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NEUROMUSCULAR RESPONSE OF THE TRUNK FOLLOWING INERTIAL-BASED PERTURBATIONS WITH WHOLE-BODY VIBRATION EXPOSURE

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By

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ABSTRACT

The purpose of this study was to evaluate the effects of vibration exposure on the neuromuscular responses to inertial-based trunk perturbations. Thirteen, male participants ($\bar{x}$ = 22.5 yrs ± 3.2) were assigned to one of two experimental groups: 1) participants not exposed to vibration (control group – CG, n=6), and, 2) participants exposed to vibration (vibration group – VG, n=7) throughout the protocol. Participants experienced 40 perurbations, of which half were in known and unknown directions. Data from trunk sEMG, motion capture markers and seat accelerometers were anaylzed. Repeated measures ANOVA with Tukey's post hoc test were used to determine statistical significance ($p<0.05$). Participants in CG had a 14% faster muscle onset time than VG. Antagonistic muscle onset times were faster than agonists in both groups. Perturbations of known direction did not show any anticipation effects both in sEMG amplitude and in $L_{4\text{-}5}$ joint angle.

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LIST OF ABBREVIATIONS

AP: Action Potential

ATP: Adenosine Triphosphate

Ca^{++}: Calcium Ion

CG: Control Group

CNS: Central Nervous System

EMG: Electromyography

EO: External Oblique

EPSP: Excitatory Post-synaptic Potentials

g: Acceleration of Gravity

GTO: Golgi Tendon Organ

hr: Hour

Hz: Hertz

IO: Internal Oblique

ISO: International Organization for Standardization

JRS: Joint Rotational Stiffness

L_4: Fourth Lumbar Vertebra

L_5: Fifth Lumbar Vertebra

LATS: Latissimus Dorsi

LBI: Low Back Injury

LBP: Low Back Pain

LES: Lumbar Erector Spinae

LHD: Load-haul Dump

m/s: Meter per Second

M1: Short Latency Response

M2: Longer Latency Response

min: Minute

ms: Millisecond

MU: Motor Unit

MUAP: Motor Unit Action Potential

MULT: Multifidus

MVC: Maximum Voluntary Contraction

MVE: Maximum Voluntary Exertion

N: Newton

OR: Odds Ratio

RA: Rectus Abdominis

RMS: Root Mean Square

S_1: First Sacral Vertebra

sec: Second

sEMG: Surface Electromyography

T_{12} : Twelfth Thoracic Vertebra

T-reflex: Tendon Tap Reflex

TES: Thoracic Erector Spinae

TVR: Tonic Vibration Reflex

μN: Micronewton

VG: Vibration Group

VWF: Vibration White Finger

WBV: Whole-body Vibration

Chapter 1

INTRODUCTION

1.1 BACKGROUND

Workplace vibration exposure is a result of human machine interactions associated with many occupations from dentistry, to gardening, which expose workers to hand-arm vibrations; to automotive mechanic and forestry occupations which expose workers to WBV exposure (Rytkönen, Sorainen, Leino-Arjas, & Solovieva, 2006; Palmer, Giffin, Bendall, Pannett, & Coggon, 2000; Neitzel & Yost, 2002). The exposure time of occupational-based vibrations can vary from less than one minute (min) of tooth drilling (Rytkönen et al., 2006) to a 12 hour shift aboard a vibrating load-haul dump (LHD) mining vehicle (McPhee, 2004). Research has shown that vibration exposure frequency and duration are direct factors that increase the risk associated with work-related injury (Bovenzi, 1996; McPhee, 2004; Eger, Salmoni, Cann, & Jack, 2006).

Whole-body vibration (WBV) exposure has been shown to have negative consequences on the digestive system, the genital/urinary system and female reproductive organs (International Organization for Standardization, 1997). However, the spine, particularly the low-back, appears to be the most common area of complaint or injury induced by chronic WBV (Bovenzi, 2009). The risk of a disc herniation and its severity, for example, were greater in people who have occupations in the transportation industry, compared to those who do not have these types of occupations (Kelsey & Hardy, 1975). It was hypothesized that this is due to the constant exposure to vibration as well as static sitting postures (Kelsey & Hardy, 1975). Odds ratios for low back pain (LBP) in the transportation industry (exposed to WBV) were 1.6-3.7 when compared to control groups who were not exposed to WBV (Bovenzi, 1996). From an economic

standpoint, health care costs related to LBP and low back injuries (LBI) in Canada are an estimated $6 to $12 billion per year (Brown et al., 2005). In light of the risk of injury associated with WBV, the International Organization for Standardization (ISO) created ISO 2631-1: Mechanical vibration and shock—Evaluation of human exposure to whole-body vibration, for periodic, random and transient vibration exposure, in an attempt to quantify WBV as it relates to reducing the incidence of WBV-related injuries. Field-based studies have compared occupational-based WBV exposures to the ISO 2631-1 standards; workers were still becoming injured despite being exposed to vibrations within these standards (Bovenzi, 2009; Grenier, Eger, & Dickey, 2010).

Significantly longer delays in the neuromotor response of muscle activation have occurred in occupations with WBV exposure, and are a theory of why LBP occurs (Arashanapalli & Wilson, 2008). In addition, vibration exposure also causes changes in the recruitment of muscle fibres (Desmedt & Godaux, 1978), potentially leading to uncoordinated responses and disturbances in stability of the trunk, thus increasing the potential for injury (Brown, Vera-Garcia, & McGill, 2006). Cholewicki et al. (2005) determined that for every millisecond (ms) delay in abdominal muscle shut-off there was a 3% increased risk of developing a LBI. It was also found that those who were susceptible to LBI had significant delays in their neuromotor reflex responses (Cholewicki et al., 2005). Presynaptic inhibition of muscle spindle fibres due to the tonic vibration reflex (TVR) is thought to be why this delay occurs (Desmedt & Godaux, 1978). TVR is an involuntary muscle reflex that causes increased muscle tone and thus, increased muscle activation to segments exposed to vibration; it is thought to affect a muscle lengthening illusion that occurs with vibration exposure (Latash, 2008). This illusion causes a person to feel as though the muscle being exposed to vibration is lengthening; it

has the potential to lead to improper positioning of the pelvis and lumbosacral spine during seated vibration due to distortion of the proprioceptive senses, which can lead to an injury (Brumagne, Lysens, Swinnen, & Verschueren, 1999).

The proprioceptive system has been described as a combination of the joint position sense, kinesthesia and the ability to sense force changes in the body (Dover & Powers, 2003). This sense is important to stability (Brumagne, Cordo, & Verschueren, 2004) as it provides feedback on the orientation of the appendicular and axial segments (Lephart, Pincivero, Giraldo, & Fu, 1997). The feedback is delivered from somatosensory receptors, specifically the muscle spindles, Golgi tendon organs (GTOs) and cutaneous mechanoreceptors. The muscle spindles sense the change in length of the muscles, as well as the rate of this length change (Shaffer & Harrison, 2007). The GTOs detect tension changes in the muscles and ligaments; they function to protect the muscle from excessive stress and strain (Lephart et al., 1997). Lastly, the cutaneous mechanoreceptors deliver information about the external environment, specifically applied pressure to the skin, and are important for the joint position sense (Shaffer & Harrison, 2007). Interestingly, all of the aforementioned somatosensory receptors have altered responses when exposed to vibration (Lundstrom & Johansson, 1986). These receptors are especially important as the information they deliver begins the cascade of events that lead to motion through voluntary or involuntary movements.

Neuromuscular responses can vary in organizational structure from mono to polysynaptic and result in simple to complex movements. Involuntary movements, also known as reflexes, are quick responses that are not consciously controlled. In comparison, voluntary movements are those which can be consciously controlled by the person performing the action, yet, they take longer to occur. Nonetheless, working in tandem, voluntary and involuntary muscle responses

collectively act to help maintain structural stability of the body (Stokes, Gardner-Morse, Henry, & Badger, 2000). For example, when static equilibrium is lost in the trunk due to a disturbance, preparatory voluntary muscle activation of the abdominal muscles increases spinal stiffness (Stokes et al., 2000), and the involuntary spinal reflex for tension modification also becomes activated (Bergmark, 1989). Both systems work together in an attempt to regain equilibrium and maintain spinal stability. Stability is vital to the health of the spine as it allows for proper transmission of the compressive and shear forces from a perturbation to the lower body, rather than solely exposing the spine to these forces and increasing its risk of injury (Cholewicki & McGill, 1996).

1.2 STATEMENT OF THE PURPOSE

The purpose of the current study was to evaluate the effects of vibration exposure on the voluntary and involuntary neuromuscular responses of muscles prior to, during and following inertial-based trunk perturbations. An outcome of this study was to provide knowledge on a possible mechanism, among the cascade of events, which causes LBP with vibration exposure. There have been many hypotheses offered on the etiology of LBP with vibration exposure, though, its true origin is still unknown (Wilder & Pope, 1996). Some of these theories are: vibration having a negative effect on metabolic costs due to muscle fatigue; vibration causing increased intradiscal pressure due to exciting resonant frequencies and; a creep response occurring in the spine tissues due to the stress of vibration (Wilder & Pope, 1996). A better understanding of the neuromuscular effects due to WBV within the human body will facilitate further knowledge and the development of strategies to reduce its negative implications. These strategies will help to prevent some of the negative financial, emotional, physical and economical impacts that LBP has on individuals and society.

1.3 HYPOTHESES

1) *Longer muscle reflex response latencies will occur during the pre-voluntary time period for those in the vibration group (VG), when compared to the non-vibration control group (CG).*

Participants in the VG were exposed to vibrations throughout the perturbation protocol; exposure time was up to 15 seconds (sec) prior to each of the 40 perturbations. Research has found that vibration exposure has led to longer latencies in the reflex response time due to a perturbation (Wilder et al., 1996; Arashanapalli & Wilson, 2008). Delays in the neuromuscular response time can lead to an increased risk of injury, as the muscular responses may become uncoordinated and negatively impact stability (Brown et al., 2006; Cholewicki et al., 2005).

2) *Surface electromyography (sEMG) onset timing for muscles whose actions oppose the direction of force of the perturbation will occur sooner than those who act with the direction of the perturbation..*

Recordings of sEMG were completed on the following seven bilateral muscles of the trunk: rectus abdominis (RA), external obliques (EO), internal obliques (IO), latissimus dorsi (LATS), thoracic erector spinae (TES), lumbar erector spinae (LES) and multifidus (MULT). Research by Cort and Potvin (2012) determined that the greatest muscle contributions to joint stability came from the muscles whose actions oppose the direction of perturbation, specifically during the pre-voluntary time period. Similar results were anticipated for this study. For example, during the forced flexion perturbation direction it was anticipated that the back extensor muscles would have the earliest onset considering their primary actions cause the trunk to oppose the direction of force caused by the perturbation.

3) *Increases in muscle activation were expected to occur during perturbations of known direction, when compared to those of unknown direction, during the pre-perturbation time period.*

An anticipation effect in the muscles, through increased activation, was expected to occur prior to the perturbation to stabilize the spine from the forced motion, in an attempt to reduce the displacement of the trunk (Vera-Garcia, Brown, Gray, & McGill, 2006; Vera-Garcia, Elvira, Brown, & McGill, 2007; Brown & McGill, 2008). This anticipation effect allows the participants to optimize the stability of their trunk, while creating the best cost-benefit relationship for compressive forces at the lumbar spine as participants reduce movement of the trunk after a perturbation, thereby reducing the increased retroactive forces from muscle activation post-perturbation (Vera-Garcia et al., 2007). It was hypothesized that increasing the muscular activation would also increase the activity of the gamma system, being the muscle spindles, which would increase the intensity of the reaction from the body, thus enhancing stability (Vera-Garcia et al., 2007).

4) *Decreased $L_{4\text{-}5}$ joint angle was expected to result during known direction perturbations in comparison to those in the unknown direction, after the perturbation occured.*

Participants' trunk angle was measured throughout the perturbation protocol during both known and unknown direction perturbations, whereby the participant's $L_{4\text{-}5}$ joint angle was estimated from the total trunk angle using the methods described in Cort, Dickey and Potvin (2013). Studies have shown that during the anticipation of a perturbation, participants have increased muscle activation, which led to increased stiffness, and a reduction in the movement of the trunk in the direction of the perturbation (Chiang & Potvin, 2001). The reduction in movement from

the perturbation may lead to a decreased risk of injury to the individual by preventing the buckling of intersegmental vertebral muscles.

Chapter 2

LITERATURE REVIEW

The following literature review will provide the reader with a comprehensive understanding of the effects of vibration on the neuromuscular system. Specifically, the variables that mediate the incidence of injuries, particularly LBP, in humans exposed to vibration will be explored, as the etiology of LBP is still unknown. This review includes a thorough explanation of the following topics: muscle mechanics, sEMG, stability, neuromuscular responses, somatosensory receptors, vibration and vibration-induced injuries.

2.1 MUSCLE MECHANICS

Force generation in muscle is produced primarily through either shortening (concentric) or lengthening (eccentric) a series of parallel viscoelastic protein-based tissues, known as sarcomeres (Enoka, 2008). Sarcomeres are the basic contractile units of muscle tissue (Figure 1). Sarcomeres are made up of thick and thin filaments of protein, known as actin and myosin, which work together to produce the muscle forces associated with a contraction. A sarcomere becomes activated indirectly by an electrical signal, known as an action potential (AP), from a motor neuron. An AP is a change in the membrane potential of excitable tissues that acts as a unit of information transmission (Latash, 2008). The result of this information transmission to the muscle tissue causes a muscle contraction, through a process known as the sliding filament theory.

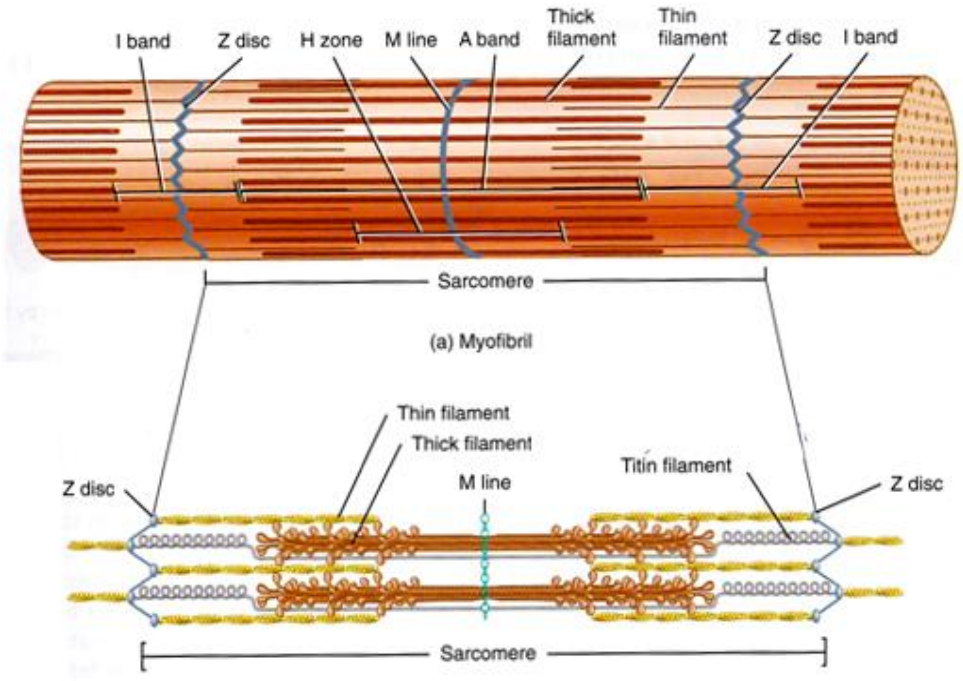

Figure 1. The arrangement of filaments within a sarcomere (Tortora & Nielsen, 2012).

2.1.1 Muscle Contraction

A motor neuron and the muscle fibres that it innervates are known as a motor unit (MU). The information transmission between the neural and muscular components of a MU results in the production of an end-plate potential at the muscular region, which can trigger a MU action potential (MUAP) (Enoka, 1994). An AP is an all-or-none response, meaning that once the end-plate potentials summate to reach a certain electrical threshold, the AP is generated. Each AP creates a muscle twitch; these twitches summate together to create a tetanus, or sustained contraction (Enoka, 1994). The primary steps that take place to create a muscle contraction are shown in Figure 2. A muscle twitch causes the release of Calcium (Ca^{++}), that was once stored in the sarcoplasmic reticulum (Enoka, 1994). The released Ca^{++} flows into the free space within the sarcomere, called the sarcoplasm. The Ca^{++} then binds to a troponin protein (found on the actin filament of the sarcomere) and triggers a reaction which changes its shape. This deformation

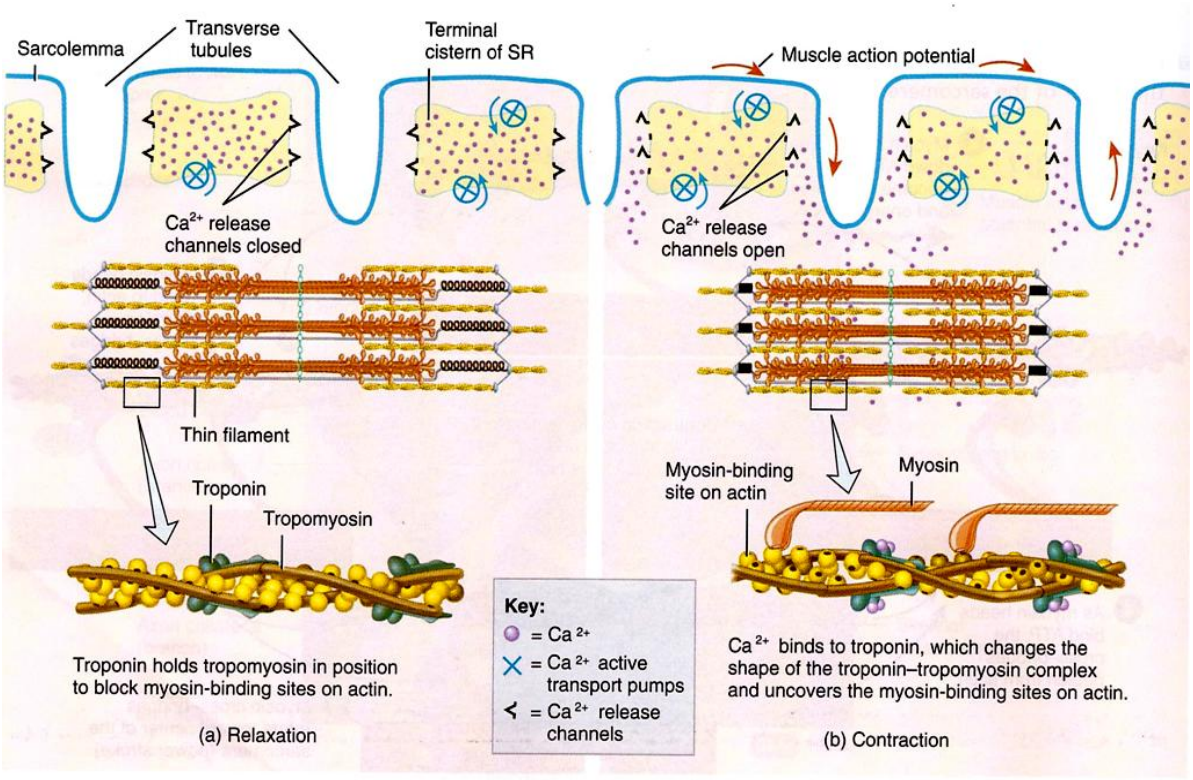

Figure 2. Steps involved in muscle contraction (Tortora & Nielsen, 2012).

initiates troponin to move the tropomyosin protein (also found on the actin filament) off the myosin binding sites of the actin filament. Once the binding sites are exposed, the myosin filament attaches itself to actin via its swiveling heads. Adenosine triphosphate (ATP) then binds to a myosin head and causes it to release from the actin filament. This ATP is hydrolyzed and the myosin head swivels and attaches to another binding site further along the actin filament. Each myosin filament is surrounded by six actin filaments; therefore, the myosin heads from one myosin filament attach to binding sites on many actin filaments simultaneously, causing continuous muscle contraction (Latash, 2008). During concentric contractions, the constant release and reattachment of the myosin heads along the actin filament cause overlap of the filaments and shortening of the sarcomere; this creates active muscle force production (Latash,

2008). The magnitude of voluntary, active muscle force, and thus tension, is mediated by the person performing the contraction and depends on the strength requirements for the effort.

2.1.2 Muscle Tension

When considering the mechanics behind increases in muscle tension, it is first important to understand the electrophysiology that occurs within a MU, thus affecting the output from the muscle. When motor neurons become excited they stimulate muscle fibres to produce MUAPs, which are APs specific to muscle tissue (Winter, 2009). If an increased force demand is required from a muscle to perform a task, the muscle responds by producing more tension. There are two ways to increase tension in an actively contracted muscle: increase the firing rate of a MU and recruiting additional MUs (Winter, 2009). Increasing the firing rate of a MU increases the number of APs that are being discharged from the unit, thus increasing the force being produced by the muscle. However, each MU has a maximum rate at which it fires; once it reaches this threshold, increasing stimulation to the unit will not cause a greater response or tension in the muscle (Winter, 2009). Increases in tension can also occur through MU recruitment. Recruitment of neighbouring MUs occurs when a single MU is reaching its maximal firing rate, but the necessary muscle force is not yet achieved (Winter, 2009). Recruitment occurs in a relatively fixed order, where the smaller, more excitable MUs are recruited first, followed by the larger, less excitable ones (Enoka, 2008). Through the recruitment of more MUs, greater muscle force and tension is produced. Furthermore, increases in the electrical charge across the muscle occur with more activation. This increase can be determined using a technique that measures the electrical activation from the muscle, known as electromyography (EMG).

2.1.3 Electromyography

The activation of MUs within a muscle, through the firing of APs, creates an electrical current that can be recorded as electrical signals; these signals quantify the activation of the muscle as a unit of voltage, in a methodological technique known as EMG. The greater the muscle activation, the greater the amplitude of electrical voltage, thus represented by the EMG signal. EMG recording is typically done using one of two methods. The first method uses indwelling-needle electrodes which require the insertion of a small wire into the muscle. The difference in voltage between the tip of the needle and the wire inserted into the muscle provides a recording of the electricity associated with a muscle contraction (Latash, 2008). The strength of this technique is that the wires are small enough to record multiple or even single MUs within a muscle (Latash, 2008). In contrast, the more common method used to record muscle activation is sEMG. The sEMG method uses mono or bipolar surface electrodes which adhere to the skin over the muscle belly. Since the recording is initiated at the skin's surface, it is not capable of measuring electrical activity of single MUs. The summation of electrical activity from all MUs in the area under the electrodes is recorded and the difference in potential between the two electrodes is amplified and recorded for that particular area of the muscle (Latash, 2008). Depending on the nature of research, one method is usually preferred over the other. Indwelling electrodes can provide specific characteristics on the activation of muscle fibres within a muscle, whereas sEMG can compare general activation of various, primarily superficial, muscles in the body. The results taken from EMG signals can be used to interpret various characteristics of a human system; commonly EMG magnitudes are used as an analog to stiffness and stability (Granata & Wilson, 2001).

2.2 STABILITY

In the context of biomechanics, the term stability has been used to classify a mechanical system's state or behaviour following a small kinematic disturbance (perturbation). Following a perturbation, if the system's behaviour is relatively the same as that observed prior to the perturbation, the system is stable (Reeves, Narendra, & Cholewicki, 2007). However, if the system's behaviour changes from its original state following the perturbation, it is considered unstable. There is no continuum on which a system can become more or less stable: stability is either present or absent (Reeves et al., 2007). Nonetheless, this definition of stability has come with some criticism (Reeves & Cholewicki, 2010). The lack of ability to quantify levels of stability, and thus system behaviour detail, does not provide the necessary information to understand how or why a system may have changed (Reeves & Cholewicki, 2010). Some researchers have attempted to address this issue by assessing stability as the change in potential energy of a system after a perturbation (Cholewicki & McGill, 1996; Brown & Potvin, 2005; Vera-Garcia et al., 2006; Brown et al., 2006; Vera-Garcia et al., 2007). However, this method is limited, as calculations can only be completed when the joint system is static (Potvin & Brown, 2005). Some researchers have used a dynamic approach where changes in joint kinematics have been used as analogs to calculate for the presence of joint stability (Chiang & Potvin, 2001). Further, another dynamic method to calculate stability quantifies the joint rotational stiffness (JRS), which is the 1st approximation of stability (Graham & Brown, 2012). A joint's ability to increase the JRS is augmented through an increase in muscle force production from both voluntary and involuntary muscle contractions (Cort & Potvin, 2012). The magnitude of these forces is often estimated through changes in sEMG amplitudes (Potvin & O'Brien, 1998). These

forces provide the joint with the capability to withstand kinematic disturbances and thus, increase joint safety through increasing the joint's stiffness.

2.2.1 Stiffness

The stiffness of the musculoskeletal system is produced both passively and actively: passive stiffness is created by ligaments, vertebral discs and elastic elements of muscle tissue that are lengthened or stretched without active muscle contraction, whereas active stiffness is generated from muscles being actively shortened or lengthened during muscle contraction. The sum of stiffness produced by these structures provides the forces that aid in maintaining joint stability. Passive tissues play a crucial role in spine stability under conditions that transmit small forces onto the spine system, such as standing in neutral posture (Cholewicki & McGill, 1996). In contrast, active stiffness increases stability in the presence of external loading at the joint, as it functions to reduce the forced movement. Active stiffness is often reported in studies from the muscles whose actions resist the direction of forced motion, known as antagonists. However, the muscles whose actions facilitate the vector of the external force, known as agonists, can increase stiffness of the system, through co-contraction, in an effort to reduce forced joint motion (Brown et al., 2006). Figure 3 explains the dynamics of the relationship between muscle activation, stiffness and stability. Figure 3*a* represents a system that contains no passive or active tissues and has no stability. Using the example of a ball, when perturbed, the ball would simply roll away as there is nothing to oppose the vector of applied force. Figure 3*b* represents a system that has only passive tissues, which is not adequate to maintain stability during exposure to large forces. The ball is at critical stability, meaning that it may stop with a small applied force, but would keep rolling with a larger applied force, as there is not enough resistance to keep the ball stable. Considering the lumbar spine as an example, a ligamentous lumbar spine buckles (becomes

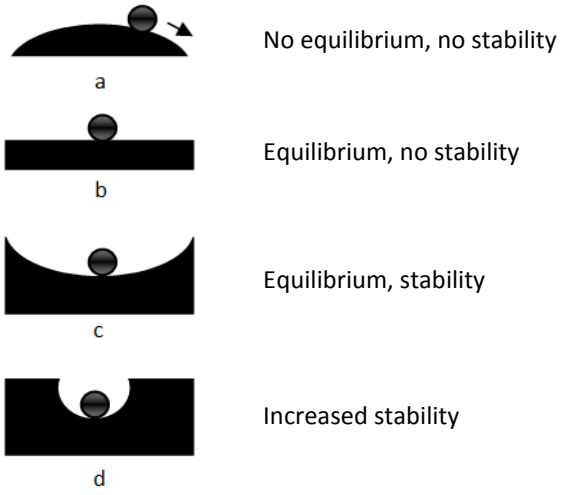

Figure 3. Degrees of stability (Reeves & Cholewicki, 2003). Diagram *a* is a system that has no equilibrium and no stability. Diagram *b* is a system that has equilibrium but no stability. Diagram *c* is a system that has equilibrium and is stable. Diagram *d* is a system that has increased stability in comparison to the previous diagrams.

unstable) with 90N of applied force (Crisco & Panjabi, 1992), whereas forces due to the weight of the torso are significantly greater than this in an adult. Figure 3*c* demonstrates a system that has both passive and active tissues. This system maintains stability by resisting a larger magnitude of force than in the previous two diagrams, but can lose stability if a critical amount of force is provided to the system. Figure 3*d* represents a system that uses passive tissues and muscle activation from agonist and antagonistic muscles. In this diagram, the ball has very little opportunity to roll away or become unstable. The addition of co-contraction to this system adds stiffness and further stabilizes the joint.

2.2.2 Co-contraction

Co-contraction, also known as coactivation, is the concurrent activation of the muscles around a joint, which involves agonist and antagonistic muscles (Enoka, 2008). A benefit of co-contraction discussed by Chiang and Potvin (2001) is that it provides increased joint stiffness by evenly distributing forces around a joint; a large concentration of force is not given to a single

muscle. However, the magnitude of stiffness is dependent on which muscles are involved and their characteristics. Brown and Potvin (2005) discuss the roles of multisegmental muscles in spine stability; these muscles are dominant moment generators and thus, provide the majority ofstiffness in the lumbar spine. They also support the notion that smaller intersegmental muscles function to maintain balance and help to coordinate the activation patterns of muscles surrounding the lumbar spine. Both types of muscles are of particular importance to increase joint stiffness, whether it comes in the magnitude or in the organization of force generation. However, the benefit of increasing stiffness at the joint to restrict movement also comes at a cost of potentially increasing the compressive loads to the spine (Vera-Garcia et al., 2006). By using this anticipation technique there is a reduction in the movement of the vertebrae, and passive and active tissues surrounding the spine post-perturbation. Otherwise, the perturbation would cause movement of these tissues, resulting in an increased reaction force from the agonist and antagonistic muscles. The increase in compression can damage both passive and active tissues of the spine if it is of high enough magnitude. Figure 4 will be used to describe this phenomenon. When there is a small amount of force required for a task, there is a small effort required by the

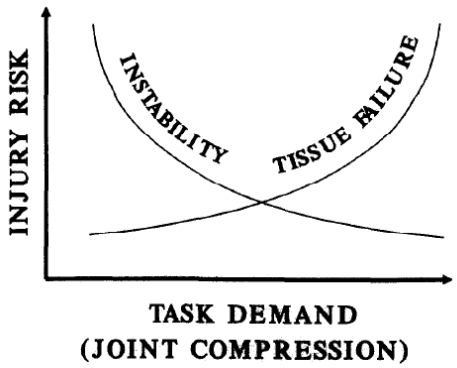

Figure 4. Hypothetical prediction of injury risk dependent on task demands where variables of instability and tissue failure both have optimal levels to reduce risk (Cholewicki & McGill, 1996).

muscles, which can lead to a loss of stability. This unstable state may lead to the sudden activation of muscles resulting in muscle spasms or overloading of a single tissue (Cholewicki & McGill, 1996). Conversely, if there is a large load demand for a task, tissue tolerances may be exceeded in an attempt to complete the task, resulting in injury. Nonetheless, the advantages and disadvantages of increased muscle activation are not only an issue because of the muscles involved, but also due to the timing of their onset.

2.2.3 Muscle Onset Timing

Initiating the activation of muscles in the torso before applying a sudden load has been shown to reduce the displacement of the torso post perturbation (Stokes et al., 2000). This pre-activation, also known as bracing, has been studied by many researchers to determine the advantages and disadvantages of the technique (Brown & McGill, 2008; Vera-Garcia et al., 2006; Brown et al., 2006; Vera-Garcia et al., 2007). A study by Brown et al. (2006) had participants perform abdominal bracing at levels equivalent to 10%, 20% and 30% of the maximum voluntary contraction (MVC) of the right EO, as well as perform a natural bracing method prior to perturbations of unknown timing. The results of the study showed the natural bracing method to be superior to the predetermined activation method for enhancing the joint stiffness, and thus stability of the system. Although the other methods of bracing also showed increased spine stiffness, individuals were often unsuccessful in achieving an optimal coordination of individual muscle contributions; the trunk muscle recruitment patterns are different for each loading situation and its specific stability needs (Brown & Potvin, 2005; Brown et al., 2006). Further, muscle preactivation has been shown to have negative effects on the muscular response to perturbations, specifically during the involuntary time period, through delays in the response time (Chiang & Potvin, 2001) and absences in their presence during this time period altogether

(Stokes et al., 2000). A more detailed look into understanding muscle responses and their role in stability is explored in the following section.

2.3 MUSCLE RESPONSE

An involuntary muscle response is associated with a contraction which cannot be changed through conscious, voluntary motor control and occurs due to an external stimulus (Latash, 2008). Involuntary responses are commonly made up of three components, collectively known as the reflex arc: an afferent neuron, a central processing unit and an efferent neuron (Latash, 2008). The afferent neuron conveys various sensory modalities (stimuli specific to a receptor) to the central nervous system (CNS). Located in the CNS, this information is then transferred to the central processing unit which consists of one or many interneurons that regulate information transmission between the afferent and efferent neurons. The efferent neuron receives information from the interneurons and relays it to the effector organ outside of the CNS. The effector organ is electrically controlled by the efferent neuron. If this effector organ is muscle tissue, for example, active muscle force is generated when it is stimulated. However, the time required for the effector organ to produce a response varies according to the number of synapses involved. A synapse is a gap between two neurons which allows the signal to travel electrochemically from one neuron to the next. When there are a larger number of synapses involved in a response, more neurons are involved and the timing of the response increases. Many reflexes associated with the involuntary responses are polysynaptic (contain two or more synapses) and have a time delay of approximately 50 ms (Latash, 2008). There are, however, circumstances where a reflex is made up of only one synapse (between the afferent and efferent neurons). This is known as a monosynaptic reflex. This reflex does not contain the same components as that of the reflex arc,

and is very quick with a typical time delay of 30 ms (Latash, 2008). These reflex types are further discussed below.

2.3.1 Monosynaptic Reflexes

Monosynaptic reflexes are considered relatively simple as they are composed of fewer elements compared to polysynaptic reflexes. For this reason, the monosynaptic reflex was the first reflex studied during the initiation of spinal pathway research (Enoka, 2008). This reflex is elicited in response to a sudden change in length of a muscle (detected through muscle spindle excitation) and can be measured through the tendon tap reflex (Figure 5). The tendon tap reflex, known as

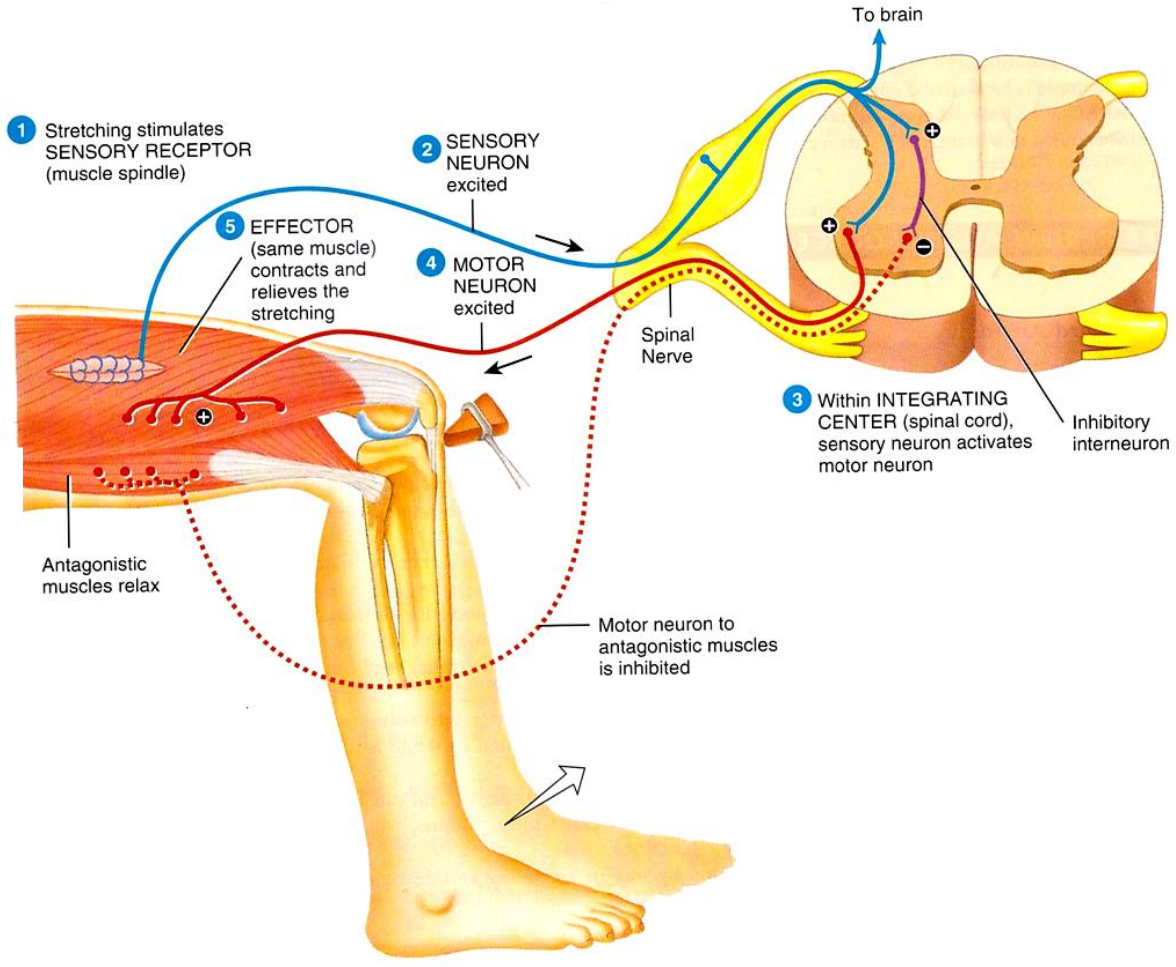

Figure 5. Tendon tap reflex showing the general components of a reflex arc (Tortora & Nielsen, 2012).

the T-reflex, is often performed on the extensor muscles that surround the knee joint. When the knee is at 90 degrees of flexion, the patellar tendon is gently impacted with a small mallet. This action causes the quadriceps tendon to deform and the muscle tissue to lengthen, thus stretching the quadriceps muscles. Muscle spindle fibres within the quadriceps muscles receive this information and elicit a synchronized response of firing APs (Latash, 2008). These APs propagate down the afferent axons until they reach a synapse with the motor endplate. A motor endplate is a region of the sarcolemma on a neuron which opens to the synaptic space between neurons. In the synaptic space, the signal is translated electrochemically from the afferent neuron to the motor endplate of the motor neurons (Latash, 2008). Once the motor neurons become excited, they produce excitatory post-synaptic potentials (EPSPs). These EPSPs summate together and, if strong enough, create an AP (Latash, 2008). This AP travels down the axons of the motor neurons in the quadriceps and signals for these muscles to contract, thus causing leg extension at the knee. There are no interneurons needed for this quick reflex to take place. In monosynaptic reflexes, a muscle twitch response begins shortly after the onset of the stimulus. This response can be broken up into two components: the short-latency response (M1) and the longer latency response (M2). The short latency response is what is produced via the homonymous motor excitation from Ia afferents. Homonymous excitation occurs when the afferent and efferent neurons responding to a stimulus are in the same muscle. The muscle spindles that sense the muscles' stretch have a very fast response and aid in the quick speed of this reflex which has a typical latency of around 30 ms (Enoka, 2008). Longer latency responses are not completely understood, but are thought to involve group II afferents, which have a slower nerve conduction velocity (Shuurmans et al., 2009). Some also believe that the longer latency

response involves processing by the motor cortex in the brain (Enoka, 2008) and would therefore not be considered part of a monosynaptic reflex, and rather, a polysynaptic reflex.

2.3.2 Polysynaptic Reflexes

As previously mentioned, polysynaptic reflexes are rather slow in comparison to monosynaptic reflexes. There are three main reasons behind the latencies of these reflexes: time of afferent conduction, central delay and time of efferent conduction (Latash, 2008). The times associated with afferent and efferent conduction refer to the speed at which the AP travels along the neural fibre, as well as the fibre's length. A large-diameter axon and presence of myelination make it easier for the neurons to become activated as they require low stimulus intensities to initiate APs. As an example, the T-reflex initiates APs in myelinated, large-diameter Ia afferent neurons; the AP travels down the axons at velocities as high as 120 m/s. The central delay is another reason for the latencies. This delay refers directly to the number of synapses involved in the reflex. Typically a delay of 0.5 ms applies for each synapse. It is important to note however, that most of our body's reflexes are polysynaptic as they are more complex in behaviour than reflexes with fewer synapses.

One polysynaptic reflex of particular interest to this study is the TVR. Tonic reflexes lead to sustained muscle contractions and emerge in response to a change in the level of a stimulus. TVR causes a tonic contraction to occur in muscles exposed to vibration and begins only a few sec after the commencement of exposure. The sEMG signals produced by this reflex appear similar to that of voluntary muscle contractions in the same muscle (Bongiovanni & Hagbarth, 1990). Furthermore, there are some MUs that fire at the same rate as the vibratory cycles (Roll & Vedel, 1982). These MUs are said to have synchronized EPSPs and their Ia afferents fire very quickly (Latash, 2008). Their response to vibration occurs so readily because these receptors

respond to vibration as a modality, which is their preferred stimulus type. A description of the receptors which are of importance to this study is provided below.

2.4 SOMATOSENSORY RECEPTORS

Receptors are specialized cells that respond to specific stimuli and, through this response change their physiological properties (Latash, 2008). As previously mentioned, they are specific to primarily one modality. For example, the afferent receptors in the nose respond to the chemical composition of substances we inhale through our nose, which we perceive as smell, whereas the receptors of the eyes respond to changes in light frequencies. However, under some circumstances (i.e. electrical or strong mechanical stimulation) receptors may respond to signals outside of their modality. The stimuli that receptors respond to are dictated based on the location and type of receptor. In the human body, there are three main types of receptors: interoceptors, exteroceptors and proprioceptors. Interoceptors obtain information from within the body, whereas exteroceptors transduce information from the external environment. Proprioceptors receive information about the arrangement of the body and limbs with respect to one another; they can only be activated through the excitation of the peripheral sensory endings (Latash, 2008). Proprioception is seen as a combination of kinaesthesia (ability to detect joint movement) and joint position sense (Shaffer & Harrison, 2007). This sense helps the neuromuscular system to perform precise movements, reflexes and maintain stability of the system (Lephart et al., 1997). The afferent information required for this sense is collected from the following somatosensory receptors: muscle spindles, GTOs, cutaneous mechanoreceptors, nociceptors, joint receptors and thermal sensors (Brooke & Zehr, 2006).

2.4.1 Muscle Spindles

Muscle spindles are a type of mechanoreceptor (receptors that respond to changes in pressure or deformation of skin or tissues of the body) found within skeletal muscle tissue (Figure 6). These spindles are arranged in a parallel fashion and have two fibre types: nuclear bag and nuclear chain fibres. They connect to skeletal muscle fibres via connective tissue and are considered intrafusal fibres (skeletal fibres are extrafusal fibres) (Enoka, 2008). During muscle contraction, muscle spindles do not provide a significant amount of force; rather, their primary role is to

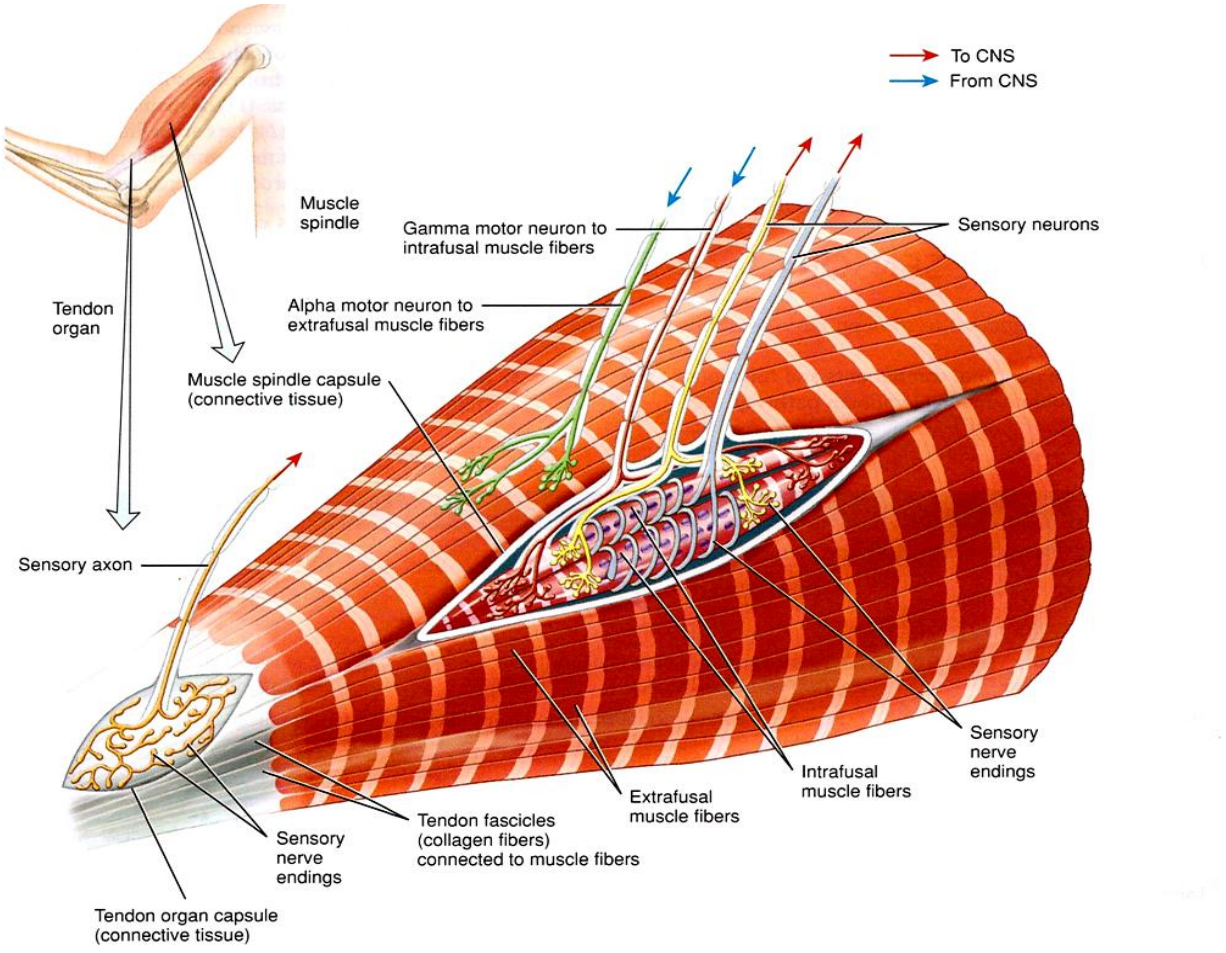

Figure 6. Muscle spindles and Golgi Tendon Organs in the muscle tissue (Tortora & Nielsen, 2012).

detect the change in length of the muscle, as well as the rate of the length change (velocity) (Enoka, 2008). However, these receptors are most important for the initiation of reflexes and voluntary movements that maintain the body's stability (Shaffer & Harrison, 2007).

Innervation of Ia afferent fibres occurs in all muscle spindles; these are among the fastest neural fibres in the body (Latash, 2008). Group II afferents, which have slower conduction velocities, are also found in some muscle spindles. The innervation of muscle spindles is actually quite unique as they are the only receptor to receive both afferent and efferent inputs. These receptors receive efferent innervation from gamma motor neurons whose signals travel at velocities around 20 m/s (Latash, 2008). Gamma motor neurons innervate the contractile proteins at the polar regions of the intrafusal fibre, which causes a stretching of the fibre. This stretch controls the sensitivity of the muscle spindles to respond to change in muscle length. In contrast to this, extrafusal fibres receive efferent information from alpha motor neurons which excite skeletal muscle fibres to contract and produce force. However, the alpha and gamma efferent systems can work in synchrony through alpha-gamma co-activation to determine and create a precise level of force needed to complete a specific task. These efferent fibres also monitor the level of feedback given by the muscle spindles. Feedback is defined as the signals arising from various peripheral receptors that provide information to the nervous system on the mechanical state of the neuromuscular system. It is important for not only producing forces, to counteract over-stretching of muscles, but also for simply monitoring the cross-sectional areas of the muscles.

Muscle spindles have an important function in the proprioceptive sense, more specifically its kinaesthetic component; however, this sense can be altered when exposed to strong mechanical stimuli such as vibration. Muscle spindles have been known to respond to vibration, firing once during each cycle from 10-120 Hertz (Hz) (Roll & Vedel, 1982). When exposed to

vibration, muscle spindles are known to create false illusions of muscle lengthening. There are two potential reasons why this illusion occurs. First, since the response of the muscle spindle receptor is dependent on the change in muscle length, the level of muscle activation has a direct relationship with the firing of the spindle (Latash, 2008). For example, during vibration exposure the muscle may elicit TVR, resulting in contraction of the skeletal muscle. Afferent muscle spindle receptors respond as if a change in length of the muscle is occurring because of the activation, thus eliciting a muscle lengthening illusion. The second potential mechanism for this illusion involves the pronounced effect that vibration has on the muscle spindles, causing them to fire extensively through activation by a strong mechanical stimulus (Latash, 2008). In either case, the increased activity of the muscle spindle receptors leads the CNS to falsely interpret that muscle lengthening is occurring. Furthermore, this illusion causes the participant to notice changes in the velocity of the muscle length change. This rate of length change is dependent on the frequency of the vibration; the greatest velocity of muscle length change was between 70-80 Hz (Roll & Vedel, 1982). However, when a participant gathers information from another source (i.e. visually sees no change in muscle length), the illusion disappears (Lackner & Taublieb, 1984).

2.4.2 Golgi Tendon Organs

GTOs are sensitive to mechanical deformation and are located both in tendons and at musculotendinous junctions surrounding extrafusal fibres (Figure 6) (Enoka, 2008). These receptors provide us with constant information on the production of active or passive muscular force at the tendon. Further, they aid in the prevention of injuries by signaling when a muscle is experiencing too much force for it to withstand. The GTOs can be easily excited during active muscle force production at thresholds as low as 30-90 μN; whereas passive forces require a

higher threshold of around 2 N before GTO activation occurs (Latash, 2008). When a muscle is stretched and produces force, its connective tissues are also stretched and excite the Ib afferents of GTOs. Signals travel down these afferents at speeds around 80 m/s to interneurons in the CNS (Shaffer & Harrison, 2007). If one interneuron is involved in this pathway, an inhibitory signal is sent to the homonymous motor neurons to stop contraction in the muscle that is creating or experiencing the force (Enoka, 2008). Conversely, if two interneurons are involved in the pathway, an excitatory signal is sent to antagonistic muscles for contraction to occur (Enoka, 2008). Both pathways work together to prevent injury by reducing extraneous stress and deformation of tissues caused by excessive force production.

2.4.3 Cutaneous Mechanoreceptors

Cutaneous receptors play an important role in providing information about the external environment. Tactile discrimination is a main function; it occurs through the detection of hair movement, stretching and indentation of skin, as well as acceleration of dermal and subcutaneous tissues (Enoka, 2008). Upon excitation, the cutaneous receptors send signals to their group II afferents, which synapse with interneurons in the CNS. The following cutaneous mechanoreceptors of the glabrous skin will be discussed in further detail: Merkel discs, Meissner corpuscles, Ruffini endings and Pacinian corpuscles.

2.4.3.1 Merkel Discs

Merkel discs have a well-defined, small receptive field that is located superficially in the cutaneous layer of the skin (Shaffer & Harrison, 2007). They respond specifically to vertical pressure on the skin (Latash, 2008). These are slow-adapting receptors that continuously deliver information on vertical pressure, until the stimulus is no longer present (Csillag, 2005). During instances of movement, these receptors are essential for providing feedback on position

awareness. For example, when someone is walking or standing they continuously know when their feet are impacting the ground, which helps with balance and planning future movements.

2.4.3.2 Meissner Corpuscles

These mechanoreceptors are found superficially in the cutaneous layer of skin (Shaffer & Harrison, 2007). They have small, well-defined receptive fields that allow them to detect fine touch (Lundstom & Johansson, 1986). Meissner corpuscles are sensitive to maintained pressure, but fade rapidly if the pressure does not change. This is an important feature of these receptors as our body must respond to competing stimuli. Those stimuli which are not pertinent to maintaining the safety or equilibrium of the body are not of critical importance for processing. An example of a response from these receptors occurs while wearing clothing; at first the clothes are sensed by the body due to the increased pressure they import on the skin, but this sensation subsides rather quickly as the maintained pressure does not change appreciably.

2.4.3.3 Ruffini Endings

Ruffini endings are fine-touch receptors and are located subcutaneously in the dermis layer (Shaffer & Harrison, 2007). They have a large receptive field and adapt slowly by continuously firing APs as a result of skin stretching over a large area (Lundstom & Johansson, 1986).

2.4.3.4 Pacinian Corpuscles

Pacinian corpuscles are located subcutaneously and respond to fine touch. They are the largest cutaneous mechanoreceptor and are innervated by only one axon (Enoka, 2008), which has a large receptive field with obscure boundaries (Shaffer & Harrison, 2007). These receptors respond to changes in pressure; specifically the acceleration properties of a stimulus (Enoka, 2008). Changes in acceleration are a major component of vibration signals; thus, this stimulus

elicits a response from Pacinian corpuscles. They respond to many vibration frequencies, but more readily to those above 50 Hz (Lundstom & Johansson, 1986).

2.4.4 Joint Receptors

Joint receptors vary both in their location around the joint and the stimuli they respond to. Specific joint receptors (or articular receptors) typically respond to small increments of joint angle displacement at the extremes of the joint range of motion, while few receptors fire at the mid-range of motion (Latash, 2008; Enoka, 2008). Due to their role in joint angle determination, the joint receptors are of particular importance for injury prevention. For example, if a joint experiences hyperextension, the joint receptors fire to signal if the joint is reaching its maximal range of motion, which aids in maintaining the integrity of the joint. The neural feedback from these receptors is useful for joint positioning, but their signals are transmitted to ascending and reflex pathways that are combined with signals from the muscles and skin (Enoka, 2008). Together, these receptors' signals combine to ensure joint safety in the body by considering the joint itself and the muscles and skin surrounding the joint.

2.4.5 Other Sensory Receptors

There are other sensory receptors that work in tandem to relay pertinent information that helps the body maintain stability; of importance to this study, afferent information interpreted by the proprioceptive sense will be further explored. The vestibular system provides information on the body's state of postural equilibrium and helps to maintain balance. This system is made up of the brain, the semicircular canals and utricle of the inner ear. Hair cells in the semicircular canals, and those of the utricle, respond to changes in head accelerations in angular and linear directions, respectively. In fact, these receptors can respond to cranial accelerations as small as $0.1^{o}/s^{2}$. As with many systems, we only become aware of this sense when the body's equilibrium is at risk;

this is not the case for all sensory systems. For example, the visual system works very differently as we continuously rely on our visual sense and are aware of this reliance on a daily basis. This sense aids in proprioception by providing us with visual, or more quantifiable, feedback of what is going on in the external environment. It is one of the most reliable senses in our body. As previously described, the illusion of muscle lengthening that often occurs with vibration disappears upon seeing that the involved limb or muscle is not actually moving (Latash, 2008). Some illusions and physiological effects that vibration causes have been discussed. However, a look into the mechanics and a further understanding of vibration is warranted for this study.

2.5 VIBRATION

Vibration occurs when an object experiences oscillatory motion. It can be found in numerous situations from disastrous earth quakes, to the sound of a piano, to the rumbling motors of large trucks. The smallest unit of vibration varies as the signal can be predictable (known as deterministic) or unpredictable (known as random or stochastic) (Bovenzi, Lindsell, & Griffin, 2000) (Figure 7). The following subsections discuss the features of these two main divisions of vibratory motion.

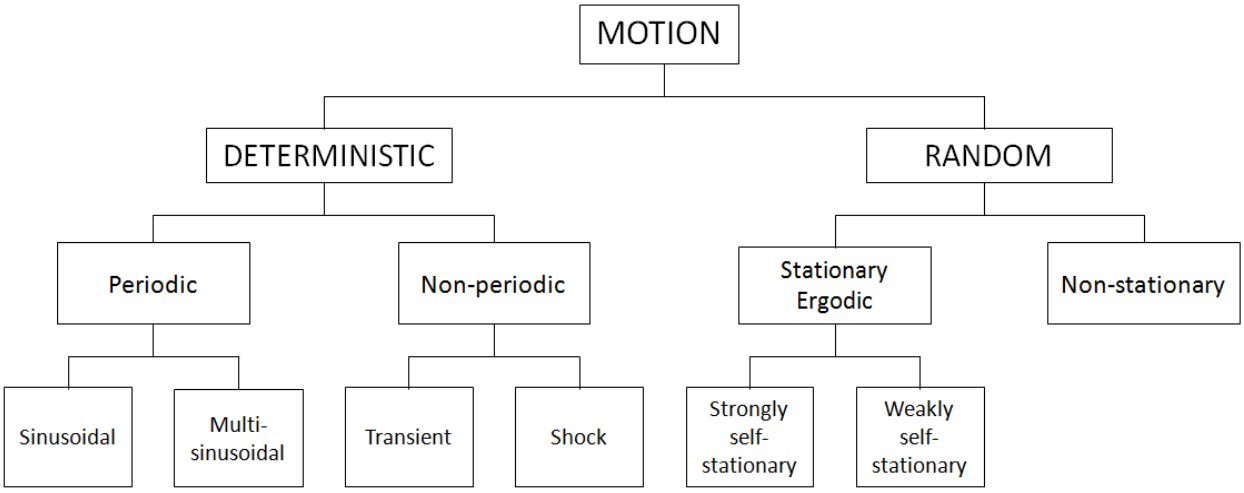

Figure 7. Categorization of oscillatory motion as described in Griffin (1990).

2.5.1 Deterministic Motions

Deterministic motions are those which can be predicted or calculated from previous information about the oscillation; they can be subdivided into periodic and non-periodic motions (Griffin, 1990). Periodic signals are made up of cycles which repeat themselves within a specific time frame; this period of time represents the length of time required for one sinusoidal cycle (sine wave) to complete. Sine waves can be represented through the following equation at time point *t*, with an instantaneous displacement *x (*Griffin, 1990*)*:

Equation 1: $x(t) = A \sin(2\pi ft + \varphi)$

where: A represents the amplitude of the sine wave, *f* is the frequency (or harmonic), and φ is the phase angle of the oscillation.

2.5.1.1 Amplitude

The amplitude of a sine wave represents the magnitude of an object's displacement at a particular point in time. Examples of varying amplitudes would be comparing vibrations experienced while seated in a compact car, where the motor causes small amplitude vibrations, to being seated in a wave water pool where the continuous waves are of larger amplitude.

2.5.1.2 Frequency

The frequency of oscillatory motion describes how many sinusoidal cycles occur within a one second time period, where the unit of measure is Hz. In order to convert the vibration into a time-based signal it must be converted from its analog form in the physical world, to a digital form where it is sampled based on a period of time and quantized (becomes discrete values) (Pelgrom, 2010). Once in digital form, the frequency content of the vibration signal can be calculated using a Fourier analysis. This analysis separates the signal into its various frequency and amplitude

components. The power of the frequencies can also be calculated by squaring the amplitude values to quantify the influence of each of the frequencies on the signal.

2.5.1.3 Phase Angle

The phase angle of a sinusoid, or the initial phase, represents the point at which a sine wave began its phase, in comparison to a reference sinusoid cycle. One cycle of a sine wave goes through 360° of phase. If a sinusoid has a phase angle of 90°, it began one quarter of the way through a typical sine wave pattern, and each cycle will start and finish at this point. In contrast, if a sine wave has a phase angle of 0° or 360°, it started its cycle at the same point as a reference sine wave and they would have identical start and end points.

Periodic sinusoidal signals are often used in research because they can be formulated to have specific frequencies and amplitudes (Griffin, 1990). This trait allows the variability of motions to be controlled for, and make it easier to build inferences from these motions. However, the specificity of such a signal makes it difficult to compare these motions to ones that occur in everyday life, as there are many variables that cause a real-life signal to be random.

2.5.2 Random Motions

Random motions consist of signals that are not predictable; rather, they are described in terms of probability (Sujatha, 2010). These types of vibration signals are present in occupations that have unpredictable variables, such as uneven driving surfaces, which may be harmful due to their unpredictable nature. For example, a worker driving on a dirt road may experience holes in the roadway which add a random characteristic to the driving motion. Nonetheless, there are ways to measure the effects and properties of random signals to get information on their characteristics and possible negative effects on health. Similar methods are applied for both deterministic and random signals.

2.5.3 Measuring Vibration

To quantify the magnitude of a vibration signal, it is common practice to examine the acceleration of the signal, which is the 2nd derivative of displacement, and most commonly the root mean square (RMS) of the acceleration is reported (Griffin, 1990). The RMS of a signal is a calculation of the square root of the mean of all the acceleration values that have been squared; it signifies the generalized mean. The ISO has created standards for the acceptable limits of occupational based vibration and utilizes a vibration's acceleration value, along with the RMS of the acceleration, to assess the severity of a vibration signal. According to ISO 2631-1 (1997), an estimated RMS of <0.45 m/s^2 has virtually no adverse health effects; whereas values of 0.45 m/s^2 to 0.90 m/s^2 may have potential health risks; and those greater than 0.9 m/s^2 are likely to result in health risks. These ISO standards are used internationally to reduce the incidence of injury in occupations with vibration exposure, more specifically whole-body vibration. However, characteristics such as vibration frequency content should also be considered as some frequencies are known to cause negative effects on the human body.

2.5.4 Natural Frequency

The tissues of the human body have been shown to resonate when exposed to vibrations of specific frequencies; this exposure has shown an increased risk of injury. When a system is exposed to a resonant frequency, the vibratory oscillations are amplified, causing increased stress and strain, which can potentially lead to mechanical fatigue or failure (Wilder, Magnusson, Fenwick, & Pope, 1994). Resonance in the human body has been studied for decades, where frequencies in the 1-20 Hz range cause resonance throughout the body (Kitazaki & Griffin, 1998); more specifically, those in the 4-6 Hz range can cause resonance in the spine (Pope & Hansson, 1992). These values are a concern when one considers that dominant frequencies

experienced in LHD trucks can range from 1-4 Hz (Kittusamy & Buchholz, 2004; Eger, Stevenson, Boileau, & Salmoni, 2008). Further negative implications and effects of vibration exposure on the human body will be discussed below for both research and occupational settings.

2.6 REPERCUSSIONS OF VIBRATION EXPOSURE

Research has shown that vibration exposures, such as those experienced in occupational settings, can lead to many negative consequences in the body, including balance. For example, WBV exposures for as little as 30 mins have caused negative effects on balance (Slota, Granata, & Madigan, 2008). These negative effects were measured through increases in kinematic variance (ellipse area, RMS radial lean angle and path length), and significant increases in non-linear stability control (Lyapunov exponent, stability diffusion analysis and Hurst rescaled range analysis) while participants were tested on a wobble chair (quiet sitting vs. vibration exposure). The authors concluded that vibration diminished postural control (Slota et al., 2008). In addition to disruptions in mobility, vibration has been shown to negatively affect various human organ functions and their surrounding tissues. For example, Penkov & Tzvetkov (1996) determined that males exposed to occupational vibration had both decreases in their sperm count and irregularities in sperm formation. Further, Bovenzi (2005) found that occupational exposures to vibrations had negative consequences on the gastro-intestinal system, peripheral veins, and neck-shoulder. Occupational vibrations transmitted through the usage of power tools, are also known to induce a disorder in the hand called vibration-induced white finger (VWF). Takeuchi, Futatsuka, Imanishi and Yamada (1986) researched the pathology of this disorder and determined that demyelination of peripheral neurons in the fingers occurred. This condition causes numbness in the fingers and negatively affects the dexterity of the fingers as well

(Takeuchi et al., 1986). The negative effects of vibration on the low back have been heavily researched and are described in detail below.

2.6.1 Vibration Exposures and Low Back Pain

Before discussing the implications of vibration on the low back and LBP, it is first important to understand why injuries are commonly occurring in this region of the body. Kimura, Steinbach, Watenpaugh and Hargens (2001) discuss the high frequency of injuries that occur at the lowest points of the lumbar spine, (i.e. the L_4-L_5 and L_5-S_1 joints). In their study, participants were exposed to compression forces similar to those experienced during upright posture (approximately 50% body weight) while in the supine position. All lumbar angles and disc heights from the joints between T_{12} through S_1 vertebrae were measured and compared. The L_4-L_5 and L_5-S_1 joint angles were the only ones that did not increase during the compression condition; further, the L_5-S_1 angle even showed a slight decrease (Kimura et al., 2001). Additionally, disc height was significantly decreased only at the L_4-L_5 region. These findings suggest that lower portions of the spine experience increased stress. Further, compression from external sources, either through the direct addition of a load, or through increased muscle activation (i.e. TVR and vibration), may increase the risk of injury to the low back.

Many studies have examined the relationship between LBP and occupational WBV produced by the engines of work vehicles (Bovenzi, 1996; Bovenzi, 2005; Seidel, Hinz, Hofmann, & Menzel, 2008; Kelsey & Hardy, 1975). Bovenzi (1996) determined that bus drivers have a significant risk of chronic LBP, being nearly two and a half times (odds ratio = 2.36) that of the control group (not exposed to WBV) for chronic LBP. Tractor drivers in the same study had a significant odds ratio for chronic LBP close to two times (odds ratio = 1.74) that of the control group. Furthermore, the percentage of workers who needed to take off time from work

due to back pain was significantly greater in the tractor driver group when compared to controls (Bovenzi, 1996). Other research has determined that among a variety of manual labour, transportation and trades occupations, only those in the truck driving industry established positive relationships with the three categories of LBP (mild, moderate and severe) based on number of hours worked (Damkot, Pope, Lord, & Frymoyer, 1984). In fact, severe pain was significantly increased for truck drivers working more than 6 hrs per week, compared to all other occupations studied (Damkot et al., 1984). This work suggests that the duration of vibration exposure on the worker has an effect on the severity of LBP. Other research supports this notion and has determined significant dose-response patterns in daily driving time and driving-related LBP for professional drivers (Tiemessen, Hulshof, & Frings-Dresen, 2008). Furthermore, a study conducted by Eger et al. (2006) determined that WBV exposures in six out of fifteen mobile mining equipment vehicles studied were over the Health Guidance Caution Zone limits indicated in the ISO 2631-1 (1997) standards for an 8-hr shift in vibrating vehicles. These results show that the drivers of these six vehicles are at much greater health risk than those experiencing vibrations under these limits. Drivers in the transportation industry, however, are not simply at a health risk because of exposure to vibration from the vehicle engine; during their shifts these drivers may also be exposed to perturbations because of variable driving surfaces (Eger et al., 2006). These perturbations cause the vibration signal originating from the engine of the vehicle to become stochastic, as the driving surface adds an additional random frequency component, from an unpredictable source. Furthermore, driving posture has been mentioned as a contributing factor for the increased rate of negative health effects, specifically LBP (Bovenzi, 1996; Kitazaki & Griffin, 1998).

The etiology of LBP during vibration exposure has been studied and various theories have been postulated. A review by Wilder and Pope (1996) cited that increased oxygen uptake occurs while sitting erect during vibration exposure. The link between increased metabolic cost due to muscle fatigue was the rationale behind this increased oxygen reuptake. It has also been cited that vibration increases intradiscal pressure in the spine, with a peak at 5 Hz, a frequency identified to cause resonance of the spine (Wilder & Pope, 1996). Another physiological change in the spine tissues due to vibration is the creep response, which is defined as the amount of deformation of an object due to induced stress or stain; it is often measured in the body through changes in body or spinal height. When height changes are measured shortly after vibration exposure, decreases in both spine and body height have been reported (Sandover, 1983; Dupuis & Zerlett, 1987; Klingenstierna & Pope, 1987; Sullivan & McGill, 1990; Pope, Wilder, & Magnusson, 1998). Hypotheses to why the height changes occur vary and include:

1) mechanical overloading from the vibration causing constant stretching and compressing of the spine, which lead to fatigue and, a reduction in the resistance response to the vibration (Dupuis & Zerlett, 1987).
2) compression from vibration causes micro-fractures at the vertebral end-plate or, subchondral trabeculae, creating a callus and decreasing the nutrient diffusion to the disc, causing degeneration (Sandover, 1983).
3) loading of vertebral joints leads to fatigue-induced breakdown of the annular lamellae, either as a failure of the collagen fibres to increased tension or because of their lack of cohesion to lamellae, leading to accelerated disc degeneration (Sandover, 1983).

In contrast, some studies have determined that hours after vibration exposure, both sitting and standing spinal height had actually increased (Klingenstierna & Pope, 1987; Sullivan & McGill,

1990). Sullivan and McGill (1990) hypothesized that this may be due to an inflammatory response in the intervertebral discs due to the mechanical stress of vibration. This stress may cause the vessels to dilate, resulting in protein-rich inflammatory exudates to enter the disc and change the osmotic gradient, which then increases water diffusion into the disc. Nonetheless, changes to the intervertebral discs are not the only physiological changes that occur due to vibration. Rather, neuromuscular changes occur in the body as well.

The stress caused by vibration exposure has also been linked to changes in the neuromuscular response of muscle activation by suppressing its timing and amplitude after an unexpected load (Santos et al., 2008; Wilder et al., 1996). A hypothesis for why this change occurs is that the response induced by vibration causes pre-synaptic effects on the terminals of Ia afferent neurons where they synapse with motor neurons (Latash, 2008). The stimuli from both the perturbation and the vibrations are competing for responses from the efferent neurons, which is hypothesized to cause delayed reflex responses. A study by Arashanapalli and Wilson (2008) also found delays in the neuromotor response of muscle around the spine during vibration exposure. A possible consequence of this delay is increased displacement of the trunk after a perturbation due to the inadequacy of muscle force production to restrict the forced motion, which could potentially lead to an injury (Arashanapalli & Wilson, 2008). However, other studies have found no change in the timing of the reflex response during vibration exposure (Santos et al., 2008). It was hypothesized that this was due to a limitation in the protocol, where the participants descended from the vibration stimulator and walked a short distance to the sudden loading apparatus, thus reducing the neuromuscular effects of the WBV protocol (Santos et al., 2008).

The means by which muscle responses are commonly made to occur in a lab setting is by using a chest harness with a sudden loading apparatus to perturb the participant; this lengthens the muscles whose actions oppose the direction of force (Cholewicki, Panjabi, & Khachatryan, 1997; Radebold, Cholewicki, Panjabi, & Patel, 2000; Stokes et al., 2000; Granata, Slota, & Bennett, 2004; Santos et al., 2008; Arashanapalli & Wilson, 2008). However, this method has its limitations. For example, in some of the studies that use this technique, the location of the loading apparatus is in a direction known to the participant (either in front, behind or beside them) (Chiang & Potvin, 2001; Stokes et al., 2000; Radebold et al., 2000). This known placement may provide an indication of the perturbation direction to the participant, leading to an unintended anticipation effect and potentially inducing pre-activation of the muscles. Another limitation of this method is that each participant is required to wear a harness or vest that is connected to an external load in order to perturb the participant. When the external load is transferred to the harness, deformation of the harness occurs, which is transferred to the body where cutaneous receptors are activated. The results from these studies may have been compromised as the mechanoreceptors are potentially activated. Therefore, it is unknown whether the response recorded in light of this methodology is due to the previous activation of the cutaneous receptors, or if it is due to the firing of the muscle spindles that occurs afterwards. It would be advantageous for those who intend to examine muscle responses initiated through the muscle spindle fibres to use a method that elicits a perturbation which results only in muscle lengthening. With the development of robotics, inertial-based perturbations have provided the means by which such work can be completed. Though previous research using a 6-DOF robot had a large focus on robot and joint testing using cadaveric specimens (Mangan, Hurtig, & Dickey, 2010; Becke & Schlegl, 2011; Kelly & DiAngelo, 2013), the current study explored its

usage in-vivo on the study participants. It was hypothesized that the rapid movement of the robotic platform in various timing and direction scenarios would reduce the opportunity for the anticipation of perturbations and thus, reduces the chance that muscles were being unintentionally pre-activated. Our study aimed to reduce this preactivation as research has determined that bracing trunk muscles reduces the necessity of an increased reflex response to enhance stability after a perturbation (Vera-Garcia et al., 2006); a primary dependent variable of this study. Furthermore, participants' reactions to random direction, inertial-based perturbations are more representative of perturbations that occur in real-life, such as those experienced when driving on uneven pavement.

Chapter 3

METHODS

3.1 PARTICIPANTS

Twenty healthy male participants between the ages 18-30 years, who had not experienced back pain in the previous 12 months, were recuited from the University population. Each participant filled out a questionnaire to assess their back health (Agius et al., 1994). Participants were also asked if they are allergic to any adhesives, tapes or rubbing alcohol. The participants were assigned to one of two experimental groups: 1) participants not exposed to vibration throughout the study (control group – CG), and, 2) participants exposed to vibration prior to and throughout the study (vibration group – VG). Following the examination of the data post collection seven participants were excluded from the study due to recording errors (such as gaps of no data) found with the sEMG data. The remaining 13 partipants' data were used for the study (CG, n=6; VG, n=7).

3.2 INSTRUMENTATION AND DATA ACQUISITION

The kinematics of the trunk, head, arms and legs were captured using a passive marker system (Vicon, Vicon Motion Systems, Los Angeles, California) and sampled at a rate of 120Hz. The placement of the passive markers on particiants is outlined in Figure 8. Additionally, fourteen channels of sEMG were recorded, using the placement protocol outlined in Cholewicki and McGill (1996). The following muscles were recorded bilaterally: RA, EO, IO, LES, TES, MULT and LATS. Disposable bipolar Ag-AgCl surface electrodes (Medi-trace disposable electrodes, Kendall, Mansfield, MA) were positioned parallel to each muscle's line of action, between the myotendinal junctions and innervation zones with an inter-electrode distance of 2.5 cm. The sEMG signals were amplified using 14 channels on the Bortec AMT-8 system (Bortec

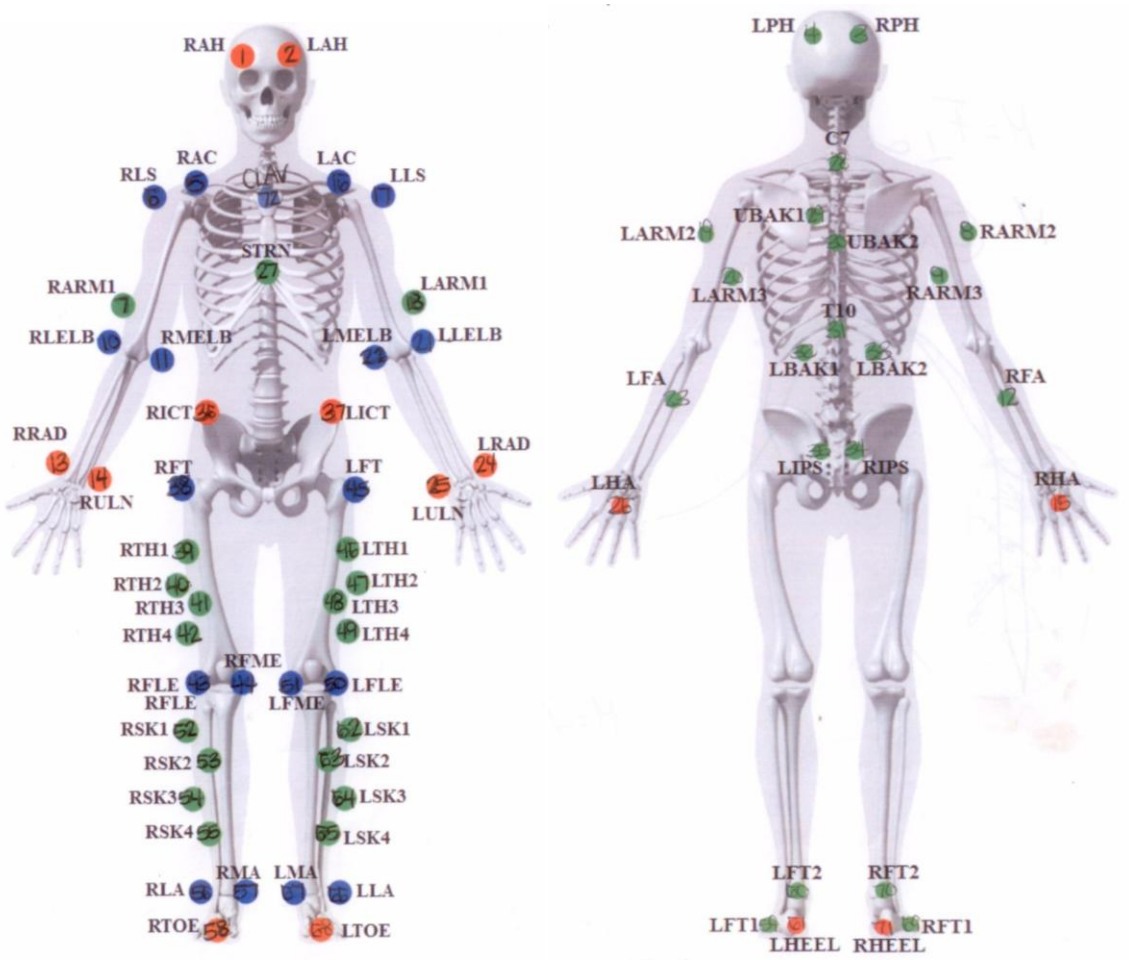

Figure 8. Marker set as it was applied to participants for the passive motion capture. There were 72 markers altogether.

Biomedical, Calgary, Canada, 10-1000 Hz, CMRR = 115dB, gain = 500-1000, input impedance =10 GΩ) at a sampling rate of 960 Hz. A parallel robotic platform (R2000 Rotopod, PRSCo, New Hampshire, USA), was used to apply tri-axial stochastic vibrations (1-8 Hz frequencies; RMS = 0.55m/s^2) (Figure 9), as well as provide the sudden frontwards, backwards, right and left, 65mm platform displacements (Figure 10) at an average acceleration of 0.6g over a 0.17 sec time period. Peak accelerations reached up to 1.6g (15.7 m/s^2). Acceleration, velocity and displacement profiles for a perturbation are shown in Figure 11. Finally, a tri-axial accelerometer (Crossbow CXL75M3, Crossbow Tehonogy Inc, Milpitas, CA) was placed on the underside ofthe robotic platform to measure acceleration and timing of the platform perturbations. An

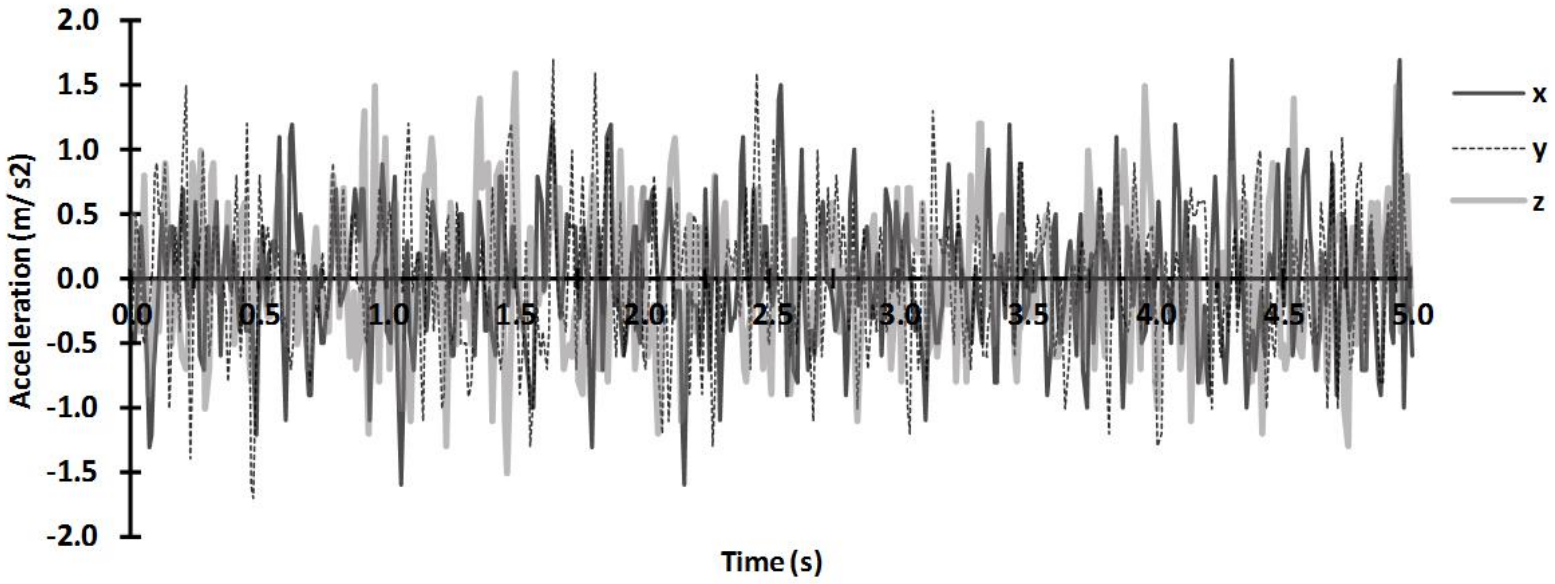

Figure 9. A graphical representation of the acceleration profiles for each axis that were used to create the robotic platform motions, which were experienced by participants in the vibration group. The average RMS acceleration in each of the axes was 0.55m/s^2.

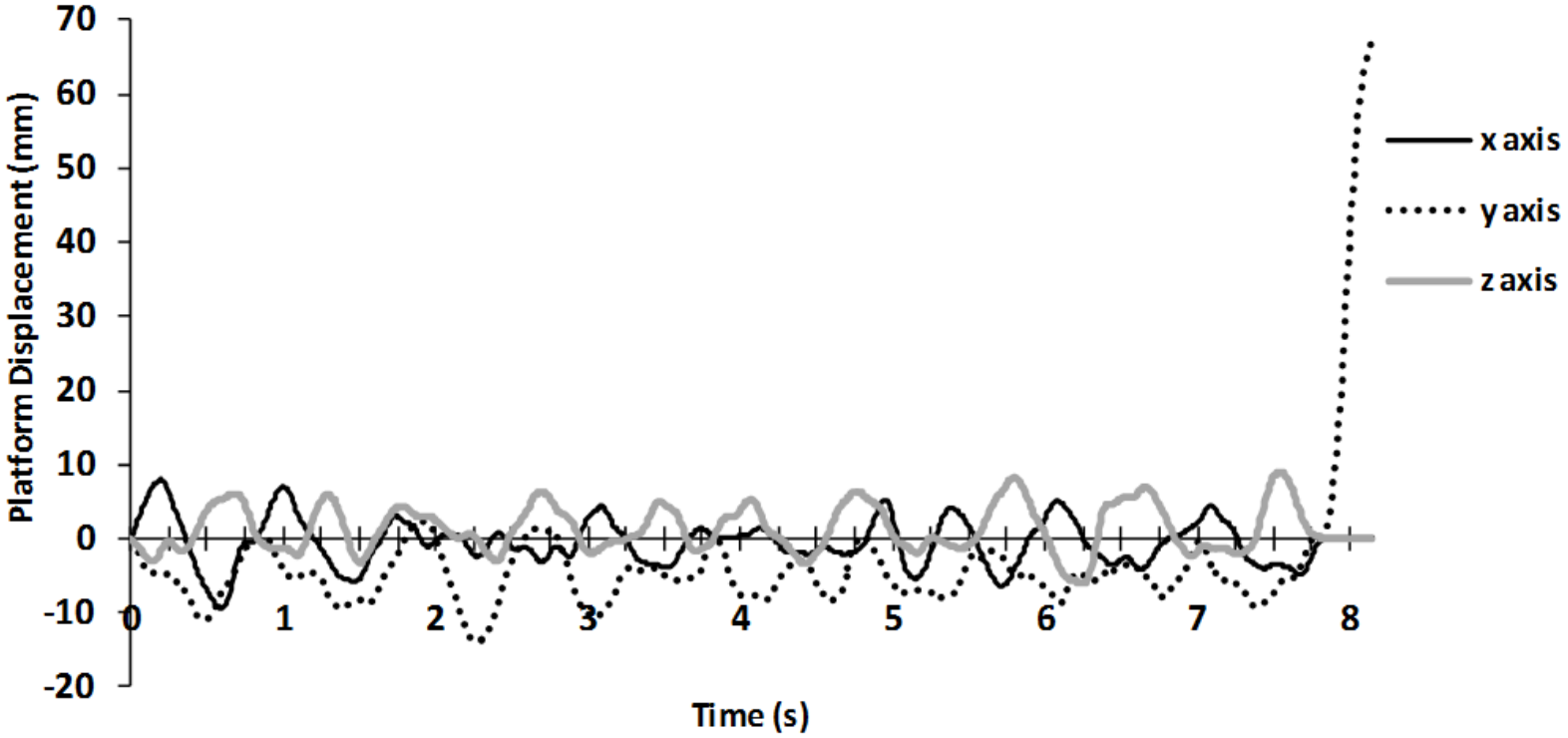

Figure 10. An example of the robotic platform displacements in each of the axes for the perturbation protocol in the back direction condition that the vibration group subjects experienced. Note that this is an example of vibration occurring from the beginning of the trial up to 7.75 seconds which is followed by the perturbation in the y axis.

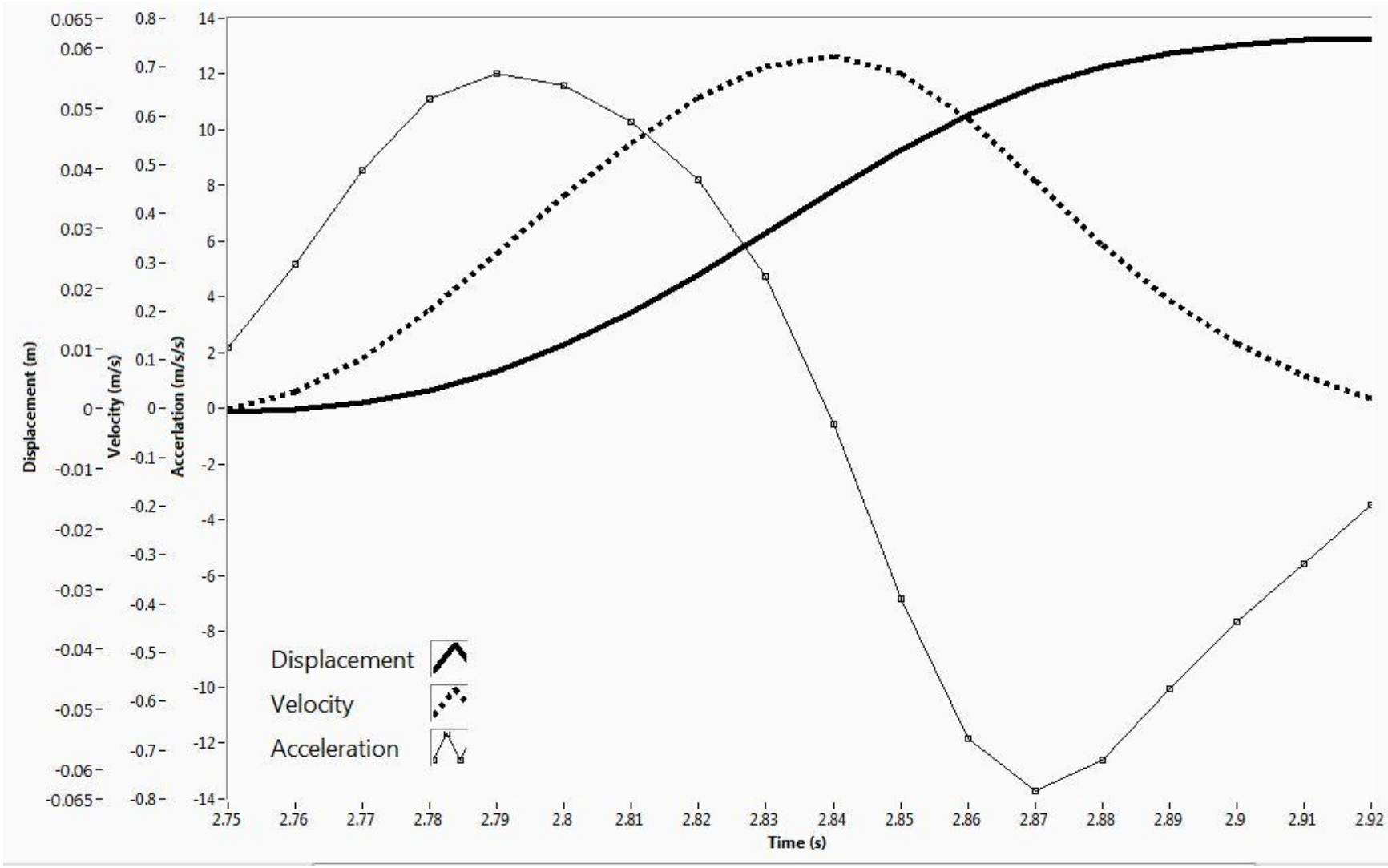

Figure 11. An example of the perturbation motion caused by the robotic platform during a perturbation in the back direction for VG. The robot is programmed to move a total of 0.065 m in a 0.17 second period. The resulting velocity and acceleration profiles are shown for the given robotic platform motion.

accelerometer was also placed on a rubber seat pad, on which the participants were seated, in order to measure the amount of vibration and magnitudes of the perturbations experienced by the participant. The rubber pad was secured to the mounted 2001 Oldsmobile Intrigue vehicle seat atop the robot. The accelerometer data were collected at a sample rate of 960 Hz.

3.3 EXPERIMENTAL PROCEDURES AND PROTOCOL

Each participant's age, height and weight was collected. Participants then provided maximal voluntary exertions (MVE) for each muscle being tested, which was used later to normalize the sEMG data collected during the experimental trials. The MVE was obtained through isometric maximal exertions of the individual's muscles. To obtain the MVEs of the abdominals (RA, EO and IO), participants laid in a supine, balancing position on pommel workout bars, replicating a 'sit-up' position with the feet placed under one of the bars (Figure 12). Participants performed a series of three maximal isomtric trunk flexion efforts that also included twist and lateral bend efforts, against the resistance of the researchers. The MVEs of the trunk extensor muscles (LES, TES and MULT) were performed as the participants lay in a prone position, balancing on the pommel workout bars with the feet placed under one of the bars (Figure 13). Participants performed a series of three maximal trunk extension efforts against the resistance of the researchers. Lastly, participants then performed a series of three upper arm adduction movements against the resistance of the researchers to obtain the MVE for the LATS. Each of the abdominal and back muscle efforts lasted 2-3 sec and rest periods were provided in between each of the efforts. The highest MVE value from the three trials, for each muscle, was used to normalize the sEMG data recorded during the experimental trials.

Following the MVEs, participants commenced the pre-perturbation protocol where they were seated on a vehicle seat, of which the seat back was removed and were told to maintain a

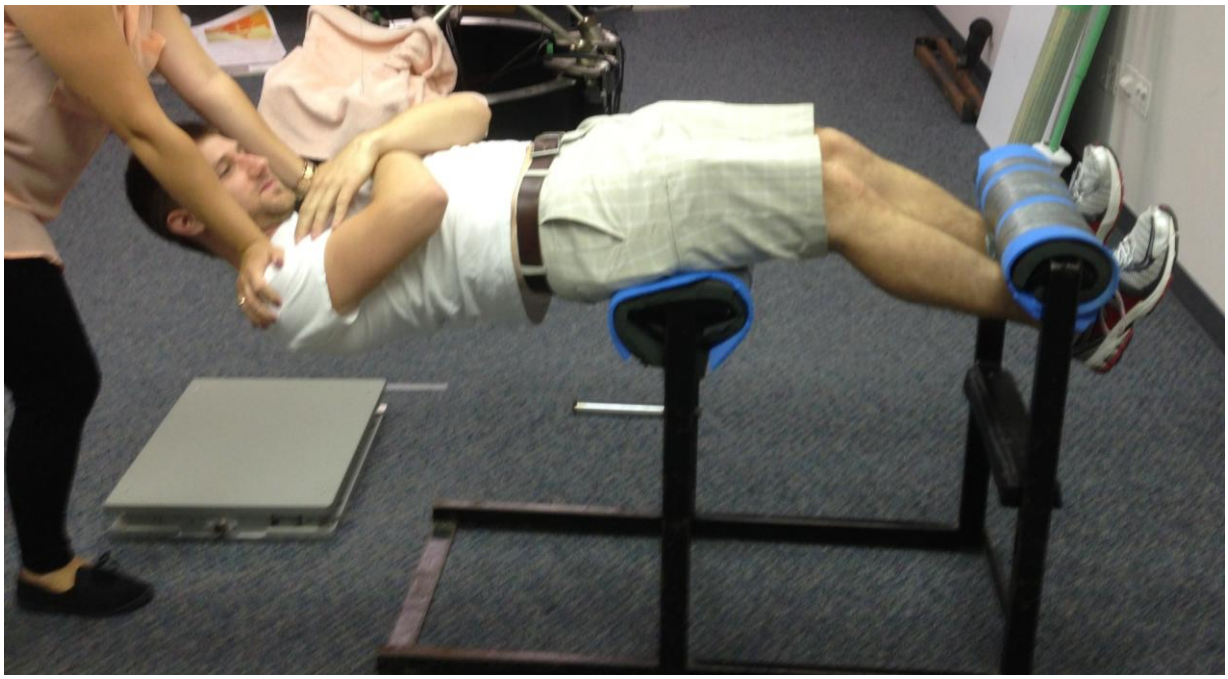

Figure 12. Collection of maximum voluntary exertions for the abdominal muscles. Participants would lay supine across pommel bars and attempt to flex forward, against the resistance of the researchers.

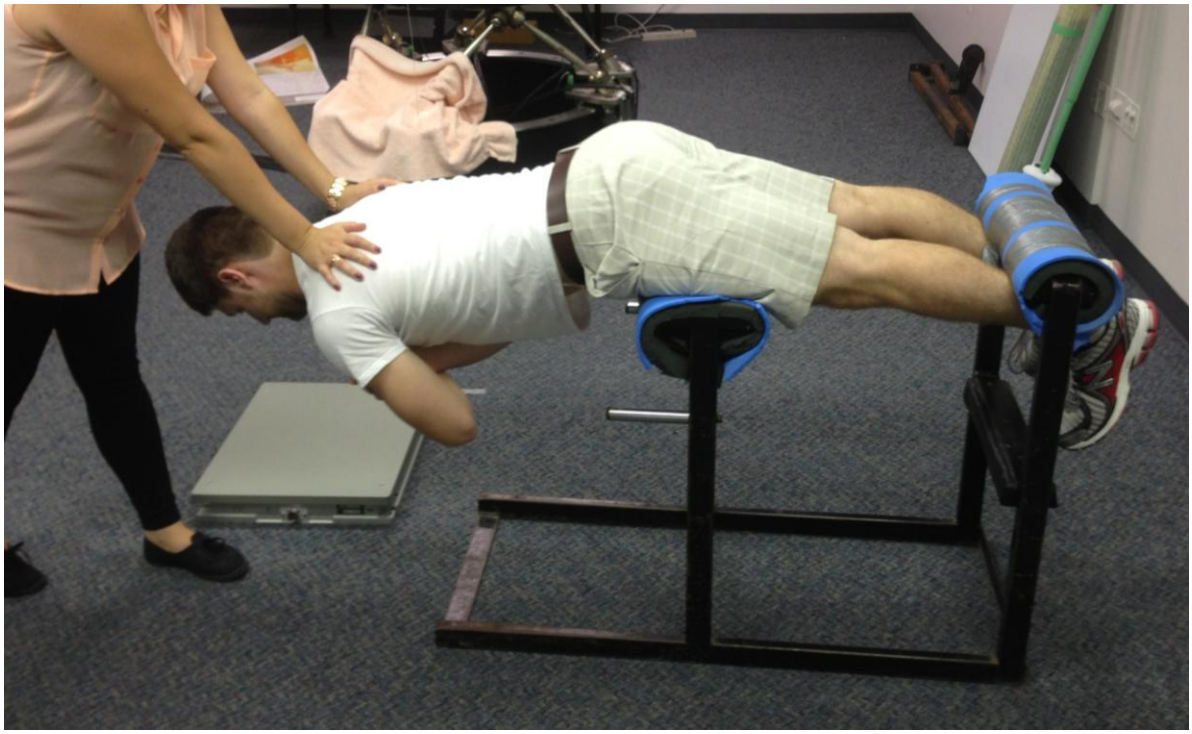

Figure 13. Collection of maximum voluntary exertions for the back extensor muscles. Participants would lay prone across pommel bars and attempt to extend back upwards, against the resistance of the researchers.

neutral spine posture for the duration of the protocol. Participants pelves were fixed to the seat via a lap belt which was placed across their proximal thigh where it joins with the pelvis; this belt was secured to the robotic platform. The belt was used to ensure participant safety, as well as to permit unrestrained motion about segments above the pelvis. For the participants within the VG, they underwent a pre-perturbation trial in this setupfor ten mins under their respective conditions (either constant vibration or quiet sitting). The vibration protocol was designed to produce vibrations with an average RMS acceleration of 0.55 m/s^2, which is under the the Health Guidance Caution Zone ISO standard for an 8-hr shift with vibration exposure (0.63 m/s^2). The CG group were also secured to the robot, but instead sat for the ten minute time period without experiencing vibration. After the pre-perturbation trial, the participants completed the perturbation protocol which included perturbations causing forced flexion, extension, right and left lateral bends of the trunk. All participants placed their hands prone on their mid-thighs, with elbows at appromimately 90º of flexion during the experimental trials (Figure 14).

All participants were exposed to ten trials of sudden known and unknown directional perturbations in each of the aforementioned four directions (total of 40 perturbations). Each perturbation was sudden, such that participants were not aware of the timing of the perturbation (average acceleration = 5.9 m/s^2; peak acceleration = 15.7 m/s^2). To accomplish this, participants were informed of the start of the trial, however, within a 15 sec period after the informed start, a randomly assigned time-based pertubation was provided by the robotic platform. For half of the trials, participants were unaware of the perturbation direction. The same perturbation profiles were used for each participant, in a randomized order. During the 15 sec window that the perturbation was elicited, the VG experienced the same vibration profile used in the pre-perturbation trial until initiation of the perturbation. Following the perturbation, the robotic

Figure 14. Participants were seated on a backless car seat and told to maintain neutral posture during both the pre-perturbation and perturbation protocols.

platform was reset to the starting position, at which time the vibration profile resumed for the next trial for the VG. At this time, the participants were once again informed of the start of the new trial. The participants in the CG were not exposed to the vibration during the 15 sec window; they experienced the perturbation while sitting quietly on the robotic platform.

3.4 DATA ANALYSIS

The tri-axial accelerometer data were dual low-pass Butterworth filtered using a 50 Hz cutoff. All sEMG data, including data collected during MVE tials and experimental trials, were

conditioned using a low-pass filter of 500 Hz (1^{st} order) and a high-pass filter of 140 Hz (6^{th} order), full wave rectified, and low-pass filtered at 2 Hz (1^{st} order) (Potvin & Brown, 2004) (known as linear enveloping). Following conditioning, all signals were down-sampled to 120 Hz. Data were also normalized to the MVEs that were obtained for each respective muscle. Data were then windowed into two sections for analysis: pre-perturbation data and perturbation data.

3.4.1 Pre-Perturbation Data

The sEMG data recorded during the pre-perturbation protocol was used to determine both the mean power frequency (MnPF) and the rectified sEMG amplitude during this protocol, for the VG and CG participants. One-second epochs of the band-passed sEMG data were used to calculate the MnPF for those time periods, which was followed by calculating the average of the MnPF for each minute. Likewise, following the linear enveloping of the sEMG data, one mintute epochs of these data were averaged to determine the sEMG magnitude for each of the ten minute periods. These data were also staticially analyzed and reported.

3.4.2 Perturbation Data

The sEMG linear enveloped amplitude and L_{4-5} joint angle data were windowed into four time periods (Figure 15) (Stokes et al., 2000): 1) *baseline*, from 500-450ms prior to perturbation, 2) *pre-perturbation*, from 50 ms prior to perturbation, 3) *pre-voluntary response*, from 25-150 ms post-perturbation (incorporating both short and medium latency neuromuscular responses), and 4) *voluntary response*, from 150-300ms post-perturbation. The mean and standard deviations for the perturbation trials sEMG and L_{4-5} joint angle data were calculated for each time period. The onset detection of sEMG were estimated based on timing of each significant muscle amplitude change following the perturbations (Stokes et al., 2000). For each trial, the sEMG onset was determined using the integration method of Santello and McDonagh (Santello & McDonagh,

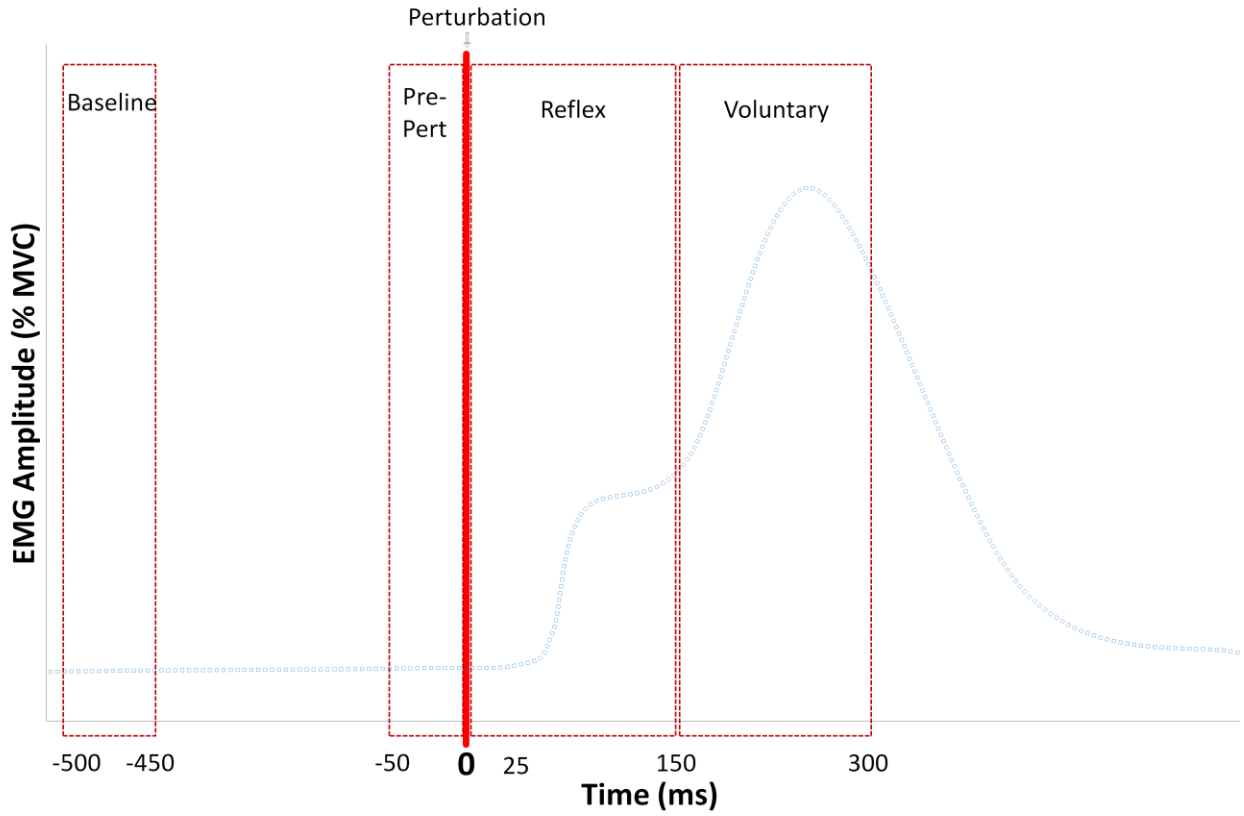

Figure 15. The four time periods are shown relative to the time profile of the perturbation (blue line).

1998). Onset timing data were removed from the analysis if the detected onset occurred more than 400 ms after the perturbation, as these responses have not been shown to be in direct response to the perturbation (Wilder & Pope, 1996).

Markers on the thoracic and lumbar regions were used to determine the relative angle of the trunk, where the thoracic segment was defined by the left acromion, right acromion, left iliac crest, right iliac crest, T4, T10 and sternum makers, and the lumbar segment was defined by the left posterior superior iliac spine, right posterior superior iliac spine, left trochanter, right trochanter, left iliac crest and right iliac crest markers. The trunk angle was calculated by using a joint coordinate system in the Visual 3D Software (Visual 3D, C-Motion Research Biomechanics, Germantown, Maryland) using an X (medio-lateral), Y (anterior-posterior), Z (up-down) cardan sequence of rotations, per Cole, Nigg, Ronsky and Yeadon (1993). All trunk

angles were calculated post-perturbation were normalized to a static standing trial, which was used as the anatomical zero. This position was chosen as the means to normalize the joint angle data due to the generalizability of the lumbar angle across participants; where a seated posture would have more variability in the lumbar angle among participants. For each of the orthogonal axes, the following percentages represent the lumbar component of the overall trunk angle measured: flexion = 72.2%, extension = 43.5%, lateral bend = 49.1% and axial twist = 5.6% (Pearcy, Portek, & Shepherd, 1984; Pearcy & Tibrewal, 1984; White & Panjabi, 1990). Furthermore, the $L_{4\text{-}5}$ joint angle was represented as a fraction of the total lumbar angle as follows: flexion = 22.4%, extension = 9.5%, lateral bend = 16.2% and axial twist = 13.3% (Pearcy et al., 1984; Pearcy & Tibrewal, 1984; White & Panjabi, 1990). The joint angles were processed with a critically damped dual-pass filter with a final cut-off of 5 Hz (2nd order). Also, we dual lowpass Butterworth filtered the tri-axial accelerometer data using a 50 Hz cut-off. Following conditioning, we down-sampled all signals to 120 Hz.

3.5 STATISTICAL ANALYSIS

3.5.1 Pre-Perturbation Protocol

The average sEMG amplitude and its standard deviation for each muscle were calculated during each minute of the pre-perturbation protocol for both experimental conditions. The average MnPF and standard deviation data were also calculated for each muscle during the pre-perturbation time period. A 4-way 2x7x2x10 analysis of variance (ANOVA), with repeated measures, was used to determine the influence of each of the four independent variables: experimental group (VG, CG), muscle (RA, EO, IO, LATS, LES, TES and MULT), muscle side (left and right) and minutes (1-10). The significance level for each ANOVA was set at $p < 0.05$. The dependent variables in this study were: sEMG amplitude, muscle MnPF and seat

accelerometer RMS acceleration. For the significant main and interaction effects, means were compared with a Tukey's HSD post hoc test. A partial eta-squared test (η^2_p) was performed on each interaction to test the effect size for any statistical effect found.

3.5.2 Perturbation Protocol

Across the two experimental conditions, the means and standard deviations of each participant were calculated for each dependent variable across the 40 trials. The mean values were used to represent each participant's response to that condition and used in subsequent statistical analysis. A 6-way 2x2x4x4x7x2 ANOVA, with repeated measures, was used to determine the influence of each of the six independent variables: vibration exposure (present and absent), direction knowledge (known and unknown), perturbation direction (left, right, frontwards and backwards), time period (baseline, pre-perturbation, pre-voluntary and voluntary), muscles (RA, EO, IO, LATS, LES, MULT, TES) and muscle side (left and right). The significance level for each ANOVA was set at $p < 0.05$. The dependent variables in this study were: sEMG amplitude, L_{4-5} joint angle and sEMG onset timing, all in response to the perturbations. For the significant main and interaction effects, means were compared with a Tukey's HSD post hoc test. A partial eta-sqaured test (η^2_p)was performed on each interaction to test the effect size for any statistical effect found.

Chapter 4

RESULTS

Data were examined in two sections, respective to the two parts of the protocol: pre-perturbation and perturbation.

4.1 PRE-PERTURBATION

4.1.1 sEMG Amplitude

The average sEMG amplitude data within each 1 minute epoch showed a significant interaction between muscle and minutes. Post hoc tests were performed on the data by comparing differences in the sEMG amplitudes for each muscle within each minute. However, post hoc analysis did not identify any significant differences between each of the minutes for any muscles (Figure 16).

4.1.2 sEMG Mean Power Frequency (MnPF)

The average sEMG MnPF within each of the minute epochs showed a significant interaction between muscle and side. Post hoc tests were performed on the data by comparing differences in the MnPF for each muscle within both respective sides (Figure 17) ($F=9.24$, $p<0.01$, $\eta^2_p=0.46$). Specifically, post hoc analysis indicated that the LEO was 33% lower than the LIO, 21% lower than the LLATS and 32% lower than the LMULT. Further, the LIO and LMULT were an average of 25% greater than LEO, 17% greater than the LLES, 14% greater than the LRA and 16% greater than the LTES muscles. On the right side, the REO and RLATS were an average of 23% lower than the RIO, 30% lower than RLES, 28% lower than the RMULT, and 32% lower than the RTES, whereas the REO was 29% lower than the RRA.

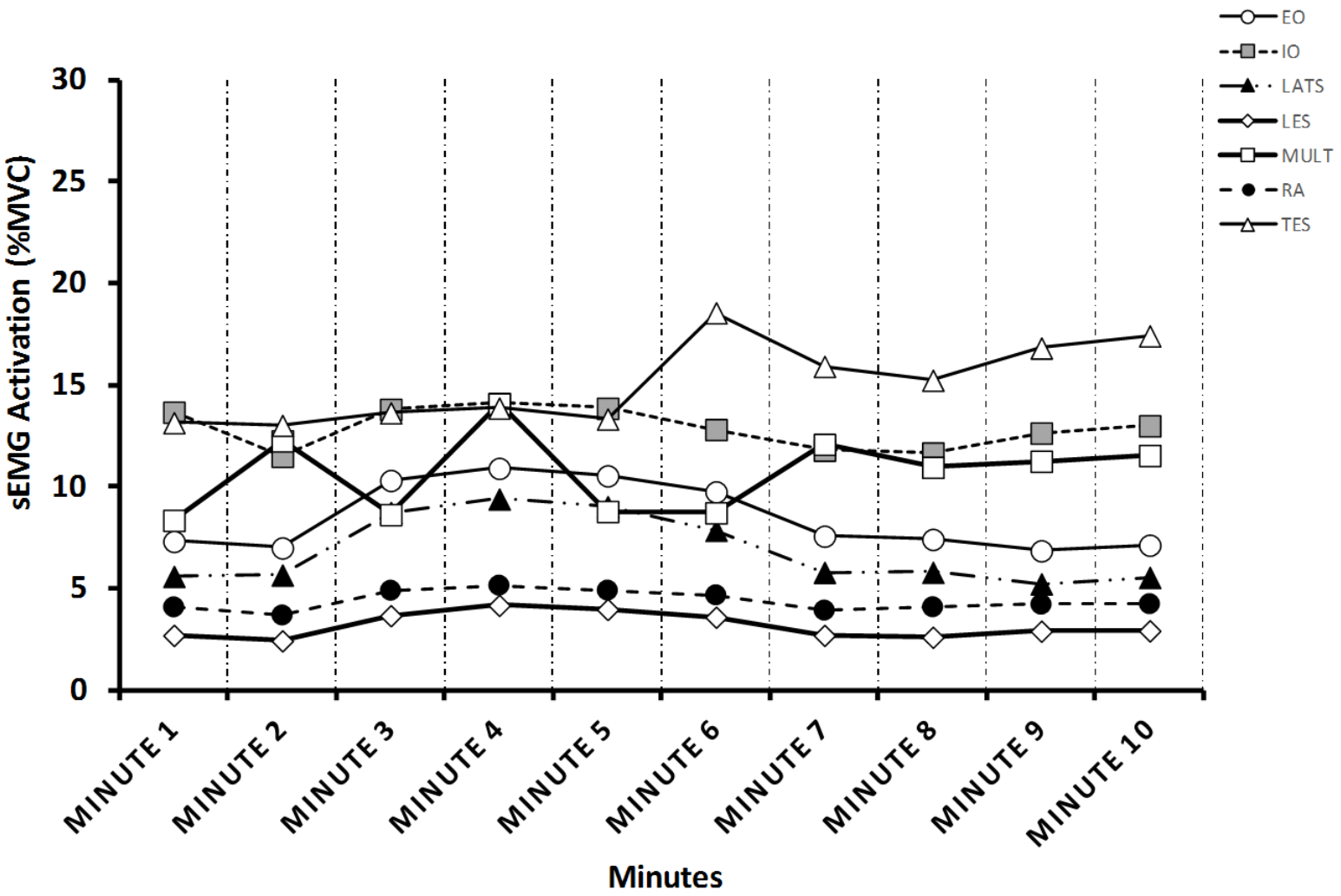

Figure 16. The average sEMG amplitude data within each of the 1 minute epochs during the pre-perturbation trial for the EO, IO, LATS, LES, MULT, RA and TES muscles. Post hoc analysis did not indicate significant differences between each of the minute epochs for any recorded muscle.

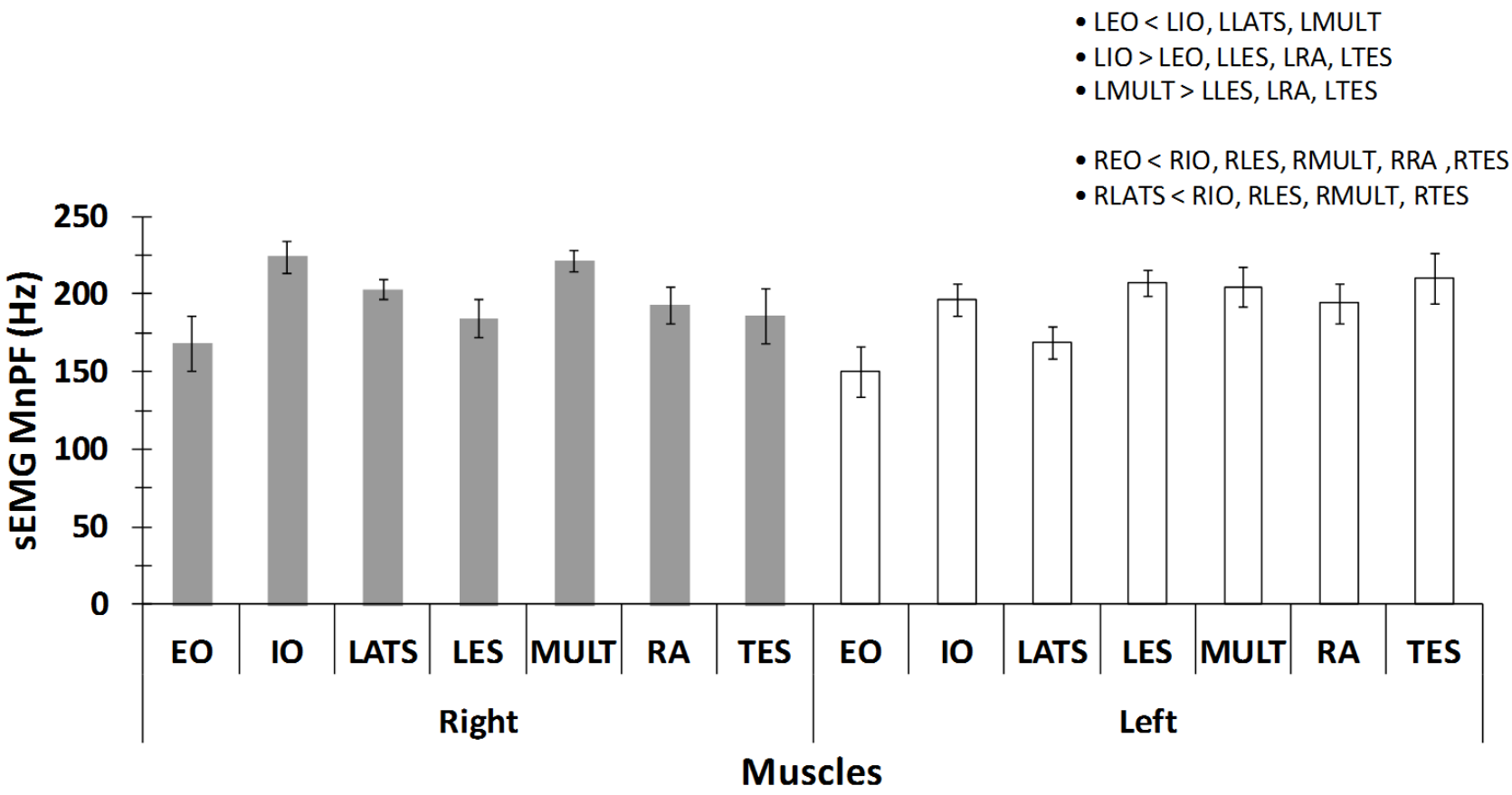

Figure 17. The mean (SD) sEMG MnPF data during the pre-perturbation trial for the EO, IO, LATS, LES, MULT, RA and TES muscles on the left and right sides. Significant differences identified by post hoc analysis are denoted in the graph for the significant muscle x side interaction.

4.1.3 Seat Acceleration RMS

The Seat Acceleration RMS showed a significant interaction between axis and minutes ($F=3.28$, $p<0.01$, $\eta^2_p=0.35$). Post hoc tests were performed on the data by comparing differences in each minute within each respective axis. Minutes 2-9 were relatively consistent across all axes, whereas minutes 1 and 10 were significantly lower. The x axis showed that minutes 1 and 10 were significantly different than each other and all other minutes (Figure 18). In particular, minute 1 was an average of 12% lower than minutes 2-9, whereas minute 10 was an average of 23% lower than minutes 2-9. Minute 10 was also 10% lower than minute 1. The Y axis showed a similar trend with minute 1 being an average of 9% lower than minutes 3, 5, 8 and 9. Minute 10 in the Y axis was an average of 15% lower than minutes 2-9 (Figure 19). The z axis showed that minute 1 was an average of 11% lower than minutes 2-8, whereas minute 10 was an average of 15% lower than minutes 2-9 as well (Figure 20).

4.2 PERTURBATION

4.2.1 Muscle Onsets

The Muscle Onsets had a significant interaction between muscle and side. Post hoc tests were performed on the data by comparing differences in muscle onset timing for each muscle within each respective side (Figure 21) ($F=2.63$, $p<0.05$, $\eta^2_p=0.19$). On the left side, the LEO had an onset time that was 12% less than the LLATS and 12% less than the LTES muscles. Furthermore, the muscle onset for the LRA was 9% faster than both the LLATS and LTES muscles. The right side showed the REO to onset faster than the RLES by 12% and RMULT by 11%. The RTES muscle onset was significantly slower than the REO by 16%, RIO by 10%, RLATS by 10% and RRA by 12%.

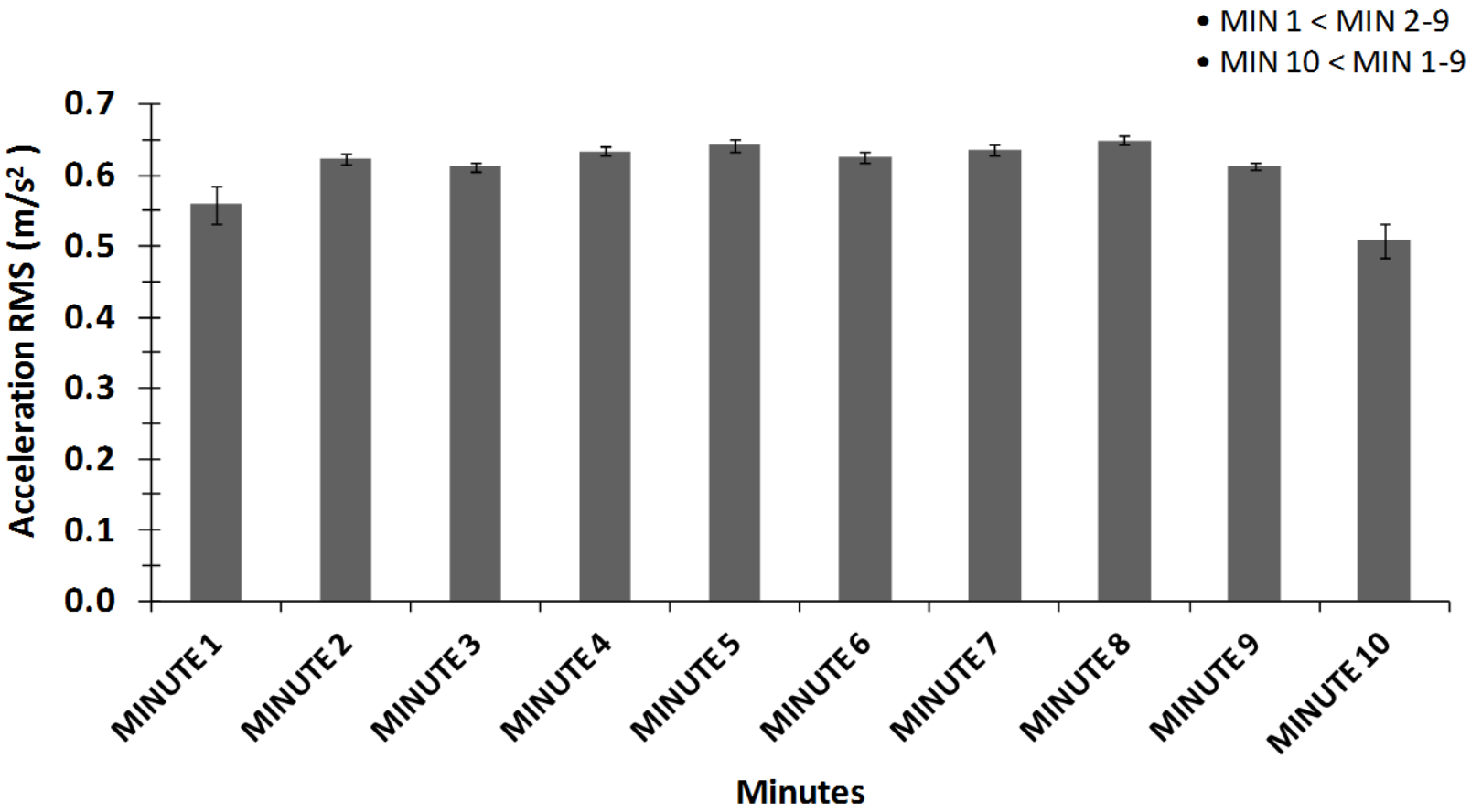

Figure 18. The mean (SD) seat acceleration RMS data within each of the 1 minute epochs during the pre-perturbation trial for the X axis. Significant differences identified from post hoc analysis are denoted on the graph for the significant axis x minutes interaction.

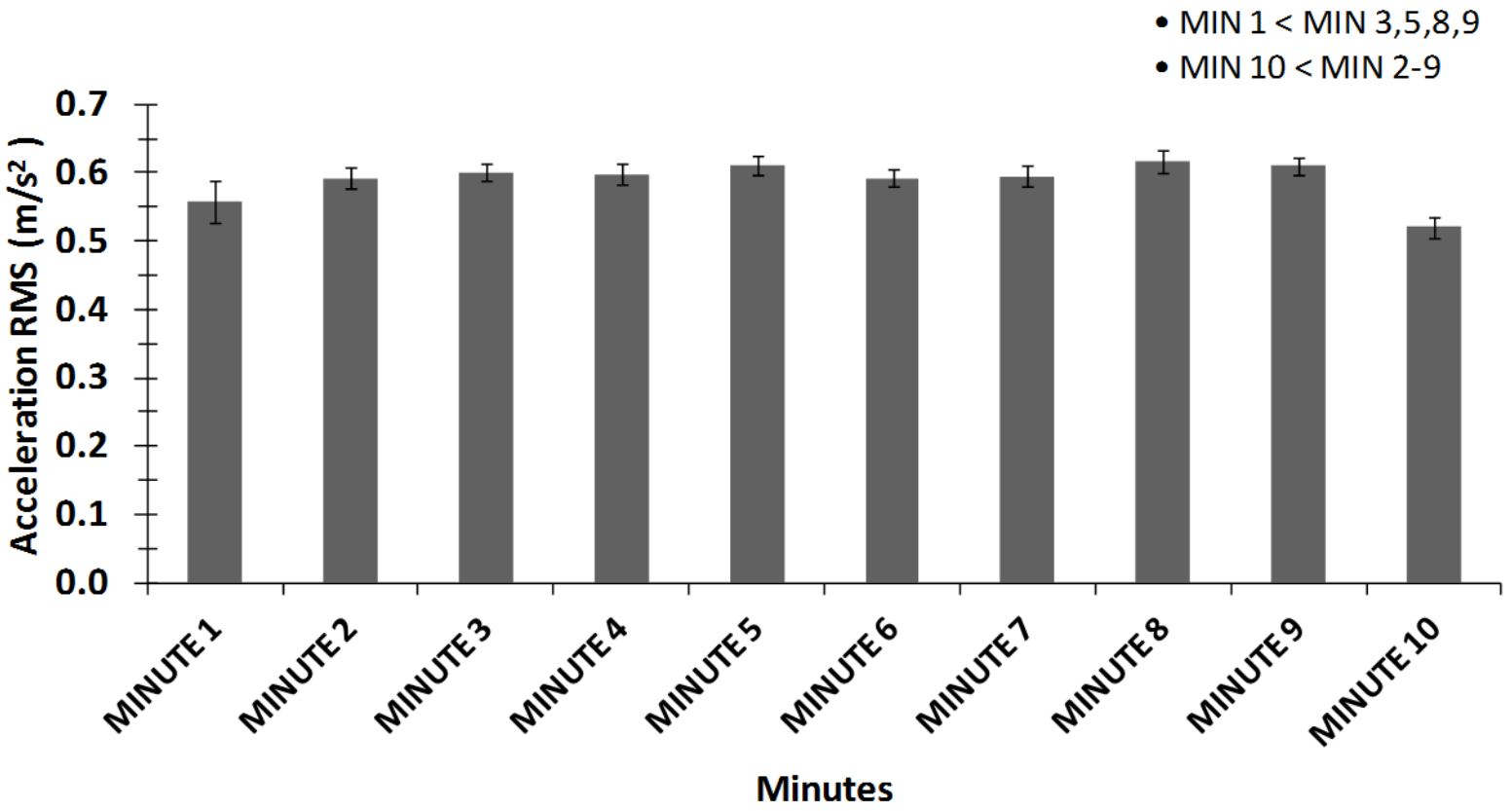

Figure 19. The mean (SD) seat acceleration RMS data within each of the 1 minute epochs during the pre-perturbation trial for the Y axis. Significant differences identified from post hoc analysis are denoted on the graph for the significant axis x minute's interaction.

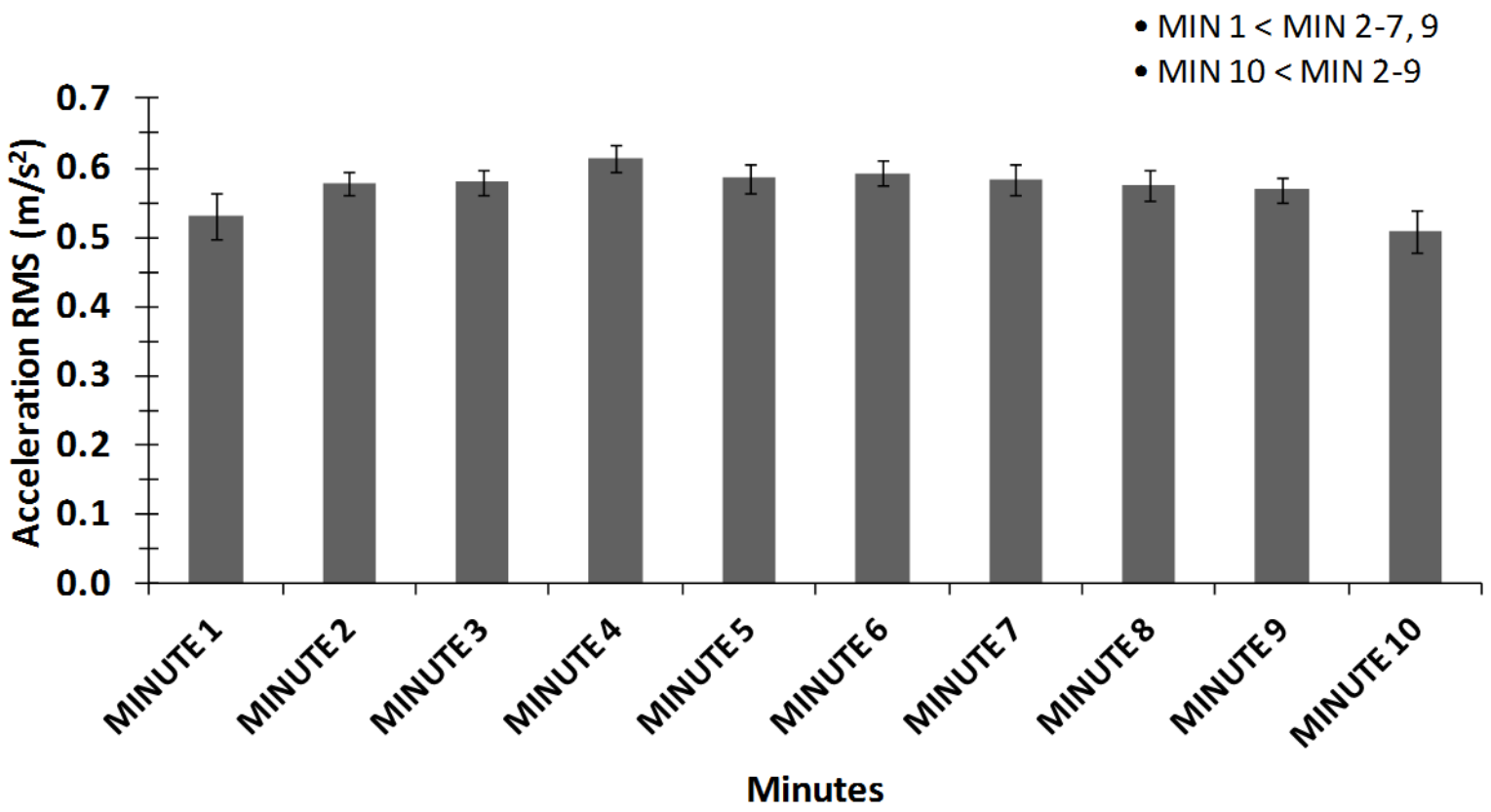

Figure 20. The mean (SD) seat acceleration RMS data within each of the 1 minute epochs during the pre-perturbation trial for the Z axis. Significant differences identified by post hoc analysis are denoted on the graph for the significant axis x minutes interaction.

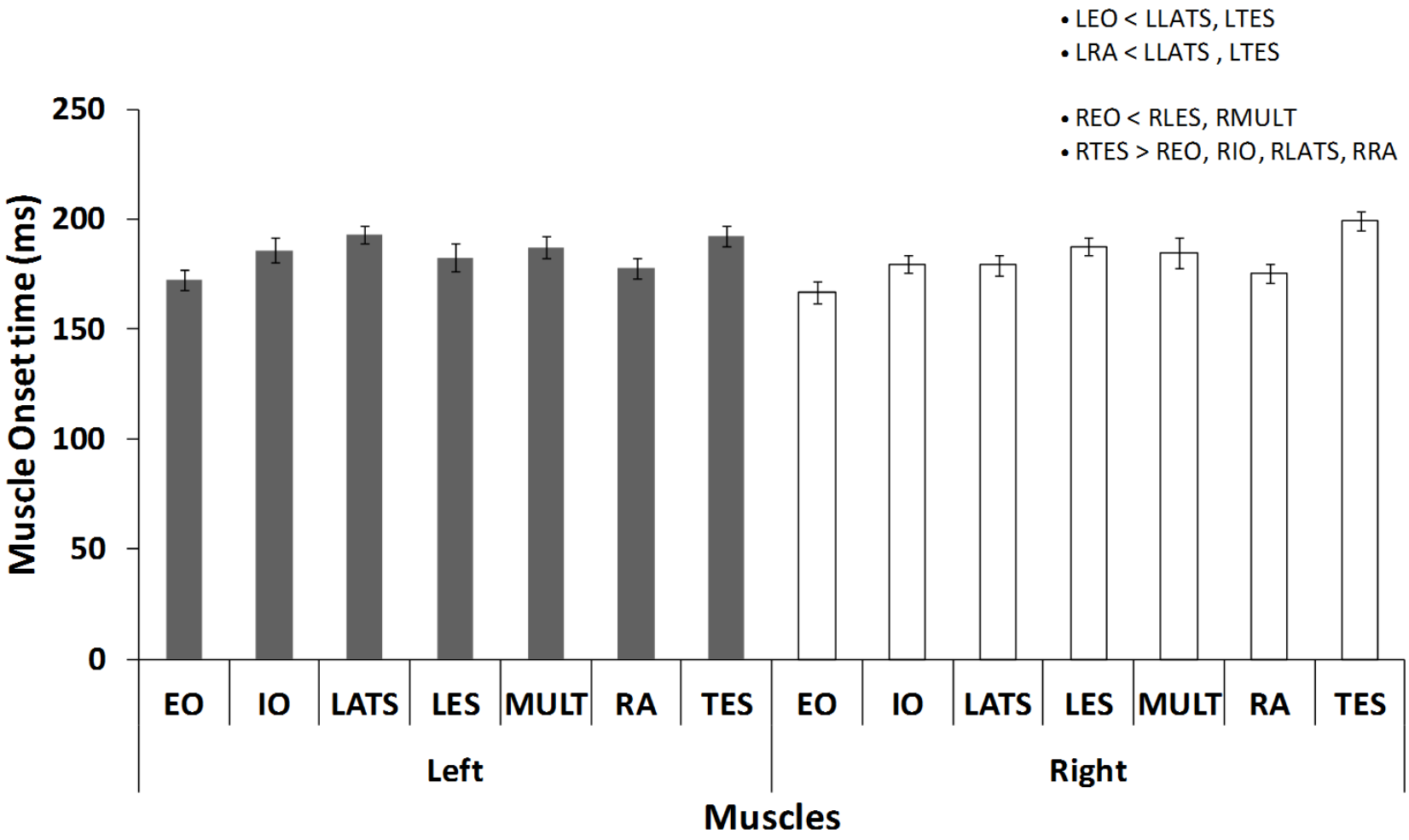

Figure 21. The mean (SD) muscle onset data for the EO, IO, LATS, LES, MULT, RA and TES muscles on both left and right sides during the perturbation protocol, showing the significant differences identified by post hoc analysis (denoted on the graph) for the interaction between muscle and side.

Another significant interaction was found between muscle and perturbation direction ($F=8.57$, $p<0.01$, $\eta^2_p=0.44$). Post hoc tests were performed on the data by comparing differences in muscle onset timing for the individual muscles within each perturbation direction. In the right direction, the TES muscle took 17% longer to activate than the EO, 13% longer than the IO and 14% longer than the RA (Figure 22). Similarly, in the left perturbation direction the TES muscle took 21% longer than the EO to activate (Figure 23). In the front perturbation direction the RA onset the quickest, being 7% faster than the IO, 16% faster than the LATS, 15% faster than the LES, 12% faster than the MULT and 17% faster than the TES (Figure 24). Lastly, in the back perturbation direction, the RA had a slower onset in comparison to the EO, LATS, LES, MULT and TES by 14%, 16%, 15%, 12% and 17%, respectively (Figure 25).

The muscle onset also showed a significant interaction between side and perturbation direction ($F=4.98$, $p<0.01$, $\eta^2_p=0.31$). Post hoc tests were performed on the data by comparing differences in muscle onset timing for the perturbation directions within each muscle side. On the right side, the front perturbation direction (169 ± 4.5 ms) had a quicker response than the back (186.3 ± 4.7 ms) and left (196.8 ± 4.2 ms) directions, whereas the right (175.1 ± 3.2 ms) direction also showed a significantly quicker response than the left direction. Interestingly, a main effect of group was also found for muscle onset (Figure 26) ($F=14.52$, $p<0.01$, $\eta^2_p=0.57$). The control group had a 14% faster muscle onset time than the vibration group.

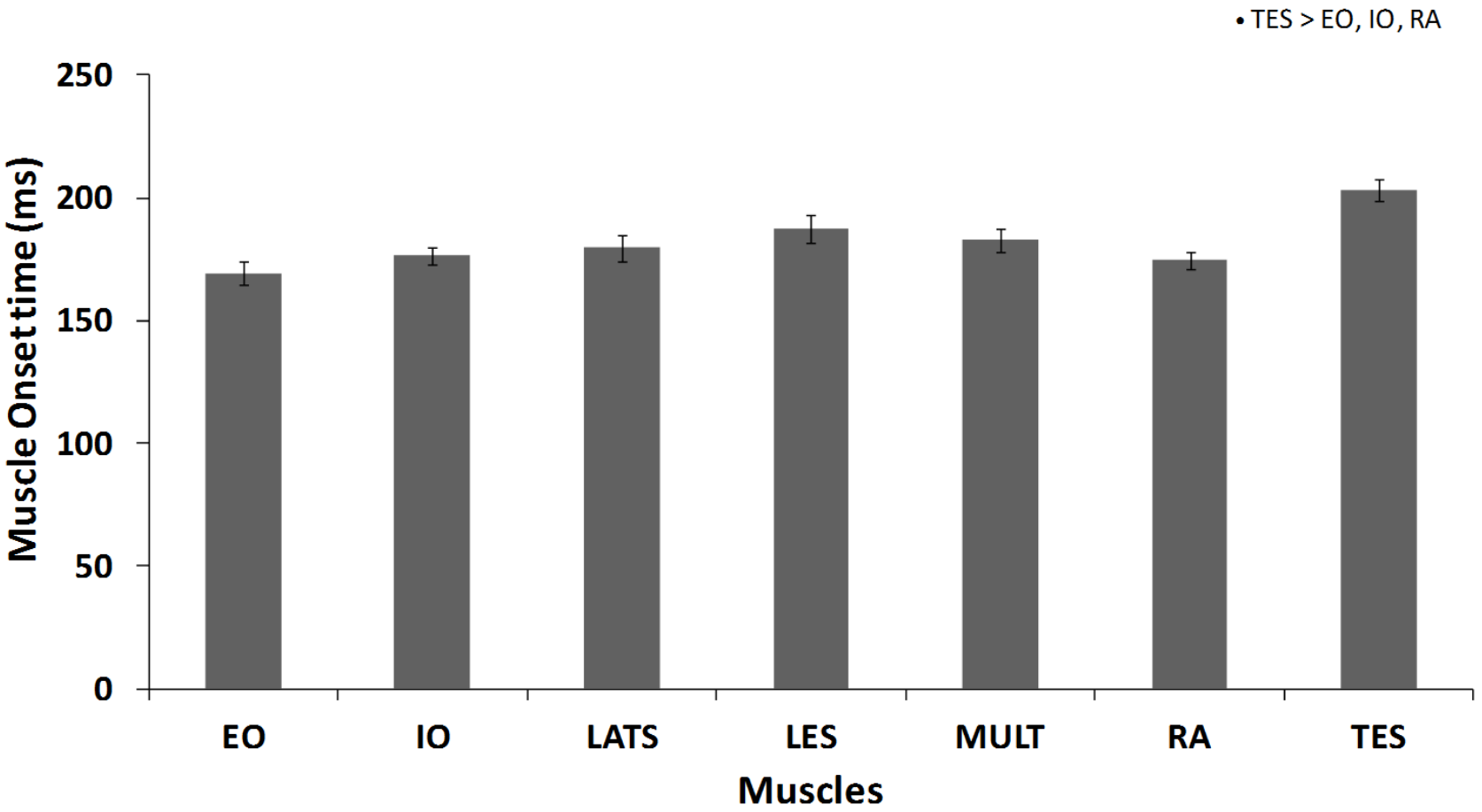

Figure 22. The mean (SD) muscle onset data for the EO, IO, LATS, LES, MULT, RA and TES muscles in the right perturbation direction, showing the significant differences identified by post hoc analysis (denoted on the graph) for the interaction between muscle and direction.

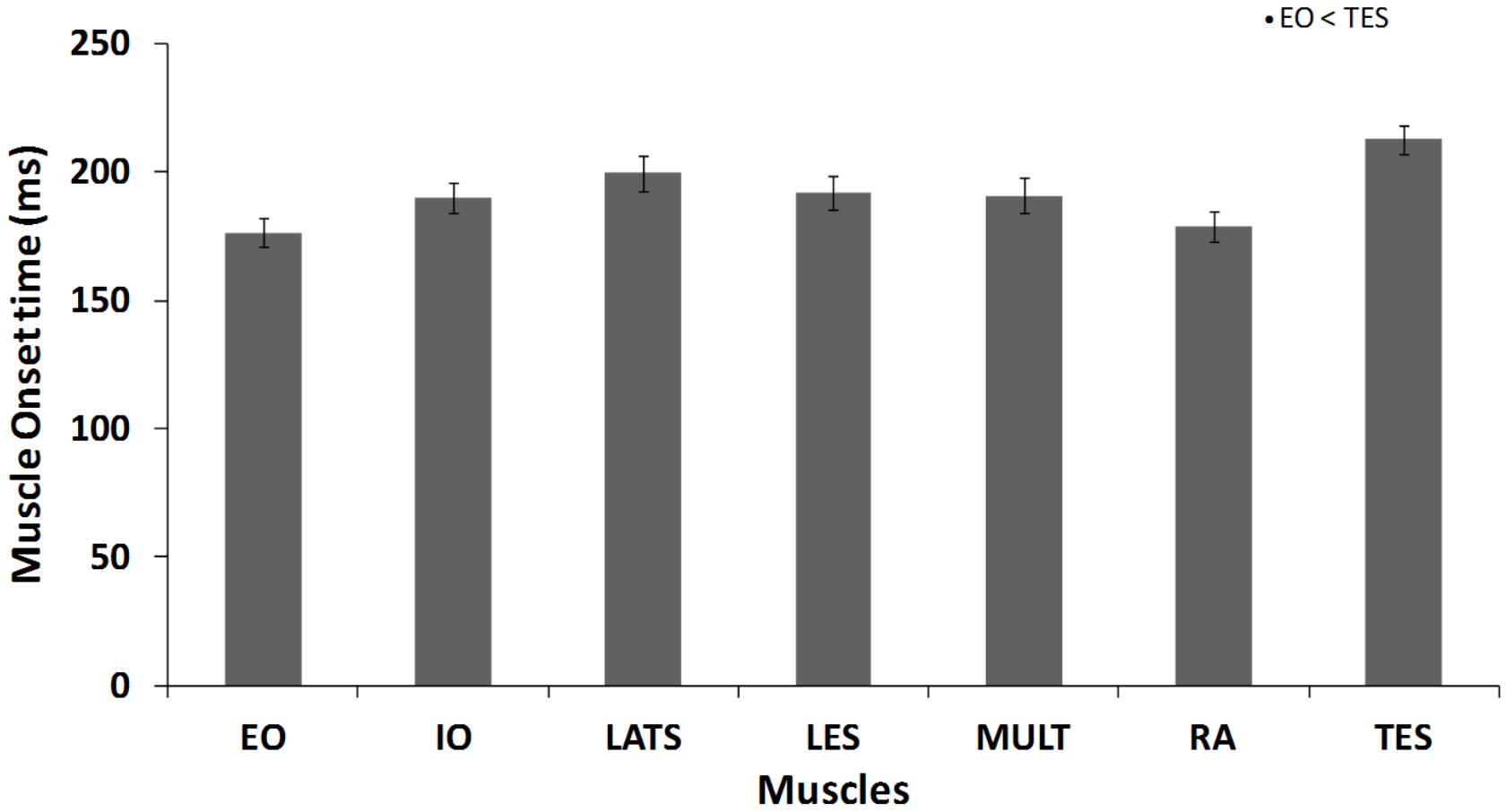

Figure 23. The mean (SD) muscle onset data for the EO, IO, LATS, LES, MULT, RA and TES muscles in the left perturbation direction, showing the significant differences identified by post hoc analysis (denoted on the graph) for the interaction between muscle and direction.

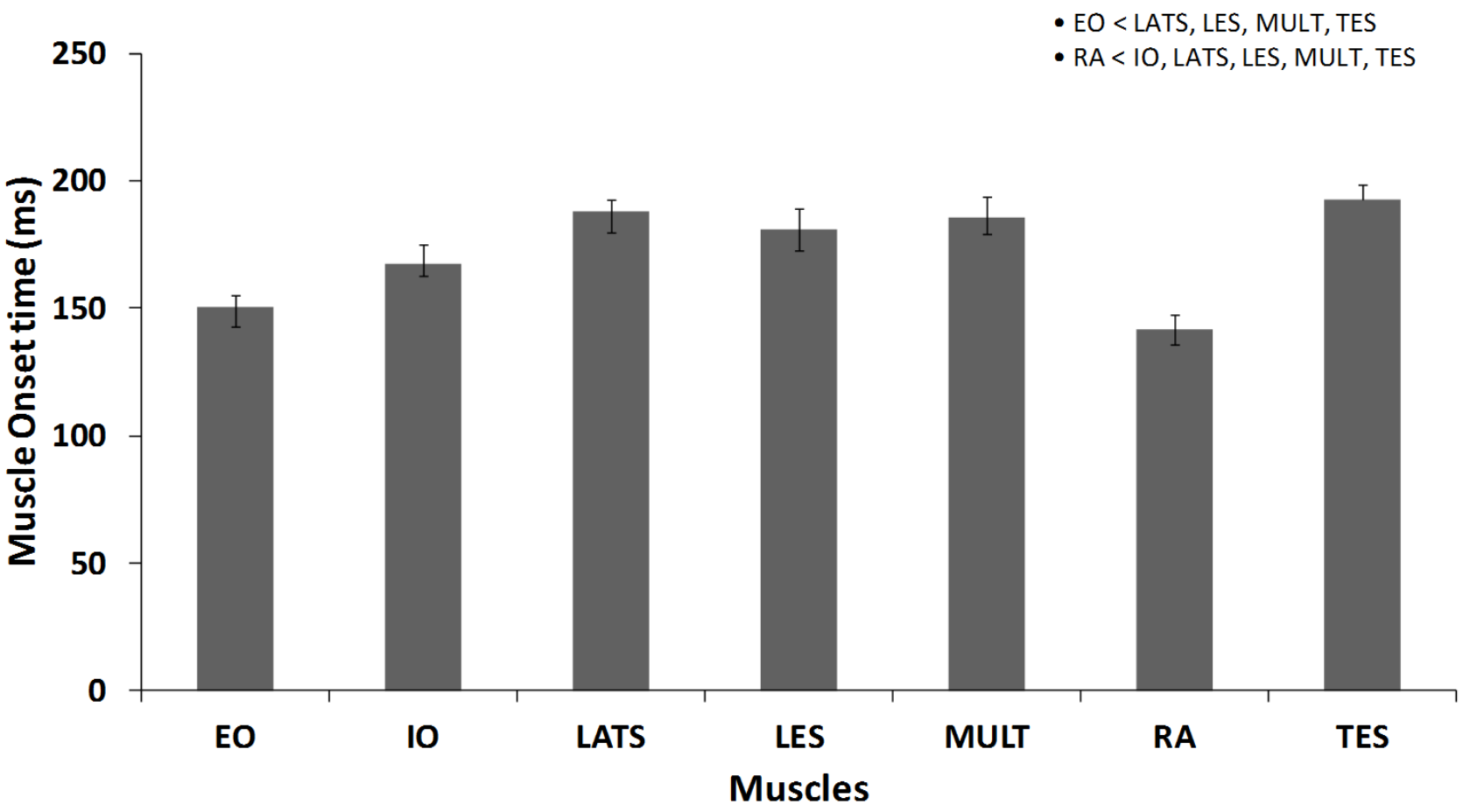

Figure 24. The mean (SD) muscle onset data for the EO, IO, LATS, LES, MULT, RA and TES muscles in the front perturbation direction, showing the significant differences identified by post hoc analysis (denoted on the graph) for the interaction between muscle and direction.

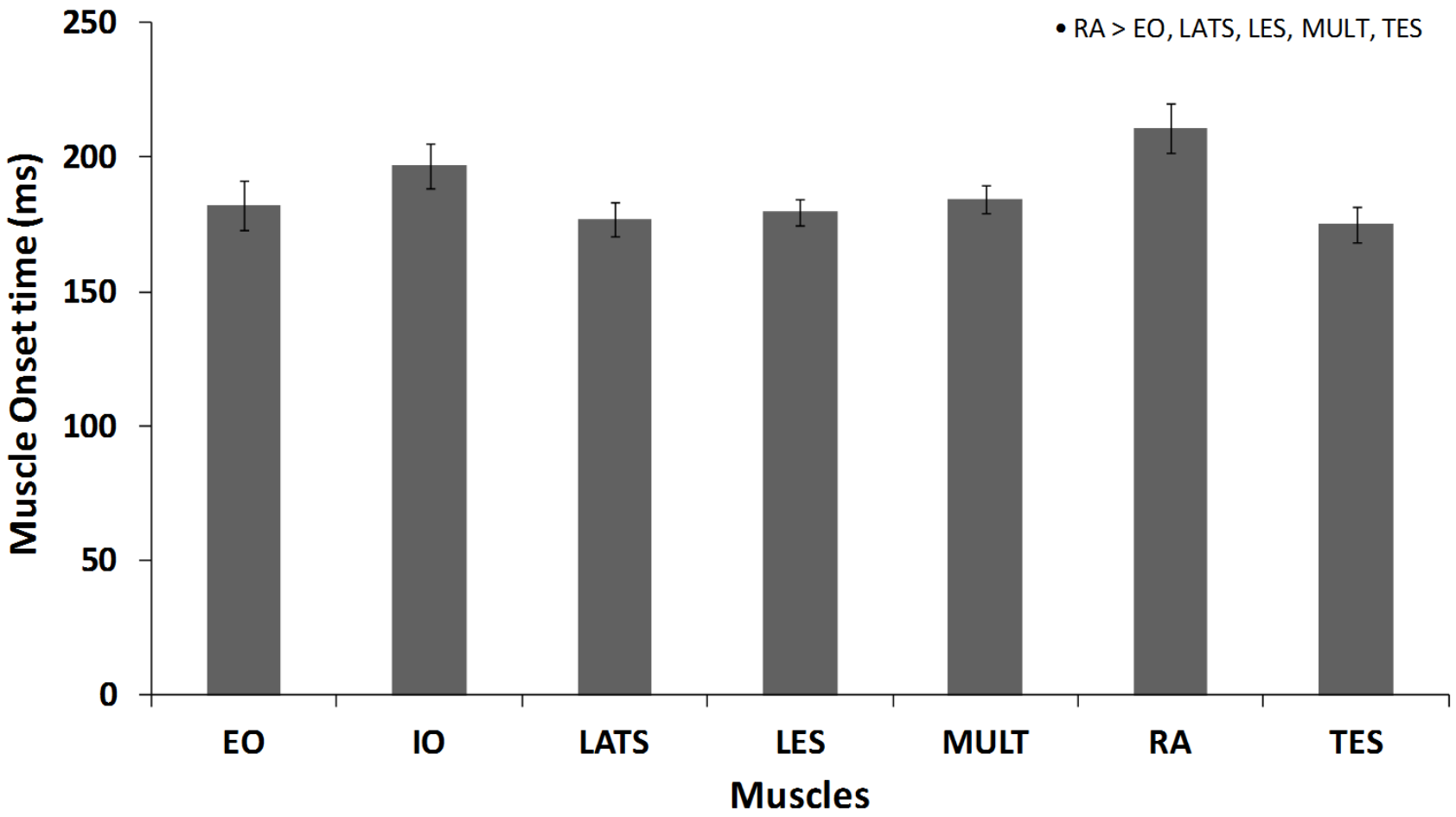

Figure 25. The mean (SD) muscle onset data for the EO, IO, LATS, LES, MULT, RA and TES muscles in the back perturbation direction, showing the significant differences identified by post hoc analysis (denoted on the graph) for the interaction between muscle and direction.

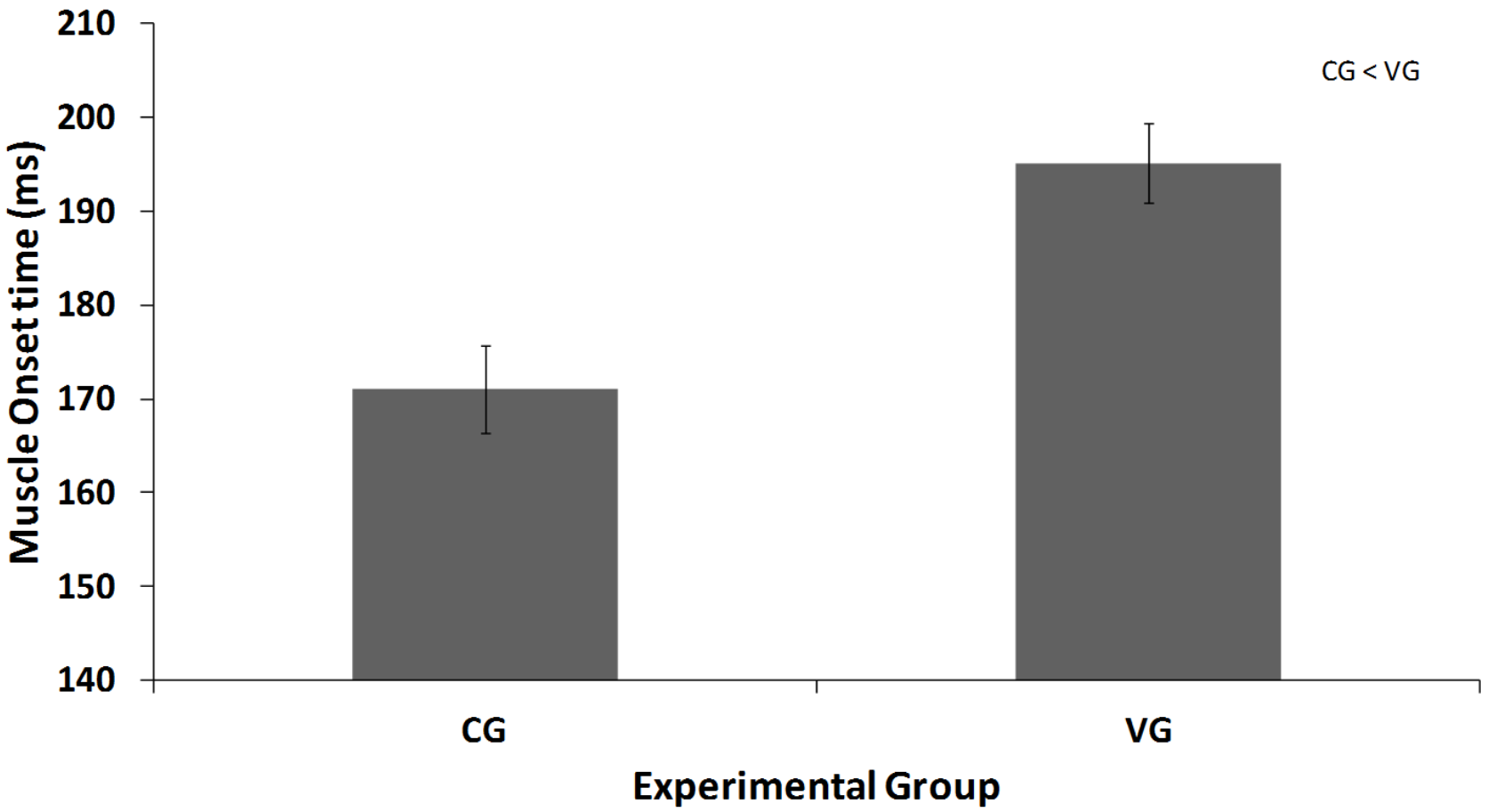

Figure 26. The mean (SD) muscle onset data during the perturbation protocol for the control group and vibration group, showing the main effect of group. Significant differences are denoted on the graph.

4.2.2. sEMG Neuromuscular Response

4.2.2.1 Baseline

The baseline time period for sEMG amplitude showed a significant interaction between muscle and side. Post hoc tests were performed on the data by comparing differences in the sEMG amplitudes for each muscle within each respective side. However, post hoc analysis did not indicate any significant differences for any muscles on either side

4.2.2.2 Pre-Perturbation

The pre-perturbation time period for sEMG amplitude showed to have a significant interaction between muscle and side. Post hoc tests were performed on the data by comparing differences in the sEMG amplitudes for each muscle within each respective side. However, upon post hoc analysis showed no significant differences between each of the muscles on either side.

4.2.2.3 Pre-Voluntary

The pre-voluntary time period for sEMG amplitude showed to have a significant interaction between muscle and side. Post hoc tests were performed on the data by comparing differences in the sEMG amplitudes for each muscle within each respective side. However, upon completing post hoc tests no significant differences were identified between each of the muscles on either side.

4.2.2.4 Voluntary

The sEMG amplitude during the voluntary time period exhibited a significant 3-way interaction of muscle, side and perturbation direction ($F=2.36$, $p<0.01$, $\eta^2_p=0.18$). Post hoc tests were performed on the data by comparing differences in sEMG amplitudes for the individual muscles

within each side and perturbation direction. The post hoc analysis showed no significant differences in the sEMG amplitude for any muscle, on the right or left sides, during the back perturbation direction (Figure 27). This was also the case for the right side in the right perturbation direction. During the front perturbation direction condition, post hoc tests identified that the sEMG amplitude of the RIO was more than four times lower than the REO, nearly four times lower than the RLATS and five times lower than the RLES muscles; whereas the RLES was 71% higher than the RMULT muscle. The sEMG amplitude of the LEO was 71% higher than the LLATS, 53% higher than the LLES and 88% higher than the LRA muscles. The LRA was four times lower than the LIO and nearly six times lower than the LMULT. Lastly, the LLATS was nearly two times lower in sEMG amplitude than the LMULT. The differences in sEMG amplitude for the muscles in the front perturbation direction are shown in Figure 28. Post hocs showed significant differences in the left perturbation direction where the RRA had greater muscle activation than the RIO by 83%, RLATS by 67% and RMULT by 78%, The left side muscles had significantly greater amplitudes for the LMULT, being 74% greater than the LLATS, 60% greater than the LLES, 90% greater than the LRA and 83% greater than the LTES (Figure 29). In the right perturbation direction, the LMULT was 70% higher in amplitude than the LLATS, 85% higher than the LRA and 73% higher than the LTES muscles (Figure 30).

4.2.2.5 Peak

The peak muscle response time for sEMG amplitude had a significant 4-way interaction between group, knowledge direction, side and muscles ($F=2.68$, $p<0.05$, $\eta^2_p=0.20$). Post hoc tests were performed on the data by comparing differences in sEMG amplitudes for the individual muscles within each side, group and direction knowledge condition. The REO in the control group

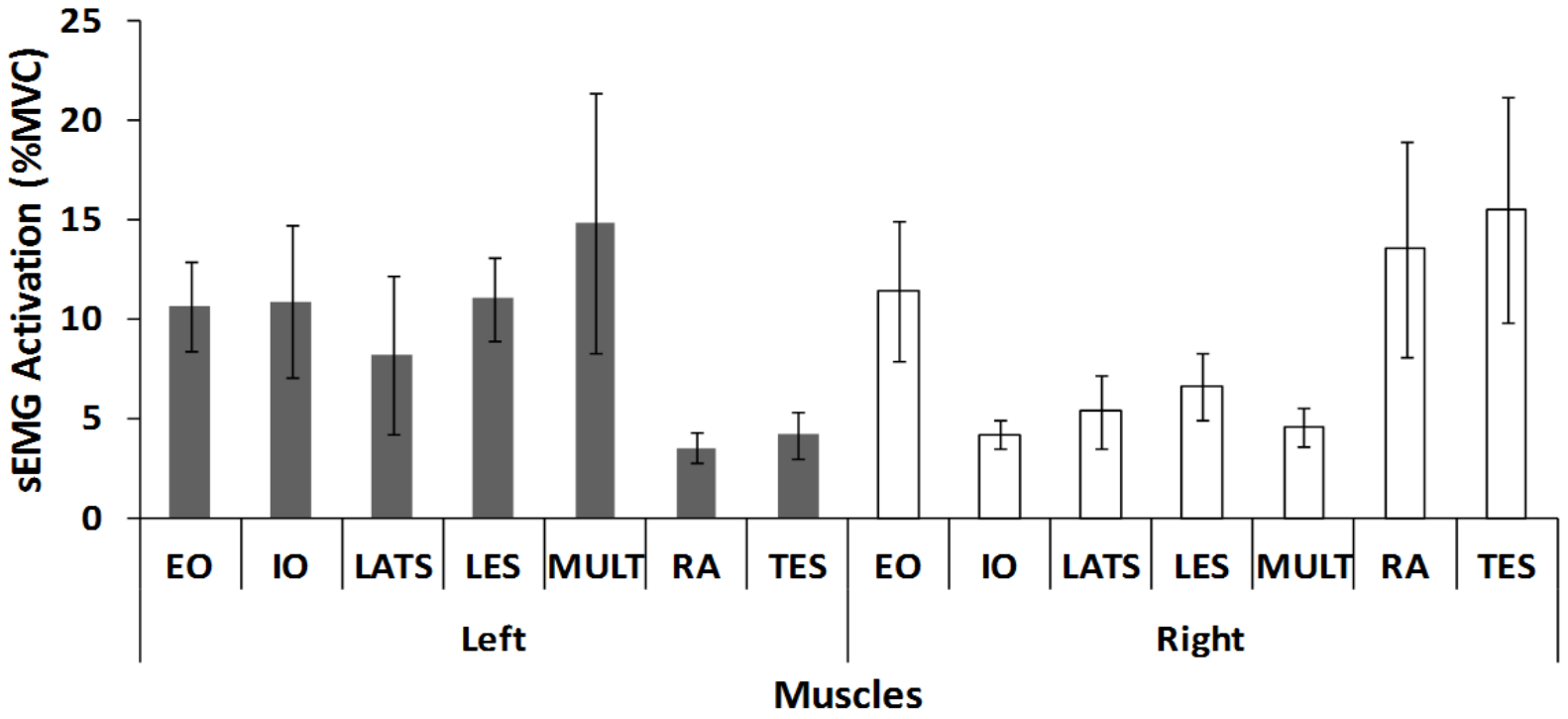

Figure 27. Mean (SD) sEMG neuromuscular response data for the left and right EO, IO, LATS, LES, MULT, RA and TES muscles during the voluntary time period when participants were perturbed in the back direction. There were significant differences in this direction upon completing post hoc analysis for the interaction between muscles, side and direction.

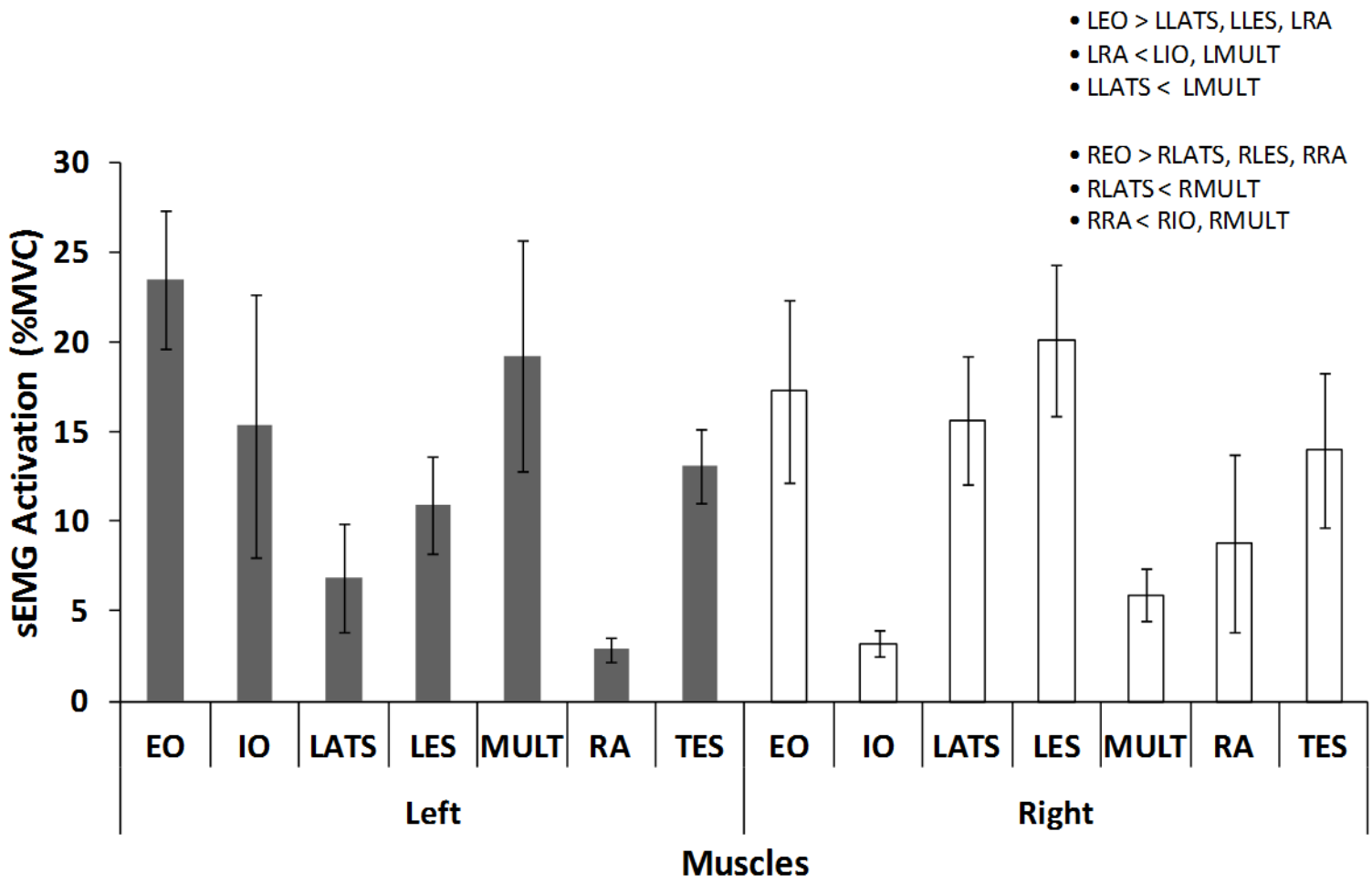

Figure 28. Mean (SD) sEMG neuromuscular response data for the left and right EO, IO, LATS, LES, MULT, RA and TES muscles during the voluntary time period when participants were perturbed in the front direction, showing the significant differences identified by post hoc analysis (denoted on the graph) for the interaction between muscles, side and direction.

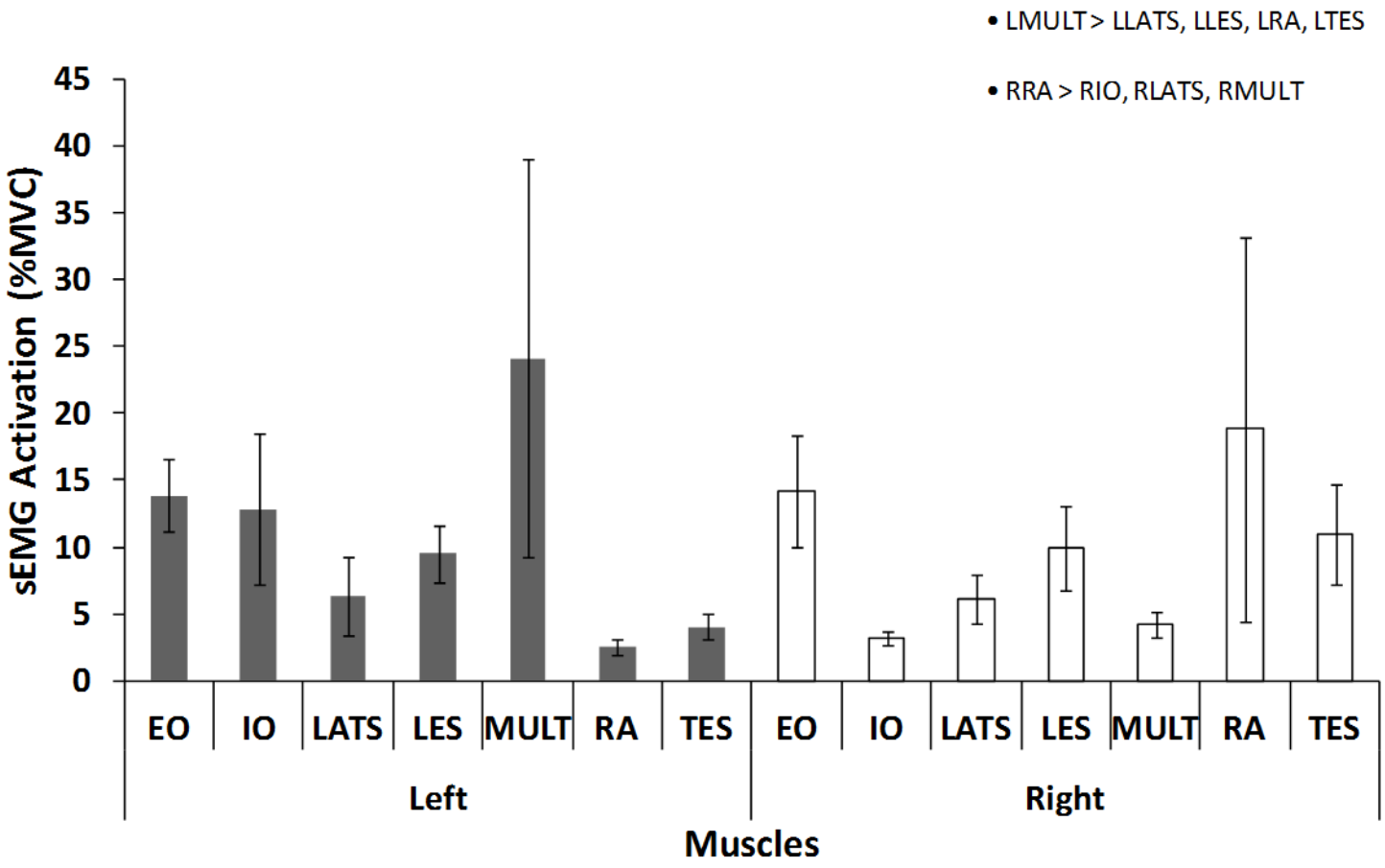

Figure 29. Mean (SD) sEMG neuromuscular response data for the left and right EO, IO, LATS, LES, MULT, RA and TES muscles during the voluntary time period when participants were perturbed in the left direction, showing the significant differences identified by post hoc analysis (denoted on graph) for the interaction between muscles, side and direction.

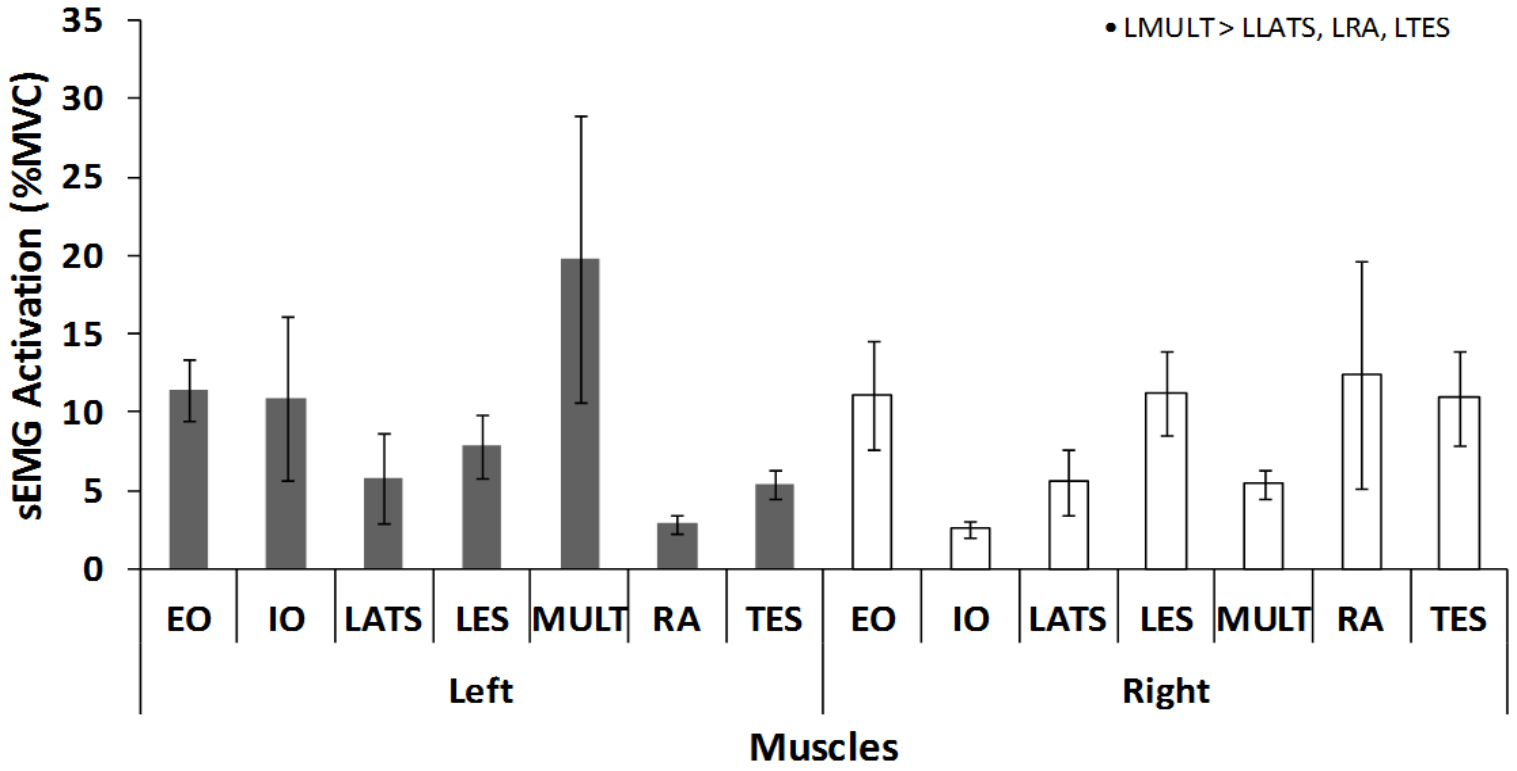

Figure 30. Mean (SD) sEMG neuromuscular response data for the left and right EO, IO, LATS, LES, MULT, RA and TES muscles during the voluntary time period when participants were perturbed in the right direction, showing the significant differences identified by post hoc analysis (denoted on the graph) for the interaction between muscles, side and direction.

perturbations on the right side for the KD condition showed was 72% higher than the RIO and 73% higher than the RRA muscles, whereas the RTES muscle was significantly higher than all other muscles by an average of 82%. Perturbations on the left side for the control group, in the KD conditions, were significantly greater for the LMULT than all other left side muscles by an average 59%. Further, the LEO, LIO and LLES were significantly greater than the LATS, LRA and LTES muscles by an average 69%. Figure 31 shows the differences in sEMG amplitudes between all muscles on both sides of the body for the control group in the KD condition. The control group perturbations on the right side for the UD condition were significantly higher for the REO than the RIO by 73% and RRA by 74%, whereas the RTES muscle was significantly higher than all other muscles by an average 82%. Perturbations on the left side for the control group, in the UD condition, were significantly greater for the LMULT by 65% compared to all other left side muscles, on average. Further, the LEO, LIO and LLES were an average of 69% greater than the LLATS, LRA and LTES muscles. Figure 32 shows the differences in sEMG amplitude between all muscles on both sides of the body for the control group in the unknown direction condition. The vibration group LIO was 68% higher than all other muscles on the left side in the known perturbation direction conditions, on average (Figure 33). The LMULT was 33% greater than the LEO, 28% greater than the LLATS, 80% greater than the LRA and 60% greater than the LTES, whereas the LEO, LLATS and LLES are an average 67% greater than both the LRA and LTES for the KD condition. The LLES was 25% greater than the LEO. On the right side the vibration group for the KD conditions showed the REO, RRA and RTES muscles were all significantly different from all other muscles, where REO was greater than the RRA by 24%, the RRA was greater than the RTES by 42%, the RTES was greater than the RIO, RLATS, RLES and RMULT by 82%, 42%, 67% and 73%, respectively. Furthermore, the

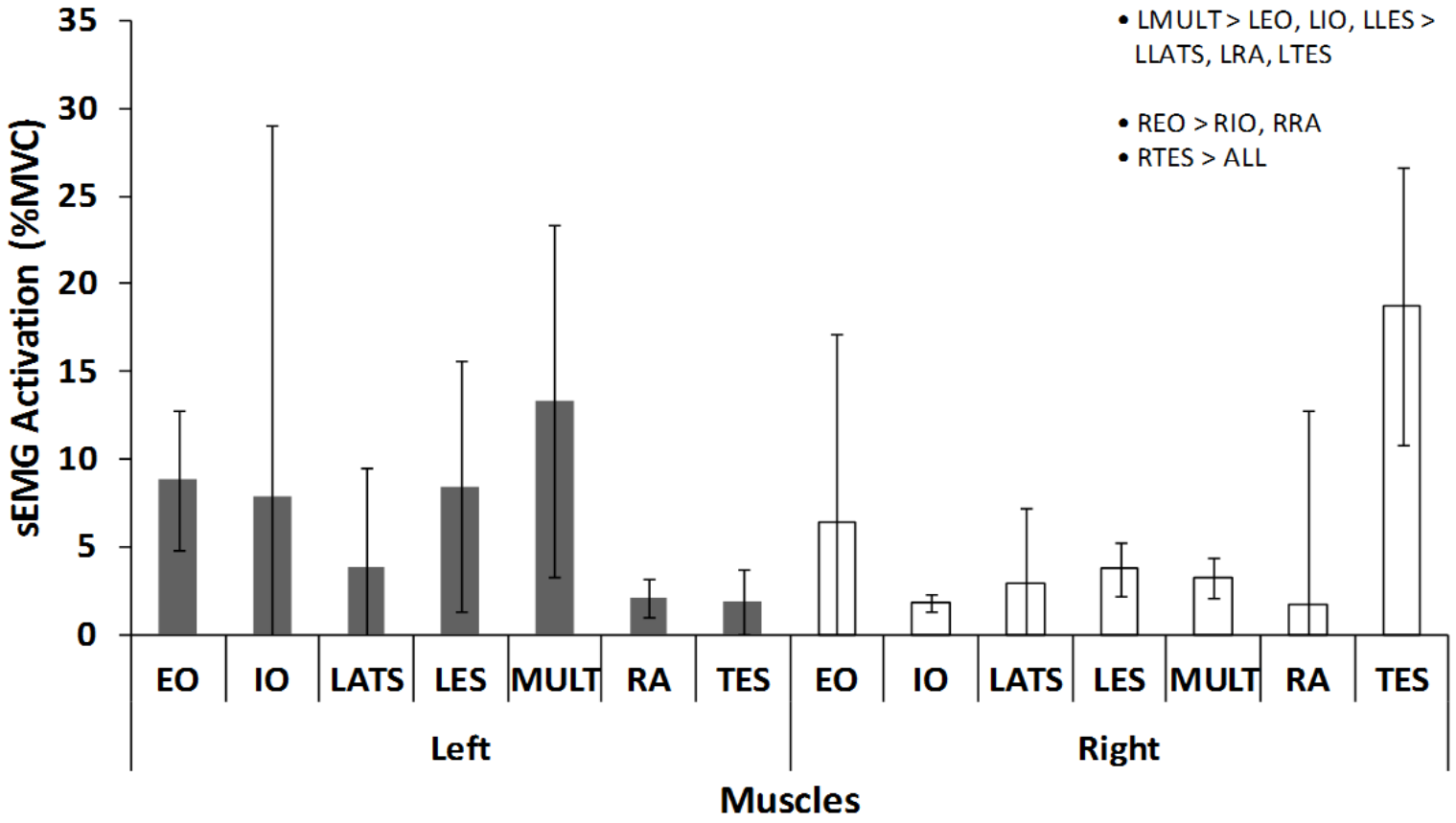

Figure 31. Mean (SD) sEMG neuromuscular response data for the left and right EO, IO, LATS, LES, MULT, RA and TES muscles during the peak time period when participants in the control group were perturbed in a known direction, showing the significant interactions identified by post hoc analysis (denoted on graph) for the interaction between group, muscles, side and direction knowledge.

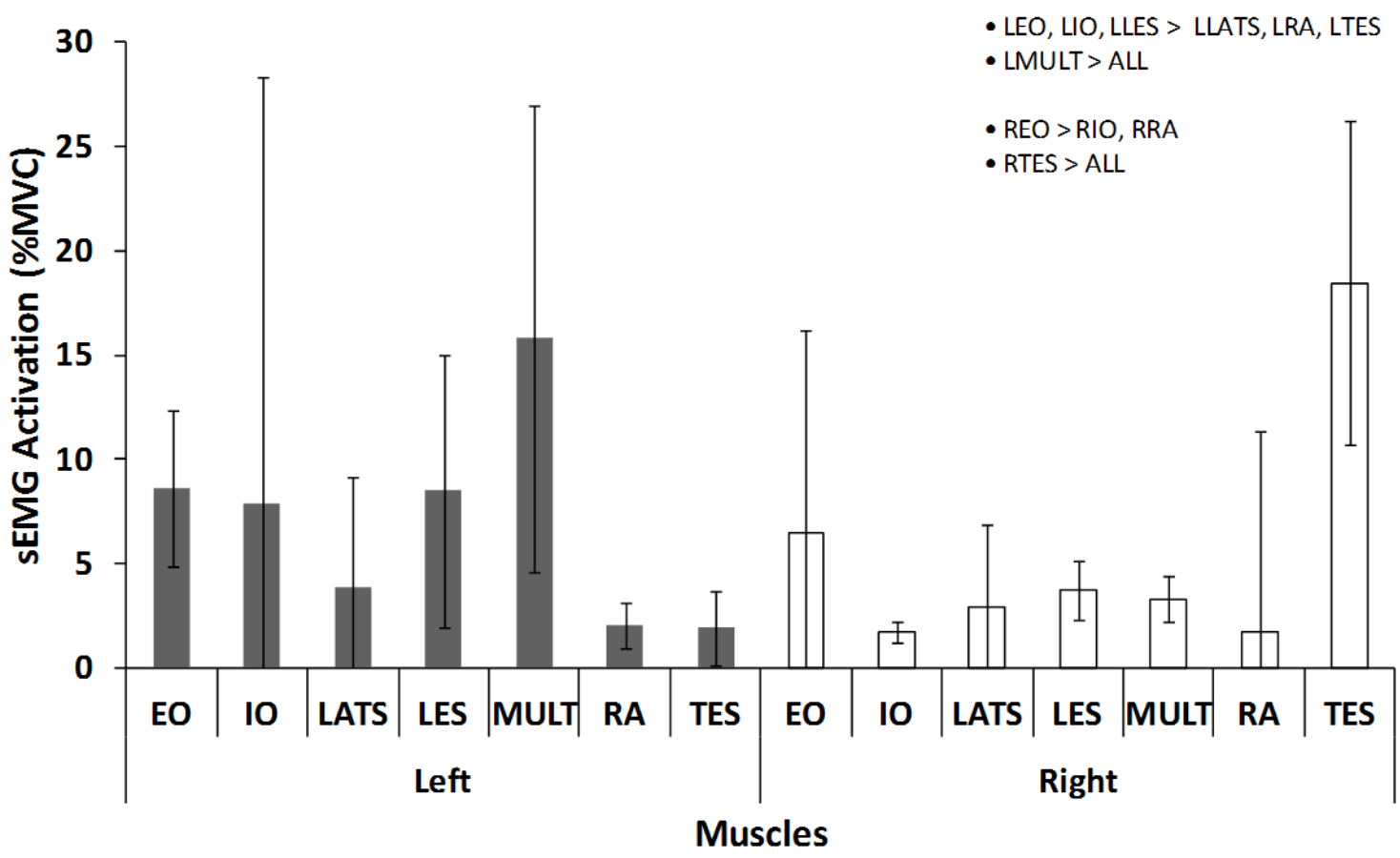

Figure 32 . Mean (SD) sEMG neuromuscular response data for the left and right EO, IO, LATS, LES, MULT, RA and TES muscles during the peak time period when participants in the control group were perturbed in an unknown direction, showing the significant differences identified by post hoc analysis (denoted on graph) for the interaction between group, muscles, side and direction knowledge.

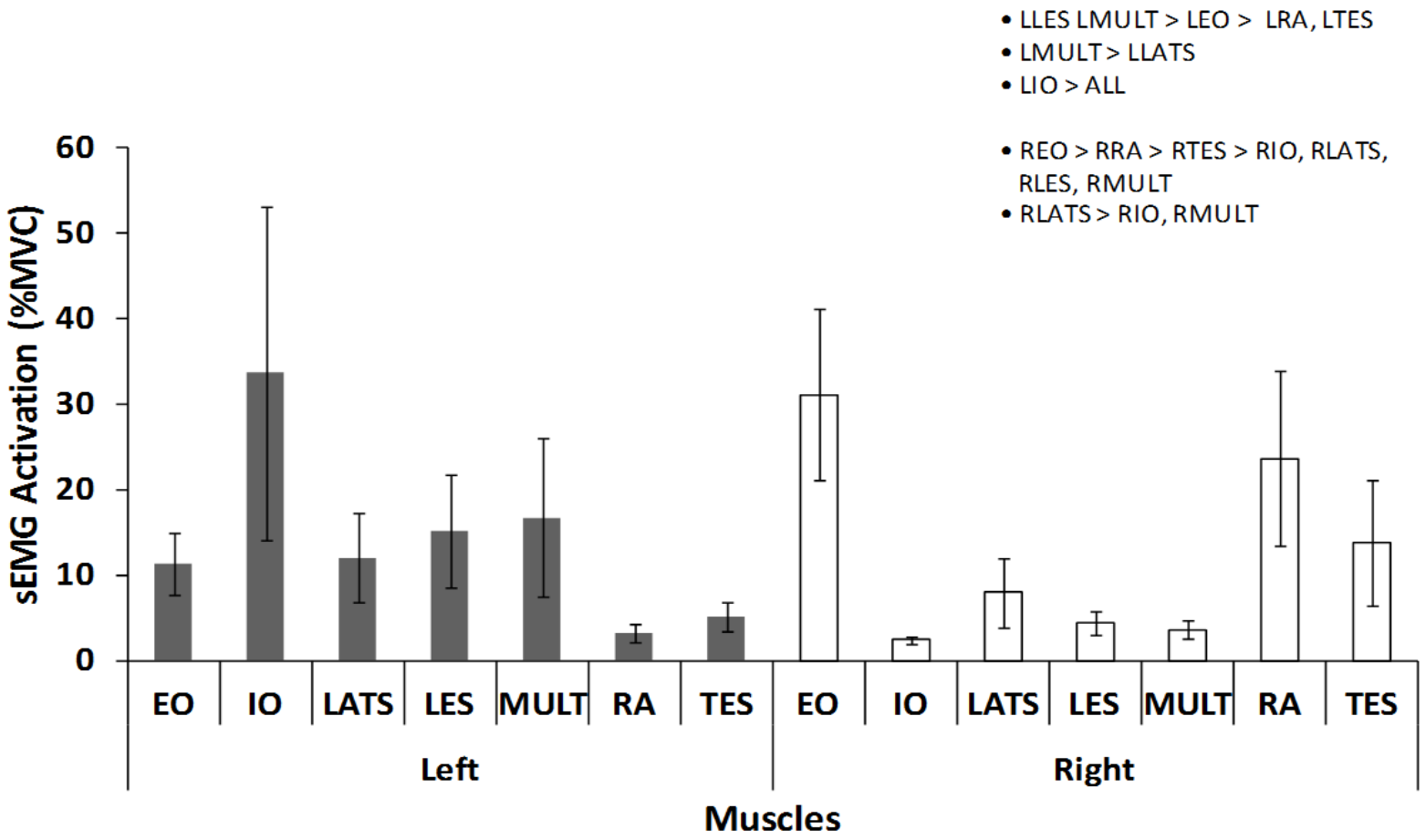

Figure 33. Mean (SD) sEMG neuromuscular response data for the left and right EO, IO, LATS, LES, MULT, RA and TES muscles during the peak time period when participants in the vibration group were perturbed in a known direction, showing the significant differences identified by post hoc analysis (denoted on graph) for the interaction between group, muscles, side and direction knowledge.

RLATS was 70% higher than the RIO and 54% higher than the RMULT. The vibration group LIO was an average 68% higher than all other muscles on the left side for the unknown perturbation direction condition (Figure 34). The LMULT was 33% greater than the LEO, 30% greater than the LLATS, 80% greater than the LRA and 69% greater than the LTES, whereas the LEO, LLATS and LLES are also an average of 67% greater than the LRA and LTES for the UD condition. On the right side, the vibration group for the UD condition showed that the REO, RRA and RTES muscles were all significantly different from all other muscles, where REO was greater than the RRA by 39%, RRA was greater than the RTES 27%, the RTES was greater than the RIO, RLATS, RLES and RMULT by 81%, 43%, 65% and 73%, respectively. Lastly, the RLATS was 68% higher than the RIO and 53% higher than the RMULT.

4.2.3 L_{4-5} Joint Angle

4.2.3.1 Baseline

The baseline time period for the L_{4-5} joint angle showed a main effect of axis ($F=163.04$, $p<0.01$, $\eta^2_p=0.94$) where the flexion-extension (Y) axis showed a significantly greater joint angle (-3.22º ± 0.24) in comparison to the lateral bend (X) (-0.01º ± 0.06) and axial twist (Z) axes (0.31º ± 0.15).

4.2.3.2 Pre-perturbation

The pre-perturbation time period for the L_{4-5} joint angle showed a main effect of axis ($F=161.09$, $p<0.01$, $\eta^2_p=0.94$) where the Y axis resulted in a significantly greater joint angle from neutral (-3.21º ± 0.24) in comparison to the X (-0.003º ± 0.06) and Z axes (0.31º ± 0.15).

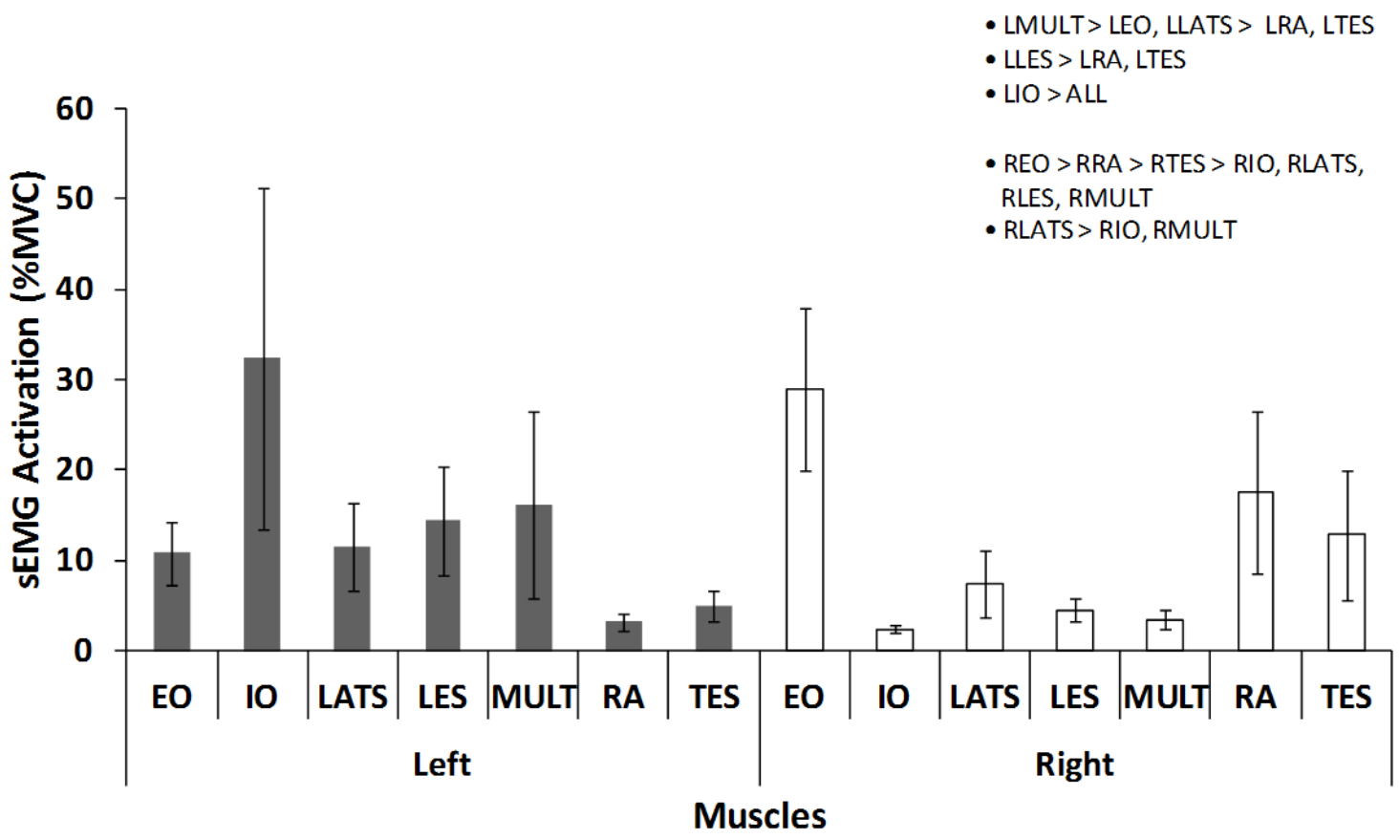

Figure 34. Mean (SD) sEMG neuromuscular response data for the left and right EO, IO, LATS, LES, MULT, RA and TES muscles during the peak time period when participants in the vibration group were perturbed in an unknown direction, showing the significant differences identified by post hoc analysis (denoted on graph) for the interaction between group, muscles, side and direction knowledge.

4.2.3.3 Pre-Voluntary

The $L_{4\text{-}5}$ joint angles for the pre-voluntary time period indicated a significant interaction between axis and perturbation direction ($F=4.45$, $p<0.01$, $\eta^2_p=0.29$). Post hoc tests were performed on the data by comparing differences in $L_{4\text{-}5}$ joint angles for the perturbation directions within each axis. In the Y axis, the front direction had a significantly smaller joint angle from neutral (-3.10º ± 0.23) than that in the left direction (-3.34º ± 0.24). Whereas in the Z axis, the left direction a showed to have a significantly greater joint angle from neutral (0.40º ± 0.17), in comparison to the right direction (0.18º ± 0.13). Due to the small differences in $L_{4\text{-}5}$ joint angles between the perturbation directions across the axes, these results were not found to be functionally relevant.

4.2.3.4 Voluntary

The voluntary time period for the $L_{4\text{-}5}$ joint angle showed a significant interaction between axis and direction ($F=12.31$, $p<0.01$, $\eta^2_p=0.53$). Post hoc tests were performed on the data by comparing differences in $L_{4\text{-}5}$ joint angles for the perturbation directions within each axis. In the Y axis, the back direction showed the greatest deviation from neutral (-3.75º ± 0.25) and was significantly larger than all other directions. Further, the front direction showed the least deviation from neutral (-3.03º ± 0.24), being significantly less than both the back (-3.75º ± 0.25) and left (-3.43º ± 0.23) directions. Due to the small differences in $L_{4\text{-}5}$ joint angles between the perturbation directions across the axes, these results were not found to be functionally relevant.

4.2.3.5 Peak

The peak response time had a significant 3-way interaction between the axis, direction and direction knowledge ($F=3.75$, $p<0.01$, $\eta^2_p=0.25$). Post hoc tests were performed on the data by

comparing differences in $L_{4\text{-}5}$ joint angles for each axis within each direction and direction knowledge condition. Upon completing post hocs there were no significant differences between the individual axes across each perturbation direction and either direction knowledge condition.

Chapter 5

DISCUSSION

The purpose of this study was to investigate the effects of WBV exposure on the voluntary and involuntary neuromuscular responses prior to, during and following inertial-based trunk perturbations. The novel methodology used in this study included the use of a robot to produce inertial-based trunk perturbations, which was used to determine that muscle sEMG onsets were delayed following a bout of vibration exposure, and that these onsets occurred sooner in muscles whose actions oppose the direction of perturbation. In addition, there were no significant differences in sEMG magnitude for perturbations of known and unknown directions, which was contrary to the original hypothesis. Lastly, the RA, EO, MULT and TES muscles all showed significantly increased sEMG during both the voluntary and peak time periods following perturbations, illustrating their vital roles in trunk stability.

5.1 MUSCLE ONSETS

Participants in the vibration group were exposed to the vibration and perturbation protocol throughout the study, whereas, the control group was simply exposed to the perturbation protocol. From this, there was a main effect of experimental group where the muscle onset timing where the vibration group onsets were significantly slower in comparison to the control group. Similar results were found by Wilder et al. (1996), who concluded that 40 min of seated WBV exposure showed significantly more delayed muscle responses to unexpected loads with both spatial and temporal anticipation. Furthermore, Arashanapalli & Wilson (2008) determined that 20 min of direct vibration exposure, to the paraspinal muscles about the L_3 joint, resulted in a slower time to peak muscle activation in response to sudden perturbations causing forced trunk

flexion. In fact, delayed muscular responses in people who are exposed to vibration and those who are at an increased risk of a LBI have been discussed in research and are thought to be correlated (Wilder et al., 1996; Arashanapalli & Wilson, 2008; Li, Lamis, & Wilson, 2008). It has been hypothesized by Brumagne et al. (1999) and Arashanapalli & Wilson (2008) that vibration exposure creates delayed neuromuscular responses due to transmission errors within the proprioceptive system, specifically affecting the muscle spindles, in the form of the muscle lengthening illusion. The CNS interprets muscle lengthening to occur with vibration exposure, via the inaccurate neuromuscular feedback from the muscle spindles, which leads to changes in the muscles' response to a disturbance or perturbation (Roll & Vedel, 1982; Brumagne et al., 1999; Arashanapalli & Wilson, 2008).

The sEMG onset timing for muscles whose actions oppose the direction of force (antagonists) from the perturbation occurred sooner in comparison to the agonistic muscles recorded in this study. Perturbations in the front direction, causing forced trunk extension, showed the RA and REO muscles to both have significantly faster muscle onset times in comparison to the other muscles measured. Both of these muscles aid in flexing the torso, an action which would oppose the forced trunk extension caused by the forward perturbation, in an attempt to regain torso equilibrium. Similar results were found in research where participants who were posteriorly loaded showed shorter onset latencies in the abdominal muscles than in the erector spinae muscles (Vera-Garcia et al., 2006). In addition, Cort & Potvin (2012) determined that the greatest contributions to joint stability were from muscles whose actions oppose the direction of perturbation. These findings support the hypothesis that the body regains equilibrium after a perturbation by first activating those muscles whose primary actions oppose the direction of forced movement.

5.2 NEUROMUSCULAR RESPONSE

It was expected that increased sEMG amplitudes would occur during perturbations of known direction, in comparison to those of unknown direction; however, this result was not found. The initial hypothesis was made with the assumption that participants would increase the sEMG amplitudes in the trunk muscles during perturbations of known direction in an attempt to increase the stiffness of the trunk, thus, reducing the destabilizing effects from the perturbations. This study, however, showed similar sEMG amplitudes for both perturbations in the known and unknown directions. A study by (Silfies, Mehta, Smith, & Karduna, 2009) examined the anticipatory effects that occurred during perturbations of known timing and found that participants without LBP increased their muscular activation prior to a trunk perturbation, in an attempt to increase stiffness, and thus stability at the spine. In contrast, Cort et al. (2013) examined the neuromuscular response to inertial-based trunk perturbations and had similar findings to the current study's, where direction knowledge did not have an effect on the neuromuscular response to trunk perturbations. Furthermore, the study by (Cort et al., 2013) also exposed participants to both temporal and spatial anticipation prior to perturbations. Since the methodology of perturbation delivery in the Cort et al. (2013) study was very similar to the current study, it is not coincidental that these studies had similar results. There may be a greater neuromuscular influence in temporal anticipation, than spatial anticipation. Though not examined in this work, Cort et al. (2013) determined that participants' sEMG activity was increased during perturbations of known timing, which has also been supported by other research (Vera-Garcia et al., 2007). It was apparent that temporal anticipation allowed the neuromuscular system to organize its activation patterns prior to a perturbation, and has been shown to maximize stability in the torso (Vera-Garcia et al., 2007).

A further examination into the neuromuscular response results from this study showed a greater sEMG response to occur in muscles whose actions oppose the direction of force from the perturbation. Specifically, the RA, EO, MULT and TES muscles had a greater neuromuscular responses to perturbations opposing the directions in which they primarily create forces. As examples, perturbations in the back direction, causing forced trunk flexion, during the voluntary time period, showed the TES and MULT muscles to have the greatest muscle activation, followed by the RA and EO muscles; for perturbations in the front direction, causing forced trunk extension, the EO had significantly greater sEMG activation, followed by the MULT. The EO is a muscle whose origin and insertion allows it to wrap around the entire trunk, with exception to the posterior region (Tortora & Nielsen, 2012). Being that it covers a large cross-sectional area both laterally and frontally, it has an effective moment arm about the vertebral joints and can be very beneficial in efficiently stabilizing the trunk in situations that require it to regain equilibrium (Crisco, III & Panjabi, 1991). The current findings show that the EO plays a significant role in regaining equilibrium of the torso after a perturbation and is supported by previously published literature. Specifically, Brown and Potvin (2007) determined that the EO was a dominant muscle in providing trunk stiffness around the flexion-extension, lateral bend and axial rotation axes. Furthermore, the oblique muscles activated at higher levels during bracing maneuvers to help maintain stability of the torso prior to a perturbation (Brown et al., 2006). Bracing maneuvers help increase trunk stability and regain equilibrium, after a perturbation, through the activation of both the agonist and antagonistic muscles involved (Brown et al., 2006; Brown & Potvin, 2007).

In this study, as previously mentioned, the EO had greater activation, followed by the MULT in the forward perturbation direction, which causes a forced extension motion of the torso. Based

on this result, it is the researcher's assumption that the MULT was acting as a counter balance to the early activation of the EO to help maintain stability of the L_{4-5} joint. Although the efficiency of smaller, intersegmental muscles is not as great as those which span multiple segments, the activation of these smaller muscles is necessary to maintain stability of the lumbar spine (Crisco & Panjabi, 1991; Cholewicki & McGill, 1996). It is the coordination of the activation of smaller muscles in conjunction with the activation of the larger, intersegmental muscles, which helps to effectively maintain stability and prevent buckling in the spine (Cholewicki & McGill, 1996). Similar results were discussed in a study by Brown and McGill (2008) where the EO and erector spinae muscles (at the L_5 level) showed increased activation from the initiation to the end of applied bending moments in flexion-extension and lateral bend directions. Furthermore, co-contraction of the muscles leads to a state of greater equilibrium and enhanced stability (Cholewicki & McGill, 1996; Vera-Garcia et al., 2006; Brown et al., 2006). Creating this state of equilibrium and stability of the muscles in the trunk allows appropriate transferring of shear and compressive forces to the lower body, thus reducing the risk of injury (Cholewicki & McGill, 1996).

5.3 PRE-PERTURBATION

The vibration exposure used in this study was within the ISO 2631-1 (1997) standards for WBV exposure, where participants in the VG were exposed to seated, tri-axial WBV at frequencies between 1-8 Hz and not exceeding RMS of 0.55 m/s^2. Though the vibration profiles that created robotic motion were designed to cause accelerations no greater than 0.55 m/s^2 about all axes, the average RMS experienced by participants, as measured by the seat accelerometer upon which the participants were seated, was 0.61m/s^2, 0.59m/s^2 and 0.57m/s^2 for the X, Y and Z axes, respectively. It was assumed that the differences between the accelerations output by the robot

and those experienced by participants were due to the amplification from the seat on which the participant was secured. Research by (Kittusamy & Buchholz, 2004) looked at the effects of vibration and seated postures in operators of earth-moving machinery. During a regular working cycle, the seats were amplifying vibration frequencies, especially those in the lower frequency range, between 1-2 Hz in the X and Y axes, and 4-8 Hz in the Z axis. In addition, a study by Burdorf and Swuste (1993) found that when measuring the transmissibility coefficient of 11 different vehicle seats, that in 29% of the cases, the vibration signal was being amplified, causing the drivers to experience greater vibration signals and RMS accelerations at the seat surface. In their study, participants were drivers of agricultural tractors, fork-lifts and lorries in the workplace portion and were two participants whose weights were 53kg and 95kg in the laboratory portion (Burdorf & Swuste, 1993). It should be mentioned, however, that though seat amplification has occured in previous research, and is hypothesized to have occurred in the current study, that the averaged vibration signals that participants experienced for minutes 2-9 in the X axis were 0.63 m/s^2, Y axis were 0.60 m/s^2 and Z axis were 0.58 m/s^2, whereas those for minutes 1 and 10 had a mean of 0.53m/s^2 across all axes due to the initiation and conclusion of the vibration.

Interestingly, no significant differences between the VG and CG for the sEMG MnPF during the 10 min pre-perturbation protocol were reported, illustrating that there were no muscle fatiguing affects in the vibration group. Studies by Potvin (1997) and Flynn et al. (2004) have found that when MnPF decreases by 15% from resting levels, this is the level at which fatigue can be measured. The decreases in the current study from min 1 (207.55 Hz ± 53.41) to min 10 (202.72 Hz ± 50) were only 2% for the CG and were <1% for VG, from minute 1 (181.49 Hz ± 42.9) to minute 10 (180.91 Hz ± 44.12). These findings contrast those of Wilder et al. (1996)

who determined that 40 min of seated 5 Hz WBV exposure to induce muscle fatigue in trunk muscles. It should be noted, however, that the vibration exposure time used in this study, in comparison to the current study, was very different for participants, where they had exposed participants to continuous vibration for 40min, rather than 10min. Though differences in the results of the current study, in comparison to the aforementioned studies, can be explained through the variation in methodology, an important variable that was not mentioned in either study was the change in MnPF from muscles prior to and following vibration exposure. The fatiguing effects in these studies were implied through changes in muscle reaction time and twitch force, which are different than the methodology used to assess fatigue in the current study. Assessment of fatigue through the decrease in MnPF has been shown in many studies as a reliable indicator of muscle fatigue, whereby the decrease is due to the recruitment of slow-twitch muscles when fast twitch muscles can no longer withstand the force or endurance requirements of the load (Dolan, Mannion, & Adams, 1995; Potvin, 1997; Flynn, Holmes, & Andrews, 2004). These findings support the notion that any reported differences between the VG and CG during the perturbation protocol were not due to fatiguing effects of the muscles, and was rather due to the direct effects of the vibration exposure on the recorded muscles. Research by Arashanapalli et al. (2008) discusses the sensitivity of the muscle spindle receptors to vibration and even using this modality as an effective tool in evaluating the role of the muscle spindles. Based on the results from the current study, and those from other studies, this work supports the hypothesis that vibration negatively affects the muscle spindle receptors as shown through delayed muscle response timings.

5.4 HYPOTHESES REVISITED

1) *Increased latencies will occur during the pre-voluntary time period for those in the VG when compared to the CG.*

The results from this study reject this hypothesis as the vibration group did not have significantly longer delays in comparison to the control group during the pre-voluntary time period. Significant delays in the muscle onset timing were determined to occur between the experimental groups, where the vibration group had longer delays to muscle onset, during the voluntary time period. This result was similar to those of Arashanapalli et al. (2008) and Santos et al. (2008) who determined that vibration exposure increased both delay in timing of the neuromuscular response in lumbar muscles at the L_1 level and the timing to peak muscle onset.

2) *sEMG onset timing for muscles whose actions oppose the direction of force of the perturbation will occur sooner than those who act with the direction of the perturbation..*

Participants were exposed to ten perturbations in each of four directions, causing forced flexion, extension and lateral bending to both left and right sides. This hypothesis was rejected as the results showed that for perturbations in the front direction, causing forced trunk extension, the RA onset was significantly faster than the LIO, LATS, LES, TES and MULT muscles. Further, the RA was the last muscle to onset during perturbations in the back direction, causing forced trunk flexion. A study by Vera-Garcia et al. (2006) who posteriorly loaded their participants, had significantly faster muscle onset timing in the abdominals than the erector spinae muscles. The proper recruitment of muscles during trunk perturbations allows the body to enable stability and maintain equilibrium of the system (Vera-Garcia et al., 2006; Brown et al., 2006).

3) *Increases in muscle activation are expected to occur during perturbations of known direction, when compared to those of unknown direction, during the pre-perturbation time period.*

The results from this study did not show any significant differences in sEMG amplitude between perturbations in a known or unknown direction, therefore this hypothesis was rejected. It is important to note, however, that the current study exposed participants not only to spatial anticipation, but also to temporal anticipation prior to the perturbations. Research by Silfies et al. (2009) has shown the importance of temporal anticipation prior to a perturbation, being that it provides participants the opportunity to increase their muscle activation as a means of creating stiffness to the system, in an attempt to better accommodate the disturbance from a perturbation. Since temporal anticipation was not an experimental variable in the current study, it is recommended that this variable be considered for research of this type in the future, to gain further knowledge on its effects on muscle responses.

4) *Decreased $L_{4\text{-}5}$ joint angle was expected to result during known direction perturbations in comparison to those in the unknown direction, after the perturbation occurs.*

The results reject this hypothesis as there were no significant differences between joint angles in either the known or unknown direction perturbations. Similar findings were reported by Cort et al. (2013) where knowledge of perturbation direction did not have an effect on any joint angle differences for perturbations of known or unknown directions. When studies by Brown et al. (2006) and Vera-Garcia et al. (2006) who examined the effects of abdominal bracing and trunk co-contraction prior to and during perturbations are examined, it was clear that trunk muscle activation is a more holistic activity done by muscles throughout the trunk, regardless of perturbation direction.

5.5 LIMITATIONS AND ASSUMPTIONS

In this study some limitations and assumptions were made regarding the fatiguing methodology, vibration timing and the generalizability of the results, all which deserve some discussion. Firstly, the amount of time that was used to determine the presence of vibration-induced fatigue in participants was shorter in comparison to that reported in other studies; the current study examined 10 min of seated WBV vibration prior to the perturbation protocol. Studies by Wilder et al. (1996) and Arashanapalli et al. (2008) had exposed their study participants to 40 and 20 min of vibration, respectively. Though the amount of time used in this study was chosen based on previous research by Wilder et al. (1994) who showed positive indices of muscle fatigue during 10 min of vibration exposure, it is unknown whether or not a longer exposure time would have changed the result of the fatigue state for those participants in the vibration group and may be worth investigating. An investigation into the effects of vibration on fatigue throughout the perturbation protocol would be worthwhile as the recovery from perturbations could have been affected by the exposure to vibration. Lastly, in consideration of the demographic of participants that were used in this study, the application of the results to a broader population may be limited as the participants in this study were young, presumably healthy, male participants, who were free from LBP. This subset may not be representative of all LHD, tractor or truck drivers as these drivers are often older than those used in the current study's sample (Wilder & Pope, 1996). Furthermore, it was determined that the older the drivers are, the more likely they are to experience LBP and a LBI, which is due to the morphological changes that may have already occurred in their spines after years of driving and vibrations exposure (Bovenzi, 1996). It is important to note, however, that though the study sample used in this research study may not be an accurate representation of all occupational drivers, or the general population, that this

relatively homogeneous sample allowed the results to not be attributed to extraneous variables such as sex, age or the presence of low back pain. With the current methodologies, the current study has built a good foundation that will facilitate future research in examining other demographics and subsets of the population, for comparison.

Chapter 6

CONCLUSION

In conclusion, the results from this study showed that stochastic WBV exposure showed significantly longer delays in the neuromotor response to inertial-based perturbations. Specifically, participants in the vibration group showed delayed sEMG onset responses during the peak time period. Results from the study show that this delay was not due to fatiguing effects from the 10 min of vibration exposure during the pre-perturbation protocol, because there were no significant differences in the MnPF values between the CG and VG experimental groups. The sEMG amplitudes were not significantly different between experimental groups during the pre-perturbation protocol, implying that the difference in the onset timing must be due to other factors or mechanisms occurring in the muscle during vibration exposure, such as the muscle spindles.

Another interesting finding from this study was that muscle onsets occurred faster in muscles acting as antagonists to the direction of force initiated by the perturbation. The RA and EO muscles both had significantly faster muscle onsets in the forward perturbation direction, which caused forced trunk extension. In addition, these muscles, along with the MULT and TES muscles, showed higher sEMG amplitudes in response to perturbations in directions which oppose the direction of their primary actions. These findings were expected and support the notion that the muscles are acting to regain equilibrium and maintain stability of the trunk post perturbation.

6.1 IMPLICATIONS FOR INDUSTRY

This study incorporated vibration exposure within international industry standards. This enabled the results of the study to be more representative to what operators of LHD vehicles may be

experiencing in the workplace. When taking that into consideration, there are findings from this research that can be used as applications in industry:

- Exploring the opportunity for seats that aid in dampening the vibration signals that are experienced by the vehicle operators. As previously discussed, it has been supported in research that seats can amplify, rather than attenuate, vibration signals that are being emitted from the vehicle motor. Looking into the basic properties of the seat and enhancing the performance of the dampening components could help with this. Studies by Wilder et al. (1994) and (Blood, Ploger, Yost, Ching, & Johnson, 2010), have examined these properties with more depth and should be used in conjunction with the results of this study to find effective ways of attenuating vibration at the seat surface.
- Another interesting application for industry would be revisiting the conservative nature of the ISO 2631-1 standards, as the participants in this study were being exposed to vibrations that were below the median RMS acceleration values that were outlined in the Health Guidance Caution Zone, though delayed neuromuscular responses were present in participants. While keeping in mind the correlation between delayed neuromuscular responses and LBP, this finding may be a concern for vehicle operators as long-term vibration exposure may increase the risk of LBI.

6.2 FUTURE RESEARCH DIRECTIONS

Future research should concentrate on the relationship between vibration, delayed muscle responses and the development of low back pain. This research study has determined that participants who do not suffer from LBP experience delayed muscle responses with the exposure to vibration. Further research is needed to decipher the links between vibration and LBP, or to

support that they are mutually exclusive from one another, though, the ladder hypothesis is not expected.

Another area that would be interesting to explore further is to understanding the exact mechanisms which lead to the delays in the muscle response with vibration exposure. Though it has been hypothesized in research that the muscle spindle receptors are delivering erroneous sensory information to the motor neurons, which is leading to this delay, it is unknown if this is the sole mechanism involved in the delayed response. Future research which isolates these somatosensory receptors, with the addition of vibration and perturbation exposure as experimental conditions, would allow researchers to verify the true extent of their involvement in the delayed muscle response.

Lastly, the effect of temporal and spatial anticipation during vibration exposure is another area of research that would be beneficial to investigate. While other studies have explored these types of anticipation during normal, seated conditions, it would be interesting to see if temporal anticipation exacerbates the muscle response to perturbations with vibration exposure. Being that truck drivers experience perturbations from potholes or uneven driving surfaces, it would be interesting to better understand how these hazards affect the neuromuscular system. Furthermore, to add to the realism and more direct correlation to real-life scenarios, it would be interesting to explore vibration exposure for longer periods of time, and mimicking the same vibration acceleration profiles as those found in the industry, to increase the transfer of laboratory based knowledge to that being experienced in industry.

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APPENDIX A

Office of the Research Ethics Board

University of Windsor
thinking forward

Today's Date: January 10, 2012
Principal Investigator: Ms. Danielle MacIntyre
REB Number: 29792
Research Project Title: REB# 11-258: Trunk muscular response following inertial-based perturbations with and without exposure to whole body vibrations: Potential implications for vehicle-related injuries
Clearance Date: January 9, 2012
Project End Date: June 01, 2012
Milestones:
Renewal Due-2012/06/01(Pending)

This is to inform you that the University of Windsor Research Ethics Board (REB), which is organized and operated according to the Tri-Council Policy Statement and the University of Windsor Guidelines for Research Involving Human Subjects, has granted approval to your research project on the date noted above. This approval is valid only until the Project End Date.

A Progress Report or Final Report is due by the date noted above. The REB may ask for monitoring information at some time during the projects approval period.

During the course of the research, no deviations from, or changes to, the protocol or consent form may be initiated without prior written approval from the REB. Minor change(s) in ongoing studies will be considered when submitted on the Request to Revise form.

Investigators must also report promptly to the REB:
a) changes increasing the risk to the participant(s) and/or affecting significantly the conduct of the study;
b) all adverse and unexpected experiences or events that are both serious and unexpected;
c) new information that may adversely affect the safety of the subjects or the conduct of the study.

Forms for submissions, notifications, or changes are available on the REB website: www.uwindsor.ca/reb. If your data is going to be used for another project, it is necessary to submit another application to the REB. We wish you every success in your research.

Pierre Boulos, Ph.D.
Chair. Research Ethics Board

c.c. Dr. Jim Dickey, Co-Investigator, University of Western
Dr. Joel Cort, Supervisor, University of Windsor

This is an official document. Please retain the original in your files.

401 Sunset Avenue, Windsor, Ontario, Canada N9B 3P4 . tel: 519.253.3000 ext. 3948 . web: www.uwindsor.ca/reb

APPENDIX B

CONSENT TO PARTICIPATE IN RESEARCH

Title of Study: **Neuromuscular response of the trunk following inertial-based perturbations with whole-body vibration exposure.**

You are asked to participate in a research study conducted by **Danielle MacIntyre and Dr. Joel Cort**, from the **Department of Human Kinetics** at the University of Windsor as a requirement of Danielle MacIntyre's **master's level thesis.**

If you have any questions or concerns about the research, please feel free to contact **Danielle MacIntyre at (519) 253-3000 ext. 4999 (**macinty2@uwindsor.ca**) or Dr. Joel Cort at (519) 253-3000 ext. 4980 (**cortj@uwindsor.ca**).**

PURPOSE OF THE STUDY

The purpose of this study is to evaluate the effects of vibration exposure on the voluntary and involuntary responses of muscles before and after trunk perturbations (small jolts). In order to achieve this purpose, subjects will be perturbed under either vibrating conditions (experimental group) or non-vibrating conditions (control group). Each subject's muscle response times will be monitored and compared between the two experimental groups.

PROCEDURES

1) Subjects:

You will be randomly assigned to one of two groups: 1) control group (CG), where subjects are not exposed to vibration during the study and, 2) vibration group (VG), where subjects are exposed to vibration prior to and throughout the protocol. There will be ten people in each of the two groups – you will not know which group you are placed into until after the study is complete. Subjects will be males aged 18-30 years old, who have not experienced back pain in the last 12 months. You will fill-out a questionnaire to identify for the presence or absence of back pain.

2) Procedures:

Information such as height, weight and age will first be taken. The investigator will then attach 14 electrodes (these measure the electrical activity of the muscles) and motion-capture markers (these will help track your movements) to the skin on your back and chest.

- Maximal contractions – *Abdominals:* You will lie on your back in a sit-up position on pommel work-out bars, while your feet are placed under one of the bars, and attempt to do a sit-up against the resistance of the researcher or research assistant. Twisting and sideways bending will be added to this contraction to turn on different abdominal muscles. *Back Muscles:* a) You will lie on your stomach on the pommel work-out bars, with your feet under one of the bars, and attempt to pull yourself up against the resistance of the researcher or research assistant, without using your arms. b) You will attempt to raise your elbows by your sides against the resistance of the investigators.
  Each of these contractions will last 2-3 seconds – you will be given a 30 second rest between contractions.
- Pre-perturbation trial – You will be seated on a backless car seat and told to maintain a normal sitting posture. This seat is mounted and secured on a robotic platform. The investigator will secure you to the seat. You will remain seated for 10 min under a vibrating or non-vibrating condition. If you are in the vibrating condition, you will experience vibrations under the current ISO standard for an 8-hour shift of a job experiencing vibration (broadband vibration of 1-8Hz, r.m.s. = 0.55 m/s^2 in each of the x, y, and z axes).
- Perturbations – While under the vibrating or non-vibrating condition and seated, you will be asked to place your hands rested on your lap, with elbows bent at 90 degrees. The researcher will signal to you that within a 15 sec time-frame, the robot will suddenly jolt you either in the frontwards, backwards, or either sideways directions. These will be short, fast jolts (moving 60mm). You will be perturbed into each of the four directions, ten times. Altogether, there will be 40 perturbations. For half of the perturbations you will be told which direction you will be going in, whereas for the remaining perturbations you will not be told the direction. All perturbations will be done in a random order.

POTENTIAL RISKS AND DISCOMFORTS

Minimal risks are anticipated – the following are possible consequences associated with this experiment:

Muscle fatigue/soreness – as with any physical activity, there is a risk of developing some muscle fatigue or soreness. Some postures that will be required for the study may cause some discomfort. You may experience some muscle soreness/discomfort during the perturbations (jolts). Any muscle soreness that may occur will usually go away within a few days after testing. After collecting the data, you will be shown some trunk and back muscle stretches in order to reduce discomfort.

Muscle and joint injury – with any exercise there is always a risk of muscle or joint injury; however, the exercises being done in this study are often used in exercise programs, and are similar to exertions you may experience at work or at home. Researchers will ensure that improper exercise/posture techniques that would put you at risk for injury are not used. Furthermore, you will not be required to maintain an exertion or posture for long periods of time (less than 30 sec).

Skin irritation – the electrodes and motion capture markers used are adhered directly to the skin. If irritation occurs, it is similar to that which may develop from the use of regular adhesive bandages and will disappear within 2-3 days after testing. You will be asked if you have had any previous reactions to adhesive bandages, tapes or rubbing alcohol – if so, you will be asked to withdraw from the study. If irritation develops during testing, the instrumentation will be removed and the skin will be cleansed with rubbing alcohol and water. The same cleansing process will be done after the testing.

POTENTIAL BENEFITS TO SUBJECTS AND/OR TO SOCIETY

By participating in the study you will have the opportunity to experience the collection procedures of both electromyography and kinematics (using the Vicon System) which may be useful in your course of study and/or future career. Also, you will increase your knowledge and understanding of experimental procedures commonly used in biomechanics research. Besides being exposed to some of the techniques and theory of human biomechanics and exercise research, there are no known benefits to participants.

The findings of the study could result in safer and more effective techniques and equipment standards for occupational workers that endure study conditions on a regular basis.

COMPENSATION FOR PARTICIPATION

You will receive a University of Windsor, Faculty of Human Kinetics research t-shirt for your participation in this study.

CONFIDENTIALITY

Any information that is obtained in connection with this study and that can be identified to you will remain confidential and will be disclosed only with your permission.

Due to the nature of the testing that will occur, participants will not be anonymous. However, the data collected from your participation will be treated as confidential. Also, your personal information will be anonymous on all documents using a coding system, which will be known only by the investigators. A file linking your name to a participant code will be stored separately from the data and will be password-protected. The password will be known only by the investigators. Data will be stored for a period of 3 years.

PARTICIPATION AND WITHDRAWAL

You choose whether or not to participate in this study. If you volunteer to be in this study, you may withdraw at any time without consequences of any kind. By volunteering for this study you are not waiving any of your legal rights. You may also refuse to answer any questions you don't want to answer and still remain in the study. The investigator may withdraw you from this research if circumstances arise which warrant doing so. If you are terminated from the study, all data collected from you will be destroyed or permanently deleted so as not to be used for any research purposes.

FEEDBACK OF THE RESULTS OF THIS STUDY TO THE SUBJECTS

Research findings will be posted online on the University of Windsor Research Ethics Board website (www.uwindsor.ca/reb) upon the completion on this study. This website is accessible to the public. Results are anticipated to be posted during the Fall of 2013.

Web address: www.uwindsor.ca/reb

Date when results are available: Fall 2013

SUBSEQUENT USE OF DATA

This data will not be used in subsequent studies.

RIGHTS OF RESEARCH SUBJECTS

You may withdraw your consent at any time and discontinue participation without penalty. If you have questions regarding your rights as a research subject, contact: Ms. Sarah Braganza, Research Ethics Coordinator, University of Windsor, Windsor, Ontario, N9B 3P4; Telephone: 519-253-3000, ext. 3948; e-mail: ethics@uwindsor.ca

SIGNATURE OF RESEARCH SUBJECT/LEGAL REPRESENTATIVE

I understand the information provided for the study **Neuromuscular response of the trunk following inertial-based perturbations with whole-body vibration exposure** as described herein. My questions have been answered to my satisfaction, and I agree to participate in this study. I have been given a copy of this form.

_____

Name of Subject

_____ _____

Signature of Subject Date

SIGNATURE OF INVESTIGATOR

These are the terms under which I will conduct research.

_____ _____

Signature of Investigator Date

APPENDIX C

Questionnaire for the Identification of Back Pain

1. a) Have you had a pain or ache in your back that interfered with your activities of daily living during the last 12 months?
   ☐ Yes ☐ No *(If no, go to question 6)*
   b) When you had that pain or ache did it go into your leg or foot?
   ☐ Yes ☐ No

2. a) Did you have that pain or ache in your back on most days of every month during the past year?
   ☐ Yes ☐ No *(If yes, go to question 3)*
   b) Have you had more than one separate attack of back pain or ache in the last 12 months?
   ☐ Yes ☐ No *(If no, go to question 3)*
   c) How many separate attacks of this back pain or ache have you had during the past 12 months?
   ☐ 2 or 3 attacks ☐ 4 or more attacks
   d) How long did the longest attack last for?
   ☐ less than a week ☐ 1 week or more

3. When your back pain or ache was at its worst during the past 12 months did you have difficulty carrying out any of the following activities?
   ☐ Walking ☐ Sitting ☐ Standing ☐ Lying ☐ Dressing yourself
   ☐ Stooping ☐ Climbing stairs ☐ Getting out of a chair

4. Have you ever been absent from work because of back pain during the past 12 months?
   ☐ Yes ☐ No

5. a) Have you seen your own doctor about your back pain during the past 12 months?
   ☐ Yes ☐ No
   b) Have you seen anybody else for treatment of your back pain during the past 12 months?
   ☐ Yes ☐ No

6. Have you had a pain or ache in your back previously, before the past 12 months?
   ☐ Yes ☐ No

Modified from: Agius et al., 2004. Questionnaire for the identification of back pain for epidemiological purposes. ***J.Occup.Env.Med.51****(11):756-760.*

APPENDIX D

Danielle MacIntyre Thesis Data Collection Sheets

Participant ID: Date:

Experimental Group: VG or CG

Subject Name : _____ Age: _____

Subject Height: _____ Subject Weight: _____

Consent Form & Questionnaire*
MVC Back and Abs (6 positions)*
Bias Trial
Markers and T-pose
10 min. vibration/quiet sitting

Pert #				Trial #	Comments:
1	KD	Front	1		
2	KD	Right	1		
3	KD	Back	2		
4	KD	Left	3		
5	KD	Left	4		
6	UD	Right	1		
7	KD	Back	1		
8	KD	Front	5		
9	KD	Back	5		
10	KD	Front	2		
11	UD	Right	3		
12	UD	Front	1		
13	KD	Back	4		
14	KD	Right	5		
15	UD	Back	1		
16	KD	Right	4		
17	UD	Front	2		
18	KD	Back	3		
19	UD	Back	2		
20	KD	Front	4		
21	UD	Front	5		
22	UD	Left	5		
23	UD	Right	4		
24	KD	Left	5		
25	UD	Front	3		
26	KD	Right	2		
27	UD	Back	5		
28	UD	Right	2		
29	KD	Left	1		
30	UD	Left	4		
31	UD	Front	4		
32	KD	Right	3		
33	UD	Left	3		
34	KD	Front	3		
35	UD	Back	3		
36	UD	Left	1		
37	UD	Left	2		
38	UD	Right	5		
39	UD	Back	4		
40	KD	Left	2		

APPENDIX E

Passive Marker Locations

Marker #	Abbr.	Location
1	RAH	Right Anterior Head LAST
2	LAH	Left Anterior Head
3	RPH	Right Posterior Head
4	LPH	Left Posterior Head
5	RAC	Right Acromion
6	RLS	Right Lateral Shoulder
7	RARM1	Right Distal Bicep
8	RARM2	Right Mid-Deltoid
9	RARM3	Right Tricep
10	RLELB	Right Lateral Elbow
11	RMELB	Right Medial Elbow
12	RFA	Right Forearm
13	RRAD	Right Radial Styloid Process
14	RULN	Right Ulnar Styloid Process
15	RHA	Right Hand
16	LAC	Left Acromion
17	LLS	Left Lateral Shoulder
18	LARM1	Left Distal Bicep
19	LARM2	Left Mid-Deltoid
20	LARM3	Left Tricep
21	LLELB	Left Lateral Elbow
22	LMELB	Left Medial Elbow
23	LFA	Left Forearm
24	LRAD	Left Radial Styloid Process
25	LULN	Left Ulnar Styloid Process
26	LHA	Left Hand
27	STRN	Sternum
28	C7	C7 Spinous Process
29	UBAK1	Left Ventral Scapular Border
30	UBAK2	T4 Spinous Process
31	T10	T10 Spinous Process
32	LBAK1	Left Lateral to T12 Spinous Process
33	LBAK2	Right Lateral to T12 Spinous Process
34	RIPS	Right PSIS
35	LIPS	Left PSIS
36	RICT	Right Iliac Crest Apex (Tubercle) CLAV

Marker #	Abbr.	Location
37	LICT	Left Iliac Crest Apex (Tubercle)
38	RFT	Right Femur Trochanter
39	RTH1	Right Proximal Ant Thigh
40	RTH2	Right Proximal Lat Thigh
41	RTH3	Right Distal Ant Thigh
42	RTH4	Right Distal Lat Thigh
43	RFLE	Right Femur Lateral Epicondyle
44	RFME	Right Femur Medial Epicondyle
45	LFT	Left Femur Trochanter
46	LTH1	Left Proximal Ant Thigh
47	LTH2	Left Proximal Lat Thigh
48	LTH3	Left Distal Ant Thigh
49	LTH4	Left Distal Lat Thigh
50	LFLE	Left Femur Lateral Epicondyle
51	LFME	Left femur Medial Epicondyle
52	RSK1	Right Proximal Fibula
53	RSK2	Right Ant Tibia
54	RSK3	Right Mid-Fibula
55	RSK4	Right Distal Ant Tibia
56	RLA	Right Lateral Malleolus
57	RMA	Right Medial Malleolus
58	RTOE	Right Ant Foot
59	RFT1	Right Lateral Foot
60	RFT2	Right Achilles
61	RHEEL	Right Heel
62	LSK1	Left Proximal Fibula
63	LSK2	Left Ant Tibia
64	LSK3	Left Mid-Fibula
65	LSK4	Left Distal Ant Tibia
66	LLA	Left Lateral Malleolus
67	LMA	Left Medial Malleolus
68	LTOE	Left Ant Foot
69	LFT1	Left Lateral Foot
70	LFT2	Left Achilles
71	LHEEL	Left Heel
72	CLAV	~~Sternal notch~~ on Sternum

VITA AUCTORIS

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