EXAMINING THE RELATIONSHIP BETWEEN CERVICAL ANTHROPOMETICS, HEAD KINEMATICS AND CERVICAL MUSCLE RESPONSES TO SUDDEN HEAD PERTURBATIONS IN COMPETITIVE ICE HOCKEY PLAYERS

By

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THE RELATIONSHIP BETWEEN CERVICAL ANTHROPOMETICS, HEAD KINEMATICS AND CERVICAL MUSCLE RESPONSES TO SUDDEN HEAD PERTURBATIONS IN COMPETITIVE ICE HOCKEY PLAYERS

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ABSTRACT

Sex differences in neck muscle characteristics may explain the higher incidence of concussions in female hockey players. The purpose of this study was to examine differences in cervical muscle characteristics between male and female hockey players when exposed to sudden head perturbations. Sixteen competitive ice hockey players (8 female) were measured for total neck volume (TNV) and head circumference/neck circumference ratio (HC/NC). Participants underwent multidirectional head perturbations (causing head flexion, extension, left and right lateral bend). Muscle onset latency, muscle activity and head acceleration data were collected before, during and after each perturbation. TNV and HC/NC showed weak relationships to head acceleration. Females displayed significantly greater head acceleration in left lateral bend and flexion despite having significantly shorter muscle onset latencies in flexion. Females displayed significantly more muscle activity in the reflex period of extension. These findings demonstrate sex differences in muscle activation and head acceleration. Further investigation of neck stiffness could place females at a greater risk of concussion.

Keywords: Perturbations, Head Acceleration, Muscle Activation, HC/NC ratio, Estimated TNV

DECLARATION

I, Chadwick Debison Larabie, declare that this thesis represents my own work except as acknowledged in the text, and that none of this material has been previously submitted for a degree at the University of Ontario Institute Of Technology, or any other University. The contribution of supervisors and others to this work was consistent with the UOIT regulations and policies. Research for this thesis has been conducted in accordance to UOIT's Research Ethics Committee.

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Finally, I would like to thank my girlfriend, Anessa Garcia, for sticking by my side through the good and bad and for believing in me every step of the way.

It was a true pleasure being part of the lab team here and it is an experience I will cherish forever. I have gained a profound appreciation for research and have the utmost respect and admiration for my supervisors.

Although this was a gradual learning process for me, my journey here allowed me to uncover new depths about myself. If I were to share advice to my future colleagues, I would recommend to make small consistent accomplishments regardless of how intimidating confusing or challenging the workload might seem. In the words of Aristotle, "*We are what we repeatedly do. Excellence, then, is not an act, but a habit*"

ABBREVIATIONS

ASR: Acoustic Startle Reflex

ANOVA: Analysis of Variance

BMI: Body Mass Index

CCR: Cervicocollic Reflex

Cm: Centimeter

CNS: Central Nervous System

COR: Center of Rotation

CSA: Cross Sectional Area

CTE: Chronic Traumatic Encephalopathy

EHI: Edinburgh Handedness Inventory

EMG: Electromyography

HC/NC: Head Circumference/Neck Circumference

K: Kilogram

IAR: Instantaneous Axis of Rotation

LLB: Left Lateral Bend

LSCL: Left Middle Scalene

LSCM: Left Sternocleidomastoid

LSPN: Left Splenius Capitis

MOI: Mechanism of Injury

MRI: Magnetic Resonance Imaging

PCS: Post-Concussion Syndrome

RLB: Right Lateral Bend

ROM: Range of Motion

RSCL: Right Middle Scalene

RSCM: Right Sternocleidomastoid

RSPN: Right Splenius Capitis

SDH: Subdural Hematoma

VCR: Vestibulocollic Reflex

OPERATIONAL DEFINITIONS

Cervical Susceptibility: The likelihood that the cervical region is more greatly influenced by a particular characteristic or attribute.

Concussion: Head injury with a temporary loss of neurologic function resulting from rapid head acceleration or impact. Synonymous with the term **mild traumatic brain injury.**

Head Acceleration: The rate of change of velocity of the head as a result of all forces (external forces and internal forces) acting on the head.

Risk Factor: Any attribute or characteristic that increases the likelihood of developing injury, in this case whiplash and concussion.

Neck Perturbation: A deviation of the neck from its regular state caused by an external influence.

Neck Stiffness: The ability of the neck to resist perturbation, which translates to a decrease in head acceleration.

Whiplash: Neck injury to muscle and ligaments due to rapid back-and-forth movement of the neck that affects acceleration and movement of the head.

THESIS OVERVIEW

Understanding the relationship between head and neck kinematics and how muscles coordinate motion of these segments is critical when investigating concussions and whiplash in competitive athletes. The neck acts as the base of support and is a factor which determines the amount of head movement in response to externally applied forces. Differences in the cervical region could result in different magnitudes and directions of head movements. Cervical variations could include physical changes in structure (skeletal geometry, musculature), or neurophysiological differences (proprioceptive, vestibular, reflex excitability) both of which affect the capacity for dynamic stabilization, excitability, timing of muscle onset and the amount of muscle activation which would subsequently formulate differential kinematic patterns of the head-neck segment in response to impact. With sufficient evidence, cervical variables that result in poor kinematic outcomes (increased susceptibility to whiplash or concussions) would be termed risk factors. Individuals who have certain risk factors may have increased susceptibility to cervical and/or head injury when exposed to unexpected perturbations. Incidence rates reveal a high concussion rate in ice hockey. This is especially concerning given that ice hockey has the largest representation of athletes involved in a high intensity sport in Canada. Within ice hockey, there is an epidemiological discrepancy between sex and concussion incidence. Females have increased concussion occurrence despite non-contact regulations. This could be due to differences in the cervical structure and physiology in females, which could translate to altered kinematic outcomes during sudden impact.

The following thesis is divided into five main sections:

1. Thesis Introduction
2. Literature Review
3. Manuscript
4. Thesis Conclusions
5. Appendices

CHAPTER 1: INTRODUCTION

INTRODUCTION

Concussions in sport have subtle signs and symptoms that often go unrecognized by athletes and coaches (Baugh et al, 2015). Other times, concussive symptoms can be ignored as they could jeopardize sporting opportunities like scholarships and playing time. This is especially concerning because athletes who undergo one concussion are subsequently more susceptible to future concussions (Bey and Oystick, 2009). In fact, delayed recovery of a concussion can result in death when a second concussive-like impact ensues while recovering from the first (Bey and Oystick, 2009). Athletes with a history of concussions increase their risk of neurodegenerative diseases, mild cognitive impairment and memory loss later in life (Gueskiewicz et al, 2003; Amen et al, 2011; De Beaumont et al, 2007).

The high speed, high intensity sport of ice hockey was the second most frequent sport played in Canada in 2010, representing an estimated 1.239 million Canadian athletes (Canada Heritage, 2010). In just regular season play, one study reported that approximately 10.7 concussions occurred per 1000 athlete exposures. In the same study, females reported 14.9 concussions per 1000 athlete exposures in comparison to 7.5 in males (Koerte, 2012). This dissociation between sexes could be due to many factors, including male reluctance to report injury (Spitzer et al, 1995), cultural and psychosocial factors. Nonetheless, females are more likely to acquire cognitive impairment, while also experiencing extended recovery times and a higher chance of post-concussive syndrome (PCS) (Broshek et al, 2005; Bazarian, 2013; Styrke et al, 2013).

One factor that could explain this discrepancy between sexes may be differences in cervical muscle activation and timing patterns, which are critical factors in resisting head

acceleration (Denny-Brown, 1941). Whiplash of the neck induces rapid translational and/or rotational movement of the head which can result in cell structure damage due to jarring of the brain (Drew et al, 2004). Greater neck stiffness when external forces are applied to a system (like checking in hockey), leads to increased robustness, or the ability to resist movement (Viano, 2007). Cervical variables that contribute to reducing head acceleration may help mitigate concussive injuries by limiting brain acceleration experienced at impact (Meaney and Smith, 2011). Hynes and Dickey (2006) suggested that the head and neck should be considered together when either concussions or whiplash were observed as the two were strongly related in a sample of ice hockey players.

Athletes that are able to prepare cervical responses pre-impact (Eckner et al, 2014) or react within a short period after impact (Schmidt et al, 2015) may be able to help dampen head acceleration. The human response to impact has been examined previously by administering low magnitude head (Schmidt et al, 2015; Tierney et al, 2005) and/or body (Blouin et al, 2003; Sacher et al, 2014) perturbations that evoke cervical muscle and head kinematic responses. Although this is not representative of high magnitude impacts experienced in hockey, it is the safest way to obtain insight into the neck's role in resisting head acceleration in response to external forces.

Before denoting sex differences in response to sudden head perturbations, the most important cervical variables that influence head acceleration need to be considered. Self-awareness is said to be a critical factor in ice hockey (CDC, 2013). Being aware of an imminent force creates time for the athlete to estimate the amplitude, timing and location of impact, thus changing head and body orientation or avoiding contact altogether. Siegmund et al (2003) showed that awareness variables affect the kinematic and muscular responses between aware and

non-aware subjects. Likewise, being aware of an incoming force gives an individual the opportunity to contract the cervical musculature, which has been shown to reduce head acceleration (Tierney et al, 2005). Interestingly, some studies that administer repeated perturbation protocols demonstrate less cervical muscle activity and increased head acceleration after successive trials (Blouin et al, 2003). This may be due to a habituation effect where the nervous system minimizes stress by adopting a neck stiffening mechanism (Blouin et al, 2003). This could also be due to risk compensation theory where athletes adjust their behavior to perceived level of risk (Hedlund, 2000; Schmidt et al, 2015).

When athletes are unware of imminent impact, neurophysiological systems (auditory, vestibular, and somatosensory) in association with cervical structures (muscles, ligaments) would be the primary mechanisms involved in reducing head acceleration. The acoustic startle reflex (ASR) has been shown to decrease muscle onset latencies when sufficient acoustic stimuli is provided (Blouin et al 2006; Yeomans and Frankland 1995). If a teammate shouts to warn of incoming impact this reflex may be activated. With the lack of auditory or visual awareness, Siegmund et al (2003) showed that subjects had larger paraspinal amplitudes and larger angular head acceleration than those who were not deprived of those senses. Thus, when impact is fully unanticipated, the cervicocollic reflex (CCR) and vestibulocollic reflex (VCR) may be the two prime reflex pathways involved in postural correction. The CCR response occurs as muscle spindles detect length change in the neck as the VCR uses the vestibular organs to sense the direction of head movement thus activating muscles to resist this movement (Keshner, 2009), Neck muscle reflexes have been shown to occur in less than 40ms as per head perturbation studies (Schmidt et al, 2015; Tierney et al, 2005). Furthermore, co-contraction of agonist and antagonist muscles may help stiffen the neck, but co-contraction has been shown to diminish as

continued perturbations are administered (Blouin et al, 2003). Reduced co-contraction may be an optimization strategy to minimize energy consumption of the central nervous system (CNS) (Blouin et al, 2003) when head acceleration can be sufficiently managed by reflexive control (Peng et al, 1995) and elastic region of muscles and ligaments in the neck (McCaw, 2014). In contrast, short cervical muscle onset latencies may not be able to efficiently increase neck stiffness in response to the speed and magnitude of concussive like impact. The involuntary reflexive attempt may generate too little muscle activity to have meaningful preventative effects.

Greater neck strength may be important in reducing head acceleration when expecting impact (Eckner et al, 2014), but strength alone, may not be sufficient when the incoming force is unexpected. Strength is affected by the time it takes for motor recruitment and rate coding to produce measurable muscle force (Cavanugh et al, 1979; Nishikawa et al, 2007), potentially making muscle onset and strength independent from each other. This may be why the relationship between neck strength and head acceleration is heavily debated in the literature (Eckner et al, 2014; Omdal 2015; Mihalik et al, 2011; Mansell et al, 2005).

With these collective principles, it is clear that the structure and function of the neck will have a large influence on head acceleration in ice hockey. Larger neck size generally reflects greater muscle cross sectional area (CSA) (Wittek et al, 2001; McNair et al, 2002) and individuals with a larger neck volume may require greater force magnitudes to induce head and neck acceleration thus compensating with a smaller muscle response to impact (Kreighbaum and Barthels, 1996). This ultimately could result in less tissue deformation and more neck stiffness. Alternatively, studies have not found a correlation between neck size and reduced head acceleration (Schmidt et al, 2015). Thus, head-neck ratios, such as the head circumference/neck circumference ratio explored by Vasavada et al (2008), could be a more noteworthy risk factor.

Individuals with a smaller neck in relative proportion to their head are prone to greater inverted pendulum loading (Stokes et al, 2000). This could translate to increased reliance on the cervical musculature for head stabilization, which could introduce muscular fatigue more quickly. This is why light-weight helmet designs are of great importance, so as not to inadvertently increase head weight.

However, it should be considered that relative head size and contact forces in female sports may offset some of these apparent differences. Regardless, this could suggest that females may be exposed to greater head and neck accelerations, which can lead to increased jarring of the brain and resultant hemorrhaging (Ommaya et al, 2002; Tencer et al, 2003). Unique sex specific factors in structure, musculature and neurophysiology of the cervical region have been found in numerous studies (Vasavada et al, 2007; 2008; Stemper et al, 2008; Tierney et al, 2005; Zheng et al, 2013). Vasavada et al (2007; 2008) and Stemper et al (2008) reported that women have smaller neck circumference, smaller vertebrae and more head mass per unit of neck muscle CSA than males. This could suggest that female cervical muscles are required to work at a greater percentage of maximum activation to stabilize the head. This could also help explain why concussive injury was more likely to occur in fatigued females (Shick et al, 2003). Zheng (2013) identified that neck volume and CSA were smaller in females. Likewise, Balkarran et al (2015) found that sternocleidomastoid volume was smaller in relative proportion to the neck in female ice hockey athletes. Interestingly, females have also been found to have shorter muscle onset latencies than their male counterparts and use a higher percentage of cervical muscle activation in response to sudden head loading, yet still experience greater head acceleration than males (Siegmund et al, 2003; Tierney et al, 2005). Thus, although females reacted faster and produced a higher amount of muscle activity, head acceleration was still greater than males. Tierney et al

(2005) attributed this difference to females having less isometric strength, head mass and cervical girth than their male counterparts, which ultimately translates to decreased neck stiffness.

These sex-related differences have yet to be investigated in varsity ice hockey athletes in response to an external force application and this may provide insight into the differences observed in concussion incidence between sexes. To the best of our knowledge, no other studies, to date, have conducted a head perturbation experiment comparing sexes in this particular athletic group. Similarly, factors such as neck strength are still heavily debated in the research (Schmidt et al, 2015; Gessel et al, 2007; Mihalik et al, 2010) and should be further explored. In addition, differences in neuromechanical variables most greatly influencing whiplash susceptibility remain inconclusive (Vasavada et al, 2008). Consequently, this study will focus on ice hockey players where player-to-player contact causes head injury despite sporting equipment and head contact penalties (Daneshvar et al, 2011; Marer et al, 2012). This research will outline the structure, musculature and neurophysiology of the cervical region as it relates to head acceleration. Cervical structure and function may be the largest predictive components for concussion rates between sexes in ice hockey. Unlike intentionally heading a ball in soccer, impact severity in hockey may be more largely derived from unexpected impact especially in the lateral directions (Hutchinson et al, 2013). This means that body checking or high velocity contact could cause direct head impact in the lateral, rear or frontal directions inducing head extension, flexion and right/left lateral bend. No previous studies have compared the neck muscle responses when perturbations are induced in a variety of directions for this particular athletic group. This thesis will address this knowledge gap.

PURPOSE AND HYPOTHESES

The purpose of this thesis was to better understand the effects of cervical anthropometrics, muscle activity and onset patterns on head acceleration between males and females during sudden externally applied perturbations. Such information may provide insight into the reason why female ice hockey players report more concussions than males. More specifically, five experimental hypotheses are proposed:

H1: Female varsity hockey players will have shorter muscle onset latencies than males when responding to multi-directional head perturbations.

H2: Female varsity hockey players will have greater relative muscle activity than males when responding to multi-directional head perturbations.

H3: Larger neck size and smaller head to neck ratios will correlate with decreased head acceleration.

H4: Female varsity hockey players will have greater head acceleration than males during sudden multi-directional head perturbations.

H5: Timing knowledge will increase cervical contraction pre-perturbation and cause earlier onset of muscle activity.

CHAPTER 2: LITERATURE REVIEW

SECTION 1: EPIDEMIOLOGY OF CONCUSSION IN SPORT

1.1 The Implications of Concussion in Sport

Concussion in sport is a serious matter which can be largely overlooked by coaches, competitive athletes and health care professionals. This is because the signs and symptoms of concussions are often subtle and not recognizable by the athlete (Khurana et al, 2012; Langlois et al, 2006). In fact, approximately 90% of concussions do not result in loss of consciousness (Broglio et al, 2011; Raleigh et al, 2009). Other times, concussion symptoms can be disregarded, especially when winning a game can be reflective of a scholarship or salary in varsity athletics or professional sports. A survey looking at 700 college players from 10 division 1 schools reported to have approximately six times more suspected concussions than diagnosed (Baugh et al, 2015). This is especially dangerous if an athlete sustains a second head injury while recovering from the first. Second impact syndrome could ensue which causes increased intracranial pressure, cerebral swelling, brain herniation and potential death. This is due to the brain's impaired ability to self-regulate blood volume when soft tissue contusions are healing (Bey and Ostick, 2009). Likewise, athletes who have a history of repeated concussions are not only more susceptible to future concussions but have a risk of neurodegenerative diseases later in life (Gueskiewicz et al, 2003). This can increase the risk of Alzheimer's dementia, memory impairment and even cause cognitive impairments such as chronic traumatic encephalopathy (CTE) (Amen et al, 2011; De Beaumont et al, 2007; Omalu et al, 2007). Iverson et al (2004) stated that "athletes with multiple concussions were 7.7 times more likely to demonstrate a major drop in memory performance than athletes with no previous concussions"

1.2 Concussive Incidence in Ice Hockey

Statistics Canada (2010) revealed that an estimated 3.3% of adolescents aged 12-19 and 2.1% of adults aged 20-64 suffered either a concussion or brain injury from 2009-2010. A study involving five emergency departments in Alberta, Canada found that 42.9% of head injuries in persons aged 15 to 19 were derived from sports (Kelly et al, 2001). In Canada, ice hockey was the second most frequent sport played in 2010 (second to golf) and represented an estimated 1.239 million persons (Canada Heritage, 2010). Of the top five most frequent sports played (golf, ice hockey, soccer, baseball and volleyball) in Canada, ice hockey appeared to have the second highest concussion rates (next to soccer) with one study estimating 10.7 concussions per 1000 athlete exposures for men and women combined in just regular season play (Canada Heritage 2010; Gessel et al, 2007; Koerte, 2012). The physical nature of ice hockey, played at an incredible speed, makes it a dangerous sport where player-to-contact and player-to-player collisions pose an imminent threat (Marer et al, 2012). Often times, these collisions directly impact the head and cause concussions despite head contact penalties (Daneshvar et al, 2011). According to Covassin et al (2003) concussions among intercollegiate ice hockey players accounted for 6.3% of practice injuries and 10.3% of game injuries. Concussions have devastating acute and chronic complications and pose a significant threat to the large representation of ice hockey players in Canada.

1.3 Epidemiological Discrepancy between Sexes for Incidence of Concussions in Ice Hockey

Within ice hockey, females are often thought to experience less concussions than males, due to non-contact regulations, but epidemiological evidence would suggest otherwise.

According to information reported to the Hockey Internal Scouting Services from 1998-2004, females reported more concussions than males (Hootman et al, 2007). More recently, other studies have also reported higher concussion rates in females (Agel and Harvey, 2010; Dick, 2009). In fact, Koerte et al (2012) found that female varsity ice hockey players experienced 14.93 concussions per 1000 athlete exposures in comparison to 7.5 concussions per 1000 athlete exposures in males. Psychosocial factors among other variables could attribute to this incidence discrepancy (i.e. male reluctance to report injury). Even so, female athletes who experienced a concussion were documented to acquire cognitive impairment more frequently, have extended recovery times and were reported to be 1.67 times more likely to have sustained post-concussion syndrome (PCS) symptoms (Broshek et al, 2005; Bazarian, 2013; Styrke et al, 2013). These epidemiological findings suggest that concussions are more of a threat to female ice hockey players; not only due to the higher incidence rate but due to the poorer recovery projection.

SECTION 2: WHIPLASH INJURY MECHANISM

2.1 Whiplash Relatedness

Hynes and Dickey (2006) showed a strong correlation between whiplash and concussions by conducting a prospective cohort observational study of twenty hockey teams in which all 13 players who experienced either an indirect (whiplash) or direct head trauma (blow to head) reported concussive symptoms. The study concluded that there was a strong association between whiplash and concussion in ice hockey, thus both should be evaluated when considering concussions. Whiplash is defined as a flexion or hyperextension injury of the neck caused by rapid acceleration/deceleration that generally occurs with impact but not always e.g. infant whiplash shake injury syndrome (Ommaya et al, 2002). This can cause pressure on nerve root ganglia, stretching of facet capsules or damage to facet articular cartilage, especially at C4-C6 which are known to have the highest rates of pinching at peak head acceleration (Ommaya et al, 2002; Tencer et al, 2003). Whiplash of the neck induces rapid translational or rotational movement of the head. This can result in brain cell structure damage and brain hemorrhaging which is a primary diagnosis of a concussion (Drew et al, 2004). With this knowledge, concussion susceptibility is largely influenced by whiplash of the neck.

2.2 Mechanism of Injury

The mechanisms of injury (MOI) for concussions in sports related contact are caused from direct trauma (e.g. blow to the head or indirect trauma via rapid acceleration), rotation or deceleration of the head from a distal impact to the body (McCrory et al, 2013). Hutchinson et al (2013) found that initial contact to the head resulted in 68% of concussions in the National Hockey League (NHL) with 47% of contact induced by elbows, shoulders and gloves to the

lateral aspect of the head (Figure 1). Sagittal and transverse head accelerations accounted for 39% of concussions (Izraelski, 2014). The athlete causing the collision was typically heavier than the player hit (65%) and the player position most susceptible to concussions was forward facing and represented 65% of all cases (Hutchinson et al, 2013).

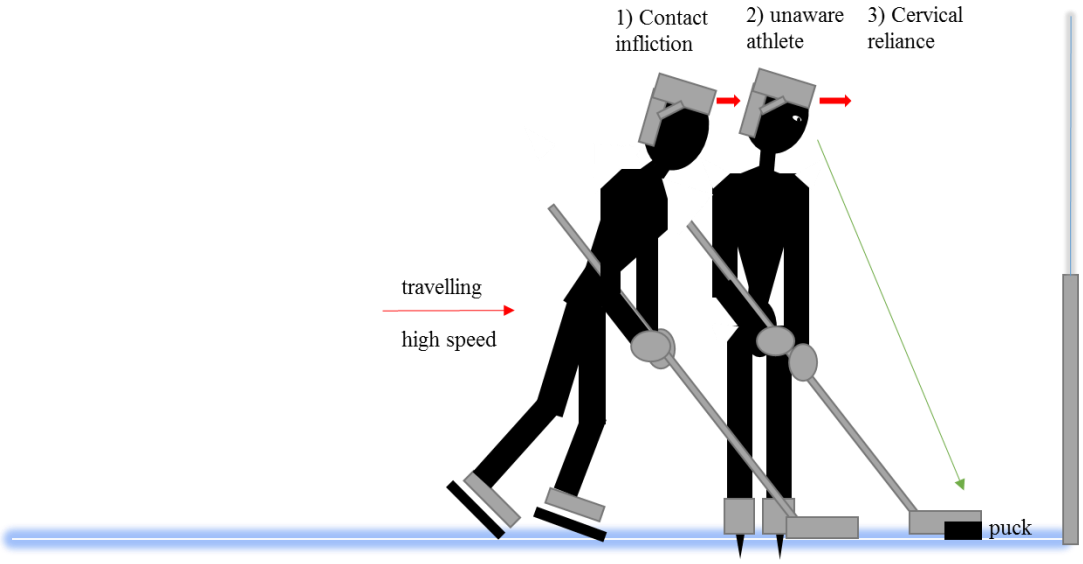

Figure 1. Imminent impact transfer to lateral aspect of the head

2.3 Head Acceleration

There is considerable evidence that concussive injuries are primarily caused by the inertial acceleration experienced by the brain at impact (Meaney and Smith, 2011). Pressure recordings observed how peak accelerations were correlated to peak pressures within the brain when subjected to impact (Gurdjian et al, 1961; Thomas et al, 1966). Transient increases in brain pressure were associated with increases in neurological dysfunction (Gurdjian et al, 1954). The patterns of strain within the brain are dependent on the direction of acceleration. Coronal plane (side-to-side) accelerations at high magnitude can cause traumatic axonal injury and are most

likely to result in loss of consciousness, whereas, sagittal plane accelerations usually results in subdural hematoma (SDH) (Goldsmith et al, 2004; Generalli, 1982). The term coup refers to brain contusion occurring at the site of impact whereas countrecoup affects the opposite part of the brain from where impact occurred. This player-to-surface or player-to-player head impact forces the brain to rebound from either side of the skull implying a countrecoup mechanism which often results in brain hemorrhaging and temporary unconsciousness (McCrory et al, 2013). The variables that impact the neck's ability to resist deformation would have the potential to mitigate this rapid head acceleration.

2.4 Neck Stiffness

The most important constant reducing whiplash of the neck is neck stiffness (Viano, 2007). The neck's ability to resist deformation in response to an applied force constitutes neck stiffness. The more rigid the spinal segments and tissues are (both passive and active structures), the more difficult to deform (Viano, 2007). Ideally, when impact ensues, an athlete would want a more 'robust' cervical region so that neck deformation through applied forces like body checking could be minimized. This would translate to a decrease in rapid head acceleration, the primary mechanism causing concussions (McCrory et al, 2013). A lack of neck stiffness can increase neck movement upon impact, thus increasing muscle length deformation and passive tension in the muscles. This can translate to larger neck angular displacement and could increase reciprocal forces in the rebounding phase of whiplash (Nordin and Frankel, 2000). Despite this, having a rigid neck when direct impact to the head occurs may prevent absorption of the brain at impact. If an individual were to move in accordance with the force administered it may lessen the severity of the applied load.

Neck stiffness is dependent on numerous factors including the reflex response pattern, mechanical properties of the neck and neck strength (Simoneau et al, 2008; Vibert et al, 2001). These components are strongly linked to musculo-tendinous stiffness which is the resultant stiffness of muscle groups crossing the joint and associated neuromuscular patterns between agonist and antagonist muscles (Cholewicki et al, 1996).

SECTION 3: SYSTEMS INVOLVED IN MECHANICAL JOINT STABILITY

The neck serves as the primary canal connecting the head to the body and acts as the base of support to the head. In order to keep the head stabilized, the passive (ligaments), active (muscle) and neural control systems (CNS and nerves) work collectively to centralize the head when at rest and in response to external disturbances (Panjabi, 1992). To do so, approximately 80 muscles work to maintain dynamic stabilization of the head through reflexive and spring-like mechanisms that act on the cervical structure to prevent excessive movement (Borst et al, 2011). Together, they create a unique arrangement that is particular to not only sex but to each individual (Vasavada et al, 2007). The understanding of a system's ability to respond to external disturbances is critical when observing sudden loading events.

3.1 Skeletal Structure

In order to better understand the active and neural systems that help stabilize the head it would first be important to observe the structure on which they act. The cervical spinal column typically consists of seven cervical vertebrae that house the enlargement of the spinal cord. The *Atlanto-Occipital* synovial joint forms the attachment between the atlas and occipital bone thus permitting flexion and extension upon the dens. The *Atlanto-Axial* pivot joint is responsible for the main articulation between the atlas and axis and allows right and left lateral rotation upon the odontoid process. The remaining cervical vertebrae (C3-C7) are facet joints that do not initiate joint sequence, but move in accordance with the Atlanto-Occipital and Atlanto-Axial joints through slight pivots (Tortora et al, 2009). Refer to Figure 2.

The mobility of the cervical column is larger than the thoracic, lumbar and sacral regions (Marieb and Hoehn, 2010). In fact, the neck can undergo a large range of motion in the sagittal, axial and coronal planes which corresponds with neck flexion, extension, and left/right lateral bend of 140° in Flexion/Extension and 180° of rotation (Abrahams, 2008; Marieb and Hoehn, 2010).

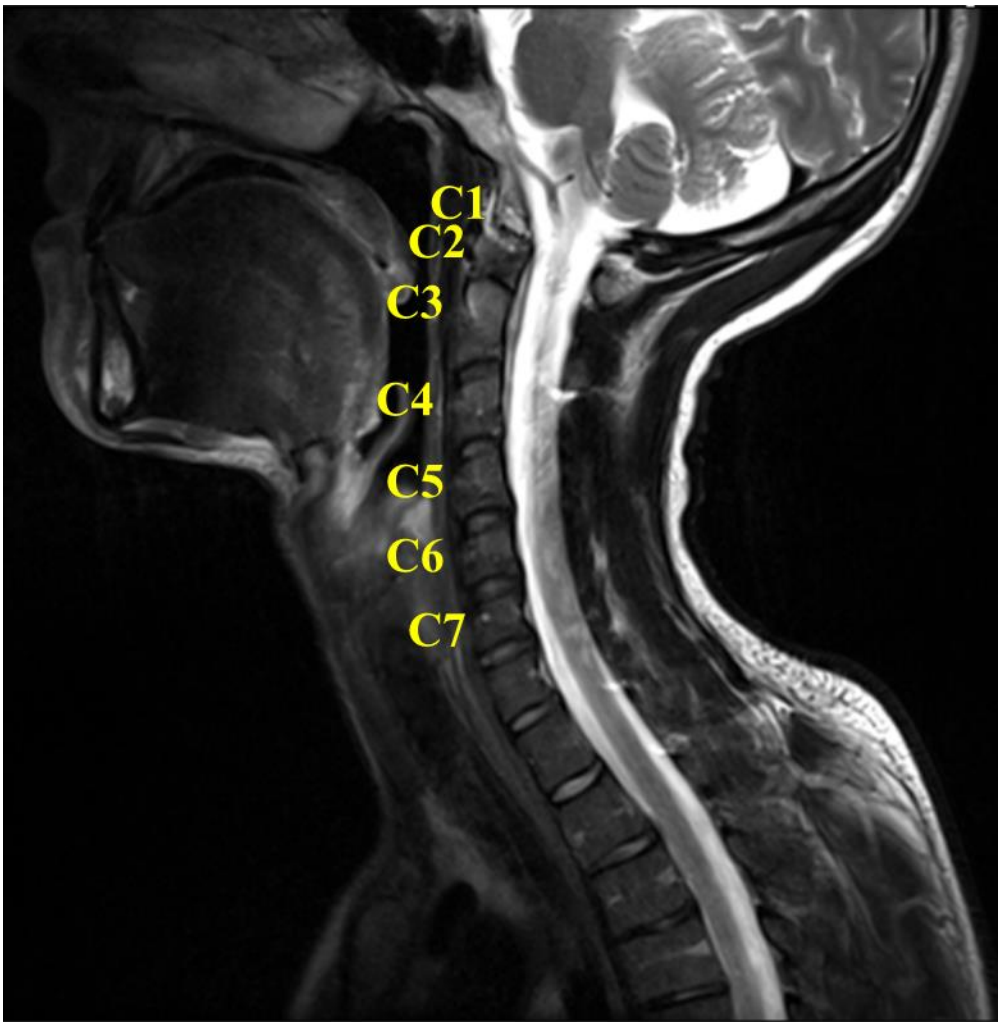

Figure 2. Sagittal slice of the cervical spinal column by Magnetic Resonance Imaging (MRI)

Fibrocartilaginous intervertebral discs lie between the vertebral bodies (except C1-C2) with a soft central component known as the nucleus pulposus and a stiffer outer ring known as

the annulus fibrosis. These discs can be compressed and act as shock absorbers that protect the vertebrae from excessive pressure (Abrahams, 2008). The cruciform, alar and apical ligaments help keep the axis and atlas in place while longitudinal ligaments resist excessive flexion (anterior longitudinal) and extension (posterior longitudinal, interspinous and nuchal ligaments). The nuchal ligament helps support the weight of the head and has a high number of elastic fibers which allows for greater stretch in neck flexion. In fact, all these ligaments are composed of collagen fibers which under tension allows them to stretch before returning to original form (Abrahams, 2008). With this information, the ligaments provide stability through reciprocal forces of passive stretching and have increased elasticity near the end range of motion (Harms-Ringdahl, 1986; McCaw, 2014). However, once tissue strain reaches a particular point, tissue stress may lead to damage (refer to figure 3).

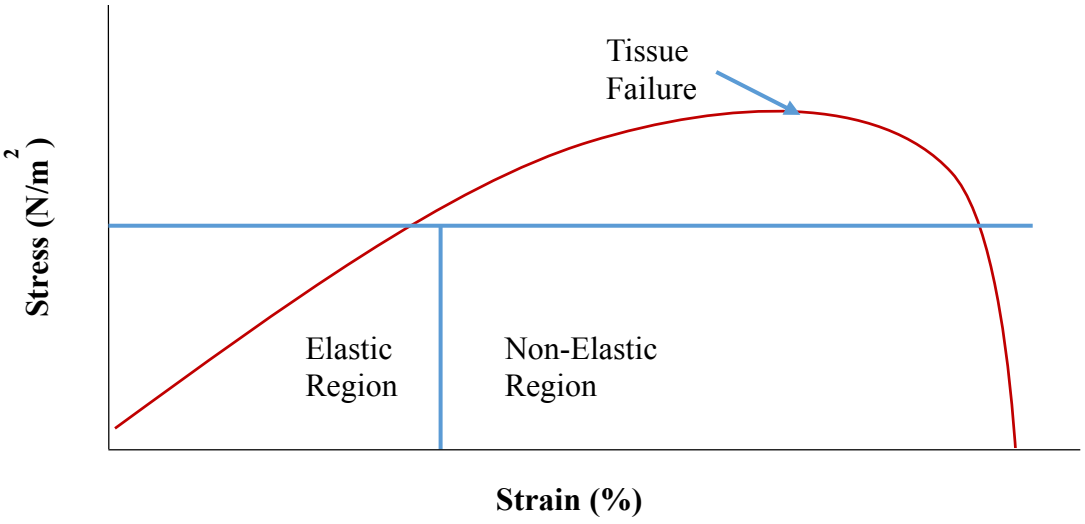

Figure 3. Stress-Strain Relationship

3.2 Cervical Musculature

The mechanical nature of a muscle suggests that muscles are able to respond to an external disturbance by taking advantage of spring and damper like properties (Hill, 1950). The musculature is often considered the essential contributor to mechanical impedance, joint rotational stiffness and overall spine stiffening (Cholewicki and McGill, 1996; Potvin and Brown, 2005). The primary muscles involved in responding to larger external disturbances are the prime movers; the major proponents influencing head and neck movement (Tortora et al, 2009). These muscles are usually located superficially and are activated more greatly in certain directions due to their specific muscle origin and insertion points in the cervical region (Abrahams, 2008; Ólafsdóttir et al, 2015). The sternocleidomastoid (SCM) muscles are powerful flexors and work in conjunction with the scalenes (Abrahams, 2008). Studies investigating the whiplash response, often observe that the SCM as a prime mover during rear-perturbation studies (Siegmund et al, 2003; Schmidt et al, 2015; Tierney et al, 2005; Sacher et al, 2014). The SCM run bilaterally originating at the sternum and clavicle and insert on the mastoid process. Simultaneous contraction of the SCM causes the muscle fibers to be pulled downwards creating neck flexion while uniform contraction of one muscle aids in lateral flexion of the neck (Tortora et al, 2009). The scalenes (SCL) originate at the transverse process of C2-C7 and insert on the first two ribs thus causing lateral flexion, amongst other functions (Abraham et al, 2008; Tortora et al, 2009). The splenius capitis (SPL) originates at the spinous process of the seventh cervical vertebrae to the fourth cervical vertebrae and inserts on the occipital bone and mastoid process to help with extension and lateral bending (Tortora et al, 2009). Simply put, agonist muscles cause motion, synergist muscles assists with motion and antagonist muscles oppose motion by resisting against the prime mover (Tortora et al, 2009). Muscle co-activation or co-contraction occurs

when a muscle is activated simultaneously with an agonist muscle and such mechanisms have been shown to help stiffen and stabilize a joint (Holmes and Kier, 2012). This is significant because when muscles undergo sudden unexpected disturbances (or perturbations) if muscle co-contraction can provide sufficient joint stiffness than muscle could resist accelerative forces.

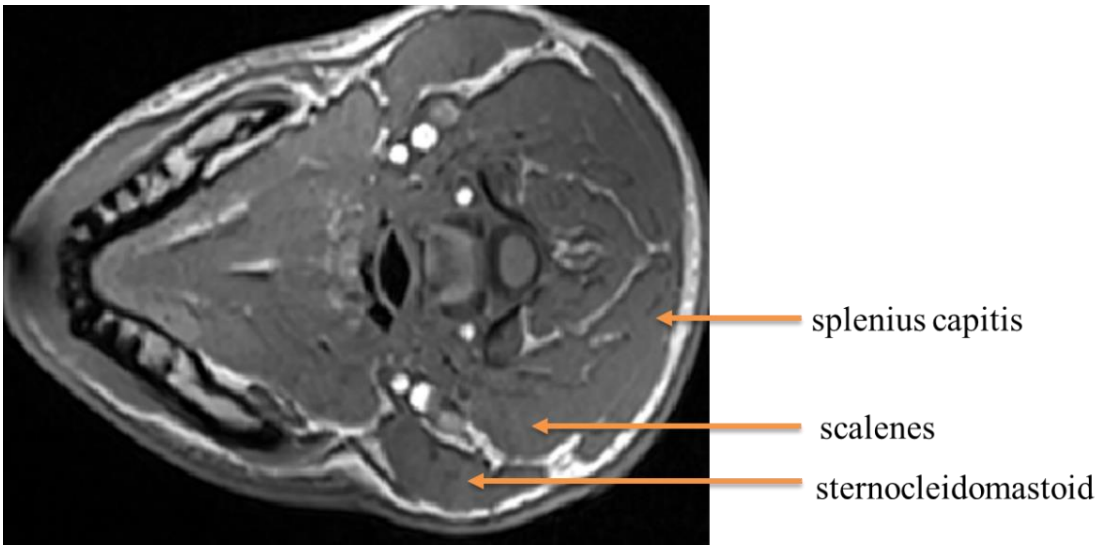

Figure 4. Axial slice of the cervical spine by MRI.

3.3 Neuromuscular System

The neuromuscular system regulates muscle contraction and maintains dynamic stabilization of the head. It gains information from proprioceptive signals like mechanoreceptors which helps coordinates activation of the cervical musculature. Sensory feedback provides a critical link to communicate mechanical information to the CNS accompanying the evolving movement for ongoing regulation (Nishikawa et al, 2007). It has been advocated that force and length feedback as per muscle spindles and Golgi tendon organs could help regulate muscular stiffness and promote stability (Nichols et al, 1976). Muscle spindles are sensory receptors

located within the belly of a muscle that detect length change and rate of length change. Upon lengthening, action potentials generated by alpha motor neurons interact with excitatory and inhibitory interneurons in the spinal cord. Upon the excitatory interneuron synapse, the muscle being stretched is activated eccentrically and produces tension while lengthening causing the stretch reflex (McCaw, 2014). Golgi tendon organs oppose the stretch reflex (i.e. neurons inhibit the contracting muscle) while activating the antagonist muscle. At the interneuron, inhibitory effects reduce activation of the motor units in the muscle developing tension as per the inverse stretch reflex while excitatory synapses cause the antagonist muscle to resist the joint action of the primary actuator (McCaw, 2014).

SECTION 4: FACTORS AFFECTING NECK PERTURBATION

4.1 Perturbation type and mechanism of delivery

Before denoting cervical differences between sexes it would first be important to identify which variables increase neck stiffness. To understand this, we would have to examine the anatomical and neurophysiological aspects of the cervical region and their biomechanical relationship in decreasing head acceleration. The biggest challenge in doing so is that experimentally, we can only draw insight into head acceleration by administering small neck perturbations. In addition, many of the experiments to date utilize different perturbation delivery mechanisms. These different delivery mechanisms can be broadly categorized as, load drops that perturb the head (refer to figure 5) and second by sled accelerations that perturb the body (refer to figure 6). Load drops generally draw insight into head impacts via sporting mechanics, while sled accelerations are typically used to develop insight on vehicle collisions. Both can be used to understand head acceleration but, mechanically can be very different. When relating to ice hockey, sled accelerations would act as indirect contact to the body and load drops would act as direct contact to the head. It's important to note that these two types of contact affect cervical neurophysiology differently. One being a bottom-up approach where primary detection occurs from the body. And the other a top-down approach where primary detection occurs from the head. With both of these mechanisms, muscle activation from the body i.e. postural control from torso, would influence resultant stiffness of the neck. To best account for this, studies involving both mechanisms typically lock body position so that only the neck is free to move which standardizes the methodology across subjects and also helps minimize joint reaction forces to other segments. In addition, this can simplify stability and stiffness calculations as the multi-

segmental muscles involved are considered at the primary joint of interest. During sled protocols, low magnitude impacts can occur from various sled velocities including 4km/hr to 8km/hr (Linder et al, 2008; Blouin et al, 2003). During load drop protocols, studies have been conducted anywhere from 1pound to 2.5% of a the participants body mass (Blouin et al, 2003; Tierney et al, 2005; Schmidt et al, 2015; Siegmund et al, 2009;). These neck perturbation devices provide insight into neck muscle activity and kinematics during sudden disturbances, which can reflect participant responses in real-life situations without risking injury.

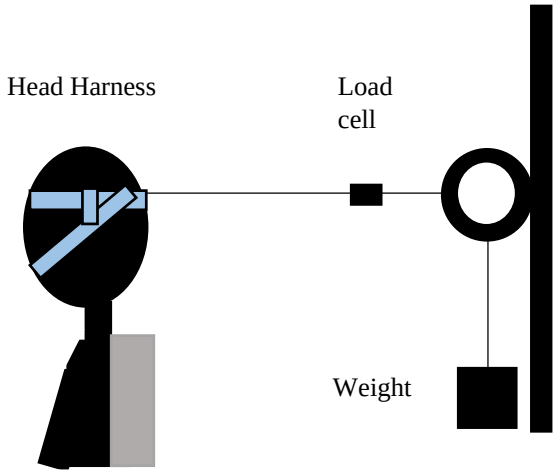

Figure 5. Head Perturbation Device

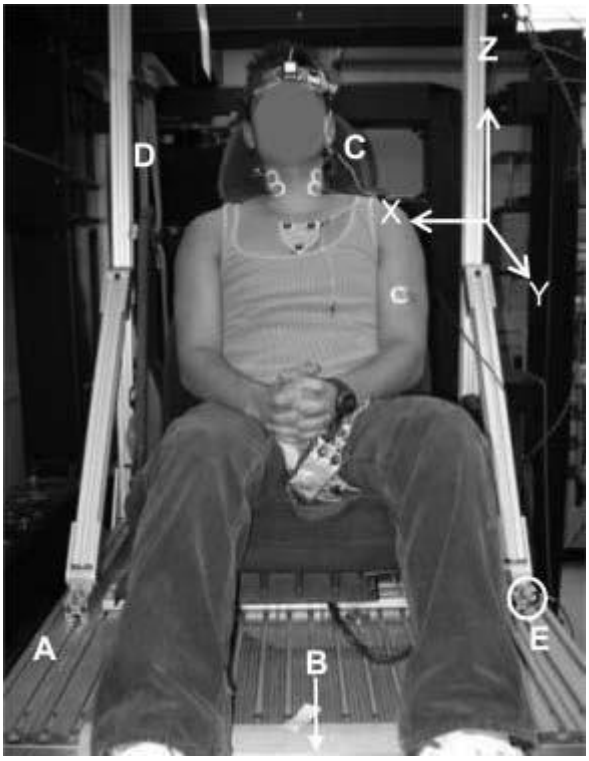

Figure 6. Sacher Sled Mechanism

Extracted from Sacher (2014). Figure 1. Subject and Experimental Apparatus

4.2 Defense Mechanisms used in Ice Hockey

Impact awareness in ice hockey is crucial to avoiding head and neck injury. The "Heads up Hockey" program (2010) is a safety initiative for all hockey players, where keeping the head up can reduce the chance of sustaining a neck injury. Having knowledge of impending impact would allow athletes to estimate the magnitude, location and timing of the external forces and could enable sufficient time to orientate head and body position and for the opportunity to contract cervical musculature (Mihalik et al, 2011; Eckner et al, 2014). These defense mechanisms rely on the ability of the visual system to gather information about the external environment and the central nervous systems ability (CNS) to integrate sensory information from head position (Abrahams, 2008). Based on this, a motor plan is executed, which contains

specifics of future movement that can shift head and body orientation (Rossetti, Desmurget and Prablanc, 1995). Being unaware of impact would inhibit the ability of athletes to invoke cervical preparatory techniques, thus relying entirely on the cervical structure (tissues, muscles, neck size) and reflex reactions of the neck muscles (Siegmund et al, 2003).

4.3 Levels of Awareness

Different awareness measures do not always affect the way one responds to a perturbation. Siegmund et al (2001) showed that individuals who were aware of the amplitude of incoming force did not have different muscular or kinematic responses than to those who were not aware. Likewise, being aware of an imminent load drop, but not knowing the precise timing of impact, did not affect muscular or kinematic responses compared to those who were given a countdown (Siegmund et al, 2003). This demonstrates that if one is expecting a perturbation, the addition of knowing the amplitude or timing of the imposed stimulus does not affect the cervical response at low magnitude. But this is not true of whiplash in hockey players as Di Fabio (1990) showed that stimulus intensity altered reflex postural responses. Siegmund et al (2003) concluded that the subjects muscle and kinematic responses were not representative of high impact situations due to the low magnitude impacts administered. Subjects who were completely deceived however (not knowing a perturbation would occur) did exhibit differences, where male cervical paraspinal amplitudes were 260% larger and angular head accelerations were 180% larger during sudden head flexion (Siegmund et al, 2003). Thus, unless a subject is completely unaware that a head impact is coming, there may not be an exaggerated response demonstrated in ‘unexpected’ conditions.

4.4 Habituation Effects of Repeated Perturbations

During some neck perturbation experiments, it was noted that subjects may have adapted to the repeated perturbations (Schmidt et al, 2015; Blouin et al, 2003). Some individuals experience decreased muscle activation and increased angular and linear accelerations of the head to perturbations (Blouin et al, 2003; Siegmund et al, 2003). This habituation effect was well demonstrated by Blouin et al (2003), where subsequent platform accelerations after the initial trial resulted in a combination of feedforward and feedback control. It was noted that subjects may have become resistant to the perturbations, adapting a feedforward strategy. It was concluded that the nervous system preferred to minimize stress rather than adopt a neck stiffening mechanism. Although repeated exposure showed a learning effect in the nervous system, it is not conducive to a better preventative strategy. With that said, Tierney et al (2005) showed that physically active males and females who contracted cervical musculature before external force was applied to the head had decreased head accelerations. This was demonstrated in the expected condition and shows that that contracting cervical musculature may lessen resultant head injury after repeated trials.

4.5 Risk Compensation Theory

Some authors have proposed that this habituation is due to risk compensation theory where athletes adjusted their behavior to perceived level of risk (Hedlund, 2000). Schmidt et al (2015) proposed that football players may have been using risk compensation when high performers (athletes with cervical variables that were thought to decrease head acceleration) had higher head accelerations than low performers in response to low magnitude head impacts. Thus, if the same magnitude of impact is considered safe to some athletes and dangerous to others,

different cervical and kinematic responses would occur as part of individualized risk behavior (Hedlund, 2000).

4.6 Effective Cervical Response to Impact

The precise mechanism behind the most effective way to respond to head impact is unknown. Kinematic responses that can help decrease the force of concussive injury needs to be more greatly observed. Irrespective of this, properly anticipating impact in ice hockey may be a critical aspect in concussion outcome. Self-awareness techniques in part with repeated experience reacting to potential concussive-like impact may decrease risk-potential. When expecting a body check, athletes may have the ability to couple the neck and torso so that the impact is minimized. Viano (2007) proposed that effectively grouping the head and torso would help link the segments together to act as one lever, thus decreasing whiplash effects.

SECTION 5: NEUROPHYSIOLOGICAL MECHANISMS INVOLVED IN HEAD IMPACT

Head impact in ice hockey affects all the neurophysiological systems (auditory, vestibular, visual and somatosensory) in an effort to maintain dynamic stabilization of the head. Physiological mechanisms that are able to react within 50ms (Laksari et al, 2015) after impact may be able to effectively aid the inertial segment of the whiplash mechanism and help dampen accelerative forces on the brain. Reflexes that exceed this time period may not provide much compensatory support to mitigate a concussion. Producing a cervical voluntary response to unexpected impact for instance would involve the motor and somatosensory cortices (Barker and Barassi, 2008) and may take too long to sufficiently aid in reducing brain damage. Reflexive responses are generally mediated by spinal and brainstem circuits (Ito et al, 1997; Blouin et al, 2007). The systems that comprise these specialized reflexes will now be examined in greater detail.

5.1 Visual System

Although the eyes can gather information regarding head orientation during head impact, Siegmund et al (2009) demonstrated that visual conditions (eyes open and closed) produced similar responses in the neck muscles during whiplash-like perturbations. If the location and magnitude of impact is known and if there are no visual cues that denote onset timing, then the eyes would have little to no effect in reducing head acceleration during unexpected perturbations. That being said, the vestibular-ocular reflex (VOR) ensures gaze stabilization is fixed and although head movements are rapid, the visual world remains unaltered (Oliveria et al, 2012). The VOR occurs by vestibular detection of head movement which sends an excitatory signal to

one side of the extraocular muscles and an inhibitory signal to the other side (Tortora et al, 2009; Barker and Barassi, 2008). This reflex allows for an individual to monitor positional sense despite external disturbances that may offset the visual field.

5.2 Auditory System

The acoustic startle reflex (ASR) has shown to decrease muscle onset latencies when sufficient acoustic stimuli is provided (e.g. loud sound in any audible range of over 85 dB) (Blouin et al 2007; Yeomans and Frankland 1995). Blouin et al (2006) compared subjects who were exposed to a whiplash-like perturbation with and without the involvement of a loud auditory signal. The group receiving the auditory stimulus had shorter muscle onset latencies and increased posterior neck muscle activity. In humans, the ACR can exhibit electromyographic (EMG) changes of the cervical muscles within 9 ms of the auditory stimulus (Gulinello, ASR Protocol, pdf.). Davis et al (1984) proposed that this neural pathway was an arrangement of synapses between the cochlear root neurons; neurons of the reticularis pontis caudalis and motor neurons in the spinal cord are associated in this reflex circuit. This is also part of the startle response, which can result in bilateral muscle activation of the SCM (Britton et al, 1991). This is just one of the many general behaviors observed in the startle response which is common to all mammalian species (Britton et al, 1991). This gives reason to the importance of verbal communication in ice hockey, especially when pre-warning teammates of incoming impact.

5.3 Vestibular System

The vestibulocollic reflex (VCR) is activated upon vestibular organ detection of head motion. Upon head tilt, the semicircular canal systems and otolith organs (utricle and saccule) in

the inner ear are stimulated (Abarahams, 2008; Oliveria et al, 2012). The semicircular canal system provides information on head rotation due to viscous fluid that displaces the fine hairs of the receptor cells. Likewise, the otolith organs provides position of the head relative to gravity by exerting pressure on nerve endings (Tortora et al, 2009; Oliveria, 2012). This mechanism activates the vestibular nerve which transmits pulses to the vestibulospinal tracts to the spinal cord initiating a postural reflex (Morningstar et al, 2005; Ito et al, 1997). When vestibular input is impaired, erroneous visual cues have a greater influence on postural sway and can severely affect postural changes if one's environment appears to be moving (Redfern et al, 2001). Horak et al (2002) compared subjects with bilateral vestibular loss to controls and found that head displacements were less prominent in controls as Creath et al (2002) seconded this motion suggesting that avestibular subjects had larger center of mass variability. Despite this, the VCR was not shown to cause shorter onset latencies or higher muscle activation in perturbation studies examining subjects with damaged vestibular apparatuses in comparison to healthy controls (Forssberg and Hirschfeld, 1994).

5.4 Somatosensory System

During direct impact to the head, proprioceptive pathways of the neck are the first detectors of head movement. The cervicocollic reflex (CCR) is activated upon proprioceptive input, sensing muscle length change in neck position (Peng et al, 1995). When a muscle spindle is stretched, it sends an immediate signal to the spinal cord and back to the muscle, which responds by muscular contraction (Ito et al, 1997). The CCR works synergistically with VCR with the utilization of spatial and body coordinate systems (Keshner et al, 2009). The VCR may be better at establishing cervical responses to low frequency neck responses than the CCR (Peng

et al, 1995). Nonetheless, both the VCR and CCR can increase damping during high-energy motion. Any muscle activity present prior to head impact would affect the response of the VCR and CCR as cervical contraction for instance could limit head motion and resultant muscle length changes.

SECTION 6: CERVICAL RISK FACTORS FOR CONCUSSION

6.1 Muscle Onset Latency

Shorter muscle onset latencies are thought to help decrease head acceleration by initiating muscle activity earlier in the disturbance, such that sufficient muscle contraction can limit head motion. However, cervical muscle onset timing and head deceleration are independent from each other. Recent head perturbation studies observing muscle activity of the SCM in forced flexion and extension perturbation protocols have observed onset latencies between 27 – 45ms (Tierney et al, 2005; Schmidt et al, 2015). Yet, Snyder et al (1975) showed that peak head deceleration as a representation of peak muscle force exceeded 130ms for both flexor and extensor muscles. Thus, although muscle onsets can occur quickly, the time to head deceleration differs for each individual. Cervical variables like muscle size and muscle strength could alter the sufficient muscle contraction needed to decrease head motion. Consequently, even though shorter muscle onset latencies appear to be advantageous, their significance in contributing to head deceleration remains inconclusive. The contribution of muscle onset timing has been debated in various studies. Snyder et al (1975) reported that muscle onsets did not contribute to neck resistance while Reid et al (1981) concluded differently. Such discrepancies may be due to factors such as stimulus intensity, which is known to alter the response amplitude of the cervical musculature (Brault et al, 2000). Recent head perturbation studies have indicated that some individuals have increased head acceleration despite having shorter muscle onset latencies (Tierney et al, 2005; Schmidt et al, 2015). Although shorter muscle onset latencies may contribute to deceleration in low magnitude head perturbations, their role in preventing concussive like impacts in ice hockey may be minimal and needs further attention.

6.2 Cervical Strength and Muscle Size

Stronger musculature has been shown to generate torque more rapidly, have increased muscle cross-sectional area, generate larger forces and have greater stiffness (Thelen et al, 1996). Cervical stiffness may also be largely dependent on the co-contraction of musculature. The measure of co-contraction is significant because it relates the ongoing dynamics of the antagonist muscles resistance against the movement carried out by agonists (Grondin and Potvin, 2009). Larger opposing forces operating on a joint will lead to increases in joint impedance (Hogan, 1984). Holmes and Kier (2012) showed this relationship with sudden perturbations and hand loading where muscle co-contraction contributed to elbow joint stiffness and stability. Interestingly, Viano (2007) did not find a correlation between neck rigidity and neck strength and presumed that neck strength was an inconclusive variable when examining concussion incidence. Similar findings have been demonstrated by Mihalik et al, (2011) who established that cervical muscle strength was not representative of decreased concussion occurrence in youth ice hockey players. Perhaps neck strength is less important when undergoing whiplash type mechanism, because unless impact is expected, a concussion would occur before the cervical muscles can sufficiently generate high bouts of activity. This contrasts with work by Omdal (2013) who identified a strong relationship between neck strength and impact severity in youth soccer players heading the ball. Performing an athletic movement that is intentional allows sufficient time to build cervical muscle tension. Neck strength studies involving head acceleration are still contradictory (Grady, 2010; Schmidt et al, 2015; Tierney et al, 2005). Despite this, muscle strength and muscle mass are positively correlated, thus larger cervical muscles, will require greater force magnitudes to overcome inertia (Chen et al, 2013; Kreighbaum and Barthels, 1996). Thus, neck strength is important as it relates to muscle

hypertrophy and cervical girth but the development of strength may take too long to generate observable stiffness effects when responding to unexpected head accelerations. This was evident in a study by Lavallee et al (2013), where the authors said "neck circumference appears predictive of neck strength" after collecting anthropometric, neck strength and endurance measurements on 91 human subjects in three bending directions (flexion, extension, lateral). This suggests that the size of the neck, as it relates to neck strength, could be a stronger predictor of decreased head acceleration when responding to unexpected head impact. Cervical muscles with larger CSA could reduce neck acceleration as seen in the relationship between girth and muscle stiffness (McNair et al, 2002; Wittek et al, 2001). In contrast to this concept, Schmidt et al (2015) did not find a good correlation with cervical size and decreased head acceleration.

6.3 Cervical Structure

Zheng (2011) demonstrated that the center of rotation (COR) of the neck was different between the sexes by intersecting perpendicular lines through standardized measurements in motion states of flexion and extension, and calculating the instantaneous axis of rotation (IAR). This is significant because particular joint regions of the cervical spine were correlated to increased susceptibility to injury (Tencer et al, 2003). Moreover, a larger HC/NC ratio is indicative of more pronounced inverted pendulum loading due to a smaller neck in relative proportion to the head (Vasavada, 2008). When a smaller neck needs to support a greater mass, there may be a need for greater dynamic stabilization to compensate for the lack of neck girth (Stokes et al, 2000). This could result in decreased neck rigidity because the base of support to the head (neck) is smaller and more susceptible to movement. Individuals with a larger ratio may be more susceptible to muscle length deformation and reciprocal head swing if they do not

sufficiently contract the neck musculature during perturbation (Vladimir, 2012; McCaw, 2014). Additionally, higher magnitudes of constant (static) muscle activity required to stabilize the head may introduce muscular fatigue more quickly. This effect is amplified if the weight and height of a helmet is larger. Ironically, helmets that are supposed to decrease concussion incidence have more cushion and support, but this added weight on a small neck may actually increase the whiplash mechanism. Light weight helmet designs are therefore very important.

6.4 Cervical Risk factors applied to Kinematic Response

Understanding cervical risk factors in relation to kinematic responses is fundamental in determining head acceleration. Schmidt et al (2015) categorized male athletes into high or low performers based on cervical characteristics thought to decrease head acceleration. High performers were categorized as having an 'advantage' for a specific risk factor i.e. shorter muscle onset latency and represented data that was above/below the median of the total participants. Other 'high performing' attributes included increased rate of torque development, higher peak torque, increased stiffness, larger CSA and less angular displacement. It's important to note that high performers could become low performers depending on their placement in the median split. The authors concluded that the odds of sustaining higher magnitude head impacts were reduced by having greater cervical stiffness and less angular displacement but these measures were not correlated with stronger or larger neck muscles.

Furthermore, high-speed cineradiography examined the intervertebral motion of subjects responding to rear impact and found that the C5 vertebra rotate higher than the C6 vertebra in comparison to normal voluntary extension movements (Siegmund, 2002). Similarly, Schafer, (1987) addressed the C4-C6 as being the highest incidence area of whiplash. If the COR

(determined by IAR) was found to occur at C4-C6, one might be more susceptible to whiplash. This coincides with Stemper et al (2011) who demonstrated that cervical anatomical dimensions, head-neck orientation at time of impact, facet joint orientation and neck muscle size/orientation influence the cervical response to rear impact. This is why safety initiative like “Heads Up” are so vital in reducing cervical injury. Having knowledge of impending impact would enable athletes to respond faster, adjust body orientation, estimate impact magnitude/location and contract cervical musculature (Eckner et al, 2014; Mihalik et al, 2011).

SECTION 7: RISK FACTOR INFLUENCE ON SEX

7.1 Sex Differences during Perturbation Anticipation

Since female varsity ice hockey have non-contact regulations, women may be less experienced at reacting to environmental threats (Hedlund, 2000). This is why training strategies directed towards improving sense of awareness may help facilitate muscle activation and effective cervical contraction. These techniques may also be taught more in males due to higher contact frequency. Cultural implications and psychosocial factors favour increased aggressiveness in males which may cause males to partake in body-checking and other forms of contact, more often resulting in concussive injury (Hedlund, 2000). More research needs to be conducted regarding risk compensation between sexes in varsity ice hockey. Observing the number of potential-concussive impacts and the proportion of impacts that resulted in concussion should be more closely examined between sexes. When examining head perturbation studies, Tierney et al (2005) showed that knowledge of force application resulted in a significant decrease in head angular acceleration in males but not for females. This study resembles Kumar et al (2000) who also observed sex differences in low velocity rear-impacts.

7.2 Sex Differences in Muscle Onset Latencies

Males generally have slower neck muscle reflex times than females (Foust et al, 1973; Tierney et al, 2005). Siegmund (1997) displayed that cervical muscle onsets occurred earlier in females than males during simulated rear end vehicle collisions. Neck perturbation studies have reported sex differences primarily in the onset of the sternocleidomastoids (Brault et al, 2000; Tierney et al, 2005). Tierney reported that females had approximately 3.8 ms shorter onset latencies during forced flexion and approximately 14 ms shorter onset latencies during forced

extension than males. This study concluded that larger head-neck angular acceleration produced shorter muscle onset latencies as per the VCR. This may be because the neck is moving earlier and therefore triggering a response, not necessarily reacting quicker. Linder et al (2008) showed that maximum head acceleration for females occurred earlier than males in response to rear impact during simulated vehicle collisions. At 4km/hr females reacted 10 ms earlier and at higher velocity impacts (8km/hr), females reacted 12 ms earlier. One potential explanation for this could be that female necks are weaker than males (relatively) and therefore, must react faster because of an earlier change in head movement. This would trigger the VCR response and partly explain resultant muscle onsets (Tierney et al, 2005). Contrastingly, since females have been shown to have smaller necks than males (Vasavada, 2008), females may react faster due to the shorter neuronal distance travelled as per nerve conduction velocity (Tortora et al, 2009; Baker and Barassi, 2008). Other factors like age and body mass index would influence nerve conduction, therefore it is important to compare similar size athletes when observing sex differences (Awang et al, 2006).

7.3 Sex Differences in Cervical Muscles

Neck strength was found to be weaker in females across numerous studies (Vasavada et al, 2008; Tierney et al, 2005). Men typically have more skeletal muscle mass than women and this has been demonstrated using ultrasound imaging (Janssen et al, 2000; Rankin et al, 2005). In addition, Zheng (2011) used magnetic resonance imaging (MRI) to demonstrate that females had significantly smaller total neck volumes than men. Studies tend to focus on total neck muscle volume or the muscles primarily activated in response to rear impact (due to the high incidence of vehicle collisions), rather than sport specific muscle analysis (especially flexed, extended or

expected states) (Rankin et al, 2005; Stemper et al 2003; Visscher et al, 1998; Zheng, 2011). A pilot study by Balkarran et al (2015) demonstrated that female ice hockey players had smaller sternocleidomastoid volumes in relative proportion to their neck in comparison to males. Balkarran et al (2015) computed bilateral volumetric estimates of the sternocleidomastoid from the C2-C5 region and found that, in females, these muscles represented 6% less of the total neck volume than males (females = 17.48 ± 2.6% TNV; males = 24.04 ± 1.1% TNV).

7.4 Sex Differences in Cervical Structure

In 2007, Vasavada et al concluded that "female necks are not uniformly scaled versions of male necks" and thus, a female model of the neck should be created. In 2008, Vasavada matched stature and neck length across sex and showed that female necks are smaller than males, but support similar size heads. Functional ratios displayed 33% more head mass per unit of neck muscle area, thus, the female neck supports significantly more weight, especially if wearing a helmet (Vasavada et al, 2008). Further data revealed that females had smaller vertebrae at C3-C5 in the anterior-posterior dimension when measured for depth and height. Similarly, Stemper et al, (2008), indicated that vertebral width and disc-facet depth were greater in men alluding to increased column stability. Females have less CSA and total neck volume when matched for height and weight and exhibit more range of motion (Vasavada et al, 2008; Zheng et al, 2013; Foust et al, 1973; Tierney et al, 2005). Posture and the position of greatest female cervical spinal curvature were also reported to be different when compared to males (Stemper et al, 2003; Visscher et al, 1998; Klinich et al, 2004). As a result, head-neck proportions like the head circumference/neck circumference ratio considered by Vasavada et al (2008) could be a more significant risk factor than muscle onset or muscle size.

7.5 Sex Differences Applied to Kinematic Response

Tierney et al (2005) subjected physically active men and women to seated head perturbations that dropped a 1kg load. During trials where impact was known, subjects were given a three second countdown and asked to prepare cervical musculature. When trials were unknown, participants were asked to relax their muscles and resist when they felt a tug. Females displayed more head accelerations than males in the known and unknown conditions for both forced flexion and forced extension (Refer to Figure 7). Inter-sex comparisons of female knowledge (known/unknown) conditions showed that head accelerations were decreased when females were expecting the perturbation for forced flexion but this was not true in forced extension. Males showed larger reductions than females in head acceleration when contracting cervical musculature for both the forced flexion and extension directions. Likewise, head accelerations were reduced for both directions when males prepared cervical musculature pre-perturbation.

Gender	Knowledge	Direction	Angular Acceleration ($°s/^2$) Mean ± SD
Female	Unknown	Forced flexion	1717.4 ± 607.9
		Forced extension	1868.0 ± 514.7
Male		Forced flexion	1275.2 ± 502.4
		Forced extension	1497.3 ± 602.8

Figure 7. Example of Head-neck segment angular acceleration data for Tierney study.

Adapted from Tierney et al (2005). Table 2: Means and standard deviations for head–neck segment kinematic data

Tierney et al (2005) found that physically active females had over 30% larger head accelerations than males and attributed this to females having less isometric strength, head mass and neck girth. Similarly, Linder et al (2008) found that females had 29% larger head acceleration (one plane) than males at 4km/hr and 9% larger head x-acceleration at 8km/hr in response to rear impact to simulate vehicle collisions whereas Siegmund et al (1997) examined similar trends. This difference in head acceleration may also be partially attributed to females having smaller C3-C5 vertebral dimensions (Vasavada, 2008) which has shown to be peak incidence area of whiplash (Tencer et al, 2003).

SECTION 8: SUMMARY, GAPS AND FUTURE WORK

On average, it would appear that females have smaller cervical musculature in relative proportions to the neck, less cervical CSA, less isometric neck strength, a greater range of head/neck motion and a smaller neck with a similar sized head, when compared to males (Vasavada et al, 2008; Zheng et al, 2013; Foust et al, 1973; Balkarran et al, 2015;Tierney et al, 2005). These anatomical factors lead to increased inverted pendulum loading and thus, increasing the effort needed to maintain head stabilization to external forces and may introduce an earlier onset of muscle fatigue (Vasavada et al, 2008; Stokes et al, 2000; Schick et al, 2003). Smaller head and neck dimensions correlate to mass, which, if less, requires less force to move (Kreighbaum and Barthels, 1996). Larger neck muscle volume may contribute to the dampening of impact forces due to the correlation to neck muscle strength (Chen et al, 2013). Likewise, smaller musculature in relative proportion to the neck may require a higher proportion of muscle activity to maintain equilibrium during a sudden disturbance. Moreover, more range of motion may decrease stability and aid in passive tension buildup of elastic properties (Nishikawa et al, 2007; Vladimir et al, 2012).

With all of this, females have shown to have shorter muscle onset latencies higher muscle activation and greater head acceleration when responding to neck perturbations (Linder et al, 2008; Tierney et al, 2005; Siegmund et al, 1997). Females therefore may have to overcompensate to make up for the less efficient cervical region when compared to men. It would therefore be important to identify which cervical risk factors aforementioned most prominently influence head acceleration in response to sudden head loading. This may help explain the higher ice hockey concussion incidence reported in females (Koerte et al, 2012). So

far, no studies have conducted a head perturbation study for this particular athletic group. Likewise there are limited head perturbation studies observing the response of cervical muscles to lateral directions which is correspondent to occur for 47% of concussions observed in the NHL (Hutchinson et al, 2013). Cervical risk factors that most greatly influence concussion susceptibility are still being debated.

Individuals that display the same anthropometric and anatomical characteristics as females may be subject to the same fate thus these principles are not specific to sex. That being said, Vasavada (2008) exclaimed that female necks are not uniformly scaled versions of male necks (Refer to Figure 8). Hormonal factors and bone density are some of the factors that are specific to sex and would affect the dynamic response of the cervical region (Dahn, 2012; Vasavada, 2008). These factors were less studied because they are not modifiable but are still important sources of sex differences.

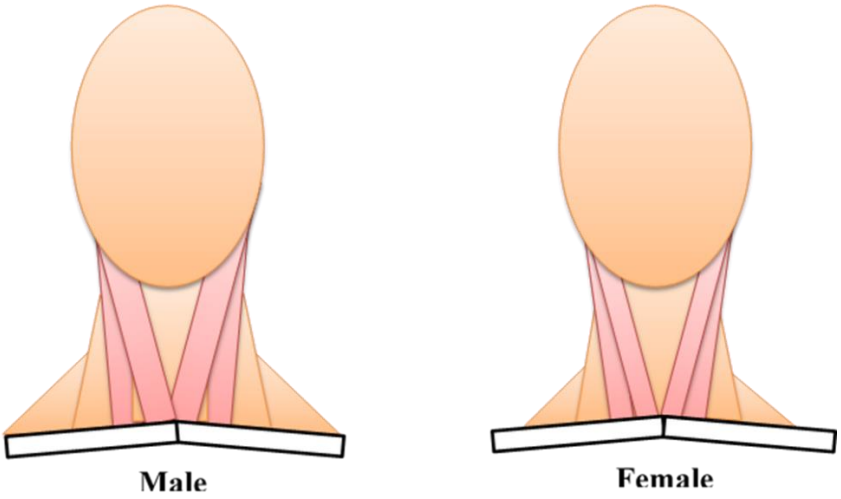

Figure 8. Cervical muscle size and girth differences between sexes in relative proportion to the head. Hypothetic dimensions created to better understand muscle relativity, the HC/NC ratio and total neck volume between sexes.

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CHAPTER 3: MANUSCRIPT

NEUROMECHANICAL DIFFERENCES IN THE RESPONSE TO HEAD PERTURBATIONS IS DIFFERENT BETWEEN FEMALE AND MALE VARSITY ICE HOCKEY PLAYERS

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1.0 ABSTRACT

BACKGROUND AND AIM: Sex differences are apparent in the prevalence of concussions in sport. The ability of the neck muscles to dampen sudden head accelerations may be one mechanism that contributes to these differences. Our aim was to examine sex differences in head acceleration and cervical muscle activity during sudden head perturbations. METHODS: 16 competitive ice hockey players (8 female) participated. Three muscles were monitored bilaterally using surface electromyography (EMG): sternocleidomastoid (SCM), scalene (SC) and splenius capitis (SPC) (Delsys, Boston, MA). Head kinematics were measured using a motion capture system (NDI, Waterloo, ON) with rigid bodies placed on the head and thorax. Cervical perturbations were induced by the release of a 1.5kg magnetized weight (dropped 15cm), attached to a wire wrapped around a height adjustable pulley secured to the participant's head. A load cell, in series with the cable, determined perturbation onset. Perturbations were delivered in 4 directions (flexion, extension, right and left lateral bend) 6 times each in randomized order. EMG was sampled at 1926 Hz, low pass Butterworth filtered at 3 Hz and normalized to muscle specific maximal voluntary excitation (%MVE). Kinematics were sampled at 64 Hz and low pass Butterworth filtered at 6 Hz; angular velocity and acceleration were calculated. Three time periods were examined: baseline (-150 to -100 ms pre-perturbation), anticipatory (-15 to 0 ms) and reflex (25 to 150 ms post-perturbation). RESULTS: Females had significantly greater head acceleration during left lateral bend (18.6%) and flexion (23.4%) perturbations, with no difference in extension. Females had significantly shorter muscle onset latencies in flexion but had longer onset latencies of the SCM and SCL muscles in left lateral bend and extension in comparison to males. Averaged across perturbation time periods, during flexion females had greater activity than males in left SPC (5.5 ± 1.8% MVE vs 3.6 ± 1.6% MVE), right SPC (3.6 ±

3.4% MVE vs 3.0 ± 2.5% MVE) and right SC (2.7 ± 1.9% MVE vs 1.6 ± 1.3% MVE), while males had greater left SCM (2.1 ± 3.1% MVE vs 1.4 ± 1.0% MVE), right SCM (1.9 ± 2.2% MVE vs 1.3 ± 0.7% MVE) and left SC (2.7 ± 3.2% MVE vs 1.9 ± 0.8% MVE). Females had greater left and right SCM and SC activity in extension, with no difference in head acceleration. There was a significant time period x sex interaction during extension, with females displaying 4.1% more overall muscle activity in the reflex period (7.2 ± 2.8% MVE vs 3.1 ± 0.9% MVE). CONCLUSIONS: There were sex differences across most variables, dependent on perturbation direction and timing knowledge, with no consistent neuromuscular strategy that could explain all directional effects. During extension perturbations, females had greater muscle activity in the reflex time period which may explain the lack of head acceleration differences between sexes. The increase in activity during reflex periods suggests a neuromuscular response to counter sudden acceleration. Further investigation of muscle onset times, joint rotational stiffness and co-contraction could reveal unique muscular strategies that place female athletes at a greater risk of concussions.

Keywords: Perturbations, Head Acceleration, Muscle Activation, HC/NC ratio, Estimated TNV

2.0 INTRODUCTION

The physical nature of ice hockey, played at an incredible speed makes it a dangerous sport where direct impact to the head is common, despite head contact penalties (Daneshvar et al, 2011). Epidemiological studies reveal that female varsity ice hockey players experience a greater number of concussions than males, despite having non-contact regulations (Angel and Harvey, 2010; Dick, 2009). Sex specific psychosocial factors, such as the under-reporting of concussions in males may contribute to these differences, but there has been limited attention given to the neurophysiological and biomechanical aspects of head acceleration between sexes in this particular athletic group (Spitzer et al, 1995; Wilcox et al, 2015). Females who experience a concussion are more prone to higher incidences of neck pain and post-concussion syndrome while having longer return to play times than males (Broshek et al, 2005; Preiss-Farzenegan et al, 2009; Bazarian et al, 2010).

Neck stiffness is widely accepted as being an important preventative method in minimizing neck movement (Hynes and Dickey, 2006; Viano et al 2007; Schmidt et al, 2015). Greater neck stiffness when external forces are applied to a system (like checking in hockey), leads to increased robustness, or the ability to resist perturbation (Reeves et al, 2007). This translates to a decrease in head acceleration, which is a primary mechanism mitigating concussion (Wilcox, 2015; Gurdjian et al, 1954).

Shorter muscle onset latencies and higher muscle activation before or in response to external force application, may lead to increased neck stiffness since linear relationships have been demonstrated in the spine for muscle activation and stiffness (Cholewicki and McGill, 1996; Potvin and Brown, 2005). Siegmund et al (2003) showed that cervical muscle activity was

greater when participants were aware of the external force delivery applied to the head. Knowledge of incoming forces will ultimately lead to an increased ability to resist sudden head perturbations. Having knowledge of an incoming head impact in ice hockey will allow the participant time to better prepare the cervical musculature to adapt and provide the required stiffness to reduce head acceleration. Without perturbation knowledge, individuals must accommodate through neuromuscular strategies that generally involve greater muscle activation post perturbation (Siegmund et al, 2003). The lack of visual or auditory cues before head impact in ice hockey may hinder the ability to establish preparatory cervical muscle techniques.

Muscle strength is strongly correlated with muscle size and studies have recommended pre-season neck strengthening routines for athletes in an attempt to reduce cervical spine injuries (Chen et al, 2013; Nash et al, 2013). An epidemiological study of 6,704 student athletes found that concussion risk was reduced for every pound of neck strength (Marer et al, 2012). Contrastingly, muscle strength is not always correlated with reduced head accelerations during head perturbation studies (Tierney et al, 2005; Schmidt et al, 2015). This suggests that recruitment strategies and muscle onset latencies need to be further studied.

Larger cervical muscle cross-sectional area (CSA) may contribute to a reduction in head acceleration (Tierney et al, 2005). Sudden forces applied to the body or head will result in neck muscle responses to stabilize the head and a smaller neck (circumference) may result in greater muscle activity to compensate for the lack of cervical CSA (McNair et al, 2002; Wittek et al, 2001). Alternately, it has been demonstrated that head impact magnitude is not reduced in athletes with larger neck muscles (Schmidt et al, 2015; Mansell et al, 2005).

Females carry more head mass per unit of neck muscle CSA than males, even when normalized for height and weight, signifying that females have smaller necks in relative proportion to their head (Vasavada et al, 2008). Tierney et al (2005) demonstrated that physically active females had greater head accelerations than males in response to an external force application, despite having greater muscle activity and shorter onset latencies. These differences could be attributed to females having smaller necks, among other variables like less cervical isometric strength and head mass.

Research has shown that females are more vulnerable to neck muscle strain, whiplash and concussions during sport contact than males (Broshek et al, 2005; Preiss-Farzenegan et al, 2009; Bazarian et al, 2010, Covassin et al, 2003). Female necks may be more susceptible to relative ice hockey contact than males and this could contribute to the epidemiological discrepancy between sexes (Koerte, 2012). The purpose of this study was to examine cervical muscle responses (magnitudes and timing) and head kinematics between male and female ice hockey players during sudden multi-directional head perturbations.

3.0 METHODS

3.1 Study Participants

Sixteen competitive ice hockey players free of head/neck injuries in the last 12 months participated in this study (table 1). Participants signed Informed Consent, completed the Edinburgh Handedness Inventory checklist and a Student-Athlete Health History Questionnaire from the University of West Florida, Athletic Training Department (UWF Athletic Training, 2009). This study was approved by the University of Ontario Research Ethics Board (REB # 13-020).

Table 1. Mean (±SD) participant demographics and anthropometrics

Characteristics	Female	Male
Current level of ice hockey competition	Varsity (n=8)	Varsity (n=4) Competitive Leagues (n=4)
Age (years)	20.60 ± 1.30	22.13 ± 1.55
Height (m)	1.69 ± 0.60	1.79 ± 0.77
Weight (kg)	71.30 ± 8.78	84.50 ± 6.19

3.2 Protocol

Anthropometrics: Five anthropometric measurements [head circumference, neck circumference, neck length, neck width and neck depth] were taken from each participant using a retractable fiberglass tape measure (Prestige Medical^{TM}). Head circumference was measured as a 360° measurement surrounding the superciliary ridge and opisthocranion (Vasavada, 2008). Neck circumference and neck width were measured at the level of the fourth cervical vertebrae,

neck length from the midpoint of the disc between C7 to TI to the midpoint of C1 and neck depth from above the thyroid cartilage perpendicular to the long axis of the neck (Cram and Criswell, 2011; Falla et al, 2002). These measurements were combined to create ratios [head circumference/neck circumference] and to estimate total neck volume (TNV) [neck length x width x depth] (Vasavada et al, 2008; Zheng et al, 2013).

Head Perturbations: Perturbations were induced in four different directions that would cause neck flexion, extension, right lateral bend or left lateral bend by attaching a wire to one of four attachment points on a head harness. Head perturbations were induced by the release of a 1.5kg magnetized weight that dropped approximately 15cm (similar to previous work, Tierney et al., 2005). The load was magnetized by an Electromagnet (EM-R175, Jobmaster Magnets, Oakville, Canada) and attached to a wire that wrapped around a height adjustable pulley system attached perpendicular to the participants head (Figure 1). A load cell (MB 100 Intercel, Trigno load cell adapter) attached in series to the wire was used to measure perturbation onset and rested on a platform, to maintain a linear cable position. Using a manual trigger, the magnet could be desensitized, allowing the weight to drop. With the wire attached to the anterior portion of the head, the release of the weight caused flexion; attached to the posterior portion of the head caused extension and attached to the sides of the head induced left and right lateral bend. The wire (Spiderwire, EZ Braid, non-resistant fishing wire), was chosen due to its low friction at the pulley interface and light weight. Prior to each perturbation, participants were instructed to relax their neck muscles and look straight ahead while seated (Figure 1).

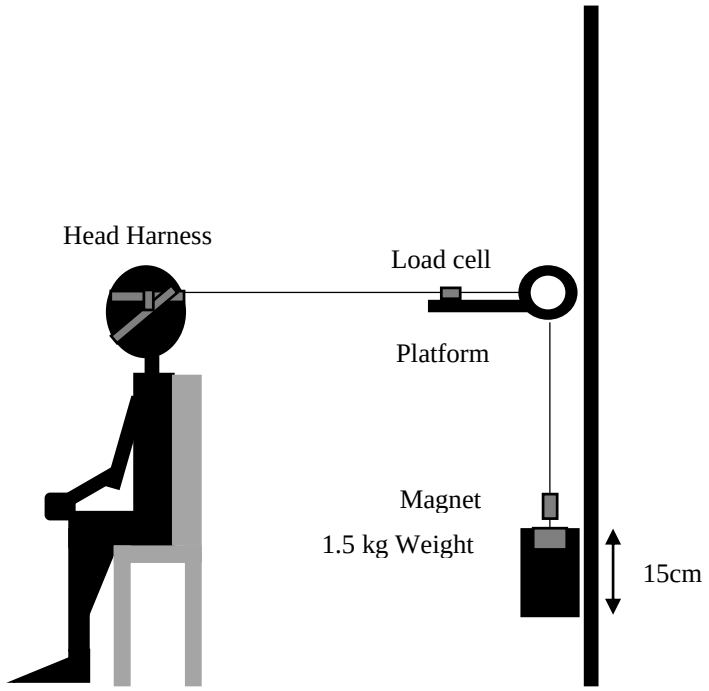

Figure 1. Experimental setup for the custom load drop device that caused head perturbations. The participant would be re-orientated to change perturbation direction.

Six perturbation trials (3 expected, 3 unexpected) were induced in each direction (flexion, extension, right and left lateral bend) for a total of 24 perturbations (12 expected, 12 unexpected), in a randomized order. Once a direction was randomly selected, all trials were completed prior to the next direction. All trials were triggered by the investigator. Expected trials consisted of a five second countdown and the participants had their eyes open. Unexpected trials occurred at a random time within a 30 second window and participants had their eyes closed. A second examiner ensured that each participant met the criteria and maintained an upright body position during each trial.

Electromyography (EMG): Prior to starting the perturbation trials, muscle activity was recorded from the Left (L) and Right (R) sternocleidomastoid (SCM), middle scalene (SCL) and splenius capitis (SPN) using wireless surface electrodes (Trigno™, Delsys Inc., Natick, MA). These muscles were chosen because the SCM acts bilaterally as a prime mover for neck flexion and unilaterally in ipsilateral neck lateral flexion and contralateral rotation, whereas the SPL is the most superficial extensor muscle, creating head and neck extension when acting bilaterally (Berhhardt et al, 1999; Cheng et al, 2014). The SCL is largely responsible for lateral flexion and has been less observed than other muscles in multi-directional perturbations (Ólafsdóttir et al, 2015). To minimize skin impedance, all electrode sites were removed of hair, lightly abraded and cleansed using an isopropyl alcohol wipe (Hermens et al, 2000). Electrodes were placed longitudinally along the muscle belly, in line with muscle fiber orientation (Sacher et al, 2014). EMG signals were differentially amplified (common mode rejection ratio >80dB, input impedance 10GΩ, band-pass filter, 20-450Hz), sampled at 1926Hz and were 16-bit A/D converted. Electrodes had a silver-contact bipolar bar with a fixed 1cm inter-electrode distance. For SCM, electrodes were placed one third of the distance between the mastoid process and sternal notch along the sternal head of the SCM; for SCL, electrodes were placed in the hollow on the side of the neck; for SPLN, underneath the cranium, parallel to the spine, lateral to spinous process over the muscle belly (Falla et al 2002; Cheng et al, 2010; Cram and Criswell, 2011).

Maximum voluntary contractions: Maximal voluntary isometric contractions were performed and used to later normalize muscle activity from each perturbation. Subjects were instructed to keep their head neutral while seated and perform 2 maximum voluntary isometric contractions for each direction against a non-movable (anterior, posterior, right and left) padded

surface. Each contraction was held for 3 seconds with two minutes of rest between each direction (Cheng et al, 2014).

Kinematics: Three dimensional kinematics of the head and thorax were also measured (3D Investigator, NDI, Waterloo, Ontario, Canada). Rigid bodies, consisting of 3 infrared markers were attached to the head and thorax of each participant. The head rigid body was attached to the opisthocranion and the thorax rigid body was attached at the midpoint between the spinous processes of the C7 and T1 vertebrae to track head and thorax movement (Potvin, 1997). The laboratory coordinate system was defined as anterior/posterior (x), medial/lateral (z), and vertical (y). All data was collected using First Principles software (1.00.03; NDI, Waterloo, ON). Assuming fixed spatial relationships, anatomical landmarks were digitized, relative to each rigid body, including, the left/right corner of the eye, left/right top of head, left/right iliac crest, left/right acromion, sternal notch and xiphoid process. Kinematic data were sampled at 64Hz and synchronized with electromyography and the load cell which were sampled at 1926Hz. By the use of a manual trigger, all three systems began recording simultaneously (Delsys Trigger Module, Delsys Inc. Boston, MA).

3.3 Data Analysis

Muscle Onset Measurement: Raw EMG signals were zero offset (DC bias removed) and full-wave rectified to ensure onset could be confirmed by visual observation. Onset threshold was defined as the first data point that exceeded a criteria of 5 x the root mean square of the baseline mean within a 0.04 second window length (Delsys EMGworks, "smart threshold" algorithm). The baseline mean consisted of taking at least one second of data before the perturbation onset. This algorithm was used to determine load cell and muscle onset times. Load

cell onset was defined as time zero and muscle onset times were calculated from this point. The load cell platform ensured no vibrational frequencies were affecting the signal and helped produce a prominent onset (figure 2). Trials were excluded if muscle onset occurred after 80 milliseconds to ensure no voluntary activity was present as Brault (2000) showed that subjects could obtain near maximal voluntary levels of muscle activation within 100ms of impact. Muscles that opposed the direction of the perturbation were termed 'opposition muscles'. Subsequently, muscle were considered non-opposition muscles if they were on the same side of the perturbation direction. This relates to Olafsdottir (2015), where cervical muscles were found to have direction specific contraction levels in some subjects but not others.

Muscle Activity: All EMG data (MVEs and perturbation trials) were low pass filtered (3Hz cutoff, dual pass, 2nd order Butterworth). The largest activation was found for each muscle from the muscle specific maximal voluntary contractions and this was used to normalize each muscle during perturbation trials as a percentage of maximum (%MVE). With load cell onset representing time 0, muscle activity was analyzed for three time periods (Matlab, R2015a, Natick, MA):

- Baseline [**A**] = -150 to -50 ms pre-perturbation
- Anticipatory [**B**] = -15 to 0 ms pre-perturbation
- Reflex [**C**] = 25 to 150 ms post-perturbation

Similar time periods have been analyzed previously for both spine (Grondin and Potvin, 2009) and upper extremity (Holmes and Keir, 2012) perturbations.

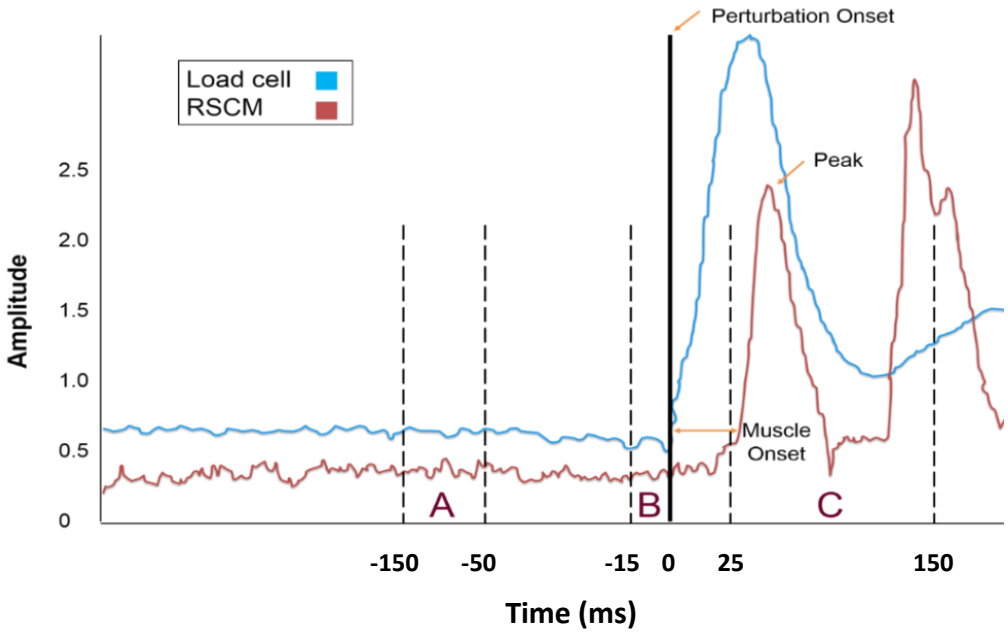

Figure 2. Representative perturbation trial for unexpected left lateral bend perturbation onset. The three time periods analyzed are indicated.

Kinematic Measurements: Kinematic data were analyzed using Visual 3D™ (C-Motion V5, Inc., Rockville, MD, USA). Data were Butterworth low pass filtered (6Hz cutoff, dual pass, 2nd order) and head acceleration was calculated (first derivative of angular velocity) as the rotation of the head relative to the thorax (°/sec^2). The peak acceleration within 100ms of the trial onset was evaluated for the primary rotation axis in all perturbation directions. For example, for flexion/extension perturbations, acceleration in the x (anterior/posterior) direction was evaluated.

3.4 Statistical Analysis

The kinematic, muscle onset and muscle activity data were averaged across the 3 trials for each experimental condition. Statistical analyses were conducted using SPSS (Windows Version 23, SPSS Inc., Chicago, IL). Independent t-tests were used to analyze anthropometric differences between sexes. Linear regression was used to examine correlations between head acceleration for HC/NC and estimated TNV to determine if neck size correlated with head acceleration. A univariate repeated measures analysis of variance (ANOVA) was used to examine the effect of perturbation direction (left lateral bend, right lateral bend, flexion, and extension) on the independent variables, sex (male vs female), condition (expected vs unexpected), muscle (left/right sternocleidomastoid, middle scalenes and splenius capitis) and time period (baseline, anticipatory, reflex). Dependent variables included, head acceleration, muscle onset latencies and maximum muscle activity. With direction removed as a variable, four separate (1 for each direction) 2 x 2 repeated measures ANOVAs were used to assess head accelerations in each direction by sex and condition. A 2 x 2 x 6 repeated measures ANOVA was used to examine muscle onset latencies in each direction by sex, condition and muscle. A 2 x 2 x 3 x 6 repeated measures ANOVA was used to measure maximum muscle activity in each direction by sex, condition, time period and muscle. Descriptive statistics and t-tests with Bonferroni correction were used to examine significant main and interaction effects. Significance was set at $P < 0.05$ for all tests. All data is reported as mean ± standard deviation.

4.0 RESULTS

4.1 HC/NC ratio and estimated TNV

Significant sex differences were found for estimated TNV [$t_{1, 16} = -3.353$, $p=0.005$] and HC/NC [$t_{1, 16} = 3.898$, $p=0.002$] with males exhibiting a 12% larger TNV (males: 1990.59 ± 246.81cm^3; females: 1563.05 ± 262.93cm^3) and a 4.6% smaller HC/NC ratio (males: 1.51 ± 0.59; females: 1.66 ± 0.10) (Figure 3).

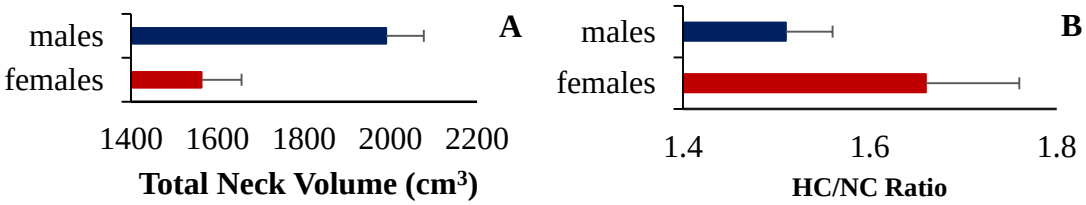

Figure 3. Comparison of males (blue) and females (red) for A) TNV and B) HC/NC ratio (mean ± standard error)

4.2 HC/NC ratio and Head Acceleration

The HC/NC ratio demonstrated weak relationships to head acceleration in left lateral bend ($r^2=0.1583$) and flexion ($r^2=0.2557$) with little to no relationship in right lateral bend or extension (figure 4).

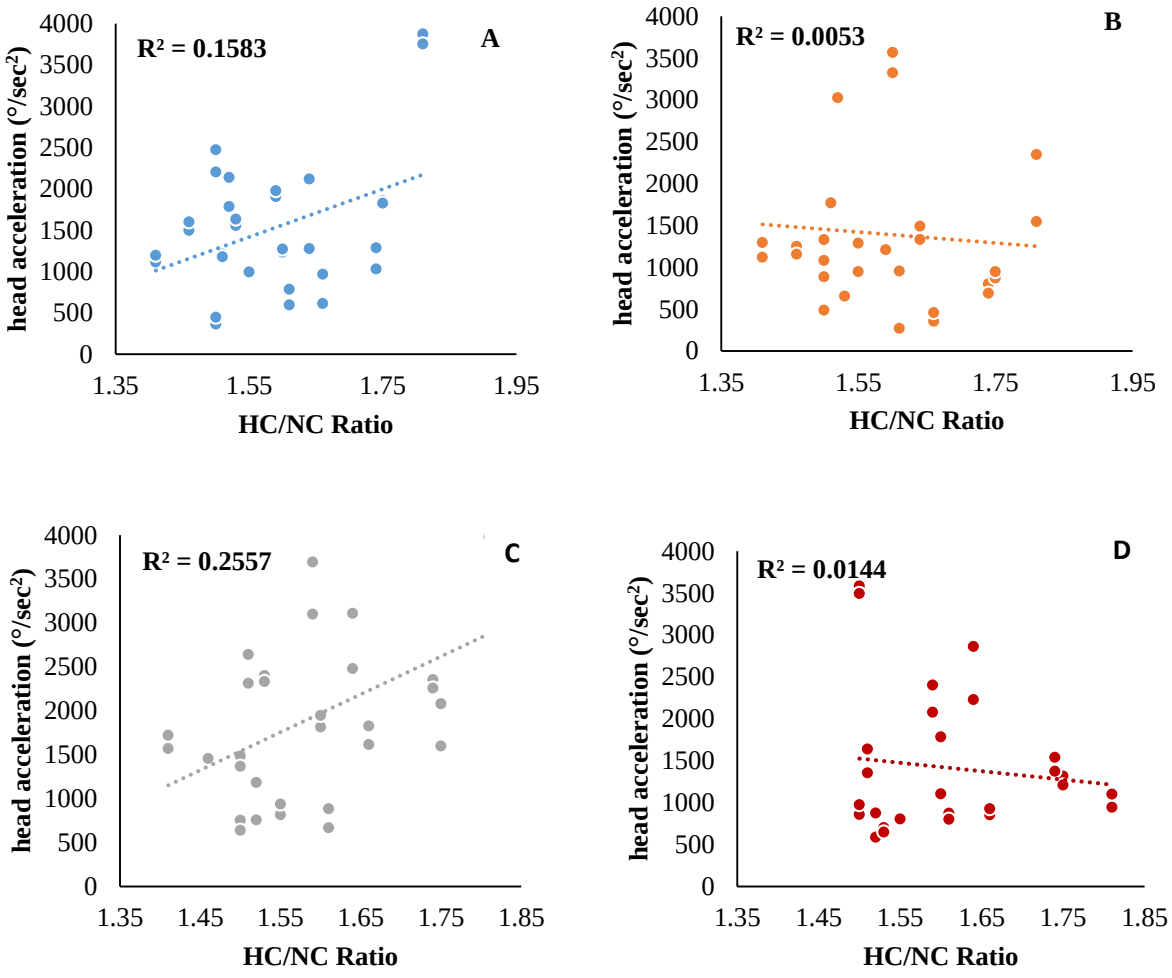

Figure 4. HC/NC ratio vs head acceleration for combined conditions (expected, unexpected) in A) left lateral bend B) right lateral bend, C) flexion D) extension.

4.3 Estimated TNV and Head Acceleration

The TNV demonstrated a weak relationship to head acceleration in left lateral bend (r^2=0.1232) with little to no relationship in right lateral bend, flexion or extension (figure 5).

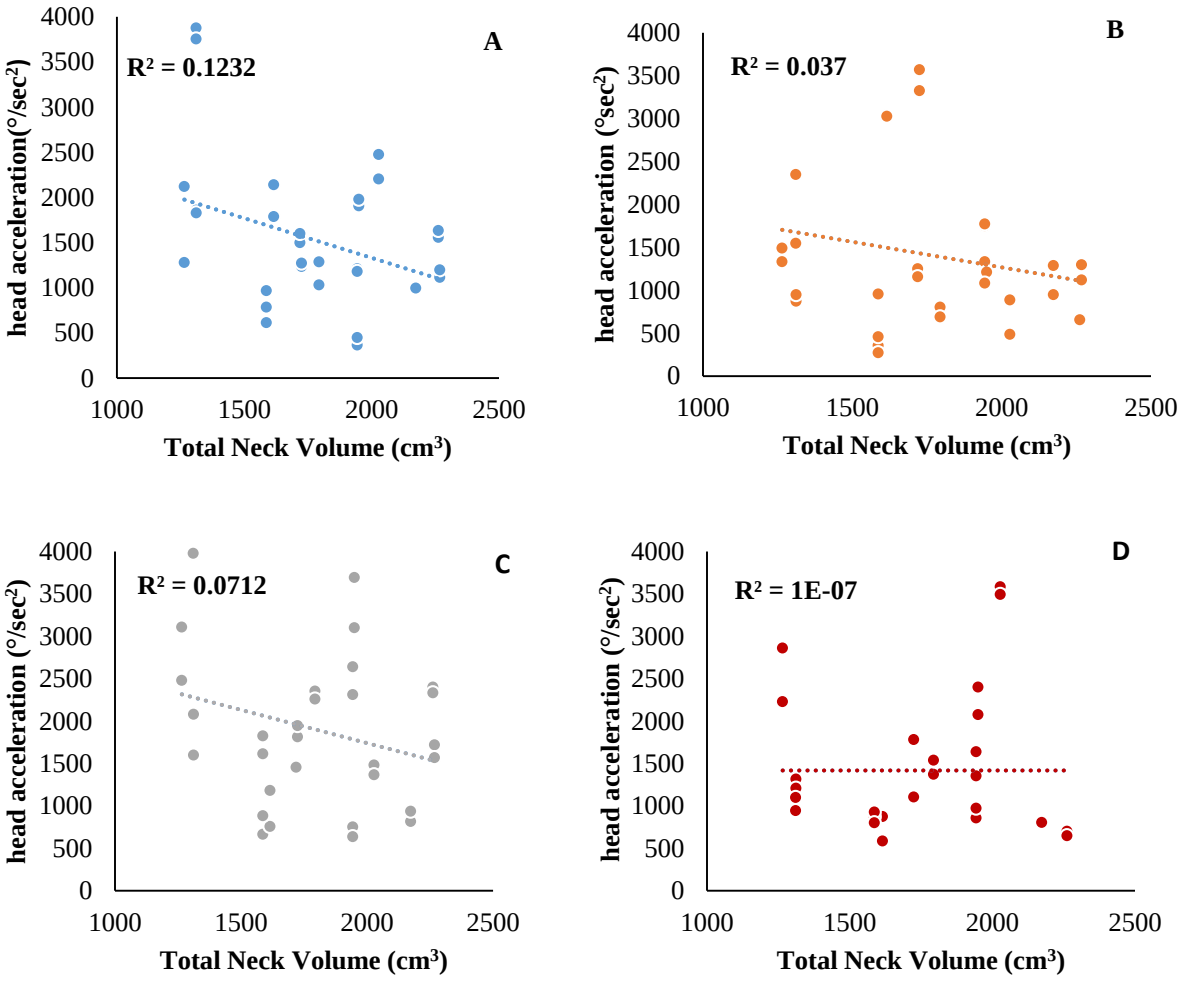

Figure 5. TNV vs head acceleration for combined conditions (expected, unexpected) in A) left lateral bend, B) right lateral bend, C) flexion and D) extension

4.4 Head Acceleration: Sex Differences

There was a significant main effect of sex for head acceleration during left lateral bend perturbations [$F_{1, 28} = 4.187$, $p=0.05$] where females induced 18.6% more head acceleration than males (females = 1807 ± 902 °/s^2; males = 1240 ± 580 °/s^2). There was also a significant main effect of sex during flexion perturbations [$F_{1, 28}=8.697$, $p=0.006$] where females induced 23.4% more head acceleration than males (females = 2360 ± 967 °/s^2; males = 1465 ± 66 °/s^2). There was a trend during right lateral bend perturbations, with females inducing 24.6% more head acceleration than males (females = 1738 ± 1264 °/s^2; males = 1056 ± 411 °/s^2). No sex differences were found for head acceleration during extension perturbations (females = 1448 ± 648 °/s^2; males = 1376 ± 1048 °/s^2). Refer to figure 6 for perturbation direction and head acceleration differences.

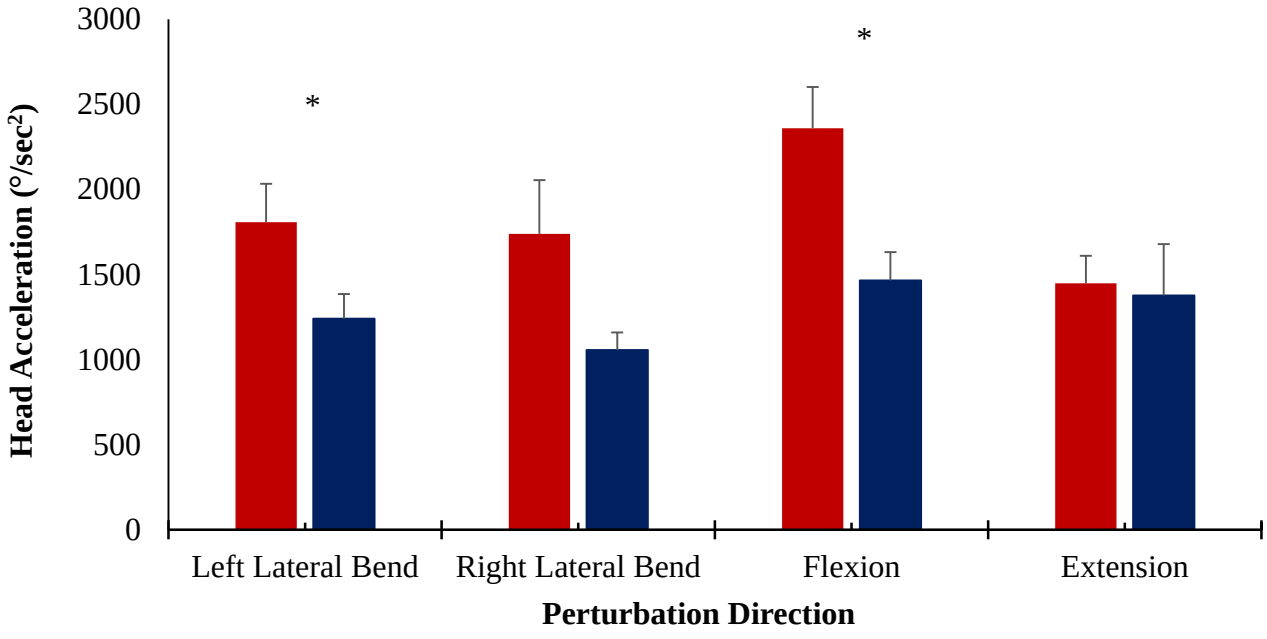

Figure 6. Head acceleration (mean ± standard error) for males (blue) and females (red) for each perturbation direction. * indicates a significant difference between males and females.

4.5 Muscle Onsets: Sex Differences

Left lateral bend: There was a significant sex X muscle interaction for left lateral bend perturbations [$F_{5,70}$ =0.2826, p=0.022] where males had shorter muscle onset times of the opposition muscles (right sternocleidomastoid, right scalene and right splenius capitis) than females (females = 57 ± 9 ms; males = 47 ± 13 ms) (Figure 7).

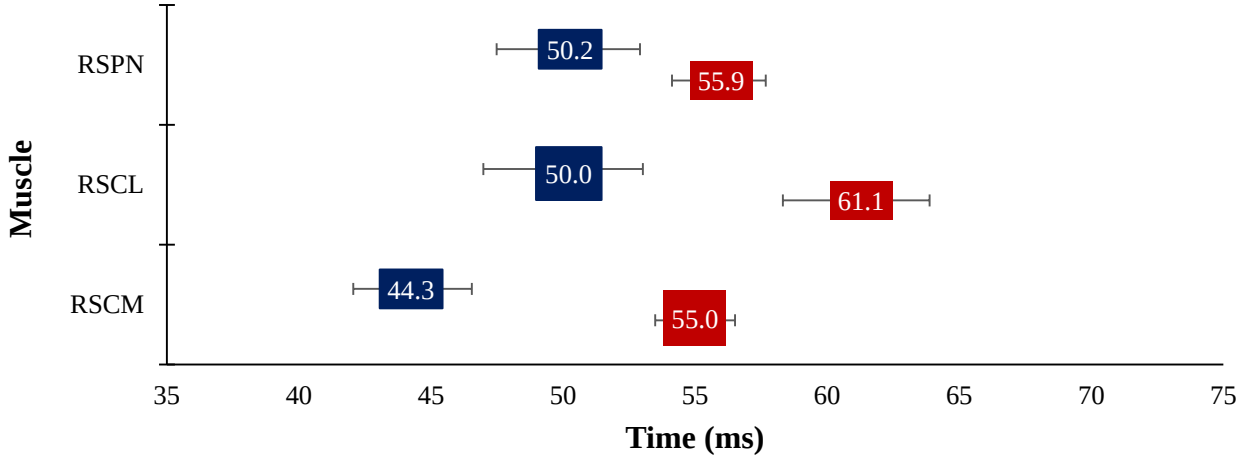

Figure 7. Muscle onset times (mean ± standard error) for the opposition muscles during left lateral bend perturbations for males (blue) and females (red) for the right (R) SPN, SCL and SCM.

In terms of the non-opposition muscles, females had shorter muscle onset times than males (left sternocleidomastoid, left scalene, left splenius capitis) during left lateral bend perturbations (females = 54 ± 11 ms; males = 63 ± 13 ms), however this was not significant and represented less than 50% of the data set (Figure 8).

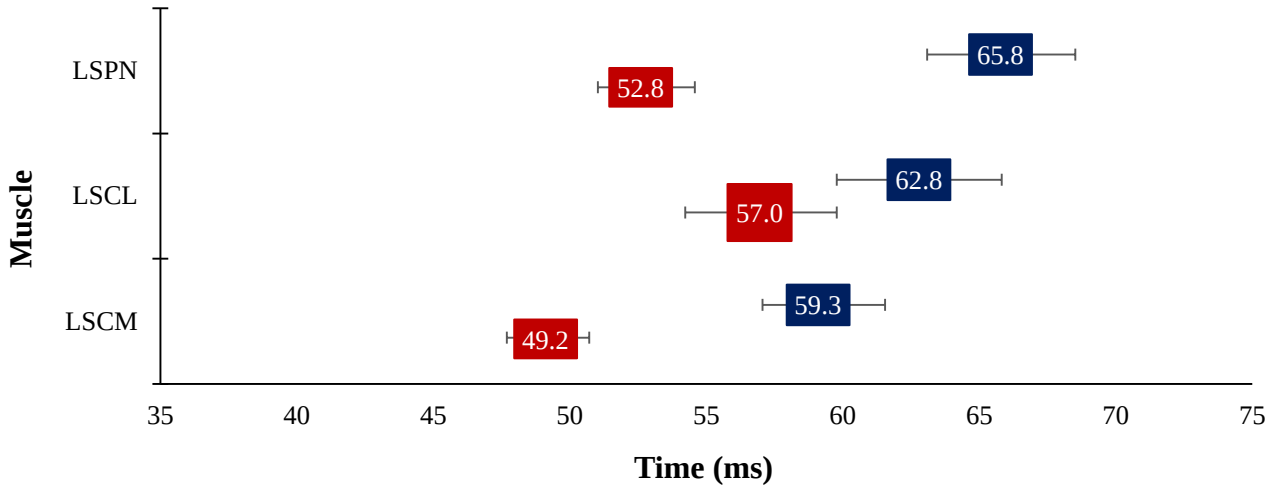

Figure 8. Muscle onset times (mean ± standard error) for the non-opposition muscles during left lateral bend perturbations for males (blue) and females (red) for the left (L) SPN, SCL and SCM.

Extension: There was a significant sex X muscle interaction for extension perturbations [$F_{5,89}$=3.118, p=0.012] where males had shorter muscle onset times of the opposition muscles (left and right sternocleidomastoid and scalene) (females, 58 ± 8 ms; males, 53 ± 9 ms) (Figure 9).

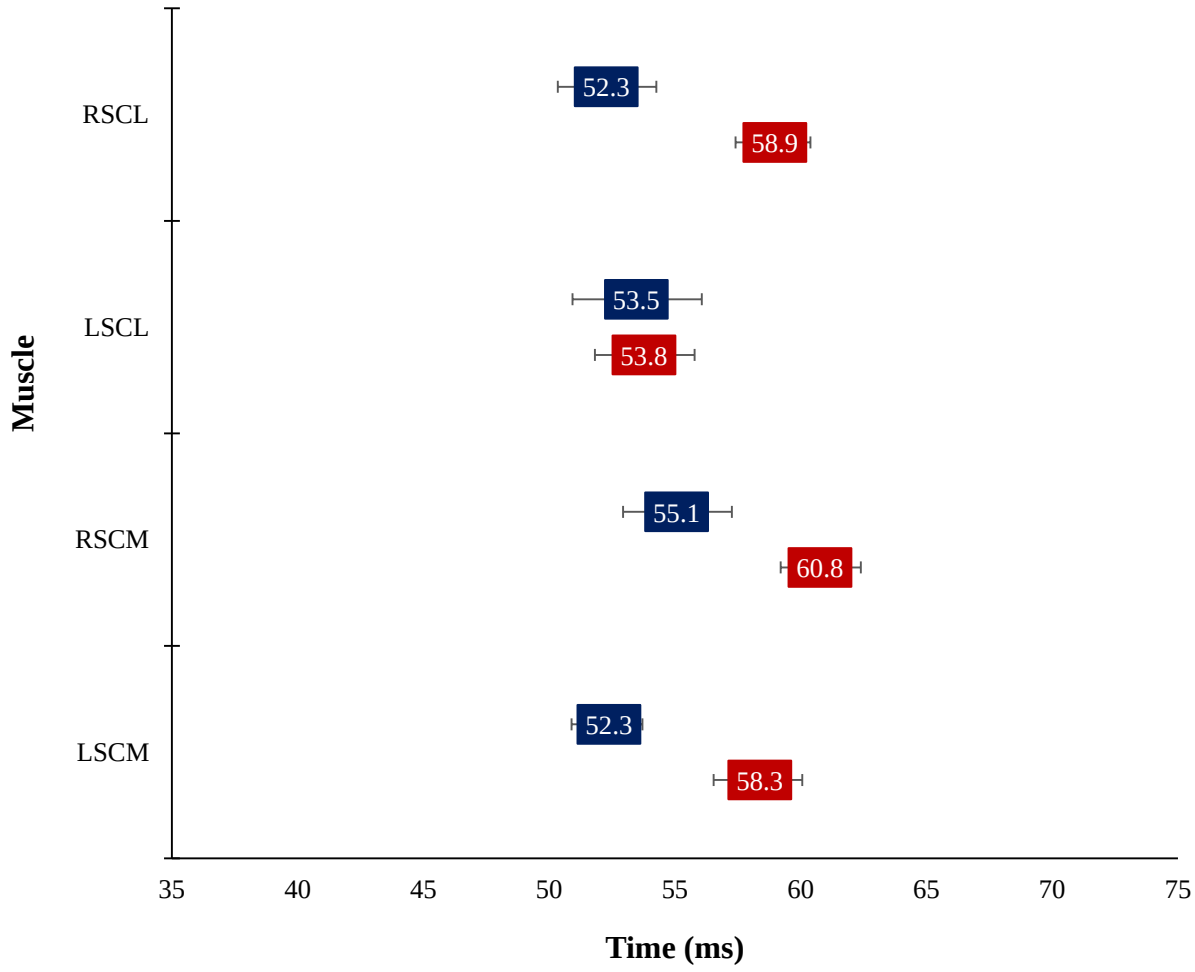

Figure 9. Muscle onset times (mean ± standard error) for the opposition muscles during extension perturbations for males (blue) and females (red) for the left (L) and right (R) SCL and SCM.

Females had shorter muscle onset times of the non-oppisition muscles (left and right splenius capitis) (females = 44 ± 7 ms; males = 55 ± 13 ms) but this was not significant due to low subject volume (Figure 10).

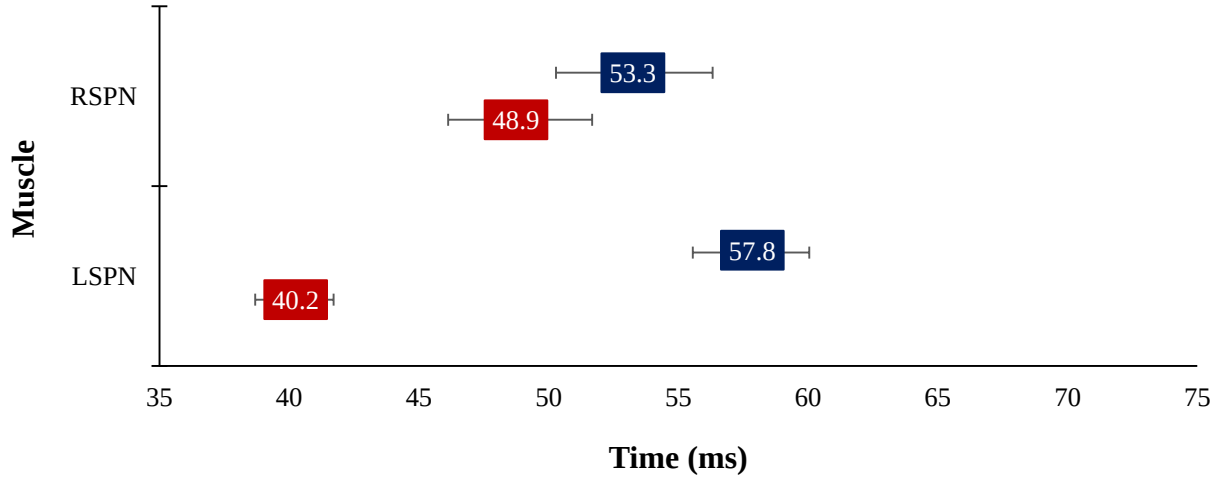

Figure 10. Muscle onset latencies (mean ± standard error) for the non-opposition muscles during extension perturbations for males (blue) and females (red) for the left (L) and right (R) SPN.

Flexion: There was a significant main effect of sex for flexion perturbations [$F_{1,66}$ =17.245, p=0.001], where, collapsed across muscles, females displayed shorter muscle onset times than males (females = 51 ± 11 ms; males = 61 ± 10 ms) (Figure 11).

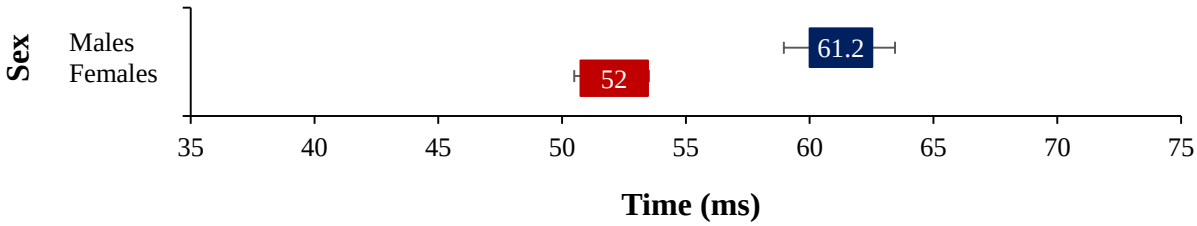

Figure 11. Muscle onset times (mean ± standard error) for males (blue) and females (red) during flexion perturbations.

4.6 Muscle Activation: **Sex Differences**

Flexion: There was a significant sex X muscle interaction for flexion perturbations [$F_{5,372}$ 4.258, p=0.001] where females displayed greater muscle activity of the left splenius capitis (females = 5.5 ± 1.8% MVE; males = 3.60 ± 1.60% MVE), right splenius capitis (females = 3.60 ± 3.40% MVE; males = 3.0 ± 2.50% MVE) and right scalene (females = 2.69 ± 1.87% MVE; males = 1.59 ± 1.34% MVE). Males displayed greater muscle activity of the left sternocleidomastoid (females = 1.44 ± 1.02% MVE; males = 2.10 ± 3.10% MVE), right sternocleidomastoid (females = 1.25 ± 0.72% MVE; males =1.86 ± 2.21% MVE) and left scalene (females = 1.86 ± 0.84% MVE; males = 2.70 ± 3.20 % MVE) (Figure 12).

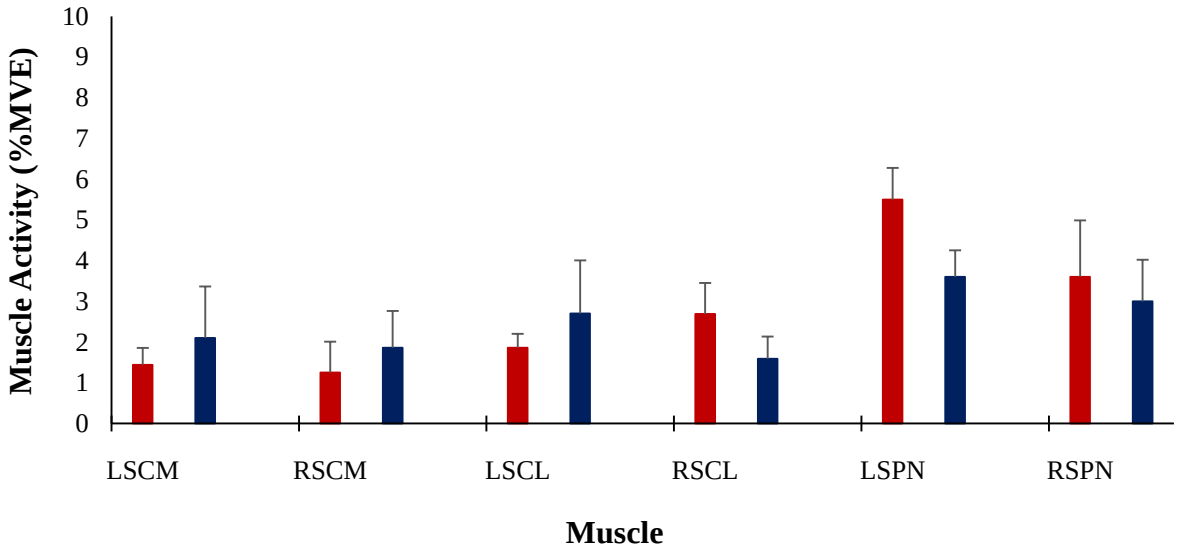

Figure 12. Sex differences in muscle activity (mean ± standard deviation) for females (red) and males (blue) for the left (L) and right (R) SCM, SCL and SPN during flexion perturbations.

Extension: There was a significant sex X muscle interaction for extension perturbations [$F_{5,372}$=2.925, p=0.013] where females exhibited greater muscle activity than males in the left sternocleidomastoid (females = 4.60 ± 7.50% MVE; males = 1.60 ± 1.10% MVE), right

sternocleidomastoid (females = 3.30 ± 4.75 % MVE, males = 1.40 ± 1.20% MVE), left scalene (females = 3.94 ± 4.70 % MVE; males 1.90 ± 1.26 % MVE) and right scalene (females =5.50 ± 8.40% MVE; males = 1.90 ± 1.20% MVE). Males and females had similar activation levels for the left and right splenius capitis during extension (Figure 13).

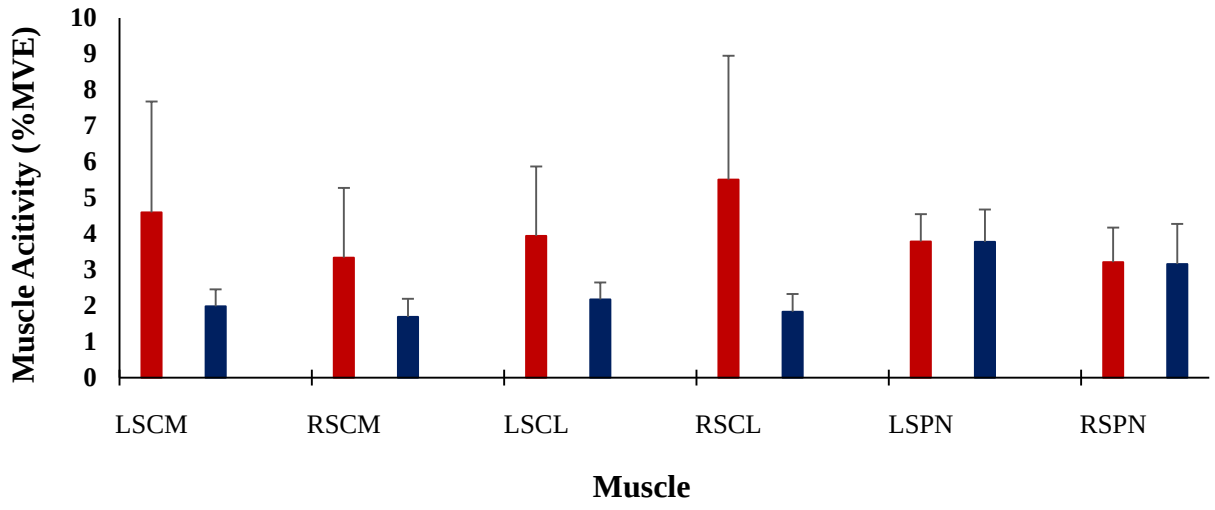

Figure 13. Sex differences in muscle activity (mean ± standard deviation) for females (red) and males (blue) for the left (L) and right (R) SCM, SCL and SPN during extension

A significant sex X time period interaction was also found for extension perturbations [$F_{2,372}$=9.786, p=0.001] where females exhibited significantly more muscle activity than males in the reflex time period (p=0.011). The mean muscle activity difference between sex for the reflex time period was 4.12% (females: 7.22 ± 2.80 % MVE; males: 3.10 ± 0.90 % MVE). The reflex time period during extension perturbations for females was signicantly greater than the male and female baseline and anticipatory time periods. No significant differences were found between sex in the baseline or anticipatory time periods (Figure 14).

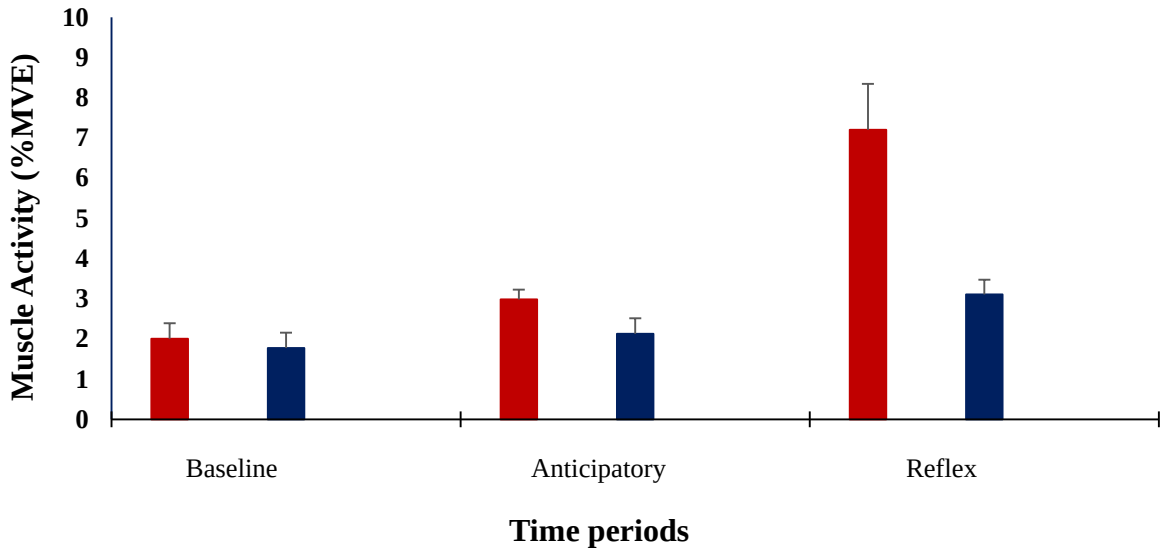

Figure 14. Sex differences in muscle activity (mean + standard deviation) for females (red) and males (blue) for the B) Baseline, A) Anticipatory and R) Reflex time periods

4.7 Muscle Activation: Non-Sex Specific Findings.

Left lateral bend: There was a significant main effect of muscle during left lateral bend perturbations [$F_{5, 330}$=12.362, p=0.001] where the left splenius captis had the greatest muscle activity (5.17 ± 3.10% MVE) followed by the right splenius capitis (4.50 ± 5.0% MVE), right scalene (3.0 ± 3.40% MVE), left scalene (2.60 ± 2.30% MVE), left sternocleidomastoid (2.0 ± 2.40% MVE) and right sternocleidomastoid (1.87 ± 2.30% MVE). There was also a significant main effect of time period for left lateral bend perturbations [$F_{2, 330}$ =20.265, p=0.001] where the reflex time period had the highest muscle activity (3.62 ± 1.31% MVE), followed by the anticipatory time period (2.17 ± 0.88% MVE) and baseline time period (1.61 ± 0.68% MVE).

Right lateral bend: There was a significant condition X time period interaction for right lateral bend perturbations [$F_{2,372}$ =5.507, p=0.004] where muscle activity was similar in the expected

versus unexpected baseline (2.08 ± 1.79% MVE and 2.02 ± 1.56% MVE, respectively) and during the anticipatory time period (2.50 ± 2.28% MVE and 2.70 ± 2.80% MVE, respectively), however, differed in the reflex time period (3.80 ± 4.20% MVE and 5.09 ± 5.20% MVE, respectively). During the reflex time period, expected and unexpected conditions were different (p=0.001), with 1.4% greater muscle activity observed in the unexpected condition. Another significant effect of muscle was found in right lateral bend perturbations [$F_{5, 372}$=5.554, p=0.001] where the left splenius capitis had the greatest muscle activity (4.66 ± 2.20% MVE), followed by the right splenius capitis (4.20 ± 4.40% MVE), right scalene (3.31 ± 4.80% MVE), left scalene (3.30 ± 2.80% MVE), left sternocleidomastoid (2.60± 3.20% MVE) and right sternocleidomastoid (2.10 ± 2.80% MVE).

Flexion: A significant main effect of time period was found during flexion perturbations [$F_{2,372}$=18.141, p=0.001] where the baseline (1.95 ± 1.10% MVE), anticipatory (2.40 ± 0.96% MVE) and reflex (3.50% ± 1.20% MVE) time period all significantly differed from each other.

Extension: A significant muscle X time period interaction was found for extension perturbations [$F_{10, 372}$=1.954, p=0.037] where the left and right splenius capitis exhibited the greatest muscle activity at baseline (3.47 ± 1.73% MVE and 2.65 ± 2.15% MVE, respectively). The left sternocleidomastoid and right scalene had the greatest muscle activity during the reflex time period (5.97 ± 8.90% MVE and 7.23 ± 9.90% MVE, respectively).

5.0 DISCUSSION

This study investigated sex differences in head acceleration, muscle onset times, muscle recruitment patterns and muscle activity magnitudes for competitive ice hockey players in response to a sudden external force application (perturbation). There were sex differences for all measures, however differences were dependent on perturbation direction and timing knowledge, with no consistent neuromuscular strategy that could explain all directional effects.

During left lateral bend perturbations, males and females had similar magnitudes of muscle activity (%MVE) during the different time periods analyzed, but differed in head acceleration and muscle onset timing. Males demonstrated shorter opposition muscle onset latencies whereas females exhibited significantly greater head acceleration. Interestingly, the differences were not bilateral. Right lateral bend perturbations demonstrated that females had greater head acceleration, despite having no differences in muscle onset latencies and muscle activity between sexes. During flexion perturbations, females had significantly greater head acceleration and significantly shorter muscle onset latencies than males. Females also displayed greater muscle activity in the SC whereas SCM produced the greatest activity for males. During extension perturbations, there were no sex differences in head acceleration and males had shorter muscle onset latencies than females. This suggests that there are neuromuscular strategies apparent during extension perturbations that would lead to this head acceleration difference not seen in the other perturbation directions. Females displayed significantly more muscle activity in the reflex time period (time period) of extension with similar activity to males in the baseline and anticipatory time periods. Larger activity in the reflex time period for females can mostly be attributed to the sternocleidomastoid and scalene muscles. These findings are related to Tierney

et al (2005), who demonstrated that physically active females displayed significantly greater head-neck segment peak angular acceleration and a greater proportion of muscle activity in flexion and extension despite having shorter onset latencies of the SCM than physically active males. This study further explored left/right lateral bend directions and observed varying sex-specific neuromuscular strategies when responding to head perturbations.

Expected and unexpected conditions did not affect head acceleration outcomes for males and females, which contradicts Tierney et al, (2005). Unless subjects are unware that a perturbation will take place, Siegmund (2001; 2003) indicated that the addition of knowing the amplitude or timing of the imposed stimulus does not affect the cervical response to low magnitude impacts. It was only when subjects were completely deceived that increases in cervical paraspinal amplitudes and angular head acceleration were observed (Siegmund et al, 2003). In our experiment, positioning the pulley on to a part of the head strap and shifting the participants' body orientation would enable the participant to know the perturbation direction being administered. Likewise, unlike other experiments (Schmidt et al, 2015), earmuffs and eye masks were not used. The magnetized weight drop and preconfigured drop time settings were thought to disable any visual or auditory cues at perturbation onset. That being said, Siegmund et al (2009) demonstrated that visual conditions (eyes open and closed) produced similar cervical kinematic and muscular responses during whiplash-like perturbations. Furthermore, we instructed participants to relax and to not pre-activate neck musculature before the load drop. Other studies have asked participants to pre-activate neck musculature for expected conditions (Tierney et al, 2005). We chose not to do this to observe if sex-specific neural strategies could help reduce head acceleration without pre-activating cervical musculature prior to the perturbation. Moreover, since the perturbation force was minimal, this may have varied the

perceived level of risk for each athlete (Hedlund, 2000). Individuals may have perceived this weight drop as low risk and therefore expressed similar cervical characteristics in the expected condition. Schmidt et al (2015) proposed that football players may have been using risk compensation when high performers (athletes with cervical variables thought to decrease head acceleration) had higher head accelerations than low performers in response to low magnitude head impacts. It's also possible that a habituation effect occurred as per the repeated perturbations. Studies observing repeated sled accelerations have shown decreased muscle activity and increased angular and linear accelerations of the head after successive perturbations (Blouin et al, 2003) Blouin et al (2003) suggested that subsequent platform accelerations after the initial trial may have resulted in a combination of feedforward and feedback control. It was concluded that the nervous system may prefer to minimize stress rather than adopt a neck stiffening strategy. All of these factors could have contributed to lack of differences shown between the expected and unexpected conditions in our study.

Muscle onsets in our study had longer latencies in some muscles than other studies but this is largely dependent on the apparatus administering the perturbations, response criteria and the onset detection algorithm. Tierney et al (2005) reported that males had an average muscle onset time of 42 ± 17.6 ms during forced flexion and 47.8 ± 16.1 ms during forced extension when examining the right sternocleidomastoid and trapezius. In our study, males exhibited right sternocleidomastoid onset times of 44.5 ± 14.6 ms during left lateral bend and 55.1 ± 10.6 ms during extension perturbations. Tierney et al (2005) reported that females had an average muscle onset time of 38.2 ± 15.5 ms during forced flexion and 33.8 ± 8.5 ms during forced extension when examining the right sternocleidomastoid and trapezius. In our study, female muscle onsets of the sternocleidomastoid were much higher than Tierney's study. Females exhibited a SCM

onset time of 60.8 ± 7.8 ms during extension and 55 ± 9.7 ms during left lateral bend. Interestingly, in the non-opposition muscles, female onset times were 40.2 ± 6.49 ms for the left splenius capitis and 48.9 ± 8.8 ms for the right splenius capitis during extension perturbations, which closely relates to the muscle onset times by Tierney et al (2005). This suggest that females displayed shorter muscle onset latencies (when compared to males) on the same-side that the perturbation direction was being administered. This was especially apparent during left lateral bend and extension perturbation directions and suggests that female's exhibit faster same side muscle strategies than males in these two directions. Thus, although non-opposition muscle onsets were represented by a small sample, neck muscle onset strategies exhibited a large variation in this study.

Females responded to the perturbation faster than males during flexion, but this was not true during extension, which differs from previous research (Siegmund et al, 1997; Brault, 2000; Linder et al, 2008; Tierney et al, 2005). More work should be conducted for individualized muscle strategies as these same side muscle strategies are not-necessarily sex-specific. These muscle onset latencies also differed greatly from the sternocleidomastoid and upper trapezius (27.45 ± 3.25 ms) of high performers in Schmidt et al (2015) during forced flexion. A key methodological difference makes direct comparison difficult, given that Schmidt et al, included a muscle pre-load prior to perturbation.

One potential mechanism for the observed variable muscle onset strategies may be due to a lack of torso strapping in attempt to replicate more natural cervical positions observed in sporting situations. Although participant heads were centered by a height adjustable pulley and monitored to maintain a relaxed position with their back against the chair, joint reaction forces and muscle activity from the torso could have contributed to the longer muscle onset latencies

observed in this study. If the torso moved before the onset of the perturbation it could help resist neck motion. Nonetheless, the various neuromuscular strategies evident in our study are important and demonstrate that impact from different directions could transpire unique cervical neuromuscular responses. Olafsdottir et al (2015) has also demonstrated similar findings during multidirectional seated perturbations as they have shown that although some muscles exhibited preferred patterns there were other muscles that varied in direction, and between/within muscles.

Shorter muscle onset latencies may play a role in decreasing head acceleration by resisting neck deformation earlier. Cervical muscle onsets that react within 50ms (Laksari et al, 2015) after impact may be able to effectively aid the inertial segment of the whiplash mechanism and minimize acceleration and transient forces to the brain. In our study, males displayed shorter muscle onset latencies of the opposition muscles during left lateral bend and extension which could have helped dampen external forces. These muscle response from males could have been more prominent if the load drop was heavier. Di Fabio (1990) showed that stimulus intensity alters reflex postural responses, and found that increased weight correlated to decreased muscle onset latency. Females displayed significantly shorter onset latencies in flexion yet had significantly more head acceleration than males. Interestingly, this could oppose the notion that shorter muscle onset latencies decrease head acceleration but rather imply that shorter muscle onset latencies are indicative of earlier load transfer to the neck (Tierney et al, 2005). Thus, as Tierney previously proposed, it may be that female necks are less efficient than males when responding to sudden head loading and therefore react faster because of the earlier change in head movement which triggers the vestibulocollic reflex (VCR) and resultant muscle onset. Tierney et al (2005) also displayed more head acceleration in females despite shorter muscle onset times. However, in our study, males had shorter opposition muscle onsets in left lateral

bend yet had significantly less head acceleration than females. Thus, it is difficult to conclude that muscle onset latencies influenced head acceleration in this study. When relating to ice hockey, these short cervical muscle onset latencies may not be able to increase resistance to the speed and magnitude of concussive like impact because the reflexive attempt may generate too little muscular activity to have preventative effects. More research should be conducted looking at the first onset between various cervical muscles in multi-directional seated perturbations for each individual rather than grouping main muscle contributors in a given direction.

Sex differences in muscle activity magnitudes were apparent during extension perturbations, where females had more than twice as much activity in the left and right sternocleidomastoid and middle scalenes during the reflex time period than males (25-150ms post-perturbation). Perhaps females over-estimated the risk of the load drop when the perturbation device was not kept in their peripheral vision, in this case directly behind them (Hedlund, 2000). In addition, the experimental load drop mass was not changed to reflect relative head mass and therefore females may have required greater muscle activity to counter the load. Females also showed decreased head accelerations during the extension conditions (compared to the other directions - left/right lateral bend, flexion). Relative sternocleidomastoid size differences in relative proportion to the neck may have also contributed to this finding. Balkarran et al (2015) demonstrated that female ice hockey players had smaller sternocleidomastoid volumes in relative proportion to their neck, when compared to males. Females in our study may have needed to use a greater percentage of muscle activation to compensate for the perturbation due to smaller muscle size. Regardless of muscle size, smaller total neck volume may attribute to the larger head accelerations observed in females as more tissue volume is correlated with increased resistance to motion (Chelboun et al, 1997; McNair et al, 2002). In conditions where

females had less muscle activity, they experienced larger head accelerations, especially in left lateral bend and flexion. More investigative work is needed to evaluate muscle volume in relative proportion to neck volume in order to better understand sex differences in competitive ice hockey players.

A larger HC/NC ratio is indicative of more pronounced inverted pendulum loading due to a smaller neck in relative proportion to the head (Vasavada, 2008). When a smaller neck needs to hold up more mass there may be greater head stabilization occurring to compensate for the lack of neck girth (Stokes et al, 2000). This could result in decreased neck stiffness because the base of support to the head (neck) is smaller and more susceptible to movement. Females exhibited a larger HC/NC ratio in our study, but there were no significant muscle activation differences during the baseline or anticipatory time periods, accounting for similar muscle responses between sexes for head-neck balance, pre-perturbation. In the reflex time period however, females may have accounted for this anthropometric difference through compensational mechanisms. Upon impact, females either used less muscle activation than males which increased head acceleration or used more muscle activation than males which decreased head acceleration, depending on the perturbation direction. With this, females may be more susceptible to muscle length deformation and reciprocal head swing if they are not sufficiently contracting neck musculature upon impact (Vladimir, 2012; McCaw, 2014). It would therefore be recommended in this study, that females increase neck girth through resistance training to better mitigate these potential anthropometric differences. This would also share importance for the use of light-weight helmets. If helmets were the same size and weight between males and females, cervical muscle fatigue might occur earlier in females due to the smaller HC/NC ratio.

Schick et al, (2003) reported that concussions are more likely to occur under fatigued conditions in female ice hockey.

Athletes who are aware of head impact but are not used to contracting neck musculature when responding to player-player collision, like in low contact sports, may be at greater risk than athletes who undergo player-player contact more frequently. Since non-contact regulations are enforced in female varsity hockey, these athletes may be less experienced at assessing the severity of impact and responding effectively. Guskiewicz (2013) found that delaying contact in youth may put athletes at greater risk in later years because they have not learned appropriate contact skills. This may also be related to risk compensation where assuming collisions will be penalized and therefore taking larger risks like skating in between two defenders (Hedlund, 2000).

The amount of force (load) used in this study was not capable of causing concussion (Wilcox et al, 2015; Rowson et al, 2012), yet was able to demonstrate sex specific differences in neck muscle response. We showed that by releasing a small (1.5kg) weight, varsity female hockey players that did not use a compensational mechanisms (increased muscle activation) were subject to larger head accelerations than males. This may lead to increased muscle loading, whiplash and concussions when larger forces are used, such is the case in sport. Not only does epidemiological evidence suggest that females are more susceptible to whiplash, their recovery times are longer and treatment is less effective, making whiplash for women much more of a threat than to males (Broshek et al, 2005; Preiss-Farzenegan et al, 2009; Bazarian et al, 2010). It is recommended that women focus on developing hypertrophy in neck muscles so that their relative proportion of muscle activation can withstand a larger or faster collision without causing concussion. This may also help produce more neck muscle co-contraction, which can ultimately lead to neck stiffness

(Holmes and Kier, 2012). More volumetric studies are needed to understand the relative proportion of the muscles in the neck.

6.0 CONCLUSION

Female varsity ice hockey players demonstrated similar levels of muscle activity to males during left/right lateral bend and flexion perturbations, but had larger head acceleration for these directions. In a similar manner, female athletes displayed similar levels of head acceleration during extension but exhibited 4.2 % more muscle activity than males. For our sample population, female athletes displayed either greater muscle activity or head acceleration than males when responding to the external force application. Males could may have displayed less head acceleration due to shorter opposition muscle onset latencies in left lateral bend and extension. That being said, females exhibited shorter muscle onset latencies during flexion yet showed significantly more head acceleration than males. The larger HC/NC ratio and smaller TNV in females may give reason as to why these sex differences exist. Few studies have looked at more than one prime muscle for a given direction. This study suggests that the necks neutral posture in ice hockey players may be prone to unique neuromuscular patterns for head impact inducing left/right lateral bend, flexion or extension of the neck. Future work should further investigate relative contributions, neck muscle size, co-contraction and head stiffness for this particular athletic group.

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CHAPTER 4: CONCLUSIONS AND FUTURE CONSIDERATIONS

CONCLUSIONS AND FUTURE CONSIDERATIONS

This study showed that female ice hockey players used significantly more cervical muscle activation (extension direction) to display similar head acceleration responses to males. If females did not exhibit significantly more muscle activity than their male counterparts they were subject to higher levels of head acceleration (left/right lateral bend and flexion directions). This is especially concerning because greater head acceleration is associated with severity of concussion (Wilcox, 2015). This highlights the need for cervical contraction pre-impact. Although timing knowledge did not alter the kinematic response in this study (perhaps due to habituation effect and low risk load drop), loud warning signals from teammates regarding collision risk could reduce the chance of concussive injury. This heightened female susceptibility to head acceleration also emphasizes the danger of competitive co-ed sporting leagues.

Muscle onset latencies differed between sexes and may play only a small role in resisting our low magnitude load drops. No consistent trends were found between shorter muscle onset latencies and reduced head acceleration. More research is needed to analyze the individual effects of cervical muscle onset latencies while observing natural sporting positions. Some of the subjects in this study could have used their torsos to help resist the perturbation which may have affected cervical muscle activity. Likewise, although the 1.5kg absolute load was thought to invoke a robust neuromuscular response between sexes, relative loading as per Schmidt et al (2015) better accommodates for body weight variability but doesn't necessarily address differences in neck size and strength.

Anthropometric measurements (TNV and HC/NC ratio) showed weak relationships in reducing head acceleration. Despite this, female necks in this study were smaller than males in

relative proportion to the head. This increases inverted pendulum loading and may induce muscular fatigue more quickly especially when wearing a helmet. More head perturbation studies should observe neck muscle fatigue between sexes.

Cervical risk factors identified in athletes pre-season would allow time to prepare proper preventative interventions like emplacing larger neck guards, conducting exercise regimes or emphasizing self-awareness techniques. This could lessen an athlete's potential risk of undergoing a concussion and minimize the chances of head and spinal injury. That being said, Mansell (2005) showed that an 8 week resistance training program did not enhance dynamic stabilization of the neck in collegiate soccer players in response to external force application despite increases in isometric cervical strength and girth. It was concluded that the training program may have been ineffective for reducing low magnitude head accelerations and plyometric training was suggested. This could have also been due to individualized risk compensation and habituation effect thus disassociating neck perturbation studies to real-life situations (Hedlund et al, 2000; Blouin et al, 2003). Nonetheless, perturbation studies have projected insight into the whiplash mechanism and have established the significance of neck stiffness and resultant decreases in head acceleration which subsequently is thought to reduce concussion. Athletes, coaches, parents and staff who are not aware of concussion severity or whom choose to lessen their importance may subject the player to second impact syndrome, cognitive impairment and neurodegenerative diseases later in life (Bey and Ostick, 2009; Gueskiewicz et al, 2003; Amen et al, 2011; De Beaumont et al, 2007). Contrastingly, being more aware of concussions may lessen sport aggressiveness, impulsivity and reluctance to engage in contact (Mihalik et al, 2011).

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CHAPTER 5: APPENDICES

Anthropometrics and Range of Motion Collection

Subject number _____Date____/____/____/

Name_____Height_____Weight_____

Anthropometrics		
Head Height	Vertical distance between the vertex and gonion	
Head Width	M-L distance at widest head point	
Head Depth	A-P distance between glabella and opisthocranion	
Head Circumference	Widest area around head – superciliary ridge to opisthocranion	
Neck Length	Vertical distance between the C7 and tragus (average of left and right ear)	
Neck Width	M-L distance of the neck above the thyroid cartilage (C4)	
Neck Depth	A-P distance of the neck above the thyroid cartilage (C4)	
Neck Circumference	C4 circumference medical tape	

Neck length/circumference	
Neck width/neck depth	
Head circumference/neck circumference	

Range of Motion (Goniometer)	
Flexion/ Extension	Instruct seated subject to perform neck flexion or extension. Set up by aligning fulcrum on auditory meatus. Measure by keeping the stable arm vertical and the moving arm in line with nostrils
Left/Right Lateral Flexion	Instruct standing subject to retain neutral posture. Align fulcrum on C7. Measure by keeping the stable arm on the spinous processes of thoracic vertebrae while the movable arm follows the cervical vertebrae. Take a neutral measurement. Instruct the subject to laterally flex their head left or right then record and minus from neutral.
F	
E	
LLB	
RLB	

Example Data Collection Spreadsheet

Subject Number_____ Date_____/_____/_____/

Camera File	EMG File	Condition (Expected/ Unexpected)	Timing (sec)	Direction	Trial
					MVC LLB
					MVC RLB
					MVC F
					MVC E
					Preliminary
					Preliminary
		E	5	E	1
		U	18	E	2
		U	11	E	3
		E	5	E	4
		U	21	E	5
		E	5	E	6
		E	5	F	7
		E	5	F	8
		E	5	F	9
		U	28	F	10
		U	6	F	11
		U	14	F	12
		U	12	RLB	13
		E	5	RLB	14
		U	31	RLB	15
		E	5	RLB	16
		U	12	RLB	17
		E	5	RLB	18
		E	5	LLB	19
		U	25	LLB	20
		U	7	LLB	21
		U	14	LLB	22
		E	5	LLB	23
		E	5	LLB	24
					Extra
					Extra

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.

Task	Left		Right	
1. Writing				
2. Drawing				
3. Throwing				
4. Scissors				
5. Toothbrush				
6. Knife (without fork)				
7. Spoon				
8. Broom (upper hand)				
9. Striking Match (match)				
10. Opening box (lid)				
Total (count checks in both columns)				

Difference	Cumulative TOTAL	Result

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 TOTAL” cell (round to 2 digits if necessary) and multiply by 100; enter the result in the “Result” cell.

Interpretation (based on Result):

below -40 = left-handed
+40 = right-handed
between -40 and +40 = ambidextrous


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