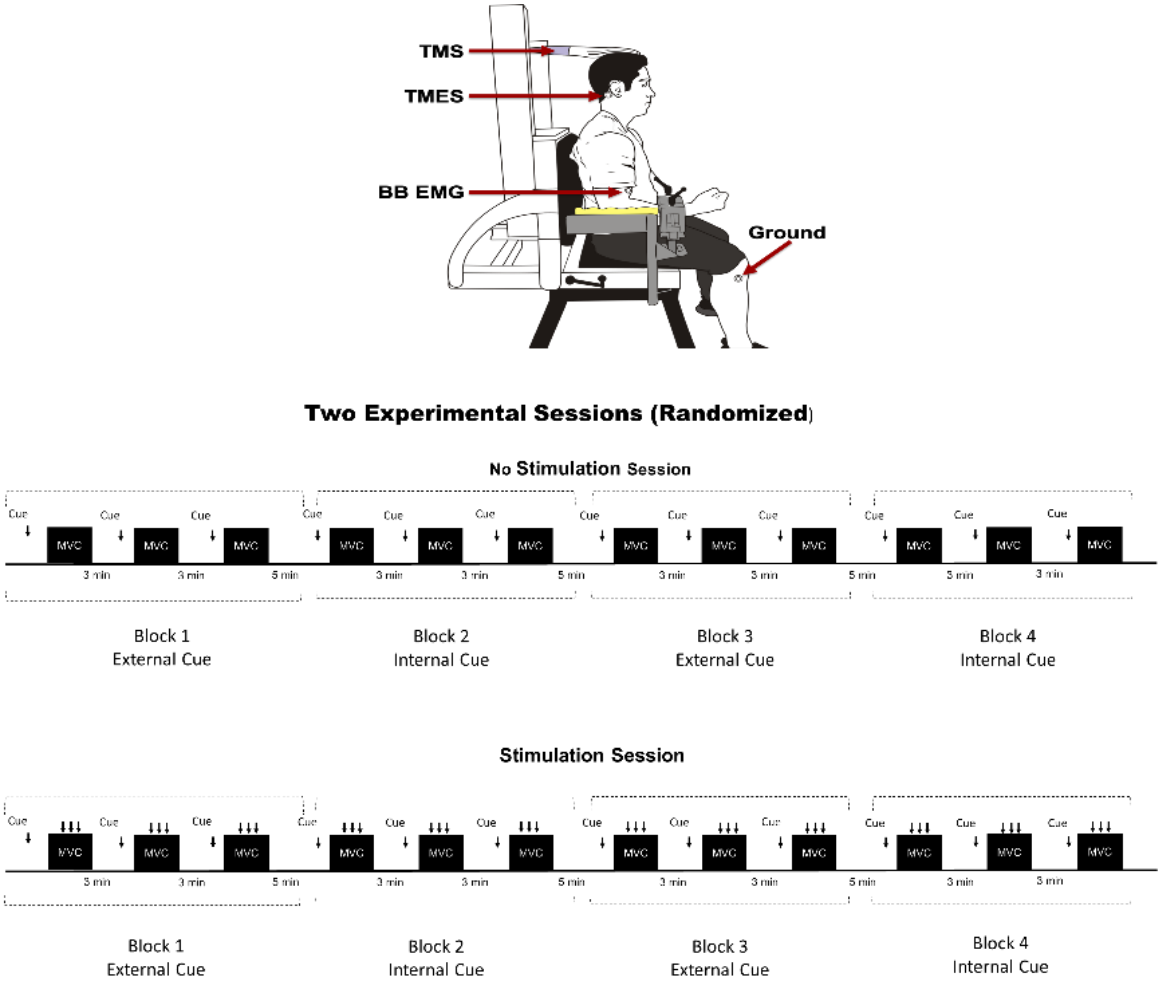
Figure 2. Peak force values for maximum voluntary isometric contractions (MVCs), measured in Newtons (N). Smaller points represent individual participant data, larger points represent mean, and error bars represent one standard deviation. (A) Peak force values for external versus internal contraction conditions, collapsed across all trials (trial 1 to trial 6) and sessions (no-stimulation, stimulation), demonstrating the significant main effect of CONDITION. (B) Peak force values for no-stimulation versus stimulation session, collapsed across all trials (trial 1 to trial 6) and conditions (external contraction, internal contraction), signifying the significant main effect of SESSION. *, statistically significant at $p \leq .05$.

Figure 3. Data expressing the relationship between muscle co-activation and MVC peak force. In panels A-B smaller points represent individual participant data, larger points represent mean, and error bars represent one standard deviation. In panels D-E points represent individual data. (A) Muscle co-activation values for external and internal contraction conditions, collapsed across trials (trial 1 to trial 6) and sessions (no-stimulation, stimulation), demonstrating the significant main effect of CONDITION. (B) Percentage of muscle co-activation/MVC peak force (co-activation per Newton force production) for external versus internal contraction

conditions, collapsed across session (no-stimulation, stimulation), illustrating the significant main effect of CONDITION. (C-D) Scatterplots demonstrating relationship between changes in MVC peak force and triceps brachii/biceps brachii co-activation across external and internal contraction conditions during the (C) no-stimulation and (D) stimulation sessions. *, statistically significant at $p \leq .05$.

3.13 Figures

Figure 1



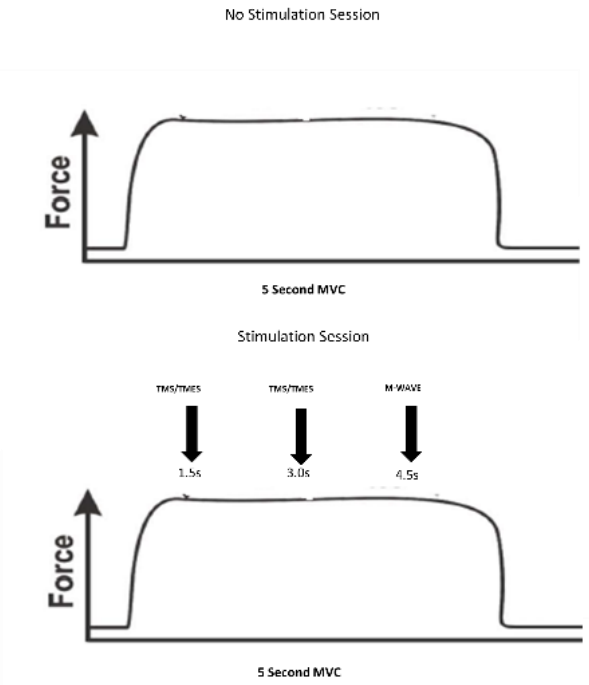


Figure 2

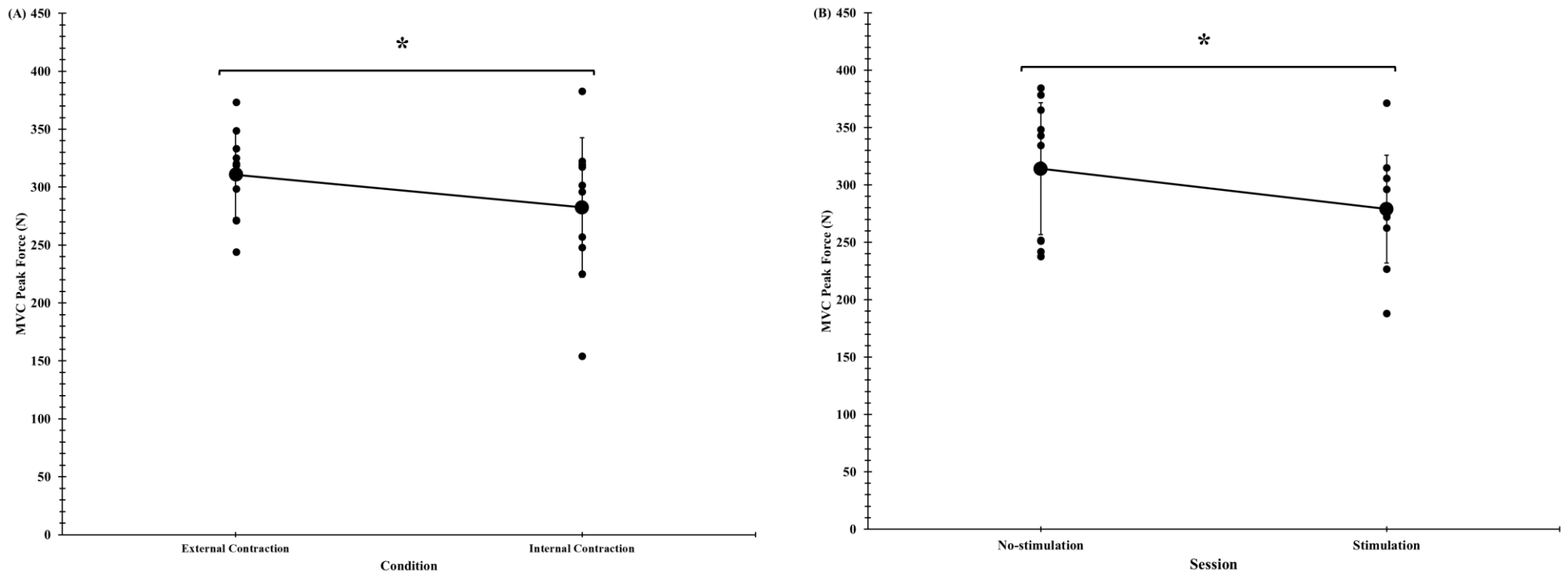
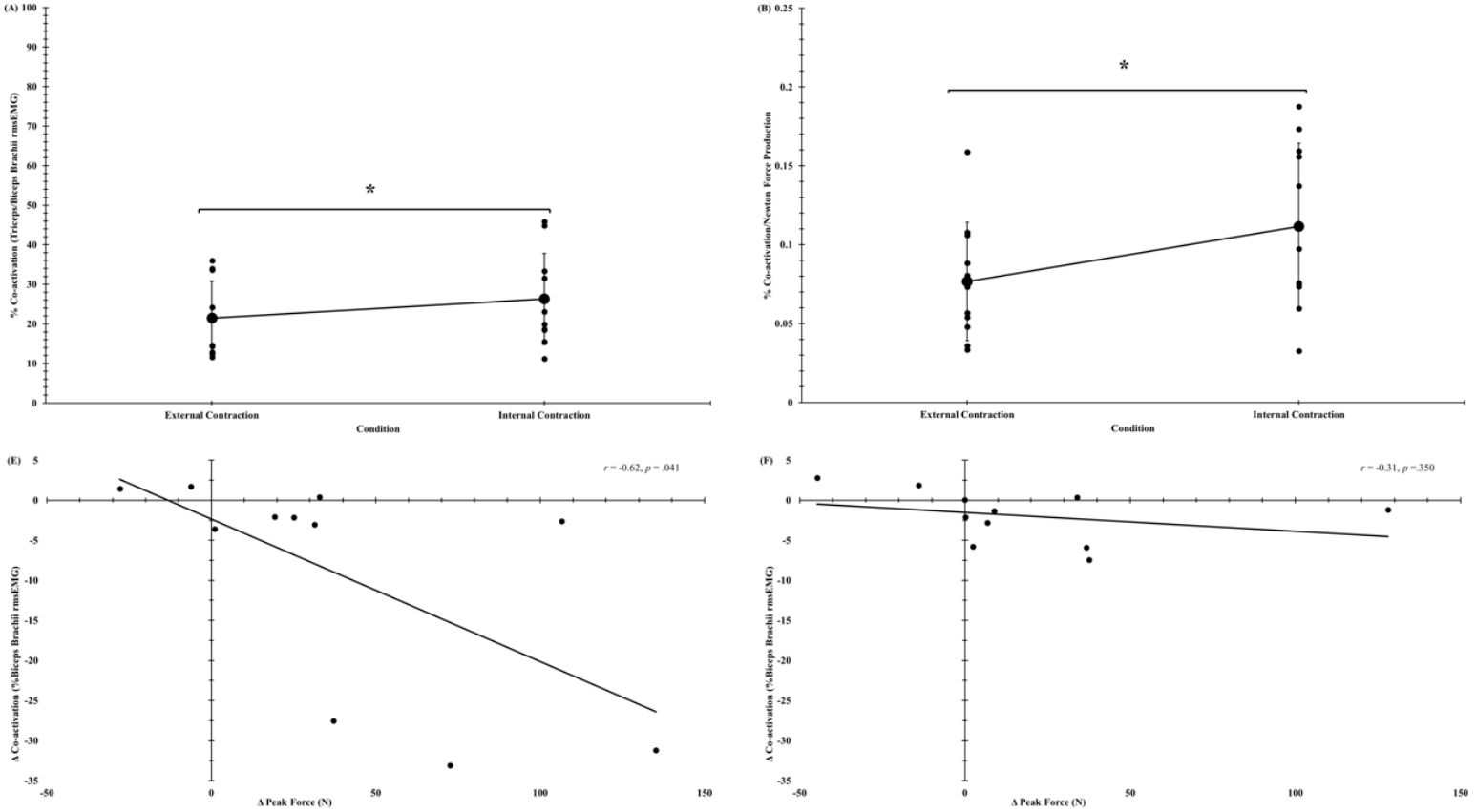

(A)
MVC Peak Force (N)
450
400
350
300
250
200
150
100
50
0
*
External Contraction
Internal Contraction
Condition
(B)
MVC Peak Force (N)
450
400
350
300
250
200
150
100
50
0
*
No-stimulation
Stimulation
Session

Figure 3


(A)
% Co-activation (Triceps/Biceps Brachii rmsEMG)
*
External Contraction
Internal Contraction
Condition
(B)
% Co-activation/Newton Force Production
*
External Contraction
Internal Contraction
Condition
(E)
r = -0.62, p = .041
Δ Co-activation (%Biceps Brachii rmsEMG)
Δ Peak Force (N)
(F)
r = -0.31, p =.350
Δ Co-activation (%Biceps Brachii rmsEMG)
Δ Peak Force (N)

Appendix A: TMS Safety Checklist

The safety of TMS continues to be supported by recent meta-analyses of published research (i.e. Machii et al., 2006; Loo et al., 2008; Janicak et al., 2008; Rossi et al., 2009). To ensure participant's safety, they were required to complete the following questionnaire prior to receiving TMS.

Magnetic Stimulation safety checklist

Please answer the following questions by circling **YES or NO.**

1. Do you suffer from epilepsy, or have you ever had an epileptic seizure? **YES/NO**

2. Does anyone in your family suffer from epilepsy? **YES/NO**

3. Do you have any metal implant(s) in any part of your body or head? (Excluding tooth fillings) **YES/NO**

4. Do you have an implanted medication pump? **YES/NO**

5. Do you wear a pacemaker? **YES/NO**

6. Do you suffer any form of heart disease? **YES/NO**

7. Do you suffer from reoccurring headaches? **YES/NO**

8. Have you ever had a skull fracture or serious head injury? **YES/NO**

9. Have you ever had any head surgery? **YES/NO**

10. Are you pregnant? **YES/NO**

11. Do you take any medication? **YES/NO**

    a. Note if taking medication, check list for contraindicated medication on next page.

12. Do you suffer from any known neurological or medical conditions? **YES/NO**

    Comments:_____

_____

_____

_____

Name: _____

Signature: _____

Date: _____

Medications contraindicated with magnetic stimulation:

1) Tricyclic antidepressants

2) Neuroleptic or Antipsychotic drugs

A) Typical antipsychotics

• Phenothiazines: • Thioxanthenes:

- Chlorpromazine (Thorazine) ○ Chlorprothixene
- Fluphenazine (Prolixin) ○ Flupenthixol (Depixol and Fluanxol)
- Perphenazine (Trilafon) ○ Thiothixene (Navane)
- Prochlorperazine (Compazine) ○ Zuclopenthixol (Clopixol and Acuphase)
- Thioridazine (Mellaril) • Butyrophenones:
- Trifluoperazine (Stelazine) ○ Haloperidol (Haldol)
- Mesoridazine ○ Droperidol
- Promazine ○ Pimozide (Orap)
- Trifluprομazine (Vesprin) ○ Melperone
- Levomepromazine (Nozinan)

B) Atypical antipsychotics

- Clozapine (Clozaril)
- Olanzapine (Zyprexa)
- Risperidone (Risperdal)
- Quetiapine (Seroquel)
- Ziprasidone (Geodon)
- Amisulpride (Solian)
- Paliperidone (Invega)

Name	Brand name
amitriptyline (& butriptyline)	Elavil, Endep, Tryptanol, Trepiline
desipramine	Norpramin, Pertofrane
dothiepin hydrochloride	Prothiaden, Thaden
imipramine (& dibenzepin)	Tofranil
iprindole	-
nortriptyline	Pamelor
opipramol	Opipramol-neuraxpharm, Insidon
protriptyline	Vivactil
trimipramine	Surmontil
amoxapine	Asendin, Asendis, Defanyl, Demolox, Moxadil
doxepin	Adapin, Sinequan
clomipramine	Anafranil

C) Dopamine partial agonists:

Aripiprazole (Abilify)

D) Others

Symbyax -A combination of olanzapine and fluoxetine used in the treatment of bipolar depression. Tetrabenazine (Nitoman in Canada and Xenazine in New Zealand and some parts of Europe Cannabidiol One of the main psychoactive components of cannabis.

Appendix B: Free and Informed Consent Form

Informed Consent Form

Title: Understanding the Neurophysiological mechanisms underlying changes in human motor performance with augmented feedback.

Researcher(s): Mr. Shawn Wiseman

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Mr. Behzad Lahouti

Masters Student

School of Human Kinetics and Recreation

Memorial University

Email: Blahouti@mun.ca

You are invited to take part in a research project entitled *"*Understanding the Neurophysiological mechanisms underlying changes in human motor performance with augmented feedback."

This form is part of the process of informed consent. It should give you the basic idea of what the research is about and what your participation will involve. **It also describes your right to withdraw from the study at any time**. In order to decide whether you wish to participate in this research study, you should understand enough about its risks and benefits to be able to make an informed decision. This is the informed consent process. Take time to read this carefully and to understand the information given to you. Please contact the lead researcher, Mr. Shawn Wiseman, if you have any questions about the study or would like more information before you consent.

It is entirely up to you to decide whether to take part in this research study. If you choose not to take part in this research or if you decide to withdraw from the research once it has started, there will be no negative consequences for you, now or in the future.

Introduction:

This research is being conducted by Mr. Shawn Wiseman, a Master's Student in the School of Human Kinetics and Recreation at Memorial University of Newfoundland. The corticospinal tract is the primary spinal tract involved in human movement. Increases in the excitability of the spinal tract has been shown to be directly related with muscular force production. There are many factors that could lead to increases in corticospinal excitability. One of these factors is performance feedback.

Purpose of study:

The purpose of this study is to examine changes in corticospinal excitability with different forms of augmented feedback.

What you will do in this study:

We will use a combination of magnetic and electrical stimulation techniques to assess corticospinal excitability after tonic contractions. You will be asked to attend two sessions totaling an estimated two hours. Both sessions will be identical except we will not be using the mentioned stimulation techniques in the second session.

GENERAL DESCRIPTION OF PROCEDURES

The first session will comprise of you performing 12 total maximum voluntary bicep contractions. You will perform these in sets of 3 with 1 minute rest given per contraction and 5 minutes rest between set. Force output will be measured for each contraction. During the contractions a combination of magnetic and electrical simulation will be used to assess the excitability of the corticospinal tract. The second session will follow the same procedure as the first but no stimulations will be used.

SPECIFIC DESCRIPTION OF STIMULATION CONDITIONS

The brain stimulation technique that we will use is referred to as transcranial magnetic stimulation and will occur over 2 different locations of the brain and spinal cord. These stimuli are not painful in any way and are designed for human research. They are completely safe and have been used extensively by Drs. Power and Button.

Skin preparation will be undertaken for all electrodes, including shaving hair off the desired area followed by cleansing with an isopropyl alcohol swab. The electrodes do contain an adhesive that allows them to stick to the skin. These impulses will be increased to obtain a maximal response and then kept consistent at 20% above that maximum.

I will gladly answer any questions or concerns you may have regarding any portion of the study if the procedures are not completely clear.

Length of time:

Participation in this study will require you to come to a lab located in the School of Human Kinetics and Recreation at Memorial for two sessions of about an hour each.

Withdrawal from the study:

You will be free to withdraw from this study at any point up until the end of the testing session. To do so you simply need to inform the researchers and you will be free to leave. Any data collected up to that point will not be used in the study and will be destroyed. In addition, you may request for the removal of your data at any time up to one year later. If you are a student, your participation in and/or

withdrawal from this study will not in any way, now or ever, negatively impact either your grade in a course, performance in a lab, reference letter recommendations and/or thesis evaluation.

Possible benefits:

The benefit of participating in his study is that you will learn about the functioning of your nervous system. You will also be aiding our basic understanding of how the nervous system responds to muscle soreness. This investigation is important because until we understand the basic mechanisms controlling nervous system excitability we cannot fully understand mechanisms of impaired motor function and potential mechanisms to improve function may have positive impact in rehabilitation after injury. The findings of this research may be used for guiding rehabilitation strategies and exercise interventions for clinical and non-clinical populations.

Possible risks:

There are several minor risks associated with participating in this study:

1) You will have electrodes placed on the front and back of your arm. These electrodes have an adhesive that has a tendency to cause redness and minor irritation of the skin. This mark is temporary (usually fades within 1-2 days) and is not generally associated with any discomfort or itching.

2) The electrical stimulations will cause twitching of the muscles and mild discomfort, but is not painful. The sensation has been described as if you flicked your arm muscles firmly with a finger. The sensation will be very brief (less than a second) and will in no way result in any harm to either muscles or skin.

3) Transcranial magnetic stimulation is used to assess brain excitability and is applied at the surface of the top of the skull and just behind the ear. This will cause activation of the brain resulting in small muscle contraction (some individuals do not experience any discomfort).

4) Post experiment muscle soreness, simlilar to that following an acute bout of exercise will be experienced by some participants.

5) Psychological risks such as nervousness or anxiety may be experienced due to the various stimulation techniques used (top of head and above collarbone). You will experience each of these stimulations on the first day of testing (familiarization trial) to deterimine whether you are comfortable to participate in the study. You will also be given the opportunity to ask any questions you have.

Each investigator is first aid certified and has access to emergency services in the unlikely event that you require medical assistance. The following address is for the University Counselling Centre should you feel the need to avail of their services.

University Counselling Centre

5th Floor University Centre, UC-5000

Memorial University of Newfoundland

St. John's NL A1C 5S7

Tel: (709) 864-8874

Fax: (709) 864-3011

Director/Associate Professor: Peter Cornish, Ph.D.

NOTE: The stimulators used for the experiment are designed for human research, are completely safe and have been used extensively by Dr. Button.

Confidentiality:

The ethical duty of confidentiality includes safeguarding participants' identities, personal information, and data from unauthorized access, use, or disclosure.

Your identity will be guarded by maintaining data in a confidential manner and in protecting anonymity in the presentation of results (see below) .

Results of this study will be reported in written (scientific article) and spoken (local and national conferences and lectures) forms. For both forms of communication only group average data will be presented. In cases where individual data needs to be communicated it will be done in such a manner that you confidentiality will be protected (i.e. data will be presented as coming from a representative subject).

Anonymity:

Anonymity refers to protecting participants' identifying characteristics, such as name or description of physical appearance. Only the researchers will be aware of your participation. In addition to Drs. Duane Button and Kevin Power, the other researchers, all masters students, required to assist with data collection are:

1. Brandon Collins
2. Lucas Stefanelli
3. Behzad Lahouti

Every reasonable effort will be made to ensure anonymity; and you will not be identified in publications without explicit permission.

Recording of Data:

There will be no video or audio or photographic recordings made during testing.

Storage of Data:

The only individuals who will access to this data are the researchers involved in this study. Data will be retained for a minimum of five years, as per Memorial University policy on Integrity in Scholarly Research after which time it will be destroyed. All data will be kept in a secured location: paper based records will be kept in a locked cabinet in the office of Dr. Button while computer based records will be stored on a password protected computer in the office of Dr. Button. The data collected as a result of your participation can be withdrawn from the study at your request up until the point at which the results of the study have been accepted for publication (~1 year post study).

Reporting of Results:

Results of this study will be reported in written (scientific article) and spoken (local and national conferences and lectures) formats. All results will be presented as group averages. In cases where individual data needs to be communicated it will be done in such a manner that your confidentiality will be protected (i.e. data will be presented as coming from a representative participant). The master's thesis will be publically available at the QEII Library upon publication.

Sharing of Results with Participants:

Following completion of this study please feel free to ask any specific questions you may have about the activities you were just asked to partake in. Also if you wish to receive a brief summary of the results then please indicate this when asked at the end of the form.

Questions:

You are welcome to ask questions at any time before, during, or after your participation in this research. If you would like more information about this study, please contact: Mr. Shawn Wiseman (ljs100@mun.ca) of Dr. Duane Button (dbutton@mun.ca).

The proposal for this research has been reviewed by the Interdisciplinary Committee on Ethics in Human Research and found to be in compliance with Memorial University's ethics policy. If you have ethical concerns about the research, such as the way you have been treated or your rights as a participant, you may contact the Chairperson of the ICEHR at icehr@mun.ca or by telephone at 709-864-2861.

Consent:

Your signature on this form means that:

- You have read the information about the research.
- You have been able to ask questions about this study.
- You are satisfied with the answers to all your questions.
- You understand what the study is about and what you will be doing.
- You understand that you are free to withdraw participation in the study without having to give a reason, and that doing so will not affect you now or in the future.
- You understand that if you choose to end participation **during** data collection, any data collected from you up to that point will destroyed.
- You understand that if you choose to withdraw **after** data collection has ended, your data can be removed from the study up to one year after the conclusion of data collection.

By signing this form, you do not give up your legal rights and do not release the researchers from their professional responsibilities.

Your signature confirms:

☐ I have read what this study is about and understood the risks and benefits. I have had adequate time to think about this and had the opportunity to ask questions and my questions have been answered.

☐ I agree to participate in the research project understanding the risks and contributions of my participation, that my participation is voluntary, and that I may end my participation.

☐ A copy of this Informed Consent Form has been given to me for my records.

☐ I would like to receive a summary of the results of the study.

_____ _____

Signature of participant Date

Researcher's Signature:

I have explained this study to the best of my ability. I invited questions and gave answers. I believe that the participant fully understands what is involved in being in the study, any potential risks of the study and that he or she has freely chosen to be in the study.

_____ _____

Signature of Principal Investigator Date