

**The Effects of Forward Head Posture on Sensorimotor Integration and Neck and
Shoulder Proprioception**

by

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The above committee determined that the thesis is acceptable in form and content and that a satisfactory knowledge of the field covered by the thesis was demonstrated by the candidate during an oral examination. A signed copy of the Certificate of Approval is available from the School of Graduate and Postdoctoral Studies.

ABSTRACT

Forward head posture (FHP) is commonly seen in today's society as a result of excess technology use. The central nervous system uses the position of the head with respect to the neck when interpreting the location of the upper limb. While experimental pain studies undoubtedly provide a link between pain and altered motor control, there is less evidence to support the potential progression to chronic pain as a consequence of altered motor control in the presence of FHP or fatigue. In this thesis, study 1 showed that individuals with FHP produced larger and more variable joint positioning errors when compared to individuals without. Study 2, used somatosensory evoked potentials to measure cortical activity related to sensorimotor integration following a motor learning task. This study was the first to report differential changes to SEP peak amplitudes in response to acute alterations in afferent input to the neck induced by FHP. With the growing use of technology in today's society, these preliminary findings support the need for further research into this potentially problematic phenomenon.

Keywords: forward head posture (FHP); sensorimotor integration (SMI); proprioception; somatosensory evoked potentials (SEPs); motor performance; technology use

AUTHOR'S DECLARATION

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Tracey A. Patrick

STATEMENT OF CONTRIBUTIONS

I hereby certify that I am the sole author of this thesis and that no part of this thesis has been published or submitted for publication. I have used standard referencing practices to acknowledge ideas, research techniques, or other materials that belong to others. Furthermore, I hereby certify that I am the sole source of the creative works and/or inventive knowledge described in this thesis.

DEDICATION

I dedicate this thesis to my Grandma. I love you more than you love me....

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First and foremost, I would like to thank my thesis supervisor Bernadette Murphy. The impact you have had on me over the past two years is far greater than a purely academic one. You gave me the freedom to explore and the opportunity to make mistakes – I will always be forever grateful for having you as a mentor throughout this entire experience.

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LIST OF ABBREVIATIONS AND SYMBOLS

APs	Action potentials
ANOVA	Analysis of variance
CEM	Cervical extensor muscle
CROM	Cervical range of motion
CNS	Central nervous system
DCL	Dorsal column lemniscus tract
DRG	Dorsal root ganglion
EEG	Electroencephalography
ERP	Event related potential
EMG	Electromyography
FHP	Forward head posture
FHT	Forward head translation
GTO's	Golgi tendon organs
IFCN	International Federation of Clinical Neurology
JPS	Joint position sense
JPSE	Joint position sense error
LMN	Lower motor neuron
M1	Primary motor cortex
μV	Microvolt
NHP	Neutral head posture
NDI	Neck disability index
NP	Neck pain
PNS	Peripheral nervous system
ROM	Range of motion
S1	Primary somatosensory cortex
SMA	Supplementary motor area

SCNP	Subclinical neck pain
SEPs	Somatosensory evoked potentials
TMS	Transcranial Magnetic Stimulation
VPL	Ventro-posterior lateral

CHAPTER 1

Introduction to Thesis

Forward head posture (FHP) is a postural disorder commonly seen in today's society as a result of excess technology use (Chiu et al. 2005, Neupane et al. 2017). FHP occurs when the head becomes positioned anterior in relation to the body's center of gravity (Neupane et al. 2017) and is known to contribute to predictable patterns of tissue overload and dysfunction (Abu El Kasem et al. 2017, Neupane et al. 2017, Pacheco et al. 2018). FHP has been shown to lead to the lengthening and weakness of the anterior cervical muscles with a corresponding shortening of the posterior cervical muscles (Lee et al. 2015, Neupane et al. 2017, Pacheco et al. 2018). These muscles, which attach directly to the cervical vertebra, play an important role in the alignment and position of the cervical spine during multi-directional movement (Chiu et al. 2005, Panjabi 1992), which in turn, provides stability and support to the head (Panjabi 1992).

In 2017, texting statistics significantly increased with the average number of monthly text messages sent in the USA being close to 781 billion (Brain 2013). Our everyday lives have become dominated by computer technology and handheld mobile technology, making FHP (or now socially know as text-neck), a growing, world-wide, health effect (Neupane et al. 2017). Approximately, one out of every 10 Canadian adults have reported to have repetitive strain injury serious enough to limit their normal daily activities (Tjepkema 2003). Past studies have indicated that neck pain impacts upper limb proprioception (De Vries et al. 2015) sensorimotor processing (Baarbé et al. 2018, Taylor and Murphy 2008, Treleaven 2008) and motor performance (Falla et al. 2004). Given that neck pain and dysfunction alter sensorimotor function, it is critical to understand if FHP also impacts sensorimotor processing.

The orientation of the head with respect to the neck plays an integral role in human movement and performance (Cohen 1961, Malmström et al. 2009) and is interconnected through neurological pathways in the brain and spinal cord which use neck position as a reference when processing multisensory input (Abrahams 1977). While studies investigating the effects of technology-induced postures on the musculoskeletal system are starting to emerge (Neupane et al. 2017), the long-term effect on sensorimotor processing and neuroplasticity remains unclear. The significance of investigating this phenomenon could enhance our understanding of how long-term sensory changes in sensory input from the neck contribute to altered sensorimotor integration and possible errors in motor performance.

The brain uses a series of excitatory and inhibitory activation patterns (feedback loops) to improve the accuracy and control of movements, and will repeat this process until otherwise instructed (Lephart et al. 1997, Riemann et al. 2002). Collectively, these pathways and structures work to influence and create a postural model known as body schema, which is defined as, the presence and awareness of one's physical occupancy in relation to their environment (Harris et al. 2015). This neural map of the body is used to generate accurate and coordinated voluntary movement based on previous experience and previously stored synchronization of muscle recruitment patterns (Doyon et al. 2003). Motor control refers to the planning and execution of movements; whereas, motor learning refers to the increasing spatial and temporal accuracy of movements with practice.

The peripheral and central nervous systems (CNS) are constantly relaying information to one another, which creates a fundamental feedback loop; whereby sensory, or afferent input, is integrated by the CNS and used for assisting in the execution of the appropriate motor output (Riemann and Lephart 2002). The ability of the CNS to perform this integration relies on its reorganization; plastic changes in the way that the CNS filters information in response to afferent input in a process termed sensorimotor integration (SMI) (Kandel et al. 2000, Riemann and Lephart 2002). These CNS alterations have been shown to persist following the period of afferent input to induce organizational changes in synaptic connectivity and strength in both the primate and human cortices (Byl et al. 1997, Classen et al. 1998, Haavik and Murphy 2011, Haavik and Murphy 2012, Haavik and Murphy 2013, Murphy et al. 2003). Input in the form of behavioural training has been shown to induce these organizational changes and the retention of such alterations reflects the reinforcement of sensorimotor skill acquisition or motor skill learning (Adams et al. 1975, Boudreau et al. 2010). The internal representation of our body map, also known as body schema, allows us to perform activities of daily life by perceiving the location of our limbs in 3D space. Human movement is guided by mechanosensory neurons located within muscles, tendons, and joints (Chapman and Goodale 2008).

Proprioceptive acuity involves both peripheral and central processing and is the product of sensory information supplied by specialized nerve endings called mechanoreceptors (Shin et al. 2017). These receptors are located in muscle, joint capsules, tendons, ligaments, and skin and are specific to providing input on joint position and velocity throughout its entire range of motion (ROM) (Grigg 1994, Imamizu et al. 2000, Kandel et al. 2000). During movement, proprioception is important for feedback (reactive control), feedforward (preparatory control) and the regulation of muscle stiffness in order to achieve specific postures required for efficient muscle balance, coordination and joint stability (Abrahams 1977, Hillier et al. 2015). An underactive proprioceptive system has been shown to lead to recruitment failure of important joint stabilizers, which results in a variety of changes including poor endpoint accuracy during movement (Zabihhosseinian et

al. 2017, Zabihhosseinian et al. 2015, Zabihhosseinian et al. 2019), reduced control during multi-segmental movements, (Sainburg et al. 1995, Sainburg et al. 1993) and an inability to perform repetitive movement sequences (Rothwell et al. 1982). If left untreated, over time, and under load, these deficits have the ability to increase an individual's chance of developing joint and muscle pain and/or injury (De Vries et al. 2015). Sensory afferents, delivered through vision and peripheral receptors, are imperative for limb orientation and coordination during movement of the arm (De Vries et al. 2015, Zabihhosseinian et al. 2015, Zabihhosseinian et al. 2019) Together these systems work synergistically with the CNS to match visual and kinesthetic input with stored motor patterns as a way to predict future limb positions and movements (Proske and Gandevia 2009).

Although considerable progress has been made in understanding the neural basis of motor control (Georgopoulos et al. 1981), little is known about the impact of chronic postural changes in sensory input from the neck and its impact on upper limb motor skill learning. Deficits in motor control of the spine can lead to poor control of joint movement, leading to the presentation of repeated microtrauma which can eventually to pain (Kapandji 1971, Panjabi 1992, Strimpakos 2011). For example, increased neural activity to the muscles of the neck muscles (upper trapezius and the levator scapulae muscles, sternocleidomastoid), due to a poor working posture of the neck or of the arms, may over time increase the compressive load on cervical segments and initiate a painful neck condition. Adopting a sustained, non-neutral spinal posture has been shown to lead to the increased activation of important head and neck stabilizers making these muscles susceptible to the development of fatigue (Rothwell et al. 1982). It has been hypothesized that changes commonly seen in muscle recruitment patterns are developed as a protective mechanism to underlying spinal instability resulting from laxity or damage to the structural tissue of the head and neck, muscle dysfunction, or reduced neuromuscular control (Kapandji 1971, Panjabi 1992). There is also evidence in the literature to suggest that muscle impairment occurs early in the history of onset spinal complaints,(De Vries et al. 2015) and that such muscle impairment does not automatically resolve even when pain symptoms improve (Daligadu et al. 2013, De Vries et al. 2015, Falla and Farina 2008)

Previous studies have demonstrated that changes in neck position and the presence of neck fatigue, in the absence of visual cues, affect the processing of incoming sensory inputs and can alter the awareness of elbow JPS (Knox and Hodges 2005, Zabihhosseinian et al. 2015). More specifically, Knox & Hodges (2005) demonstrated that spatial position and orientation of upper limb segments were dependent on the position of the head and neck. Furthermore, fatiguing the cervical extensor muscles (CEM) has been shown to impact accuracy of elbow joint position matching in healthy participants (Zabihhosseinian et al. 2015), indicating that altered afferent input from the neck subsequent to fatigue may impair upper limb proprioception. Since FHP has become ubiquitous in our

technology focused society (Neupane et al. 2017) and given the aforementioned impact of altered sensory impact from the neck on upper limb proprioception, it is important to understand if FHP also has the potential to impact both neck and upper limb proprioception.

OBJECTIVES AND HYPOTHESES

Research Objectives

The aims of this thesis were to determine whether FHP impacts:

1. Neck proprioception accuracy as compared to a Non_FHP control group.
2. Glenohumeral (shoulder) joint proprioception accuracy as compared to a Non_FHP control group.
3. Motor tracing performance (accuracy) as compared to a Non_FHP control group.
4. Cortical activity related to somatosensory integration following performance of a novel motor tracing task, when compared to a Non_FHP control group.

Research Hypothesis

1. Individuals with FHP will demonstrate worse cervical JPS accuracy when compared to a Non_FHP control
2. Individuals with FHP will demonstrate worse glenohumeral joint position sense accuracy, when compared to a Non_FHP control group.
3. Following motor skill acquisition, individuals with FHP will demonstrate worse motor performance and differences in SEP peaks related to SMI as compared to a Non_FHP control group.

OVERVIEW

This thesis is divided into the following chapters:

1. Literature Review
2. Proposed Research
3. Manuscript 1
4. Manuscript 2
5. Thesis Summary
6. Appendices (including data tables, participant consent form, and questionnaires)

Chapter 1. LITERATURE REVIEW

1.1 INTRODUCTION TO LITERATURE REVIEW

In 2000/2001, one in twenty Canadians (2.3 million people) 20 years of age or older reported having severe enough symptoms of repetitive strain injury to limit their usual activities, and the majority of these injuries (83%) affected the upper extremity (neck, shoulder, elbow, forearm, wrist and hand) (McLean 2005). In addition, Chiu et al. (2002) found that approximately 60% of individuals with neck pain had FHP. Neck and shoulder pain are a common workplace complaint worldwide, with estimates of incidence ranging from 20% to 50% over a twelve-month period (Wilson 2002). More recently, texting statistics significantly increased with the average number of monthly text messages sent in the USA being close to 781 billion (Brain, 2013). Our everyday lives have become dominated by computer technology and handheld mobile technology, making FHP (or now socially know as text-neck), a growing, world-wide, health effect (Neupane, Ali et al. 2017). Approximately, one out of every 10 Canadian adults have reported to have repetitive strain injury serious enough to limit their normal daily activities (Tjepkema 2003). Past studies have indicated that neck pain impacts upper limb proprioception (De Vries et al. 2015) sensorimotor processing (Taylor and Murphy 2008, Treleaven 2008, Baarbé, et al. 2018) and motor performance (Falla, et al. 2004). Given that neck pain and dysfunction alter sensorimotor function, it is critical to understand if FHP also impacts sensorimotor processing.

The orientation of the head with respect to the neck plays an integral role in human movement and performance (Cohen 1961, Malmström, et al. 2009) and is interconnected through neurological pathways in the brain and spinal cord which use neck position as a reference when processing multisensory input (Abrahams 1977). While studies investigating the effects of technology-induced postures on the musculoskeletal system are starting to emerge (Neupane, et al. 2017), the long-term effect on sensorimotor processing and neuroplasticity remains unclear. What is currently available is starting to identify the significance of investigating this phenomenon as further investigation could enhance our understanding of how long-term sensory changes in sensory input from the neck contribute to altered sensorimotor integration and possible errors in motor performance.

The brain uses a series of excitatory and inhibitory activation patterns (feedback loops) to improve the accuracy and control of movements, and will repeat this process until otherwise instructed (Lephart et al. 1997, Riemann et al. 2002). Collectively, these pathways and structures work to influence and create a postural model known as body schema, which is defined as, the presence and awareness of one's physical occupancy in relation to their environment (Harris,

Carnevale et al. 2015). This neural map of the body is used to generate accurate and coordinated voluntary movement based on previous experience and previously stored synchronization of muscle recruitment patterns (Doyon et al. 2003). Motor control refers to the planning and execution of movements; whereas, motor learning refers to the increasing spatial and temporal accuracy of movements with practice.

The periphery and central nervous systems (CNS) are constantly relaying information to one another, which creates a fundamental feedback loop; whereby sensory, or afferent input, is integrated by the CNS and used for assisting in the execution of the appropriate motor output (Riemann and Lephart 2002). The ability of the CNS to perform this integration relies on its reorganization; plastic changes in the way that the CNS filters information in response to afferent input in a process termed sensorimotor integration (SMI) (Kandel, Schwartz et al. 2000, Riemann and Lephart 2002). These CNS alterations have been shown to persist following the period of afferent input to induce organizational changes in synaptic connectivity and strength in both the primate and human cortices (Byl, Merzenich et al. 1997, Classen, Liepert et al. 1998, Murphy, Taylor et al. 2003, Haavik and Murphy 2011, Haavik and Murphy 2012, Haavik and Murphy 2013). Input in the form of behavioural training has been shown to induce these organizational changes and the retention of such alterations reflects the reinforcement of sensorimotor skill acquisition or motor skill learning (Adams, Gopher et al. 1975, Boudreau, Farina et al. 2010). The internal representation of our body map, also known as body schema, allows us to perform activities of daily life by perceiving the location of our limbs in 3D space during static and dynamic movements (Chapman and Goodale 2008). An accurate body schema is an essential contributor to the execution of accurate and efficient limb movements; which is highly dependent on the awareness of limb location during the initial phases of movement (Chapman and Goodale 2008).

Proprioceptive acuity involves both peripheral and central processing and is the product of sensory information supplied by specialized nerve endings called mechanoreceptors (Shin, Kim et al. 2017). These receptors are located in muscle, joint capsules, tendons, ligaments, and skin and are specific to providing input on joint position and velocity throughout its entire range of motion (ROM) (Grigg 1994, Imamizu, Miyauchi et al. 2000, Kandel, Schwartz et al. 2000). During movement, proprioception is important for feedback (reactive control), feedforward (preparatory control) and the regulation of muscle stiffness in order to achieve specific postures required for efficient muscle balance, coordination and joint stability (Abrahams 1977, Hillier, Immink et al. 2015). An underactive proprioceptive system has been shown to lead to recruitment failure of important joint stabilizers, which results in a variety of changes including poor endpoint accuracy during movement (Zabihhosseinian, Holmes et al. 2015, Zabihhosseinian, Holmes et al. 2017, Zabihhosseinian, Yelder et al. 2019),

reduced control during multi-segmental movements, (Sainburg, Poizner et al. 1993, Sainburg, Ghilardi et al. 1995) and an inability to perform repetitive movement sequences (Rothwell, Traub et al. 1982). If left untreated, over time, and under load, these deficits have the ability to increase an individual's chance of developing joint and muscle pain and/or injury (De Vries, Ischebeck et al. 2015). Sensory afferents, delivered through vision and peripheral receptors, are imperative for limb orientation and coordination during movement of the arm (De Vries, Ischebeck et al. 2015, Zabihhosseinian, Holmes et al. 2015, Zabihhosseinian, Yelder et al. 2019) Together these systems work synergistically with the CNS to match visual and kinesthetic input with stored motor patterns as a way to predict future limb positions and movements (Proske and Gandevia 2009).

Although considerable progress has been made in understanding the neural basis of motor control (Georgopoulos, Kalaska et al. 1981), little is known about the impact of chronic postural changes in sensory input from the neck and its impact on upper limb motor skill learning. Deficits in motor control of the spine can lead to poor control of joint movement, leading to the presentation of repeated microtrauma which can eventually to pain (Kapandji 1971, Panjabi 1992, Strimpakos 2011). For example, increased neural activity to the muscles of the neck muscles (upper trapezius and the levator scapulae muscles, sternocleidomastoid), due to a poor working posture of the neck or of the arms, may over time increase the compressive load on cervical segments and initiate a painful neck condition. Adopting a sustained, non-neutral spinal posture has been shown to lead to the increased activation of important head and neck stabilizers making these muscles susceptible to the development of fatigue (Rothwell, Traub et al. 1982). It has been hypothesized that changes commonly seen in muscle recruitment patterns are developed as a protective mechanism to underlying spinal instability resulting from laxity or damage to the structural tissue of the head and neck, muscle dysfunction, or reduced neuromuscular control (Kapandji 1971, Panjabi 1992). There is also evidence in the literature to suggest that muscle impairment occurs early in the history of onset spinal complaints, (De Vries, Ischebeck et al. 2015) and that such muscle impairment does not automatically resolve even when pain symptoms improve (Falla and Farina 2008, Daligadu, Haavik et al. 2013, De Vries, Ischebeck et al. 2015)

Previous studies have demonstrated that in the absence of visual cues, changes in the positioning of the neck, in the presence of neck fatigue, altered the awareness of elbow JPS (Knox and Hodges 2005, Zabihhosseinian, Holmes et al. 2015). More specifically, (Knox & Hodges, 2005), demonstrated that spatial position and orientation of upper limb segments were dependent on the position of the head and neck. Furthermore, fatiguing the cervical extensor muscles (CEM) has been shown to impact accuracy of elbow joint position matching in healthy participants (Zabihhosseinian, Holmes et al. 2015), indicating that altered

afferent input from the neck subsequent to fatigue may impair upper limb proprioception. Since FHP has become ubiquitous in our technology focused society (Neupane, Ali et al. 2017) and given the aforementioned impact of altered sensory impact from the neck on upper limb proprioception, it is important to understand if FHP also has the potential to impact both neck and shoulder proprioception.

1.2 THE SOMATOSENSORY SYSTEM: AN OVERVIEW

The somatosensory system is a complex grouping of sensory neurons and pathways that respond to changes with respect to the body as it interacts with its environment (Classen et al. 1998, Ferrell and Craske 1992, Kandel et al. 2000). Somatosensation refers to the detection, discrimination, and recognition of the body with respect to its environment (Classen et al. 1998, Ferrell and Craske 1992, Kandel et al. 2000). The brain recognizes the incoming sensory information and responds by sending out a response that is specific to the imposed demand (Classen et al. 1998, Ferrell and Craske 1992, Kandel et al. 2000). Therefore, the somatosensory system allows an individual to interact with their environment and allows an individual to perceive its position during human movement (Classen et al. 1998, Ferrell and Craske 1992, Kandel et al. 2000).

The brain acts as the central control station for all biological processes and human movement patterns by encoding and integrating sensory information from the skin, eyes, ears and joints, which allow an individual to respond to their environment (Classen et al. 1998, Ferrell and Craske 1992, Kandel et al. 2000). This information is transmitted in a systematic, controlled and highly structured pathway system for further processing (Classen et al. 1998, Ferrell and Craske 1992, Kandel et al. 2000). For the purpose of this thesis the area of focus will remain within the peripheral nervous system and sensorimotor system; however, it is worth noting that human movement and performance would not be as effective without the contribution of visual and auditory stimuli.

1.2.1 Neuroanatomy of somatosensation

Sensory receptors are tiny structures located throughout the body that convert stimuli into action potentials (APs) (Classen et al. 1998, Ferrell and Craske 1992, Kandel et al. 2000). These nerve impulses, which are classified according to their structure and function, are located at the distal ends of peripheral neurons (Classen et al. 1998, Ferrell and Craske 1992, Kandel et al. 2000) and allow skeletal muscle to communicate with higher levels of the CNS (Classen et al. 1998, Ferrell and Craske 1992, Kandel et al. 2000). Sensory receptors have their cell bodies located outside the spinal cord, within the dorsal root ganglia, and are

highly specialized (Classen et al. 1998, Ferrell and Craske 1992, Kandel et al. 2000). Peripheral sensory axons, also called, afferents, are classified according to their axon diameter (Classen et al. 1998, Ferrell and Craske 1992, Kandel et al. 2000).

The somatosensory system sub-serves three main functions which include: exteroception (perception of the external environment), interoception (perception of the internal environment) and proprioception (the perception of the body/peripheral extremities in relation to 3D space) (Kandel et al. 2000). These three main functions encompass sensations such as touch, pressure, motion, vibration, and pain (Classen et al. 1998, Kandel et al. 2000). Pacinian Corpuscles, are specialized touch receptors located in the skin and detect vibration and deep pressure (Kandel et al. 2000). Proprioceptors are found in skeletal muscle, tendons, ligaments and joint capsules and detect muscle length (stretch), muscle tension and joint position during static and dynamic movement on a conscious (walking and talking) or unconscious level (seated posture) (Kandel et al. 2000). Proprioceptors are innervated by dorsal root ganglion (DRG) neurons, which are classified as large diameter, heavily myelinated axons; meaning they conduct action potentials rapidly (Kandel et al. 2000). Muscle spindles, are specialized stretch receptors that detect change in muscle length and found in most skeletal muscle (Kandel et al. 2000). They appear in large numbers in postural muscles and intrinsic muscles of the hand (Kandel et al. 2000). When communicating to the CNS muscle spindles detect two characteristics of muscle stretch: 1) determines whether the length of the muscle is changing, and 2) how fast is the rate change (or how quickly is the muscle moving) (Kandel et al. 2000).

Muscle spindles are innervated by specialized motor neurons called gamma motor neurons, which synapse onto the spindles of muscle fibers (Kandel et al. 2000). Stimulation of these fibers pull, or lengthens, the central part of the spindle fiber and makes the location stiff; creating a change in muscle tension (Kandel et al. 2000). When spindles are stiff they become more sensitive to stretch; therefore, gamma motor neurons control how sensitive the spindles are to muscle stretch (Kandel et al. 2000). The sensitivity causes a change in the number of action potentials being sent to the brain and thus this information is used to determine the angle of the joint as well as provide information on the speed and direction of movement (Kandel et al. 2000). Golgi Tendon Organs (GTO's) are found in the tendons of skeletal muscle, located near the junction between the muscle and its tendon (Kandel et al. 2000). The primary function of GTO's is to detect muscle tendon tension, meaning, when a muscle contracts, the change in muscle length creates tension changes in the muscle tendon (Kandel et al. 2000). The change in tension changes the rate at which action potentials are sent to the CNS (Kandel et al. 2000). Taken together, this group of specialized receptors respond to the mechanical stress placed on the joint

capsule and ligaments and send important information to the brain, via the spinal cord, about joint position and limb movement (Kandel et al. 2000). Therefore, it is plausible to assume that changes in spinal posture, over an extended period of time, has the ability to alter the stimulation rate of these postural receptors, subsequently thus, altering the way the brain interprets the body's location and position, subsequently effecting the way it integrates the sensory information (Kandel et al. 2000).

Transmission of sensory information requires a complete somatosensory pathway in order to keep the ascending information organized (Kandel et al. 2000). A somatosensory pathway consists of an afferent (sensory) neuron, located in a peripheral nerve, that carries the action to the central nervous system (CNS) and a neuron that transmits the AP up through the spinal cord to the brain for further integration (Kandel et al. 2000). When the sensation is perceived consciously, an additional neuron is included in the pathway and is responsible for conveying the sensation to the primary somatosensory cortex (Kandel et al. 2000). For the purposes of this review, the Dorsal-Column Leminiscal (DCL) pathway will be the sensory tract of focus, as this pathway conveys muscle spinal afferent information. Short latency somatosensory evoked potentials (SEPs) can be used to assess processing in this pathway, as the 1A information from muscle spindles is responsible for generating short latency SEPs (Cruccu et al. 2008).

1.2.2 Dorsal Column Leminiscal (sensory) Pathway

The role of sensation in movement is complex. The DCL pathway conveys both mechanoreception and proprioception (Cruccu et al. 2008, Kandel et al. 2000) to help coordinate and control both voluntary (walking) and involuntary (maintenance of posture) muscle contraction and to help regulate muscle tension (Cruccu et al., 2008; Kandel et al., 2000). The pathway of the DCL tract begins with peripheral receptor stimulation of a first-order neuron in the dorsal root ganglia (Cruccu et al., 2008; Kandel et al., 2000). This afferent volley is sent via the ipsilateral posterior column in the spinal cord, where central branches either terminate within the spinal grey matter or ascend via the dorsal column the spinal cord to synapse on second-order neurons in the thalamus. (Cruccu et al., 2008; Kandel et al., 2000). Third order neurons arise in the thalamus and project to the parietal cortex where the information is integrated into appropriate motor responses (Cruccu et al., 2008; Kandel et al., 2000). Local branches can activate local reflex circuits while the ascending branches carry information into the brain, where this information becomes raw material for the perception of touch, position sense, pain and temperature (Cruccu et al., 2008; Kandel et al., 2000). This orderly somatotopic distribution is maintained throughout the entire ascending somatosensory pathway (Cruccu et al., 2008; Kandel et al., 2000).

Sensory fibers from the lower body are located medially; fibers from the trunk, arm, shoulder and neck occupy progressively more lateral areas (Kandel et al. 2000). At the cervical level, the axons which are forming the dorsal column are divided into 2 bundles: a medially situated gracile (slender) fascicle and a more laterally situated cuneate (wedge shaped) fascicle (Kandel et al., 2000). Primary afferent fibers from the lower extremities and trunk pass in the medial, gracile fasciculus, and primary afferents from the upper extremities and associated trunk and neck travel in the lateral, cuneate fasciculus (Kandel et al., 2000). These axons in the two bundles ascend to the medulla where they terminate in the gracile and cuneate nuclei, respectively (Kandel et al., 2000). These nuclei give rise to axons which decussate and ascend in the medial lemniscus and terminate in the thalamus (Kandel et al., 2000). The thalamus is an egg-shaped structure which allows for the projection information between various cortical and subcortical structures. The cells in the ventro-posterior lateral (VPL) thalamus process somatosensory information and their axons project to the primary somatosensory cortex (S1). Other portions of the thalamus participate in motor functions, transmitting information from the cerebellum and basal ganglia to the motor regions of the frontal lobe (Kandel et al., 2000).

Conscious awareness of sensation is integrated in the primary somatosensory cortex (S1) which is located on the post central gyrus of the parietal lobe (Kandel et al., 2000). This area of the cortex is strategically organized based on body region to form a sensory map known as the somatosensory homunculus (Kandel et al., 2000); which is also found in the motor cortex with the same organizational structure (Kandel et al., 2000). The largest areas of this map are developed to body parts that have a high density of sensory receptors with the highest level of sensitivity (fine motor skills (Kandel et al., 2000). This arrangement allows us to localize sensations coming from these regions with great precision and accuracy, that is, the more area that is represented in this region, the greater precision that limb has with respect to movement abilities (Kandel et al., 2000). The S1 is divided into various regions known as Brodmann's areas (Crucchi et al. 2008). Brodmann's classification refers to sections of the cerebral cortex that are classified based on the structure and organization of cells (Crucchi et al. 2008). Brodmann areas 1, 2, and 3 represent the S1, and area 4 represents the primary motor cortex (MI) (Crucchi et al. 2008). However, only Brodmann area 3 should be referred to as S1 as it receives the majority of the thalamocortical projections from incoming sensory feedback (Viaene et al. 2011).

1.3 SENSORIMOTOR INTEGRATION (SMI)

In everyday life, we go about activities, sub-consciously, using a variety of motor skills that have been acquired gradually through practice and repetitive interactions with our environment (walking, driving, speaking, writing) (Kandel et

al., 2000). Every action we perform requires the motor system and the coordinated actions of peripheral, spinal, cerebellar and cerebral regions which is further refined by our intentions and goals (Kandel et al. 2000, Strimpakos 2011). Sensorimotor integration (SMI) is a complex process whereby sensory input from the CNS in response to a change in the body's environment (Kandel et al. 2000, Strimpakos 2011) is processed by the CNS and then used to modify outgoing motor commands. Both sensory afferents and the corresponding motor outputs are processed by a variety of distinct pathways which are active simultaneously and used to create an internal representation of the external environment (Kandel et al. 2000, Strimpakos 2011). The mechanism of SMI involves the incorporation of proprioceptive, kinesthetic and somatic sensation which results in the selection of a suitable motor output for a given environmental demand (Kandel et al. 2000, Strimpakos 2011). The ability to integrate this information into an appropriate motor response makes the sensory motor system essential during movement control (Kandel et al. 2000, Strimpakos 2011).

The motor components of the sensorimotor system consist of a central axis and two associate areas which contribute to stability and control of the limbs and torso during movement (Strimpakos 2011). The central axis corresponds to the three levels of motor control: spinal cord, brain stem, and cerebral cortex (Matthews, 1997). Whereas, the two associate areas, the cerebellum and basal ganglia, are responsible for modulating and regulating the motor commands (Ghez et al. 1991). Accurate function of both systems and integration of the sensorimotor information are required to perform precise and purposeful movements; therefore, optimal functioning of these pathways is necessary for smooth and accurate limb movements (Kandel et al. 2000, Strimpakos 2011).

The components giving rise to functional joint stability must be flexible and adaptable and the process of maintaining functional joint stability is accomplished through a complementary relationship between static and dynamic components (Strimpakos 2011). Ligaments, joint capsule, cartilage, friction, and the bony geometry within the articulation within the spine comprise the static, passive, components (discussed further in subsection 1.4.1 of this review) (Johansson and Magnusson 1991, Lew et al. 1993, Panjabi 1992). Dynamic contributions arise from feedforward and feedback (neuromotor) control over the skeletal muscles crossing the joint (Johansson and Magnusson 1991, Lew et al. 1993, Panjabi 1992). Underlying the effectiveness of the dynamic restraints are the biomechanical and physical characteristics of the joint, which include, the range of motion (ROM), muscle strength and muscle endurance of the joint, in its entirety (Kandel et al. 2000, Lew et al. 1993, Panjabi 1992). The performance of fine and gross motor skills relies on the synergistic relationship between the (CNS) and peripheral nervous system (PNS) (Kandel et al. 2000, Lew et al. 1993, Panjabi 1992). The integration of sensory input is performed by neural networks, therefore, when individuals are presented with a change in external dynamics,

adaptation is gradually experienced (Lew et al. 1993, Panjabi 1992). The fundamental feedback system of SMI develops an internal representation of the external world and is used to create a generalizable “blue-print” to act a starting point for novel movements (Kandel et al. 2000, Lew et al. 1993, Panjabi 1992).

The primary motor (M1) cortex is one of the main areas involved in motor function. M1 is located in the frontal lobe of the brain, along the precentral gyrus (Kandel et al, 2000). The role of M1 during SMI is to generate neural impulses that control the execution of movement (Kandel et al, 2000). Signals from M1 cross the body’s midline to activate skeletal muscles on the opposite side of the body, meaning that the left hemisphere of the brain controls the right side of the body, and the right hemisphere controls the left side of the body (Kandel et al, 2000). Every part of the body is represented in the M1, and these representations are arranged somatotopically (Kandel et al., 2000). M1 receives somatosensory information via the thalamus and indirectly from various somatosensory cortical areas. Whereas the primary somatosensory cortex (S1) forms part of the descending pyramidal tract which stimulate lower motor neurons (LMN) and cause muscle contraction (Kandel et al. 2000, Strimpakos 2011). M1 is organized as motor maps (much like S1) consisting of somatotopically arranged representations of muscle synergies which are represented by the weight of neural connections (termed the motor homunculus) (Kandel et al. 2000, Keller and Asanuma 1993). The cortical areas in which the movement representations are imbedded have strong interconnections (Keller and Asanuma 1993), that are highly dynamic and capable of rapid organization (Hess and Donoghue 1994). The specific modifications involved in spatial and temporal organizations of muscle synergies are responsible for the smooth and accurate movement sequence during task performance (Hammond 2002). Neural representation of a given task sequence strengthens within M1 increases as movements are learned; therefore, the expansion of cortical motor map encoding for a specific movement or skill is enhanced, neurologically and physiologically, with practice (Schieber and Santello 2004).

1.3.1 Cerebellar functions and Cortical interactions in SMI

The cerebellum and basal ganglia relay loops cannot independently initiate motor activity, however, they are essential for the execution of coordinated motor control (Kandel et al., 2000). The cerebellum coordinates movement and postural control by operating entirely at a subconscious level. The ability to plan and modify motor activities in real time can only be accomplished through the comparison of the intended movement with the outcome movement or the goal of the task (Bloedel and Bracha 1995, Doyon et al. 2003, Kandel et al. 2000). This is accomplished through the continuous inflow of information from the motor control areas of the brain which is integrated and adjusted by the CNS and peripheral

sensory areas located throughout the body (Bloedel and Bracha 1995, Doyon et al. 2003). The outer layer of the cerebellum is grey matter, which consists of three cortical layers. The outer and inner layers contain interneurons which contribute to motor coordination via reciprocal and recurrent inhibition, and the middle layer contains Purkinje cell bodies which inhibit or reduce cerebellar nuclei activity (Kandel et al., 2000).

Two different sensory fibers convey information to the cerebellar cortex; mossy fibers which ascend from the spinal cord, and climbing fibers which originate from the medulla (Kandel et al., 2000). Both fibers influence the activity of the cerebellum through their unique pathways (Kandel et al., 2000). Mossy fibers convey somatosensory information such as equilibrium and balance and synapse with interneurons that convey information to Purkinje cells (Kandel et al., 2000). The climbing fibers relay information regarding movement errors during a task performance and synapse with Purkinje dendrites (Kandel et al., 2000). The integration of these fibers enables the cerebellum to evaluate movements that are occurring according to the motor plan already stored in memory, or, if movement patterns are off, the cerebellum is able to coordinate the corrections which are necessary to improve task performance. If the motor command needs to be adjusted, the cerebellum sends feedback to the motor areas of the motor cortex, to correct the motor output in order to make the movement smoother and more accurate (Peterburs and Desmond 2016). Postural control provides orientation and balance to body segments which is achieved by central commands to the lower motor neurons which stimulate skeletal muscle (Kendal et al., 2000). Sensory input is used in both feedback and feedforward mechanisms, which provides information about the state of the system and transmits anticipatory impulses which prepare the body for movement, respectively (Kendal et al., 2000). The position of the head, with respect to the neck, affects muscular activation and is signaled by neck proprioception, vestibular and visual systems in order to orient a horizontal reference point which the brain uses as a guiding system for subsequent movement (Kendal et al., 2000) Activity of the cervical joint receptors and neck muscle stretch receptors elicits neck reflexes and regulates control of the limbs with respect to the head via alterations in sensory information (Knox and Hodges 2005), suggesting that FHP, could have the ability to alter sensory input from the neck to the CNS, subsequently adjusting motor output being sent to the muscle.

Cerebellar lesions have been shown to impair the intracortical processing of somatosensory stimuli without affecting the arrival of the somatosensory volley to S1 (Restuccia et al. 2006) et al., 2007). The cerebellum's role is to detect mismatched sensorimotor information responsible for the modulation and coordination of human movement (Apps and Garwicz 2005, Doyon et al. 2002). The cerebellum provides constant feedback about the current state of the body during on-going movement to update and improve muscle activation patterns

(Apps and Garwicz 2005, Doyon et al. 2002). Andrew et al. (2018), recently demonstrated impairments in motor learning and differential changes in cortical SEP peaks related to SMI in individuals with recurrent neck pain. Changes in afferent information help to contribute to joint position sense, therefore, and the cerebellum plays a critical role in effective SMI by guiding and adjusting limb movements, as well creating an internal awareness of joint position sense (Feldman and Latash 1982, Feldman and Latash 2005).

Observations from previous studies have shown increases in early SEP peak amplitudes related to the cerebellum following motor-skill training (Andrew et al. 2018, Dancy et al. 2016). Individuals with cerebellar lesions demonstrate a decreased ability to control and coordinate voluntary movements in addition to alterations in joint reflexes (Bloedel and Bracha 1995, Bloedel and Bracha 1997, Marshall 1984, Thach et al. 1992). This provides evidence to support the dynamic role that the cerebellum plays in SMI. Furthermore, cerebellar impairment has been shown to minimize the stored after-effects of learning which is essential to skill retention (Tseng et al. 2007, Werner et al. 2009). The cerebellum contributes to the efficiency of neuromuscular control through its ability to provide real-time task specific adjustments to motor commands which help to update our movement patterns; therefore, increasing the likelihood of it being stored in long-term memory (Ghez et al. 1991, Ghez and Krakauer 1991, Johansson and Magnusson 1991).

Transcranial magnetic stimulation (TMS) is a non-invasive method which measures the excitability of the CNS through the cerebellum-M1 pathway. TMS, therefore, is an effective technique used to help map the activation and inhibition patterns of motor output relative to SMI (Barker et al. 1985, Peterchev et al. 2012). Magnetic stimulation over the cerebellum, when coupled with a conditioning test stimulus over the contralateral motor cortex, can be used to investigate changes in cerebellar outputs following skill acquisition (Doyon et al. 2002). A number of TMS studies have shown changes in MI excitability in response to motor learning of both skilled and non-skilled repetitive movements (Jull et al. 2007, Knox et al. 2006, Knox et al. 2006, Knox and Hodges 2005, Strimpakos et al. 2006). Doyon et al (2002), proposed that in order to learn a new skill, the cerebellum needs to disinhibit in order to learn and store new motor programs (Doyon et al. 2002). The process of disinhibition has been described as the changes in the neural activity of the descending motor pathway, which was shown to be altered instantly, following magnetic stimulation over the cerebellum (Ugawa et al. 1995). Recently, a TMS study which combined cerebellum and M1 stimulation demonstrated the functional connectivity and plasticity between the cerebellum and the M1 (Baarbé et al. 2013). The findings of this study were able to demonstrate the ability of the cerebellum to disinhibit in order to acquire a new motor skill (Baarbé et al. 2013). Given that muscle contraction is initiated in the motor cortex and descends to skeletal muscle, it is plausible to assume that

changes to sensory information throughout this pathway, in the presence of FHP, may produce results similar to movement performance similar to studies of muscular fatigue (Gandevia et al. 1996, LETAFATKAR et al. 2009, Taylor et al. 2006).

1.4 PROPRIOCEPTION AND BODY SCHEMA

From the perspective of joint stability, neuromuscular control refers to the unconscious activation of dynamic restraints occurring in preparation for, and in response to, joint movement (Burke 2007, Matthews 1988, Strimpakos 2011). Although neuromuscular control underlies all motor activities in some form, it is not easily separated from the neural commands controlling the overall motor program (Sherrington 1952, Strimpakos 2011). Similar to Panjabi's stability model (1992), Sherrington (1952) describes proprioception as the ability of the CNS to regulate the body's posture and position through the control of postural equilibrium and segmental (joint) stability. Although he considered vestibular information to be proprioceptive with respect to the head, Sherrington (1952) clearly outlined the functions and pathways of those receptors throughout the periphery and CNS. According to Matthews (1982), Sherrington described four sub modalities of proprioception (which he termed muscle sense): (1) posture, (2) passive movement, (3) active movement, and (4) resistance to movement. Therefore, proprioception correctly describes all afferent information arising from the periphery and areas of the body that contribute to postural control, joint stability. Without it, we lose our ability to accurately associate and coordinate our limb movements throughout as we navigate throughout our environment.

Piaget (1936) was the first Psychologist to develop a systematic theory of cognitive development with three fundamental modules: schemas, adaptation processes, and stages of development (Piaget 1936). Piaget defined body schema as an organized perception of one's past experiences and acquired knowledge, which is linked to a mental representation or "blue print" of the world as we experience it. This concept is used to describe the relationships formed between our experiences and how our body subsequently reacted, as a way to help recognize and respond to re-occurring situations more effectively in the future (Piaget 1936, Wadsworth 1996). Therefore, schemas act as an 'index card or a list of instructions' filed in the brain meant to outline quick and accessible details on how to react to received stimuli or information (Wadsworth, 1996).

In order to control the movement of the limbs through our environment, the brain is continually screening the position and movement of the body with regard to nearby objects (Gibson 1966, Popper and Eccles 1977, Popper and Eccles 2012). Vertical body orientation, corrective postural reactions, and anticipatory postural adjustments are all organized based on this internal representation

(Sharples et al. 2016). Head position is one of the main factors in the organization of sensory information for upper limb joint position sense (JPS) as the CNS uses the position of the head, relative to the neck, when interpreting the position of the arm in 3D space (Paulus and Brumagne 2008). JPS is the most commonly used measure of proprioception and is described as the conscious awareness of limb position during both passive and active movements and without vision (Riemann and Lephart 2002). Previous work by Knox and Hodges (2005) demonstrated that changes in head and neck position, in the absence of visual cues, has the ability to affect the processing of incoming sensory inputs, subsequently altering the awareness of elbow JPS. This postural body schema remains stable during gravitational changes, even when mechanoreceptive and vestibular inputs are significantly decreased (Sakihara et al. 2003). In order to pursue an intended goal, we need to have awareness, in the form of an integrated neural representation of all body parts, in order to assess the body's relative motion throughout a given movement (Popper & Eccles, 1977; 2012). Thus, body schema is considered a postural model of the body that modifies and continuously updates limb position during movement, in relation to the position of other body parts and relies heavily on the accurate awareness of prior movement performance (Head et al. 1920).

1.4.1 Joint Position Sense (JPS)

Spatial accuracy is described as the precision required during aiming movements for which the position of the movement's end point is important to the overall performance measure (Willingham, 1998). Temporal accuracy can be described as the type of accuracy needed during rapid movements in which accuracy of the movement time is important to the performance measure (Willingham, 1998). Joint position sense (JPS) is an important characteristic of proprioception and is defined as the ability of an individual to recognize the position of a joint in relation to space or other body segments (Selfe, Callaghan et al. 2006, De Vries, Ischebeck et al. 2015). Clinically, joint position sense error (JPSE) is associated with joint proprioception and is used to assess the ability of an individual to actively or passively perceive the position of a joint (Selfe, Callaghan et al. 2006, De Vries, Ischebeck et al. 2015). JPSE is measured by testing the ability of a blindfolded participant to accurately relocate the position of a joint relative to a predefined target angle (referred to as the neutral position of a joint) (Selfe, Callaghan et al. 2006). The difference between the presented and reproduced position is the repositioning error (Selfe, Callaghan et al. 2006, De Vries, Ischebeck et al. 2015). Current literature shows that JPSE is a relevant measure when it is used correctly. All studies which calculated the JPSE over at least six trials showed a significantly increased JPSE in the neck pain group. This strongly suggests that the number of repetitions is a major element in correctly performing the task. Based on a protocol that sets a threshold of variation in magnitudes of <5% of the SD for the cumulative mean for at least three straight trials, (Selfe et

al., 2006; Vries et al., 2015) it is suggested that the average of the fourth, fifth, and sixth trials should be used to accurately measure shoulder proprioception. (Selfe et al., 2006; Vries et al., 2015).

Examples of joints commonly used in the literature in which JPSE has been used for testing proprioception are: 1) the shoulder (Anderson and Wee 2011), 2) the knee (Knoop et al. 2013), and 3) the ankle (Nakasa et al. 2008). For the purpose of this study measurements for the JPSE of the neck and shoulder in individuals with and without FHP was assessed.

1.5 MOTOR LEARNING AND ACQUISITION

A motor skill is defined as the coordinated and sequential movement of skeletal muscles; whereas, motor skill learning is described as the intent to learn a new motor skill through repetitive practice (Willingham 1998). When humans perform goal-directed actions, they strive to achieve their goal quickly, accurately and with minimal effort (Willingham 1998). The idea is to complete the task without interfering with the performance of ongoing movements, (walking and talking), while maintaining sufficient attentional resources in order to respond appropriately to environmental demands (Kendal et al., 2000) Cortical changes occur dynamically throughout our lifespan and are updated and remodeled as we familiarize our self with our continuously changing environment (Boudreau et al. 2007). Motor skills can be classified as either gross or fine motor skills, with gross motor skills being involved in the movement and coordination of large muscle groups (such as arms, legs and torso) and fine motor skills which are involved in the use of smaller muscle groups (such as those that act on the wrists, hands, fingers and toes) (Kandel et al. 2000, Willingham 1998).

The acquisition of a new motor skill follows two distinct stages with continued practice: first, there is an early, fast learning stage in which performance improves rapidly within a single training session; later, there is a slower learning stage in which incremental gains in performance are seen over several sessions of practice (Ungerleider et al. 2002). The latter stage involves processes of practice-independent learning known as consolidation (Karni and Sagi 1993, Maquet et al. 2003, Walker et al. 2003). The retention of a skill is dependent on the amount of practice however, long periods of non-practice or use of a skill has the ability to undo the connections that were developed during practice. Typically, performance levels of gross motor skill remain unchanged during long periods of non-use, however, retention loss during long periods of non-practice for fine motor skills appear much sooner (Dayan and Cohen 2011). Motor skill acquisition is usually measured by a reduction in reaction time and the number of errors, or accuracy, with the retention of these improvements generally assessed 24 to 48 hours after motor skill acquisition (Dayan & Cohen, 2011). There is strong evidence that the cerebellum is highly active in motor adaptation and learning, especially in the early stages of skill acquisition (Doyon et al. 2002, 2003).

Altered afferent input in the form of behavioural training has been shown to induce organizational changes in both the animal and human cortex (Byl et al. 1997, Classen et al. 1998). The alterations in the afferent input, which was shown to induce these changes in the CNS does not have to be of long duration. In fact, repetitive muscle contraction activities have been shown to have an effect on neural activity with as little as 20 minutes of repetitive finger contractions (Murphy et al. 2003). The effects seen in the neural activity of the prefrontal cortex and brainstem for up to 20 minutes following the cessation of the finger activity supports the hypothesis that the cerebellum has neuroplastic capabilities with repetitive practice (Murphy et al., 2003). The mechanisms which underlie these use-dependent representations, however, are not well understood and must be further investigated.

1.6 NEUROPLASTICITY

Neuroplasticity is an intrinsic and fundamental neurophysiological adaptation that refers to changes in the structure, function and organization within the CNS which occurs continuously throughout a person's lifetime (Boudreau et al. 2007, Boudreau et al. 2010, Kleim et al. 2003, Pascual-Leone et al. 2005). Research over the past decade has demonstrated that altered afferent input to the CNS leads to plastic changes in the way that the CNS processes information and responds to subsequent input (Brasil-Neto et al. 1993, Byl et al. 1997, Haavik-Taylor and Murphy 2007, Pascual-Leone and Torres 1993, Rothwell et al. 1999). Neuroplastic changes have been demonstrated in response to experience and behaviour (Pascual-Leone and Torres 1993, Recanzone et al. 1992, Tyč et al. 2005), motor learning (Bayona et al. 2005) (Adkins et al. 2006, Kleim et al. 2003, Kleim et al. 1998, Nudo et al. 2000, Plautz et al. 2000), pain (Flor 2002, Rittig-Rasmussen et al. 2014, Shigemoto et al. 1997), injury (Elbert and Rockstroh 2004, Hamilton and Pascual-Leone 1998) and sensory stimuli (Hamdy et al. 1998, Jenkins and Merzenich 1984, Merzenich and Jenkins 1993). Cortical changes can be brief, which reflects the adaptability of the sensorimotor system to respond in response to rapid changes in the internal and environmental demands (Classen et al. 1998, Hayashi et al. 2002).

Neuroplastic changes in sensory-motor areas are stimulus driven, therefore, lasting neuroplastic changes typically occur when the practice of a skill is repetitive, relevant and is isolated from distraction or noise (Byl et al. 1997, Jenkins et al. 1990, Pascual-Leone and Torres 1993, Remple et al. 2001, Tyč et al. 2005). Neuroplastic changes have been observed in different areas of the CNS including the spinal cord, subcortical and cortical areas. The cerebellum is known to undergo neuroplastic changes following motor training and is responsible for the modulation and adjustments required after a new skill is learned, in order to update the motor circuitry (Baarbé et al. 2013). In fact, the cerebellum plays a key role in the prediction of movement outcomes is required

for online (or real time) movement correction when learning a new task (Kleim and Jones 2008). The cerebellum is also responsible for using online corrections to update body schema in the feedforward model of motor control (Kleim & Jones, 2008), which allows for corrections to be made prior to the time that is physically required to complete a task or goal (Pascual-Leone et al. 2005).

There is a growing body of evidence that reveals structural and functional changes within the CNS of people with chronic musculoskeletal disorders (MSK) (Boudreau et al. 2010, Pelletier et al. 2015, Stanton et al. 2016). These changes are described to be initially beneficial to help the body adapt to the adjustment in sensory information, however, overtime, these changes are thought to be influential in the neurophysiology of the condition (Pelletier et al. 2015). Due to the reciprocal role of the cerebellum in SMI and the coordination of limb movements it is possible that altered sensory input from the neck has the ability to alter sensory integration and processing, which is subsequently expressed as adjusted or adapted movement patterns (Pelletier et al. 2015).

1.7 PANJABI'S STABILITY MODEL

Spinal stability is the basic requirement for the transfer of power forces between the upper and lower limbs, the active generation of forces in the trunk, the prevention of early biomechanical deterioration of spine components and the reduction of the energy expenditure during muscle action (Guillot et al. 1990, Haher et al. 1993). Instability is typically considered as an increase in individual joint motion during various body movements and is often associated with the occurrence of back and/or nerve root pain (Haher et al. 1993). Bones, disks and ligaments contribute by playing a structural role and by acting as transducers through their mechanoreceptors (Haher et al. 1993). Mechanoreceptors send proprioceptive impulses to the central nervous system which coordinates muscle tone, movement and reflexes (Kandel et al. 2000). Damage to any spinal structure gives rise to some degree of instability (Guillot et al. 1990, Haher et al. 1993).

Panjabi's model (1992) described a unique concept of spinal stability and its respective training parameters. More specifically, Panjabi described the functional ability of the spine to be dependent on the stability between three subsystems; the active system (muscles), the passive system (joints and soft tissue), and the neural system (neural conduction) (Panjabi 1992). The basic biomechanical functions of the spinal system are as follows: a) to allow movements between body parts, b) responsible for carrying external loads and c) the protection of the spinal cord and its nerve roots – making mechanical stability of the spine necessary when performing any of the described functions (Panjabi, 1992). A stabilized system, according to Panjabi, would be one to provide sufficient stability of the spine by coordinating subsystems to match the ever-changing stability demands from changes in spinal posture via static and

dynamic loading (Panjabi, 1992); this process has been designed, developed and optimized to reach the goal of spinal stabilization.

Panjabi (1992), proposed that the failure of one subsystem would result in the compensation of the remaining two, which overtime, would influence further alterations in motor control, eventually leading to chronic injury. The end result would therefore lead to impaired movement, regardless of the original cause and the ability to improve these weaknesses would help to restore the communication between three significant variables of human movement (Hoffman and Gabel 2013). This model has been used to generate hypotheses for a number of different low back interventions (Hoffman and Gabel 2013). With the growing incidence of neck pain this stability model could be of similar use to assist with the conceptualization and development of clinical interventions when addressing neck pain; especially, since the characteristics between upper and lower spinal segments are physiologically similar.

The ability of the spinal system to respond to dysfunction is one manifestation of its adaptability. It is believed that under circumstances of unusually demanding loading conditions, there may be a functional reserve that can be called upon to enhance spinal stability beyond its normal physiological level. The consequence, however, may become detrimental to the individual components of the spinal system if kept constant overtime, leading to abnormal muscle loading, accelerated degeneration of spinal structures and tissue and muscle fatigue; all of which, can lead to acute or chronic injury (Panjabi, 1992).

1.7.5 The Passive System: Relationship to altered neck function

According to Panjabi (1992), the passive subsystem maybe be caused by mechanical injury, such as, overloading a normal structure rapidly, or, the normal loading of a weakened structure (Panjabi, 1992). These injurious situations can lead to overstretching of the ligaments, development of tears and fissures in the annulus, development of microfractures in the end plates and/or extrusion of disc material into the vertebral bodies (Panjabi, 1992). Development of anyone of these injuries has the ability to alter the load-bearing and stabilizing capacity of the passive system, leading to compensatory changes in the active system which could lead to acute or chronic injury (Panjabi, 1992). Different joints can be classified based on their structural stability or mobility. In fact, it is well known that a joint with greater mobility will be less stable in order to move efficiently through the given range of motion (Hoffman and Gabel 2013). The most prominent and identifiable symptom of FHP is the excessive extension of the cervical vertebrae causing postural deformities and biomechanical changes in the structure and function of the joints associated with the neck (Abrahams 1977). While in a seated position, individuals with FHP have been found to counterbalance the strain placed on their neck by combining lower cervical flexion with excessive upper cervical extension (16). In this particular study, the weakness from the

head and neck transitioned down the kinetic chain and influenced the position of the shoulders (Kwon et al. 2015). The authors described the posture of individuals with FHP to also have rounding in their shoulders when seated, which altered the muscle lengths of the back and chest, further increasing the torque and strain placed on the cervical spine articulations (Kwon et al. 2015).

The cervical region is known to be the most mobile section of the vertebral column in order to support the various movements of the head (Knoop et al. 2013). Even though each joint is mobile, it relies heavily on the muscular system for stability in order to perform a movement with efficiency and stability (Hoffman and Gabel 2013). One study discussed the effects of neck pain on other areas of the body, including shoulder pain and muscle weakness in the arms (Shin et al. 2017). The results of the study indicated that there was a negative correlation between neck pain and shoulder joint range of motion (Shin et al. 2017). In addition to these findings, it was also observed that participants also demonstrated a reduction in muscle strength during shoulder abduction in the presence of neck pain (Shin et al. 2017). In addition to this, Kwon et al. (2015) reported that due to the alignment change of the scapula, in relation to the thoracic spine, FHP was associated with decreases in the recruitment times and patterns of prime movers of the scapulothoracic joint. As a result, the alteration restricted the individual's shoulder range of motion putting further strain on the neck and shoulder during upper limb movement (Kwon et al. 2015).

1.7.6 The Active Subsystem: Motor Control Dysfunction of the Cervical Spine

According to Panjabi, deterioration of active subsystem components leads to a reduced ability of spinal muscles and tendons to receive or carry out neural commands (Panjabi, 1992). Efficient communication of the active subsystem is needed in order to provide accurate feedback on muscle tension information to the neural control unit (Panjabi, 1992). Under normal conditions, the active subsystem produces coordinated and adequate muscle tension during static and dynamic movements; however, deformation to the integrity of the system can result in disuse of important spinal muscles, degeneration of tissue and/or joint structure leading to injury (27). The dysfunction of any part of the active subsystem can compromise its ability to provide both compensatory help to the passive system when needed, as well as, support the passive system during unexpected dynamic or abnormally large external loads (27).

Muscle fibers can be classified as one of two main types; slow-twitched (type 1) muscle fibers or fast-twitched (type 2) muscle fibers. Slow-twitch fibers are known to contribute to stability-based movements due to their high aerobic capacity and are their ability to withstand low-force, isometric or dynamic contractions (Hoffman and Gabel 2013). Neck pathology studies have shown evidence of a transformation, or shift of the deep cervical muscle fibers from type 1 to type 2

(stronger but less aerobically efficient) in the presence of pain and dysfunction of the cervical spine (Hoffman and Gabel 2013). The concept of the body to recruit stronger muscles is understandable, however, with strength comes lack of endurance. The inability of type 2 muscle fibers to maintain its contraction over a lengthy period of time would therefore explain the fatigue observed in clinical studies (O'Riordan et al. 2014).

The deep flexors of the posterior aspect of the cervical spine act like dynamic ligaments and are known to contribute to the control and alignment of the cervical vertebra; and so, are vital in maintaining ideal cervical alignment (Falla and Farina 2007, Jull et al. 2007). Previous research has identified alterations and impairments within the activation patterns of the deep cervical flexors of individuals suffering from NP, and those with FHP (Falla and Farina 2007, Harman et al. 2005, Jull et al. 2007). These studies have also shown that a result of the alteration causes the superficial muscles of the cervical spine to activate in place of the deeper cervical structures resulting in early fatigue, over-activity and pain (O'Riordan et al. 2014). In a recent review, it was acknowledged that individuals who suffer from NP also showed a reduced ability to relax the superficial cervical muscles while in a rested state (Falla and Farina 2007).

Increased activity of the superficial cervical muscles of individuals with NP was seen during performance of low load tasks suggesting that a compensation strategy was demonstrated by the CNS as a way to make up for weakness and inhibition of the deep cervical muscles (Falla et al. 2004). Taken together, these findings show that not only were these muscles working harder during multi-directional movements, they were also seen to be over active and stimulated during periods of rest. Similar findings were demonstrated in individuals with FHP, with alternations further reducing their ability to maintain an upright sitting posture that favoured ideal cervical alignment (Caneiro et al. 2010).

1.7.7 The Neural Subsystem: Altered Motor Control

Dysfunction of the neural subsystem, according to Panjabi, is described as compensations that cause sensory error and contributes to excessive muscle tension leading to soft tissue injury, alteration in neural control and/or pain (Panjabi, 1992). An example of this would be the alterations in the firing patterns of spinal muscles in a manner that is undesirable to the system. These alterations include: firing patterns that are too small or large and/or firing patterns that are too early or too late. This is believed to be the result of either faulty information transmitted from sensory receptors or due to deficits found within the neural control unit itself (Panjabi, 1992).

Alterations and motor control abnormalities have been well documented in individuals with NP (Jull et al. 2004). Recently, similar results are being documented in those with FHP (Neupane et al. 2017). The proposed theory of

neck pain and its relationship with FHP is described as an alteration of afferent input sent to the brain from the peripheral nervous system (PNS) (Neupane et al. 2017). The evidence supports this theory as changes observed in the cortical area devoted to processing PNS input has been well documented in the literature of individuals with FHP and NP (Neupane et al. 2017, Snodgrass et al. 2014). Neuroplasticity can be defined as an adaptable, intrinsic response of the CNS to ever changing environmental, physiological or psychological stimuli (Haavik and Murphy 2012). Evidence is emerging in favour of the theory which suggests that the CNS reorganizes how it responds to a given stimulus in the presence of musculoskeletal pain or dysfunction (Snodgrass et al. 2014).

When an individual is learning a new task, adaptive neuroplastic behaviour of the CNS will allow the individual to acquire and store the new task for future recall (Shin et al. 2017). Conversely, maladaptive neuroplastic behaviour, which is observed in the presence of pain and dysfunction, has an opposite and adverse effect on skill acquisition (Shin et al. 2017); whether it is new or familiar. Recognizing and accepting these neuroplastic behaviours as a modifiable variable of motor control is important to the understanding of sensorimotor integration (SMI) and the treatment of neurophysiological dysfunction (Shin et al. 2017). Studies have shown that effective sensorimotor integration patterns of the CNS are essential to how the body responds physically to a given phenomenon (Strimpakos 2011). Therefore, understanding both adaptive and maladaptive behaviour observed in the brain activity of healthy individuals and those with FHP would be beneficial to our understanding of the development and neurological alterations underlying neck fatigue and SCNP.

1.8 FORWARD HEAD POSTURE AND TECHNOLOGY USE

FHP is an acute structural disorder which occurs when the head becomes positioned anteriorly, in relation to the body's center of gravity (Neupane et al. 2017). FHP is measured as the sagittal distance from C7 to the nose or the angle between C7 and the ear relative to horizontal with most studies reporting FHP as the measurement reflecting head and neck position with respect to the trunk (Neupane et al. 2017). The head is considered to be dynamically balanced on the cervical spine when tragus of the ear is located over the acromion process of the scapula (Kapandji 1971). From a biomechanical perspective, this joint, which is referred to as the atlanto-occipital joint, can be considered a first-class lever system (Kapandji 1971). With respect to the human body, the head would act as the lever, and the atlanto-occipital joint would be considered the fulcrum, which is placed directly over the center of the lever (ie the cervical spine) (Kapandji 1971). Having the head in this position allows for quick and rapid movements, however, this mobility comes at the cost of strength and force production from the surrounding muscles which act directly on the cervical spine (reference). Studies have shown that FHP is known to contribute to predictable patterns of tissue

overload and dysfunction; resulting in a reoccurring cycle of pain and discomfort (Audette et al. 2010, Neupane et al. 2017). It is believed that a more extended upper cervical angle (UCA), (commonly observed in FHP) is developed in response to a shift in activation patterns between cervical flexors and extensors, in order to maintain eye gaze; which becomes larger when there is an increased anterior translation of the head (Silva et al. 2009) Although FHP is commonly observed in patients with cervical pain and is frequently reported in studies related to cervical pain, (Yip et al. 2008) the strength of the relationship between FHP and cervical pain is still a topic of discussion (Audette et al. 2010, Neupane et al. 2017).

FHP has been shown to lead to the lengthening and weakness of the anterior cervical muscles with a corresponding shortening of the posterior cervical muscles (Lee et al. 2015). These muscles, which attach directly to the cervical vertebra, play an important role in the alignment and position of the cervical spine during multi-directional movement (Panjabi, 2012), which in turn, provides stability and support to the head (Knoop et al. 2013). According to Kapandji (1971), for every inch of forward head translation an additional 10 pounds is added to the total weight of head carriage on the cervical spine. The muscles which attach directly to the cervical vertebrae play an important role in the alignment and position of the head during multi-directional movement (Falla and Farina 2007), by providing stability and support to the neck (Ravi et al. 2016). In order to achieve ideal neck movement, each section of the cervical spine should be properly aligned and mechanically stable in order to reduce the impact of gravitational forces, in addition to, forces accumulated during postures of an individual's activities of daily life (Falla et al. 2004).

Work-related musculoskeletal disorders (WMSD) are a significant health problem worldwide, with intensive computer users particularly affected (Buckle and Devereux 2002, Gerr et al. 2004, Tittiranonda et al. 1999). Neck pain and dysfunction has been reported in individuals who spend majority of their workday in a seated position (Neupane et al. 2017). The sitting patterns used during computer use has been shown to alter the mechanical structure of the cervical spine, leading to weakness and pain (Rossignol et al. 1987, Szeto and Lee 2002, Szeto et al. 2002). Adopting a sustained, non-neutral spinal posture has been shown to alter the muscle activity of the head and neck (Szeto et al. 2009, Vakili et al. 2016). Therefore, an exaggerated forward head posture over time may place stress on specific cervical regions and their musculoskeletal system by increasing flexion of the lower cervical spine with a corresponding extension of the upper cervical spine (Friction et al. 1985, Gossman et al. 1982). In support of this, Falla et al. (2007) reported that individuals with chronic neck pain demonstrated a reduced ability to maintain an upright sitting posture.

More recently, there is a growing interest in musculoskeletal (MSK) disorders related to smartphone use (Neupane et al. 2017). A review recently reported that 79% of the population between the age of 18-44, admitted to having their cell phones on them for the majority of their day (Neupane et al. 2017). The motion of looking down at your phone can lead to postural changes of the upper back and neck which could lead to the development of upper back pain, shoulder pain and tightness if left over time. Despite the growing evidence between MSK disorders and FHP (or text-neck), there is little discussion with respect to the sensory changes and the neurological adaptation that occurs in the presence of long term or extreme cases of FHP. This sudden increase of reported neck pain cases has placed a tremendous strain on the health care system as these individuals are said to use twice as many services when compared to the rest of the population (Lee et al. 2015). A modern-day lifestyle which includes increased technology use and poor work station set-up has exacerbated the occurrence of neck pain (Carroll et al. 2008, Carroll et al. 2009); therefore, investigation into the neurophysiological properties of this phenomena would be beneficial to our understanding of neck posture and its influence on SMI.

1.8.1 Effect of altered neck sensory inputs on SMI and motor control

The brain uses a series of excitatory and inhibitory activation patterns (feedback loops) to improve the accuracy and control of movements, and will repeat this process until otherwise instructed (Riemann and Lephart 2002). The neural control subsystem receives its information from the sensory receptors (as mentioned above) located in and around the joint that respond to the mechanical stress placed on the joint capsule and ligament (Panjabi, 2012). Based on the sensory information provided, the brain determines specific requirements needed for the maintenance of spinal stability and, therefore, communicates with the muscles of the spine to achieve stabilization before limb movement begins (Panjabi, 2012). The neural control system accomplishes this through the measurement of individual muscle tension provided by muscle spindles and golgi tendon organs. Instantaneous adjustments are made in accordance to the stability requirements and are constantly updated until ideal spinal posture, movements and loads are achieved (Panjabi, 2012). Collectively, these pathways and structures work to influence and create a postural model known as body schema, which is defined as, the presence and awareness of one's physical occupancy in relation to their environment. This neural map of the body is used to generate accurate and coordinated voluntary movement based on previous experience and previously stored synchronization of muscle recruitment patterns (Doyon et al. 2003).

In order to produce coordinated and accurate limb movements, the ability of the spinal system to remain stable is necessary for agonist muscles to function efficiently (Panjabi 1992, Strimpakos 2011) The CNS therefore, has the task of

continuously and simultaneously monitoring and adjusting the forces in each of the muscles surrounding the spinal column and adjusting to the ever-changing demands of the environment (Strimpakos 2011). Muscles which attach to the cervical spine have a high density of sensory receptors, which are located in the joint capsules of the cervical spine, as well as the highest density of proprioceptors in the neck muscles (ref) which project to the CNS and play an important role in SMI. Kebaetse et al., (1999), associated FHP with the shortening of the neck extensor muscles with the subsequent tightening of the anterior neck muscles. Furthermore, FHP was also associated with alterations in the activity of shoulder muscles (Kebaetse et al. 1999, Kwon et al. 2015, Lee et al. 2015), which, over time, could potentially affect upper limb performance.

Fatigue

CEM fatigue has been shown to affect postural control, possibly due to a decrease in central drive and/or inconsistencies in proprioceptive feedback during task performance (Gosselin et al. 2004). Proprioceptive feedback is produced by muscle spindles during muscle contraction and has been previously shown to be affected by muscle fatigue (Zabihhosseinian et al. 2015). Allen & Proske, (2006) demonstrated that upper limb positional error increased with the effort exerted from fatigue. The authors concluded that the positional errors were due a decline in muscle spindle activity, thereby adjusting the motor unit firing rate, subsequently altering the recruitment patterns of the muscle (Macefield et al. 1991). Deficits in motor control of the spine can lead to poor control of joint movement, leading to the presentation of repeated microtrauma which can eventually to pain (Panjabi, 1992). For example, increased neural activity to the muscles of the neck muscles (upper trapezius and the levator scapulae muscles, sternocleidomastoid), due to a poor working posture of the neck or of the arms, may over time increase the compressive load on cervical segments and initiate a painful neck condition. Therefore, adopting a sustained, non-neutral spinal posture, such as FHP, could lead to the increased activation of important head and neck stabilizers, making these muscles susceptible to the development of fatigue (Falla et al. 2004).

More recently, Zabihhosseinian et al., (2015), demonstrated that CEM fatigue was able to change recruitment patterns of cervical neck extensors (Zabihhosseinian et al. 2015), reduce the accuracy of upper limb motor performance (Zabihhosseinian et al. 2015), and alter the accuracy of an eye-hand coordination task (Zabihhosseinian et al. 2019). Collectively, these studies suggest that neck fatigue has the ability to impact the internal feedforward models of the brain, which ultimately affects the way our body interprets our environment (Zabihhosseinian et al. 2019). CEM fatigue has also been found to alter cortical and cerebellar processing techniques, in response to novel motor skill acquisition, (Zabihhosseinian et al. 2019), however, the effect of FHP on SMI

and task performance are still unknown. It has been hypothesized that changes commonly seen in muscle recruitment patterns are developed as a protective mechanism to underlying spinal instability resulting from laxity or damage to the structural tissue of the head and neck, muscle dysfunction, or reduced neuromuscular control (Kwon et al. 2015, Shin et al. 2017) There is also evidence in the literature to suggest that muscle impairment occurs early in the history of onset spinal complaints,(Kwon et al. 2015) and that such muscle impairment does not automatically resolve even when pain symptoms improve (Jull et al. 2008, Kwon et al. 2015). Individuals with mild to moderate neck pain have been found to have decreased muscle endurance (Lee et al. 2004, Lee et al. 2005), decreased range of motion (Lee et al. 2004) impaired cervical kinesthesia (Lee et al. 2005) and may also impact upper limb proprioception (Haavik and Murphy 2011). Recent neurophysiological studies suggest individuals with SCNP may have altered cerebellar processing (Daligadu et al. 2013). This suggests that this population was unable to cope with an external demand, such as fatigue, due to the cortical alterations associated with mild-moderate neck pain. Previous studies have shown that altered sensory input from the neck resulted in neurophysiological changes in both cerebellar-related SEPs (Andrew et al. 2018), and cerebellar disinhibition measured via TMS (Baarbé et al. 2018) subsequent to a motor learning task. Changes in sensory input is also known to affect the activity of 1A fibers (Bolton 1998), as well as impacting upper limb proprioception (Knox et al. 2006, Zabihhosseinian et al. 2015). Both pain and neck muscle fatigue have been shown to contribute to alterations in both proximal and distal limb and forearm position sense (Baarbé et al. 2018, Bolton 1998, Haavik and Murphy 2011, Knox et al. 2006, Wall et al. 2002, Zabihhosseinian et al. 2015) and alterations in motor unit recruitment patterns and activity has been linked with neck pain (Kristjansson et al. 2003, Revel et al. 1991, Roren et al. 2009); however, the extent to which FHP alters SMI and influences subsequent motor performance remains unknown.

Fatiguing contractions lead to changes in all levels of the motor pathway (Taylor and Gandevia 2001). Faulty cervical spinal alignment, such as FHP, is usually associated with shortening of the posterior neck extensor muscles and tightening of the anterior neck muscles, as well as the shoulder muscles, affecting scapular position and kinematics (Kebaetse et al. 1999). The change in muscle activation following sustained changes in cervical posture can result in localized muscle fatigue, which, may enhance the signs and symptoms of repetitive strain injury in the cervical spine and glenohumeral-scapular complex (McLean 2005). Sustained and prolonged muscle contraction has been shown to lead to the development of chronic cervical and shoulder pain syndrome (Edwards 1988, Sluiter et al. 2001); with progressive structural changes observed in the postures of computer workers, which have been shown to be associated with the increased activity of the trapezium muscle (Kleine et al. 1999); a primary neck extensor.

SCNP represents recurrent neck pain of greater than 3 months in duration (Haavik and Murphy 2011, Lee et al. 2008, Lee et al. 2004, Lee et al. 2005). Andrews et al., (2018), recently demonstrated that SCNP has the ability to impact motor learning, as well as, SEP peaks related to SMI through changes in cortical activity associated with the cerebellar-M1 pathway (Andrew et al. 2018). Therefore, it is plausible to assume that other forms of altered input from the neck, such as the changes in muscle activity in the presence of FHP, are likely to impact upper limb SMI, task acquisition and subsequent motor performance. A TMS study investigating changes in cerebellar-inhibition found that following motor acquisition, those with (SCNP) had no capacity to disinhibit inhibitory cerebellar projections to the motor cortex, relative to healthy controls who showed significant disinhibition (Baarbé et al. 2018). Alterations in sensory information processing due to SCNP, appears to have altered the capacity for plasticity of the cerebellum to motor and somatosensory cortex pathways (Doyon et al. 2003, Doyon et al. 2002). A previous study found that CEM fatigue altered neck motor control as measured by the flexion relaxation response (Zabihhosseinian et al. 2015). Following fatigue, the CNS attempted to stabilize the cervical spine by increasing CEM muscle activation even during the relaxation phase of the response in a healthy control group. Interestingly, in the same study, an SCNP group had differential neuromuscular activation with less CEM muscle activity following fatigue, possibly because their neuromuscular system was already maximally activated to decrease the risk of further injury (Zabihhosseinian et al. 2015). Several variables have been identified as risk factors for the development of shoulder and neck pain, including the prolonged, sustained posture of the cervical spine, repetitive use of the arm, and work with the arm in an elevated position (Kanchanomai et al. 2011). Lifting and overhead work in the sagittal plane are often performed in construction and assembly line workplaces for loading and unloading, and FHP is commonly associated with the typically assumed posture during computer and cell phone use (Burgess-Limerick et al. 1999, Kanchanomai et al. 2011, Neupane et al. 2017)

Neck Pain (NP)

Altered afferent input due to recurrent neck pain and associated neck joint dysfunction has been suggested by Haavik and Murphy (2012) to lead to altered afferent processing of incoming sensory information from the upper limb, leading to disordered sensorimotor integration, with the potential to interfere with motor sequence acquisition. In a previous postural study, the onset of neck pain was predicted by the position of a computer screen and whether it was level with the eyes (Villanueva et al. 1997). Higher computer screen heights were found to be related to increases in neck extension (Villanueva et al. 1997), subsequently altering the muscle activity of cervical extensions and flexors (Seghers et al. 2003). The authors concluded that the prolonged use of a computer that was not at eye level, may increase the possibility of developing neck pain versus a screen

that was at eye level. The distance between the individual and the computer screen may also influence neck symptoms, head posture and neck muscle activity. Rempel (2007) found that the viewing tasks on a computer display led participants to make postural adjustments to their torsos and heads that brought them closer to the display. That is, the amount of the postural adjustment and foreshortening of the viewing distance increased with farther viewing distances (Rempel et al. 2007).

NP is considered a chronic episodic condition, characterized by persistent, recurrent, or fluctuating pain and disability (Côté et al. 2004). Musculoskeletal pain occurs due to changes in muscle length when assuming a poor posture for a prolonged, repetitive movements period (Bergqvist et al. 1995), therefore, potential mechanisms common to FHP may alter the length and tension of muscles which attach to the neck. Despite previous work examining the relationship between FHP and the development of neck pain, there is still a universal disconnect between the effects of FHP on the recruitment patterns and cortical activity of the CNS and subsequent effects on upper limb performance. Repetitive strain injuries (RSI) and occupational overuse injuries (OOI) prevalence has had a marked increase in prevalence among Canadian adults since the late 1990s (Côté et al. 1998, Tjepkema 2003). Furthermore, neck pain is a common health problem associated with significant disability in the general population (Côté et al. 1998, Manchikanti et al. 2009), most of whom typically devote the majority of their day to technology use (Neupane et al. 2017).

If FHP is related to NP, then understanding the potential preliminary changes that occur before the development of pain would be beneficial in reducing the substantial demand this spinal dysfunction places on the health care system and better assist clinicians with adequate rehabilitation programs specific to these neural changes.

1.9 EVENT- RELATED POTENTIALS (ERPs)

An event-related potential (ERP) is a fixed electrophysiological response to an internal or external stimulus, which can be defined as the voltage fluctuation in response to the processing and/or perception of a given stimulus (Picton et al. 2000). The most basic form of electrical communication between cells in the human body is the action potential (Passmore et al. 2014); which is defined as, the movement of ions (Na⁺, K⁺, Ca⁺⁺, and Cl⁻) through neuronal membranes in the direction governed by the membrane potential (Kandel et al. 2000). Responses of the PNS and CNS to internal and external stimuli initiates the propagation of electrical activity within the cellular membrane (Kandel et al. 2000) and can be produced through the external stimulation of a peripheral nerve (Passmore et al. 2014). Scalp electrodes record the difference in electrical activity of post-synaptic potentials, which is the summation of extracellular currents (Kandel et al., 2000). Excitatory inputs from afferent nerve potentials

result in an influx of Na⁺ and Ca⁺⁺ ions towards an excitatory synapse, resulting in an outward current flowing in the opposite direction (Kandel et al., 2000). The outward current, known as an inhibitory potential, involves Cl⁻ and K⁺ ions flowing in the opposite direction of the afferent activity-- usually following excitation after a brief temporal delay (Kandel et al., 2000). The balance between the two potentials contribute to the fine tuning of neural integration and is essential when performing controlled movement patterns of skeletal muscles (Kandel et al., 2000).

Signal fluctuations are filtered and averaged using continuous EEG measurements for clinical diagnoses and treatment interventions (Picton et al. 2000). Since EEG reflects the summation of thousands of simultaneous neural processes, the brain's response to a specific stimulus is rarely visible in a continuous EEG recording, due to the low amplitude response of a single event (Kandel et al., 2000). Therefore, ERPs that are recorded over time, using a recurring stimulus permits the use of an averaging technique which is effective for cancelling out noise and spontaneous EEG artifacts (Picton et al. 2000). Taken together, this process is known to enhance the amplification of a given stimulus and provides valuable insight to the sequential process of neural integration (Picton et al. 2000). ERPs are used in research to develop an understanding of how an individual's CNS will respond to an ever-changing environment (Passmore et al. 2014). These responses can be further broken down in to individual classifications based on the neural pathway of interest (Passmore et al. 2014). The somatosensory system is comprised of PNS and the CNS which detect senses of touch, vibration, temperature, pain and kinesthetic awareness (Passmore et al. 2014). Therefore, somatosensory evoked potentials (SEPs), can be used to examine sensory nerve pathways between the extremities (PNS) and the dorsal column of the spinal cord (CNS) (Passmore et al. 2014). The dorsal column is the primary pathway to the cerebral cortex, which is the area of the brain associated with higher cognitive functions such as thoughts and actions (Passmore et al. 2014).

1.9.1 Somatosensory Evoked Potentials (SEPs)

The International Federation of Clinical Neurophysiology (IFCN) recently updated its guidelines of recommended standards for the acquisition of somatosensory evoked potentials (Cruccu et al. 2008). These findings have been used to generate recommended SEP stimulating and recording parameters which are described in detail in the following section of this review (Cruccu et al. 2008). It is well known that the CNS can reorganize in response to altered afferent input (Murphy et al. 2003). Decreased sensory input (due to nerve damage, disuse etc.) has been shown to decrease the size of the brain map (known as the motor and sensory homunculus) relative to the body part injured (Murphy et al. 2003).

Whereas increased sensory input can expand the size of the neural region devoted to that body part; which is made possible due to the plastic nature of the brain (Murphy et al. 2003). These changes in neuronal activity can be measured using somatosensory evoked potentials (SEPs), which includes, the changes in pathways between the cerebellum and SI and cerebellum-M1 before and after motor acquisition (Passmore et al. 2014). Somatosensory evoked potentials (SEPs) are elicited by stimulating the peripheral nerve at a distal end of a joint, (e.g. the median nerve at the wrist) (Tolikas 2005). Fine touch, proprioception, and vibration sense are conducted rostrally through the ipsilateral cuneate tract in the dorsal columns of the spinal cord before synapsing in the nucleus cuneatus in the lower brainstem. From here, fibers project through the brainstem to the contralateral ventral posterior lateral nucleus of thalamus (VPL) (Berger and Blum 2007). The contralateral VPL contains extensive connections to the contralateral somatosensory cortex in the parietal lobe, relative to the site of stimulation (Berger and Blum 2007). SEPs are widely-utilized and have been used as an intraoperative monitoring method for more than 30 years (Nash et al. 1977). SEPs are also used when examining the effect of different treatments and drug interventions on the central nervous system (CNS), and used to monitor the level of the CNS where these subsequent changes occur (Passmore et al. 2014).

Peripheral Nerve Stimulation

It is possible to stimulate virtually any sensory nerve in the body and record its associated neural activity. The stimulation of sensory receptors in the skin initiates the activation of peripheral sensory nerves, which extend through the brachial plexus to the dorsal root ganglia and ascend up to the cerebral cortex for further integration (Berger and Blum 2007). According to the updated IFCN guidelines, SEP stimulation should be applied on the surface of the skin over a peripheral nerve using a constant electrical pulse lasting between 0.1-0.2 ms (Cruccu et al. 2008). The intensity of the pulse should produce a clear and tolerable sensation of at least 2-3 times the sensory threshold, with the rate of stimulation being issued between 3-5Hz (Cruccu et al. 2008). Due to the accessibility and reliability of the stimulation and recording sites and the existence of ample normative data supporting the effectiveness of its measurement, both the median (upper body sensory information) and posterior tibial nerve (lower body sensory information) are the most commonly used peripheral nerve in clinical research (Berger and Blum 2007). For most clinical situations, the most important potentials examined are those derived from the activity of the cervical spinal cord, relay nuclei, and the cerebral cortex (Berger and Blum 2007).

Signal Recording

Skin preparation is recommended but can be challenging for individuals who have a lot of hair. It is recommended that individuals should arrive to the recording session with freshly washed and dried hair without any conditioner (Passmore et al. 2014). This is important for good skin conductance while avoiding conduction bridges between electrodes (Passmore et al. 2014). To eliminate skin potentials, the impedance at the scalp–electrode junction will need to be reduced to less than 5000 Ω to preserve the quality of the EEG signal (Cruccu et al. 2008). Analysis time should be at least 100ms after stimulation, since most SEP components peak before 50-100ms (Cruccu et al. 2008). Lastly, IFCN guidelines suggest an average of 500 trials to be recorded for a minimum of two blocks of stimulation to ensure reproducibility of the SEP wave of interest (Cruccu et al. 2008).

For standard clinical SEP readings, it is recommended that a minimum of four, one centimeter, surface recording electrodes be placed as per the 10-20 international EEG system guidelines (Klem et al. 1999). Electrodes should be attached to the scalp with an accuracy of within 5 mm (Picton et al. 2000). This standardized placement is expressed as a percentage of distance between key landmarks of the skull and is identified by a letter corresponding to the region of the brain associated with the EEG measurement (F; frontal, P; parietal, T; temporal and O; occipital (Klem et al. 1999). However, for the purpose for this thesis, we recorded differential changes in SEP peak amplitudes using a 64-electrode EEG cap.

The exact locations of the electrodes, using an EEG cap, can be determined relative to anatomical landmarks indicated by the 10/20 international EEG system. The point which intersects the midpoint of both the anterior-posterior and lateral coronal planes is labelled Cz indicating the relative center point of the individuals' skull (measured at 50% distance from nasion to inion; A-P plane and tragus to tragus; coronal plane) (Klem et al. 1999). From here, additional electrode placement will fall between 10% of the total measurement and (labeled Fp), and 20% intervals of the total measurement and labeled with a letter corresponding to the region of the brain (Figure 1) (Klem et al. 1999). An active electrode is placed ipsilateral to the site of stimulation and a reference (ground electrode) should be identified as the scalp electrode Fz (Cruccu et al. 2008).

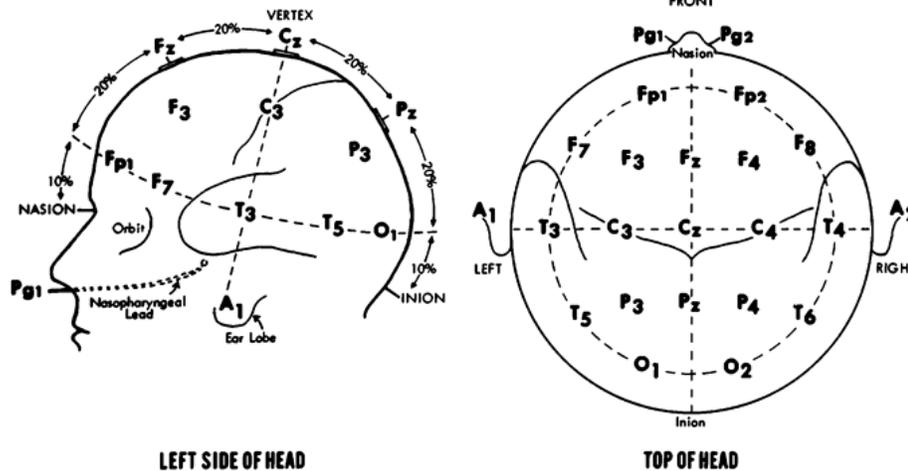


Figure 1. Electrode placement using the IFCN 10/20 system. Retrieved from: <https://instrumentationforum.com/t/electrode-10-20-system/5486>

1.9.3 SEP peak nomenclature

Amplitudes and latencies of SEP peaks represent processing at different levels of the CNS of incoming sensory information (Passmore et al. 2014). For example, the amplitude of a given waveform represents the magnitude of the incoming information from the PNS and the latency reflects the anatomical location of the signal along the somatosensory pathway (Passmore et al. 2014). Waveform reproducibility is made possible by averaging several controlled stimuli to time locked trials (Passmore et al. 2014). The resulting waveform can therefore be analyzed using peaks and troughs, presented at different time points, relative to the onset of stimulation (Passmore et al. 2014). Waveform peaks are assigned a letter and number representing the polarity and stimulation latency respectively and whose nomenclature is based upon the waveform's direction of deflection (Passmore et al. 2014). For example, the N20 peak represents a waveform that results in a negative deflection at 20ms post stimulus. Another convention commonly used would be to label the waveform in the opposite manner; meaning that an upward deflection as negative and a downwards deflection as positive (Cruccu et al. 2008, Hayward et al. 1991, Mauguiere et al. 1999, Nuwer et al. 1994). For the purpose of this thesis, the latter convention will be applied. The latency of the SEP peak represents the transmission time between the point of stimulation and the activity of the neural generator, in milliseconds. There may, however, be slight variations due to characteristics such as, age and height, however, the overall latency title (N20) is used to enable the comparison between waveforms. Both the latency, and SEP peak amplitude values of a given peak can be used to interpret changes in neural activity (Passmore et al. 2014). Therefore, alternations in peak latency and peak amplitude are used to represent alterations in neural transmission and can be used clinically as a way to measure

an experimental condition or test for a potential neuropathy (Mauguiere et al. 1999). The SEP peak components of interest for the purposes of this research are N9, N18, N20, N24, P25 and N30.

1.9.4 SEP peak neural generators

SEP peaks are generally recorded from a number of sites throughout the CNS, however, the specific region of the brain or spinal cord, that generate these potentials is termed the neural generator of that given peak (Valeriani et al. 1998). The flow of ion distribution across cellular membranes of active neural elements give rise to the differences in the amplitude of the each potential and is measured in voltages (Mauguiere et al. 1999). Thus, SEP peak amplitudes are used to measure changes in the activity of these underlying neural generators, with greater peak amplitudes being recorded at sites closest to the neural generator of interest (Mauguiere et al. 1999, Valeriani et al. 1998). The following section will focus on the neural generators of various SEP peaks and the assurance of their location validity

N9

The N9 peak is recorded at Erb's point (also known as the brachial plexus) which is located within the soft triangle of the neck (Nuwer et al. 1994). The electrode placement for this site should be placed in the soft triangle of the neck, which is bordered inferiorly by the clavicle, medial by the sternocleidomastoid, laterally by the trapezes muscle and is future exposed by having an individual rotate their head to the opposite side of interest (Nuwer et al. 1994). The N9 peak represents a near-field potential which is generated by afferent action potentials in the brachial plexus and is strongly influenced by changes in upper limb position (Nuwer et al. 1994). Previous work has demonstrated that The N9 component reflected changes in amplitude in the presence of abnormalities during stimulation of the median nerve (Nakanishi et al. 1983, Synek and Cowan 1982). The results of this study support the theory that the neural generator of this potential is located in the peripheral pathways either close to or in the brachial plexus.

Measuring the amplitude of this peak provides researchers a way to ensure that the afferent volley of information within the sensory nerve is stable between pre and post recording during the analysis process of the signal (Nuwer et al. 1994). IFCN guidelines require that the N9 be within $\pm 20\%$ of each other when comparing pre-post changes in the amplitudes of subsequent upstream neural generators (Nuwer et al. 1994). If the volley of N9 is considered stable, the researchers can have confidence in the subsequent changes in SEP peak amplitudes as being related to a change in the activity of the neural generator.

N18

The N18 SEP peak is recorded from the contralateral frontal cephalic site (Fc') (Rossi et al. 2003) and is a long-duration potentials of post-synaptic activity (Desmedt and Cheron 1981). The neural generator of the N18 peak, is thought to arise in the brain stem, located above the spinal cord, but below the cortex and receives its input from the medial lemniscus which represents a composite waveform with multiple generators (Noël et al. 1996, Sonoo et al. 1991). More specifically, this peak it is thought to reflect alterations in cerebellar activity through changes in sensory input coming from the dorsal column medial lemniscus tract (Nuwer et al. 1994, Sonoo et al. 1991). The lateral aspect of this tract, known as the cuneate nucleus, transmits upper limb sensory information to the cerebellum and plays an important role in SMI by channeling and transmitting various types of somatic information through the nervous system (Kandel et al., 2000). The cerebellum works to coordinate these signals from the spinal cord through its relationship with the accessory inferior olives of the medulla, and its projections to M1; together these pathways work to regulate motor coordination by initiating and regulating motor functions by adapting to somatosensory input during skill acquisition, thereby, enhancing motor learning (Haavik and Murphy 2013, Marshall 1984, Pascual-Leone and Torres 1993). Additional evidence to support the dorsal column nuclei playing an important role in the generation of the N18 peak comes from several cases of patients with pontine lesions, high cervical brain-stem, and thalamic lesions who demonstrated profound disturbances of deep sensation, which is relayed through the DCL (Sonoo et al. 1992, Sonoo et al. 1991). These researchers concluded that the cuneate nucleus was the most probable neural generator of the widespread N18 far field potential. Manzano et al. (1998) also found the dorsal column nuclei important for the generation for N18.

N20

The N20 is a near-field potential which is recorded over the contralateral parietal cortex (Berger and Blum 2007). The neural generators for this peak are thought to arise from thalamocortical projections from the ventral posterior lateral thalamus (Berger and Blum 2007) and is known to reflect the earliest cortical activity in the primary somatosensory cortex (S1) (Desmedt and Cheron 1980, Nuwer et al. 1994).

N24

The N24 peak, which has been previously studied using dipole analysis (Waberski et al. 1999), reflects the activity of the neuronal pathways between the cerebellum and S1, and has been shown to reflect changes in cerebellar output (Restuccia et al. 2006, Rossi et al. 2003)(Restuccia et al. 2001; Rossi Et al. 2003). Using dipole source localization Waberski et al. (1999) localized the source of the N24 SEP component near the posterior wall of the central sulcus (area 3b), close to the location of N20. At low stimulus rates the N24 can often be

difficult to isolate since it typically appears as a notch on the N30's ascending slope. However, increasing the stimulus rate has been shown to selectively decrease the N30 component allowing the individualization of the N24 which has been shown to not attenuate at higher stimulus rates (Fujii et al. 1994, García-Larrea et al. 1992, Haavik-Taylor and Murphy 2007). The N24 has also been shown to be absent in those with cerebellar lesions which indicate that it reflects the pathway between the cerebellum and the primary somatosensory cortex (Restuccia et al. 2006). The cerebellum is involved in control of limb movements, and uses a feedforward internal model to predict the sensory consequences of different planned movements (Manto et al. 2012), thus, the analysis of the N24 peak is significant when trying to measure SMI activity in a clinical population.

N30

The N30 component of somatosensory evoked potentials has been recognized as a crucial index of brain sensorimotor processing and has been increasingly used clinically. The N30 is recorded from the contralateral frontal cephalic (Fc') at the same recording electrodes used to measure the N18 and N24 peak (Desmedt and Cheron 1981, Restuccia et al. 2006, Rossi et al. 2003). This peak originates in the post-central cortical regions and is reflective of the complex subcortical and cortical loop that connects the basal ganglia, thalamus, pre-motor and primary motor cortex (Kaňovský et al. 2003, Rossini 1990). The presence of a N30 generator in the motor and premotor cortex is supported by a large set of evidences coming from experimental (Chéron and Borenstein 1987, Desmedt and Cheron 1980, Taylor and Murphy 2008) and clinical studies (Mauguière and Desmedt 1991, Mauguière et al. 1983, Rossini 1990, Slimp et al. 1986). More recently, an EEG source localization (swLORETA) method demonstrated that the frontal N30 SEP peak was localized in the overlapping areas in M1, specifically, the motor cortex, and extending to the pre-motor and prefrontal cortices (Cebolla et al. 2011). Therefore, the N30 SEP peak is considered a marker of sensorimotor processing and is representative of multiple neural generators (Mauguière et al. 1983, Rossini 1990, Waberski et al. 1999). The connection between the basal ganglia relay loops and the N30 SEP peak are largely supported by anatomical studies which have demonstrated that the area of the SMA related to the arm received its neural input from projections of the ventrolateral thalamus, which is known to be the termination site for efferent information coming from the basal ganglia (Pierantozzi et al. 1999, Pierantozzi et al. 2000, Rossini et al. 1987, Schell and Strick 1984, Wiesendanger and Wiesendanger 1985). Conversely, the N30 peak amplitude has also been shown to decrease in Parkinson's Disease patients (Pierantozzi et al. 1999, Pierantozzi et al. 2000). Taken together, these studies demonstrate that the basal ganglia

may be responsible for both altering SMA activity as well as the changes observed in the N30 peak.

1.10 SUMMARY

Activities of daily life often require the coordination of both gross and fine motor skills, which are possible through the adaptive nature of our sensorimotor system (Riemann and Lephart 2002). The sensorimotor system is a highly structured and complex network which involves the stimulation of afferent pathways from external sources (Kandel et al. 2000, Riemann et al. 2002). The summation and integration of subsequent action potentials into the CNS, and the excitation of appropriate efferent pathways resulting in the execution of a motor command (Kandel et al. 2000, Riemann et al. 2002). FHP is adopted gradually as a consequence of adaptive or compensatory cervical adjustments in response to the repetitive and long-term exposure to the postures commonly associated with computer and cellphone use (Neupane et al. 2017). In this adaptive state, individuals with FHP are not getting the correct sensory information from the neck, when then is likely to impact the body schema of the upper limb relative to the neck. As a result, sensory information coming from the peripheral system, which has gradually become adjusted, is interpreted by the cerebellum as being correct; especially if this posture is repeated in excess, over time (Baarbé et al. 2018, Caneiro et al. 2010, Manto et al. 2012, Seghers et al. 2003, Villanueva et al. 1997), resulting in altered upper limb motor performance (Falla et al. 2004, Zabihhosseinian et al. 2015).

The CNS uses the position of the head and neck when interpreting the position of the upper limb and altered input from neck muscles may affect the sensory inputs to the CNS, and consequently may impair the awareness of upper limb joint position. It is generally believed that FHP is a contributor to the development of chronic neck, shoulder, and even jaw pain (Haughie et al. 1995); however, the impact of FHP on upper limb kinematics and subsequent motor control is not known. In the absence of joint stability, compensatory mechanisms most likely arise from the dynamic restraints of the joint, as well as motor adaptations at proximal and distal segments, therefore, it is possible that FHP is enough to alter sensory information from the neck subsequently impacting the accuracy of limb performance via the impact of the altered input on the postural body schema.

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Chapter 2. PROPOSED RESEARCH

The proposed research framework will investigate differences in the relationship between sensorimotor integration and proprioception between healthy (Non_FHP) and forward head posture (FHP) humans by running two separate, but related studies.

The first study aims to examine how FHP influences proprioception by comparing joint positioning errors of the neck and shoulder between individuals with and without FHP.

The second study utilizes somatosensory evoked potential techniques to investigate patterns of brain activity related to sensorimotor integration (SMI) before and after a motor tracing task.

These studies investigate the possibility of FHP subsequently impacting motor performance of the upper limb by altering the sensory information to the neck. The proposed research framework incorporates motor behaviour and neurophysiological processes to form a comprehensive study on SMI and upper limb performance in the presence of FHP.

CHAPTER 3. MANUSCRIPT 1

Title: The effects of forward head posture on neck and shoulder proprioception

Authors: Patrick, T. A., and Murphy, B. A.

3.1 Introduction

Proprioception is a complex and multifaceted term used to describe an individual's ability to sense and perceive both body position and limb motion in 3-D space (Proske and Gandevia 2012). Proprioception is an essential component of the somatosensory system (Abrahams 1977) and is an important source of feedback for promoting neural plasticity (Daligadu et al. 2013, Haavik-Taylor and Murphy 2007, Legget et al. 2016, Pascual-Leone et al. 2005). During the most basic task such as standing at a desk, maintaining an upright posture against the force of gravity is dependent on the effectiveness of the body's ability to recruit and sustain the correct muscle forces and activation patterns over a given period of time. Therefore, proprioception can be thought of as the body's link to the outside world as it provides ongoing feedback to the central nervous system (CNS) regarding changes to our posture and/or environment (Proske and Gandevia 2012).

From a biomechanical perspective, as the connection between the neck and the head is the atlanto-occipital joint (which can be considered a first-class lever system) (Kapandji 1971), the head is the lever, and the atlanto-occipital joint would be considered the fulcrum, which is placed directly under the center of the skull (lever). The neck muscles would then provide the force to move the lever on the fulcrum (Kapandji 1971). Having the head in this position allows for quick and rapid movements however, this mobility comes at the cost of strength and force production from the surrounding muscles which act directly on the cervical spine (Panjabi 1992).

Proprioceptive acuity involves both peripheral and central processing and is the product of sensory information supplied by specialized nerve endings called mechanoreceptors (Proske and Gandevia 2012). These receptors are located in muscle, joint capsules, tendons, ligaments, and skin and are specific to providing input on joint position and velocity throughout a joint's entire range of motion (ROM) (Proske and Gandevia 2012). During movement, proprioception is important for feedback (reactive control), feedforward (preparatory control) and muscle stiffness regulation in order to achieve specific postures required for efficient muscle balance, coordination and joint stability (Abrahams 1977, Milner et al. 2007, Riemann et al. 2002).

An underactive proprioceptive system has been shown to lead to recruitment failure of important joint stabilizers, which results in a variety of changes including poor endpoint accuracy during movement, (Needle et al. 2013) reduced control during multi-segmental movements, (Sainburg et al. 1995, Sainburg et al. 1993) and an inability to perform repetitive movement sequences (Rothwell et al. 1982). Proprioceptive deficits, left untreated, over time and with repetitive loading, have the ability to increase an individual's chance of developing joint and muscle pain

and/or injury (De Vries et al. 2015). Sensory feedback originating through vision and peripheral mechanoreceptors, are imperative for limb orientation and coordination during movement of the arm (De Vries et al. 2015, Zabihhosseinian et al. 2015). Together these systems work synergistically with the CNS to match visual and kinesthetic input with stored motor patterns as a way to predict future limb positions and movements (Proske and Gandevia 2009).

Cervical proprioception is the sense of position of the head or neck in space, describing the complex interaction between afferent and efferent receptors to monitor head and neck position and movement (Laskowski et al. 2000). In the cervical spine, this sense has its neurological basis in muscle spindles (Proske and Gandevia 2012) and, to a lesser extent, in tendon organs (Golgi receptors) (Hogervorst and Brand 1998), cutaneous receptors, and joint receptors (Grigg 1994, Lephart et al. 1997, McCloskey 1978, Proske et al. 2000). The cervical muscles provide information to (Bolton 1998) and receive information from the central nervous system (Hellström et al. 2005, Prud'Homme and Kalaska 1994). Neck muscles contain many sensory receptors which connect and integrate with sensory information coming from the vestibular and visual system pathways (Bolton 1998).

Afferent information from the cervical muscles converges in the vestibular nuclei, where the head movement-related information from the visual and vestibular system also converges (Corneil et al. 2002). Recently (Malmström et al. 2009), showed that accurate head-on-trunk orientation can be achieved without vestibular information. This suggests that proprioceptive information of the cervical spine is important for head-on-trunk orientation. The complex integration of sensory afferents between these pathways allows us to have unconscious control of our postural muscles as well as conscious control of voluntary movements (Bolton 1998), making head position one of the main factors in the organization of sensory information for upper limb joint position sense (JPS) (Paulus and Brumagne 2008). Therefore, it is likely that prolonged deviations in neck posture would alter neck sensory input to the CNS, possibly leading to faulty upper limb movements overtime.

Forward head posture (FHP) occurs when the head becomes protracted or anterior in relation to the body's center of gravity (Neupane et al. 2017). This maladaptive position is known to contribute to predictable patterns of tissue overload and dysfunction, resulting in a reoccurring cycle of pain and discomfort (Neupane et al. 2017). More specifically, FHP has been shown to contribute to the lengthening and weakness of the anterior cervical flexor muscles with a corresponding shortening of the posterior cervical extensor muscles (Lee et al. 2015, Neupane et al. 2017). Musculoskeletal pain can result from changes in sensory feedback from peripheral receptors when poor posture is a prolonged for an extended period of time or repeated excessively (Bergqvist et al. 1995).

Greenfield et al (1995) reported that participants diagnosed with shoulder overuse injuries demonstrated an increase in forward head translation in comparison with those who had an ideal head positioning. Spinal alignment is also thought to affect scapular position and shoulder girdle function during static and dynamic movements. Faulty cervical spinal alignment such as FHP has been shown to be associated with the shortening of the posterior neck extensor muscles and tightening of the anterior neck muscles, as well as impacting axio-scapular muscles affecting scapular position and kinematics (Kebaetse et al. 1999). To prevent shoulder pain associated with abnormal neck posture researchers have stressed maintenance of a neutral head posture (NHP) during arm movement and functional activity (Edmondston et al. 2007, McLean 2005).

Previous studies have demonstrated that changes in neck position and the presence of neck fatigue in the absence of visual cues affect the processing of incoming sensory inputs and can alter the awareness of elbow JPS (Knox and Hodges 2005, Zabihhosseinian et al. 2015). (Knox and Hodges 2005), demonstrated that spatial position and orientation of upper limb segments were dependent on the position of the head and neck. Furthermore, fatiguing of the neck extensor muscles has been shown to alter the accuracy of elbow JPS during a matching task in healthy participants (Zabihhosseinian et al. 2015), indicating that altered sensory input from the neck subsequent to fatigue may impair upper limb proprioception (Zabihhosseinian et al. 2015).

Since FHP has become ubiquitous in our technology focused society (Neupane et al. 2017) and given the aforementioned impact of altered sensory impact from the neck on upper limb proprioception, it is important to understand if FHP also has the potential to impact both neck and upper limb proprioception. Therefore, the aim of the present study was to further investigate:

1. The effect of FHP on neck proprioception
2. The effect of FHP on glenohumeral (shoulder) joint proprioception

It was hypothesized that individuals with FHP would:

1. Demonstrate differences in cervical JPS accuracy when compared to a Non_FHP control due to the structural relationship of the head with respect to the neck
2. Demonstrate alterations in glenohumeral joint position sense, due to the role of the neck in upper limb motor control, when compared to a Non_FHP control group.

3.2. METHODS

3.2.1 Eligibility

Participants were volunteers recruited from the student population of Ontario Tech University, in Oshawa, Canada. In order to be eligible for the study all participants had to be free of neurological and/or spinal pathology, experience little to no neck pain/discomfort and be right hand dominant. Ethical approval was obtained and the research complied with the Declaration of Helsinki. Participants signed and completed an informed consent and eligibility questionnaires on the day of testing.

3.2.2 Study Overview and Questionnaires

Before the experiment began, all participants completed the Edinburgh Handedness Questionnaire, which assessed which hand was most dominant (right, left, or ambidextrous). The Neck Disability Index questionnaire (NDI) was used as a way to exclude neck pain participants from this study. The NDI is both a valid and reliable tool and has been used extensively in research as a way to assess the impact of neck pain on the performance of activities of daily life (Vernon and Mior 1991). The NDI consists of 10 questions with each having an ordinal scale with six potential response. Each response describes a greater degree of disability, ranging from 0 to 5. Scores are summed to provide a total score (Vernon and Mior 1991). No neck pain was defined as a score between 0-4; mild pain was defined as 5-14; moderate 15-24 and severe as 25-34 (Vernon and Mior 1991). The NDI's total percentage score can be calculated by multiplying the total summed score by two and expressing the result as a percentage (Hains et al., 1998). Since neck pain has been shown to alter proprioception (De Vries et al. 2015) it was decided a priori to exclude any participant who scored an NDI of >10 (20%) during baseline assessment.

Each participant completed a shoulder and neck repositioning task of a previously presented angle. The cervical joint position sense error (JPSE) was assessed by testing the ability of a blindfolded participant to accurately reproduce a previously presented angle (neck rotation to the right and left over the course of 3 trials). The glenohumeral (shoulder) joint JPSE was assessed by testing the ability of a blindfolded participant to accurately replicate a previously presented angle of shoulder internal rotation over the course of 3 trials. Participants who experienced neck pain or discomfort on the day of testing were rescheduled in order avoid pain impacting results. This study was approved by the University of Ontario Institute of Technology Research Ethics Board.

3.2.3 Baseline Measurements

Both FHP and non-FHP participants had the degree of FHP assessed based on an average of three forward head translation (FHT) measurements. FHT was

measured using the C-ROM device, a well-documented instrument for which normal values have been established for head and neck range of motion (Youdas et al. 1992) (Figure 3 C).

Postural measurements were calculated from the anatomical landmarks: a) the tragus of the external ear, b) the acromion process of the scapula and c) the C7 spinous process; as indicated in (Figure 1). FHT is measured by using the attachment arm and measuring the distance of the tragus in relation to the acromion process. C7 is identified as a landmark for the FHT attachment arm which will allow the deviation of the tragus to be accurately measured with respect to the position acromion process. FHT of approximately one inch (2.5 cm) is estimated to increase head carriage by approximately 10 pounds, and FHP as an FHT measurement of ≥ 2 centimeters (Neupane et al. 2017).

3.2.4 Forward Head Translation Assessment

Participants were instructed to sit upright in a straight-back chair and asked to sit with their sacrum and shoulders touching the chair (maintaining the lumbar curve) with their arms hanging by their side and feet flat on the floor. Next, the C-ROM device was placed on the participants head and the participants head adjusted in a position which would allow the sagittal dial on the C-ROM device to read zero degrees; indicating a neutral head position (Figure 1).

Two measurements were used to calculate FHT:

1. Ideal Posture

For this position, the participants head was positioned with the tragus directly over the acromion process. This position was held for 5 seconds and recorded a total of 3 times.

2. Resting Posture

After ideal posture was recorded the participant was asked to relax for 5 seconds. This position was then measured and repeated 3 times.

Forward Head Translation Calculation

FHT (cm) = Resting Position (cm) – Ideal Position (cm)

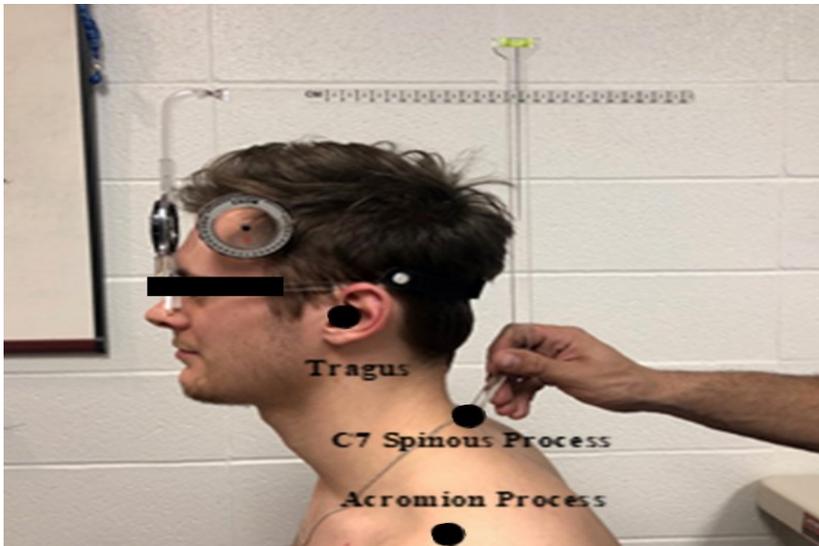


Figure 1. Forward Head Translation Measurement using the C-ROM device

3.2.5 Forward Head Posture Group

Fifteen participants with FHP (6 males and 9 females) aged 21.9 (± 2.9) years were used for this study. Inclusion criteria for the FHP group was an average FHT of ≥ 2 centimeters during their baseline assessment. Participants in the FHP group were allowed to experience mild to moderate neck pain/discomfort during activities of daily life, however, they were to be asymptomatic during the day of testing.

3.2.6 Non-Forward Head Posture Group

Fifteen Non_FHP participants (6 males and 9 females) aged 21.7 (± 3.3) years were used as a control group for this study. Inclusion criteria for the Non_FHP group was an average FHT of < 2 centimeters, in addition to, an NDI score of ≤ 4 (8%) during their baseline assessment.

3.3. EXPERIMENTAL PROTOCOL

3.3.1 Joint Position Sense

Joint position sense (JPS) is an important characteristic of proprioception and is defined as the ability on an individual to recognize the position of a joint in relation to space or other body segments (De Vries et al. 2015, Selfe et al. 2006). Clinically, joint position sense error (JPSE) is associated with joint proprioception and is used to assess the ability of an individual to actively or passively perceive the position of a joint (De Vries et al. 2015, Selfe et al. 2006). JPSE is measured by testing the ability of a blindfolded participant to accurately relocate the position of a joint relative to a predefined target angle (referred to as the neutral position of a joint) (Selfe et al. 2006). The difference between the presented and

reproduced position is the repositioning error (De Vries et al. 2015, Selfe et al. 2006).

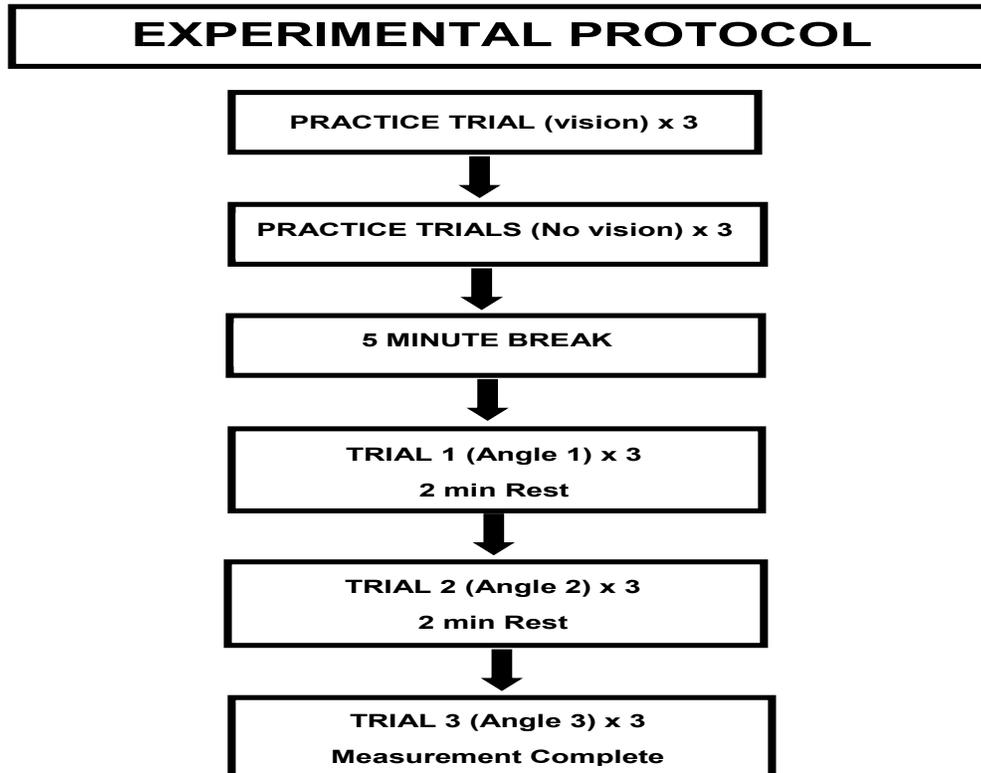


Figure 4. Experimental Protocol. Both cervical and glenohumeral JPSE measurements followed the same procedure.

3.3.2 Shoulder Proprioception Task

A purpose-built device, which assisted in moving the shoulder joint into humeral internal/external rotation, was used to measure JPSE. Glenohumeral rotation was selected for two reasons. The first is that it assesses humeral movement relative to the torso with minimal scapular assistance, minimizing any potential impact of neck muscle fatigue on axio-scapular muscles. Secondly, a number of occupational tasks rely on glenohumeral rotation making it important to understand if FHP has the potential to impact the accuracy of glenohumeral JPS. The device was adjusted to a height which allowed the olecranon process of the right ulna to be in contact with the rotating arm of the device. This placed both the elbow and shoulder joint into approximately 90° of shoulder and elbow flexion as well as scapular abduction (Figure 2A). Once the arm was in this position the participant was asked to adjust their torso location (stepping forward or backwards) in order to create a position which felt the most natural during shoulder internal/external rotation. Once the starting position was established, each participant completed 2 practice trails before starting the task: 1) a practice

trial with vision (visual feedback) and, 2) a warm-up trial without vision (specific to the task). During the task each participant was asked to reproduce each target angle over a series of three attempts. (figure 2B). The target angle was held for 3 seconds and moved back to neutral. In order to eliminate any predictable cues generated by soft tissue stretch, movement time of cervical rotation and repetition of movement, the task was performed in the mid-range of the joints ROM. The target angle for cervical rotation was set between 40-50° and each target angle was selected using an online randomized number generator program (used through google search engine). In order to minimize the development of fatigue, a minute rest break was given between all trials. An overview of the experimental protocol is listed in *Figure 4*



Figure 2. Shoulder JPS matching task. (A) Starting position (neutral). (B) Shoulder positioned at randomized angle.

3.3.3 Neck Proprioception

Neck JPSE was measured using the C-ROM device, a reliable and valid tool, which is able to measure cervical ROM throughout various planes of movement (Audette et al. 2010) (See figure A,B,C). The measurement began with the

participant seated upright in a back supported chair with their hands by their sides and their feet flat on the floor. Starting from a neutral position (Figure 3A), the head was rotated to a randomized angle which was also repeated on the opposite side. Similar to the shoulder, each participant completed 2 practice trials before starting the task: 1) a practice trial with vision (visual feedback) and, 2) a warm-up trial without vision (specific to the task). During the task each participant was asked to reproduce each target angle over a series of 3 attempts. (Figure 3B). The target angle was held for 3 seconds and moved back to neutral. In order to eliminate any predictable cues generated by soft tissue stretch, movement time of cervical rotation and repetition of movement, the task was performed in the mid-range of the joints ROM. The target angle for cervical rotation was set between 20-50° and each target angle was selected using an online randomized number generator program (used through google search engine). In order to minimize the development of fatigue, a minute rest break was given between all trials. An overview of the experimental protocol is listed in *Figure 4*

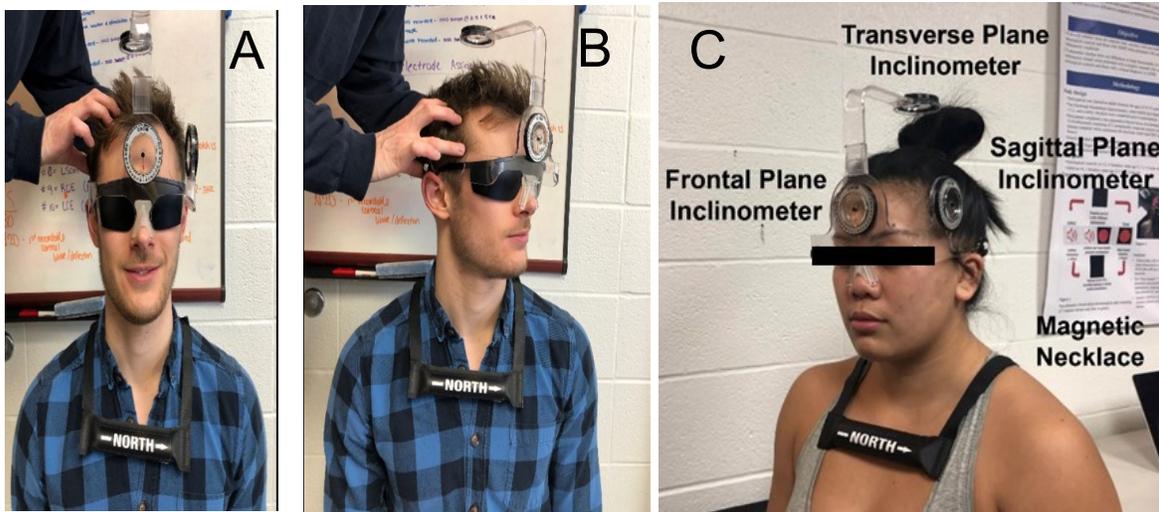


Figure 3. Neck JPS matching task. (A) Starting Position (Neutral) (B) Head rotated to a randomized angle (C) Reference

3.4 DATA ANALYSIS

All measurements were averaged using Microsoft Excel.

3.4.1 Absolute, Constant and Variable Error

The accuracy of the angle reproduction was measured using absolute, constant and variable errors (Knox and Hodges 2005). Absolute error was defined as the overall deviation between a presented angle and target angle without considering the direction of error (Knox and Hodges, 2005). Constant error was defined as

the deviation between the presented and reproduced angle, with respect to the direction of the error (Knox and Hodges, 2005). Variable error was defined as a measure of the consistency between trials and is the standard deviation (SD) of the mean constant error (Knox and Hodges, 2005). The average of three trials for each calculation was used to measure the overall JPSE. Absolute, constant and variable error were calculated using Eqs. 1, 2 and 3 (Abramowitz and Stegun, 2012).

$$\text{Absolute error : } \sum \left| \frac{[X - X_0]}{N} \right| \quad (1)$$

$$\text{Variable error : } \sqrt{\frac{\sum (X_0 - M)^2}{N}} \quad (2)$$

$$\text{Constant error : } \sum \frac{[X - X_0]}{N} \quad (3)$$

3.4.2 Statistical Analysis

Univariate analysis of variance (ANOVA) was performed for the analysis of neck and shoulder JPSE between Non_FHP and FHP participants using SPSS (SPSS v.25, IBM Corporation, Armonk, NY, USA). Statistical significance was set at $p \leq 0.05$ for all analysis. All data was tested for normality (Shapiro–Wilk’s test), homoscedasticity of variance (Levene’s test of equality), and sphericity (Mauchly’s test), where applicable. All numeric values are expressed as mean \pm standard deviation (SD). Effect sizes were classified as small (0.01), moderate (0.06), and large (>0.14) (Richardson 2011). Moderate and large effect sizes were considered indicators of statistically meaningful results (Richardson 2011). In addition, a paired-sample t test was used to evaluate whether there were differences between the right and left sides during neck rotation. Outliers were calculated by SPSS as ± 1.5 times the interquartile range between the 25th and 75th percentiles removed from analysis.

3.5. RESULTS

3.5.1 Neck Joint Position Sense Error

The assumptions of normality were met for both groups. There were no significant differences between the JPSE of the right and left side of the neck for both the Non_FHP group ($p= 0.48$) and the FHP group ($p=0.52$). Based on these results, both the left and right side were pooled, averaged and used for the overall measurement of cervical JPSE.

Absolute Error

For absolute error there was a significant effect of GROUP ($F_{1,29} = 40.64$, $p = 0.001$; $\eta^2 = 0.610$) and SEX ($F_{1,29} = 8.143$, $p = 0.008$; $\eta^2 = 0.239$). The mean JPSE for the Non_FHP group was $1.38 \pm 0.84^\circ$ and $3.71 \pm 1.28^\circ$ for the FHP group. In addition, JPSE for the Non_FHP group had a mean error of $0.76 \pm 0.39^\circ$ and $1.8 \pm 0.8^\circ$ for males and females respectively, whereas for the FHP group the mean JPSE was $3.09 \pm 0.73^\circ$ and $4.12 \pm 1.43^\circ$ for males and females respectively.

There was no GROUP by SEX interaction ($p = 0.993$).

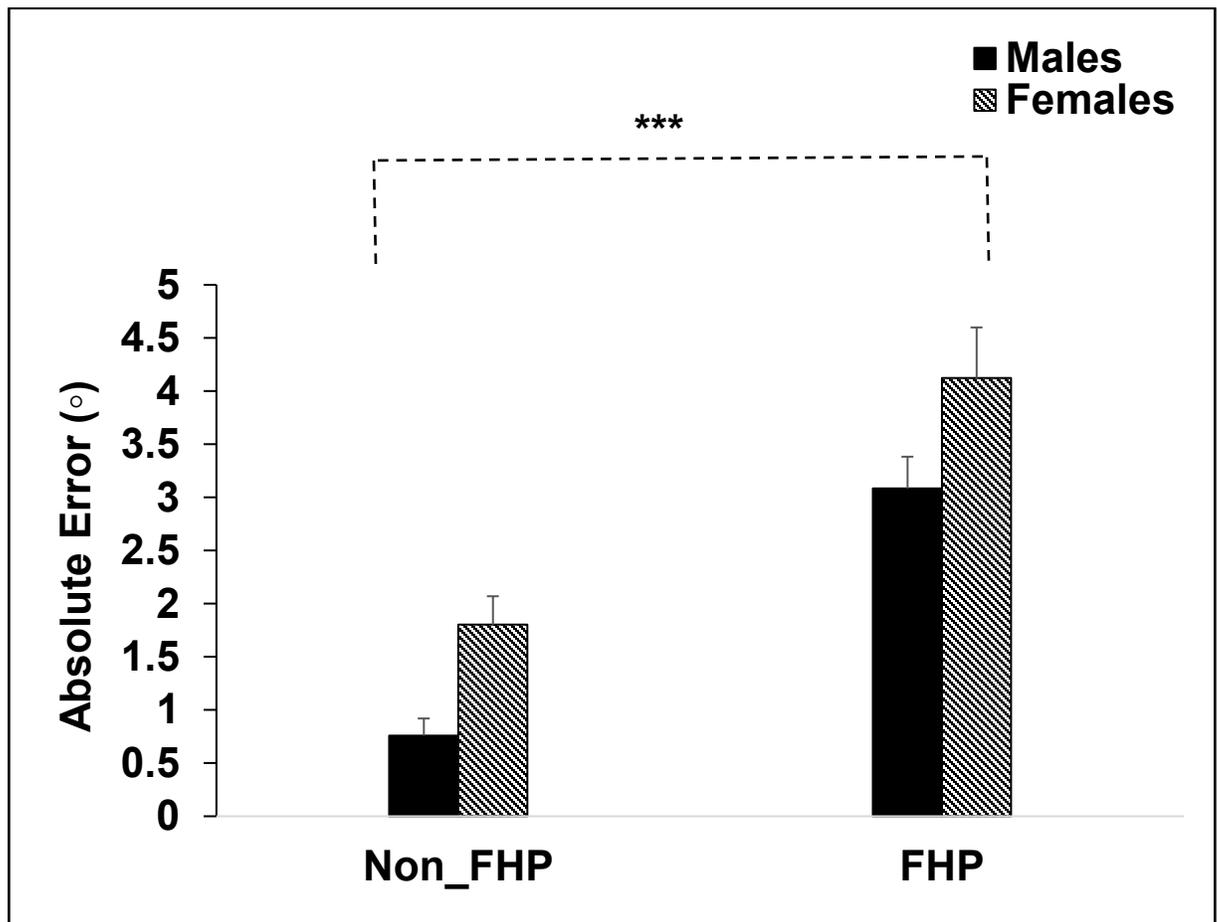


Figure 5. Absolute joint position errors for the neck. Error bars represent the standard error of the mean.

Constant Error

No significant main effect or meaningful effect size was found for GROUP ($p=0.391$), or SEX ($p=0.611$), nor was there a GROUP by SEX interaction ($p=0.538$).

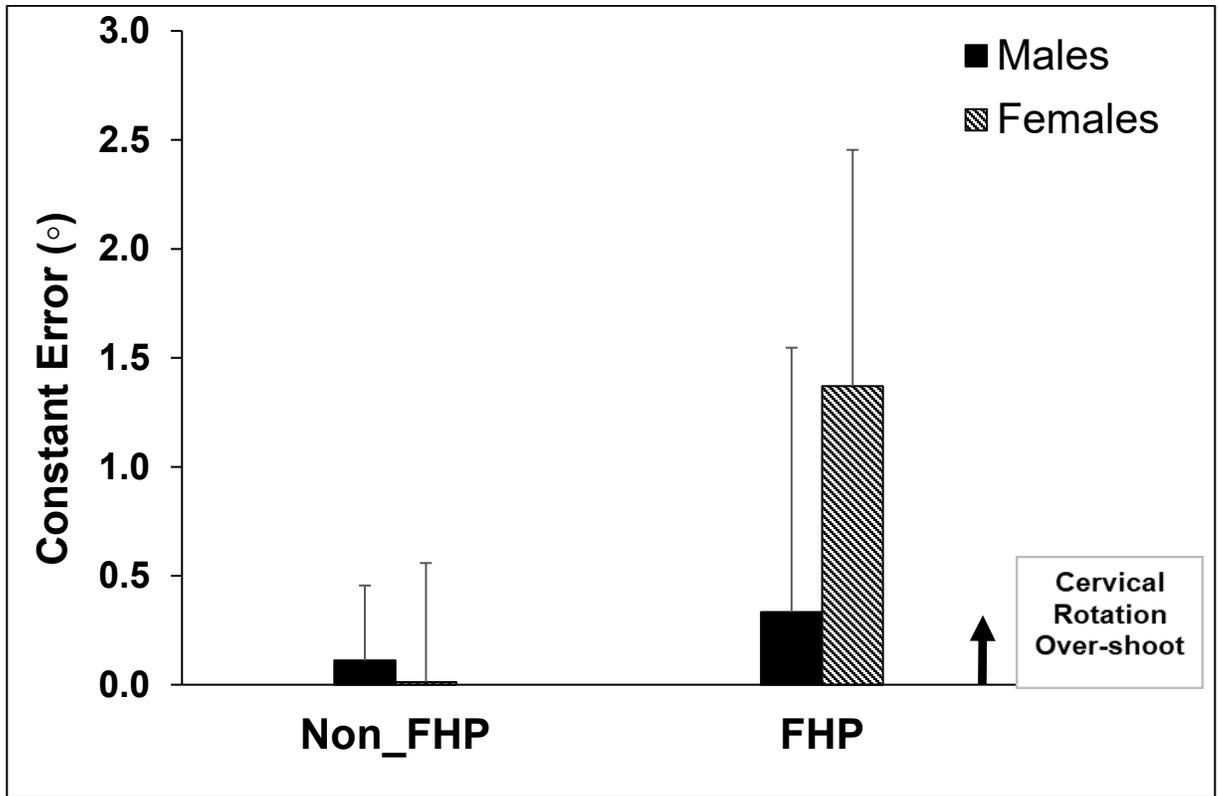


Figure 6. Constant joint position errors for the neck. Error bars represent the standard error of the mean.

Variable Error

For variable error there was a significant effect of GROUP ($F_{1,29}= 18.234$, $p=0.001$; $\eta^2= 0.412$) and SEX ($F_{1, 29}= 4.68$, $p=0.04$; $\eta^2=0.153$). That is, the mean JPSE for the Non_FHP group was $1.01 \pm 0.48^\circ$ and $2.17 \pm 0.98^\circ$ for the FHP group. In addition, JPSE for the Non_FHP group had a mean error of $0.6 \pm .24^\circ$ and $1.28 \pm 0.41^\circ$ for the males and females respectively. For the FHP group, the mean JPSE was $1.86 \pm 0.56^\circ$ and $2.38 \pm 1.17^\circ$ for the males and females respectively.

There was no GROUP by SEX interaction ($p=0.771$)

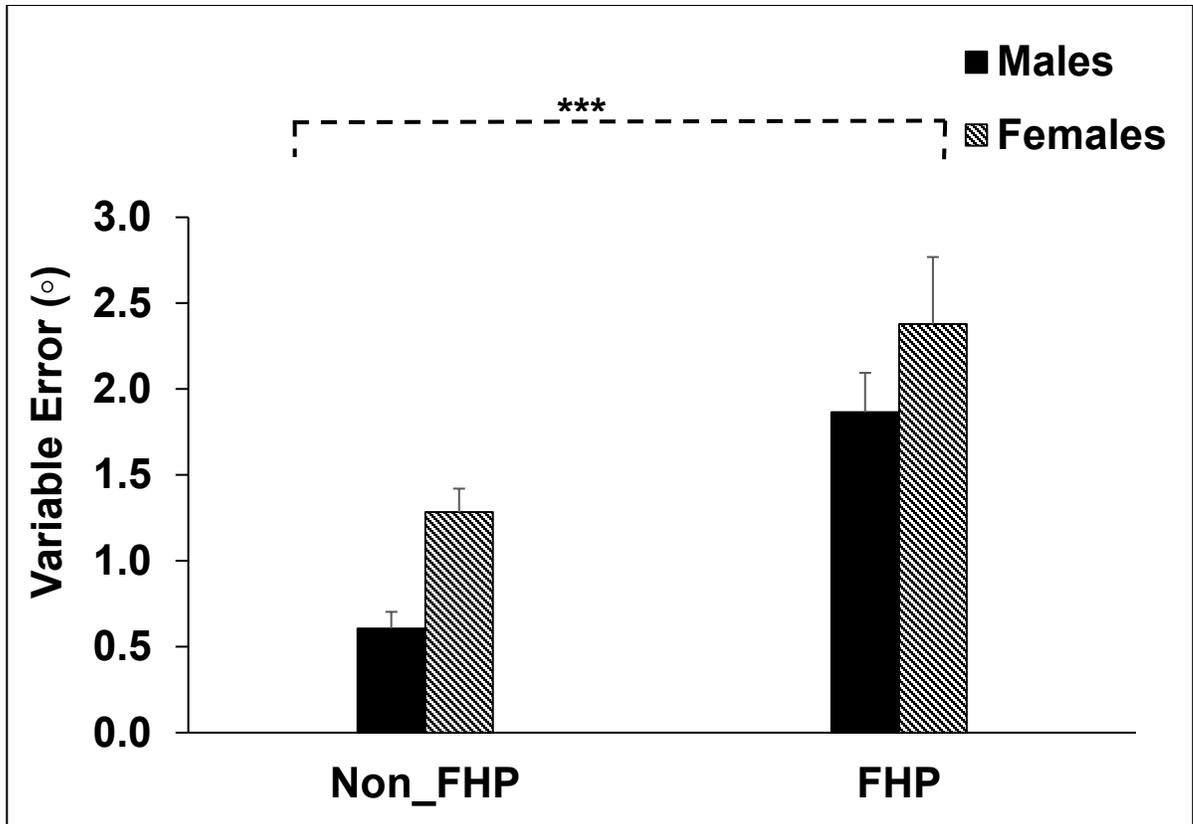


Figure 7. Variable joint position errors for the neck. Error bars represent the standard error of the mean. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$, and **** $p < 0.0001$

3.5.2 Shoulder Joint Position Sense Error

Absolute Error

For absolute error there was a significant effect of GROUP ($F_{1,29} = 6.54$, $p = 0.017$; $\eta^2 = 0.201$). That is, the mean JPSE for the non_FHP group was $3.95 \pm 1.15^\circ$ and $5.32 \pm 1.62^\circ$ for the FHP group.

No significant main effect or meaningful effect size was found for SEX ($p = 0.469$) or SEX by GROUP interaction ($p = 0.972$).

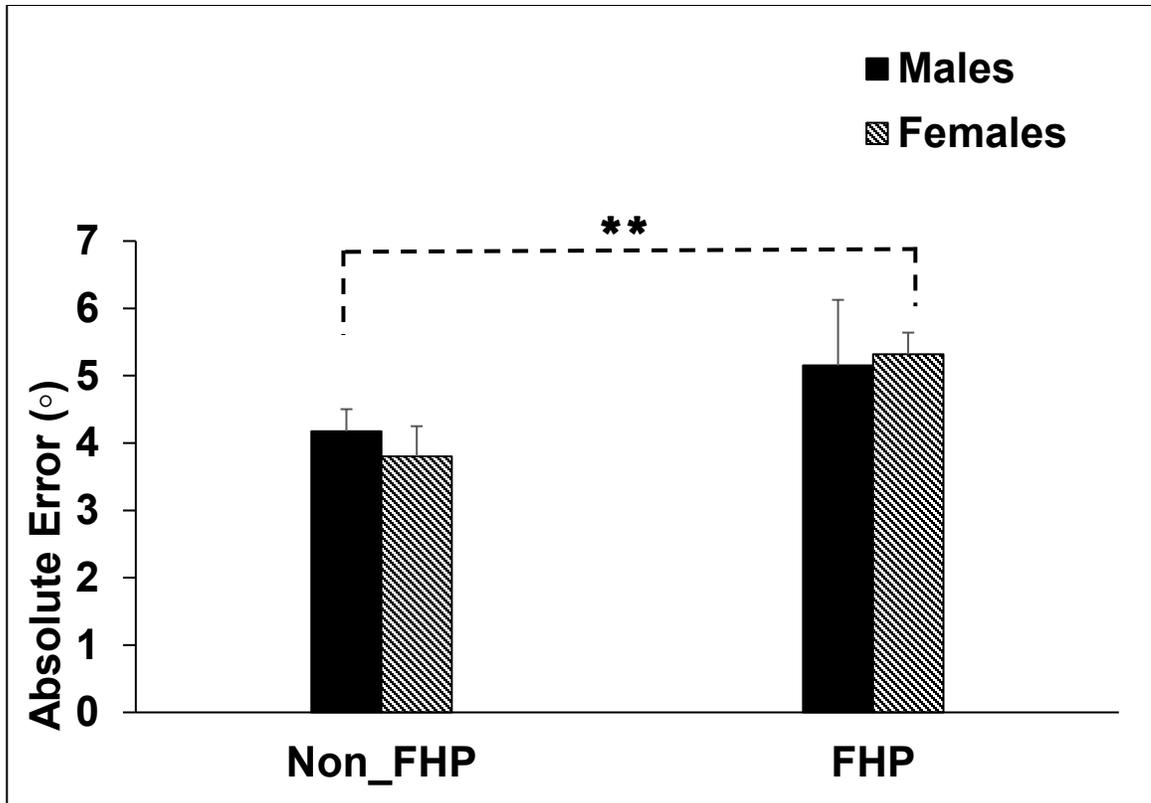


Figure 8. Absolute joint position errors for the shoulder. Error bars represent the standard error of the mean. * $p < 0.05$, ** $p < 0.01$

Constant Error

For constant error there was non-significant GROUP effect with a medium effect size ($F_{1,29} = 3.85$, $p = 0.06$; $\eta^2 = 0.129$) however, there was a significant GROUP by SEX interaction ($F_{1,29} = 5.15$, $p = 0.032$; $\eta^2 = 0.165$). That is, the mean JPSE for the Non_FHP group was $0.67 \pm 0.29^\circ$ and $2.41 \pm 3.73^\circ$ for the FHP group. More specifically, the JPSE for the Non_FHP group had a mean error of $-0.02 \pm 3.18^\circ$ and $1.14 \pm 2.79^\circ$ for the males and females respectively. For the FHP group, the mean JPSE was $4.86 \pm 3.68^\circ$ and $0.79 \pm 2.91^\circ$ for the males and females respectively. No significant main effect or meaningful effect size was found for SEX ($p = 0.218$)

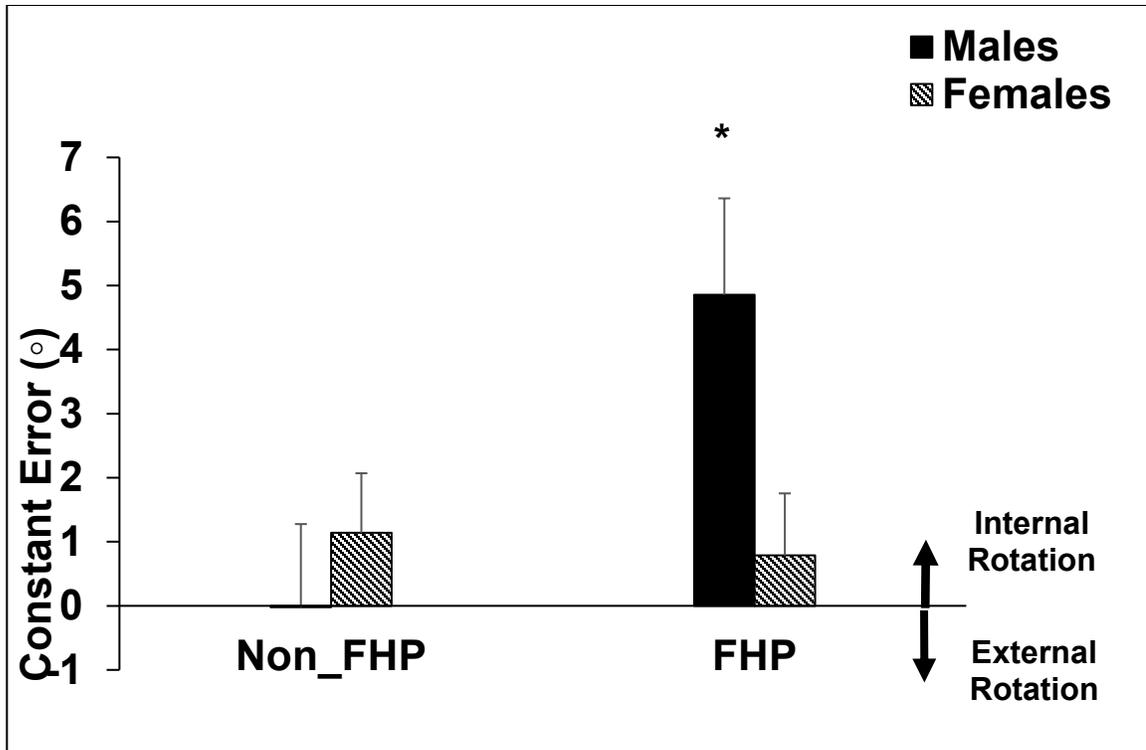


Figure 9. Constant joint position errors for the shoulder. Error bars represent the standard error of the mean. * $p < 0.05$

Variable Error

For variable error there was a significant GROUP effect ($F_{1,29} = 4.227$, $p = 0.05$; $\eta^2 = 0.140$). The mean JPSE for the Non_FHP group was $2.92 \pm 1.09^\circ$ as compared to $3.97 \pm 1.38^\circ$ for the FHP group. No significant main effect or meaningful effect size was found for SEX ($p = 0.166$) or SEX by GROUP interaction ($p = 0.143$).

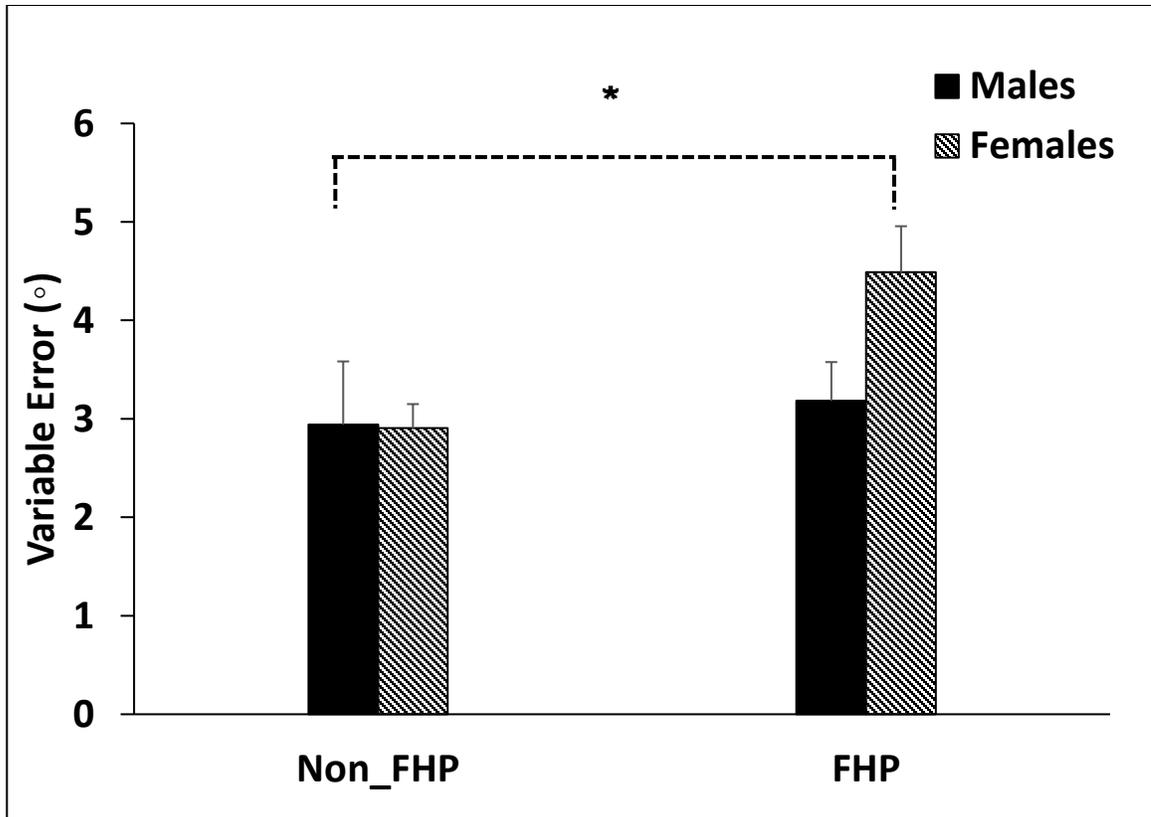


Figure 10. Variable joint position errors for the shoulder. Error bars represent the standard error of the mean. * $p < 0.05$

3.6 DISCUSSION

This study sought to investigate the effect of FHP on cervical spine and glenohumeral joint (shoulder) proprioception in young adults by measuring the accuracy of their JPS. The results of this study demonstrated significant differences in the absolute, constant and variable error for cervical JPS and significant differences in the absolute and variable error for shoulder JPS, indicating that FHP can decrease the accuracy of both neck and the upper limb JPS.

3.6.1 Neck Joint Position Sense

This work is in agreement with previous studies showing that neck fatigue impacted elbow JPS (Zabihhosseinian et al. 2015) and performance of a shoulder aiming task which relied on proprioception (Zabihhosseinian et al. 2019). The changes in the neck fatigue group were suggested to be due to the impact of fatigue on sensory input from the neck muscles to the CNS. Fatigue has been shown to alter the firing rate of 1A afferents during sustained submaximal contractions (Macefield et al. 1993), indicating that fatigue-induced excitation of slowly conducting muscle afferents is significant in mediating the

fatigue-induced inhibition of motoneuron output (Gandevia 2001, Hayward et al. 1991, Taylor et al. 1996). FHP sits at the intersection of fatigue and SCNP. It has the chronic changes in sensory input from the neck that would occur in SCNP, but may have similar changes to the acute changes induced by the neck fatigue studies. It is interesting that it shows similar findings to both works. Several studies in recent decades have examined the relationship between proprioceptive deficits in non-specific (acute) and chronic neck pain however, there is generally no consensus on the relationship between the two. In an upright posture the primary function of the cervical spine is to orient the head against the opposing forces of gravity (Grigg 1994, Panjabi 1992, Singla and Veqar 2017) and, is considered to be the most mobile section of the vertebral column (Panjabi 1992, Singla and Veqar 2017). The muscles which attach directly to the cervical vertebra play an important role in the alignment and position of the head during multi-directional movement (Panjabi 1992, Singla and Veqar 2017) by providing stability and support to the neck (Abu El Kasem et al. 2017, Côté et al. 2004, Panjabi 1992). In order to achieve efficient static and dynamic movement each segment of the cervical spine should be properly aligned and mechanically stable in order to reduce the impact of gravitational forces (Panjabi 1992, Singla and Veqar 2017, Strimpakos 2011).

Cervical spine posture is considered to have a significant influence on the perception of upper limb position due to the reliance of CNS on the position of the head as a way to conceptualize the relative position of each body segment (Cohen 1961, Paulus and Brumagne 2008, Strimpakos 2011). Neck muscles have numerous sensory receptors that are responsible for postural control systems during voluntary muscle contraction (Jull et al. 2007). During upper limb movement, kinesthetic and visual inputs are constantly matched against the brain's internal map or "schema" of the body as a way to predict the future position of the limb (Grigg 1994). In the absence of visual feedback muscle spindles are responsible for limb proprioception (Proske and Gandevia 2009) therefore, is it plausible to assume that changes in the posture of the cervical spine have the ability to subsequently adjust the way internal and external sensory information is processed, leading to inaccuracy during movements of the upper limb.

In the current study, the FHP group had worse absolute and variable error for neck JPS (i.e. individuals with FHP not only had a higher JPSE for the neck when compared to the non_FHP group they were also more variable). Several studies have demonstrated the effect that cervical proprioceptors have on head orientation and cervical posture (Karlberg et al. 1995, LETAFATKAR et al. 2009, Pyykkö et al. 1989, Revel et al. 1994) therefore, it is possible that the JPSE observed between groups in this study could be related to altered input from neck muscles consequently impacting the accuracy of subsequent neck and limb movement during the task performance.

The results of this study also revealed that regardless of the group women, on average, produced significantly more error in neck JPS when compared to men. Several studies have shown that women exhibit lower neck muscle strength when compared to men (Chiu and Lo 2002, Jordan et al. 1999, Staudte and Dühr 1994, Ylinen and Ruuska 1994) which could contribute to these sex differences. Several studies have shown that greater muscular strength was associated with improved balance and control at the knee (Keays et al. 2003, Park et al. 2010, Wang et al. 2016). Furthermore, a strength training study conducted by (Salles et al. 2015), found to improve JPS at the shoulder, possibly due to the ability of the strength program to restore muscle balance between reciprocal muscles (Swanik et al. 2002). Since muscle spindles have been shown to be the primary proprioceptors involved in JPS (Proske 2005) and are also involved in the generation of muscle tension (Brooks et al. 2013, Luu et al. 2011, Savage et al. 2015), it is possible that there may be a relationship between JPSE and muscular strength.

Individuals with NP have also been reported to have larger errors in the JPS of the head and upper limb during movement performance, showing less accurate position sense of their head and upper limbs (Haavik and Murphy 2011, Lee et al. 2005, Stanton et al. 2016). It was suggested that the neck pain altered the proprioceptive input being sent to the CNS (Paulus and Brumagne 2008), subsequently effecting task performance. Taken together, these findings support our hypothesis that FHP would be sufficient enough to alter the body's ability to orient and monitor the position of the head in the presence of FHP.

3.6.2 Shoulder JPSE

To our knowledge, this is the first study to demonstrate that the altered neck position associated with FHP is able to impact upper limb proprioception likely due to altered afferent input from the neck. The results of the current study found significant main effects for absolute and variable error, suggesting that the FHP group had larger and more variable shoulder JPSE when compared to the Non_FHP group. Furthermore, a significant group by sex interaction was observed for males in the FHP group for constant error, indicating that males were more likely to overshoot the indicated target during task performance. These results support hypothesis two, which suggested that differences would be seen in upper limb JPS as a result of the altered afferent input from the neck due to FHP.

The relationship between alterations in neck posture and subsequent effects on motor control of the neck and upper limb are an important topic due to the increases in global cellphone use (Neupane et al. 2017). Although FHP is commonly observed in patients with neck pain and dysfunction, and is frequently reported in studies related to NP (Audette et al. 2010), the strength of the relationship between FHP and cervical discomfort is still a topic of discussion.

Studies of individuals who lack proprioceptive sense (due mainly to large fiber neuropathy) highlight the importance of proprioceptive feedback during movement. Deficits observed included poor endpoint accuracy (Messier et al. 2003), reduced control of multi-segmental dynamics (Sainburg et al. 1995, Sainburg et al. 1993), and an inability to perform extended movement sequences (Rothwell et al. 1982). Participants in both groups had either no pain or minimal pain based on the inclusion criteria suggesting that the postural changes of FHP are sufficient to impact the accuracy of both head and upper limb JPS.

Gandevia & Burke (1992), originally proposed that the repositioning or relocation of a joint after active positions or motions may rely more on central motor programs rather than a memory of proprioceptive coordinates. The findings of this study support this theory. The decrease in the shoulder JPS accuracy observed in the FHP group could be related to a change in the activation patterns of muscles afferents coming from the neck. The habitual slumped position that is adopted during prolonged sitting tasks has been documented to lead to the development of FHP, affecting the kinematics of the cervical spine (White and Sahrman 1994). Muscle fatigue has been shown to increase the spontaneous discharge in muscle afferents thereby sensitizing their response to muscle stretch and movement (Hayward et al. 1991). Neck pain is associated with impaired sensorimotor function which includes alterations in shoulder kinematics (Zabihhosseinian et al. 2017), impaired upper limb proprioception (Zabihhosseinian et al. 2017), and reduced cervical range of motion (Sjölander et al. 2008). More recently, Zabihhosseinian et al (2019), has demonstrated the effects of neck muscle fatigue on eye hand coordination and upper limb JPS (Zabihhosseinian et al. 2015). Taken together with the results of the current study, the evidence suggests that altered sensory input from the neck, whether due to altered posture, pain or fatigue, appears to impair upper limb proprioception.

3.6.3 Limitations

There were some limitations of the study which need to be acknowledged. First, the shoulder JPS matching task was a conscious exercise. Proprioception is usually an unconscious sensory process, and so, should be measured in a way that prevents an individual from consciously perceiving where their limb is moving. A second limitation was that we did not control for height differences between subjects as our main focus was to assess preliminary differences between groups. Some of the taller participants tended to have larger errors, and future work should consider height matching between groups. Lastly, another limitation could be the participation of athletes in the study. Individuals with more movement skill may have better proprioception due to their bodies ability to recalibrate their movements faster (Muaidi et al. 2009). Since participants were recruited through campus it is possible that some participants could have been

high-performance athletes, and future studies should determine athletic experience in advance and match for it between groups.

3.7 CONCLUSION

This study found that the presence of FHP lead to significant increases in joint position error for both the neck and glenohumeral joint. Future studies should measure whether these individuals show EMG changes indicative of fatigue relative to non-FHP individuals, as well as assessing performance on upper limb visuomotor hand-eye tracking tasks to understand if these proprioceptive deficits impact upper limb performance.

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CHAPTER 4. MANUSCRIPT 2

Title: The effects of forward head posture on sensorimotor integration

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4.1 INTRODUCTION

Forward head posture (FHP) is commonly seen as a result of excess cell phone and technology use (Chiu et al. 2005, Neupane et al. 2017). The orientation of the head with respect to the neck plays an integral role in human movement and performance (Knox and Hodges 2005, Malmström et al. 2009). Head orientation is fundamental for accurately integrating visual, vestibular, somatosensory and auditory inputs via interconnected pathways in the brain and spinal cord, as we associate, process and respond to our ever-changing environment (Baumann et al. 2015, Mousavi-Khatir et al. 2018). FHP occurs when the head is positioned anteriorly in relation to the body's center of gravity (Neupane et al. 2017) and is known to contribute to predictable patterns of tissue overload and dysfunction, resulting in a reoccurring cycle of pain and discomfort (Cohen 1961, De Vries et al. 2015, Hansraj 2014, Neupane et al. 2017). FHP has been shown to lead to the lengthening and weakness of the anterior cervical muscles with a corresponding shortening of the posterior cervical muscles (Lee et al. 2015). These muscles, which attach directly to the cervical vertebra, play an important role in the alignment and position of the cervical spine during multi-directional movement (Chiu et al. 2005) which in turn, provide stability and support to the head (Chiu et al. 2005).

In 2017, texting statistics significantly increased with the average number of monthly text messages sent in the USA being close to 780 billion (Brain 2013). Our everyday lives have become dominated by computer technology and handheld mobile devices making FHP (or now socially know as text-neck) a growing, world-wide, health concern (Neupane et al. 2017). While studies investigating the effects of technology-induced postures on the musculoskeletal system are starting to emerge (Neupane et al. 2017), the long-term effect on sensorimotor processing and neuroplasticity remains unclear. Currently, available research is starting to identify the significance of investigating this phenomenon as it could enhance our understanding of how long-term sensory changes in sensory input from the neck contribute to altered sensorimotor integration and possible errors in motor performance.

The brain uses a series of excitatory and inhibitory activation patterns (feedback loops) to improve the accuracy and control of movements and will repeat this process until otherwise instructed (Myers and Lephart 2000, Proske et al. 2000). Collectively, these pathways and structures work to influence and create a postural model known as body schema, which is defined as the presence and awareness of one's physical occupancy in relation to their environment. This neural map of the body is used to generate accurate and coordinated voluntary movement based on previous experience and previously stored synchronization of muscle recruitment patterns (Doyon et al. 2003). Motor control refers to the planning and execution of movements whereas motor learning refers to the

increasing spatial and temporal accuracy of movements with practice. Although considerable progress has been made in understanding the neural basis of motor control (Bizzi et al. 1995, Georgopoulos et al. 1981), little is known about the impact of chronic postural changes in sensory input from the neck and its impact on upper limb motor skill learning. Deficits in motor control of the spine can lead to poor control of joint movement leading to the presentation of repeated microtrauma which can eventually to pain (Kapandji 1971, Panjabi 1992). For example, increased neural activity to the muscles of the neck muscles (upper trapezius, the levator scapulae muscles and sternocleidomastoid) due to a poor working posture of the neck or of the arms may, over time, increase the compressive load on cervical segments and initiate a painful neck condition. Adopting a sustained, non-neutral spinal posture has been shown to lead to the increased activation of important head and neck stabilizers making these muscles susceptible to the development of fatigue (Falla et al. 2004). It has been hypothesized that changes commonly seen in muscle recruitment patterns are developed as a protective mechanism to underlying spinal instability resulting from laxity or damage to the structural tissue of the head and neck, muscle dysfunction, or reduced neuromuscular control (Kwon et al. 2015, Shin et al. 2017). There is also evidence in the literature to suggest that muscle impairment occurs early in the history of onset spinal complaints (Kwon et al. 2015) and that such muscle impairment does not automatically resolve even when pain symptoms improve (Jull et al. 2008, Kwon et al. 2015).

Sensorimotor integration (SMI) refers to the ability of the brain to integrate sensory (afferent) from the periphery and motor (efferent) information when generating task-specific motor outputs (Lephart et al. 1997, Myers and Lephart 2000). Sensory input is filtered by the central nervous system (CNS) according to its destination and entry point in the spinal cord and is processed by higher cortical structures which are involved in maintaining joint stability and muscle coordination during movement (Lephart et al. 1997, Myers and Lephart 2000). The sensorimotor relay is broken down into groupings of related ascending and descending pathways which begin and end at the cellular level and contribute to subconscious postural control and conscious voluntary movement (Lephart et al. 1997, Myers and Lephart 2000). Short latency, somatosensory evoked potentials (SEPs) (<30 milliseconds) are elicited in response to stimulation of peripheral nerves or their receptors, and provide a way to non-invasively assesses SMI as properties of both the peripheral and central nervous system can be recorded at various anatomical locations where afferent projections of the stimulated nerves terminate (Rapuno 2009).

The goal of motor skill acquisition and learning is to enhance the performance of a given task with as little conscious awareness as possible; an example of this would be the maintenance of posture (Gandevia et al. 1990, Gardner 1953). These changes are known to develop slowly overtime requiring repeated

exposure to a stimulus (Gandevia et al. 1990, Gardner 1953). The long-term adaptations which result are better known as neuroplasticity which describes the ability of the cerebral cortex to alter and restructure its neural connections based on an experience or in response to an environmental demand (Buonomano and Merzenich 1998). The development of new synaptic connections is known to be adaptive (beneficial to the organism such as learning a new motor skill) or maladaptive (harmful to the organism, i.e. resulting in poor movement patterns and/or overuse injuries). This plasticity occurs as a result of a reoccurring stimulus or motor-skill training in the form of planned practice (Boudreau et al. 2007, Boudreau et al. 2010, Classen et al. 1998, Hluštík et al. 2004, Kleim et al. 2003, Koeneke et al. 2006, Perez et al. 2004, Solodkin et al. 2004, Ungerleider et al. 2002). Somatosensory Evoked Potentials (SEPs) have been used in previous research to measure changes and alterations in SMI in various experimental paradigms involving motor acquisition tasks (Andrew et al. 2018, Baarbé et al. 2013, Dancey et al. 2016) as a way to measure neural plasticity in response to motor skill learning which can be defined as the ability to increase spatial and temporal accuracy of movements with practice (Willingham 1998).

SEP peak amplitudes related to the cerebellum have been observed to change following motor-skill training (Andrew et al. 2018, Dancey et al. 2016). Individuals with cerebellar lesions demonstrate a decreased ability to control and coordinate voluntary movements in addition to alterations in joint reflexes (Bloedel and Bracha 1995, Bloedel and Bracha 1997, Kleim et al. 1998, Thach et al. 1992). This provides evidence to support the dynamic role that the cerebellum plays in SMI. Furthermore, cerebellar impairment has been shown to minimize the stored after-effects of learning which is essential to skill retention (Tseng et al, 2007). The cerebellum contributes to the efficiency of neuromuscular control through its ability to provide real-time task specific adjustments to motor commands which help to update our movement patterns therefore increasing the likelihood of it being stored in long-term memory (Ghez et al. 1991, Ghez and Krakauer 1991, Wadell et al. 1991).

Neck fatigue has been shown to impair upper limb motor performance outcomes in conjunction with differential changes in SEP peak amplitudes related to SMI (Paulus and Brumagne 2008). This suggests that areas dedicated to SMI such as the cerebellum may be impacted by CEM fatigue. In contrast, task performance and skill retention has been shown to significantly improve in the presence of enhanced proprioceptive sensory information and feedback (Adams et al. 1975). There is a growing body of evidence which supports the theory that alterations in SMI and proprioception, in the presence of altered neck function, has the ability to impair motor performance and accuracy of upper limb movements (Zabihhosseinian et al. 2017, Zabihhosseinian et al. 2015, Zabihhosseinian et al. 2019). Previous studies have shown that altered sensory input from the neck resulted in neurophysiological changes in both cerebellar-related SEPs (Andrew

et al. 2018), and cerebellar disinhibition measured via TMS, subsequent to a motor learning task (Baarbé et al. 2018). Changes in sensory input from the neck is also known to affect the activity of 1A fibers (Bolton 1998), as well as impact upper limb proprioception (Knox and Hodges 2005, Zabihhosseinian et al. 2015).

Neck pain and neck muscle fatigue have been shown to contribute to alterations in proximal and distal limb and forearm position sense (Baarbé et al. 2018, Bolton 1998, Haavik and Murphy 2011, Knox and Hodges 2005, Wall et al. 2002, Zabihhosseinian et al. 2015). Also, alterations in motor unit recruitment patterns and activity has been linked with neck pain (Kristjansson et al. 2003, LETAFATKAR et al. 2009, Revel et al. 1991). Currently, the extent to which FHP alters SMI and the influence it has on subsequent motor performance remains unknown. Therefore, the aim of the present study is to further investigate:

1. The effect of FHP on motor tracing performance (accuracy) when compared to a Non_FHP control group.
2. The effect of FHP on changes in cortical activity related to somatosensory integration following performance of a novel motor tracing task, when compared to a Non_FHP control group.

It was hypothesized that following motor skill acquisition, individuals with FHP would demonstrate differences in motor performance and cortical activity related to SMI when compared to a Non_FHP control group.

4.2. METHODS

4.2.1. Participant overview

The study received approval from the Ontario Tech University Research Ethics Board and was conducted in accordance with the Declaration of Helsinki. Students between 18-35 years with no known neurological conditions were recruited from the University of Ontario Institute of Technology through campus posters and word of mouth. In order to be eligible for the study all participants had to be free of neurological and/or spinal pathology, experience little to no neck pain/discomfort and be right hand dominate. Participants signed and completed an informed consent and additional questionnaires on the day of testing. Right hand dominance was confirmed using the Edinburgh Handedness Inventory (Cohen 1961).

The Neck Disability Index questionnaire (NDI) was used as a way to exclude neck pain participants from this study. The NDI is both a valid and reliable tool and has been used extensively in research as a way to assess the impact of neck pain on the performance of activities of daily life (Vernon and Mior 1991). The NDI consists of 10 questions with each having an ordinal scale with six potential response. Each response describes a greater degree of disability,

ranging from 0 to 5. Scores are summed to provide a total score (Vernon & Mior, 1991). No neck pain was defined as a score between 0-4, mild pain was defined as 5-14, moderate 15-24 and severe as 25-34 (Vernon and Mior 1991). To ensure that participants had minimal neck pain which could confound the results, participants with NDI scores greater than 10 were excluded.

Changes in SEP peak amplitude were measured both pre- and post-intervention for both FHP and non_FHP groups. Additionally, to determine motor training effects, accuracy pre- and post-acquisition phase as well as accuracy during a retention test was measured. The methods followed for this study were similar to those used in a previous study performed by Andrew et al. (2015; 2018). Participants in both groups performed the same training intervention.

4.2.3. Experimental protocol

This study was a between group experimental design comparing the effects of the motor training on a group with FHP to a Non_FHP group. All participants were required to attend two sessions. Day 1 consisted of the SEP measurements which were measured pre-acquisition (baseline) and post motor acquisition training. Day two consisted of a follow-up session to measure retention using the same test performed pre- and post-learning on day 1. A schematic of the protocol design can be seen in Fig. 4. All participants performed the same training intervention. Double baseline measurements were performed for pre-SEP measurements through the stimulation of the median nerve (2.5 and 5Hz). Immediately following baseline SEP measures, participants were required to perform a repetitive tracing task using only their right thumb (Figure 1)

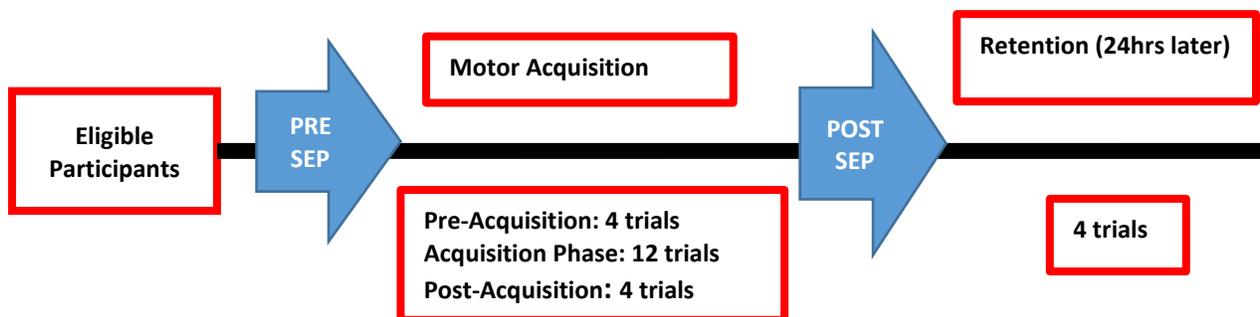


Figure 1. Experimental Protocol

4.2.4. SEP Stimulation parameters

Stimuli consisted of electrical square pulses, 200 μ s in duration, delivered at a constant intensity and frequencies of both 2.5 Hz and 5 Hz through Ag/AgCl EMG conductive adhesive surface electrodes (Meditrace™ 130, Kendall, and Mansfield, MA, USA) (impedance <5 k Ω) connected to the cathode and anode of the stimulator (Digitimer DS7A constant current, 0-100MA, 100-400V, 50-2000 μ s, Welwyn Garden city, England). Electrodes were placed on the skin overlying the median nerve of the right wrist between the tendons of flexor pollicis longus and palmaris longus. The cathode was placed 2 cm proximal to the wrist crease and the anode on the wrist crease in order to prevent anodal block (Crucchi et al. 2008). This position allows for movement of the APB through stimulation of the motor branches of the median nerve that mainly innervates this muscle. SEP peak amplitudes were measured from the averaged 1500 sweeps of the waveforms.

The intensity of the electrical stimuli was delivered until motor threshold was reached for each participant which was defined as the lowest possible stimulation intensity that elicited a visible and constant thumb twitch (approximately 10 mm) of the APB muscle. At this stimulus intensity, all SEP components peaking before 50 ms post stimulus can reach their maximal amplitude (Crucchi et al. 2008). The goal of stimulating above motor threshold ensured that the fastest conducting group 1a afferents responsible for much of the short latency SEPs, are being stimulated (Gandevia et al. 1996, Gandevia et al. 1984) The activity in group 1a muscle afferents project to the cerebral cortex following the median nerve stimulation (Gandevia et al. 1984). Somatosensory evoked potentials were recorded at 2 different rates to enable optimal conditions to record both the N24 and N30 SEP peaks. Using the slower rate of 2.5 Hz does not lead to SEP peak attenuation whereas the faster rate, 5 Hz, attenuates the N30 SEP peak allowing for the N24 SEP peak to be accurately identified and measured (Fujii et al. 1994)(Haavik and Murphy 2013).

4.2.5. Recording parameters

A Waveguard™ 64-electrode EEG cap (ANT Neuro, Netherlands) was used to collect surface brain electrical activity in response to the stimulation of the median nerve. The Waveguard™ cap was connected to a TMSi REFA-8 amplifier with 64 EEG channels, four bipolar channels, and four auxiliary channels, which was run through asaLab™ (Netherlands) to collect and record each session. An additional electrode was placed on the ipsilateral Erb's point in order to record N9; a reference ear clip was placed on the ipsilateral earlobe. Both channels were connected to the same TMSi REFA-8 amplifier.

During data collection the participants were asked to sit with their eyes closed while remaining quiet sitting as still as possible while the lights in the room were turned off. To ensure participant comfort, the data were collected in a quiet room with the participants seated in a comfortable but rigid office chair. Each SEP peak was amplified (gain 10,000), filtered (0.2–1000 Hz) and saved on a laboratory computer for analysis. SEPs analysis was completed on a separate laptop using Advanced Source Analysis (ASA™; Netherlands), Matlab™ (Natick, MA, USA), and SPSS® (Armonk, New York, NY, USA). Figure 2 (Montage of 64 electrode cap)

4.2.6. Tracing task parameters

The motor acquisition tracing task was run and analyzed through a custom-written (C++) Leap Motion software tool (Leap Motion, Inc., San Francisco, CA, USA). Traces were formed by a series of continuous sinusoidal-pattern waves composed of colored dots which moved vertically down a monitor while the participant attempted to trace each dot as it passed the horizontal axis. This horizontal axis has a single dot with the same radius as the dots composing the sinusoidal-pattern waves. Four unique preselected sinusoidal-pattern waves were designed to change in complexity through the variation and randomization of both the frequency and amplitude of the sinusoid, allowing for unpredictability throughout the duration of the trace (Andrew et al, 2015).

Completion of this tracing task has been shown not to result in hand muscle fatigue (Holland et al. 2014), while allowing continuous learning to occur (Andrew et al. 2015). Each trial required the participant to continuously adjust the velocity and range of motion of the thumb (abduction/adduction) throughout the trace. This movement allowed for a sweep of the thumb to occur from left to right, utilizing the abductor pollicis brevis (APB) muscle. Colour coding of the dots provided trace accuracy feedback with green representing a perfect trace and yellow demonstrating some error. The sinusoidal-pattern waves are close together at the beginning and get wider at the end of each trial (Figure 2)

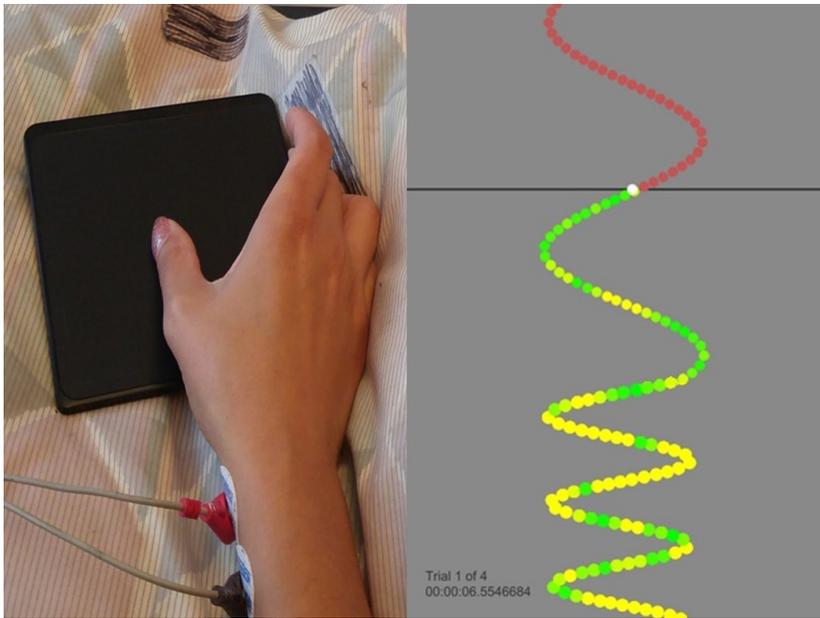


Figure 2. Motor acquisition tracing task (Custom Leap Motion Software Tool). A continuous sinusoidal wave will move vertically down the screen and participants attempted to trace/copy each dot as it passed the black horizontal axis. Colour coding of the dots show trace accuracy (Green = accurate; Yellow = not accurate)

Participants were seated in front of a desk upright in a back supported chair in front of lap top and a trace pad. This scenario was used in order to simulate an environment common to students. Precaution to stabilize the arm and isolate the thumb during task performance was utilized in order to prevent shoulder fatigue and discomfort. The height of the armrest was set to allow the shoulder to remain in a neutral position and the table's platform was level with the armrest. The position and direction of the hand was marked to ensure a consistent position during median nerve stimulation. The thumb was positioned on an external wireless touchpad (Logitech, Inc., Fremont, CA) to perform the tracing task. No obvious delay time occurred between the participant's movements on the external tracing pad and the movement of the traces observed on the monitor.

During each session the participant completed three blocks of training for the pre-acquisition, post-acquisition, and retention trials; with each of the four trace conditions performed once in approximately 3-minutes time duration. For the acquisition phase, each version was performed three times for a total of 12 traces taking approximately 10-minutes. Prior to the start of each block, for each participant, the order of the task version was pseudo-randomized to control for possible order effects in completing the different tasks (Holland et al. 2017). Previous work using the same tracing task has shown that the simplest trace was easy to trace for all participants and the hardest was challenging (Holland et al. 2017). This was done to ensure that motor learning would occur. Motor error was analyzed by determining the average distance of the attempted trace from

the original template trace. Improvement in performance refers to a decrease in the percentage of error throughout the task.

Immediately following the motor task intervention post-SEP measurements were taken in an identical fashion to the pre-measurements. The post-SEP measurements took approximately 10 mins at 2.5 Hz and about 5 mins at 5 Hz for a total post-measurement time of approximately 15 mins. In addition to the SEP measurements, behavioural measurements of accuracy or error rate were also measured pre- and post-acquisition phase. 24 h following the first session participants came in for retention test for the tracing task which consisted of tracing versions 1–4 one time each (duration of approximately 2 min in total). During this time, SEPs were not collected but accuracy was measured. This was done to support the main focus of measuring the immediate capacity of the CNS to respond to the acquisition of a novel task. It is not possible to standardize changes on different days or any potential deviations in the placement of recording or stimulation sites (Figure 1).

4.3 DATA ANALYSIS

4.3.1. Performance Data Analysis

Accuracy of the task was measured by a customized program and was calculated based on the average distance the participant's trace attempt was to the actual trace presented (which would represent a "perfect" trace). The sinusoidal tracing task data was exported to Excel™ (Microsoft Office version 16). The mean percent error from each trial was then averaged and compared. A percent error of 100% would indicate that on average, the tracing accuracy of the participant was a width of about one dot away from the presented perfect trace. Therefore, for all participants any tracing data which resulted in an error greater than 100% was filtered out of the trace. This removed any potential outliers which could have potentially skewed data resulting from participant distraction or boredom which may have occurred during the task.

4.3.2. Neurophysiological Data Analysis

A customized MATLAB code was used for all SEP peak analysis. Post motor acquisition SEP peaks were normalized to pre-intervention values to enable comparison of proportional changes for all participants. In keeping with our pre-established protocols, only trials with a stable peripheral nerve volley (N9 SEP peak) were included for statistical analysis. For this criterion to be achieved, the N9 SEP peak was to differ by no more than $\pm 20\%$ pre- and post-intervention trials (Nuwer et al. 1994). This percent variation ensured any potential changes observed in the SEP peaks known to have central generators were indeed due to the motor training intervention and not due to an altered afferent volley due to

changes in arm position. The brachial plexus, also known as Erb's point, on the anterior aspect of the neck is used as a recording site for the peripheral N9 as it is the last recordable point of entry for afferent activity before it enters the spinal cord (Nuwer et al. 1994).

The amplitudes of the SEP peaks were measured from the peak of interest to the preceding or succeeding peak of opposite deflection in accordance with international recommendations (Nuwer et al. 1994) and previous studies which outline the optimal sites for recording (Cheron et al. 2007, Rossi et al. 2003, Sonoo et al. 1991). The amplitudes and latencies of the following SEP components were identified and measured: the peripheral N9, the spinal N13, the far-field N18 (P14–N18 complex), the parietal N20 (P14–N20 complex) and P25 (N20–P25 complex), the frontal N24 (P22–N24 complex), and the frontal N30 (P22–N30 complex). The latencies were recorded from the time of stimulation onset to their maximal peak or trough for each of the SEP components. The N24 SEP peak is often observed as a notch which resides on the upward slope of the N30 SEP peak (Larrea et al. 1992). This particular SEP peak's amplitude is only measured using the faster rate measurement trials of 5 Hz as the higher stimulation rate allows for attenuation of the N30 peak thus allowing for appropriate identification and measurement of the underlying N24 SEP peak (Fujii et al. 1994, Haavik and Murphy 2013).

4.3.3. Statistical Analysis

To investigate and compare the mean difference of accuracy both pre and post the acquisition phase and during retention of both motor tasks, a 2×3 repeated measures ANOVA was performed comparing pre-acquisition, post-acquisition and retention phases for both FHP and Non_FHP groups. The interactive effect of FHP on post-motor training changes in each SEP peak amplitude was tested with a 2×2 repeated measures ANOVA with time (pre-acquisition vs. post-acquisition) as the repeated measure and group (Non_FHP vs. FHP) as the between subjects' factor.

Both behavioural and neurophysiological data was analyzed using SPSS (SPSS v.25, IBM Corporation, Armonk, NY, USA). Statistical significance was set at $P \leq 0.05$ for all analysis. All data was tested for normality (Shapiro–Wilk's test), homoscedasticity of variance (Levene's test of equality), and sphericity (Mauchly's test), where applicable. All numeric values are expressed as mean \pm standard deviation (SD). Effect size was classified as small (0.01), moderate (0.06), and large (>0.14). Moderate and large effect sizes were considered indicators of statistically meaningful results (Richardson 2011). Outliers were calculated by SPSS as ± 1.5 times the interquartile range between the 25th and 75th percentiles (added to 75th percentile or subtracted from the 25th percentile) and were removed from analysis.

4.4. RESULTS

4.4.1. Participants

Out of the 43 eligible participants, a total of 16 (6M; 10F) participants were able to be included in the SEP peak analysis and 41 participants (21M; 20F) were able to be included in the analysis of performance data (tracing task). The assumptions of normality were met for both groups and results was reported using the Huynh-Feldt correction . As mentioned in our SEP inclusion criteria, in order to be confident with any central changes the N9 peripheral volley must not increase to an amplitude larger than 20% of what was recorded pre-motor learning. During analysis, both groups showed no statistical difference for the N9 peak ($p = 0.31$) however, many N9 traces in the FHP group had excessive “noise” which meant that the N9 could not be confidently measured. Therefore, these participants were not able to be included in the subsequent SEP peak analysis. Furthermore, SPSS identified 1 male and 1 female in the FHP group as outliers and were subsequently removed from analysis.

Representative traces from an individual participant seen in Fig. 5 are indicative of the group changes observed in our analysis. Mean group comparisons are shown in Fig. 6.

4.4.2. Performance Data

When examining performance measures of the tracing task, accuracy was defined by the average percentage of error at pre-acquisition, post-acquisition and retention. Both post-acquisition and retention time posts were normalized to each participant’s baseline value to account for inter-participant baseline variability and to allow for between- group comparisons.

The 3x2 repeated measures ANOVA for the time period of pre-acquisition and retention showed a significant interactive TIME x GROUP effect [$F_{1,37} = 1.581 = 3.890$, $p = 0.035$, $\eta^2 = 0.095$]. The pre-planned contrasts indicated that the group difference in improvement relative to baseline was significant ($F = 6.069$, $p = 0.019$) at the 24hr retention period, where the Non_FHP group improved their accuracy by 7.61%, and the FHP by 1.39%. These changes are demonstrated in Figure 3.

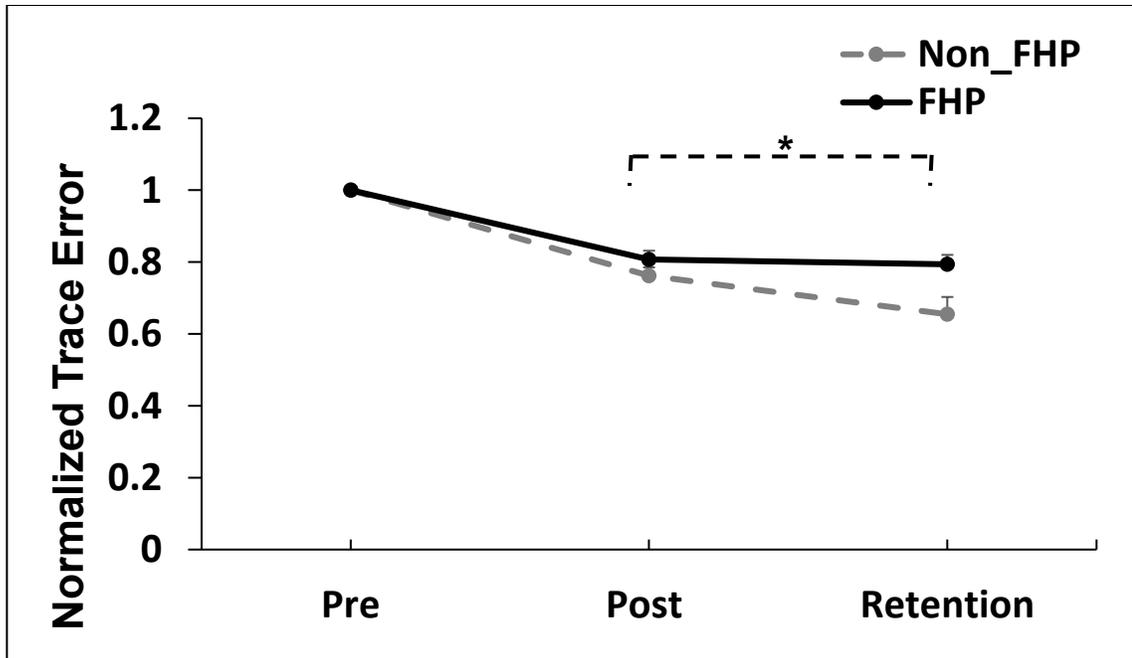


Figure 3. Normalized mean proportional trace error changes by groups (Non_FHP vs FHP). Both groups improved in accuracy after motor acquisition tracing tasks and during the retention ($p=0.019$). The Non_FHP group outperformed the FHP group in both post- motor acquisition and during the retention tracing tasks ($*p \leq 0.05$). Error bars represent the standard error of the mean.

4.4.3. Neurophysiological data

Far-field N18

Following motor acquisition there was a significant main effect of TIME [$F_{1,12} = 8.503$, $p = 0.013$, $\eta^2 = 0.415$] and a trend towards a significant interaction of TIME x GROUP [$F_{1,12} = 3.87$, $p = 0.073$, $\eta^2 = 0.244$]. The FHP group had a mean percent increase of $41 \pm 20\%$, with a mean percent increase of $10 \pm 39\%$ in the Non_FHP group. No significance or meaningful effect size was found for a TIME by SEX ($p = 0.624$) interaction (Figure 3 and 4).

Parietal N20

Following motor acquisition there was a significant interactive TIME x GROUP effect [$F_{1,12} = 12.361$, $p = 0.004$, $\eta^2 = 0.507$] observed. Additionally, a significant effect of TIME was also observed following motor learning [$F_{1,12} = 8.036$, $p = 0.015$, $\eta^2 = 0.401$]. The amplitude of the N20 peak for the FHP group had a mean percent decrease of $2 \pm 8\%$ versus a $22 \pm 16\%$ increase for the Non_FHP group (Figure 3 and 4).

Frontal N24

Following motor acquisition there was a significant interactive TIME x GROUP effect [$F_{1,12} = 8.67$, $p = 0.012$, $\eta^2 = 0.419$]. The amplitude of the N24 peak of the

FHP group had a mean percent increase of $15 \pm 10\%$, while the Non_FHP group decreased $3 \pm 15\%$ following the tracing task.

P25

Following motor acquisition there was a significant main effect of TIME [F1,12 = 13.81, $p = 0.003$, $\eta^2 = 0.535$] with no significant interactive effect of TIME by GROUP ($p = 0.624$) or TIME x SEX ($p = 0.253$). The FHP group had a mean percent decrease in amplitude of $9 \pm 8\%$, while the Non_FHP group decreased by $7 \pm 7\%$ following the tracing task (Figure 3 and 4).

Frontal N30

Following motor acquisition there was a significant interactive TIME x GROUP [F1,12 = 35.227, $p = 0.001$, $\eta^2 = 0.746$] and TIME by SEX [F1,12 = 4.98, $p = 0.045$, $\eta^2 = 0.293$] effect observed. The amplitude for the FHP group had a mean percent increase of $17 \pm 3\%$, while the Non_FHP group increased $4 \pm 5\%$ following the tracing task. The amplitude for males increased by $14 \pm 8\%$, while the females increased $9 \pm 8\%$ following the tracing task. No significance was found for a three-way TIME x SEX by GROUP interaction ($p = 0.816$) (Figure 3 and 4).

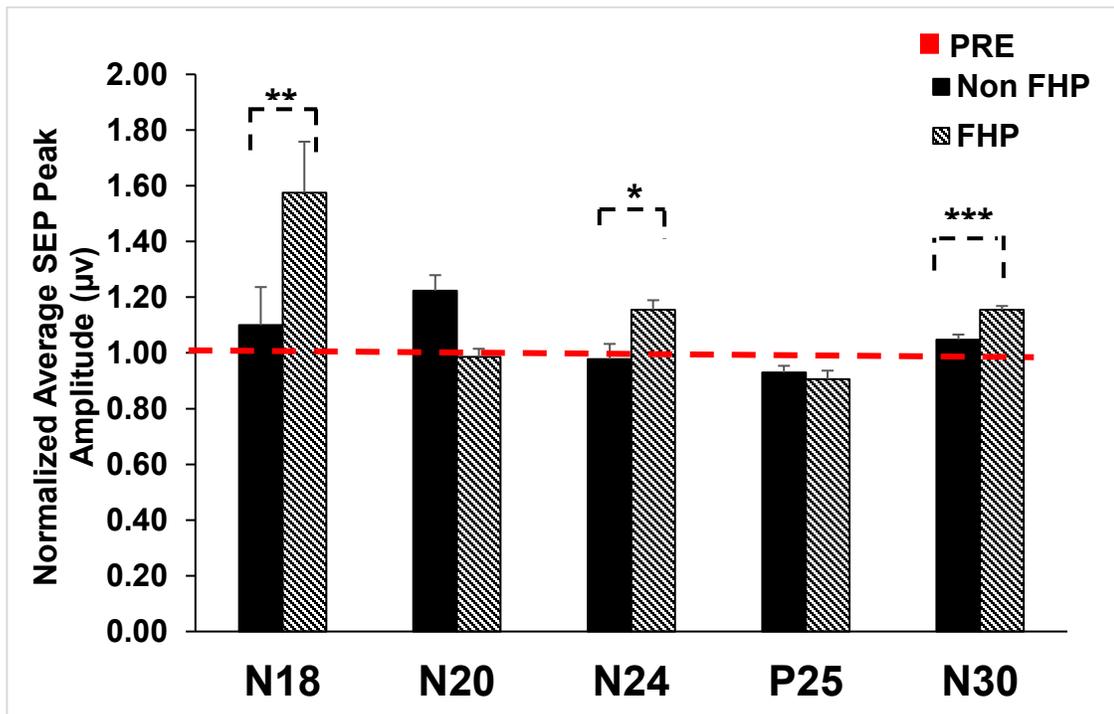


Figure 4. Averaged and normalized SEP Peak amplitude (ratios relative to baseline dotted line) for the Non_FHP vs. FHP groups following motor acquisition. Significant interactive effects between groups were found for N18, N20, N24 and N30 SEP peaks. **Note:** TIME by SEX interaction was observed for the N30 peak ($p = 0.045$). The amplitude for males increased by $14 \pm 8\%$, while the females increased $9 \pm 8\%$ following the tracing task. Error bars represent the standard error of the mean. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

4.5. DISCUSSION

This study sought to investigate the effect of FHP on sensorimotor processing and motor skill acquisition in young adults by measuring changes in early SEP peak amplitudes and motor performance. It should be noted that due to unanticipated issues with electrical noise we could only analyze the data of sixteen participants therefore, additional data should be collected to confirm these findings and validate any existing trends within the data.

The outcomes of this study had two novel findings with respect to our hypotheses. While both FHP and Non_FHP improved following motor acquisition, only the Non_FHP group continued to improve at retention whereas the FHP group plateaued. FHP was also shown to alter early SEP peak amplitudes in peaks known to reflect SMI (N18, N24 and N30) differentially to the Non_FHP group following a complex motor tracing task. Neck muscle fatigue has been shown to alter upper limb performance measures (Zabihhosseinian et al. 2015, Zabihhosseinian et al. 2019) however, little is documented with respect to FHP. This study is the first to report differential SEP peak amplitude changes in response to acute alterations in afferent input to the neck induced by FHP. While the experimental pain studies undoubtedly provide a link between pain and altered motor control (Arendt-Nielsen and Graven-Nielsen 2008, Falla and Farina 2008), there is less evidence to support the impact of FHP on altered motor control. With the growing demand of technology in today's society ((Neupane et al. 2017), these preliminary findings support the need for further research into this potentially problematic phenomenon.

In a sub-group of 8 FHP and 8 Non_FHP (6M;10F), SEP data was able to be collected to help understand the possible influence of FHP on neurophysiological processing in response to motor skill acquisition. Differential changes between FHP and Non_FHP groups were seen in N18, N24 and N30 SEP peaks.

The acquisition of a new motor skill follows two distinct stages with continued practice. First, there is an early, fast learning stage in which performance improves rapidly within a single training session. Later, there is a slower learning stage in which incremental gains in performance are seen over several sessions of practice (Ungerleider et al. 2002). In the current study, both groups were able to improve their performance rapidly from pre to post acquisition however, no further improvements were seen in accuracy measures in the FHP group, suggesting that FHP impairs upper limb motor learning. Andrews et al. (2018), initially investigated the impact of SCNP on SMI and found that changes in sensory information from the neck were associated with alterations in cerebellar processing in response to motor learning. More recently, a study conducted by Zabihhosseinian et al. (2019), found that altered afferent input from the neck induced by fatigue, impaired performance accuracy on a similar task as the one used in the current study. This is similar to what was observed in both this study

and in the first study of this thesis which saw significant differences between upper limb and neck proprioception accuracy in those with FHP versus those in the control group. Previous work has also shown alterations in cerebellar to M1 pathway following a motor learning task in the presence of SCNP (Baarbé et al. 2018, Daligadu et al. 2013) which supports the findings of this study.

It has been hypothesized that changes in muscle recruitment patterns are an adaptation to an underlying spinal instability resulting from muscle ligament laxity or damage, muscle dysfunction or reduced neuromuscular control (Jull et al. 2004, Jull 2000, Jull et al. 2008, Panjabi 1992). This maladaptive posture is adopted gradually as a consequence of adaptive or compensatory cervical adjustments in response to the repetitive and long-term exposure to the postures commonly associated with computer and cellphone use (Neupane et al. 2017). In this adaptive state, individuals with FHP are not getting the correct sensory information from the neck when there is likely to impact the body schema of the upper limb relative to the neck. As a result, sensory information coming from the peripheral system which has gradually become adjusted is interpreted by the cerebellum as being correct, especially if this posture is repeated in excess over time (Andrew et al. 2018, Baarbé et al. 2018), resulting in altered upper limb motor performance.

The neck is a coordinated network of nerves, bones, and muscles which are coordinated, controlled and influenced through the communication of the brain with the outside world (Neupane et al. 2017). Neck muscles contain a high density of sensory receptors with neural connections that project to the vestibular and oculomotor systems (Jull et al. 2007, Winters et al. 2012). During movement the brain will rely on these systems in order to plan, control, and adjust limb movement (van Beers et al. 1996). Sensory inputs are matched against the brain's internal map or "body schema" to predict the future position of the limb (Proske and Gandevia 2009). Therefore, alterations in afferent input from the neck to the CNS is likely to lead to a distorted body schema which influences the awareness of upper limb movement in 3D space (Guerraz et al. 2001, Haavik and Murphy 2011). Previous work has demonstrated that alterations in sensory input as a result of neck pain or fatigue has the ability to affect upper limb motor performance (Helgadottir et al. 2011, HelgadoTTir et al. 2010, Zabihhosseinian et al. 2015), and alter body schema of the upper limb in relation to the neck (Knox and Hodges 2005) however, this is the first study to document similar results in FHP.

Motor skill acquisition requires processing of somatosensory information from a motor task and the ability of brain to integrate this information with the appropriate motor command (Willingham 1998). Visual and proprioceptive information is used to fine-tune and improve the efficacy of the motor task performed which is referred to as sensorimotor integration (Ungerleider et al.

2002, Willingham 1998). The cerebellum is of specific interest when discussing SMI as one of its primary function is to modify voluntary movement (Andrew et al. 2018) and is recently emerging as a primary contributor in the role of sensory integration and internal feedback associated with SMI (Andrew et al. 2018). The thalamus, which is known to have projections to the primary somatosensory area (S1), works with the cerebellum to develop and internal representation of the external world (Andrew et al. 2018). This is an important pathway in SMI as it allows an individual to gradually adapt to a task by reducing errors in motor performance in real time (Andrew et al. 2018).

The cerebellum is known to be highly involved in sensorimotor integration, both receiving information from the motor cortex regarding motor commands and integrating this with peripheral sensory information and expected sensory information due to past experience (Manzoni 2007, Timmann et al. 2010), to enable finetuning and improved efficacy in task performance. There is a large body of evidence that reveals structural and functional changes within the CNS of people with chronic musculoskeletal disorders (Manto and Bastian 2007). These changes may initially be beneficial, but as they persist they are thought to be maladaptive.

This study is the first to report differential SEP peak amplitude changes in response to alterations in sensory input from the neck induced by FHP. With the growing use of technology in today's society (Neupane et al. 2017), these preliminary findings support the need for further research into this potentially problematic phenomenon.

4.5.1. N18 SEP peak changes

In the current study, both groups showed a significant increase in the N18 SEP peak amplitude following motor acquisition. There was no significant difference found between the Non_FHP and FHP group following motor acquisition however, the large effect size indicates there may have been greater cerebellar activation associated with FHP during SMI.

The neural generator of the N18 peak is thought to arise in the brain stem, located above the spinal cord but below the cortex (Noël et al. 1996, Sonoo et al. 1991). More specifically, this peak it is thought to reflect alterations in cerebellar activity through changes in sensory input coming from the dorsal column medial lemniscus tract (Nuwer et al., 1994; Sonoo et al., 1991). The lateral aspect of this tract, known as the cuneate nucleus, transmits upper limb sensory information to the cerebellum and plays an important role in SMI by channeling and transmitting various types of somatic information through the nervous system (Kandel et al. 2000). The cerebellum works to coordinate these signals from the spinal cord through its relationship with the accessory inferior olives of the medulla and its projections to M1; together these pathways work to regulate motor coordination

by initiating and regulating motor functions by adapting to somatosensory input during skill acquisition thereby, enhancing motor learning (Haavik and Murphy 2013, Marshall 1984, Pascual-Leone and Torres 1993). The interactive effect observed for this peak in the current study may reflect alterations in the way individuals with FHP process information in the cerebellum-M1 pathway when learning a new skill.

A key function of the cerebellum is to modify sensory output to the somatosensory cortex through disinhibition or a decrease in cerebellar activity and is starting to become recognized in the literature as activity associated with the early stages of learning (Baarbé et al. 2013, Daligadu et al. 2013, Doyon et al. 2003). It has been suggested that in order to acquire and enhance a new skill the cerebellum must first allow an influx of sensory information to be sent to the higher cortices in order to provide feedback for real time movement adjustments. A recent SEPs study conducted by Andrews, et al. (2018) observed a larger increase in the N18 SEP peak amplitude of individuals with SCNP following a complex task when compared to a healthy control. The findings of this study support the findings of the current study; therefore, it is plausible to suggest the larger increase of cerebellar activity is due to the increased afferent information coming from the neck.

4.5.2. N24 SEP peak changes

In the current study, the FHP group showed a significant increase in the N24 SEP peak amplitude in response to the tracing task vs the Non_FHP group which significantly decreased following motor acquisition.

The N24 peak which has been previously studied using dipole sourced analysis reflects the activity of neuronal pathways between the cerebellum and S1, and has been shown to reflect changes in cerebellar output (Restuccia et al. 2006, Rossi et al. 2003). The cerebellum is involved in control of limb movements and uses a feedforward internal model to predict the sensory consequences of different planned movements (Manto et al. 2012). When trying to learn an unfamiliar task or when trying to correct errors in a newly learned skill, the efficient communication between the cerebellum, midbrain structures (basal ganglia, thalamus) and higher cortices (S1 and M1) is necessary to produce smooth and accurate motor performance (Karni, 1996; Doyon, 1997; Sanes & Donoghue, 2000; Baarbé et al., 2014). As we learn something new, cells that send and receive information about the task become more and more efficient (Baarbé et al. 2013, Doyon et al. 2003, Karni 1996, Sanes and Donoghue 2000). Each and every time we learn something new our brain forms new connections and makes existing neural pathways stronger or weaker; a term known as neural plasticity (Doyon et al. 2003, Karni 1996, Sanes and Donoghue 2000). The cerebellum does not encode motor memory but rather is involved and activated

in the earlier stage of motor learning (Flament et al. 1996) with its role related to the acquisition of internal models during motor learning (Imamizu et al. 2000).

The decrease in the N24 SEP peak in the Non_FHP group is consistent with previous studies that found decreases following a simple automatic repetitive typing task (Andrew et al. 2015) and after a motor learning tracing task similar to the one used in the current study (Andrew et al. 2018). Furthermore, a more recent study demonstrated that the N24 SEP peak showed a larger decrease for the tracing task in comparison to the repetitive typing task. The authors proposed that the decreases observed were likely due to the ability of complex tracing tasks relying heavily on cerebellar pathways (Doyon et al. 2003) therefore, the decreased N24 amplitude in the Non_FHP group of the current study is likely related to decreased cerebellar nuclei activity (Doyon et al. 2002), which was also associated with the greater improvement in motor acquisition.

The increase in the N24 in the FHP group is consistent with a previous study conducted by Andrew et al (2018), which investigated the long-term changes to sensory input from the neck of participants with neck pain following a motor training task. Their results found similar increases in the N24 SEP peak in response to motor skill acquisition when compared to a healthy control, potentially reflecting increases in cerebellar-SI processing in individuals with neck pain (Andrew et al., 2018). The changes in the amplitude related to cerebellar processing indicates that altered sensory input from the neck is likely to impact the expected neuroplastic changes in cerebellar pathways in response to motor acquisition [30].

4.5.3. N30 SEP peak changes

The N30 SEP peak component is considered a marker of sensorimotor processing and is generated through a complex cortical-subcortical loop which links the basal ganglia, thalamus, pre-motor areas, and the primary motor cortex (Mauguiere et al., 1983; Rossini et al., 1989, Rossini et al., 1987; Waberski et al., 1999). It is suggested that this peak reflects the process of sensorimotor integration (Rossi et al., 2003). In the current study, increases were seen in both groups however, the amplitude was significantly greater in the FHP group. These findings are similar to previous SCNP (30) and experimental pain studies (72) which saw similar increases following motor acquisition. Males were also seen to have a significantly larger amplitude in the N30 SEP peak vs females, which to our knowledge, is the first time this was observed in this paradigm.

The greater N30 SEP peak in the FHP group post- motor acquisition is thought to reflect attempts by the SMI integration network to integrate the altered neck muscle sensory feedback following fatigue resulting in altered excitability in pathways relevant to motor learning (30, 44). Altered movement patterns in the cervical spine were also found for both SCNP and chronic pain groups indicating

changes in motor control strategies (Andrews et al., 2018). They concluded that the changes observed were not in fact related to a history of neck trauma or to current pain, but more likely due to long-lasting effect on pain and its ability to create plastic changes in our feedforward and feedback pathways (Andrews et al., 2018). These results help to support our second novel finding with regards to SEP peak changes found in the FHP group in response to motor learning.

4.5.4 Limitations

Although great care was taken when positioning the participant, unexpected electrical noise in the N9 recordings meant that a number of traces could not be analyzed because it could not be confirmed that the N9 was a reliable part of the inclusion criteria for subsequent peak analysis. This 'noise' may have been due to EMG contamination in the FHP group and future studies should consider recording the peripheral nerve volley at a more distal site such as the elbow. Participants were also required to sit for long periods in an upright position. Having the participant in a more relaxed and supported neck position may have helped to reduce fatigue and associated EMG activity, which could have interfered with N9 peak.

4.6 CONCLUSIONS

The results of this study provide preliminary support for the importance of head posture and the influence cervical postural deviations have on cerebellar activity and subsequent SMI. In support of our hypothesis, the postural changes associated with FHP altered afferent information from the neck was demonstrated through differential changes observed in the magnitudes of cortical peaks related to SMI (N18, N24 and N30). It has been suggested that alterations in muscle recruitment patterns are developed as a protective mechanism to underlying spinal instability, which result from laxity or damage to the structural tissue of the head and neck, muscle dysfunction, or reduced neuromuscular control (Kwon et al. 2015, Shin et al. 2017). Therefore, the differences in performance accuracy seen in the FHP group could be outcome of faulty sensory processing, subsequently altering upper limb performance. The inability of the FHP group to further improve their accuracy following a 24-hour retention could also suggest that the change in cervical posture also has the ability to alter pathways and mechanisms associated with consolidation and storage of short-term memory. Due to the novelty of this research and the small sample size, additional data should be collected to confirm these findings and validate any existing trends within the data. Future work should aim to investigate the changes in visual orientation in response to changes in cervical posture. Since the primary role of the visual system is to help create a map of our surroundings (Adams et al. 1975, Matthews 1988) it would be beneficial to our understanding of the effects FHP has on the visual component of SMI and its subsequent effect on motor performance.

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CHAPTER 5. THESIS SUMMARY

The orientation of the head with respect to the neck plays an integral role in human movement and performance (Cohen 1961, Guerraz et al. 2001) and is interconnected through neurological pathways in the brain and spinal cord, which, use the neck position as a reference when processing multisensory input (Cohen 1961, Malmström et al. 2009). While studies investigating the effects of technology-induced postures on the musculoskeletal system are starting to emerge (Neupane et al. 2017), the long-term effect on sensorimotor processing and neuroplasticity remains unclear. What is currently available is starting to identify the significance of investigating this phenomenon as further investigation could enhance our understanding of how long-term sensory changes in sensory input from the neck contribute to altered sensorimotor integration and possible errors in motor performance.

In everyday life, we go about activities using a variety of motor skills that have been acquired gradually through practice and repetitive interactions with our environment (walking, driving, speaking, writing) (Kandel et al. 2000). Every action we perform requires the motor system and the coordinated actions of peripheral, spinal, cerebellar and cerebral regions which is further refined by our intentions and goals (Kandel et al. 2000, Strimpakos 2011). Sensorimotor integration (SMI) is a complex process whereby sensory input from the CNS in response to a change in the body's environment (Kandel et al. 2000, Strimpakos 2011) is processed by the CNS and then used to modify outgoing motor commands. Both sensory afferents and the corresponding motor outputs are processed in by a variety of distinct pathways which are active simultaneously and used to create an internal representation of the external environment (Kandel et al. 2000, Strimpakos 2011). The mechanism of SMI involves the incorporation of proprioceptive, kinesthetic and somatic sensation which results in the selection of a suitable motor output for a given environmental demand (Kandel et al. 2000, Strimpakos 2011). The ability to integrate this information into an appropriate motor response makes the sensory motor system essential during movement control (Kandel et al. 2000, Strimpakos 2011).

FHP is adopted gradually as a consequence of adaptive or compensatory cervical adjustments in response to the repetitive and long-term exposure to the postures commonly associated with computer and cellphone use (Neupane et al. 2017). In this adaptive state, individuals with FHP are not receiving the correct sensory information from the neck, which is then likely to impact the body schema of the upper limb relative to the neck. As a result, sensory information coming from the peripheral system, which has gradually become adjusted, would be interpreted by the cerebellum as being correct; especially if this posture is repeated in excess, over time (Baarbé et al. 2018, Caneiro et al. 2010, Manto et

al. 2012, Seghers et al. 2003, Villanueva et al. 1997), resulting in altered upper limb motor performance (Falla et al. 2004, Zabihhosseinian et al. 2015).

The results of the first study found significant main effects for absolute, constant and variable error during a JPS matching task; suggesting that individuals in the FHP group had larger, and more variable shoulder JPSE when compared to the Non_FHP group. Furthermore, a significant two-way interaction was observed for males in the FHP group for constant error, indicating that males were more likely to overshoot the indicated target during task performance. These results support hypothesis two, which suggested that differences would be seen in upper limb JPS as a result of the altered afferent input from the neck due to FHP. This work agrees with previous studies showing that neck fatigue impacted elbow JPS (Villanueva et al. 1997, Zabihhosseinian et al. 2017, Zabihhosseinian et al. 2015, Zabihhosseinian et al. 2019). The changes in the neck fatigue group were suggested to be due to the impact of fatigue on sensory input from the neck muscles to the CNS. Fatigue has been shown to alter the firing rate of 1A afferents during sustained submaximal contractions (Macefield et al. 1991), indicating that fatigue-induced excitation of slowly conducting muscle afferents is significant in mediating the fatigue-induced inhibition of motoneuron output (Gandevia 2001, Hayward et al. 1991, Taylor et al. 1996)

Proprioceptive acuity involves both peripheral and central processing and is the product of sensory information supplied by specialized nerve endings called mechanoreceptors (Grigg 1994, Laskowski et al. 2000, Matthews 1988). These receptors are located in muscle, joint capsules, tendons, ligaments, and skin and are specific to providing input on joint position and velocity throughout its entire range of motion (ROM) (Gandevia et al. 1990, Hillier et al. 2015, Matthews 1988). During movement, proprioception is important for feedback (reactive control), feedforward (preparatory control) and the regulation of muscle stiffness in order to achieve specific postures required for efficient muscle balance, coordination and joint stability (Hillier et al. 2015, Milner et al. 2007, Riemann and Lephart 2002). An underactive proprioceptive system has been shown to lead to recruitment failure of important joint stabilizers, which results in a variety of changes including poor endpoint accuracy during movement (Needle et al. 2013), reduced control during multi-segmental movements, (Sainburg et al. 1995, Sainburg et al. 1993) and an inability to perform repetitive movement sequences (Rothwell et al. 1982).

Cortical neuroplastic changes are associated with altered motor function or behaviour (Sanes and Donoghue 2000). The periphery and central nervous systems (CNS) are constantly relaying information to one another, which creates a fundamental feedback loop; whereby sensory, or afferent input, is integrated by the CNS and used for assisting in the execution of the appropriate motor output (Karni 1996, Riemann and Lephart 2002). The ability of the CNS to perform this

integration relies on its reorganization; plastic changes in the way that the CNS filters information in response to afferent input in a process termed sensorimotor integration (SMI) (Cohen 1961, Karni 1996). These CNS alterations have been shown to persist following the period of afferent input to induce organizational changes in synaptic connectivity and strength. Altered afferent input in the form of behavioural training has been shown to induce organizational changes in both the animal and human cortex (Byl et al. 1997, Classen et al. 1998, Paulus and Brumagne 2008). The alterations in the afferent input, which was shown to induce these changes in the CNS does not have to be of long duration; in fact, repetitive muscle contraction activities have been shown to have an effect with as little as 20 minutes of repetitive finger contractions (Murphy et al. 2003). Cervical extensor muscle (CEM) fatigue has been shown to affect postural control, possibly due to a decrease in central drive and/or inconsistencies in proprioceptive feedback during task performance (Gosselin et al. 2004).

When an individual is learning a new task, adaptive neuroplastic behaviour of the CNS will allow the individual to acquire and store the new task for future recall (Hillier et al. 2015). Conversely, maladaptive neuroplastic behaviour, which is observed in the presence of pain and dysfunction, has an opposite and adverse effect on skill acquisition (Hillier et al. 2015) – whether it is new or familiar. Recognizing and accepting these neuroplastic behaviours as a modifiable variable of motor control is important to the understanding of sensorimotor integration (SMI) and the treatment of neurophysiological dysfunction (Hillier et al. 2015). Studies have shown that effective sensorimotor integration patterns of the CNS are essential to how the body responds physically to a given phenomenon (Daligadu et al. 2013, Haavik and Murphy 2011, Merzenich and Jenkins 1993). Therefore, understanding both adaptive and maladaptive behaviour observed in the brain activity of healthy individuals and those with FHP would be beneficial to our understanding of the development and neurological alterations underlying neck fatigue and SCNP.

The results of the second study had two novel findings with respect to our hypotheses. While both FHP and Non_FHP improved following motor acquisition, only the Non_FHP group continued to improve at retention, whereas the FHP group plateaued. FHP was also shown to alter early SEP peak amplitudes in peaks known to reflect SMI (N18, N24 and N30) differentially to the Non_FHP group following a complex motor tracing task. Neck muscle fatigue has been shown to alter upper limb performance measures (Zabihhosseinian et al. 2015, Zabihhosseinian et al. 2019); however, little is documented with respect to FHP. This study is the first to report differential SEP peak amplitude changes in response to acute alterations in afferent input to the neck induced by FHP. While the experimental pain studies undoubtedly provide a link between pain and altered motor control (Arendt-Nielsen and Graven-Nielsen 2008, Falla and Farina 2008), there is less evidence to support the potential progression to chronic pain

as a consequence of altered motor control in the presence of FHP or fatigue. With the growing use of technology in today's society (Neupane et al. 2017), these preliminary findings support the need for further research into this potentially problematic phenomenon.

The preliminary findings of this thesis provide new insight on the importance of head posture and the influence cervical deviations have on cerebellar activity and subsequent SMI. In support of our hypothesis, the postural changes associated with FHP were associated with the reduced proprioceptive abilities of the neck and shoulder during a joint positioning task. The second study further supported these findings by demonstrating differential changes in the magnitudes of cortical peaks related to SMI which further supports the theory that alterations to sensory information coming from the neck is enough to alter subsequent motor performance.

Taken together, the two studies of this thesis provide novel information regarding neural plasticity and motor learning adaptations in the presence of FHP. Our everyday lives have become dominated by computer technology and handheld mobile devices, making FHP (or now socially know as text-neck), a growing, world-wide, health effect (Neupane et al. 2017). With the recent increases in the prevalence of musculoskeletal, repetitive strain and overuse disorders reported in the neck (Tjepkema 2003) it is critical to identify contributing factors to these problems before they become long-term. In doing so, clinicians will be able to intervene with the appropriate preventative measures, and in doing so, reduce the financial strain these complaints are putting on the health care system.

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CHAPTER 6. APPENDICES

Appendix A. Participant Consent Form



RESEARCH ETHICS BOARD
OFFICE OF RESEARCH SERVICES

Title of Research Study: The Effects of Forward Head Posture on Sensorimotor Integration and Head and Neck Proprioception.

You are invited to participate in a research study entitled: **The effects of Forward Head Posture on Sensorimotor Integration and Head and Neck Proprioception**. This study has been reviewed the University of Ontario Institute of Technology Research Ethics Board [REB # 14965] and originally approved on [October 29th, 2018]. The study is being conducted by **Dr. Bernadette Murphy and Dr. Paul Yelder**, in conjunction with MHS candidate **Tracey Patrick** from the Faculty of Health Sciences at the University of Ontario Institute of Technology (UOIT), in Oshawa, Ontario, Canada. All researchers involved will have signed confidentiality agreements and completed the TCSP II tutorial on research ethical concerns.

Purpose and Rationale:

Ideal posture can be described as having the head positioned directly over the shoulders. This keeps the weight of the head over the body's center of gravity, therefore, minimizing strain on the spine. Forward head posture (FHP) occurs when the head becomes positioned anterior in relation to the body's center of gravity (no longer over the spine). FHP is a common postural disorder in individuals experiencing neck pain, and it is known to contribute to patterns of tissue overuse, which, over time, could lead to changes in the way that the brain processes information about your body and formulates appropriate movement patterns.

Proprioception is the sense that people have of knowing where their body is in their environment and is guided by receptors in the skin, muscles, and joints. These receptors connect with the brain through the spine, a communication pathway known as the Central Nervous System (CNS). These "body maps" create what is known as body awareness, which help us move through our environment safely. Pain and joint dysfunction has been shown to reduce the brain's ability to process proprioceptive information, however, little information is documented in the literature about the role that forward head posture could potentially play in the development of pain and neck dysfunction.

Research has found that neck pain affects the way that people move, and their awareness of head and upper arm positioning; however, little is known about the effects of forward head posture on shoulder and neck proprioception.

The purpose of this study is to investigate the neurophysiological function of the central nervous system in individuals with, and without, FHP in order to gain a better understanding of how FHP affects neck and shoulder proprioception.

In order to do this we will need to collect some information about the way your brain process signals from your hand and forearm muscles during rest and during a series of tasks. We will also get you to complete some questionnaires, which will provide information regarding your current functional capacity, level of neck discomfort (if any), and general well-being. We want to show how FHP affects movement; our hope is that this research will better define the entity of “forward head posture” and observe how it affects our sensorimotor pathways (pathway between the muscles and the brain).

Information for Participants:

For this study, we are seeking right-hand dominate individuals, aged 18-35 years, who:

1. Have no neck or shoulder injuries or neurological conditions
2. Have had no history of intermittent neck or shoulder pain or discomfort in the past six months
3. Have not received treatment of the head or shoulder in the past 6 weeks.

To participate in this study, you will also need to complete an eligibility checklist with one of the researchers.

If you volunteer to participate in the study, **you will participate in a total of four data collection sessions:** two at the start of the study and two identical sessions at the end of the study (8 weeks later). Please take your time reading this information and if you think of any specific questions regarding the study that you may contact anyone of the researchers at any time (**contact information is provided below**)

Measurement sessions:

Session 1:

At **both baseline and follow-up**, you will be asked to complete 7 questionnaires that provide the researcher with information about:

1. Your current functional capacity
2. Level of neck pain (if any)
3. General well-being.

We will then ask you to participate in a **free postural assessment** recorded by a Cervical Range of Motion Instrument (CROM), which is used to identify the presence of FHP. A series of photos will also be taken to document postural assessment before and after the intervention. ****Note: these pictures are for the researchers to make measurements on and they will not be reproduced for publication purposes, without your consent and without first masking any identifying features. A separate consent form will be presented to you for the use of these photos and you can still participate in this research even if you do not wish for these de-identified photos to be used in publications.**

This session should take no longer than **an hour**. You will then be given a chance to review the study details and ask any questions in advance of your participation. We will also use this time to book you in for your second measurement session, and allocate you into one of two groups based on your CROM measurement:

1. Forward Head Posture Group
2. Healthy control group (no FHP).

Session 2

At **both baseline and follow-up**, you will also perform a series of tests to determine any differences in the communication pathways between the brain and your muscles. Brain activity will be recorded before and after you:

1. Perform a finger tracing task on a computer
2. While seated in a chair at rest.

Brain activity during these tasks will be recorded through the use of an EEG cap (explained below). While participating in these tasks we will also be measuring the muscle activity in the muscles of your upper back and neck through the use of surface electrodes (**explained below**).

Data collection on day two, will take approximately **2-3 hours to complete**. These two data collection sessions (one hour, and 2 to 3 hours) will then be repeated for the FHP groups at the follow-up session (e.g. post exercise or control intervention).

Payment or Reimbursement:

If you are a student enrolled in approved Kinesiology courses you may also have the opportunity to earn 2% extra credit toward your final grade (**see attached list**). If you are interested in this option, the investigator will provide you with additional information. If you opt for extra course credit as compensation, the Masters' student Tracey Patrick will handle this information confidentially and your instructor will not be informed of your extra credit until your course is already complete. If you are not interested in this option, or you are not enrolled in any of the eligible courses, you will be compensated with a \$10 Tim Hortons gift card

Physiological Measurements:

1. **Neck muscle function:** the strength of Sternocleidomastoid (SCM) and trapezius, (two neck muscles), will be recorded using surface electromyography (EMG). Prior to EMG electrode placement, standard preparations, including preparing the surface of the skin with standard EMG preparation tape to exfoliate the top layer of dead skin cells, and cleansing with alcohol will be performed. Surface recording electrodes will be placed over the SCM and trapezius muscles. Following electrode preparation, you will perform maximal voluntary contractions. These maximal voluntary contractions will require you to shrug your shoulders as hard as you can while a force transducer measures your strength, and to turn your head to the right and left and push as hard as you can against a force transducer
2. **Brain Activity:** a 64-electrode cap, which is non-invasive will be used to record brain activity during rest and a finger tracing task. The cap is placed on your head over your hair. We do need to apply electrode gel to your scalp which will need to be wiped off after the completing the measurement.

NOTE: This will require identifying landmarks on the body often covered up by a shirt or sweater. In order to access these landmarks we will need to have you in a loose fitted t-shirt, or a tank-top/sports-bra if you are comfortable.

Proprioception Measurements:

1. **Accuracy of your awareness of the position of your shoulder:** Participants will place their arm in a device made to assist with moving the shoulder joint into various positions:
 - a. Transverse protraction/retraction (moving your arm/shoulder blade forward and backwards with the elbow positioned at 90 degrees to the shoulder)
 - b. Humeral internal/external rotation. (Rotating the arm inwards and outward with the elbow positioned at 90 degrees to the shoulder; like you are throwing a ball)
2. **Head tilt, neck and shoulder range of motion (ROM):** a goniometer (which measures the degrees of movement) will be placed on your head and you will be asked to move in various ranges of neck and head movements

Before and/after photographs of head tilt and neck and shoulder ROM will be documented.

Risks and Benefits:

The surface EMG techniques have low risks such as the person getting a skin irritation from the alcohol swab or electrode gel. These are uncommon and not serious. You may also experience mild discomfort as your skin is prepared for the electrodes by shaving the skin with a razor, or lightly abrading with special tape, and then wiping the area with alcohol. The surface EMG techniques have low risks such as the person getting a skin irritation from the alcohol swab or electrode gel. If a skin irritation develops in response to the electrode gel or adhesive, the area will be cleaned immediately. It should be noted that these reactions are uncommon and the irritation is gone within a few hours due to the short time the electrodes are in contact with the skin.

Participation in this study, may cause mental-fatigue and potential boredom while performing the task, however, as a student this is not outside the normal risk associated with day-to-day life. Participation will take approximately 2.5-3 hours. Some participants could experience anxiety or stress as a result of their responses to the questionnaires. If this happens to you, please discuss any discomfort or distress with the researcher(s) who can help you find appropriate resources to help you.

Storage of Data/ Confidentiality:

All of your data files will be coded so that they can be kept in confidence and not directly linked to you. Your personal information will not be disseminated. All questionnaire data will be locked and stored in the Human Neurophysiology and Rehabilitation laboratory that only the research team will have access to. All electronic files will be anonymized, and stored on a computer that is locked in the Human Neurophysiology and Rehabilitation laboratory. While other lab group members will have access to this computer, your data: a) will only consist of number series' from which you cannot be identified, and b) will be recoded and removed of personal identifiers. Only frequency counts, means, and standard deviations calculated over many participants will be published. We will only include images of your head and neck posture where identifying features are covered, and if you have signed the second consent form to allow these images to be used.

Results Dissemination: The data from this research will be submitted to scientific conferences and peer reviewed journals. All published data will be coded so that you are not identifiable.

Right to Withdraw:

You may withdraw your consent at any time and discontinue participation without penalty. If you have any questions concerning the research study or experience any discomfort related to the study, please contact the researcher Tracey Patrick at tracey.patrick@uoit.net, Dr. Bernadette Murphy at Bernadette.murphy@uoit.ca, or Dr. Paul Yelder at paul.yelder@uoit.ca. Any questions regarding your rights as a participant, complaints or adverse events may be addressed to Research Ethics Board through the Research Ethics Coordinator – researchethics@uoit.ca or 905.721.8668 x. 3693. By consenting, you do not waive any rights to legal recourse in the event of research-related harm.

Participant Concerns and Reporting:

If you have any questions concerning the research study or experience any discomfort related to the study, please contact the researcher or the Principle Investigator (PI)

Tracey Patrick (researcher)

[905]-767-1325

tracey.patrick@uoit.net.

Dr. Bernadette Murphy (PI)

905-721-8668 extension 2778.

Bernadette.Murphy@uoit.ca

Any questions regarding your rights as a participant, complaints or adverse events may be addressed to Research Ethics Board through the Research Ethics Coordinator – researchethics@uoit.ca or 905.721.8668 x. 3693.

Thank you very much for your time and help in making this study possible. If you have any queries or wish to know more please contact:

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Please read the following before signing the consent form and remember to keep a copy for your own records.

1. I understand that taking part in this study is voluntary (my choice) and that I am free to withdraw from the study up until the end of the data collection session without a reason and that this will in no way affect my academic progress.
2. This consent form will be kept in a locked area in the Neurophysiology and Rehabilitation Research Laboratory at UOIT, Oshawa, Ontario for a period of seven years before being destroyed.
3. The data collected in this study will be coded so that it is confidential from the consent form and stored in a locked area at UOIT, Oshawa, Ontario for a period of seven years before being destroyed.
4. I have read and I understand the information sheet for volunteers taking part in the study. I have had the opportunity to discuss this study. I am satisfied with the answers I have been given.
5. I understand that I can withdraw any data I supply up to the completion of my final measurement session
6. I understand that my participation in this study is confidential and that no material which could identify me will be used in any reports on this study.
7. I have had time to consider whether to take part.
8. I know who to contact if I have any side effects to the study.

I give consent for the data from this study to be used in future research as long as there is no way that I can be identified in this research. (Tick one) **YES** **NO**

I would like to receive a short report about the outcomes of this study (tick one)

YES **NO**

Consent to Participate:

1. I have read the consent form and understand the study being described;
2. I have had an opportunity to ask questions and my questions have been answered.
I am free to ask questions about the study in the future;
3. I freely consent to participate in the research study, understanding that I may discontinue participation at any time without penalty. A copy of this Consent Form has been made available to me.

(Name of Participant) (Date)

(Signature of Participant)/ (Signature of Researcher)

APPENDIX B

Edinburgh Handedness Inventory

Please indicate your preferences in the use of hands in the following activities *by putting a check in the appropriate column. Where the preference is so strong that you would never try to use the other hand, unless absolutely forced to, put 2 checks.* If in any case you are really indifferent, *put a check in both columns.*

Some of the activities listed below require the use of both hands. In these cases, the part of the task, or object, for which hand preference is wanted is indicated in parentheses.

Please try and answer all of the questions, and only leave a blank if you have no experience at all with the object or task.

	Left	Right
1. Writing	<input type="checkbox"/> <input type="checkbox"/>	<input type="checkbox"/> <input type="checkbox"/>
2. Drawing	<input type="checkbox"/> <input type="checkbox"/>	<input type="checkbox"/> <input type="checkbox"/>
3. Throwing	<input type="checkbox"/> <input type="checkbox"/>	<input type="checkbox"/> <input type="checkbox"/>
4. Scissors	<input type="checkbox"/> <input type="checkbox"/>	<input type="checkbox"/> <input type="checkbox"/>
5. Toothbrush	<input type="checkbox"/> <input type="checkbox"/>	<input type="checkbox"/> <input type="checkbox"/>
6. Knife (without fork)	<input type="checkbox"/> <input type="checkbox"/>	<input type="checkbox"/> <input type="checkbox"/>
7. Spoon	<input type="checkbox"/> <input type="checkbox"/>	<input type="checkbox"/> <input type="checkbox"/>
8. Broom (upper hand)	<input type="checkbox"/> <input type="checkbox"/>	<input type="checkbox"/> <input type="checkbox"/>
9. Striking Match (match)	<input type="checkbox"/> <input type="checkbox"/>	<input type="checkbox"/> <input type="checkbox"/>
10. Opening box (lid)	<input type="checkbox"/> <input type="checkbox"/>	<input type="checkbox"/> <input type="checkbox"/>
TOTAL(count checks in both columns)	<input type="text"/>	<input type="text"/>

Difference	Cumulative TOTAL	Result
<input type="text"/>	<input type="text"/>	<input type="text"/>

Scoring:

Add up the number of checks in the “Left” and “Right” columns and enter in the “TOTAL” row for each column. Add the left total and the right total and enter in the “Cumulative TOTAL” cell. Subtract the left total from the right total and enter in the “Difference” cell. Divide the “Difference” cell by the “Cumulative

APPENDIX C

The Neck Disability Index

This questionnaire has been designed to give your therapist information as to how your neck pain has affected your ability to manage in everyday life. Please answer every question by placing a mark in the **ONE** box that applies to you. We realize that 2 of the statements may describe your condition, but please mark only the **ONE** box that most closely describes your current condition.

Neck Pain Intensity

- I have no pain at the moment.
- The pain is very mild at the moment.
- The pain is moderate at the moment.
- The pain is fairly severe at the moment.
- The pain is very severe at the moment.
- The pain is the worst imaginable at the moment.

Concentration

- I can concentrate fully when I want to with no difficulty.
- I can concentrate fully when I want with slight difficulty.
- I have a fair degree of difficulty in concentrating when I want to.
- I have a lot of difficulty in concentrating when I want to.
- I have a great, great deal of difficulty in concentrating when I want to.
- I cannot concentrate at all.

Personal Care (eg washing, dressing)

- I can look after myself normally without causing extra pain.
- I can look after myself normally but it causes extra pain.
- It is painful to look after myself, and I am slow and careful
- I need some help, but manage most of my personal care.
- I need help every day in most aspects of self-care.
- I do not get dressed, I wash with difficulty, and stay in bed

Work

- I can do as much work as I want too.
- I can only do my usual work, but no more.
- I can do most of my usual work, but no more.
- I cannot do my usual work.
- I can hardly do any work at all.
- I cannot do any work at all.

Lifting

- I can lift heavy weights without extra neck pain
- I can lift heavy weights, but it gives extra neck pain
- Neck pain prevents me from lifting heavy weights off the floor, but I can manage if they are conveniently positioned, for example on a table
- Neck pain prevents me from lifting heavy weights, but I can manage light to medium weights if they are conveniently positioned
- I can lift only very light weights
- I cannot lift or carry anything

Driving

- I can drive my car without any neck pain at all.
- I can drive my car as long as I want, with slight pain in my neck.
- I can drive my car as long as I want, with moderate pain in my neck.
- I cannot drive my car as long as I want, because of moderate pain in my neck.
- I can hardly drive at all because of severe pain in my neck.
- I cannot drive my car at all because of the pain in my neck.

Reading

- I can read as much as I want, with no pain in my neck.
- I can read as much as I want, with slight pain in my neck.
- I can read as much as I want, with moderate pain in my neck.
- I cannot read as much as I want, because of moderate pain in my neck.
- I can hardly read at all because of severe pain in my neck.
- I cannot read at all because of pain in my neck.

Sleeping

- I have no trouble sleeping.
- My sleep is barely disturbed (sleepless less than 1 hr).
- My sleep is mildly disturbed (sleepless 1-2 hrs).
- My sleep is moderately disturbed (sleepless 2-3 hrs).
- My sleep is greatly disturbed (sleepless 3-5 hrs).
- My sleep is completely disturbed (sleepless 5-7 hrs).

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Headaches

Recreation

<ul style="list-style-type: none"> <input type="radio"/> I have no headaches at all. <input type="radio"/> I have slight headaches that come infrequently. <input type="radio"/> I have moderate headaches that come infrequently. <input type="radio"/> I have moderate headaches that come frequently. <input type="radio"/> I have severe headaches that come frequently. <input type="radio"/> I have headaches almost all the time. 	<ul style="list-style-type: none"> <input type="radio"/> I am able to engage in all my recreational activities, with no neck pain at all. <input type="radio"/> I am able to engage in all my recreational activities, with some pain in my neck. <input type="radio"/> I am able to engage in most, but not all of my usual recreational activities, because of pain in my neck. <input type="radio"/> I am able to engage in few of my usual recreational activities, because of pain in my neck. <input type="radio"/> I can hardly engage in any recreational activities because of pain in my neck. <input type="radio"/> I cannot engage in any recreational activities at all because of pain in my neck.
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Vernon, H. and S. Mior, *The Neck Disability Index: A Study of Reliability and Validity*. Journal of Manipulative and Physiological Therapeutics, 1991. **14**(7): p. 409-415.