Long-term effects of concussion: Investigating the relationship between sensory gating and motor learning

by

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Author's Declaration

I hereby declare that I am the sole author of this thesis. This is a true copy of the thesis, including any required final revisions, as accepted by my examiners.

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Abstract

Motor learning relies on the sensorimotor system to interpret, adapt, and integrate sensory inputs to guide motor behavior. A key tenet of the somatosensory system is the ability to inhibit irrelevant information from the CNS to protect higher cortical areas from information overload (sensory gating). Movement and attention can contribute to this process through movementrelated gating and relevancy-based facilitation, respectively. Sensory gating occurs at early cortical processing stages and has been shown to be impacted in individuals with a history of concussion. Past work from our lab found a delay in relevancy-based facilitation in a group with a history of concussion compared to healthy controls. The current work aimed to understand the behavioral manifestations that result from disruptions to relevancy-based gating modulations at early cortical processing stages in the concussion population. A total of 40 participants were recruited to participate in this study with 25 in the concussion history group (Hx) and 15 in the control group (No-Hx). This study consisted of 2 experimental sessions that occurred 24 hours apart. During session 1, somatosensory-evoked potentials (SEPs) were elicited via median nerve stimulation while subjects performed a task that manipulated their focus of attention toward or away from proprioceptive feedback. Subjects then completed an implicit motor sequence learning task relying solely on proprioceptive cues. Individuals performed a retention test at session 2, followed by a visual attentional blink (AB) task. The AB is a phenomenon elicited by the rapid presentation of sequential targets which results in reduced accuracy of detecting the second target at the expense of detecting the first target. The No-Hx performed the implicit learning task at session 1 and a retention test at session 2 because SEP and AB data were compared to control data previously collected by this lab. SEP data replicated past work showing an absence of relevancy-based facilitation at early cortical processing stages (N20-P27) that

emerged at later processing stages. Our Hx showed evidence of relevancy-based facilitation at either the P50-N70 or the N70-P100 consistent with past work that found this to occur at the N70-P100. Performance on the learning task was not significantly different between the Hx and No-Hx. Performance on the AB task revealed greater AB magnitude in the Hx compared to the No-Hx. Collectively, these results suggest a compensatory strategy in the Hx that enables them to learn to the same degree as controls. However, when the attentional system is taxed with high temporal demands there are decrements in performance. These results are of particular importance given that these individuals are at an increased risk of sustaining subsequent concussions, and musculoskeletal injuries.

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List of abbreviations

AB: Attentional blink
AMPA: alpha-amino-3-hydroxy-5-methyl-4-isoazolepropionic
APB: Abductor pollicis brevis
AtDCS: Anodal transcranial direct current stimulation
ATP: Adenosine triphosphate
BA: Brodmann area
CaMKII: Calcium-dependent protein kinase II
CSP: Cortical silent period
cTBS: Continuous theta burst stimulation
DCML: Dorsal column medial lemniscus system
DLPFC: Dorsolateral prefrontal cortex
EEG: Electroencephalography
EMG: Electromyography
ERN: Error-related negativity
ERP: Event-related potential
fMRI: Functional magnetic resonance imaging
ISI: Inter-stimulus interval
lPFC: Lateral prefrontal cortex
LICI: Long-interval intracortical inhibition
LTD: Long-term depression
LTP: Long-term potentiation
MEP: Motor evoked potential
MI: Primary motor cortex
mPFC: Medial prefrontal cortex
Ne: Error negativity
NMDA: N-methyl-D-aspartate
PAS: Paired associative stimulation
PFC: Prefrontal cortex
preSMA: Pre-supplementary motor area
RMSE: Root mean square error
RSVP: Rapid serial visual presentation
rTMS: repetitive transcranial magnetic stimulation
SI: Primary somatosensory cortex
SII: Secondary somatosensory cortex
SEP: Somatosensory evoked potential
SMA: Supplementary motor area
SRT: Serial reaction time
SVIPT: Sequential visual isometric pinch task
TBI: Traumatic brain injury
TMS: Transcranial magnetic stimulation
VPL: Ventral posterior lateral

1.0 INTRODUCTION

Concussive brain injuries are the result of direct or indirect biomechanical forces acting on the head which result in widespread neurological and functional disturbances (McCrory et al., 2017; Romeu-Mejia et al., 2019). While symptoms typically resolve within 7-10 days postinjury, evidence for long-term deficits in asymptomatic individuals with a history of concussion is an emerging theme in the literature (Giza & Hovda, 2014; Chmielewski et al., 2021).

The ability to interact with our environment is an essential component of performing activities of daily living. When actions are repeatedly performed, such as pouring your morning coffee, this leads to relatively permanent changes in the involved brain regions that govern that skilled behavior (motor learning). The neurophysiological underpinnings that enable us to do so rely upon the nervous system's ability to interpret, adapt, and integrate sensory inputs to guide motor behavior. The brain must also attenuate irrelevant afferent stimuli (sensory gating) so the system is not overloaded and can focus on the accurate and relevant sensory information needed to guide motor output.

These processes – motor learning and sensory gating – have been shown to be impacted in asymptomatic individuals with a history of concussion. Whether these processes are related, has yet to be elucidated. Given the pertinent role of sensory feedback in guiding voluntary movement and updating motor plans, perhaps a relationship exists between observed deficiencies in sensory gating and motor learning in asymptomatic individuals with a history of concussion. This study, therefore, aims to investigate this knowledge gap.

Investigating these processes will deepen our understanding of the long-term effects of concussions. This is of particular importance given the impact these deficiencies may have upon

11 activities of daily living, as well as the observation that these individuals are at an increased risk of sustaining subsequent concussions, and musculoskeletal injuries.

2.0 LITERATURE REVIEW

2.1 Concussion

2.1.1 Definition & Epidemiology

The most recent consensus statement defines a concussion as a type of functional traumatic brain injury (TBI) in which an external biomechanical force applied directly or indirectly to an individual causes a functional disturbance and/or microstructural injury (McCrory et al., 2017), with the absence of macroscopic injury (Giza & Hovda 2014; MacFarlane & Glenn, 2015). Functional disturbances describe disruptions of cellular or physiological function. This may include ionic shifts, metabolic changes, and impaired neurotransmission. Microstructural injuries describe microscopic changes at the cellular level only detectable through advanced imaging techniques, such as diffusion tensor imaging (Giza & Hovda, 2014; MacFarlane & Glenn, 2015).

Concussive brain injuries are a growing public health concern (World Health Organization, 2006) estimated to affect 6 per 1000 people a year (Hon et al., 2019). However, due to a lack of consensus in the defining criteria of concussion and under-reporting of injuries, it is difficult to capture the true incidence of the condition (Cassidy et al., 2004). Of particular concern, athletes with a history of concussion have an increased risk of sustaining subsequent concussions and musculoskeletal injuries when returning to play (Giza & Hovda, 2014; Chmielewski et al., 2021; MacFarlane & Glenn, 2015). This finding is suggestive of long-term functional alterations not detectable by standard clinical tests.

Concussions commonly result in the sudden onset of transient neurological changes and dysfunction. However, evidence of long-lasting deficits is an emerging finding in the literature. The resultant symptoms of concussion are thought to reflect underlying injury to the brain (see section 2.1.2) and are largely tied to functional disturbances as opposed to structural abnormalities (McCrory et al., 2017).

2.1.2 Pathophysiology & Symptomatology

A complex cascade of events is initiated immediately following the concussive blow, leading to acute and chronic consequences. This is termed the neurometabolic cascade of concussion, which may underly the host of clinical symptomatology (Giza & Hovda 2014; MacFarlane & Glenn, 2015). First elucidated in animal models, concussion continues to be established in human studies as a neurometabolic cascade of events involving "bioenergetic challenges, cytoskeletal and axonal alterations, impairments in neurotransmission, vulnerability to delayed cell death, and chronic dysfunction" (Giza & Hovda, 2014).

Biomechanical injury leads to mechanoporation of neuronal lipid membranes, which in turn leads to potassium efflux, and sodium and calcium influx (ionic flux) (Giza & Hovda, 2014). This ionic flux triggers voltage- and/or ligand-gated ion channels which cause a diffuse "spreading depression-like" state. This process is thought to underly acute post-concussive impairments such as migraine headaches, photophobia, and phonophobia (Giza & Hovda, 2014). As a result of the ionic flux, adenosine triphosphate (ATP)-requiring membrane ionic pumps must work continuously to restore cellular homeostasis, leading to an imbalance between energy supply and demand. The calcium influx, which occurs early and persists longer relative to the other ionic disturbances, results in calcium sequestration into mitochondria to restore homeostasis. This results in mitochondrial dysfunction, which inhibits oxidative metabolism and further exacerbates the energy crisis. This increased stress causes a shift in metabolic pathways and generates free radicals which are thought to underly vulnerability to repeated injury, particularly within the first 10 days postinjury (Giza & Hovda, 2014). Following the initial stage of hyperglycolysis and metabolic uncoupling, glucose metabolic rates enter a stage of impaired metabolism lasting 7-10 days that has been linked to impaired spatial learning (Giza & Hovda, 2014). In addition, biomechanical stretching damages neurofilaments and microtubules which

provoke axonal dysfunction and/or disconnection which has been linked to impaired cognition and slowed reaction time (Giza & Hovda, 2014).

2.2 Motor Learning

2.2.1 Techniques & Definitions

This section provides a brief overview of motor learning as numerous studies have identified motor learning abnormalities in the concussion population. Motor learning describes a set of internal processes, driven by practice or experience, that leads to a relatively permanent change in motor behavior (plateau) (Krakauer et al., 2019). Generally, motor learning involves procedural or implicit learning. Implicit learning occurs without subjects' explicit awareness and subjects are typically unable to articulate what they have learned. Procedural learning describes the phenomena of acquiring the ability to perform a skill, such as learning to ride a bike. On the other hand, explicit motor learning describes conscious awareness of a learned behavior. An example of explicit motor learning would be a basketball player being coached on shooting a free throw. The key distinction between implicit and explicit learning is the conscious awareness of the strategies and movements involved. As this study involves implicit learning of a motor sequence, this section will focus on the neurological underpinnings of this type of learning.

Continuous sequential actions, such as the learning task employed in this study, requires that the motor system learns to initiate the correct time-varying sequence of actions to accomplish an action with accuracy (Krakauer et al., 2019). Improvement in this type of learning task can come from two sources: sequence-specific learning and general improvement/understanding of task dynamics (Vidoni & Boyd, 2009). Sequence-specific learning involves the acquisition and retention of the ability to perform a specific pattern of movements. This reflects learning the transitional muscle activations required to perform the task with accuracy. Acquisition describes a

type of online learning whereby an individual acquires a motor skill. Retention refers to a sustained change in ability that is typically measured after allowing time for the storage and consolidation of a learned skill. On the other hand, general improvement/understanding of task dynamics involves improvement on the nonspecific elements of the task and therefore reflects general task learning. An example used by Vidoni & Boyd
(2009) describes sequence-specific learning as signing one's name, where proper execution of one letter leads to the execution of the next. Whereas general improvement/understanding of task dynamics reflects learning the required grip force to hold a pen. The use of a random (i.e different every time) and repeated (i.e., identical every time) sequence in learning tasks enables researchers to identify the learning that is specific to the practice of the motor sequence as opposed to learning that arises from familiarity with the task and is more generalizable (Vidoni & Boyd, 2009).

2.2.1 Overview of Neuroanatomical & Neurophysiological Mechanisms of Motor Learning

Long-term potentiation (LTP) and long-term depression (LTD) are forms of synaptic plasticity, critical to the initial stages of motor learning that enable sustained changes in ability (Cirillo, 2021). LTP is a process by which synaptic connections between neurons become stronger with frequent activation. Conversely, LTD is a process by which the strength of synaptic connections decreases due to infrequent activation. One form of synaptic plasticity believed to underpin motor learning is N-methyl-D-aspartate (NMDA) receptor-dependent LTP (Malenka & Bear, 2004). This form of LTP is initiated by the release of glutamate from the pre-synaptic cell which binds to alpha-amino-3-hydroxy-5-methyl-4-isoazolepropionic (AMPA) receptors on the post-synaptic cell. Activation of AMPA receptors allows sodium (Na+) to enter the cell causing the post-synaptic neuron to depolarize. This post-synaptic potential expels magnesium (Mg2+) from the voltage-gated NMDA receptor channel, allowing calcium (Ca2+) and sodium (Na+) to

enter the cell. The rapid influx of Ca2+ activates calcium-dependent protein kinase II (CaMKII) which initiates the phosphorylation of additional AMPA receptors on the post-synaptic cell. The increased quantity of AMPA receptors on the post-synaptic cell allows more glutamate to bind to its receptors, therefore facilitating the opening of more NMDA receptors at a given time. This ultimately leads to a larger excitatory post-synaptic potential. In addition, persistent activity between nerve cells can lead to the creation of new synapses (synaptogenesis). In contrast, the slow influx of Ca2+ initiates mechanisms of LTD. This slow influx of Ca2+ activates calcineurin which removes AMPA receptors from the post-synaptic cell membrane.

Motor learning involves vast brain regions, including non-primary motor regions. First, the frontal cortex directs attention to task-relevant stimuli. The supplementary motor area (SMA) and pre-supplementary motor area (preSMA) are key regions involved in sequence learning (Krakauer et al., 2019). Neurons in preSMA and SMA can be (1) sequence selective, (2) movement selective, or (3) rank-order selective. Sequence selective neurons will fire prior to the initiating of a sequential action. Movement-selective neurons fire prior to a specific sub-element of a sequence and are most prominent within the SMA. Rank-order selective neurons fire prior to the temporal position of a sub-element of a sequence, regardless of what that sub-element is. Rank order neurons are most prominent in the pre-SMA. The SMA is primarily involved in the planning and control of movement sequences, whereas the preSMA is primarily involved in the learning phase of a sequence (Krakauer et al., 2019).

In addition, the basal nuclei are thought to play a role in modulating sequence-specific performance. Krakauer and colleagues (2019), suggest that the basal nuclei contribute to the organization of discrete elements into a sequence and the automaticity with which the sequential actions are performed. Furthermore, the authors suggest that the basal nuclei control the discharge of an action sequence, as opposed to storing or initiating the entire sequence itself.

The cerebellum is believed to contribute to the process of learning before the changes become relatively permanent in the primary motor cortex (MI) (Krakauer et al., 2019). The cerebellum contributes to motor control and learning in two major ways: (1) through the use of a forward model, and (2) by means of online feedback control (Krakauer et al., 2019; Scott, 2004). The forward model enables the cerebellum to act in advance of sensory feedback arising from movement by relying on an internal model to create a feedforward control for movement. Through online feedback control, the cerebellum integrates the efferent copy and sensory feedback (via ascending spinocerebellar tracts) and projects to motor planning regions of the cortex to update the motor plan originating in M1. In addition, the lateral cerebellum, in conjunction with parietal and cingulate motor cortical areas, is thought to be involved in the storage and refinement of newly acquired sensorimotor representations (Seidler, 2010).

The M1 is a dynamic and integral component in the control of voluntary movement and motor learning. Permanent changes induced by motor learning are typically reflected by changes in this brain region. Transient levels of activation in GABAergic neurons in M1 influence LTP-like mechanisms; increased levels of GABAergic inhibition prior to motor learning reduces LTP-like mechanisms, whereas reduced levels of GABAergic inhibition prior to motor learning promotes LTP-like mechanisms (Cirillo, 2021). The somatotopic organization in M1 can be modified by experience (use-dependent plasticity) and is thus an optimal site for motor learning (Cirillo, 2021) and motor acuity (Krakauer et al., 2019).

2.2.3 Concussion & Motor Learning

De Beaumont and colleagues (2012) used a paired associative stimulation (PAS) protocol to investigate whether increased GABAergic inhibition contributes to motor learning impairments in individuals with a history of repeated concussions via its effects on LTP/LTD-like mechanisms.

The sample consisted of 13 active football athletes aged 19-27 years who sustained at least 2 concussions at least 9 months prior to testing. PAS is a non-invasive brain stimulation technique that involves paired stimulation over a peripheral nerve with single-pulse transcranial magnetic stimulation (TMS) over contralateral M1. The resulting action potential barrage ascends through the dorsal column medial lemniscus system (DCML) and arrives at the primary somatosensory cortex (SI) which then projects to M1. Single-pulse TMS is applied at the instant the signal arrives at M1. The underlying theory is that repeated paired stimulation will produce a plastic alteration of the excitability of the receptive motor neurons in M1, which essentially mimics LTP/LTD-like mechanisms. De Beaumont et al. (2012) used a modified version of the serial reaction time (SRT) task consisting of 10 sequence blocks and 4 random blocks. Sequence-specific learning was assessed by contrasting participants' median reaction time across sequence block 10 and random block 4. The total training-related learning was assessed by contrasting median response time across sequence blocks 1 through 10. In each trial, participants had to respond to a symbol on the computer screen with the corresponding finger, the learning component was in the sequence of symbols presented that subsequently required implicit learning of a patterned motor output (sequence-specific blocks). Relative to healthy controls, De Beaumont et al. (2012) found that athletes with a history of multiple concussions displayed attenuated PAS-induced LTP/LTD-like plasticity which was correlated with decreased implicit motor learning in the SRT task as indexed by slower reaction time. However, there were no significant differences in response accuracy between groups. The reduced effect of PAS was also accompanied by enhanced long-interval intra-cortical inhibition (LICI) and longer cortical silent period (CSP). LICI and CSP are both inhibitory measures thought to be modulated by $GABA_B$ receptor activity (Lefebvre et al., 2015). The authors interpret this finding to be indicative that enhanced GABAergic inhibition in persons with a history of concussion might be partly related to

observed deficiencies in motor learning. Findings from this study may imply that repeated concussions cause a functional disturbance to the mechanisms that underpin adaptation and motor learning.

Beaulieu and colleagues (2019) investigated motor sequence learning using the SRT task in asymptomatic athletes with a history of multiple concussions to determine whether concussive effects on cognitive control were associated with sequence learning. Electroencephalography (EEG) was continuously recorded in all participants to capture the Error Negativity (Ne), also known as Error-related Negativity (ERN). The Ne/ERN is a type of event-related potential (ERP) that occurs 50-100 ms following an overt error. The Ne/ERN is thought to reflect a signal from the anterior cingulate cortex and supplementary motor regions of the medial prefrontal cortex (mPFC) to the lateral prefrontal cortex (lPFC) indicating an error has occurred. The Ne/ERN thus reflects the cognitive operations involved in top-down processes concerned with improving performance and promoting learning (De Beaumont et al., 2012; 2013; Brush et al., 2018; Beaulieu et al., 2019). The authors' results indicate that the Ne/ERN amplitude was significantly attenuated with task progression in athletes with a history of concussion, but not healthy controls, despite evidence of motor learning indexed by decreased reaction times relative to baseline in both groups. However, relative to controls, athletes with a history of concussion displayed slower reaction times during the SRT task. This may suggest that motor sequence learning in athletes with a history of concussion was not associated with modulation of the Ne/ERN amplitude. Given that the Ne/ERN reflects awareness of an error, lack of modulation of this ERP may imply that individuals with a history of concussion do not rely on cognitive control processes during a motor learning task.

Investigating implicit motor learning in adults (aged 50-70 years) with a history of concussion (experienced 3-24 months prior to testing), Bourassa and colleagues (2021) used a

modified version of the SRT task described previously (De Beaumont et al., 2012; 2013). Of interest, this is the only study discussed that recruited participants based on a physician's diagnosis of concussion, as opposed to self-reporting in the studies described previously (De Beaumont et al., 2012; 2013, Tremblay et al., 2014; Beaulieu et al., 2019; Cantarero et al., 2020). Relative to age and education-matched controls, the concussion history group improved significantly less than controls at sequence blocks of the SRT task (training-related learning). This finding implies that the concussion history group did not reach the same level of learning (indexed by decreased reaction time) as controls when training on a repeated sequence. However, there was no significant difference between groups on sequence-specific learning (sequence block 10 – random block 4). This lack of between-group differences is in stark contrast to findings from studies using the same task (De Beaumont et al., 2012; 2013). The authors interpreted this observation as being due to a selection bias induced by strict recruitment criteria that led to a sample that was healthier than the general population. Furthermore, participants in the present study had sustained a maximum of 1 concussion, whereas participants in the aforementioned studies (De Beaumont et al., 2012; 2013) had sustained multiple concussions. This finding implies that a history of multiple concussions might be a critical factor in observed motor learning deficiencies and that a single concussion might not impact motor learning to the same extent.

In a study by Cantarero et al (2020), researchers investigated the relationship between concussion acuity on motor learning. In this study, participants were separated into three groups: (1) athletes with no history of concussion, (2) athletes in the acute stage of concussion (<2 weeks post-concussion), and (3) athletes in the chronic state of concussion (> 1-year post-concussion). The experiment was divided into a training day and a retention day. On both days, participants performed the Sequential Visual Isometric Pinch Task (SVIPT), where participants had to

squeeze an isometric force transducer between the thumb and index finger to control the movement of a cursor. The learning component was the force output needed to navigate from a 'home' position through 5 'gates' quickly and accurately. The speed-accuracy trade-off was used to assess behavioral performance. Retention was also measured using neurophysiological measures of occlusion – transient blocking of artificially induced LTP-like plasticity following learning which indicates the usage of neuroplasticity during learning (Cantarero et al., 2021). To measure levels of occlusion of LTP-like plasticity, the authors used TMS to then quantify motor evoked potential (MEP) amplitude changes in M1, as well as anodal transcranial direct current stimulation (AtDCS) to induce LTP-like plasticity in M1. MEP amplitudes were recorded before, immediately after, and at 5-minute increments (up to 25 minutes) following AtDCS to obtain participants' baseline. This process of MEP recordings following AtDCS was repeated immediately after training on the SVIPT on a separate training day. The authors defined occlusion as the aftereffects evoked by AtDCS on MEP amplitude after motor training.

Cantarero et al. (2020) found that participants in both the acute and chronic stages of concussion, relative to healthy controls, had impaired occlusion as well as reduced retention of the motor skill that was correlated with the number of previous concussions. Results from this study show that impaired motor learning exists as early as 2 weeks post-concussion and that this continues to manifest 1-year post-concussion.

Taken together, results from these studies are mixed. While some suggest that concussive brain injuries result in functional disturbances to the brain regions involved in motor skill learning, others show similar performance between concussed and healthy controls.

2.3 Overview of Somatosensory System in the Upper Limb

The somatosensory system is a critical neuronal network underpinning human perception and movement. This section provides an overview of the somatosensory system as it relates to the nature of the learning task and sensory gating.

Somatosensation begins at the level of peripheral receptors (mechanoreceptors) that convert stimuli into electrical signals, a process called transduction. For proprioception, muscle spindle fibers, Golgi tendon organs, and joint capsules signal a change in muscle length, muscle force, and joint angle, respectively. For cutaneous (touch) sensation, four distinct mechanoreceptors respond to various types of stimuli in terms of magnitude, and how that stimulus changes with time. The local stimulus that is transduced by the appropriate mechanoreceptor transmits this information to first-order neurons (large diameter afferent fibers) which enter the spinal cord via the dorsal root. The majority of these neurons ascend to the medulla, via the dorsal columns of the spinal cord, where they synapse onto the cuneate nucleus in the medulla. The second-order neurons decussate (internal arcuate fibers) in the tegmentum and ascend via the medial lemniscus to synapse onto neurons in the ventral posterior lateral (VPL) nucleus of the thalamus. From there, third-order neurons project as the internal capsule and the corona radiata (once the fibers leave the thalamus) to synapse with neurons in layer IV of the primary somatosensory cortex (S1). The route taken by these ascending afferents is referred to as the DCML pathway. Housed within S1 are three functional areas: Brodmann areas (BAs) 1, 2, and 3, which are further subdivided into 3a and 3b. BA 3a, in conjunction with area 1, receives proprioceptive information, while area 3b receives cutaneous information. It should also be noted that both S1 and the DCML are arranged somatotopically.

2.3.1. Importance of Somatosensory Feedback to Motor Learning

Somatosensory feedback plays a critical role not only in guiding voluntary movement and updating motor plans but also in motor learning. Learning new motor skills requires an adaptive neural system, relying heavily on the sensorimotor system to adapt to changing body size, position, or environmental parameters. The axons of neurons in layer V of the primary motor cortex provide the major output of the neocortex to control movement via projections in the corticospinal tract to motor neurons in the ventral horn of the spinal cord. Motor information carried in the corticospinal tract is modulated by sensory information and information from other motor regions such as the cerebellum and basal nuclei. Vidoni et al. (2010) demonstrated the role of S1 in motor learning. In their study, researchers employed 1 Hz repetitive transcranial magnetic stimulation (rTMS) over S1 to transiently disrupt cutaneous somatosensation and proprioception in the wrist/hand during an implicit motor learning task. The purpose of 1 Hz rTMS was to transiently suppress cortical excitability, meaning a reduction in sensory afferents being able to excite S1. Compared to a control group that received sham rTMS, the experimental group displayed poorer performance in the learning task, showed more errors, and poorer retention of the learned motor behavior on a day where no rTMS was administered. This shows that altered somatosensation impairs motor learning, therefore somatosensation is an integral component involved in motor learning. Furthermore, altered somatosensation during learning may lead to an inaccurate motor plan (or internal model) and thus hinder the acquisition of the motor skill. This has important implications as it relates to the system's need for accurate somatosensation during learning, which may be confounded by an overabundance of stimuli not being attenuated and/or facilitated.

2.4 Sensory Gating

2.4.1 Movement-Related Gating & Relevancy-Based Facilitation

Sensory gating describes the intrinsic ability of the brain to attenuate irrelevant afferent stimuli. The purpose of this is so as not to overload the system with an overabundance of incoming sensory afferents, allowing the most relevant information to result in the greatest response in the S1. Sensory gating occurs at multiple levels of the nervous system. This includes peripheral receptors, the spinal cord, the thalamus, as well as various cortical regions (Knight et al., 1999). In addition, sensory gating can be influenced by movement (both passive and active), as well as task relevancy.

Movement-related gating describes the attenuation of somatosensory responses, measured by the amplitude of SEPs, to stimuli during movement (Brown et al., 2015). This occurs during both active and passive movements, evidenced by smaller SEPs compared to a rest condition. However, if the sensory feedback is task-relevant, greater activity is seen in S1 (Brown et al., 2015; Tennant et al., 2021; Knight et al., 1999).

Movement-related gating results from peripheral (centripetal) and central (centrifugal) mechanisms. Centripetal gating describes a bottom-up gating mechanism arising from sensory receptor discharge (reafference) (Brooke et al., 2004). In contrast, centrifugal gating describes a top-down gating mechanism proceeding out from motor control centers. Cortical mechanisms of movement-related gating are thought to arise from the influence of efferent projections from M1 on sensory afferents at the level of the spinal cord by altering levels of primary afferent depolarization, at the level of the dorsal column nuclei via presynaptic inhibition, as well as thalamically through connections with the thalamic reticular nucleus and VPL of the thalamus (Knight et al., 1999; Brown et al., 2015).

Attentional mechanisms have also been shown to influence sensory gating both during movement and at rest. The prefrontal cortex (PFC) has been shown to modulate somatosensory information during movement-related gating (Brown et al., 2015). In their study, researchers employed continuous theta burst stimulation (cTBS) to transiently inhibit the DLPFC. Brown et al. (2015) manipulated the relevance of somatosensory afferents during passive wrist movements. Compared to participants' baseline SEPs (pre-cTBS), SEPs following cTBS over DLPFC resulted in attenuated SEPs during task-relevant conditions. Results from this study demonstrate the important role of the PFC in relevancy-based facilitation during movement-related gating. This is an important consideration given that the frontal lobe is particularly vulnerable to concussive forces (Eierud et al., 2014) and past work (discussed in the next section) has found evidence of aberrant sensory gating abilities in the concussion population.

2.4.1 Concussion & Sensory Gating

Research in persons with a history of concussion has demonstrated a disruption in the mechanistic selection of relevant information (sensory gating) to guide the motor act (Adams et al., 2020; Tennant et al., 2021). In one study, Adams and colleagues (2020) used EEG to capture ERPs in participants with and without a history of concussion during a sensory grading task. In blocks of trials, participants had to produce a scaled motor response by squeezing a pressure sensitive bulb to various amplitudes of vibrotactile or visual stimuli presented alone or concurrently. While healthy controls demonstrated an enhanced somatosensory-evoked N70 ERP in response to relevant stimuli, and attenuated N70 to irrelevant stimuli, the concussion history group did not show any modulation of the N70. Specifically, in the concussion group, the loss of modulation of the N70 was related to a decreased facilitation of relevant stimuli and reduced attenuation to irrelevant stimuli. Furthermore, Adams et al. (2020) found that the presence of a

distractor stimulus had a significant effect on task accuracy in the concussion group, but not the healthy control group. Taken together, results from this study indicate that individuals with a history of concussion have impaired sensory gating, and this influenced the accuracy of their scaled motor response.

Similar deficits in relevancy-based modulation of early ERPs and their relationship to task accuracy were observed in the same task after cTBS was applied to the contralateral dorsolateral prefrontal cortex (DLPFC) in healthy participants (Adams et al., 2019). cTBS reduces cortical excitability, therefore applying cTBS over DLPFC downregulates cortical activity in this region meaning it has a lesser effect on the areas it projects to. The authors found that the tactile-evoked N70 ERP was modulated by task relevance pre- but not post-application of cTBS to DLPFC. The N70 amplitude to both task-relevant and task-irrelevant conditions was reduced post-cTBS – driven primarily by a loss of facilitation to task-relevant stimuli. These results indicate that the N70 is modulated by attention, subserved by the DLPFC. Given the role of the DLPFC in relevancy-based facilitation shown here and by Brown and colleagues (2015), combined with the fact that the frontal lobe is particularly vulnerable to concussive forces (Eierud et al., 2014), these results may explain gating disturbances observed in the concussion population. Concussive brain injuries might disrupt the DLPFC's ability to modulate the amount of tonic inhibition it normally exerts over SI. This may result in a loss in ability to selectively facilitate relevant sensory afferents.

Similarly, Tennant and colleagues (2021) found that an individual's ability to up or downregulate afferent somatosensory information at early cortical processing stages is lost in persons with a history of concussion. EEG was used to capture somatosensory-evoked potentials (SEPs) in the contralateral S1 elicited by stimulation of the left median nerve. SEPs are a specific type of ERP that are time-locked to a sensory event, in this case, median nerve stimulation.

Subjects performed a task that manipulated their focus of attention toward (task-relevant) or away (task-irrelevant) from proprioceptive feedback. Early cortical processing stages were indexed by the N20 and P27 SEPs, which represent the arrival of somatosensory information to contralateral S1, specifically Brodmann's area 3b and area 1, as well as area 3a. Tennant et al. (2021) found that healthy controls displayed relevancy-based modulation of the N20-P27 SEPs; increased amplitudes to task-relevant somatosensory afferents and decreased amplitudes to task-irrelevant somatosensory afferents. Of interest, the concussion history group did not display relevancy based modulation of incoming somatosensory information at early cortical processing stages; N20-P27 SEP amplitudes to task-relevant and task-irrelevant somatosensory afferents were not significantly different. However, the concussion group displayed the same modulation pattern seen in the control group at the N20-P27 at the N70-P100. Findings from this study are in line with that of Adams et al (2020), where individuals with a history of concussion displayed an impaired ability to attenuate irrelevant afferent stimuli while facilitating relevant afferent stimuli based on task-relevancy. In both studies, ERP and SEP amplitudes were not modulated by task relevancy.

2.5 Attentional Blink

The speed at which individuals process successive stimuli in their environment is a critical factor underpinning voluntary actions and their outcomes. The attentional blink (AB) is a phenomenon elicited by the rapid presentation of sequential targets (200-500ms) which results in the reduced accuracy of detecting the second target (T2) at the expense of detecting the first target (T1) (Arasanz et al. 2012). Two main theories are suggested to explain the deterioration of detecting T2: Raymond et. al's (1992) Attentional Gating Theory and Chun and Potter's (1995) Two-Stage Model Theory (Zivony & Lamy, 2021). According to the Attentional Gating Theory,

the attentional processes needed for high-level perceptual processing are disrupted during the blink and T2 is therefore not identified as a target. According to the Two-Stage Model theory, the AB reflects a structural limitation in working memory (WM) encoding that occurs after T2 has been attended to and identified as a target. This view encompasses the "bottleneck" theory in which WM can only encode one item (or one chunk of items at a time), resulting in a bottleneck through which items cannot pass. This leaves T2 in a vulnerable state in which it can be overwritten by lagging stimuli before being encoded in WM (Zivony & Lamy, 2021). To our knowledge, no studies have investigated the AB in the post-concussion population. We therefore sought to examine the AB in the concussion population given the need for accurate processing of stimuli for guiding voluntary actions.

3.0 RATIONALE

Previous research has shown evidence of disturbances to sensory gating abilities in individuals with a history of concussion (Adams et al., 2020; Tennant et al., 2021). Specifically, Tennant and colleagues (2021) observed a delay in relevancy-based facilitation in a group with a history of concussion compared to healthy controls. In addition, numerous studies have shown that individuals with a history of concussion do not attain the same level of learning as healthy controls (De Beaumont et al., 2012; 2013, Beaulieu et al., 2019; Bourassa et al., 2021; Cantarero et al., 2021). Given the importance of somatosensation to motor learning (Vidoni et al. 2010), it seems plausible that disruptions to the gating system could impact motor learning abilities in this population. However, it is unknown whether the gating abilities are directly related to motor learning abilities.

The purpose of this study was to confirm disruptions to relevancy-based gating modulations at early cortical processing stages in the concussion population and identify the behavioral manifestations that result from those. An implicit motor sequence learning task that required reliance on proprioceptive cues was used to investigate motor learning ability. In addition, the AB task was used to provide insight into how the attentional system behaves when taxed with heightened attentional demands in the temporal domain. The sensory gating task used by Brown et al. (2015) and Tennant et al. (2021) was used in the present study to investigate participants' sensory gating abilities.

4.0 OBJECTIVES & HYPOTHESES

The objectives and corresponding hypotheses for this study are as follows:

Objective 1: To replicate disruptions to relevancy-based gating modulations at early cortical processing stages in the concussion population and determine whether this has an impact on motor learning.

Hypothesis 1a: Based on previous work (Tennant et al., 2021), it was hypothesized that the concussion history group would demonstrate smaller SEP amplitudes in response to task irrelevant sensory information compared to task-relevant sensory information and a rest condition at the N70-P100 but not at early cortical processing stages (N20-P27). This would be reflective of a delay in relevancy-based gating modulations as this normally occurs at the N20P27 in healthy controls.

Hypothesis 1b: Based on previous work (De Beaumont et al., 2012; 2013, Beaulieu et al., 2019; Bourassa et al., 2021; Cantarero et al., 2021), it was hypothesized that performance on the implicit motor sequence learning task that required reliance on proprioceptive inputs would be worse in the concussion history group compared to the healthy control group.

Objective 2: To understand how the attentional system behaves when taxed with heightened attentional demands in the temporal domain using the AB task.

Hypothesis 2: Given the role of the DLPFC in selective attention and sensory processing (Brown et al., 2015), combined with evidence of the frontal lobe being particularly vulnerable to concussive forces (Eierud et al., 2014), it was hypothesized that the concussion history group would display poorer performance on the AB task compared to control data.

5.0 METHODS

5.1 Participants

Sample size for the concussion group was calculated with an *a priori* power calculation using G*Power 3.1 software (v3.1.9.6; Faul, Erdfelder, Lang, & Bruchner, 2007; 2009). Using the "Linear multiple regression: Fixed model, R^2 deviation from zero" setting in the F-test family, sample size was calculated with the following specifications: large effect size f^2= 0.35, alpha error probability = 0.05, power = 0.8, and one predictor (sensory gating). This calculation yielded an *a priori* sample size of 25 participants. Sample size for the control group for the motor learning task was selected based on past work by Tennant et al. (2021) who observed differences in their concussion and control groups. We, therefore, recruited 15 participants for the control group. The control group used for the analysis of the AB data in the present study was from the pre-cTBS group from data previously collected by Arasanz and colleagues (2012). This sample consisted of 3 separate groups of 15, resulting in a total of 45 participants.

A total of 40 participants were recruited to participate in this study. Inclusion criteria for the control group were as follows: participants must have no history of neurological impairments or sensorimotor deficits, as well as have no allergies to adhesive gels or electrode pads. Additional inclusion criteria for the concussion history group were as follows: participants must have sustained at least one clinically diagnosed concussion, must have been clinically cleared to return to both physical and cognitive activities, and be free from concussion-related symptoms.

Participant demographics are summarized in Table 1. The control group consisted of 15 participants (9 females and 6 males ranging from 19-30 years, $M_{age} \pm SD_{age}$ = 21.73 $\pm$ 3.43 years). The concussion history group consisted of twenty-five individuals who sustained at least one medically diagnosed concussion (17 females and 8 males ranging from 18-30 years, $M_{age} \pm$

SD_{age} = 22.64 ± 2.89 years, $M_{concussion}$ ± $SD_{concussion}$ = 2.52 ± 2.31). On the University of Waterloo Health History Questionnaire, concussed participants reported an average symptom score of 14.64 (SD = 15.82) out of a possible 126. At the time of participation in this study, an average of 42.88 (SD = 39.71) months elapsed since the individual's most recent concussion. Informed written consent was obtained before beginning the study. This study was approved by the University of Waterloo Research Ethics Board.

	Concussion History		Control	
Demographic	Mean	SD	Mean	SD
Age (years)	22.64	2.89	21.73	3.43
Sex	8M, 17F		6M, 9F	
Medically Diagnosed Concussions	2.52	2.31	-	-
Last Concussion (months)	42.88	29.71	-	-

Table 1. Participant Demographics. SD = standard deviation.

5.2 Questionnaires

Upon arrival in the lab, participants were given an information letter and consent form to review and sign. Once consent was obtained, each participant completed the Edinburgh Handedness Questionnaire (See Appendix A) to determine hand dominance for the task. A modified version of the University of Waterloo Health History Questionnaire (See Appendix B) was given to individuals in the concussion history group to collect participant characteristics following concussion. This form has been used previously by Tennant et al. (2021) to gather information pertaining to the heterogeneous nature of concussions. The modification made by Tennant et al. (2021) includes additional questions to collect information relating to neurological conditions/disorders, or upper limb nerve injuries which could confound results, as well as the number of medically diagnosed concussions.

5.3 Experimental Design & Procedures

A schematic overview of the experimental protocol can be seen in Figure 1. This study followed a repeated measures design and consisted of two experimental sessions. For the concussion history group, experimental session 1 consisted of a sensory gating (see 5.4.1 Sensory Gating) task followed by the motor learning task (see 5.4.1 Motor Learning). Experimental session 2, which took place 24 hours after session 1 consisted of a retention test followed by completion of the attentional blink task (see 5.4.3 Attentional Blink). The control group performed the motor learning task at experimental session 1 (~1.5 hours), followed by the retention test 24 hours later at experimental session 2.

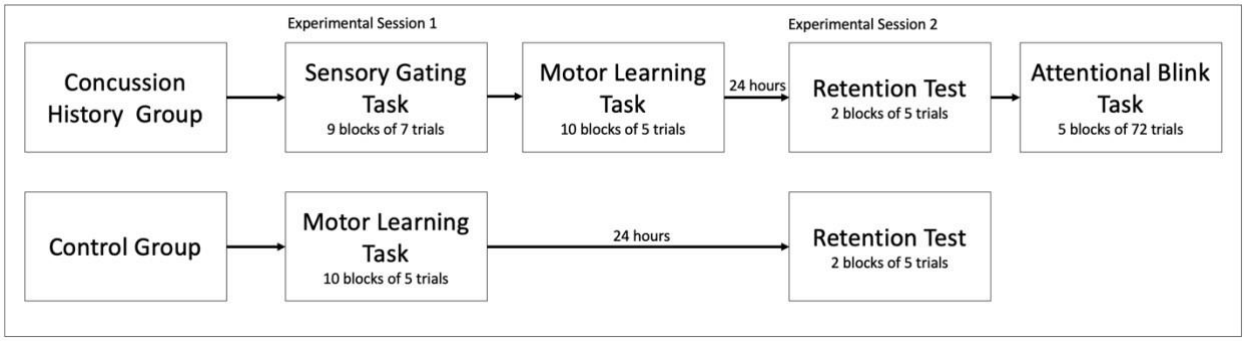

Figure 1. Experimental Protocol. The concussion group performs the sensory gating and motor learning task at experimental session 1 followed by a retention test and attentional blink task at experimental session 2 (24 hours later). The control group performs the motor learning task at experimental session 1 followed by a retention test at experimental session 2 (24 hours later).

Following consent and questionnaire completion, participants were fitted and prepped for a 32-channel EEG cap (Quik-Cap, Compumedics Neuroscan, NC, USA) worn by both the concussion and control groups for the duration of experimental session 1. To optimize the contact between the electrode and the scalp, the hair under each electrode site was moved out of the way and the skin underneath was abraded. This was done using the blunt end of a syringe. A conductive gel was injected into each of the electrodes to allow for electrical signals from the

brain to conduct from the scalp to the electrode. Preparation was complete once each electrode had an impedance of < 5 kohms.

Participants in the concussion group were also prepped for median nerve stimulation in order to generate SEPs during the sensory gating task (see 5.4.1 Sensory Gating). For this, the participant's non-dominant wrist and thumb were prepped using an abrasive gel to remove dead skin and dirt and then sterilized with alcohol. Surface electromyographic (EMG) recording electrodes were placed over the sterilized areas; two over the abductor pollicis brevis (APB). Next, a bar electrode was placed with the anode distal to APB over the sterilized area on the wrist, and the motor threshold was determined. This was done by stimulating participants' median nerve starting with imperceivable intensities and gradually increasing the intensity until a just-visible twitch was seen in APB five times out of ten consecutive trials while the muscle was at rest. Once the motor threshold was determined, the intensity was trialed with each participant to familiarize them with it and to confirm that the stimulation was tolerable and did not cause any discomfort.

Following all preparatory steps, participants performed the task(s) – described in the next section. For the duration of the task(s), participants were seated comfortably in a chair and rested the medial aspects of both forearms on a custom-made device for wrist movement in the transverse plane while grasping the handles of the device with both hands (Figure 2).

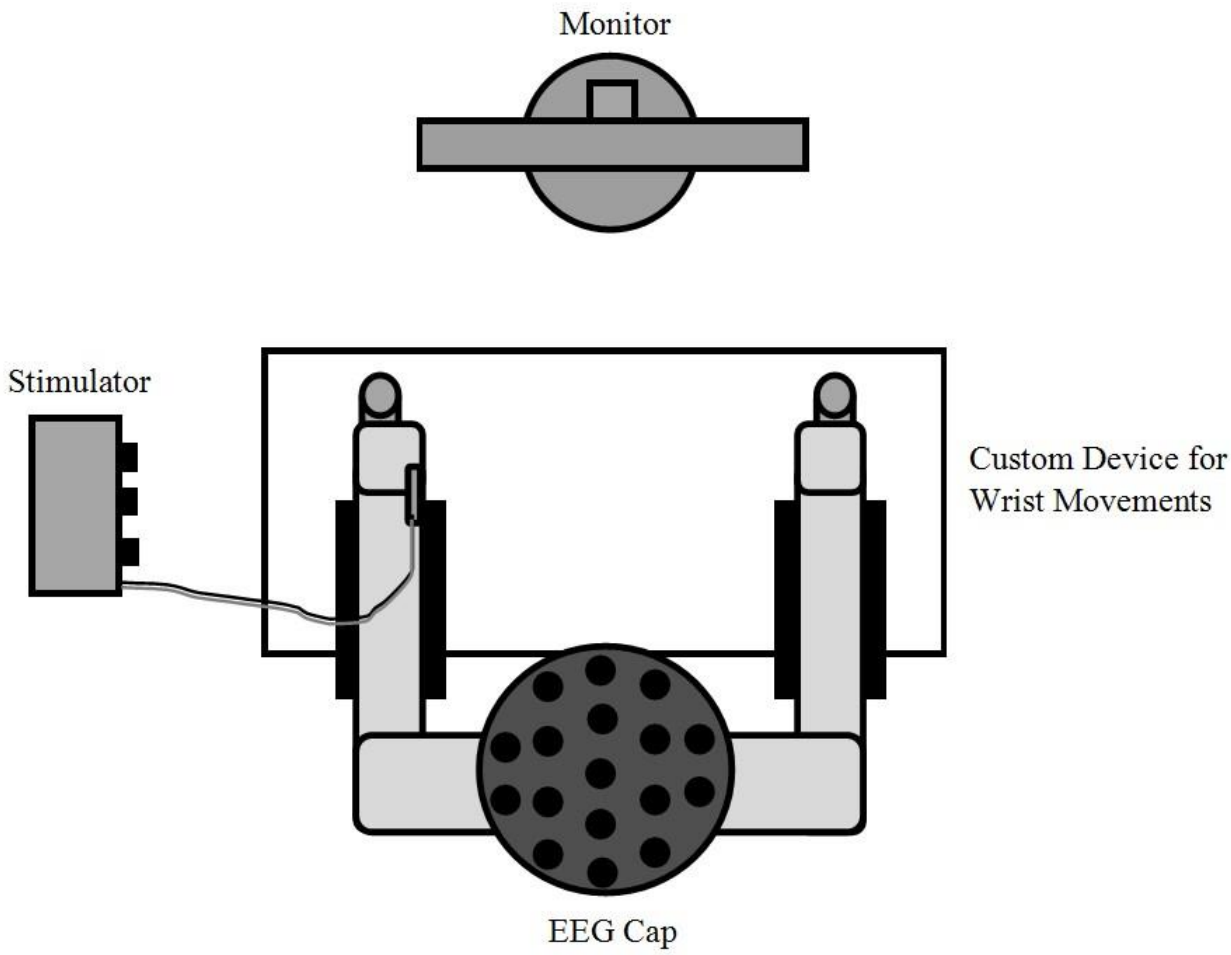

Figure 2. Experimental Setup at Experimental Session 1 (Tennant et al., 2021). Subjects were seated with bilateral aspects of medial forearms rested on a custom device for wrist movements. Concussion group only: subjects' non-dominant wrist was connected to a peripheral nerve stimulator.

5.4 Experimental Tasks

5.4.1 Sensory Gating

This task served to replicate the findings of Tennant et al. (2021) and was based on the protocol used by Brown et al. (2015) and Tennant et al. (2021). EEG was used to examine SEPs elicited by median nerve stimulation. A bar electrode was placed on the participants' nondominant wrist, just above the median nerve. A square wave pulse (0.5 ms) was delivered (Digitimer DS7R constant current stimulator, Fort Lauderdale, FL, USA) at an intensity of motor threshold. To

ensure changes in SEP amplitude were the direct result of task manipulations, Mwave amplitude in APB was recorded for offline analysis. As M-waves result from efferent nerve stimulation, this will ensure that any observed changes in SEPs are due to task manipulations as opposed to a change in the number of stimulated fibers. EEG was recorded throughout the task.

A custom LabView program (National Instruments, Austin, Texas, USA) was used to guide the experimenter during passive movements. The experimenter followed the sinusoidal trace on the computer monitor which resulted in wrist flexion and extension of participants' nondominant wrist. Participants' eyes remained closed during each movement condition (outlined below) to ensure reliance on proprioceptive feedback. Conditions were presented to participants in separate blocks, each containing 7 trials. Each condition (block) was performed three consecutive times, for a total of 9 blocks performed for this task. Subjects' eyes remained closed throughout all conditions. Nerve stimulation occurred throughout each condition and was paused when participants re-created the movement during the active condition, and during breaks. In each condition, 147-294 nerve stimuli were delivered, although the exact number may have differed for each condition and participant, as the interstimulus intervals were randomly generated between 500 and 1000 ms. The order of conditions was counterbalanced across participants. SEPs were taken during the following three conditions:

1) Rest: The subject's non-dominant wrist was stimulated while they mentally counted backward by sevens from a given starting number while maintaining their wrist in a neutral wrist position.
2) Passive: Subjects stimulated wrist was passively moved through a series of flexion and extension movements (within 60 degrees) for seven seconds while they mentally counted backward by sevens from a given starting number.

3) Active: The subject's stimulated wrist was passively moved through a series of flexion and extension movements (within 60 degrees) for seven seconds. Once their wrist was returned to the neutral starting position, subjects were instructed to mirror the passive movements with their non-stimulated wrist.

5.4.2 Motor Learning

Both the concussion history and control groups performed the motor learning task. Participants were instructed to actively match the movement of their nondominant wrist (passively moved by the experimenter) with their dominant wrist while their eyes remain closed. A target waveform generated in LabView (National Instruments, Austin, Texas, USA) was used to guide the experimenter through the series of wrist flexion/extension movements of the participant's nondominant wrist. The target waveform consisted of a 15s random and 15s repeated segment (Figure 3). Each series of wrist movements lasted 30 seconds (1 trial) and was repeated 5 times within each block. Participants were given a 10-second rest period between each trial. There was no nerve stimulation during this task. A total of 10 blocks were performed at experimental session 1 and 2 blocks of the same task were performed during the retention test at experimental session 2. EEG was recorded throughout the task at experimental session 1 but not at session 2.

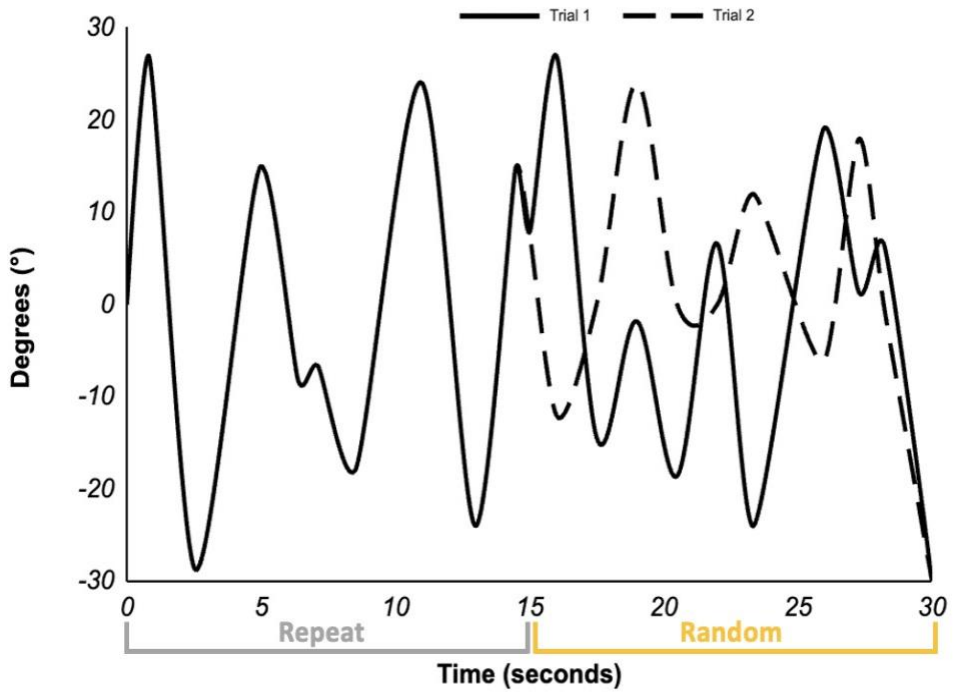

Figure 3. Overview of the motor learning task. A 30s target waveform consisting of a 15s random and 15s repeated segment was used to guide the experimenter through a series of wrist flexion/extension movements of the participant's nondominant wrist. Participants were asked to actively match the movement of their nondominant wrist with their dominant wrist while their eyes remain closed.

5.4.3 Attentional Blink

The protocol for the AB task was identical to that used by Arasanz and colleagues (2012). Participants were seated comfortably at a viewing distance of 35 cm from a computer screen. Stimuli were presented in black on a grey background as uppercase letters (9.1 cd/m^2) spanning a visual angle of 16.3□ by 12.5□ using EPrime software (Psychology Software Tools Inc, USA). Letters were presented in rapid serial visual presentation (RSVP; 120 ms/letter) for 120 ms per letter with no blank inter-stimulus interval (ISI). Each trial began with a small white dot at center fixation lasting 180 ms. Within each trial, two target letters were embedded amongst the string of distractor letters. The T1 was either a white H or S and the T2 was either a black X or Y. There were no repeated letters within the string and distractors were any letter of the alphabet excluding

T1 and T2 letters. T1 occurred 7-15 letters after the central fixation cue. T2 occurrences varied between no (lag 1), one (lag 2), two (lag 3), three (lag 4), five (lag 6), or seven (lag 8) distractors after T1. Lags 1-4 were short lags occurring within 480 ms of T1, while lags 6 and 8 were long lags occurring at least 720 ms after T1. In approximately one-third of trials, a distractor letter replaced T1 to act as the control condition where no AB effect should be present.

Participants performed 5 blocks of 72 trials. Participants were instructed to attend to the center of the screen, where the fixation cue appeared to indicate the start of the trial. Participants had to identify whether the white target letter (T1) was an H or S or did not occur at all and whether the black target letter (T2) was an X or Y. Manual responses were prompted by separate screens of instructions following the RSVP of letters. For T1, participants were instructed to press "H", "S", or "N" on the keyboard to indicate whether they saw H, S, or neither letter, respectively (Figure 4). For T2, participants were instructed to press "1" if they saw X and "2" if they saw Y. Participants were told to place importance on accuracy and to guess on trials where they are unsure.

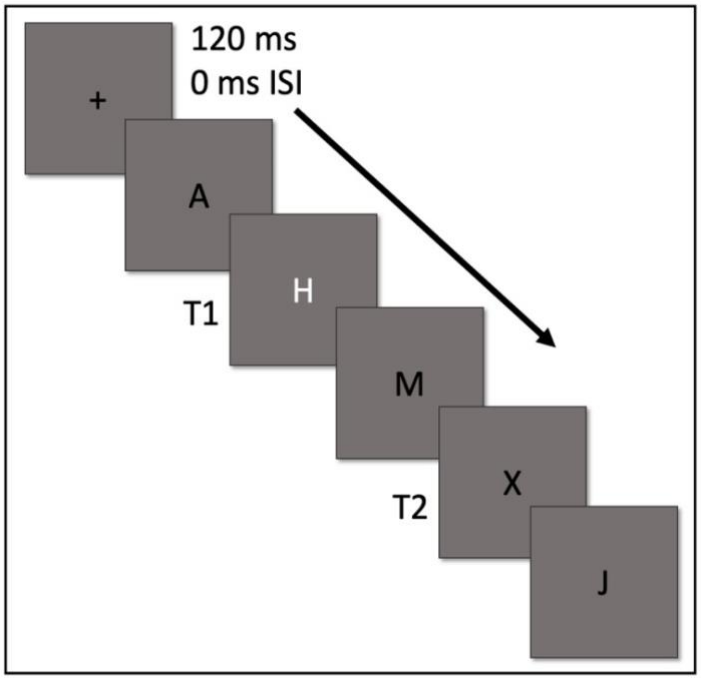

Figure 4. Example of stimuli used in attentional blink task.

5.5 Data Analysis

5.5.1 Sensory Gating

EEG was recorded from a 32-channel cap (Quik-Cap, Compumedics Neuroscan, NC, USA) using the International 10-20 system. The main electrode of interest was Cp4 (righthanded individuals) and Cp3 (left-handed individuals), which rests on the scalp directly above the somatosensory cortex contralateral to the nerve stimulation. Electrodes were re-referenced to the average of both mastoids, and all channel impedances were maintained at less than 5 kΩ. Data was digitized at 1000 Hz and band-pass filtered at 0.1-200 Hz (SynAmps2 Compumedics Neuroscan, NC, USA).

The motor task program run in LabView (National Instruments, Austin, Texas, USA) sent event codes to the running EEG file denoting the delivery of peripheral nerve stimulation to the median nerve. EEG data was analyzed using Neuroscan (Compumedics Neuroscan, NC, USA) software. The primary electrode of interest was Cp4 for right-handed individuals and Cp3 for left-handed individuals, as this electrode is situated contralateral to the site of median nerve stimulation. Continuous data files were epoched from -100 to 400 ms for each median nerve stimulation and baseline corrected to the 100 ms pre-stimulation interval. All epochs were manually inspected for noise and artifacts which can be produced by biological and/or nonbiological sources of noise. For each potential of interest, epochs were averaged within each condition for each participant to extract values in microvolts. Based on previous research (Brown et al., 2015; Tennant et al., 2021), the latency ranges for each of the potentials of interest were as follows:

- N20: Greatest negativity between 17-23 ms
- P27: Greatest positivity between 24-31ms
- P50: Greatest positivity between 40-60 ms

- N70: Greatest negativity between 60-80 ms
- P100: Greatest positivity between 80-120 ms

Given that the amplitude of a cortical potential may be influenced by the preceding potential, peak-to-peak amplitudes were used for analysis (N20-P27, P50-N70, & N70-P100). Peak-topeak amplitudes were determined by calculating the difference in amplitude between the two potentials and taking the absolute value of the difference.

5.5.2 Motor Learning

Participant behavioral data for the motor learning task was analyzed using a custom LabView (National Instruments, Austin, Texas, USA) program. The voltages measured from the potentiometers (recorded at a rate of 100 Hz) embedded in the handles of the wrist movement device were plotted over time to generate two waveforms: the target and the response. For each trial, the program generated a difference waveform by plotting the voltage difference between the target and the response handle. Next, the root mean square (RMS) of the difference waveform was calculated within the LabView program to provide a root mean square error (RMSE) value for the entire 30 s waveform, the 15 s repeat, and the 15 s random segments. The average of all 5 trials within each block was used for analysis. The difference in RMSE scores for the last 2 blocks of training on day 1 (average of 10 trials) and the retention test on day 2 (average of 10 trials) for the 15 s repeat segment was used as an index of retention to infer motor learning.

5.5.3 Attentional Blink

Participants' responses during the AB task were recorded using EPrime software (Psychology Software Tools Inc, USA). For T2 detection accuracy, only trials with a correct response for T1 were used for analysis. This is because if participants incorrectly identified T1,

we can't say for certain that T1 was observed by the participants and therefore whether they processed it prior to processing T2, so there would be no attentional blink. For each lag, T2 detection accuracy was calculated by summing the total number of correct T2 responses and dividing that by the total number of responses before multiplying by 100 to produce an accuracy score (T2 % accuracy).

5.6 Statistical Analysis

All statistical analyses were conducted using RStudio (version 2022.07.1.554; RStudio Team, 2022).

For the SEP data, QQ plots were inspected to ensure the data did not violate the assumption of normality. Four separate one-way repeated measures ANOVAs were conducted to determine the effect of condition (Rest, Passive, Active, Selective Relevancy-Based Enhancement) on SEP amplitude (dependent variable). For the Selective Relevancy-Based Enhancement condition, SEP amplitudes were extracted on a case by case basis based on the component of the SEP in which in participants showed the greatest amplitude difference between the Passive and Active conditions. For ANOVAs with a significant main effect of condition, pairwise t-tests with Bonferroni corrections for multiple comparisons were used to elucidate which conditions differed. In addition,, a one-way repeated measures ANOVA was used to confirm no significant differences in m-wave amplitude across conditions.

For the behavioural data analysis, retention was used to infer motor learning ability. Individual participants' retention was calculated by taking the difference between the last two blocks of training at Experimental session 1 and the two blocks performed at Experimental session 2. A one-tailed independent samples t-test was performed for the 15 s random, 15 s repeat, and 30 s segment of the waveform to test our *a priori* hypothesis that the concussion group would perform worse than the control group.

For the attentional blink data, Lag 8 was dropped from the analysis because of a ceiling effect. QQ plots were inspected to ensure the data did not violate the assumption of normality. Levene's test revealed that the data did not violate the assumption of homogeneity of variances. Therefore, a two-way mixed measures ANOVA was conducted to assess the effect of Group (Concussion, Control) and Lag (Lag 1, Lag 2, Lag 3, Lag 4, Lag 6) on T2 Detection Accuracy. Group was treated as a between subject's factor and Lag was treated as a repeated measure. Pairwise t-tests were used to interpret significant main effects.

6.0 RESULTS

6.1 Delay in Relevancy-Based Gating Modulations in the Concussion Group

Table 2 displays mean SEP amplitudes and standard deviations separated by potential (N20-P27, P50-N70, N70-P100, Max Gating Diff (max difference between active and passive)) and condition (Rest, Passive, Active).

	N20-P27		P50-N70		N70-P100		Max Gating Diff	
Condition	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Rest	3.525	1.766	5.922	2.938	7.536	3.184	6.04	3.12
Passive	1.969	1.322	3.86	2.527	4.701	3.46	3.56	2.66
Active	1.741	1.176	4.506	2.681	4.761	3.341	4.92	3.11

Table 2. Mean SEP amplitudes for each potential and condition. SD = standard deviation.

N20-P27

A one-way repeated measures ANOVA was conducted to determine the effect of condition (Rest, Passive, Active) on the N20-P27 SEP amplitude. The one-way repeated measures ANOVA revealed a significant effect of condition [$F(2, 48) = 21.379$, $E = 0.96$, $p.adj < 0.001$, $\eta^2=0.239$] (Figure 5A). Pairwise t-tests revealed that N20-P7 SEP amplitude was significantly greater during the rest condition compared to the active and passive conditions [$p.adj < 0.001$ and $p.adj < 0.001$, respectively]. However, SEP amplitude was not different between the active and passive conditions [$p.adj = 1$]. Therefore, SEP amplitude was greatest at rest but did not differ between passive or active conditions.

P50-N70

A one-way repeated measures ANOVA was conducted to determine the effect of condition (Rest, Passive, Active) on the P50-N70 SEP amplitude. The one-way repeated measures ANOVA revealed a significant effect of condition [$F(2, 48) = 11.349$, $E = 0.99$, $p.adj < 0.001$, $\eta^2=0.095$] (Figure 5B). Pairwise t-tests revealed that SEP amplitude was significantly greater during the rest condition compared to the active and passive conditions [$p.adj = 0.012$ and $p.adj < 0.001$, respectively]. However, SEP amplitude was not different between the active and passive conditions [$p.adj = 0.45$]. Therefore, SEP amplitude was greatest at rest but did not differ between passive or active conditions.

N70-P100

A one-way repeated measures ANOVA was conducted to determine the effect of condition (Rest, Passive, Active) on the N70-P100 SEP amplitude. The one-way repeated measures ANOVA revealed a significant effect of condition [$F(2, 48) = 20.069$, $E = 0.94$, $p.adj < 0.001$, $\eta^2=0.141$] (Figure 5C). Pairwise t-tests revealed that SEP amplitude was significantly greater during the rest condition compared to the active and passive conditions [$p.adj < 0.001$ and $p.adj < 0.001$, respectively]. However, SEP amplitude was not different between the active and passive conditions [$p.adj = 1$]. Therefore, SEP amplitude was greatest at rest but did not differ between passive or active conditions.

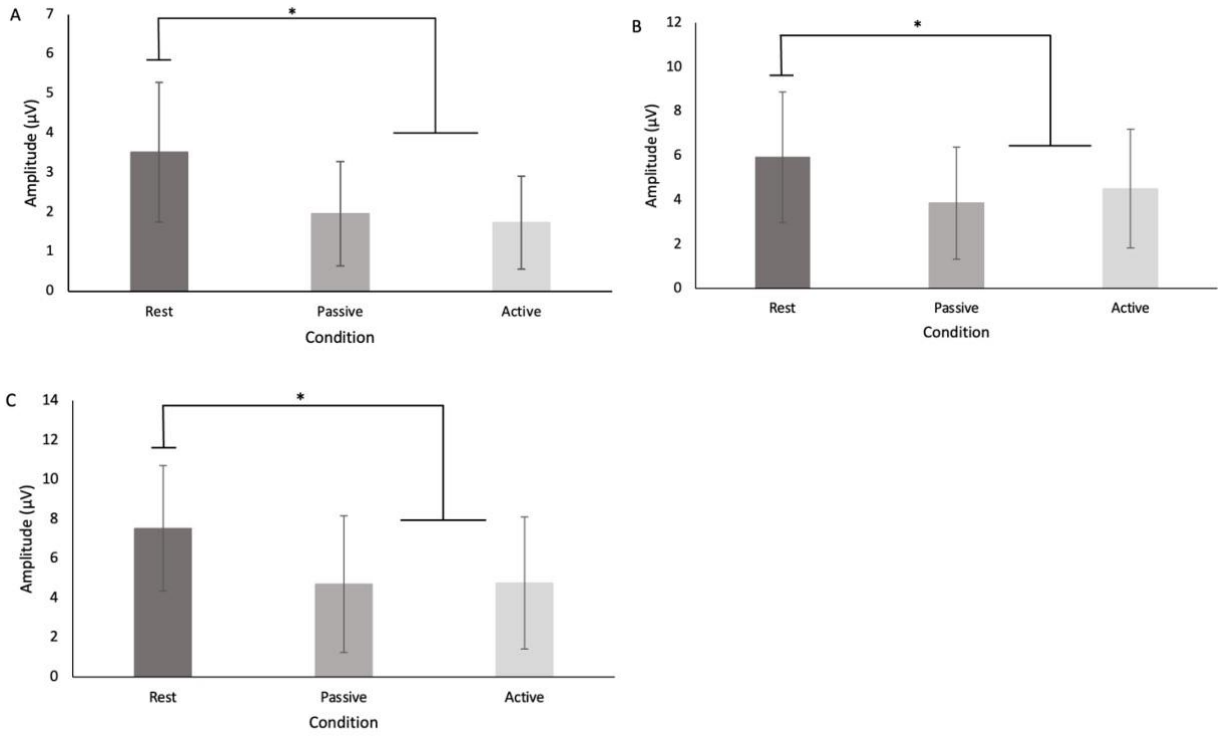

Figure 5. Mean SEP Amplitude by Condition. (A) N20-P27, (B) P50-N70, (C) N70-P100. Error bars denote standard deviation.

Selective Relevancy-Based Enhancement

Upon closer inspection of individual subjects' P50-N70 and N70-P100 SEP amplitudes, we observed that subjects showed evidence of relevancy-based enhancement at either the P50N70 (n=18) or the N70-P100 (n=7). SEP amplitudes for all three conditions (Rest, Passive, Active) in which subjects showed evidence of relevancy-based facilitation were grouped for further analysis. Figures 7A and 7B show the grand average EEG trace for individuals who showed evidence of relevancy-based facilitation at the P50-N70 and the N70-P100, respectively.

A one-way repeated measures ANOVA was conducted to determine the effect of condition (Rest, Passive, Active) on the collapsed P50-N70 & N70-P100 SEP amplitudes. The one-way repeated measures ANOVA revealed a significant effect of condition [$F(2, 48) =$

15.134, E = 0.96, p.adj < 0.001, $\eta^2 = 0.107$] (Figure 6). Pairwise t-tests revealed that SEP amplitude was significantly greater during the rest and active condition compared to the passive condition [p.adj < 0.001 and p.adj = 0.008, respectively]. However, SEP amplitude was not different between the active and rest conditions [p.adj = 0.065]. This finding is in line with past work that observed a delay in movement-related gating and relevancy-based facilitation in the concussion population (Tennant et al., 2021).

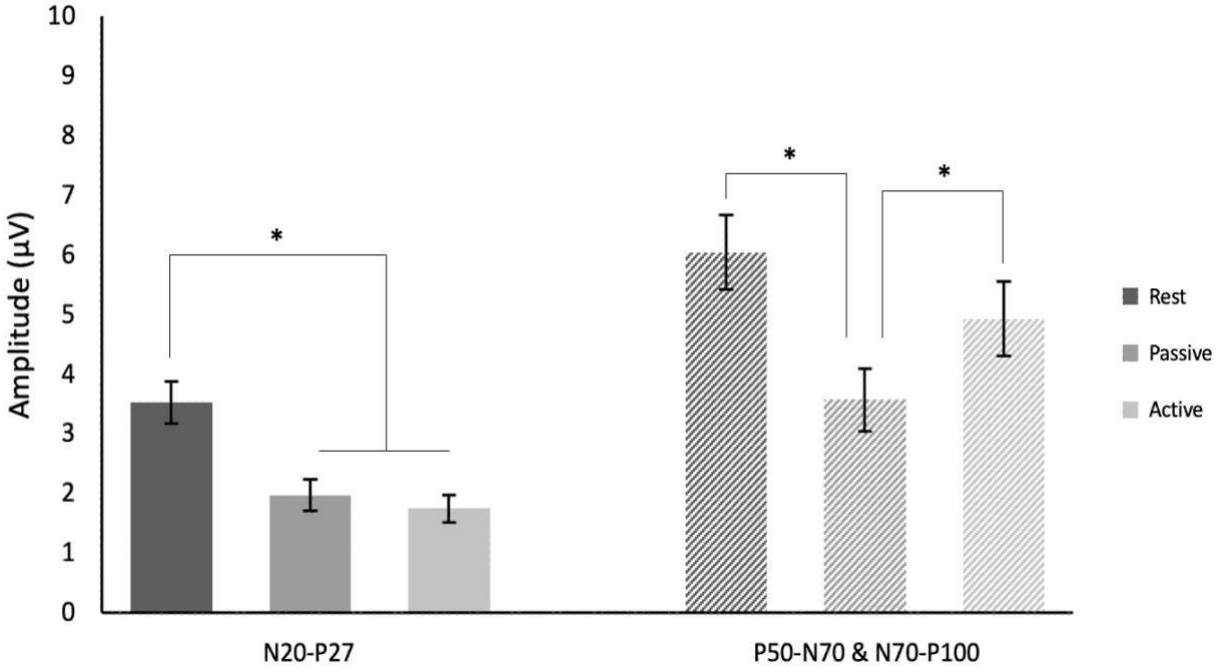

Figure 6. Mean SEP Amplitude N20-P27 and P50-N70 & N70-P100. Mean SEP amplitudes (μV) for each condition separated by SEP latency (N20-P27 – solid bars; P50-N70 & N70-P100 – hatched bars) recorded from electrode CP4 (n=22) and CP3 (n=3). Error bars represent ± SEM. * denotes p < 0.05.

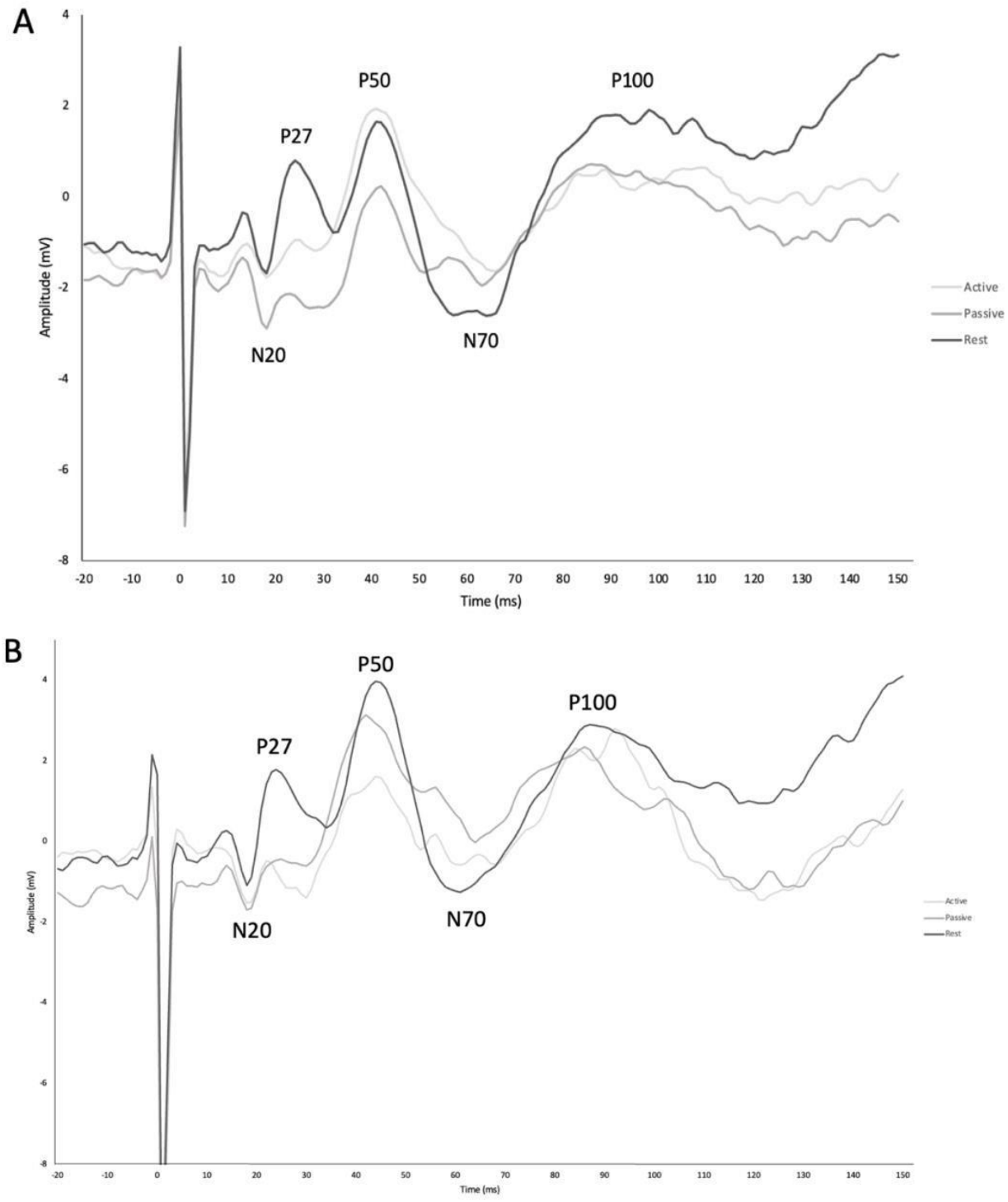

Figure 7. Grand Average Waveforms. The grand average EEG trace during Rest, Passive and Active conditions for the individuals who showed relevancy-based enhancement at (A) P50-N70 (n=18) and (B) N70-P100 (n=7). Potentials of interest are shown, recorded from electrode CP4 or CP3 for right- or left-handed individuals, respectively.

6.2 Effect of Concussion on Implicit Motor Sequence Learning

A one-tailed independent samples t-test was conducted to determine whether the concussion history group had worse retention than the control group on the 30 s segment, 15 s repeated segment, and 15 s random segment of the motor learning task (as indexed by a lower difference in RMSE between the average of the last 2 blocks at Experimental session 1 and the 2 retention blocks from Experimental session 2). Due to a recording malfunction, the data from subject 7 was excluded from this analysis. The t-test revealed no significant difference in retention for the 30 s segment [t=1.38, df=37, p=0.911, Concussion=0.031$\pm$0.045, Control= 0.014$\pm$0.025], no significant difference in retention for the 15 s repeated segment [t=1.35, df=37, p=0.908, Concussion=0.026$\pm$0.046, Control=0.008$\pm$0.027], and no significant difference in retention for the 15 s random segment [t=1.33, df=37, p=0.904, Concussion=0.036$\pm$0.045, Control=0.019$\pm$0.026]. Therefore, both groups performed similarly on the implicit motor sequence learning task (Figure 8a).

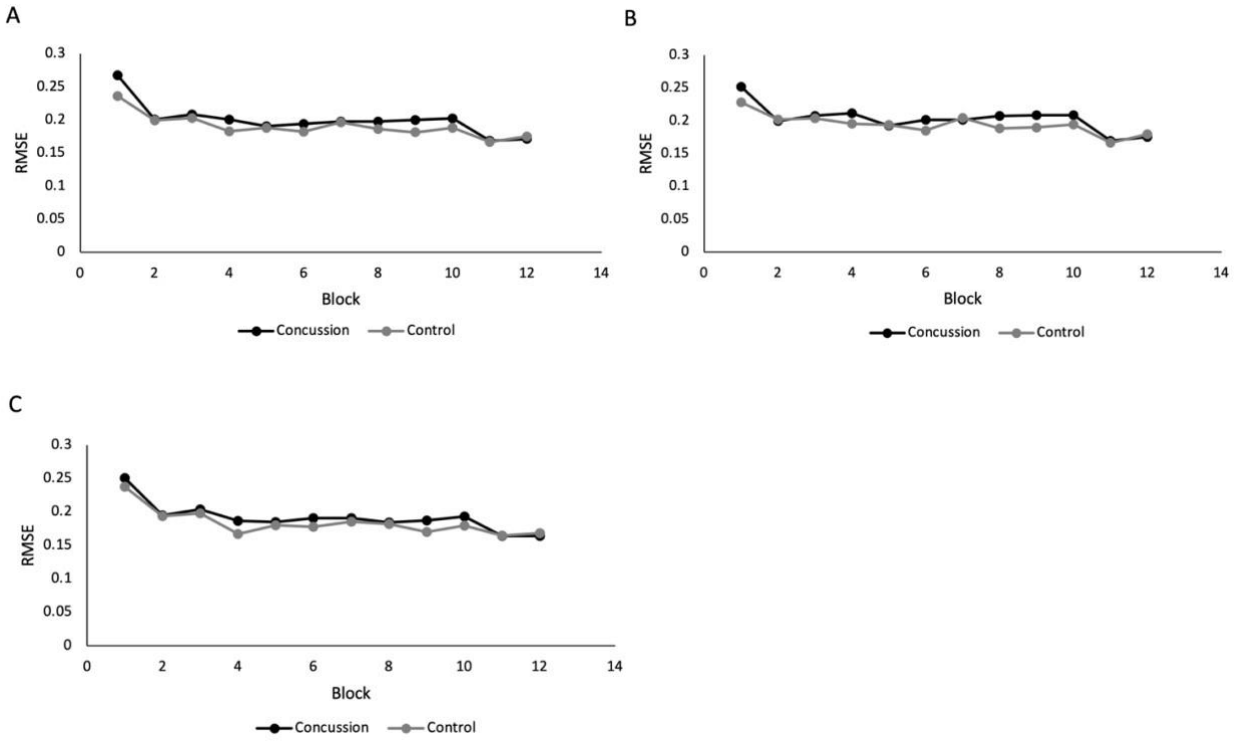

Figure 8. Mean RMSE Values. (A) 30s segment, (B) Random Segment, (C) Repeated Segment.

6.3 Effect of Concussion on Attentional Blink Task

Table 3 displays mean T2 Detection Accuracy as a percentage. A two-way mixed measures ANOVA was conducted to assess the effect of Group (Concussion, Control) and Lag (Lag 1, Lag 2, Lag 3, Lag 4, Lag 6) on T2 Detection Accuracy. Group was treated as a between subject's factor. Lag was treated as a repeated measure. The ANOVA revealed a main effect of Group [$F(1, 68) = 9.761$, $p = 3.00e\text{-}03$, $\eta^2 = 0.082$] and a main effect of Lag [$F(4, 272) = 47.452$, $p = 3.09e\text{-}30$, $\eta^2 = 0.209$] (Figure 9). The interaction was not significant [$F(4, 272) = 0.065$, $p = 9.92e\text{-}01$, $\eta^2 = 0.00036$]. The main effect of Group was broken down with pairwise t-tests which revealed that T2 Detection Accuracy was significantly greater in the control group compared to the concussion history group [$p < 0.001$, Control = 0.861 ± 0.008, Concussion = 0.796 ± 0.01, mean $\pm$ standard error]. The main effect of Lag was broken down with pairwise t-tests which revealed that T2 Detection Accuracy decreased from Lag 1 to Lag 2, followed by successive increases from Lag 2 onward (all p-values < 0.001). Therefore, the magnitude of the AB was greater in the concussion history group compared to the control group.

	Concussion		Control	
Lag	Mean	SD	Mean	SD
1	78.0	11.1	84.1	14.2
2	70.7	12.8	77.7	12.7
3	78.2	9.2	85.1	9.9
4	83.7	8.9	89.9	8.7
6	87.5	8.3	93.6	5.8
8	97.3	3.0	98.0	3.3

Table 3. Mean T2 % Accuracy. Numbers reflect the percentage.

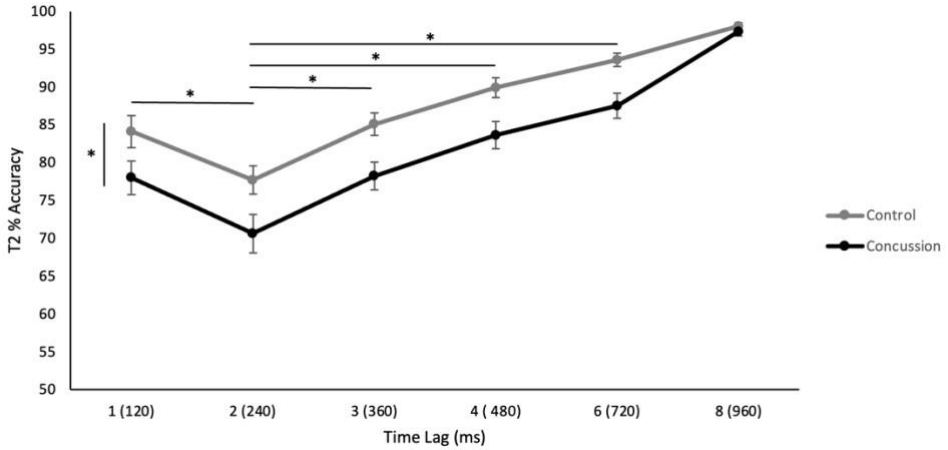

Figure 9. Performance in detecting T2 for the Control and Concussion History Group. Performance (Mean % accuracy), error bars denote SEM.

7.0 DISCUSSION

7.1 Summary

The current study aimed to confirm disruptions to relevancy-based gating modulations at early cortical processing stages in the concussion population and determine whether this has an impact on motor learning. As well as to understand how the attentional system behaves when taxed with heightened attentional demands in the temporal domain using the AB task. SEPs were elicited via median nerve stimulation during a sensory gating task that manipulated the relevance of proprioceptive stimuli induced by the experimenter. Consistent with our hypothesis and past work by Tennant et al. (2021), the concussion history group demonstrated a delay in relevancybased facilitation to task-relevant stimuli that typically occurs at the N20-P27 component of the SEP in healthy participants. An implicit motor sequence learning task that required reliance on proprioceptive cues delivered by the experimenter was administered to determine participant's motor learning ability – a potential behavioral manifestation of hypothesized impaired sensory gaiting ability. Contrary to our hypothesis, the concussion history and control group performed similarly on this task. Indicating that despite aberrant sensory gating abilities, the concussion history group can compensate for sensory processing alterations and obtain the same level of motor learning as their healthy counterparts. Finally, the AB task was administered which consisted of manual responses by participants to indicate which letter they saw for T1 and T2. Consistent with our hypothesis, the concussion history group had lower accuracy in detecting T2 compared to control data. This suggests that concussive brain injuries impact attentional processing or that these individuals have an impaired ability to allocate attentional resources.

7.2 Delay in Relevancy-Based Enhancement in the Concussion Population

The results from the sensory gating task indicate that individuals with a history of concussion have a delay in the relevancy-based enhancement of relevant proprioceptive stimuli. For context, the task used in the present study was identical to that used by Tennant et al. (2021) and Brown et al. (2015). Tennant and colleagues (2021) observed a delay in relevancy-based enhancement in their concussion group compared to their healthy control group. The present study replicates these findings, although we did not have a control group because we used the results from Tennant et. al (2021) as a reference. In their study, researchers observed relevancybased enhancement (as indexed by increased SEP amplitude) at the N20-P27 component of the SEP during the active condition in which participants had to replicate the passive movement of their non-dominant wrist induced by the experimenter with their dominant hand compared to the passive condition where the passive movement was not replicated. This relevancy-based enhancement was not seen by Tennant et al. (2021) at the N20-P27 component for the concussion history group, consistent with the findings of the present study. The authors found evidence of relevancy-based enhancement at the N70-P100 component of the SEP. This is consistent with our results showing evidence of relevancy-based enhancement at the P50-N70 component of the SEP for 18 participants while the other 7 participants in our concussion history group showed this at the N70-P100 component of the SEP. Overall, the concussion history group in the present study demonstrated the same modulation pattern at later components of the SEP (P50-N70 and N70-P100) as the healthy control participants showed at the earlier N20-P27 component of the SEP in the study from Tennant et al. (2021).

These results suggest a delay, as opposed to an absence, in the ability to effectively process somatosensory information post-concussion. Since the present concussion group demonstrated effective movement-related gating, evidenced by decreased SEP amplitudes for the

passive and active movement conditions compared to rest, the ability to facilitate relevant somatosensory information is likely altered. This could be due to disruptions in the mechanisms involved in relevancy-based facilitation. The N20 and P27 components of the SEP are generated in SI and represent the arrival of somatosensory information to BAs 3b and 1 (Yamaguchi & Knight, 1990). While the N70 and P100 components of the SEP are generated in SI and SII, respectively, and represent later sensory processing (Hamalainen et al., 1990). Past work by Brown and colleagues (2015) revealed an important role of the DLPFC in the relevancy-based facilitation of task-relevant sensory information. Given the notion that frontal brain areas are particularly vulnerable to concussive forces (Eierud et al., 2014), our results, combined with evidence from past work (Brown et al., 2015; Tennant et al., 2021) may suggest that concussive injuries alter normal DLPFC function during these types of tasks. This may explain why evidence of relevancy-based facilitation was delayed, but not absent, and seen at either the P50N70 or the N70-P100 but not the N20-P27 component of the SEP. The observed delay in relevancy-based facilitation that occurs at later components of the SEP is likely to be mediated by the DLPFC (Bolton & Staines, 2011; Adams et al., 2019). The application of cTBS over DLPFC in healthy participants abolished relevancy-based differences in the tactile-evoked N70 component of the SEP (Adams et al., 2019). In another study, cTBS over DLPFC resulted in attenuation of relevancy-based modulation of the P100 post-cTBS compared to pre-cTBS (Bolton & Staines, 2011). Results from the present study suggest that concussions impact the DLPFC's ability to modulate somatosensory information based on task relevancy at the earliest cortical processing stages (as indexed by the N20-P27 component of the SEP). Evidence of relevancy-based enhancement at later components of the SEP (P50-N70 and N70-P100) suggest the use of a compensatory strategy occurring at later processing stages in SI and SII.

7.3 Preserved Motor Learning Ability in the Concussion Population

Despite an impaired ability to facilitate relevant sensory information, the concussion history group demonstrated similar performance on the motor learning task as the control group. RMSE values across all blocks were not significantly different between the concussion history and control group. This was contrary to our hypothesis that the concussed group would display poorer performance on this task given that (1) efficient somatosensory processing is integral to efficient motor control and motor learning (Vidoni et al. 2010), and (2) past work showing deficits in motor learning abilities in the concussed population (De Beaumont et al., 2012; Beaulieu et al., 2019; Bourassa et al., 2021; Cantarero et al., 2021).

The importance of somatosensory feedback to motor learning cannot be overlooked, therefore a compensatory mechanism is likely at play which may explain why the concussion history group performed similarly to the control group in the present study. For example, a longitudinal functional magnetic resonance imaging (fMRI) study in 15 varsity athletes observed increased activation in bilateral DLPFC during the performance of the n-back task (for n = 1, 2, and 3) at 72 hours, 2 weeks, and 2 months post-injury compared to healthy controls (Dettwiler et al., 2014). They also observed increased activation of the inferior parietal lobe 72 hours and at 2 weeks post-injury, but not at the 2-month time point compared to healthy controls. Performance on the n-back task was not significantly different between the concussion and healthy control group. Perhaps the recruitment of additional brain resources enabled the concussion group in the present study to attain the same level of performance as healthy controls.

An alternative explanation for the absence of hypothesized performance deficits in the concussion history group compared to the control group could be due to the nature of the task used in the present study compared to other studies that observed motor learning deficits in the concussion population (De Beaumont et al., 2012; Beaulieu et al., 2019; Bourassa et al., 2021;

Cantarero et al., 2021). It is possible that measures such as the speed-accuracy trade-off and reaction time, which both encompass the speed of motor output, as well as occlusion, are more sensitive to detecting subtle deficits in motor learning ability that we simply were not able to quantify with the task used in the present study. The concussion population in the aforementioned studies (Section 2.2.3 Motor Learning & Concussion), as well as the present study, demonstrate an ability to compensate for subtle deficits in motor learning ability that enables them to achieve similar performance as healthy controls.

7.4 Concussion Impacts Performance on the Attentional Blink Task

The results from the AB task revealed that individuals with a history of concussion display poorer performance on the task compared to healthy controls. This was true for all Lags (Lag 1, Lag 2, Lag 3, Lag 4, Lag 6) except for Lag 8 (960ms between T1 and T2), where the concussion group reached similar T2% accuracy as controls. These results support our hypothesis that the control group would have greater performance than the concussion group on the AB task.

The control group used for the analysis of the AB data in the present study was from the pre-cTBS group from data previously collected by Arasanz and colleagues (2012). Their study consisted of 3 groups: a control group that received sham cTBS over the cerebellum, a group that received cTBS over the left cerebellum, and a group that received cTBS over the right cerebellum. Participants performed the AB task twice: pre-cTBS and post-cTBS. The authors observed significantly worse performance on the AB task for the right cerebellar cTBS group, similar to the concussion group in the present study, compared to the left and sham groups. Since T2% accuracy was reduced even at Lag 1, the authors suggest that the right cerebellum, which is involved in both the cognitive and motor domains, may be responsible for modulating attentional resources allocated to T1 to readily detect T2. Given the similarity in performance between the

right cerebellar cTBS group from Arasanz and colleagues (2012) and the concussion group from the present study, concussions may impact right cerebellar function.

Results from the present study lend support to the two main theories thought to underpin performance decrements during the AB: Raymond et. al's (1992) Attentional Gating Theory and Chun and Potter's (1995) Two-Stage Model Theory (Zivony & Lamy, 2021). Results from the sensory gating task suggest that concussions impact the DLPFC's ability to modulate somatosensory information based on task relevance. It is well known that the DLPFC also plays a role in aspects of attention and working memory – which have both been shown to be impacted post-concussion (Giza & Hovda, 2014). According to Raymond et. al's (1992) Attentional Gating Theory, the attentional processes needed for high-level perceptual processing are disrupted during the blink and T2 is therefore not identified as a target (Zivony & Lamy, 2021). Since T2% accuracy was reduced even at Lag 1, when no distractors were present between T1 and T2, this may suggest a disruption in the allocation of attentional resources subserved by the DLPFC. According to Chun and Potter's (1995) Two-Stage Model Theory, the AB reflects a structural limitation in WM encoding that occurs after T2 has been attended to and identified as a target (Zivony & Lamy, 2021). Recall that at Lag 8, when there was 960 ms between T1 and T2, the concussion history group achieved similar T2% accuracy as controls. This may suggest that enough time had elapsed following the processing of T1, resulting in adequate neural resources available for encoding T2 into WM. Our results can be explained by both the aforementioned theories thought to underpin the AB. This may suggest that concussive brain injuries further exacerbate the disruptions to attentional engagement and WM encoding that can be observed in neurologically healthy populations during the AB.

7.5 Limitations

The following limitations should be considered when interpreting the results of this study. Firstly, it is possible that wrist movement during the passive and active conditions of the sensory gating task resulted in positional changes of the electrode used to stimulate the participant's median nerve. Positional changes of the electrode could lead to different areas of the nerve being stimulated, meaning a different number of stimulated fibers of that nerve. This could affect the amplitude of SEPs recorded at the cortical level, meaning that SEP amplitude changes between conditions could not be solely attributed to task manipulations. However, the experimental set-up used in the present study was identical to that used by Brown et al. (2015) who monitored M-wave activity throughout the task and observed no significant differences in M-wave amplitude between conditions, which was also seen in our study. Our offline analysis of M-wave amplitude revealed no significant differences between conditions. Because the diameter of the efferent fibers is the same as the afferent fibers, analysis of M-wave amplitudes ensures similar stimulus intensities since large diameter fibers are recruited first – indicating a constant number of stimulated fibers between conditions.

Secondly, the concussion history group performed the sensory gating task prior to completing the motor learning task at experimental session 1, whereas the control group only performed the motor learning task. Given the similarities between the tasks, it is possible that the concussion history group gained learning experience prior to completing the learning task, which may explain the lack of differences between the concussion history and control group seen in our results. However, past work by Tennant and colleagues (2021) observed no evidence of learning during the sensory gating task between the concussion and control groups. Therefore, it is unlikely that the present concussion history group gained learning experience prior to completing the motor learning task in the present study. Although, a control group for the sensory gating task

would provide stronger evidence as to whether learning did or did not occur in the present sample.

7.6 Future Directions

Results from this thesis suggest that individuals with a history of concussion have longterm alterations in their ability to effectively process somatosensory stimuli and allocate attentional resources. Some individuals showed relevancy-based facilitation at the P50-N70 component while others show this at the N70-P100 component of the SEP. The N70 component of the SEP is thought to be generated in SI, while the P100 component is thought to be generated in SII (Hamalainen et al., 1990). It would be interesting to understand what is driving these group differences in relevancy-based facilitation. Future research with a larger sample size could attempt to provide insight such as whether this relates to the nature of the concussive blow, time since injury, number of concussions, or physical activity level, for example.

The results from the present study showed no difference in performance on the motor learning task. The purpose of this task was to identify whether there was a behavioral cost to the observed delay in relevancy-based facilitation. While there appears to be no cost to performance on the task used in the present study, it is possible that the task was not challenging enough, and the concussion group was able to compensate. Perhaps a more challenging task that taxes the somatosensory system may help elucidate the true cost of these gating disturbances. However, this task does lack external validity. Future research using a more ecologically valid learning paradigm would provide insight as to whether gating disturbances affect performance in the “real world”.

Finally, results from the AB task revealed lower T2% detection accuracy in the concussion history group compared to the control group. Future studies using EEG during the AB task would

be useful in understanding the specific neural processes that are contributing to performance decrements.

7.7 Conclusion

This thesis aimed to replicate disruptions to relevancy-based gating modulations at early cortical processing stages in the concussion population and identify the behavioral manifestations that result from those. An implicit motor sequence learning task that required reliance on proprioceptive cues was used to investigate motor learning ability. In addition, the AB task was used to provide insight into how the attentional system behaves when taxed with heightened attentional demands in the temporal domain. The sensory gating task used by Brown et al. (2015) and Tennant et al. (2021) was used in the present study to investigate participants' sensory gating abilities.

The results suggest that concussive brain injuries impact the processing of relevant somatosensory feedback, but this does not affect motor learning. The N20-P27 peak-to-peak component of the SEP generated in S1 revealed an absence in the facilitation of relevant sensory information that normally occurs here. However, evidence of compensatory mechanisms is suggested by the facilitation of relevant sensory information seen at either the P50-N70 or the N70-P100 peak-to-peak components of the SEP. This observed delay in relevancy-based facilitation of somatosensory information may suggest a compensatory mechanism or adaptation that enabled the concussion history group to obtain similar performance to the control group on the motor learning task.

Results from the AB task suggest that concussions result in increased AB magnitude, as demonstrated by poorer performance observed in the concussion history groups compared to the control group across all Lags aside from Lag 8. The concussion history group achieved similar

performance to the control group for Lag 8, which was a longer lag with greater time between T1 and T2. Thus, it appears that taxing the attentional system in the temporal domain results in performance deficits in the concussion population.

A greater understanding of the mechanisms involved in sensory gating alterations and attentional processing in the concussion population are important avenues for future research. Further insight into these processes may help to improve rehabilitative techniques and reduce the likelihood of sustaining subsequent concussions and musculoskeletal injuries.

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Appendix A: Edinburgh Handedness Questionnaire

Edinburgh Handedness Inventory[1]

Participant ID: _____

Please indicate with a check (✓) your preference in using your left or right hand in the following tasks.

Where the preference is so strong you would never use the other hand, unless absolutely forced to, put two checks (✓✓).

If you are indifferent, put one check in each column (✓ | ✓).

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

Task / Object	Left Hand	Right Hand
1. Writing		
2. Drawing		
3. Throwing		
4. Scissors		
5. Toothbrush		
6. Knife (without fork)		
7. Spoon		
8. Broom (upper hand)		
9. Striking a Match (match)		
10. Opening a Box (lid)		
Total checks:	LH =	RH =
Cumulative Total	CT = LH + RH =	
Difference	D = RH – LH =	
Result	R = (D / CT) × 100 =	
Interpretation: (Left Handed: R < -40) (Ambidextrous: -40 ≤ R ≤ +40) (Right Handed: R > +40)		

[1] Oldfield, R. C. (1971). The assessment and analysis of handedness: The Edinburgh inventory. *Neuropsycholologia*, *9*, 97-113.

Appendix B: Modified University of Waterloo Health History Questionnaire

Participant ID_____M/F
Age_____

1. Aside from concussion, have you been diagnosed with any other neurological conditions (ADHD, depression, anxiety, etc.)?
a) _____yes _____no
b) If yes, please list:
_____

2. Have you sustained nerve damage to either of your upper limbs:
a) In the past 6 months?
_____yes _____no
b) That resulted in permanent damage?
_____yes _____no

3. At what age did you begin playing organized sport? _____

4. How many years have you played your sport? _____

5. Do you wear a mouth guard while playing?
_____yes _____no

If yes, what kind?
_____stock _____boil & bite
_____custom, front teeth _____custom, all

6. Have you suffered from neck pain within the past 6 months? _____yes _____no

7. Have you suffered a concussion?
_____yes _____no _____not sure

8. If yes to #7,
a) How many times total? _____
b) How many were clinically diagnosed?
_____
c) How many while playing sport in the past 6 months? _____
d) Date of last concussion? _____
e) Have you been clinically cleared to return to full physical and cognitive activity since the most recent concussion? _____yes _____no

Date_____
Sport/Position_____

f) How long did symptoms last (for most recent concussion)?
_____ 1-3 days _____ 4-7days
_____ 8-10 days _____ 11-14 days
_____ more than 14 days
g) After the most recent concussion, how long did you refrain from physical activity?
_____ 1-3 days _____ 4-7 days
_____ 8-10 days _____ 11-14 days
_____ more than 14 days

9. Have you ever been knocked unconscious?
_____yes _____no

10. If yes to #9,
a) How many times in the past 6 months? _____
b) What is the longest duration you've been knocked unconscious? _____

11. In the past 6 months, after being hit in the head in, have you experienced any of the following symptoms?
_____ confusion _____getting 'dinged'
_____headaches _____balance problems
_____nausea _____getting 'bell rung'
_____dizziness _____ringing in ears
_____blurry vision _____poor memory
_____other: _____

12. In regards, to how you feel NOW, please rate the following:

	None			Mild			Severe
Headache	0	1	2	3	4	5	6
"Pressure in head"	0	1	2	3	4	5	6
Neck pain	0	1	2	3	4	5	6
Nausea/vomiting	0	1	2	3	4	5	6
Dizziness	0	1	2	3	4	5	6
Blurred vision	0	1	2	3	4	5	6
Balance problems	0	1	2	3	4	5	6
Sensitivity to light	0	1	2	3	4	5	6
Sensitivity to noise	0	1	2	3	4	5	6
Feeling slowed down	0	1	2	3	4	5	6
"Don't feel right"	0	1	2	3	4	5	6
Hard to concentrate	0	1	2	3	4	5	6
Feeling "in a fog"	0	1	2	3	4	5	6
Trouble remembering	0	1	2	3	4	5	6
Fatigue/low energy	0	1	2	3	4	5	6
Confusion	0	1	2	3	4	5	6
Drowsiness	0	1	2	3	4	5	6
Trouble falling asleep	0	1	2	3	4	5	6
More emotional	0	1	2	3	4	5	6
Irritability	0	1	2	3	4	5	6
Nervous/anxious	0	1	2	3	4	5	6

13. Do the above symptoms get worse with physical activity? _____yes _____no

14. Do the above symptoms get worse with mental activity? _____yes _____no