Motor performance of participants with chronic neck pain and asymptomatic participants pre- and post-spinal manipulation using separate eye and head movement Fitts' tasks

by

Geoffrey Gelley

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List of Abbreviations:

ANOVA	Analysis of variance
APB	Abductor pollicis brevis
CBI	Cerebellar inhibition
CCPA	Canadian Chiropractic Protective Association
CE	Constant error
CNS	Central nervous system
COR	Cervico-ocular reflex
CSP	Cortical silent period
EI	Extensor indices

FT	Fitts' task
HVLA	High velocity low amplitude
ID	Index of difficulty
LICI	Long-interval cortical inhibition
MEP	Motor evoked potential
MT	Movement time
OKR	Optokinetic reflex
PA	Peak acceleration
PV	Peak velocity
ROM	Range of motion
RT	Reaction time
SCNP	Sub-clinical neck pain
SEP	Somatosensory evoked potential
SICF	Short-interval cortical facilitation
SICI	Short-interval cortical inhibition
SM	Spinal manipulation
SMT	Spinal manipulative therapy
TMS	Transcranial magnetic stimulation
ttPA	Time to peak acceleration
ttPV	Time to peak velocity
VAS	Visual analog scale
VE	Variable error
VOR	Vestibulo-ocular reflex
WAD	Whiplash associated disorder

Abstract

Background: Spinal manipulation (SM) is an intervention for chronic neck pain. The proposed mechanisms responsible for the benefits of SM have been investigated, and previous Fitts' task (FT) studies that measured motor performance in participants with chronic neck pain have reported improved head movement time following cervical SM.

Objective: The primary purpose was to examine the effect of cervical SM on eye and head movement tasks in chronic neck pain participants who were symptomatic or asymptomatic at the time of the study. A secondary purpose was to determine if the eye and head movement time/index of difficulty (MT/ID) slopes will be different for the amplitude and width conditions.

Method: This is an observational study that involves a cervical SM intervention on symptomatic participants (n=20) and asymptomatic controls (n=20). All participants completed an eye movement and head movement FT before and after cervical SM, to identify behavioral and kinematic outcome variables related to changes in eye and head movements.

Results: During the eye movement FT, there was no significant effect of SM on MT. During the head movement FT, there was a significant SM x Group interaction on MT of the symptomatic group. There was a significant effect of SM on MT for the amplitude and width conditions within the symptomatic group. Within the asymptomatic group, there was a significant SM x side interaction for the amplitude condition such that MT increased following SM for right horizontal head movements but decreased following SM for left horizontal head movements. The saccade MT/ID slope was asymmetrical with amplitude being steeper than width. The head MT/ID slope was symmetrical for amplitude and width slopes.

Conclusion: There was no effect of SM on MT during the eye movement FT. However, the significant effects of SM on MT during the head movement FT indicate that participants demonstrated changes in the performance of head rotations following SM, suggesting potential neuromuscular and sensorimotor adaptations. The asymmetry for the amplitude and width conditions for saccades suggests these elements of Fitts' Law are separable rather than interdependent. The head movement task produced symmetrical amplitudes and widths that are consistent with Fitts' Law.

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Dedication

The completion of my PhD in Applied Health Sciences has been both motivating and challenging. Trying to balance my responsibilities as a chiropractic clinician and a PhD student has allowed me to discover my true capabilities. Throughout all this, my wife Alayna and my son Reese have been my foundation. It is only through their love, support, and understanding that I managed to stay focused and on purpose.

Introduction

Background

Cervical spine pain is a serious public health problem. According to the Global Burden of Disease study of 291 musculoskeletal conditions, neck pain ranks twenty-first in terms of the overall burden and fourth in terms of overall disability on society (Hoy et al., 2014; Safiri et al., 2020). Due to its prevalence, neck pain is a common reason for patients to present to health care providers such as chiropractors, physiotherapists, and primary care physicians (Bussières et al., 2016; Gross et al., 2010).

Cervical spine pain is defined as neck pain, with or without referred pain into the upper extremities, that is of at least one day's duration (Haldeman, Carroll, Cassidy, Schubert, & Nygren, 2008; Hurwitz, Randhawa, Yu, Côté, & Haldeman, 2018). The duration of neck pain can be described as either of recent onset of 0-3 months (acute to subacute), or persistent (chronic) pain that is greater than 3 months (Bussières et al., 2016).

Various spinal structures including the intervertebral discs, facet joints, myofascial tissues, and ligaments are common sources of spinal pain (Urits et al., 2019; Vlaeyen et al., 2018). However, the ability to reliably identify the specific tissue responsible for spinal pain has been called into question and therefore chronic neck and low back pain are considered to be non-specific in origin when there is no identifiable pathology (Hartvigsen et al., 2018; Vlaeyen et al., 2018). Given that chronic neck pain affects individuals and society as a whole (Hoy et al., 2014; Safiri et al., 2020), evidence-based, effective, and safe interventions are required in the management of this condition (Hartvigsen et al., 2018).

Spinal manipulation

Spinal manipulation (SM) is a widely administered and evidence-based manual intervention for acute and chronic spinal pain (Beliveau et al., 2017; Gross et al., 2010). While mostly provided by chiropractors (Beliveau et al., 2017), SM is also used by physiotherapists and osteopaths (Fryer, Morris, & Gibbons, 2004; Gross et al., 2010; Reed, Liebschner, Sozio, Pickar, & Gudavalli, 2015). The SM research conducted to date has been extensive and has vastly increased our understanding of some aspects of the mechanisms involved with the benefits of this therapeutic intervention (Reed, Cranston, Onifer, Little, & Sozio, 2017; Reed, Long, Kawchuk, & Pickar, 2014). In fact, numerous studies have reported that biomechanical and neurophysiological mechanisms likely underlie the benefits of SM (Bialosky et al., 2014; Boal &

Gillette, 2004; Gay, Robinson, George, Perlstein, & Bishop, 2014; Gyer, Michael, Inklebarger, & Tedla, 2019; Herzog, 2010; Pickar & Bolton, 2012; Pickar & Wheeler, 2001; Reed et al., 2014). However, the exact and complete mechanisms responsible for the benefits of SM such as pain modulation, tissue healing and repair, and restoration of functional ability, to a large degree remain uncertain (Bialosky et al., 2014; Gorrell, Conway, & Herzog, 2020; Gyer et al., 2019).

Spinal manipulation is typically applied to hypomobile segments of the spine. Chiropractors examine these hypomobile segments via manual palpation to identify changes in tissue texture and tone, elicit pain or tenderness of the paraspinal tissues, and to find restrictions of spinal segmental motion (Fryer et al., 2004; Haneline, Cooperstein, Young, & Birkeland, 2008; Stochkendahl et al., 2006). The beneficial effects of a chiropractor delivering a spinal manipulative thrust includes restoring normal spinal intersegmental motion and normalizing aberrant afferent input from the spinal segments (Haavik & Murphy, 2012; Pickar & Bolton, 2012; Reed et al., 2015).

Spinal manipulation is a mechanical intervention that is typically delivered by hand utilizing a high velocity, low amplitude (HVLA) thrust applied to hypomobile spinal segments (Gelley, Passmore, & MacNeil, 2015; Herzog, 2010; Reed et al., 2014). The initial studies on SM were primarily focused on measuring the applied forces and vertebral displacements that occurred during the manipulative thrust (Herzog, 2010; Reed et al., 2014) to assist in explaining the analgesic and hypoalgesic effects of SM (Gyer et al., 2019). The thrust kinetics can be visualized as a force/time history characterized by a pre-load phase, a thrust phase (rapid rise to peak force), and a resolution phase. Neural responses are increased with higher peak forces, faster rates of force application, and shorter thrust durations (Gorrell et al., 2020; Reed, Cao, Long, Kawchuk, & Pickar, 2013). A 2015 study provided a description of the acceleration properties of the manipulative thrust and reported that acceleration amplitudes varied significantly at various spinal levels, with relatively little differences in the acceleration latencies (Gelley et al., 2015).

The biomechanical changes that occur with SM are thought to be due to vertebral displacement and the mechanical response of the tissues to the manipulative input (Evans & Lucas, 2010). The vertebral displacement during the manipulative thrust is complex and often results in several adjacent vertebral segments being mobilized (Gyer et al., 2019; Ross, Bereznick, & McGill, 2004). It has been proposed that biomechanical changes elicited by SM

include: (1) the release of entrapped synovial folds or meniscoids; (2) the restoration of buckled motion segments; (3) the reduction of articular and periarticular adhesions; and (4) the normalization of hypertonic muscles by reflexogenic effects (Cramer, Budgell, Henderson, Khalsa, & Pickar, 2006; Gyer et al., 2019). Even though the above biomechanical changes in motion have been quantified, there is no convincing evidence the biomechanical changes are lasting, and in fact they are mostly transient in nature (Bialosky, Bishop, Price, Robinson, & George, 2009; Funabashi, Kawchuk, Vette, Goldsmith, & Prasad, 2016).

The success of a SM is often judged by clinicians and patients to occur in the presence of a cracking sound, commonly known as cavitation (Dunning et al., 2013; Gorrell et al., 2020). Joint cavitation during SM was once thought to be related to the collapse of a bubble within the synovial fluid as the articular surfaces are separated (Evans & Lucas, 2010; Kawchuk et al., 2015). The bubble collapse theory of cavitation was contradicted by a 2015 study that used Magnetic Resonance Imaging (MRI) to identify that the joint cracking is related to cavity inception. Specifically, as the joint surfaces resist separation during manipulation there is a subsequent formation of gas cavities within the synovial fluid. This process of cavity inception has been termed tribonucleation (Kawchuk et al., 2015). Previous studies have reported that cavitation during SM is associated with clinical benefits; however, there is controversy within the literature as to the role of cavitation (Gorrell et al., 2020; Herzog, 2010). The occurrence of cavitation during SM has been associated with an attenuation of the stretch reflex of the lumbar erector spinae muscles (Clark et al., 2011) and a reduction in corticospinal and spinal reflex excitability (Fryer & Pearce, 2012). These studies are important to quantify and characterize the manipulative input to the spinal tissues, as this may provide some insight as to how the specific mechanisms of SM may produce a clinical benefit and also take into consideration the non-specific effects of SM (Gevers-Montoro, Provencher, Descarreaux, Ortega de Mues, & Piché, 2021). The cavitation associated with SM makes it difficult to blind participants to the intervention and, therefore, study participants are often required to be unfamiliar with SM to help ensure that blinding is successful (Puhl, Reinhart, Rok, & Injeyan, 2011).

Integral to the principles of treating spinal pain is the premise that ongoing spinal segmental pain alters afferent input resulting in increased excitability of spinal interneurons (George, Bishop, Bialosky, Zeppieri, & Robinson, 2006; Sillevis, Cleland, Hellman, & Beekhuizen, 2010), and alters cortical processing due to an intense nociceptive barrage entering

the CNS (Flor, Braun, Elbert, & Birbaumer, 1997; Haavik-Taylor & Murphy, 2007; Haavik & Murphy, 2011, 2012; Haavik Taylor & Murphy, 2010). The spinal segmental displacement induced by the manipulative thrust suggests that mechanical processes may also contribute to the neurophysiologic effects of SM (Gyer et al., 2019; Herzog, 2010; Pickar, 2002; Pickar & Bolton, 2012; Reed et al., 2014), and therefore there has been a paradigm shift toward a neurophysiologic mechanism of SM (Gyer et al., 2019).

It is thought that either the thrust input or the changes in spine biomechanics affect afferent input to the CNS or peripheral nervous system (Pickar & Bolton, 2012). The thrust force of SM imparts changes to sensory input and subsequently modifies neural integration; either by directly affecting reflex activity, and/or by affecting central neural integration within motor, nociceptive, and possibly autonomic neuronal pools (Bialosky et al., 2014; Gyer et al., 2019; Haavik & Murphy, 2012; Pickar, 2002). Individuals with chronic pain have different cortical responses to pain than healthy individuals, which can result in increased pain sensitivity (Coronado et al., 2012; Millan, Leboeuf-Yde, Budgell, & Amorim, 2012). de Camargo et al., (2011) report that SM results in increased mechanical pain thresholds in patients with neck pain and therefore the clinical effectiveness of SM could result from the reduction of central sensitization.

It has been reported that SM produces altered EMG activity in paraspinal muscles (Herzog, 2010; Lehman, 2012; Pickar, 2002; Pickar & Bolton, 2012). SM may also alter ascending and descending pain-modulating elements of the nociceptive system (Boal & Gillette, 2004; Savva, Giakas, & Efstathiou, 2014) and may elicit a reduction in temporal summation of pain (Bishop et al., 2015; Randoll et al., 2017).

According to several studies, the HVLA thrust of SM evokes discharges from muscle spindle Ia afferents and high threshold mechanoreceptors (Lehman, 2012; Pickar, 2002; Pickar & Bolton, 2012; Reed et al., 2014), and increases corticospinal excitability (Niazi et al., 2015). The discharge from these afferents appears to be related to the preload and rate of force application of the SM thrust that subsequently affects neuromuscular responses and muscle spindle discharge (Nougarou, Dugas, Deslauriers, Page, & Descarreaux, 2013; Reed et al., 2014) and loads spinal tissues such as joint capsules, ligaments, and intervertebral discs (Funabashi et al., 2016). More specifically, when the peak force is increased (Nougarou et al., 2013), or the pre-load force is decreased (Nougarou, Dugas, Loranger, Page, & Descarreaux, 2014; Reed et al., 2014), or when

the stimulus latency is of short duration (Page, Nougarou, & Descarreaux, 2016; Pickar & Bolton, 2012), the amplitude of neuromuscular responses and muscle spindle discharge will increase. These studies report a variety of neural responses occurring in the peripheral and central nervous systems following SM. What is not clear is whether these neurophysiologic changes following SM are indicative of a clinically meaningful outcome or merely transient neural responses (Gyer et al., 2019). The above studies also serve to better clarify as to how the biomechanical input of SM affects the neurophysiologic processes, and could further enhance our understanding of the clinical effectiveness of SM.

It has been established that SM is able to mediate autonomic responses such as changes in: skin blood flow, blood pressure, pupillary reflex response, and heart rate variability (Sillevis et al., 2010; Wirth et al., 2019). These autonomic nervous system changes after SM would suggest a sympatheto-excitatory response involving the sympathetic nervous system (Gyer et al., 2019). However, there is conflict in the literature regarding whether activation of the sympathetic nervous system following SM (Benarroch, 2006) since others report no change in sympathetic activity after SM (Benarroch, 2006; Sillevis et al., 2010). Of special interest to health care providers that perform SM is the potential modulatory effect of the sympathetic nervous system on pain and inflammation which may help explain some of the neurophysiologic effects observed after SM (Bialosky et al., 2009; Pickar, 2002).

Patient-based and performance-based outcome measures

Spinal manipulation research has utilized a variety of patient-based (Khorsan, Coulter, Hawk, & Choate, 2008) and performance-based outcome measures (Passmore & Descarreaux, 2012) to track patient symptoms and function. Of the patient-based measures, the most commonly cited in the literature include: Oswestry Pain & Disability Index (Oswestry), Roland-Morris Low Back Pain & Disability Questionnaire, Short Form 36 (SF-36), The Numeric Pain Rating Scale (NPRS), Vernon-Mior Disability Index (NDI), and the Visual Analog Scale (VAS) (Khorsan et al., 2008). Passmore & Descarreaux (2012) reviewed performance-based outcome measures which included: biomechanically constrained performance measures; spinal range of motion and spinal stiffness; neurologically constrained performance measures; force production, EMG, heart rate variability, and perceptual motor and complex task-constrained performance measures; pressure algometry (perceptual); head repositioning accuracy; Fitts' task and other complex motor skill tasks. These patient-based and performance-based outcome measures have

been used to measure how a mechanical event such as SM can impact the objective and subjective changes in patients with spinal pain (Khorsan et al., 2008; Passmore & Descarreaux, 2012).

Fitts' Law

Fitts' Law tasks can be used as perceptual motor outcome measures as Fitts' Law is one of the most studied and robust laws of motor behavior (Beamish, Bhatti, MacKenzie, & Wu, 2006). First published by Paul Fitts (1954), the Law is identified mathematically as MT= a + b (ID) where ID = $\log_2(2A/W)$. Movement time (MT) is defined as the interval between the onset of the movement and the termination (offset) of the movement; ID is the index of difficulty of the task; A is the amplitude (distance) of movement; W is the width of the target; and a/b are constants. The ID is measured in binary digits referred to as bits (Schmidt & Lee, 2011) and describes the fundamental trade-off between movement accuracy and speed. Schmidt and Lee (2011) define a bit as "the amount of information required to reduce the original amount of uncertainty by half." Fitts' Law has an adjustable index of difficulty (ID) which is derived from the alteration of the amplitude of the movement (A) or the size of the target (W).

Fitts' Law predicts there is a linear relationship between ID and MT wherein MT will increase with Fitts' tasks that have a higher ID. Furthermore, when the size of the designated target decreases, an individual will trade speed for accuracy thereby increasing MT to reach the target (Fitts, 1954; Fitts & Peterson, 1964; Passmore, Burke, Good, Lyons, & Dunn, 2010; Passmore & Descarreaux, 2012; Smith, Dainoff, & Smith, 2006). Fitts' Law is also resistant to learning (Beamish et al., 2006; Schmidt & Lee, 2011), indicating the task could be repeated over time with only small measurable changes being associated with the individual's physical ability to perform the task (Passmore et al., 2019).

Fitts' tasks have been used to attain functional measurements of motor performance in those patients with neck pain as compared to a healthy control group (Lersa, Stinear, & Lersa, 2005; Passmore & Descarreaux, 2012; Smith et al., 2006), or in asymptomatic participants with palpable cervical joint restrictions (Passmore et al., 2010). A significant improvement in MT during a Fitts' task occurred in participants with neck pain (Lersa et al., 2005; Passmore & Descarreaux, 2012; Smith et al., 2006) and asymptomatic participants (Passmore et al., 2010) after receiving SM.

The mechanism by which SM improves MT during a Fitts' task in patients with neck pain is not well understood. The mechanism of effect of SM on MT could be biomechanical and/or neurophysiological in origin. Support for a biomechanical mechanism is based on increased cervical ROM along with improved MT during a Fitts' task (Passmore et al., 2010; Passmore & Descarreaux, 2012; Smith et al., 2006). Whereas a change in a behavioral finding such as MT performance suggests a neurophysiologic basis as MT is less impacted by task difficulty and may neurologically, rather than biomechanically, be related to the site of manipulation (Marchand, Cantin, Murphy, Stern, & Descarreaux, 2014; Passmore et al., 2010; Treleaven, 2008). The neurologic component of SM and motor performance have been explored via changes in motor control and cortical somatosensory evoked potentials, whereby these changes were found to be directly related to cervical SM (Haavik & Murphy, 2012; Passmore, Murphy, & Lee, 2014).

Cervico-ocular reflex and cervical proprioception

The cervico-ocular reflex (COR) is a reflexive eye response that is elicited by neck rotation. The COR is responsive to movements of the head in relation to body trunk movements (de Vries et al., 2016; Ischebeck et al., 2018). The COR acts concurrently with the vestibulo-ocular reflex (VOR) and the optokinetic reflex (OKR), acting on the extraocular muscles to maintain a stable vision of our surroundings on the retina (Montfoort et al., 2006; Treleaven, 2008; Treleaven, Jull, & Grip, 2011). The upper cervical spine is densely innervated by mechanoreceptors and muscle spindles and therefore provides an important role in proprioception (Treleaven, 2008, 2017), and controlling the movement of the eyes and body posture to coordinate eye and head movement (Li & Peng, 2015). The upper cervical afferents project to the superior colliculus which serves as a reflex center between the visual system and neck movement and is thereby responsible for contributing to eye-head coordination (Treleaven et al., 2011).

The COR receives input from the afferents of the facet joints and muscle spindles of the cervical spine from C1 to C3 (H. V. Heikkila & Wenngren, 1998; Hikosaka & Maeda, 1973; Treleaven, 2008). The reflex pathway of the COR is not entirely known, but is theorized to be derived from cervical afferents that ascend ipsilaterally in the spinal cord, then decussate to the contralateral side in the brainstem at the level of the inferior olive, and then project to the vestibular nuclei (H. V. Heikkila & Wenngren, 1998; Hikosaka & Maeda, 1973; Ischebeck et al., 2018; Sato, Ohkawa, Uchino, & Wilson, 1997). Increased activity of cervical muscle spindles

results in excitation of contralateral vestibular nuclei neurons via a disynaptic pathway relaying in the central cervical nucleus and decussating in the spinal cord (Sato et al., 1997).

Changes in oculomotor control have been associated with cervical spine dysfunction due to altered neck proprioception directly related to aberrant sensorimotor information from cervical mechanoreceptor activity (Gimse, Tjell, Bjorgen, & Saunte, 1996). Palmgren et al., reported that SM can improve cervical spine proprioception, as measured by improved sensorimotor integration and improved eye-tracking control (Palmgren, Sandstrom, Lundqvist, & Heikkila, 2006). Further, it has been established that spinal manipulation alters sensorimotor integration (Haavik & Murphy, 2012) and affects the specific areas of the brain that are associated with oculomotor control (Lelic et al., 2016). Sensorimotor integration (SMI) – within the context of this thesis – is the process by which the CNS coordinates afferent information from different parts of the body and integrates with the motor system to control movement.

Gimse et al. (1996) and Treleaven (2017) reported that altered proprioception of the facet joints and deep muscles of the neck plays a significant role in the pathogenesis of whiplash associated disorders (WAD). A variety of symptoms such as headache, neck pain, visual disturbances, tinnitus, dizziness, and fatigue are associated with WAD (Eck, Hodges, & Humphreys, 2001; Johnston, Daye, & Thomson, 2017; Treleaven, 2008). These symptoms are thought to be related to an imbalance between altered proprioception from the cervical spine and normal afferent input from the vestibular and visual systems (Ischebeck et al., 2017; Treleaven, 2008, 2017).

Dysfunction of the proprioceptive system in WAD patients may affect oculomotor function (de Vries et al., 2016; H. V. Heikkila & Wenngren, 1998; Ischebeck et al., 2017) as well as eye-head coordination, gaze stability, and saccadic eye movements (Gimse et al., 1996; Treleaven, 2008; Treleaven et al., 2011). It has also been reported that proprioceptive dysfunction can result in poor motor control might be a factor in individuals with non-traumatic neck pain (Treleaven, 2008). Therefore, it is reasonable to consider that altered proprioception from muscles of the cervical region could also induce changes in oculomotor control and saccadic eye movements in patients with non-traumatic mechanical neck pain.

Sensorimotor integration

The research of Haavik and Murphy (Haavik & Murphy, 2012; Passmore, Murphy, et al., 2014), reports that cervical spine segmental dysfunction creates a state of altered afferent input

from the cervical region, which then contributes to altered somatosensory filtering and processing due to central neuroplastic changes within the cortex and cerebellum. These authors further suggest that the altered somatosensory changes result in aberrant afferent feedback from the spine and extremities leading to altered sensorimotor integration of this afferent input, which then contributes to altered motor control with subsequent pain and disability. Moreover, Haavik & Murphy propose that SM delivered to dysfunctional cervical joints normalizes the afferent input from the neck, thereby normalizing somatosensory integration to produce normal motor control and reduce pain and disability. Haavik & Murphy's research provides evidence that utilizing somatosensory-evoked potentials (SEPs) can identify neurophysiologic changes after cervical SM. Specifically, their SEPs work revealed a decrease in the parietal N20 and frontal N30 SEPs waveform components which are integral to quantifying SMI (Haavik-Taylor & Murphy, 2007; Haavik Taylor & Murphy, 2010). They also incorporated transcranial magnetic stimulation (TMS) into their research methods (Daligadu, Haavik, Yielder, Baarbe, & Murphy, 2013; Taylor & Murphy, 2008) to further illustrate changes in sensorimotor integration following cervical SM.

To date, the use of extremity (Smith et al., 2006) and head (Passmore et al., 2010; Passmore & Descarreaux, 2012) movement Fitts' tasks studies have noted changes in MT. However, the express purpose of these studies was not to differentiate between a biomechanical or neurophysiologic mechanism for SM. While both are changed with SM intervention, it has not been established that either head/extremity ROM or MT have been determined to have a biomechanical and/or neurophysiologic underpinning.

It has been determined that COR is propagated from the afferents of the upper cervical spine (Hikosaka & Maeda, 1973; Kelders et al., 2005), and that dysfunction of the cervical joints can lead to changes in oculomotor function (Gimse et al., 1996; H. V. Heikkila & Wenngren, 1998; Kelders et al., 2005) and subsequent changes in saccadic eye movements (Treleaven, 2017; Treleaven et al., 2011).

Saccades

The visual and perceptual outcomes of eye movements are of interest to basic scientists, and clinicians such as neurologists and ophthalmologists. Furthermore, the study of eye movements has made a contribution to the motor control knowledge base (Robinson, 1986). Saccades are rapid conjugate eye movements that shift our gaze to move the fovea of both eyes

in the same direction between successive fixation points (Kowler, 2011; Leigh & Zee, 2015), and respond with appropriate motor behaviors (Robinson, 1986). Saccades allow objects of interest to be focused on the fovea, which sees images in greater detail and acuity. The termination of a saccade is a fixation point that holds the image steady on the fovea to ensure detail and acuity. Collectively, saccades and fixations are part of eye movements that enable us to navigate our environment (Leigh & Zee, 2015).

The amplitude, velocity, and duration of saccades demonstrate a consistent relationship. For example, large amplitude saccades will have a greater velocity and longer duration than saccades of smaller amplitude. However, the duration of most saccades is less than 70 milliseconds (Leigh & Zee, 2015). The decision making process and control of a saccade involves a number of cortical areas (Kennard, 2011). Even though the study data on the saccade circuitry is extensive, there are still questions on where and how the saccade is generated, and the contribution of the respective areas within the circuitry (Moren, Shibata, & Doya, 2013).

The saccade generating circuits of the brainstem receive commands from cortical areas, in particular the frontal eye fields. These commands are directed to the superior colliculus, which is a pivotal structure in integrating and relaying these commands (Leigh & Zee, 2015; Moren et al., 2013). The performance of a horizontal saccade originates in the caudal pons, in particular the paramedian pontine reticular formation (PPRF) (Horn, 2006), which receives projections from the superior colliculus (Leigh & Zee, 2015). There is a delay of approximately 200 msec between the stimulus for a saccade, the release from its fixation, and its actual performance. This time delay allows for neural processing in the retina, cerebral cortex, superior colliculus, and cerebellum (Leigh & Zee, 2015), with the rostral pole of the superior colliculus having a prominent role in the release of the fixation (Dorris & Munoz, 1995, 1998).

Horizontal saccades of both eyes require reciprocal innervation to the medial rectus (Oculomotor nerve – Cranial nerve III) and lateral rectus muscles (Abducens nerve – Cranial nerve VI) (Optican, Rucker, Keller, & Leigh, 2008). Saccade generation requires critical input from two types of neurons: burst cells and omnipause cells (Sparks & Mays, 1990). The horizontal oculomotor muscles via the motoneurons and interneurons of the right abducens nucleus, receive saccadic commands from excitatory burst neurons in the PPRF (Buttner & Buttner-Ennever, 2006; Horn, 2006; Optican et al., 2008). The excitatory burst neurons also project to inhibitory burst neurons to inhibit the motoneurons and interneurons of the left

abducens nucleus (Moren et al., 2013). Contralateral excitatory and inhibitory burst neurons are also inhibited by the inhibitory burst neurons (Optican et al., 2008; Yoshida, Iwamoto, Chimoto, & Shimazu, 1999).

Burst cells within the PPRF trigger a pulse-step to initiate a saccade. The pulse is a velocity command that moves the eyes to the target, while the step is an eye position command that holds the eyes at the target (Leigh & Zee, 2015). The burst cell activity creates the saccadic pulse to generate the forces necessary to overcome the orbital tissue resistance, while the saccadic step reflects the higher level of tonic innervation of the agonist eye muscle which holds the eye in its new position against the orbital elastic forces (Leigh & Zee, 2015). There is a transition period between the pulse and the step, with this change in input for the saccade being referred to as a pulse-slide-step (Optican et al., 2008).

Interneurons within the PPRF will project to the contralateral medial rectus muscles via fibers that decussate and ascend in the medial longitudinal fasciculus (Leigh & Zee, 2015). The excitatory burst neurons in the PPRF also excite latch neurons which will inhibit omnipause neurons until the saccade is completed (Missal & Keller, 2002). Latch neurons are located in the reticular formation, while the omnipause neurons are located within the pontine nucleus raphe interpositus (RIP) (Buttner & Buttner-Ennever, 2006). Omnipause neurons discharge continuously, but as their names suggest, will pause just before and during a saccade (Leigh & Zee, 2015). The omnipause neurons would otherwise inhibit the excitatory burst neurons if they were not completely turned off when the saccade was initiated (Optican et al., 2008).

Inhibitory burst neurons will also receive projections from excitatory burst neurons. The saccade neural circuitry also receives input from inhibitory burst neurons which are located just caudal to the abducens nucleus in the nucleus paragigantocellularis dorsalis of the dorsomedial portion of the rostral medulla (Horn, 2006; Strassman, Highstein, & McCrea, 1986). The inhibitory burst neurons receive inhibitory inputs from the omnipause neurons and contralateral inhibitory burst neurons and contralateral excitatory inputs from the superior colliculus (Shinoda, Sugiuchi, Izawa, & Takahashi, 2008).

For a right horizontal saccade, the motoneurons in the right abducens nucleus drive the right lateral rectus muscle. Abducens internuclear neurons within the right abducens nucleus send axons to the left oculomotor nucleus of the left eye to drive the medial rectus muscle. Inhibitory burst neurons decussate across the midline to the contralateral abducens nucleus to

inhibit the contralateral abducens motor neurons during horizontal saccade, thereby inhibiting antagonist muscle activity (Strassman et al., 1986). Neurons distributed throughout the medial vestibular nuclei and nucleus prepositus hypoglossi, located bilaterally within the brainstem as well as the cerebellar flocculus, serve as neural integrators to control eye position. In other words, the excitatory burst neurons provide velocity information to these brainstem neurons to integrate velocity signals to position signals (Leigh & Zee, 2015). This is equivalent to mathematical integration. This position signal is then relayed to the abducens neurons that control the lateral rectus muscles (McFarland & Fuchs, 1992). The omnipause neurons will then inhibit the excitatory burst cells, and hence latch neuron inhibition of OPN, to maintain eye fixation once the saccade has been completed (Missal & Keller, 2002).

Rationale for the thesis

The principal investigator of this thesis has frequently noted, over 30 years of experience as a chiropractic clinician, that a number of patients with neck pain will often complain of associated symptoms of blurred vision, diplopia, or reduced visual acuity. Other studies (Treleaven, 2008, 2017; Treleaven et al., 2011) have also reported similar visual disturbances associated with neck pain. The PI of this thesis has also noted these visual disturbances often improve after the patient undergoes a trial of cervical SM. While these patient outcomes observed by the PI are anecdotal, they have provided an impetus to question why and how these visual disturbances improve following a trial of cervical SM and provide a background for the rationale of this thesis.

Altered neck proprioception associated with cervical spine joint dysfunction has been reported to be related to aberrant afferent input from cervical mechanoreceptors that can result in changes in oculomotor control (Cade, Jones, Holt, Penkar, & Haavik, 2021). This oculomotor dysfunction has been associated with changes in saccadic eye movements (Gimse et al., 1996; Treleaven, 2008; Treleaven et al., 2011). Fitts' Law has been found to robustly apply to saccades, wherein MT increases linearly with higher indexes of difficulty (Chi & Lin, 1997; Heath, Samani, Tremblay, & Elliott, 2016; Wu, Kwon, & Kowler, 2010). Given the sensorimotor changes observed in patients with neck pain (Bialosky et al., 2014; Haavik & Murphy, 2012; Passmore, Murphy, et al., 2014; Pickar, 2002; Pickar & Bolton, 2012), this study will serve to identify potential changes in saccade MT related to motor performance and sensorimotor changes, that may occur following cervical SM.

This study used an eye-tracker system to record and measure the MT of the participants' eyes (horizontal saccades) during the performance of a computer-based Fitts' task. Using an eye-tracker device to measure saccades is a commonly used method when investigating eye movements that follow a target (Gadotti et al., 2016). The method involved with an eye movement Fitts' task have been established in the literature (Chi & Lin, 1997; Heath et al., 2016). Any change in MT performance following SM could suggest a neurophysiologic mechanism, as eye movements are controlled by the CNS. The current evidence that altered motor control (Haavik & Murphy, 2011, 2012) and deficits in eye movement and the COR (Kelders et al., 2005; Treleaven et al., 2011) are observed in some patients with neck pain supports the exploration of this experimental method.

Abnormal cortical processing of afferent information has been proposed to be paramount in the development and persistence of functional performance impairment in patients with neck pain (Haavik-Taylor & Murphy, 2007; Haavik & Murphy, 2011, 2012; Haavik Taylor & Murphy, 2010). Therefore, it follows to reason that the improvements noted by Haavik and Murphy (2012) and Treleaven et al., (2011) might result in the subsequent improvement in MT of the eyes during a Fitts' task following SM is provided to patients with chronic neck pain.

This study will include a head movement Fitts' task, which has been previously used to identify a reduction in head MT in asymptomatic participants (Descarreaux, Passmore, & Cantin, 2010) and in participants who had chronic neck pain (Passmore & Descarreaux, 2012), after receiving SM. The head movement Fitts' task will serve as a biomechanical comparator to the eye movement Fitts' task, which in theory has a neurophysiologic basis as a measure of motor performance (Chi & Lin, 1997).

Deficits in the control of head and eye movements have been observed in patients with neck pain (Bexander & Hodges, 2012; Johnston et al., 2017; Treleaven, 2008). Neck pain patients have also demonstrated improved head repositioning after spinal manipulation (H. Heikkila, Johansson, & Wenngren, 2000; Palmgren et al., 2006). Cervical sensorimotor control can be used as an assessment of cervical spine dysfunction through the measurement of the control and accuracy of head movements (de Zoete, Osmotherly, Rivett, & Snodgrass, 2020).

There is an inter-relationship between the COR and alteration in sensorimotor integration as the COR function remains altered due to aberrant cervical afferent input resulting in impaired sensorimotor integration (Ischebeck et al., 2017; Treleaven, 2008, 2017). The use of both head

and eye movement tasks is justified as the process of eye-head coordination involves compensatory eye movements and cervical spine motor control that requires the integration of saccades and the COR (Ischebeck et al., 2016; Johnston et al., 2017).

Since the COR involves cervical afferents (H. V. Heikkila & Wenngren, 1998; Hikosaka & Maeda, 1973; Treleaven, 2008) that project to the superior colliculus (Treleaven et al., 2011), dysfunction of these segments can affect oculomotor function (de Vries et al., 2016; H. V. Heikkila & Wenngren, 1998; Ischebeck et al., 2017) and in particular saccadic eye movements (Gimse et al., 1996; Treleaven, 2008; Treleaven et al., 2011).

In addition, cervical spine segmental dysfunction alters somatosensory integration due to neuroplastic changes within the cortex and cerebellum (Haavik & Murphy, 2012; Passmore, Murphy, et al., 2014). These authors also assert that SM applied to these dysfunctional cervical segments normalizes afferent input from the cervical spine and thereby normalizes sensorimotor integration. Saccade commands are integrated within the superior colliculus which then projects to the PPRF to initiate horizontal saccades involving the oculomotor muscles (Horn, 2006). The integrated neural circuitry for the COR, sensorimotor integration, and horizontal saccades offers a mechanism as to how saccadic MT is increased in patients with neck pain, but also serves to explain how SM when applied to dysfunctional cervical joints can alter oculomotor function (Cade et al., 2021), and hence decreased saccade MT.

There is a growing body of evidence regarding the impact of cervical SM or any adaptations that may occur regarding cortical processing and altered motor control. The eye-tracker has been used to explore whether patients with chronic neck pain have improved eye MT during a Fitts' task and to quantify their motor performance. The proposed experiments will build on previous Fitts' task models that measured motor performance in patients with neck pain to quantify the biomechanical and neurophysiologic underpinning of the altered motor performance as demonstrated by functional changes following SM (Passmore et al., 2010; Passmore & Descarreaux, 2012; Smith et al., 2006).

This study will provide a better understanding of performance-based outcome measures to the wider scientific community for future investigation of motor control and CNS changes that may occur following SM in patients with cervical spine pain. This proposed investigation may further delineate the possible interplay between the biomechanical and neurophysiologic mechanisms of cervical SM during a Fitts' task by quantifying the changes in MT of the head

and MT of saccades, which may benefit the understanding of motor performance beyond the effects of learning and practice. A clear delineation of a biomechanical versus a neurophysiologic mechanism to explain altered motor performance during a Fitts' task, pre and post SM, is unlikely due to the multimodal mechanisms associated with spinal manipulation (Passmore et al., 2010). However, this study will provide data on changes in head MT and saccade MT following SM, wherein head movement is a combination of biomechanical and neurophysiologic mechanisms, and saccades involve a neurophysiologic mechanism.

Additionally, we will also explore the kinematic variables of movement to provide the details associated with the planning, execution, and online control of movement (Passmore, Johnson, et al., 2014; Passmore et al., 2019; Passmore et al., 2015). The current evidence suggests that the effects of SM are related to a cascade of neurophysiologic mechanisms which are beyond the observed biomechanical changes (Schmid, Brunner, Wright, & Bachmann, 2008). Basic science and clinical research seems to dichotomize biomechanics and neurophysiology into separate and distinct phenomena when in fact they are very much interconnected (Triano et al., 2013).

It has been reported that goal-directed reaching (Heath, Weiler, Marriott, Elliott, & Binsted, 2011) and saccadic eye movements (Heath et al., 2016) demonstrate MT values that increase with greater target amplitudes, but are refractory to changes in target width. More specifically, the MT/ID slopes are steeper for movement amplitude than for target width. In addressing this issue, it has been reported that MT values increase with increasing movement amplitude (Al-Aidroos, Fischer, Adam, & Pratt, 2008; Heath et al., 2016; Heath et al., 2011). On this basis, we will contrast if eye and head movements have interdependent or separable MT/ID relations for amplitude and width conditions within symptomatic and asymptomatic chronic neck pain groups.

This study may serve to better explain the biomechanical and neurophysiologic mechanisms associated with the changes in motor performance during eye and head movement Fitts' tasks. The measurable biomechanical effects of SM are not in dispute, but these effects do not completely explain the clinical benefit of SM, therefore, additional measures are required to explain the clinical effectiveness of SM. Studies have established that the kinetics and kinematics of SM appear to have a relationship with the subsequent neurophysiologic effects (Gyer et al., 2019; Herzog, 2010; Pickar, 2002; Pickar & Bolton, 2012; Reed et al., 2014); that is, a unique

dose-dependent neurophysiologic response (Bishop et al., 2015; Cambridge, Triano, Ross, & Abbott, 2012; Nougarou et al., 2013). Understanding the biomechanical and neurophysiologic mechanisms that underly SM may help explain the etiology of neck pain, as well as identify the appropriate patients to receive and respond favorably to SM. Identification of the mechanisms of action of SM could facilitate a greater acceptance of SM, along with the utilization of this manual therapy in a clinical setting (Bishop et al., 2015).

Objectives

The purpose of this study was to examine the effects of manipulation of the cervical spine on eye and head movement tasks in a population of chronic neck pain participants who were symptomatic or asymptomatic at the time of the study.

The specific objectives of this study are:

1. To determine if cervical spine manipulation has an effect on the time needed to complete task-oriented eye and head movements.
2. To determine if the time needed to complete task-oriented eye and head movements is different within symptomatic and asymptomatic groups.

It is hypothesized that:

1. The time needed to complete eye and head movement tasks will be influenced by cervical spine manipulation.
2. The time needed to complete eye and head movement tasks will decrease for symptomatic participants following cervical spine manipulation.

An additional purpose of this study was to determine the difference in MTs with increasing IDs during specific amplitude and width conditions for task-oriented eye and head movements in the same study population.

The specific objectives of this portion of the study are:

3. To determine if the saccade MT/ID slope will be different for the amplitude condition compared to the width condition within symptomatic and asymptomatic groups.
4. To determine if the head MT/ID slope will be different for the amplitude condition compared to the width condition within symptomatic and asymptomatic groups.

It is hypothesized that:

3. Saccades will demonstrate a steeper MT/ID slope as MT increases with increasing ID values for the amplitude condition compared to the width condition within symptomatic and asymptomatic groups.
4. Head MT/ID slopes will be similar as MT increases with increasing ID values for the amplitude and width conditions within symptomatic and asymptomatic groups.

Review of Literature

Movement time during a Fitts' task

Smith and colleagues (2006) set out to determine the effect of SM on MT of the upper extremity by using a mouse cursor during a computerized Fitts' task (FT). Participants (n=10) in the intervention group received HVLA manipulation to areas of spinal joint dysfunction. The intervention group participants demonstrated a significant improvement ($p<0.05$) in MT following SM and had reduced MT compared to the no intervention group. However, one participant in the control group did have reduced MT. The average improvement in MT for the intervention group was 183 msec (9.2%), and 29 msec (1.7%) was seen in MT for the one participant in the no intervention group. Smith's study is identified as a randomized controlled trial, yet the randomization process and the method of treatment allocation concealment is not described. As such, the investigators enrolling participants in this study could possibly foresee assignments and thus introduce selection bias to the study, and it becomes difficult for differences in MT to be confidently attributed to SM.

A single-blind trial (Passmore et al., 2010) was conducted on 15 asymptomatic participants with segmental restrictions at the C1-2 level who were randomly assigned to either a SM group or no intervention group. Movement time during a FT, as well as active cervical ROM, were measured pre- and post-SM in the intervention group. The same outcome measurements were also performed in the no intervention group. When the segmental restriction at C1-2 was in the direction of the FT movement, it was considered congruent, whereas if the movement was opposite, it was considered incongruent. For the SM group, active cervical ROM increased post-SM ($p<0.02$). Further, significant group differences were present for MT in the congruent ($F_{8,48} = 2.83$; $p<0.02$; $\eta_p^2 = .320$) and incongruent ($F_{8,48} = 2.31$; $p<0.05$; $\eta_p^2 = .278$) conditions in the SM group, as compared to the no intervention group. The study authors concluded that SM increases active cervical ROM and further promotes the performance of neck rotation during a FT. In addition, they concluded that a linear relationship exists, as predicted by Fitts' Law, between ID and MT. It would be informative to see how the results of this study would be reported with the addition of a neck pain group.

These studies (Passmore et al., 2010; Smith et al., 2006), report the quantity of movement in participants based on ROM and MT. It would be of interest to examine the quality

of participant performance during the Fitts' task through utilization of the kinematics of motor performance after SM.

A (2010) study by Descarreaux, Passmore & Cantin, addresses the kinematics of motor performance during a Fitts' task in participants with chronic neck pain and with a healthy control group. Participants were required to perform a head movement Fitts' task under four ID conditions. The quality of head movement during the Fitts' task also included the measurement of acceleration and deceleration phase duration. Movement time increased with the increasing ID in both groups, which is consistent with the speed-accuracy trade-off of Fitts' Law. Decreased motor performance was noted in chronic neck pain patients who had an increase in MT ($p=0.034$) and deceleration phase duration ($p=0.012$), as compared to healthy controls during the head movement Fitts' task. The participants with chronic neck pain were able to complete the Fitts' task as accurately as healthy controls, but to maintain accuracy the neck pain group increased their MT by increasing the duration of the deceleration phase to get as near to the target as possible.

The speed-accuracy trade-off of Fitts' Law was reported in an experiment that examined saccades between targets (Chi & Lin, 1997). In this study, the saccadic eye movements of participants ($n=10$) were tracked under different ID conditions. Being one of the earliest studies to use of eye-tracker technology during a Fitts' task, it is notable that saccade MT was significantly related to target width and amplitude. However, the speed-accuracy trade-off was contrary to that predicted by Fitts' Law in that a longer MT occurred with a larger target amplitude, and a larger target width rather than a smaller target width.

In contrast, Wu et al, (2010) reported on an oculomotor Fitts' task that was consistent with Fitts' Law in the time taken to complete a saccade in a four-target sequencing task. This study's results showed that the duration of the saccade sequence lengthened with increasing ID for amplitude and width manipulations, whereas the MT for the primary saccades within each sequence scaled to the amplitude manipulations of the ID. Participants in this study utilized a strategy of secondary saccades to correct the aiming errors of the primary saccades. The primary saccade was the first saccade from one target to the next target. Secondary saccades are error correcting saccades used to offset the aiming errors of the primary saccades. The frequency of these secondary saccades increased with larger target amplitudes but decreased with larger target sizes. What this study suggests is that to achieve a higher accuracy, participants required the use

of secondary corrective saccades rather than increasing the MT to aim the primary saccades more carefully. They concluded Fitts' Law was best predicted by the frequency of the secondary saccades rather than the primary saccades. Wu et al. provide a rather restrictive conclusion regarding secondary saccades. Perhaps a more obvious conclusion to his study would be that primary saccades do not adhere to Fitts' law.

A recent study (Heath et al., 2016) used a video-based eye-tracking system to examine whether MT for saccades was based on the unitary nature of Fitts' Law, whereby target size and amplitude both contribute to the ID. More specifically, they contrasted ID to MT while altering the target widths and amplitudes. Reported results demonstrated that MT increased with increasing ID within the changing width and amplitude conditions. The MT for saccades demonstrated robust linear relationships with amplitude (R^2 = .99) and width (R^2 = .76). Further, the ID/MT slope was steeper for the amplitude condition (b =18) versus width (b = 2). These results indicate that saccade MT for a given ID was greatly influenced by amplitude, rather than by target width. They further stated that these separable slopes were counter to Fitts Law that asserts that the MT for any two responses is equivalent given a constant ratio between target amplitude and width. Moreover, the conclusion of this study implied that the width and amplitude components of Fitts' Law are separable rather than interdependent, and that the amplitude condition was associated with greater information processing demands than the width condition.

Both Wu et al. (2010) and Heath et al. (2016) conclude that saccade MT increases with a more difficult ID. However, the Heath study goes on further to report that the speed-accuracy trade-off for amplitude and width conditions are separable and not interdependent as MT was more influenced by movement amplitude than target width.

Cervico-ocular reflex (COR) and neck pain

Patients (n=8) with whiplash-associated disorder (WAD) and age-matched controls (n=8) underwent cervico-ocular reflex (COR) testing to measure proprioceptive deficit related to the facet joints or deep cervical muscles (Kelders et al., 2005). This study used an apparatus that passively rotated the participant's body on a chair with the head fixed, and subsequently induced slow compensatory eye movements and fast correcting saccades. The gain of the response was defined as "the amplitude of the eye velocity fit divided by the peak velocity of the chair movement" (Kelders et al., 2005). Patients diagnosed with WAD had significantly increased

COR gain values at a wider range of stimulus peak velocities, with a maximum difference at lower frequencies ($p<0.037$) when compared to healthy controls. Kelders et al. concluded that the sensitivity of the COR measurement is satisfactory to detect the presence of cervical spine injury.

A subsequent study also utilized an infrared eye-tracking device to measure the eye movements of 13 patients and 18 age-matched healthy controls to determine whether the reported increase gain of the COR in WADs is accompanied by changes in the vestibular-ocular reflex (VOR) and optokinetic reflex (OKR) (Montfoort et al., 2006). This study used the same methodology as Kelders et al (2005). Based on their analysis, Montfoort and colleagues reported a significant increase in COR gain in whiplash patients with no change in VOR or OKR.

A cross-sectional study involving participants with non-specific neck pain (n=37) and healthy controls (n=30) used and infrared eye-tracking device to measure COR while the participant was in a dark room on a rotating chair (de Vries et al., 2016). The experimental design was identical to the above studies (Kelders et al., 2005; Montfoort et al., 2006). Analysis of Covariance (controlling for age and sex) demonstrated that participants with non-specific neck pain had increased COR gain ($F_{1,62}=4.15$, $p=.046$) when compared with healthy controls. This study reports that the increased COR gain in the participants with non-specific neck pain could be related to altered afferent input from the cervical spine. They also suggested that patients with neck pain tend to avoid cervical end range of motion which could alter afferent information from the cervical segments and affect the gain of the COR.

Ischebeck et al. (2017) conducted a cross-sectional study of 91 chronic neck pain patients subdivided into a WAD neck pain group, a traumatic neck pain group, and a non-traumatic neck pain group. A comparison was made with 30 healthy controls. COR gains were measured with the participant's head fixed and sitting on a rotating chair in a darkened room. Neck pain patients had a higher COR gain compared with healthy controls. However, the COR gain did not differ between the three neck pain groups. They concluded that the etiology of the patient's neck pain was not associated with oculomotor dysfunction.

A 2011 study (Treleaven et al.) investigated eye-head coordination and gaze stability in participants with chronic WAD (n=20) and asymptomatic controls (n=20). Using wireless motion sensors and electro-oculography, this group was able to measure unconstrained head rotation, head rotation during gaze stability, and sequential eye and head movements. The WAD

group had significantly less maximal eye angle to the left end range of head movement during the gaze stability task, and decreased velocity of head movement in head-eye co-ordination and gaze stability tasks compared to the control group ($p < 0.01$). Gaze stability and head-eye coordination disturbances may be related to deficits in COR activity associated with decreased head ROM and neck pain. The study authors acknowledge that electro-oculography has relatively low resolution and indicated it might be necessary to use a faster sampling rate and/or a different technology to track eye movements.

These studies all report an alteration in COR gain in patients with neck pain that is most likely the result of impaired neck range of motion and altered proprioception from dysfunctional cervical facet joints. Further, these studies confirm the possibility of eye movement impairments in patients with neck pain. A pragmatic 2008 study by (Montfoort, Van Der Geest, Slijper, De Zeeuw, & Frens) verified this concept by measuring the eye movements of whiplash patients and healthy controls that wore a rigid cervical collar for 2 hours. Both the reduced neck mobility group and the whiplash group demonstrated an increase in the COR gain suggesting that increased COR gain in WAD patients may be related to reduced neck mobility. The resulting increased COR gain in healthy controls using a rigid cervical collar was replicated in a 2018 study (Ischebeck et al.). However, this does not rule out alteration in cervical muscle proprioception which will require more sophisticated experiments to fully address this issue.

The above studies measured the COR in isolation via eye stabilization reflexes. However, the COR is influenced by visual, vestibular, and cervical motor control (Kelders et al., 2005; Montfoort et al., 2006; Montfoort et al., 2008). Therefore, it is impossible to influence COR gain intentionally and use COR as a proxy for oculomotor function. But if patients decrease their neck range of motion (e.g. pain, altered motor control, kinesiophobia), the oculomotor system has to compensate for the reduced afferent input from the cervical spine with an increase in COR gain (de Vries et al., 2016; Ischebeck et al., 2018). A limitation of the above COR studies is their use of a 250 Hz sampling frequency for saccades which could result in the reduced accuracy of capturing peak saccade velocities. Peak velocity capture can be restored to 95% accuracy with sampling rates greater than 250 Hz (Mack, Belfanti, & Schwarz, 2017).

Sensorimotor Integration

Somatosensory-evoked potentials and cervical spine manipulation

Haavik-Taylor and Murphy (2007) utilized somatosensory-evoked potentials (SEPs) to examine the immediate neurophysiologic effects of cervical SM on participants with subclinical neck pain (SCNP). Somatosensory-evoked potential components were collected in response to median nerve stimulation at the wrist of the participant at baseline and for 30 minutes after the interventions. There was a significant decrease in the amplitude of the parietal N20 ($p=0.006$) and frontal N30 SEP ($p=0.009$) components in response to manipulation, with these changes lasting approximately 20 minutes. The N20 SEP component is generated in the primary somatosensory cortex and is the earliest marker of cortical processing (Rossi et al., 2003). The primary somatosensory cortex corresponds to Brodmann's area 3b in the parietal lobe (Rossi et al., 2003). The N30 SEP component is related to complex cortical and subcortical loops linking the basal ganglia, thalamus, pre-motor areas, and primary motor cortex. The N30 peak is thought to reflect early sensorimotor integration (SMI). The authors advise that the method of recording SEPs potentials in this study does not exclude the possibility that subcortical changes did occur. There were no significant changes in either latency or amplitude of the SEPs components in the passive head movement group.

A more complex study design by Taylor & Murphy (2008) investigated the effects of SM on dysfunctional cervical joints, and the subsequent changes in the intrinsic inhibitory interactions within the sensorimotor system. Three SEPs trials were carried out with stimulation of the median nerve, ulnar nerve, and simultaneous stimulation of both nerves pre/post SM. Various SEPs components obtained from simultaneous median and ulnar stimulation divided by the individual stimulation of the median and ulnar nerves were used to calculate SEPs ratios. There was a significant ($p=0.003$) decrease in the ratio for the cortical N30 component, after manipulation of the cervical spine. No significant changes were noted during passive head movement controls. This study reports that the N30 cortical ratio change reflects the ability of the CNS to attenuate the dual peripheral nerve input (defined as "surround-like inhibition"), on the basis that there was a decrease in the amplitude of the N30 SEP peak after cervical SM. The attenuation or suppression of this dual nerve input allows the body to perceive the dual stimuli as separate and process them accordingly.

A follow-up SEPs study (Haavik Taylor & Murphy, 2010) set out to determine the resulting response of the CNS to a motor task after SM to dysfunctional cervical joints. This study repeated the dual peripheral nerve stimulation technique. The experimental group received cervical SM just prior to a typing task, while the control group was exposed only to the typing task. There was a significant increase in the median and ulnar nerve stimulation ratio (MU/M+U) for the N20 and N30 components after the typing task. However, for the group that received cervical SM prior to the motor task, the MU/M+U ratios for the N30 SEPs component decreased significantly ($p=0.005$). From these data, this study concluded that SM prior to the motor training task resulted in a significant decrease in the dual SEPs ratio for one of the cortical SEPs components and was most likely related to the ability of the CNS to filter and integrate the dual peripheral nerve input.

The data from these SEPs studies has demonstrated that SM of dysfunctional cervical joints can alter somatosensory processing and early SMI of input from the upper limb, as demonstrated via alterations in the amplitude of N20 and N30 cortical SEPs peaks. In addition, the dual peripheral nerve studies indicate that patients with SCNP have a reduced ability to suppress dual nerve input, but this ability improves after cervical SM which increases the surround-like inhibition. The above research group's extensive work with respect to altered SMI related to dysfunctional cervical joints was performed on participants with subclinical neck pain. Response to their work includes the criticism that subclinical neck pain is not generalizable to a neck pain population wherein different SEPs changes may be observed after SM in those patients with neck pain. Some of their experiments did not include a control group with SCNP and therefore these studies are unable to decisively conclude that any observed SEPs changes are related to SM.

The SEPs protocols in the above studies were able to demonstrate changes in the N20 and N30 amplitudes following SM. However, the specific areas of the brain involved with the changes in these SEPs components is not identified due to the poor spatial resolution of the EEG measures used in these studies. Similar to the findings of Haavik-Taylor & Murphy (2007), Lelic et al., (2016) observed reduced SEP amplitude in the N30 brain region after SM compared to sham-manipulated controls. Further the use of swLORETA (standardized weighted Low-Resolution Brain Electromagnetic Tomography) as a brain source localization technique enabled detection of the pre-frontal cortex as being the source of change in SMI as demonstrated by

reduced activity of 20.2 ± 12.2% (p=0.03) following SM. The N30 has several complex inputs with the prefrontal cortex being one of the known sources of the N30 SEPs component. This study suggests that SM potentially improves motor performance via changes in the prefrontal cortex.

One of the main issues involving the research of Haavik and Murphy is the question of how long the change in N30 lasts after SM. It has been demonstrated that the N30 change is present for at least 20-30 minutes after SM (Haavik Taylor & Murphy, 2007). To expand on this further, a pilot study (Haavik, Niazi, Holt, & Murphy, 2017) was performed to investigate the potential long-term changes in SMI that may occur following cervical SM. The authors opine that by using a dual SEPs protocol and comparing the SEP ratios serves as a protective measure against the variations that may occur in placement of the recording and stimulating electrodes. Electrode placement variability can affect SEP amplitudes, in comparison to measuring SEP data evoked from stimulation of a single peripheral nerve. Moreover, the dual SEPs method is an ideal technique to measure the long-term sensorimotor integration changes that may occur with spinal manipulation.

Haavik et al. (2017) reported that chronic neck pain patients (n=6) were provided with 12 weeks of cervical SM, resulting in a significant decrease in the MU/M +U ratio ($p<0.001$) and the MU amplitude ($p<0.031$) for the cortical P22-N30 SEP component. There was also a significant decrease in the visual analog scale (VAS) following SM and at 12 weeks. There was no change in the control group. This study proposes that the decreased frontal N30 peak is related to the filtering of sensory information from the upper limb (surround-like inhibition of the dual peripheral input). The conclusions of this study should be viewed with caution due to the small sample size. Another important limitation to note was the control group was not matched for the same 12-week period as the group receiving cervical SM. As such, the changes in dual SEP ratios after 12 weeks may have occurred in the control group as well.

Transcranial magnetic stimulation and cervical spine manipulation

Through the utilization of Transcranial Magnetic Stimulation (TMS), it has been reported that SM of dysfunctional cervical joints can alter sensorimotor input from the upper limb (Daligadu et al., 2013; Taylor & Murphy, 2008). Single and paired pulse TMS protocols were used to explore the inhibitory and facilitatory reciprocal actions within the sensorimotor cortex on participants with SCNP after cervical SM (Taylor & Murphy, 2008). The study outcome

measures included: short-interval intracortical inhibition (SICI) and short-interval intracortical facilitation (SICF), cortical silent periods (CSP), and motor evoked potentials (MEP) to measure corticomotor excitability (Haavik et al., 2021).

The evoked potentials of the target muscles are knowns as MEPs. Changes in the size of MEPs are an indication of upper and lower motoneuron excitability and their ability to activate the corticospinal tract that projects to the target muscle (Gyer et al., 2019; Haavik et al., 2021). When the TMS stimulus is delivered during the target muscle contraction, the MEP is followed by an interruption of voluntary muscle contraction known as a cortical silent period (CSP). Any change in the size of the MEP or CSP reflects a change in motor control (Haavik et al., 2021). MEPs were recorded from the abductor pollicus brevis (APB) and extensor indices (EI) muscles of the upper extremity following cervical SM. In addition, F-wave activity was recorded at the wrist of the participant to assess for changes in spinal excitability following median nerve stimulation. The F-wave is a late response that follows the motor response elicited by TMS. The F-wave provides a means of examining transmission between stimulation sites of the arm and leg and the related motoneurons of the cervical or lumbosacral spinal cord (Klomjai, Katz, & Lackmy-Vallee, 2015).

A significant decrease in SICI ($p=0.02$) and increase in SICF ($p=0.003$) for the APB muscle in comparison to head movement controls were said to reflect changes in SMI of the upper extremity. There was a significant shortening ($p=0.01$) of the CSP in the APB muscle. Conversely, it was observed that for the EI muscle there was a significant decrease ($p=0.001$) in SICF and a significant lengthening ($p=0.01$) of the CSP. No MEPs or F-wave response changes were identified, suggesting there were no lasting changes in spinal excitability after SM. There were also no changes observed in the passive head movement controls. This study suggests that an increased CSP in the APB muscle may be representative of intracortical inhibition as the greater duration of the CSP is reflective of a greater SICI.

A study by Daligadu and colleagues (2013), investigated patients with SCNP to determine whether alterations in cerebellar efferent activity exist, and if SM prior to motor sequence learning would affect sensorimotor integration between the cerebellum and motor cortex. TMS was used to test an experimental group ($n=10$) before and after a combined intervention of SM and motor sequence learning (typing random numbers on a keypad). The same protocol was also applied to 10 healthy control participants who did not receive the SM

intervention. Dependent measures included: short-interval cortical inhibition (SICI), long-interval cortical inhibition (LICI), and cerebellar inhibition (CBI). Short-interval cortical inhibition and LICI were recorded to determine the level of cortical inhibition, whereas CBI was measured to ascertain if there was any inhibitory effect of the cerebellum on the motor cortex. A significant improvement in task performance ($p<0.0001$) was reported for the experimental group, as exhibited by a 19% reduction in reaction time and a significant decrease in CBI. They report that significant changes in reaction time indicates that motor learning was acquired. Interestingly, the control group demonstrated an improvement in task performance with a 25% decrease in reaction time ($p<0.001$) but no change in CBI. A significant decrease in SICI was also found in the control group, with no change in the experimental group. CBI in the control group was not affected by the motor learning sequence task, but CBI was significantly altered ($F_{1,6} = 7.92$; $p<0.05$) in the experimental group after SM and motor sequence learning. Moreover, cervical SM in patients with SCNP led to a pattern of cerebellar modulation similar to asymptomatic individuals. The findings of this study suggest that SM to dysfunctional cervical joints may improve upper limb task performance and movement by reducing intracortical inhibition. The results of this study remain uncertain as, without a passive head movement control group, the observed changes related to cervical SM are speculative. The presence of a passive head movement or sham manipulation would more adequately control for the non-specific physiologic effects that occur with cervical SM.

The participants in the above two TMS studies had SCNP that was hypothesized may lead to motor control changes and subsequent perpetuation of their clinical symptoms. These studies provide evidence to support that cervical SM influences afferent input from the upper extremity due to a central mechanism of action involving altered neural processing at the cortical level of the brain as well as the cerebellum. How the cerebellum interacts with the motor cortex in patients with recurrent neck pain requires further investigation.

A recent study again utilized a TMS protocol to examine the connectivity between the cerebellum and motor cortex (Baarbe, Yielder, Haavik, Holmes, & Murphy, 2018). This study set out to further investigate cerebellar inhibition (CBI), whereby TMS was provided over the cerebellum prior to stimulation of the motor cortex, resulting in inhibited MEPs. This protocol is compared to TMS stimulation of the motor cortex alone to identify CBI. Since healthy controls have been shown to demonstrate reduced CBI following motor acquisition, this study

hypothesized that CBI would not be reduced, to the same extent, in patients with recurrent neck pain. Further, in patients with neck pain, cervical SM would reduce CBI following a motor acquisition task that required typing on a keyboard. Cerebellar inhibition was measured at baseline and then again following the motor task in a SM group, sham control group, and a no intervention control group. After the motor control task, the sham manipulation group remained inhibited (58 ± 33% of test MEP) while the healthy controls were disinhibited (98 ± 49% of test MEP). In comparison, the SM group underwent cerebellar facilitation (146 ± 95% of test MEP, $p<0.001$). This study concluded that there was greater inhibition of the neck sham group as compared to the healthy control group, suggesting that neck pain may alter the interaction between the cerebellum and motor cortex due to impaired SMI. In addition, SM may reverse the inhibitory effects of neck pain as indicated by a change to facilitation. One of the strengths of this study is that it did include a neck pain control group to differentiate whether neck pain or SM was responsible for the reduced CBI following the motor control task.

Haavik and Murphy's extensive work with respect to altered SMI related to dysfunctional cervical joints was performed on participants with SCNP. The above SEPs and TMS studies do not provide conclusive evidence that SM to dysfunctional cervical joints was the exact cause of the alteration in cortical SEPs peak amplitudes, or cortical and cerebellar inhibition. Moreover, it has not been established that these are either maladaptive or adaptive responses (Haavik & Murphy, 2012). Further research is required to clarify the neurophysiology of these cortical changes and their etiology with respect to a participant's clinical presentation and their ability to undertake their activities of daily living (Haavik-Taylor & Murphy, 2007; Haavik Taylor & Murphy, 2010). More specifically, does altered SMI result in pain in a healthy population (Haavik & Murphy, 2012)? Future studies should be sure to include a no intervention control group. The inclusion of a no intervention control group would have assisted these studies to explore whether any observed changes in cortical SEPs peak amplitudes, or cortical and cerebellar inhibition following SM may be due to cervical intervertebral restrictions.

Extensive research has been completed on the mechanisms of SM (Bialosky et al., 2014; Gevers-Montoro et al., 2021; Gyer et al., 2019; Herzog, 2010; Pickar & Bolton, 2012). By providing a better understanding as to how these SM mechanisms attenuate spinal pain, clinicians and researchers can better determine which mechanisms may affect specific conditions and how this translates into clinical benefits. The evidence presented thus far would suggest that

SM induces biomechanical changes that in turn influence neurophysiologic mechanisms. Insofar as these neurophysiologic mechanisms are concerned, the main effects are related to spinal cord mechanisms including segmental mechanisms of pain inhibition and reduction in the temporal summation of pain (Gevers-Montoro et al., 2021).

Whether the beneficial effect is biomechanical, neurophysiological, or likely a combination of both, these experiments have not been able to identify clinically relevant changes that can be conclusively related to SM (Honoré, Leboeuf-Yde, & Gagey, 2018). Translational research is required to close the gap between basic and clinical research. In the future, randomized controlled trials on the efficacy of SM on spinal pain should include neurophysiologic variables as outcome measures to bridge the gap between experimental and clinical research findings (Clark et al., 2018).

Therefore, this thesis study will examine the effect of manipulation of the cervical spine on eye and head movement tasks in a population of chronic neck pain participants to further delineate the possible interplay between the biomechanical and neurophysiologic mechanisms of cervical SM during a Fitts' task by quantifying the changes in saccade MT and head rotation MT which may benefit the understanding of motor performance. Head movement is a combination of biomechanical and neurophysiologic mechanisms, while saccades involve a neurophysiologic mechanism.

Methods

This is an observational between-groups study that involves a pre and post intervention design utilizing cervical spine manipulation on participants with chronic neck pain and asymptomatic controls.

Participants

One group of participants (n=20; 3 males, 17 females) were symptomatic for chronic neck pain and between the ages of 18 and 40 years (32.5 ± 6.91). All neck pain participants were recruited from the Principal Investigator's chiropractic clinic using poster advertisements. Inclusion criteria for the chronic neck pain participants included neck pain between the superior nuchal line and the first thoracic spinal segment for at least 3 months and have palpable spinal segmental fixations at C1-7. Each participant's neck pain had to be reproducible by neck movements and/or provocative tests. These participants had to have normal or corrected-to-normal vision.

A second group of asymptomatic participants (n=20; 6 males, 14 females) of the same age range (30.8 ± 5.57) were also included. The asymptomatic participants were also recruited from the Principal Investigator's chiropractic clinic through poster advertisements. The asymptomatic participants had to have a history of neck pain between the superior nuchal line and the first thoracic spinal segment of no less than 3 months but were not experiencing neck pain during data collection. They were also required to have palpable spinal segmental fixations at C1-7. These participants had to have normal or corrected-to-normal vision.

Spinal manipulation was provided by an experienced chiropractic clinician (Principal Investigator) to all participants. Patients of the Principal Investigator's clinic had already undergone a comprehensive health history and physical examination prior to testing. Participants were excluded if they had any contraindications to spinal manipulation. Further exclusion would occur if there was any indication of current serious cervical spine conditions such as progressive neurologic deficits, trauma, infection, tumor, osteoporosis, inflammatory spondyloarthropathy, fracture, vestibular/inner ear dysfunction, and a history cervical spine surgery. Participants were excluded from the study if they had been diagnosed with conditions that may affect eye movements: an autonomic disorder such as Horner's syndrome, current ocular and/or retinal disease, diabetes, a history of head trauma, or were currently using opioids, recreational drugs or

have a history of substance abuse.

The participants reviewed and signed informed consent prior to participation in the study in accordance with the Declaration of Helsinki. The World Medical Association developed the Declaration of Helsinki as a statement of ethical principles to safeguard and protect the health of participants volunteering in research studies (Carlson, Boyd, & Webb, 2004). All procedures were approved by the University of Manitoba Biomedical Research Ethics Board [HS23591 (B2020:010)].

All participants completed a 10 cm visual analog scale (VAS) to score their baseline pain intensity at the start of the experimental session (pre-SM). Participants were instructed to assess and indicate the severity of their pain on a 0-10 cm horizontal line (0 = no pain and 10 = worst pain imaginable). A VAS score was also obtained to evaluate any change in each participant's pain immediately after the SM. VAS pain scoring is commonly used and has been shown to exhibit high test-retest correlations (Pool, Ostelo, Hoving, Bouter, & de Vet, 2007; Rosier, Iadarola, & Coghill, 2002). The Neck Disability Index (NDI) was obtained at the start of the experimental session to quantify neck disability related to work and physical activity. The NDI has been reported as a valid and reliable self-reporting outcome measure (Hains, Waalen, & Mior, 1998; Pool et al., 2007; Vernon & Mior, 1991).

Sample Size

Sample size calculations were performed a priori using G*Power v3.1.9.7 software (G*Power© Heinrich-Heine-University, Dusseldorf, Germany) and were based upon an estimated moderate effect size of 0.75 for differences in MT amplitude. For a statistical power level of beta= 0.95 and an alpha < 0.05, a minimum of 20 participants per group would be required.

Instrumentation

Once the consent forms were signed, the participants completed a Visual Analog Scale (VAS) and Neck Disability Index (NDI) questionnaire to obtain baseline pain and disability scores, respectively. Using a counter-balanced order, all participants completed an eye movement and head movement Fitts' task. A counter-balanced experimental design was used so that all participants would eventually undergo the Fitts' task opposite to which they were originally assigned.

Eye Movement Fitts' Task

This portion of the study utilized the EyeLink 1000® (SR Research Version 1.5.0, Mississauga, Ontario, Canada) which is a tower mounted eye tracking device with up to 1000 Hz binocular sampling rate or a monocular 2000 Hz sampling rate. The EyeLink 1000® tracks a user's eye movements via high-speed fiber optic cameras that capture the corneal reflection (Fig. 1). The system consists of two computers: the host computer is dedicated to data collection while the second computer is used for display purposes to present the Fitts' task to the participants. The host computer monitor was visible only to the research assistant and provided real-time saccadic eye movements. The computers were connected via an Ethernet link that allowed the sharing of information between the two computers.

Before the data was gathered for the eye movement Fitts' task, the eye tracker was calibrated using a 9-point calibration and validation performed by the display computer while the participants looked through the eye tracking apparatus and fixated their eyes on black circles on a white background displayed on a 24-inch computer screen (Dell LCD monitor, 1920 x 960 pixels resolution [4.04 pixels/mm]). The fovea of the eye is not located directly opposite the pupil but is 4° to 8° higher. The optical axis (or pupillary axis) is not the same as the visual axis (or line of sight). Calibration becomes necessary as the offset of the fovea differs from person to person (Drewes, 2010).

The targets were displayed on a computer screen as a white circle at the center of a black background to precue the start of the trial, followed by another white circle with variable randomized foreperiods of 1500 to 2100 ms to avoid participant anticipation. Seven different ID conditions were presented by four target diameters (0.5 cm, 1.0 cm, 2.0 cm, and 4.0 cm) and four amplitude distances (1.6 cm, 3.2 cm, 6.5 cm, and 12.9 cm). The calibration sequence, drift correction, and Fitts' task were built using the SR Research Experiment Builder software (SR Research Version 1.5.0, Mississauga, Ontario, Canada).

Eye Movement Fitts' Task Protocol

During the eye movement Fitts' task, participants were seated in a chair positioned in front of a computer monitor with a chin and forehead rest used to restrict head movement. Participants were advised to not apply any eye makeup prior to participation as makeup can interfere with the ability of the eye-tracker camera system to track eye movements. The

participants fixed their gaze on a central white circle, then moved their eyes on the horizontal axis when the central circle disappeared, and a target white circle appeared randomly to the right or left. The participants were instructed to maintain their eye position until the target circle disappeared.

Saccade accuracy is influenced by the instructions provided to a participant, with more accurate saccades being made when accuracy is emphasized over speed (Leigh & Zee, 2015). Therefore, to avoid influencing the participants' performance, they were instructed simply to perform horizontal eye movements as quickly and accurately as possible.

All conditions were conducted in a random order to avoid a sequence effect. An eye drift correction occurred partway through the trials. The post-saccadic drift, or glissade, is related to a mismatch in the sizes of the phasic (pulse) and tonic (step) components of the saccade. Glissades tend to be more frequent in fatigued participants (Leigh & Zee, 2015). The participants had the opportunity to perform 6 practice movements prior to any trial data being collected.



Figure 1. EyeLink 1000® (SR Research Version 1.5.0, Mississauga, Ontario, Canada)

Head Movement Fitts' task

The equipment for the head movement portion of the study was based on a modification of two previous studies (Descarreaux et al., 2010; Passmore et al., 2010). The Headmouse Nano (Origin Instruments Corporation, Grand Prairie, Texas), an infrared optical sensor that tracks head movements, was utilized. The headmouse filtered and transformed the participant's head movements to control a mouse cursor on a 34-inch computer monitor (MSI 34" curved monitor, 3440 x 1720 pixels resolution [4.35 pixels/mm]). The headmouse was mounted to the top portion of the monitor (Fig. 2). A cable connection powered the headmouse via a computer USB port. Participants were seated in a chair that was positioned 60 cm from the computer monitor. Plastic safety glasses worn by the participant had a reflective passive marker (5 mm dot) attached to the nose bridge. The location of the reflective marker was captured and tracked by the Headmouse Nano at a frequency of 80 Hz and a 10 msec latency.

A custom software package (E-prime, version 2.0, Psychology Software Tools Inc, USA) was used to present the Fitts' stimuli. The targets were displayed on a computer screen as a white circle at the center of a black background to cue the start of the trial, followed by another white circle with variable randomized foreperiods of 1500 to 2100 ms to avoid participant anticipation. Seven different ID conditions were presented by four target diameters (1.2 cm, 2.4 cm, 4.8 cm, and 9.6 cm) and four amplitude distances (4.5 cm, 8.3 cm, 15.5 cm, and 29.0 cm).

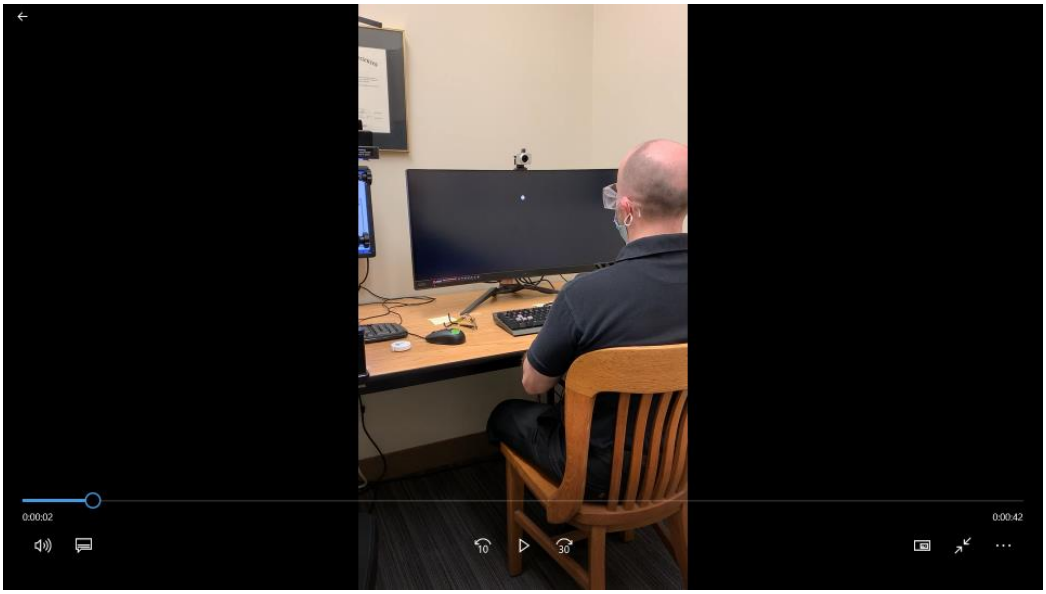




Figure 2. Headmouse Nano (Origin Instruments Corporation, Grand Prairie, Texas)

Head Movement Fitts' Task Protocol

During the head movement Fitts' task, the participants were required to use head movements to control the headmouse cursor. From a neutral head position with the cursor pointed at the central white circle, the participant rotated their head, and thereby the cursor, on the horizontal axis when the target white circle appeared randomly to the right or left. The participants were instructed to maintain their head position until the target disappeared. The participants were instructed simply to perform horizontal head movements as quickly and accurately as possible. To limit the use of sensory feedback, the participants were instructed to not correct their head movement once it was initiated.

All conditions were conducted in a random order to avoid a sequence effect. The participants had the opportunity to perform 6 practice movements prior to any trial data being collected.

The principal investigator, a chiropractor with 32 years' experience, assessed the cervical spine of all participants. To minimize the possible confounding effects of using multiple examiners that can lead to poor inter-examiner reliability, only a single chiropractor was used to assess the cervical spines of all the participants (Lersa et al., 2005). The following criteria was used to detect areas of spinal dysfunction: abnormal joint end feel, abnormal quality of resistance to motion, and reproduction of local or referred pain (Fryer et al., 2004; Haneline et al., 2008; Stochkendahl et al., 2006). The participants received a single high velocity and low amplitude (HVLA) cervical spine rotary manipulation to the previously identified palpable cervical segmental fixation. Prior to the manipulation, the participant was placed supine on a chiropractic treatment table with their arms at the sides of their body. The index finger of the chiropractor's contact hand was placed on the lamina of the restricted cervical segment. The chiropractor then rotated the participant's head contralaterally to the contact hand until the barrier of the cervical segments passive end range was reached. The chiropractor's other hand was placed behind the participant's head to induce gentle neck rotation contralateral to their thrusting hand. The chiropractor delivered a HVLA thrust, with the thrust vector directed towards the participant's opposite eye (Bergmann, 2011). The presence of an audible joint cavitation for any participant during SM was documented by the principal investigator. There is anecdotal evidence that cavitation is related to the successful delivery of a HVLA manipulation (Gorrell et al., 2020).

Once the neck pain and asymptomatic groups had undergone SM, they then completed the post-intervention eye movement and head movement Fitts' tasks. Additionally, the participants completed another VAS questionnaire.

Calculation of indexes of difficulty and viewing angles

The index of difficulty (ID), measured in bits for each trial, was pre-determined by the amplitude of the movement task and the size of the target (Table 1 and 2). The movement amplitude, target size, and appearance on either side of the central target circle was presented in random order to avoid a sequence effect. Seven different ID presentations were represented by four target sizes and four amplitudes. There was a need to have overlapping IDs to identify if performance is due to the relative contributions of movement amplitude or target width. The different target widths and amplitudes were selectively combined to produce 2 conditions for each experiment. For the eye movement width condition (0.5 cm, 1.0 cm, 2.0 cm, and 4.0 cm diameters), each target was presented at a common amplitude of 12.9 cm, whereas for the amplitude condition (1.6 cm, 3.2 cm, 6.5 cm, and 12.9 cm), each target amplitude was presented with a common target width of 0.5 cm. For the head movement width condition (1.2 cm, 2.4 cm, 4.8 cm, and 9.6 cm diameters), each target was presented at a common amplitude of 29.0 cm, whereas for the amplitude condition (4.5 cm, 8.3 cm, 15.5 cm, and 29.0 cm), each target amplitude was presented with a common target width of 1.2 cm.

Calculations, based on the Pythagorean Theorem, produced viewing angles of 1.6, 3.2, 6.5, and 12.9 degrees based on a viewing distance of 80 cm, a viewing height of 48 cm, and four amplitude distances for the eye movement Fitts' task. The same method was used to calculate viewing angles of 4.5, 8.3, 15.5, and 29.0 degrees based on a viewing distance of 60 cm, a viewing height of 48 cm, and four amplitude distances for the head movement Fitts' task.

Exploring the relative importance of movement amplitude and target width allowed inferences to be made on the movement strategies used by participants to complete the task. For each ID condition, a block of 8 trials was completed. The same number of IDs (7) were presented on either side of the central target circle for a total of 112 trials. An eye movement trial was deemed successful when the participant's saccade velocity fell below a threshold of 30°/s for 28 msec (Heath et al., 2016), which corresponds to the participant's eye fixation being within the target limits. A head movement trial was deemed successful if the mouse cursor was pointed

within the target limits and the head movement velocity dropped below 5% of peak velocity (Sjölander, Michaelson, Jaric, & Djupsjöbacka, 2008) for at least 200 msec.

Table 1. *Index of difficulty ([log_2 (2A/W)] = ID) per eye movement.*
The target width corresponding to the amplitude of motion was calculated to obtain the index of difficulty. An asterisk (*) denotes overlapping IDs. The different target widths and amplitudes were selectively combined to produce 2 eye movement conditions: for the width condition (.5 cm, 1.0 cm, 2.0 cm, and 4.0 cm diameters), each target was presented at a common amplitude of 12.90 cm, whereas for the amplitude condition (1.60 cm, 3.20 cm, 6.50 cm, and 12.90 cm), each target amplitude was presented with a common target width of 0.5 cm.

	Target width (cm) = W				
Amplitude (cm) = A		1.60	3.20	6.50	**12.90**
	0.5	2.68	3.68	4.70	5.69*
	1.0				4.69
	2.0				3.69
	4.0				2.69

Table 2. *Index of difficulty ([log_2 (2A/W)] = ID) per head movement.*
The target width corresponding to the amplitude of motion was calculated to obtain the index of difficulty. An asterisk (*) denotes overlapping IDs. The different target widths and amplitudes were selectively combined to produce 2 head movement conditions: for the width condition (1.2 cm, 2.4 cm, 4.8 cm, and 9.6 cm diameters), each target was presented at a common amplitude of 29.0 cm, whereas for the amplitude condition (4.5 cm, 8.3 cm, 15.5cm, and 29.0 cm), each target amplitude was presented with a common target width of 1.2 cm.

	Target width (cm) = W				
Amplitude (cm) = A		4.50	8.30	15.50	**29.00**
	1.2	2.91	3.79	4.69	5.60*
	2.4				4.60
	4.8				3.60
	9.6				2.60

Data Analysis

The saccade raw data was imported to a Microsoft Excel template and plotted. The raw data was then analyzed offline to identify each saccadic event. Each saccadic event was extracted as per the stimulus onset and offset time from the SR Research Experiment Builder software (SR Research Version 1.5.0, Mississauga, Ontario, Canada). Each saccadic event then underwent a Second Order Zero Phase Butterworth Low Pass Filter at 50 Hz. The raw data was filtered so that the process of numerical differentiation is less prone to cumulative error. Each filtered event was also plotted. The filtered events served to provide the gaze angles to calculate the magnitude of displacement as well as the onset/offset time of the saccades. Saccade movement onset was determined from the filtered displacement data when velocity and acceleration exceeded 30°/sec and 8,000°/sec^2, respectively. Saccade offset was determined when saccade velocity fell below a threshold value of 30°/sec for 28 msec (Heath et al., 2016; Leigh & Zee, 2015). A 5-point central finite differentiation algorithm was used to calculate velocity from the displacement data. Acceleration data was similarly obtained from the velocity data. Following the differentiation process, final plots were made that included gaze angle, velocity, peak velocity, saccade onset/offset, and saccade duration.

The raw head movement data was also imported to Microsoft Excel and examined offline. The raw data trials were sorted by target width and amplitude. The discrete trial events from E-prime were then plotted. The raw data was low-pass filtered using a dual-pass fourth-order Butterworth filter with a cut-off frequency set at 6 Hz. The raw data was filtered so that the process of numerical differentiation was less prone to cumulative error. A 5-point central finite differentiation algorithm was used to calculate velocity from the headmouse displacement data. The onset of the participant's head movement was determined from the filtered displacement data when the velocity of the head movement was above 5% peak velocity (Sjölander et al., 2008). Acceleration data was similarly differentiated from the velocity data.

The dependent variables analyzed for the eye movement and head movement Fitts' task included:

Behavioral Measures

- Movement time (MT) is the time [saccade (msec); head (sec)] between saccade or head movement onset and offset.

- Reaction time (RT) is the time interval [saccade (msec); head (sec)] between the presentation of the randomized target and the initiation of eye or head movement.
- Constant error (CE) is a measure of average bias (degrees), which is the average error of the participant's achieved eye or head movement amplitude from the actual target amplitude. CE is a measure of movement accuracy. A positive CE corresponds to overshooting the target while a negative CE corresponds to undershooting the target.
- Variable error (VE) is the standard deviation of target amplitude scores achieved by the participant based on their average score. VE is a measure of movement consistency.

Kinematic Measures

- Peak velocity (PV) is the peak of the saccade or head movement velocity amplitude [saccade (m/s); head (degrees/sec)] in the horizontal movement direction, and the time to peak velocity (ttPV) measured as the time [saccade (ms); head (sec)] from the onset of velocity to its peak amplitude.
- Peak acceleration (PA) is the peak of the saccade or head movement acceleration amplitude [saccade (m/s^2); head ($degrees/s^2$)] in the horizontal movement direction, and the time to peak acceleration (ttPA) measured as the time [saccade (ms); head (sec)] from the onset of acceleration to its peak amplitude.

Statistical Analysis

All descriptive statistics are expressed as mean ± SD. The data were analyzed using Jamovi. (*jamovi* [Computer Software]. (2019). Retrieved from https://www.jamovi.org). The differences in mean values for baseline data were calculated using independent student t-tests with a significance set at $p<0.05$.

All participants completed seven independent experimental conditions of amplitude and width to assess the effects of SM on eye and head movements. As an initial step, data generated from these conditions were collapsed into a single grand mean for each participant to conduct a between-group analysis of the dependent variables for the eye movement and head movement tasks respectively. Data from the amplitude and width conditions were collapsed into a single grand mean as this study is using Fitts' task as a means to examine the main effects of SM and Group on performance-based outcomes in symptomatic and asymptomatic participants. There

were baseline differences in pain (VAS) between the symptomatic and asymptomatic groups that allowed this analysis to be conducted.

The next step used the data from the seven experimental conditions to conduct a within-group analysis of the dependent variables for the eye movement and head movement tasks. We had collapsed the amplitude and width data into a grand mean during the between-group analysis. Therefore, the dependent variables were analyzed within each symptomatic and asymptomatic group to identify the main effects of the amplitude and width conditions. As such, the within-group analysis is able to assess the effects of SM under separate amplitude and width conditions on the performance-based outcome measures within the symptomatic and asymptomatic groups.

All between-group dependent variables were compared between the symptomatic and asymptomatic groups with a 2 (spinal manipulation: pre, post) x 2 (group: symptomatic, asymptomatic) repeated measures analysis of variance (ANOVA).

The within-group dependent variables were compared within each symptomatic and asymptomatic group with a repeated measures analysis of variance (ANOVA).

Eye:

- 2 (spinal manipulation: pre, post) x 2 (side of eye movement: left, right) x 4 (amplitude: 1.60, 3.20, 6.50, 12.90 cm).
- 2 (spinal manipulation: pre, post) x 2 (side of eye movement: left, right) x 4 (width: 0.5, 1.0, 2.0, 4.0 cm).

Head:

- 2 (spinal manipulation: pre, post) x 2 (side of head movement: left, right) x 4 (amp: 4.5, 8.3, 15.5, 29.0 cm).
- 2 (spinal manipulation: pre, post) x 2 (side of head movement: left, right) x 4 (width: 1.2, 2.4, 4.8, 9.6 cm).

The final step was to compare the effect of the amplitude and width conditions (Fitts' parameter) on the MT/ID relationship during the eye and head movement tasks. Specifically, this analysis compared the effect of achieving the same range of ID values by changing amplitude or by changing width. There was no main effect of SM included in this analysis. Movement time was compared within each symptomatic and asymptomatic group with a repeated measures

analysis of variance (ANOVA). The asterisk (*) denotes overlapping IDs for the amplitude and width conditions for the eye (5.69^{*}) and head (5.60^{*}) movement tasks.

Eye:

- 2 (Group: symptomatic, asymptomatic) x 2 (Fitts' Parameter: amplitude condition, width condition) x 3 (ID: 2.69, 3.69, 4.69 bits).

Head:

- 2 (Group: symptomatic, asymptomatic) x 2 (Fitts' Parameter: amplitude condition, width condition) x 3 (ID: 2.60, 3.60, 4.60, bits).

The overlapping IDs for the amplitude and width conditions, for both the eye and head movement tasks, were excluded from the ANOVA. Inclusion of these overlapping IDs would have required duplicating these values for the amplitude and width conditions, which would have reduced the variability in the data within the groups and would have been a violation of an ANOVA assumption.

Given the dependent linear relationship between amplitude or width to the index of difficulty, an analysis was completed to calculate the slope of the MT/ID function and correlation coefficient (R^2) of the MT during the amplitude and width conditions against the four IDs for the eye (ID: 2.69, 3.69, 4.69, 5.69^{*} bits) and head (ID: 2.60, 3.60, 4.60, 5.60^{*} bits) movement tasks. This analysis was used to determine if the saccade and head MT/ID slopes would be different for the amplitude condition compared to the width condition within symptomatic and asymptomatic groups.

Whenever a significant interaction was observed, pair-wise comparisons were made using Tukey's HSD post-hoc test. For all analyses, statistical significance was set as $p<0.05$. Estimate of effect sizes for ANOVA were reported based on partial eta-squared ($_p\eta^2$).

Results

Participant baseline demographics are outlined in Table 3. The differences in mean values for baseline data were calculated using independent student t-tests with a significance set at $p<0.05$.

Table 3. *Participants' characteristics*

Demographics	Asymptomatic	Symptomatic	Mean difference (95% CI)	p-value
N (males/females)	20 (6/14)	20 (3/17)		
Age (years; mean ± SD)	30.8 ± 5.57	32.5 ± 6.91	-1.45 (-5.47 - 2.57)	0.470
VAS (cm; mean ± SD)	0.570 ± 1.31	2.79 ± 1.53	-2.22 (-3.13 - -1.30)	< 0.001*
NDI (mean ± SD)	4.85 ± 4.98	11.1 ± 5.31	-6.25 (-9.54 - -2.96)	< 0.001*
Last SM visit before data collection (Days; mean ± SD)	54.8 ± 57.3	41.9 ± 56.3	12.95 (-23.41 – 49.31)	0.474
Pain duration (Months; mean ± SD)	15.5 ± 17.6	20.6 ± 19.7	5.05 (-17.00 – 6.90)	0.398

Visual Analog Scale (VAS)

Descriptive statistics are outlined in Table 4. SM did not alter reported pain levels in the experimental group experiencing neck pain. There was no significant difference in the pre/post VAS scores after spinal manipulation within the symptomatic group ($t_{(1,19)} = 0.442$; $p = 0.332$).

Table 4. Descriptive statistics for VAS in symptomatic group

	Pre-SM	Post-SM
N	20	20
Missing	0	0
Mean	2.77	2.63
Median	2.80	2.15
Standard deviation	1.53	1.77
Minimum	0.100	0.00
Maximum	5.70	6.30

Eye Movement Fitts' Task

Movement Time (MT)

Between-Group Analysis

Table 5. Descriptive statistics for saccade MT (msec)

	Group	Pre-SM	Post-SM
N	Asymp	20	20
	Symp	20	20
Missing	Asymp	0	0

Table 5. Descriptive statistics for saccade MT (msec)

	Group	Pre-SM	Post-SM
	Symp	0	0
Mean	Asymp	55.6	59.3
	Symp	57.6	56.4
Median	Asymp	54.4	57.3
	Symp	57.5	57.8
Standard deviation	Asymp	7.29	11.0
	Symp	7.00	5.47
Minimum	Asymp	43.7	44.9
	Symp	42.0	42.0
Maximum	Asymp	71.3	88.6
	Symp	77.0	64.3

Spinal manipulation did not alter saccade MT in either experimental group. There were no significant main effects of SM ($F_{(1,38)} = 0.939$; $p = 0.339$; $\eta^2_p = 0.024$) or Group ($F_{(1,38)} = 0.051$; $p = 0.823$; $\eta^2_p = 0.001$) on MT. There was no significant SM x Group interaction ($F_{(1,38)} = 3.374$; $p = 0.074$; $\eta^2_p = 0.082$).

Within-Group Analysis – Symptomatic

Within the symptomatic group, MT increased with larger target amplitudes. There was a significant main effect for the different amplitudes at the common target width of 0.50 cm on MT ($F_{(3,57)} = 254.23$; $p < 0.001$; $\eta^2_p = 0.930$)(Fig. 3; Error bars in all figures represent standard error (SE); different colour paired asterisks * denote pairwise differences). Tukey's HSD post-hoc testing noted that there was a significant increase in MT ($p < 0.001$) throughout all amplitude comparisons. There were no significant main effects for left and right saccades on MT ($F_{(1,19)} =$

0.259; p= 0.616) and SM on MT ($F_{(1,19)}$ = 1.14; p= 0.299). There were no significant amplitude x side, amplitude x SM, side x SM, and amplitude x side x SM interactions.

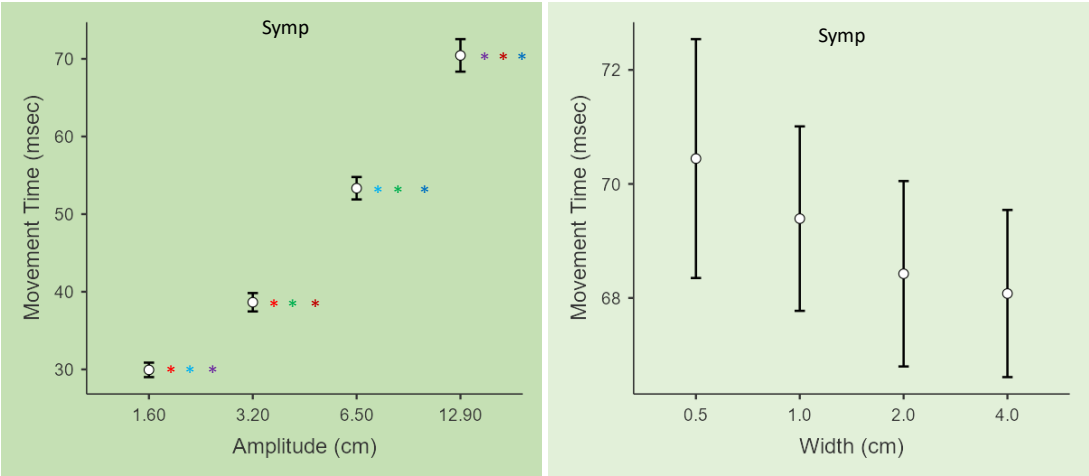


Figure 3. Main Effects of Amplitude & Width on Symptomatic Saccade Movement Time

Saccade MT did not change appreciably as target widths became larger. There was no significant main effect of target width on saccade MT (Fig. 3) in the symptomatic group ($F_{(3,57)}$ = 1.95; p = 0.133). There were no significant main effects for left and right side horizontal eye movements on MT ($F_{(1,19)}$ = 0.165; p= 0.689) and SM on MT ($F_{(1,19)}$ = 0.335; p= 0.570). There were no significant width x side, width x SM, side x SM, and width x side x SM interactions.

Within-Group Analysis – Asymptomatic

There was a significant main effect of amplitude on MT at the common target width of 0.50 cm ($F_{(3,57)}$ = 188.44; p < 0.001; η^2_p = 0.908)(Fig. 4). Pairwise comparisons resulted in a significant difference (p < 0.001) between all amplitude comparisons. Saccade MT increased as the target amplitudes became larger. There were no significant main effects of side and SM on MT. There were no significant amplitude x side, SM x side, SM x amplitude and amplitude x side x SM interactions.

Saccade MT decreased as the target widths increased from 0.50 to 1.0 cm. There was a significant main effect of width on MT at the common target amplitude of 12.90 cm ($F_{(3,57)}$ = 3.35; p = 0.025; η^2_p = 0.150)(Fig. 4). Post-hoc testing noted there was a significant difference (p < 0.001), but a very limited effect, between the 0.50 and 1.0 cm target width. There were no significant differences during pairwise comparisons of the remaining target widths. There were

no significant main effects of side or SM on MT. There were no significant side x SM, width x side, SM x width, and side x SM x width interactions.

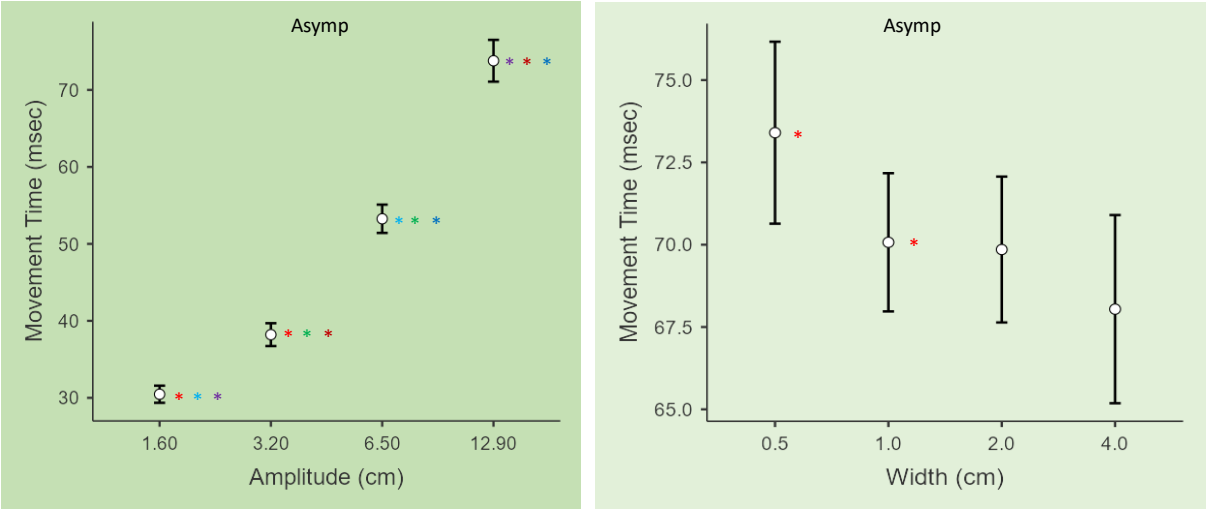


Figure 4. Main Effects of Amplitude & Width on Asymptomatic Saccade Movement Time

In summary, saccade MT was not altered following SM in either the symptomatic or asymptomatic groups. Movement amplitude affected saccade MT but there was only a minimal influence of target width on saccade MT. Furthermore, the main effects of SM and Group also had no effect on saccade MT within either experimental group.

Reaction Time (RT)

Between-Group Analysis

Table 6. Descriptive statistics for saccade RT (msec)

	Group	Pre-SM	Post-SM
N	Asymp	20	20
	Symp	20	20
Missing	Asymp	0	0

Table 6. Descriptive statistics for saccade RT (msec)

	Group	Pre-SM	Post-SM
	Symp	0	0
Mean	Asymp	179	179
	Symp	178	175
Median	Asymp	175	180
	Symp	172	169
Standard deviation	Asymp	22.9	22.1
	Symp	21.3	19.5
Minimum	Asymp	152	146
	Symp	148	136
Maximum	Asymp	224	226
	Symp	225	215

Saccade RT was not significantly changed following SM in either experimental group. There were no significant main effects of SM ($F_{(1,38)} = 0.976$; $p = 0.329$; $\eta^2_p = 0.025$) or Group ($F_{(1,38)} = 0.085$; $p = 0.772$; $\eta^2_p = 0.002$) on RT. There was no significant SM x Group interaction ($F_{(1,38)} = 0.659$; $p = 0.422$; $\eta^2_p = 0.017$).

Within-Group Analysis – Symptomatic

Within the symptomatic group saccade RT decreased as target amplitudes increased (Fig. 5). There was a significant main effect for the different amplitudes at the common target width of 0.50 cm on RT ($F_{(3,57)} = 20.91$; $p < 0.001$; $\eta^2_p = 0.524$). Tukey's HSD post-hoc comparisons noted that there was a significant decrease in RT when the 1.60 cm amplitude was compared to the 3.20 cm amplitude ($p = 0.001$), the 6.50 cm amplitude ($p < 0.001$), and the 12.90 cm amplitude ($p < 0.001$). There was also a significant decrease in RT when the 3.20 amplitude was compared to the 6.50 cm amplitude ($p = 0.001$). There were no significant differences in RT when the 3.20 and 12.90 cm ($p = 0.056$), and the 6.50 and 12.90 cm ($p = 1.000$) were compared. There were no significant main effects for left and right side eye movements on RT ($F_{(1,19)} = 0.856$; $p= 0.367$), and SM on RT ($F_{(1,19)} = 0.547$; $p= 0.468$). There were no significant amplitude x side, amplitude x SM, side x SM, and amplitude x side x SM interactions.

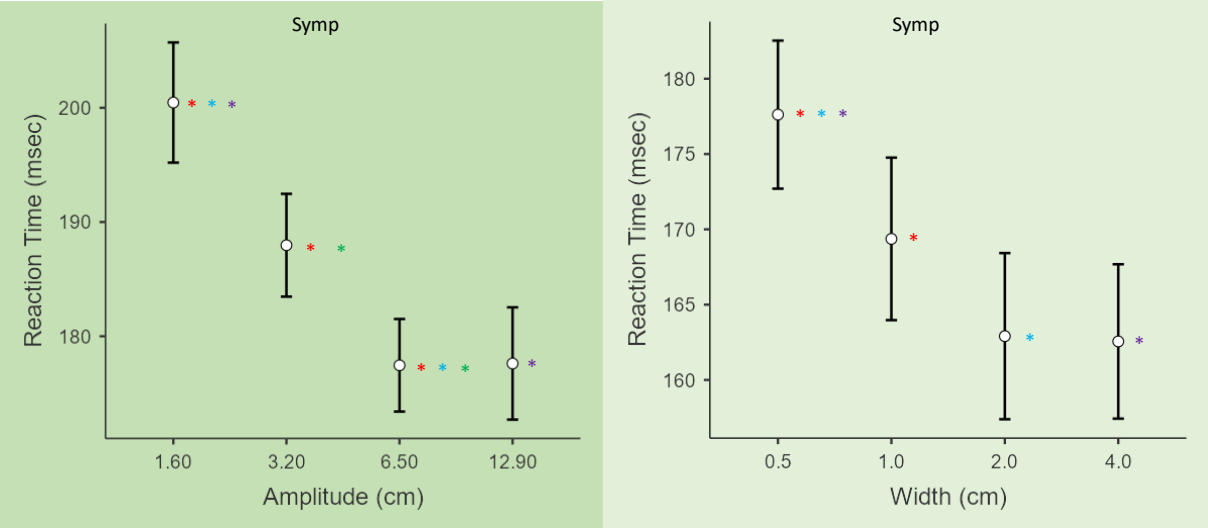


Figure 5. Main Effects of Amplitude & Width on Symptomatic Saccade Reaction Time.

Within the symptomatic group saccade RT decreased as target widths increased (Fig. 5). There was a significant main effect for the different target widths at the common amplitude of 12.90 cm on RT ($F_{(3,57)} = 13.18$; $p < 0.001$; $\eta^2_p = 0.410$). Pairwise comparisons noted that there was a significant decrease in RT when the 0.5 cm width was compared to the 1.0 cm width ($p = 0.011$), the 2.0 cm width ($p = 0.002$), and the 4.0 cm amplitude ($p < 0.001$). There were no significant post-hoc differences between the 1.0 cm width compared to the 2.0 and 4.0 cm widths, and between the 2.0 and 4.0 cm widths. There were no significant main effects for left and right side on RT ($F_{(1,19)} = 0.516$; $p= 0.481$) and post-SM on RT ($F_{(1,19)} = 3.89$; $p= 0.063$). There were no significant amplitude x side, amplitude x SM, side x SM, and amplitude x side x SM interactions.

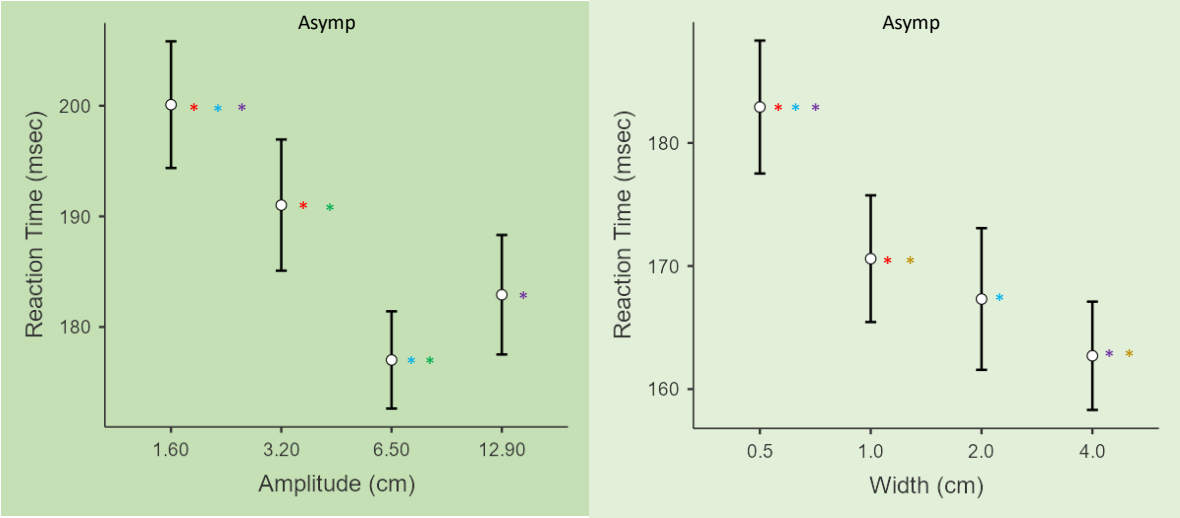


Figure 6. Main Effects of Amplitude & Width on Asymptomatic Saccade Reaction Time

Within-Group Analysis - Asymptomatic

Within the asymptomatic group saccade RT decreased as target amplitudes increased (Fig. 6). There was a significant main effect for the different amplitudes at the common target width of 0.50 cm on RT ($F_{(3,57)} = 13.62$; $p < 0.001$; $\eta^2_p = 0.418$). Tukey's HSD post-hoc testing noted that there was a significant decrease in RT when the 1.60 cm amplitude was compared to the 3.20 cm amplitude ($p = 0.027$), the 6.50 cm amplitude ($p < 0.001$), and the 12.90 cm amplitude ($p = 0.016$). There was also a significant decrease in RT when the 3.20 amplitude was compared to the 6.50 cm amplitude ($p < 0.001$). There were no significant differences in RT when the 3.20 and 12.90 cm ($p = 0.272$), and the 6.50 and 12.90 cm ($p = 0.337$) were compared. There were no significant main effects for side on RT ($F_{(1,19)} = 1.27$; $p= 0.063$) and SM on RT ($F_{(1,19)} = 0.09$; $p= 0.774$). Likewise, there were no significant amplitude x side, amplitude x SM, side x SM, and amplitude x side x SM interactions.

Target width influenced saccade RT such that RT decreased as target width increased (Fig. 6). There was a significant main effect for target width on RT at the common target amplitude of 12.90 cm ($F_{(3,57)} = 19.39$; $p < 0.001$; $\eta^2_p = 0.505$). More specifically, pairwise comparisons noted that there was a significant decrease in RT when the 0.5 cm target width was compared to the 1.0 ($p = 0.003$), 2.0 ($p = 0.001$), and 4.0 ($p < 0.001$) cm target widths, and when the 1.0 cm target width was compared to the 4.0 ($p = 0.017$) target width. RT decreased as the target widths became larger in the asymptomatic group with no further effect related to SM or

the side of eye movement. There was no significant change in RT when the 1.0 cm target width was compared to the 2.0 cm target width ($p = 0.485$) and when the 2.0 cm target width was compared to the 4.0 cm target width ($p = 0.324$). There were no significant main effects for side ($F_{(1,19)} = 0.021$; $p= 0.886$) and SM ($F_{(1,19)} = 0.273$; $p= 0.607$) on RT. There were no significant width x side, width x SM, side x SM, and width x side x SM interactions.

In summary, SM did not later saccade RT in either experimental group. Saccade RT was reduced with larger movement amplitudes and target widths in both the symptomatic and asymptomatic groups.

Constant Error (CE)

Between-Group Analysis

Table 7. Descriptive statistics for saccade CE (degrees).

	Group	**Pre-SM**	**Post-SM**
N	Asymp	20	20
	Symp	20	20
Missing	Asymp	0	0
	Symp	0	0
Mean	Asymp	-0.572	-0.533
	Symp	-0.497	-0.420
Median	Asymp	-0.422	-0.508
	Symp	-0.557	-0.519
Standard deviation	Asymp	0.475	0.409
	Symp	0.437	0.529
Minimum	Asymp	-1.91	-1.58
	Symp	-1.43	-1.10
Maximum	Asymp	0.0698	0.177
	Symp	0.137	1.01

The eye movement bias to overshoot or undershoot the target was not influenced by SM for the experimental groups. There were no significant main effects of SM ($F_{(1,38)} = 0.176$; $p = 0.677$; $\eta^2_p = 0.005$) or Group ($F_{(1,38)} = 0.008$; $p = 0.927$; $\eta^2_p = 0.000$) on CE. There was no significant SM x Group interaction ($F_{(1,38)} = 0.077$; $p = 0.783$; $\eta^2_p = 0.002$).

Within-Group Analysis – Symptomatic

There was a bias for the symptomatic group to undershoot the target as movement amplitudes became larger. There was a significant main effect of amplitude on CE at the common target width of 0.5 cm ($F_{(3,57)} = 21.37$; $p < 0.001$; $\eta^2_p = 0.529$) (Fig. 7). Pairwise comparisons noted a significant decrease in CE between the 1.6 cm and the 3.20 cm ($p = 0.012$), 6.50 cm ($p = 0.002$), and 12.90 cm ($p < 0.001$) movement amplitudes. Significant differences were also seen between the 3.20 cm and the 6.50 cm ($p = 0.005$), the 3.20 cm and the 12.90 cm ($p < 0.001$), and the 6.50 cm and 12.90 cm ($p = 0.006$) movement amplitudes. There were no significant main effects of side ($F_{(1,19)} = 0.025$; $p = 0.876$) or SM ($F_{(1,19)} = 0.632$; $p = 0.436$) on saccade CE. There were no significant amplitude x side, amplitude x SM, side x SM, and amplitude x side x SM interactions.

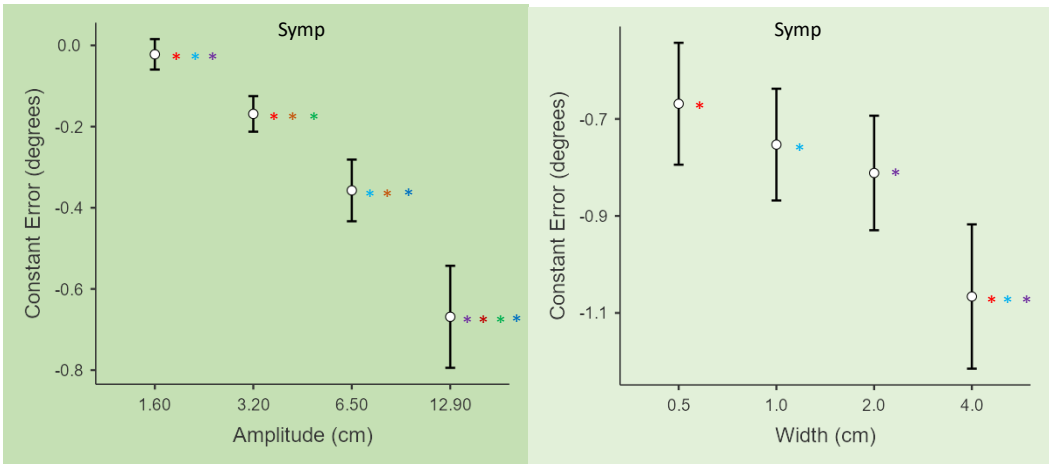


Figure 7. Main Effects of Amplitude & Width on Symptomatic Saccade Constant Error

There was a bias for the symptomatic group to undershoot the target as target widths became larger. There was a significant main effect of width on CE at the common target amplitude of 12.90 cm ($F_{(3,57)} = 12.58$; $p < 0.001$; $\eta^2_p = 0.398$) (Fig. 7). Pairwise comparisons noted significant differences between the 0.5 cm and 4.0 cm ($p = 0.001$) and between the 1.0 cm and the 4.0 cm ($p = 0.012$) and between the 2.0 cm and 4.0 cm ($p = 0.004$) target widths. There

were no significant differences between the 0.5 cm and 1.0 cm and 2.0 cm target widths and between the 1.0 and 2.0 cm target widths. There were no significant main effects for side ($F_{(1,19)}$ = 0.268; p = 0.611) or SM ($F_{(1,19)}$ = 0.410; p = 0.530) on CE. There were no significant width x side, width x SM, side x SM, and width x side x SM interactions.

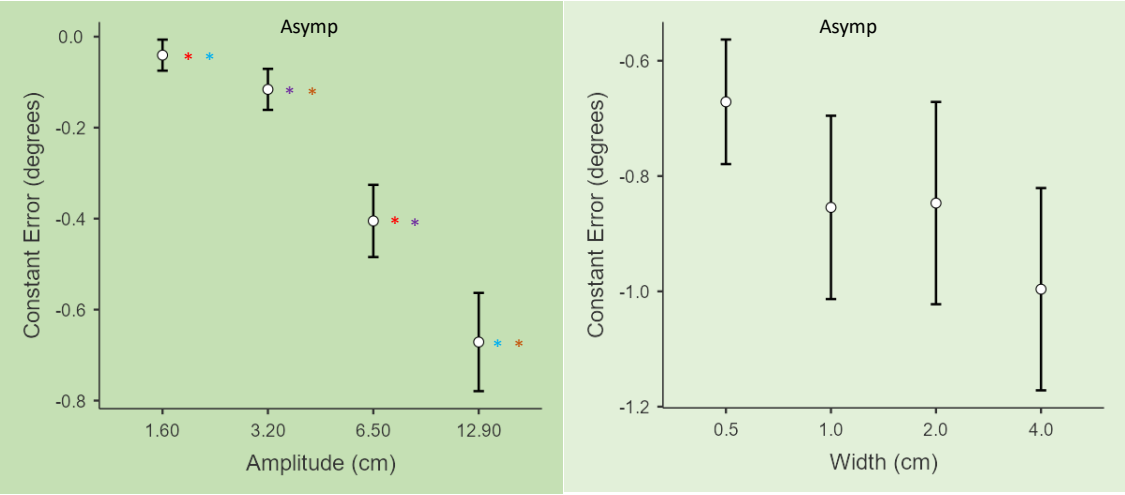


Figure 8. Main Effects of Amplitude & Width on Asymptomatic Saccade Constant Error

Within-Group Analysis – Asymptomatic

There was a bias for the asymptomatic group to undershoot the target as movement amplitudes became larger. There was a significant main effect of amplitude on CE ($F_{(3,57)}$ = 24.41; p < 0.001; η^2_p = 0.562) (Fig. 8) at the common target width of 0.5 cm. Pairwise comparisons noted a significant difference between the 1.60 cm compared to the 6.50 cm (p = 0.001) and 12.90 cm (p < 0.001) amplitudes. There were also significant differences between the 3.20 cm compared to the 6.50 cm (p < 0.001) and 12.90 cm (p < 0.001) target amplitudes. There were no differences between the 1.60 cm and 3.20 cm, and between the 6.50 cm and 12.90 cm amplitudes. There were no significant main effects of side and SM on CE. There were no significant amplitude x side, SM x side, SM x amplitude and amplitude x side x SM interactions.

There was a bias for the asymptomatic group to undershoot the target as target widths became larger. There was a significant main effect of width on CE ($F_{(3,57)}$ = 3.01; p < 0.038; η^2_p = 0.138) (Fig. 8) at the common target amplitude of 12.90 cm. However, there were no significant differences between any of the width comparisons on post hoc testing. There were no significant

main effects of SM and side on CE. There were no significant side x SM, width x side, SM x width, and side x SM x width interactions.

In summary, SM had no influence on undershooting or overshooting the target for the symptomatic or asymptomatic groups. There was an increase in CE with larger movement amplitudes and some indication of an increased CE with larger target widths. This increase in saccade CE reflects a bias towards undershooting the target.

Variable Error (VE)

Between-Group Analysis

Table 8. Descriptive statistics for saccade VE (degrees).

	Group	Pre-SM	Post-SM
N	Asymp	20	20
	Symp	20	20
Missing	Asymp	0	0
	Symp	0	0
Mean	Asymp	9.53	9.60
	Symp	9.59	9.53
Median	Asymp	9.70	9.66
	Symp	9.56	9.52
Standard deviation	Asymp	0.511	0.450
	Symp	0.447	0.443
Minimum	Asymp	8.07	8.44
	Symp	8.68	8.91
Maximum	Asymp	10.2	10.2
	Symp	10.3	10.7

Saccade movement consistency was not influenced by SM in either experimental group. There were no significant main effects of SM ($F_{(1,38)} = 0.018$; $p = 0.895$; $\eta^2_p = 0.000$) or Group ($F_{(1,38)} = 1.684$; $p = 0.202$; $\eta^2_p = 0.042$) on VE. There was no significant SM x Group interaction ($F_{(1,38)} = 0.077$; $p = 0.783$; $\eta^2_p = 0.002$).

Within-Group Analysis – Symptomatic

Within the symptomatic group saccade VE increased as target amplitudes increased (Fig. 9). There was a significant main effect for the different amplitudes at the common target width of 0.50 cm on VE ($F_{(3,57)} = 23.63$; $p < 0.001$; $\eta^2_p = 0.554$). The symptomatic groups' saccades became less consistent as target amplitudes became larger. Tukey's HSD post-hoc testing noted that there was a significant increase in VE ($p < 0.001$) for comparisons between the 1.60 and 12.90 cm amplitudes, the 3.20 and 12.90 cm amplitudes, and the 6.50 and 12.90 cm amplitudes. There was also a significant increase in VE with the comparison between the 3.20 and 6.50 cm amplitudes ($p = 0.025$). The remaining amplitude comparisons were not significant. There were no significant main effects for left and right sided horizontal eye movements on VE ($F_{(1,19)} = 0.067$; $p= 0.798$) and post-SM on VE ($F_{(1,19)} = 0.016$; $p= 0.902$). There were no significant amplitude x side, amplitude x SM, side x SM, and amplitude x side x SM interactions.

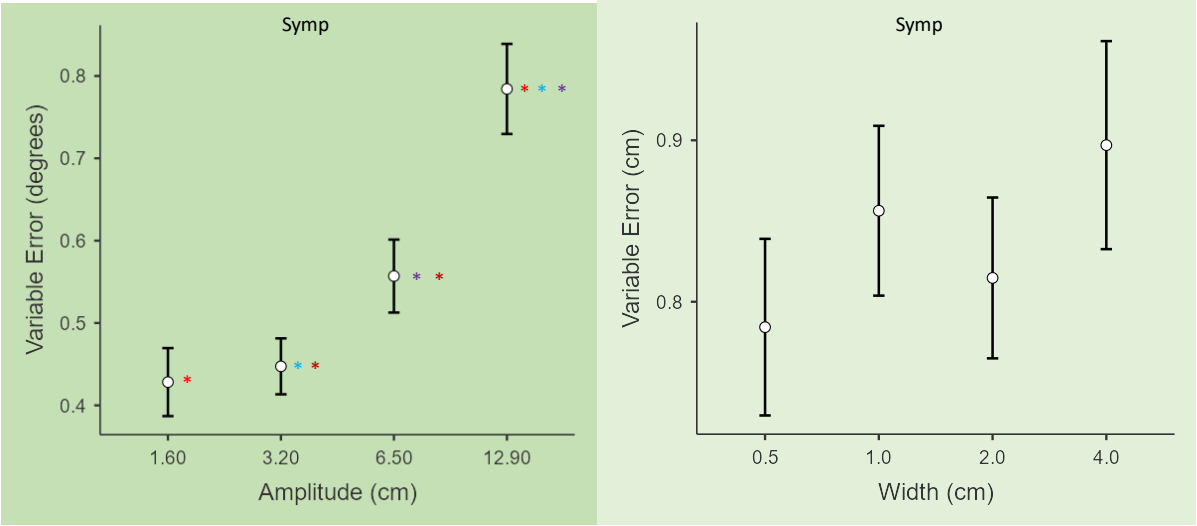


Figure 9. Main Effects of Amplitude & Width on Symptomatic Saccade Variable Error

Target width had no influence on saccade consistency within the symptomatic group. There were no significant main effects for the different target widths at the common amplitude of 12.90 cm on saccade VE ($F_{(3,57)} = 1.44$; $p = 0.241$) (Fig. 9). There were no significant main effects for left and right sided horizontal eye movements on VE ($F_{(1,19)} = 0.184$; $p= 0.673$), and post-SM on VE ($F_{(1,19)} = 0.137$; $p= 0.716$). There were no significant amplitude x side, amplitude x SM, side x SM, and amplitude x side x SM interactions.

Within-Group Analysis – Asymptomatic

Within the asymptomatic group saccade VE increased as target amplitudes increased. There was a significant main effect of target amplitude on VE at the common target width of 0.50 cm ($F_{(3,57)} = 23.97$; $p < 0.001$; $\eta^2_p = 0.558$)(Fig. 10). During pairwise comparisons there were significant differences for VE between the 1.60 and 6.50 cm ($p = 0.014$), and 1.60 and 12.90 cm ($p < 0.001$) amplitudes. There were also significant differences between the 3.20 and 12.90 cm ($p < 0.001$), and 6.50 and 12.90 cm ($p = 0.001$) amplitudes. Variable error increased as the target amplitudes became larger indicating the asymptomatic participants' saccades became less consistent in their movements across the larger target amplitudes. There were no significant main effects of SM and side on CE. There were also no significant SM x side, SM x amplitude, side x amplitude, and SM x side x amplitude interactions.

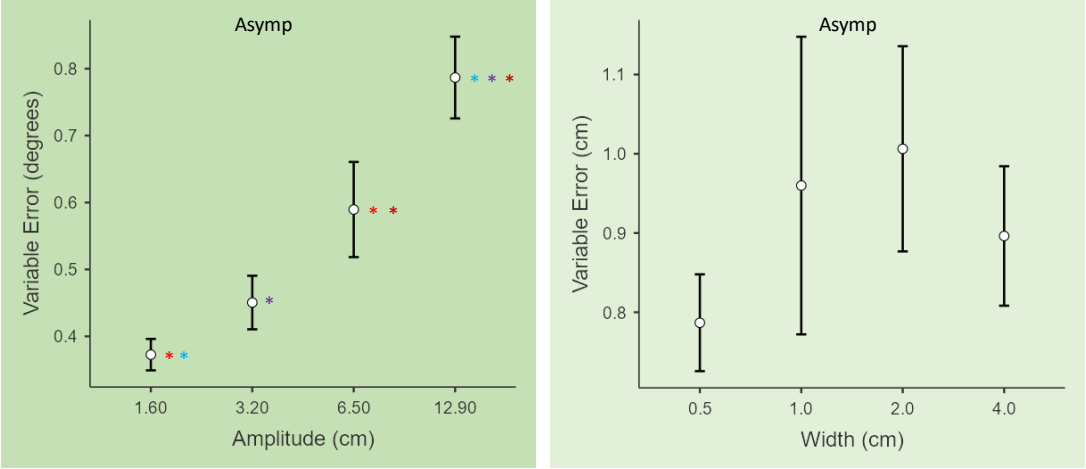


Figure 10. Main Effects of Amplitude & Width on Asymptomatic Saccade Variable Error

Saccade movement consistency was not influenced by target width, SM, or the direction of eye movement. There were no significant main effects of width ($F_{(3,57)} = 1.39$; $p = 0.256$) (Fig. 10), side ($F_{(1,519)} = 0.97$; $p = 0.337$), or SM ($F_{(1,19)} = 0.07$; $p = 0.796$) on VE at the common target amplitude of 12.90 cm. There were no significant SM x side, SM x width, side x SM, and SM x side x width interactions.

In summary, saccade movement consistency was not altered by SM in either experimental group. There was an increased VE with larger movement amplitudes but no main effect of target width on VE. The increase in VE during the amplitude condition corresponds to less consistent eye movement.

Peak Velocity (PV)

Between-Group Analysis

Table 9. Descriptive statistics for saccade PV (m/s).

	Group	Pre-SM	Post-SM
N	Asymp	20	20
	Symp	20	20
Missing	Asymp	0	0
	Symp	0	0
Mean	Asymp	390	369
	Symp	362	364
Median	Asymp	373	375
	Symp	361	353
Standard deviation	Asymp	114	78.7
	Symp	81.2	82.2
Minimum	Asymp	192	179
	Symp	224	222
Maximum	Asymp	779	489
	Symp	530	536

Saccade PV was not altered following SM in the symptomatic or asymptomatic groups. There were no significant main effects of SM ($F_{(1,38)} = 0.925$; $p = 0.342$; $\eta^2_p = 0.024$) or Group ($F_{(1,38)} = 0.406$; $p = 0.528$; $\eta^2_p = 0.011$) on PV. There was no significant SM x Group interaction ($F_{(1,38)} = 1.306$; $p = 0.260$; $\eta^2_p = 0.033$).

Within-Group Analysis – Symptomatic

Within the symptomatic group, saccade PV increased as target amplitudes became larger (Fig. 11). There was a significant main effect for the different amplitudes at the common target width of 0.50 cm on PV ($F_{(3,57)} = 138.74$; $p < 0.001$; $\eta^2_p = 0.880$). Pairwise comparisons demonstrated that PV was significantly different ($p < 0.001$) at all amplitudes. There were no significant main effects for side or SM on PV.

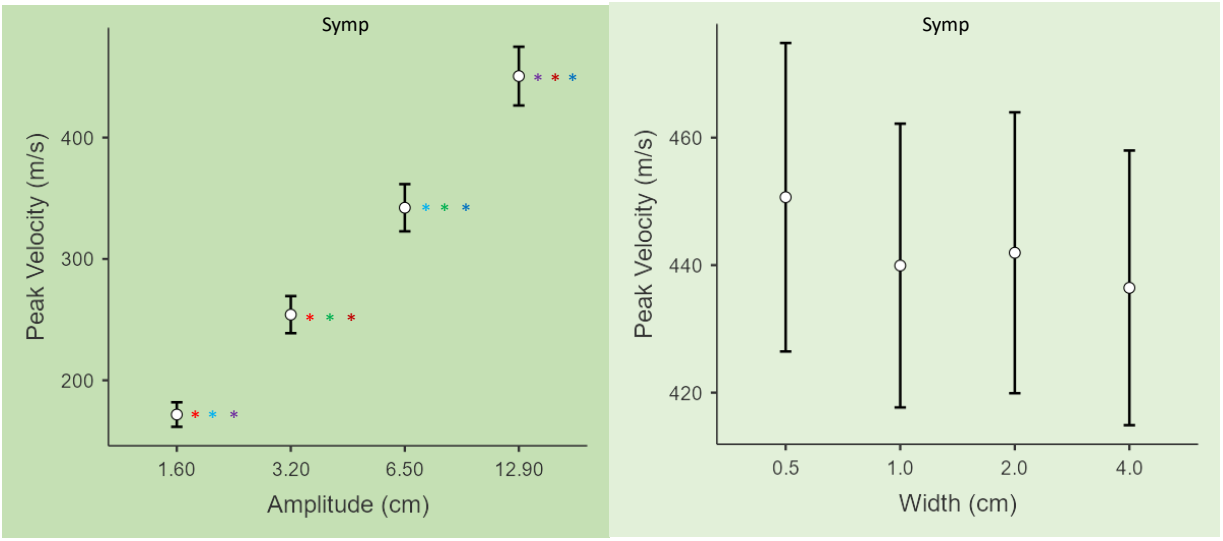


Figure 11. Main Effects of Amplitude & Width on Symptomatic Saccade Peak Velocity

There was a significant amplitude x side interaction ($F_{3,57)} = 2.85$; $p = 0.046$; $\eta^2_p = 0.130$) (Fig. 12) on PV. There was an increase in PV between all target amplitudes for leftward and rightward saccades. Differences in PV also existed in pairwise comparisons between left and right saccades except when the same saccade movement amplitudes were compared to one another. There were no significant main effects of width (Fig. 11), side, or SM on PV. There were also no significant side x SM, side x amplitude, SM x amplitude, or side x SM x amplitude interactions.

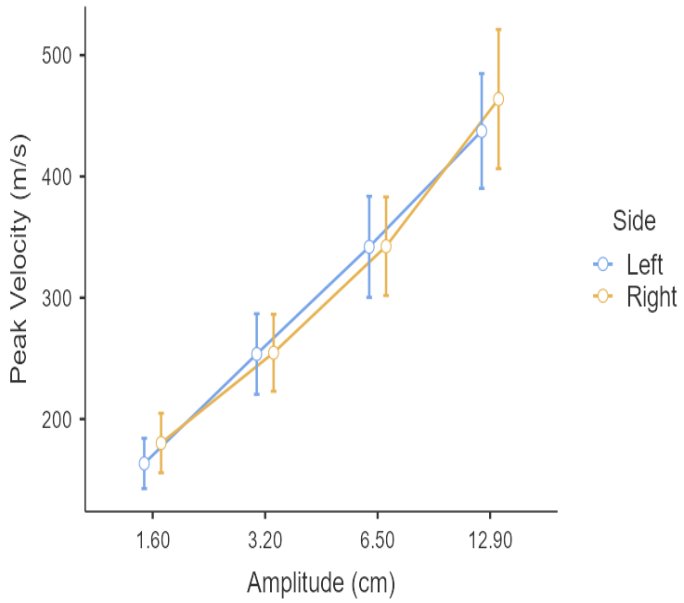


Figure 12. Amplitude x Side Interaction on Symptomatic Saccade Peak Velocity

Within-Group Analysis – Asymptomatic

Saccade PV increased as the target amplitudes became larger. There was a significant main effect of amplitude on PV at the common target width of 0.50 cm ($F_{(3,57)} = 51.01$; $p < 0.001$; $\eta^2_p = 0.729$)(Fig. 13). Post-hoc testing noted there was a significant difference ($p < 0.001$) between all amplitude comparisons except for between the 3.20 and 6.50 cm amplitudes. There were no significant main effects of side or SM on PV. There were no significant SM x side, SM x amplitude, side x SM, and SM x side x amplitude interactions.

There were no significant main effects of width (Fig. 13), side, or SM on saccade PV at the common target amplitude of 12.90 cm. There were no significant SM x side, SM x width, side x SM, and SM x side x width interactions.

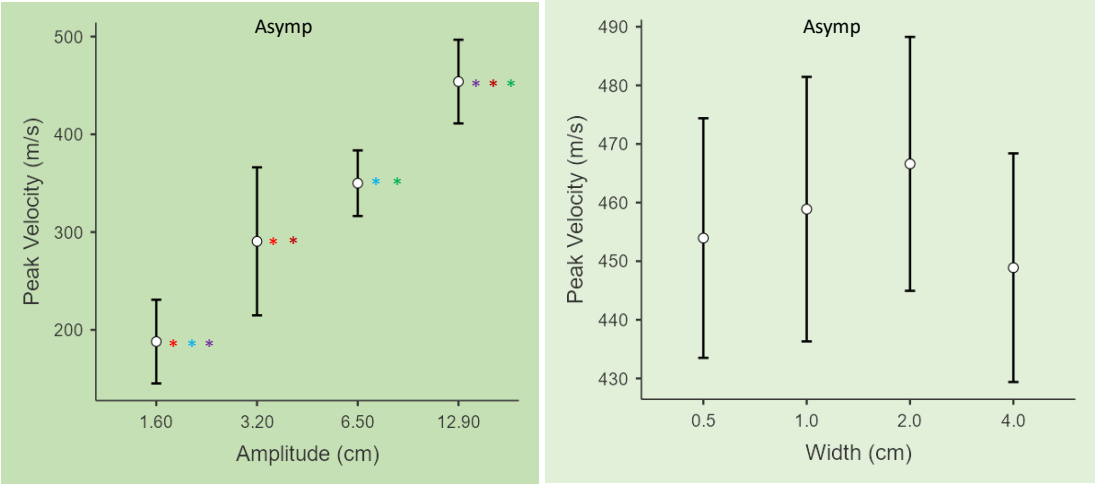


Figure 13. Main Effects of Amplitude & Width on Asymptomatic Saccade Peak Velocity

In summary, there was no alteration in PV following SM for either experimental group. There was an increase in PV with larger saccade movement amplitudes but there was no influence of target width on PV.

Time to Peak Velocity (ttPV)

Between-Group Analysis

Table 10. Descriptive statistics for saccade ttPV (msec).

	Group	Pre-SM	Post-SM
N	Asymp	20	20
	Symp	20	20
Missing	Asymp	0	0
	Symp	0	0
Mean	Asymp	18.8	20.5
	Symp	17.5	16.2
Median	Asymp	17.0	16.9
	Symp	15.9	15.0
Standard deviation	Asymp	7.28	7.90
	Symp	5.18	3.85
Minimum	Asymp	13.2	12.8
	Symp	12.5	13.0
Maximum	Asymp	44.5	42.0
	Symp	29.5	28.4

There was a significant SM x Group interaction ($F_{(1,38)} = 7.66$; $p = 0.009$; $\eta^2_p = 0.168$)(Fig. 14) on ttPV indicating a reduction in ttPV for the symptomatic group following SM and an increase in ttPV for the asymptomatic group following SM. However, there were no significant pairwise comparisons on post-hoc testing. There were no significant main effects of SM ($F_{(1,38)} = 0.107$; $p = 0.745$; $\eta^2_p = 0.003$) or Group ($F_{(1,38)} = 2.07$; $p = 0.158$; $\eta^2_p = 0.052$) on ttPV.

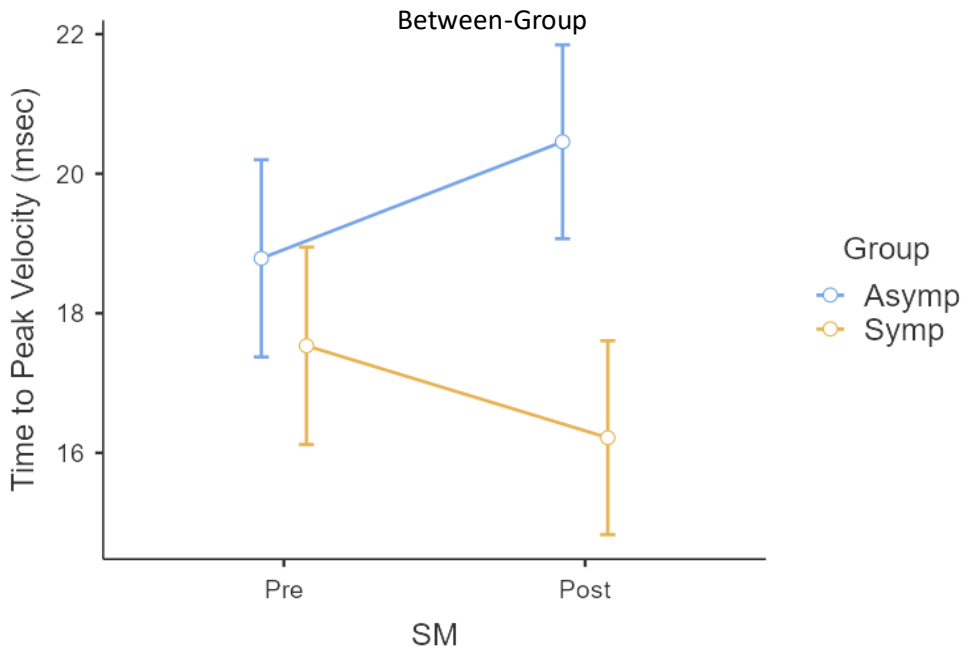


Figure 14. SM x Group Interaction on Saccade Time to Peak Velocity

Within-Group Analysis – Symptomatic

For the symptomatic group, ttPV occurred later in the saccade as movement amplitude increased and was in keeping with the longer MT for the larger movement amplitude. There was a significant main effect for the different amplitudes at the common target width of 0.50 cm on ttPV ($F_{(3,57)}$ =6.47; $p < 0.001$; $\eta^2_p = 0.254$)(Fig. 15). Pairwise comparisons demonstrated that ttPV was significantly different between the 1.60 and 6.50 cm amplitudes ($p = 0.026$), and the 1.60 and 12.90 cm amplitudes ($p = 0.005$). There were no differences between the 1.60 and 3.20 cm amplitudes, the 3.20 and 6.50 cm amplitudes when compared to the 12.90 cm amplitude. There were no significant main effects for side or SM on ttPV. There were no significant side x amplitude, side x SM, amplitude x SM, and side x amplitude x SM interactions.

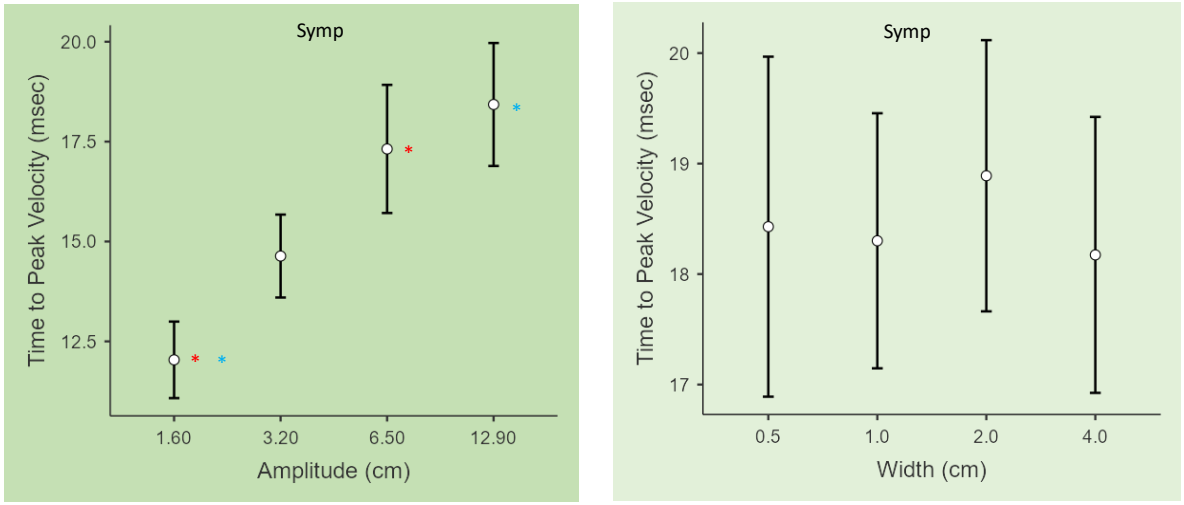


Figure 15. Main Effects of Amplitude & Width on Symptomatic Saccade Time to Peak Velocity.

Target width had no effect on altering saccade ttPV for the group experiencing neck pain. There was a no significant main effect for the different widths at the common target amplitude of 12.90 cm on ttPV ($F_{(3,57)}$ =0.233; p = 0.873; η^2_p = 0.012)(Fig. 15). There were no significant main effects for side or SM on ttPV. There were no side x SM, side x width, SM x width, or side x SM x width interactions.

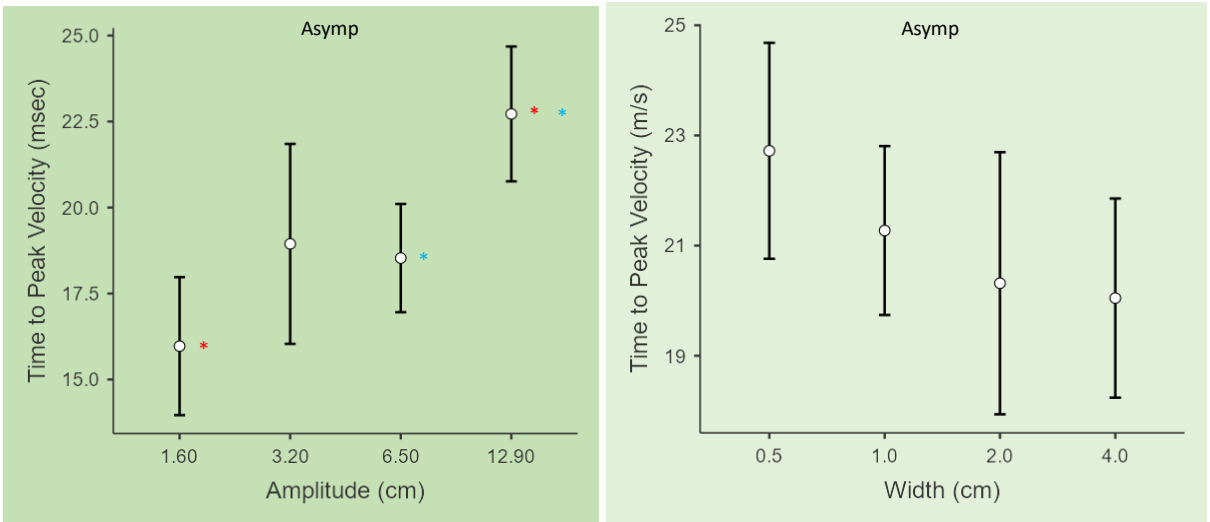


Figure 16. Main Effects of Amplitude & Width on Asymptomatic Saccade Time to Peak Velocity

Within-Group Analysis – Asymptomatic

For the asymptomatic group, ttPV occurred later in the saccade as movement amplitude increased and was in keeping with the longer MT for the larger movement amplitude. There was a significant main effect of amplitude on ttPV at the common target width of 0.50 cm ($F_{(3,57)}$ = 4.12; p = 0.010; η^2_p = 0.178)(Fig. 16). Pairwise comparisons noted there was a significant difference between the 1.60 and 12.90 cm amplitudes (p = 0.007), and the 6.50 and 12.90 cm amplitudes (p =0.006). The remaining amplitude comparisons were not significant. Generally, saccade ttPV increased as the target amplitudes became larger. There were no significant main effects of side or SM on PV. There were no significant SM x side, SM x amplitude, side x SM, and SM x side x amplitude interactions.

There was a significant decrease in ttPV following SM indicating it took longer to reach peak velocity for the width condition. There was no significant main effect of width on ttPV at the common target amplitude of 12.90 cm ($F_{(3,57)}$ =22.82; p < 0.001; η^2_p = 0.219)(Fig. 17). There was no significant main effect of side on ttPV. There was a significant main effect of SM on ttPV during the width condition ($F_{(1,19)}$ =5.33; p = 0.032; η^2_p = 0.546)(Fig. 16). There were no significant side x SM, width x side, SM x width, and side x SM x width interactions.

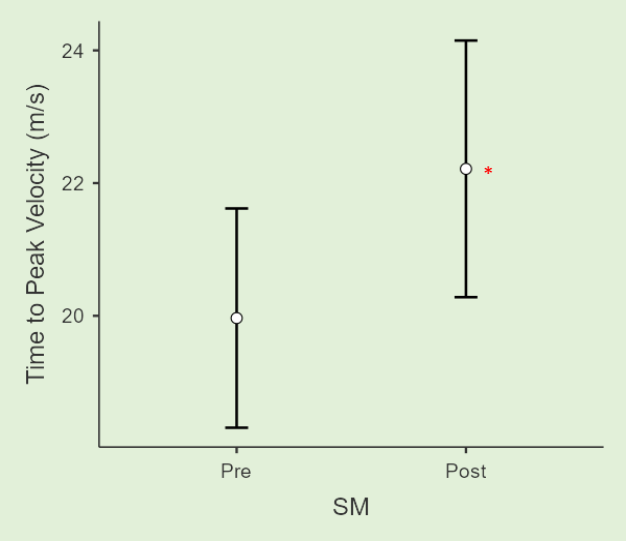


Figure 17. Main Effect of SM on Time to Peak Velocity (Width Condition)

In summary, SM did not alter ttPV for the amplitude condition in either the symptomatic or asymptomatic groups. There was a significant SM x Group interaction on ttPV indicating there was a significant reduction in saccade ttPV following SM within the symptomatic group.

There were main effects for movement amplitude on ttPV; however, ttPV showed no width effects in either group. There was a significant main effect of SM on ttPV during the width condition within the asymptomatic group.

Peak Acceleration (PA)

Between-Group Analysis

Table 11. Descriptive statistics for saccade PA (m/s^2).

	Group	Pre-SM	Post-SM
N	Asymp	20	20
	Symp	20	20
Missing	Asymp	0	0
	Symp	0	0
Mean	Asymp	42312	39791
	Symp	37168	37566
Median	Asymp	41414	39916
	Symp	38162	37305
Standard deviation	Asymp	22339	13202
	Symp	10894	10876
Minimum	Asymp	15655	13811
	Symp	12701	12396
Maximum	Asymp	128085	68768
	Symp	58671	59041

Saccade PA did not change following SM in either experimental group. There were no significant main effects of SM ($F_{(1,38)} = 0.296$; $p = 0.589$; $\eta^2_p = 0.008$) or Group ($F_{(1,38)} = 0.716$; $p = 0.403$; $\eta^2_p = 0.018$) on PA. There was no significant SM x Group interaction ($F_{(1,38)} = 0.560$; $p = 0.459$; $\eta^2_p = 0.015$).

Within-Group Analysis – Symptomatic

There was a significant main effect for the different amplitudes at the common target width of 0.50 cm on PA ($F_{(3,57)} = 61.63$; $p < 0.001$; $\eta^2_p = 0.764$), indicating that within the symptomatic group saccade PA increased as target amplitudes increased (Fig. 18). Pairwise

comparisons demonstrated that PA was significantly different ($p < 0.001$) at all amplitudes. There were no significant main effects for side or SM on saccade PV. There were no significant amplitude x side, amplitude x SM, side x SM, or amplitude x side x SM interactions on PA.

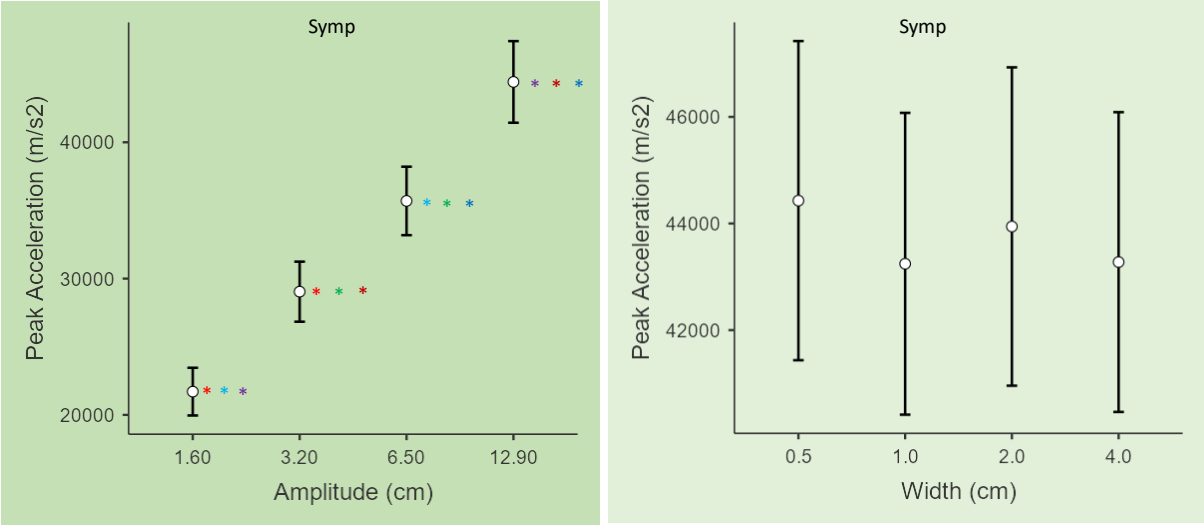


Figure 18. Main Effects of Amplitude & Width on Symptomatic Saccade Peak Acceleration

There were no significant main effects of width (Fig. 18), side, or SM on saccade PA. There were also no significant side x SM, side x width, SM x width, or side x SM x width interactions.

Within-Group Analysis – Asymptomatic

For the asymptomatic group, saccade PA increased as the target amplitudes became larger. There was a significant main effect of amplitude on PA at the common target width of 0.50 cm ($F_{(3,57)} = 8.59$; $p < 0.001$; $\eta^2_p = 0.311$)(Fig. 19). Post-hoc testing noted there was a significant difference between the 1.60 cm and the 3.20 ($p = 0.013$), 6.50 ($p = 0.043$), and 12.90 cm ($p < 0.001$) target amplitudes respectively. There was also a significant difference between the 6.50 and 12.90 cm ($p = 0.002$) amplitudes. There was no significant difference between the 3.20 cm amplitude when compared to the 6.50 and 12.90 cm amplitudes. There were no significant main effects of side or SM on PA. There were no significant SM x side, SM x amplitude, side x amplitude, and SM x side x amplitude interactions.

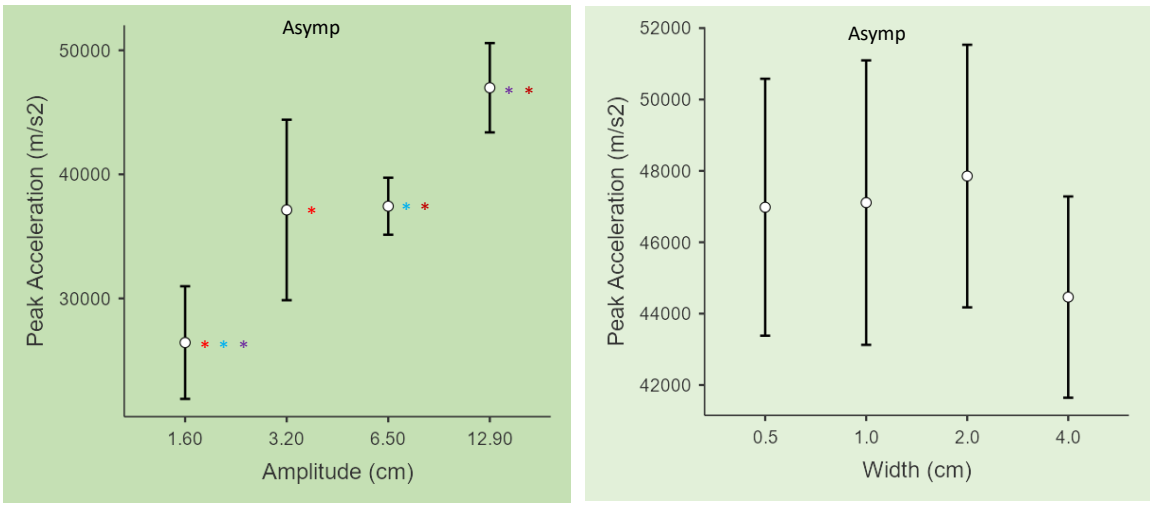


Figure 19. Main Effects of Amplitude & Width on Asymptomatic Saccade Peak Acceleration

Saccade PA was not altered by movement amplitude or the horizontal direction of eye movement. SM had no influence on saccade PA. There were no significant main effects of width (Fig. 19), side, or SM on PA at the common target amplitude of 12.90 cm. There were no significant SM x side, SM x width, side x SM, and SM x side x width interactions on PA.

In summary, saccade PA was not altered following SM in either experimental group. There was a significant influence of movement amplitude on PA, but there was no effect of target width on PA.

Time to Peak Acceleration (ttPA)

Between-Group Analysis

Table 12. Descriptive statistics for saccade ttPA (msec).

	Group	Pre-SM	Post-SM
N	Asymp	20	20
	Symp	20	20
Missing	Asymp	0	0
	Symp	0	0
Mean	Asymp	10.2	11.0
	Symp	9.27	8.23
Median	Asymp	8.78	8.27
	Symp	7.97	7.06
Standard deviation	Asymp	6.16	5.67
	Symp	3.72	3.07
Minimum	Asymp	5.57	6.09
	Symp	4.36	5.16
Maximum	Asymp	33.2	24.0
	Symp	19.0	15.2

Saccade ttPA did not change following SM in either experimental group. There were no significant main effects of SM ($F_{(1,38)} = 0.057$; $p = 0.813$; $\eta^2_p = 0.001$) or Group ($F_{(1,38)} = 1.74$; $p = 0.196$; $\eta^2_p = 0.044$) on ttPA. There was no significant SM x Group interaction ($F_{(1,38)} = 2.013$; $p = 0.164$; $\eta^2_p = 0.050$).

Within-Group Analysis – Symptomatic

Within the symptomatic group, the ttPA was not influenced by target width or the side of horizontal eye movement. Further, ttPA was not altered following SM. There was no significant main effect for the different amplitudes at the common target width of 0.50 cm on ttPA ($F_{(3,57)} = 1.16$; $p = 0.334$; $\eta^2_p = 0.057$)(Fig. 20). There were no significant main effects of side or SM on ttPA. There were no significant side x amplitude, side x SM, amplitude x SM, or side x amplitude x SM interactions on ttPA.

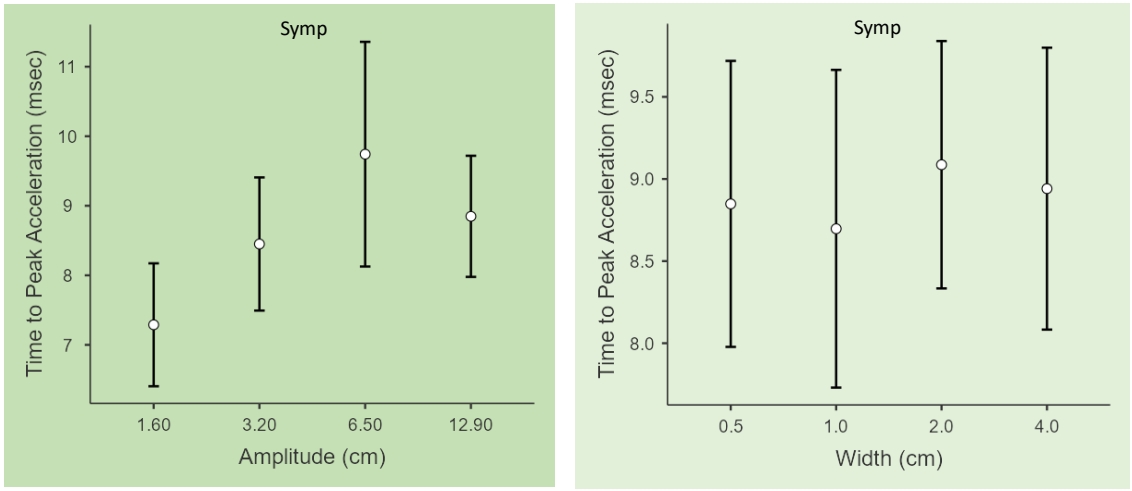


Figure 20. Main Effects of Amplitude & Width on Symptomatic Saccade Time to Peak Acceleration

There was no significant main effect for the different widths at the common target amplitude of 12.90 cm on saccade ttPA ($F_{(3,57)}$ =0.070; p = 0.976; η^2_p = 0.004)(Fig. 20). There were no significant main effects for side or SM on ttPA. There were no side x SM, side x width, SM x width, or side x SM x width interactions.

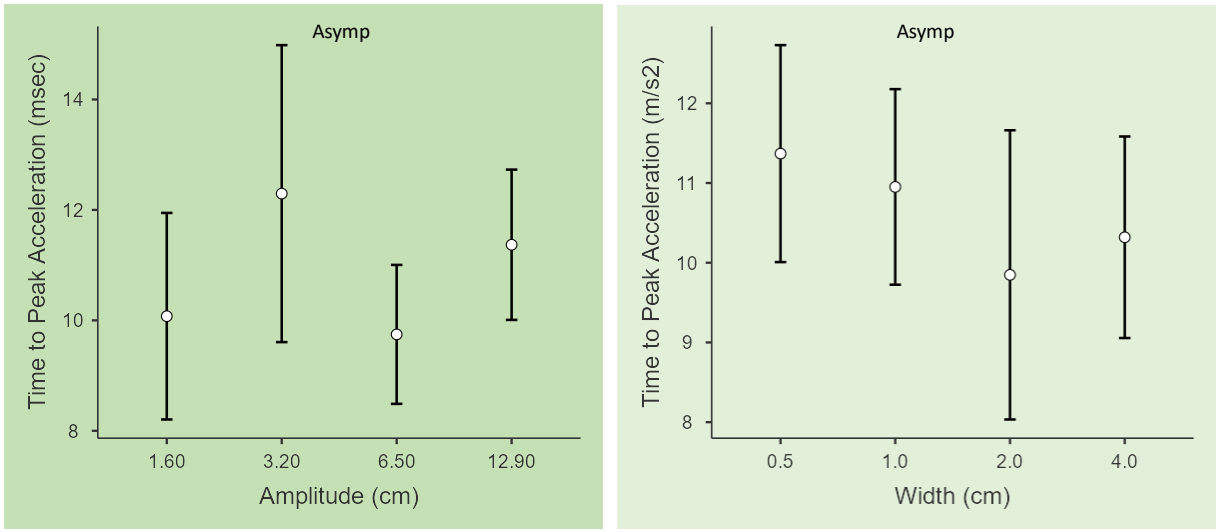


Figure 21. Main Effects of Amplitude & Width on Asymptomatic Saccade Time to Peak Acceleration

Within-Group Analysis – Asymptomatic

Saccade ttPA was not altered by amplitude, SM, or the side of horizontal eye movement. There was no significant main effect of amplitude on ttPA at the common target width of 0.50 cm ($F_{(3,57)} = 0.709$; $p = 0.551$; $\eta^2_p = 0.036$) (Fig. 21). There were no significant main effects of side or SM on ttPA. There were no significant side x amplitude, side x SM, amplitude x SM, and SM x side x amplitude interactions.

There was no significant main effect of width on ttPA at the common target amplitude of 12.90 cm ($F_{(3,57)} = 0478$; $p = 0.699$; $\eta^2_p = 0.025$)(Fig. 21). There were no significant main effects of side or SM on ttPA. There were no significant side x width, side x SM, width x SM, and side x SM x width interactions.

In summary, there were no SM or Group main effects on ttPA. There were no movement amplitude and target width main effects on ttPA in either group.

Head Movement Fitts' Task

Movement Time (MT)

Between-Group Analysis

Table 13. Descriptive statistics for head MT (sec).

	Group	Pre-SM	Post-SM
N	Asymp	20	20
	Symp	20	20
Missing	Asymp	0	0
	Symp	0	0
Mean	Asymp	0.938	0.938
	Symp	0.964	0.911
Median	Asymp	0.937	0.926
	Symp	0.966	0.897
Standard deviation	Asymp	0.124	0.126
	Symp	0.125	0.116
Minimum	Asymp	0.763	0.746
	Symp	0.663	0.666
Maximum	Asymp	1.20	1.14
	Symp	1.18	1.10

There was a significant main effect of SM on head MT ($F_{(1,38)} = 5.30$; $p = 0.027$; $\eta^2_p = 0.122$) and a significant SM x Group interaction on MT ($F_{(1,38)} = 5.67$; $p = 0.022$; $\eta^2_p = 0.130$)(Fig. 22). Post-hoc testing showed a reduced MT following SM occurred within the symptomatic group ($p = 0.011$), whereas MT in the asymptomatic group was not changed ($p = 1.000$). There was no significant main effect for Group on MT ($p = 0.994$).

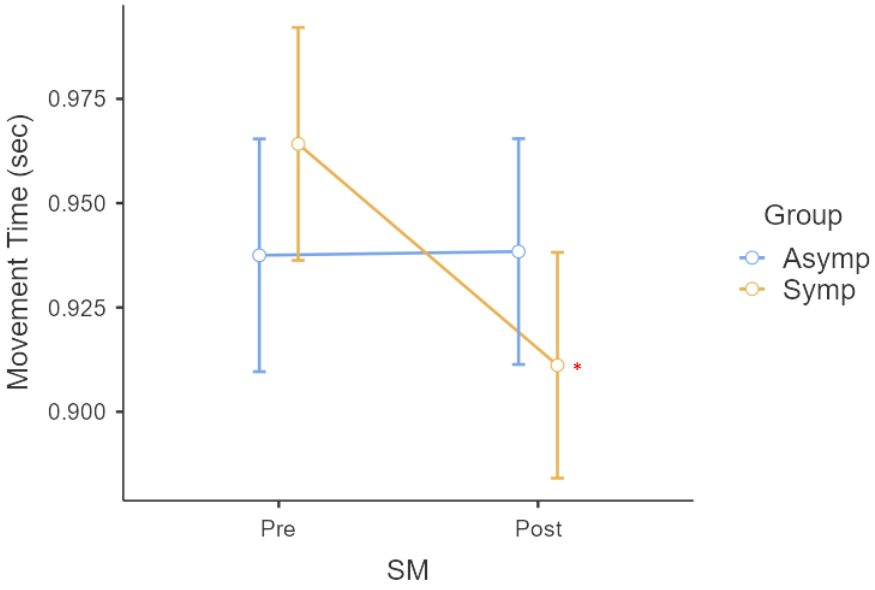


Figure 22. SM x Group interaction on Head Movement Time

Within-Group Analysis – Symptomatic

Head MT increased with larger movement amplitudes and head MT decreased at each movement amplitude following SM in the symptomatic group. There were significant main effects of amplitude for the four target amplitudes at the common target width of 1.2 cm on MT ($F_{(3,57)} = 28.33$; $p < 0.001$; $\eta^2_p = 0.599$) (Fig. 23) and there was a significant main effect of SM on MT ($F_{(1,19)} = 8.81$; $p = 0.008$; $\eta^2_p = 0.317$) (Fig. 24). Post-hoc testing noted that MT increased with larger amplitudes and significant differences were found when 4.5cm was compared to 8.3 cm ($p = 0.008$), 15.5 cm ($p < 0.001$), and 29.0 cm ($p < 0.001$), and 8.3 cm was compared to15.5 cm ($p = 0.014$) and 29.0 cm target amplitudes ($p = 0.001$). Post-hoc comparisons were not significant for the 15.5 to 29.0 cm amplitudes.

There was no significant main effect of side on MT ($F_{(1,19)} = 0.143$; $p = 0.709$). There was a significant amplitude x side interaction ($F_{(3,57)} = 3.07$; $p = 0.035$; $\eta^2_p = 0.139$) (Fig. 25). Pairwise comparisons showed differences in head MT between the left 4.5 cm and right 15.5 cm amplitudes ($p = 0.002$); left 4.5 cm and right 29.0 cm amplitudes ($p < 0.001$); left 8.3 cm and right 29.0 cm amplitudes ($p = 0.034$); left 15.5 cm and right 4.5 cm amplitudes ($p < 0.001$); left 29.0 cm and right 4.5 cm amplitudes ($p < 0.001$); and left 29.0 cm and right 8.3 cm amplitudes ($p = 0.006$). This interaction reveals that MT increased with larger target amplitudes when the

participant rotated their head to the left or right and the main effect of amplitude was different between the left and right side. There were no significant differences for the amplitude x SM, side x SM, and amplitude x side x SM interactions.

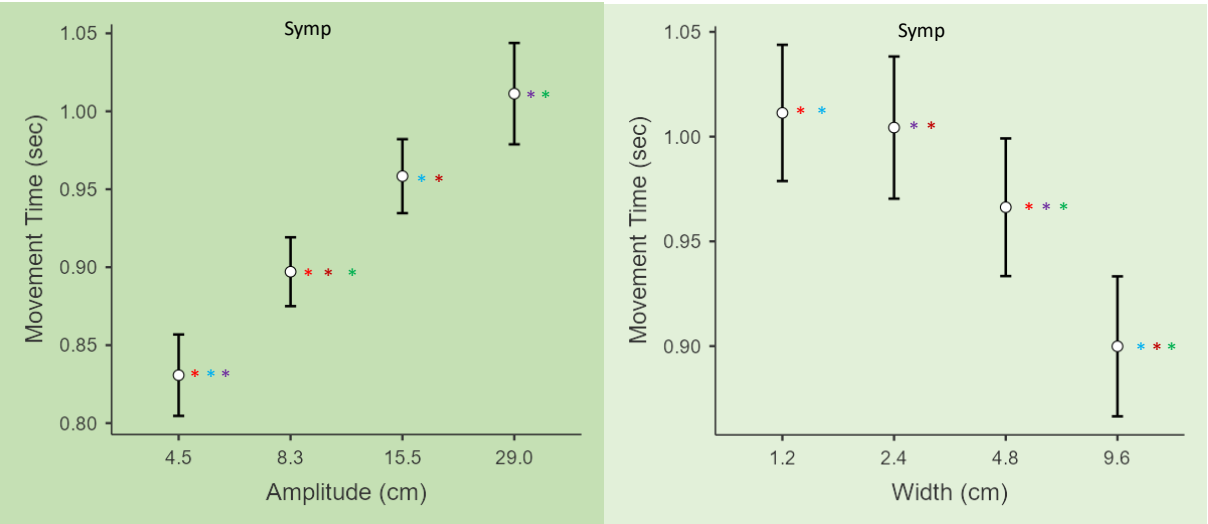


Figure 23. Main Effects of Amplitude & Width on Symptomatic Head Movement Time

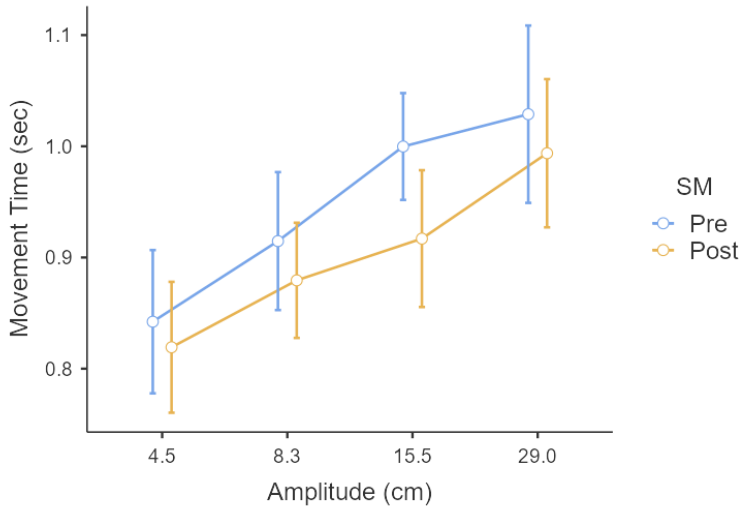


Figure 24. Main Effects of Amplitude and SM on Symptomatic Head Movement Time

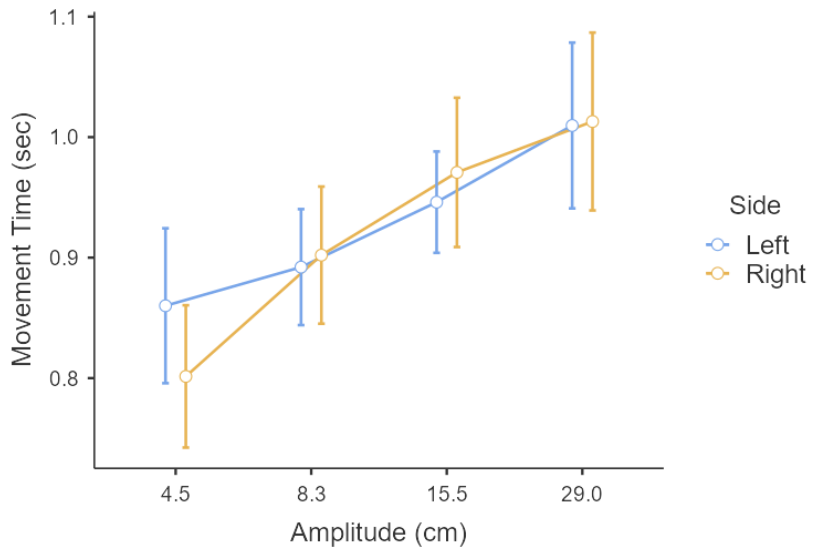


Figure 25. Amplitude x Side Interaction on Symptomatic Head Movement Time

MT decreased following SM and MT decreased with larger target widths. There were significant main effects of target width ($F_{(3,57)} = 21.63$ 3; $p < 0.001$; $\eta^2_p = 0.532$) (Fig. 23) and SM ($F_{(1,19)} = 7.85$; $p = 0.011$; $\eta^2_p = 0.292$) (Fig. 26) on MT for the four target widths at the common target width of 29.0 cm. Post-hoc testing noted significant differences between the 1.2 and 4. 8 cm ($p = 0.036$), the 1.2 and 9.6 cm ($p < 0.001$), the 2.4 and 4.8 cm ($p = 0.046$), the 2.4 and 9.6 cm ($p < 0.001$), and the 4.8 and 9.6 cm ($p < 0.001$) target widths. There was no significant pairwise comparison between the 1.2 and 2.4 cm target widths. There were no significant effects for the width x SM, and side x SM, side x width and width x side x SM interactions. The increased MT with longer amplitudes and decreased MT with larger target widths is predicted by the Fitts' Law speed-accuracy trade-off principle.

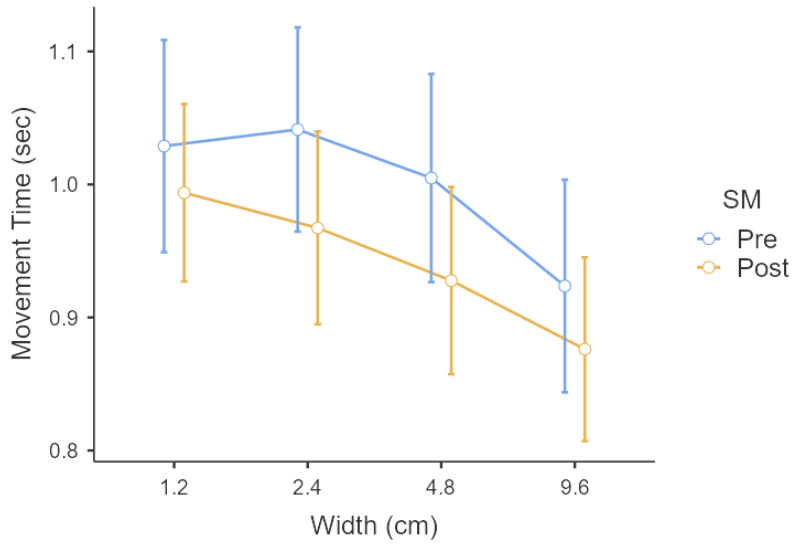


Figure 26. Main Effects of Width and SM on Symptomatic Head Movement Time

Within-Group Analysis – Asymptomatic

There was a significant main effect of target amplitude at the common target width of 1.2 cm on MT ($F_{(3,57)} = 45.36$; $p < 0.001$; $\eta^2_p = 0.705$) (Fig. 28), whereby MT increased with larger target amplitudes. Post hoc pairwise comparisons noted a significant difference between all the amplitudes: 4.5 and 8.3 cm ($p < 0.001$), 4.5 and 15.5 cm ($p < 0.001$), 4.5 and 29.0 cm ($p < 0.001$), 8.3 and 15.5 cm ($p = 0.047$), 8.3 and 29.0 cm ($p < 0.001$), and 15.5 and 29.0 cm ($p = 0.015$). There were no significant MT main effects for side ($F_{(1,19)} = 0.242$; $p= 0.628$) and SM ($F_{(1,19)} = 0.402$; $p= 0.533$). There was a significant SM x side interaction ($F_{(1,19)} = 5.73$; $p = 0.027$; $\eta^2_p = 0.232$) such that MT increased post-SM for right rotational head movements but decreased post-SM for left rotational head movements (Fig. 27). There were no significant pairwise comparisons for this interaction. The improvement in MT for the left head movements indicates that the asymptomatic group displayed an improved ability to rotate their head in this direction. There were no significant amplitude x side, amplitude x SM, or amplitude x side x SM interactions.

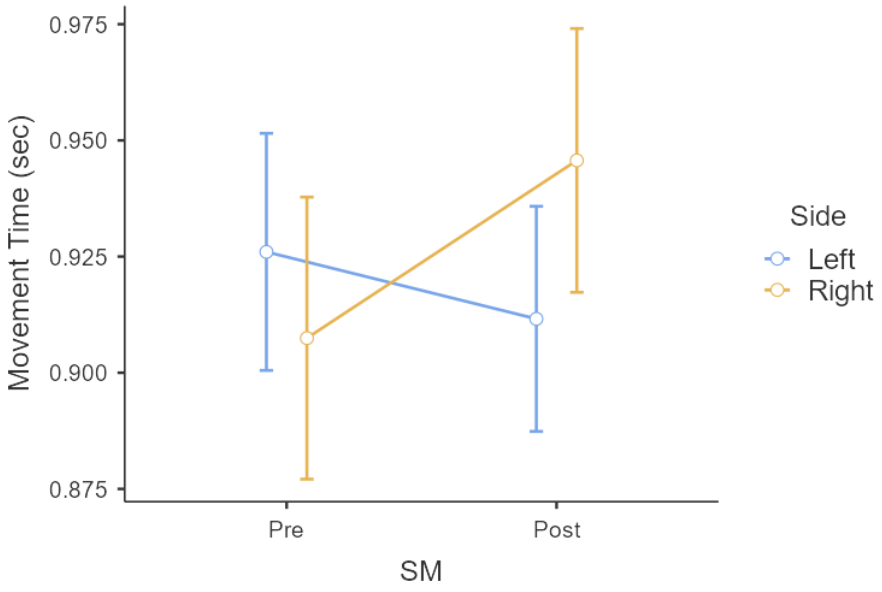


Figure 27. SM x Side Interaction on Asymptomatic Head Movement Time

There was a significant main effect of the four target widths at the common target amplitude of 29.0 cm on MT ($F_{(3,57)} = 18.421$; $p < 0.001$; $\eta^2_p = 0.492$), whereby MT decreased as target width increased (Fig. 28). Asymptomatic patients had shorter MTs when they moved their heads to larger width targets. This pattern was not affected by the direction of head movement or by SM. Pairwise comparisons noted a significant difference between the following widths: 1.2 and 4.8 cm ($p = 0.020$), 1.2 and 9.6cm ($p < 0.001$), 2.4 and 4.8cm ($p = 0.044$), 2.4 and 9.6 cm ($p < 0.001$), 4.8 and 9.6 cm ($p = 0.010$). There was no difference between the 1.2 and 2.4 cm widths ($p = 0.467$). There were no significant main effects on MT for side ($F_{(1,19)} = 0.082$; $p= 0.778$) and SM ($F_{(1,19)} = 0.195$; $p= 0.664$). There were no significant width x side, width x SM, side x SM, or width x side x SM interactions.

The increased MT with longer amplitudes and decreased MT with larger target widths is predicted by the Fitts' Law speed-accuracy trade-off principle.

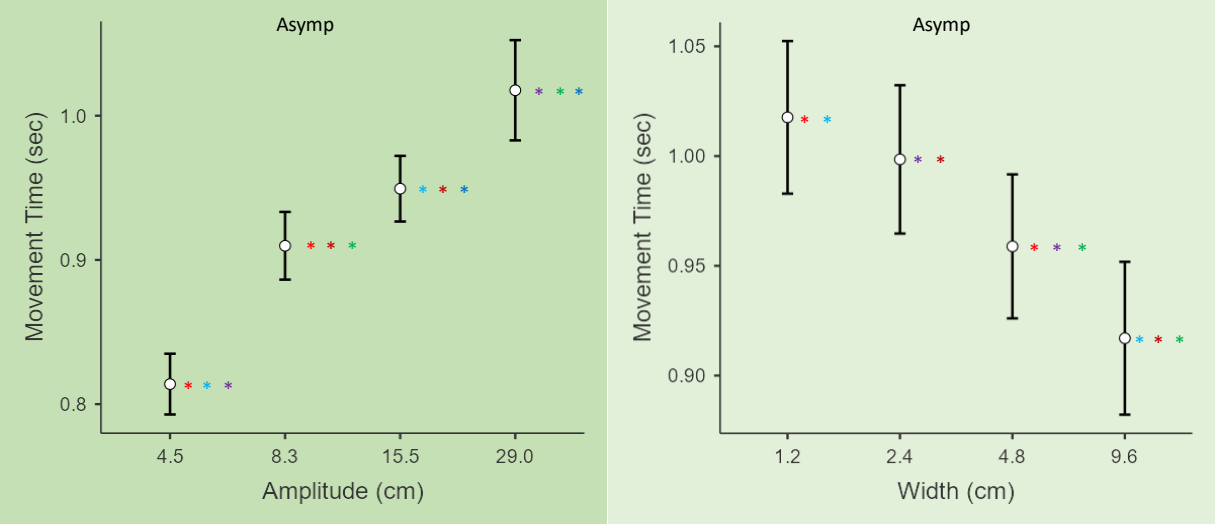


Figure 28. Main Effects of Amplitude & Width on Asymptomatic Head Movement Time

In summary, head MT was altered during the amplitude and width conditions following SM in the symptomatic group but not the asymptomatic group. There was an overall effect of SM on MT but there were no significant pairwise comparisons for the asymptomatic group.

Reaction Time (RT)

Between-Group Analysis

Table 14. Descriptive statistics for head RT (sec).

	Group	Pre-SM	Post-SM
N	Asymp	20	20

Table 14. Descriptive statistics for head RT (sec).

	Group	Pre-SM	Post-SM
	Symp	20	20
Missing	Asymp	0	0
	Symp	0	0
Mean	Asymp	0.253	0.247
	Symp	0.248	0.240
Median	Asymp	0.257	0.251
	Symp	0.250	0.241
Standard deviation	Asymp	0.0373	0.0306
	Symp	0.0235	0.0243
Minimum	Asymp	0.183	0.190
	Symp	0.209	0.203
Maximum	Asymp	0.353	0.297
	Symp	0.287	0.287

There was a significant main effect of SM on RT ($F_{(1,38)} = 9.950$; $p = 0.003$; $\eta^2_p = 0.208$)(Fig. 29) indicating that there was a reduction in head movement RT following SM. There was no significant main effect for group ($F_{(1,38)} = 0.472$; $p = 0.496$; $\eta^2_p = 0.012$) or SM x Group interaction ($F_{(1,38)} = 0.157$; $p = 0.695$; $\eta^2_p = 0.004$).

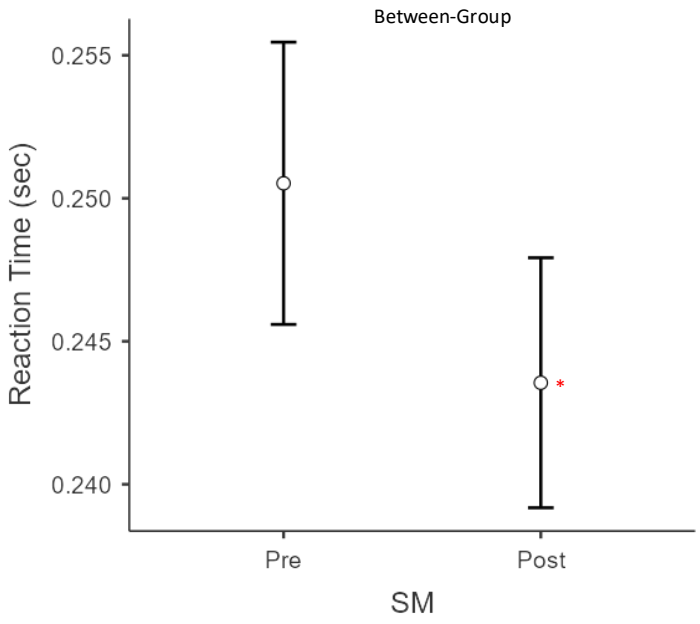


Figure 29. Main Effect of SM on RT Based on Between-Group Analysis

Within-Group Analysis – Symptomatic

Head movement RT was not influenced by increasing movement amplitudes. There were no significant main effects on RT for the different amplitudes at the common target width of 1.2 cm ($F_{(3,57)} = 0.710$; p= 0.550) (Fig. 32). There was also no significant main effect on RT for left and right sided rotational head movements ($F_{(1,19)} = 0.108$; p= 0.747). There was a significant main effect of SM on RT ($F_{(1,19)} = 7.81$; p= 0.012; $\eta^2_p = 0.291$)(Fig. 30) indicating that symptomatic participant RT decreased during head movement following SM. There were no significant amplitude x side, amplitude x SM, side x SM, and amplitude x side x SM interactions.

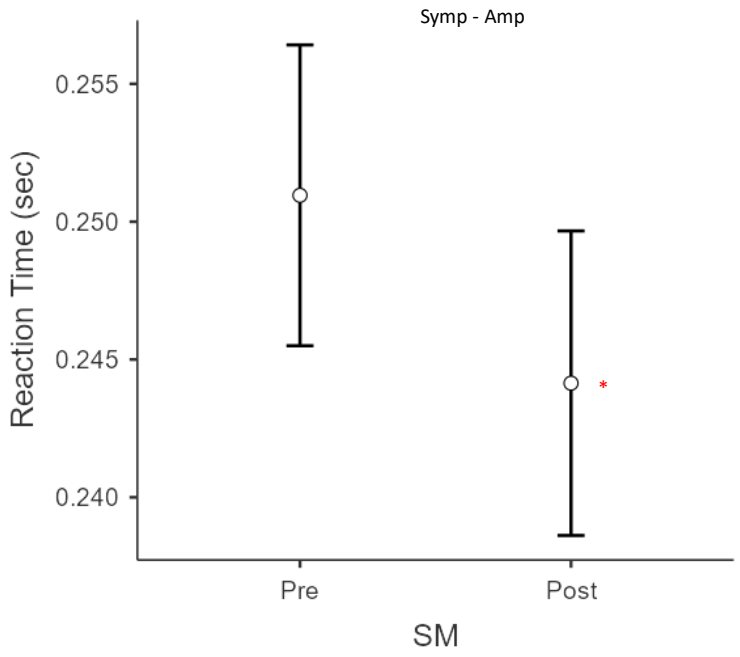


Figure 30. Main Effect of SM on Symptomatic Head Reaction Time for Amplitude Condition

RT decreased following SM and RT also decreased with larger target widths during the rotational head movement task. There were significant main effects of SM ($F_{(1,19)} = 6.24$; p= 0.022; $\eta^2_p = 0.247$)(Fig. 32) and width ($F_{(3,57)} = 12.37$; $p < 0.001$; $\eta^2_p = 0.394$) (Fig. 31) on RT at the common target amplitude of 29.0 cm. Pairwise comparisons demonstrated that RT decreased from the 1.2 to 4.8 cm (p = 0.046), 1.2 to 9.6 cm (p < 0.001), 2.4 to 9.6 cm (p = 0.009), and 4.8 to 9.6 cm (p = 0.008) target widths. There was no significant main effect on RT related to the side of horizontal head movement ($F_{(1,19)} = 2.59$; p= 0.124). There were no significant amplitude x side, width x SM, side x SM, and width x side x SM interactions.

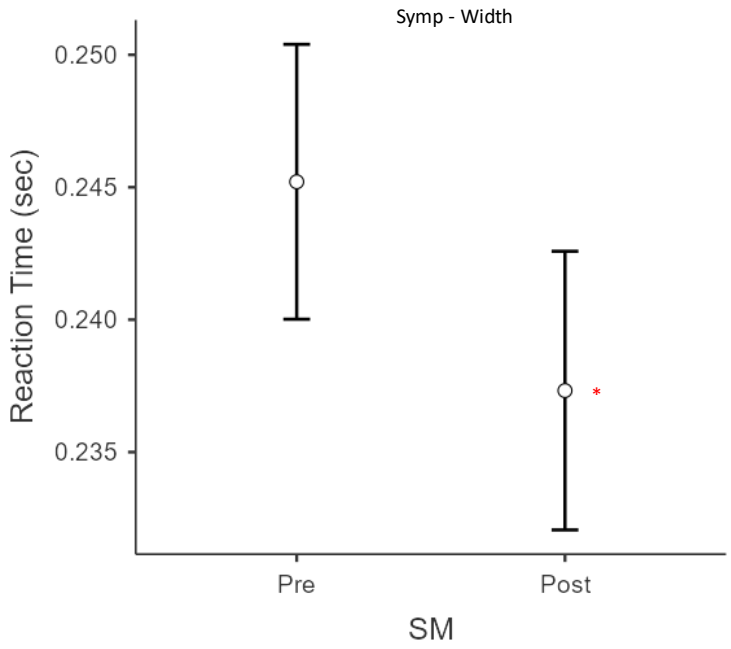


Figure 31. Main Effect of SM on Symptomatic Head Reaction Time for Width Condition

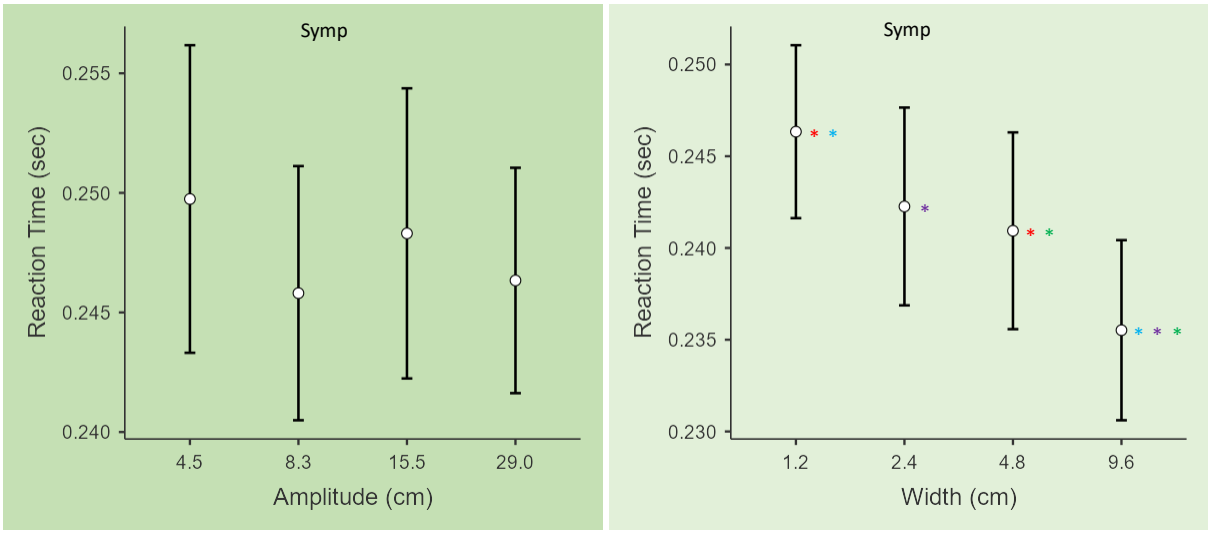


Figure 32. Main Effects of Amplitude & Width on Symptomatic Head Reaction Time

Within-Group Analysis – Asymptomatic

The asymptomatic group's head RT was not altered by amplitude, the side of rotational head movement, or following SM. There were no significant main effects for the different amplitudes at the common target width of 1.2cm on RT ($F_{(3,57)} = 0.329$; $p = 0.804$) (Fig. 33). There were also no significant main effects for left and right sided rotational head movements on

RT ($F_{(1,19)}$ = 2.77; p= 0.113) and post-SM ($F_{(1,19)}$ = 2.03; p= 0.171). Likewise, there were no significant amplitude x side, amplitude x SM, side x SM, and amplitude x side x SM interactions.

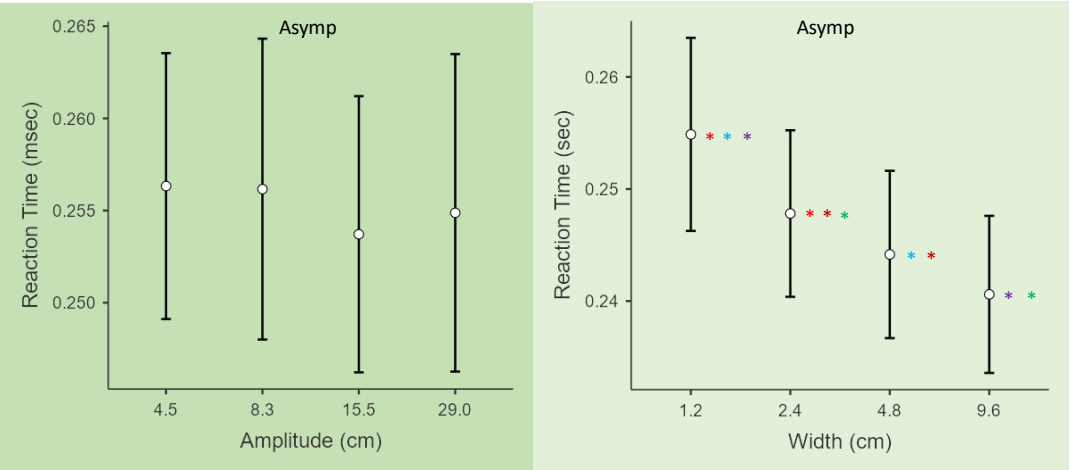


Figure 33. Main Effects of Amplitude & Width on Asymptomatic Head Reaction Time

There was a significant main effect for target width on RT ($F_{(3,57)}$ = 14.222; p < 0.001; η^2_p = 0.428) (Fig. 33) at a common target amplitude of 29.0 cm, indicating that RT decreased as target width increased. More specifically, pairwise comparisons noted that there was a significant decrease in RT when the 1.2 cm target width was compared to the 2.4 (p = 0.034), 4.8 (p = 0.007), and 9.6 (p = 0.001) cm target widths, and when the 2.4 cm target width was compared to the 4.8 (p = 0.032) and 9.6 cm (p = 0.007) target widths. Within the asymptomatic group, RT became shorter as the target widths became larger with no further effect related to SM or the side of head movement. There was no significant change in RT when the 4.8 cm target width was compared to the 9.6 cm target width (p = 0.166). There were no significant RT main effects for side ($F_{(1,19)}$ = 0.088; p= 0.361) and SM ($F_{(1,19)}$ = 3.75; p= 0.165). There was a significant side x SM x width interaction ($F_{(3,57)}$ = 3.139; p = 0.032; η^2_p = 0.142) (Fig. 34). Pairwise comparisons noted a significant decrease in RT for leftward head movements between the 2.4 cm and 9.6 cm target widths. There were no significant width x side, width x SM, and side x SM interactions.

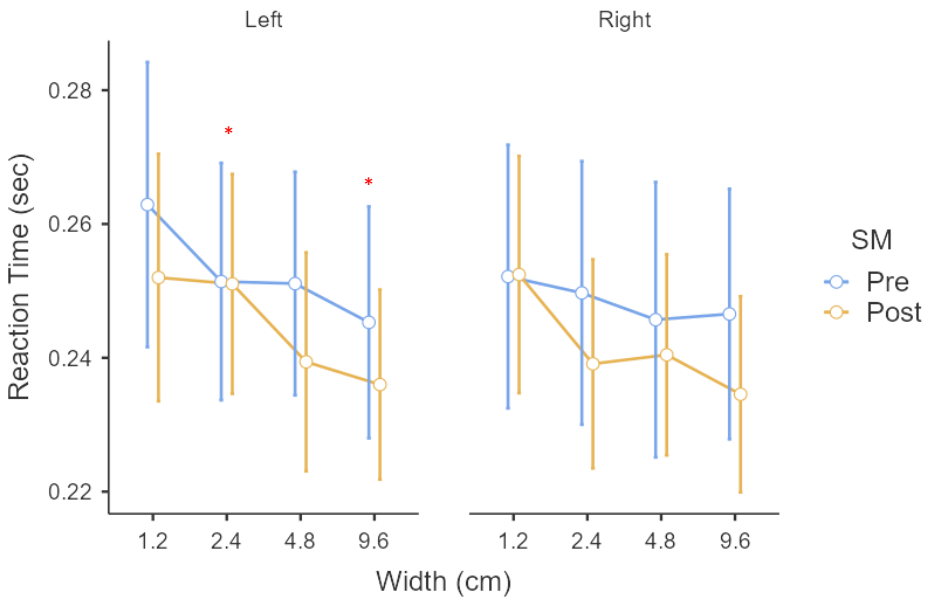


Figure 34. Side x SM x Width Interaction on Asymptomatic Head Reaction Time

In summary, there was a significant reduction in head movement RT following SM, but there was no effect of Group on RT. There was a reduction in head movement RT following SM within the symptomatic group as movement amplitudes and target widths became larger. Within the asymptomatic group there was no effect of movement amplitude on RT, but RT did decrease with larger target widths. There was no effect of SM within the asymptomatic group as movement amplitudes and target widths became larger.

Constant Error (CE)

Between-Group Analysis

Table 15. Descriptive statistics for head CE (degrees).

	Group	Pre-SM	Post-SM
N	Asymp	20	20

Table 15. Descriptive statistics for head CE (degrees).

	Group	Pre-SM	Post-SM
	Symp	20	20
Missing	Asymp	0	0
	Symp	0	0
Mean	Asymp	0.00730	-0.0360
	Symp	-0.0417	0.0609
Median	Asymp	-0.0445	-0.00350
	Symp	-0.0255	0.0110
Standard deviation	Asymp	0.330	0.148
	Symp	0.165	0.257
Minimum	Asymp	-0.459	-0.410
	Symp	-0.438	-0.329
Maximum	Asymp	1.17	0.123
	Symp	0.256	0.938

A bias to undershoot or overshoot the target was not altered following SM in either experimental group. There were no significant main effects of SM ($F_{(1,38)} = 0.577$; $p = 0.452$; $\eta^2_p = 0.015$) or Group ($F_{(1,38)} = 0.140$; $p = 0.710$; $\eta^2_p = 0.004$) on CE. There was no significant SM x Group interaction ($F_{(1,38)} = 3.498$; $p = 0.069$; $\eta^2_p = 0.084$).

Within-Group Analysis – Symptomatic

The main effect of amplitude on CE demonstrated an undershoot for all amplitudes except for an overshoot at the 29.0 cm amplitude within the symptomatic group. There was significant main effect of target amplitude ($F_{(3,57)} = 329.73$ 3; $p < 0.001$; $\eta^2_p = 0.946$) (Fig. 35) on CE for the four target amplitudes at the common target width of 1.2 cm. Pairwise comparisons noted significant differences between the 4.5 cm and 29.0 cm ($p = 0.018$), the 8.3 cm and 29.0 cm ($p = 0.003$), and the 15.5 cm and 29.0 cm ($p = 0.002$) target amplitudes. There were no significant pairwise comparisons between the 4.5 cm and 8.3 cm, the 4.5 cm and 15.5 cm, and the 8.3 cm and 15.5 cm target widths. There were no significant main effects of SM ($F_{(1,19)} = 3.40$; $p = 0.081$) or side ($F_{(1,19)} = 3.80$; $p = 0.066$) on CE.

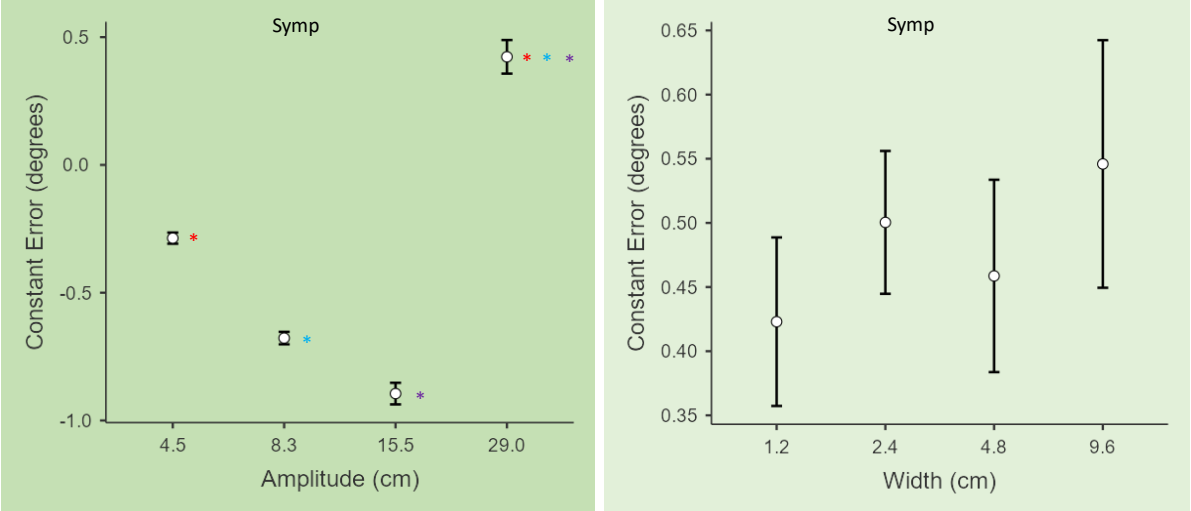


Figure 35. Main Effects of Amplitude & Width on Symptomatic Head Constant Error

There was a significant amplitude x side interaction ($F_{(3,57)} = 8.57$; $p < 0.001$; $\eta^2_p = 0.311$) (Fig. 36) that demonstrated CE increased in a negative direction during left and right head rotational movements from the 4.5 cm to 8.3 cm and 15.5 cm amplitudes thereby indicating a bias to an undershoot between these target amplitudes. In contrast, CE was increased in a positive direction from the 15.5 cm to the 29.0 cm target amplitude for left and right head movements, indicating there was a bias to an overshoot between these target amplitudes. Post-hoc testing demonstrated significant differences ($p < 0.001$) between the following comparisons of head movement amplitudes: left 4.5 cm to the right 8.3 cm, 15. 5cm, and 29.0 cm; left 8.3 cm to the right 15.5 cm and 29.0 cm; left 15.5 cm to right 29.0 cm; and left 29.0 cm to the right 4.5 cm, 8.3 cm, and 15.5 cm. There were no significant differences for the amplitude x SM, and side x SM, and width x side x SM interactions.

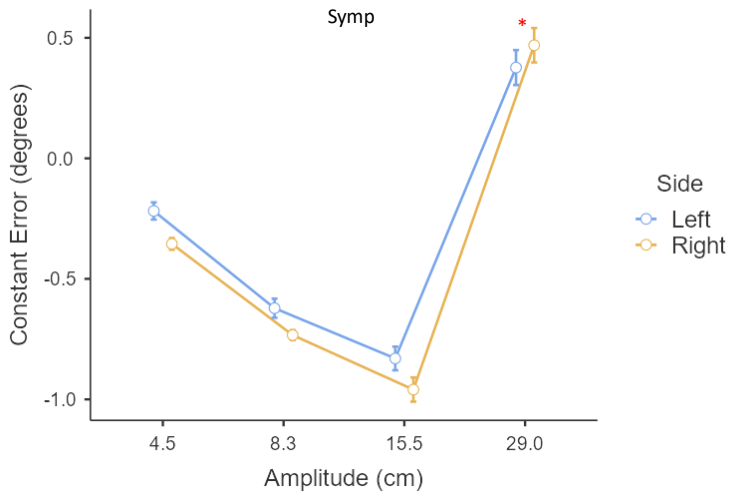


Figure 36. Amplitude x Side Interaction on Symptomatic Head Constant Error

There were no significant main effects of target width ($F_{(3,57)} = 1.31$; $p = 0.279$; $\eta^2_p = 0.065$) (Fig. 35) and side ($F_{(1,19)} = 2.34$; $p = 0.143$; $\eta^2_p = 0.110$) on CE for the four target widths at the common target amplitude of 29.0 cm. There was a significant main effect of SM on CE ($F_{(1,19)} = 7.87$; $p = 0.011$; $\eta^2_p = 0.293$)(Fig. 37). There was a significant side x width interaction (Fig. 38) ($F_{(1,19)} = 3.47$; $p = 0.022$; $\eta^2_p = 0.154$) that demonstrated CE tended to demonstrate a greater bias to an overshoot for leftward head movements compared to rightward head movements. There were no significant differences for the SM x width, and side x width, and width x side x SM interactions.

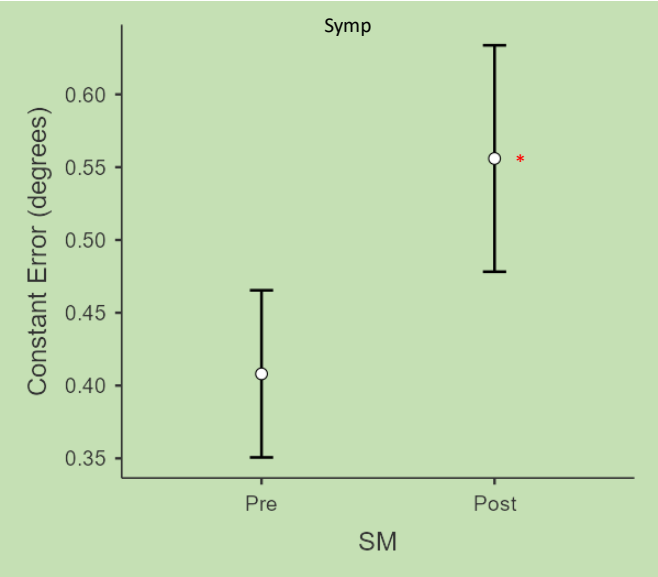


Figure 37. Main Effect of SM on Symptomatic Head Constant Error

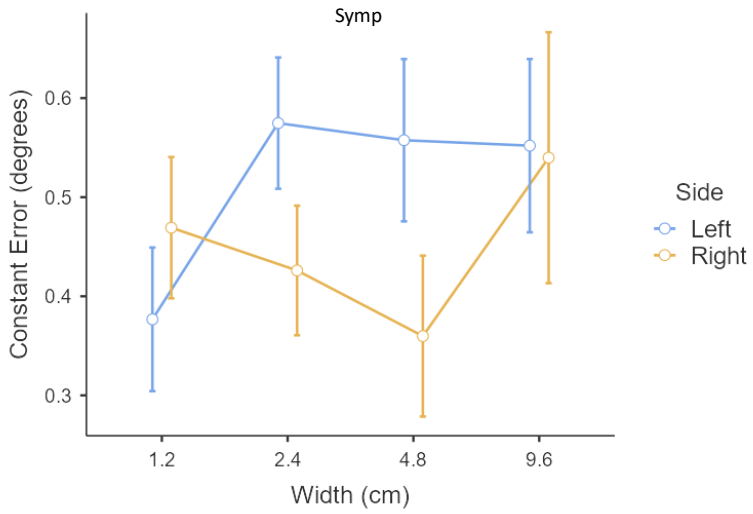


Figure 38. Side x Width Interaction on Symptomatic Head Constant Error

Within-Group Analysis – Asymptomatic

Head movement CE was less for leftward compared to rightward head movements. There were significant main effects for amplitude ($F_{(3,57)} = 107.89$; $p < 0.001$; $\eta^2_p = 0.850$)(Fig. 39) and the side of horizontal head movement on CE ($F_{(1,19)} = 15.55$; $p < 0.001$; $\eta^2_p = 0.450$) (Fig. 40). All pairwise comparisons were significant ($p < 0.001$) between all movement amplitudes except for between the 8.3 cm and 15.5 cm amplitudes. There was no significant main effect of SM on CE ($F_{(1,19)} = 1.342$; $p = 0.261$). There were no significant amplitude x side, amplitude x SM, side x SM, and amplitude x side x SM interactions.

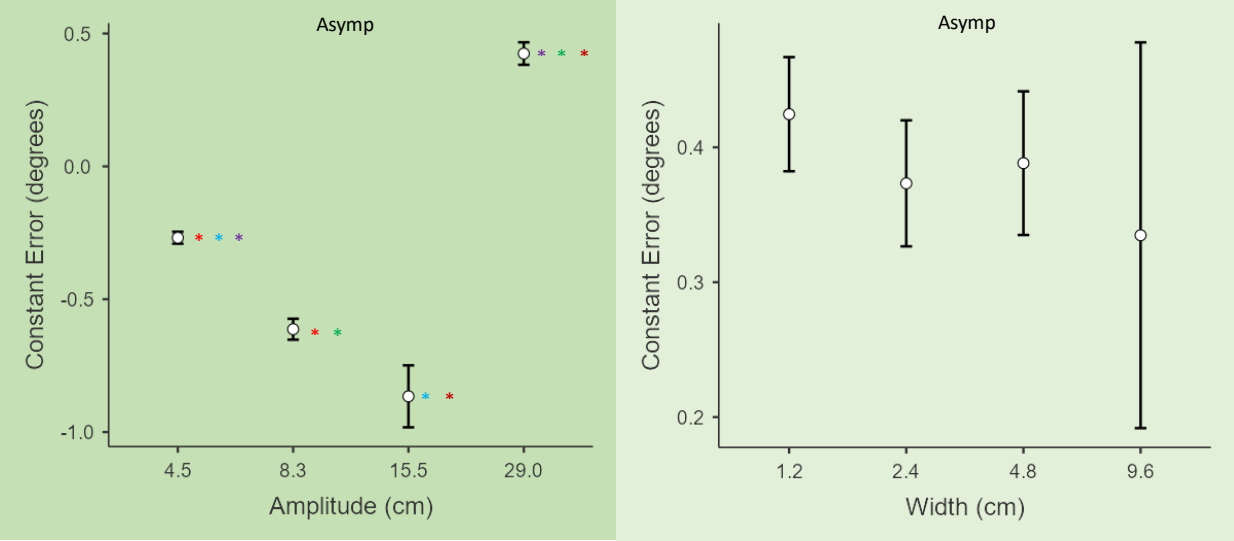


Figure 39. Main Effects of Amplitude & Width on Asymptomatic Head Constant Error

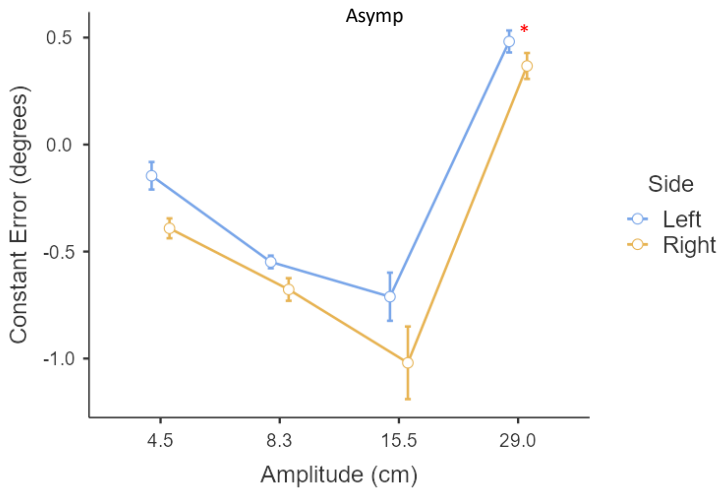


Figure 40. Main Effects of Amplitude and Side on Asymptomatic Head Constant Error

There was a significant main effect of side on CE ($F_{(1,19)} = 5.46$; $p = 0.031$; $\eta^2_p = 0.223$) (Fig. 40). There were no significant main effects for width ($F_{(3,57)} = 0.281$; $p = 0.839$) (Fig. 39) and SM ($F_{(1,19)} = 0.681$; $p = 0.420$). There was a significant SM x width interaction for CE for the four target widths at the common target width of 29.0 cm ($F_{(3,57)} = 3.19$; $p = 0.030$; $\eta^2_p = 0.144$)(Fig. 41). There were no significant differences identified on pairwise comparisons pre- and post-SM at the four target widths. There was a trend for CE to decrease following SM at all

target widths except for the 2.4 cm width indicating that there was an error bias due to a reduction in target overshoots.

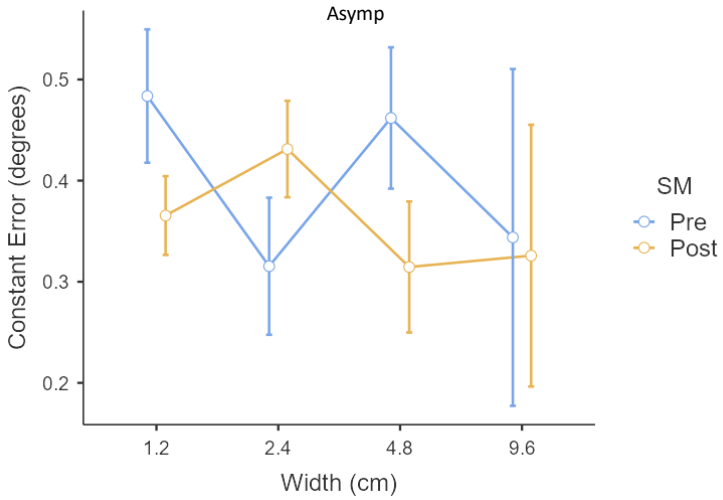


Figure 41. SM x Width Interaction on CE within Asymptomatic Group Head for Head Task

In summary, head movement accuracy was not changed following SM in either the symptomatic or asymptomatic groups. The main effect of amplitude on CE demonstrated an undershoot for all amplitudes except for an overshoot at the 29.0 cm amplitude within the symptomatic and asymptomatic groups. There was no main effect of target width on CE within both groups. There was a larger overshoot post-SM compared to pre-SM during the width condition within the symptomatic group. There was a greater overshot bias for left head rotations compared to right head rotations within the symptomatic group. Finally, CE was shown to decrease post-SM at all target widths except for the 2.4 cm width indicating that there was an error bias due to a reduction in target overshoots.

Variable Error (VE)

Between-Group Analysis

Table 16. Descriptive statistics for head VE (degrees).

	Group	Pre-SM	Post-SM
N	Asymp	20	20
	Symp	20	20
Missing	Asymp	0	0
	Symp	0	0
Mean	Asymp	23.3	23.1
	Symp	23.1	23.3
Median	Asymp	23.1	23.1
	Symp	23.1	23.2
Standard deviation	Asymp	0.894	0.182
	Symp	0.208	0.282
Minimum	Asymp	22.5	22.7
	Symp	22.7	22.8
Maximum	Asymp	26.9	23.4
	Symp	23.5	24.2

The consistency of head movements was not altered in either experimental group following SM. There were no significant main effects of SM ($F_{(1,38)} = 0.019$; $p = 0.893$; $\eta^2_p = 0.000$) or Group ($F_{(1,38)} = 0.001$; $p = 0.972$; $\eta^2_p = 0.000$) on VE. There was no significant SM x Group interaction ($F_{(1,38)} = 2.282$; $p = 0.139$; $\eta^2_p = 0.057$).

Within-Group Analysis – Symptomatic

There was a significant main effect of target amplitude ($F_{(3,57)} = 37.44$; $p < 0.001$; $\eta^2_p = 0.663$) (Fig. 42) on VE for the four target amplitudes at the common target width of 1.2 cm. Post-

hoc testing noted significant differences between the 4.5 cm and 15.5 cm ($p = 0.012$), the 4.5 cm and 29.0 cm ($p < 0.001$), the 8.3 cm and 29.0 cm ($p < 0.001$), and the 15.5 cm and 29.0 cm ($p < 0.001$) target amplitudes. This demonstrates that VE generally increased during head movement to the larger amplitude indicating that participants became less consistent in their head movements over larger target amplitudes. In particular, there was inconsistency at the 29.0 cm movement amplitude due to the greater variability. There was no significant pairwise comparison between the 4.5 cm and 8.3 cm, and the 8.3 cm and 15.5 cm target amplitudes. There were no significant main effects of side ($F_{(1,16)} = 1.18$; $p = 0.294$) or SM ($F_{(1,16)} = 0.232$; $p = 0.637$) on VE. There were no significant amplitude x side, amplitude x SM, side x SM, and amplitude x side x SM interactions.

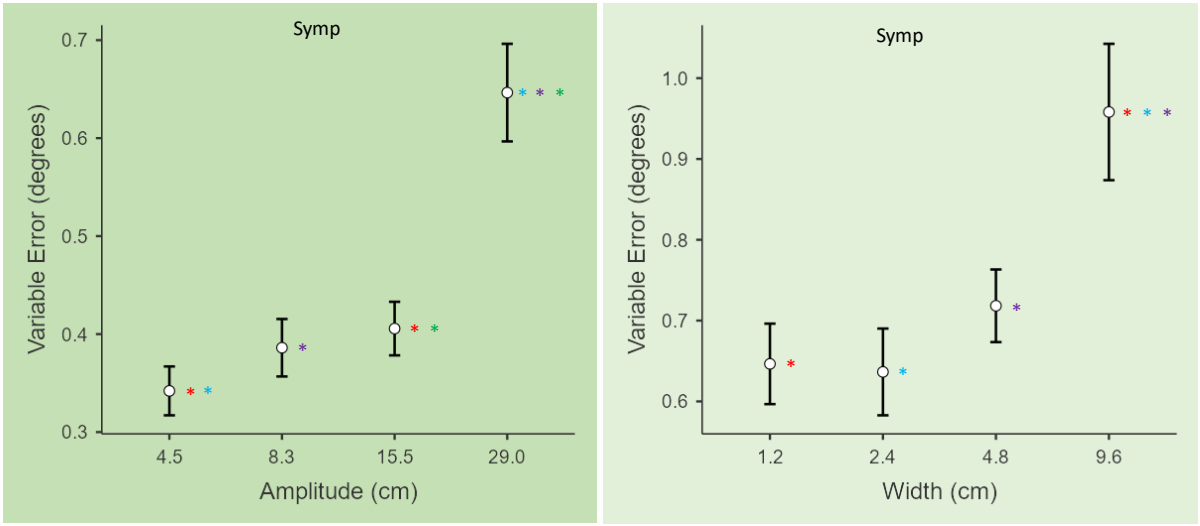


Figure 42. Main Effects of Amplitude & Width on Symptomatic Head Variable Error

Head movement became less consistent with larger target widths for the symptomatic group. There was a significant main effect of target width ($F_{(3,57)} = 12.28$; $p < 0.001$; $\eta^2_p = 0.392$) (Fig. 42) on VE for the four target widths at the common target amplitude of 29.0 cm. Pairwise comparisons noted significant differences between the 1.2 cm and 9.6 cm ($p = 0.004$), the 2.4 cm and 9.6 cm ($p < 0.001$), and the 4.8 cm and 9.6 cm ($p = 0.010$) target widths. There was no significant pairwise comparisons between the 1.2 cm and 2.4 cm, the 1.2 cm and 4.8 cm, and the 2.4 cm and 4.8 cm target widths. There were no significant main effects of side ($F_{(1,19)} = 0.223$; $p = 0.642$) and SM ($F_{(1,19)} = 0.036$; $p = 0.852$). There was a significant SM x side x width interaction ($F_{(3,57)} = 3.50$; $p = 0.021$; $\eta^2_p = 0.156$)(Fig. 43) demonstrated post-SM as compared to

pre-SM: VE decreased on leftward and increased on rightward head movements at the 1.2 cm width, VE increased on bilateral head movements at the 2.4 cm width, VE increased on head movements to the left but decreased on movements to the right at the 4.8 cm width. Lastly, at the 9.6 cm target width VE post-SM decreased with head movements to the left but increased with right head movements. These results indicate that following SM there was

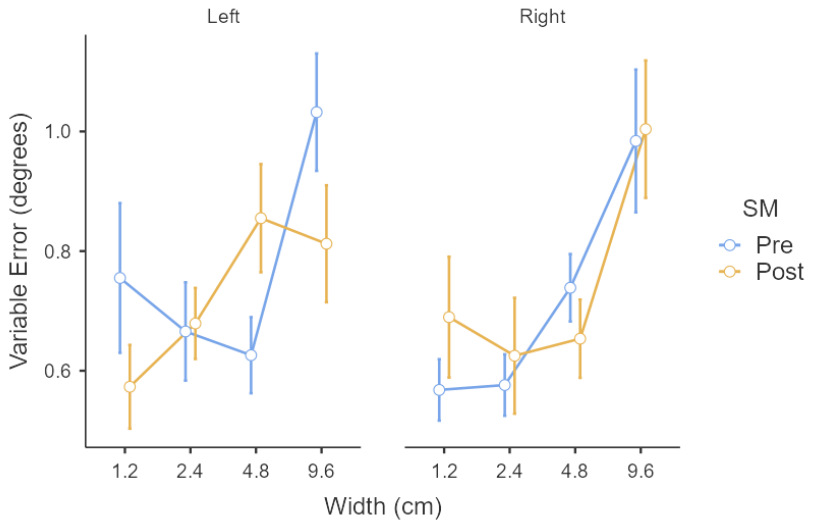


Figure 43. Width x Side x SM Interaction on VE within Symptomatic Group for Head Task

Within-Group Analysis – Asymptomatic

There was a significant main effect on VE for the four target amplitudes at the common target width of 1.2 cm ($F_{(3,57)} = 3.70$; $p = 0.017$; $\eta^2_p = 0.163$)(Fig. 44). Pairwise comparisons noted that VE increased with larger amplitudes. Specifically, VE increased from the 4.5 to 29.0 cm amplitude ($p = 0.010$) and the 8.3 to 29.0 cm amplitude ($p = 0.001$). There was no significant difference for VE between the 4.5 and 8.3 cm, the 4.5 and 15.5 cm, the 8.3 and 15.5 cm, and the 15.5 and 29.0 cm amplitudes. Head movement inconsistency was greater at the 15.5 cm and 29.0 cm amplitudes due to the larger variability at these data points. There were no significant main effects on VE for side ($F_{(1,19)} = 1.14$; $p= 0.299$), and SM ($F_{(1,19)} = 0.204$; $p= 0.657$). There were no significant differences for the amplitude x side, amplitude x SM, side x SM, and amplitude x side x SM interactions.

There was a significant main effect on VE for the four target widths at the common target amplitude of 29.0 cm ($F_{(3,57)} = 9.13$; $p < 0.001$; $\eta^2_p = 0.325$)(Fig. 44). Pairwise comparisons noted

that VE increased with larger target widths. In fact, VE increased from the 1.2 to 9.6 cm width ($p < 0.001$), the 2.4 to 9.6 cm amplitude ($p = 0.027$), and the 4.8 to 9.6 cm amplitude ($p = 0.002$). There was no significant difference on VE between the 1.2 and 2.4 cm, 1.2 to 4.8 cm, and 2.4 to 4.8 cm widths. There were no significant main effects on VE for side ($F_{(1,19)} = 0.094$; $p = 0.763$), and SM ($F_{(1,19)} = 2.43$; $p = 0.136$). There were no significant differences for the amplitude x side, amplitude x SM, side x SM, and amplitude x side x SM interactions.

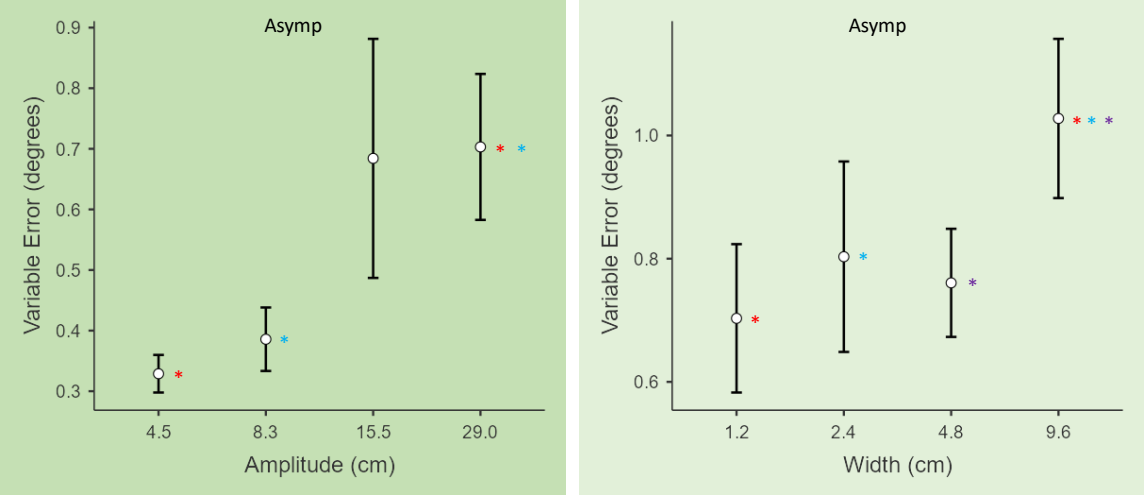


Figure 44. Main Effects of Amplitude & Width on Asymptomatic Head Variable Error

In summary, head movement consistency was not altered in either experimental group following SM. There was an increase in VE within the symptomatic and asymptomatic groups as movement amplitudes and target widths became larger. Within the symptomatic group there was a significant SM x Side x Width interaction indicating that following SM there was less consistency during rotational head movement to the left or right as target widths increased.

Peak Velocity (PV)

Between-Group Analysis

Table 17. Descriptive statistics for head PV (degrees/sec).

	Group	Pre-SM	Post-SM
N	Asymp	20	20
	Symp	20	20

Table 17. Descriptive statistics for head PV (degrees/sec).

	Group	Pre-SM	Post-SM
Missing	Asymp	0	0
	Symp	0	0
Mean	Asymp	58.0	56.5
	Symp	60.5	62.4
Median	Asymp	57.1	54.7
	Symp	59.4	58.9
Standard deviation	Asymp	13.6	11.3
	Symp	16.6	15.5
Minimum	Asymp	38.9	39.3
	Symp	39.8	38.6
Maximum	Asymp	83.7	77.8
	Symp	108	107

Head movement PV was not altered in either group following SM. There were no significant main effects of SM ($F_{(1,38)} = 0.031$; $p = 0.861$; $\eta^2_p = 0.001$) or Group ($F_{(1,38)} = 0.907$; $p = 0.347$; $\eta^2_p = 0.023$) on PV. There was no significant SM x Group interaction ($F_{(1,38)} = 3.063$; $p = 0.088$; $\eta^2_p = 0.075$).

Within-Group Analysis – Symptomatic

The participants' approach was to rotate their head faster towards the target over a larger amplitude. There was a significant main effect on PV for the four target amplitudes at the common target width of 1.2 cm ($F_{(3,57)} = 255.80$; $p < 0.001$; $\eta^2_p = 0.931$)(Fig. 45). Additionally, post-hoc testing noted that PV increased with larger amplitudes during all amplitude comparisons ($p < 0.001$). There were no significant main effects of PV for side ($F_{(1,19)} = 0.542$; $p= 0.471$) and SM ($F_{(1,19)} = 0.080$; $p= 0.780$). There were no significant differences for the amplitude x side, amplitude x SM, side x SM, and amplitude x side x SM interactions.

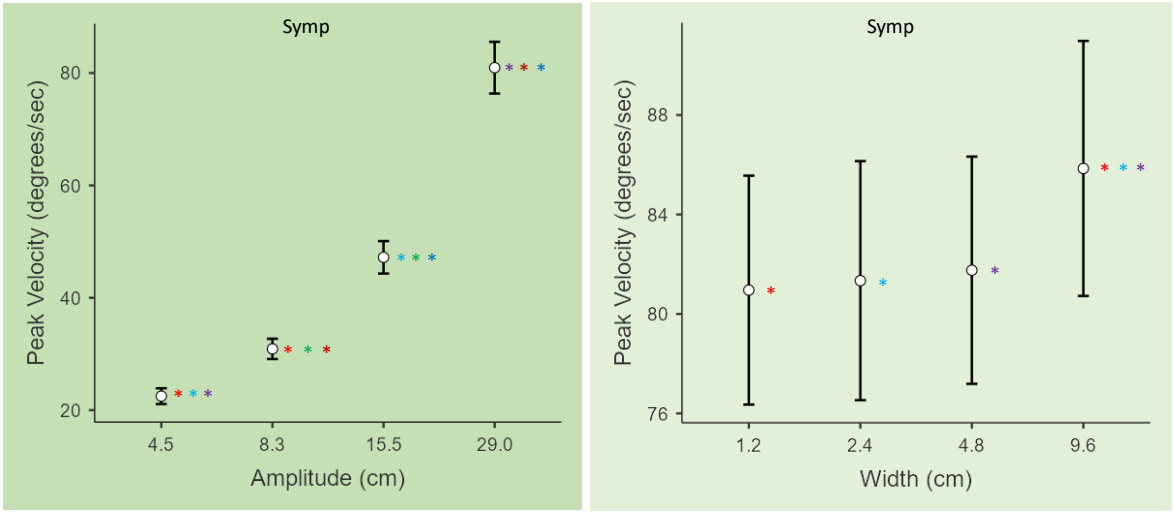


Figure 45. Main Effects of Amplitude & Width on Symptomatic Head Peak Velocity

There was a significant main effect on PV for the four target widths at the common target amplitude of 29 cm ($F_{(3,57)} = 9.42$; $p < 0.001$; $\eta^2_p = 0.331$) (Fig. 45). Post-hoc testing noted a significant increase in PV as target widths increased from 1.2 cm to 9.6 cm ($p = 0.002$), 2.4 cm to 9.6 cm ($p = 0.007$), and 4.8 cm to 9.6 cm ($p = 0.018$). The participants' approach was to rotate their head faster toward a target of larger width. There were no significant main effects on PV for side ($F_{(1,19)} = 0.011$; $p= 0.917$) and SM ($F_{(1,19)} = 2.40$; $p= 0.138$). There were no significant differences for the amplitude x side, amplitude x SM, side x SM, and amplitude x side x SM interactions.

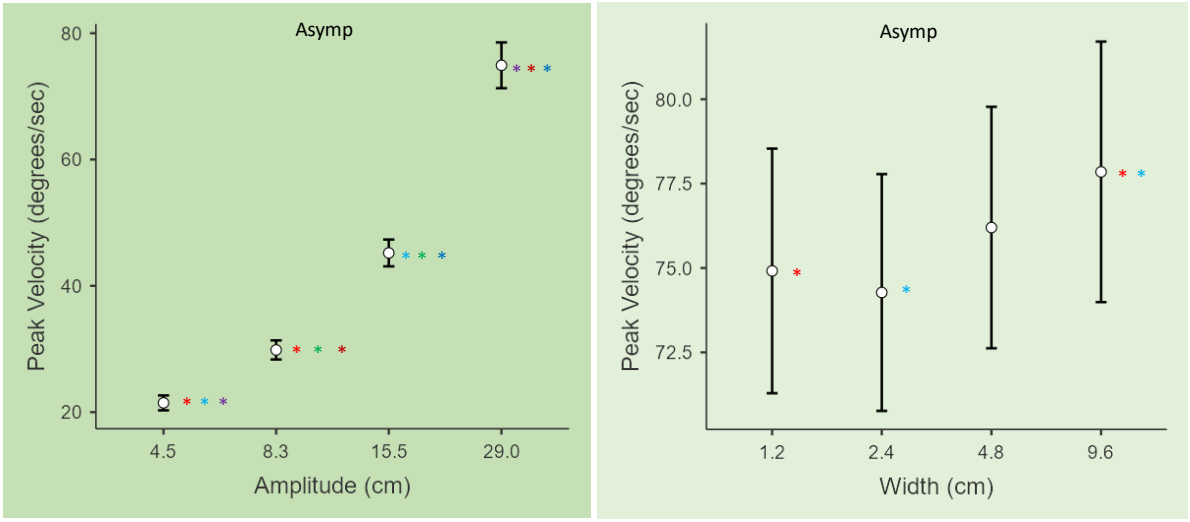


Figure 46. Main Effects of Amplitude & Width on Asymptomatic Head Peak Velocity

Within-Group Analysis – Asymptomatic

The participants' approach was to rotate their head faster towards the target over a larger amplitude. There was a significant main effect on PV for the four target amplitudes at the common target width of 1.2 cm ($F_{(3,57)} = 372.58$; $p < 0.001$; $\eta^2_p = 0.951$)(Fig. 46). Additionally, post-hoc testing noted that PV increased with larger amplitudes during all amplitude comparisons ($p < 0.001$). There were no significant main effects on PV for side ($F_{(1,19)} = 0.716$; $p= 0.408$) and SM ($F_{(1,19)} = 4.25$; $p= 0.183$). There were no significant differences for the amplitude x side, amplitude x SM, side x SM, and amplitude x side x SM interactions.

There was a significant main effect for the four target widths at the common target amplitude of 29 cm on PV ($F_{(3,57)} = 8.44$; $p < 0.001$; $\eta^2_p = 0.308$) (Fig. 46). Pairwise comparisons noted a significant increase in PV as target widths increased from 1.2 to 9.6 cm ($p = 0.012$) and 2.4 to 9.6 cm ($p = 0.001$). There were no significant main effects on PV for side ($F_{(1,19)} = 1.17$; $p= 0.294$) and SM ($F_{(1,19)} = 0.067$; $p= 0.798$). There were no significant differences for the amplitude x side, amplitude x SM, side x SM, and amplitude x side x SM interactions.

In summary, head movement PV was not altered in either group following SM. There was a significant increase in PV with increasing movement amplitudes within both the symptomatic and asymptomatic groups. There was also a significant increase in PV during the width condition within both the symptomatic and asymptomatic groups; however, the increase in PV was only significant toward the largest target width.

Time to Peak Velocity (ttPV)

Between-Group Analysis

Table 18. Descriptive statistics for head ttPV (sec).

	Group	Pre-SM	Post-SM
N	Asymp	20	20
	Symp	20	20
Missing	Asymp	0	0
	Symp	0	0

Table 18. Descriptive statistics for head ttPV (sec).

	Group	Pre-SM	Post-SM
Mean	Asymp	0.262	0.244
	Symp	0.245	0.227
Median	Asymp	0.261	0.242
	Symp	0.233	0.226
Standard deviation	Asymp	0.0681	0.0451
	Symp	0.0515	0.0421
Minimum	Asymp	0.185	0.180
	Symp	0.156	0.160
Maximum	Asymp	0.457	0.318
	Symp	0.335	0.321

There was a significant main effect of SM on ttPV ($F_{(1,38)} = 12.30$; $p < 0.001$; $\eta^2_p = 0.244$)(Fig. 47) indicating that ttPV was reduced following SM. There was no significant main effect of Group ($F_{(1,38)} = 1.17$; $p = 0.287$; $\eta^2_p = 0.030$) on ttPV and there was no significant SM x Group interaction ($F_{(1,38)} = 0.059$; $p = 0.981$; $\eta^2_p = 0.000$).

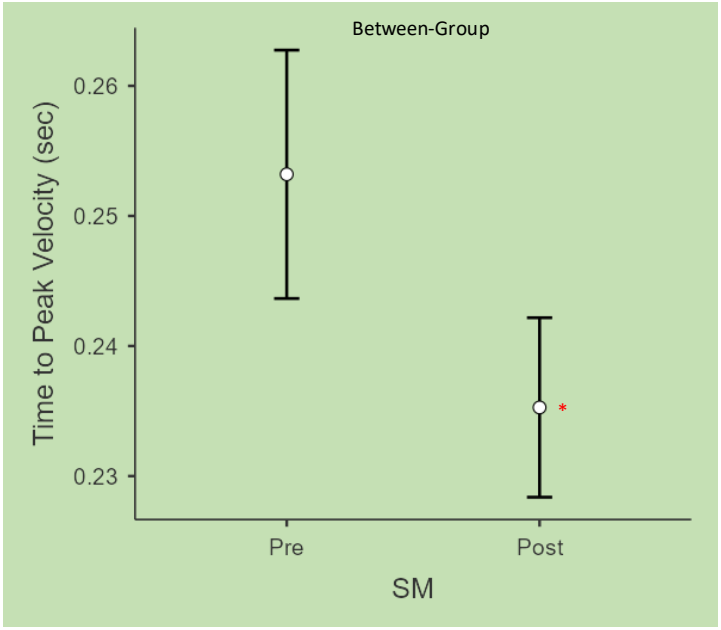


Figure 47. Main Effect of SM on Time to Peak Velocity for Between-Group Analysis

Within-Group Analysis – Symptomatic

The symptomatic participants' head ttPV increased as movement amplitudes became larger. There was a significant main effect for amplitude on ttPV ($F_{(3,57)} = 2.74$; $p = 0.052$; $\eta^2_p = 0.126$); however, there were no significant pairwise post-hoc comparisons (Fig. 48). There were no significant main effects for SM or side on ttPV. There were no significant differences for the amplitude x side, amplitude x SM, side x SM, and amplitude x side x SM interactions.

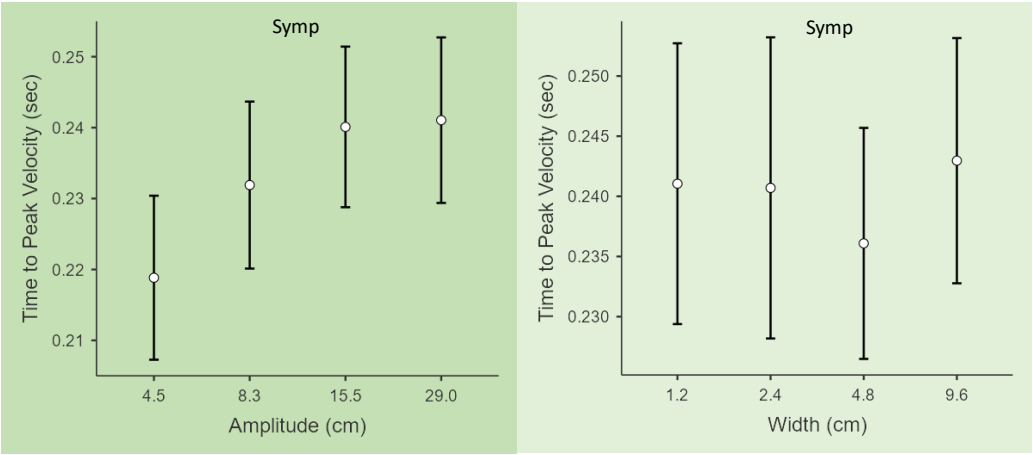


Figure 48. Main Effects of Amplitude & Width *on Symptomatic Head Time to Peak Velocity*

There was a significant main effect of SM for target width at the common amplitude of 29.0 cm on ttPV ($F_{(1,19)} = 8.07$; $p = 0.010$; $\eta^2_p = 0.298$) (Fig. 49), whereby ttPV decreased following SM. There were no significant main effects on ttPV for side of horizontal head movement or target width ($F_{(3,57)} = 0.857$; $p = 0.469$; $\eta^2_p = 0.043$) (Fig. 48). There were no significant side x width, side x SM, width x SM, and side x SM x width interactions.

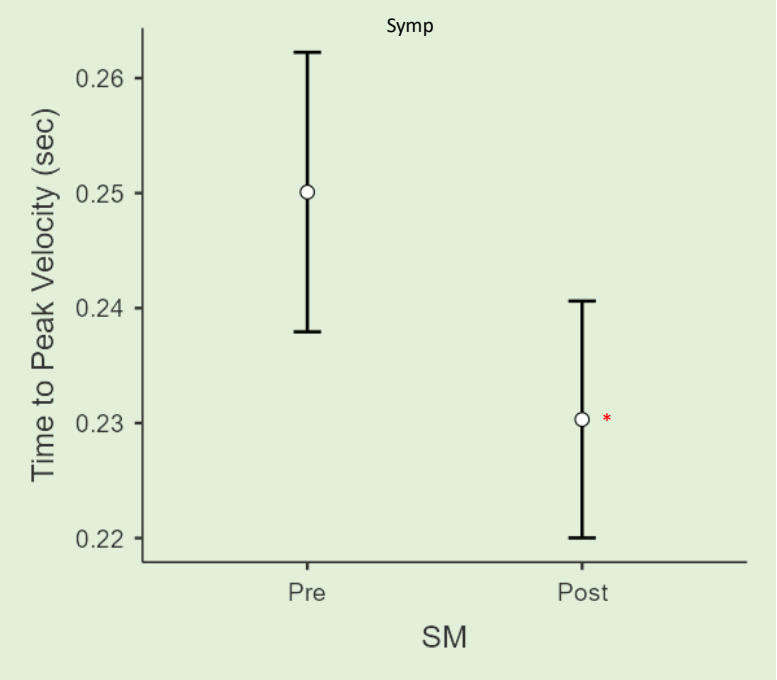


Figure 49. Main Effect of SM on Symptomatic Head ttPV (Width Condition)

Within-Group Analysis – Asymptomatic

The asymptomatic participants took longer to reach PV as target amplitudes increased. There was a significant main effect of the four target amplitudes at the common target width of 1.2 cm on ttPV ($F_{(3,57)} = 15.96$; $p < 0.001$; $\eta^2_p = 0.457$) (Fig. 50). Post-hoc testing noted that ttPV increased with larger amplitudes and significant differences were found when 4.5cm was compared to 15.5 cm ($p = 0.002$) and 29.0 cm ($p < 0.001$), and 8.3 cm was compared to the 15.5 cm ($p = 0.024$) and 29.0 cm ($p = 0.001$). There were not any differences between the 4.5 and 8.3 cm amplitudes, and the 15.5 and 29.0 cm amplitudes for ttPV. There was a significant main effect of SM on ttPV ($F_{(3,57)} = 5.55$; $p = 0.029$; $\eta^2_p = 0.226$) indicating that ttPV decreased following SM (Fig. 51). There was no significant main effect on ttPV for side ($F_{(1,19)} = 3.39$; $p= 0.081$). There were no significant differences for the amplitude x side, amplitude x SM, side x SM, and amplitude x side x SM interactions.

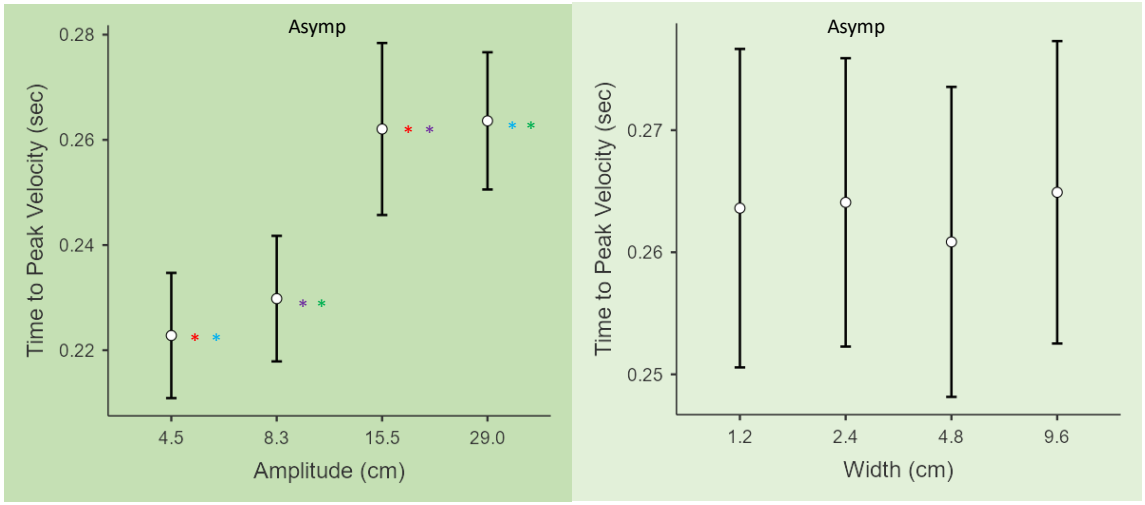


Figure 50. Main Effects of Amplitude & Width *on Asymptomatic Head Time to Peak Velocity*

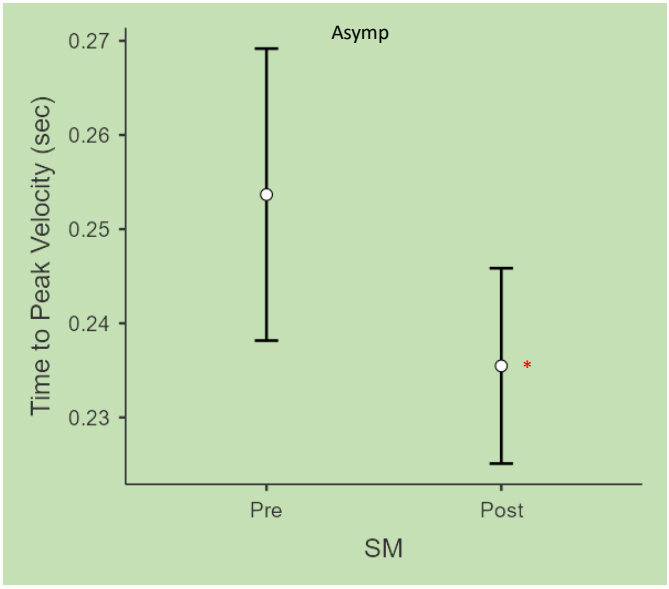


Figure 51. Main Effect of SM on Asymptomatic Head ttPV for Amplitude Condition

There was no significant main effect on ttPV for target width at the common target amplitude of 29.0 cm ($F_{(3,57)} = 0.335$; $p = 0.800$; $\eta^2_p = 0.017$) (Fig. 50). There were also no significant main effects of SM or side on ttPV. There were no significant side x width, side x SM, SM x width, and side x width x SM interactions.

In summary, there was a significant reduction in ttPV following SM noted during the between-group analysis. There was a significant main effect of SM on ttPV for the width

condition within the symptomatic group and for the amplitude conditions within the asymptomatic group.

Peak Acceleration (PA)

Between-Group Analysis

Table 19. Descriptive statistics for head PA (degrees/s^2).

	Group	Pre-SM	Post-SM
N	Asymp	20	20
	Symp	20	20
Missing	Asymp	0	0
	Symp	0	0
Mean	Asymp	444	439
	Symp	497	519
Median	Asymp	403	417
	Symp	471	473
Standard deviation	Asymp	140	122
	Symp	184	170
Minimum	Asymp	264	266
	Symp	267	272
Maximum	Asymp	710	667
	Symp	1054	1020

There were no significant main effects of SM ($F_{(1,38)} = 0.588$; $p = 0.448$; $\eta^2_p = 0.015$) or Group ($F_{(1,38)} = 1.910$; $p = 0.175$; $\eta^2_p = 0.048$) on PA. There was no significant SM x Group interaction ($F_{(1,38)} = 1.491$; $p = 0.230$; $\eta^2_p = 0.038$).

Within-Group Analysis – Symptomatic

There was a significant main effect of amplitude at the common target width of 1.2 cm on PA ($F_{(3,57)} = 101.52$; $p < 0.001$; $\eta^2_p = 0.842$)(Fig. 52). Additionally, post-hoc testing noted that PA increased with larger amplitudes during all amplitude comparisons ($p < 0.001$). There were no significant main effects on PA for side ($F_{(1,19)} = 0.152$; $p= 0.695$) and SM ($F_{(1,19)} = 0.728$; p=

0.404). There were no significant differences for the amplitude x side, amplitude x SM, side x SM, and amplitude x side x SM interactions.

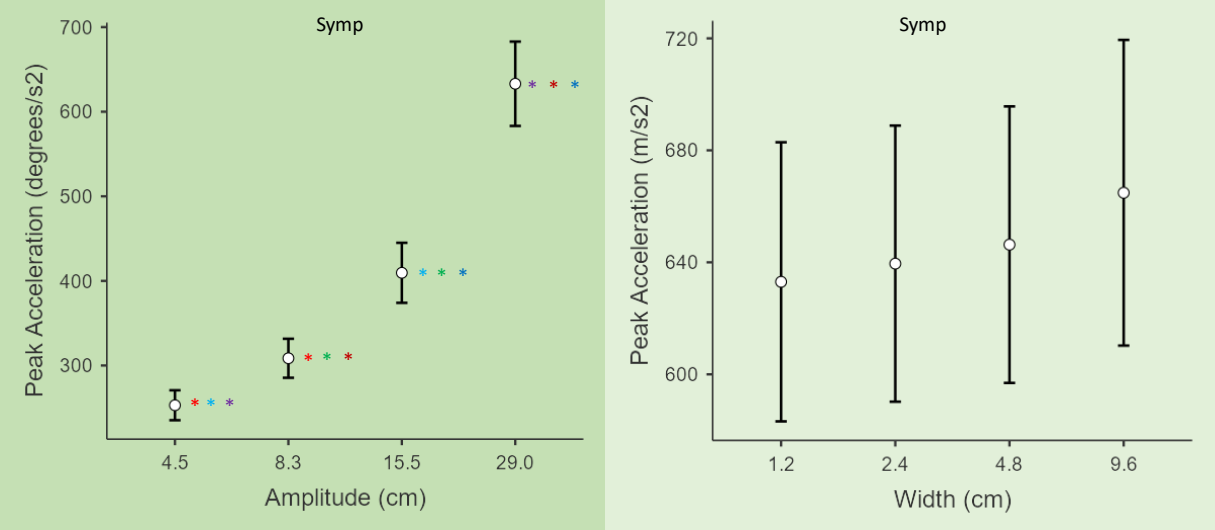


Figure 52. Main Effects of Amplitude & Width on Symptomatic Head Peak Acceleration

Symptomatic group head PA was not altered by target width, the side of rotational head movement, or following SM. There was no significant main effect of target width at the common target amplitude of 29 cm on PA ($F_{(3,57)} = 2.21$; $p = 0.097$) (Fig. 52). There were also no significant main effects for side ($F_{(1,19)} = 0.003$; $p= 0.958$) and SM ($F_{(1,19)} = 2.42$; $p= 0.136$) on PA. There were no significant differences for the amplitude x side, amplitude x SM, side x SM, and amplitude x side x SM interactions.

Within-Group Analysis – Asymptomatic

There was a significant main effect on PA for the four target amplitudes at the common target width of 1.2 cm ($F_{(3,57)} = 129.83$; $p < 0.001$; $\eta^2_p = 0.872$)(Fig. 53). Specifically, post-hoc testing noted that PA increased with larger amplitudes during all amplitude comparisons ($p < 0.001$). There were no significant main effects on PA for side ($F_{(1,19)} = 0.798$; $p= 0.383$) and SM ($F_{(1,19)} = 0.453$; $p= 0.509$). There were no significant differences for the amplitude x side, amplitude x SM, side x SM, and amplitude x side x SM interactions.

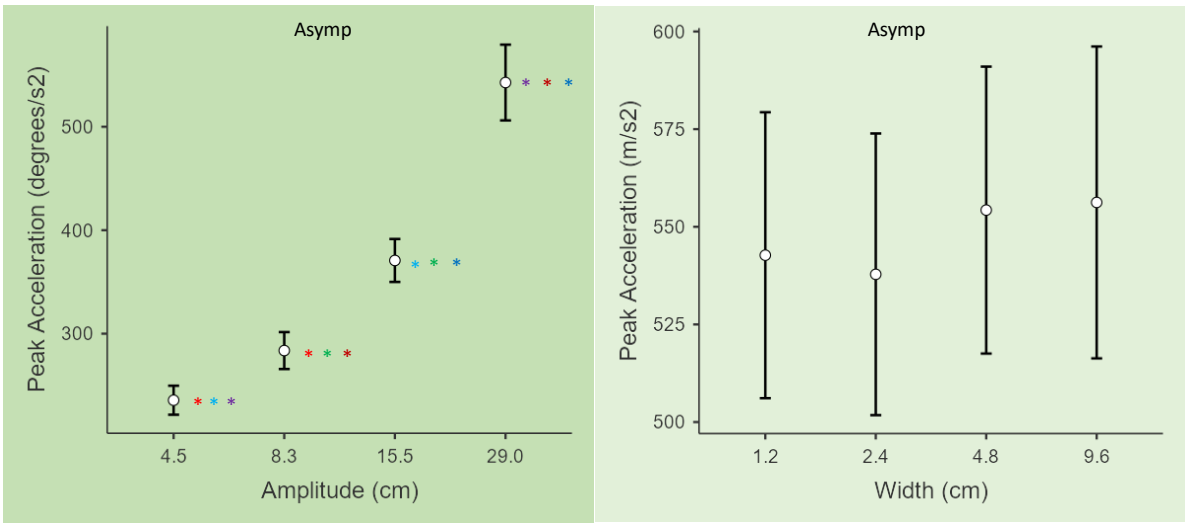


Figure 53. Main Effects of Amplitude & Width *on Asymptomatic Head Peak Acceleration*

Asymptomatic group head PA was not altered by target width, the side of rotational head movement, or following SM. There was no significant main effect on PA for the four target widths at the common target amplitude of 29 cm ($F_{(3,57)} = 2.18$; $p = 0.100$; $\eta^2_p = 0.103$) (Fig. 53). There were also no significant main effects of PA for side ($F_{(1,19)} = 0.24$; $p= 0.879$) and SM ($F_{(1,19)} = 0.100$; $p= 0.755$). There were no significant differences for the amplitude x side, amplitude x SM, side x SM, and amplitude x side x SM interactions.

In summary, PA during rotational head movements was not altered following SM in either experimental group. There was a significant main effect of amplitude on PA within the symptomatic and asymptomatic groups such that PA increase with larger movement amplitudes.

Time to Peak Acceleration (ttPA)

Between-Group Analysis

Table 20. Descriptive statistics for head ttPA (sec).

	Group	Pre-SM	Post-SM
N	Asymp	20	20
	Symp	20	20
Missing	Asymp	0	0
	Symp	0	0
Mean	Asymp	0.168	0.152
	Symp	0.162	0.136
Median	Asymp	0.158	0.132
	Symp	0.145	0.124
Standard deviation	Asymp	0.0825	0.0561
	Symp	0.0712	0.0551
Minimum	Asymp	0.0790	0.0860
	Symp	0.0900	0.0830
Maximum	Asymp	0.441	0.298
	Symp	0.388	0.333

There was a significant main effect of SM on ttPA ($F_{(1,38)} = 6.954$; $p = 0.012$; $\eta^2_p = 0.155$)(Fig. 54) indicating a reduction in ttPA following SM. There was no significant main effect of Group ($F_{(1,38)} = 0.308$; $p = 0.582$; $\eta^2_p = 0.008$) on ttPV and there was no significant SM x Group interaction ($F_{(1,38)} = 0.051$; $p = 0.537$; $\eta^2_p = 0.010$).

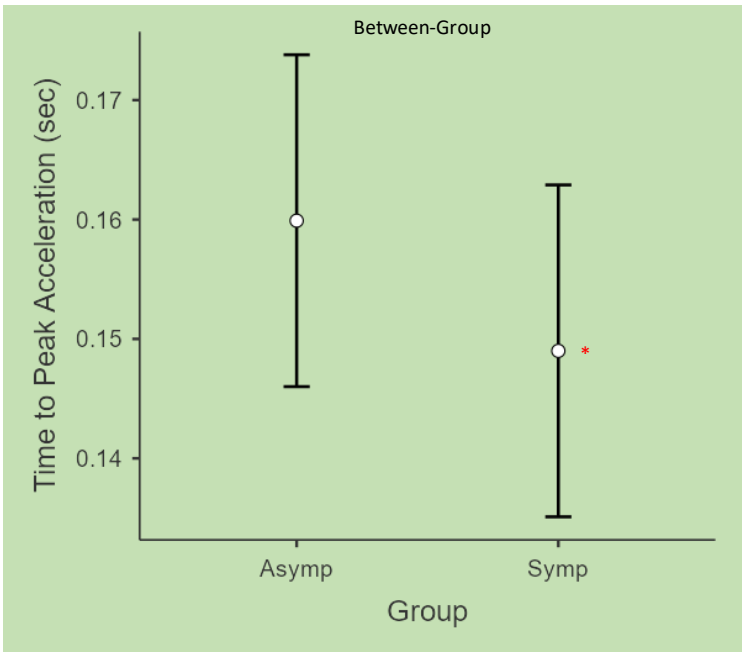


Figure 54. Main Effect of SM on Peak Acceleration for Between-Group Analysis

Within-Group Analysis – Symptomatic

There was a significant main effect for target amplitude at the common target width of 1.2 cm on ttPA ($F_{(3,57)} = 11.06$; $p < 0.001$; $\eta^2_p = 0.368$) (Fig. 55). Pairwise comparisons noted that ttPA decreased with larger amplitudes and significant differences were found when 4.5cm was compared to 15.5 cm ($p = 0.011$) and 29.0 cm ($p = 0.003$), and when 8.3 cm was compared to 15.5 cm ($p = 0.016$) and 29.0 cm ($p = 0.006$). There were not any differences between the 4.5 cm and 8.3 cm amplitudes, and the 15.5 cm to 29.0 cm amplitudes for ttPA. There were no significant main effects on ttPA for side ($F_{(1,19)} = 0.003$; $p = 0.954$) or SM ($F_{(1,19)} = 3.85$; $p = 0.064$). There were no significant differences for the amplitude x side, amplitude x SM, side x SM, and amplitude x side x SM interactions.

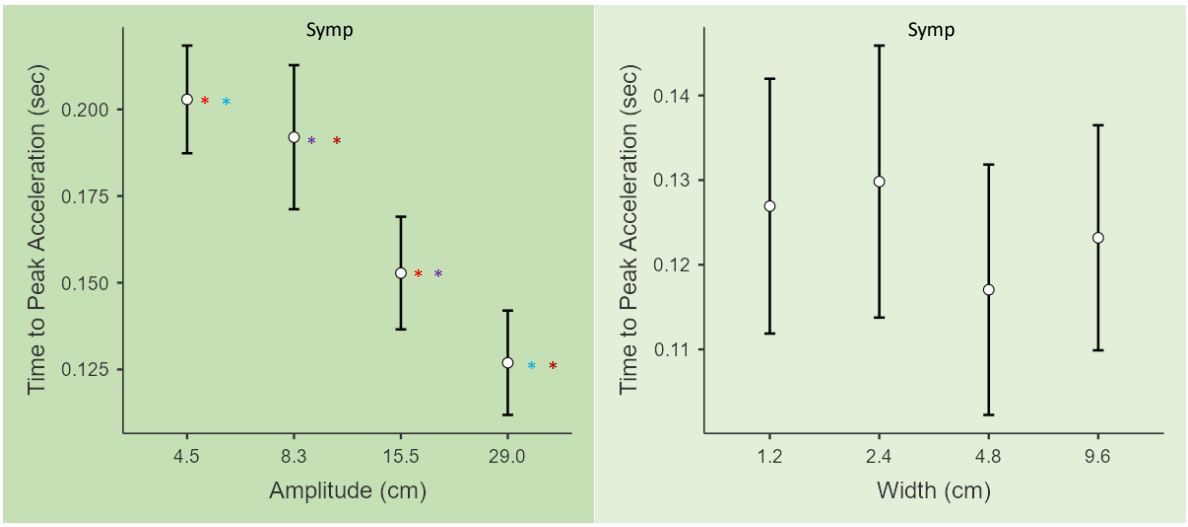


Figure 55. Main Effects of Amplitude & Width on Symptomatic Head Time to Peak Acceleration

There was a significant main effect of SM on ttPA for the width condition at the common target amplitude of 29.0 cm ($F_{(1,19)} = 16.86$; $p < 0.001$; $\eta^2_p = 0.470$)(Fig. 56) whereby ttPA decreased following SM. There were no significant main effects on ttPA for side ($F_{(1,19)} = 0.499$; $p = 0.489$) and target width ($F_{(3,54)} = 1.23$; $p = 0.309$) (Fig. 55). There were no significant side x SM, side x width, SM x width, or side x SM x width interactions.

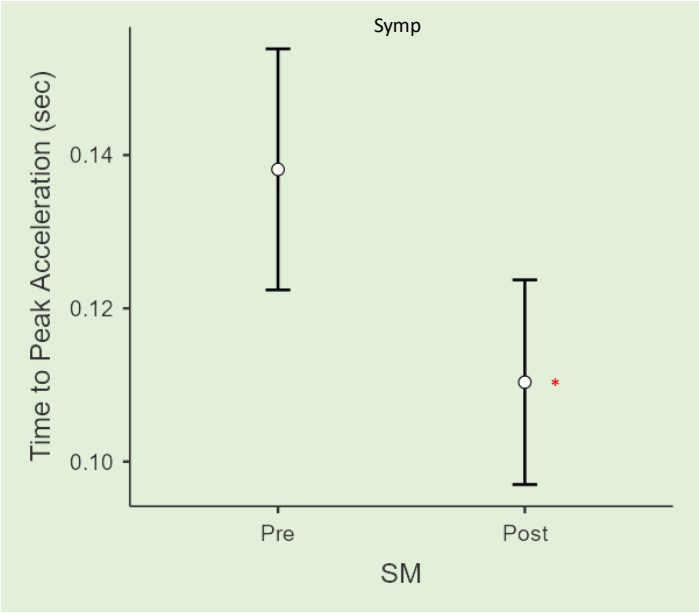


Figure 56. Main Effect of SM on Symptomatic Head ttPA for Width Condition

There was a significant SM x side interaction on ttPA within the symptomatic group for the width condition ($F_{(1,19)} = 6.35$; $p = 0.021$; $\eta^2_p = 0.250$)(Fig 57). Head PA was reached in a shorter time following SM for both right and left rotational head movements.

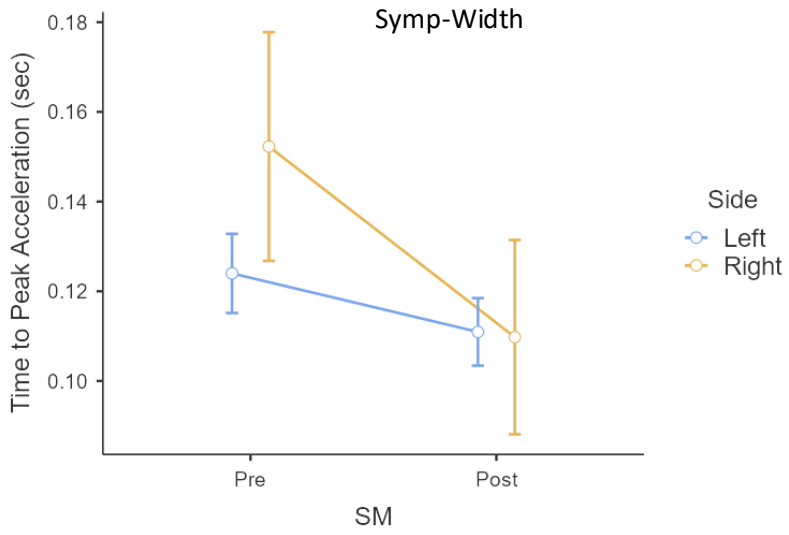


Figure 57. SM x Side Interaction on ttPV within Symptomatic Group for Width Condition

Within-Group Analysis – Asymptomatic

There was a significant main effect on ttPA for the four target amplitudes at the common target width of 1.2 cm ($F_{(3,57)} = 10.25$; $p < 0.001$; $\eta^2_p = 0.350$) (Fig. 58). Post-hoc testing noted that ttPA decreased with larger movement amplitudes. Significant differences were found when 4.5cm was compared to 8.3 cm ($p = 0.036$) and 29.0 cm ($p < 0.001$) and 8.3 cm was compared to 29.0 cm ($p = 0.004$). There was also a significant difference when the 15.5 cm amplitude was compared to the 29.0 cm amplitude ($p = 0.038$). There were not any pairwise differences between the 4.5 and 15.5 cm amplitudes and the 8.3 and 15.5 cm amplitudes for ttPA. There were no significant main effects on ttPA for side ($F_{(1,19)} = 2.74$; $p= 0.114$) and SM ($F_{(1,19)} = 1.18$; $p= 0.292$). There were no significant differences for the amplitude x side, amplitude x SM, side x SM, and amplitude x side x SM interactions.

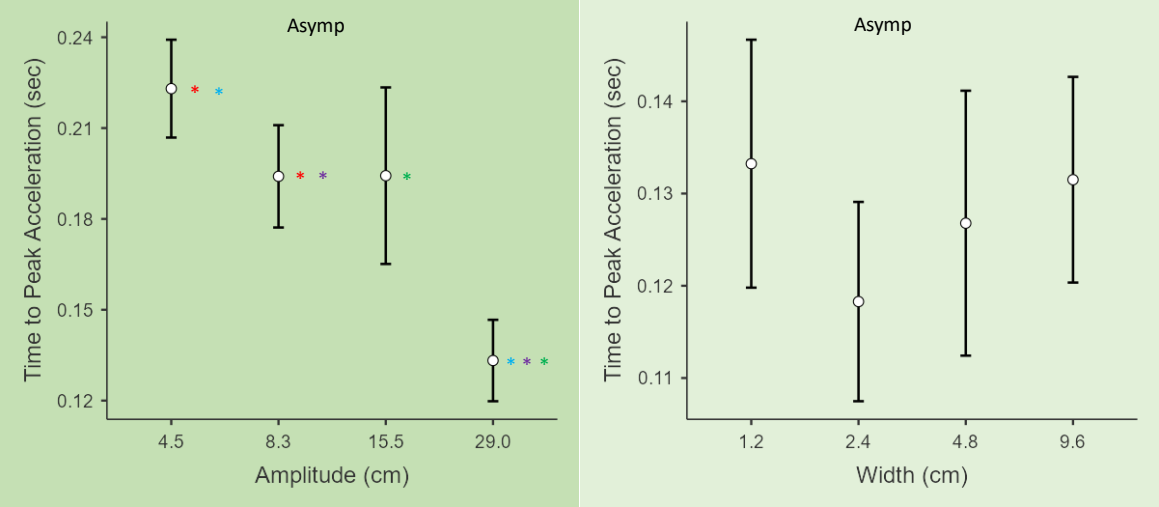


Figure 58. Main Effects of Amplitude & Width on Asymptomatic Head Time to Peak Acceleration

Head movement ttPA was not affected by target width, the direction of rotational head movement, or following SM. There was no significant main effect of target width on ttPA at the common target amplitude of 29.0 cm ($F_{(3,57)} = 2.15$; $p = 0.103$; $\eta^2_p = 0.102$) (Fig. 58). There were no significant main effects of SM ($F_{(1,19)} = 3.43$; $p = 0.080$) or side ($F_{(1,19)} = 0.900$; $p= 0.355$) on ttPA. There were no significant side x width, side x SM, SM x width, and side x width x SM interactions.

In summary, there was a significant effect of SM reducing ttPA but no effect of Group on ttPA. There was a significant main effect of amplitude on ttPA within the symptomatic and asymptomatic groups. There was no significant main effect of width within the symptomatic and asymptomatic groups. Lastly, there was a significant main effect of SM on ttPA during the width condition within the symptomatic group.

Between-Group Analysis for Eye and Head Movement Times

An additional purpose of this study was to determine the difference in MTs with increasing IDs during specific amplitude and width conditions for task-oriented eye and head movements in the same study population. The objective for this analysis was to determine if the saccade and head MT/ID slopes would be different for the amplitude condition compared to the width condition within symptomatic and asymptomatic groups.

There were significant main effects of Fitts Parameter ($F_{(1,38)} = 663.82$; $p < 0.001$; $\eta^2_p = 0.946$) and ID ($F_{(2,76)} = 270.43$; $p < 0.001$; $\eta^2_p = 0.877$) on saccade MT. The mean value for

saccade MT was shorter for the amplitude condition than for the width condition and saccade MT increased linearly with increasing IDs within both the symptomatic and asymptomatic groups (Fig. 59). There was also a significant Fitts Parameter x ID interaction on saccade MT ($F_{(2,76)} = 120.73$; $p < 0.001$; $\eta^2_p = 0.761$) (Fig. 59). Pairwise comparisons note a significant increase ($p < 0.001$) in saccade MT during the amplitude condition between all IDs; however, there was no significant increase in saccade MT during the width condition with increasing IDs.

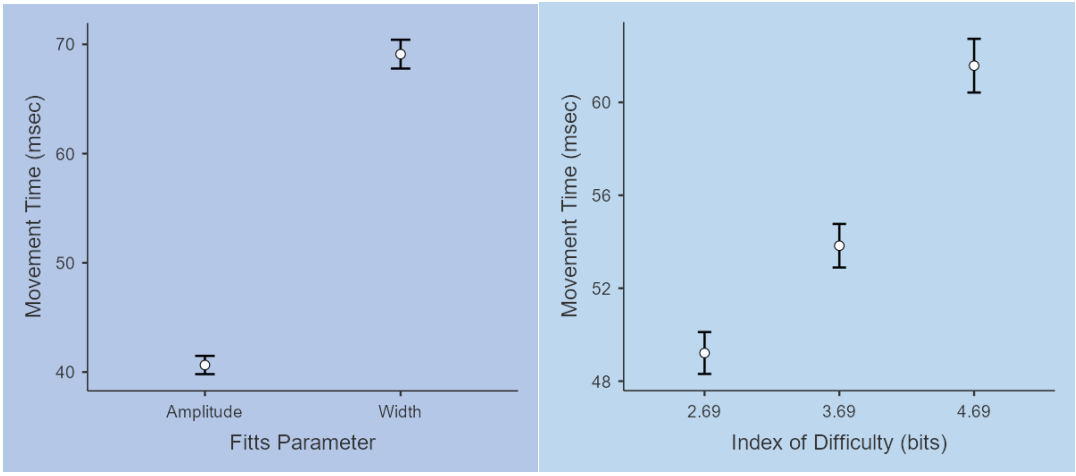


Figure 59. Main Effects of Fitts Parameter and Index of Difficulty on Saccade Movement Time

Figure 60 presents saccade MT as a function of ID for the amplitude and width conditions for the symptomatic and asymptomatic groups respectively. The solid amplitude condition and width condition lines and the associated linear regression equations are also presented in the graph. This figure demonstrates robust linear amplitude increases as a function of increasing ID (Symp: R^2=0.981, slope=13.62 msec; Asymp: R^2=0.962, slope=14.50 msec). Whereas saccade MTs for the width condition, while still demonstrating robust linear increases, did not reliably vary with ID (Symp: R^2=0.955, slope=0.79 sec; Asymp: R^2=0.891, slope=1.64 sec).

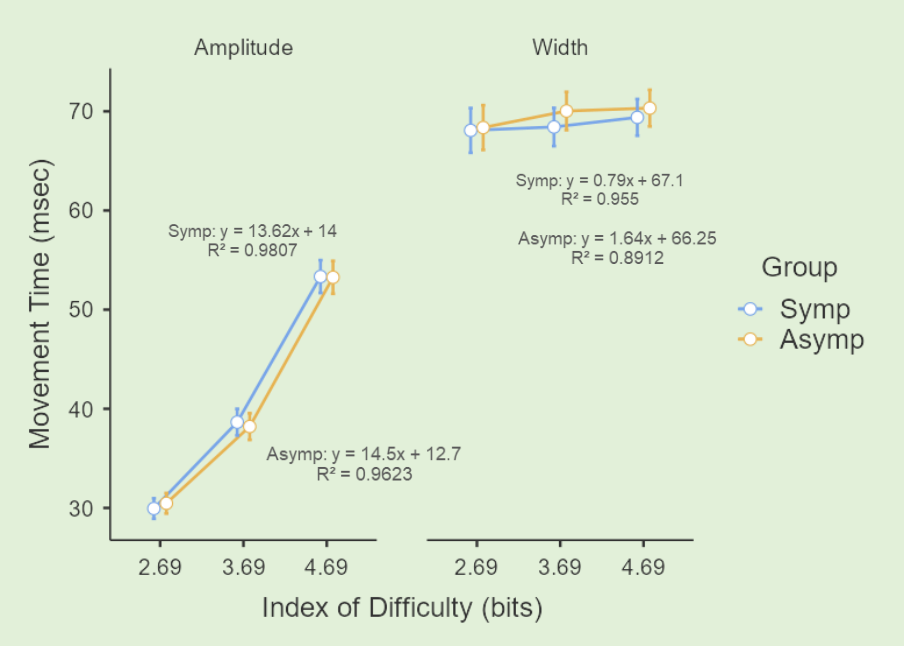


Figure 60. Fitts Parameter x Index of Difficulty Interaction on Saccade Movement Time

There were significant main effects of Fitts Parameter ($F_{(1,38)} = 20.07$; $p < 0.001$; $\eta^2_p = 0.346$) and ID ($F_{(2,76)} = 119.72$; $p < 0.001$; $\eta^2_p = 0.759$) on head MT. The mean value for head MT was shorter for the amplitude condition than for the width condition and head MT increased with increasing IDs within both the symptomatic and asymptomatic groups (Fig. 61). There was also a significant Fitts Parameter x ID interaction on head MT ($F_{(2,76)} = 3.27$; $p = 0.043$; $\eta^2_p = 0.079$)(Fig.61). Pairwise comparisons note a significant increase ($p < 0.001$) in head MT during the amplitude condition between all IDs. There was also significant increase in MT during the width condition with increasing IDs between the: 2.60 bit and 3.60 bit ($p < 0.001$), 2.60 bit and 4.60 bit ($p < 0.001$), and 3.60 bit and 4.60 bit ($p = 0.003$) IDs.

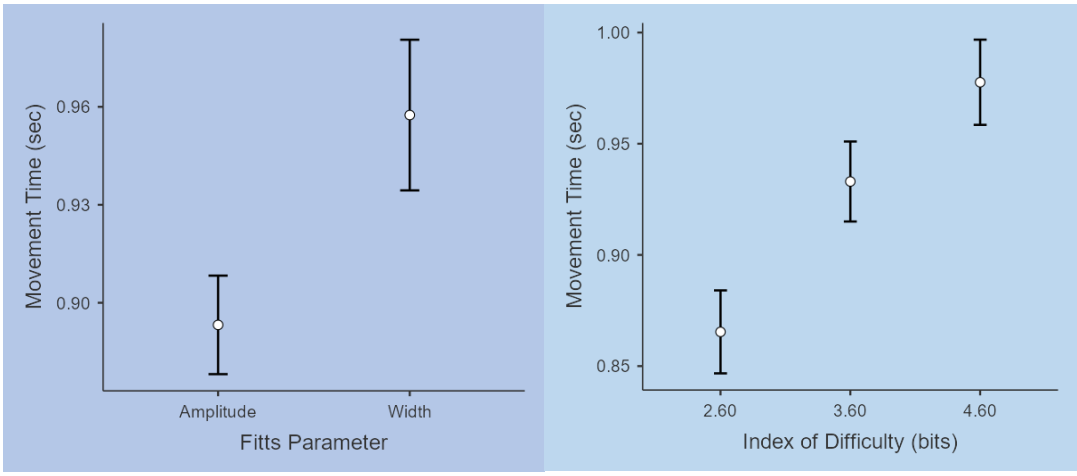


Figure 61. Main Effects of Fitts Parameter and Index of Difficulty on Head Movement Time

Figure 62 presents head MT as a function of ID for the amplitude and width conditions for the symptomatic and asymptomatic groups respectively. The solid amplitude condition and width condition lines and the associated linear regression equations are also presented in the graph. This figure demonstrates that amplitude increased linearly as a function of increasing ID (Symp: R^2=0.998, slope=0.06 sec; Asymp: R^2=0.974, slope=0.07 sec). MTs for the width condition also increased linearly as a function of ID (Symp: R^2=0.888, slope=0.04 sec; Asymp: R^2=0.978, slope=0.03 sec).

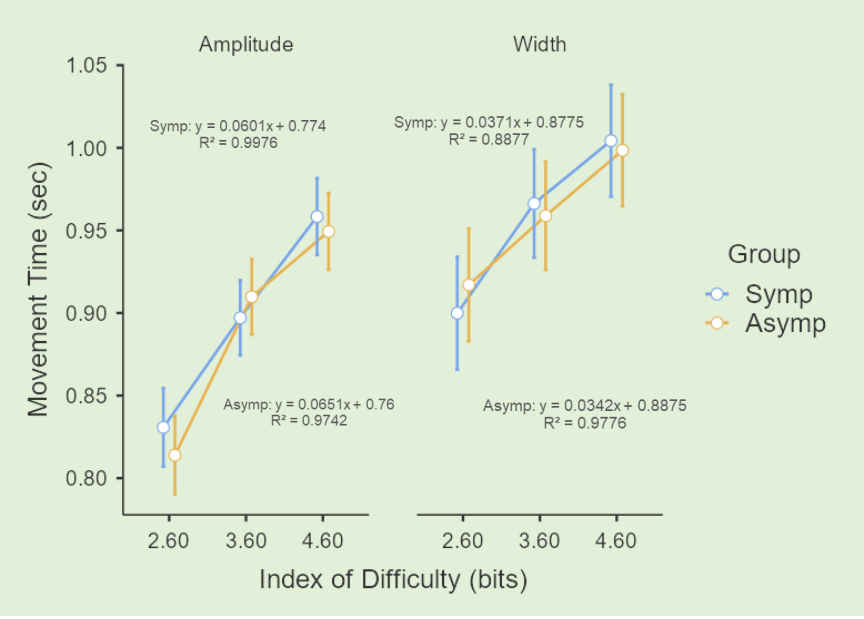


Figure 62. Fitts Parameter x Index of Difficulty Interaction on Head Movement Time

In summary, the average slopes of MT for the amplitude condition were of a steeper gradient than the width condition (asymmetric) for the symptomatic and asymptomatic groups during the eye movement task. However, the MT slopes were similar (symmetric) for the amplitude and width conditions during the head movement task for the symptomatic and asymptomatic groups.

Discussion

The purpose of this study was to examine the effect of manipulation of the cervical spine on eye and head movement tasks in a population of chronic neck pain participants who were symptomatic or asymptomatic at the time of the study.

The results of this study determined that:

1. The time needed to complete an eye movement task was not influenced by cervical spine manipulation.
2. The time needed to complete a task-oriented eye movement was no different in symptomatic participants compared to the asymptomatic participants.
3. The time needed to complete a head movement task was influenced by cervical spine manipulation within the symptomatic group.
4. The time needed to complete task-oriented head movements was reduced within the symptomatic group for both the amplitude and width conditions.

Eye Movement Fitts' Task

Saccades have been described as inherently ballistic eye movements that allow the individual to gather visual information from their surrounding environment. The initial phase of the saccade appears to be ballistic, and any movement errors are corrected through additional secondary saccades. (Al-Aidroos et al., 2008; Heath et al., 2016; Leigh & Zee, 2015). Recent research, however, contradicts that the saccade is ballistic because it can be modified by the CNS to respond to visual information while the eye is decelerating toward the end of the saccade. It is suggested that saccades only appear ballistic due to their high velocities and short durations (Leigh & Zee, 2015).

The utilization of an eye-tracker device to measure saccades is a commonly used method to record eye movements that follow a target. Other eye-tracking devices such as electro-oculography are prone to artifact contamination due to electrode placement that can result in the capture of poor-quality data (Gadotti et al., 2016). This thesis study used a high-speed fiber optic eye-tracking device that provides accurate eye movement metrics. Eye-tracker devices have been frequently utilized to capture the eye movements during a Fitts' task (Al-Aidroos et al., 2008; Chi & Lin, 1997; Heath et al., 2016; Wu et al., 2010).

Altered behavioral or kinematic performance of the saccade following SM could be related to a neurophysiologic mechanism (Leigh & Zee, 2015). Changes in oculomotor control related to aberrant afferent input from dysfunctional cervical spine mechanoreceptors (Gimse et al., 1996) is related to alterations in sensorimotor integration (Cade et al., 2021; Treleaven, 2017) and alterations in COR gain (Treleaven et al., 2011). The reflex pathway of the cervico-ocular reflex (COR) in part is derived from cervical afferents that project to the superior colliculus which serves as a reflex center between the visual system and neck movements and is therefore responsible for eye-head coordination (Treleaven et al., 2011). Alterations in the COR have been reported in patients with mechanical neck pain (Treleaven, 2017). The COR and sensorimotor integration provide an integrated neural circuitry for horizontal saccades (Gimse et al., 1996; Treleaven et al., 2011) and may offer an explanation as to how saccadic MT is increased in patients with neck pain. It may also explain how SM, when applied to dysfunctional cervical joints, could alter oculomotor function (Lelic et al., 2016; Palmgren et al., 2006) and hence decrease saccade MT. This is a theoretical proposition and does not imply that changes to saccade MT following SM are exclusively a neurophysiologic mechanism.

Movement Time (MT)

Between-Group

Based on a search of the literature, no other study has been found to have reported on the effect of SM on saccade MT. It has been theorized that a modulatory effect of SM on a behavioral measure, such as saccadic MT, would suggest the mechanism of SM is neurophysiologic in nature (Passmore et al., 2010). Since saccades are controlled by the CNS, a neurophysiologic mechanism would be required to alter saccade movement. It has been reported that SM alters sensorimotor integration (Haavik & Murphy, 2012) and that SM can also have an influence on specific areas of the brain that are associated with eye movement control (Lelic et al., 2016). Given these relevant research findings, it is plausible that SM could alter eye movement in symptomatic and asymptomatic chronic neck pain participants.

The results of the between-group analysis of this thesis study demonstrate no significant main effect of SM on saccade MT. Based on the aforementioned studies, it was unexpected that SM did not influence saccade MT. This thesis study used a counter-balanced order for participants performing the eye and head Fitts' task before and after SM. As a result, several

participants would have completed the head movement Fitts' task prior to the eye movement Fitts' task after undergoing SM. It was initially thought that the time elapsed between the manipulation and the performance of the eye task for some participants in this study may have allowed the effects of SM on cortical changes, and hence oculomotor control, to diminish so that no significant changes were seen in saccade MT. However, a posteriori statistical analysis was conducted to compare those participants who underwent the eye movement Fitts' task immediately following SM to those participants that underwent the head movement Fitts' task following SM. There was no difference in saccade MT in participants who underwent the eye movement or head movement Fitts' tasks immediately following SM in either the symptomatic or asymptomatic groups.

A plausible reason that SM may have had no effect on saccade MT was that the participant was in a relatively neutral position while their head was fixed by a chin and head rest during the eye movement Fitts' task. This neutral head position may have served as a passive intervention (similar to cervical traction) to reduce any influence the dysfunctional cervical restrictions could have on the cortical changes that modulate oculomotor control, and any subsequent changes to saccade MT or other saccade dependent variables. This is a reasonable postulation as changes in oculomotor control related to aberrant afferent input from dysfunctional cervical spine biomechanics could be related to alterations in sensorimotor integration (Cade et al., 2021).

Lastly, SM may have had no effect on eye movements despite the numerous potential mechanisms by which SM may impact saccades. In particular, none of the participants in this thesis study had any symptoms related to COR deficits, which might also why SM had no effect on saccade MT.

Within-Group

The results of this thesis study are consistent with Heath et al. (2016) and demonstrate that the amplitude condition produces robust relationships to saccade MT within both groups (Figs. 3 and 4), as compared to the width condition which had less robust yet significant effects on MT in the asymptomatic (Fig. 4) but not the symptomatic group (Fig. 3). Studies have reported that eye movement amplitude almost entirely determines saccade MT. Larger amplitudes take longer to complete and therefore movement amplitude can challenge saccade processing demands (Al-Aidroos et al., 2008; Heath et al., 2016). The performance of saccade

sequences supports the speed-accuracy trade-off whereby larger target amplitudes and smaller target widths have a longer MT (Wu et al., 2010). It has been reported that saccade movement amplitude imposed greater information processing demands than target width and that saccade MT was greatly influenced by amplitude, rather than by target width. This discrepancy indicates that the movement amplitude and target width components of Fitts' Law are indeed separable and not interdependent (Heath et al., 2016).

There was also no main effect of SM on saccade MT for the symptomatic or asymptomatic groups during either the amplitude or width condition. This may again be related to the neutral head position of the participants during the eye movement Fitts' task.

Peak Velocity (PV), Time to Peak Velocity (ttPV), Peak Acceleration (PA), and Time to Peak Acceleration (ttPA)

Between-Group

This study observed no significant main effect difference of SM on PV and PA between the symptomatic and asymptomatic groups. There was a significant SM x Group interaction on ttPV; however there were no significant pairwise comparisons on post-hoc testing indicating this to be a weak interaction and possibly a type I error. There was also no change in ttPA following SM in either of our study groups.

In summary, SM had no effect on the kinematic measures related to eye movements between the symptomatic and asymptomatic groups. There was also no effect of SM on saccade MT from the between-group analysis. Movement time and these kinematic measures are all inter-related outcomes since they are derived from the same source and should generally move in the same direction. These results are contrary to this study's hypothesis that SM would influence eye MT and by extension SM would also influence these kinematic outcome measures.

Within-Group

Peak velocity increased within both the symptomatic and asymptomatic groups as movement amplitudes became larger (Figs. 11 and 13) and this is consistent with the assumption that saccade peak velocities will increase with larger saccade amplitudes (Jóhannesson Ó, Edelman, Sigurþórsson, & Kristjánsson, 2018). This effect of movement amplitude on PV agrees with discrete aiming such as in a Fitts' task; to get as close to the target as quickly as possible.

As MT increases with longer movement amplitudes there may be a corresponding increase in PV. The increase in MT and PV of the saccade with increasing movement amplitude has been termed the "main sequence" (Harris & Wolpert, 2006). Therefore, for the participants to reach the target as quickly as possible, but still exhibit a longer MT, would suggest they spend more time reaching PV so as to maintain accuracy and reduce the magnitude of a late movement correction. This assertion is supported by this thesis study data that demonstrates ttPV increases as movement amplitudes became larger and therefore saccades reach PV over a longer period for the larger amplitudes, which supports the speed-accuracy trade-off of Fitts' Law, to allow eye movements to reach the target as quickly as possible and maintain accuracy.

Additionally, there was a significant 2-way interaction between amplitude and side (left or right horizontal saccade) on PV in the symptomatic group (Fig. 12). The decomposition of this interaction demonstrated that PV was greater for rightward than leftward saccades at the 1.6 cm and 12.90 cm amplitudes. For the most part, people read from left to right and therefore we are highly practiced in rightward saccades and perhaps this helps to explain the effect of this interaction on PV. In contrast, a 2012 study divided 45 participants into 4 groups according to their hand and eye dominance. The direction of saccades in these groups noted left-right asymmetries in PV regardless of the dominant hand. Leftward saccades had longer durations, larger sizes, and greater peak velocities than rightward saccades. The authors of this study suggested the asymmetries in PV based on the direction of the saccade could be explained by nasotemporal differences in some participants and eye dominance for other participants (Vergilino-Perez et al., 2012). The nasotemporal asymmetries in saccade velocity are more predominant with smaller saccades, whereby temporally directed saccades are faster than nasal directed saccades (Jóhannesson Ó et al., 2018; Jóhannesson & Kristjánsson, 2013).

The main effect of movement amplitude demonstrated that saccade PA increased with larger amplitudes within the symptomatic and asymptomatic groups (Figs. 18 and 19). This aligned with the higher PV as acceleration is the rate of change of velocity and since velocity was increasing during saccades over these longer amplitudes, the participants' eye movement also had to be accelerating in the same direction. This relationship between amplitude and PA has also been reported by other investigators (Al-Aidroos et al., 2008; Leigh & Zee, 2015).

This thesis study reports that ttPA was not significantly decreased with increasing movement amplitudes, but as amplitude increases so does PA. This would suggest that saccades

are faster when the target is further away, but the saccade has a tendency to take longer to reach PA for closer targets. This thesis study also reports that ttPA did not significantly decrease as target widths increase; however, PA does not increase with larger target widths. This could suggest there is some restraint in the saccadic system, otherwise the saccade would not be able to slow down in enough time to hit the target. It is interesting that a saccade to larger target widths has a tendency to occur in a shorter time and might suggest the eye could fixate at any point within the target. Furthermore, this thesis study also reports that RT did not change appreciably with larger target widths and therefore RT may serve as the constraining factor to explain a reduction in ttPA with increasing target widths.

Within both the symptomatic and asymptomatic groups, ttPV increased with larger movement amplitudes but not with larger target widths (Figs 15 and 16). That is, the participants moved their eyes more quickly over larger amplitudes over a longer duration, possibly indicating there was a change in the online control of movement. Evidence supports that online control of eye movement can occur since the saccade is not entirely ballistic because it can be modified by the CNS to respond to visual information while the eye is slowing toward the end of the saccade (Leigh & Zee, 2015).

Within the asymptomatic group there was a significant main effect of SM on ttPV during the width condition (Fig. 17). Following SM there was an increase in ttPV and might suggest that SM may have an influence on the online control of eye movements.

Since acceleration is a derivative of velocity, it was expected that ttPA, similar to ttPV, would also decrease with larger amplitudes and target widths in both groups. It has been reported that a reduction in ttPV corresponds to a change in the duration of the neuromotor signal, and PA has been postulated to correspond to a change in the magnitude of the neuromotor signal, for online movement control (Al-Aidroos et al., 2008).

The ttPV and PA are the most reflective of the neuromotor signal of horizontal saccades (Abrams, Meyer, & Kornblum, 1989). Typically, horizontal saccades require the contraction of a single agonist muscle with simultaneous inhibition of the antagonist muscles. The friction and elastic properties of the orbital tissues, rather than antagonist muscle contraction, allows the eye to decelerate. The forces that produce the saccade are generated by this agonist muscle contraction. It is from this muscle activity that the duration and magnitude of the neuromotor signal that initiates the saccade are obtained. The duration and magnitude of the neural control

signal is reflected by the kinematics of the eye movement (Leigh & Zee, 2015). As the saccade decelerates, saccade ttPV provides the neuromotor signal's duration. The force magnitude that generates the eye movement is reflected by acceleration of the eye with PA providing an indication of the magnitude of the neuromotor signal (Al-Aidroos et al., 2008). In short, PA and ttPV reflect the neuromotor control that the visuomotor system uses to drive the saccade.

Reaction Time (RT)

Between-Group

Saccade RT occurs in response to a stimulus such as the sudden appearance of a target (Leigh & Zee, 2015). RT is measured to determine the time it takes for a person to process information to produce the required response (Lersa et al., 2005; Schmidt & Lee, 2011). More specifically, RT involves the CNS activity required for stimulus processing, decision making, and movement programming (Schmidt & Lee, 2011). Saccade RTs are controlled by the brain centers involved with saccade programming, such as the superior colliculus and frontal eye fields (Dorris & Munoz, 1995; Leigh & Zee, 2015; Montenegro & Edelman, 2019).

It has been reported that saccade training using a gap paradigm has resulted in improvements in RT, and this improved performance is related to the ability to disengage eye fixation. It is theorized that the gap paradigm decreases saccade RT by disengaging the inhibitory fixation of the omnipause neurons before the target appears, and therefore the eye movements can react more quickly to the appearance of the target. Changes related to fixation disengagement are mediated by the superior colliculus and frontal eye fields (Montenegro & Edelman, 2019).

This is a premise that has been reported by other researchers who note that the reduction in RT requires the deliberate insertion of a temporal gap between the disappearance of the initial fixation target and the appearance of the new saccade target. This temporal gap involves a delay of approximately 100-200 ms between the stimulus for a saccade, the release from its fixation, and its actual performance. (Dorris & Munoz, 1995, 1998; Leigh & Zee, 2015; Moren et al., 2013). This time delay allows for neural processing in the retina, cerebral cortex, superior colliculus, and cerebellum (Leigh & Zee, 2015), with the rostral pole of the superior colliculus having a prominent role in the release of the fixation (Dorris & Munoz, 1995, 1998).

The data from this thesis research did not report any significant change in saccade RT

following SM. This thesis study did not utilize a temporal gap between the central fixation target and the appearance of a new target. However, there is really no reason to suspect that SM would have had an effect on RT with the use of a gap paradigm. A likely possibility is that SM simply had no effect on the brain centers involved with saccade central programming and processing, and any subsequent changes to saccade RT.

Within-Group

This thesis study found that saccade RT decreased as movement amplitudes and target widths became larger within the symptomatic and asymptomatic groups (Figs. 5 and 6). The reduction in RT would indicate that the main effects of movement amplitude and target width might have an influence on the planning of movement. This is consistent with the expectation that RT would increase with smaller target widths due to increasing accuracy and precision demands (Fitts & Peterson, 1964; Schmidt & Lee, 2011). Even though we did not examine the effect of ID on RT; the expectation would be for RT to increase with a higher ID so as to maintain accuracy. In contrast, other studies have reported that RT is refractory to amplitude and width manipulations and hence saccade generation is not influenced by motor planning (Heath et al., 2016; Wu et al., 2010).

This thesis study reported that RT was not significantly changed by the direction of the saccade. This is consistent with the findings by Chi & Lin (1997). In contrast, another study reported a shorter RT for leftward saccades in a right eye dominant group and no difference in the left eye dominant group (Lazarev & Kirenskaya, 2008), and a second study reported that RT was shorter for leftward saccades in the left eye dominant group but no clear results were obtained for the right eye dominant group (Kolesnikova, Tereshchenko, Latanov, & Shulgovskii, 2010). This thesis study did not track eye dominance and, as such, can neither confirm nor refute the role of eye-hand dominance.

The changes in RT related to the main effects of amplitude and width may be indicative of a facilitation of movement planning during the ballistic phase of the saccade. This thesis study reports that RT was decreased for larger amplitude movements and wider target widths, and according to Al-Aidroos et al., (2008), this suggests the visuomotor system can modulate the planning of the saccadic eye movement.

Constant Error (CE) & Variable Error (VE)

Between-Group

Constant error (CE) is a measure of the average error of the participant's achieved eye movement amplitude from the actual target amplitude. In other words, how close did they get to the target? A positive CE corresponds to overshooting the target while a negative CE corresponds to undershooting the target. Saccadic eye movements demonstrate this range effect such that there are overshoots to the nearest target and undershoots to the farthest target (Al-Aidroos et al., 2008; Vergilino-Perez et al., 2012). Variable error (VE) is the standard deviation of target amplitude scores achieved by the participant based on their average score. A reduction in end-point variability (VE) indicates that the participant was able to be more consistent in landing near the target using a primary movement (Elliott et al., 2017). In the between-group analysis of this thesis study, no significant main effects of SM or Group on CE or VE were observed during horizontal saccades.

Since saccades are under neural control, the effect of SM on saccade CE and VE would likely involve a neurophysiologic mechanism. The fact that there was no difference in CE and VE following SM might suggest that eye movements were not influenced by the altered afferent input from the dysfunctional cervical segments to which the manipulation was provided. Spinal manipulation may also have had no effect on saccade CE and VE as the influence of the dysfunctional cervical intersegmental restrictions was minimized because the participant's head was in a relatively neutral posture as their head was fixed by a chin and head rest during the eye movement Fitts' task. While not deliberate, this fixed head posture may have served as a passive intervention similar to cervical traction. Another possibility is that CE and VE are simply independent of the influence of cervical SM. Finally, the altered afferent input that can occur with dysfunctional cervical segments may not have been present in these participants to begin with, and therefore SM would not influence the CE or VE of horizontal eye movements.

Within-Group

Within both the symptomatic and asymptomatic groups there were significant changes in CE during the amplitude and width conditions (Figs. 7 and 8). CE generally increased in a negative direction (larger undershoot) with larger amplitudes and larger target widths in both groups; therefore, horizontal saccades became less accurate as movement amplitudes and target

widths became larger. However, it appears there was a greater influence of movement amplitude on CE than was observed by target width as the MT/ID slope is steeper during the amplitude condition than during the width condition. This thesis study also observed a reduced saccade accuracy toward larger targets at a larger amplitude. In contrast, Chi & Lin (1997) reported that the accuracy of the saccade was greater towards larger targets over smaller movement amplitudes. The investigators of another study suggest that saccades are optimized for speed and accuracy in accordance with Fitts' law, and their observation that slower saccades are less accurate than saccades of normal velocity indicates a violation of Fitts' law (Gopal, Jana, & Murthy, 2017).

Of interest is the main effect of movement amplitude on CE whereby CE increased with larger target amplitudes. This is not an artifact that can be attributed to the influence of larger targets as target width was held constant at 0.5 cm during the amplitude condition. A larger target width could bias an undershoot if the eye fixation is at the proximal border of the target (Leigh & Zee, 2015). However, during the width condition this could be an artifact as the amplitude is held constant at 12.9 cm. Therefore, the participants' approach could be to undershoot the target by fixing their eyes at the proximal border of the target. Additionally, during the width condition, the CE values are clustered around the CE value that was observed during the 12.9 cm movement amplitude. Again, this can be attributed to the 12.9 cm amplitude that was held constant during the width condition and further informs this movement amplitude's influence on a bias to undershoot. A future study could examine if the width main effect might be more prominent if tested at a smaller fixed amplitude.

Variable error increased with larger amplitudes within both groups (Fig. 9 and 10) indicating that saccades became less consistent as movement amplitudes increased, which is in agreement with a study that reported that saccade VE tended to increase with larger movement amplitudes (Al-Aidroos et al., 2008). During optimal movement, the participant will move closer to the target using a primary movement and reduces the need for a corrective submovement. This movement strategy improves the precision of the movement and allows for a reduction in end-point variability. The participants of this thesis study were instructed to move their eyes to the target as quickly and accurately as possible, which may have contributed to the increase in VE as end-point variability increases in a linear fashion with movement velocity (Elliott et al., 2010; Elliott et al., 2017).

Head Movement Fitts' Task

Discrete manual movements are typically comprised of an initial ballistic phase followed by corrective submovements. The second phase of the movement is a homing phase that allows for visual and proprioceptive feedback to correct any discrepancy in manual movement (Elliott et al., 2017; Meyer, Abrams, Kornblum, Wright, & Smith, 1988).

It was thought the ballistic phase was pre-programmed and that no correction in movement or accuracy could occur at this time. Elliott et al., argue that some aspects of the initial so-called ballistic phase are subject to online control. This form of control can occur early in the movement by comparing the velocity and direction of limb movement to an internal model of sensory expectations in what is termed "impulse control". Following this early ballistic phase, late corrections of the trajectory could be made to hit the target and these late corrections take into consideration the mass of the effector (the head in this thesis study) and any influences of gravity that may reduce the need for corrective submovements that can be time-consuming and energy depleting (Elliott et al., 2010; Elliott et al., 2017).

The Fitts paradigm has been used previously to attain a functional measurement of motor performance in those patients with neck pain as compared to a healthy control group (Lersa et al., 2005; Passmore & Descarreaux, 2012; Smith et al., 2006), or in asymptomatic participants with palpable cervical joint restrictions (Passmore et al., 2010). The head movement Fitts' task is a useful perceptual motor behavior task to identify the presence of neuromuscular impairments related to cervical spine dysfunction (Descarreaux et al., 2010). The index of difficulty (ID) and MT relationship of the head movement task agrees with the linear speed-accuracy trade-off seen in previous Fitts' task research (Fitts, 1954; Fitts & Peterson, 1964; Passmore et al., 2010; Passmore & Descarreaux, 2012; Smith et al., 2006).

An important finding of these studies is not only a decrease in motor performance in chronic neck pain patients but also decreased motor performance in a group of asymptomatic participants who have a history of neck pain. Presumably on this basis, a head movement Fitts' task could be useful to assess the neuromuscular and motor performance changes in patients with subclinical neck pain (Descarreaux et al., 2010). More specifically, a head movement Fitts' task, through the analysis of various behavioral and kinematic measures, allows for the functional assessment of disability in participants with chronic neck pain (Descarreaux et al., 2010).

Movement Time (MT)

Between-Group

The symptomatic group experienced a reduction in head MT following SM (Fig. 23), a finding that could be explained as an improved ability of these participants to rotate their head along the horizontal axis. This result is consistent with our hypothesis that MT would be reduced after SM in symptomatic participants. Spinal manipulation did not have the same effect on MT for the asymptomatic group (Fig. 23). This change in head movement is consistent with the pain-adaptation model that notes increased joint ROM due to decreased muscular forces. Painful movement is related to the pain and decreased activity of the agonist muscles and increased activity of the antagonist muscles. This adaptation to pain will demonstrate a reduced velocity of the movement and reduced agonist muscular forces at the painful site (Hodges & Tucker, 2011; Lund, Donga, Widmer, & Stohler, 1991). Previous research has also reported that patients with neck pain have impaired head rotation due to altered cervical proprioception (Harvie et al., 2015; Paulus & Brumagne, 2008).

Within-Group

Movement time increased with larger movement amplitudes and smaller target widths within the symptomatic and asymptomatic groups (Figs. 24 and 29). These results are predicted by the Fitts' Law speed-accuracy trade-off principle (Fitts, 1954; Fitts & Peterson, 1964). However, some specific differences within both groups were evident during the amplitude and width conditions. Within the symptomatic group during the amplitude condition, MT increased with larger target amplitudes when the participant rotated their head to the left or right (Fig. 26), but during the width condition MT decreased following SM with larger target widths (Fig. 27). During the amplitude condition, MT increased following SM for right rotational head movements, but decreased following SM for left rotational head movements within the asymptomatic group (Fig. 28). It should be emphasized that these are weak interactions as there were no significant pairwise comparisons during post-hoc testing. Therefore, these interactions should be interpreted with caution as they may indicate a novel or chance finding rather than a meaningful outcome.

The alteration of head MT following SM is consistent with our hypothesis that MT would be reduced after SM in symptomatic participants. Related findings by Passmore (2010) noted

significant group differences in the SM group for MT in direction congruent (head rotated in same direction as side of intervertebral restriction) conditions ($p < 0.02$) and direction incongruent (head rotated in opposite direction to the side of intervertebral restriction) conditions ($p < 0.05$) when compared to a no intervention group.

The findings of Passmore et al., and the changes in the behavioral measure of MT in this thesis study suggest that cervical SM could be associated with an interplay between biomechanical and neurophysiologic mechanisms. Following SM, an increase in active cervical ROM could be related to a biomechanical mechanism and an improvement in MT could be related to a neurophysiologic component (Passmore et al., 2010). Previous studies have reported alterations in cortical somatosensory evoked potentials during a motor training task (Haavik Taylor & Murphy, 2010) or alterations in cerebellar output during motor sequence learning (Daligadu et al., 2013).

Peak Velocity (PV), Time to Peak Velocity (ttPV), Peak Acceleration (PA), and Time to Peak Acceleration (ttPA)

Between-Group

This thesis study did not report any between-group differences in PV or PA following SM. It was assumed that the symptomatic group would have demonstrated a lower PV and PA, at least at baseline, due to the higher VAS score and would have demonstrated an increase in PV and PA following SM. Therefore, it was unexpected that the symptomatic group would have a higher PV and PA values than the asymptomatic group. Furthermore, PV and PA were increased rather than decreased following SM in the asymptomatic group.

Sjolander et al (2008) reported that the PV was lower in patients with neck pain, in contrast to a control group, during a head rotation repositioning task. Patients with chronic neck pain are expected to have slower movements than asymptomatic participants, as this may be a motor control adaptation developed to avoid or reduce painful movement. This adaptation can be achieved by reducing agonist muscle activity and increasing antagonist muscle activity, or possibly to increase both agonist and antagonist muscle activity due to protective guarding. This alteration in motor control would be consistent with the pain-adaptation model that requires that the amplitude and velocity of movement be reduced to decrease the muscular forces of the agonist muscle during the movement (Hodges & Tucker, 2011; Lund et al., 1991). Because we

did not observe any change in PV or PA following SM, we are unable to be certain that a motor control adaptation was present due to pain despite the symptomatic group having the higher baseline VAS score. This discrepancy could be related to a floor effect where the level of pain on the VAS score was too low to discern any detectable difference in PA or PV following SM.

This thesis study did report a reduction in ttPV (Fig. 47) and a significant reduction in ttPA (Fig. 54) following SM in the between-group analysis. The reduction in ttPV following SM found in this this study could be related to improved online control of rotational head movement consistent with reports of SM reducing some of the muscular forces via muscular reflexogenic responses that are often associated with spinal pain (Cramer et al., 2006; Gyer et al., 2019). Arguably, this improved online control of movement is occurring during the initial primary submovement of the head, instead of occurring during the expected corrective submovement (of which we did not analyze). This is in agreement with previous reviews that indicate that the ballistic phase of movement is not as ballistic as once thought and therefore the direction and velocity of movement can be modified before the end of the primary movement (Elliott et al., 2010; Elliott et al., 2017).

Cervical muscle hypertonicity was not quantified in any of the participants in this thesis study. The chiropractic clinician used manual palpation to examine hypomobile segments to identify changes in tissue texture and tone, elicit pain or tenderness of the paraspinal tissues, and to find intervertebral restrictions of the cervical intervertebral segments. As such, it would be speculative, but not unreasonable, that muscular forces could affect head movement, particularly in the symptomatic group, and that SM could reduce these muscular forces and alter outcome measures such as ttPV.

Within-Group

Within both the symptomatic and asymptomatic groups there was an increase in PV with longer amplitudes and larger target widths (Figs. 46 and 47). This is consistent with discrete aiming, in which a reasonable movement strategy is to approach the target as quickly as possible, most likely resulting in an undershoot, which can then be corrected with the appropriate acceleration or deceleration to reach the target. By utilizing this method, participants can plan their movements to minimize the need for a late movement correction, as would occur with an overshoot (Elliott et al., 2017). However, the observation that PV increased with larger movement amplitudes, or larger target widths translates into higher ID values for amplitude and

lower ID values for width. In consideration of Fitts' Law, MT would increase with longer movement amplitudes and smaller target width (both equate with a higher ID). Peak velocity follows this pattern for longer amplitudes in that it may take longer to move to a farther target, but movement is at a higher velocity. This thesis study reports that PV increases while moving to larger targets (low ID). This is inconsistent with Fitts' Law where PV, similar to MT, would be expected to decrease with larger target widths. While this inconsistency between Fitts' Law and the PV data from this thesis study is of interest, it should be noted that there are very small differences and small effect sizes (η^2_p) for width compared to amplitude.

Peak acceleration will increase with increasing head movement amplitudes as greater muscular force is required for larger amplitudes. PV also increases for larger amplitudes. As such, fast movements require more muscular force than slow movements of the same amplitude. Therefore, there will be an increase in PA associated with these increased muscular forces.

There was no main effect of amplitude on ttPV within the symptomatic group (Figs. 48), which is unusual since PV did increase with larger movement amplitudes. A more consistent relationship was found during the width condition, such that there was no main effect of width on ttPV (Figs. 48 and 50), which aligns with the minimal change in PV observed at the various target widths.

There was a reduction in ttPV following SM within the symptomatic (Fig. 49) and asymptomatic (Fig. 51) groups. For the symptomatic group, ttPA was decreased following SM during the width condition (Fig. 56) and there was also a reduction in ttPA with a SM x side interaction within the symptomatic group (Fig. 57).

These findings are supportive of the possibility that SM was able to modulate the rate of movement during online control. The reduced ttPA would suggest that SM may have had an influence on the ability of participants to control their head movement. If ttPA is reduced that means they are reaching PA at an earlier point. It has been reported that during a head movement Fitts' task, chronic neck pain patients use a motor control adaptation to maintain accuracy, similar to that of healthy controls, by increasing the duration of the deceleration phase of MT; that is, they decreased their ttPA (Descarreaux et al., 2010).

Reaction Time (RT)

Between-Group

There was a significant reduction in head RT following SM (Fig. 30). A reduction in RT after SM could suggest that the motor planning processes of rotational head movement might be influenced by cervical SM. Both groups were assessed for intervertebral restrictions that are associated with cervical spine dysfunction and SM was directed at these restricted segments. The intervertebral cervical restrictions of the participants in this study could have been associated with altered motor planning processes due to aberrant afferent input resulting in altered sensorimotor integration.

It is relevant to note that the reduction in RT following SM when compared to pre-SM (Table 14) noted a small 10 msec difference with a small effect size ($\eta^2_p = 0.208$). Even though the difference was statistically significant, the reduction in RT could be a novel or chance finding and might suggest a type I error.

It has been suggested that the motor planning processes may be related to cortical processes that may be impaired by cervical spine dysfunction (Lersa et al., 2005). When using a probe task, Lersa and colleagues reported that multiple sites of cervical spine dysfunction altered cortical processing. A probe task involves a participant performing the primary task of interest when a secondary task is introduced to challenge their capacity to process information. Simultaneous presentation of the secondary task with the primary task requires the participant to use additional capacity to process information and will lower performance on the secondary task in relation to the amount of capacity required by the primary task (Schmidt & Lee, 2011). In the Lersa study, the site(s) of cervical spine dysfunction were assessed for tissue compliance over the facet joints, tone of the cervical musculature, and pain or tenderness. Evidence from this study reports probe task RT being significantly longer for the group with two sites of cervical spine dysfunction (448.8 ± 15.8 ms) as compared to a group with one site of cervical spine dysfunction (404.0 ± 19.4 ms).

This thesis study utilized a randomized variable foreperiod and randomized side of target appearance to prevent the participant from anticipating when and where the target would appear on the computer screen. Due to the use of this randomized process, it is unlikely the reduction in RT was due to participant anticipation and therefore was potentially a response to SM. A study by Haavik & Murphy (2010) suggests that cervical spine manipulation not only alters cortical

integration of dual somatosensory inputs, but also alters the way the central nervous system responds to subsequent motor training tasks. On this basis, SM applied to the cervical segmental restriction may have had some influence on the planning process of a motor behavior task such as horizontal head movement.

Within-Group

Reaction time decreased with increases in target width within the symptomatic and asymptomatic groups (Figs. 33 and 34), which may indicate different motor planning processes are utilized as the target widths become larger (Schmidt & Lee, 2011). It is also possible that since both groups were assessed for intervertebral restrictions that are associated with cervical spine dysfunction, that these restrictions could be associated with the decreased RT found in both groups with head movement during the width condition. It has been suggested that the motor planning processes may be related to cortical processes that may be impaired by cervical spine dysfunction (Lersa et al., 2005).

Furthermore, it was interesting to find that RT increased during the width condition and not during the amplitude condition (Figs. 33 and 34). It is possible the participants were responding to the appearance of the target width, rather than responding to the movement amplitude. This could possibly explain the influence of target width on head movement RT.

This thesis study found that RT was reduced in the symptomatic group, but not the asymptomatic group, following SM (Figs. 31 and 32). There was also a reduction in RT within the asymptomatic group as indicated by a 3-way interaction during the width condition (Fig. 35). This interaction did not report any significant pairwise comparisons during post-hoc testing and should be considered a weak outcome and could possibly be a chance finding even though the overall significance was $p< 0.05$. The reduction in RT following SM observed in the symptomatic group suggests that the change in RT was pain related. Moreover, the reduction in RT could also be due to reduced aberrant cervical afferent input related to improved sensorimotor integration. On this basis, SM applied to neck pain participants or to the site of cervical segmental restriction may have had some influence on motor planning of a motor behavior task such as rotational head movement.

Constant Error (CE) & Variable Error (VE)

Between-Group

Constant error indicates the participant's bias towards undershooting or overshooting the target and reflects the intrinsic biases in the motor control system (Sjölander et al., 2008). There was no significant change in CE in either the symptomatic or asymptomatic groups which might suggest that pain was not a factor influencing movement accuracy in the symptomatic group. However, the expectation was that SM would reduce CE in the symptomatic group due to the reduction of cervical intersegmental dysfunction, resulting in improved neuromuscular control and sensorimotor integration (Haavik et al., 2021; Haavik & Murphy, 2012). It is also plausible that one session of SM is not adequate to influence the aberrant output related to cervical spine dysfunction and a longer course of SM may be required to reduce this aberrant afferent output to influence a change in CE. Finally, SM may have not influenced CE as the participants in this thesis study did not want to compromise accuracy in order to maximize their speed.

Variable error indicates the variability of the participant's performance at the end-point of their head rotation, and has been reported to be the result of the sensitivity of sensorimotor processes (Sjölander et al., 2008). By examining VE, we can determine if end-point variability is a characteristic associated with chronic neck pain and if SM has any influence on VE and therefore the consistency of head movement in these patients. There was no significant difference in VE between the groups after SM in this thesis study, but the expectation was that head movement end-point variability would be reduced post-treatment when SM was applied to the cervical intervertebral restriction.

Within-Group

Within the symptomatic group, there was a significant amplitude x side interaction (Fig. 37) on CE whereby the participant's head movement to the left or right, overshot the targets at the largest amplitude and undershot the targets at the smaller amplitudes. This finding is inconsistent with the assertion by Elliott et al., that greater spatial variability is a characteristic of larger amplitude movements and are associated with greater undershooting in comparison to smaller amplitude movements (Elliott et al., 2017). In keeping with Elliott's assertion, it was unexpected that participants would overshoot targets at the largest amplitude. This overshoot at the 29.0 cm movement amplitude is likely an artifact which could possibly be explained by the

edge of the monitor serving to draw the participant to an overshoot. Another possibility is that the participant is moving their head so fast over the 29.0 cm amplitude that their decrease in speed is inadequate and they overshoot the target. Moreover, this possible effect of the 29.0 cm amplitude (which was held constant during the width condition) appears to be consistent with the bias to overshoot at all target widths. That is, the 29.0 cm movement amplitude seems to have an influence on the bias to an overshoot.

This thesis study also found changes in CE during head movements under the width condition within both the symptomatic and asymptomatic groups (Figs. 36 and 40). There was a significant main effect of SM on CE in the symptomatic group (Fig. 38) and a significant SM x width interaction in the asymptomatic group (Fig. 42). Following SM, the symptomatic group demonstrated an increase in CE related to overshooting the target resulting in reduced head movement accuracy. Following SM, the asymptomatic group CE was generally reduced at specific target widths indicating that the asymptomatic participants overshot to a lesser extent as the target widths increased and therefore had greater head movement accuracy.

A 2010 study analyzed head repositioning accuracy and reported that CE decreased after a muscle contraction task, as the normal overshoot was reduced from 7.1 degrees to 4.6 degrees for the 30-degree target and 2.2 to 1.4 degrees for the neutral head position. The study proposed that the muscle contraction task reduced unilateral neck muscle tension and CE was thereby reduced due to increased sensitivity of the cervical proprioceptors (Malmström et al., 2010). The muscle contraction task that reduced neck muscle tension is similar to the effects of SM on reducing muscle tension via a reflexogenic effect (Cramer et al., 2006; Gyer et al., 2019).

One of this study's inclusion criteria was the presence of neck pain and palpable intersegmental restrictions related to hypertonic muscles and segmental joint dysfunction in the symptomatic group. SM could reduce intersegmental dysfunction at this cervical level and possibly result in improved neuromuscular control and sensorimotor integration (Haavik et al., 2021; Haavik & Murphy, 2012). Therefore, the expectation would have been to improve head movement accuracy resulting in a reduction in CE.

All the participants in this thesis study received a spinal manipulative thrust to cervical intersegmental restrictions, which could have resulted in increased cervical ROM in the horizontal direction with a subsequent increase in ability of head rotation. This increase in head rotation is consistent with the pain-adaptation model that notes increased joint ROM due to

decreased muscular forces (Hodges & Tucker, 2011; Lund et al., 1991). Therefore, the increase in head rotation due to a reduction in muscular forces may have allowed for greater ability of the symptomatic and asymptomatic participants to rotate their heads, resulting in a larger overshoot with a corresponding increase in CE.

This differences in CE within the symptomatic and asymptomatic groups could also be accounted for by the possible nonlinearity of the headmouse during maximal head rotations, such that the amount of head rotation is underestimated at the largest target amplitude and may be challenging the tracking range of the headmouse. The horizontal field of view for the Headmouse Nano is 72° (36° left or right) at the 60 cm viewing distance. The calculated angle at the 29.0 cm amplitude is 25.8° which is certainly within the 36° tracking range. However, the 29.0 cm amplitude may still be challenging the linearity of the headmouse as PV does increase with larger movement amplitudes and the head rotation velocity at this amplitude may have been enough to overcome the tracking ability of the headmouse.

Within the symptomatic group there was an increased VE associated with head movement during the amplitude condition (Fig. 43), especially with the longer amplitudes, indicating the end-point variability increased with the longer amplitudes. This is consistent with a Fitts' task as the longer movement amplitudes indicate a higher ID that requires greater information processing demands that can result in significantly less movement consistency due to a more variable performance (Lersa et al., 2005). There was greater inconsistency at the 29.0 cm movement amplitude as was seen with the large SE indicating greater variability at this amplitude. The asymptomatic group had greater variability at the 15.5 cm and 29.0 cm amplitudes as demonstrated by a large SE. Therefore, there was greater inconsistency at the larger movement amplitudes which could be associated with the higher PV at these amplitudes making it more difficult for the participant to move the headmouse cursor to a consistent fixation point on the target.

A previous study observed that VE was different between control, insidious neck pain, and WAD groups (Sjölander et al., 2008). Sjölander also reported no change in head movement end-point variability during left and right head rotations during a head repositioning task. Malmström (2010) reported that VE did not change with head movements to the side of activated muscle contraction or to the non-activated side. The muscle contraction task that reduced neck

muscle tension is similar to the effects of SM on reducing muscle tension via a reflexogenic effect (Cramer et al., 2006; Gyer et al., 2019).

Similar to their research findings on RT, Lersa et al (2005) reported that VE tended to be higher in the group with two sites of cervical spine dysfunction versus a group with only one site. This thesis study only assessed for one site of cervical spine dysfunction, and it was at this cervical segment that the manipulation was provided. As such, this thesis study was not designed to assess if VE, or any of the other outcome variables, would be different in the presence of two sites of dysfunction versus only one site of dysfunction. An area of further investigation would be to examine if VE, or any of the outcome variables, would be altered if SM was provided at two sites of cervical dysfunction versus only one site.

In summary, the eye movement Fitts' task between-group analysis did not find any significant main effect of SM or Group on MT. The neutral position of the participant's head on the head/chin rest or the absence of COR deficits may have reduced the influence of SM on saccade MT. There was also no significant effect of SM or Group on the remaining dependent variables, which may be simply explained by the fact that the symptomatic and asymptomatic groups were not that different from each other.

The within-group analysis was conducted to determine if the main effects of amplitude, width, and SM had any influence on the dependent variables. The within-group analysis found that there was a significant increase in MT with increasing amplitudes, a finding that is consistent with Fitts' Law. The significant influence of movement amplitude on MT is also likely related to the nature of eye movement during the saccade which serves to maintain the fovea's position on the target to ensure visual detail and accuracy. This increase in MT with increasing amplitudes, along with a corresponding increase in PV with larger movement amplitudes, suggests that participants move their eyes quickly over larger amplitudes, but they have a longer MT to maintain accuracy. The increase in MT and PV of the saccade with increasing movement amplitude has been termed the main sequence. Movement amplitude also influenced ttPV and PA which may indicate an effect on the execution of the saccadic eye movement and further delineate the neuromotor control of the visuomotor system that generates the saccadic eye movement. In addition, movement amplitude and target width had a significant effect on RT, thereby having a possible influence on the planning of eye movement and that

saccades are not an entirely ballistic movement. Finally, there was a reduced saccade accuracy and consistency with larger movement amplitudes with a participant bias towards an undershoot.

During the head movement Fitts' task there was a significant effect of SM on MT in the symptomatic group compared to the asymptomatic group. There was also a significant effect of SM on MT within the symptomatic group during the amplitude and width conditions. MT increased with larger amplitudes and smaller target widths, which is again consistent with Fitts' Law. During examination of the kinematic variables, there was no change in PV or PA following SM, but a significant reduction in ttPV and ttPA following SM, suggesting that SM may have had an influence on the online control of head movement. The improved online control of movement could be related to SM reducing the muscular forces and might suggest the initial primary submovement is not as ballistic as once thought. The findings were different for the within-group analysis that reports PV was increased at larger target amplitudes along with a significant change in CE and VE. Therefore, the participants rotated their head quickly toward the target resulting in an undershot, indicating a reduction in movement accuracy and increased end-point variability, which would be followed by the appropriate corrective submovement. The increase in VE with larger movement amplitudes was consistent with Fitts' Law as a higher ID requires greater information processing that can then result in reduced movement consistency. The reduction in ttPV following SM was only significant within the symptomatic group during the width condition, suggesting that the online control of head movement was altered following SM. RT was reported to be reduced, between and within groups following SM, suggesting that SM may have had an influence on the motor planning processes of head movement with subsequent alterations in cortical processing that was originally impacted by impaired cervical spine intersegmental dysfunction.

Eye and Head Movement Amplitude and Width Comparisons

Fitts' Law asserts that the combined effects of movement amplitude and target width (Index of Difficulty) defines MT for discrete movements (Fitts, 1954; Fitts & Peterson, 1964). Moreover, Fitts' Law theorizes that, regardless of the manipulation of the amplitude and width combinations (2A/W), discrete movements yield equivalent linear MTs for the same ID. It has been argued that the myriad of research in direct support of Paul Fitts' seminal work utilized short movement amplitudes and small target widths and therefore a small range of IDs (Heath et al., 2011). Therefore, some researchers are of the opinion that there is no direct evidence to

support the interdependent nature of MT/ID relations across a larger range of movement amplitudes and target widths (Heath et al., 2011).

Amplitude and width conditions (Fitts Parameter) were manipulated to examine the MT/ID relationship within symptomatic and asymptomatic groups during an eye movement Fitts' task. A Fitts Parameter x ID interaction (Fig. 66) on saccade MT identified a significant increase in saccade MT between all IDs during the amplitude condition but not during the width condition. Results from this thesis study demonstrate that saccade MT for the amplitude (mean=40.6 msec) and width (mean=69.1 msec) conditions were dissimilar but produced robust linear relationships with ID within both the symptomatic (amplitude: R^2=0.981; width: R^2=0.955) and asymptomatic (amplitude: R^2=0.962; width: R^2=0.891) groups. Notably, even though ID values were similar between the amplitude and width conditions, the average MT/ID slope for saccades were asymmetrical with the amplitude condition (Symp: slope=13.62; Asymp: slope=14.5) having a steeper slope than for the width condition (Symp: slope=0.79; Asymp: slope=1.64).

The decomposition of the Fitts Parameter x ID interaction and the differences in the slopes for the amplitude and width conditions is contrary to Fitts' Law that asserts the MT increases as a function of increasing ID given a constant ratio between amplitude and width. These results indicate that saccade MT for a given ID was greatly influenced by amplitude rather than by target width.

The results of this thesis study are consistent with Heath et al. (2016) who reported that the width and amplitude components of Fitts' Law are separable rather than interdependent, and that the amplitude condition may be associated with greater information processing demands than the width condition. Additionally, this thesis study along with Wu et al. (2010) and Heath et al. (2016) reports that saccades demonstrate reliable speed-accuracy relations as noted by a lengthening of saccade MT with increasing ID.

The head movement task also identified a significant Fitts Parameter x ID interaction (Fig. 68) on head MT whereby MT increased with increasing IDs during the amplitude and width conditions. The index of difficulty (ID) and MT relationship of the head movement task agrees with the linear speed-accuracy trade-off seen in previous Fitts' task research wherein increasing the ID decreases the speed (increased MT) (Fitts, 1954; Fitts & Peterson, 1964; Passmore et al., 2010; Passmore & Descarreaux, 2012; Smith et al., 2006).

Amplitude and width conditions were again manipulated to examine the MT/ID relationship within symptomatic and asymptomatic groups during a head movement Fitts' task. Results from the present study demonstrate that head MT for the amplitude (mean=0.893 sec) and width (mean=0.957 sec) conditions were similar and produced robust linear relationships with ID within both the symptomatic (amplitude: R^2=0.998; width: R^2=0.888) and asymptomatic (amplitude: R^2=0.974; width: R^2=0.978) groups. In contrast to the eye tracking task, the ID values were similar between the amplitude and width conditions, with the average MT/ID slope for head movement being symmetrical for amplitude condition (Symp: slope=0.06 sec; Asymp: slope=0.07 sec) and the width condition (Symp: slope=0.04 sec; Asymp: slope=0.03 sec) within the symptomatic and asymptomatic groups.

The symmetrical MT/ID slopes for head movement during the amplitude and width conditions of this thesis study are consistent with Fitts' Law that indicates discrete movements, such as head or reaching movements, are associated with the same ID results in equivalent linear MTs regardless of the manipulation of the amplitude and width combinations (Fitts, 1954; Fitts & Peterson, 1964). The symmetrical linear movement times might suggest that the amplitude and width conditions may both be associated with greater information processing demands during the head movement task. In contrast, a study that utilized a discrete reaching task reported that amplitude and width manipulations produced robust linear MT/ID relations; however, the MT/ID slopes were steeper for the amplitude condition versus the width condition (Heath et al., 2011). Heath and colleagues concluded that the amplitude and width components are separable rather than interdependent and is contrary to Fitts' Law that asserts the dependability of the 2A/W ratio yields equivalent MTs. The differences in the amplitude and width slopes in this thesis study compared to Heath may simply be related to the differences in movement when comparing a head movement task to an arm reaching task. Furthermore, this thesis study recruited chronic neck pain patients while the participants in Heath's study were healthy university students.

The eye movement Fitts' task results are consistent with our hypothesis that saccades will elicit a fixed MT/amplitude relation such that MT increases will be greater with increasing ID values for the amplitude condition than those associated with the width condition within symptomatic and asymptomatic groups. The head movement Fitts' task results were also consistent with our hypothesis that head MT will increase similarly during either the amplitude or width condition with increasing ID values within symptomatic and asymptomatic groups.

Discreet task-oriented eye movements yield asymmetrical MTs across amplitude and width manipulations of the ID and these eye movements elicit separable MT/ID relations within symptomatic and asymptomatic chronic neck pain groups. The task-oriented head movements in contrast produce symmetrical MTs across amplitude and width manipulations of ID and elicit interdependent MT/ID relations within symptomatic and asymptomatic chronic neck pain groups.

Limitations

The behavioral and kinematic outcomes from this study were measured after a single session of cervical SM. While significant outcomes were reported from the head movement Fitts' task, a single session of SM during this task does not always equate with clinically meaningful results. Therefore, future studies should utilize a therapeutic trial of several sessions of SM over a clinically relevant period. For example, it has been reported that a course of cervical spine manipulation at 3 times per week for 6 weeks (18 visits) was the most effective dose for patients with cervicogenic headache (Haas et al., 2018). The behavioral and kinematic outcomes should then be measured over several time points to establish if any changes in these outcomes are transient responses, or if they persist for longer periods.

A power analysis was conducted a priori to allow for calculation of an appropriate sample size based on published saccade MT effect sizes. However, beyond MT, the sample size of the study may not have been large enough to demonstrate the true effect of SM on the behavioral and kinematic measures of eye movement. The between-group variation due to differences in baseline VAS for pain could have obscured small differences in behavioral and kinematic measures that were not detected following SM, resulting in a potential type II error. However, the within-group comparisons, despite the baseline VAS differences, would reduce any random (error) variation to allow us to see the differences more clearly in outcomes related to SM. This type of design also lowers the risk of a type II error.

The symptomatic group reported relatively low levels of pain at baseline. Higher levels of pain may have resulted in different or more pronounced sensorimotor adaptations that could provide more robust behavioral and kinematic changes, or at least clearer differences between the symptomatic and asymptomatic groups following SM. The minimal clinically important difference (MCID) for the VAS is 15 mm/1.5 cm (Hägg, Fritzell, & Nordwall, 2003; Xia et al., 2016). The symptomatic group had a pre-SM mean VAS score of 2.77 cm and post-SM mean

VAS of 2.63 cm. A mean difference of 0.14 cm does not satisfy the MCID for the VAS and could represent a floor effect, with no chance of further improvement as the pain scores were too low to start with. A future study should include participants with higher pain score on the VAS so that the MCID is more likely to be achieved following SM.

Another limitation may be the forehead and chin support that was used by the participant during the eye movement Fitts' task. This neutral head position may have served as a passive traction intervention that reduced the influence the dysfunctional cervical restrictions could have on the cortical changes that modulate eye movements and resulted in non-significant between-group changes to saccade MT. Furthermore, any changes in performance-based outcomes identified in the within-group analysis may be related to a reduction in the neurophysiologic influence of cervical joint dysfunction due to the neutral head position rather than any potential influence of SM.

This study did not explore the impact of the dominant eye on the behavioral or kinematic changes that may occur with the direction of eye movements. A previous study suggested the asymmetries in PV based on the direction of the saccade could be explained by nasotemporal differences in some participants and eye dominance for other participants (Vergilino-Perez et al., 2012). Future studies could incorporate this comparison when utilizing an eye movement Fitts' task.

This thesis study is also limited by the absence of a healthy control group with no history of neck pain. The presence of a healthy control group would have assisted this study to explore whether any observed changes in behavioral or kinematic measures following SM may be due to cervical intervertebral restrictions.

Additionally, the patients who volunteered for this thesis study were a convenience sample recruited from the principal investigator's chiropractic clinic; therefore, any results from this study may not be generalizable to other patient populations. The participants in this study were familiar with treatment from the PI, and their professional relationship with the PI and his clinic may have influenced their performance. However, testing participants who are familiar with cervical SM could serve to reduce any apprehension the participant may experience during the manipulation and any subsequent neurophysiologic effect, such as a startle response, that could be a confounder for any of the outcome measures.

Conclusion

The primary purpose of this study was to examine the effect of cervical spine manipulation on eye and head movement tasks in a population of chronic neck pain participants who were symptomatic or asymptomatic at the time of the study.

The non-significant MT outcome from the eye movement Fitts' task is contrary to the study's hypothesis that the time needed to complete eye movement tasks will be different between and within symptomatic and asymptomatic groups following cervical SM. This requires the acceptance of the alternate hypothesis that the time needed to complete eye movement tasks do not change following SM between and within symptomatic and asymptomatic groups. During the head movement Fitts' task, MT was decreased in the symptomatic group compared to the asymptomatic group following SM for the between-group analysis. This is consistent with our hypothesis that head movement times will be different between symptomatic and asymptomatic groups. Both the between-group and within-group analysis indicated head MT was also decreased within the symptomatic group following SM, which was predicted by our hypothesis that head MT will decrease following SM within the symptomatic group. Thus, we demonstrated that SM could improve head movements in patients experiencing neck pain.

This study quantified other behavioral and kinematic measures and reported no main effect of SM on these measures as outlined in the between-group and within-group analyses for the eye movement task. The between-group analysis for the head movement task demonstrates that SM had a significant main effect on RT, ttPV, and ttPA. By comparison, the within-group analysis reports that SM had a significant main effect: on RT during the amplitude and width conditions within the symptomatic group; on ttPV during the amplitude and width conditions within the symptomatic and asymptomatic groups; and on ttPA during the width condition within the symptomatic and asymptomatic groups. This study demonstrates that SM may have an effect on the motor planning process and online control of rotational head movement.

A secondary purpose of this study was to determine if discreet task-oriented eye and head movements yield symmetrical or asymmetrical MTs across amplitude and width manipulations of the ID and whether these eye and head movements elicit interdependent or separable MT/ID relations within symptomatic and asymptomatic groups.

Saccade MT during the eye movement task was associated with reliable speed-accuracy trade-offs during the amplitude and width conditions. However, there was an asymmetry of the

MT/ID relations with the slope of the amplitude condition being steeper than that of the width condition. This asymmetry between the amplitude and width conditions for saccadic eye movements suggests that these elements of Fitts' Law are separable rather than interdependent. In contrast, this thesis study found that the head movement task produced symmetrical MTs across amplitude and width manipulations of ID and elicited interdependent MT/ID relations within the symptomatic and asymptomatic chronic neck pain groups.

This thesis study expands the knowledge of motor control and CNS changes that may occur following cervical SM, for future investigation of these performance-based outcome measures by the wider scientific community. A clear delineation of biomechanical versus neurophysiologic mechanisms to explain altered motor performance during a Fitts' task following SM is unlikely due to the multimodal mechanisms associated with spinal manipulation (Passmore et al., 2010).

Exploring the behavioral and kinematic variables of movement provides further knowledge of the planning, execution, and online control of movement (Passmore, Johnson, et al., 2014; Passmore et al., 2019; Passmore et al., 2015). Current evidence suggests that the effects of SM are related to a cascade of neurophysiologic mechanisms which are beyond the biomechanical changes that are observed at the site of the manipulative thrust. Specifically, a number of studies report that spinal manipulation/mobilization can activate various areas of the CNS beyond the specific joints and spinal segments to which the manual therapy is delivered in order to reduce pain by modulating the sympathetic nervous systems and specific areas of the brain such as the periaqueductal grey region (Schmid et al., 2008). Basic science and clinical research appear to dichotomize biomechanics and neurophysiology into separate and distinct phenomena, when in fact they are very much interconnected (Triano et al., 2013). As such, this thesis study serves to quantify the behavioral and kinematic changes following SM that are associated with changes in motor performance during a Fitts' task which could be related to the proposed interplay between distinct biomechanical and neurophysiologic mechanisms.

The biomechanical effects of SM are not in dispute, but these effects do not completely explain the clinical benefit of SM; therefore, additional mechanisms are required to explain the clinical effectiveness of SM. The biomechanical effects of SM include: the forces generated during SM; the force-time histories of SM; and the vertebral displacements that occur during SM. The neurophysiologic effects of SM include muscle spindle afferent responses, muscle

reflexogenic responses, central motor excitability, and neuroplastic brain changes to name a few (Gevers-Montoro et al., 2021; Gyer et al., 2019; Herzog, 2010; Pickar & Bolton, 2012). Studies have established that the kinetics and kinematics of SM appear to have a relationship with the subsequent neurophysiologic effects (Gyer et al., 2019; Herzog, 2010; Pickar, 2002; Pickar & Bolton, 2012; Reed et al., 2014).

Understanding the biomechanical and neurophysiologic mechanisms that underly SM may assist in a better understanding of patient responses to SM, as well as identify the appropriate patients to receive and respond favorably to SM. Identification of the mechanisms of action of SM may also provide greater acceptance of SM, along with the proper utilization of this manual therapy in a clinical setting (Bishop et al., 2015). By providing a better understanding as to how the mechanisms of SM attenuate spinal pain, clinicians and researchers can better determine which mechanisms may affect specific conditions, and how this translates into clinical benefits. Translational research is required to close the gap between basic and clinical research. In the future, randomized controlled trials on the efficacy of SM for spinal pain should include neurophysiologic outcomes such transcranial magnetic stimulation and somatosensory-evoked potentials (Clark et al., 2018), as well as performance-based outcome measures such as a Fitts' task (Passmore & Descarreaux, 2012) to bridge the gap between experimental and clinical research findings.

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Appendix A

Research Participant Information and Consent Form

Title of Study: Motor performance of asymptomatic and chronic neck pain participants pre- and post- spinal manipulation using an eye and head movement Fitts' task

Principle Investigator: Geoff Gelley DC, MSc, FRCCSS(C)
12-845 Dakota St.
Winnipeg, MB R2M 5M3
(204) 254-0130

You are being asked to participate in a research study. Please take your time to review the consent form and discuss any questions you may have with the study staff. You may take your time to make your decision about participating in this study and you may discuss it with your friends, family, or (if applicable) your doctor before you make your decision. This consent form may contain words that you do not understand. Please ask the study staff to explain any words or information that you do not clearly understand.

Purpose of Study

The purpose of this research study is to measure the head and eye movements of participants, with and without neck pain, before and after spinal manipulative therapy (SMT).

A total of 40 participants will be recruited for this study.

Study procedures

All participants are being recruited for this study from a chiropractic clinic in the City of Winnipeg.

Dr. Gelley is conducting a research study to determine the head and eye movement times, before and after the delivery of SMT, in participants with and without neck pain.

Prior to consent, Dr. Gelley will have determined your eligibility for the study. If you consent to be a participant in this study, one of the clinic staff members will obtain your date of birth and gender. No other requirements are needed for this study.

You can stop participating at any time. Your decision not to participate or withdraw from the study will not affect your chiropractic care. However, if you decide to stop participating in this study, we encourage you to talk to the study staff first.

If you choose to participate in this study, you will be asked to do the following:

- *For those participants with neck pain, you will be asked to complete 2 short questionnaires.*
- *During the eye movement portion of the study, you will be seated on a chair in front of a computer monitor. You will be looking into an eye-tracker system that has small cameras to measure your eye movements.*
- *You will be asked to fixate your gaze on a central target that will appear on the computer. You will then direct your gaze to another target that will appear on the screen. You will repeat this a total of 56 times. If we are not able to capture the movement of your eyes during part 1, you will be excluded from the eye movement portion of the study.*
- *During the head movement portion of this part of the study, you will be seated on a chair in front of a computer monitor. You will be wearing safety glasses that will have a reflective marker (to move a cursor on the computer monitor) attached to the nasal bridge of the glasses.*
- *You will be asked to fixate your gaze on a target that will appear on the computer monitor with the cursor on the target. You will then move the cursor by turning your head to another target that will appear on the monitor. You will repeat this a total of 56 times.*
- *You will then be positioned on your back on a chiropractic treatment table. Dr. Gelley will perform spinal manipulation to one side of your neck. All participants will then complete another questionnaire. All participants will then repeat another 54 trials each of the eye movement and head movement tasks.*
-

This visit will take approximately 90 minutes.

Risks and Discomforts

There are potential risks of low incidence associated with cervical spine manipulation. You should note:

- *While uncommon, some patients may experience short term aggravation of symptoms, or muscle strains and ligament sprains as a result of cervical spine manipulation.*
- *Although extremely remote, disc injury, vertebral fracture, and stroke have also been known to occur following cervical spine manipulation.*
- *No additional risks or discomfort are expected to occur because of your participation in this study. You will be allowed to rest between trials if needed, or you can discontinue at any time.*
- *Pre-treatment diagnostic imaging is neither a standard of chiropractic care nor contemplated in this study design.*

Benefits

There may or may not be direct benefit to you from participating in this study as specific therapeutic benefits cannot be assured. We hope the information learned from this study will help advance the scientific understanding of the relationship of cervical spine manipulation, and eye and head movements.

Costs

All the procedures that will be performed as part of this study are provided at no additional cost to you.

Payment for participation

You will receive no other payment or reimbursement for any expenses related to taking part in this study.

Confidentiality

Information gathered in this research study may be published or presented in public forums; however, your name and other identifying information will not be used or revealed. Despite

efforts to keep your personal information confidential, absolute confidentiality cannot be guaranteed. Your personal information may be disclosed if required by law. All data files including those with personal information will bear only your assigned study number. A separate document will contain the master list of only names and study numbers.

The data collected from you during this study may be shared in an anonymized or de-identified form with academic journals for publication purposes or other researchers according to international guidelines. The data may also be stored by the academic journal under an open access policy in which case it may be used by other researchers for further data analysis and research purposes. Before publishing/sharing any data, it will be reviewed with the Research Ethics Board or oversight committee to ensure full compliance with privacy legislation.

ClinicalTrials.gov is a website that provides information about federally and privately supported clinical trials. A description of this clinical trial will be available on http://ClinicalTrials.gov. This website will not include information that can identify you. At most, the website will include a summary of the results. You can search this website at any time.

Organizations that may inspect and/or copy your research records for quality assurance and data analysis include groups such as: The Faculty of Graduate Studies at the University of Manitoba and The Applied Health Sciences PhD Program of The University of Manitoba.

The Biomedical Research Ethics Board of the University of Manitoba may review records related to the study for quality assurance purposes.

All records will be kept in a locked secure area and only those persons identified will have access to these records. If any of your research records need to be copied to any of the above, your name and all identifying information will be removed.

Voluntary Participation/Withdrawal from the Study

Your decision to take part in this study is voluntary. You may refuse to participate, or you may withdraw from the study at any time. ***Your decision not to participate or withdraw from the study will not affect your chiropractic care.*** *If the study staff feels that it is in your best interest to withdraw you from the study, they will remove you without your consent.*

We will tell you about any new information that may affect your health, welfare or willingness to stay in the study.

Health Care for Injury Related to the Study

In the case of injury resulting from this study, necessary treatment will be available at no additional cost to you.

You are not waiving any of your legal rights by signing this consent form or releasing the investigator(s), or the sponsors(s) from their legal and professional responsibilities.

Questions

You are free to ask any questions that you may have about your treatment and your rights as a research participant. If any questions come up during or after the study, or if you have a research-related injury, contact the study principal investigator: Dr. Geoff Gelley at (204)254-0130 or umgelley@myumanitoba.ca.

For questions about your rights as a research participant, you may contact The University of Manitoba, Bannatyne Campus, Biomedical Research Ethics Board at (204) 789-3255.

Do not sign this consent form unless you have had a chance to ask questions and have received satisfactory answers to all your questions.

Statement of Consent

I have read this consent form. I have had the opportunity to discuss this research study with the research study staff. I have had my questions answered by them in language I understand. The risks and benefits have been explained to me. I believe that I have not been unduly influenced by any study team member to participate in the research study by any statements, or implied statements. Any relationship (such as employer, supervisor or family member) I have with the study team has not affected my decision to participate. I understand that I will be given a copy of this consent form after signing it. I understand that my participation in this study is voluntary and that I may choose to withdraw at any time. I freely agree to participate in this research study.

I understand that information regarding my personal identity will be kept confidential, but that confidentiality is not guaranteed. I authorize the inspection of any of my records

that relate to this study by The University of Manitoba Research Ethics Board for quality assurance purposes.

By signing this consent form, I have not waived any legal rights that I have as a participant in a research study.

I agree to be contacted for future follow-up in relation to this study: Yes__ No__

Participant signature _____ Date _____

Participant printed name _____

Relationship (if any) to study team members _____

I, the undersigned, have fully explained the relevant details of this research study to the participant named above and believe that the participant has understood and has knowingly given their consent.

Person obtaining consent _____ Date _____

Person obtaining consent
printed name _____

COVID-19 Consent

Research Project Title: Motor performance of asymptomatic and chronic neck pain participants pre- and post- spinal manipulation using an eye and head movement Fitts' task.

Principal Investigator and contact information: Geoff Gelley, DC, MSc, FRCCSS(C)
12-845 Dakota St
Winnipeg, MB R2M 5M3
(204) 254-0130
umgelley@myumanitoba.ca

Research Advisor and contact information: Brian MacNeil, PhD
(204) 977-5635
brian.macneil@umanitoba.ca

Co- Advisor and contact information: Steven Passmore, DC, PhD
(204) 474- 6552
steven.passmore@umanitoba.ca

This document contains important information about in-person research during the COVID-19 public health crisis. COVID-19 (also called SARS-CoV2) is an illness caused by the coronavirus. Coronaviruses are most commonly spread from an infected person through a) respiratory droplets when you cough or sneeze; b) close personal contact, such as touching or shaking hands; or c) touching something with the virus on it, then touching your eyes, nose or mouth before washing your hands.

The University of Manitoba is committed to taking measures to protect the health and safety of their campuses and the wider community. Your safety is important to us. The university has suspended most research that cannot be conducted remotely or virtually. This project requires in-person visits. Therefore, it is important to understand that your participation in this study may increase your exposure to COVID-19.

Our project has been approved to proceed by the Research Ethics Board, our Faculty, the COVID Recovery Response Team, the COVID Recovery Steering Committee, and the University Provost. To gain approval, we created policies to ensure the safety of the research team and participants. These plans were reviewed and approved by the parties above. These precautions include:

- *All study research personnel are wearing 3-ply reusable or disposable masks. You are also required to wear a mask.*
- *We also require all our employees to screen themselves for symptoms daily before they come into work, and we will screen you for symptoms the day of your visit.*
- *We will provide you with a screening questionnaire. Please complete this on the morning of your visit. If you answer yes to any of the questions, please call us to reschedule your visit. You should not attend any visits if you are not feeling well or exhibiting any symptoms of COVID-19, if you have been a close contact with someone with COVID-19 (or awaiting tests results) or have been told by a health official to self-isolate.*
- *We are following meticulous infection control practices, including disinfection, wearing gloves, and hand washing.*
- *We are limiting the number of visitors accompanying people for their study visits and have rearranged and/or removed furniture in our waiting areas to enforce strict physical distancing practices.*
- *We ask that you attend the study alone. Accommodation can be made if necessary. Please let us know ahead of time if you require someone to attend with you.*
- *We are also being careful about who we ask to come for in-person study visits, and when possible are using telephone calls to reduce the number of study participants coming to our research areas at the same time.*
- *Please use the parking lot in front of the clinic and phone (204) 254-0130 to let us know you have arrived, and we will let you know when you can enter the clinic. You must wear a mask upon entering the clinic. If you do not have a mask, one will be provided for you.*

COVID-19 is a serious health threat, and the situation is evolving rapidly. If you feel that you are from a group that is more vulnerable to COVID-19 effects (e.g., senior (over the age of 60 years), immuno-compromised), please discuss your participation with the research team before

providing your consent. You are under no obligation to participate and can change your mind about participating in the research at any time and without consequence.

The University of Manitoba is closely watching the situation in Manitoba and may restrict in-person research at any time. We will continue to keep you informed as to changes that may occur to this study.

There is a possibility that during your participation in the study you could encounter someone with COVID-19. We are required to collect your personal contact information that we must retain in order to follow up with you and/or conduct contact tracing if you may have been exposed to COVID-19 in coming to the research site. ***We cannot guarantee anonymity as the personal contact information identifies you as a participant and we may be required to disclose this information in the event of possible exposure.*** *Your contact information will be kept separately from data collected through the research study to allow for de-identification of the research data. You maintain your right to withdraw from the study at any time, including your research data. If you do withdraw from the study, we will still need to continue to maintain your contact information and will only give it to the University's Environmental Health and Safety (EHS) Office and/or Manitoba Health if required for contact tracing. Please note, Manitoba Health or the University's EHS office will not have access to your research data.*

If you have questions regarding this study, measures we are taking to keep all parties safe, or have any concerns, please do not hesitate to ask. You can contact any of the above-named researchers or the Bannatyne Campus Research Ethics Board office at shelly.rempel-rossum@umanitoba.ca

Your signature on this form indicates that you have understood to your satisfaction the information regarding participation and the COVID-19 risk and agree to participate. In no way does this waive your legal rights nor release the researchers, sponsors, or involved institutions from their legal and professional responsibilities. Your continued participation should be as informed as your initial consent, so you should feel free to ask for clarification or new information throughout your participation.

Participant Name _____

Contact Information (phone # or email): _____

Participant's Signature _____ *Date:* _____