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The effects of muscle belly vibration at varying muscle lengths on corticospinal  
excitability: A TMS Study

by

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## **Abstract**

The application of vibration to a muscle is known to activate its muscle spindle receptors; the same receptors that are activated when the muscle is lengthened. Both muscle length and vibration are known to affect corticospinal excitability. However, their combined effects on corticospinal excitability have not been established. Thus, single pulse transcranial magnetic stimulation was applied before and after first dorsal interosseous muscle vibration at four different muscle lengths. Post-vibration motor evoked potentials were significantly higher and tonic vibration reflexes were elicited at the longest muscle length condition. As both muscle length and vibration are known to increase muscle spindle activation, the current results suggest increased corticospinal excitability when these conditions are combined may be due to increased spindle activity. More research is needed to further understand the mechanisms within the corticospinal pathway.

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## List of Symbols, Abbreviations and Nomenclature

Symbol	Definition
A/D	Analogue to Digital
Ag	Silver
AgCl	Silver Chloride
AVR	Antagonistic Vibratory Response
CNS	Central Nervous System
CT	Computerized Tomography
DEXA	Dual Energy X-Ray Absorptiometry
EMG	Electromyography
FDI	First Dorsal Interosseous
g	Acceleration measured in Gravity
Hz	Hertz
m	Metres
MCP	Metacarpophalangeal
MEP	Motor Evoked Potential
mm	Millimetres
mV	Millivolts
M1	Short Latency Stretch Reflex
M2	Long Latency Stretch Reflex
n	Sample Size
N	Newtons
PET	Positron Emission Tomography
rMT	Resting Motor Threshold
s	Seconds
SD	Standard Deviation
TMS	Transcranial Magnetic Stimulation
TVR	Tonic Vibration Reflex
WBV	Whole Body Vibration
$Z_{\alpha}$	Z-score corresponding to desired significance level
$Z_{\beta}$	Z-score corresponding to desired power level
$\Delta$	Difference between means
$\mu\text{V}$	Microvolts

## CHAPTER ONE: INTRODUCTION AND REVIEW OF LITERATURE

### *1.1 General Introduction*

The use of muscle vibration is becoming increasingly popular in the fields of exercise physiology, as well as rehabilitative and preventative medicine (Rittweger, 2010). There are generally two vibration protocols used in these fields, either whole body vibration (WBV) or local muscle vibration. During WBV, the subject stands on an oscillating platform and the mechanical oscillations from the platform are transferred throughout a series of muscles, joints, and spring-like connective tissues (Rittweger, 2010). Acute bouts of WBV improve muscular power in athletes (Cochrane & Stannard, 2005; Cormie, Deane, Triplett, & McBride, 2006) and strength performance over time (Rønnestad, 2004). Local muscle vibration involves an oscillating disc or probe placed upon the muscle belly or tendon, such that only one muscle is vibrated. Local muscle vibration is known to improve dexterity in hemi-paretic stroke patients (Liepert & Binder, 2010) and sensorimotor organization in individuals with dystonia (Rosenkranz et al., 2008). Given that these disorders are known to affect the central nervous system (CNS), these improvements observed with vibration suggest that they are neurally mediated. Furthermore, researchers have attributed the vibration-induced performance enhancements in the exercise field to neural adaptation as well (Rittweger, 2010). Despite its uses in exercise prescription and medicine, there are also known negative effects of vibration such as “vibration syndrome” in which symptoms include pain, numbness and tingling, as well as loss of strength and dexterity due to the development of nerve damage (Rosen, Bjorkman, & Lundborg, 2011). This suggests a need to better understand the

influences of vibration on the system. Much of the existing research on muscle vibration has been directed towards understanding the underlying neurophysiology of its effects at the level of the muscle, and at the numerous levels along the pathways leading to the level of the cerebral cortex.

### ***1.2 Vibration and muscle spindle activity***

Vibration, applied to either the muscle belly or its tendon, is arguably the most potent mechanical stimulus for activating primary (type Ia) muscle spindle receptors i.e., receptors found in the muscles that detect muscle length and changes in muscle length (Smith & Brouwer, 2005). Muscle spindles thus provide information to the central nervous system regarding their respective muscles and joints' position within space, termed "proprioception". The repetitive oscillation placed upon the muscle belly or tendon produces a series of rapid muscle lengthening and shortening cycles which continuously activates its muscle spindle primary endings (type Ia fibres) sending afferent messages to the spinal cord and supraspinal centers (Roll, Vedel, & Ribot, 1989). In fact, microneurographic recordings have shown that the discharge rates of most muscle spindle Ia afferents mimic the frequency of the vibratory stimuli up to approximately 100 Hz (Roll, et al., 1989), which suggests the influence of vibration on spindle activity. Furthermore, there is evidence of increased group II afferent receptor activity during muscle vibration, however the group II afferents do not fire synchronously with the vibration frequency consistently above 30 Hz (Fallon & Macefield, 2007; Roll & Vedel, 1982). There is evidence that vibration of a muscle in a stretched position activates muscle spindle receptors at a higher rate than vibration of a muscle in a neutral position

(Burke, Hagbarth, Lofstedt, & Wallin, 1976). This suggests that muscle spindles, whose primary action is to monitor muscle length changes, are still sensitive to muscle length while being activated by vibration.

### ***1.3 Muscle length and reflex activity***

It has long been understood that when stretch of a skeletal muscle excites its muscle spindle primary endings, it evokes a tonic contraction in that same muscle. This is known as the classic stretch reflex (Liddell & Sherrington, 1924). Spindle endings also act on inhibitory interneurons that inhibit the antagonists of the stretched muscle to better facilitate the movement brought on by the stretch reflex. In humans, there is evidence of two distinct components to a stretch reflex: the short latency (M1) stretch reflex which is considered spinal in origin, and the long latency (M2) stretch reflex that has a more complex and debated origin (Matthews, 1991). It has been suggested that the extra delay characterizing the long latency reflex response is a result of afferent information traveling to the somatosensory cortex and being relayed to the motor cortex which in turn mediates the M2 response (see Figure 1.1). Thus, the long latency portion of the stretch reflex has been termed the transcortical reflex pathway (Matthews, 1991). Although it is suggested to be cortically mediated, the latency of the M2 reflex response is nonetheless shorter than that of the voluntary reaction time (Pearson, 2000), indicating that it does not reflect a voluntary action and is still a reflex.

Palmer and Ashby (1992) provided direct evidence to support the link between the transcortical pathway and the observed M2 by examining the excitability of the motor cortex during stretch of the flexor pollicis longus muscle. They found that stimulating the

cortex using transcranial magnetic stimulation (TMS; see below) at a time corresponding to when the M2 would be generated cortically produced a larger facilitation of the M2 response in the muscle than the sum of cortical stimulation or muscle stretch alone, suggesting a cortical influence to the long latency reflex. This phenomenon did not occur when cortical stimulation was superimposed on the short latency reflex response, suggesting the short latency reflex, M1, was not cortically-mediated (Palmer & Ashby, 1992). Through research such as this, it has become clear that muscle spindle activity influences more than just spinal pathways.

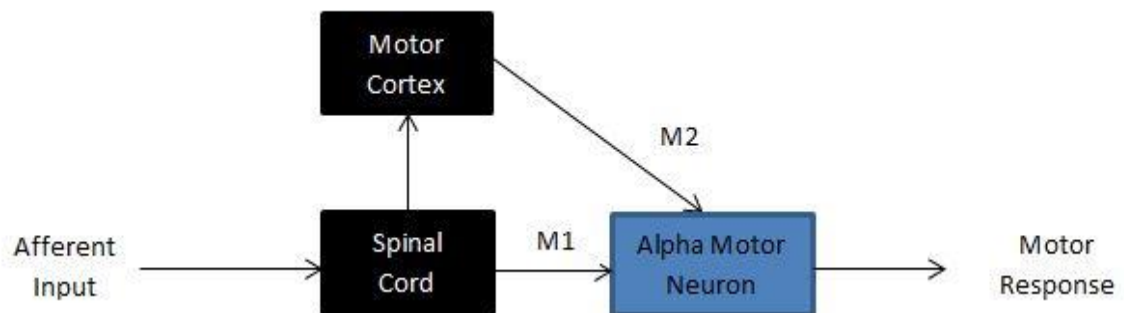


Figure 1.1. Schematic depiction of the M1 and M2 reflex pathways.

#### ***1.4 Vibration and reflex activity***

There is evidence that suggests that adding vibratory stimuli to a muscle will influence its stretch reflex characteristics. For example, prolonged vibration has shown to depress muscle spindle discharge in leg muscles, suggesting that a habituation effect occurs in the muscle spindle which leads to a decrease in stretch reflex amplitude when elicited during muscle vibration (Bove, Nardone, & Schieppati, 2003). In fact, resting spindle discharge rate has been shown to decrease following as little as 30 s vibration of

lower leg muscles (Ribot-Ciscar, Rossi-Durand, & Roll, 1998). H-Reflex (an electrically induced reflex analogous to the mechanically induced spinal stretch reflex; Palmieri, Ingersoll, & Hoffman, 2004) amplitude is widely known to be suppressed or diminished during or immediately following vibration, due to decreased activation of the Ia pathway (Ekblom & Thorstensson, 2011; Hagbarth & Eklund, 1966). However, there is evidence that these habituation effects are not as robust in upper extremity muscles. Shinohara et al (2005) found that 30 minutes of vibration of the first dorsal interosseous muscle, an intrinsic hand muscle, increased stretch reflex amplitude and motor unit discharge rate following vibration. These authors attribute the increases in stretch reflex amplitude to increases in muscle spindle activity rather than adaptations in the Ia afferent pathway thought to be responsible for decreasing the H-reflex, as the H-reflex does not involve the muscle spindle receptors (Shinohara, et al., 2005). The above findings suggest that there is a contrast between the behaviour of upper and lower limb muscle afferent activity.

Similar to the effects of muscle stretch, high frequency muscle vibration (> 20 Hz) at a peak to peak amplitude of approximately 1.0 mm can result in tonic contraction in the vibrated muscle, termed the tonic vibration reflex (TVR, Eklund and Hagbarth, 1966). The TVR is generally referred to as an example of a myotatic stretch reflex (Matthews, 1966b). Similar to the stretch reflex, the TVR also results in inhibition of the vibrated muscle's antagonists (Eklund & Hagbarth, 1966) and the repeated tendon taps of vibration can elicit both a short latency and a long latency response in the vibrated muscle (Romaine, Vedel, Azulay, & Pagni, 1991). This suggests, as it is suggested with the classic stretch reflex, that the TVR is at least partly cortically mediated.

Evidence of a TVR suggests that vibration excites alpha motor neurons.

Synchronization between motor unit discharge rates and the frequencies of muscle vibration have been observed up to vibration frequencies of ~150 Hz (Martin & Park, 1997). As both vibration and muscle length are known to affect muscle spindle activity, researchers have examined the influence of vibration of a muscle at varying muscle length on force output and electromyographic (EMG) response. For instance, Eklund (1971) discovered that the TVR elicited by vibrating the elbow flexors (as measured by torque generated by the biceps) diminished when the biceps brachii muscle was in a shortened position, as opposed to a lengthened position, and increased when it was in a lengthened position versus a shortened position. This suggests that muscle length can have a large effect on the ability of vibration to elicit a TVR (Eklund & Hagbarth, 1966). While the aforementioned results all suggest that muscle length plays a role in the amount of neural drive to a muscle during vibration, the origin of the neural drive, i.e. the extent to which it originates from spinal reflex or descending drive, remains unclear.

### ***1.5 Vibration and cortical activity***

Muscle vibration has been shown to increase activity in the somatosensory areas of the cortex as measured by electroencephalography (Munte et al., 1996) and positron emission tomography (PET; Radovanovic et al, 2002). It has also been shown that biceps tendon vibration in monkeys elicited a concurrent increase in excitatory motor cortical cell response (Fourment, Chenneville, Belhaj-Saif, & Maton, 1996) providing a direct connection between muscle vibration and motor cortical activity (Fourment, et al., 1996).

There is evidence to suggest the link between vibration and cortical activity is due to the influence of vibration on muscle spindle activity. Specifically, vibration induces proprioceptive illusions of muscle lengthening in the vibrated muscle (Goodwin, McCloskey, & Matthews, 1972; Izumizaki, Tsuge, Akai, Proske, & Homma, 2010). Researchers use psychophysical techniques to quantify individuals' subjective experience (perception) of movement by, for instance, administering questionnaires post-vibration (Steyvers, Levin, Van Baelen, & Swinnen, 2003) and requiring non-verbal reporting of the experience such as pressing buttons on a computer keyboard (Seizova-Cajic & Azzi, 2010) to indicate velocity or direction of the perceived movement. More typically though, study participants are asked to reproduce the illusory limb position or movement experienced in their vibrated limb with their non-vibrated limb, or with the vibrated limb post-vibration (Goodwin, et al., 1972; Kito, Hashimoto, Yoneda, Katamoto, & Naito, 2006; Rogers, Bendrups, & Lewis, 1985), to provide a quantitative measure of the illusion.

Differences in muscle length are known to affect the strength, vividness, and duration of proprioceptive illusions. Izumizake et al (2010) found that the perceived speed of the movement of the vibrated arm was influenced by moving the contralateral reference arm into either flexion (reduced speed) or extension (increased speed) in a position matching task. This suggests that the incidence or strength of illusions may be influenced by the initial resting length of the vibrated muscle. Roll and Vedel (1982) concluded that a higher frequency of vibration elicits a higher velocity of perceived movement up to 80 Hz, beyond which perceived movement velocity decreases. These findings are consistent with the findings that muscle spindle afferents are activated

synchronously with increasing vibration frequency (Roll, et al., 1989), as muscle spindles are known to encode movement to the central nervous system. In addition to static position, vibration has also been found to influence proprioception during actual movement, such that vibration of a muscle that is lengthening increases the perceptual speed of its movement (Sittig, Denier van der Gon, & Gielen, 1987). In many cases of proprioceptive illusions, there is an additional illusory aftereffect whereby the limb is felt to return to its normal resting position following vibration (Kito, et al., 2006). As previously mentioned, it was found that 30 s of vibration decreased the firing rate of muscle spindle primary endings for approximately 8-10 s following the cessation of vibration (Ribot-Ciscar, et al., 1998). These results suggest that the illusory aftereffect is mediated by spindle activity, like the vibratory illusion, as the reduction or cessation of muscle spindle activation typically encodes muscle shortening in the central nervous system.

Other sensory information can also influence the parameters of the proprioceptive illusions experienced by individuals. Illusions are significantly diminished when the subject is able to see the vibrated arm during the vibration, as the vision of the arm being stationary will conflict with the proprioceptive signal (Seizova-Cajic & Azzi, 2011). It has also been found that the illusory aftereffect is decreased in intensity if the subject is distracted versus when the subject is focused on the perceived movement (Seizova-Cajic & Azzi, 2010). There is evidence that other types of afferent input can also influence the strength of illusions. For example, localized skin stretch over a metacarpophalangeal joint akin to the stretch that would occur during finger flexion has shown to elicit similar illusions, and this phenomenon is stronger when applied in concert with vibration

(Collins, Refshauge, & Gandevia, 2000). Furthermore, stimulation between tactile and proprioceptive (i.e. muscle spindle activation) modalities that are designed to elicit the same illusion will induce stronger and faster illusions, however combined stimulation designed to elicit conflicting illusions are processed depending on the intensities of the stimuli. When two types of conflicting stimuli are given at equal intensities, the participant will favour the tactile information at low and medium intensities, however the participant will favour proprioceptive information at high intensities (Blanchard, Roll, Roll, & Kavounoudias, 2011). These results all suggest that the intensity of proprioceptive illusions is dependent on other factors that could potentially influence the ability of the CNS to integrate multiple sensory signals.

During proprioceptive illusions, an increase in muscle activity can occur in the muscle antagonistic to the vibrated muscle, termed the “Antagonistic Vibratory Response” (AVR; Gilhodes, Roll, and Tardy-Gervet, 1986), or the “inverted TVR” (Feldman & Latash, 1982a) which is in direct contrast to the antagonist inhibitory muscle response during a TVR. The AVR appears to be only possible when the individual is experiencing proprioceptive illusion (Calvin-Figuere, Romaguere, Gilhodes, & Roll, 1999). However, the AVR does not always occur during proprioceptive illusions (Kito, et al., 2006). Interestingly, it has been shown that when an AVR (and movement illusion) is observed with muscle vibration, an auditory or visual distractor will actually reverse the muscle activity to the vibrated muscle, simultaneously developing a TVR and eliminating the movement illusion (Feldman & Latash, 1982b). Furthermore, upon the cessation of the distraction, the AVR resumes while the TVR vanishes (Feldman & Latash, 1982b). This suggests that the AVR is cortically mediated, as it disappears when perceptual focus

is taken off of the vibration stimuli, giving way to a lower level mechanism (i.e. TVR). Through PET, Naito and Ehrsson (2001) found increased brain activity during proprioceptive illusion in the primary sensory and motor cortices, supplementary motor cortex, and cingulate motor areas contralateral to the limb being vibrated, which are the same brain areas active during voluntary movement. This finding also supports the idea that the AVR is caused by the proprioceptive illusion and is cortically mediated.

### ***1.6 Changes in corticospinal excitability with muscle length changes***

Transcranial magnetic stimulation (TMS) is a tool that can be used to directly measure changes that occur in the excitability of the corticospinal tract. Use of TMS involves strategically placing an electromagnetic coil upon the scalp directly above the area of interest in the motor cortex. This coil produces an electromagnetic pulse inducing an electrical current in any nearby conductors, which in this case are cortical tissue (Davey, 2008). This pulse excites an area in the motor cortex eliciting a muscular response, i.e., a motor evoked potential (MEP), recorded by EMG of the muscle(s) innervated by that particular area of the motor cortex. Changes in MEP amplitude prior to and following an intervention protocol provide an indication of the changes within the corticospinal pathway occurring as a result of an intervention (Rosenkranz, Pesenti, Paulus, & Tergau, 2003; Rösler & Magistris, 2008).

There are a variety of TMS protocols that provide different information about the changes occurring in the CNS. For instance, single pulse TMS protocols have been used to measure corticospinal excitability and, when paired with measures of reflex activity, for cortical mapping (Muellbacher, Ziemann, Boroojerdi, & Hallett, 2000). This protocol

has been used to examine the effects of changes in muscle length on corticospinal excitability. Corticospinal excitability has been shown to increase when muscles are passively shortened (Coxon, Stinear, & Byblow, 2005) and decrease when passively lengthened (Coxon, et al., 2005; Edwards, Thickbroom, Byrnes, Ghosh, & Mastaglia, 2002). Similarly, corticospinal excitability is known to be inhibited when the muscle is at a static lengthened position, and facilitated when the muscle is at a static shortened position (Lewis, Byblow, & Carson, 2001), suggesting a negative relation between corticospinal excitability and muscle length. Furthermore, more corticospinal facilitation is observed when passively moving through different joint angles as compared to being in a static joint position (Lewis & Byblow, 2002). This suggests that movement has more of an effect on corticospinal excitability than does static muscle position. The authors attribute these results to potential influences of the Ia inhibitory pathway, as paired pulse measures (See Section 4.2.2) revealed no differences in intracortical inhibition (Lewis, et al., 2001). Furthermore, Lewis and Byblow (2002) found that muscle length has no effect on corticospinal excitability when the muscle is actively contracted. This suggests that further research is needed to fully understand the relation between corticospinal excitability and muscle length.

### ***1.7 Changes in corticospinal excitability with vibration***

Vibration parameters vary across studies and vibration can elicit a number of different neurophysiological, perceptual and behavioral responses. These can be divided into 3 distinct classes (see Table 1.1). First, at vibration displacements at or above approximately 1.0 mm peak to peak, a TVR may occur (Matthews, 1966a). However if

the muscle is already performing a submaximal contraction or it is free to shorten as a product of contraction, the TVR is known to occur at vibrations with lower peak to peak displacements (Goodwin, et al., 1972). During a TVR, the corticospinal excitability response, measured by peak to peak MEP amplitude in the vibrated muscle, increases (Claus, Mills, & Murray, 1988). Although it is known that antagonist muscle activity decreases when an agonist muscle undergoes a TVR (Eklund & Hagbarth, 1966; Matthews, 1966b), the antagonist corticospinal excitability response is unknown. Although one might suggest that excitability levels in the antagonist muscle might decrease during an agonist TVR, no known studies have examined the corticospinal influence on the antagonist muscle.

Table 1.1. Summary Table of corticospinal excitability responses to various vibration protocols.

	Causing a TVR	Causing Illusory Movements	Illusory Aftereffect	Causing neither a TVR or Illusory Movements
MEP Response Vibrated Muscle	↑Claus et al, 1988	↓Kito et al, 2006	↑Kito et al, 2006	↑Rosenkranz and Rothwell, 2003
MEP Response Vibrated Muscle's Antagonist	<b>Unknown</b>	↑Kito et al, 2006	↓Kito et al, 2006	↓Siggelkow et al, 2009

Second, when vibrating at a displacement subthreshold for eliciting a TVR (i.e., ~0.5-0.8 mm peak to peak), proprioceptive illusions are elicited (Goodwin, et al., 1972). This phenomenon occurs when vision is occluded and the muscle is kept in an isometric condition. In situations in which vibration elicits illusory muscle lengthening,

corticospinal excitability decreases. Furthermore, there is an increase in the antagonist MEP response (Kito, et al., 2006). This is likely because the illusory lengthening mimics contraction of the vibrated muscle's antagonist (Calvin-Figuere, et al., 1999). This illusory antagonist contraction supports the presence of an AVR whereby the antagonist muscle actually contracts if the illusory movement is strong enough. As previously mentioned, if a proprioceptive illusion occurs, as soon as vibration subsides the vibrated muscle feels as if it is contracting and the joint is returning to normal, which is termed the illusory aftereffect (Goodwin, et al., 1972; Kito, et al., 2006). During the illusory aftereffect, corticospinal excitability in the vibrated muscle increases, similar to that during a TVR response, and corticospinal excitability measured in the antagonist muscle decreases (Kito, et al., 2006).

Lastly, when the vibration displacement is smaller than that necessary to elicit a TVR or illusory movements, changes in MEP responses can still be observed. Rosenkranz et al. (2003) report that the displacement necessary to modulate MEP response without eliciting TVR or illusory movements is variable across subjects, between 0.2 and 0.5 mm peak to peak. Like during a TVR, the muscle vibration that does not cause a TVR or illusory movements causes an increase in MEP response, but also a decrease in the antagonist MEP response (Rosenkranz, et al., 2003; Rosenkranz & Rothwell, 2003; Siggelkow et al., 1999), as if it is a "precursor" to a TVR response.

Additionally, prolonged vibration has been shown to activate both the mechanisms of an AVR via illusory movements, *and* a precursor to a TVR, i.e. increased MEP in the vibrated muscle, however these effects only developed after the vibration ceased. Specifically, Steyvers and colleagues (2003) found that 30 minutes of muscle

tendon vibration resulted in a gradual increase in MEP amplitude post vibration, becoming significantly different from baseline at 25 minutes following vibration. Furthermore, there was an increase in excitability found in its antagonist muscles 10 minutes post vibration which continued to 55 minutes post vibration. The authors concluded that the vibration amplitude used did not reach threshold to elicit a TVR but did surpass the threshold for an AVR, and as expected, was coupled with the illusion of joint movement throughout the vibration protocol. It is unclear how altering muscle length can affect these mechanisms for a period of time following vibration.

### ***1.8 Rationale***

Of the studies that have examined muscle vibration, there is no literature that examines the corticospinal effects of vibration at different muscle lengths in spite of the evidence that both manipulations have been known to separately affect both muscle spindle activity and corticospinal excitability. Thus, the purpose of this study was to combine factors of muscle vibration and muscle length and evaluate their effect on corticospinal excitability. Muscle spindle primary afferents fire synchronously with muscle vibration consistently up to ~100 Hz (Roll, et al., 1989) and 75 Hz vibration has been shown to have the greatest influence in increasing MEP amplitude during vibration compared to 20 Hz and 120 Hz vibration (Steyvers, Levin, Verschueren, & Swinnen, 2003). Taken together, it appears that vibration produces the greatest degree of central effects when it elicits greater peripheral afferent responses. As muscle spindle afferents are known to fire more at lengthened positions during vibration (Burke, et al., 1976) it may be expected that the corticospinal excitability would be increased under these

conditions. Therefore, we hypothesize that there would be larger increases in corticospinal excitability following vibration at longer versus shorter muscle lengths.

A secondary objective was to determine the extent to which a TVR occurs, and whether changes in muscle length alter any relation between TVR and corticospinal excitability. Vibration parameters were chosen so as not to elicit a reflex response, however it is unclear how muscle lengths could affect this. We hypothesize that if a TVR occurs, there would be a positive relation between TVR and corticospinal excitability.

Results from this study just as others before it will potentially offer new perspectives for professionals in medicine and occupational therapy in developing protocols that assist in the recovery from disorders that affect the motor cortex. Furthermore, these results can offer further insight into the prescription of exercise for the purpose of increasing athletic and motor skill performance, and augmenting strength during strength training.

## CHAPTER TWO: PILOT TESTING

Extensive piloting was required before initiating the actual data collection given the technical complexities of the project, specifically: 1) issues surrounding the integration of multiple measurement tools including force sensors, accelerometry, vibration shaker, Transcranial Magnetic Stimulation (TMS) and electromyography (EMG; see Figure 2.1), and 2) the design of an appropriate manipulandum to fit different hand sizes and ensure consistent hand/finger postures (Figure 2.2).

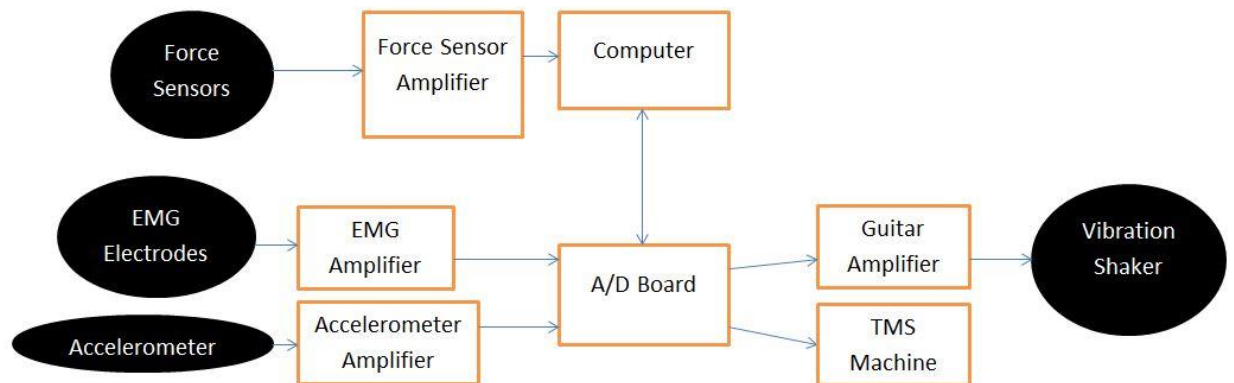


Figure 2.1. Schematic diagram depicting the equipment needed to be synchronized for successful data collection.

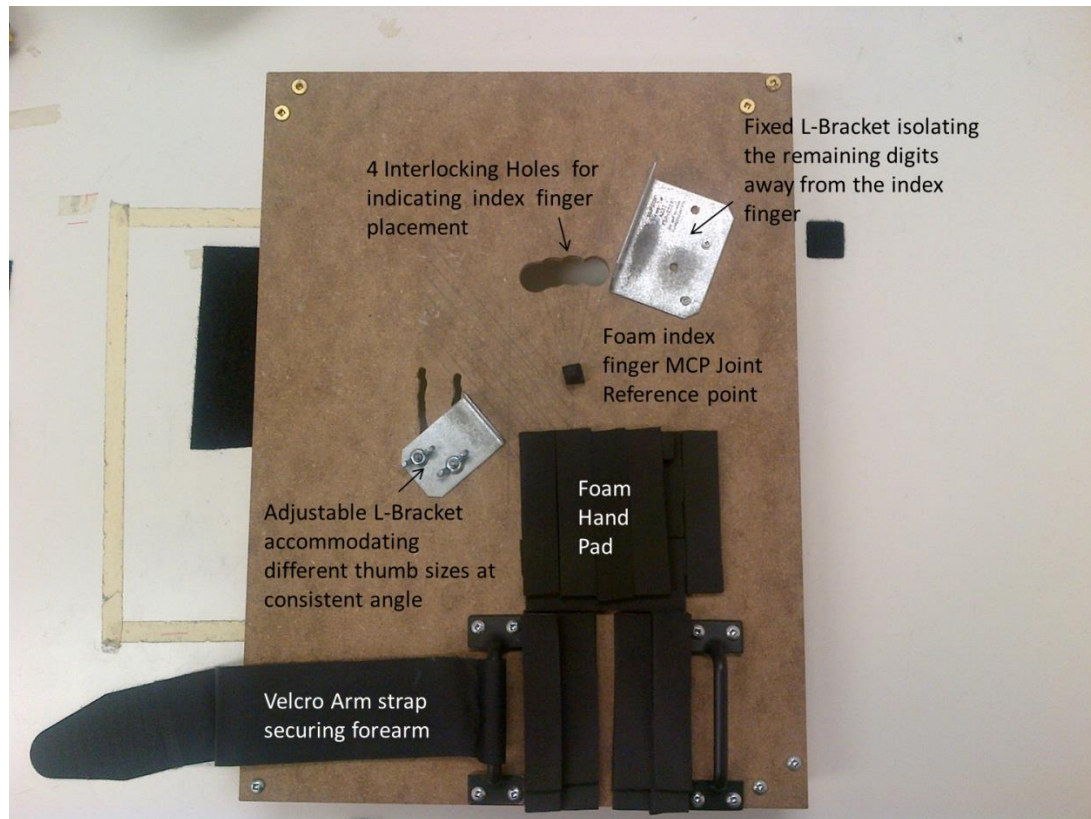


Figure 2.2. The experimental manipulandum.

### 2.1 Shaker

The vibration was produced by an electromagnetic shaker that was driven by a sinusoidal signal generated from a custom-built algorithm in LabView (National Instruments, Austin, TX). The peak to peak amplitude of this signal could be modified to change the vibration displacement. However, this signal from LabView required additional amplification. The most cost effective method of amplifying this signal was found to be a 38 watt electric guitar amplifier (Fender Electronics, Corona, CA). To verify the consistency of the amplification over time as well as determine the relation between the peak to peak amplitude of the input to the amplifier to its output, we varied

the input signal and measured the output once a day for 10 days. We observed that the input-output relation was consistent across days, but the relation was nonlinear. After determining the amplifier's consistency and observing the nonlinear relation between the amplifier's input and output, we needed to determine the relation between the peak to peak amplitude of the signal generated by LabView and the peak to peak displacement of the vibration from the shaker. To do so, a custom made mono-axial accelerometer was placed upon the contact surface of the vibrating probe and a series of trials were performed in which the input signal amplitude was varied and the vibration acceleration was recorded (measured in voltage, corresponding to acceleration measured in gravity, g). The same procedures were performed over a number of days to ensure consistency. Upon conversion of g to  $\text{m}\cdot\text{s}^{-2}$  using MATLAB (Mathworks, Natick, MA) the trace was integrated twice to yield the peak to peak displacement of the probe in metres, which was then converted to millimetres. The LabView signal amplitude that yielded a peak to peak displacement of the vibration probe of 0.5 mm was used for the study.

## ***2.2 Transcranial Magnetic Stimulation/Electromyography***

The most time consuming aspect of piloting was ensuring a good signal to noise ratio of the EMG recordings. We encountered three different sources of noise which first needed to be identified and then addressed. First, noise occurred during movement of the electrode leads. We determined that the lead wires were too small in diameter and had limited shielding. Thus, we wound the wires of both the active and reference electrodes together and taped them with electrical tape to provide extra shielding from the outside environment and reduce excessive movements of the wires. This did not sufficiently

address the movement artefact issue. Recognizing the limitations of these electrodes, we purchased new electrodes with thicker, tangle-proof leads, which eliminated this particular noise issue.

Simultaneous to this first noise issue, was significant stimulus artefact produced by the TMS. Transcranial magnetic stimulation produces a temporary magnetic field designed to induce electrical current in neighboring excitable tissue. However, this magnetic field can also be detected by electrodes placed anywhere in its vicinity. In our case, the width of this stimulus artefact interfered with the motor evoked potentials (MEPs) potentially confounding our results (Figure 2.3). We tried a number of measures to reduce this stimulus artefact. For instance, on advice from other TMS experts, we clipped our gold plated EMG electrodes, removing a small section to break the circular current generated on the electrode by the magnetic pulses emitted by the TMS machine. This did not reduce the stimulus artefact as expected. Thus, we then replaced the gold plated electrodes with Ag-AgCl disk electrodes as they are the most commonly used in this type of research. Again, this did not reduce the stimulus artefact. After consulting with other EMG experts within the faculty, it was determined that having both the experimenter and participant on the same electrical potential during the TMS pulses would minimize the width of the stimulus artefact. Therefore, a large aluminum mesh screen, grounded to the EMG amplifier, was placed on the floor to allow both the experimenter and participant to place their feet on and share the same potential. This rectified the issue with wide stimulus artefact.

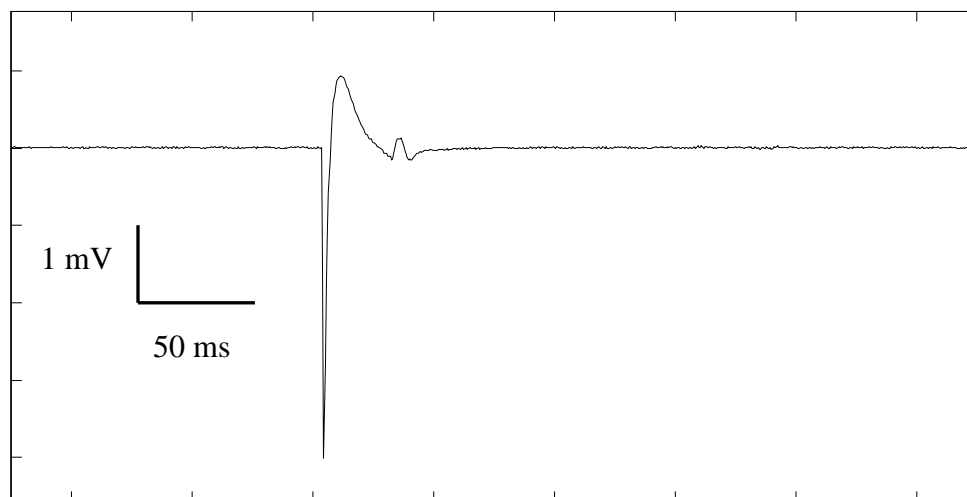


Figure 2.3. MEP with abnormally large stimulus artefact. Note the bleeding from the artefact into the MEP, as well as the large peak to peak amplitude of the artefact.

It is well established that the presence of computers and other electrical equipment that draw power from the outlets introduces very high amplitude 60 Hz interference in neurophysiological recordings (Figure 2.4). Typically, researchers use a 60 Hz notch filter when recording EMG to reduce this noise. Unfortunately, it was known a priori that any online 60 Hz notch filtering of the EMG signal would affect the peak to peak amplitude and introduce excessive rippling in the MEPs (Figure. 2.5), and thus filtering was not a viable option. In addition the use of the aluminum mesh screen to minimize the stimulus artefact from the TMS pulses acted as a large antenna for the ambient 60 Hz noise, thereby increasing its amplitude in our EMG. Without the mesh, we encountered TMS stimulus artefact, however with it, we encountered significant 60 Hz noise.

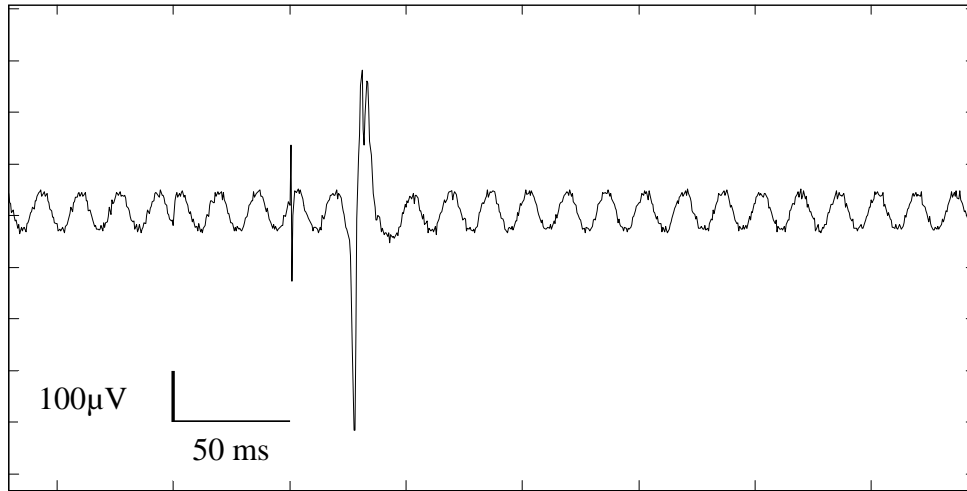


Figure 2.4. An MEP with 60 Hz noise potentially interfering with the amplitude measurements.

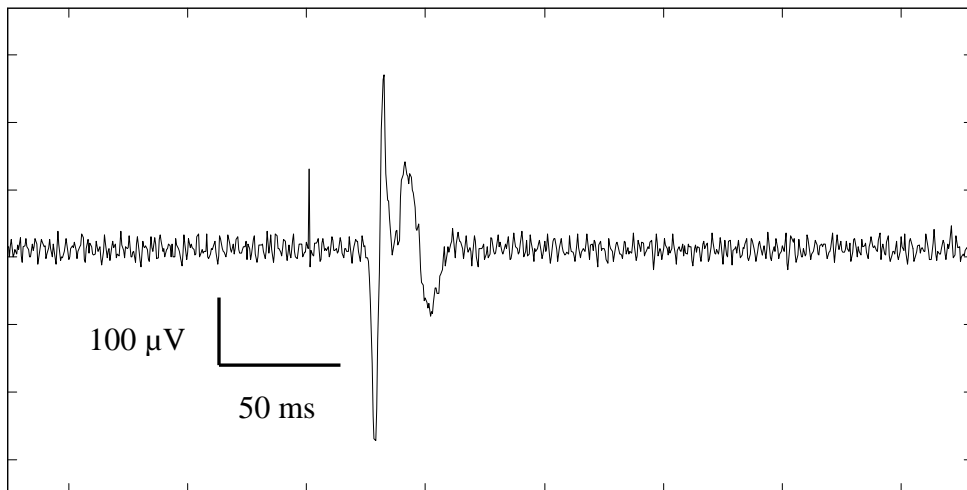


Figure 2.5. An MEP with an online 60 Hz line filter. Note the excessive rippling of the MEP.

To address the 60 Hz noise, we attempted to isolate the source by testing all possibilities. First, we purchased a “line conditioner” to eliminate any 60 Hz noise from the power outlets and ensured all experimental equipment was connected to the conditioner to eliminate the possibility of ground loops. Second, we used different wires, electrodes, EMG channels and A/D board inputs to ensure that the noise was not introduced by any of these components. We also attempted to shield the A/D system using an aluminum mesh screen. Third, the presence of powerful imaging machines in nearby laboratories and the sport medicine clinic immediately underneath the Motor Neurophysiology Lab (e.g., DEXA, x-ray, Extreme CT) was thought to be a possible source for ambient noise within our lab, however by timing our EMG data collection while these large machines were powered off did not consistently make a difference. It should be noted, however, that when the EMG system was used on weekends, there appeared to be lower amplitude of this noise. To further test the ambient noise within the laboratory, separate EMG systems were tested in the lab, some of which showed similar line interference, whereas others did not. Fourth, our EMG system was brought into other laboratories and rooms within the faculty building, however line interference was still present, suggesting that the interference was not localized to the laboratory. Ultimately, we determined, through consultation with our electrical technician, that the distance from the electrodes to the amplifier were too long and included too many connections. This allowed greater opportunity for outside interference to enter the system prior to reaching the amplifier which resulted in the 60 Hz noise being amplified in addition to the signal. The best solution to this problem was to have the EMG amplifier modified such that the electrode wires enter directly into the EMG amplifier, thereby eliminating all extraneous

connections and reducing the distance between the source of the signal and the amplifier. This system was then used for the data collection of the study. See Figure 2.6 for a usable MEP.

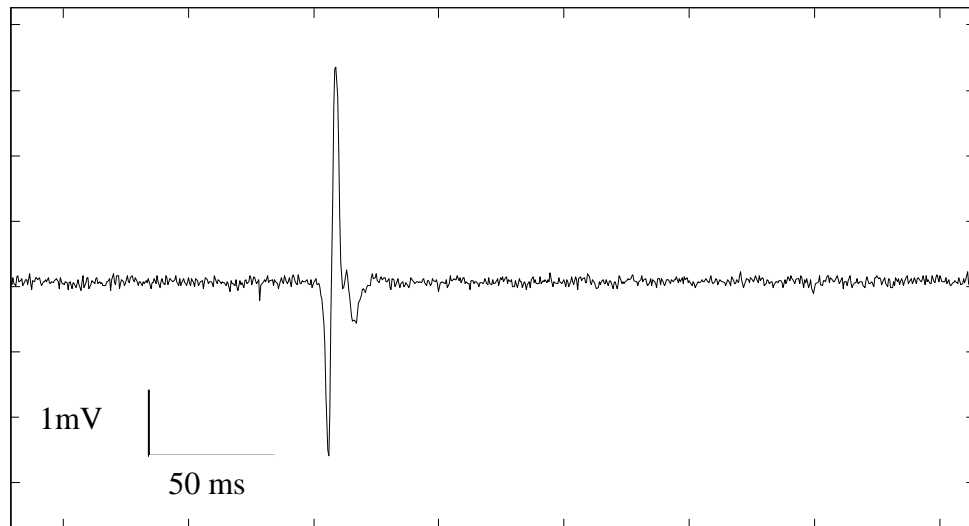


Fig. 2.6. A usable MEP. Note the small amplitude stimulus artefact, lack of 60 Hz noise and limited rippling.

### ***2.3 Experimental Manipulandum***

We used a custom built manipulandum which was necessary to ensure a consistent stable placement of the hand with the ability to isolate the FDI muscle at different muscle lengths (Figure 2.2). To accommodate different hand sizes, a single reference point at the index finger metacarpophalangeal (MCP) joint was fabricated, while an adjustable L-bracket and an adjustable ring was used for the thumb and index finger placement, respectively. Another fixed L-bracket was used to isolate the remaining

fingers away from the index finger range of motion. A Velcro strap was used to comfortably stabilize the forearm in a foam channel during the experiments.

To determine the index finger's range of motion in the abduction/adduction plane, a sample of the students and staff at the Human Performance Lab had their right hands measured for finger length, hand length, and range of motion of the index finger in the abduction/adduction plane with the remaining digits held stable. It was determined that the maximum range of adduction/abduction about the index finger MCP joint was  $30^\circ$ , in the direction of  $20^\circ$  abduction, and  $10^\circ$  adduction, with  $0^\circ$  (neutral) being the index finger in line with the second metacarpal bone. From this information, it was determined that the optimal joint angles to examine effects of FDI muscle length were  $20^\circ$ ,  $10^\circ$ ,  $0^\circ$ , and  $-10^\circ$ , fully abducted/shortened to fully adducted/lengthened, respectively. It was also determined that the optimum distance to place the adjustable ring to isolate the index finger was 7 cm from the MCP joint. With this distance, the manipulandum could accommodate the widest variety of hand and finger shapes and sizes for both male and female participants. Unfortunately, the maximum diameter of the ring excluded some potential male participants with larger widths of index finger.

The adjustable ring was anchored to a tri-axial force sensor (Nano-17, ATI Industrial Automation, Apex, NC, Resolution 0.00625 N) a distance of 57 mm above the table, which necessitated raising the rest of the hand and arm so as to allow for a comfortable position within the apparatus. To facilitate this, a  $\frac{1}{4}$ " width wooden board was mounted with 1x1 feet to allow the force sensor and mount to slide in underneath the board and have the force sensor itself go through four holes drilled through the board which correspond to the four chosen index finger positions.

## **CHAPTER THREE: THE EFFECTS OF MUSCLE BELLY VIBRATION AT VARIOUS LENGTHS ON CORTICOSPINAL EXCITABILITY**

### ***3.1 Introduction***

Vibration, either of the whole body or isolated muscles/tendons, has gained popularity as a supplement to traditional resistance training (Cormie, et al., 2006; Jordan, Norris, Smith, & Herzog, 2005; McBride et al., 2010; Rittweger, 2010) and has been examined as a possible rehabilitation tool for such movement disorders as stroke (Liepert & Binder, 2010; Marconi et al., 2011), Parkinsons disease (King, Almeida, & Ahonen, 2009) and dystonia (Rosenkranz, et al., 2008). The effect of muscle/tendon vibration on muscle spindles, i.e., receptors found in the muscles that detect muscle length and changes in muscle length, are well established (Burke, et al., 1976; Roll & Vedel, 1982; Roll, et al., 1989). Mechanical oscillations at a certain amplitude and frequency repeatedly activate muscle spindles producing neural adaptations at both the local spinal and supraspinal levels (Kito, et al., 2006). Despite its potential uses in exercise prescription and medicine, much is still unknown about the effects of vibration on the Central Nervous System (CNS). Specifically, it is unclear how and to what degree changes in muscle length affect the neural adaptations brought on by muscle vibration. Since the muscle spindles activated by muscle vibration are also differentially activated by muscle lengths, it is possible that the length of the vibrated muscle may effect excitability within the CNS affecting the efficacy of vibration protocols in medicine and exercise prescription.

Research has shown that both vibration and muscle length can affect all levels of the sensorimotor system. Specifically, mechanical vibration of a muscle activates muscle spindle afferents (Burke, et al., 1976; Fallon & Macefield, 2007; Roll & Vedel, 1982), which are known to increase activation when the muscle is lengthened and reduce activation when a muscle is shortened. During vibration, primary muscle spindle afferents have been shown to be activated at a 1:1 ratio to the vibration frequency up to ~100 Hz (Roll & Vedel, 1982), providing a direct link between vibration and muscle spindle activity. Furthermore, muscle spindle activity increases when the muscle undergoes passive stretch while being vibrated, as well as when it is vibrated at an increased static muscle length (Burke, et al., 1976). This suggests that there is a combined effect of both vibration and muscle length on muscle spindle activity.

Under certain vibration parameters, the excitation of muscle spindles induced by vibration causes an increase in activation of the homonymous muscle, known as the tonic vibration reflex (TVR, Eklund and Hagbarth, 1966). The TVR is similar to the classic stretch reflex (Matthews, 1966a), elicited by stretch of muscle. Observation of TVR presence indicates that vibration induces motor unit activity, and in fact Martin and Park (1997) demonstrated that motor units are activated synchronously with muscle vibration frequencies up to ~150 Hz. As muscle spindle activation increases with muscle length, so does the amplitude of the TVR response (Eklund, 1971; Eklund & Hagbarth, 1966). The effects that these reflex changes, due to vibration and muscle length, have on corticospinal excitability are unclear.

The application of vibration to a muscle belly or tendon has been shown to alter corticospinal excitability either during or following vibration (Claus, et al., 1988; Forner-Cordero, Steyvers, Levin, Alaerts, & Swinnen, 2008; Rosenkranz & Rothwell, 2003; Steyvers, Levin, Van Baelen, et al., 2003). Research on nonhuman primates has demonstrated that motor cortical cells fire synchronously with vibration (Fourment, et al., 1996), which suggests that the motor cortex is at least partly involved in the changes in corticospinal excitability in response to vibration. Rosenkranz et al. (2003) found that muscle vibration reduced intracortical inhibition, further supporting the idea of a cortical link to muscle vibration. Individuals sometimes perceive that the vibrated muscle is lengthening (Roll, et al., 1989), commonly referred to as proprioceptive illusions, again suggesting a cortical influence. In terms of muscle length, there is evidence that both static and dynamic muscle length changes have an effect on corticospinal excitability (Chye, Nosaka, Murray, Edwards, & Thickbroom, 2010; Edwards, et al., 2002; Lewis, et al., 2001). Since both changes in muscle length and vibration are known to affect muscle spindle afferent activity, it is thought that the changes in corticospinal excitability from each of these two manipulations are influenced by activation in the Ia afferent pathway (Lewis, et al., 2001; Steyvers, Levin, Verschueren, et al., 2003). However to this point no study has examined the concomitant effects of both vibration and muscle length changes on corticospinal excitability.

Thus, the primary aim of this study was to determine to what degree changes in muscle length affect corticospinal excitability induced by local muscle vibration in healthy adults. The hypothesis was that there would be larger increases in corticospinal excitability following vibration at longer versus shorter muscle lengths. A secondary

objective was to determine the extent to which a TVR occurs, and whether changes in muscle length alter any relation between TVR and corticospinal excitability. The hypothesis was that if a TVR occurred, there would be a positive relation between TVR and corticospinal excitability.

### **3.2 Methods**

#### *3.2.1 Participants*

Fourteen right handed participants (8 male; mean age:  $26.6 \pm 2.87$  years), as determined by the Edinburgh Handedness Inventory (Oldfield, 1971; Appendix A), without history of highly skilled hand use (e.g. professional musician, professional athlete, surgeon, etc.) participated in this study. Further screening was done to exclude individuals with a history of neuromuscular disorders and/or brain injury/condition (e.g. concussion or epilepsy, etc.) using the Transcranial Magnetic Stimulation Adult Safety Screen (TASS, Keel et al, 2001; Appendix B). Ethics approval was obtained from the Conjoint Health Research Ethics Board at the University of Calgary. Participants gave written, informed consent (Appendix C) to the procedures in accordance with the Declaration of Helsinki.

#### *3.2.2 Electromyography*

Surface electromyography (EMG) was recorded from the right First Dorsal Interosseous (FDI) muscle of each participant using Ag-AgCl electrodes (Grass-Astromed, West Warwick, RI) affixed in a monopolar belly tendon montage (Moller, Arai, Lucke, & Ziemann, 2009). A ground electrode was also placed on the right radial styloid process. Each electrode area was prepared by abrading the skin using sandpaper

and cleansed using isopropyl alcohol prior to affixing the electrodes. Electromyographic signals were amplified (x1000) with a bandwidth of 30 Hz - 1 kHz (Grass-Astromed, West Warwick, RI), and digitized at 2 kHz using custom software (Labview, National Instruments, Austin, TX).

### *3.2.3 Transcranial Magnetic Stimulation*

Prior to the start of the experimental protocol, the optimal location on the scalp to elicit a motor evoked potential (MEP) in the FDI muscle, the FDI 'hotspot', was determined. To do this, participants were fitted with an elastic latex swim cap pulled tightly onto the head. The vertex of the head was determined as the point half the distance between the nasion and inion and half the distance between the auditory openings along the interaural line (Jasper, 1957). A 4 cm x 4 cm square grid with 1 cm increments was drawn on the cap, with the centre of the grid 6 cm lateral to the vertex along the interaural line on the left side of the head (Forner-Cordero, et al., 2008; Smith & Brouwer, 2005). Each point on the grid could be potential stimulation sites. The FDI 'hotspot' was determined as the site on the grid that elicited the highest MEP at a given stimulation intensity. To determine this, the experimenter began presenting low intensity (30% of maximum) transcranial magnetic stimulation (TMS; Magstim 200, Dyfed, UK) pulses at a single location through a figure of eight coil (Diameter: 90 mm). The stimulus intensity was increased until an MEP (see Figure 2.6) in the FDI muscle was visible (Smith & Brouwer, 2005). Stimulation began at the centre of the grid and then spiraled out in 1 cm increments until the hotspot was determined. After determining the hotspot, the resting motor threshold (rMT), i.e., the minimum stimulator output required to elicit a consistent

MEP in the FDI muscle was determined. Specifically, MT was defined as the minimum stimulation intensity required to elicit an MEP  $\geq 50 \mu\text{V}$  (peak to peak) in 5 out of 10 successive pulses (Rossini, 1994) when stimulating at the hotspot. An aluminum mesh screen, grounded to the EMG amplifier, was placed underneath the participant and experimenter to allow them to share the same electrical potential, thereby reducing the stimulus artefact caused by the TMS pulses (see Section 2.2).

#### *3.2.4 Vibration*

Vibration was administered to the FDI muscle (see below) with an electromagnetic shaker (V201, Ling Dynamic Systems, Royston, UK) powered by a sinusoidal signal generated by custom made software (Labview, National Instruments, Austin, TX). Two cylindrical probes (length 4 cm and 13 cm, diameter 0.4 cm) separated by a tri-axial force sensor (Nano-17, ATI Industrial Automation, Apex, NC, Resolution 0.00625 N) were fashioned, with the short probe attached to the shaker, and the longer probe designed to apply vibration to the FDI muscle of the participant (Figure 3.1). Note that vibration was applied to the muscle belly as opposed to the tendon as the proximity of the FDI tendon to the metacarpophalangeal joint would likely cause discomfort and provide very little lengthening/shortening of the muscle.

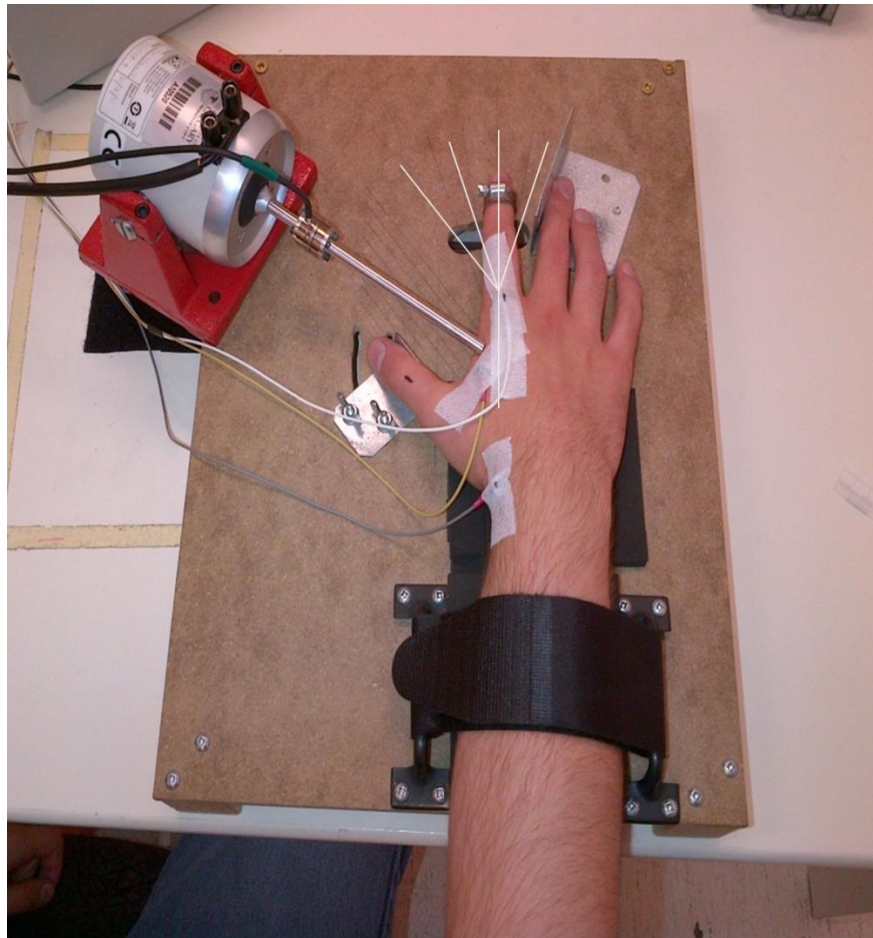


Figure 3.1. The experimental setup, with the participant's hand fastened into the manipulandum with vibration probe attached. The 4 joint angle positions have been superimposed on top of the hand.

### *3.2.5 Experimental Protocol*

The experimental protocol included four conditions, each condition consisted of a different static muscle length of the FDI. To ensure consistency within and across participants, a custom built manipulandum isolated the right FDI muscle at these four possible lengths whilst keeping the rest of the hand in a stable consistent position (Figure 3.1). As the FDI is an index finger abductor, muscle length was changed by rotating the

index finger about the metacarpophalangeal (MCP) joint in the abduction/adduction plane. To ensure the accuracy of the changes in joint angle (i.e., muscle length) we maintained a consistent placement of the MCP joint on the manipulandum throughout the experimental conditions. To maintain the prescribed joint angle and to stabilize the index finger, the index finger was secured in an adjustable ring attached to a tri-axial force sensor (Nano-17, ATI Industrial Automation, Apex, NC, Resolution 0.00625 N). The force sensor fit securely into the surface of the manipulandum through one of four holes that corresponded to abduction/adduction of the index finger at  $-10^\circ$ ,  $0^\circ$  (neutral position),  $10^\circ$ , and  $20^\circ$  (FDI muscle lengthened to shortened, respectively). Note that the  $0^\circ$  (neutral position) was defined with the index finger in line with the second metacarpal bone. The angle between the thumb interphalangeal and the index MCP joints referenced from the radial styloid process was held constant at  $40^\circ$  measured with a manual goniometer. Additionally, the angle between the index MCP joint and the ulnar styloid process referenced from the radial styloid process was held constant throughout the experimental conditions, to ensure consistency in the ulnar-radial deviation of the wrist. These measurements were performed with the index finger in the neutral ( $0^\circ$ ) position. The forearm was fastened down onto a channel of foam to keep it in a stable and comfortable position (Figure 3.1).

At the start of the experimental session, the participant was instructed to remove his/her left shoe and place his/her foot flat on the aluminum mesh screen on the floor under the experimental table. The participant was then seated in an upright posture, with the right arm comfortably extended on the manipulandum. Once the EMG electrodes were affixed, and the FDI hotspot and MT established, the hand was securely placed in

the manipulandum by aligning the MCP joint with the reference point and inserting the index finger into the adjustable ring. The vibration probe was then set perpendicular to the belly of the FDI muscle with a static force of ~0.5 N. The participant was instructed to keep his/her right hand as relaxed as possible and close their eyes throughout the protocol to eliminate the influence of visual information on any proprioceptive illusions elicited by vibration. Each experimental condition began with the experimenter presenting a series of six single TMS pulses to the FDI hotspot at 120% of the predetermined rMT (see above), each pulse spaced one minute apart. Following the first six TMS pulses (five minutes in duration), ten trains of 80 Hz vibration (25 s on, 5 s off) at 0.5 mm peak to peak displacement (Steyvers et al, 2003) were applied to the FDI muscle for a total duration of five minutes. Note that the 80 Hz vibration frequency was chosen to elicit the most consistently high muscle spindle activation (Roll & Vedel, 1982), and the peak to peak displacement of the vibrating probe was chosen so as not to elicit a TVR (Siggelkow, et al., 1999). After the five minute vibration period, an additional six TMS pulses were applied each spaced one minute apart. Six TMS pulses were chosen for the pre- and post-vibration blocks as it has been shown that blocks of 5 or more pulses have higher levels of reliability (Kamen, 2004). Thus, each experimental condition lasted fifteen minutes. Throughout the protocol, the experimenter provided verbal communication to the participant to avoid being startled by the presentation of vibration or TMS pulses.

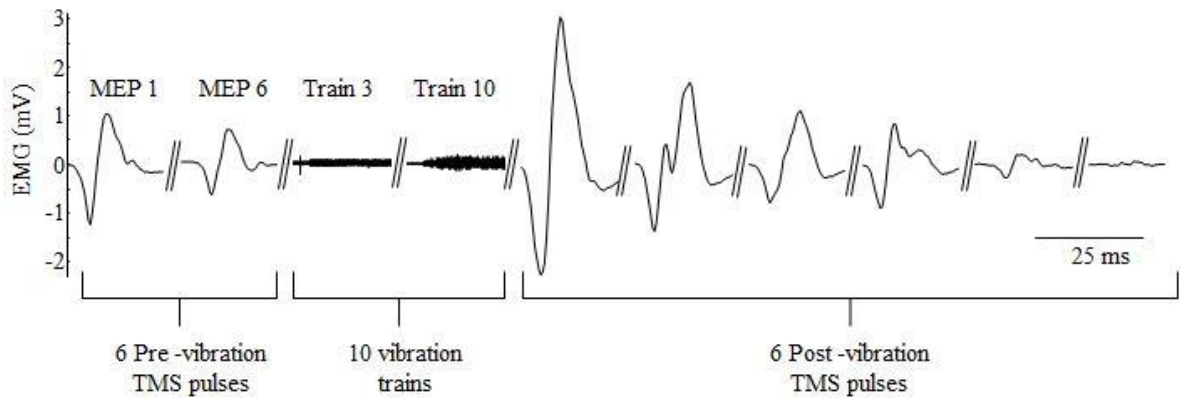


Figure 3.2. Schematic description of the experimental protocol.

Once each condition was finished, the participant was instructed to open his/her eyes, and the vibration probe was removed from the FDI muscle. The participant was then given a questionnaire (Steyvers et al, 2003) determining whether any proprioceptive illusions were elicited during the vibration periods. Specifically, participants were asked whether they felt movement in the index finger and/or other digits (Occurrence). If so, they were then asked to rate the 1) amplitude of the movement illusion (Strength), 2) clarity or “lifelike” feeling of the movement (Vividness), and 3) describe when the illusion started and ended (Duration; Appendix D). After approximately five minutes, the next experimental condition began until all four conditions were completed. The presentation order of the four experimental conditions, i.e., muscle lengths, was randomly assigned. The entire experimental session of pre-screening, participant preparation, and the four experimental conditions lasted approximately two hours.

### 3.2.6 Analysis

Normalization of the raw EMG data was required to reduce the across-subject variability associated with EMG and TMS (e.g., due to differences in skin conductivity, motor map variability, etc). Pre-vibration peak to peak MEP amplitudes ( $MEP_{pre}$ ) for each experimental condition were averaged and expressed relative to the the average MEP amplitude at the neutral ( $0^\circ$ ) condition. Each post-vibration peak to peak MEP amplitude ( $MEP_{post}$ ,  $n = 6$ ) from each condition was expressed relative to its respective average  $MEP_{pre}$ , and then averaged.

The TVR can be measured using either EMG response, or force generated by the reflex contraction (Eklund & Hagbarth, 1966; Marsden, Meadows, & Hodgson, 1969). The current study examined both abduction and adduction forces; the abduction force being an indication of TVR activation. The abduction/adduction force from the index finger during the vibration period was low pass filtered at 5 Hz. The force from the first 5 s during the vibration period (i.e., the first “off” period during vibration occurring between the last pre-vibration TMS pulse and the first vibration train) was averaged and subtracted from the force over the remainder of the vibration period, to remove any isolated effect of muscle length change, as there is no vibration occurring at this time. The force was then averaged across time for each experimental condition, separately. All pre-processing was performed using Matlab software (Mathworks, Natick, MA).

For statistical analysis, three separate one-way repeated measures ANOVAs were performed to determine the effects of Condition (4 joint angles:  $-10^\circ$ ,  $0^\circ$ ,  $10^\circ$ ,  $20^\circ$ ) on the dependent measures of  $MEP_{pre}$ ,  $MEP_{post}$ , and abduction/adduction force. Additionally, a two-way repeated measures ANOVA was performed to determine the effects of both

Condition and Time (6 post-vibration MEPs) on  $MEP_{post}$ . Post-hoc analyses were performed on any significant main effects using a Bonferroni correction. One sample t-tests were performed to compare  $MEP_{post}$  and its respective pre-vibration baseline for each Condition to determine the effects of vibration alone, on corticospinal excitability. A Bonferroni correction was applied to these tests to account for multiple comparisons, which resulted in an alpha level for significance occurring at  $p \leq 0.0125$ .

Given results of the ANOVA for abduction/adduction force, forces were separated into abduction or adduction forces depending on muscle length condition. Linear regression analyses were performed to determine the extent of the relation between the average abduction or adduction force and 1) average  $MEP_{post}$ , and 2) the first  $MEP_{post}$  for each experimental condition. Qualitative data from the participants' post-vibration proprioceptive illusion questionnaire were compiled and examined in concert with their corresponding mean  $MEP_{post}$  and abduction/adduction force. Alpha level for ANOVA and linear regressions were set at  $p \leq 0.05$ . All statistical analyses were performed using IBM SPSS Statistics v.19 (Armonk, NY).

### **3.3 Results**

#### **3.3.1 $MEP_{pre}$**

Qualitatively, the baseline MEP levels ( $MEP_{pre}$ ) increased as muscle length decreased, and exhibited large across-subject variability particularly at the two shortest muscle length conditions (Figure 3.3). However, the one-way ANOVA showed no significant effects of Condition on  $MEP_{pre}$  ( $F [1.117, 14.518] = 1.044, p > 0.05$ ).

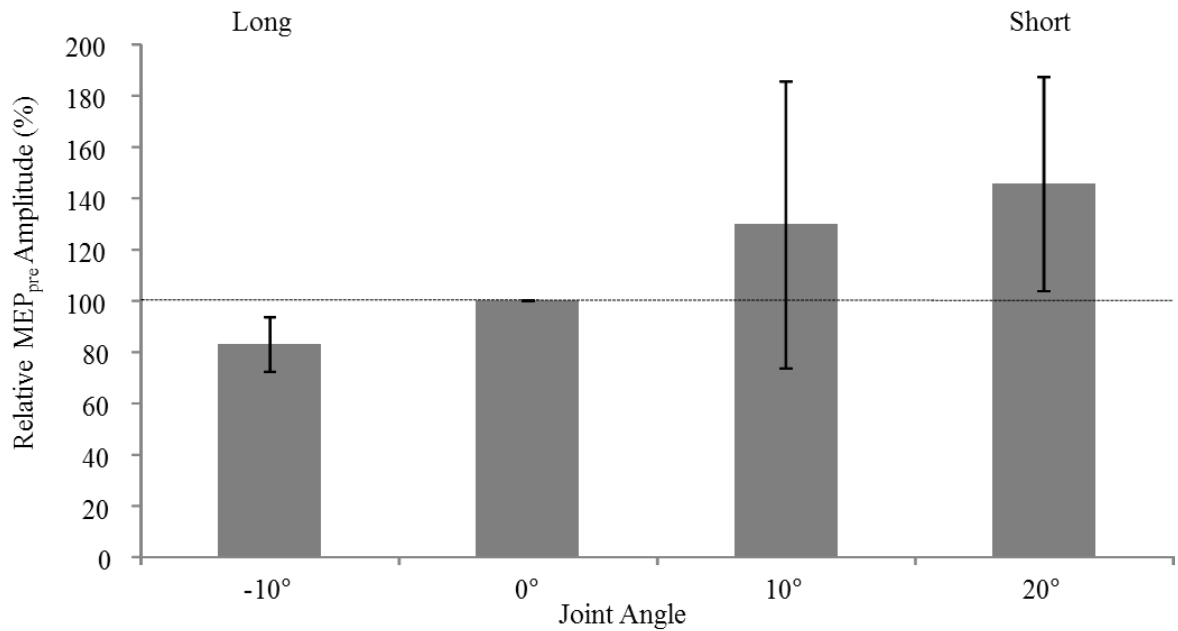


Figure 3.3. Average MEP<sub>pre</sub> amplitudes expressed relative to the 0° condition.

### 3.3.2 MEP<sub>post</sub>

Each MEP<sub>post</sub> condition increased from baseline, with the two longest muscle length conditions (-10° and 0°) showing similar values, and further stepwise decreases in MEP<sub>post</sub> as the muscle length decreased (Figure 3.4). The one-way ANOVA performed on MEP<sub>post</sub> across conditions showed no significant main effects ( $F [3, 39] = 0.872, p > 0.05$ ). However, the one-sample T-test performed on MEP<sub>post</sub> for each condition showed a significant increase from baseline, but only for the -10° condition ( $t [13] = 3.21, p \leq 0.01$ ). The other conditions were not significantly different from baseline. Figure 3.5 shows the data for all individual participants to illustrate the across-subject variability.

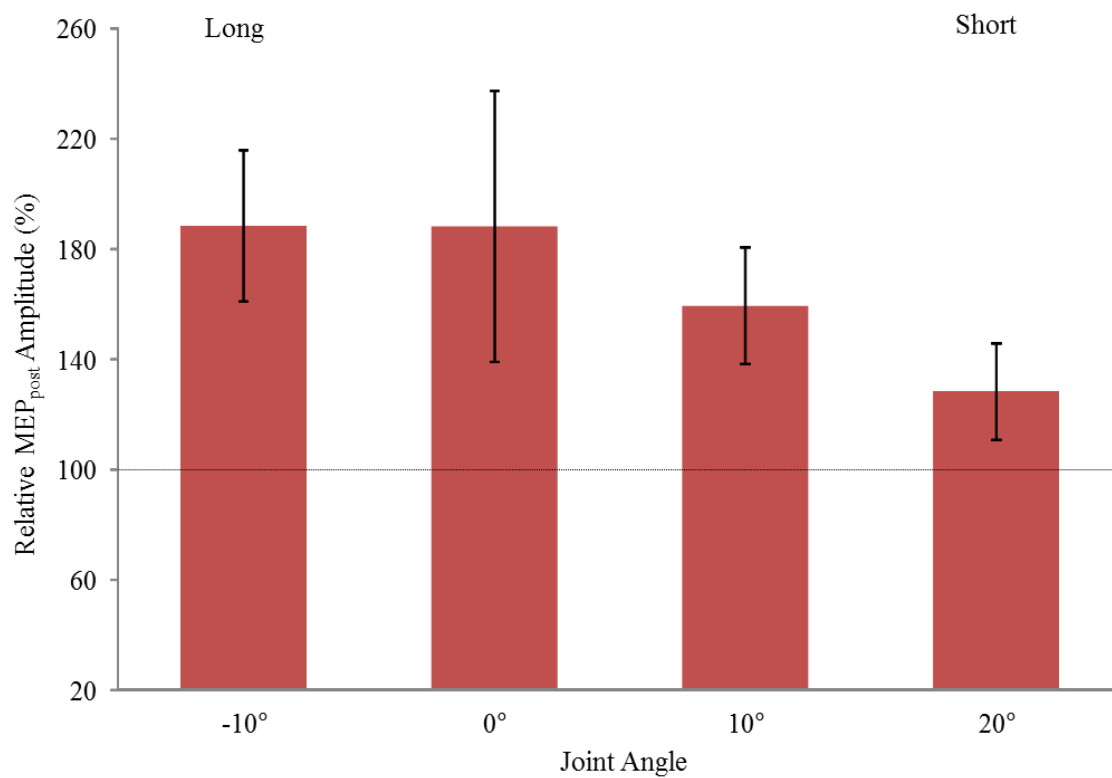


Figure 3.4. Average MEP<sub>post</sub> amplitude across joint angle condition expressed relative to pre-vibration MEP amplitude.

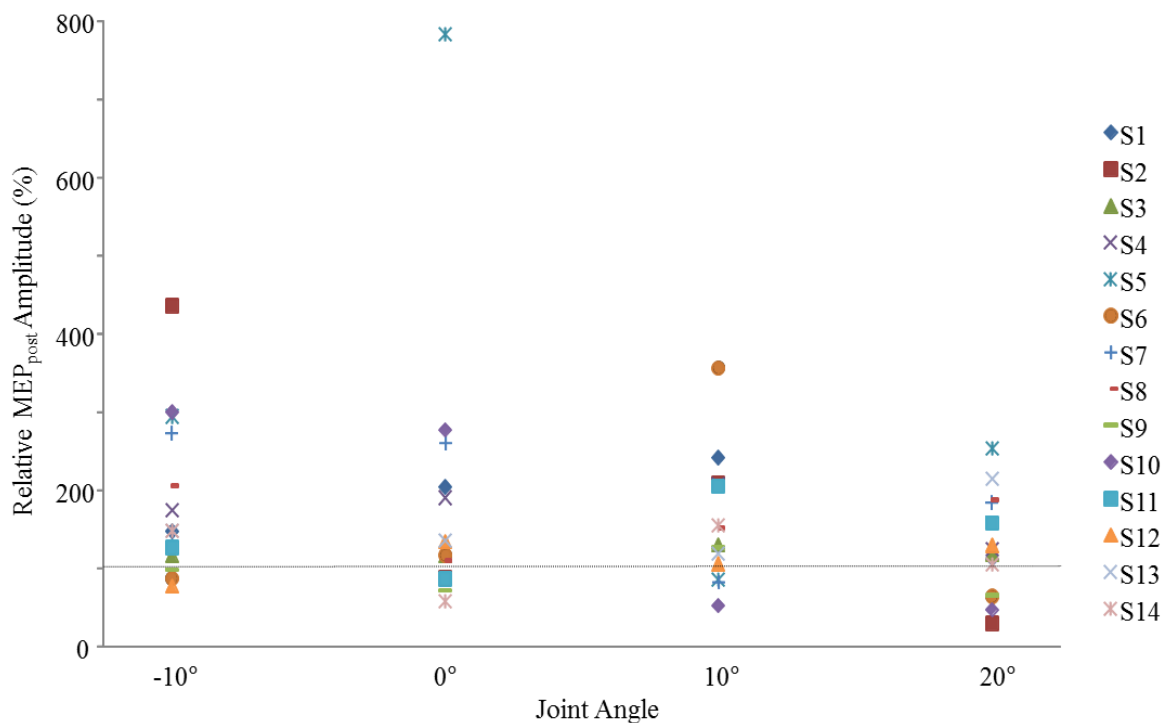


Figure 3.5. Average  $MEP_{post}$  amplitudes for each participant during each experimental condition. Note the horizontal line represents pre-vibration  $MEP$  amplitude at the same joint angle.

The results of  $MEP_{post}$  over time are shown in Figure 3.6. The first  $MEP_{post}$  after vibration appears to be higher for the  $-10^\circ$  and  $0^\circ$  condition, and then returns to the range of values of the remaining  $MEP_{post}$  amplitudes. The two-way ANOVA showed a significant main effect of Time ( $F [2.223, 28.902] = 5.537, p < 0.01$ ), with no significant main effect of Condition ( $F [3, 39] = 0.887, p > 0.05$ ). Post hoc analysis on the time effect showed no significant pairwise comparisons. There was also no interaction between Time and Condition ( $F [15, 195] = 1.105, p > 0.05$ ).

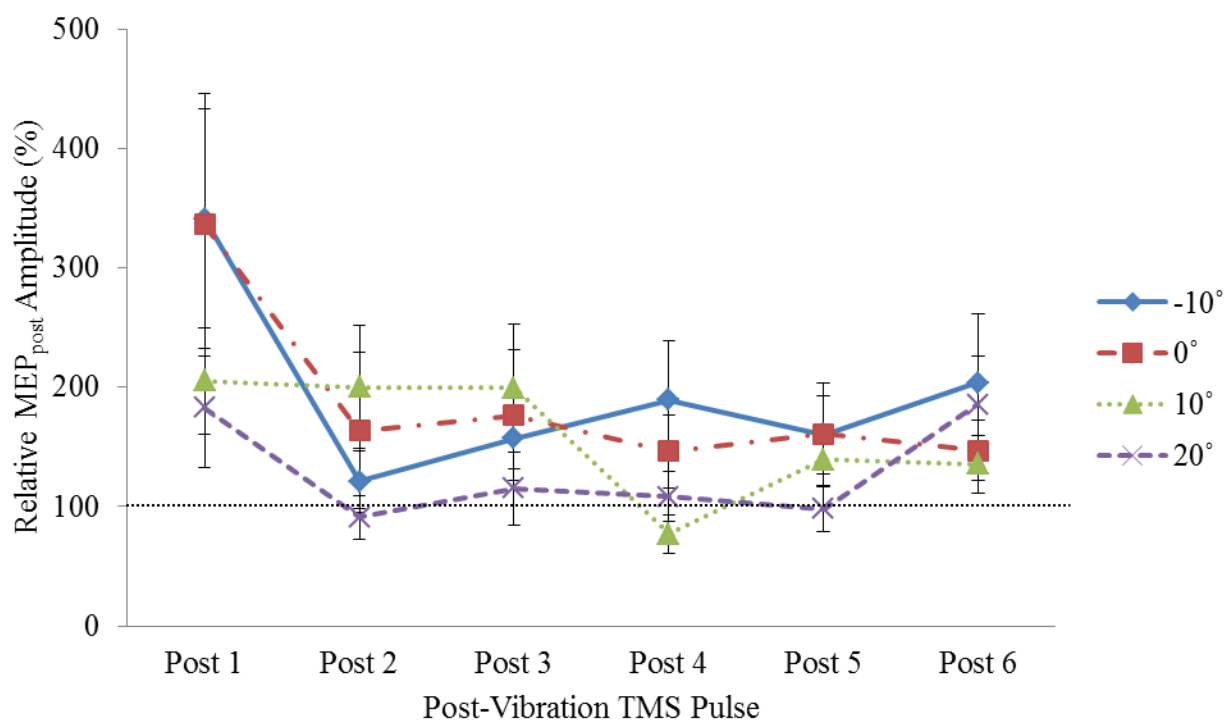


Figure 3.6.  $MEP_{post}$  for each time point across joint angle conditions. Note: 100% represents pre-vibration MEP amplitude.

### 3.3.3 Abduction/Adduction Force

A comparison of the mean abduction/adduction force produced during vibration at each joint angle is shown in Figure 3.7. Only the  $-10^\circ$  condition showed an abduction force, while the remaining conditions showed adduction. A one-way ANOVA showed a main effect of Condition ( $F [1.222, 15.892] = 5.075, p < 0.05$ ). Post-hoc analysis revealed no significant pairwise comparisons.

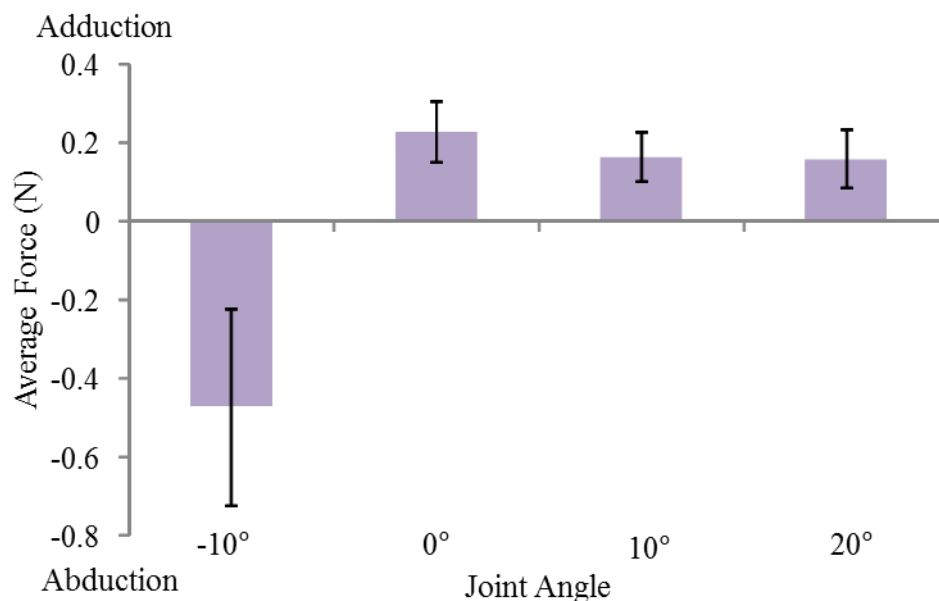


Figure 3.7. Abduction/adduction force produced during vibration for each experimental condition. Note that negative forces are abduction (FDI muscle shortening direction) and positive forces are adduction (FDI muscle lengthening direction).

#### 3.3.4 Relations between Abduction/Adduction Force and $MEP_{post}$

Given the results in Section 3.3.4 and Figure 3.7 above, regression analyses relating abduction force to mean  $MEP_{post}$  and first  $MEP_{post}$  after vibration were performed for the -10° condition (Figure 3.8). There appears to be a slight negative relation between abduction force and  $MEP_{post}$  in both analyses, but neither were significant. Given that this analysis was performed to determine the relation between MEP amplitude and only abduction force, only those participants that exhibited abduction force were included. Note that one participant showed adduction force during the -10° condition and thus was removed from this analysis to prevent possible confounds.

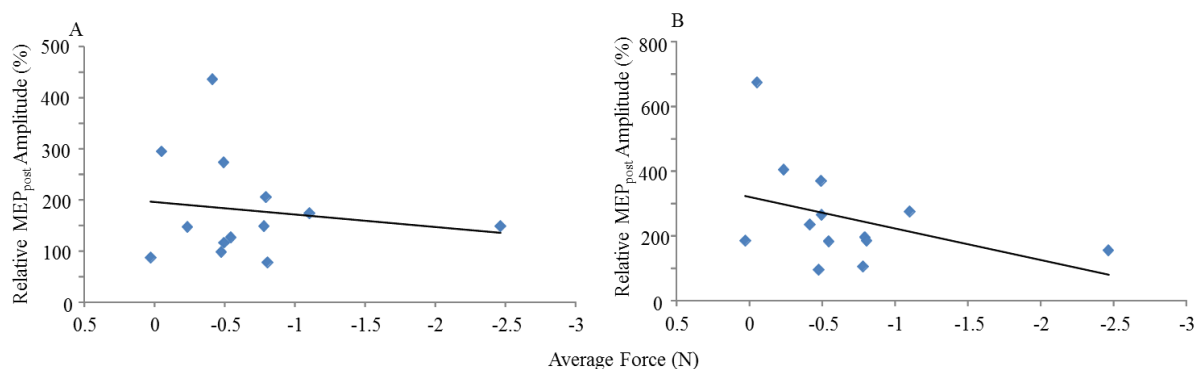


Figure 3.8. Relations between abduction force (representative of TVR) during vibration and (A) mean MEP<sub>post</sub>, and (B) the first MEP<sub>post</sub> after vibration, for the  $-10^\circ$  condition. Note that abduction forces are expressed as negative forces (FDI muscle shortening direction).

Regression analyses relating adduction force during vibration to mean MEP<sub>post</sub> and first MEP<sub>post</sub> after vibration were performed for the  $0^\circ$ ,  $10^\circ$ , and  $20^\circ$  condition (Figure 3.9). At the neutral muscle length condition ( $0^\circ$ ) there appears to be no relation between adduction force and MEP<sub>post</sub> in both analyses, however a positive relation seems to develop as muscle length shortened. In fact, the relation with adduction force and mean MEP<sub>post</sub> in the  $20^\circ$  condition was significant ( $r^2 = 0.46$ ,  $p < 0.05$ ), while the relation with adduction force and first MEP<sub>post</sub> after vibration showed a trend ( $r^2 = 0.40$ ,  $p = 0.067$ ). Given that this analysis was performed to determine the relation between MEP amplitude and only adduction force, only those that exhibited adduction force were included in the analysis, thus participants exhibiting abduction force during these conditions were removed from the analysis to prevent possible confounds.

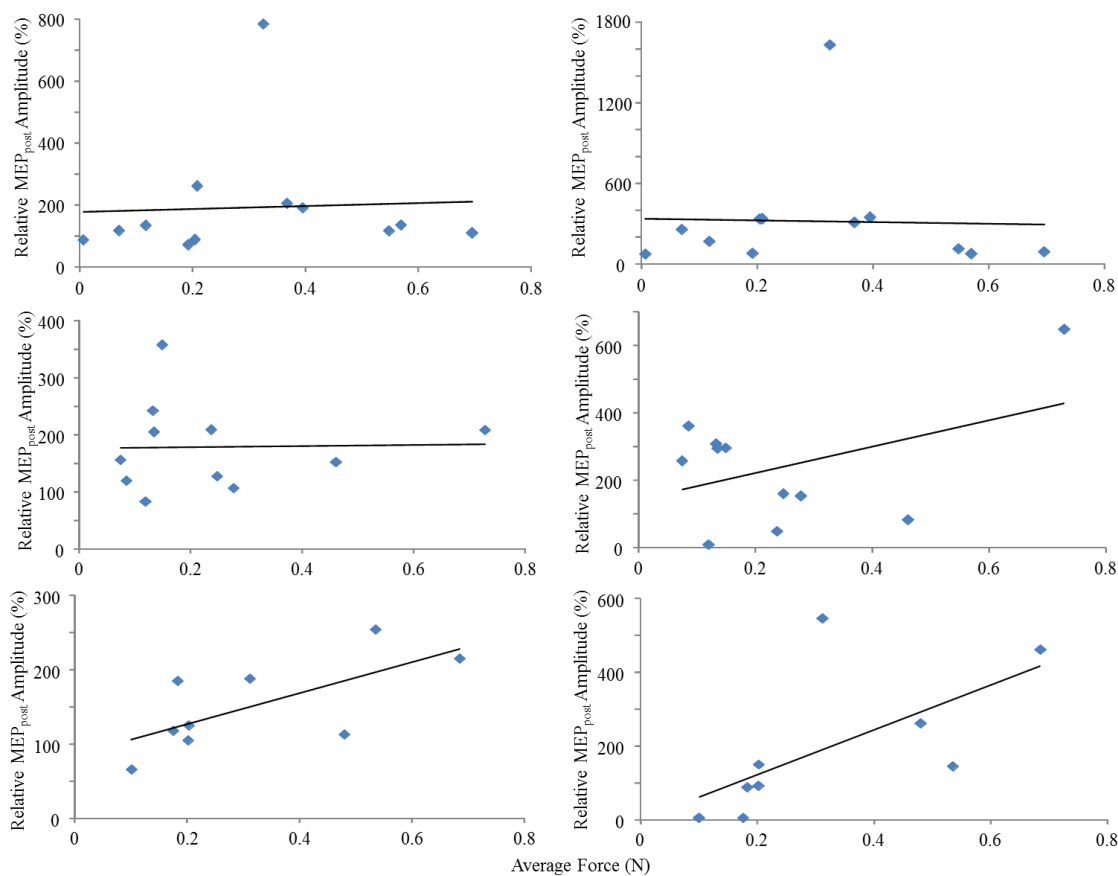


Figure 3.9. Relations between adduction force during vibration and the mean  $MEP_{post}$  and the first  $MEP_{post}$  amplitude (left and right, respectively) for  $0^\circ$ ,  $10^\circ$ , and  $20^\circ$  (top to bottom, respectively).

### 3.3.5 Proprioceptive Illusions

A total of 6 out of the 14 participants experienced some type of movement illusion induced by the muscle vibration in at least one of the experimental conditions. The results of the questionnaires (see Appendix) given to the participants are shown in Table 3.1. There were ten cases of an adduction illusion during vibration, which corresponds to the illusion of muscle lengthening. There were 6 cases of an abduction illusion which corresponds to muscle shortening, and 2 cases of a flexion illusion, which is a secondary action of the FDI muscle. There was also one participant that felt the index

finger move in both abduction and adduction throughout the vibration. Out of all the cases of movement illusions, 12 of them were accompanied by force in the same direction as illusion, while 4 were accompanied by force in the opposite direction.

Flexion force was not measured.

Table 3.1. A compilation of the proprioceptive illusions (if any) elicited by vibration experienced with the corresponding MEP<sub>post</sub> for each participant in each experimental condition. Note: S: Study Participant Abd: Abduction, Add: Adduction, Dir.: Direction of illusion, Flex: Flexion, Both: Both directions, N/A: Not applicable

S	-10°			0°			10°			20°		
	Dir.	Force (N)	MEP ± SD (%)	Dir.	Force (N)	MEP ± SD (%)	Dir.	Force (N)	MEP ± SD (%)	Dir.	Force (N)	MEP ± SD (%)
1	N/A	-0.23	147.22 ± 137.27	N/A	0.37	204.35 ± 67.28	N/A	0.13	241.95 ± 119.49	N/A	0.48	112.63 ± 103.42
2	Flex	-0.41	435.83 ± 310.35	Add	0.20	88.00 ± 121.98	Abd	0.24	208.60 ± 98.26	Abd	-0.11	29.75 ± 18.62
3	N/A	-0.50	116.40 ± 80.17	N/A	0.55	116.00 ± 106.00	N/A	-0.12	130.60 ± 108.96	N/A	0.18	118.01 ± 113.44
4	Abd	-1.10	174.20 ± 69.38	Add	0.39	190.69 ± 117.03	Add	0.73	207.50 ± 218.01	Add	0.20	124.96 ± 46.30
5	N/A	-0.05	294.52 ± 242.03	N/A	0.33	784.07 ± 514.98	N/A	-0.05	85.38 ± 39.60	Flex	0.54	253.79 ± 124.08
6	Add	0.03	87.42 ± 74.04	Add	0.07	116.80 ± 77.39	Add	0.15	356.87 ± 375.86	Add	-0.06	64.26 ± 90.25
7	N/A	-0.49	273.13 ± 108.48	N/A	0.21	260.56 ± 86.33	N/A	0.12	82.49 ± 67.42	N/A	0.18	184.52 ± 177.49
8	N/A	-0.79	205.78 ± 98.60	N/A	0.70	109.99 ± 109.21	N/A	0.46	151.74 ± 88.71	N/A	0.31	187.42 ± 178.51
9	N/A	-0.48	98.43 ± 80.40	N/A	0.19	71.66 ± 35.16	N/A	0.25	127.05 ± 46.58	N/A	0.10	65.37 ± 31.62
10	N/A	2.00	300.83 ± 570.75	N/A	-0.35	277.57 ± 294.72	N/A	-0.20	52.30 ± 52.44	N/A	-0.26	47.17 ± 47.16
11	Flex	-0.55	127.06 ± 70.14	Both	0.01	86.59 ± 50.58	Add	0.13	204.80 ± 99.24	Add	-0.08	158.22 ± 196.29
12	Abd	-0.80	77.33 ± 56.32	N/A	0.12	134.31 ± 76.43	Abd	0.28	106.20 ± 59.68	Abd	-0.16	129.73 ± 120.93
13	N/A	-0.78	148.17 ± 155.42	N/A	0.57	135.33 ± 83.06	N/A	0.09	119.18 ± 131.06	N/A	0.68	214.78 ± 166.99
14	N/A	-2.46	148.21 ± 33.97	N/A	-0.17	58.00 ± 28.08	N/A	0.07	155.49 ± 72.98	N/A	0.20	105.04 ± 45.70

### 3.4 Discussion

This is the first study to examine muscle length as a potential modulating factor of corticospinal excitability following muscle vibration. There were two main findings.

First, there were no significant differences in corticospinal excitability after vibration across muscle lengths, and vibration only had a significant effect on corticospinal excitability when the muscle was in a lengthened position. Second, the tonic vibration reflex was only observed in the longest muscle length condition, however, it was unrelated to corticospinal excitability. These findings will be discussed in relation to the underlying physiology and literature in this area.

#### *3.4.1 Corticospinal Excitability as a Function of Vibration and Muscle Length*

In this study, muscle length and vibration were used as interventions as each are known to affect afferent activity, and in turn, corticospinal excitability. To evaluate the effects of static muscle length alone, corticospinal excitability prior to vibration was examined. The results revealed no differences in corticospinal excitability across muscle lengths prior to vibration. This is contrary to Lewis et al. (2001) who found greater excitability during a static and relaxed condition when the wrist flexor was held in a relaxed and shortened as compared to lengthened position. These authors suggest that the increase in excitability observed in their study is due to a reduced muscle spindle output during the condition when the muscle is shortened. This may suggest that in the case of muscle length, muscle spindle output and corticospinal excitability are negatively related. Note that despite the lack of significance in the current study, the relation observed between muscle length and corticospinal excitability mirrored that of Lewis et al. (2001).

Corticospinal excitability following vibration was compared to corticospinal excitability prior to vibration at each individual muscle length to evaluate the effects of vibration alone. Corticospinal excitability increased from baseline following vibration

for each muscle length, however this was only statistically significant at the longest muscle length condition. Larger corticospinal excitability during and after vibration is supported by previous research (Claus, et al., 1988; Kossev, Siggelkow, Schubert, Wohlfarth, & Dengler, 1999; Rosenkranz, et al., 2003) and is thought to be due to increases in afferent input from the affected muscles (Rosenkranz & Rothwell, 2003). Thus, according to these authors there is a positive relation between spindle activity and corticospinal excitability as a function of vibration. In fact, research has shown that the highest increases in corticospinal excitability elicited by vibration are found when the vibration frequency elicits the highest amount of muscle spindle discharge (Siggelkow, et al., 1999; Steyvers, Levin, Verschueren, et al., 2003), which is the same vibration frequency used in this study. This further supports the idea that the increases in corticospinal excitability found following vibration are most likely due to increased afferent activity travelling to the central nervous system (CNS). Note that the influence of spindle activity on corticospinal excitability when induced by muscle length as compared to vibration differs (see above). This may suggest that the CNS may be processing the afferent information from these two manipulations differently.

The current results showed a significant increase in corticospinal excitability after vibration in only the longest muscle length position, which suggests a differential effect of muscle length and vibration on corticospinal excitability. Burke et al. (1976) reported that vibration of a muscle in a lengthened position activates muscle spindles at a higher rate than vibration of a muscle in the neutral position. Therefore, the larger corticospinal excitability observed after vibration at the lengthened muscle position is perhaps due to increases in spindle activity. If so, the positive relation between spindle activity and

corticospinal excitability as a function of vibration may indicate that vibration has a larger effect on corticospinal excitability than muscle length, which shows a negative relation between the assumed spindle activity and corticospinal excitability. The fact that the remaining muscle length conditions did not exhibit an increase in corticospinal excitability may suggest that at these shortened muscle length conditions, there was an insufficient increase in muscle spindle activity. Despite seeing the differences in corticospinal excitability following vibration for only the longest muscle length, significant differences in corticospinal excitability were not found across muscle length conditions. It is likely that this is due to large intra- and inter-subject variability observed (see section 3.4.3). As such, further research is needed to understand this relationship.

After vibration, corticospinal excitability decreased over time with the first time point exhibiting the highest level of corticospinal excitability, particularly at the longest and the neutral muscle lengths. Despite the decreasing corticospinal excitability over time, the time points remained slightly above baseline. This suggests potential lingering effects of the vibration following an acute increase in corticospinal excitability immediately following the vibration.

#### *3.4.2 Tonic Vibration Reflex and Muscle Length*

Abduction/adduction force generated during vibration differed across muscle lengths. A tonic contraction of the FDI elicited by vibration corresponds to an abduction force and would indicate a tonic vibration reflex (TVR; Matthews, 1966b). This was only observed in the longest muscle length condition, which supports the early findings by Eklund and Hagbarth (1966) and Eklund (1971) that the TVR force produced by the

biceps brachii muscle was largest at the most lengthened muscle position. The presence of TVR is thought to be mediated by muscle spindle activity (Matthews, 1966a), and muscle vibration of a stretched muscle has been shown to yield larger muscle spindle output compared to vibration at a normal muscle length (Burke, et al., 1976). Therefore, it is likely that the TVR produced by the lengthened muscle in the current study is due to the increased muscle spindle activation compared to the non-stretched muscle lengths. This study employed peak to peak vibration amplitude of 0.5 mm and muscle belly vibration rather than muscle tendon vibration, both of which are not known to elicit a tonic vibration reflex on a regular basis. Thus, it is possible that the increased sensitivity of the muscle spindles at the longest muscle length position was required to elicit a TVR, and may be the explanation for why the TVR was not elicited at the other muscle lengths. It is interesting to note that at the longest muscle length condition, more participants reported feeling the index finger moving in the abduction direction, which is in the opposite direction of proprioceptive illusions generally elicited by vibration (Goodwin, et al., 1972). This may suggest that the presence of reflex activity is strong enough to produce a conscious awareness of the muscle activity in the participant, thus leading them to report index finger abduction. There was no relation between TVR and corticospinal excitability in the lengthened muscle condition. This lack of relation is contrary to the findings of Claus et al, (1988) who found that the presence of a TVR facilitates corticospinal excitability during vibration. The differences between the two results could be due to different methodologies. Specifically, Claus et al (1988) examined corticospinal excitability during vibration, whereas in the current study, corticospinal excitability was

examined after vibration. This is relevant because the TVR is known to dissipate within the few seconds after termination of vibration (Marsden, et al., 1969).

The force results observed at the other muscle lengths suggest an opposite effect of vibration, such that it produced adduction force during the vibration period. This may indicate the presence of an antagonistic vibratory response (AVR), whereby vibration causes a force to be produced in the muscle antagonistic to the muscle vibrated (Calvin-Figuere, et al., 1999). This response is generally accompanied with a vibration-induced illusion of movement (Calvin-Figuere, et al., 1999) which was frequently observed in this study, although there were many occasions that participants did not report any proprioceptive illusions and still elicited an adduction force. This may suggest, contrary to suggestions by Calvin-Figuere, et al. (1999), that an AVR could potentially occur without the conscious movement illusion accompanying it. Given the force responses that were observed in both the abduction and adduction directions, the results seen here suggest that when measuring the TVR induced force, it may be worthwhile to ensure that force is being measured in the direction of action of both the vibrated muscle and its antagonist muscles, in case an AVR is elicited.

There was a positive relation between adduction force during vibration and corticospinal excitability following vibration that appeared to strengthen as muscle length decreased. At these muscle lengths, more participants reported incidence of adduction illusions than abduction illusions. In many cases, the incidence of proprioceptive illusion is followed by an illusory aftereffect, whereby the participant feels the illusion of the joint position returning to normal (Kito, et al., 2006). As corticospinal excitability was measured following vibration, it is possible that excitability was measured during the

illusory aftereffect. During the illusory aftereffect, it has been shown that corticospinal excitability in the vibrated muscle is increased (Kito, et al., 2006), similar to the results seen here.

### *3.4.3 Limitations*

The most significant limitation of the current study was the large across-subject variability which led to reduced statistical power. This is likely due to several issues, including 1) small subject numbers (see sample size calculation in Section 4.1), 2) the small number of MEPs collected for each experimental condition and 3) variability in TMS coil placement. The sample size calculation performed suggested 11 participants, thus we believe the main reasons for the variability are due to the second and third issues above. Specifically, it is well known that there is a large amount of intra-subject variability (Maeda et al, 2002; see Table 1) from pulse to pulse in many TMS paradigms, and the number of MEPs collected for each individual may not have been enough to elicit a consistent MEP average. The large amount of variability exhibited within the participant responses potentially led to unstable and inaccurate mean measures of excitability. This could in turn lead to inaccurate and highly variable means across participants. The other limiting factor is the fact that the stimulating coil was manually held on the optimal stimulating site during the experiments, meaning that the coil placement was subject to experimenter error in the coil position. This could lead to further variability across the MEPs collected.

#### *3.4.4 Conclusion*

While there is evidence that suggests that muscle length and vibration can influence corticospinal excitability, the concomitant effects of each have not been established. The results seen in this study suggest that vibration at longer muscle lengths can increase corticospinal excitability, and this difference is most likely attributed to the known increase in muscle spindle activation. However, evidence obtained during this study suggests that there are other factors involved in modulating corticospinal excitability, which are not clearly understood. Lack of statistical power is likely responsible for some of the lack of clarity in these results. Future research should be directed at further understanding the relationship between muscle vibration and muscle lengths, as the findings may prove useful for practitioners in the medical, rehabilitative, and exercise community.

## CHAPTER FOUR: GENERAL DISCUSSION

The increase in the public interest of vibration as an exercise tool in recent years can be attributed to the beneficial effects observed by those in the fitness industry. Lesser known benefits have been seen by clinicians in a rehabilitative setting. The potential benefits are vast, including improvements in bone density, increased growth hormone release, oxygen metabolism and adaptations within the nervous system. The enthusiasm seen by the fitness and rehabilitation public towards vibration has encouraged many researchers to further study its effects on the function of the nervous system. While there are significant benefits seen by vibration as a rehabilitative or exercise tool, there are also known detriments to long term exposure to vibration. Unfortunately, there has not been a systematic approach to studying vibration, which has led to unclear and conflicting results. The current study set out to fill one of the many gaps in the vibration literature.

Muscle vibration is known to excite muscle spindle afferent receptors, whose physiological role is to signal position sense by altering discharge rate based on muscle length. Vibration-induced increases in muscle spindle activation are known to influence spinal reflexes, and can also influence cortical processing. Both vibration and muscle length position are known to influence corticospinal excitability, however until this current study, the combined effects of both has never been evaluated.

The current study used single pulse transcranial magnetic stimulation to evaluate how both vibration and length of a small hand muscle together affect corticospinal excitability. It was found that corticospinal excitability measures increased following vibration and vibration-induced reflexes occurred when the muscle was at its most

lengthened position. Incidences of movement illusions induced by vibration were also present, with the largest incidence of movement illusions occurring in shorter muscle length conditions. Thus, it appears that the effects of vibration on the central nervous system differ as a function of muscle length. Therefore, the length of a muscle can be an important variable to consider when administering vibration.

Specific adaptations observed in this study are generally attributed to an increase in muscle spindle activity during vibration at the longer muscle lengths rather than the shorter muscle lengths. Corticospinal excitability measures the excitability of all neural tissue along the corticospinal tract, therefore the precise area(s) of adaptation along the corticospinal pathway is/are not discernible. Future research using other TMS protocols could provide more insight into specific locations of adaptation (see below).

The observations from this study and others like it may be very useful for individuals in the medical, rehabilitative, exercise, and research community, where vibration is used. Practitioners might like to consider muscle posture as a variable when using vibration as a method of treatment. Depending on the desired results, muscle vibration may have more robust effects if it is used at a specific muscle length or limb position. Similarly, if researchers are interested in evaluating the effects of proprioceptive illusions, they might wish to consider a muscle length where they are more commonly elicited. As this stream of research continues to gather momentum and popularity, there are certainly some areas of research in need of additional examination to further aid professionals in the fitness and performance realm. Further discussion of those areas is presented below.

#### 4.1 Limitations

The most significant limitation of the current study was the large across-subject variability which led to reduced statistical power. This is likely due to several issues, including 1) small subject numbers, 2) variability in joint angle determination due to different hand sizes across participants, 3) the small number of MEPs collected for each experimental condition and 4) variability in TMS coil placement. First, the sample size calculation performed (Equation 1) suggested 11 participants, thus we believe there were enough participants to achieve significance.

$$\begin{aligned} n &= (SD^2 \times [Z_\alpha + Z_\beta]^2) / \Delta^2 \\ n &= (98^2 \times [1.96 + 0.84]^2) / (86)^2 \\ n &= 10.18, \text{ rounded up to } 11. \end{aligned} \quad \text{Equation 1}$$

Where  $n$  is sample size,  $Z_\alpha$  is the Z score corresponding to 0.05,  $Z_\beta$  is the Z score corresponding to 0.80,  $\Delta$  is the difference between the two means, and SD is the standard deviation of the means. Note the data for SD and  $\Delta$  were taken from Siggelkow et al. (1999).

It is well known that there is a large amount of inter- and intra-subject variability from pulse to pulse in many TMS paradigms (Maeda, et al., 2002), and the number of MEPs collected for each individual may not have been enough to elicit a consistent response. Other researchers have employed pulses in blocks of trials to reduce the intra-subject variability, and enhance reliability/consistency of the measurement. In cases where time points are necessary, several trials at each time point are repeated to have a larger number of pulses available for averaging. Previous researchers have used blocks of 60 trials (Steyvers, Levin, Van Baelen, et al., 2003), while others have used as low as 5

(Kamen, 2004). It has been reported that blocks of 5 or more averaged trials have good to high reliability, however the optimal number of trials is 10 (Bastani & Jaberzadeh, 2012), which suggests a possibility for inconsistent MEP measures in the current study.

The other limiting factor is the fact that the stimulating coil was manually held on the optimal stimulating site during the experiments. Specifically, the positioning of the TMS coil on the subject's head was done by simply aligning it with marks drawn on the rubber cap and the experimenter physically holding the coil in place. As the potential stimulation sites are spaced one cm<sup>2</sup> apart, this method leaves a great deal of empty space on the scalp that potentially is more localized to a specific motor hotspot. Next, the fact that the experimenter was required to hold the coil in place on the head of the study participant likely introduced error in angular positioning of the coil, potentially producing larger intra-subject variability in MEPs. There are commercially available systems that use 3-D infrared markers to establish and maintain the optimal coil position throughout the testing protocol (i.e. Brainsight, Northern Digital TMS Manager). Brainsight also uses functional magnetic resonance imaging to identify the hotspot on the cortex for the particular muscle which is then able to be integrated into the coil positioning systems for ensuring the most possible consistency of coil placement. These systems come with multi-jointed clamping arms available as well to ensure that the coil stays in the correct spot, once the position is chosen. Unfortunately, the costs of these systems make their purchase unlikely for many laboratories that wish to use TMS as a tool for studying neurophysiology.

Another possible source of inter- and intra-subject variability is the fact that individuals have widely varying hand sizes and experimental manipulandum used for the study needed to account for this. As the device was required to accommodate a variety of hand sizes, the four joint angles used in this study were chosen such that the range of motion between the longest and shortest muscle length conditions accommodated as many individuals as possible, while still maintaining a meaningful a range of motion. It is very likely that the actual range of motion of every participant is somewhat different than what was chosen for the study. It is therefore possible that a  $-10^{\circ}$  joint angle for one individual might be well within normal range of motion, while in another individual it could be at the limit of range of motion. This could certainly have an impact on across-subject variability in our results. It is also possible that the position of the vibrating probe that pressed upon the muscle might have influenced the effects of vibration (and therefore intra-subject variability) as the probe needed to be individually placed against the muscle every trial. Due to hand size variability, the position of the probe could have been placed in slightly different areas on the muscle belly, which in turn could affect muscle spindle receptors differently. Although measures were taken to affix the probe to the largest part of the muscle belly, it is possible that there were individual differences with respect to probe placement, potentially leading to a differential stretch of the FDI muscle.

Many studies presently utilize EMG response when measuring the effects of a TVR (Nakajima, Izumizaki, Sekihara, Atsumi, & Homma, 2009), as it gives a more direct measure of muscle activity, rather than force production. However, the most effective way to measure this is by expressing EMG relative to the EMG produced during a maximal voluntary contraction, which greatly reduces variability across subjects. As

studying TVR was not a primary aim of this study, MVCs were not measured, rendering this method of analysis unusable. Therefore, force analysis was used for the measure of TVR.

#### ***4.2 Future Directions***

Future research in this area should be directed towards systematically examining the variables related to muscle length, vibration, and corticospinal excitability. However, it is of utmost importance to rectify the limitations mentioned above as these future endeavours are pursued. In that vein, the top priority for studying the effects of muscle length and vibration would be to make every effort to reduce intra-subject variability in the motor evoked potential responses in the future. Further potential research pathways are discussed below.

##### ***4.2.1 Microneurography***

Although Burke et al (1976) determined that muscle spindle discharge due to vibration can be altered by lengthening the vibrated muscle, there has yet to be a study performed that looks at both spindle discharge, through microneurography, and corticospinal excitability when vibrating a muscle at varying lengths. Therefore, it would be beneficial for a study to evaluate both these variables together such that the changes in both corticospinal excitability and muscle spindle discharge and their potential relationship could be more directly evaluated.

##### ***4.2.2 Paired pulse TMS paradigms***

Single pulse TMS is used to evaluate corticospinal excitability throughout the corticospinal tract from the cortex to the muscle. Thus single pulse TMS cannot

determine the level at which adaptations occur along the corticospinal tract. However, paired pulse TMS is a measurement paradigm that is able to more specifically evaluate effects of adaptation (Kujirai et al., 1993) at the cortical level. Previous paired pulse TMS studies have shown that vibration can decrease intracortical inhibition, which leads to an increase in corticospinal excitability (Rosenkranz & Rothwell, 2003), however the compound effects of vibration and muscle length on specific cortical adaptations has yet to be evaluated.

#### *4.2.3 Static versus dynamic movement*

Previous research has demonstrated that corticospinal excitability is modulated more so when a muscle is dynamically lengthening and shortening rather than being held in a static position (Lewis, et al., 2001), however there has yet to be a study examining the effects of vibration on corticospinal excitability during movement. Therefore, the first step is to systematically add vibration to a protocol which has examined corticospinal excitability through passive range of motion, such as the study performed by Lewis et al, (2001). Next, add vibration to a protocol which has examined corticospinal excitability through active range of motion, such as the study performed by Chye et al (2010). The effects of dynamic movement on corticospinal excitability are further enhanced when the muscle is undergoing a contraction compared to when it is passively going through range of motion. However, vibration under these conditions has not been evaluated. Thus, it may be worthwhile to examine how vibration of an active muscle compared to a passive muscle undergoing dynamic movement, which would encompass a large number of variables known to influence muscle spindle activity.

#### *4.2.4 Agonist versus antagonist relations*

As mentioned in Chapter One, there have been studies that evaluated the effects of agonist and antagonist muscle pairs on corticospinal excitability following vibration. Given that corticospinal excitability measured in agonist and antagonist muscles differ in response to vibration, it would be worthwhile to examine muscle pairs under any manipulation (see above).

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## APPENDIX A: EDINBURGH HANDEDNESS INVENTORY

Last Name: \_\_\_\_\_ Given Names: \_\_\_\_\_

Date of Birth: \_\_\_\_\_ Sex: \_\_\_\_\_

Please indicate your preferences in the use of hands in the following activities by putting '+' in the appropriate column. Where the preference is so strong that you would never try to use the other hand unless absolutely forced to, put '++'. If in any case you are really indifferent, put '+' in both columns. Some of the activities require both hands. In these cases the part of the task, or object, for which hand preference is wanted is indicated in brackets. Please try to answer all of the questions, and only leave a blank if you have no experience at all of the object or task.

		LEFT	RIGHT
1.	Writing		
2.	Drawing		
3.	Throwing		
4.	Scissors		
5.	Toothbrush		
6.	Knife (without fork)		
7.	Spoon		
8.	Broom (upper hand)		
9.	Striking Match (match)		
10.	Opening Box		
i.	Which foot do you prefer to kick with?		
ii.	Which eye do you use when using only one?		

LQ Value =

The LQ value is the total number of +'s for the RIGHT hand boxes, less the total number of +'s for the LEFT hand boxes, divided by the total +'s in both RIGHT and LEFT hand boxes.

**APPENDIX B: TRANSCRANIAL MAGNETIC STIMULATION ADULT  
SAFETY SCREEN (TASS)**

**Transcranial Magnetic Stimulation Adult Safety Screen (TASS)**

Name:
Age:
Date:

*Please answer the following:*

Have you ever:

Had an adverse reaction to TMS? Yes No

Had a seizure? Yes No

Had an electroencephalogram (EEG)? Yes No

Had a stroke? Yes No

Had a serious head injury (include neurosurgery)? Yes No

Had any other brain-related condition? Yes No

Had any illness that caused brain injury? Yes No

Do you have any metal in your head (outside the mouth) such as shrapnel, surgical clips, or fragments from welding or metalwork? Yes No

Do you have any implanted devices such as cardiac pacemakers, medical pumps, or intracardiac lines? Yes No

Do you suffer from frequent or severe headaches? Yes No

Are you taking any medications? Yes No

Are you pregnant, or is it possible that you may be pregnant? Yes No

Does anyone in your family have epilepsy? Yes No

Do you need further explanation of TMS and its associated risks? Yes No

*If you answered **yes** to any of the above, please provide details (use reverse if necessary):*

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## **APPENDIX C: INFORMED CONSENT**

**TITLE:** The effects of muscle belly vibration at varying muscle lengths on corticospinal facilitation and inhibition in normal healthy adults.

**SPONSOR:** University of Calgary

**INVESTIGATORS:** Dr. Jamie Johnston (Professor of Kinesiology)  
Michael Lane

This consent form is only 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. If you would like more detail about something mentioned here, or information not included here, please ask. Take the time to read this carefully and to understand any accompanying information. You will receive a copy of this form.

### **BACKGROUND**

It is widely understood that the immediate increase in strength and performance by resistance training is due to an adaptation in the nervous system, rather than an increase in muscle contractile tissue. However, the mechanism by which this occurs is not clearly understood. The proprioceptive system is used to provide feedback to the brain about the body's position in space. There is evidence that suggests vibrating a muscle alters proprioceptive input to the nervous system changing its activity. After a certain period of time, this alteration in the nervous system's activity can lead to increases in performance parameters in target muscles, such as force production, rate of force development, and declines in fatigue.

### **WHAT IS THE PURPOSE OF THE STUDY?**

The purpose of this study is to examine how applying vibration to an intrinsic hand muscle can alter the neural activity responsible for activating that muscle, and how the neural activity changes at different muscle lengths. The results from this study will allow us to better understand the physiology of the nervous system and foster developments in the fields of exercise prescription, sport science, neural rehabilitation, and basic medicine.

### **WHAT WOULD I HAVE TO DO?**

If you decide to participate, you will be asked to participate in four sessions, the first session lasting approximately 1.5 hours, while the additional sessions approximately 60 minutes. For each session, you will sit comfortably at a table and we will affix your right hand to an apparatus that isolates an intrinsic muscle in your hand, the first dorsal interosseous (FDI) muscle. This muscle will be vibrated using a probe attached to an electromagnetic piston that will repeatedly compress the muscle. Note that this procedure

is entirely noninvasive, and at most you will feel a tingly sensation. This should never be painful or uncomfortable. Before and after vibration, we will apply transcranial magnetic stimulation (TMS) to the area of your motor cortex that is responsible for your right hand FDI. The first session will be spent locating the correct area of your motor cortex that is responsible for the right FDI, which will require single TMS pulses at low intensity on various areas on your motor cortex. Note that this entire procedure is noninvasive, however you may experience transient feelings of dizziness, giddiness or disorientation. Note while there might be brief disorientation after TMS, there are no known serious or long term risks for healthy individuals and that it is believed that TMS is safe and well tolerated. The experimental session will be performed at University of Calgary's Neurophysiology of Movement Laboratory, Kinesiology B room 2223. Approximately 10 subjects will be participating in this study.

### **WHAT ARE THE RISKS?**

While the experimental sessions may be tedious, there are no risks to you. There are no risks of electric shock from electromyography and the intensities and frequencies of the transcranial magnetic stimulation used in the present study are safe and pose no risk to a healthy individual. Any individuals who have suffered seizures or have had a previous brain injury or implanted medical device in their body are not eligible for TMS, as the magnet may induce adverse effects. If you fall into this category, you are not eligible for this study. However, you may experience an unnatural sensation at the time of stimulation that may cause you minor discomfort, feelings of dizziness, giddiness or disorientation. If you are tired and need a break, please inform the experimenter and a break will be provided. In addition, rest breaks will be scheduled throughout the study.

### **WILL I BENEFIT IF I TAKE PART?**

If you agree to participate in this study there may not be a direct benefit to you. However, the investigators believe that the results of the proposed research will have important implications for sport science developments and neural rehabilitation using muscle vibration as a tool.

### **DO I HAVE TO PARTICIPATE?**

You absolutely are under no obligation to participate. It is okay for you to say no at any time prior to or during your participation. If you agree to participate now, you are still free to withdraw from the study at any time. Your participation is voluntary, greatly appreciated and nonparticipation or withdrawal from the study will not affect your relationship with the University of Calgary or the investigators.

### **WILL I BE PAID FOR PARTICIPATING, OR DO I HAVE TO PAY FOR ANYTHING?**

You will receive \$30 to compensate you for the time you have spent completing this study. In addition, the investigators will provide you with any information regarding the background, purpose, design, or any other details of the study. The investigators will provide this information upon your request at a mutually convenient time. There will be no financial burden for your participation in this study.

**WILL MY RECORDS BE KEPT PRIVATE?**

All information obtained in this study is strictly confidential. The results of this research study may be used in reports, presentations, and publications, but the researchers will not identify you. In order to maintain confidentiality of your records, the investigators will employ only experiment numbers to refer to the data from each subject and all records will be stored in a locked filing cabinet accessible to only those researchers listed above. This identification and only this will accompany any communications, published or unpublished regarding these experiments.

**IF I SUFFER A RESEARCH-RELATED INJURY, WILL I BE COMPENSATED?**

In the event that you suffer injury as a result of participating in this research, no compensation will be provided to you by the University of Calgary or the Researchers. You still have all your legal rights. Nothing said in this consent form alters your right to seek damages.

**SIGNATURES**

Your signature on this form indicates that you have understood to your satisfaction the information regarding your participation in the research project and agree to participate as a subject. In no way does this waive your legal rights nor release the investigators, or involved institutions from their legal and professional responsibilities. You are free to withdraw from the study at any time without jeopardizing your health care. If you have further questions concerning matters related to this research, please contact:

Dr. Jamie Johnston (403) 220-3649

If you have any questions concerning your rights as a possible participant in this research, please contact: Director, Office of Medical Bioethics, University of Calgary, at 403-220-7990.

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**Participant's Name**

**Signature and Date**

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**Investigator/Delegate's Name****Signature and Date**

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**Witness' Name****Signature and Date**

A signed copy of this consent form has been given to you to keep for your records and reference.

## APPENDIX D: POST-VIBRATION PROPRIOCEPTIVE FEEDBACK

### QUESTIONNAIRE

1. Did you sense any kind of index finger movements during the vibration periods?

Circle One: **YES**    **NO** If yes, in what direction? \_\_\_\_\_

2. Did you sense any other digit movements during the vibration periods?

Circle one: **YES**    **NO** If yes, which digit(s)? \_\_\_\_\_

In what direction(s)? \_\_\_\_\_

**If you answered "NO" to both questions 1 and 2, go to question 9.**

3. In your opinion, describe how strong/intense these movements felt (circle one):

VERY WEAK

VERY STRONG

1      2      3      4      5      6      7      8      9      10

4. In your opinion, describe how real/vivid these movements felt (circle one):

NOT VIVID

VERY VIVID

1      2      3      4      5      6      7      8      9      10

5. When did the sense of movement start?

a) Started immediately at onset of vibration

b) Started in the middle of the entire 5 minute vibration period

6. When did the sense of movement end?

a) Ended immediately at termination of vibration

b) Lasted longer than vibration period

7. Did you ever sense a change in movement direction? Circle one:    **YES**    **NO**

Are there any other sensations in your hand that you felt during the vibration period?

Please explain:

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